17th International Congress on Psychopharmacology & Child and Adolescent Psychopharmacology / Psychotherapy

Overcoming Treatment Resistance

Sueno Deluxe Belek, Antalya / Türkiye
April 15th-18th, 2026



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&

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SCIENTIFIC PROGRAM

15 April 2026, Wednesday

14:30-15:00

15:00-16:00

HALL 1

OPENING CEREMONY

Speeches of the Congress Presidents and the TAP President - Ayşe Sakallı Kani, Alperen Bıkmazer, Allan Young, Kemal Sayar

OPENING CONFERENCE
OPENING SPEECH
Session Chairs: Xinmin Li - Allan Young

Medical Potential of Cannabis & Pyschodelics: Policy, Challenges & Future Directions - Lin Lu

16:00-16:30 *COFFEE BREAK*

16:30-18:00

HALL 1

SYMPOSIA ORAL SESSION-1

IT TAKES A VILLAGE: INNOVATIVE DEVELOPMENTS IN EFFECTIVE INTERVENTIONS IN PTSD THROUGH MULTIDISCIPLINARY

RESEARCH IN THE CLINICAL CONTEXT

Session Chair: Andrew Greenshaw

New Pathways of Care for War-Related PTSD in Ukraine: Integrating Ketamine-Assisted Group Therapy and 3MDR – Oksana Gryschenko

Behavioral- and Psycho-Therapeutic Advances For PTSD, From Youth To Adults - Rakesh Jetly

Applications of Assistive AI Agents For Remote Access and Continuity Of Care - Venkat Bhat

HALL 2

PLENARY LECTURE-1

Session Chair: Kemal Sayar

Difficult to Treat Depression – Christopher Aiken

HALL 3

PLENARY LECTURE-9

Session Chairs: Feyza Arıcıoğlu - Mesut Çetin

Recent Advances in Neuropsychopharmacology - Allan Young

HALL 4

SYMPOSIA ORAL SESSION-2

TREATMENT-RESISTANT COMORBIDITIES IN SCHIZOPHRENIA AND THEIR CLINICAL MANAGEMENT

Session Chair: Alpay Ateş

Treatment-Resistant Depression in Schizophrenia: Differential Diagnosis, Clinical Course, and Evidence-Based Therapeutic Strategies - Özcan Uzun

Anxiety Disorders in Schizophrenia: Mechanisms of Resistance, Clinical Burden and Management. - Çağrı Öğüt

Obsessive-Compulsive Symptoms in Schizophrenia: Treatment Resistance, Antipsychotic Interactions and Management - İbrahim Gündoğmuş

Substance Use Disorders in Schizophrenia: Resistance Pathways, Relapse Risks and Management - Burak Okumuş

HALL 5

SYMPOSIA ORAL SESSION-3

CURRENT DEVELOPMENTS IN EARLY DEMENTIA DIAGNOSIS

Session Chair: Ece Yazla Asaf

Clinical Evaluation and Screening in the Early Diagnosis of Dementia - Buket Koparal

Neuropsychological Assessment in the Early Diagnosis and Differential Diagnosis of Dementia - Ece Yazla Asaf

Use of Anti-Amyloid Therapies in the Treatment of Mild Cognitive Impairment - Turceun İleri Akdoğan

What are Liquid Biomarkers in the Early Diagnosis of Dementia? Who Should We Look for and How? - Ali Dayi

HALL 6

SYMPOSIA ORAL SESSION-4

FAMILY ACCOMMODATION AS A TRANSDIAGNOSTIC MECHANISM IN CHILD AND ADOLESCENT PSYCHOPATHOLOGY

Session Chair: Alperen Bıkmazer

Interrupting the Trauma Loop: Familial Accommodation in PTSD - Fatma Zehra Bıçakcı

Adaptive Support or Reinforced Rigidity? Family Accommodation in ASD - Büşra Nur Dambasan Gündüz

Parental Support or Symptom Alignment? Accommodation Dynamics in ADHD - Sena Ciminli

Family-Driven Meal Modifications and Accommodation in Eating Disorders - Melike Ayşegül Kara Karaman

HALL 7

WORKSHOP-1

DEEP BRAIN STIMULATION IN PSYCHIATRY: REVISITING THE PAST, DEFINING THE FUTURE

Session Chair: Umut Mert Aksoy

From Psychosurgery to Network Psychiatry: Conceptual Shifts in Deep Brain Stimulation-Umut Mert Aksoy

Precision, Connectivity, and the Surgical Architecture of Psychiatric DBS - Tanju Uçar

Preparing the Mind for Modulation: Psychiatric Selection, Ethics, and Integration in DBS - Özge Doganavsargil Baysal

HALL 8

SYMPOSIA ORAL SESSION-5

SSRIS EFFECTIVENESS IN CHILD AND ADOLESCENT PSYCHIATRY: WHERE DO WE STAND?

Session Chair: Şahin Bodur

Is the Monoamine Hypothesis of Depression Still Valid? - Burçin Özlem Ateş

The Role of SSRIs in the Management of Anxiety Disorders: Where Do We Stand? - Duygu Bilgili Can

The Role of SSRIs in the Management of Obsessive-Compulsive Disorder: Where Do We Stand? - Hazal Selin Soyugür

The Role of SSRIs in the Management of Post-Traumatic Stress Disorder: Where Do We Stand? - Yasemin Güney

18:00-18:30 *COFFEE BREAK*

18:30-20:00

HALL 1

SYMPOSIA ORAL SESSION-6

PSYCHEDELICS AND PSYCHOSTIMULANTS: MOLECULAR MECHANISMS, THERAPEUTICS, AND SOCIETAL IMPACT IN A NEW ERA OF RESEARCH

Session Chairs: Lin Lu - Xinmin Li

Gut–Brain Axis Mechanisms Underlying MDMA-Induced Stress Resilience - Kenji Hashimoto

The Chemical Biology of Psychoactive Substances - Xiaohui Wang

Why Relapse Risk Grows: The Consolidation of Remote Addictive Memory - Xiaoxing Liu

At the Crossroads of Innovation and Challenge: The Science and Policy of Psychedelics - Meng Jin

HALL 2

DOUBLE CONFERENCE-1

Session Chairs: Kemal Sayar - Erdoğdu Akça

The Role of Clinical Judgment in Psychiatry - Giovanni Andrea Fava

A Novel Discovery Platform for Targeted Drug Repurposing: Application for Psychiatric Disorders - Michael Berk

HALL 4

SYMPOSIA ORAL SESSION-7

IN THE SHADOW OF ATTACHMENT: RETHINKING SEPARATION ANXIETY DISORDER ACROSS THE LIFELONG

Session Chair: Rümeysa Yeni Elbay

The Origins of Attachment: The Biopsychosocial Roots of Separation Anxiety in the Perinatal Period - Rümeysa Yeni Elbay

The Adult Face of Attachment: Lifelong Clinical and Relational Manifestations of Separation Anxiety - Nehir Mutlusoy Eraslan

The Intergenerational Transmission and Healing of Anxiety: Epigenetic Mechanisms, Therapeutic Approaches and Prevention - Hazal Demirer

HALL 5

ORAL PRESENTATIONS SESSION-1

Session Chair: Ömer Fatih Sönmez

The Role of Maintenance ECT in Treatment-Resistant Schizophrenia: A Case Series - Hazal Berfin Acim

Investigating Nomophobia in Adolescents: Examining its Relationship with Intolerance of

Uncertainty and Emotion Regulation Difficulties – Preliminary Findings - Şule Kaplan

Clinicians' Compliance with Turkish Psychiatric Association Guideline Recommendations in the Treatment of Bipolar Depression: a Retrospective Study From Turkey, Ankara - Oğuzhan Arat

Sociodemographic and Developmental Characteristics of Adults with Intellectual Disability with and without Autism, Followed in an Adult Neurodevelopmental Psychiatry Outpatient Clinic –

Berra Öztürk

The Effect of Antidepressant Treatment on Glycemic Control and Lifestyle Behaviors in Patients with Type 2 Diabetes Mellitus and Depression - Elif Akgöz Güzel

Evaluation of the Relationship Between Social Media Addiction and Emotion Regulation Difficulties,

Eating Behaviors and Orthorexia Nervosa in Adolescents - Merve Sağdur Şimşek

Supersensitivity Psychosis with Tardive Dyskinesia in an Adolescent with Early-Onset Schizophrenia: A Case Report – Ceren Sena Coşkuner Akyüz

Examining the mediating role of sleep quality in the relationship between impulsivity and emotion regulation among adolescents referred to child and adolescent psychiatry clinics –

Hande Günal Okumuş

HALL 6

SYMPOSIA ORAL SESSION-8

PSYCHOPHARMACOLOGY MANAGEMENT IN AUTISM SPECTRUM DISORDER

Session Chair: Şahin Bodur

Psychopharmacological Management of Autism Spectrum Disorder: General Principles –

Özgün Türe Tekin

Efficacy and Safety of Antiepileptic Drugs in the Management of Behavioral Issues in Autism Spectrum Disorders – Pınar Ayaroğlu

Safety and Efficacy of Atomoxetine in Children and Adolescents With Autism Spectrum Disorder –

Nevin Kılınç

Efficacy and Safety of Alpha-2 Agonists in Autism Spectrum Disorder - Süeda Aybike Sönmez

HALL 7

SYMPOSIA ORAL SESSION-9
PHARMACOLOGICAL TREATMENT RESISTANCE IN AUTISM SPECTRUM DISORDER: FROM NEUROBIOLOGY TO CLINICAL MANAGEMENT
Session Chairs: Özalp Ekinci - Serkan Güneş
Resistance Mechanisms in the Treatment of Irritability and Aggression in ASD - Emine Merve Kalınlı
The Effect of Glutamatergic and GABAergic System Imbalance on Treatment Response-Muhammed Albayati
Pharmacological Dose Optimization and Combination Strategies - Serkan Güneş
The Complementary Role of Behavioral and Special Education Interventions - Özalp Ekinci

HALL 8

MEET THE EXPERT-1
Session Chair: Ali Saffet Gönül
Beyond Childhood:"Bridging Neurodiversity and Psychopharmacology: Rethinking Treatment Resistance in Adult ADHD and Autism Spectrum Disorder" - Umut Mert Aksoy

16 April 2026 Thursday

08:00 – 09:00

HALL 3

COURSE-1
CLINICAL MASTERY IN PSYCHOPHARMACOLOGY: ANTIDEPRESSANT PRESCRIBING IN THE PRESENCE OF COMORBID PHYSICAL DISEASES
Ömer Yanartaş

HALL 4

SESSION OF STARS - 1
Session Chair: Burak Baykara
VR-Based Intervention for Problematic TikTok Use in University Students: Effects on Metacognition and Psychological Symptoms - Iman Ehnech Seran
Maintenance Treatment Profiles of Bipolar Disorder Patients Without Criminal Responsibility- Hatice Öztürk
Fear of Childbirth in Pregnancy: The Role of Prenatal Bonding, Attachment Style, and Trauma - Hazal Yavuzlar Civan
Reducing peer bullying in children who stutter through an ACT-informed group intervention: Preliminary findings – Merve Aydoğuş
Retrospective Evaluation of Sociodemographic and Clinical Characteristics of Patients with Catatonia Hospitalized in an Inpatient Psychiatric Unit - Gizem Anıl Yunusoğlu
Psychotropic Recommendations Following Child and Adolescent Psychiatry Consultation in a Pediatric Burn Unit: A Five-Year Retrospective Review - Ece Pabuscu
Association of Metabolic Control with Diabetes-Related Distress and Health-Related Quality of Life in Adolescents with Type 1 Diabetes Mellitus: A Cross-Sectional Study - Ece Pabuscu
Depression, Anxiety, Hopelessness, and Alexithymia in Parents of Children Undergoing Postnatal Meningomyelocele Surgery: A Multicenter Cross-Sectional Study - Mehmet Özdemir

HALL 5

SESSION OF STARS - 2
Session Chair: Tuncay Sandıkçı
Foundations and Physiology of Heart Rate Variability - Ümmügülsüm Gündoğdu
Predictors of Burnout in Parents of Children with Autism: The Importance of Physical Space - Şeyma Aker
Evaluation of insight in major depressive disorder: A comparativeestudy of insight scales-Zehra A. Güvenç
Storytelling in Child Psychiatry Education: "If Fluoxetine Could Talk" as an Innovative Teaching Method - Öznur Adıgüzel Akman
Glasgow Antipsychotic Side Effect Scale (GASS): Adaptation and Validity Reliability Study for Child and Adolescent Versions (Child Form for 6 -13 years and Adolescent Form for 13-17 years) - Zeynep Kemik
The Relationship Between Metacognition and Childhood Adverse Experiences in Patients with Bipolar Disorder and a History of Suicide Attempts - İnci Timur Pekgöz
Serum and Urinary Metabolomic Alterations in Bipolar Disorder:An Exploratory UHPLC–MS Study-Talha Ağaç

HALL 6

Session Chair: Selma Tural Hesapçıoğlu

Investigation of the Relationship Between Interoception and Eating Behaviors in Children and Adolescents With Attention Deficit Hyperactivity Disorder - Zeliha Çetin

The Relationship of Odor Perception And Facial Expression Recognition Skills With Aggression And Impulsivity In Patients With Schizophrenia - Ahmet Çöpür

WISC-IV Cognitive Profile, Subindices, and Index Discrepancy in Children Presenting With Attention and/or Learning Difficulties: Associations With Clinical Variables - Hande Çelik

Circadian Gene Interactions (CLOCK and PER3) Differentially Predict Lithium and Valproate Response in Bipolar I Disorder - Burcu Yatkın Güney

The Relationship Between Home Internet Use, Familial Sociodemographic Characteristics, and Clinical Severity (Clinical Global Impression) in Children and Adolescents with Attention-Deficit/Hyperactivity Disorder During Follow-up - Ahmet Güleç

Sociodemographic and Clinical Differences Between Children With Biotinidase Deficiency and Epilepsy: A Retrospective Comparative Study - Merve Okuyucu

HALL 7

SESSION OF STARS - 4

Session Chair: Mustafa Solmaz

Longitudinal Links Between Intolerance of Uncertainty, Changes in Emotion Regulation, and Non-Suicidal Self-Injury in Adolescents With a History of NSSI - Sena Ciminli

Substance P, neuropeptide Y, neuropeptide S and spexin in children with tic disorders: associations with sensory sensitivity and allergy - Meryem Macit Efe

The Investigation of Attention Deficit Hyperactivity Disorder Comorbidity and Risk Factors in Children Diagnosed with Congenital Heart Disease - Nurşen Bozkoyun

Sleep Problems and Behavioral Symptoms in Preschool Children with Autism Spectrum Disorder: A Cross-sectional Study - Tuğba Günay

Evaluation of the Post-Discharge Effectiveness of the SAMBA Program Implemented During Compulsory and Voluntary Hospitalization of Patients with Alcohol and Substance Use Disorders - Ahmet Arif Özyurt

Social-Cognitive Vulnerability in Neurofibromatosis Type 1: Theory of Mind, Autism-Like Traits, Executive Functions, and MRI-Based Brain Volumetric Correlates - Tuğba Acehan

Memory Functions in the Schizophrenia Spectrum: First-Episode Psychosis and Chronic Schizophrenia Compared with Healthy Controls - Fatma Naz Akarca

09:00 – 10:30

HALL 1

PLENARY LECTURE-4

Session Chair: Aynur Görmez

Stigma in mental health, focusing on the MENA region - Suhaila Ghuloum

HALL 2

DOUBLE CONFERENCE-2

Session Chair: Erol Göka

Phenomenology of Obsessive-Compulsive Disorder - Mümin Kazım Yazıcı

Cognitive-Behavioral Conceptualization of Obsessive-Compulsive Disorder Subtypes - Hakan Türkçapar

HALL 4

SYMPOSIA ORAL SESSION-10

ARTIFICIAL INTELLIGENCE–ASSISTED CHILD AND ADOLESCENT PSYCHIATRY: BEYOND TREATMENT RESISTANCE

Session Chair: Tuba Mutluer

Pharmacogenetics and Artificial Intelligence: Personalized Medication Selection in Treatment-Resistant Cases – Merve Altunsoy

Data-Driven Clinical Decisions: Predicting Treatment Response with Machine Learning - Yasemin Yeter Demir Üstündağ

AI-Assisted Psychotherapies and Digital Phenotyping - Hatice Elvan Keskin

Ethics, Data Security, and Clinical Integration: Boundaries of AI Use in Child and Adolescent Psychiatry - Gökçe Kırlı Öcal

HALL 5

SYMPOSIA ORAL SESSION-11
ADDRESSING TREATMENT RESISTANCE IN PTSD AND COMPLEX PTSD
Session Chair: Yasir Şafak
PTSD and Complex PTSD: Unpacking the Roots of Treatment Resistance - İbrahim Gündoğmuş
Childhood Trauma, High Trauma Load, and the Roadblocks to PTSD Recovery - Hasan Ünver
PTSD with Comorbidities: Untangling the Factors That Fuel Treatment Resistance - Kübra Özcan Çetin
Personality Patterns and PTSD: Cracking the Code of Treatment Resistance - Mehmet Rıdvan Varlı

HALL 6

SYMPOSIA ORAL SESSION-12
CHILDREN DRAWN TO CRIME AND VICTIMS OF CRIME
Session Chair: Selma Tural Hesapçıoğlu
The Concept of Children Drawn to Crime and Victims of Crime - Görkem Erçin Esen
Statistics on Children Drawn to Crime - Ayşe Gülden Kaya
Psychiatric Conditions in Children Drawn to Crime and Victims of Crime - Seda Kafalı
Solution Proposals for the Relationship Between Crime and Children - Merve Okuyucu

HALL 7

SYMPOSIA ORAL SESSION-13
IN THE SHADOW OF ALGORITHMS: THE PSYCHIATRIC, TECHNOLOGICAL AND ARTIFICIAL INTELLIGENCE DIMENSIONS OF
DIGITAL ADDICTIONS
Session Chair: Erdoğdu Akça
Technology and the Attention Economy: Designing Behavior - Halil İbrahim Eren
Clinical Aspects and Neurobiological Basis of Digital Addictions - İbrahim Halil Deniz
Artificial Intelligence and the Future of Addiction - Turgut Hacıoğlu
Clinical Management of Digital Addictions and Modern Psychiatric Approaches - Ayten Yüksek

HALL 8

MEET THE EXPERT-2
FROM PIXELS TO PRACTICE: CLINICAL CLASSIFICATION AND ACT INTERVENTION IN PORNOGRAPHY USE
Session Chair: Hasan Turan Karatepe
Making Sense of Pornography Use: From Clinical Classification to Tailored Interventions - İlker Aktürk

HALL 3

PLENARY LECTURE-5
Session Chair: Ayşegül Yıldız
Clinical Predictors and Prognostic Factors for Progress in First-Episode Psychosis - Celso Arango

10:30 - 10:45 *COFFEE BREAK*

10:45 – 11:45

HALL 1

KEYNOTE SPEECH
Session Chairs: Feyza Arıcıoğlu - Mesut Çetin
Arketamine: The Overlooked Driver of Ketamine's Rapid and Sustained Antidepressant Effects - Kenji Hashimoto

11:45 - 12:00 *COFFEE BREAK*

12:00 – 13:00

HALL 1

SATELLITE SYMPOSIUM - 1 / JOHNSON & JOHNSON

BYANNLI®: Antipsychotic Used Every 6 Months - Özcan Uzun, Mesut Yıldız

13:00 – 14:00

HALL 1

LUNCH

HALL 2

POSTER PRESENTATION SESSİON 1 / PLASMA -1
Session Chair: Mesut Yıldız
**Mirtazapine-Induced Hypotension in a Patient With Psychosomatic Depression - Ömer Osman Bazna*
**A Case of Refeeding Syndrome (RFS) Developing in Result of Successful Treatment of Catatonia in Major Depressive Disorder: The Importance of Predicting RFS as a Probable Complication - Elif Gül Dönmez*
**Trauma-Triggered Olfactory Hallucination During a Manic Episode: Amygdala-Mediated Sensory Memory Reactivation Following Earthquake Exposure - Hatice Şahin*
**Computer-Assisted Forensic Psychiatric Interview: A Case Report - Ahmet Çöpür*
**When psychosis comes first: Huntington's disease with acanthocytosis mimicking schizophrenia – a case-based review – Esra Acar*
**Serotonin Syndrome Following the Addition of Linezolid in a Patient Receiving SSRI Pharmacotherapy - Hasan Berat Civelek*
**Resolution of Antipsychotic-Induced Hyperprolactinemia After Switching from Risperidone to Lurasidone: A Case Report - Ezgi Topaloğlu Civelek*
**The Unbroken Dyad: One Self, Two Bodies—A Joint Suicide Attempt in Dizygotic Twins and Review of the Literature – Nisa Oyartı Karabulut*
**A Life-Threatening Complication: Pulmonary Embolism Following Zuclopenthixol Acetate - Abdullah Ayaz*

HALL 3

POSTER PRESENTATION SESSİON 1 / PLASMA -2
Session Chair: Yasin Kavla
**Mirtazapine induced visual hallucinations in an adolescent: a case report - Cumali Yüksekkaya*
**Combined Use of Clozapine and Monthly Long-Acting Injectable Paliperidone Palmitate: A Case Report - Merve Işıklı*
**MTHFR A1298C Polymorphism in Psychosis with Neurodevelopmental Red Flags: A Case Report - Yusuf Özgür Memiş*
**Substance/Medication-Induced Psychotic Disorder Following Propranolol Treatment: A Pediatric Case - Hande Çelik*
**Melanopsin-Mediated Pupillary Responses in Remitted Bipolar Disorder Using Chromatic Pupillometry: A Preliminary Report - Işık Batuhan Çakmak*
**Evolution of Buprenorphine/Naloxone Maintenance Therapy into Intravenous Misuse: A Case of Pharmacodynamic Destabilization, Hepatitis C Seropositivity, and Psychotic Symptoms - Betül Gizem Cevahir*
**Progression from selective mutism to bipolar disorder: a 10-year clinical follow-up of a case - Sabriye Erdem*
**Dose-dependent fluoxetine-associated amenorrhea without hyperprolactinemia: a case report - Aslıhan Gümüşlü Ağcabay*
**Neuroleptic Malignant Syndrome Complicated by Severe Hyponatremia in Schizophrenia: A Case Report - Kübranur Yerlikaya*

HALL 4

POSTER PRESENTATION SESSİON 1 / PLASMA -3
Session Chair: Ömer Yanartaş
Clozapine-associated stuttering with EEG abnormalities: Resolution with valproate and long-term stability — A case report - Bedirhan Şenol
Manic episode triggered by discontinuation of Aripiprazole in an adolescent diagnosed with Kleine–Levin Syndrome: Kleine–Levin Syndrome or Mood Disorder? - İlayda Söğüt
When Imaging Is Silent, Clinical Clues Speak: A Case Of Poretti–Boltshauser Syndrome with Normal Brain MRI - Hatice Gülşen
Cotard Syndrome in a 50-Year-Old Female - Begüm Özsaraç
A Systemic and Relapse-Prevention Approach to Alcohol Use Disorder in a Surgeon: Partner-Involved Treatment – Ümmühan Özkal

Paliperidone-Induced Toxic Hepatitis in a Patient with Psychosis: A Case Report - Pınar Ertunç
Onset of Motor Tics Following Citicoline Use in a Child With Dopaminergic Sensitivity: A Case Report - Merve Gül Açıkgöz
Resolution of brain fog in a patient with bipolar depression through shift from fluoxetine-olanzapine combination to vortioxetine and olanzapine in 3 days - Atıf Arpaçukuru
Guanfacine in a Patient with ADHD and Hypertension: A Single Solution for Two Conditions? - Ceyda Bıyıklı
Diagnostic Value of a Structured Mental State Examination With Sign Language Interpretation: A Case of Schizophrenia with Preschool-Onset Total Deafness and Speech Impairment - Bengisu Nermin Yedievli

HALL 5

POSTER PRESENTATION SESSİON 1 / PLASMA -4
Session Chair: Hasan Turan Karatepe
Severe developmental language disorder without autism in 16p11.2 deletion syndrome: a longitudinal case report – Şevval Öztürk Algan
A Neurodevelopmental Disorder Associated With A Novel Nonsense Variant In Pıdd1: A Case Report - Hayriye Rumeysa Erdem
A Rare but Reversible Adverse Effect of Escitalopram: Drug-Induced Alopecia - Ezgi Şişman
Co-occurrence of Sleep Attacks and Neuropsychiatric Symptoms: Initial Experience of Kleine–Levin Syndrome in Our Clinic - Yağmur Yetimoğlu
Severe Restrictive Anorexia Nervosa with Multi-Organ Failure: A Clinical Case Analysis - Feyza Nur Gümrükçü
Recurrent Black Hairy Tongue Linked to Olanzapine: A Case of Challenge and Dechallenge - İlay Dalkıran
Severe Intellectual Disability and Epilepsy Associated with a Homozygous ATP8A2 Splice-Region Variant: A Case Report from a Multiplex Family - Görkem Erçin Esen

HALL 6

POSTER PRESENTATION SESSİON 1 / PLASMA -5
Session Chair: Erdoğdu Akça
Sertraline Treatment in a Patient With Anxiety Disorder Presenting With Pathological Laughter: A Case Report - Salim Çağatay Kağızman
Sertraline-Associated Acute Urinary Retention Following Dose Escalation: A Case Report - Salim Çağatay Kağızman
Pharmacological management of inappropriate sexual behaviors in a adolescent with Autism Spectrum Disorder: use of GnRH agonists - Zehra Özge Karaca
Escitalopram Discontinuation Related Brain Zaps in Panic Disorder: A Case Report - İmren Kurt Sabitay
Escitalopram-Responsive Ménière's Disease with Symptom Recurrence after Discontinuation: A Case Report - İmren Kurt Sabitay
Pseudologia Fantastica: Clinical and Forensic Challenges - Mualla Keskinsezer
Acute Urinary Retention Following Transition from Oral to Depot Risperidone - Mualla Keskinsezer

14:00 – 15:30

HALL 1

PLENARY LECTURE-6
Session Chairs: Mücahit Öztürk - Onur Burak Dursun
Reconceptualising ADHD as neurodivergence: Implications for science and practice - Edmund Sonuga-Barke

HALL 2

PLENARY LECTURE-7
Session Chair: Ayşe Sakallı Kani
Increasing Acces to Psychotherapy for Perinatal Depression Collaborative and Cost-Effective - Ryan J. Van Lieshout

HALL 3

SYMPOSIA ORAL SESSION-14
ARTIFICIAL INTELLIGENCE AND DIGITAL THERAPEUTICS IN PSYCHIATRY: FROM DEPRESSION TREATMENT TO YOUTH MENTAL HEALTH AND AI SIMULATION
Session Chair: Yanbo Zhang
AI Standardized Patients: Transforming Psychiatry Training through Simulation - Yuqi Wu
Using AI Responsibly in Psychiatric Practice: A Clinician's Appraisal Framework - Yanbo Zhang

HALL 4

SYMPOSIA ORAL SESSION-15
PLACEBO EFFECT IN PSYCHIATRY
Session Chair: Şahin Bodur
Placebo overview in psychiatry - Nazlıcan Tekin
Psychotherapy and the placebo effect - Cansel Azizağaoğlu
Antidepressants and the placebo effect - Burcu Akın Sarı
ADHD medications and the placebo effect - Müberra Üçüncü

HALL 5

SYMPOSIA ORAL SESSION-16
THE FEMALE PHENOTYPE IN CHILD PSYCHIATRY: NEURODEVELOPMENTAL DISORDERS, DISTINCT PRESENTATIONS AND BIOLOGICAL INFLUENCES
Session Chair: Gözde Yazkan Akgül
The Camouflage Effect: Understanding Autism Spectrum Disorder in Girls - Gözde Yazkan Akgül
Hidden in the Classroom: Specific Learning Disorders in the Female Population - Emine Ayhan
Riding the Wave: The Impact of Menstrual Cycles on Cognition and Emotion in Girls with ADHD - Yaşam Gizem Uluman
The Monthly Storm: Premenstrual Syndrome and Emotional Dysregulation in Adolescence - Esma Nur Bozyel

HALL 6

DOUBLE CONFERENCE-3
FROM MADNESS TO MAJOR PSYCHOSES: THE PROBLEMS OF CLASSIFICATION IN PSYCHIATRY
Session Chair: Mümin Kazım Yazıcı
Epistemological and Ontological Bases of Psychiatric Classification Systems - Cengiz Arca
Approaches to Classification in Psychiatry and the Critique of Contemporary Systems - İbrahim Aylak

HALL 7

MEET THE EXPERT-3
WHEN AND HOW SHOULD EFFECTIVE TMS TREATMENT BE ADMINISTERED FOR DEPRESSION?
Session Chair: Alpay Ateş
MEET THE EXPERT-3 - Alpay Ateş

HALL 8

COURSE-2
USER GUIDE FOR ANTIPSYCHOTICS
Session Chair: Özcan Uzun
User Guide for Antipsychotics - Özcan Uzun

15:30 – 15:45 COFFEE BREAK

15:45 – 16:45

HALL 1

SATELLITE SYMPOSIUM - 2 / SANOVEL
Session Chair: Mesut Çetin
LURASIDONE IN SCHIZOPHRENIA AND BIPOLAR DEPRESSION: REAL-WORLD CASE EXPERIENCES FROM CLINICAL PRACTICE - Köksal Alptekin

16:45 – 18:15

HALL 1

PLENARY LECTURE-8
Session Chairs: Hakan Türkçapar - Rümeysa Yeni Elbay
The Neurobiology of Social Dysfunction: a Transdiagnostic and Translational Approach - Martien Kas

HALL 2

PLENARY LECTURE-11
Session Chair: David N. Osser
Psychopharmacology Algorithm for Generalized Anxiety Disorder in Older Adults - Eran Metzger

HALL 3

SYMPOSIA ORAL SESSION-17
PSYCHEDELICS AND KETAMINE FOR DEPRESSION AND PTSD, FROM ANIMAL STUDIES TO CLINICAL COMPETENCE
Session Chairs: Xinmin Li - Yanbo Zhang
Cultural Perspective of Psychedelics for psychiatric Disorders - Yanbo Zhang
Reopening Critical Periods: Psychedelics, Sigma-1 Receptor Signaling, and Late-Life Neuroplasticity - Xiaohui Wang
Healing the Fragmented Self: Psychedelic-Assisted Psychotherapy for Trauma and Identity Integration - Yanbo Zhang

HALL 4

SYMPOSIA ORAL SESSION-18
INTOLERANCE OF UNCERTAINTY AS A TRANSDIAGNOSTIC FACTOR IN CHILDHOOD PSYCHOPATHOLOGY
Session Chair: Alperen Bıkmazer
Intolerance of Uncertainty as a Transdiagnostic Factor and Intolerance of Uncertainty in Children with Anxiety Disorders – Tuba Sürücü
Intolerance of Uncertainty in Children with Hoarding Disorder - Ecren Parlak Kuvvet
Intolerance of Uncertainty in Children with Post-Traumatic Stress Disorder - Özlem Nida Erbaşı
Intolerance of Uncertainty in Children with Eating Disorders - Cansu Çınar

HALL 5

SYMPOSIA ORAL SESSION-19
IMPULSIVITY AND COMPULSIVITY IN PSYCHIATRIC DISORDERS: A DIMENSIONAL CLINICAL APPROACH
Session Chair: Ömer Faruk Demirel
Impulsivity and Compulsivity in Psychiatric Disorders: A Dimensional Clinical Approach - Salim Çağatay Kağızman
Diagnoses Across the Spectrum: Clinical Manifestations of Impulsivity and Compulsivity - Ece Yazla Asaf
Clinical Interventions for Impulsive and Compulsive Symptoms: From CBT to Pharmacotherapy - İrem İlter
From Impulsivity to Compulsivity: Developmental Processes and a Transdiagnostic Future-Mehmet Sertçelik

HALL 6

SYMPOSIA ORAL SESSION-20
ARTIFICIAL INTELLIGENCE IN PSYCHOPHARMACOLOGY: APPLICATIONS, EXPERIENCES AND FUTURE PERSPECTIVES
Session Chairs: Selma Tural Hesapçıoğlu - Muhammed Tayyib Kadak
Artificial Intelligence Applications in Psychopharmacology: Current Status from Diagnosis to Treatment - Yasin Çalışkan
Digital Natives and Mental Health: The Use of Artificial Intelligence in Young People's Psychiatric Treatment Processes – Yavuz Meral
Artificial Intelligence in Psychopharmacology: Ethical Issues, Challenges and Future Trends - Tuncay Sandıkçı
AI-Driven Prediction of Drug-Drug Interactions in Psychopharmacology: A New Era of Safety-AbasHaşimoğlu

HALL 7

COURSE-3
CURRENT TREATMENT APPROACHES IN GAMBLING DISORDER: CBT, PHARMACOTHERAPY, AND NEUROMODULATION
Session Chair: Bahadır Geniş
Current Evidence on Pharmacotherapy and Neuromodulation in the Treatment of Gambling Disorder - Muhammed Hakan Aksu
Case-Based Cognitive Behavioral Therapy in Gambling Disorder - Bahadır Geniş

HALL 8

COURSE-4
WORKING WITH ATTENTION DEFICIT AND HYPERACTIVITY THROUGH SCHEMA THERAPY: A PRACTICAL MODE-BASED FRAMEWORK
Session Chairs: Gökçen Güven - Abdullah Bozkurt
Working with Attention Deficit and Hyperactivity through Schema Therapy: A Practical Mode-Based Framework – Gökçen Güven
Working with Attention Deficit and Hyperactivity through Schema Therapy: A Practical Mode-Based Framework – Abdullah Bozkurt

	18:15 – 18:30 COFFEE BREAK
	18:30 – 20:00
	HALL 1
	PLENARY LECTURE-2 *Session Chairs: Mesut Çetin - Feyza Arıcıoğlu* *An Appraisal of Some New and Newer Medications for Major Depression and Schizophrenia - David N. Osser*
	HALL 2
	PLENARY LECTURE-10 *Session Chair: Tomas Hajek* *The Biological Cost of Latency: Linking Diagnostic Delay to Metabolic Burden and Brain Aging in Bipolar Disorder – Ayşegül Yıldız*
	HALL 3
	JOINT SESSION *Psychiatry and Spirituality - Kemal Sayar, Erol Göka, Hayrettin Kara*
	HALL 4
	SYMPOSIA ORAL SESSION-21 *POST-SSRI SEXUAL DYSFUNCTION (PSSD): CLINICAL FEATURES, MECHANISMS, AND CURRENT DEBATES* *Session Chair: Erdoğdu Akça* *Clinical Spectrum and Phenomenology of PSSD - Cansun Şahin Çam* *Neurobiological Mechanisms and Pathophysiology - Ebru Şahan Aydın* *Diagnostic Challenges, Management Strategies and Future Directions - Ahmet Özercan*
	HALL 5
	SYMPOSIA ORAL SESSION-22 *ANTIPSYCHOTIC-INDUCED WEIGHT GAIN: FROM MECHANISMS TO GLP-1–BASED STRATEGIES* *Session Chair: Burcu Akın Sarı* *Mechanisms of Antipsychotic-Induced Weight Gain (AIWG) - Halenur Teke* *Intervention strategies for AIWG: Lifestyle, Switching and Pharmacologic Augmentation - Mustafa Dinçer* *Overview of Glucagon-Like Peptide-1 Receptor Agonists (GLP-1 RAs): Pharmacokinetic/Pharmacodynamic considerations, feasibility, and choice - Özge Demircan Tulacı* *Clinical Evidence on GLP-1 RAs: Effects on Glucose Dysregulation and AIWG –* *Furkan Mavi*
	HALL 6
	SYMPOSIA ORAL SESSION-23 *OVERCOMING TREATMENT RESISTANCE IN DEPRESSION* *Session Chair: Ali Saffet Gönül* *The Neurobiology of Depression: Overcoming Treatment Resistance - Ali Saffet Gönül* *Dilemma in Schizophrenia: Negative Symptom or Depression? - Aslı Ceren Hınç* *Recognizing Depression with Mixed Features - Furkan Yazıcı*
	HALL 7
	SYMPOSIA ORAL SESSION-24 *PTSD AND COMPLEX PTSD IN CHILDREN AND ADOLESCENTS* *Session Chair: Abdurrahman Cahid Örengül* *PTSD and Complex PTSD in Children and Adolescents: Assessment and Measurement Tools - Rana Selin Küçükardalı* *PTSD and Complex PTSD in Children and Adolescents: Emotional Difficulties - Beyza Karal* *PTSD and Complex PTSD in Children and Adolescents: Neuropsychological Challenges - Afife Özkal* *PTSD and Complex PTSD in Children and Adolescents: Treatment Approaches –* *Abdurrahman Cahid Örengül*

HALL 8

SYMPOSIA ORAL SESSION-25

ADHD IN WOMEN FROM THE PERSPECTIVE OF HORMONAL CYCLES: NEUROBIOLOGY, CLINIC AND TREATMENT OPTIONS

Session Chair: Ayşe Sakallı Kani

The menstrual cycle and ADHD in the reproductive period: PMS, other comorbidities and treatment approaches – Ayşe Süleyman

ADHD Mothers: Management of ADHD during pregnancy and lactation - Didem Aykaç İzi

ADHD in elder women: premenopausal and postmenopausal period - Didem Kılıç

17 April 2026 Friday

08:00 – 09:00

HALL 1

COURSE-5

ALL OR NOTHING! UNDERSTANDING PERFECTIONISM FROM AN ACT PERSPECTIVE

Session Chair: Hasan Turan Karatepe

Tuğba Nur Sayımlar Erginay, Zülal Çelik

HALL 2

ORAL PRESENTATIONS SESSION-2

Session Chair: Ömer Yanartaş

Abuse Of Unexpected Drugs: A Case Series - İlknur Gündoğdu

Healthcare Workers' Knowledge, Attitudes, and Stigma About ADHD - Hatice Gülşen

The Attitudes, Beliefs and Knowledge of Parents About Methylphenidate Treatment - Hilal Yazıcı Kopuz

Psychological Correlates of Pain Severity in Adolescents with Non-Cardiac Chest Pain: A Multivariate Analysis - Armağan Aral

Valuation Of The Relationship Between Online Risky Sexual Behaviors And The Attitudes Of The Parents Of Adolescents With Attention Deficit And Hyperactivity Disorder - Veysel Özdağ

Perceived Competence to Initiate Clozapine Treatment Among Child and Adolescent Psychiatrists: The Role of Institutional Support and Professional Experience - Tevhide Ekin Süt

Parental burnout is related to attention-deficit/hyperactivity disorder symptoms in a large sample of Turkish mothers – Sarper İçen

Investigation of Cognitive Function, Activities of Daily Living, and Depression in Adults Aged 65 Years and Older - Pınar Ertunç

Caffeine Use Disorder and Attention-Deficit/Hyperactivity Disorder Symptoms in Medical Students: A Cross-Sectional Study - Elif Küçük

HALL 3

Session Chair: Gökhan Ünal

Gender-Based Assessment of Academic Procrastination, Diagnosis and Cognitive Performance in Children - Ayfer Karabulut

Mental Health Literacy, Depression, Perceived Stress, and Psychological Help-Seeking During Pregnancy - İlknur Kiraz

Theory of Mind, Alexithymia, and Perceived Loneliness in Adolescents with Attention-Deficit/Hyperactivity Disorder – Ayşe Selma Yenen Menderes

Astrocytic Extracellular Matrix Proteins in ADHD: The Role of Thrombospondin-1, Sparc, and Sparcl-1 - Nilüfer Yıldız Kısmet

Psychometric Properties of the Turkish Version of the Bipolar Self-Efficacy Scale (BPSES) - Cansu Ünsal Mavi

Eating Problems in Children with ASD and Maternal Mental Health: The Mediating Role of

Maternal Quality of Life - Büşra Nur Dambasan Gündüz

The Structural Framework of Anhedonia: A Transdiagnostic Analysis of Striato-Cortical Circuits - Mehmet Akif Şakiroğlu

Knowledge, Clinical Expectations, and Ethical Attitudes of Child and Adolescent Psychiatrists in Türkiye toward Xanomeline–Trospium (KarXT): Preliminary Findings - Müge Mutlu

HALL 4

ORAL PRESENTATIONS SESSION-4

Session Chair: Şahin Bodur

The Effect of Comorbid Autism Traits on Perceived Social Support, Loneliness and Suicide Risk in Adolescents Diagnosed with Social Anxiety Disorder - Elif Burcu Bağçeli

Predictors of Smartphone Distraction: The Contributions of Emotion Regulation Difficulties, Anticipatory Pleasure, and ADHD Inattentive Symptoms - Burak Okumuş

From stethoscopes to screens: prevalence and associated factors of internet addiction among healthcare workers in Mogadishu, Somalia – a cross-sectional study - Nur Adam Mohamed

Neuroepigenetic-Regulating Long Non-Coding RNA Polymorphisms in Newly Diagnosed Psychosis - Ayşe Döndü

Relationship Between Maternal Emotion Regulation Difficulties, Feeding Practices, and Preschool Malnutrition: A Cross-Sectional Comparison - Ayşegül Demir Çevirici

Examining the Relationship Between Death Literacy and Death Anxiety - Doğancan Sönmez

Cultural discontinuity, cultural resilience, and mental health in the era of Large Language Model: Civilizational disruption and reconstruction in the context of technological revolution - Chengyi Liu

Comparison of Digital and Traditional Mindfulness-based Interventions for major depressive disorder: A Systematic Review

and Network Meta-Analysis of Randomized Clinical Trials - Chengyi Liu

HALL 5

ORAL PRESENTATIONS SESSION-5

Session Chair: Tuncay Sandıkçı

Examining the relations of mindfulness and metacognitive processes in adolescents with obsessive–compulsive disorder - Doğa Sevinçok

Emotional problems, alexithymia, body image and interoception in adolescent girls with anorexia nervosa - Dila Elif Sema Mert Kılıç

Cognitive Disengagement Syndrome and Executive Function Difficulties in Children with Specific Learning Disorder: A Preliminary Study - Ekrem Özçopur

Intergenerational Transmission of Post-Migration Stress and Peer Exclusion in Second-Generation Syrian Refugee Children in a Shared Low-Resource District: A Longitudinal Mixed-Methods Study - Tayfun Kara

Maternal Smartphone Addiction, Parenting Stress, and Children's Perceived Emotional Neglect and Attachment Insecurity - Tayfun Kara

Polypharmacy and comorbidity burden in adolescents with autism spectrum disorder in an inpatient psychiatric unit - Dila Elif Sema Mert Kılıç

Public Attitudes Toward the Use of Artificial Intelligence in Mental Health: A Cross-Sectional Online Survey Study - Begüm Eser Uyar

HALL 6

ORAL PRESENTATIONS SESSION-6

Session Chair: Yasir Şafak

Evaluation of the Relationship Between Social Appearance Anxiety and Body Image with Obesity and Lipid Profile in Patients with Obsessive-Compulsive Disorder - Elif Nur Donbaloğlu

Short-Form Video Platforms in Child and Adolescent Psychiatry: A Bibliometric Analysis of Research Trends and the Shift Toward Psychopathological Focus - Meryem Seçen Yazıcı

The Relationship Between Psychosocial Risk, Psychiatric Symptoms, and Post-Traumatic Growth With Quality of Life in Organ Transplant Recipients: Preliminary Findings From a Longitudinal Study - Aslı Ceren Hınç

Anticipated Stigma in Adolescents with Chronic Illness: Validation of the Turkish CIASS-A and Associations with Psychosocial Outcomes - Muhlise Özkan Namlı

Metabolic Syndrome and Cognitive Function in Bipolar I Disorder: A Pilot Cross-Sectional Study Accounting for Illness Burden and Treatment - Mehmed Ediz Çelik

Aggression and Impulsivity in Methamphetamine and Alcohol Use Disorders: A Comparative Preliminary Study - Özge Akyürek Bolat

Transition to Long-Acting Injectable Aripiprazole in Chronic Psychiatric Disorders: A Retrospective Analysis of Clinical Functioning and Caregiver Burden - Hazan Tomar Bozkurt

The Invisible Barrier to Mandatory Treatment: The Relationship Between Internalized Stigma and Treatment Motivation in Probationers - Hüseyin Ozan Torun

HALL 7

ORAL PRESENTATIONS SESSION-7

Session Chair: Yasin Kavla

Irritability in generalized anxiety disorder: preliminary cross-sectional findings on its associations with theory of mind and psychological symptoms - Hakan Yılmaz

Evaluation of the Treatment Efficacy and Tolerability of Caripiprazine in Schizophrenia and Bipolar Disorder: Case Series - Sinem Zeynep Yıldız

Cross-Validated Bipolar Disorder Classification and Risk Stratification Using Penalized Logistic Regression and Machine Learning in a Single-Center Pilot Case–Control Study - Mehmed Ediz Çelik

Drug-Related Suicide Deaths: Medicolegal Analysis of sociodemographic and clinical characteristics - Halil İbrahim Eren

Depression Severity as the Primary Predictor of Functional Outcomes: The Role of Coping and Metacognitive Beliefs in Adjustment Disorder - Halil İbrahim Eren

An Investigation of the Relationship Between Reading-Writing Performance and Executive Functions, Neurocognitive Profile, and Functionality in Children with Specific Learning Disorder and Comorbid AttentionDeficit/Hyperactivity Disorder – Ebrar Onar

Admission Augmentation and Polypharmacy Patterns as Pragmatic Indicators of Treatment Resistance in Patients Hospitalized for a Depressive Episode: Associations With Electroconvulsive Therapy Use - Muhammed Can Altınay

Investigation of Factors Associated with Suicide Attempts in Patients with Major Depressive Disorder - Fatma Nur Meral Altınay

HALL 8

ORAL PRESENTATIONS SESSION-8

Session Chair: Abas Haşimoğlu

The Protective Role of Self-Compassion Against Psychiatric Morbidity in Dermatological Diseases: A Cross-Sectional Pilot Study - İbrahim Hakkı Karakuş

Early Changes in Inflammatory Indices Obtained from Hemogram After Electroconvulsive Therapy - Hürmet Tosun

Intrusive verbal thought and intrusive visual imagery in depressive and anxiety disorders: The role of serotonergic medication - Ezgi Cellat

Multidimensional Assessment of Acne Vulgaris: Links Between Psychiatric Symptoms, Disordered Eating, and Dermatologic Severity - Ayşe Döndü

Exploring the lived experiences of young adults after the death of their mother - Iman Shafique

Masculine Honor Ideology and Aggression Across Genders - Ayesha Rai

Guanfacine-Associated Stuttering in Autism Spectrum Disorder: The First Reported Case in the Literature - Burak Kamış

The Effectiveness of Nonpharmacological vs. Pharmacological Interventions in Major Depression, Bipolar Disorder, and Schizophrenia - Yasin Tayem

09:00 – 10:30

HALL 1

RATIONAL DRUG USE SYMPOSIUM

COMORBID MEDICAL DISEASES AND PSYCHIATRIC DISORDERS: MASTERING RATIONAL PSYCHOPHARMACOLOGY – CLINICAL

PEARLS AND PITFALLS

Session Chairs: Mustafa Solmaz - Gökhan Ünal

Rational Psychopharmacology: Pharmacodynamics and Pharmacogenetics - Gökhan Ünal

Rational Psychopharmacology in Cardiovascular Diseases: Drug–Drug and Drug–Disease Interactions - Veysel Oktay

Rational Psychopharmacology in Metabolic Diseases: Drug–Drug and Drug–Disease Interactions - Erdoğdu Akça

Rational Psychopharmacology in Neurological and Renal Diseases: Drug–Drug and Drug–Disease Interactions - Ömer Yanartaş

HALL 2

PLENARY LECTURE-12

Session Chair: Mümin Kazım Yazıcı

Responses to the Theses of Dostoevsky's The Gambler - Erol Göka

HALL 3

SYMPOSIA ORAL SESSION-26

LITHIUM: NEW FINDINGS ABOUT THE OLDEST TREATMENT

Session Chair: Rebecca Strawbrıdge

Neuroprotective Effects of Lithium – Food for Thought - Tomas Hajek

Temporospatial Lithium Kinetics in the Brain Using Repeat ^{7}LiMRI Imaging - Philipp Ritter

Mind the Gap: Rethinking Lithium Monitoring for the 21st Century - Rebecca Strawbrıdge

HALL 4

SYMPOSIA ORAL SESSION-27

THE SCIENTIFIC BASIS OF COGNITIVE ENHANCEMENT IN NEURODEVELOPMENTAL DISORDERS: THE CONTEXT OF INTELLECTUAL DISABILITY

Session Chair: Tayfun Kara

Intellectual Disability within the Neurodevelopmental Disorders - Orhan Kocaman

Major Approaches to Cognitive Enhancement: Concepts and Mechanisms - Tayfun Kara

Pharmacological and Neurotechnological Interventions: Possibilities and Limitations - Fahri Çelebi

HALL 5

SYMPOSIA ORAL SESSION-28

POSTPARTUM PATERNAL DEPRESSION

Session Chair: Rümeysa Yeni Elbay

Postpartum Paternal Depression - Talha Ağaç

The Effect of Postpartum Paternal Depression on Mother - İrem Yazıcı Karabulut

The Effect of Postpartum Paternal Depression on Children - Nihal Serdengeçti

HALL 6

SYMPOSIA ORAL SESSION-29

BEHAVIORAL ADDICTIONS IN DEVELOPING MINDS

Session Chair: Muhammed Tayyib Kadak

Theoretical Controversies and Conceptual–Diagnostic Boundaries in Behavioral Addictions - Büşra Arslan

Shared Neurobiology, Neurodevelopmental Windows, and Pediatric Vulnerability in Behavioral Addictions - Meltem Küçükdağ

Game or Addiction? Assessing Gaming and Gambling Disorders in Children and Adolescents - Meryem Seçen Yazıcı

Hidden Behavioral Addictions in Youth: Compulsive Buying, Exercise, Hypersexual Behavior, and Binge Eating - Ceren Tosun

HALL 7

SYMPOSIA ORAL SESSION-30

ATTENTION DEFICIT HYPERACTIVITY DISORDER: TREATMENT FROM CHILDHOOD TO ADULTHOOD

Session Chair: Akın Tahıllıoğlu

Attention Deficit Hyperactivity Disorder: Treatment in Childhood - Akın Tahıllıoğlu

Management of Psychiatric Comorbidities in Attention Deficit Hyperactivity Disorder - Fadime Bayraktar

Cognitive Behavioral Therapy for Attention Deficit Hyperactivity Disorder - Nilay Bilgin

HALL 8

MEET THE EXPERT-5

PHENOMENOLOGY AT THE INTERSECTION: CASE-BASED EXPLORATION OF SCHIZO-OBSESSIVE DISORDER WITH EXPERT INSIGHT

Konuşmacılar - Kemal Sayar, Ayşe Süleyman

10:30 – 10:45 COFFEE BREAK

10:45 -11: 45

HALL 4

SESSION OF STARS - 5

Session Chair: Yasin Çalışkan

Self-Acceptance as a Moderator of the Relationship Between Emotion Dysregulation and Emotional Eating in Adolescents with Major Depressive Disorder - Eda Çelik

Comparison of Telomere Length in Patients with Schizophrenia Using Clozapine - Elif Ateş Budak

Evaluation of Drug-Related Suicide Attempts in Terms of Age, Gender, and Time - Barış Sağlık

Investigation of Plasma Amino Acid Profiles in Children Diagnosed with Obsessive–Compulsive Disorder Using LC–MS/MS Analysis - Özgür Esmeray

The Effect of Child-Centered Play Therapy on Self-Compassion and Emotion Regulation Skills in Children with Cancer – Şaziye Senem Başgül

Meaning in Life Profiles and Mental Health Outcomes Among Medical Students: A Latent Profile Analysis - Esat Soylu

The Role of Emotion Regulation and Sleep Quality in the Relationship Between Problematic Digital Media Use and Functioning in Adolescents Diagnosed with Attention Deficit Hyperactivity Disorder - Nadide Fettahlıoğlu

Psychiatrists' Preferences for Long-Acting Injectable Antipsychotics and Their Attitudes Toward New-Generation Psychotropic Medications: How Open Are Psychiatrists to Innovation? - Muratcan Agaçe

The Impact of Perfectionism on Emotional Dysregulation in Adults with ADHD - Nilay Bilgin

HALL 5

SESSION OF STARS - 6

Session Chair: Abas Haşimoğlu

Comparison of Clinical and Socio-Demographic Characteristics and Rorschach Test Findings of Offenders Diagnosed with Schizophrenia and Substance-Induced Psychosis in Turkey: A Retrospective Study - Mustafa Akan

Social Skills in Specific Learning Disorder: Associations with Executive Functions, Theory of Mind, and Emotion Regulation - Tuba Karakuş Aslan

Social and Psychiatric Risk Profiles of Juveniles Involved in Sexual Offenses: Analysis of 82 Forensic Cases - Neşe Kavruk Erdim

Meaningful Gains with Small Data: Poisson-Port, a Novel Neural Network Architecture—Validated on Two Challenging Psychiatric Datasets (Depression Severity and Suicide-Risk Classification) - Ece Yazla Asaf

Carbon Footprint of Antipsychotic Treatment in Schizophrenia: A Comparative Analysis of Oral and Long-Acting Injectable Antipsychotics - Sakine Aktaş

Somatization, Coping Styles, Anxiety, and Sleep Habits in Children And Adolescents With Alopecia Areata - Meltem Küçükdağ

Potential Association Between SNAP-25 rs362998:C>T and Methylphenidate Treatment Response in ADHD: A Next-Generation Sequencing Study - Zeynep Gizem Todurga Seven

Evaluation of Dopamine Transporter DAT1 Gene Variations in Turkish Children with ADHD Treated with Methylphenidate Adverse Effects - Zeynep Gizem Todurga Seven

Comparison of Disease Duration and Length of Hospitalization with Hematological Inflammatory Markers in Patients with Schizophrenia - Feride Uysal

HALL 8

SYMPOSIA ORAL SESSION-31

FROM POISON TO ANTIDOTE: PSYCHIATRIC OUTCOMES OF THE ELEMENTS

Session Chair: Özcan Uzun

Essential Trace Elements and Psychiatric Outcomes - Özcan Uzun

Heavy Metals and Mental Health - Meryem Sebla Ertuğrul

Use of Trace Elements in Psychiatric Disorders - Esra Yalım

11:45 – 12: 45

HALL 1

SATELLITE SYMPOSIUM - 3 / ABDİ İBRAHİM OTSUKA

REDEFINE EXPECTATIONS WITH REXULTI: REAL-WORLD CASE EXPERIENCES IN NEUROPSYCHIATRIC DISORDERS - Ali Saffet Gönül, Sibel Çakır

12:45 – 13:45

HALL 1

LUNCH

HALL 2

POSTER PRESENTATİONS SESSİON-2 / PLASMA 1

POSTER PRESENTATİONS SESSİON 2 / PLASMA 1

Session Chair: Filiz Kulacaoğlu

Case Report of a Patient with Autism Spectrum Disorder Carrying a MYT1L Variant - Saliha Baykal

Suicide Attempt with Oleander Ingestion in an Adolescent - Saliha Baykal

Late Recognition of Autism Spectrum Disorder in a 55-Year-Old Man Followed as Schizoaffective Disorder: Diagnostic Pitfalls,Polypharmacy Burden and Suicide Risk - Nursel Şeker

Bright-Light Therapy in a Case of Non-Seasonal Recurrent Resistant Depression in a Geriatric Patient - Nursel Şeker

"Is More Better?": Liver Enzyme Elevation Following Self-Escalated Naltrexone in Gambling Disorder - Hüseyin Ozan Torun

Catatonia as the First Manifestation of Dementia with Lewy Body (DLB): A Case Report - Şerife Kübra Öztaş

Dissociative Amnesia in the Context of Prolonged Grief Disorder: A Case Report - Kübra Sever

Five-Session Written Exposure Therapy in a Young Woman with PTSD, Borderline Personality Disorder, and Bulimia Nervosa: Feasibility and Short-Term Clinical Outcomes - Miray Yıldızdal

Epilepsy-Related Encephalopathy in an Advanced Age Bipolar Disorder Case - Miray Yıldızdal

HALL 3

POSTER PRESENTATİONS SESSİON 2 / PLASMA 2

Session Chair: Rümeysa Yeni Elbay

Hyperacusis After Escitalopram Treatment: A Case Report - Kübra Özcan Çetin

Brexpiprazole-Induced Recurrent Hiccups During Antidepressant Augmentation: A Case Report - Mehmet Rıdvan Varlı

Misdiagnosed Parkinsonism and Dementia: A Case of Bipolar Depression Presenting with Catatonia - İbrahim Hakkı Karakuş

Fluoxetine-induced myokymia: a rare adverse effect - Ömer Osman Bazna

Kleptomania and Hoarding Disorder with Animal Hoarding: A Challenging Case - Selin Karakaya

Comparative risk of incident irritable bowel syndrome in PTSD Patients Treated with SSRIs vs. SNRIs: a real-world TriNetX cohort study - Chao Chung Ho

Individuation process and family conflict in a young woman diagnosed with MS: An ACT-focused archetypal analysis – Yasemin Günay

Pediatric Schizoaffective Disorder: A Case Report - Berk Uğurlu

Guanfacine-Responsive Chronic Motor Tic Disorder With Comorbid Obsessive–Compulsive Disorder in an Adolescent: A Case Report - Ayşe Merve Benk Ayaz

HALL 4

POSTER PRESENTATİONS SESSİON 2 / PLASMA 3

Session Chair: Gökhan Ünal

A metabolic trap mimicking antipsychotic-induced motor side effects: 22q11.2 deletion syndrome and hypocalcemia management - Tuğçe Diren Uyanık

Fluoxetine-Associated Leukocytoclastic Vasculitis: A Case Report in an Adolescent - Beyza Nur Top Karakılınç

Somatic Delusions and Comorbid Obsessive Symptoms in Early-Onset Psychosis: A Case Report - Ceylin Köksal Cotard Syndrome Following Methamphetamine Use: A Rare Case with a Family History of Psychotic Disorder - Merve Sena Kırmacı

Schizophrenia-Like Behavioral Alterations in a Prenatal Poly(I:C)-Induced Model of Rats - Burcu Gül Akkoç

Cervical dystonia following dental extraction in a 13-year-old girl with autism spectrum disorder: a case report - Sevde Taşcı

Low-Dose Aripiprazole Augmentation and Thermoregulation Management in Generalized Anxiety Disorder with Dissociative Features Following Pediatric Hodgkin's Lymphoma: A Case Report - Selinay Üçeş

From Screening to Care: A Digital Mental Health Clinic Model for Schools Serving Children and Adolescents - Chengbin Guan

HALL 5

POSTER PRESENTATİONS SESSİON 2 / PLASMA 4

Session Chair: Şaziye Senem Başgül

Probable Ocular Migraine–Like Visual Phenomena After Escitalopram Dose Escalation: A Case Report - Ezgi Şişman

Management of Acute Psychotic Mania with Severe QTc Prolongation in an Adolescent with Substance Use: A Case Report - Sedanur Serdar Şimşek

Skin Damage While Attention Improves in Autism Spectrum Disorder: Finger Picking Following Methylphenidate Treatment -Öznur Adıgüzel Akman

Aripiprazole-Induced Late-Onset Visual Disturbance: A Case Report - Ayla Canlı İşler

Bipolar Disorder Accompanying Sjögren's Syndrome - Pınar Durmaz

Guanfacine Treatment for Polypharmacy-Resistant Behavioral Problems in Autism - Nurşen Bozkoyun

Transdiagnostic Distribution of Autistic Traits in Obsessive–Compulsive Disorder, Panic Disorder, Autism Spectrum Disorder, and Neurotypical Controls - Yunus Akkeçili

Catatonia associated with Wernicke encephalopathy after gastrointestinal resection: a case report - Ferize Yıldız Kaymak

HALL 6

POSTER PRESENTATİONS SESSİON 2 / PLASMA 5

Session Chair: Ayşe Kurtulmuş Çalış

Catatonia as a Prelude to Mania: A Bipolar Case Report - Buket Doğan

Cyclic Vomiting Syndrome in an Adolescent With Autism Spectrum Disorder: A Case Report Highlighting Behavioral Management Challenges - Abdullah Ayaz

A case of rapid nocturnal enuresis resolution after fluoxetine initiation - Hilal Tüter Mavi

Delusional Parasitosis Presenting with the Matchbox Sign in an Elderly Patient: A Case Report - Sema Çağal

Diagnostic Challenges in Wernicke's Encephalopathy: A Case with Atypical MRI Findings - Fatima Malikova

Autism Spectrum Disorder in a Female Adolescent: Diagnostic Challenges and Overlapping Presentations - Ebru Işık

Juvenile Huntington's Disease with early and atypical psychiatric manifestations - Meliha Rehnüma Karaca Çolaker

Sporadic Creutzfeldt-Jakob disease initially presenting as treatment-resistant depression with catatonic features: a case report - Büşra Söylemez Karakuş

HALL 7

POSTER PRESENTATİONS SESSİON 2 / PLASMA 6

Session Chair: Şahin Bodur

Artificial Intelligence as a Theme in Psychotic Delusions: A Case Report - Bahar Akgün

Cognitive Disengagement Syndrome in Cornelia de Lange Syndrome: Expanding the Neuropsychiatric Phenotype - Mehmet Ali Karaali

Electroconvulsive Therapy Experience in a Case of Autism with Treatment-Resistant Severe Behavioral Symptoms: A Case Report - Esma Candan Aksoy

Epistaxis Associated with Risperidone Dose İncrease: A Case Report - Beyzanur Gökçe Ceylan

Management of ADHD Symptoms in a Child with Taussig–Bing Anomaly, Cerebral Palsy, and Epilepsy: A Case Report – Şükret Alev

Hyperprolactinemia and Reversible Pituitary Lesion During Early Antipsychotic Treatment in First-Episode Psychosis: A Case Report - Fatma Çetinkaya Yamak

Attitudes Toward Regular Oral Medication Use and Medication Adherence in a Community Sample - Melike Düzgün

The Healing Power of Being Seen: A Case Report Illustrating the Psychiatric Implications of Family Disclosure in Gender Dysphoria - Ezgi Şişman

**Psilocybin-Induced Manic Episode: An Analysis via Neural Network Connectivity and Cortico-Striatal Dopaminergic Interaction - Mehmet Akif Şakiroğlu*

13:45 – 15:15

HALL 1

PLENARY LECTURE-13
Session Chair: Ali Saffet Gönül
Advancing Schizophrenia Research with Big Data: Findings from the ENIGMA Schizophrenia Working Group - Theodorus Van Erp

HALL 2

Session Chairs: Feyza Arıcıoğlu - Ömer Faruk Demirel
How Assistive AI Solutions Based on Large Language Models Will Improve Mental Health - Andrew Greenshaw

HALL 3

SYMPOSIA ORAL SESSION-32
Session Chair: Aynur Görmez
Clinically-Based Critical Issues in the Use of Psychedelics in Treatment-Resistant Depression - Giuseppe Bersani
Mental Health in the AI Era: Clinical Practice Through a Cross-Cultural Lens - Xinmin Li

HALL 4

SYMPOSIA ORAL SESSION-33
NEXT-GENERATION BIOMARKERS IN DEPRESSION: FROM MOLECULAR TRACES TO CLINICAL PRACTICE
Session Chair: Doğancan Sönmez
Biomarker Use in Clinical Practice: Is Targeted Antidepressant Treatment Possible? - Saniye Gökçe Saykal Yıldız
Cystatin C, Inflammation, and Depression: Mechanism or Marker - İbrahim Sefa Güleç
Inflammatory and Neurotrophic Biomarkers in Depression: CRP, IL-6, BDNF, and Beyond - Doğancan Sönmez
Wearable Technologies and Digital Phenotyping: Behavioral Biomarkers in Depression - Salim Çağatay Kağızman

HALL 5

SYMPOSIA ORAL SESSION-41
UNDERSTANDING DEATH THROUGH DIFFERENT LENSES
Session Chair: Mümin Kazım Yazıcı
The Concept of Death at the Intersection of Psychiatry, Philosophy, and Mythology - Serhat Çıtak
Death Anxiety from the Perspective of Cognitive Theory - Hakan Türkçapar
Dying While Living: Where Life Touches Death - Erol Göka

HALL 6

SYMPOSIA ORAL SESSION-35
BREAKING TREATMENT RESISTANCE IN AUTISM SPECTRUM DISORDER: CLINICAL AND MULTIDISCIPLINARY APPROACHES WITH THE ÇÖZGEM MODEL
Session Chair: Ayşegül Efe
Standard Treatments in ASD and Defining Resistance - Tuğba Acehan
Psychopharmacological Approaches to Resistant Symptoms in ASD - Huriye Berna Devecioğlu
Complementary and Alternative Treatments for Resistant Symptoms in ASD – Current Evidence - Esra Güngör Bağlıcakoğlu
Multidisciplinary Approaches to Treatment Resistance and the ÇÖZGEM Model - Merve Canlı

HALL 7

SYMPOSIA ORAL SESSION-36
CHILD AND ADOLESCENT PSYCHIATRIC DISORDERS: INSIGHTS INTO NEUROINFLAMMATORY MECHANISMS
Session Chair: Özge Gizli Çoban
The Role of Neuroinflammation in Autism Spectrum Disorder: From Pathophysiology to Clinical Implications - Ayşenur Özer Güvenç
Neuroinflammatory Pathways in Attention-Deficit/Hyperactivity Disorder - Berivan Gündürü
Inflammatory Mechanisms in Cognitive Disengagement Syndrome - Emine Esra Varkal Toprak
Neuroinflammatory Responses to Childhood and Adolescent Trauma - Berhan Akdağ

HALL 8

ORAL PRESENTATIONS SESSION-9

Session Chair: Yavuz Meral

The Relationship of Intolerance of Uncertainty, Psychological Flexibility, Repetitive Thinking, and Self-Criticism with Executive Functions in Individuals Diagnosed with Attention Deficit Hyperactivity Disorder - Tuğba Nur Sayımlar Erginay

Determınıng levels of anxıety, depressıon, and prenatal attachment ın patıents in the prenatal perıod admıtted to famıly health centers - Fadime Şİmşek

Evaluation of the Relationship Between Repetitive Negative Thinking and Quality of Life in Women Diagnosed with Endometriosis - Berra Öztürk

Emotional Blunting Associated With Antidepressant Treatment: A Cross-Sectional Study - Melike Düzgün

Knowledge, Skills, and Attitudes Toward Psilocybin Use in Child and Adolescent Mental Health Professionals: Preliminary Results - Batuhan Er

Clinical and Sociodemographic Comparison of Bipolar Depression and Major Depressive Disorder - Pınar Öztürk

Differences and Similarities Between Mothers and Fathers in Perceptions of Child Sleep and Psychosocial Functioning – Şefika Nurhüda Karaca Cengiz

Evaluation of comorbid psychiatric symptoms in patients presenting with sleep disorders during the COVID-19 pandemic - Keziban Turgut

Evaluation of Psycholeptic and Psychoanaleptic Drug Use in Pregnant Women - Sultan Kurt Yıldırım

Patterns of Complementary And Alternative Medicine Use Among Children And Adolescents With Autism Spectrum Disorder - Miraç Barış Usta

Psychometric Properties of the Abridged Version of the Autism-Spectrum Quotient (AQ-28) in a Turkish Non-Clinical Sample - Zeynep Nur Demirok

15:15 – 15:30 COFFEE BREAK

15:30 – 17:00

HALL 1

SYMPOSIA ORAL SESSION-39

COGNITIVE NEUROPSYCHOPHARMACOLOGY: RECENT ADVANCES

Session Chair: Dimosthenis Tsapekos

Prefrontal-Limbic Interactions and Antidepressant Mechanisms - Laith Alexander

Neural Signatures of Emotional Biases in Major Depressive Disorder and Their Potential Clinical Application - Diede Fennema

The Association Between Cognitive Performance and Sleep Spindles in Euthymic Individuals With Bipolar Disorder – Michail Kalfas

Cognitive and Treatment-Related Modulation of Human Hippocampal Neurogenesis in Mood Disorders - Megan Boreham

HALL 2

DOUBLE CONFERENCE-4

CLINICAL INSIGHTS FROM THE MASTERS PRACTICAL TIPS, GOLDEN RULES

Session Chairs: Eyüp Sabri Ercan - Mücahit Öztürk

HALL 3

PLENARY LECTURE-15

Session Chairs: Ali Saffet Gönül - Mesut Yıldız

New Approaches to Treatment Resistance in Psychotic Disorders - Dost Öngür

HALL 4

SYMPOSIA ORAL SESSION-40

RESISTANCE TO SUBSTANCE USE DISORDER TREATMENT; MANAGING COMORBIDITIES

Session Chair: Damla İşman Haznedaroğlu

Overcoming Resistance to Substance Use Disorder Treatment - Hüseyin Ozan Torun

The Relationship Between Substance Use Disorder, ADHD, and Bipolar Disorder - Furkan Yazıcı

Recovery of Substance Induced Psychosis: a Machine Learning Model from a Multicenter Study - Umut Kırlı

HALL 5

SYMPOSIA ORAL SESSION-61
BLIND SPOTS IN FUNCTIONING AND TREATMENT RESISTANCE IN ADHD: NEURO-MOTIVATION, THE ANXIETY–PERFORMANCE CYCLE, TEMPERAMENT AND UNDER-DISCUSSED CLINICAL PHENOMENA
Session Chair: İbrahim Gündoğmuş
A Neuro-Motivational Perspective in ADHD: What Do Reward Circuits Tell Us? Lack of Motivation or Altered Reward Sensitivity? - Kübra Özcan Çetin
The Performance Paradox in ADHD: How the Motivation–Anxiety Cycle Sabotages Treatment and How CBT Helps - Onur Cem Demir
Temperament Profiles in ADHD: Individual Differences Shaping Functioning and the Course of Treatment - Mehmet Rıdvan Varlı
Under-Discussed Clinical Phenomena in ADHD: Time Blindness, Hyperfocus, Social Anxiety, Masking, Rumination – Buket Kuruçay Yazar

HALL 6

SYMPOSIA ORAL SESSION-42
SIDE EFFECTS AND MANAGEMENT OF CLOZAPINE, THE REVOLUTIONARY DRUG IN PSYCHIATRY
Session Chair: Ayşegül Taşdelen Kul
Agranulocytosis; Is the Risk Really Great? - Cansu Ünsal Mavi
Hypersalivation and Constipation; GI Side Effects - Esra Yalım
Sedation and Seizures; What Should We Do? - Beyza Erdoğan Aktürk
Myocarditis; The Importance of Screening - Merve Işıklı

HALL 7

SYMPOSIA ORAL SESSION-43
HEART RATE VARIABILITY AND CHILD PSYCHIATRY: CLINICAL AND NEUROBIOLOGICAL IMPLICATIONS
Session Chair: Ümmügülsüm Gündoğdu
Foundations and Physiology of Heart Rate Variability - Ümmügülsüm Gündoğdu
Heart Rate Variability and Psychiatric Disorders: Diagnostic Categories and Clinical Correlates of HRV - Elif Naz Bedir
Clinical Applications and Interventions of Heart Rate Variability in Child and Adolescent Psychiatry-Ece Balcı
Use of Propranolol in Non-Akathisic Pediatric Psychiatric Patients: A Retrospective Evaluation-Zeynep Çelik

HALL 8

SYMPOSIA ORAL SESSION-44
AI-AUGMENTED PSYCHIATRY IN TREATMENT-RESISTANT CASES: FROM PERSONALIZED PHARMACOLOGY TO INTEGRATED DIGITAL ECOSYSTEMS
Session Chair: Burçin Özlem Ateş
AI-Assisted Psychopharmacology: Personalized Medication Selection in Treatment-Resistant Cases - Sarper İçen
Enhancing the Therapeutic Process Through Chatbots and AI Assistants - Nagihan Özer
AI-Driven Self-Management and Monitoring Systems in Daily Life - Şefika Nurhüda Karaca Cengiz
An Integrated AI Ecosystem for Treatment-Resistant Cases: Collaboration Between Clinicians, Patients, and Technology - Yusuf Selman Çelik

17:00 – 17:30 COFFEE BREAK

17:30 – 19:00

HALL 1

PLENARY LECTURE-16
Session Chairs: Ayşegül Yıldız - Ayşe Kurtulmuş Çalış
Beyond the Scale: Neuropsychiatric Weight of Obesity in Severe Mental Illness - Tomas Hajek

HALL 2

MEET THE EXPERT-6
MEET THE EXPERT
Session Chair: Ayşe Sakallı Kani
Treating Perinatal Mental Disorders: Lessons from the Development of an International Clinical Practice Guideline - Ryan J. Van Lieshout

HALL 3

SYMPOSIA ORAL SESSION-45

STRESS AND NEUROPSYCHIATRIC DISORDERS: FROM MOLECULAR MECHANISMS TO TRANSLATIONAL THERAPEUTICS

Session Chairs: Xingshun Xu - Xinmin Li

The Role of GluN3 Subunit in Dysregulation of Stress Response - Weiwen Wang

Lateral Habenular Tet2 Mediates Social Deficits - Xingyun Xu

HALL 4

SYMPOSIA ORAL SESSION-46

INTEGRATED PERSPECTIVES ON MILITARY PSYCHIATRY: FROM CLINICAL PRACTICE TO OPERATIONAL READINESS

Session Chairs: Mesut Çetin - Ayhan Güler

Selection, Screening, and Psychological Readiness - Onat Yılmaz

Clinical Presentations and Diagnostic Challenges (PTSD and Adjustment Disorders.) - Özgür Maden

Clinical Presentations and Diagnostic Challenges (Psychosis, Bipolar Disorder, and Malingering) - Alparslan Asil Budaklı

Administrative and Operational Applications (Medical Board Procedures, Suicide Prevention, and Fitness for Weapon Possession.) - Hasan Toğul

HALL 5

SYMPOSIA ORAL SESSION-47

ULTRA-HIGH-RISK FOR PSYCHOSIS: EARLY DIAGNOSIS, INTERVENTION METHODS AND FUTURE PERSPECTIVES

Session Chair: Kamil Nahit Özmenler

Ultra-High Risk for Psychosis: Diagnostic Criteria and Clinical Assessment Methods - Kamil Nahit Özmenler

Neurobiological and Genetic Underpinnings of Ultra-High Risk for Psychosis - Zehra Uçar Hasanlı

Current Pharmacological Treatment Approaches in Ultra-High Risk for Psychosis - Hatice Ayça Kaloğlu

Psychosocial Interventions for Ultra-High Risk for Psychosis: Early Intervention Strategies, Psychotherapy, and Community-Based Models - Ezgi Özserezli

HALL 6

SYMPOSIA ORAL SESSION-48

THE OVERLOOKED PSYCHODYNAMIC DIMENSION OF ATTENTION-DEFICIT/HYPERACTIVITY DISORDER

Session Chair: Erol Göka

From Neurobiological Foundations to Psychodynamic Patterns in Attention-Deficit/Hyperactivity Disorder - Işık Batuhan Çakmak

Psychodynamic Defense Mechanisms in Attention-Deficit/Hyperactivity Disorder - Rabia Nazik Ekinci

HALL 7

SYMPOSIA ORAL SESSION-49

UNDERSTANDING JUVENILE OFFENDERS: CONCEPTUAL, DEVELOPMENTAL, NEUROBIOLOGICAL, AND FORENSIC PSYCHIATRIC PERSPECTIVES

Session Chairs: Selma Tural Hesapçıoğlu - Alperen Bıkmazer

Juvenile Offenders: From Concept to Reality — A Global and Turkish Perspective - Aybala Nur Balık

Neurobiological Foundations of Antisocial Behavior in Juvenile Offenders - Büşra Yıldız Bayındır

Criminal Responsibility and Forensic Psychiatric Evaluation in Juvenile Offenders - Neşe Kavruk Erdim

Psychopharmacological Management of Impulsivity, Aggression, and Comorbidities in Juvenile Offenders: Evidence-Based Approaches - Melik Yiğit Bayındır

HALL 8

MEET THE EXPERT-7

ASPERGER SYNDROME IN ADULTHOOD: DELAYED, DISMISSED AND OVERLOOKED CASES – IS REMEDIATION STILL POSSIBLE?

Session Chair: Aynur Görmez

Asperger Syndrome in Adulthood: Delayed, Dismissed, and Overlooked Cases – Is Remediation Still Possible? - Aynur Görmez

19:00 – 20:30

HALL 1

PLENARY LECTURE-17
Session Chair: Ayşe Kurtulmuş Çalış
Psychiatry Education - Yanbo Zhang

HALL 2

PLENARY LECTURE-18
Session Chairs: Andrew Greenshaw - Mesut Çetin
Repurposing Glucagon-receptor Peptide -1 Agonist in the Prevention and treatment of Mental Disorders - Roger McIntyre

HALL 3

SYMPOSIA ORAL SESSION-50
CLINICAL APPROACH TO PATIENTS WITH SLEEP COMPLAINTS IN OUTPATIENT SETTINGS: PRACTICAL CONSIDERATIONS
Session Chair: Yavuz Selvi
The Tired Patient: Understanding and Differentiating Sleep Complaints in Psychiatry Clinic-Ekrem Furkan Uçak
Effective Assessment Sleep Complaints under Time Constraints: Selection and Application of Clinical Scales - Samet Kaya
Exploring Sleep Problems From A Psychiatrist's Perspective: What Do Sleep Complaints Reveal About Underlying Psychiatric Disorders? - Bilal Sağlıyan

HALL 4

SYMPOSIA ORAL SESSION-51
WHAT IS THE FUTURE OF RESEARCH ON HIGH-RISK PSYCHOSIS?
Session Chair: Aslı Enzel Koç
Is Early Diagnosis Possible with Digital Health Technologies in Individuals at Clinically High Risk of Developing Psychosis? - Sema Çağal
Is Early Diagnosis Possible with Digital Health Technologies in Individuals at Clinically High Risk of Developing Psychosis? - Turceun İleri Akdoğan
What is the Prevalence and Impact of Comorbid Mental Disorders in Individuals at Clinically High Risk for Psychosis? - Aslı Enzel Koç
The Challenge of Treating the Clinically High-Risk Group for Psychosis - Şule Gül Uysal

HALL 5

SYMPOSIA ORAL SESSION-52
RECONSIDERATION OF DIAGNOSIS IN TREATMENT RESISTANCE
Session Chair: Ayşe Nur İnci Kenar
Do We Call ADHD Bipolar Disorder? - Ayşe Nur İnci Kenar
Do We Call Adults with Autism Psychosis? - Ayşe Döndü
What Does the Current Literature Say About the Diagnostic Confusion? - Selin Balki Tekin

HALL 6

ORAL PRESENTATIONS SESSION-10
Session Chair: Gökhan Ünal
Lamotrigine Co-Administration May Reduce Effectiveness of Intravenous Ketamine in the Treatment of Major Depressive Episodes - Irem Hacısalihoglu Aydın
Clinical and Inflammatory Determinants of Length of Hospital Stay in Patients with Bipolar Disorder Manic Episodes – Pelin Göksel
Uncoupling Physiological Resistance from Chronicity: Longitudinal Phenotyping of Seizure Efficiency in Electroconvulsive Therapy - Yusuf Çiçek
Symptom Predominance and Duration of Untreated Psychosis in First-Episode Schizophrenia: Retrospective Analysis of an Inpatient Sample - İbrahim Akbaş
Inflammatory and Lipid-Related Biomarkers in First-Episode Schizophrenia and Methamphetamine-Induced First Episode Psychosis - İrem Bostan Özgündüz
Use of Propranolol in Non-Akathisic Pediatric Psychiatric Patients: A Retrospective Evaluation-Zeynep Çelik
The Evaluation of Galectin-1, Galectin-3, Galectin-4, IL-33 and sST2 Levels in First Episode Psychosis and in Schizophrenia - Hüseyin Uçar

Clinical Display and Comorbid Diseases of the Posttraumatic Embitterment Disorder in Anxiety Outpatient Clinic - Bahar Arslan

Rapid Antidepressant and Anti-Suicidal Effects of Ketamine Infusion in Unipolar and Bipolar Depression: Real-World Clinical Outcomes - Mehmet Güneş

Early symptom onset and clinical course in Pediatric Obsessive-Compulsive Disorder: Gender differences and severity predictors - Ali İşitmez

Examining the relationship between parental attitudes of individuals who became parents through assisted reproductive technologies and their children's self-perception, anxiety, and depressive symptoms - Pınar Coşkun

HALL 7

SYMPOSIA ORAL SESSION-54

ATTENTION DEFICIT HYPERACTIVITY DISORDER AND EMOTION REGULATION: DIAGNOSTIC STATUS, TREATMENT STRATEGIES, AND FUNCTIONAL OUTCOMES

Session Chairs: Abdullah Bozkurt - Saliha Baykal

Should Emotion Regulation Difficulties Be Included Among the Diagnostic Criteria for Attention Deficit Hyperactivity Disorder? - Saliha Baykal

Psychopharmacological Treatment of Emotion Regulation Difficulties in Attention Deficit Hyperactivity Disorder – Abdullah Bozkurt

Psychotherapeutic Interventions for Emotion Regulation Difficulties in Attention Deficit Hyperactivity Disorder - Gökçen Güven

The Impact of Emotion Regulation Skills on Parenting in Adults with Attention Deficit Hyperactivity Disorder - Hazan Tomar Bozkurt

Emotion Regulation Difficulties in Adult Attention Deficit Hyperactivity Disorder: A Mediating Mechanism of Functional Impairment? - Özge Akyürek Bolat

HALL 8

COURSE-6

HIDDEN DETERMINANTS OF TREATMENT RESISTANCE: COUPLE DYNAMICS AND THERAPEUTIC DEADLOCKS

Treatment Resistance or Relationship Dynamics? Clinical Differentiation - Rabia Nazik Ekinci

Working with Treatment Resistance in Couple Interviews and Couple Therapy - Merve Yiğit Başkan

21:30 – 22:00 AWARD CEREMONY

18 April 2026 Saturday

09:30 – 11:00

HALL 1

Session Chairs: Alperen Bıkmazer - Hicran Doğru

Towards Psychiatric Utility of Neuroimaging: Still a Long Way to Go - Francisco Xavier Castellanos

HALL 2

SYMPOSIA ORAL SESSION-55

MEANING LIFE IN PSYCHIATRY

Session Chair: Berhan Akdağ

Meaning in Life or Meaning of Life? - Berhan Akdağ

Purpose in Life and Mental Health - Fatma Yıldırım

Life Coherence and Mental Health - Sema Öznur Çeltik

Existential Significance and Mental Health - Tuğçe Cansu Şahin

HALL 3

SYMPOSIA ORAL SESSION-64

COMPLEX CLINICAL PRESENTATIONS IN THE AUTISM SPECTRUM – RISK FACTORS AND PROTECTIVE INTERVENTIONS

Session Chair: Halime Tuna Çak Esen

Psychotic Symptoms and Autism Spectrum: Risk and Protective Factors - Melike Karaçam Doğan

Trauma and Autism Spectrum: Risk Factors and Protective Interventions - Ecem Selin Akbaş Aliyev

Catatonia and Autism Spectrum: Risk Factors and Early Intervention - Makbule Esen Öksüzoğlu

HALL 4

SYMPOSIA ORAL SESSION-56
NEW PERSPECTIVES IN SEX THERAPY: OVERCOMING RESISTANCE TO TREATMENT WITH UNDEREXPLORED TOPICS AND INNOVATIVE TECHNIQUES
Session Chair: Yasir Şafak
Theoretical and clinical perspectives on resistance in the therapeutic process - Hatice Ayça Kaloğlu
Strategies to overcome resistance: creative and innovative interventions in sex therapy - Başak Şahin
Employing lesser-known techniques to overcome treatment resistance - Yasir Şafak

HALL 5

SYMPOSIA ORAL SESSION-57
CHALLENGES IN WOMEN'S MENTAL HEALTH
Session Chair: Filiz Kulacaoğlu
Monthly Recurring Cycle: Premenstrual Dysphoric Disorder - Sena Ece Ilgın
The Threshold Period in Bipolar Disorder: Pregnancy and the Postpartum Period - Aslı Ceren Hınç

HALL 6

SYMPOSIA ORAL SESSION-58
GRAY ZONES IN CHILD AND ADOLESCENT PSYCHIATRY: BLURRING OF DIAGNOSTIC BOUNDARIES
Session Chair: Ayşegül Tuğba Hıra Selen
Executive Dysfunction and Emotional Dysregulation: A Holistic Approach to Differential Diagnosis - Ayşegül Tuğba Hıra Selen
Withdrawal Symptoms: Psychotic Prodrome, Depression, or Anxiety? - Müge Mutlu
Aggression and Impulsivity: Bipolar Spectrum, ADHD, or Disruptive Mood Dysregulation Disorder? - İrem Cihanyurdu Erdem
Obsessive and Repetitive Behaviors: Obsessive–Compulsive Disorder or Autism Spectrum Disorder? - Melike Uysal

HALL 7

COURSE-7
PSYCHIATRIC EVALUATION, INITIAL INTERVIEW AND INTERDISCIPLINARY APPROACH IN CHILDREN AND ADOLESCENTS
Session Chair: Şaziye Senem Başgül
Psychiatric Evaluation, Initial Interview and Interdisciplinary Approach in Children and Adolescents - Şaziye Senem Başgül

HALL 8

SYMPOSIA ORAL SESSION-59
DISRUPTIONS OF THE BRAIN'S INTERNAL CLOCK: TIME PERCEPTION ALTERATIONS IN ADHD, DEPRESSION, PSYCHOSIS AND ADDICTIVE DISORDERS
Session Chair: Rabia Nazik Ekinci
Foundations of Time Perception and the Concept of Time Blindness in ADHD - Rabia Nazik Ekinci
The Flow and Freeze of Time in Mood Disorders - Turceun İleri Akdoğan
Temporal Disintegration in Psychotic Disorders - Bedirhan Şenol
Digital Addictions and the Distortion of Time in the Modern Age - Mert Şenel

11:00 – 11:15 COFFEE BREAK

11:15 – 12:45

HALL 1

SYMPOSIA ORAL SESSION-60
SELF-COMPASSION IN CLINICAL AND TRANSDIAGNOSTIC CONTEXTS: ADHD, MOOD DISORDERS, TRAUMA, OTHER PSYCHOPATHOLOGIES AND PSYCHOLOGICAL FLEXIBILITY
Session Chair: Aliye Tuğba Bahadır
Kristin Neff's Self-Compassion Model and Self-Compassion in Attention-Deficit/Hyperactivity Disorder - Çağla Hacıalioğlu
Self-Compassion and Its Emotion-Regulatory Role in Mood, Anxiety, and Eating Disorders - Beyza Kuyumcu
Self-Compassion and Trauma: The Role of Self-Compassion in the Healing Process - İrem Cihanyurdu Erdem
Self-Compassion as a Transdiagnostic Construct: Shared Mechanisms and Psychological Flexibility - Aliye Tuğba Bahadır

HALL 5

SYMPOSIA ORAL SESSION-63

GROWING UP IN THE DIGITAL WORLD: PARENTING, PRIVACY AND SOCIAL MEDIA BEHAVIORS IN CHILDREN AND ADOLESCANT AND IMPACT ON ADULT PSYCHIATRY

Session Chair: Tuba Mutluer

Social Media Use in Children and Adolescant: Emotional, Behavioral and Clinical Reflections - Ayşegül Duman Kurt

Callous-Unemotional Traits and Impulsivity in Digital Interaction: Risky Challenges, Cyberbullying, and Clinical Findings – Hilal Aydemir

Digital Parenting and Sharenting: A New Space Between Childhood and Parenting - Hatice Gülşen

The İmpact Of Social Media Use in Childhood and Adolescense on Adult Psychiatry - Ayşe Ergüner Aral

HALL 8

SYMPOSIA ORAL SESSION-66

THE PRESENT AND FUTURE OF ECT IN THE AGE OF ARTIFICIAL INTELLIGENCE: GUIDELINE UPDATES, SIDE-EFFECT MANAGEMENT, PERSONALIZED APPLICATIONS

Session Chair: Rabia Nazik Ekinci

History and Neurobiological Mechanisms of ECT - Işık Batuhan Çakmak

The Role of ECT in Current Clinical Guidelines and Treatment-Resistant Conditions - Ezgi Cellat

Current ECT Application Protocols and Management of Side Effects - Ayşe Gülden Kaya

Future Directions and Innovations in ECT - Ahmet Çöpür

12:45 – 13:00 CLOSING SESSION

İçindekiler

Scientific Program **3**

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Outstanding Research Award Nominees Brief Reports

17th International Congress on Psychopharmacology & Child and Adolescent Psychopharmacology / Psychotherapy

0096 - Social and Psychiatric Risk Profiles of Juveniles Involved in Sexual Offenses: Analysis of 82 Forensic Cases

Neşe Kavruk Erdim

Council of Forensic Medicine, istanbul, Turkey

Introduction

Juvenile sexual offending represents an important forensic and public health concern. Research suggests that such behaviors arise from complex interactions between developmental vulnerabilities, family environment, and broader social contexts^ Meta- analytic studies indicate that adolescents who commit sexual offenses constitute a heterogeneous group with diverse psychosocial and developmental characteristics.[2]

Previous research has linked adolescent sexual offending to multiple risk domains, including antisocial behavior, family dysfunction, exposure to violence, and developmental vulnerabilities.[1,3] In addition, adolescents involved in the juvenile justice system frequently present with elevated rates of psychiatric disorders and behavioral problems, particularly externalizing conditions such as attention-deficit/hyperactivity disorder (ADHD), conduct disorder, and substance use disorders.[4,5]

Despite the growing international literature, empirical data from non-Western forensic contexts remain limited. This study examined the sociodemographic, psychosocial, and psychiatric characteristics of juveniles referred for forensic psychiatric evaluation for sexual offenses in Turkey and explored factors associated with abuse severity and recurrence.

Methods

This retrospective cross-sectional study included juveniles referred to the Sixth Specialization Board of the Council of Forensic Medicine, istanbul, for forensic psychiatric evaluation following allegations of sexual offenses between January 2022 and July 2023.

The sample consisted of 82 male juveniles. Data were obtained from forensic case files, psychiatric evaluation reports, and judicial records using a structured extraction form.

Variables included sociodemographic characteristics, family background, psychiatric history, substance use, and offense-related factors. Offense-related variables included victim characteristics, victim-offender relationship, abuse context (intrafamilial vs. extrafamilial), penetration, abuse repetition, and additional offending.

Psychiatric diagnoses were based on clinical assessments conducted during forensic evaluations. Statistical analyses were performed using IBM SPSS Statistics (version 26). Categorical variables were compared using chi-square or Fisher's exact tests, and odds ratios (OR) were calculated where appropriate. Statistical significance was set at $p < 0.05$. Ethics approval was obtained from the Education and Scientific Research Commission of the Council of Forensic Medicine (October 2021). All data were anonymized prior to analysis.

Results

The mean age of the juveniles was 15.6 ± 1.2 years, and all participants were male. Most were enrolled in high school (85.4%), and 14.6% attended middle school. School dropout was reported in 21 juveniles (25.6%), and parental separation or disrupted family structure was present in 30.5% of the sample.

Psychiatric diagnoses were identified in eight juveniles (9.8%), most commonly attention- deficit/hyperactivity disorder (ADHD; n = 5), followed by ADHD with conduct disorder (n =2) and obsessive-compulsive disorder (n = 1). Behavioral risk factors included smoking (30.5%) and alcohol use (4.9%).

Additional offending was documented in 12 juveniles (14.6%), including repeated sexual abuse (n = 3) and non-sexual offenses (n = 9), such as theft, assault, and attempted homicide. Prior detention was recorded in six juveniles (7.3%), and a family history of crime was reported in 24.4% of cases (Table 1).

Table 1. Demographic and Psychosocial Characteristics of the Study Sample (N = 82)

Variable	n (%) / Mean ± SD
Age (years)	15.6 ± 1.2
Male sex	82 (100)
Education level	
Middle school	12 (14.6)
High school	70 (85.4)
School dropout	21 (25.6)
Parental separation or disrupted familey structure	25 (30.5)
Any psychiatric diagnosis	8 (9.8)
Smoking	25 (30.5)
Alcohol use	4 (4.9)
Additional offending	12 (14.6)
Prior detention	6 (7.3)
Family history of crime	20 (24.4)

Note. Values are presented as n (%) unless otherwgse specgfged. Age gs presented as mean ± SD. Addgtgonal offendgng gncludes repeated sexual abuse (n = 3) and non-sexual offenses (n = 9).

The characteristics of sexual abuse cases are summarized in Table 2. Victims were predominantly female (56.1%), followed by males (42.7%); one case involved victims of both sexes (1.2%). Abuse occurred more frequently in extrafamilial contexts (62.2%) than in intrafamilial contexts (37.8%). The most common victim-offender relationship was acquaintances or neighbors (42.7%), followed by siblings or cousins (20.7%) and other relatives (17.1%). Penetration was reported in 72.0% of cases, and repeated abuse occurred in 58.5% of the sample. Anal penetration was the most frequently reported form of penetration (45.1%), followed by vaginal (24.4%), oral (14.6%), and digital penetration (3.7%). Multiple acts could be reported within a single case.

Table 2. Characteristics of Sexual Abuse Cases (N = 82)

Variable	n (%)
Victim sex	
Female	46 (56.1)
Male	35 (42.7)
Both sexes	1 (1.2)
Abuse context	
Intrafamilial	31 (37.8)
Extrafamilial	51 (62.2)
Victim-offender relationship	
Sibling / cousin	17 (20.7)
Other relative	14 (17.1)
Acquaintance / neighbor	35 (42.7)
Friend / partner	14 (17.1)
Stranger	2 (2.4)
Penetration	
Yes	59 (72.0)
No	23 (28.0)
Repeated abuse	
Single episode	34 (41.5)
>2 episodes	48 (58.5)

Note. Penetration refers to oral, vaginal, anal, or digital acts. Repeated abuse was defined as two or more abuse episodes.

Several contextual and familial factors were significantly associated with abuse severity and recurrence **(Table 3).** Penetration was significantly more common among male victims than among female victims (88.6% vs. 60.9%; OR = 4.98, p = .006). Juveniles with a family history of crime were significantly more likely to engage in additional offending (25.0% vs. 6.5%; OR = 4.83, p = .035). Repeated abuse was significantly more frequent in intrafamilial contexts than in extrafamilial contexts (74.2% vs. 49.0%; OR = 2.99, p = .037).

Table 3. Factors Associated With Abuse Severity and Recurrence (N = 82)

Variable	Category	n (%)	OR	p-value
Victim sex	Female (n = 46) Male (n = 35)	Penetration, n (%): 28 (60.9) Penetration, n (%): 31 (88.6)	4.98	.006
Family history of crime	Absent (n = 62)	Additional offending, n (%): 4 (6.5)		
	Present (n = 20)	Additional offending, n (%): 5 (25.0)	4.83	.035
Abuse context	Extrafamilial (n = 51)	Repeated abuse, n (%): 24 (49.0)		
	Intrafamilial (n = 31)	Repeated abuse, n (%): 23 (74.2)	2.99	.037

Note. p values were calculated using Pearson $\%^2$ or Fisher's exact tests as appropriate. OR indicates odds ratio. Penetration refers to anal, vaginal, oral, or digital penetration. Additional offending refers to offenses committed by the juvenile other than the index sexual abuse offense. Repeated abuse was defined as >2 episodes of abuse.

Discussion

This study provides a forensic psychiatric analysis of juveniles involved in sexual offenses in a non-Western context, examining the demographic, contextual, and psychiatric characteristics of 82 cases referred for evaluation in Turkey.

The demographic structure of the sample is consistent with previous research. All offenders were male adolescents with a mean age of approximately 15-16 years, corresponding to the mid-adolescent developmental period commonly reported in studies of juvenile sexual offending.[2] Developmental models suggest that impulsivity, immature self-regulation, and peer influences during adolescence may contribute to sexually inappropriate behaviors.[1]

Educational disruption was also notable in the sample, with approximately one-quarter of the juveniles having dropped out of school. School problems and academic disengagement have been identified as important risk domains associated with persistent delinquent behavior among adolescents.[3]

The findings highlight the importance of contextual and relational factors in juvenile sexual offenses. Most victims were known to the offender, and intrafamilial abuse accounted for over one-third of cases. This pattern is consistent with previous research indicating that child sexual abuse is often perpetrated by individuals known to the victim, including family members and acquaintances.[6] Such findings suggest that juvenile sexual offending frequently occurs within familiar social environments rather than involving strangers.[2,6] The higher rate of repeated abuse in intrafamilial contexts may partly reflect the close relational context of these offenses, where continued contact between the offender and the victim may increase the likelihood of repeated incidents.

Another notable finding was the higher rate of penetration among male victims. Although juvenile sexual offenses often involve non-penetrative behaviors, previous research indicates that adolescent sexual offenders constitute a heterogeneous group and that patterns of abuse, including the type of sexual act and the characteristics of victims, may vary across cases.[2,6] These differences may be related to variations in victim vulnerability, power dynamics, or contextual factors within close social or familial relationships.

Juveniles with a family history of crime were significantly more likely to engage in additional offending. This finding is consistent with meta-analytic evidence showing that family environment—including parental criminality,

poor supervision, family dysfunction, and exposure to violence—is an important risk domain for juvenile delinquency.[3] Recent research also indicates that family and parenting risk factors such as inadequate supervision, inconsistent discipline, and family conflict are closely interconnected and may jointly contribute to delinquent behavior among adolescents.[7]

Psychiatric diagnoses were identified in a minority of cases in this study, with ADHD being the most frequently recorded diagnosis. Previous studies have shown that externalizing

disorders, including ADHD and conduct disorder, are commonly observed among adolescents involved in the juvenile justice system.[4] The relatively low rate of psychiatric diagnoses may reflect reliance on forensic case records rather than structured diagnostic interviews.

The present findings also support the view that adolescent sexual offending often overlaps with broader patterns of delinquent behavior rather than representing a distinct form of criminal activity. Earlier research has shown that many juveniles who commit sexual offenses also engage in other antisocial behaviors.[8] This interpretation is consistent with evidence indicating high rates of psychiatric and behavioral disorders among adolescents involved in the juvenile justice system.[5] Although the present study examined repeated abuse within the index offense rather than long-term recidivism, longitudinal and meta-analytic studies suggest that sexual recidivism among juveniles is relatively low, with average rates around 5%.[9]

These findings contribute to the limited empirical literature on juvenile sexual offending in non-Western forensic contexts, where most existing research has been conducted in North America and Western Europe.

Several limitations should be considered. First, the retrospective design limits the ability to establish causal relationships between risk factors and offending behavior. Second, the data were derived from forensic case files and judicial records, which may not capture all psychosocial variables relevant to adolescent development. Third, the sample included only juveniles referred for forensic psychiatric evaluation, which may limit generalizability. Finally, all offenders in the sample were male, limiting the applicability of the findings to female juveniles involved in sexual offenses.

Despite these limitations, this study provides insight into the social and psychiatric characteristics of juveniles involved in sexual offenses within a forensic context. These results support risk-informed forensic psychiatric assessments and underscore the importance of incorporating developmental, familial, and contextual factors—particularly family-based risk assessment—into evaluations and preventive intervention strategies for adolescents at risk of repeated or more severe offending.

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0102 - A Case of Refeeding Syndrome (RFS) Developing in Result of Successful Treatment of Catatonia in Major Depressive Disorder: The Importance of Predicting RFS as a Probable Complication

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Introduction

Catatonia is a complex neuropsychiatric syndrome characterized by a distinct cluster of psychomotor abnormalities, including wide variety of symptoms. Although historically viewed as a subtype of schizophrenia, one of the current diagnostic frameworks such as DSM-5-TR recognize catatonia as an independent syndrome that may occur in the context of affective, psychotic, or medical illnesses (1).

The estimated prevalence of catatonia ranges from 9% to 30% among psychiatric inpatients, particularly those with major depressive disorder and bipolar affective disorders. In patients with prolonged nutritional restriction due to catatonia, the reintroduction of feeding may precipitate refeeding syndrome (RFS) — a potentially fatal condition characterized by rapid shifts in electrolytes, especially hypophosphatemia, hypokalemia, and hypomagnesemia.

Here, we present the case of a 54-year-old woman with major depressive disorder and catatonia, who experienced a full recovery after a combination of pharmacotherapy and electroconvulsive therapy (ECT), but subsequently developed refeeding syndrome upon relatively fast and robust resumption of oral nutrition. This case underscores the need for consideration of refeeding syndrome as a clinical entity during recovery of catatonic patients who had experienced prolonged fasting prior to effective treatment.

Case

A 54-year-old woman was brought to the emergency department with severe withdrawal characterized by lying in bed, not eating, not moving, and staring at the ceiling. Her relatives reported that she had developed a gradually worsening depressive state after the death of her husband one year earlier, marked by depressed mood, anhedonia, social withdrawal, and psychomotor retardation. She had previously been hospitalized following a suicide attempt by self-suffocation and received electroconvulsive therapy (ECT) and pharmacotherapy without remission.

On admission, she was cachectic and severely dehydrated with a body weight of 33 kg (BMI: 12.12). Mental state examination revealed mutism, verbal negativism ,rigidity, catalepsy, and posturing, consistent with catatonic stupor. Neurological and medical causes of catatonia were excluded through neuroimaging, EEG, and laboratory investigations.

The patient met DSM-5-TR criteria for catatonia associated with severe non-psychotic major depressive disorder with melancholic features. Initial treatment with alprazolam produced only transient improvement. ECT was initiated on day 5 and resulted in gradual clinical improvement, with resumption of oral intake.

However, 72 hours after refeeding, she developed hypophosphatemia, hypokalemia, and hypomagnesemia consistent with refeeding syndrome. Electrolyte replacement, thiamine supplementation, and gradual nutritional support were initiated. Laboratory parameters normalized within two weeks, and the patient was discharged with a weight of 38 kg (BMI: 16.0)



Figure 1. Clinical Timeline of Hospital Course

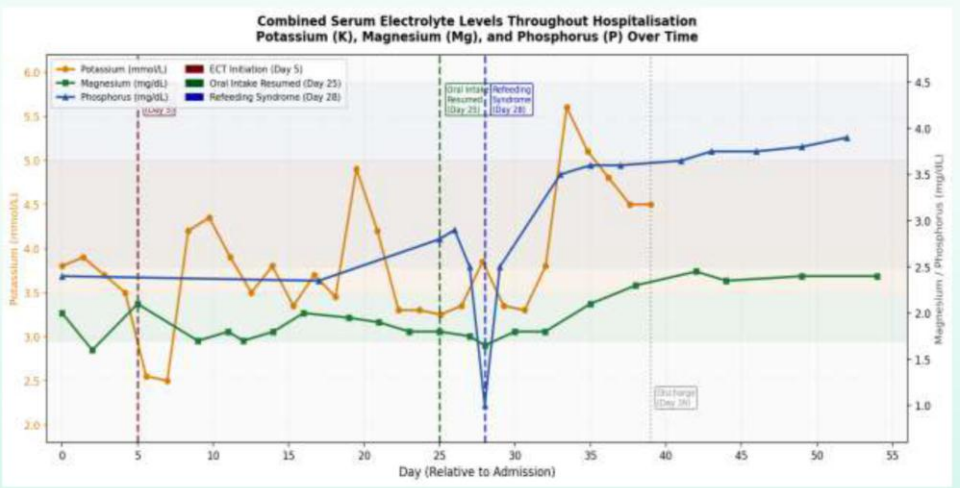
Conclusion

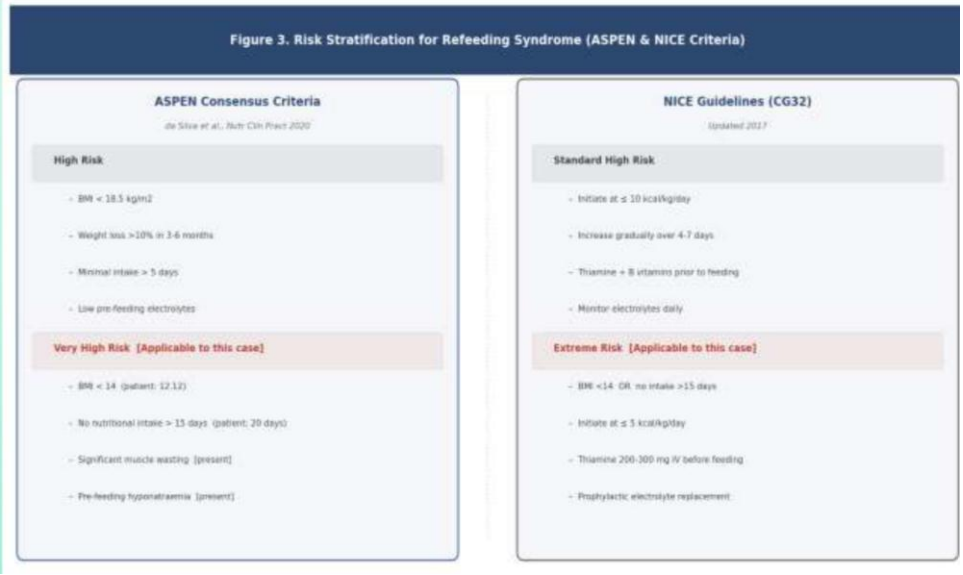
This case highlights a clinically significant overlap between psychiatric and metabolic pathology: catatonia secondary to major depressive disorder successfully treated with electroconvulsive therapy (ECT), followed by the development of refeeding syndrome (RFS). While RFS is well described in eating disorders such as Anorexia Nervosa, reports of its occurrence in patients with catatonia after recovery and reintroduction of nutrition are extremely limited.

RFS results from a metabolic shift from catabolism to anabolism after carbohydrate intake. Increased insulin secretion leads to intracellular shifts of phosphate, potassium, and magnesium, resulting in electrolyte disturbances that may cause severe complications including arrhythmias, seizures, heart failure, or respiratory failure. In this case, severe hypophosphatemia developed within 72 hours after refeeding, emphasizing the need for close biochemical monitoring in high-risk patients.

Guidelines such as those from American Society for Parenteral and Enteral Nutrition (ASPEN) and National Institute for Health and Care Excellence (NICE) recommend careful risk assessment, thiamine supplementation, and gradual caloric reintroduction in patients with prolonged malnutrition.

This case suggests that catatonia should also be considered a potential high-risk condition for RFS due to prolonged nutritional deprivation and abrupt refeeding after clinical recovery.





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0109 – Lamotrigine Co-Administration May Reduce Effectiveness of Intravenous Ketamine in the Treatment of Major Depressive Episodes

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Background: Intravenous (IV) ketamine has a therapeutic window. Thus, coadministration with a drug that may share a similar mechanism of action may alter the effective dose. Both ketamine and lamotrigine inhibit the N-methyl-D-aspartate (NMDA)- associated sodium channel in an activity-dependent manner so that co-administration of the two agents may alter the apparent effective dose of ketamine in treatment for depression. There are data suggesting that coadministration of ketamine and lamotrigine may reduce the effectiveness of the ketamine treatment. This was investigated in a new cohort of IV ketamine treated patients.

Methods: We retrospectively evaluated Montgomery Asperg Depression Rating Scale (MADRS) scores in patients receiving IV ketamine with (n = 7) and without (n = 49) coadministration with lamotrigine. The extent of improvement from baseline to 24 hours after the third round of IV ketamine was compared with an unpaired, two-tailed t-test.

Results: Patients receiving lamotrigine (baseline 28.3 ± SD 11.4; after 3rd treatment 18.3 ± 31.4; ns) did not improved to the same extent as patients not receiving lamotrigine (baseline 27.8 ± SD 6.2; after 3rd treatment 10.8 ± 9.0; $P < 0.0001$). The extent of improvement was non-significantly greater in patients not receiving concomitant lamotrigine (total improvement 16.9 ± 9.0 vs. 10.0 ± 1.38, $P = 0.07$, $t = 2.01$). Conclusions: While the data are limited by the small sample of lamotrigine -co-treated patients, they support the concept of a therapeutic window for ketamine, and possibly also for lamotrigine. Co-administration of the two medications should be avoided or minimized.

Key Words: Ketamine; Lamotrigine; Major Depression; Treatment response

Introduction

Major depressive disorder (MDD) is a common and frequently disabling disorder). Amine reuptake inhibiting antidepressants are the mainstay of treatment, but their effectiveness is often inadequate and emergence of treatment resistance to antidepressants is a significant problem. Intravenous (IV) ketamine has been found to mitigate treatment-resistant depression (TRD). Use of IV ketamine is limited for several reasons, including the fact that it is not approved by the United States Food and Drug Administration (FDA).

IV ketamine appears to have a therapeutic window so that after peak efficacy is achieved higher doses are less effective.[1] Peak utility occurs predominantly at a subanesthetic, sub- hallucinogenic dose. Because of this phenomenon, coadministration of medications that share a similar mechanism may result in reduced efficacy. This has already been demonstrated for coadministration of benzodiazepines in individuals receiving IV ketamine.[2] Since both ketamine and lamotrigine both inhibit the #-methyl-D-aspartate (NMDA)-associated sodium channel it

is important to determine if coadministration my reduce efficacy of one or both agents.[3] Previous reports have been mixed.

Methods

This was a post hoc, retrospective evaluation of clinical data of individuals participating in the Bio-K study. Bio-K was an open-label, non-randomized, single-arm, multi-site clinical trial, performed to identify putative blood-based biomarkers of clinical response to IV ketamine for adults (18-65 years of age) with TRD. It was conducted at four sites: Johns Hopkins Hospital, Mayo Clinic (Rochester, Minnesota), University of Michigan, and Pine Rest Christian Mental Health Services. The study was approved by the Institutional Review Board for each study site (IRB #16-009737). Full description and primary outcome results have been previously presented.[4] Briefly, subjects were included based on the following criteria: DSM-5 diagnoses of either major depressive disorder (MDD) or bipolar disorder (I or II), current depressive symptom severity at least 'moderately severe' (Patient Health Questionnaire-9 Item total score > 15), undergoing a current episode of treatment-resistant depression (defined as failure of at least 2 prior antidepressant or mood stabilizing medications within the current depressive episode) established by the Antidepressant Treatment History Form: Short-Form (ATHF-SF). Failed treatments were defined as at least 8 weeks of adequate-dose antidepressants or 6 sessions of electroconvulsive therapy (ECT).

Subjects were excluded if they had psychotic symptoms, active substance use disorder, cannabis use, or unstable medical conditions. Because of previous reports that concomitant benzodiazepines may interfere with ketamine response[2], patients who had ongoing prescription of > 4 mg lorazepam equivalents daily were excluded and any morning dose of benzodiazepine was held. Other prior psychotropic medications were continued during the study. The details of medications were previously presented in Singh et al. (2024).5 Cut point of >2mg lorazepam equivalents (total) daily, based preliminary reports that >3mg discriminated non-responders from responder.

Ketamine treatment occurred over 11 - 13 days with three infusions of IV ketamine at one to five-day intervals, dosed at 0.5 mg/kg, and using either 40- or 100-minute infusion time (determined by collaborative clinical decision making).[4]

Depressive symptoms were measured using the Montgomery Asberg Depression Rating Scale (MADRS) (Montgomery and Asberg 1979), administered at baseline (pre-treatment), and then thrice within each infusion session (pre-infusion, immediate post-infusion, and 24-hours post- infusion).

Results

Of the 102 subjects screened for Bio-K, 75 were enrolled, but one withdrew. Of the remaining 74 subjects, 18 were excluded from the current analysis because they were receiving a benzodiazepine. A total of 56 subjects were included in this analysis, seven with and 49 without coadministration of lamotrigine. Patients receiving lamotrigine did not improved to the same extent (baseline 28.3 ± SD 11.4; after 3rd treatment 18.3 ± 31.4; ns) as patients not receiving not receiving (baseline 27.8 ± SD 6.2; after 3rd treatment 10.8 ± 9.0; $P < 0.0001$) (Figure 1). However, due to the small sample size, the extent of improvement was non-significantly different two groups (total improvement 16.9 ± 9.0 vs. 10.0 ± 1.38, $P = 0.07$, $t = 2.01$). Limiting the dosage of coadministered benzodiazepine to < 2 mg of lorazepam equivalents appears to have mitigated the potential loss of efficacy of ketamine (Figure 1).

Discussion

Medications that inhibit glutamate nearly always have a therapeutic window. This is because their mechanism of action is dependent on selective inhibition of the inhibitory y-amino- butyric acid (GABA) interneurons.[6,7] The quantity of postsynaptic GABAergic interneurons circling back to inhibit the glutamatergic pyramidal cell greatly outnumbers the number of postsynaptic target neurons. Consequently, a relatively low dose of an inhibitor of the NMDA- associated sodium channel (whether it is ketamine or lamotrigine) will selectively inhibit the GABAergic interneurons, allowing the glutamateric pyramidal cell to be more active.[6,7] When a drug with a similar mechanism is used, the additive effect of the two drugs may exceed the therapeutic window. This is believed to be the underlying cause of the interfering effect of benzodiazepines[2], and may also occur when lamotrigine is added to dextromethorphan (both of which work by inhibition of the NMDA-associated sodium channel). It is proposed that when lamotrigine and ketamine are coadministered, the blockade of postsynaptic glutamate signal loses its selectivity and the treatment becomes less effective. The effect may be small enough that it cannot be adequately

captured by the seven patients receiving concomitant lamotrigine, but may have contributed to the non-significant difference in response (Figure 1).

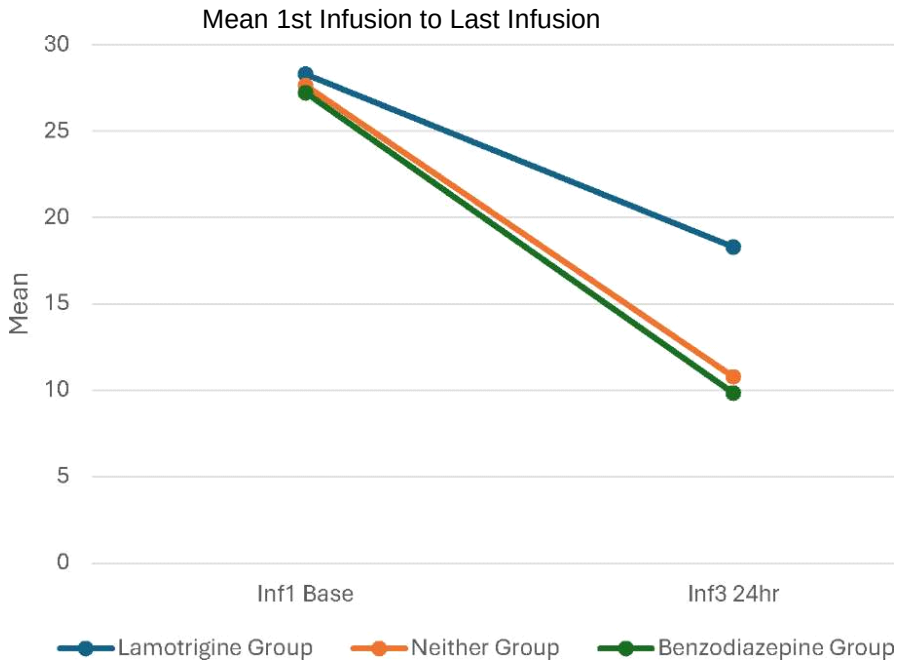


Figure 1. Difference between baseline and final evaluation in patients treated with IV ketamine (0.5 mg/kg) and cotreated with lamotrigine (LAM, n = 7), a benzodiazepine (BNZ, n = 18), and neither (n = 49).

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0110 - Somatization, Coping Styles, Anxiety, and Sleep Habits in Children and Adolescents with Alopecia Areata

Objective

Alopecia areata (AA) is a chronic inflammatory condition characterized by non-scarring hair loss, frequently emerging during childhood and adolescence. Although it does not result in significant physical morbidity, its psychosocial burden can be substantial. Previous studies have suggested associations between AA and psychiatric comorbidities; however, data regarding somatization, coping styles, and sleep habits in pediatric populations remain limited (1).

This study aimed to evaluate somatization symptoms, coping styles, anxiety levels, and sleep habits in children and adolescents with AA compared with healthy controls, and to examine the relationships among these variables within the patient group.

Methods

This cross-sectional study included 80 children and adolescents aged 8-18 years, consisting of 40 patients diagnosed with alopecia areata and 40 healthy controls matched for age and sex. Participants in the AA group were recruited from the Dermatology Outpatient Clinic of Düzce University, while controls were selected from the general population. All participants had normal intellectual capacity and no chronic systemic illness.

Sociodemographic characteristics, including prenatal and perinatal factors, family structure, academic performance, and peer relationships, were collected using a structured form completed by both parents and participants.

Somatization symptoms were assessed using the Children's Somatization Inventory-24 (CSI- 24). Coping styles were evaluated using the Coping Orientation to Problems Experienced (COPE) scale, which includes problem-focused and emotion-focused coping dimensions and their respective subscales. Sleep habits were assessed using the Children's Sleep Habits Questionnaire (CSHQ), which includes eight subdomains. Anxiety symptoms were evaluated using the Screen for Child Anxiety Related Disorders (SCARED), which measures panic disorder, generalized anxiety disorder, separation anxiety, social anxiety, and school refusal.

Disease severity in the AA group was assessed using the Severity of Alopecia Tool (SALT) score.

Statistical analyses were performed using SPSS version 22. Normality was evaluated using the Shapiro-Wilk test and skewness-kurtosis values. Between-group comparisons were conducted using independent samples t-tests, Mann-Whitney U tests, one-way ANOVA, and generalized linear models where appropriate. Associations between categorical variables were analyzed using chi-square tests with Monte Carlo simulation or Fisher's exact test. Correlations between continuous variables were evaluated using Pearson correlation coefficients. Reliability of the scales was assessed using Cronbach's alpha coefficients. A p value < 0.05 was considered statistically significant.

Ethical approval was obtained from the Düzce University Ethics Committee (Approval No: 2023/130), and written informed consent was obtained from the parents of all participants prior to enrollment.

Results

The study included 80 participants with a mean age of 12.5 ± 3.1 years. There were no statistically significant differences between the AA and control groups in terms of age or sex distribution ($p > 0.05$).

No significant differences were observed between groups regarding whether the pregnancy was planned, mode of delivery, breastfeeding duration, regular medication use, or presence of chronic illness ($p > 0.05$). However, birth complications, preterm delivery, and maternal smoking during pregnancy were significantly more frequent in the AA group ($p < 0.05$).

Significant differences were identified in psychosocial variables. Daily sibling conflict was reported more frequently in the AA group. Additionally, the AA group demonstrated poorer peer relationships and lower academic performance compared with controls ($p < 0.05$).

The mean CSI-24 somatization score was significantly higher in the AA group compared with controls (13.1 ± 8.8 vs. 6.7 ± 4.2, $p < 0.001$). Cronbach's alpha values for CSI-24 were 0.761 in the patient group and 0.722 in the control group.

Regarding coping styles, emotion-focused coping strategies were significantly more frequently used in the AA group ($p = 0.014$). Among the subscales, the helpless coping approach was significantly higher in the AA group ($p = 0.021$). No significant differences were observed between groups in problem-focused coping, self-confident, optimistic, submissive, or social support-seeking approaches ($p > 0.05$).

Sleep assessment revealed significantly higher total CSHQ scores in the AA group compared with controls ($p = 0.003$). Among subscales, bedtime resistance ($p = 0.012$), sleep anxiety ($p = 0.004$), and night wakings ($p = 0.001$) were significantly higher in the AA group. No statistically significant differences were found for other subscales. Cronbach's alpha values for the CSHQ were 0.515 in the patient group and 0.748 in the control group.

Anxiety assessment demonstrated significantly higher scores in the AA group for separation anxiety ($p = 0.004$), generalized anxiety disorder ($p = 0.004$), social anxiety disorder ($p < 0.001$), and total anxiety score ($p < 0.001$). No significant differences were found for panic disorder or school refusal subscales ($p > 0.05$).

Correlation analyses within the AA group showed that CSI-24 scores were positively correlated with panic disorder, generalized anxiety disorder, social anxiety disorder, school refusal, and total anxiety scores ($p < 0.05$), whereas no significant correlation was observed with separation anxiety ($p > 0.05$).

CSI-24 scores were also positively correlated with parasomnia scores ($p < 0.05$). No significant correlations were identified between CSI-24 and other sleep subscales.

Emotion-focused coping was positively correlated with panic disorder, generalized anxiety, social anxiety, school refusal, and total anxiety scores ($p < 0.05$). Problem-focused coping showed negative correlations with generalized anxiety and school refusal ($p < 0.05$).

The helpless coping approach demonstrated significant positive correlations with multiple anxiety domains and total anxiety scores ($p < 0.001$). Self-confident and optimistic coping approaches were negatively correlated with anxiety measures ($p < 0.05$).

CSI-24 scores were positively correlated with emotion-focused coping and its subcomponents, particularly helpless and submissive approaches, in the AA group ($p < 0.05$), while no significant associations were observed in the control group.

No significant associations were found between SALT scores and somatization, anxiety, coping styles, or sleep variables ($p > 0.05$).

Discussion

This study demonstrated that children and adolescents with alopecia areata exhibit significantly higher levels of somatization, anxiety, and sleep disturbances compared with healthy controls. In addition, maladaptive coping strategies, particularly emotion-focused and helpless approaches, were more frequently observed in the AA group.

The absence of a relationship between disease severity and psychological variables suggests that the psychological burden associated with AA may be independent of the extent of hair loss. This finding supports previous literature indicating that the visibility and social meaning of the condition, rather than clinical severity, may play a more critical role in psychological outcomes.

The higher prevalence of emotion-focused coping strategies in the AA group, together with their positive correlations with anxiety and somatization, suggests that these coping patterns may contribute to the persistence or exacerbation of psychological symptoms (2). In contrast, problem-focused coping strategies demonstrated negative associations with anxiety domains, indicating a potentially protective role (3).

Sleep disturbances, particularly bedtime resistance, sleep anxiety, and night wakings, were significantly more common in the AA group. The observed associations between sleep problems and anxiety symptoms further highlight the bidirectional relationship between sleep and emotional regulation in this population (4) .

The findings related to somatization are consistent with previous studies demonstrating that psychological distress in children often manifests through physical symptoms. The significant correlations between somatization, anxiety, and maladaptive coping strategies suggest a complex interaction in which emotional difficulties may be expressed somatically, particularly in individuals with limited adaptive coping skills.

From a clinical perspective, these findings emphasize the importance of comprehensive evaluation in children with AA, including not only dermatological assessment but also psychological and behavioral screening. Early identification of maladaptive coping styles and psychological symptoms may facilitate timely interventions and improve overall outcomes.

This study has several limitations. Its cross-sectional design precludes causal inferences, and the relatively small sample size may limit generalizability. Additionally, psychiatric symptoms were assessed using self-report measures rather than structured clinical interviews. Future studies with longitudinal designs and larger samples are needed to further elucidate these relationships.

Conclusion

Children and adolescents with alopecia areata exhibit increased somatization, anxiety, and sleep disturbances, along with a greater tendency to use emotion-focused coping strategies. These findings highlight the importance of integrating psychological assessment and intervention into the routine management of pediatric AA.

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0112 - Maintenance Treatment Profiles of Bipolar Disorder Patients Without Criminal Responsibility

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Introduction

Bipolar disorder is a chronic psychiatric condition characterized by recurrent manic and depressive episodes and requires long-term maintenance treatment to prevent relapses. The main goals of maintenance therapy are to prevent new episodes, reduce residual symptoms, preserve functioning, and improve quality of life. In clinical practice, mood stabilizers, second- generation antipsychotics, and combinations of these medications are frequently used for this purpose [1]. Current guidelines recommend that maintenance treatment in bipolar disorder be individualized and that treatment strategies be tailored according to the dominant pattern of mood episodes and clinical features of the illness [2,3].

In forensic psychiatry settings, the treatment of patients with bipolar disorder involves not only symptom control but also the maintenance of behavioral stability and the management of potential risks. Therefore, pharmacological treatment decisions may involve different dynamics than in general psychiatric practice. However, data on post-discharge maintenance treatment patterns among patients with bipolar disorder treated in high-security forensic psychiatric units are limited.

The aim of this study is to describe the post-discharge maintenance treatment profiles of patients with bipolar disorder who were treated in a high-security forensic psychiatric unit and found not criminally responsible due to their illness, and to examine the associations between these treatment patterns and sociodemographic, clinical, and offense-related variables.

Method

This study was designed as a retrospective, descriptive study. The study sample consisted of patients who were hospitalized in a high-security forensic psychiatric unit between 2019 and 2024 and were diagnosed with bipolar disorder according to the DSM-5 diagnostic criteria. The records of a total of 107 patients whose treatment was completed and who were discharged during the specified time period were retrospectively reviewed. The study was approved by the local ethics committee (07.10.2025, no. 612)

Data were obtained by reviewing patients' electronic and physical medical records, discharge summaries, and forensic reports. Using a data collection form developed by the researchers, patients' sociodemographic characteristics, clinical features, offense-related variables, and maintenance treatments prescribed at discharge were recorded.

The World Health Organization Anatomical Therapeutic Chemical/Defined Daily Dose (ATC/DDD) system was used to evaluate the total pharmacological load [4]. The defined daily dose for each medication was calculated, and the patients' total daily medication load was determined as the sum of these values.

The severity of the index crime was assessed using the Violence Profile for Current Offence (VPCO) [5]. This profile classifies crimes as non-violent, minimally violent, moderately violent, moderately severe violent, and violently violent.

The Clinical Global Impression (CGI) scale was used to assess the patients' clinical status. Illness severity at admission was evaluated using the CGI-Severity (CGI-S) subscale, while clinical improvement at discharge was assessed using the CGI-Improvement (CGI-I) subscale.

Statistical analyses were performed using the SPSS software. Descriptive data were presented as mean, standard deviation, and percentage. Relationships between continuous variables were assessed using Spearman's correlation analysis, and $p<0.05$ was considered statistically significant.

Results

The mean age of the 107 patients included in the study was 40.14 ± 11.29 years. Fifty-seven percent of the patients resided in urban areas, and approximately one-third had a history of alcohol or substance use. The mean age at illness onset was 25.91 ± 8.54 years. The mean CGI-S score at admission was 3.7 ± 0.7, while the CGI-I score at discharge was 2.6 ± 0.5. The patients' mean number of psychiatric hospitalizations was 3.96 ± 3.79. The mean age at the time of the index offense was 35.99 ± 11.33 years. According to the VPCO, 61.7% of the offenses were classified as moderately violent, 26.2% as minimally violent, and 12.1% as non- violent. No offenses in the moderately severe or severely violent categories were observed in the study sample. The mean length of stay in the high-security forensic psychiatric unit due to the index offense was 62.67 ± 36.68 days.

At discharge, 95.3% of the patients were receiving antipsychotic treatment, with 92.5% using oral antipsychotics. The proportion of patients receiving long-acting injectable antipsychotics (LAIs) was 18.6%. Clozapine use was observed in only two patients (1.9%). Among oral antipsychotics, the most frequently prescribed were olanzapine (42.7%) and risperidone (31.6%), followed by aripiprazole (10.2%) and amisulpride (1.9%). Quetiapine was prescribed to 40.9% of patients, but only 7.4% received it within the antipsychotic dose range. Among LAIs, the most commonly used were zuclopenthixol depot (7.4%), paliperidone palmitate (6.5%), and aripiprazole (4.6%). Regarding mood stabilizers, valproate was the most frequently used (56.7%), followed by lithium (19.5%) and carbamazepine (1.9%). When treatment combinations were evaluated, 67.3% of patients were on a combination of an antipsychotic and a mood stabilizer. The proportion of patients receiving antipsychotic monotherapy was 29.9%, while 3.7% were treated with a combination of two mood stabilizers. When pharmacological load was assessed, the mean total daily dose was 2.00 ± 0.96 DDD. The mean daily dose of antipsychotics was 1.50 ± 0.90 DDD, and the mean daily dose of mood stabilizers was 0.82 ± 0.44 DDD.

Correlation analyses revealed a significant positive relationship between hospitalization length and total daily dose (r_s=0.342, $p<0.001$). Similarly, a significant positive correlation was observed between hospitalization length and antipsychotic dose (r_s=0.278, p=0.005). A significant positive correlation was also found between the CGI-S score at admission and total daily dose (r_s=0.289, p=0.003). In contrast, no significant relationship was observed between the severity of the index offense and medication doses. Likewise, there was no significant association between the CGI-I score at discharge and antipsychotic doses.

Discussion

This study demonstrates that post-discharge maintenance treatment in patients with bipolar disorder who were treated in a high-security forensic psychiatric unit and found not criminally responsible is largely antipsychotic-based. Antipsychotic use was observed in the vast majority of patients, and a significant proportion of treatment strategies involved combinations with mood stabilizers. These findings indicate that treatment decisions in forensic psychiatric settings encompass broader clinical goals beyond the control of mood symptoms, including the maintenance of behavioral stability and the management of potential risks. The addition of antipsychotics to mood stabilizers has been reported to be more effective in reducing violent offenses compared with the use of mood stabilizers alone [6,7]. The regulatory effects of antipsychotics on agitation, impulsivity, and behavioral disinhibition may provide a significant advantage for clinicians in this setting.

When the relationships between medication doses and clinical and forensic variables were examined, it was observed that the pharmacological load was associated not with the severity of the index offense but rather with the patients' current clinical status. In particular, the association of total medication dose with length of stay and clinical severity at admission suggests that treatment intensity is determined more by the current psychopathology and the need for clinical stabilization than by prior offending behavior. Since longer hospitalizations often reflect more complex or treatment-resistant clinical presentations, the implementation of more intensive pharmacological interventions in these patients may be expected.

In contrast, it is noteworthy that no significant association was observed between the severity of the index offense and medication dosages. This result indicates that, in forensic psychiatric practice, pharmacological treatment is guided not as a response to prior offending behavior but as an intervention aimed at managing the patient's current clinical condition. Furthermore, the lack of a significant association between antipsychotic doses and clinical

improvement at discharge suggests that higher antipsychotic doses do not necessarily lead to better clinical outcomes, underscoring the importance of optimizing dose strategies in maintenance treatment.

The retrospective and single-center design of the study may limit the generalizability of the findings. In addition, the absence of post-discharge clinical follow-up data precluded the evaluation of the long-term outcomes of maintenance treatment.

This study demonstrates that maintenance treatment for patients with bipolar disorder in high- security forensic psychiatric units is frequently antipsychotic-based and often involves combination therapies. The findings suggest that, in forensic psychiatric settings, treatment strategies are guided not only by symptom control but also by a multidimensional clinical approach encompassing behavioral stability and risk management. To obtain more robust evidence in this field, prospective and comparative studies involving forensic psychiatric populations are needed.

Keywords: Bipolar Disorder, Criminal Responsibility, Forensic Psychiatry, Maintenance Treatment

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Table 1. Sociodemographic and clinical characteristics of the sample

	Patient group (n = 107) n (%) or mean ± SD
Age, years	40.14 ± 11.29
Age at illness onset, years	25.91 ± 8.54
Age at index offense, years	35.99 ± 11.33
Duration of illness at index offense, years	10.31 ± 9.73
Hospitalization length, days	62.67 ± 36.68
CGI-Severity at admission	3.7 ± 0.7
CGI-Improvement at discharge	2.6 ± 0.5
Residence	
Rural area	46 (43.0%)
City	61 (57.0%)
Substance use history	32 (29.9%)
Self-mutilation history	10 (9.3%)
Suicide attempt history	3 (2.8%)
Severity of index offense (Taylor scale)	
Non-violent	13 (12.1%)
Minimal violence	28 (26.2%)
Moderate violence	66 (61.7%)
Moderately severe violence	0
Severe violence	0

Note: M: Mean, SD: Standard deviation, n: number of cases, %: percentage, CGI: Clinical global impression

Table 2. Pharmacological treatment profiles and medication load

	Patient group (n = 107) n (%) or mean ± SD (range)
Total daily medication dose (DDD)	2.00 ± 0.96
Antipsychotic dose (DDD)	1.50 ± 0.90
Mood stabilizer dose (DDD)	0.82 ± 0.44
Antipsychotic treatment	
Any antipsychotic use	102 (95.3)
Oral antipsychotic use	99 (92.5)
Long-acting injectable antipsychotic use	20 (18.6)
Clozapine use	2 (1.9)
Oral antipsychotics	
Olanzapine	46 (42.7)
Risperidone	34 (31.6)
Aripiprazole	11 (10.2)
Amisulpride	2 (1.9)
Quetiapine (any dose)	44 (40.9)
Quetiapine at an antipsychotic dose	8 (7.4)
Long-acting injectable antipsychotics	
Zuclopenthixol depot	8 (7.4)
Paliperidone palmitate	7 (6.5)
Aripiprazole LAI	5 (4.6)
Mood stabilizers	
Valproate	61 (56.7)
Lithium	21 (19.5)
Carbamazepine	2 (1.9)
Treatment combinations	
Antipsychotic monotherapy	32 (29.9)
Antipsychotic + mood stabilizer	72 (67.3)
Dual mood stabilizer therapy	4 (3.7)

Note: DDD = Defined Daily Dose; a standard unit used to evaluate and compare drug utilization, applied within the ATC (Anatomical Therapeutic Chemical) classification system; LAI = Long-acting injectable antipsychotic.

0115 - Fear of Childbirth in Pregnancy: The Role of Prenatal Bonding, Attachment Style, and Trauma

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Objective

Fear of childbirth (FoC) is a significant source of psychological distress during pregnancy, yet it is often conceptualized primarily as an obstetric issue rather than as a reflection of broader psychological and relational processes. Pregnancy represents a profound psychological transition involving changes in identity, emotional regulation, and relational expectations. Central to this process is prenatal bonding, defined as the affective relationship a woman develops with her fetus.

FoC, also referred to as tokophobia, ranges from moderate anticipatory anxiety to severe, clinically significant fear that can influence delivery preferences and birth experiences [1]. While previous research has linked FoC to obstetric outcomes and general psychopathology, less attention has been paid to the relational and psychological mechanisms underlying it. In particular, prenatal bonding and adult attachment style may play central roles in shaping how women experience and anticipate childbirth.

Attachment theory provides a framework for understanding individual differences in stress regulation and threat perception. Internal working models influence how individuals respond to uncertainty, pain, and dependency—conditions that are highly relevant during pregnancy [2]. Insecure attachment has been associated with increased psychological vulnerability, including heightened childbirth fear [3].

Trauma exposure has also been identified as a risk factor for FoC. However, evidence suggests that the impact of trauma may depend more on how it is psychologically processed than on exposure alone, potentially operating through relational pathways such as attachment.

This study aimed to examine whether prenatal bonding, adult attachment style, and trauma exposure independently predict fear of childbirth. We hypothesized that (1) stronger prenatal bonding would be associated with lower FoC, (2) insecure attachment would independently predict higher FoC, and (3) trauma exposure would not independently predict FoC once relational variables were taken into account.

Methods

This cross-sectional study was conducted at the Obstetrics and Gynecology outpatient clinic of Bakırköy Dr. Sadi Konuk Training and Research Hospital. Ethical approval was obtained from the relevant institutional review board, and written informed consent was obtained from all participants.

Eighty pregnant women attending routine prenatal care were recruited consecutively. Inclusion criteria were being pregnant and able to complete self-report measures. Women with cognitive impairment or inability to provide informed consent were excluded.

Fear of childbirth was assessed with the Wijma Delivery Expectancy Questionnaire Version A (W-DEQ-A), prenatal bonding with the Prenatal Attachment Inventory (PAI), and adult attachment with the Relationship Scales Questionnaire (RSQ); participants were categorized as having secure or insecure attachment styles.

Trauma exposure was evaluated using a checklist of events meeting PTSD Criterion A and coded dichotomously. Sociodemographic and obstetric variables were collected using a structured form.

Hierarchical linear regression analyses were performed to examine predictors of FoC. Trauma exposure was entered in the first model, prenatal bonding in the second model, and attachment style in the third model. A final model included the interaction term between prenatal bonding and attachment style.

Results

The sample characteristics and descriptive statistics are presented in Table 1. The sample had a mean maternal age of 28.46 years (SD = 6.05) and a mean gestational age of 29.93 weeks (SD = 7.91). Approximately half of the participants reported trauma exposure (n = 38, 49.4%), and 60.5% were classified as insecurely attached (n = 46).

Table 1. Sample characteristics and descriptive statistics

Variable	***n* (%) or Mean (SD)**
Maternal age (years)	28.46 (6.05)
Gestational week	29.93 (7.91)
Trauma exposure (PTSD Criterion A)	
Yes	38 (49.4%)
No	39 (50.6%)
Attachment style	
Secure	30 (39.5%)
Insecure	46 (60.5%)
PAI score	62.29 (13.38)
W-DEQ-A Score	46.35 (25.97)
Previous traumatic birth experience* (n=43)	
Yes	14 (32.6%)
No	29 (67.4%)

PAI: Prenatal Attachment Inventory, W-DEQ-A: Wijma Delivery Expectancy Questionnaire Version A
*Assessed only among women with a previous childbirth experience

Results of the hierarchical regression analyses are presented in Table 2. In the first regression model, trauma exposure was not significantly associated with fear of childbirth ($\beta = 0.01$, $p = 0.92$).

In the second model, prenatal bonding was added and emerged as a significant predictor, with higher bonding associated with lower FoC ($\beta = -0.45$, $p < 0.001$), explaining a substantial proportion of variance ($\Delta R^2 = 0.20$).

In the third model, attachment style was included. Prenatal bonding remained the strongest predictor ($\beta = -0.43$, $p < 0.001$), while insecure attachment independently predicted higher FoC ($\beta = 0.25$, $p = 0.017$). The model explained 26% of the variance in fear of childbirth ($R^2 = 0.26$).

In the final model, the interaction between prenatal bonding and attachment style was not statistically significant ($\beta = -0.38$, $p = 0.34$), and did not meaningfully increase explained variance ($\Delta R2 = 0.01$), indicating no moderation effect.

Table 2.Hierarchical regression analyses predicting fear of childbirth (Wijma-A scores)

	Dependent variable: Wijma-A Scores			
	Model 1 β (p)	**Model 2** β (p)	**Model 3** β (p)	**Model 4** β (p)
PTSD Criteria A	0.01 (0.92)	-0.06 (0.57)	-0.05 (0.63)	-0.05 (0.64)
PAI score		-0.45 (<0.001)	-0.43 (<0.001)	-0.06 (0.89)
Attachment Style (Secure/Insecure)			0.25(0.017)	0.25 (0.02)
Interaction between attachments (Prenatal Bonding X Attachment Style)				-0.38(0.34)
R^2	0.0	0.2	0.26	0.27
AR^2	-	0.2	0.06	0.01

PTSD: Posttraumatic stress disorder, PAI: Prenatal Attachment Inventory

In an exploratory analysis among multiparous women (n = 43), those with a history of traumatic birth (n = 14) showed higher FoC scores compared to those without (n = 29), although this difference did not reach statistical significance (p = 0.055).

Discussion

This study demonstrates that fear of childbirth is more strongly associated with relational and psychological processes during pregnancy than with trauma exposure alone. Prenatal bonding emerged as the most robust predictor of FoC, suggesting that the emotional connection a woman develops with her fetus plays a central role in shaping her anticipation of childbirth.

Stronger prenatal bonding may function as a psychological buffer, reducing threat perception and facilitating emotional integration during pregnancy. This finding is consistent with previous research linking maternal-fetal attachment to lower childbirth fear and improved perinatal outcomes [4,5]. Unlike attachment style, prenatal bonding represents a dynamic and potentially modifiable process, making it a clinically meaningful target for intervention.

Insecure attachment independently predicted higher FoC, supporting theoretical models that emphasize the role of internal working models in stress regulation [2]. Pregnancy involves conditions that activate attachment-related expectations, including vulnerability, dependence on care, and uncertainty. Women with insecure attachment may experience these conditions as threatening, leading to heightened fear responses [3].

Trauma exposure did not independently predict FoC once relational variables were included. This finding suggests that trauma may influence childbirth fear not through exposure alone, but through its psychological sequelae. In particular, posttraumatic stress symptoms may play a more critical role, as they can amplify threat perception, disrupt the sense of bodily safety, and shape expectations of care during childbirth. More broadly, these findings indicate that focusing solely on the presence or absence of traumatic events may be insufficient; rather, how these experiences are subjectively processed and integrated into a woman's relational and emotional world may be more relevant for understanding FoC. From this perspective, FoC may be better understood as a manifestation of altered relational and affective regulation processes, rather than a direct consequence of past traumatic events. This distinction highlights the importance of emphasizing individual psychological processing and relational mechanisms when evaluating FoC.

From a clinical perspective, screening for impaired prenatal bonding and attachment insecurity during routine antenatal care may improve early identification of women at risk for FoC. FoC is also clinically relevant, as it

has been associated with adverse obstetric outcomes, including increased rates of elective cesarean delivery, prolonged labor, and greater use of obstetric interventions [1]. Addressing and preventing FoC may therefore contribute not only to improved psychological well-being but also to more favorable obstetric outcomes. Interventions that enhance emotional engagement with pregnancy and address relational expectations may thus be more effective than approaches focused solely on obstetric education [1].

Although the cross-sectional design limits causal interpretation and the relatively small multiparous subgroup may have reduced statistical power, these findings offer a meaningful reframing of fear of childbirth from a primarily obstetric concern to a relational and psychological process. By demonstrating the central role of prenatal bonding and attachment processes in shaping fear, the study highlights the importance of individualized psychological assessment in perinatal care, emphasizing that it is not merely the presence of adverse experiences but how they are subjectively perceived and integrated into a woman's relational and emotional world that may be most relevant. Integrating this perspective into routine care may improve both psychological well-being and obstetric outcomes.

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0118 - Reducing Peer Bullying in Children Who Stutter Through an ACT-Informed Group Intervention: Preliminary Findings

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Objective

Stuttering is considered a multidimensional communication disorder involving genetic, neurological, cognitive, emotional, and environmental factors. Children who stutter (CWS) experience significantly higher levels of negative peer interactions, which are associated with increased anxiety, emotional distress, reduced social participation, and lower self-esteem, thereby amplifying the overall psychosocial burden of stuttering [1]. In line with the growing recognition of the emotional, cognitive, and behavioral processes underlying stuttering, there has been a shift from speech-centered interventions toward more comprehensive, psychosocially informed treatment frameworks. However, despite being frequently reported as a significant concern in CWS, peer bullying is still seldom incorporated as a direct and explicit therapeutic target within intervention programs for this population [2].

Within this evolving therapeutic landscape, Acceptance and Commitment Therapy (ACT) has emerged as a promising approach for addressing the broader psychosocial dimensions of stuttering. Given that many individuals who stutter place a high value on fluent communication, ACT provides a relevant framework by emphasizing acceptance and psychological flexibility, redirecting intervention from an exclusive focus on symptom reduction toward engagement in meaningful, value-based communication [3]. In addition, ACT's emphasis on psychological flexibility and acceptance of difficult internal experiences may offer a useful framework for helping children cope more effectively with bullying-related stressors and social challenges. Consistent with this perspective, ACT has shown promising effects in individuals who stutter, particularly in improving psychosocial functioning, enhancing engagement in meaningful communication, and reducing avoidance behaviors [4]; however, empirical studies examining its application in CWS remain scarce.

The primary objective of this pilot study was to evaluate the feasibility and preliminary outcomes of an ACT-based group intervention integrating structured bullying-focused psychoeducation for early school-age CWS. It was hypothesized that participation in the

program would be associated with (1) reduced bullying experiences, (2) improved quality of life, (3) decreased psychosocial impact of stuttering, alongside (4) improvements in emotional and behavioral functioning.

Methods

Participants: The study included four children (3 males, 1 female) aged 7-8 years with a history of developmental stuttering of at least 12 months. Participants were recruited from a private speech and language therapy clinic through parent referral. Two participants had comorbid attention-deficit/hyperactivity disorder (ADHD), while the remaining participants had no additional diagnoses.

All participants were native Turkish speakers and had no sensory or language impairments that would interfere with participation. Ethical approval was obtained, and informed consent was provided by all parents.

Measurements: Multiple standardized instruments were used to assess both speech-related and psychosocial outcomes. Health-related quality of life was measured using the Pediatric Quality of Life Inventory (PedsQL 4.0), which evaluates physical, emotional, social, and school functioning. Peer bullying experiences were assessed with the Violence/Bullying in Schools Scale (VBSS). Emotional and behavioral functioning was evaluated through parent report using the Child Behavior Checklist (CBCL 6-18). The impact of stuttering on daily life was assessed using the Overall Assessment of the Speaker's Experience of Stuttering - School Age (OASES-S). Stuttering severity was measured using the Stuttering Severity Instrument, Fourth Edition (SSI-4), which evaluates frequency, duration, and secondary behaviors. In addition, speech samples consisting of approximately 300 syllables were obtained through spontaneous conversational tasks and analyzed by an experienced speech- language pathologist.

Intervention Procedure: The intervention consisted of eight weekly group sessions (60 minutes), co-led by a speech-language pathologist and a child and adolescent psychiatrist trained in Acceptance and Commitment Therapy (ACT). A pre-post design was employed, with assessments conducted one week before and after the intervention. The program systematically targeted ACT's six core processes—present-moment awareness, acceptance, cognitive defusion, self-as-context, values, and committed action—integrated with stuttering- related psychoeducation and communication-based activities.

The intervention followed a structured progression across sessions. In the first session, group cohesion was established through introductions, rule-setting, and psychoeducation on stuttering, alongside brief mindfulness and breathing exercises to support contact with the present moment. The second session focused on acceptance and cognitive defusion, incorporating child-adapted exercises such as "I am having the thought that..." and visual metaphors (e.g., thoughts as passing clouds) to help participants observe internal experiences without over-identifying with them. In the third session, committed action was introduced through role-play and assertiveness training, allowing children to practice value-consistent responses in challenging social situations. The fourth session emphasized emotional awareness and acceptance, integrating mindfulness exercises (e.g., "leaves on a stream") and voluntary stuttering to promote desensitization and reduce experiential avoidance. The fifth session further targeted cognitive defusion using metaphor-based activities (e.g., "passengers on the bus"), supporting children in relating more flexibly to negative thoughts. In the sixth session, values clarification was introduced, guiding participants to identify personally meaningful directions in communication and social relationships. The seventh session focused on self-as-context, encouraging children to distinguish between their experiences and their sense of self, supported by reflective storytelling and body-based awareness exercises. Finally, the eighth session aimed to consolidate therapeutic gains through personal narratives, reinforcement of committed action, and reflection on value-based behavioral changes in everyday communication.

Results

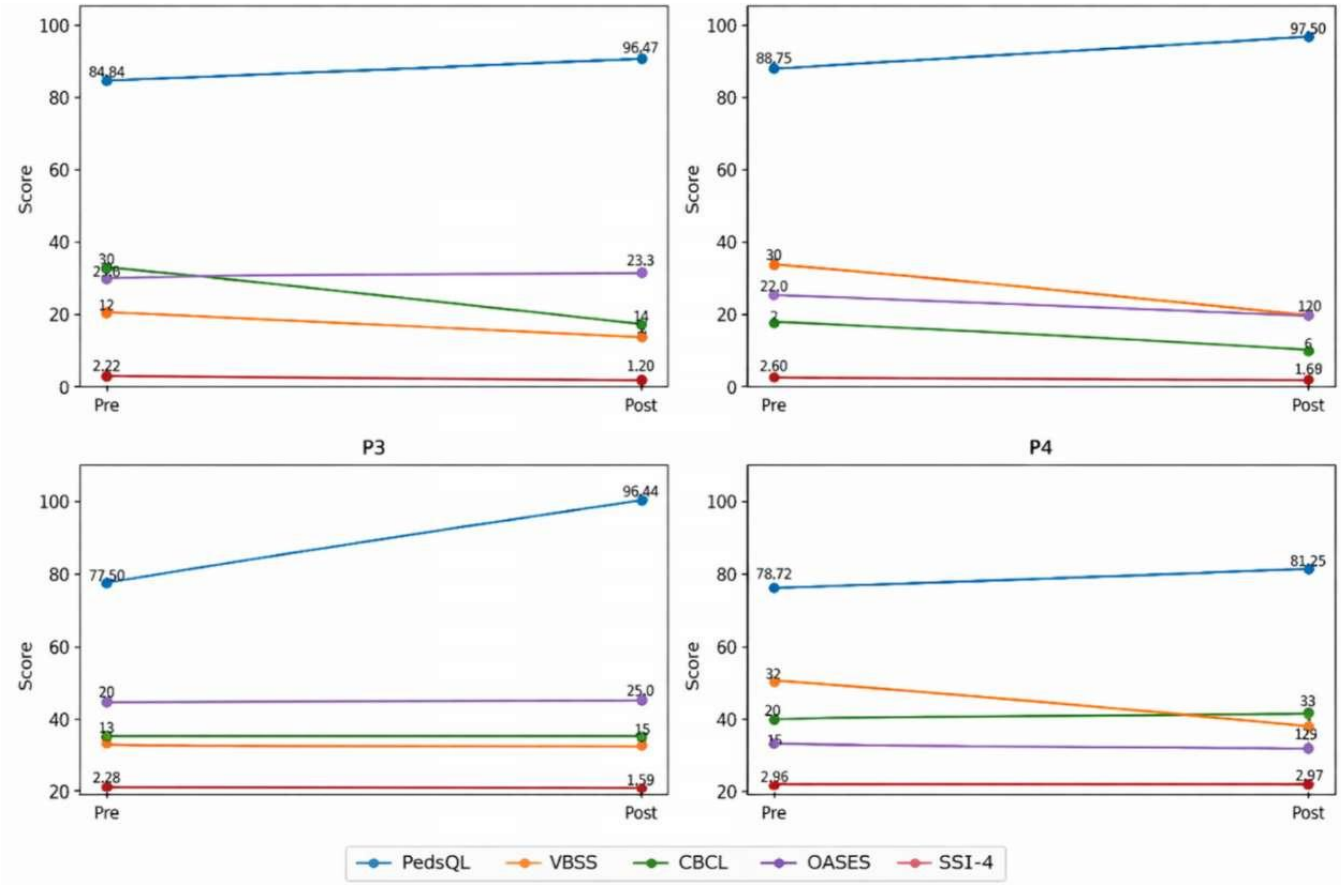
Preliminary inspection of the data indicated pre-to-post changes across outcome domains. As shown in Table 1, descriptive statistics revealed increased quality-of-life scores (PedsQL) alongside decreases in peer bullying experiences (VBSS), emotional-behavioral problems (CBCL), the overall impact of stuttering (OASES), and stuttering severity (SSI-4).

Table 1. Group-level descriptive statistics for pre- and post-intervention outcome measures

Measure	N	Pre Mean ± SD	Pre Median (Min-Max)	Post Mean ± SD	Post Median (Min-Max)
PedsQL	4	81.95 ± 5.82	81.17 (76.72-88.75)	91.91 ± 7.95	93.99 (81.25-98.44)
VBSS	4	22.75 ± 8.85	24.5 (12-30)	14.25 ± 2.50	14.5 (11-7)
CBCL	4	22.75 ± 10.14	23.0 (13-32)	16.75 ± 10.94	14.5 (6-32)
OASES	4	2.38 ± 0.39	2.22 (2.12-2.96)	1.96 ± 0.68	1.65 (1.57-2.97)
SSL-4	4	23.55 ± 6.78	25.3 (14.6-29.0)	20.58 ± 8.17	21.0 (11.0-29.3)

Individual outcome trajectories (Figure 1) further illustrated overall patterns of improvement across participants while also highlighting variability in treatment response. Overall, these findings suggest improvements in quality of life, accompanied by reductions in psychosocial difficulties and the impact of stuttering following the intervention.

Figure 1. Individual pre- and post-intervention scores across outcome measures for P1- P4



Discussion

The present pilot study examined the feasibility and preliminary outcomes of an ACT-based group intervention integrating bullying-focused psychoeducation for school-age CWS. To our knowledge, this study is among the first to integrate ACT within an intervention framework specifically targeting CWS. Overall, the findings suggested improvements across several psychosocial domains, including reduced bullying victimization, fewer emotional-behavioral difficulties, and a lower psychosocial impact of stuttering, alongside improved health-related quality of life.

These findings are consistent with evidence indicating that stuttering is associated with significant psychosocial burden, including increased emotional difficulties and vulnerability to negative peer experiences [1]. Previous research has shown that children who stutter are at increased risk of anxiety, reduced self-esteem, and bullying victimization, underscoring the need for interventions that extend beyond speech fluency [1]. In this context, the present findings support the clinical relevance of directly targeting peer bullying within intervention frameworks.

The observed improvements may be understood within the framework of psychological flexibility, a central process targeted by ACT. Psychological inflexibility and avoidance are known to exacerbate communication difficulties and negative emotional responses in individuals who stutter [5]. ACT aims to reduce experiential avoidance by fostering acceptance and engagement in value-based communication [3]. From an ACT perspective, the goal is not necessarily to eliminate difficult experiences such as bullying but to support more flexible responding while maintaining engagement in meaningful communication behaviors [6]. In line with this framework, the reductions observed in bullying scores in the present study may be interpreted not solely as a decrease in exposure, but as an indication of improved coping with peer-related stressors among children who stutter. More specifically, increased psychological flexibility may have enabled participants to reinterpret bullying

experiences, reduce avoidance, and respond more adaptively in social interactions, thereby diminishing the perceived impact of bullying on their daily functioning.

Existing research further supports the relevance of ACT in stuttering. ACT-based interventions have been associated with reductions in anxiety and avoidance, as well as improvements in psychosocial functioning and communication participation [4]. Extending this literatüre, the present findings suggest that developmentally adapted ACT interventions may also be applicable in younger children who stutter.

Several limitations should be considered. The small sample size and absence of a control group limit the generalizability of the findings and preclude causal conclusions. In addition, the short follow-up period does not allow evaluation of the long-term maintenance of treatment effects. Future studies should investigate the effectiveness of ACT-based group interventions in larger, controlled samples and examine psychological flexibility as a potential mechanism of change.

In conclusion, the present pilot study provides preliminary evidence that ACT-based group interventions incorporating bullying-focused psychoeducation may improve psychosocial outcomes in children who stutter. Targeting psychological flexibility alongside peer-related experiences may represent a clinically meaningful approach for addressing the broader psychosocial dimensions of stuttering.

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0119 - Trauma-Triggered Olfactory Hallucination During a Manic Episode: Amygdala-Mediated Sensory Memory Reactivation Following Earthquake Exposure

Introduction

Traumatic experiences are strongly associated with a wide range of psychiatric outcomes, including mood disorders, dissociative symptoms, and post-traumatic stress phenomena. Sensory triggers play an important role in the reactivation of traumatic memories. Among sensory modalities, olfaction occupies a unique neurobiological position due to its direct connections with limbic structures, particularly the amygdala and hippocampus. Unlike visual and auditory inputs, olfactory signals reach emotional memory circuits without thalamic filtering, which allows odors to evoke powerful emotional memories (1,2).

Previous research has demonstrated that odor-evoked memories are often vivid, emotionally intense, and strongly linked to autobiographical experiences. In trauma- exposed individuals, olfactory cues may act as potent triggers that reactivate previously encoded traumatic experiences (1,5). This phenomenon has been frequently described in post-traumatic stress disorder, where trauma-related odors can provoke intense emotional responses and sensory flashbacks (2,5).

Manic episodes are characterized by increased limbic activation and reduced prefrontal inhibitory control. Neuroimaging studies indicate that bipolar mania is associated with hyperactivity in the amygdala and decreased regulatory activity in the prefrontal cortex (4). This imbalance may lower the threshold for emotionally charged sensory experiences.

The present case describes a patient who developed an acute manic episode accompanied by persistent olfactory hallucinations following a mild earthquake. The olfactory perception specifically involved the smell of dead bodies, corresponding directly to a traumatic sensory experience during a previous earthquake in which the patient had been trapped under rubble and exposed to decomposing bodies. This case provides an example of trauma-associated olfactory memory reactivation occurring during a manic episode and highlights the potential role of amygdala-mediated sensory memory in such phenomena.

Case Presentation

The patient was a 38-year-old married male with a university education and active employment. He had no prior history of psychotic disorders and no known neurological conditions. Approximately five years before the current presentation, he experienced a major earthquake during which he remained trapped under collapsed structures for an extended period. During that time he was exposed to the smell of decomposing bodies. Following the event, he reported temporary sleep disturbances and heightened startle responses but did not receive professional psychiatric treatment.

Two days before admission, a mild earthquake occurred in the city where he lived. Within hours after the tremor, family members observed a marked behavioral change. The patient developed severe insomnia, increased talkativeness, agitation, elevated mood, and grandiose ideas. Shortly afterward he began repeatedly stating that he could smell "dead bodies" in the environment. The perceived odor had no identifiable external source and could not be confirmed by others.

During psychiatric examination the patient was fully oriented and conscious. However, he displayed pressured speech, accelerated thought processes, increased goal-directed activity, and reduced insight. His mood was elevated and irritable. The olfactory perception was persistent and distressing and was interpreted as an olfactory hallucination.

Neurological examination revealed no focal deficits. Routine laboratory investigations were within normal limits. There was no evidence of infection, metabolic disturbance, substance use, or seizure activity. Brain imaging did not

reveal structural abnormalities. Based on the clinical findings, the patient was diagnosed with a manic episode accompanied by olfactory hallucinations.

Discussion

This case illustrates a rare intersection between traumatic sensory memory and mood- related limbic activation. Olfactory hallucinations are relatively uncommon in psychiatric disorders and are more frequently associated with neurological conditions such as temporal lobe epilepsy or neurodegenerative diseases. When they occur in psychiatric contexts, they are most often linked to psychotic disorders.

In this case, however, the olfactory hallucination appeared closely related to a previously experienced traumatic odor. The smell of decomposing bodies perceived during the manic episode corresponded directly to the sensory experience the patient had during the earlier earthquake. This suggests that the hallucination may represent a reactivation of trauma-encoded sensory memory rather than a primary psychotic phenomenon.

From a neurobiological perspective, the olfactory system has privileged access to limbic emotional memory circuits. Olfactory signals are transmitted from the olfactory bulb to the piriform cortex and directly to the amygdala and hippocampus. The amygdala plays a central role in fear conditioning and emotional memory encoding (2,3). During traumatic events, intense sensory inputs may become strongly associated with emotional responses through amygdala-hippocampal networks.

When a new stressor resembling the original traumatic context occurs, these networks may be reactivated even in the absence of the original sensory stimulus. In this case, the mild earthquake may have acted as a contextual trigger that activated trauma-associated olfactory memory stored within limbic circuits.

Manic episodes may further facilitate such reactivation through limbic disinhibition. Neuroimaging studies of bipolar disorder indicate increased activity in the amygdala and decreased regulatory control from the prefrontal cortex during mania (4). Reduced top- down inhibitory control may allow emotionally encoded sensory memories to emerge in the form of vivid perceptual experiences.

In this context, mania may not generate the hallucination itself but may release trauma- encoded sensory memory through limbic disinhibition. The olfactory hallucination observed in this patient may therefore represent the convergence of two processes: reactivation of trauma-associated sensory memory and mood-related limbic activation.

Conclusion

This case highlights the potential role of trauma-encoded olfactory memory in the emergence of unusual perceptual experiences during mood episodes. In individuals with prior exposure to disaster-related trauma, new stressors may reactivate emotionally encoded sensory memories through amygdala-mediated pathways. Clinicians should consider trauma-associated sensory memory reactivation when evaluating atypical perceptual symptoms in mood disorders, particularly when such symptoms are linked to specific traumatic experiences.

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0121 – Computer Assisted Forensic Psychiatric Interview A Case Report

Introduction

Speech is the primary means of establishing dialogue among human beings. The inability to speak due to physical impairment is a loss of faculty frequently observed, particularly following cerebrovascular accidents (CVA). Approximately 88% of stroke survivors experience some degree of communication impairment, while aphasia develops in one-third of cases. The loss of speech ability negatively impacts participation in daily life and may lead the individual to feel "unimportant, unnoticed, and invisible".

Computer-mediated speech is a frequently utilized method for facilitating communication in such individuals. The use of computer-mediated communication in the fields of medicine and psychiatry has demonstrated an upward trend in recent years. In the field of psychiatry, computer-assisted psychotherapy applications, in particular, are gaining prevalence and are recommended as first-line treatments in certain countries. In the realm of psychotherapy, computer-mediated communication is typically employed as a tool to overcome the barrier of distance via the internet between two verbal individuals. However, no cases regarding psychiatric assessment conducted via computer-mediated means for a patient who has lost the ability to speak due to a CVA have been encountered in the literature. This presentation describes the forensic psychiatric assessment of a patient who lost the ability to speak due to a CVA and communicates through specialized computer hardware and software. Consent was obtained from the individual via email.

Case Presentation

E.H., a 43-year-old male. A report was requested by the court regarding the necessity of continuing the guardianship of the patient, who became tetraplegic and unable to speak following an accident. Due to the patient's ability to communicate only with the aid of specialized equipment, an in-home assessment was requested. In accordance with this request, an assessment was conducted at the patient's residence by an expert committee consisting of two psychiatrists and one neurologist.

Anamnesis obtained from the patient and his spouse revealed that the patient, who had no known history of psychiatric illness, sustained head trauma two years prior due to a fall from a bicycle. Following the trauma, a vertebral artery dissection occurred, subsequently leading to pontine and cerebellar infarction. He was monitored in the intensive care unit in an unconscious state for a period. A tracheostomy and percutaneous endoscopic gastrostomy (PEG) were performed. He was hospitalized three times and underwent physical therapy and rehabilitation. During his medical follow-up, he regained consciousness and no longer required respiratory support. He was discharged in a tetraplegic state. Some time later, it became evident that the patient comprehended spoken language and was attempting to respond. The patient, whose neck and eye movements were preserved, began to communicate with his environment through a method where letters were shown individually, and he signaled by closing his eyes when the correct letter was reached. To facilitate communication, he began utilizing a specialized computer program and hardware.

The patient, who was a computer programmer prior to the accident, developed a software application similar to the one he utilized for communication and began employing it personally. He advanced his communication capabilities by navigating a cursor across a virtual keyboard displayed on the computer screen; this was achieved through neck movements tracked by specialized hardware worn on his head, while character selection was executed via a chewing motion on the desired letter. He endeavored to maintain social activities within the home environment and commenced regular Arabic grammar instruction. He had been placed under the guardianship of his spouse on the grounds of inability to manage his own affairs; however, he and his spouse subsequently petitioned the court for the revocation of the guardianship, as the arrangement complicated the management of financial matters. Throughout the legal proceedings, he personally submitted his requests to the court in written form.

The mental status examination was conducted via the hardware and computer software utilized for communication. It was observed that the patient comprehended the inquiries and was capable of responding by typing via the computer interface. The mental status examination revealed the patient to be conscious, cooperative, and oriented, with intact attention and memory. Intelligence was clinically within normal limits. Abstraction ability was preserved; he was able to interpret proverbs at an abstract level. Judgment was intact. He was able to perform mathematical calculations. The thought process was normal. Thought content was dominated by vital themes; no delusions were detected. No perceptual disturbances were described. Affect was evaluated as restricted, attributed to neurological impairment.

Sufficient conviction was established to reach a conclusion during the forensic psychiatric assessment conducted via computer-assisted interview, and a report was issued stating that guardianship was not required from a mental health perspective.

Conclusion

The loss of speech and motor function does not equate to a loss of mental faculties. The loss of speech ability may be misperceived as cognitive deterioration or demential processes, as illustrated in the presented case, potentially leading to the unjust placement of patients under guardianship or the deprivation of their civil rights. Even in cases of severe physical disability, provided that appropriate technological hardware and adaptations are available, it is possible to conduct a sound evaluation of an individual's capacity to exercise civil rights. This case presentation demonstrates that in cases of severe physical disability:

1. Complex cognitive processes can be clearly demonstrated through technology, as evidenced by the patient developing his own communication software;
2. Computer-assisted communication tools can be effectively utilized in forensic psychiatric assessment;
3. Transcending standard examination techniques in patients with communication limitations may be essential for the accurate determination of legal capacity and the preservation of patient autonomy.

0125 - Retrospective Evaluation of the Sociodemographic and Clinical Characteristics of Patients with Catatonia Hospitalized in an Inpatient Psychiatric Unit

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Introduction:

Catatonia is a neuropsychiatry syndrome characterized by motor, behavioral, and autonomic symptoms and may occur in association with various psychiatric and medical disorders (1). Although historically it has been most commonly associated with schizophrenia, current literature indicates that catatonia can also occur in mood disorders, neurological diseases, and various general medical conditions (2). Delayed diagnosis of catatonia may lead to serious complications such as dehydration, malnutrition, and increased morbidity. Therefore, early recognition and appropriate treatment are of great clinical importance.

The clinical and demographic characteristics of catatonia have shown heterogeneous findings across different studies. Moreover, the differences between catatonia associated with schizophrenia and catatonic presentations related to non- schizophrenic diagnoses have not been fully clarified in clinical practice.

The aim of this study was to retrospectively evaluate the sociodemographic and clinical characteristics of patients hospitalized with a diagnosis of catatonia in a university hospital psychiatry clinic. Additionally, the study aimed to compare catatonic patients diagnosed with schizophrenia and those with other psychiatric diagnoses in terms of clinical characteristics, treatment history, and severity of catatonia.

Methods

This retrospective study was conducted by reviewing the medical records of patients who were hospitalized with a diagnosis of catatonia in the Psychiatry Clinic of Akdeniz University Faculty of Medicine Hospital between November 5, 2019, and November 5, 2024. During this period, the records of 1,972 patients who were hospitalized in the psychiatry ward were screened. Among them, 25 patients who met the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) criteria for catatonia and satisfied the study inclusion criteria were included in the sample.

The inclusion criteria were: being between 18 and 65 years of age, having been hospitalized in the psychiatry ward with a diagnosis of catatonia, and having complete medical records. Patients with substantially missing clinical data were excluded from the study.

Patient records were reviewed to collect sociodemographic and clinical variables, including age, sex, marital status, referral source, hospitalization status, previous hospitalization history, number of previous hospitalizations, history of catatonia, history of and response to electroconvulsive therapy (ECT), history of and response to benzodiazepine (BDZ) treatment, presence of medical comorbidity, and family history of psychiatric disorders.

The severity of catatonia was evaluated using the KANNER Catatonia Rating Scale. Ethical approval for the study was obtained from the Akdeniz University Medical Scientific Research Ethics Committee with decision number TBAEK-79 on January 1, 2025, and the study was conducted in accordance with the principles of the Declaration of Helsinki.

Statistical analyses were performed using IBM SPSS Statistics version 25 (SPSS Inc., Chicago, IL, USA). Since the sample size in at least one of the groups was below 30, non-parametric tests were preferred. The Mann-Whitney U test was used for comparisons between two independent groups. For categorical variables, Pearson's chi-square test, Fisher's exact test, Fisher-Freeman-Halton exact test, and Yates' continuity correction were applied where appropriate. Post-hoc Bonferroni correction was performed when necessary. A p value < 0.05 was considered statistically significant.

Results

Among the 25 patients included in the study, 11 patients (44%) were diagnosed with schizophrenia.

When patients with and without schizophrenia were compared, no statistically significant differences were found between the groups in terms of age ($p = 0.609$) and sex ($p = 0.081$). However, the proportion of single individuals was significantly higher in the schizophrenia group ($p = 0.012$). The analysis results of the sociodemographic variables are presented in Table 1.

According to the analysis, the only variable that showed a statistically significant difference between the groups was the number of electroconvulsive therapy (ECT) sessions administered ($p = 0.033$). Patients diagnosed with schizophrenia received a significantly higher number of ECT sessions compared with patients with other diagnoses.

Several clinical variables were numerically higher in the schizophrenia group, including psychiatric outpatient clinic referrals, history of previous hospitalization and number of hospitalizations, presence of a history of catatonia, presence of prior ECT treatment, full response rate to ECT, response to benzodiazepine (BDZ) treatment, absence of medical comorbidity, and family history of psychiatric illness. However, these differences were not statistically significant ($p > 0.05$). The analysis results of the clinical variables are presented in Table 2.

Regarding the severity of catatonia, the total KANNER Catatonia Scale score was found to be lower in the schizophrenia group compared with the non-schizophrenia group. Nevertheless, the schizophrenia group showed higher scores in several symptom subdomains, including immobilization-stupor, mutism, staring, catalepsy, grimacing, mannerism, waxy flexibility, eating and drinking disturbances, reduced eye contact, automatic obedience, and ambitendency. Despite these differences, no statistically significant differences were observed between the groups in terms of total scale scores or subscale scores ($p > 0.05$).

Discussion

In this study, the sociodemographic and clinical characteristics of patients hospitalized with catatonia in a university hospital were retrospectively evaluated, and catatonia cases associated with schizophrenia were compared with catatonic patients with other psychiatric diagnoses.

During the study period, catatonia was identified in 25 out of 1,972 patients (1.26%) hospitalized in the psychiatry ward. This rate appears to be lower than that reported in some studies in the literature. Differences between studies may be related to variations in diagnostic criteria for catatonia, sample characteristics, and methodological differences.

In our study, no significant differences were observed between the groups in terms of age or sex. This finding supports the notion that catatonia is not restricted to a specific demographic profile and may present with a similar demographic distribution across different psychiatric diagnoses (2).

In contrast, a significant difference was observed in marital status. The higher proportion of single individuals in the schizophrenia group may be explained by the early onset, chronic course, and marked impairment in social functioning associated with schizophrenia (3). It is well known that social withdrawal, negative symptoms, and cognitive impairment in schizophrenia can adversely affect interpersonal relationships and the likelihood of marriage (4).

One of the most important findings of this study was that the number of ECT sessions was significantly higher in the schizophrenia group. This finding may suggest that schizophrenia-associated catatonia may follow a more resistant or more severe clinical course. Previous studies have also reported that catatonia associated with schizophrenia may respond less favorably to benzodiazepine treatment and may require more intensive ECT protocols (2,5). In this context, our findings support the idea that schizophrenia-associated catatonia may have different treatment requirements.

Although several clinical variables, including history of psychiatric outpatient visits, number of previous hospitalizations, history of catatonia, and history of ECT treatment, were numerically higher in the schizophrenia group, these differences did not reach statistical significance. This may indicate that catatonia can present with severe clinical manifestations regardless of the underlying psychiatric diagnosis (6).

In addition, the limited sample size may have contributed to the lack of statistically significant findings for these variables.

Examination of the KANNER Catatonia Scale results showed that although some symptom subdomains were higher in the schizophrenia group, total scale scores were similar between the groups. This finding suggests that the symptom pattern of catatonia may vary depending on the underlying diagnosis rather than the overall severity of the syndrome. Previous literature has reported that motor inhibition symptoms such as stupor, mutism, and catalepsy may be more prominent in schizophrenia-related catatonia, whereas agitation and emotional reactivity may be more frequent in catatonia associated with mood disorders (2,6).

This study has several limitations. The retrospective design, limited sample size, and single-center setting may restrict the generalizability of the findings. Future studies with larger samples and prospective designs may provide a better understanding of the clinical characteristics of catatonia across different psychiatric diagnoses.

In conclusion, catatonia is a heterogeneous syndrome that can occur in various psychiatric disorders. The most important finding of our study is that patients with schizophrenia-associated catatonia required a higher number of ECT sessions, suggesting that treatment requirements for catatonia may vary depending on the underlying psychiatric diagnosis. Recognizing the different clinical patterns of catatonia may help clinicians develop more individualized diagnostic and treatment strategies.

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Table 1. Sociodemographic Characterictics According to Comorbid Psychiatric Diagnosis

Variables	Comorbid Psychiatric Diagnosis			
	Schizophrenia (n=11)	Other Diagnoses(n=14)	Test Statistic	p
Age (years)	41(21-51)	38,5(26-59)	67.5	0,609[M]
Gender			4.588	0,081*
Male	6(54,5)	2(14,3)		
Female	5(45,5)	12(85,7)		
Marital Status			8.214	**0,012***
Married	1(9,1)[a]	9(64,3)[b]		
Single	9(81,8)[a]	5(35,7)[b]		
Divorced	1(9,1)[a]	0(0)[a]		

*Pearson Chi-Square Test, Fisher's Exact Test, Fisher's Freeman Halton Exact Test, Yates Correction, Post-Hoc Bonferroni Correction, *n(%).*[v]*Mann* Whitney U Test, *Med(IQR).*Superscript letters ([ab]) inticate subgroups that are not significantliy differens from each other at $p<0.05$.

Table 2.Clinical Parameters According to Comorbid Psychiatric Diagnosis

Variables	n_1/n_2	Comorbid Psychiatric Diagnosis		Test Statictic	P*
		Schizophrenia	Other Diagnoses		
Referral Source	11/14			0.649	0,623*
Emergency Department		8(72,7)	12(85,7)		
Psychiatry Outpatient		3(27,3)	2(14,3)		
Clinic					
Hospitalization Status	11/14			1.708	0,487*
Outpatient		0(0)	2(14,3)		
Inpatient		11(100)	12(85,7)		
Histroy of Previous Hospitalization	11/14			1.326	0,414*
Yes		8(72,7)	7(50)		
No		3(27,3)	7(50)		
Number of Previous Hospitilzations	11/14	2(0-5)	0,5(0-2)	51.5	0,166[M]
History of Catatonia	11/14			0.000	>0,999*
Yes		6(54,5)	7(50)		
No		5(45,5)	7(50)		
History og ECT	11/14			2.231	0,227*
Var		8(72,7)	6(42,9)		
Yok		3(27,3)	8(57,1)		
Response to ECT	8/6			4.489	0,055*
Full Response		8(100)	3(50)		
Partial Response		0(0)	1(16,7)		
No Response		0(0)	2(33,3)		
Number of ECT	11/14	10(0-12)	0(0-8)	38.5	**0,033[μ]**
History of Benzodiazepine Use	11/14			1.857	0,288*
Yes		8(72,7)	13(92,9)		
No		3(27,3)	1(7,1)		
Response to Benzodiazepines	8/13			0.493	>0,999*
Full Response		3(37,5)	4(30,8)		
Partial Response		4(50)	6(46,2)		
No Response		1(12,5)	3(23,1)		
Medical Comorbidity	11/14			0.698	0,604*
Yes		1(9,1)	3(21,4)		
No		10(90,9)	11(78,6)		
Familiy History of Psychiatric Disorder	11/14			0.172	>0,999*
Yes		4(36,4)	4(28,6)		
No		7(63,6)	10(71,4)		

*Pearson Chi-Square Test, Fisher's Exact Test, Fisher's Freeman Halton Exact Test, Yates Correction, n(%).

[μ]Mann Whitney U Test, *Med(IQR)*

0132 - Kleptomania and Hoarding Disorder with Animal Hoarding: A Challenging Case

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Introduction

Kleptomania is an impulse control disorder characterized by a recurrent failure to resist urges to steal objects that are not needed for personal use or monetary value, and it is frequently comorbid with many psychiatric conditions [1]. Patients with kleptomania typically experience increasing tension or excitement before the act, followed by relief and satisfaction after the theft [2]. However, they often experience guilt afterward, as the behavior conflicts with their social, professional, and family values and obligations. Diagnostic features include a strong and uncontrollable urge to steal. Despite considerable efforts to resist, patients are often unable to control these impulses and may engage in repeated theft behaviors, typically occurring several times per year. The incidence of kleptomania is very low in the general population, standing at about 0.3% - 0.6%. Approximately 4%-24% of the arrested shoplifters are reportedly kleptomania patients. In addition, more women get this disorder than men, with the female-to- male ratio being about 3:1 [3]. Nonetheless, the etiology and pathogenesis of kleptomania remain inconclusive. The possible etiological factors of kleptomania include genetic, familial, personality-related, social, and physiological factors [4].

Kleptomania is frequently associated with several psychiatric comorbidities, particularly mood disorders, anxiety disorders, obsessive-compulsive related disorders, and substance use disorders. Major depressive disorder is among the most commonly reported comorbid conditions [3].

Given the phenomenological and comorbid overlap between impulse control disorders and obsessive-compulsive related disorders, hoarding disorder may also be observed in patients with kleptomania. Hoarding disorder is classified under obsessive-compulsive and related disorders and is characterized by persistent difficulty discarding possessions regardless of their value, excessive acquisition, and marked clutter that compromises living areas. Though patients with hoarding disorder often hoard newspapers, old clothes, bags, and documents, any material can be hoarded. The main reasons why they cannot discard them are that those items are recognized as useful or beautiful, or they are strongly attached to them. Intense emotional attachment of subjects to objects is an essential feature of hoarding disorder[5]. A population- based study found that the overall prevalence of provisional hoarding disorder was around 2% From an etiological perspective, familial factors, neurodevelopmental disorders, and genetic conditions such as Huntington's disease have been suggested to contribute to the development of hoarding disorder [6].

Furthermore, animal hoarding a subtype of hoarding disorder is not recognized as a distinct diagnosis in DSM; however, it is defined by the accumulation of an unusually large number of animals, failure to provide adequate care, and consequent impairment in health, safety, and social or occupational functioning[6].

Treatment adherence is often low in individuals with these complex and difficult-to-manage conditions. To our knowledge, the co-occurrence of kleptomania and hoarding disorder has rarely been reported in the literature. Here, we present a 45-year-old woman with kleptomania, hoarding disorder including animal hoarding, and major depressive disorder, who has been under our follow-up for two years.

Case presentation

The patient presented with a more than 20-year history of collecting garbage, stealing small items such as cleaning supplies, fabric materials, and inexpensive accessories that were not needed for personal use, difficulty discarding household items, marked deterioration in self- care, and intense guilt and sadness following the loss of a pet. Additionally, she reported

having adopted more than 15 cats. Further clinical interviews revealed a history of traumatic life experiences which were considered possible contributing factors to her emotional dysregulation and psychiatric symptoms. Additionally, she had a history of multiple suicide attempts.

Mental status examination revealed reduced self-care, an anxious appearance, and partial insight, with no psychotic features observed. The Beck Depression Inventory score indicated severe depression. MMPI findings suggested impulsivity, emotional lability, difficulties in anger control, and a potential tendency toward behaviors violating social norms. Neurological evaluation and brain magnetic resonance imaging findings were within normal limits.

Treatment was initiated with clomipramine, followed by dose escalation and augmentation. Additionally, monthly supportive psychotherapy sessions were conducted By the third month of follow-up, the treatment regimen was adjusted to clomipramine 225 mg/day, risperidone 1 mg/day, and lamotrigine 100 mg/day. By the sixth month of follow-up, a marked reduction in depressive symptoms and an improvement in self-care were observed. Although she experienced intermittent passive suicidal ideation during the follow-up period, no suicide attempts were reported. Kleptomania behaviors, which initially occurred 4-5 days per week, decreased to a few times per month, and the patient began returning the items she had taken. Due to persistent irritability and ongoing behaviors causing harm to the environment, the risperidone dose was subsequently increased to 3 mg/day.

At the second year of follow-up, she demonstrated improved insight into her condition. Re- evaluation with the MMPI indicated that her psychological difficulties were still prominent and that her ego strength remained insufficient to cope effectively with these problems. However, a significant reduction in anxiety levels was observed. Additionally, a reduced tendency toward somatization was noted. Assessment with the Beck Depression Inventory revealed that she continued to exhibit moderate depressive symptoms.

Consistent with clinical observations, improvements were noted in the patient's self-care, sleep pattern, and motivation to participate in social activities. Additionally, at the end of the two- year follow-up, an improvement in self-esteem and a reduction in feelings of guilt were observed. Although she experienced a few severe episodes of stealing during the follow-up period, the frequency of stealing behavior decreased to once or twice every few months, and the patient completely stopped collecting items from garbage. She was also able to discard a substantial amount of accumulated belongings. Similarly, a reduction in the number of animals and objects accumulated by the patient was observed, along with behaviors aimed at decreasing her existing hoarding.

In conclusion, although the patient's treatment is still ongoing, significant improvement has been observed in her mood symptoms, kleptomania behaviors, and hoarding behaviors. Verbal informed consent was obtained from the patient for the publication of this case report.

Conclusion

Kleptomania is a rare and often underrecognized psychiatric disorder. The presence of comorbid conditions such as major depressive disorder and hoarding disorder may further complicate the clinical course, resulting in difficulties in treatment adherence and long-term management.

According to the existing literature, familial risk factors and adverse childhood traumatic experiences may contribute to its etiology. The addition of guilt and shame associated with kleptomania to depressive symptoms may increase the risk of suicidal ideation and behaviors. Although it may represent one of the most critical aspects requiring careful clinical evaluation and management, the assessment of suicide risk in such complex cases has not been sufficiently emphasized in the literature. In this context, comprehensive psychiatric assessment, a supportive psychotherapeutic approach, enhancement of the patient's insight into her illness, and effective management of guilt and shame are of critical importance, particularly in socially isolated patients such as our case.

In terms of medical treatment, inconsistent results have been reported with different medical treatment approaches, including SSRIs and SNRIs, while some studies have demonstrated the potential benefit of naltrexone [7].

On the other hand, in individuals with kleptomania or hoarding disorder, personality characteristics such as lack of empathy, low self-esteem, and social isolation, as well as intense stress, anxiety, depressive symptoms, and difficulty expressing negative emotions, highlight the importance of including psychotherapy as part of a multimodal treatment approach [4].

As demonstrated in our case, even partial clinical improvement may provide clinically relevant information. This case highlights the importance of individualized and long-term follow-up in patients with complex psychiatric comorbidities. We hope that this case may be helpful for clinicians in managing similar cases, which can be challenging both clinically and forensically. The patient's follow-up is ongoing, and further studies are needed to improve treatment approaches for this population.

Keywords: Animal Hoarding, Hoarding Disorder, Impulse Control Disorders, Kleptomania

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0136 - Psychotropic Recommendations Following Child and Adolescent Psychiatry Consultation in a Pediatric Burn Unit: A Five-Year Retrospective Review

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Objective

Pediatric burns are complex clinical conditions extending beyond tissue injury, involving pain, repeated wound care procedures, visible bodily involvement, prolonged hospitalization, and substantial family-related stress. Children and adolescents with burn injuries are considered a psychologically vulnerable group, particularly regarding anxiety, acute stress symptoms, trauma-related reactions, and longer-term psychosocial difficulties, and psychological support for both the child and family should be considered an integral part of care (1,2). Data from Turkiye likewise suggest that psychopathology in pediatric burn patients is clinically significant and may be associated with burn percentage and the number of affected body regions (3).

Pediatric consultation-liaison psychiatry addresses psychiatric symptoms arising in the context of medical illness and hospitalization, including adjustment difficulties, behavioral dysregulation, treatment nonadherence, and the need for psychopharmacological support (4). In this context, psychiatric consultation in a burn unit may contribute not only to diagnostic clarification but also to symptom relief and treatment continuity. This study aimed to examine the pattern of psychotropic recommendations following child and adolescent psychiatry consultation in a pediatric burn unit, together with consultation reasons, clinical impressions, and reconsultation needs.

Methods

This single-center retrospective descriptive chart review included patients hospitalized in the pediatric burn unit between November 2020 and November 2025 who received child and adolescent psychiatry consultation during admission. Variables examined were age, sex, burn etiology, burn extent and depth, affected body regions, surgical history, length of stay, day of psychiatric consultation, consultation reasons, clinical impressions/preliminary diagnoses, recommended non-pharmacological and pharmacological interventions, and reconsultation characteristics. Data were summarized using descriptive statistics. Correlation analyses evaluated the relationships between number of reconsultations and length of stay, number of operations, and burn percentage. Additional group comparisons were conducted between patients who received any pharmacological recommendation at any point during hospitalization and those who did not, with respect to length of stay and reconsultation.

Results

During the five-year study period, a total of 1,171 patients were hospitalized in the pediatric burn unit, and 8 patients died during this period. The psychiatric consultation sample consisted of 174 consultations across 106 admissions.

In the consultation sample, mean age was 105.32±60.42 months, and 59.4% of patients were male. The most common burn etiologies were scald burns (46.7%, n=50) and flame burns (43.9%, n=47). Extensive burns were present in 78.5% of cases, and second-degree burns in 70.1%. Facial involvement was observed in 44.3%, genital involvement in 25.5%, trunk involvement in 54.0%, upper extremity involvement in 66.0%, and lower extremity involvement in 60.0%. 60.7% of patients underwent surgery. Mean length of hospital stay was 29.88±25.23 days, and the mean day of psychiatric consultation was 9.84±12.40. In this sample, 104 patients were discharged alive and 2 died (1.9%); functional loss was documented in 24.3% of cases.

Table l.Sociodemographic and clinical characteristics of the consultation sample

Variable	Value
Age (months), mean ± SD	105.32 ± 60.42
Male sex, n (%)	63 (59.4)
Burn etiology, n (%)	
Scald burn	50 (46.7)
Flame burn	47 (43.9)
Contact burn	4 (3.7)
Electrical burn	3 (2.8)
Chemical burn	2 (1.9)
Burn extent, n (%)	
Extensive burn	84 (79.2)
Localized burn	22 (20.7)
Burn degree, n (%)	
Second-degree burn	75 (70.1)
Mixed second- and third-degree burn	28 (26.2)
Third-degree burn	3 (2.8)
Burn localization, n (%)	
Facial involvement	47 (44.3)
Genital involvement	27 (25.5)
Trunk involvement	58 (54.0)
Upper extremity involvement	70 (66.0)
Lower extremity involvement	64 (60.0)
Surgery performed, n (%)	65 (60.7)
Length of stay (days), mean ± SD (min-max)	29.88 ± 25.23 (2-139)
Day of consultation, mean ± SD (min-max)	9.84 ± 12.40 (1-97)
Discharge status, n (%)	Alive 104 (97.2) / Deceased 2 (1.9)
Functional loss, n (%)	26 (24.3)

The most common consultation reason was agitation and treatment nonadherence (48.6%, n=52). Among clinical impressions/preliminary diagnoses, adjustment disorder (41.1%, n=44) ranked first, followed by general psychiatric evaluation (39.3%, n=42). Acute stress disorder or post-traumatic stress disorder was identified in 11.2% (n=12), anxiety or depression in 2.8% (n=3), substance use in 3.7% (n=4), and delirium in 0.9% (n=1). Non-pharmacological interventions ranked first among recommended interventions (51.4%, n=55). Among pharmacological recommendations, antipsychotics accounted for 27.3% (n=29), antihistamines for 10.3% (n=11), benzodiazepines for 6.5% (n=7), and selective serotonin reuptake inhibitors (SSRIs) for 3.7% (n=4). Among cases in which psychotropic treatment was recommended, the most frequently used agents were risperidone (54.9%, n=28/51) and hydroxyzine (21.5%, n=11/51). When the entire hospitalization period, including reconsultations, was considered, the number of patients who received at least one pharmacological recommendation increased to 58

(54.7%). Reconsultation was required in 30.8% (n=33) of cases. The number of reconsultations was positively correlated with length of stay (p=0.393, p<0.001), number of operations (p=0.439, p<0.001), and burn percentage (TBSA) (p=0.347, p<0.001). Length of stay was also positively correlated with number of operations (p=0.787, p<0.001) and burn percentage (p=0.683, p<0.001).

Additional analyses showed that patients who received a pharmacological recommendation at any point during hospitalization had a significantly longer length of stay (36.0±30.3 days; median 24.0 [14.25-50.5]) than those who did not (22.5±14.4 days; median 19.5 [11.0-33.25], p=0.031). Reconsultation was also more frequent in the pharmacologically treated group (44.8% vs. 20.8%, p=0.013), and the number of reconsultations was higher (0.98 vs. 0.23, p=0.006). Pharmacological treatment requirement was positively associated with length of stay (p=0.228, p=0.019), number of reconsultations (p=0.302, p=0.002), and number of operations (p=0.258, p=0.008), whereas its association with burn percentage was not significant (p=0.155).

Table 2. Reconsultation and related clinical variables

Association	P	p
Number of reconsultations ↔ length of stay	0.393	<0.001
Number of reconsultations ↔ number of operations	0.439	<0.001
Number of reconsultations ↔ burn percentage (TBSA)	0.347	<0.001
Length of stay ↔ number of operations	0.787	<0.001
Length of stay ↔ burn percentage (TBSA)	0.683	<0.001
Pharmacological treatment requirement ↔ length of stay	0.228	0.019
Pharmacological treatment requirement ↔ number of reconsultations	0.302	0.002
Pharmacological treatment requirement ↔ number of operations	0.258	0.008

Discussion

This five-year review suggests that the need for child and adolescent psychiatry consultation in the pediatric burn unit becomes most visible in the domains of behavioral regulation and treatment collaboration. The predominance of agitation and treatment nonadherence as consultation reasons, together with the high frequency of adjustment disorder as the leading clinical impression, indicates that psychiatric need in this setting often reflects emotional and behavioral symptoms that complicate acute medical care rather than severe enduring psychiatric conditions. This is consistent with pediatric consultation-liaison psychiatry literature, in which consultations are frequently requested because of difficulty adjusting to medical illness, treatment nonadherence, and the need for psychopharmacological support (4).

The intervention pattern supports this interpretation. Non-pharmacological approaches were the most common initial recommendation, suggesting that psychiatric input was primarily organized around environmental regulation, supportive interviewing, behavioral de-escalation, and team collaboration. However, the fact that nearly half of the patients received a pharmacological recommendation at the first evaluation, and more than half did so when the entire hospitalization period was considered, indicates that psychosocial and behavioral interventions alone were insufficient in a substantial subgroup. In these cases, psychotropic treatment appears to have been used primarily to reduce symptoms that interfered with wound care, procedures, ward routine, and overall treatment adherence.

Within this framework, the prominence of risperidone and hydroxyzine may be understood as reflecting a short-term, symptom-oriented strategy. Marzullo (2014) emphasized that agitation in children should be approached within a broad differential framework and that pharmacological intervention may be considered in selected cases to improve safety, behavioral control, and continuation of medical care(5). In our sample, psychotropic recommendations seem to have functioned less as diagnosis- specific long-term treatment strategies and more as

pragmatic consultation-liaison interventions aimed at enhancing treatment adherence, reducing behavioral dysregulation, and maintaining continuity of care.

It is also noteworthy that patients requiring pharmacological treatment had longer hospital stays and higher reconsultation needs. The positive associations between pharmacological treatment requirement and length of stay, number of reconsultations, and number of operations suggest that psychopharmacological intervention was not selected routinely, but rather in patients with more complex clinical courses and greater behavioral burden. Likewise, the association of reconsultation number with length of stay, number of operations, and TBSA suggests that psychiatric need often extends beyond a single bedside assessment. These findings support the value of earlier and more longitudinal psychiatric involvement in patients with more severe burn burden and more complicated inpatient courses.

Our results are consistent with the literature identifying pediatric burn patients as a psychologically vulnerable population. Woolard et al highlighted the important psychological consequences of pediatric burns(1), while Bakker et al emphasized the emotional burden experienced by both children and families, especially in the early period after injury(2). Kara9etin et al also demonstrated associations between burn- related clinical factors and psychopathology in a Turkish sample(3). Our findings extend this literature by showing that psychiatric consultation in pediatric burn settings should be understood not only in relation to trauma-related diagnoses, but also in terms of treatment adherence, behavioral regulation, psychotropic treatment need, and reconsultation patterns.

The main limitations of this study are its retrospective design, single-center setting, and inclusion of only those patients for whom psychiatric consultation was requested. Accordingly, the findings reflect not the prevalence of psychopathology among all pediatric burn patients, but rather the pattern of clinical need requiring psychiatric intervention. In the future, studies could be designed to include psychiatric evaluations and monitoring of all patients followed in the pediatric burn unit. Nevertheless, the study is noteworthy in systematically presenting 5 years of institutional data and in making visible the psychotropic recommendations given particularly in the context of adjustment-related treatment nonadherence.

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0141 - Symptom Predominance and Duration of Untreated Psychosis in First-Episode Schizophrenia: A Retrospective Inpatient Study

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Introduction

Schizophrenia may emerge through markedly different clinical trajectories. In some individuals, onset is acute and characterized by florid, disruptive positive symptoms such as hallucinations, delusions, and disorganized behaviour that rapidly precipitate clinical attention (1). In others, the illness develops insidiously, with a prolonged prodromal phase dominated by negative symptoms including social withdrawal, avolition, and affective flattening (2). These early changes are often subtle, nonspecific, and easily misattributed to psychosocial stressors or personality traits, thereby delaying recognition and prolonging the duration of untreated psychosis (DUP). Thus, DUP is not solely a measure of healthcare delay but is closely intertwined with illness phenomenology and pathways to care.

This heterogeneity reflects longstanding conceptualizations of schizophrenia as a spectrum rather than a unitary disorder (3). Bleuler emphasized the diversity of schizophrenic presentations, and earlier diagnostic systems attempted to capture this variability through subtypes such as paranoid and disorganized schizophrenia (4). Although such subtyping has been largely abandoned in contemporary nosology (2), differences in onset patterns—acute versus insidious—may still represent clinically meaningful dimensions.

DUP has consistently been associated with poorer symptomatic, functional, and quality-of-life outcomes (5-7). Evidence suggests a non-linear relationship, with early delays exerting disproportionately harmful effects (6). However, the interpretation of DUP remains complex. Longitudinal studies demonstrate heterogeneous trajectories across symptom domains (7), and more recent work highlights the role of methodological confounds such as lead-time bias (8). Within this context, negative symptom predominance may contribute to prolonged DUP through reduced symptom visibility and impaired help-seeking behavior.

The present study aimed to examine whether symptom predominance during the untreated phase of psychosis is associated with differences in DUP in patients hospitalized for first- episode schizophrenia (FES). We hypothesized that negative symptom predominance would be associated with significantly longer DUP compared to positive symptom predominance.

Methods

Study Design and Setting

This retrospective case-control study was conducted at Ba§ak§ehir £am ve Sakura City Hospital, a tertiary psychiatric center in Istanbul, Turkiye. Ethical approval was obtained from the institutional Scientific Research Ethics Board (No. 1; approval date: 24 September 2025; decision no: 288).

Participants

Electronic medical records were reviewed to identify patients admitted with a first episode of psychosis between January 2020 and December 2022. Inclusion criteria were: (i) age between 15 and 45 years, (ii) inpatient admission for an acute psychotic episode, (iii) no prior diagnosis of a psychotic disorder, (iv) no history of antipsychotic treatment or psychiatric hospitalization, and (v) a confirmed diagnosis of schizophrenia at index hospitalization or during follow-up.

Exclusion criteria included organic psychoses, affective psychoses, substance-induced psychotic disorders, concomitant substance use disorders and neurological or medical conditions that could confound psychiatric presentation.

Data Collection and Measures

84 individuals were identified who met the operational definition of first-episode psychosis and all inclusion criteria following a meticulous exclusion process. For these participants, the following demographic and clinical variables were extracted from the detailed case descriptions in their initial admission files and recorded using standardized data collection forms developed by the research team: sex (male/female), age at illness onset (years), duration of first hospitalization (days), duration of untreated psychosis (weeks), predominant symptom profile during the untreated psychosis period (positive vs. negative symptom predominance), marital status at illness onset (married/single), educational level at illness onset (none/primary/middle/high/university), employment status at illness onset (employed/unemployed), living arrangements at illness onset (with parents and/or siblings; with spouse and/or children; living alone), receipt of electroconvulsive therapy during the index admission (yes/no), and smoking status at illness onset (smoker/non-smoker).

Patients were categorized into two groups according to symptom predominance during the untreated phase:

- **Positive symptom predominance** (e.g., delusions, hallucinations, disorganized behavior)
- **Negative symptom predominance** (e.g., social withdrawal, affective flattening, avolition)

Symptom predominance was determined through clinical records at admission and retrospective evaluation of symptom trajectories prior to treatment. All cases were independently evaluated from a phenomenological perspective by both authors under blinded conditions. In instances of disagreement regarding symptom predominance, the final classification was determined by the senior clinician (OG).

Statistical Analysis

Statistical analyses were conducted using Jamovi (Version 2.6.45.0). Continuous variables were assessed for distributional properties. Between-group comparisons were performed using the Mann-Whitney U test due to non-normal distribution of DUP. Statistical significance was set atp < 0.05. Effect size was calculated to estimate the magnitude of differences.

Results

Systematic sampling was conducted by excluding cases diagnosed with manic episodes with psychotic features, depressive episodes with psychotic features, substance-induced psychosis, organic mental disorders, and peripartum psychosis from the institutional database. This process identified 84 patients who were admitted to our facilities with a clinical presentation consistent with first-episode psychosis and who were subsequently diagnosed with schizophrenia during long-term follow-up. None of the patients reported alcohol or substance use at illness onset or at the time of the present assessment.

Certain demographic and clinical characteristics of these initial 84 patients are presented in Table 1 and 2. Patients who suffered more predominantly from negative symptoms throughout their pre-diagnosis untreated psychosis phase had longer DUP (98.2±59.85 vs. 33.7±55.19, statistic: 242, p<0.001, effect size: 0.623) (Table 3).

Discussion

In this study, we found a clear association between longer duration of untreated psychosis (DUP) and negative symptom predominance during the untreated phase of first-episode schizophrenia. Patients presenting with predominantly negative symptoms experienced substantially longer delays before treatment initiation compared to those with positive symptom predominance. This relationship was not accompanied by differences in age at illness onset or duration of first hospitalization, suggesting that DUP is more closely linked to early illness phenomenology than to general markers of severity or service-related factors.

These findings intersect with three major theoretical frameworks regarding the meaning of DUP: as a causal determinant of outcome (5,6), as an epiphenomenon of illness characteristics such as mode of onset and premorbid trajectory (9), and as an artifact influenced by lead-time bias (8). The traditional view conceptualizes DUP as a modifiable risk factor, with longer untreated periods exerting a deleterious effect on illness trajectory. Evidence supporting a curvilinear relationship—where early delays are particularly harmful—has reinforced the notion of a critical intervention window (6). Within this framework, prolonged DUP in patients with negative symptom

predominance could be interpreted as contributing directly to poorer outcomes through cumulative biological or psychosocial effects.

However, our findings more strongly support an alternative interpretation: that DUP is, at least in part, an epiphenomenon of illness onset type. Negative symptom predominance is inherently characterized by insidious onset, reduced behavioral disruption, and diminished help-seeking, all of which contribute to delayed recognition (10). In contrast, positive symptoms are typically disruptive and rapidly prompt clinical attention (10). Thus, DUP may not simply reflect treatment delay but rather differences in illness visibility and trajectory, with negative symptom profiles generating longer untreated periods.

This interpretation is further reinforced by the lead-time bias hypothesis, which posits that patients with shorter DUP are identified earlier in their illness course, whereas those with longer DUP present at a more advanced stage (8). Longitudinal evidence suggests that when illness course is aligned to psychosis onset rather than treatment initiation, the predictive value of DUP diminishes, supporting its role as an index of illness stage rather than a causal factor (8). At the same time, other long-term studies indicate that shorter DUP remains associated with better outcomes across multiple domains, suggesting a complex and domain-specific relationship (7).

Taken together, these perspectives suggest a hybrid model in which DUP reflects a complex interplay between illness phenomenology, detection processes, and methodological factors. Within this framework, negative symptom predominance may represent a distinct illness pathway characterized by insidious onset and delayed detection. Although contemporary diagnostic systems have moved away from subtyping, our findings support the relevance of phenomenology-informed stratification, where early symptom profiles may index meaningful variation within the schizophrenia spectrum.

Clinically, these results underscore the need to expand early detection strategies beyond overt psychotic symptoms to include subtle negative and prodromal features. At the same time, caution is warranted in interpreting DUP as a purely modifiable prognostic factor. Future longitudinal studies, particularly those anchoring analyses to symptom onset, are needed to disentangle causal effects from illness trajectory and detection bias.

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Tables

Table 1. Demographic Characteristics of Total Sample of Inpatients Diagnosed as First Episode Schizophrenia (n=84)

Gender	Male	59	70.2%
	Female	25	29.8%
Marital Status at Illness Onset	Single	67	79.8%
	Married	17	20.2%
Level of Education	None	4	6.1%
	Primary	10	15.2%
	Middle	17	25.8%
	High	21	31.8%
	University	14	21.2%
Working Status at Illness Onset	Working	22	26.2%
	Not Working	62	73.8%
Co-Living Status at Illness Onset	With Parents and/or Siblings	63	75.0%
	With Spouse	16	19.0%
	Alone	5	6.0%
	Mean+/-SD	Minimum	Maximum
Age (Years)	28.5+/-7.70	18	48

Table 2. Clinical Characteristics of Total Sample of Inpatients Diagnosed as First Episode Schizophrenia (n=84)

Dominant Clinical Features During the Period of Untreated Psychosis	Positive Symptoms Dominant	64	76.2%
	Negative Symptoms Dominant	20	23.8%
Tobacco Use Disorder at Illness Onset	Yes	51.2	26.2%
	No	48.8	73.8%
ECT Receival Status During First Hospitalization	Yes	10	11.9%
	No	74	88.1%
	Mean+/-SD	Minimum	Maximum
Duration of Untreated Psychosis (Weeks)	49.0+/-62.4	1	312
Duration of First Hospitalization (Days)	31.0+/-16.5	4	87

Table 3. Comparison of Age at Illness Onset, Duration of First Hospitalization, and Duration of Untreated Psychosis Among First Episode Schizophrenia Patient by Positive or Negative Symptom Predomination Throughout Duration of Untreated Psychosis

	GV	n	Mean+-SD	Statistic*	p	ES
Age at Illness Onset (Years)	Positive Symptoms Dominant	64	28.9+-8.01	557	0.383	-0.130
	Negative Symptoms Dominant	20	27.2+-6.66			
Duration of First Hospitalization (Days)	Positive Symptoms Dominant	64	31.4+-15.88	561	0.409	-0.123
	Negative Symptoms Dominant	20	29.9+-18.75			
Duration of Untreated Psychosis (Weeks)	Positive Symptoms Dominant	64	33.7+-55.19	242	<0.001	0.623
	Negative Symptoms Dominant	20	98.2±59.85			

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0146 - Serum Substance P, Neuropeptide Y, Neuropeptide S and Spexin Levels in Children with Tic Disorders: Associations with Sensory Sensitivity and Allergic Inflammatory Markers

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Objective

Tic disorders are neurodevelopmental conditions characterized by sudden, rapid, recurrent motor movements or vocalizations that typically emerge during childhood [1]. These disorders are associated with alterations in cortico-striato-thalamo-cortical (CTSC) circuits as well as dysregulation in neurotransmitter and neuropeptide systems [2]. Increasing evidence also suggests a potential role of immune and inflammatory mechanisms in the pathophysiology of tic disorders [3].

Clinical and epidemiological studies have indicated that tic disorders may co-occur with immune-related conditions such as allergic diseases and heightened sensory sensitivity. Sensory hypersensitivity is frequently reported in children with tic disorders and may contribute to tic expression or exacerbation [4]. At the same time, allergic inflammation and immune activation have been proposed as potential contributors to neurodevelopmental psychiatric conditions through neuroimmune interactions [3, 4].

Neuropeptides are important neuroendocrine mediators involved in stress regulation, immune modulation, and neuroinflammatory processes. neuropeptide Y (NPY) has been associated with stress regulation and immune function [5], whereas substance P (SP) plays a role in neurogenic inflammation and sensory processing [6]. Neuropeptide S (NPS) is involved in arousal and stress responses [5], and spexin (SPX) has recently been described as a neuropeptide involved in metabolic and neuroendocrine regulation [7].

This study aimed to evaluate serum levels of substance P, neuropeptide Y, neuropeptide S, and spexin in children and adolescents with tic disorders and to investigate their associations with sensory sensitivity, allergic inflammatory markers, and tic severity.

Methods

Participants

This cross-sectional study included 40 children and adolescents aged 8-18 years diagnosed with tic disorders according to DSM-5 diagnostic criteria and presenting with active tic symptoms. A control group consisting of 40 age- and sex-matched healthy participants without a history of psychiatric or neurological disorders was also included. Participants were recruited from a child and adolescent psychiatry outpatient clinic.

individuals with intellectual disability, autism spectrum disorder, psychotic disorders, mood disorders, substance use disorders, chronic neurological or systemic medical illnesses (e.g., cardiovascular, metabolic, genetic, endocrine, or rheumatologic disorders), severe head trauma, or organic brain injury were excluded. Participants with obesity (BMI >30 or weight >97th percentile), a history of infection within the previous month, recent antibiotic use, or corticosteroid or immunomodulatory medication use within the previous six months were also excluded.

Clinical assessments

Sociodemographic data were collected using a structured sociodemographic data form. Tic severity was evaluated using the Yale Global Tic Severity Scale (YGTSS). Sensory sensitivity was assessed using the 12-item self-report short form of Highly Sensitive Child Scale (HSC). The presence of allergic diseases was assessed using the International Study of Asthma and Allergies in Childhood (ISAAC) questionnaire.

Laboratory measurements

Blood samples were collected from all participants to evaluate serum levels of neuropeptides and allergic inflammatory markers. Serum concentrations of substance P, neuropeptide Y, neuropeptide S, and spexin were measured using enzyme-linked immunosorbent assay (ELISA) according to the manufacturer's protocols. In addition, allergic inflammatory parameters including immunoglobulin E (IgE) levels and eosinophil counts were assessed.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics version 26.0. Descriptive statistics were calculated for all variables. Group differences were analyzed using independent samples t-tests or Mann-Whitney U tests, depending on data distribution. Categorical variables were compared using the Pearson chi-square test. Associations between neuropeptide levels, allergic inflammatory parameters, and clinical variables were examined using Spearman correlation analysis. Generalized linear models (GLM) were used to identify factors associated with neuropeptide levels, and logistic regression analysis was performed to evaluate predictors of tic severity. A p value < 0.05 was considered statistically significant.

Results

The study included 40 children and adolescents with tic disorders and 40 healthy controls. Among the patients, 82.5% were diagnosed with Tourette syndrome and 17.5% with chronic motor tic disorder.

At least one psychiatric comorbidity was present in 77.5% of the participants. The most frequent comorbid conditions were attention deficit hyperactivity disorder (ADHD) (60%) and obsessive-compulsive disorder (OCD) (35%).

Comparisons of serum neuropeptide and allergic inflammatory parameters between the tic disorder and control groups are presented in Table 1. Serum NPY levels were significantly higher in the tic disorder group compared with controls ($p = 0.003$). Serum SPX levels were also higher in the patient group; however, this difference did not reach statistical significance ($p = 0.060$). No statistically significant differences were observed between the groups with respect to SP, NPS, IgE levels, or eosinophil counts. The distribution of serum neuropeptide levels in both groups is illustrated in Figure 1.

Correlation analyses revealed that serum NPY levels were negatively correlated with motor tic severity, vocal tic severity, overall impairment, and total tic severity scores. In addition, serum SPX levels were negatively correlated with overall impairment scores, while serum IgE levels were positively correlated with motor tic severity and overall impairment scores. A significant positive association was also observed between serum SP levels and the "discomfort with sensory stimuli" subscale of the 12-item self-report form of the Highly Sensitive Child Scale.

Logistic regression analysis, controlling for potential confounding variables including age, BMI, ADHD, and OCD, demonstrated that serum SPX and NPY levels were significant predictors of tic severity (Table 2).

Discussion

The present study investigated the relationship between neuropeptide systems, allergic inflammatory parameters, sensory sensitivity, and tic severity in children and adolescents with tic disorders. The findings suggest that neuropeptide systems, particularly NPY and SPX, may be involved in the biological mechanisms underlying tic disorders.

One of the most notable findings of the present study was the significantly higher serum NPY levels observed in the tic disorder group compared with controls. Interestingly, despite these elevated levels, serum NPY levels were negatively correlated with tic severity and emerged as an independent predictor of tic severity. This pattern may indicate a compensatory neurobiological response. NPY is known to exert inhibitory and stress-buffering effects within CSTC circuits and has been associated with resilience to stress-related neuropsychiatric conditions [8]. Increased NPY levels in children with tic disorders may therefore represent an adaptive response aimed at counteracting heightened neuronal excitability and stress-related dysregulation. However, the negative association between NPY levels and tic severity suggests that this compensatory system may become insufficient as tic symptoms become more severe. Previous studies in high-stress populations, including military personnel exposed

to extreme stress, have shown that although NPY levels increase during acute stress, prolonged or overwhelming stress may lead to reductions in NPY levels, possibly reflecting the limits of this regulatory mechanism [9].

A similar pattern was observed for SPX. Although serum SPX levels were higher in the tic disorder group, they were negatively associated with clinical severity. This finding suggests that SPX may not merely reflect disease presence but may represent activation of a regulatory system engaged in response to stress and emotional burden. Experimental evidence indicates that SPX interacts with neuroendocrine stress pathways, including CRH and serotonergic systems involved in emotional regulation [7]. In this context, increased serum SPX levels in patients with tic disorders may reflect a compensatory response aimed at modulating stress- related neurobiological processes.

The positive correlations between serum IgE levels and tic severity measures further support the potential contribution of immune-related mechanisms to the pathophysiology of tic disorder. Previous studies have suggested that immune activation and inflammatory processes may influence neurodevelopmental psychiatric conditions through neuroimmune interactions [3].

Another interesting finding was the association between serum SP levels and sensory sensitivity, particularly the discomfort response to sensory stimuli. SP is known to be involved

in neurogenic inflammation and sensory processing, which may explain its association with sensory hypersensitivity observed in the present study [6].

Several limitations should be considered when interpreting these findings. First, the cross- sectional design of the study limits causal interpretations. Second, the relatively modest sample size may have limited the ability to detect smaller effect sizes. Additionally, serum neuropeptide levels may not fully reflect central nervous system activity. Despite these limitations, the present study provides novel evidence suggesting that neuropeptide systems and immune- related processes may be associated with the clinical characteristics of tic disorders.

Conclusion

In conclusion, the findings of this study suggest that neuropeptide systems and allergic inflammatory mechanisms may be associated with the clinical presentation of tic disorders in children and adolescents. The inverse relationships observed between NPY and SPX levels and tic severity may indicate potential regulatory roles of these neuropeptides, whereas the positive associations between IgE levels and tic severity support the contribution of immune-related mechanisms. Further longitudinal studies with larger samples are needed to clarify the role of these biological pathways in the pathophysiology of tic disorders.

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Tables and Figures

Table 1. Comparison of allerjic inflammatory parameters and serum neuropeptide levels between the tic disorder and control groups

Variable	Tic disorder group		Control group		Test stat.	p
	Mean (SD)	Median (min-max)	Mean (SD)	Median (min-max)		
Eosinophil	0,21 (0,18)	0,17 (0,04 - 1,07)	0,25 (0,22)	0,21 (0,03 - 1,3)	1,358	0,170
lgE	2964,3 (822,3)	3218,06 (1019,0- 4839,7)	2595,38 (991,9)	2730,22 (58,0 - 3649,9)	1,689	0,090
Substance P	465,67 (514,3)	251,36 (78,5 - 2150,0)	389,17 (464,8)	254,09 (53,1 - 2277,8)	0,674	0,500
Neuropeptide Y	1185,34 (643,0)	1433,28 (105,9 - 1980,8)	745,76 (606,0)	601,84 (83,3 - 1920,2)	2,942	**0,003***
Neuropeptide S	775,81 (95,6)	786,27 (533,0 - 917,6)	730,98 (144,7)	749,49 (305,3 - 990,5)	1,477	0,140
Spexin	531,4 (494,9)	294,36 (100,1 - 2168,5)	327,2 (293,8)	240,71 (83,4 - 1539,5)	1,833	0,060

Table 2. Logistic regression analysis of variables predicting tic severity

	B	SE	95 % CI		Wald χ^2	Exp(B)	p
			Lower CI	Upper CI			
Age	0,017	0,016	0,987	1,049	1,212	1,017	0,271
BMI	0,083	0,122	0,855	1,381	0,457	1,086	0,499
Spexm	-0,004	0,002	0,993	1,000	4,829	0,996	**0,028***
Neuropeptide Y	-0,002	0,001	0,997	1,000	5,095	0,998	**0,024***
Constant	-1,165	3,058	-	-	0,145	0,312	0,703

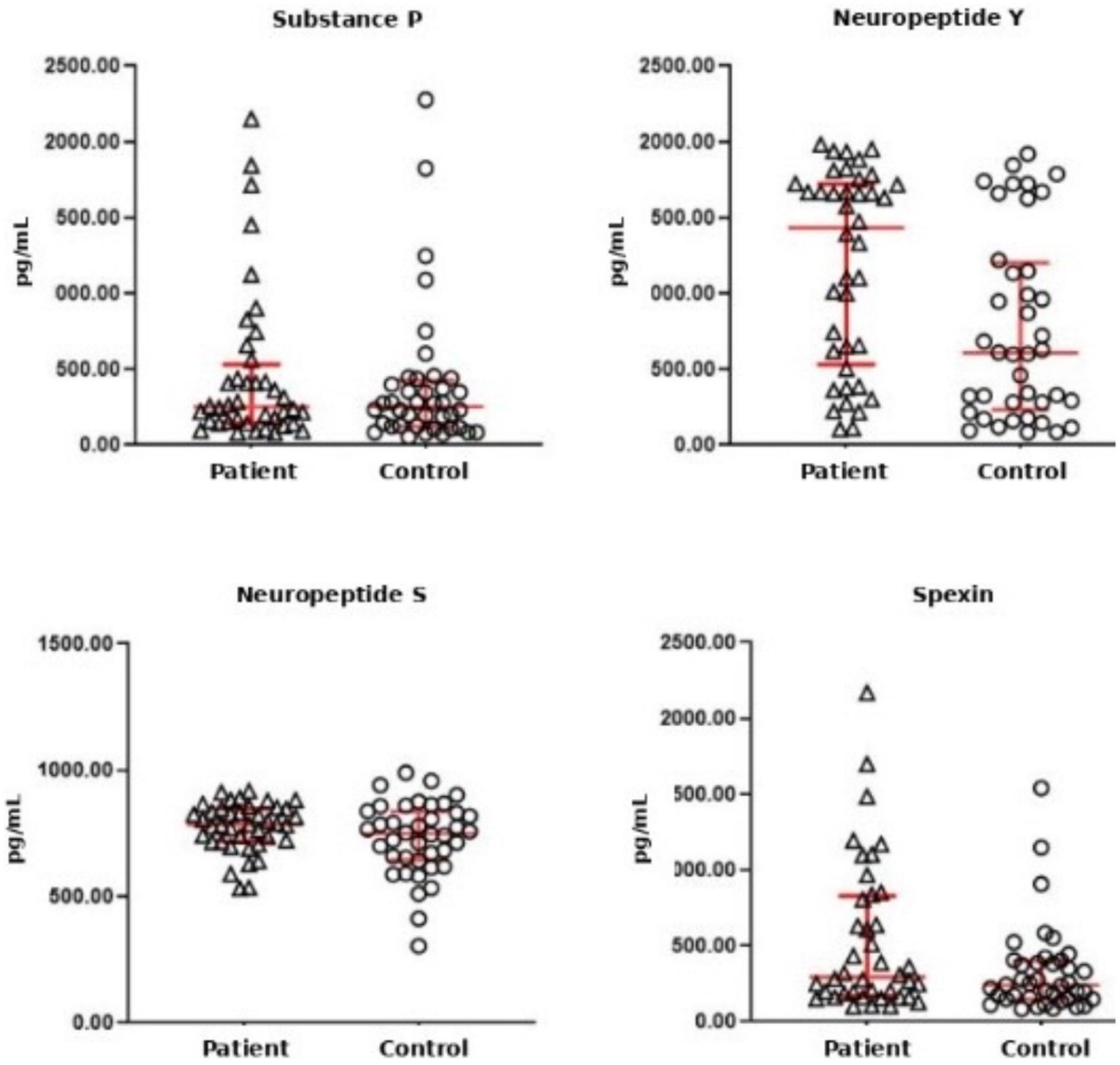


Figure 1. Comparison of serum neuropeptide levels between the tic disorder and control groups.

The red horizontal line represents the median value, and the short vertical bars indicate the interquartile range (Q1-Q3).

0153 - Kalp Hizi Değişkenliğinin Temelleri ve Fizyolojisi

Kalp hızı değişkenliği (HRV), ardışık kalp atışları arasındaki zaman aralıklarının (R-R aralıkları) sürekli değişim göstermesidir. HRV, otonom sinir sisteminin (OSS) kalp üzerindeki anlık etkilerini yansıtan önemli bir fizyolojik göstergedir; anlık kalp hızı ve R-R mesafelerindeki bu doğal dalgalanmalar, sempatik ve parasempatik sinir sistemleri arasındaki etkileşimin bir sonucudur. Normalde sağlıklı bireylerde kalp atımları arasındaki süreler tamamen eşit değildir; solunum gibi ritmik süreçler nedeniyle kalp hızı inspirasyon sırasında artar, ekspirasyon sırasında yavaşlar. Bu olgu solunumsal sinüs aritmisi (respiratory sinus arrhythmia, RSA) olarak bilinir ve HRV'nin en belirgin bileşenlerinden biridir. HRV'nin varlığı, kalbin çevresel ve içsel uyaranlara karşı adaptif yanıt verebilmesini sağlar ve bu nedenle yüksek HRV genellikle kardiyovasküler sağlığın ve otonomik esnekliğin bir göstergesi, düşük HRV ise stres, hastalık veya regülasyon kapasitesinin azalması ile ilişkilidir.

Otonom Sinir Sistemi ve HRV: Sempatik-Parasempatik Denge

Kalp hızı değişkenliğinin altında yatan temel fizyoloji, otonom sinir sisteminin iki dalı olan sempatik ve parasempatik (vagal) sinirlerin kalbe yaptıkları zıt fakat koordineli etkilerden kaynaklanır. Parasempatik sinirler (vagus siniri), istirahat halinde baskın olan dal olup kalp hızını yavaşlatır; sempatik sinirler ise kalp hızını hızlandırır ve vücudu strese yanıt vermeye hazırlar. Sağlıklı bir bireyde sinüs düğümü, bu iki sistemin tonik aktivitesinin dengesi altında çalışır.
Parasempatik (vagal) etki hızlı ve anlık gerçekleşir: Vagus sinirinden gelen tek bir uyarı bile kalp döngüsü içinde hemen iletilip bir-iki atım içinde kalp hızını yavaşlatabilir. Bu sayede vagal sistem, solunum gibi hızlı döngülere uyum sağlayarak HRV'nin yüksek frekanslı bileşenini (RSA) oluşturur. Sempatik etki ise görece yavaş ve kalıcıdır: Sempatik uyarının kalp hızını belirgin şekilde artırması birkaç saniyelik gecikmeyle olur (etkinin başlaması ~1-5 saniye, maksimuma ulaşması 20-30 saniye sürebilir) ve etkisi vagal çekilmeye göre daha uzun sürer. Sempatik aktivite, HRV'nin düşük frekanslı bileşenine kısmen katkıda bulunur, ancak tek başına HRV yaratmada parasempatik kadar hızlı değildir.

Kalpte OSS etkileri "accentuated antagonism" prensibiyle işler: Parasempatik ve sempatik sistemler birbirlerinin etkisini karşılıklı olarak modüle eder. Vagal aktivite, sempatik etkinin kalp üzerindeki etkisini anında baskılayabilir (yüksek vagal tonus, sempatik artışı maskeler), sempatik aktivite ise vagal etkinin geri çekilmesine yol açarak kalp hızını artırır. Bu dinamik denge sayesinde organizma, çevresel değişimlere uygun kalp hızı tepkileri verir: Örneğin ayağa kalkınca veya strese girince sempatik tonus artar (HRV azalır, kalp hızı yükselir), dinlenme veya güven hissinde vagal tonus artar (HRV yükselir, kalp hızı yavaşlar).

Özetle, HRV'nin fizyolojik temeli sempatik-parasempatik dengedeki anlık kaymalardır. HRV, kalp üzerinde baskın vagal frenin anlık değişimleri ile ortaya çıkar; vagus siniri devredeyken kalp atımları arasındaki aralıklar sürekli ufak dalgalanmalar gösterir. Sempatik sistem devraldığında ise kalp atımları daha ritmik ve tekdüze olur (HRV düşer). Bu nedenle yüksek HRV genellikle sağlıklı bir vagal tonus ve esnek bir OSS tepkisinin göstergesidir, düşük HRV ise sempatik aşırı aktivite veya yetersiz vagal modülasyon belirtisi olabilir.

Vagal Tonus ve Polivagal Teori

Vagal tonus, vagus sinirinin kalp üzerindeki etkinliğinin derecesini ifade eden bir kavramdır. Klinik olarak vagal tonus genellikle HRV ile ölçülür, çünkü HRV özellikle yüksek frekans bileşeniyle vagus aktivitesine duyarlıdır. Parasempatik aktivitenin ana taşıyıcısı olan n. vagus, kalpte sinüs düğümü ve AV düğümü inhibe ederek kalp atışlarını yavaşlatır. Dinlenme durumunda yüksek vagal tonus, kalbin düşük hızda ve değişken aralıklarla atmasını (yüksek HRV) sağlar. Bu, bedenin "güvenli ve sosyal" bir durumda olduğunun göstergesi olarak da yorumlanır.

Polivagal teoriye göre vagus siniri iki farklı dallanma gösterir: Miyelinli ventral vagus (kalbe ve yüz kaslarına giden kısım) ve myelinsiz dorsal vagus (daha ilkel, "donma" tepkisiyle ilişkili kısım). Ventral vagus, sosyal etkileşim ve duygusal düzenleme ile bağlantılı olup kalp hızını anlık olarak ayarlayan "vagal fren"i oluşturur. Yüksek ventral vagal tonus, bireyin stres altında bile kalp hızını çabucak düşürüp sakinleşebilmesini ve sosyal olarak etkileşimde kalabilmesini sağlar. Porges, vagal tonusu yüksek bireylerin daha iyi sosyal işlevsellik ve öz-

düzenleme becerilerine sahip olduğunu öne sürmüştür. Nitekim araştırmalarda, dinlenik halde ölçülen yüksek HF-HRV (RSA) değerlerinin daha iyi başa çıkma stratejileri, duygusal denge ve sosyal bağlılıkla pozitif ilişki gösterdiği bulunmuştur.

Polivagal teori, OSS'nin sadece iki durumlu (sempatik vs parasempatik) olmadığını, farklı vagal devrelerin farklı davranışsal durumları desteklediğini vurgular. Örneğin, tehlike anında ventral vagal aktivite geri çekilip sempatik sistem devreye girerken (savaş-kaç tepkisi), aşırı kaçınılmaz tehditte dorsal vagus devreye girip "donma" veya bayılma tepkisine yol açabilir. HRV ölçümleri, özellikle RSA bileşeni, polivagal teoride ventral vagal fonksiyonun bir göstergesi olarak kullanılır. Bu teori klinik psikolojide travma, anksiyete ve sosyal ilişki sorunlarını anlamada yeni bir çerçeve sunmuştur.

HRV Ölçüm Yöntemleri (ECG, PPG, Holter)

HRV'yi ölçmek için önce kalp atımları arası milisaniye cinsinden aralıkların hassas şekilde kaydedilmesi gerekir. En yaygın yöntem, elektriksel kalp aktivitesini kaydeden Elektrokardiyografi (EKG)'dir. EKG'den elde edilen R-R aralıkları HRV analizlerinin altın standardıdır. Klinik ve araştırma ortamında HRV genellikle şu yöntemlerle ölçülür:

• Kısa süreli EKG kayıtları: Genellikle 5 dakikalık dinlenik EKG kayıtları kullanılır. Bu süre, HRV'nin standart kısa dönem analizine yeterli görülmüştür ve özellikle HF ile LF bileşenlerini hesaplamak için asgari gerekliliktir. Kısa kayıtlar ile anlık OSS durumu değerlendirilir; örneğin yatarken vs stres altındayken 5 dk'lık HRV ölçümleri karşılaştırılabilir. Hatta bazı çalışmalar 1 dakikaya kadar kısa kayıtların bile özellikle RMSSD gibi metrikler için güvenilir olabileceğini göstermiştir, ancak genel kabul 5 dakikanın altında inmemektir.

• Uzun süreli EKG (24 saat Holter): Taşınabilir Holter cihazlarıyla 24 saat boyunca sürekli EKG kaydı almak, HRV'nin tam bir günlük siklusunu analiz etme imkânı verir. Göğüs Kayışı (Kalp Hızı Monitörü): Sporcu ve ev kullanımı için yaygın olan göğüs bantları, kalp atım anlarını algılayan elektrotlar içerir. Bu cihazlar EKG sinyalinin kendisini değil, genellikle R-R aralıklarını kaydeder. Göğüs kayışları ile HRV takibi pratik olsa da, cilt ile temasın bozulması veya hareket artefaktları nedeniyle EKG kadar hassas olmayabilir.

• Fotopletismografi (PPG): Parmak ucu veya kulak memesi gibi bölgelere ışık gönderip geri yansıyan ışık miktarını ölçerek nabız dalgasını izleyen optik sensörlerdir. PPG, atımlar arasındaki nabız dalga geçiş sürelerini (PTT) ölçer ve bu ölçüm genellikle R-R aralığına yakın bir sonuç verir. Dinlenme durumunda PPG ile ölçülen nabız aralıkları, EKG'den türetilen R-R aralıklarına oldukça yakın bir doğrulukta HRV hesaplamaya olanak sağlar. Nitekim, istirahat halinde PPG tabanlı akıllı saat/cihaz uygulamalarının HRV ölçümleri ile EKG temelli ölçümler arasında iyi bir uyum saptanmıştır. Ancak, stres veya egzersiz durumlarında PPG sinyali bazı sınırlılıklara sahiptir: Artan sempatik aktiviteyle damar tonusu değişip nabız dalgasının yayılma hızı etkilenebilir, bu da PPG ile ölçülen atım aralıklarında ilave varyasyonlara yol açar. Yapılan karşılaştırmalarda, PPG yönteminin dinlenme koşullarında HRV için uygun olduğu, fakat yüksek stres altında veya ani değişimlerde EKG kadar güvenilir olmadığı bildirilmiştir.

• Giyilebilir Cihazlar ve Uygulamalar: Son yıllarda HRV ölçümü yapan ticari cihazlar (akıllı saatler, telefon uygulamaları vb.) yaygınlaşmıştır. Bu "kendini takip" cihazlarının bazıları göğüs bandı veya PPG sensörü ile veri toplar ve kullanıcılara stres düzeyi, toparlanma indeksi gibi metrikler sunar. Ancak tüketici cihazlarının çoğu özel (proprietary) algoritmalar kullandığından, elde edilen HRV değeri standart metriklerden farklı olabilir.

Klinik Yorumlama ve Önemi

HRV parametrelerinin klinik anlamı, kalbin otonom sinir sistemi tarafından düzenlenmesindeki etkinlik ve vücudun strese uyum sağlama kapasitesi ile yakından ilişkilidir. Genel bir kural olarak, yüksek HRV iyi bir prognostik işaret kabul edilirken, düşük HRV birçok hastalık durumda kötü prognozla ili şkil endirilmi ştir. Önemli HRV göstergelerinin klinik yorumları kısaca şöyle özetlenebilir:

• **SDNN (toplam varyasyon):** Yüksek SDNN, hem sempatik hem parasempatik sistemin geniş bir modülasyon aralığı kullandığını, kalbin geniş yelpazede adaptasyon yapabildiğini gösterir. Düşük SDNN ise genellikle azalmış global otonomik değişkenlik belirtisidir; örneğin ileri kalp yetmezliği hastalarında veya otonom nöropatilerde SDNN düşer.

- **RMSSD & HF (parasempatik tonus):** Bu iki parametre güçlü şekilde korele olduğundan, kısa dönem vagal aktivitenin göstergesi olarak birlikte yorumlanırlar. Yüksek RMSSD veya HF gücü, sağlam bir vagal fren ve iyi bir dinlenik OSS durumu demektir. Özellikle genç ve atletik bireylerde RMSSD/HF yüksek, stres altındaki veya hastalıklı bireylerde düşüktür. Klinik ortamda RMSSD, örneğin yoğun bakım hastalarında ağrı veya ağrı kesici etkinliğinin takibinde ya da sporcularda toparlanma düzeyinin değerlendirilmesinde kullanılmaktadır. Düşük RMSSD (<~10-15 ms kısa ölçümlerde) genellikle yüksek sempatik aktivite veya düşük vagal tonus anlamına gelebilir.

- **LF gücü (barorefleks ve karışık etki):** LF bileşeni hem sempatik hem parasempatik etkileri içerdiğinden yorumlanması en zor olan parametrelerdendir. Barorefleks fonksiyonunu ve damar tonusundaki yavaş salınımları yansıttığı için, LF gücünün azalması barorefleks duyarlılığının bozulduğuna işaret edebilir (örn. diyabetik otonom nöropatide LF düşer). Öte yandan, akut stres veya ortostatik stres altında LF yükselirken HF düşer; bu, sempatik aktivitenin göreceli artışını gösterir. Ancak tek başına LF yüksekliği, mutlaka sempatik aşırı aktivite demek değildir; aynı anda vagal komponent de bulunabileceği için, LF artışı bazen adaptif bir barorefleks cevabın göstergesi olabilir. Bu nedenle klinikte LF'ye tek başına bakarak karar verilmez, diğer parametrelerle birlikte değerlendirilir.

- **LF/HF Oranı (sempatovagal denge):** Özellikle geçmiş literatürde bu oran, "sempatik- parasempatik denge" indeksidir diye anılsa da günümüzde dikkatle ve sınırlı şekilde kullanılmaktadır. Örneğin, kronik stresli bir bireyde LF/HF oranının sürekli yüksek olması, vagal tonusun düşük kalıp sempatik etkinliğin baskın sürdüğünü düşündürebilir. Fakat akut koşullarda (egzersiz sırasında) LF/HF doğal olarak artacağından, durumsal faktörler göz önüne alınmalıdır. Ayrıca bazı kalp ritmi düzensizlikleri veya solunum paternindeki değişimler de LF/HF oranını etkileyebilir. Kısacası, LF/HF yorumlaması bağlama bağımlıdır: Yalnızca istirahat durumundaki ölçümlerde ve diğer bulgularla birlikte kullanıldığında faydalı olabilir. Tek başına bir otonom denge skoru olarak değerlendirildiğinde hatalı sonuçlara götürebilir.

Sonuç olarak, HRV'nin klinik önemi, otonom sinir sisteminin esnekliğinin ve kardiyovasküler sağlığın bir göstergesi olmasından gelir. Yüksek HRV, iyi bir adaptif kapasite, stresle başa çıkabilme ve genel olarak sağlıklı bir OSS fonksiyonu ile ilişkilidir.

0154 - When Psychosis Comes First: Huntington's Disease With Acanthocytosis Mimicking Schizophrenia - A Case-Based Review

Esra Acar, Hüseyin Köse, Filiz Kulaçaoğlu Öztürk

Background

Huntington's disease (HD) is an autosomal dominant neurodegenerative disorder caused by an expanded CAG trinucleotide repeat in the HTTgene, leading to progressive neuronal loss predominantly affecting the basal ganglia and cerebral cortex. (1) Clinically, HD is characterized by a triad of motor, cognitive, and psychiatric symptoms. While motor manifestations are traditionally considered the defining features of the disease, psychiatric symptoms are common, contribute substantially to disease burden, and may precede motor findings by several years. Psychiatric manifestations in HD include depression, irritability, apathy, obsessive-compulsive behaviors, and less frequently, psychosis. (2) Depression is the most prevalent psychiatric symptom, whereas psychotic symptoms—characterized by delusions and/or hallucinations—are reported in a minority of patients, with prevalence estimates ranging from 3% to 11%. (3) Despite their relative rarity, psychotic symptoms are clinically significant and associated with poorer outcomes, increased caregiver burden, and higher rates of hospitalization. Historically, and in some cases even in modern clinical practice, HD has been misdiagnosed as schizophrenia or other primary psychotic disorders due to the prominence and early onset of psychiatric symptoms. This diagnostic overlap is particularly problematic when psychosis precedes motor signs or dominates the clinical picture, leading to prolonged exposure to antipsychotic medications. Such treatments may mask the underlying neurodegenerative process or exacerbate motor symptoms, further delaying accurate diagnosis.

Beyond Huntington's disease, the evaluation of adult-onset chorea requires consideration of other hereditary neurodegenerative disorders, most notably neuroacanthocytosis syndromes. Neuroacanthocytosis (NA) syndromes comprise a group of rare neurodegenerative disorders characterized by progressive neuropsychiatric involvement and the presence of acanthocytes in peripheral blood. Among these, X-linked McLeod syndrome and autosomal recessive chorea-acanthocytosis represent the two most well-defined clinical entities. (4) However, acanthocytosis has also been sporadically reported in patients with genetically confirmed Huntington's disease. While its prevalence in HD appears low and remains poorly defined, the coexistence of acanthocytosis may further obscure the diagnostic pathway, particularly in cases dominated by psychiatric symptoms and atypical clinical trajectories.(5)

Greater awareness of neurodegenerative disorders presenting with chronic psychosis is essential to reduce misdiagnosis, avoid inappropriate treatment, and facilitate timely recognition of underlying neurological pathology.

Case Presentation

Here, we present a 58-year-old man with a long-standing diagnosis of schizophrenia who presented with a six-month history of progressively worsening gait disturbance, dysarthria, and involuntary choreoathetoid movements, accompanied by exacerbation of psychotic symptoms. The patient's psychiatric history dates back to 2013, when he experienced his first psychiatric hospitalization at another medical institution; however, detailed clinical records from that admission were unavailable. He subsequently underwent a second psychiatric hospitalization in October 2018, during which he presented with persecutory and religious delusions, auditory hallucinations, and a history of medication non-adherence for approximately six months. Clozapine was initiated due to extrapyramidal adverse effects associated with prior antipsychotic treatments; however, it was discontinued following the development of tachycardia, raising concern for clozapine-associated myocarditis. He was subsequently discharged on olanzapine 20 mg/day and transferred to long-term institutional care, where he remained predominantly calm and cooperative.

Over the six months preceding the current admission, he developed progressive gait and speech impairment, purposeless and bizarre movements, and worsening psychotic symptoms. He was evaluated at another medical institution and treated with haloperidol oral drops, after which his neurological and psychiatric symptoms markedly deteriorated.

At presentation, his regular medications included olanzapine 20 mg/day, aripiprazole 30 mg/day, sertraline 25 mg/day, haloperidol oral drops (2x5 drops/day), and biperiden 2 mg/day. Zuclopenthixol decanoate injections had been administered until three months prior and were subsequently discontinued. He had no history of alcohol or illicit substance use, chronic medical illness, allergies, or prior surgical interventions. Family history was remarkable for an early-onset movement disorder in his father and for a similar movement disorder in his sister with symptom onset in her 50s; notably, no genetic testing had been previously performed in either case. On admission, the patient was alert, oriented, and cooperative, with preserved self-care. His speech was dysarthric and poorly organized. Affect was blunted, and mood was dysphoric. He exhibited probable auditory and visual hallucinations, with markedly impaired insight and judgment. Neurological examination revealed gait instability and prominent choreoathetoid movements involving the face and upper extremities. Muscle tone and deep tendon reflexes were within normal limits, and no focal neurological deficits were detected. Routine laboratory investigations were unremarkable. Non-contrast cranial CT, diffusion- weighted MRI, and conventional brain MRI showed no acute or structural abnormalities. Neurology consultation excluded an acute neurovascular event.

Treatment was initiated as a simplified antipsychotic regimen with olanzapine and quetiapine. A neurology consultation was obtained, during which peripheral blood smear examination for neuroacanthocytosis and genetic testing for Huntington's disease were recommended. Hematology consultation demonstrated marked acanthocytosis on peripheral blood smear, with acanthocyte counts increasing from approximately 10-15% on the baseline smear to nearly 50% on the induced smear, consistent with neuroacanthocytosis.

Subsequent molecular genetic analysis identified a pathogenic CAG repeat expansion in the HTT gene, with a repeat length of 15/40 (NP_002750.1:p.Gln18[15];[40] (±1)), confirming the diagnosis of Huntington's disease.

The patient was discharged on olanzapine 20 mg/day and quetiapine 100 mg/day, with planned close neurology and psychiatry outpatient follow-up. Following the adjustment of the antipsychotic regimen, partial improvement in choreoathetoid movements was observed at discharge. Written informed consent for publication was obtained from the patient's relatives, and appropriate genetic counseling was provided to the family.

Discussion

This case exemplifies a neurodegenerative disorder presenting with predominant psychiatric symptomatology, initially misclassified as a primary psychotic disorder. The late emergence of progressive choreoathetoid movements, together with clinical deterioration following exposure to dopamine antagonists and a contributory family history, constituted critical indicators suggestive of an underlying neurodegenerative etiology. Such presentations reflect the well- documented phenotypic overlap between Huntington's disease and primary psychiatric disorders, particularly in cases in which psychiatric manifestations precede overt motor findings.

The demonstration of marked acanthocytosis on peripheral blood smear, in conjunction with the identification of a pathogenic CAG repeat expansion in the HTT gene, established the diagnosis of Huntington's disease. Although acanthocytosis is classically associated with neuroacanthocytosis syndromes, its occurrence in Huntington's disease has been sporadically reported and may further obscure the diagnostic process, particularly in cases with atypical clinical trajectories. (5)

From a clinical perspective, this case underscores the necessity of maintaining a high index of suspicion for neurodegenerative disorders in patients with long-standing psychotic diagnoses who subsequently develop progressive movement abnormalities. The phenomenon of paradoxical clinical worsening under dopamine antagonist therapy represents an additional diagnostic clue and should prompt comprehensive neurological and genetic evaluation. Early recognition is essential to prevent prolonged misdiagnosis, limit inappropriate pharmacological exposure, and enable timely multidisciplinary management.

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0157 - Comparison of Disease Duration and Length of Hospitalization with Hematological Inflammatory Markers in Patients with Schizophrenia

Objective:

This study aimed to investigate the relationship between disease duration, length of hospitalization, and hematological inflammatory markers in patients with schizophrenia.

Methods:

A total of 69 patients aged 18-65 years who were hospitalized with a diagnosis of schizophrenia between January 2022 and August 2025 were included in this retrospective study. Hematological inflammatory markers including NLR, MLR, PLR, SII, MHR, and NHR were calculated from routine laboratory parameters obtained within the first 24 hours of hospitalization. Relationships between variables were analyzed using Pearson correlation analysis.

Results:

A moderate positive correlation was found between disease duration and number of hospitalizations ($r = 0.473$, $p < 0.001$). Disease duration showed a significant negative correlation with MHR ($r = -0.262$, $p < 0.05$). Length of hospitalization was positively correlated with SII ($r = 0.280$, $p < 0.05$) and LDL levels ($r = 0.433$, $p < 0.001$). Strong positive correlations were also observed among several inflammatory indices including NLR, PLR, and SII.

Conclusion:

The findings suggest that inflammatory processes in schizophrenia may be more closely associated with clinical severity and hospitalization-related indicators rather than the chronic duration of the illness. Hematological inflammatory indices may serve as practical and accessible biomarkers in clinical settings.

Introduction

Schizophrenia is a complex psychiatric disorder that arises from the interaction of genetic, neurobiological, and environmental factors (1). In recent years, the stress-inflammation model has attracted increasing attention among the proposed models explaining the pathophysiology of schizophrenia (2). Studies have shown the presence of biomarkers indicating chronic inflammation and immune system activation in this disorder (3). In addition, exposure to influenza, toxoplasma, or other infections during the prenatal period has been suggested to trigger the development of schizophrenia in genetically predisposed individuals (4).

Lymphocytes, neutrophils, and monocytes are cells that play important roles in the inflammatory response (5). Moreover, there is evidence suggesting that platelets may contribute to the pathophysiology of schizophrenia through serotonin-related mechanisms and inflammation (6). HDL, on the other hand, is a lipoprotein with anti-inflammatory properties that can modulate inflammatory processes by regulating cholesterol efflux from peripheral tissues (5).

Ratios derived from routine hemogram parameters, such as the neutrophil-to-lymphocyte ratio (NLR), monocyte-to-lymphocyte ratio (MLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), monocyte-to-HDL ratio (MHR), and neutrophil-to-HDL ratio (NHR), are used as indicators of peripheral inflammation (7-9). These indices have been reported to correlate well with other inflammatory markers such as C-reactive protein (CRP) and cytokine levels (10). However, it remains unclear whether these markers are more closely related to the chronic duration of the disease or to indicators such as clinical severity and the need for hospitalization.

The aim of this study was to investigate the relationship between disease duration, length of hospitalization, and hematological inflammatory markers in patients diagnosed with schizophrenia. In addition, we aimed to evaluate the potential of these markers to reflect clinical severity and duration of hospital stay.

Methods

A total of 69 patients aged 18-65 years who were hospitalized in the inpatient psychiatry unit of Kartal Dr. Lutfi Kirdar City Hospital, Istanbul, between January 2022 and August 2025 and diagnosed with schizophrenia according to ICD-10 criteria were included in this study.

Patients who were discharged due to refusal of treatment or rehospitalized within two weeks after discharge were excluded. For patients with more than one hospitalization during the study period, only the first admission was included in the analysis. In addition, patients with alcohol or substance use disorders and those hospitalized following a suicide attempt were excluded because of the potential effects of these conditions on inflammatory parameters.

Clinical data and laboratory parameters were retrospectively reviewed through the hospital electronic medical record system. Complete blood count and biochemical parameters were obtained from samples collected within the first 24 hours of hospitalization. The majority of blood samples were collected between 08:00 and 09:00 in the morning.

Ethics approval for the study was obtained from Kartal Dr. Lutfi Kirdar City Hospital on 24.02.2026 with decision number 202610|0.99125138.

Statistical Analysis

Statistical analyses were performed using SPSS. Continuous variables were presented as median and 25%-75% percentiles, while categorical variables were expressed as numbers and percentages. Relationships between variables were evaluated using Pearson correlation analysis, and correlation coefficients were reported as r values. Statistical significance was set at $p < 0.05$.

Results

Of the 69 patients included in the study, 68.1% were male and 31.9% were female. Of the participants, 73.9% were single and 85.5% were unemployed. Physical illness was present in 26.1% of the patients, 36.2% were using psychiatric medication, and 20.3% had a family history of psychiatric illness.

The median age of the patients was 37 (29-48) years. The median disease duration was 8 (3-20) years, the median number of hospitalizations was 2 (1-3), and the median length of hospitalization was 20 (14-28) days.

According to Pearson correlation analysis, a moderate positive and statistically significant relationship was found between disease duration and number of hospitalizations ($r = 0.473$, $p < 0.001$). A significant negative correlation was observed between disease duration and MHR ($r = -0.262$, $p < 0.05$). Length of hospitalization was positively and significantly correlated with SII ($r = 0.280$, $p < 0.05$). In addition, a positive significant correlation was found between length of hospitalization and LDL levels ($r = 0.433$, $p < 0.001$).

Significant relationships were also identified among inflammatory parameters. Strong positive correlations were found between NLR and PLR ($r = 0.739$, $p < 0.001$) and between NLR and SII ($r = 0.856$, $p < 0.001$). Similarly, a strong positive correlation was observed between PLR and SII ($r = 0.716$, $p < 0.001$). A very strong positive correlation was found between triglycerides (TRG) and VLDL ($r = 0.949$, $p < 0.001$). Lower-level but significant relationships were also detected between HDL and some inflammatory parameters.

Discussion

This study investigated the relationship between disease duration, length of hospitalization, and hematological inflammatory markers in patients with schizophrenia. The findings showed that the number of hospitalizations increased as disease duration increased. In addition, a negative relationship was found between disease duration and MHR. Furthermore, the positive relationship between length of hospitalization and SII suggests that inflammatory processes may be associated with clinical severity and the acute phase of the illness.

Because of the relationship between the immune system and schizophrenia, inflammatory indices such as NLR, MLR, and PLR are widely used in schizophrenia research (11-13). The relative stability of these indices over time and their independence from age and sex suggest that they may serve as potential biomarkers reflecting the biological burden of the disorder (14).

The positive association found in our study between disease duration and number of hospitalizations is consistent with studies showing that schizophrenia is a chronic disorder characterized by recurrent relapses (15,16). As disease duration increases, relapses may also increase, which may lead to a higher frequency of hospitalization.

MHR has been reported in the literature as a novel biomarker reflecting systemic inflammation and may be higher in patients with schizophrenia than in healthy controls (17,18). However, the negative relationship found in our study between disease duration and MHR suggests that the inflammatory response may vary across different stages of the disease.

The positive correlation between length of hospitalization and SII suggests that the inflammatory response may be associated with clinical severity. A study conducted in hospitalized psychiatric patients regardless of diagnosis reported that a high inflammatory response was significantly positively associated with length of hospital stay (19,20).

In conclusion, the findings of this study suggest that inflammatory processes may be more closely related to clinical severity and the need for hospitalization than to the chronic duration of the illness.

This study has several limitations. The retrospective and single-center design, together with the limited sample size, may restrict the generalizability of the findings. In addition, potential confounding factors such as antipsychotic treatment, metabolic status, and lifestyle factors could not be fully controlled.

Nevertheless, one of the strengths of this study is the combined evaluation of disease duration, length of hospitalization, and hematological inflammatory indices. Furthermore, the fact that these parameters are derived from routine laboratory tests suggests that they may be easily applicable biomarkers in clinical practice.

Keywords: Schizophrenia, inflammation, NLR, SII, hospitalization, biomarkers

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0161 - Predictors of Burnout in Parents of Children with Autism: The Importance of Physical Space

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Introduction: Autism Spectrum Disorder (ASD) is a neurodevelopmental condition marked by impairments in social interaction and communication, alongside restricted interests and repetitive behaviors [1]. Affecting individuals from early childhood throughout their lives, ASD significantly impacts social, academic, and daily functioning. Global prevalence rates, such as 3.2% among 8-year-old children in the United States in 2022, underscore its widespread impact [1].

The lifelong nature of ASD profoundly affects not only the child but also the entire family system [2]. Parents of children with ASD often experience elevated stress levels compared to those raising typically developing children or children with other developmental challenges. The unique difficulties, such as the child's lack of social reciprocity, limited eye contact, and emotional sharing, can lead to unfulfilled emotional bonds and deep disappointment for parents[2]. Furthermore, the continuous demands of caregiving, including managing cognitive inadequacies, irritability, hyperactivity, learning and language problems, and the need for constant supervision, significantly contribute to parental stress[2].

Chronic stress in this context can culminate in parental burnout, a distinct syndrome characterized by overwhelming exhaustion related to the parenting role, emotional distancing from the child, and a sense of ineffectiveness in one's parental identity[3, 4] This state contrasts sharply with previous parental attitudes, transforming the parenting role into an unsustainable burden. Key risk factors for parental burnout in families of children with ASD include the unpredictability of behaviors, difficulties in accessing educational and health services, societal prejudices, and a lack of social support[4]. The "Balance between Risks and Resources" theory posits that parental burnout arises from an imbalance between excessive parenting demands and insufficient resources[5] Consequences extend beyond parental well- being, impacting parenting quality, marital conflict, and increasing the risk of neglect or violence towards children[6]

While early research on parental burnout primarily focused on the child's diagnostic characteristics and the care burden, contemporary literature has shifted towards examining parents' internal and external resources [7]. Recent studies highlight the role of sociodemographic variables and internal resources like psychological resilience and perceived social support in mitigating burnout[7]. However, there remains a gap in understanding how specific physical living conditions, beyond general socioeconomic status, might influence parental burnout. This study aims to address this gap by investigating the relationship between physical living space and burnout levels in parents of children with ASD within a Turkish sample, thereby contributing to the development of more holistic support recommendations.

Methods

Research Design and Participants: This study employed a relational survey model, a quantitative research method, to explore burnout levels in parents of children diagnosed with ASD and their relationship with various sociodemographic variables. The study sample comprised 131 parents of children with ASD who were voluntarily recruited from the Ondokuz Mayis University (OMU) Faculty of Medicine Child and Adolescent Mental Health and Diseases outpatient clinic and the Autism Application and Research Center (OGEM) within OMU. Ethical approval was obtained from the Ondokuz Mayis University Clinical Research Ethics Committee (Decision No: 2025/141, Date: 22.04.2025).

Data Collection Instruments: Data were collected through online surveys via Google Forms, utilizing two primary tools:

- **Sociodemographic Data Form:** Developed by the researchers, this form gathered comprehensive personal and household information. Variables included parents' age, marital status, education level, occupation, number of household members, **number of rooms in the house,** child's age at diagnosis, school status, types of spec ial education received (e.g., speech therapy, occupational therapy, sensory integration), and psychiatric medication treatment.
- **Maslach Burnout Inventory (MBI):** The MBI, originally developed by Maslach and Jackson in 1981, was adapted to the parenting role to measure parental burnout levels [3]. The Turkish validity and reliability studies were conducted by Ergin and Qam [8]. The scale consists of 22 items across three subdimensions: Emotional Exhaustion (9 items), Depersonalization (5 items), and Reduced Personal Accomplishment (8 items). Items were rated on a 5-point Likert scale. For scoring, items in the Personal Accomplishment subdimension were reverse-scored, where a high score indicates low burnout. Higher scores in Emotional Exhaustion and Depersonalization indicate increased burnout. The scale items were rephrased to be specific to the parenting role (e.g., "my child," "care of my child")[4] The Cronbach's alpha internal consistency coefficient for the scale was .93.

Statistical Analysis: Data analysis was performed using IBM SPSS Statistics v21. Prior to analysis, data were checked and coded. The total burnout score, calculated by summing the subdimension scores (emotional exhaustion + personal accomplishment + depersonalization), served as the dependent variable. Hierarchical multiple linear regression analysis was applied to identify predictors of the total burnout score. Continuous variables were directly included, while categorical variables were handled using a dummy variable approach, with one level designated as the reference category for comparisons. Regression results were reported with unstandardized coefficients (B), standard errors (SE), standardized coefficients (β), t-values, p-values, and 95% confidence intervals. Model fit was assessed using R and adjusted R2 values. The significance level was set at $p < 0.05$ for all two-tailed tests.

Results

Parent and Household Characteristics: The study involved 131 parents, predominantly mothers (77.9%) and married individuals (90.1%). The mean age for mothers was 37.72 ± 7.15 years, and for fathers, 40.79 ± 8.82 years. High school graduation was the most common education level for both parent groups. Most mothers (84.7%) were unemployed, while 43.5% of fathers worked as laborers. The average number of children per family was 2.02 ± 0.93, and the average number of rooms in residences was 3.79 ± 0.71. Children's average age was 8.4 ± 4.14 years, with 74.0% being male. The majority (87.0%) attended school, primarily special education classes (50.4%). Most children received special education support (93.1%) and psychiatric medication treatment (60.3%).

Descriptive Statistics and Regression Analysis: The mean total burnout score for the sample was 50.57 ± 9.26 (Median = 51.0). Subscale scores were: Emotional Exhaustion (13.85 ± 9.44), Depersonalization (5.42 ± 4.53), and Personal Accomplishment (37.97 ± 7.19). Spearman correlation analysis indicated strong positive correlations between total burnout score and Emotional Exhaustion (rho = 0.909) and Depersonalization (rho = 0.734).

Crucially, the multiple linear regression analysis revealed that most sociodemographic variables (income, education level, parental age) did not significantly predict parental burnout. However, the **"number of rooms in the house" was identified as the only statistically significant predictor ($p < 0.05$).** Specifically, an increase in the number of rooms in the household was significantly associated with a decrease in total parental burnout scores. This finding suggests that greater physical living space acts as a protective factor against burnout in parents of children with ASD.

Discussion

This study provides novel insights into the predictors of parental burnout in families of children with ASD, particularly highlighting the overlooked role of physical living space. Contrary to the emphasis in some literature on socioeconomic indicators, our findings suggest that traditional sociodemographic variables like income and education level may not be the primary drivers of burnout in this specific population within the Turkish context.

Instead, the **"number of rooms in the house" emerged as a uniquely significant protective factor,** indicating that adequate physical space serves as a crucial "spatial resource" for parents.

This finding aligns with the "Balance between Risks and Resources" theory, suggesting that a larger living space can be considered a valuable resource that helps parents manage the inherent demands and stressors associated with raising a child with ASD[5]. A greater number of rooms likely provides opportunities for individual family members, especially parents, to have private areas for self-regulation, respite, and personal time. This ability to withdraw and recharge, even within the home environment, can significantly mitigate the emotional exhaustion and detachment characteristic of parental burnout. For parents navigating the continuous and often unpredictable challenges of ASD, having a dedicated personal space may be more critical for mental well-being than financial status alone.

The implications of these findings are substantial for the development of targeted support strategies. Current interventions often focus on psychological resilience, social support networks, and access to specialized services. While these remain vital, our study suggests that improving the quality of life through spatial arrangements should also be considered. This could involve advocating for housing policies that support families with special needs children in acquiring larger living spaces or promoting home modifications that create designated private areas. Furthermore, the emphasis on physical space underscores the need for accessible respite services outside the home, offering parents additional opportunities for personal time and stress reduction.

It is important to acknowledge the limitations of this study. The cross-sectional design prevents the establishment of causal relationships. The reliance on self-report measures for burnout and sociodemographic data may introduce bias. The sample, drawn from a specific university setting in Turkey, limits generalizability to broader populations. Future research could benefit from longitudinal designs, objective measures of living space quality, and diverse samples to validate and expand upon these findings. Despite these limitations, this study offers a compelling argument for considering physical environment as a significant, yet often neglected, determinant of parental well-being in families affected by ASD.

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0163 - Rapid Antidepressant and Anti-Suicidal Effects of Intravenous Ketamine Infusion in Unipolar and Bipolar Depression: Real-World Clinical Outcomes

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Objective

Treatment-resistant depression (TRD), encompassing both unipolar and bipolar forms, represents a major public health concern due to its association with high morbidity, marked functional impairment, and an increased risk of suicide. The delayed onset of action of conventional antidepressant treatments poses significant clinical challenges, particularly in patients at acute risk of suicide. In recent years, ketamine, an N-methyl-D-aspartate (NMDA) receptor antagonist, has attracted considerable attention because of its rapid onset of action and pronounced anti-suicidal effects [1]. However, real-world data regarding the efficacy and safety of ketamine across different diagnostic subgroups remain limited.

The aim of this study was to evaluate, based on real-world clinical data, the effects of intravenous ketamine infusion on depressive symptoms, anxiety levels, suicidal ideation, overall clinical severity, and functional status in patients diagnosed with severe unipolar and bipolar depression.

Methods

This study is an ethics committee-approved, retrospective, naturalistic real-world investigation conducted at a tertiary care psychiatric center. Clinical records were reviewed retrospectively, and data from patients who received intravenous ketamine infusion were analyzed.

Approximately 350 patient files were screened, and 100 patients who had received at least six ketamine infusion sessions were included in the study. The sample consisted of 79 patients diagnosed with unipolar depression and 21 patients diagnosed with bipolar depression. All participants were undergoing treatment for a severe depressive episode and had previously demonstrated an inadequate response to multiple pharmacological treatments.

Clinical assessments were performed using multidimensional, standardized rating scales:

- Montgomery-Asberg Depression Rating Scale (MADRS): severity of depressive symptoms
- MADRS Suicidal Ideation Item: level of suicidal ideation
- Hamilton Anxiety Rating Scale (HAM-A): severity of anxiety symptoms
- Clinical Global Impression - Severity and Improvement (CGI-S/I): overall clinical status
- Brief Functional Assessment Scale: psychosocial functioning
- Young Mania Rating Scale (YMRS): assessment of manic symptoms

The study did not include a control group, and the research design was structured to reflect outcomes observed in routine clinical practice.

Results

The sociodemographic and clinical characteristics of the patients included in the study indicate that the sample comprised individuals with chronic, complex, and high-risk depressive disorders observed under real-world clinical conditions. Of the participants, 57% were female; the mean age was 39.3 ± 11.9 years, mean age at illness onset was 29.7 ± 10.9 years, mean illness duration was 9.5 ± 7.5 years, and mean years of education was 9.5 ± 5.4 years. Comorbid medical conditions were present in 29% of the patients. These findings suggest that the results are grounded in a clinically representative sample with strong external validity.

The clinical profile of the sample reflected a population with severe and high-risk depression. Sixty-one percent of the patients met criteria for treatment-resistant depression, 37% had a history of suicide attempts, 29% had

previously received electroconvulsive therapy, and 35% reported a history of psychiatric hospitalization. These data indicate that ketamine treatment was predominantly utilized in patients requiring advanced-stage or rapid-acting therapeutic interventions. Despite the high-risk clinical profile, no cases of manic switch or psychotic exacerbation were observed, supporting the notion that ketamine can be administered safely with appropriate patient selection and close clinical monitoring.

Clinical outcomes demonstrated that ketamine infusions were associated with early-onset and sustained improvements in depressive symptoms, suicidal ideation, anxiety, overall clinical severity, and functional status. The mean Montgomery-Asberg Depression Rating Scale (MADRS) total score decreased from 36.2 ± 7.6 to 17.5 ± 8.7 by the sixth session, indicating a clinically significant improvement from the severe to the moderate-mild range. Scores on the MADRS suicidal ideation item declined from 3.3 ± 1.3 to 1.1 ± 0.8, with a particularly rapid anti-suicidal effect observed after the first two sessions. Hamilton Anxiety Rating Scale (HAM-A) scores decreased from 23.5 ± 9.3 to 11.8 ± 6.4, reflecting a marked reduction in comorbid anxiety symptoms. The decrease in Clinical Global Impression-Severity (CGI-S) scores (from 4.8 ± 0.9 to 3.2 ± 1.0) further supported global clinical improvement. A reduction in Brief Functional Assessment Scale scores from 50.8 ± 9.8 to 34.6 ± 11.4 indicated that symptomatic improvement translated into enhanced daily functioning. Young Mania Rating Scale (YMRS) scores remained low and stable throughout the sessions (from 2.5 ± 1.5 to 3.8 ± 1.5), suggesting the absence of treatment-emergent manic episodes. Standardization of the data using z-scores and their presentation in an integrated graphical format enabled a comprehensive visualization of the temporal dynamics of multidimensional clinical improvement (Figure 1).

Safety data indicated that adverse effects associated with ketamine were largely transient and tended to diminish across successive sessions. The most frequently observed adverse effects were dizziness and orthostatic symptoms, reported in 74% of patients during the first infusion session; these decreased markedly to approximately 30% in subsequent sessions. Dissociative symptoms were observed in 46% of patients during the initial sessions, declining to 19% and 8% in later sessions, respectively. Perceptual disturbances, including derealization and depersonalization, were reported in 43% of patients during the first sessions and decreased to 17% and 7% in subsequent sessions, following a short-lived and self-limited course. Somatic adverse effects such as headache and nausea-vomiting occurred less frequently, were generally of mild to moderate severity, and resolved spontaneously without requiring clinical intervention. Symptoms of anxiety and agitation were observed in 20-23% of patients in the early sessions but declined to 3-4% in later sessions and did not constitute a persistent clinical problem. No patient developed psychotic symptoms or showed a clinically significant increase in YMRS scores during the study period, underscoring the absence of manic switch, particularly in individuals with bipolar depression (Table 1).

In summary, intravenous ketamine infusions emerged as a safe treatment option in a high-risk, treatment-resistant depressive population, with adverse effects that were predominantly transient and diminished over time. The treatment was associated with early and clinically meaningful improvements in depressive symptoms, suicidal ideation, anxiety, and functional outcomes, without evidence of manic or psychotic adverse effects (Figure 2).

Discussion

This retrospective study based on real-world data demonstrates that intravenous ketamine infusion is associated with rapid-onset and multidimensional clinical improvement in patients with severe unipolar and bipolar depression. Following ketamine treatment, significant reductions were observed in depressive symptoms, anxiety levels, and functional impairment, as reflected in improvements across the Montgomery-Asberg Depression Rating Scale (MADRS), Hamilton Anxiety Rating Scale (HAM-A), Clinical Global Impression-Severity (CGI-S), and the Brief Functional Assessment Scale [2]. Notably, the rapid decline observed in the MADRS suicidal ideation item supports the acute anti-suicidal effects of ketamine. Considering that the therapeutic effects of conventional antidepressants typically emerge over several weeks, ketamine's onset of action within hours to days represents a critical advantage in high-risk patient populations [3].

The observation of comparable clinical responses in both unipolar and bipolar depression suggests that ketamine's therapeutic effects may be relatively independent of traditional diagnostic classifications, indicating a potential transdiagnostic effect profile. This effect is thought to be mediated by neurobiological mechanisms involving glutamatergic neurotransmission, synaptic plasticity, and brain-derived neurotrophic factor (BDNF) [4]. Ketamine has been shown to increase glutamate release through NMDA receptor antagonism, leading to enhanced synaptic

connectivity [5]. These neurobiological processes are believed to underlie not only improvements in depressive symptoms but also broader benefits in anxiety levels and functional outcomes.

Safety data indicated that the adverse effects of ketamine were predominantly transient and decreased across successive treatment sessions. Although dizziness, orthostatic symptoms, and dissociative experiences were more frequent during the initial infusions, their incidence declined markedly in subsequent sessions [6]. The absence of manic switch or psychotic exacerbation throughout the study period further supports the safe administration of ketamine in patients with bipolar depression when appropriate patient selection and clinical monitoring are ensured [7,8].

Because these findings reflect routine clinical practice, they encompass a broader diversity of patients and treatment-related variables compared with the controlled and idealized settings of randomized controlled trials, thereby enhancing external validity. The high proportion of treatment-resistant patients, individuals with a history of suicide attempts, and those previously treated with electroconvulsive therapy indicates that ketamine may be an effective and tolerable therapeutic option even in the most difficult-to-treat subgroups of depression [9].

Conclusion

This ethics committee-approved study demonstrates that intravenous ketamine infusion is associated with rapid, clinically meaningful, and multidimensional improvement in patients with severe unipolar and bipolar depression. Ketamine treatment produced beneficial effects on depressive symptoms, suicidal ideation, anxiety, and functional capacity, with similar response patterns observed across diagnostic categories. The absence of manic switch or psychotic exacerbation during the study period suggests that ketamine represents a safe treatment option when administered under appropriate clinical supervision. These findings indicate that ketamine may provide a valuable addition to clinical practice as a rapidly acting intervention, particularly for patients with treatment-resistant depression and those at acute risk of suicide [8,10].

Keywords: Bipolar Disorder; Depressive Disorder, Treatment-Resistant; Ketamine; Real-World Data; Suicidal Ideation

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Figure 1. Standardized (z-score) trajectories across ketamine sessions Unipolar vs Bipolar (solid vs dashed)

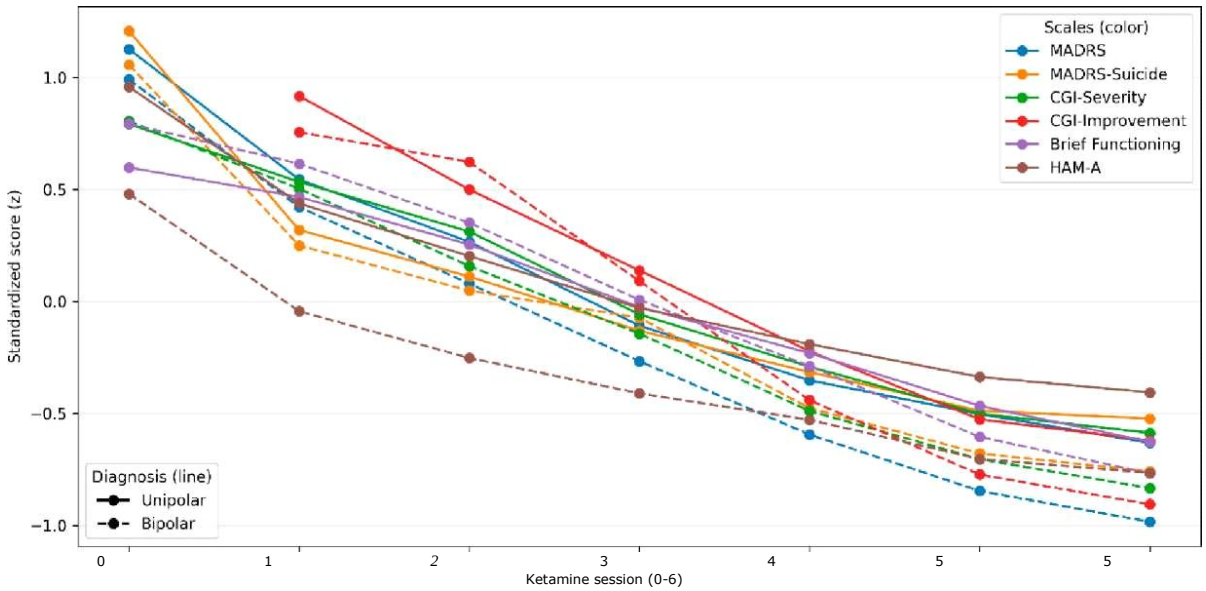


Figure 2.

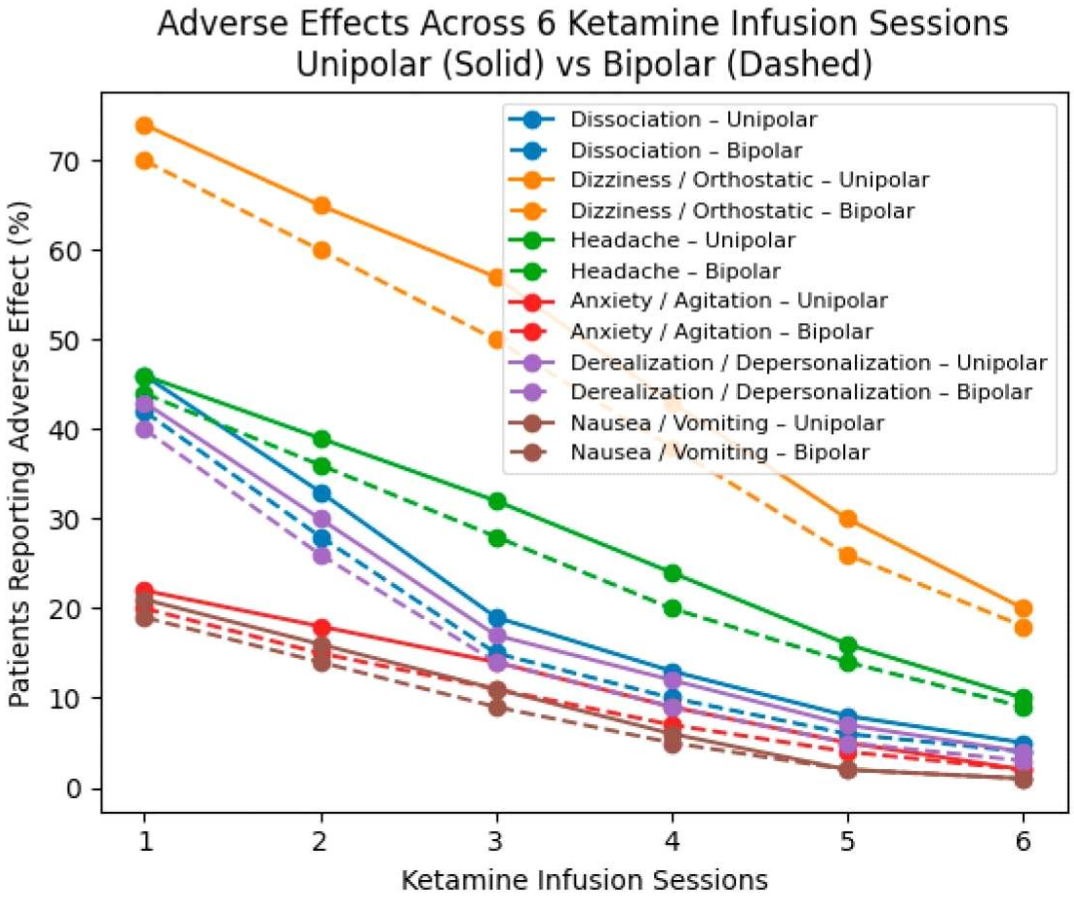


Table 1. Safety and Tolerability Profile Across Ketamine Infusion Sessions (Real-World Clinical Data, n=100)

Adverse Effect	Session 1 (%)	Session 3 (%)	Session 3 (%)	Clinical Interpretation
Dissociation	46	19	8	Transient, dose-related, rapidly attenuating
Dizziness / Orthostatic symptoms	74	57	30	Most common early effect, decreases over time
Headache	46	32	16	Mild-moderate, self-limiting
Anxiety / Agitation	20-23	11-14	3-4	Early onset, no persistence
Derealization / Depersonalization	43	17	7	Expected ketamine-related effect, short-lived
Nausea / Vomiting	21	11	2	Low frequency, clinically manageable
Psychotic symptoms	0	0	0	**Not observed**
Manic switch (YMRS increase)	-	-	-	**Not observed**

**** Adverse effects were predominantly transient, non-progressive, and diminished with repeated infusions, with no evidence of psychosis or manic switch.

0179 - The Impact of Perfectionism on Emotional Dysregulation in Adults with ADHD

Objective

Attention-Deficit/Hyperactivity Disorder (ADHD) is a complex, lifelong neurodevelopmental disorder affecting approximately 2.5% of the adult population. While the core diagnostic criteria established by the DSM-5 primarily focus on inattention, hyperactivity, and impulsivity; contemporary clinical research increasingly emphasizes the role of emotional dysregulation (Ed) as a core clinical phenotype. In adults with ADHD, ED is characterized by heightened emotional lability, irritability, and a diminished capacity to modulate emotional responses to routine daily stressors. To compensate for pervasive executive function deficits—such as impaired working memory and cognitive flexibility—and to mitigate the stigma associated with perceived "failure," many patients develop sophisticated compensatory mechanisms. One of the most prevalent yet clinically complex strategies is perfectionism. Perfectionism is traditionally bifurcated into two dimensions: "High Standards," which can be adaptive, and "Discrepancy," which is inherently maladaptive. The latter represents the subjective gap between an individual's unattainable goals and their actual performance, frequently precipitating a chronic cycle of self-criticism and profound emotional distress. Although clinical observations often suggest a correlation between these constructs, empirical research examining the direct impact of perfectionism on emotion regulation in adults with ADHD remains virtually nonexistent. This study aims to fill this critical gap by testing the hypothesis that perfectionism, specifically its maladaptive component, serves as a significant and independent predictor of emotional dysregulation in this population.

Method

The study sample consisted of 65 adults (18-60 years) diagnosed with ADHD via SCID-5 interviews. Exclusion criteria included psychiatric comorbidities, neurological disorders, and substance use. Participants completed the Wender-Utah Rating Scale (WURS), Adult ADHD Self-Report Scale (ASRS), Difficulties in Emotion Regulation Scale-16 (DERS-16), and the Short Form of the Almost Perfect Scale-Revised (SAPS). Statistical analyses involved Pearson correlations and multiple linear regression to evaluate the predictive role of perfectionism on emotion regulation, while controlling for age, sex, and ADHD symptom severity. Ethical approval was obtained from the Ege University Faculty of Medicine Clinical Research Ethics Committee under approval number 25-4T/86, approval date April 17, 2025. All participants provided informed consent.

Results

The study sample (N = 65) had a mean age of 29.82 ± 10.55 years. Mean scores for ADHD symptom severity were 57.32 ± 7.20 on the ASRS and 56.57 ± 10.53 on the WURS. A significant, strong positive correlation was found between total perfectionism (SAPS) and emotional dysregulation (DERS-16) ($r = .67$, $p < .001$). Subscale analysis revealed that the "Discrepancy" dimension (representing maladaptive perfectionism) exhibited a stronger association with emotional dysregulation ($r = .65$, $p < .001$) compared to the "High Standards" dimension ($r = .47$, $p < .001$). Additionally, DERS-16 scores correlated significantly with WURS ($r = .33$, $p < .001$) and ASRS ($r = .31$, $p < .001$) scores. The multiple linear regression model explained 48% of the total variance in emotional dysregulation (Adj. $R2 = .48$, $F(4,60) = 15.74$, $p < .001$). Notably, after controlling for age, sex, and ADHD symptom severity (ASRS), perfectionism remained the strongest independent predictor of emotional dysregulation ($\beta = .58$, $p < .001$). When perfectionism was included in the model, the direct effect of current ADHD symptom severity (ASRS) on emotional dysregulation fell below the threshold of statistical significance ($p = .075$), and demographic factors showed no significant impact.

Discussion

To our knowledge, this is the first clinical study to empirically demonstrate that perfectionism is a primary independent predictor of emotional dysregulation (ED) in adults with ADHD. Our findings indicate that perfectionism outweighs the impact of core ADHD symptoms—such as inattention and hyperactivity—in explaining the variance of emotional instability within this population. Specifically, the regression model revealed

that once perfectionism was accounted for, the direct effect of current ADHD symptom severity on emotional dysregulation fell below the threshold of statistical significance. This suggests that the emotional volatility frequently observed in clinical ADHD phenotypes may be driven more by internal cognitive-evaluative processes than by the neurobiological core of the disorder itself.

The subscale analysis provides deeper insight into this relationship, showing that the "Discrepancy" dimension—representing maladaptive perfectionism or the perceived gap between one's performance and personal standards—exhibited a significantly stronger association with ED compared to the "High Standards" dimension. This implies that for an adult with ADHD, the psychological distress is not necessarily derived from having high aspirations, but rather from the chronic, self-critical perception of failing to meet those aspirations. Given that individuals with ADHD often struggle with executive dysfunction, they may adopt perfectionism as a compensatory strategy to avoid the stigma of failure; however, this strategy appears to be counterproductive, leading to a cycle of frustration and emotional reactivity.

These results underscore a critical clinical necessity: the routine screening for perfectionistic traits in adults diagnosed with ADHD. Standard pharmacological treatments or organizational coaching may not be sufficient if the underlying driver of emotional distress is a maladaptive perfectionistic schema. Therefore, integrating targeted psychotherapeutic interventions—such as Cognitive Behavioral Therapy (CBT) specifically adapted for perfectionism—into standard ADHD care could significantly enhance emotional regulation and improve functional outcomes. By addressing the cognitive "discrepancy" gap, clinicians can help patients develop more realistic self-evaluations, thereby reducing the emotional friction that characterizes the daily lives of many adults with ADHD. Ultimately, this study highlights that managing perfectionism is not just an elective addition to treatment, but a fundamental component of holistic ADHD care that can lead to superior functional recovery and improved quality of life.

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0181 - Insight in Major Depressive Disorder: A Comparative Study of Clinical, Cognitive, and Metacognitive Dimensions and Their Associations with Symptom Severity

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Abstract

Introduction: Major Depressive Disorder (MDD) is a mood disorder characterized by persistent feelings of sadness, alterations in thought, speech, and psychomotor activity, accompanying psychophysiological changes, impairments in cognitive functioning, anhedonia, loss of interest in daily activities, and pervasive thoughts of worthlessness and hopelessness (1). MDD is the most prevalent psychiatric disorder, often recurrent in nature, and associated with significant functional impairment. According to the World Health Organization, it is projected to become the leading cause of global disease burden by 2030 (2).

Despite extensive research, there is no clear consensus on the conceptualization of insight in psychiatry. While early approaches considered insight as a unidimensional construct focused solely on illness awareness, later models proposed a multidimensional structure including awareness of illness, the ability to recognize one's experiences as abnormal, and treatment adherence (3). Subsequently, the recognition that symptoms are attributable to a mental disorder was added as a fourth component (4). More recent perspectives conceptualize insight as a multidimensional construct encompassing clinical, cognitive, and metacognitive domains (5). Examining insight within these dimensions enables a more individualized and patient- centered approach to intervention (6).

Although insight has been widely studied in various psychiatric disorders, it has received relatively limited attention in MDD. However, impaired insight may play a significant role in the chronic course of depression. Identifying patients at risk for poor insight and targeting insight improvement may positively influence the trajectory of the disorder (7).

Metacognition refers to an overarching system that enables individuals to be aware of and regulate their cognitive processes, including perception, memory, and thinking, in line with specific goals (8). Previous studies have emphasized that negative thinking patterns observed in MDD are strongly influenced by metacognitive processes (9). Metacognitive insight can be defined as the ability to evaluate oneself as a subject experiencing a mental disorder in a consistent and reflective manner (5).

To date, no study has simultaneously examined the relationships between multiple insight measures and depressive symptom severity, nor the predictive value of different insight dimensions and metacognitive functions on symptom severity in MDD.

Therefore, the aim of the present study was to investigate the correlations among different insight scales, evaluate their relationships with depression severity, and determine their predictive value for depressive symptoms.

Hypotheses

- **H1:** There is a significant relationship between clinical, cognitive, and metacognitive insight levels and depressive symptom severity in patients with MDD.
- **H2:** Impairments in metacognitive functions are associated with increased symptom severity as assessed by both clinicians and patients.
- **H3:** Different insight scales are not strongly correlated with each other and do not demonstrate full concordance.
- **H4:** Clinical insight is the strongest predictor of clinician-rated depression severity, whereas self-reflection and cognitive insight are the strongest predictors of self- reported symptom severity.

Methods: This study was conducted between June 1, 2023, and December 31, 2023, at the Psychiatry Clinic of Sakarya University Training and Research Hospital. Ethical approval was obtained from the Sakarya University Faculty of Medicine Ethics Committee on May 31, 2023 (approval number: E-249232-183).

A priori power analysis was conducted using G*Power (version 3.1.9.7 for Windows). With an alpha level of 0.05 and a statistical power of 0.95, assuming a medium effect size (d = 0.5), the minimum required sample size was calculated as 56 participants.

Patients diagnosed with MDD according to DSM-5 criteria and currently in remission (not meeting diagnostic criteria for at least two months) were included. Exclusion criteria comprised intellectual disability, psychotic disorders, organic mental disorders, substance or alcohol use disorders, significant risk factors for bipolar disorder, and a history of treatment with antipsychotics or mood stabilizers.

Structured Clinical Interview for DSM-5 (SCID-1) assessments were conducted by a psychiatry resident with three years of training under the supervision of experienced clinicians. A total of 80 patients (40 males, 40 females) meeting the inclusion criteria were enrolled.

Clinician-administered measures included: Hamilton Depression Rating Scale (HDRS), Brown Assessment of Beliefs Scale (BABS), Scale to Assess Unawareness of Mental Disorder (SUMD), Scale to Assess the Three Components of Insight (SAI).

Self-report measures included: Beck Depression Inventory (BDI), Beck Cognitive Insight Scale (BCIS), Self-Reflection and Insight Scale (SRIS), Metacognition Questionnaire (MCQ).

Statistical analyses were performed using IBM SPSS Statistics 22.0. Group comparisons were conducted using chi-square tests and independent samples t-tests. ANOVA with post-hoc tests (Tukey, Scheffe, Gabriel) was used for comparisons involving more than two groups. Relationships between continuous variables were analyzed using Pearson and Spearman correlation coefficients. Linear regression analyses were conducted to examine predictive relationships.

Results: The findings of the study demonstrated the relationships between symptom severity and insight dimensions, as well as their predictive capacity. A moderate, positive, and statistically significant correlation was found between clinician-rated (HDRS) and self- reported (BDI) depression severity ($r = 0.569$, $p < 0.001$).

In line with our hypotheses, clinical insight (SAI) was negatively correlated with HDRS scores ($r = -0.408$, $p < 0.001$), whereas unawareness of illness (SUMD) was positively correlated with HDRS scores ($r = 0.376$, $p = 0.001$). Metacognitive dysfunction (MCQ) showed a weak but significant positive correlation with HDRS scores ($r = 0.260$, $p = 0.020$). BDI scores were weakly but significantly negatively correlated only with SRIS ($r = -0.263$, $p = 0.018$). Structural validity analyses revealed a very strong negative correlation between SAI and SUMD ($r = -0.888$, $p < 0.001$). MCQ was weakly but significantly positively correlated with BCIS ($r = 0.254$, $p = 0.023$) and SRIS ($r = 0.222$, $p = 0.048$).

Multiple linear regression analyses confirmed the unique contributions of insight dimensions in predicting symptom severity. The model including SAI, MCQ, and BABS explained 24.6% of the variance in HDRS scores ($R2 = 0.246$), with SAI emerging as the strongest predictor ($\beta = -0.410$), accounting for 15.8% of the variance. In the model predicting BDI scores, SRIS and MCQ explained 10.6% of the variance ($R2 = 0.106$), with SRIS identified as the strongest predictor ($\beta = -0.265$).

Overall, clinical insight (SAI) primarily predicted clinician-rated symptom severity, whereas cognitive insight and self-reflection (SRIS) were associated with self-reported severity. MCQ functioned as a common factor influencing both types of assessments. (Table.1)

Discussion: The present study provides novel evidence regarding the multidimensional nature of insight in MDD and its relationship with symptom severity. Our findings indicate that clinical insight (SAI), cognitive insight (BCIS, SRIS), and metacognitive functioning (MCQ) are distinct yet interrelated constructs that differentially predict depressive symptoms, depending on the method of assessment (clinician-rated vs. self-reported).

A key finding is the strong negative correlation between SAI and SUMD, confirming that these clinical insight measures assess opposing dimensions of illness awareness, yet remain structurally consistent (3). Cognitive insight

measures (BCIS, SRIS) demonstrated weak but significant correlations, supporting the multidimensional conceptualization of insight in MDD (4).

Contrary to the "Depressive Realism" hypothesis, which posits heightened self-awareness in depression (10), our results revealed that more severe depressive symptoms were associated with lower insight across all dimensions. This pattern aligns with recent models suggesting that excessive rumination and dysfunctional metacognitive beliefs (MCQ) may impair patients' ability to accurately evaluate their illness, creating a self-reinforcing cycle that diminishes insight (6, 8).

Regression analyses further clarified that clinical insight (SAI) was the strongest predictor of clinician-rated symptom severity (HDRS), whereas self-reflection and cognitive insight (SRIS) were more predictive of self-reported severity (BDI). Metacognitive dysfunction (MCQ) emerged as a common factor affecting both measures, highlighting its central role in symptom appraisal and insight formation in MDD (4,6).

These findings have important clinical implications. Interventions targeting not only mood symptoms but also metacognitive and cognitive insight may improve both symptom management and illness awareness, potentially reducing relapse risk and enhancing functional outcomes (11). Moreover, assessment of insight in MDD should adopt a multidimensional approach rather than relying solely on clinician observation or self-report.

Limitations of the study include the cross-sectional design, which precludes causal inference, and the relatively modest sample size. Future studies with longitudinal designs and larger samples are warranted to clarify the temporal relationships between depressive symptom severity, multidimensional insight, and metacognitive processes.

In conclusion, our findings support the view that insight in MDD is a multidimensional construct encompassing clinical, cognitive, and metacognitive domains. Recognizing the unique contributions of each dimension is essential for both research and clinical practice, and interventions aimed at enhancing insight may yield significant benefits in symptom reduction and patient self-management.

Keywords: Insight, insight scales, major depressive disorder, symptom severity. metacognition

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Table 1. Insight Scales: Correlations and Predictive Value for Depressive Symptoms

Insight Scale	Domain	Correlation with Other Scales	Correlation with Symptom Severity	Predictive Value (Regression)
SUMD	Clinical	Negative with SAI	↑ HDRS with | SUMD	Strongest predictor of HDRS
SAI	Clinical	Negative with SUMD	↑ HDRS with t SAI	Moderate predictor of HDRS
BABS	Clinical	Positive with MCQ	Not significant	Third predictor of HDRS
BCIS	Cognitive	Positive with MCQ & SRIS	↓ BDI with ↑ BCIS	Predictor of BDI (moderate)
SRIS	Cognitive	Positive with MCQ & BCIS	↓ BDI with ↑ SRIS	Strongest predictor of BDI
MCQ	Metacognitive	Positive with BCIS & SRIS	↑ HDRS & ↑ BDI with ↑ MCQ	Predictor of both HDRS & BDI

* The title has been updated following the abstract submission to more accurately reflect the scope and dimensions of the study.

0185 - Serotonin Syndrome Following the Addition of Linezolid in a Patient Receiving SSRI Pharmacotherapy: A Brief Report

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Abstract

Background: Serotonin syndrome (SS) is a potentially life-threatening condition caused by excessive serotonergic activity, most commonly resulting from drug interactions. Linezolid, an oxazolidinone antibiotic with reversible monoamine oxidase inhibitor (MAOI) activity, can precipitate SS when co-administered with selective serotonin reuptake inhibitors (SSRIs). Recognition may be delayed in hospitalized patients because the clinical features overlap with sepsis and delirium.

Methods: We describe the case of a 58-year-old man with major depressive disorder in remission, maintained on escitalopram 20 mg/day, who developed SS approximately 48 hours after linezolid 600 mg twice daily was added for a diabetic foot infection. Diagnosis was established using the Hunter Serotonin Toxicity Criteria.

Results: The patient presented with agitation, hyperthermia, tachycardia, hypertension, diaphoresis, hyperreflexia, tremor, and spontaneous clonus. Following discontinuation of both linezolid and escitalopram and initiation of supportive care including benzodiazepines, symptoms resolved within 48-72 hours.

Conclusion: This case underscores the importance of medication reconciliation and multidisciplinary communication when prescribing linezolid to patients receiving serotonergic agents. Clinicians should consider SS in the differential diagnosis of fever and agitation in hospitalized patients, particularly when neuromuscular findings such as clonus are present.

Keywords: Drug-induced toxicity, escitalopram, linezolid, serotonin syndrome, SSRI interaction

Main Points

1. Linezolid possesses reversible, non-selective monoamine oxidase inhibitor activity that is frequently overlooked outside psychiatric practice, creating a significant risk for serotonin syndrome when combined with SSRIs.

2. In hospitalized patients with active infections, the clinical features of serotonin syndrome— including fever, agitation, and altered mental status—may be misattributed to sepsis or delirium, leading to delayed diagnosis.

3. The Hunter Serotonin Toxicity Criteria provide a practical bedside tool for diagnosing serotonin syndrome; the presence of spontaneous clonus in a patient receiving a serotonergic agent is highly suggestive.

4. Systematic medication reconciliation at every prescribing step and timely multidisciplinary consultation between infectious diseases and psychiatry teams are essential to prevent potentially life-threatening drug interactions.

Introduction

Serotonin syndrome (SS) is a potentially life-threatening toxidrome resulting from excessive serotonergic activity in the central and peripheral nervous systems. It classically presents with a triad of altered mental status, autonomic instability, and neuromuscular hyperactivity.[1,2] The condition most frequently arises from pharmacological

interactions between two or more serotonergic agents, and its severity ranges from mild tremor and diarrhea to life-threatening hyperthermia, rhabdomyolysis, and multi-organ failure.[3]

Linezolid is an oxazolidinone-class antibiotic widely used for the treatment of resistant gram- positive infections, including methicillin-resistant Staphylococcus aureus (MRSA) and vancomycin-resistant Enterococcus (VRE). Beyond its antimicrobial properties, linezolid acts as a reversible, non-selective monoamine oxidase inhibitor (MAOI).[4,5] This pharmacological property is frequently underrecognized by non-psychiatric prescribers, creating a clinically significant risk when linezolid is co-administered with serotonergic antidepressants such as selective serotonin reuptake inhibitors (SSRIs). In hospitalized patients with active infections, the overlapping clinical features of SS—fever, tachycardia, and altered mental status—may be misattributed to sepsis or delirium, thereby delaying appropriate recognition and management.[6]

In this brief report, we present the case of a patient on long-term escitalopram therapy who developed SS following the addition of linezolid for a diabetic foot infection. We discuss the diagnostic approach using the Hunter Serotonin Toxicity Criteria and emphasize the critical role of medication reconciliation and multidisciplinary collaboration in preventing this potentially fatal drug interaction.

Case Presentation

A 58-year-old man was admitted to the infectious diseases ward for the management of a diabetic foot infection. His medical history was notable for type 2 diabetes mellitus and major depressive disorder (MDD) in sustained remission. He had been receiving escitalopram 20 mg/day for several years with good adherence and reported no active psychiatric symptoms at the time of admission. Baseline vital signs were within normal limits (temperature 37.4°C, heart rate 84 beats per minute [bpm], blood pressure 130/80 mmHg).

Due to persistent infection despite ongoing antimicrobial therapy, the infectious diseases team initiated linezolid 600 mg intravenously twice daily. Approximately 48 hours after linezolid initiation, the patient developed marked psychomotor restlessness, inability to remain still, increased speech output, fluctuating confusion, and profuse diaphoresis. His temperature rose to 39.1°C, heart rate increased to 118 bpm, and blood pressure elevated to 150/90 mmHg. Neurological examination revealed prominent lower-extremity hyperreflexia (3+), generalized tremor, myoclonic jerks, and sustained ankle clonus with intermittent spontaneous clonic episodes.

Initially, progression of sepsis was considered as the primary etiology. However, laboratory parameters did not fully support this hypothesis: white blood cell count was 11,200/mm^3 and C- reactive protein was 62 mg/L, both comparable to prior values. Creatine kinase was mildly elevated at 310 U/L. Blood cultures obtained at the time of symptom onset did not yield any new pathogens. Given the combination of acute mental status change and prominent neuromuscular findings disproportionate to the infectious picture, a psychiatric consultation was requested.

On psychiatric assessment, the patient exhibited marked agitation and distractibility. Sustained ankle clonus and diffuse hyperreflexia were confirmed on examination. A focused medication review identified the recent addition of linezolid to ongoing SSRI therapy as the probable precipitant. Based on the Hunter Serotonin Toxicity Criteria—specifically, the presence of a serotonergic agent combined with spontaneous clonus, associated agitation, and hyperthermia—a diagnosis of SS was established.[2]

In collaboration with the infectious diseases team, both linezolid and escitalopram were promptly discontinued. Supportive management was initiated, including intravenous fluid resuscitation, active cooling measures, and benzodiazepine administration for agitation and neuromuscular hyperactivity. The patient demonstrated rapid clinical improvement; autonomic instability and neuropsychiatric symptoms diminished substantially within 48 hours, and neuromuscular hyperactivity fully resolved within 72 hours. An alternative antibiotic regimen was selected for the ongoing management of the diabetic foot infection. The patient was subsequently discharged in stable condition, and psychiatric follow-up was arranged for reassessment of antidepressant therapy.

Discussion

This case illustrates the clinical significance of the pharmacological interaction between linezolid and SSRIs and highlights the diagnostic challenges posed by SS in the setting of active infection. The MAOI activity of linezolid,

although well-documented in the pharmacological literature, remains underappreciated in routine clinical practice, particularly among non-psychiatric prescribers.[4,5]

The Hunter Serotonin Toxicity Criteria, which demonstrate a sensitivity of 84% and specificity of 97%, provide a practical and reliable bedside diagnostic framework.[2] In our patient, the combination of a serotonergic agent exposure, spontaneous clonus, agitation, and hyperthermia fulfilled these criteria. Notably, the presence of clonus—particularly spontaneous and sustained ankle clonus—is considered one of the most reliable clinical indicators of SS and serves as a key distinguishing feature from sepsis-related encephalopathy and neuroleptic malignant syndrome.[1,7]

A systematic review by Woytowish and Maynard reported that the incidence of SS in patients receiving concurrent linezolid and serotonergic agents ranges from 0.24% to 4%, depending on the study population and the specific serotonergic agent involved.[8] Although this incidence may appear low, the potentially fatal consequences of SS warrant a high index of clinical suspicion. The United States Food and Drug Administration (FDA) issued a drug safety communication in 2011 advising against the concomitant use of linezolid with serotonergic psychiatric medications unless no acceptable alternatives exist.[9]

Several factors contributed to the initial diagnostic uncertainty in this case. First, the patient was being treated for an active infection, making fever and tachycardia expected findings. Second, the altered mental status could plausibly have been attributed to sepsis-associated delirium. Third, the MAOI properties of linezolid may not have been immediately recognized by the primary treating team. These factors underscore the importance of systematic medication reconciliation at every prescribing step and the value of early psychiatric consultation when unexplained neuromuscular findings accompany mental status changes in medically ill patients.[10]

The management of SS primarily involves discontinuation of the offending serotonergic agents and provision of supportive care. Benzodiazepines are recommended for agitation and may also attenuate neuromuscular hyperactivity. In severe cases, cyproheptadine, a serotonin antagonist, may be considered, although evidence for its efficacy remains limited to case reports and case series.[1,3] In our patient, prompt discontinuation of both linezolid and escitalopram, combined with supportive measures, resulted in complete resolution of symptoms within 72 hours, consistent with the expected time course given the reversible nature of linezolid's MAOI activity and the pharmacokinetic profile of escitalopram.

This report has several limitations inherent to single-case observations. The Naranjo Adverse Drug Reaction Probability Scale was not formally applied, although the temporal relationship, clinical presentation, and resolution upon drug withdrawal strongly support a causal association. Additionally, serotonin levels were not measured, as there is no readily available laboratory test for SS; the diagnosis remains clinical.[11]

Conclusion

Linezolid, through its reversible MAOI activity, can precipitate serotonin syndrome when combined with SSRIs. In hospitalized patients with active infections, the emergence of fever, agitation, and altered mental status should not be attributed solely to sepsis, particularly when neuromuscular findings such as clonus and hyperreflexia are present. This case reinforces the necessity of thorough medication reconciliation, awareness of non-obvious drug interactions, and timely multidisciplinary communication between infectious diseases and psychiatry teams to ensure early recognition and prompt management of this potentially life-threatening condition.

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0186 - Resolution of Antipsychotic-Induced Hyperprolactinemia After Switching from Risperidone to Lurasidone: A Case Report

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Short Title: Lurasidone Switch for Hyperprolactinemia
Previous Presentation: This case was previously presented as a poster at the 2026 International Congress of Psychiatry.

Main Points

- Risperidone-induced hyperprolactinemia can cause clinically significant reproductive and sexual dysfunction even in psychiatrically stable patients.
- Switching from risperidone to lurasidone resulted in rapid prolactin normalization within 8 weeks without compromising psychotic symptom control.
- Lurasidone's favorable receptor-binding profile with moderate D2 occupancy makes it a suitable prolactin-sparing alternative in patients requiring long-term antipsychotic therapy.
- Routine prolactin monitoring and proactive assessment of sexual and reproductive side effects should be integral components of long-term antipsychotic management.
- Individualized, biologically informed treatment strategies can effectively balance symptom control with endocrine safety and quality of life.

Abstract

Objective: Antipsychotic-induced hyperprolactinemia is a common but often underrecognized adverse effect that can significantly impair quality of life. This brief report describes the successful management of risperidone-induced hyperprolactinemia by switching to lurasidone in a psychiatrically stable patient with schizophrenia.

Case: A 43-year-old woman with an 8-year history of schizophrenia, stable on risperidone 4 mg/day for three years, presented with secondary amenorrhea, loss of libido, and marital distress due to sexual dysfunction. Serum prolactin was markedly elevated at 80 ng/mL. Pituitary imaging and other investigations excluded alternative etiologies. A cross-tapering strategy was implemented, gradually discontinuing risperidone while initiating lurasidone 40 mg/day. Prolactin levels declined to 28 ng/mL at week 4 and normalized to 16 ng/mL by week 8, with resumption of menstrual cycles and improvement in sexual functioning. Positive and Negative Syndrome Scale scores remained stable throughout.

Conclusion: Switching from risperidone to lurasidone effectively resolved hyperprolactinemia and associated reproductive and sexual dysfunction without compromising psychiatric stability. This case underscores the importance of routine prolactin monitoring and individualized antipsychotic selection in long-term management.

Keywords: Amenorrhea, antipsychotic switching, hyperprolactinemia, lurasidone, risperidone, sexual dysfunction

Introductıon

Antipsychotic-induced hyperprolactinemia is one of the most prevalent yet frequently overlooked adverse effects of antipsychotic pharmacotherapy. It occurs primarily through blockade of dopamine D2 receptors in the tuberoinfundibular pathway, which disrupts the tonic dopaminergic inhibition of prolactin secretion from the anterior pituitary gland (1). The prevalence of hyperprolactinemia varies considerably across antipsychotic agents, with potent D2 receptor antagonists such as risperidone and paliperidone being the most commonly implicated (2).

The clinical consequences of sustained hyperprolactinemia extend beyond endocrine disruption. In women, elevated prolactin levels may manifest as menstrual irregularities including amenorrhea, galactorrhea, sexual dysfunction with diminished libido, and infertility. In men, gynecomastia, erectile dysfunction, and decreased spermatogenesis may occur. Long-term hyperprolactinemia has also been associated with decreased bone mineral density and potential cardiovascular risk (1,3). Beyond these biological effects, the psychosocial impact of sexual and reproductive dysfunction can be substantial, contributing to reduced treatment adherence, impaired quality of life, and relationship difficulties.

When antipsychotic-induced hyperprolactinemia is identified, management strategies include dose reduction, addition of a dopamine agonist, or switching to a prolactin-sparing antipsychotic agent (3). Among second-generation antipsychotics, lurasidone has been characterized by moderate D2 receptor occupancy and a favorable receptor-binding profile that includes potent 5- HT2A and 5-HT7 antagonism, with minimal prolactin-elevating potential (4). This pharmacological profile makes lurasidone a rational alternative in patients experiencing clinically significant hyperprolactinemia.

We present the case of a psychiatrically stable woman with schizophrenia who developed symptomatic hyperprolactinemia during long-term risperidone treatment, which resolved following a switch to lurasidone.

Case Presentation

A 43-year-old married woman with an 8-year history of schizophrenia presented to the outpatient psychiatry clinic with complaints of secondary amenorrhea persisting for 6 months, significant loss of libido, and progressive marital distress attributed to sexual dysfunction. She had been clinically stable on risperidone 4 mg/day for the preceding three years, with no recent hospitalizations, symptom exacerbations, or medication changes. She denied galactorrhea, visual disturbances, or headaches.

Laboratory evaluation revealed a markedly elevated serum prolactin level of 80 ng/mL (normal range: 2-29 ng/mL). Thyroid function tests, renal function panels, and hepatic function tests were all within normal limits. A pregnancy test was negative. Pituitary magnetic resonance imaging (MRI) demonstrated no evidence of adenoma or other structural pathology. A comprehensive medication review identified no other agents associated with prolactin elevation. Based on these findings, the clinical picture was consistent with risperidone-induced hyperprolactinemia.

Given the patient's sustained psychiatric stability and the significant impact of hyperprolactinemia on her quality of life, a decision was made to switch antipsychotic therapy. A cross-tapering strategy was implemented: risperidone was gradually reduced and discontinued over a two-week period while lurasidone 40 mg/day (administered with food to ensure adequate absorption) was simultaneously initiated and maintained.

Follow-up assessments demonstrated a progressive decline in prolactin levels. At week 4, serum prolactin had decreased to 28 ng/mL. By week 8, prolactin levels had normalized to 16 ng/mL. Concurrently, the patient reported resumption of regular menstrual cycles and marked improvement in libido and overall sexual functioning. Importantly, no exacerbation of psychotic symptoms was observed during the transition period or subsequent follow-up. Positive and Negative Syndrome Scale (PANSS) total scores remained stable throughout the observation period. No new adverse effects attributable to lurasidone were reported.

Discussion

This case illustrates the clinically meaningful impact of antipsychotic-induced hyperprolactinemia and demonstrates the efficacy of switching to a prolactin-sparing agent as a management strategy. Risperidone is well established as one of the most potent prolactin- elevating antipsychotics due to its high affinity for D2 receptors and its limited penetration of the blood-brain barrier at the pituitary level, resulting in sustained tuberoinfundibular D2 blockade (1,2). In our patient, prolonged risperidone use led to a prolactin level nearly three times the upper limit of normal, with consequent amenorrhea and sexual dysfunction.

The decision to switch to lurasidone was based on its pharmacological profile. Lurasidone exhibits moderate D2 receptor affinity with a dissociation rate that allows for sufficient dopaminergic tone in the tuberoinfundibular pathway, thereby minimizing prolactin elevation (4). Clinical trials and post-marketing data have consistently demonstrated that lurasidone is associated with minimal or no clinically significant prolactin increase compared

with risperidone and paliperidone (2,4). In our case, the rapid normalization of prolactin levels within 8 weeks supports these pharmacological observations.

The successful resolution of amenorrhea and sexual dysfunction following the switch highlights the reversibility of risperidone-induced hyperprolactinemia when the causative agent is removed. This finding is consistent with previous reports demonstrating that prolactin levels typically normalize within weeks of discontinuing a prolactin-elevating antipsychotic or switching to a prolactin-sparing alternative (3). The maintenance of psychiatric stability throughout the transition, as evidenced by stable PANSS scores, further supports the clinical utility of lurasidone as an effective antipsychotic with a favorable endocrine profile.

Several important clinical implications emerge from this case. First, routine monitoring of prolactin levels should be considered in patients receiving antipsychotics known to elevate prolactin, particularly risperidone and paliperidone. Second, clinicians should proactively inquire about sexual and reproductive side effects, as patients may not spontaneously report these symptoms due to embarrassment or lack of awareness of their association with medication. Third, when hyperprolactinemia is identified, switching to a prolactin-sparing antipsychotic such as lurasidone, aripiprazole, or quetiapine should be considered, particularly in patients who are psychiatrically stable (2,3).

A limitation of this report is that it describes a single case without a control condition, and the generalizability of these findings requires confirmation through larger controlled studies. Additionally, longer-term follow-up data would be valuable to assess the durability of prolactin normalization and sustained psychiatric stability.

Conclusion

This case demonstrates that switching from risperidone to lurasidone can effectively resolve antipsychotic-induced hyperprolactinemia and its associated reproductive and sexual consequences without compromising psychotic symptom control. The findings underscore the importance of individualized, biologically informed treatment strategies that balance efficacy with endocrine safety. Routine prolactin monitoring and proactive assessment of sexual and reproductive side effects should be integral components of comprehensive long-term antipsychotic management.

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0188 - Storytelling in Child Psychiatry Education: "If Fluoxetine Could Talk" as an Innovative Teaching Method

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Objective

Medical education requires the effective structuring of learning due to its high cognitive load and conceptual complexity. Students are expected not only to acquire theoretical knowledge but also to interpret and apply this knowledge within a clinical framework. These demands highlight the importance of approaches that go beyond simple knowledge acquisition and support meaning-making and cognitive organization.

Among innovative educational approaches, storytelling (narrative-based) has gained increasing attention due to its ability to engage both cognitive and emotional processes. Presenting information within a coherent and meaningful context facilitates the integration of fragmented knowledge and promotes deeper learning. In this respect, **narrative-based learning** has been conceptualized as an educational approach that structures knowledge through meaningful narratives. In addition, the narrative medicine framework underscores the role of such approaches in enhancing empathy and clinical reasoning in medical education [1].

Psychopharmacology education presents particular challenges, as it requires learners to integrate abstract neurobiological mechanisms with clinical outcomes. Core concepts—such as mechanisms of action, receptor interactions, and pharmacokinetic processes—are often learned in a fragmented and rote manner. This fragmentation may limit knowledge retention and hinder the application of knowledge in clinical settings. Therefore, psychopharmacology education requires pedagogical approaches that support meaningful and structured knowledge organization.

In this study, storytelling is reconceptualized as "narrative-based psychopharmacology education," a structured and theoretically grounded pedagogical approach that extends beyond modifying content delivery to fundamentally transforming how knowledge is cognitively organized by learners. Within this framework, the present study investigates the effects of a **researcher-developed, story-based educational intervention,** entitled ***"Worlds Colored by the Journey with Fluoxetine,"*** centered on the commonly used active ingredient fluoxetine in child psychiatry clinical practice, on medical students' learning processes and academic performance, in comparison with traditional teaching methods. Furthermore, the study evaluates students' views and attitudes toward this innovative learning approach.

Methods

This study was designed as a controlled pretest-posttest educational intervention. A total of 54 third-year medical students were included. Third-year students were specifically selected because they possess general pharmacological knowledge but have not yet received structured education in psychopharmacology. This allowed for a clearer evaluation of the effect of the intervention.

Participants were allocated into two groups matched for age and sex: a theoretical education group (n=27) and a narrative-based education group (n=27).

In the narrative-based group, an original narrative text entitled **"Worlds Colored by the Journey with Fluoxetine,"** developed by the researcher, was used. In this narrative, psychopharmacological information related to fluoxetine was presented within a structured and chronological storyline. Mechanism of action, indications, contraindications, adverse effects, and pharmacokinetic properties were integrated within a coherent contextual framework. In the theoretical education group, the same content was delivered using a traditional didactic method.

Both groups were taught under comparable classroom conditions, for equal durations, and by the same instructor. The only difference between groups was the format of content delivery.

Participants' knowledge levels were assessed using a 19-item multiple-choice fluoxetine knowledge test administered before and after the intervention. The test covered the mechanism of action, indications, contraindications, adverse effects, pharmacokinetic properties, and clinically relevant drug interactions.

In addition, students' attitudes toward the educational method were evaluated using a structured questionnaire.

Statistical analyses were performed using SPSS software. Between-group comparisons were conducted using independent samples t-test or Mann-Whitney U test, while within-group comparisons were analyzed using appropriate paired tests, including the Wilcoxon signed-rank test when necessary. A p-value of <0.05 was considered statistically significant.

Due to non-normal distribution, statistical analyses were conducted using median values, while figures present mean scores for visualization purposes.

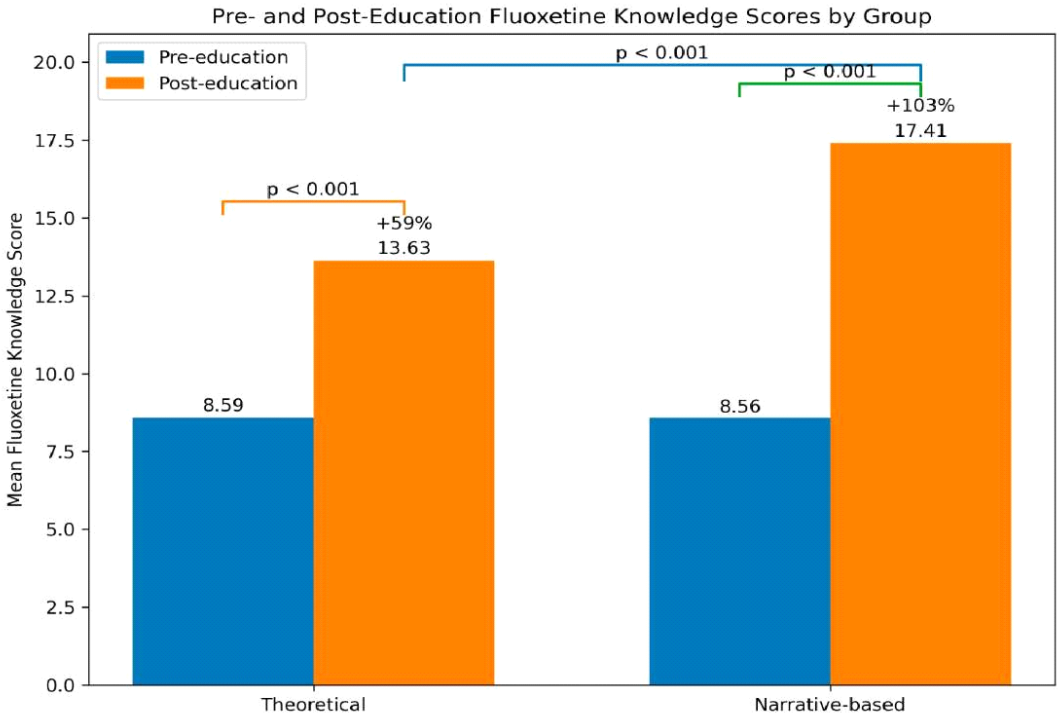
Results

The sociodemographic characteristics of the participants did not differ significantly between the groups ($p > 0.05$).

There was no statistically significant difference between the theoretical and narrative- based education groups in the pre-education fluoxetine knowledge test scores ($p = 0.967$).

Following the educational intervention, a statistically significant increase in knowledge scores was observed in both groups ($p < 0.001$ for both groups). In the pre-post comparison, mean scores in the theoretical group increased from 8.59 ± 3.31 to 13.63 ± 2.98, while in the narrative-based group they increased from 8.56 ± 3.31 to 17.41 ± 1.53 In the post-education comparison, knowledge scores were significantly higher in the narrative-based education group compared to the theoretical group ($p < 0.001$). (Figure 1).

Figure 1. Pre- and Post-Education Fluoxetine Knowledge Scores by Group

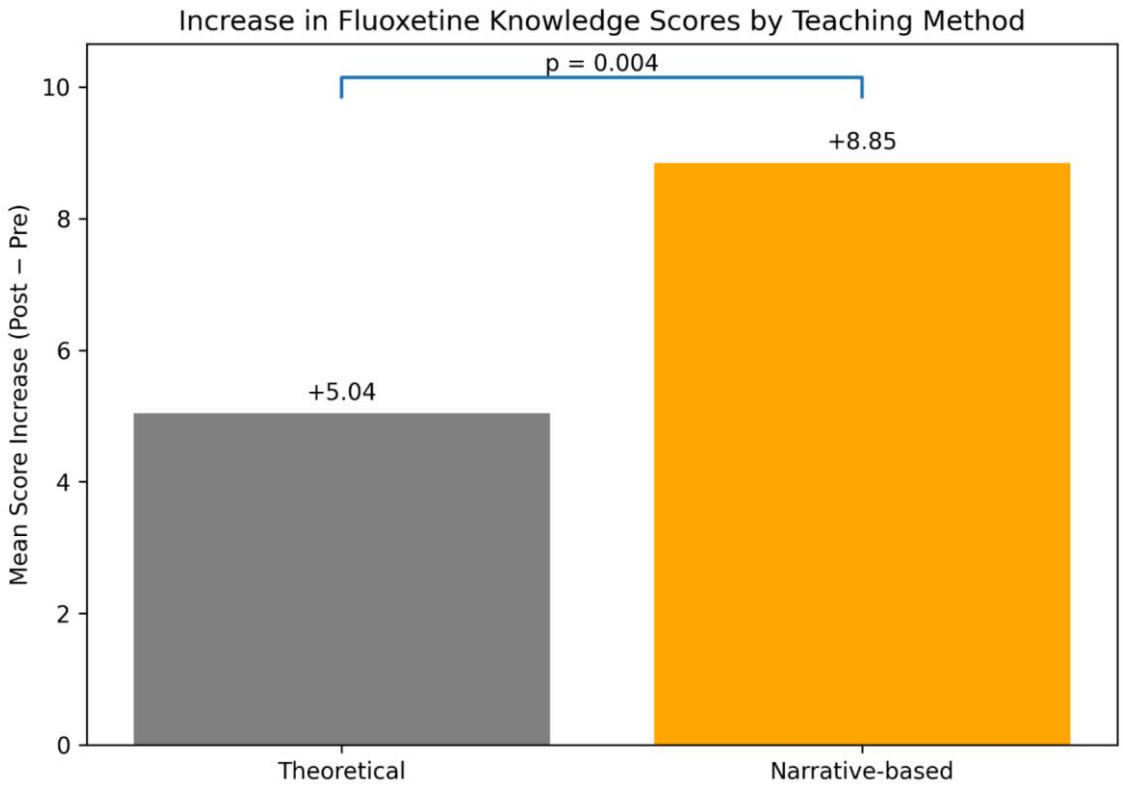


Both groups showed significant increases in knowledge after education ($p < 0.001$). The narrative-based group demonstrated greater improvement than the theoretical group.

When knowledge gain (post-pre difference) was evaluated, the mean increase was +5.04 in the theoretical group and +8.85 in the narrative-based group, with a significantly greater improvement in the narrative-based group (p =

0.004) (Figure 2). Median-based analyses showed approximately 50% and 80% increases, respectively. Figures present mean values, while percentage changes are based on median values.

Figure 2. Increase in Fluoxetine Knowledge Scores by Teaching Method



Mean increase in fluoxetine knowledge scores (post-pre). The narrative-based group showed a significantly greater increase than the theoretical group (p = 0.004).

Most students reported that narrative-based learning facilitated understanding, supported the contextualization of theoretical knowledge, contributed to the development of empathy, and should be used more widely in education. Detailed attitudinal findings are presented in Table 1.

Table 1. Students' Perceptions of Narrative-Based Psychopharmacology Education

Statement	Yes n (%)	No n (%)
Facilitates understanding of topics	52 (96.3)	2 (3.7)
Enhances empathy skills	49 (90.7)	5 (9.3)
Makes class time inefficient	12 (22.2)	42 (77.8)
Supports long-term learning (perceived)	54 (100)	0 (0)
Makes clinical preparation difficult	11 (20.4)	43 (79.6)
Helps contextualize theoretical knowledge	52 (96.3)	2 (3.7)
Increases classroom interaction	38 (70.4)	16 (29.6)
Difficult to assess and grade	18 (33.3)	36 (66.7)
Should be used more in education	49 (90.7)	5 (9.3)
Effective when brief and structured	54 (100)	0 (0)

Note. Students' responses to the storytelling-based psychopharmacology education approach. Results are presented as frequencies and percentages. The item regarding long-term learning reflects students' subjective perceptions.

Discussion

This study demonstrates that adapting storytelling to the content and cognitive complexity of psychopharmacology—conceptualized here as "narrative-based psychopharmacology education"—can produce a significant positive effect on medical students' learning processes and academic performance, compared with traditional teaching methods. These findings suggest that this approach may represent a cognitively transformative model of learning.

Narrative-based learning has been widely emphasized in the literature as supporting learning by presenting information within a meaningful and contextual structure. Information delivered through storytelling has been shown to enhance attention, reduce cognitive load, and improve knowledge retention [2]. Similarly, the narrative medicine approach highlights the positive effects of narratives on the development of empathy and clinical reasoning [3].

The findings of this study are consistent with the existing literature and indicate that a narrative-based approach can also be effective in an abstract and complex field such as psychopharmacology. In particular, the marked increase in learning observed in the narrative-based education group suggests that this approach may enhance both the depth and scope of learning.

Psychopharmacology education inherently requires the integration of complex relationships between neurobiological mechanisms and clinical outcomes. The narrative-based framework may facilitate this process by embedding information within a coherent and temporally structured context, thereby supporting more holistic knowledge organization. This interpretation is also consistent with cognitive load theory, which emphasizes the importance of structured information processing in learning [4]. In addition to its effects on academic performance, the high level of positive student attitudes toward the intervention suggests that this approach may enhance the overall learning experience. In particular, students' perceptions regarding its contribution to empathy development are in line with findings from the narrative medicine literature [1,5].

The study has several limitations. First, long-term learning outcomes were not objectively assessed and were based on students' subjective evaluations. Second, the single-center design and relatively small sample size may limit the generalizability of the findings. Despite these limitations, the study provides an original contribution by experimentally evaluating a narrative-based approach to structuring psychopharmacology education.

In conclusion, the findings of this study provide evidence that a story-based educational approach, centered on fluoxetine, can enhance medical students' learning processes and academic performance compared with traditional teaching methods. Additionally, the consistently positive student views and attitudes toward this approach underscore its potential as a pedagogically robust and engaging strategy for psychopharmacology education.

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0204 - Psilocybin-Induced Manic Episode: An Analysis via Neural Network Connectivity and Cortico-Striatal Dopaminergic Interaction

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Objective

Although psilocybin has emerged as a promising therapeutic agent for treatment-resistant depression in recent years [1], its risk profile in individuals with bipolar spectrum disorders remains poorly understood. Since current clinical trials systematically exclude patients on the bipolar spectrum, prospective data regarding the risk of mania in this population are lacking [2,3]. Evidence for psilocybin-induced mania primarily relies on case reports and retrospective series [2].

Case-based evidence in the literature suggests that psilocybin may trigger manic or mixed episodes, particularly in individuals with diagnosed bipolar disorder, a strong family history, or subclinical bipolar predisposition [3,4]. While the number of reported cases is limited, it is emphasized that the relative risk is clinically significant for those with known or suspected bipolar diathesis [2]. In this study, we discuss a case of a first manic episode following psilocybin use, analyzed in light of current neuroscientific literature.

Case Presentation

A 33-year-old female lawyer (G.O.D.) presented to our clinic with increased irritability, decreased need for sleep, increased psychomotor activity, and paranoid ideation following psilocybin use during a trip to Amsterdam. Symptoms had persisted for approximately two months and showed a clear temporal relationship with psilocybin ingestion. Her medical history included a three-year history of psychoactive substance use and a prior pre-diagnosis of dissociative disorder, for which she had used aripiprazole. Mental state examination revealed poor self-care, pressured speech, loosened associations (derailment), paranoid delusions, impaired judgment, and a lack of insight. Acute stabilization was achieved with Haloperidol and Biperiden. Following symptom control, the patient was discharged in remission with a maintenance treatment plan of Aripiprazole (10 mg/day) and Lithium (600 mg/day).

Neurobiological Discussion

The case of G.O.D. serves as a critical example of a "triggered" first manic episode following psilocybin exposure, aligning with risk projections in the literature. Cases reported by Halim et al. [3], involving a Bipolar II patient shifting to full mania (Bipolar I) after psilocybin, and Hendin & Penn [4], involving an undiagnosed woman with a family history developing acute mania, parallel our findings. The common denominator in these cases is that psilocybin acts as a "precipitant" in neurobiologically predisposed individuals, bringing latent bipolarity to a clinical level.

5-HT2A and Dopaminergic Cross-talk: Psilocybin (via its active metabolite psilocin) increases glutamatergic output by activating 5-HT2A receptors on cortical pyramidal neurons [5, 6]. As demonstrated by Vollenweider et al. [7] through PET studies, this process secondarily triggers striatal dopamine release, increasing D2 receptor occupancy and provoking manic phenotypes. The pressured speech and increased psychomotor activity observed in our patient can be considered clinical reflections of this hyperdopaminergic state.

Network Connectivity and Control Energy: Neuroimaging data indicate that psilocybin disrupts Default Mode Network (DMN) integrity and causes cortical desynchronization [8]. **Salience Network Shift:** 5-HT2A activation can affect the salience network via the claustrum and Anterior Cingulate Cortex (ACC), leading to aberrant salience attribution to external stimuli [9]. The paranoid delusions and faulty external attributions found in our case can be explained by this inter-network connectivity dysfunction. **Flattening of the Energy Landscape:** The "flattening of the control-energy landscape" hypothesis proposed by Singleton et al. [10] suggests that the brain can transition between states more effortlessly. This provides a theoretical framework for the loosened associations and manic lability observed in our patient.

Literature Evidence and Emphasis on First Episode: The most significant factor distinguishing or supporting our case is the induction of a first manic episode by a psychoactive substance (psilocybin) at age 33. As highlighted by Gard et al. [2] in their systematic review, persistent manic symptoms following psilocybin use are typically clustered in individuals with a "bipolar diathesis." Retrospectively, the patient's prior pre-diagnosis of dissociative disorder and aripiprazole use may have been indicators of a subclinical mood spectrum. In this instance, psilocybin lowered the "threshold," making the full manic state manifest.

Conclusion

Psilocybin may trigger latent bipolar disorder or exacerbate existing conditions in susceptible individuals. Clinicians must screen for hallucinogen use in cases of atypical psychosis or mania. In individuals on the bipolar spectrum, rigorous screening and prophylactic mood- stabilizing strategies prior to psilocybin use should be discussed as a clinical necessity.

Keywords: Psilocybin, Mania, Bipolar Disorder, 5-HT2A Receptor, Default Mode Network (DMN)

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0207 - The Relationship of Intolerance of Uncertainty, Psychological Flexibility, Repetitive Thinking, and Self-Criticism with Executive Functions in Individuals Diagnosed with Attention Deficit Hyperactivity Disorder

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Objective

Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by developmentally inappropriate levels of inattention, hyperactivity, and impulsivity that impair functioning(l). A large-scale study published in 2023, including 21 million adults, reported a prevalence of 3.10% in adults(2).

Intolerance of uncertainty (IU) is a transdiagnostic construct referring to the tendency to perceive uncertain, unpredictable, or uncontrollable situations as threatening and distressing(3). IU has been associated with multiple psychiatric disorders and externalizing symptoms(4-7). However, research on IU in ADHD populations is limited. Studies suggest that individuals with ADHD show higher IU than healthy controls(8,9).

Another relevant construct is psychological flexibility, which refers to the ability to remain in contact with the present moment and to adapt behavior in accordance with personal values(10). Although direct comparisons in ADHD populations are lacking, intervention-based studies have assessed changes in psychological flexibility(11)

Similarly, perseverative thinking, particularly in the form of Repetitive Negative Thinking (RNT), is characterized by repetitive, intrusive, and difficult-to-disengage thought patterns that consume cognitive resources(12). Although well studied in mood and anxiety disorders, it is also linked to ADHD symptoms and executive dysfunction(13,14).

In addition, self-criticism is a maladaptive form of self-relation, characterized by judgmental thoughts about the self, accompanied by intense negative emotions, and linked to multiple psychopathologies(15-21). Although not directly studied in ADHD using self-report measures, indirect evidence suggests increased vulnerability due to negative self-perception, lower self-esteem, and higher exposure to external criticism in individuals with ADHD(22-25).

These transdiagnostic psychological processes may be particularly important given the central role of executive functions in ADHD. Executive functions are higher-order cognitive processes involved in goal-directed behavior, including inhibition, working memory, attention regulation, and planning(26), and are frequently impaired in ADHD(27).

The aim of this study is to examine the relationship between intolerance of uncertainty, psychological flexibility, repetitive thinking, and self-criticism and the severity of ADHD symptoms and executive functions in individuals diagnosed with ADHD. Additionally, it aims to compare these processes between individuals with ADHD and a control group without ADHD. Given the limited research in this area, the findings may help inform clinical assessment and intervention targets. As data collection is ongoing, the present report presents preliminary results from the ADHD group only.

Methods

This study employed a quantitative, cross-sectional design. Although the broader study includes both ADHD and control groups, data collection is ongoing; therefore, the present brief report presents preliminary analyses conducted within the ADHD group only.

Participants in the ADHD group were recruited from the outpatient psychiatry clinic of Goztepe Prof. Dr. Suleyman Yalçın City Hospital. The sample consisted of adults aged 18-65 years who met DSM-5 criteria for ADHD, as confirmed by the Structured Clinical Interview for DSM-5/Clinician Version (SCID-5/CV). Individuals with intellectual disability, autism spectrum disorders, psychotic disorders, bipolar disorder, or substance use disorders were excluded. To minimize the confounding effects of mood and anxiety symptoms, only participants

scoring <7 on the Hamilton Depression Rating Scale and <5 on the Hamilton Anxiety Rating Scale were included. Participants using psychotropic medication were required to be on a stable dose for at least one month prior to participation. All participants completed self-report measures assessing intolerance of uncertainty, psychological flexibility, repetitive thinking, self-criticism, ADHD symptom severity, and executive functioning, including the Intolerance of Uncertainty Scale-Short Form (IUS-12), the Acceptance and Action Questionnaire-II (AAQ-II), the Perseverative Thinking Questionnaire (PTQ), the Forms of Self-Criticizing/Attacking and Self-Reassuring Scale (FSCRS), the Adult ADHD Self-Report Scale (ASRS), and the Adult Executive Functioning Inventory (ADEXI). Neuropsychological tests were administered but are not included in this preliminary report.

Pearson correlation analyses were conducted to examine associations between intolerance of uncertainty, psychological flexibility, repetitive thinking, self-criticism, ADHD symptom severity, and executive function impairments. Multiple linear regression analyses were performed to identify independent predictors of ADHD symptom severity and executive function impairment. ADHD symptom severity (ASRS total score) and executive function impairment (ADEXI total score) were entered as dependent variables in separate models, while IUS-12, AAQ-II, PTQ, and FSCRS total scores were entered as independent variables. Ethical approval for the study was obtained from the Non-Interventional Clinical Research Ethics Committee of Goztepe Prof. Dr. Suleyman Yalfin City Hospital (Decision No:2025/0201).

Results

The following results are based on preliminary analyses conducted within the ADHD group, as data collection is ongoing. The sample consisted of 78 participants diagnosed with ADHD, of whom 59% were women (n = 46). The mean age of the participants was 25.8 years (SD = 8.39).

Correlation analyses revealed that all psychological variables were moderately and positively associated with ADHD symptom severity. Specifically, the ASRS total score showed significant moderate positive correlations with the IUS-12 total score (r = .53), AAQ-II total score (r = .42), PTQ total score (r = .53), and FSCRS total score (r = .40). Similarly, executive function impairment was moderately and positively associated with all psychological variables. The ADEXI total score demonstrated significant moderate positive correlations with the IUS-12 total score (r = .51), AAQ-II total score (r = .47), PTQ total score (r = .53), and FSCRS total score (r = .43). All reported correlations were statistically significant at the $p < .01$ level.

Multiple linear regression analyses were conducted to examine which psychological variables predicted ADHD symptom severity and executive function impairment. In the first model, ASRS total scores were entered as the dependent variable, and the overall model was statistically significant, $F(4,73)=9.79$, $p<.001$, explaining 34.9% of the variance in ADHD symptom severity. Among the predictors, the IUS-12 total score emerged as the only significant independent predictor of ASRS scores ($B=0.44$, $SE=0.16$, $t=2.71$, $p=.008$, $\beta=.32$). The PTQ total score showed a trend-level association ($p=.059$), whereas AAQ-II and FSCRS total scores did not contribute significantly to the model ($p > .05$).

To examine predictors of executive function impairment, ADEXI total scores were entered as the dependent variable in the second model. The regression model was also statistically significant, $F(4,73)=9.73$, $p<.001$, accounting for 34.8% of the variance in executive function impairment. Consistent with the first model, the IUS-12 total score was the only variable that significantly predicted ADEXI scores ($B=0.29$, $SE=0.13$, $t=2.33$, $p=.023$, $\beta=.28$). The contributions of AAQ-II, PTQ, and FSCRS total scores were not statistically significant ($p > .05$).

Discussion

The present findings indicate that intolerance of uncertainty, psychological inflexibility, repetitive thinking, and self-criticism are moderately associated with both ADHD symptom severity and subjective executive function impairments in adults with ADHD. Among these variables, intolerance of uncertainty emerged as the only independent predictor of both ADHD symptom severity and executive dysfunction. These findings suggest that intolerance of uncertainty may play a central role in the cognitive and behavioral difficulties observed in ADHD. From a transdiagnostic standpoint, avoidance and engagement in risky or impulsive behaviors have been linked to intolerance of uncertainty(7, 28). Since executive dysfunction has been linked to both avoidance and impulsivity, and impulsivity and executive deficits are central characteristics of ADHD, these findings point to a possible mechanism through which IU may be linked to executive difficulties in this population(27,29,30). Notably,

intolerance of uncertainty has been shown to interfere with executive functioning in trauma-exposed and non-trauma populations(31); however, no studies to date have directly examined this relationship in individuals with ADHD.

Although psychological inflexibility, repetitive thinking, and self-criticism were not significant predictors in regression models, their significant positive associations with ADHD symptoms and executive dysfunction highlight their potential clinical relevance. These findings align with prior literature in ADHD populations, suggesting that higher psychological flexibility may serve a protective role for executive functioning; however, research in this area remains very limited(32).

The observed associations between perseverative thinking and executive dysfunction are also consistent with previous reports, which have documented positive correlations between ADHD symptom severity and ruminative thinking, as well as links between various forms of repetitive negative thinking (RNT) and impairments in executive functions(15,16). Despite this, studies directly examining these relationships in ADHD are scarce, underscoring the need for further investigation.

Moderate correlations between self-criticism and executive function impairments align with evidence from non-ADHD populations showing that self-criticism is associated with reduced

cognitive flexibility and attentional control(33,34), however, to our knowledge, this is the first study to demonstrate this relationship in an ADHD sample. These processes may still contribute indirectly or interactively to ADHD-related impairments, suggesting that they may represent important targets for future research and psychotherapeutic interventions.

Importantly, the present results are based on preliminary analyses restricted to self-report measures of executive functioning. Future analyses incorporating neuropsychological test data will provide a more comprehensive understanding of the relationships between these variables.

Overall, the findings support the relevance of transdiagnostic psychological processes in understanding ADHD and suggest that targeting intolerance of uncertainty as a particularly salient factor. These results also underscore the importance of dimensional approaches in conceptualizing ADHD beyond core symptom domains. From a clinical perspective, while intolerance of uncertainty appears to be especially important, other related psychological processes examined in this study should also be considered during patient follow-up and treatment planning. Addressing these processes through psychotherapeutic interventions may contribute to reductions in symptom severity as well as improvements in executive functioning in individuals with ADHD.

Keywords: Attention Deficit Disorder with Hyperactivity, Intolerance of Uncertainty, Repetitive Thinking, Self-Criticism, Executive Function

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0208 – Determining Levels of Anxiety, Depression, and Prenatal Attachment in Patients in The Prenatal Period Admitted to Family Health Centers

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1. Introduction and Objective

Depression is one of the most common psychiatric disorders and leads to significant functional impairment. The literature indicates that 13% of women experience at least one depressive episode during their lifetime. Depression during pregnancy is often not easily recognized and may be overlooked.

Anxiety disorders, like depression, are among the commonly encountered conditions in women; however, their onset typically occurs during the reproductive period or earlier. Therefore, women with anxiety disorders experience pregnancy during the course of their illness.

Attachment is one of the fundamental elements of human development and represents a critical period affecting an individual's social, emotional, and cognitive development. The attachment process begins in the womb and is completed during the early years of life.

Primary healthcare services, due to their accessibility and low cost, play a vital role in protecting and monitoring maternal and fetal health during pregnancy, as well as in the early detection of possible complications.

This study aimed to determine the levels of anxiety and depression—which are frequently observed during pregnancy and are thought to affect prenatal attachment—among pregnant women presenting to a family health center, and to investigate the relationship between these variables.

Materials and Methods

The study was conducted with pregnant women in the prenatal period who were over 18 years of age, applied to a family health center, and consented to participate in the study. Individuals with comorbid diseases, those with prior psychiatric consultations, and those who did not consent to participate were excluded from the study. After obtaining sociodemographic data and informing participants about the study, the Beck Depression Inventory, Beck Anxiety Inventory, and Prenatal Attachment self-report scales were administered.

Results

Three scales were administered to the participants. The mean scores of these scales were found to be as follows: Beck Depression Inventory score 10.61±8.76, Beck Anxiety Inventory score 9.13±9.08, and prenatal attachment mean score 91.78±11.13.

When the data were examined, the proportion of individuals in the mild-moderate-severe groups was found to be 44.9% (n=79) for depression and 44.3% (n=78) for anxiety. These individuals require further evaluation in terms of psychiatric comorbidities.

It was found that Beck Anxiety Inventory scores were correlated with age, and anxiety symptoms increased as the age of the pregnant woman increased. When prenatal attachment scores were evaluated, an inverse correlation with age was observed, indicating that attachment scores decreased as age increased, and this relationship was statistically significant.

Participants' number of children was statistically compared with their scale scores. As the number of children increased, Beck Depression and Anxiety scores increased, while prenatal attachment scores decreased in a statistically significant manner.

It was observed that an increase in Beck Depression Inventory scores led to an increase in Beck Anxiety Inventory scores, and this relationship was statistically significant.

It was also found that there was an inverse relationship between Beck Depression Inventory scores and prenatal attachment scores; as depression scores increased, prenatal attachment scores decreased, and this relationship was statistically significant.

5. **Discussion**

This study is important in emphasizing the necessity of screening individuals who present to primary healthcare services during the prenatal period in terms of depression and anxiety, and in demonstrating that nearly half of pregnant women fall within this risk group. Through such screening, psychiatric conditions arising during the prenatal period and the associated morbidity and mortality may be prevented, and prenatal attachment as well as the mother-infant relationship in the postpartum period may be strengthened.

In the study, Beck Depression and Anxiety Inventories were administered, and it was observed that nearly half of the participants exhibited symptoms according to these scales. It was statistically determined that individuals in this risk group had lower prenatal attachment scores. This highlights the importance of psychiatric screening of pregnant women and the identification of at-risk groups. These rates may increase with screening at the primary care level and referral to specialists. Family physicians play a crucial role at this level.

Considering the cut-off points of the Beck Depression Inventory, it was observed that 44.9% of participants scored above 9 points and exhibited minimal or higher levels of depressive symptoms. Our findings are consistent with studies in the literature that use the Beck Depression Inventory for screening depression during the prenatal period. Nearly half of women in the prenatal period may exhibit some depressive symptoms. Studies conducted by Lanceaster et al. on depression and risk factors during pregnancy indicate that depression is a public health problem during this period. Therefore, these individuals should be invited for further evaluation and, if necessary, referred to specialist physicians. These findings demonstrate the importance and value of depression screening in primary care and contribute to the literature.

According to the cut-off points of the Beck Anxiety Inventory, the proportion of individuals scoring above 8 points and exhibiting anxiety symptoms was found to be 44.3%. It is known that anxiety and depression are often comorbid conditions, and the presence of one may predispose to the other. Overlooking this condition may delay diagnosis. This once again highlights the importance of screening in primary healthcare services.

The relationship between participants' age and scale scores was statistically analyzed. The analysis showed that as age increased, Beck Depression Inventory scores did not change at a statistically significant level. Studies examining the relationship between age and depression during the prenatal period report varying results. This may be due to the small sample size, the relatively young mean age of the participants, and the tendency of higher-risk pregnancies at advanced ages to be managed in secondary or tertiary healthcare services.

When Beck Anxiety Inventory scores were examined, a correlation with age was observed, indicating that anxiety symptoms increased with age during the prenatal period. As maternal age increases, the risk of pregnancy-related complications and the awareness of responsibilities also increase, which may raise the likelihood of exhibiting anxiety symptoms.

When participants' number of children was compared with Beck Depression and Anxiety scale scores, it was observed that depression and anxiety scores increased significantly in those with three or more children. It can be concluded that having three or more children may be considered a risk factor for both depression and anxiety. When the literature is examined, there are numerous studies indicating that an increase in the number of children and births constitutes a risk factor [41,58]. These results may be explained by the physical and psychological burden imposed on women by an increasing number of children, previous negative childbirth and postpartum experiences, and insufficient social support.

When the relationship between prenatal attachment scale scores and the number of children was examined, an inverse relationship was observed. It was found that prenatal attachment scores decreased after two children, and this difference was statistically significant. This finding is consistent with data in the literature. The fact that having a first child or experiencing this process less frequently leads to greater expectations and excitement during pregnancy may explain this difference. From a different perspective, having two or more children and thus having experience may lead individuals to approach events from a less emotional perspective. In addition, having multiple

children, repeated childbirth experiences, being of more advanced age, and awareness of the risks associated with the process due to past experiences may have influenced attachment scores.

The interaction between scale scores was statistically analyzed. It was observed that an increase in Beck Depression Inventory scores led to an increase in Beck Anxiety Inventory scores, and this increase was statistically significant. The literature emphasizes the high probability of comorbidity between these two psychiatric conditions. The findings of our study are consistent with this literature.

It was observed that there was an inverse relationship between increases in Beck Depression and Anxiety scale scores and prenatal attachment scores; as depression and anxiety increased, prenatal attachment decreased, and this relationship was statistically significant. These findings are consistent with studies in the literature.

Prenatal attachment is a process with a high emotional load that increases as the mother anticipates her baby, feels its movements, hears its heartbeat, sees its images during check-ups, and makes plans and dreams about the baby. However, the presence of anxiety and depression, or even the presence of depressive and anxious symptoms that do not reachdiagnostic levels, negatively affects the attachment process. The importance of attachment in human life, as well as its impact on personality development and the formation of healthy generations, is evident. This once again emphasizes the importance of this study.

The most important limitation of our study is that it was conducted within a cross-sectional time frame and in a specific region. This may have affected the incidence of psychiatric disorders that exhibit seasonal variation. Additionally, conducting the study in a limited region reduced the proportion of sociodemographic data available for comparison within the sample. Data were collected through self- report scales completed by the participants, which may have influenced the responses given while filling out the scales.

7. Conclusions and Recommendations

Nearly half of women during pregnancy exhibit at least one or several symptoms of anxiety or depression.

Simply asking women about their condition (by administering a questionnaire) was sufficient for them to express it. This indicates how often psychiatric symptoms are overlooked, remain unexpressed unless asked, and reflects help-seeking behavior.

Our sample consists of a population with a relatively younger mean age compared to the literature, generally of middle income level, and with a lower rate of employment. All of these factors constitute risk factors for depression and anxiety. This further emphasizes the importance of screening for depression and anxiety during pregnancy at the societal level.

High anxiety and depression scores during pregnancy negatively affect prenatal attachment. In psychiatric approaches, many problems are emphasized to be associated with insecure attachment. Therefore, ensuring that pregnant women receive adequate psychiatric support is essential not only for women's health but also for infant health and public health.

In primary healthcare services, which are the most easily accessible and free of charge, it is extremely important—both for maternal and infant health and for the development of strong future generations— to screen women in the prenatal period not only for physical illnesses but also from a psychiatric perspective, to provide information regarding their anxieties and negative thoughts, and to refer them to specialist physicians when necessary.

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0210 - Adaptation and Psychometric Validation of the Child and Adolescent Forms of the Glasgow Antipsychotic Side-Effect Scale

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Objectıve

Antipsychotic medications are widely used in children and adolescents for the treatment of various psychiatric disorders including primarily psychotic disorders, bipolar disorder, conduct disorder, Tourette syndrome and autism spectrum disorder. However, both short and long term use of antipsychotics may be associated with a broad range of adverse effects including extrapyramidal symptoms (EPS), sedation, weight gain, metabolic disturbances, endocrine changes, gastrointestinal complaints and increased cardiovascular risk (1, 2). Despite the increasing use of antipsychotics in children and adolescents, evidence regarding which adverse effects predominate across specific developmental stages remains limited (3). Furthermore, to date, there is no validated and easily applicable antipsychotic specific side- effect screening instrument available for children and adolescents in our country.

The Glasgow Antipsychotic Side-Effect Scale (GASS) developed by Waddell and Taylor, is a brief self-report instrument designed to assess antipsychotic related adverse effects (4). The Turkish validity and reliability of the adult version have previously been established (5). In this context, the present study aimed to adapt the child (6-12 years) and adolescent (13-17 years) forms of the GASS into Turkish and to evaluate their validity and reliability.

Methods

This single-centre, cross-sectional study was conducted between 2 June 2025 and 1 September 2025 at the Child and Adolescent Psychiatry Outpatient Clinic of Istanbul Medeniyet University, Goztepe Prof. Dr. Suleyman Yalcin City Hospital. The study sample

consisted of parents of children aged 6-12 years and adolescents aged 13-17 years who met the inclusion and exclusion criteria and had been receiving antipsychotic treatment for at least three months. Most items of the scale assess adverse effects experienced during the preceding week.

Sample size was determined in accordance with recommendations from the measurement and factor analysis literature, targeting at least 5-10 participants per item. Psychiatric diagnoses were assessed using the semi-structured Schedule for Affective Disorders and Schizophrenia for School-Aged Children - Present and Lifetime Version (K-SADS-PL-DSM-5). Questionnaires for children were completed with parental assistance whereas adolescent forms were completed jointly by adolescents and their parents.

To enhance data quality, attention-check items were embedded within the questionnaires to identify inattentive responding. Participants who failed these items or provided incomplete responses were excluded. After excluding 16 participants, the final sample consisted of 264 participants.

Ethical approval was obtained from the Istanbul Medeniyet University Ethics Committee (Approval No: 2025/09-32). Data were analysed using IBM SPSS version 23, AMOS version 24, and JASP version 0.19.3.0. Exploratory and confirmatory factor analyses were conducted, and statistical significance was set at $p < 0.05$.

Results

The final sample comprised of 264 participants including 132 children (CF) and 132 adolescents (AF). The mean age was 9.42 years in the CF group and 15.58 years in the AF group, with an overall mean age of 12.5 years. ADHD was the most common diagnosis (60.2%) followed by depressive disorders (17.8%), ASD (11.7%) and

generalized anxiety disorder (6.4%). Atypical antipsychotics were the most frequently prescribed medications in both groups.

Exploratory factor analysis (EFA) for AF was conducted using the principal components analysis with Varimax rotation. According to established criteria, diagonal values in the anti- image correlation matrix exceeded 0.50 and factor retention required at least three items per factor with no cross-loading. Based on these criteria, items Glasgow 3,4,6,7,10,15,18,19,21, and 22 were removed. The final model consisted of 12 items with a four-factor structure. The

KMO measure was 0.754 and Bartlett's test of sphericity was significant ($\chi2=456.807$, $p < 0.001$), explaining 64.74% of the total variance. CFA demonstrated acceptable model fit (CMIN/DF=2.073, GFI=0.895, CFI=0.876, RMSEA=0.091, SRMR=0.074). Cronbach's α ranged from 0.587 to 0.748. All standardized path coefficients were statistically significant ($p<0.05$) (Tables 1-2).

For the CF, EFA using principal components analysis with Varimax rotation resulted in the removal of items Glasgow 4, 11, 16, and 18. The final model consisted of 14 items with a four-factor structure. The KMO measure was 0.724 and Bartlett's test was significant ($\chi2=526.042$, $p < 0.001$), explaining 59.05% of the variance. Confirmatory factor analysis using WLSMV indicated good model fit (CFI=1.000, RMSEA=0.000). Cronbach's α values ranged from 0.551 to 0.671. Tukey's test indicated that the use of a total score was not supported ($F=121.256$, $p<0.001$). Weight gain and sedation were the most frequently reported discomfort items in both forms. (Tables 3-4)

Discussion

In the present study we developed and evaluated the Turkish versions of the GASS for both children (CF, ages 6-12) and adolescents (AF, ages 13-17). The findings demonstrated that both forms yielded a four-factor structure that was clinically interpretable. Importantly, our analyses did not support the use of a single total score; instead, a subscale-based interpretation appeared to better reflect the multidimensional nature of antipsychotic-related adverse effects. Given the high rate of atypical antipsychotic use within our sample, it is also likely that the observed factor structure largely reflects the side effect profile associated with second generation agents.

Our results further suggest that the pattern of reported side effects varies depending on both developmental stage and the source of information. In both adolescent self-reports and parent- proxy reports, weight gain and sedation were the most frequently reported symptoms likely because they are more observable in daily life. In contrast, extrapyramidal, autonomic and more subjective complaints were less frequently reported. This pattern indicates that side effect assessment in pediatric populations is shaped not only by the presence of symptoms but also by how noticeable they are, how they are interpreted and who reports them. In this context, relying on a single informant may overlook clinically relevant experiences.

Although antipsychotic-related adverse effects in children and adolescents are heterogeneous, they remain poorly studied and are often underreported in research, leading to underrecognition in clinical practice (6). Moreover in youths with neurodevelopmental conditions such as ADHD, ASD and intellectual disability, adverse effects particularly EPSs may be obscured by complex behavioral features; therefore the high prevalence of these conditions in our sample may likewise have hindered their identification and distinction from behavioral manifestations (7).

The EFAs indicated that the factor structures of both the CF and AF differed psychometrically from the system based grouping of side effects in the original adult scale. Developmental differences in pharmacokinetics, pharmacodynamics, and physiology as well as differences in how symptoms are perceived and reported, likely contribute to this discrepancy.

Consistent with the multidimensional structure of the scale, internal consistency was evaluated at the subscale level. A four-factor solution was confirmed for both forms and the results of the Tukey test of additivity indicated that combining all items into a single total score was not appropriate. For the AF, Cronbach's alpha values ranged from 0.587 to 0.748, while for the CF they ranged from 0.551 to 0.671. These values fall within an acceptable range particularly given the heterogeneous nature of pediatric populations and the broad range of clinical domains covered by the scale.

Importantly, although internal consistency was not reported in the original GASS study, subsequent validations including the Arabic version and later adaptations have demonstrated acceptable to good reliability, indicating that translation and adaptation do not adversely affect the scale's consistency.

The construct validity of both forms was supported by the EFA findings, which consistently identified a four-factor structure. Although some previous adaptations of the original scale have reported a higher number of factors, our results suggest that a reduced-item Turkish version comprising 14 items for the CF and 12 items for the AF is sufficient. This more concise structure may also improve feasibility in routine clinical practice.

Adverse effects in children and adolescents may be underreported or overlooked due to normalization, privacy concerns, or limited awareness. Internal symptoms (e.g. blurred vision, palpitations) are particularly difficult to detect without self-report, while observable effects

such as enuresis may not be linked to medication use by caregivers. Moreover, the short assessment window may hinder recognition of transient changes such as weight variation. Notably, up to 55% of pediatric adverse drug reactions may go unreported, highlighting substantial gaps in detection (8).

Consistent with this, several studies indicate that such symptoms may be associated with antipsychotic use yet remain underrecognized in clinical practice.

This study addresses an important gap by providing a developmentally sensitive, informant- specific tool for assessing antipsychotic-related side effects in pediatric populations.

Keywords: Antipsychotic, child, adolescent, side effect, validity, reliability, scale adaptation

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Table 1. EFA results according to the adolescent form.

	Factor 1	Factor 2	Factor 3	Factor 4	Extraction	Anti-image correlation coefficient
Glasgow 11	0,782				0,674	0,783
Glasgow 12	0,784				0,699	0,701
Glasgow 17	0,739				0,631	0,852
Glasgow 1		0,813			0,702	0,750
Glasgow 2		0,831			0,795	0,721
Glasgow 9		0,623			0,660	0,812
Glasgow 5			0,692		0,470	0,732
Glasgow 14			0,701		0,587	0,761
Glasgow 20			0,621		0,741	0,650
Glasgow 8				0,731	0,501	0,728
Glasgow 13				0,586	0,618	0,774
Glasgow 16				0,817	0,691	0,716
Eigenvalue VEO CVER	2,145 17,875 17,875	2,060 17,165 35,039	1,784 14,870 49,910	1,780 14,830 64,740		

VEO: Variance explained ratio, CVER: Cumulative variance explained ratio, K-M-O=0.754; Bartlett's Test (χ^2= 456.807; p<0.001)

Table 2. CFA Results According to the Adolescent Form

			β^1 (%95 GA)	**β^2 (%95 CI)**	**S. error**	**Test Statistics**	**P**	**R^2**
Glasgow 11	<--	F 1	0,693 (0,499 - 0,871)	0,54 (0,306 - 0,845)	0,082	6,598	**<0,00 1**	0,48 1
Glasgow 12	<--	F 1	0,639 (0,403 - 0,819)	0,639 (0,365 - 0,925)	0,103	6,234	**<0,00 1**	0,40 8
Glasgow 17	<--	F 1	0,79 (0,61 - 0,953)	1 (1 - 1)	---	---	---	0,62 3
Glasgow 1	<--	F 2	0,664 (0,384 - 0,788)	1,515 (0,604 - 2,424)	0,245	6,178	**<0,00 1**	0,44 1
Glasgow 2	<--	F 2	0,896 (0,665 - 1,04)	1,79 (0,906 - 3,023)	0,275	6,508	**<0,00 1**	0,80 3
Glasgow 9	<--	F 2	0,628 (0,459 - 0,892)	1 (1 - 1)	---	---	---	0,39 5
Glasgow 5	<--	F 3	0,642 (0,344 - 0,876)	1,744 (0,763 - 5,054)	0,376	4,635	**<0,00 1**	0,41 2
Glasgow 14	<--	F 3	0,636 (0,352 - 0,919)	1,29 (0,559 - 4,121)	0,279	4,617	**<0,00 1**	0,40 4
Glasgow 20	<--	F 3	0,561 (0,258 - 0,826)	1 (1 - 1)	---	---	---	0,31 5
Glasgow 8	<--	F 4	0,546 (0,163 - 0,855)	1,482 (0,392 - 5,478)	0,383	3,872	**<0,00 1**	0,29 8
Glasgow 13	<--	F 4	0,707 (0,362 - 1,059)	1,317 (0,689 - 4,591)	0,320	4,119	**<0,00 1**	0,50 0
Glasgow 16	<--	F 4	0,505 (0,158 - 0,821)	1 (1 - 1)	---	---	---	0,25 5

β^1: Standardized beta coefficient, β^2: Unstandardized beta coefficient CI: Confidence Interval

Table 3. EFA results according to the child form

	Factor 1	Factor 2	Factor 3	Factor 4	Extraction	Anti-Image correlation coefficient
Glasgow 6	0,855				0,784	0,701
Glasgow 13	0,865				0,782	0,681
Glasgow 14	0,568				0,447	0,781
Glasgow 7		0,731			0,715	0,754
Glasgow 8		0,645			0,544	0,798
Glasgow 12		0,795			0,693	0,759
Glasgow 17		0,549			0,487	0,768
Glasgow 3			0,707		0,58	0,698
Glasgow 5			0,698		0,632	0,793
Glasgow 10			0,647		0,506	0,832
Glasgow 15			0,538		0,403	0,736
Glasgow 1				0,804	0,689	0,649
Glasgow 2				0,795	0,645	0,705
Glasgow 9				0,451	0,361	0,870
Eigenvalue	2,332	2,116	2,082	1,738		
VEO	16,655	15,113	14,874	12,416		
CVER	16,655	31,768	46,642	59,058		

VEO: Variance explained ratio, CVEO: Cumulative variance explained ratio, K-M-O=0,745;
Bartlett's Test (χ^2 = 526,042; p<0,001)

Table 4. DFA results according to the child form

Factor	Item	β^1	95% Confidence Interval Lower Limit	Upper Limit	S. Error	P	R^2
F1	Glasgow 6	0,964	0,813	1,115	0,077	**<0,001**	0,928
	Glasgow 13	0,988	0,758	1,217	0,117	**<0,001**	0,976
	Glasgow 14	0,769	0,564	0,973	0,104	**<0,001**	0,591
F2	Glasgow 7	0,901	0,718	1,084	0,093	**<0,001**	0,812
	Glasgow 8	0,803	0,611	0,995	0,098	**<0,001**	0,644
	Glasgow 12	0,793	0,649	0,938	0,074	**<0,001**	0,629
	Glasgow 17	0,715	0,553	0,877	0,083	**<0,001**	0,511
F3	Glasgow 3	0,800	0,600	1,001	0,102	**<0,001**	0,640
	Glasgow 5	0,893	0,626	1,161	0,136	**<0,001**	0,798
	Glasgow 10	0,862	0,655	1,068	0,105	**<0,001**	0,742
	Glasgow 15	0,726	0,537	0,916	0,097	**<0,001**	0,528
F4	Glasgow 1	0,730	0,480	0,980	0,127	**<0,001**	0,533
	Glasgow 2	0,734	0,482	0,985	0,128	**<0,001**	0,538
	Glasgow 9	0,750	0,539	0,962	0,108	**<0,001**	0,563

β^1: Standardized beta coefficient S: Standart

0213 - Serum and Urinary Metabolomic Alterations in Bipolar Disorder: An Exploratory UHPLC-MS Study

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Objective

Bipolar disorder (BD) is a chronic and recurrent psychiatric disorder characterized by alternating episodes of mania, hypomania, and depression. The disorder is associated with significant morbidity, functional impairment, and increased mortality. Despite advances in psychiatric research, the diagnosis of BD still relies primarily on clinical interviews and symptom-based diagnostic criteria, which may lead to delayed diagnosis and inappropriate treatment in many patients.[1] The lack of objective biological markers further complicates early detection and monitoring of the disorder.

Recent advances in metabolomics have provided new opportunities to explore biochemical alterations associated with psychiatric illnesses. Metabolomics refers to the comprehensive analysis of small molecules and metabolic intermediates within biological systems.[1] Because metabolic profiles reflect the integrated effects of genetic, environmental, and physiological factors, metabolomic approaches may help identify disease-specific biomarkers and reveal pathophysiological mechanisms underlying complex psychiatric disorders.

In recent years, mass spectrometry-based metabolomics has emerged as a powerful tool for identifying metabolic alterations associated with neuropsychiatric disorders. Ultra high- performance liquid chromatography-mass spectrometry enables the detection and semiquantitative analysis of numerous metabolites simultaneously, providing insight into altered biochemical pathways. However, metabolomic studies investigating BD remain relatively limited, and the results across different populations have shown considerable variability. Previous studies have suggested that metabolic alterations in BD may involve lipid metabolism, amino acid metabolism, oxidative stress pathways, and energy metabolism.[2] Nevertheless, the available findings have not yet resulted in clinically applicable biomarkers.

Another important challenge in BD research is understanding the biological differences between different mood states, particularly manic and euthymic phases.[1] Identifying metabolic signatures that differentiate these states may help improve diagnostic accuracy and enable the prediction of mood episode transitions. Such biomarkers could also contribute to personalized treatment strategies and early interventions.

The present study aimed to investigate the metabolomic profiles of patients with BD during manic and euthymic states using serum and urine samples and to compare these profiles with those of healthy controls. Specifically, the study sought to identify metabolites that may distinguish BD patients from healthy individuals and to explore potential metabolic markers associated with different mood states. In addition, the study aimed to identify metabolic

pathways that may be implicated in the pathophysiology of BD and contribute to the development of novel diagnostic and therapeutic approaches.

Methods

Study Design and Participants

This study was conducted as a metabolomic profiling investigation involving patients diagnosed with bipolar disorder and healthy control participants. The study protocol was approved by the Ethics Committee of the University of Health Sciences Hamidiye Scientific Research Ethics Board.

Participants aged between 18 and 65 years who presented to the Psychiatry Clinic of Bagcilar Training and Research Hospital and were diagnosed with Bipolar Disorder Type I according to DSM-5 diagnostic criteria were eligible for inclusion in the study. Clinical assessments were conducted by experienced psychiatrists.

To classify the clinical status of the patients, standardized psychiatric rating scales were used. The Hamilton Depression Rating Scale was applied to evaluate depressive symptoms, and the Young Mania Rating Scale was used to assess manic symptom severity. Based on clinical evaluation and scale scores, patients were categorized into manic and euthymic groups.

Sample Collection

Biological samples were collected from both patient groups and healthy control participants. Serum samples were obtained from 33 patients experiencing a manic episode, 25 patients in a euthymic state, 40 healthy control participants. Urine samples were obtained from 22 patients during manic episodes, 31 healthy control participants .All biological samples were collected under standardized conditions and stored appropriately until analysis.

Metabolomic Analysis

Metabolomic analyses were performed using ultra high-performance liquid chromatography coupled with mass spectrometry (UHPLC). This technique allows highly sensitive detection and semi-quantitative measurement of a wide range of metabolites within biological samples. UHPLC is widely used in metabolomic research due to its high sensitivity, broad analytical range, and ability to detect metabolites present at very low concentrations.

The collected serum and urine samples underwent chromatographic separation followed by mass spectrometric detection. Detected metabolites were identified by comparing their mass- to-charge ratios and fragmentation patterns with known metabolite databases.

In addition to metabolite identification, pathway analysis and statistical comparisons between groups were performed to determine metabolites that differed significantly between bipolar disorder patients and healthy controls.

Statistical Analysis

Statistical analyses were conducted to evaluate differences in metabolite levels among the groups. Comparisons were performed between: manic patients and healthy controls, euthymic patients and healthy controls, manic and euthymic patients. Metabolites showing statistically significant differences were further analyzed to determine their potential roles in metabolic pathways related to bipolar disorder. Enrichment analyses were also performed to identify metabolic pathways potentially involved in the pathogenesis of BD.

Results

Sociodemographic Characteristics

The study included a total of 98 participants whose serum samples were analyzed: 40 healthy controls, 25 euthymic patients, and 33 manic patients.

Serum Metabolomic Findings

Metabolomic analyses of serum samples revealed several metabolites that differed significantly between bipolar disorder patients and healthy controls. Comparisons between patients with BD and healthy controls revealed alterations primarily in lipid-related metabolites, including primary fatty acid amides, phosphatidylcholine derivatives, and acylcarnitines. Following adjustment, oleamide and 2-arachidonoylglycerol were found to be significantly associated with the euthymic state relative to the control group. In manic patients, several lipid metabolites exhibited consistent directional changes compared to the control group, such as Decanoyl-L- carnitine (C10:0 acylcarnitine), 1,2-Dilinoleoyl-sn-glycero-3-phosphocholine, and 1,2-dioleoyl- sn-glycero-3-phosphatidylcholine. Direct comparison between the manic and euthymic states identified a limited number of metabolites, suggesting metabolic differences related to the state, such as C10:1 acylcarnitine. These results suggest potential disruptions in phospholipid metabolism in manic episodes and in phenylalanine metabolism, retrograde endocannabinoid signalling, and thermogenesis in euthymic states.

Table 1 Confounder-adjusted logistic regression results for the healthy controls versus euthymic group

Metabolite	m/z	Adjusted OR	95% CI	ρ- value	q- value
2-Arachidonoylglycerol	379.2813	4.31	1.58-11.75	**0.004**	0.046
Oleamide	282.2787	3.15	1.39-7.16	**0.006**	0.046
1 -pentadecanoyl-2-hydroxy-sn- glycero-3-phosphocholine	482.3235	1.89	0.86-4.16	0.113	0.328
Palmitamide	256.263	1.67	0.86-3.24	0.129	0.328
1 -Palmitoyl-2-hydroxy-sn- glycero-3-phosphoethanolamine	454.2922	1.66	0.81-3.41	0.165	0.358
1 -Stearoyl-2-linoleoyl-sn- glycero-3-phosphocholine	808.5831	1.53	0.81-2.91	0.192	0.358
trans-3'-Hydroxycotinine	193.0971	0.40	0.11-1.53	0.183	0.358
Stearamide	284.2944	1.51	0.79-2.87	0.213	0.372
1-Hexadecyl-2-(5Z,8Z,11Z,14Z-eicosatetraenoyl)-sn-glycero-3- phosphocholine	768.5892	1.33	0.69-2.57	0.395	0.614
Phytanic acid	359.3149	0.76	0.41-1.42	0.394	0.614
Linoleoylglycine	338.266	1.25	0.68-2.29	0.469	0.656
Alpha-N-Phenylacetyl-L- glutamine	265.118	0.81	0.42-1.59	0.547	0.696
Hippuric acid	180.0654	0.86	0.46-1.60	0.628	0.703
3-Indoleacetic acid	176.0705	1.00	0.48-2.06	0.995	0.995
Isostearic acid	331.2837	0.99	0.54-1.81	0.968	0.995

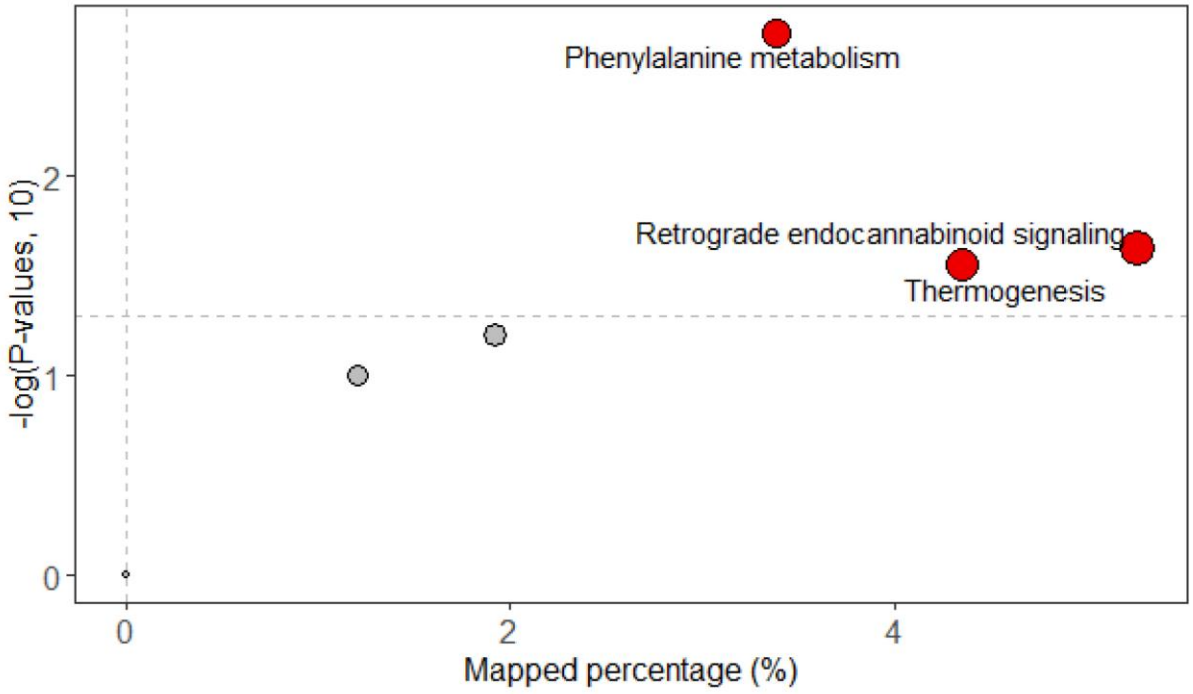
Table 2. Confounder-adjusted logistic regression results for healthy controls versus manic patients

Metabolite	m/z	Adjusted OR	95% CI	ρ- value	q- value
Decanoyl-L-carnitine (C10:0 acylcarnitine)	316.2481	0.31	0.12-0.77	**0.012**	0.076
1,2-Dilinoleoyl-sn-glycero-3- phosphocholine	782.568	2.75	1.16-6.50	**0.022**	0.076
1,2-dioleoyl-sn-glycero-3- phosphatidylcholine	786.5995	2.47	1.04-5.87	**0.041**	0.095
Oleamide	282.2787	1.94	0.98-3.86	0.058	0.102
Phosphocholine	184.0731	1.72	0.89-3.32	0.105	0.136
Palmitoyl sphingomyelin	703.5738	2.18	0.82-5.75	0.117	0.136
H-Pro-Hyp-OH	229.118	1.04	0.57-1.88	0.898	0.898

Table 3. Confounder-adjusted logistic regression results for the Manic versus Euthymic groups

Metabolite	mz	Adjusted OR	95% CI	p-value	q-value
C10:1 acylcarnitine	314.2322	3.03	1.37-6.69	**0.006**	0.015
Decanoyl-L-carnitine (C10:0 acylcarnitine)	316.248	1.73	0.93-3.21	0.083	0.112
Phe-Phe	313.154	1.78	0.91-3.49	0.093	0.112

Figure 1 Pathway Impact Analysis for Control and Euthymic Groups



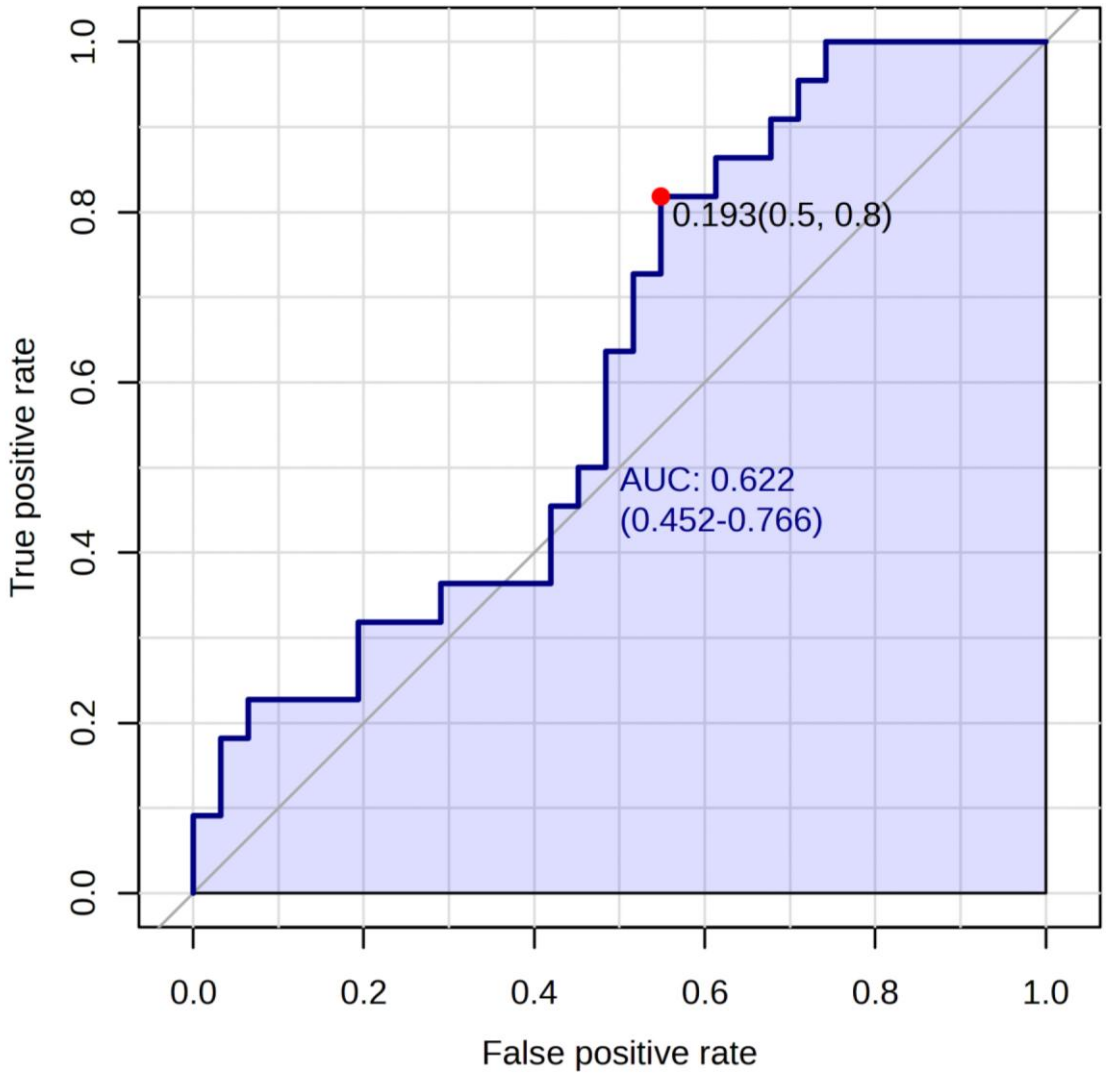
Unline Metabolomic Findings

Urine metabolomic analysis also revealed significant differences between manic BD patients and healthy controls. Several lipid-related metabolites were found to be increased in the patient group. The metabolites showing increased levels in manic patients included, 1,2-dioleoyl-sn- glycero-3-phosphatidylcholine, and 1 -stearoyl-2-linoleoyl-sn-glycero-3-phosphocholine.

Table 4. Comparison of Urinary Metabolites Between Control versus Manic Groups

Compound Name	Adduct	Mz Match Score	Total Score	Fold Change	p value
Palmitamide	(M+H)+	0.996646	0.883824	1.02	0.307
Oleamide	(M+H)+	0.999457	0.842868	1.08	0.698
1,2-dioleoyl-sn- glycero-3-phosphatidylcholine	(M+H)+	0.998371	0.812094	0.70	**0.048**
1-Stearoyl-2-linoleoyl-sn-glycero-3-phosphocholine	(M+H)+	0.998371	0.805729	0.70	**0.048**

Figure 2 ROC analysis and Area Under Curve (AUC) result of 1,2-dioleoyl-sn-glycero-3-phosphatidylcholine and 1 -Stearoyl-2-linoleoyl-sn-glycero-3-phosphocholine



These findings indicate that alterations in lipid metabolism may be detectable not only in serum but also in urine samples, suggesting systemic metabolic dysregulation in bipolar disorder.

Pathway Analysis

Pathway enrichment analyses suggested that several metabolic pathways may be involved in BD pathophysiology. These pathways included lipid metabolism, fatty acid metabolism, and phospholipid metabolism. Disruptions in these pathways may influence neuronal membrane structure, neurotransmitter release, and cellular signaling mechanisms.

Discussion

The present study investigated metabolomic alterations in bipolar disorder by comparing serum and urine metabolite profiles of manic and euthymic patients with those of healthy controls. The findings revealed significant metabolic differences between BD patients and healthy individuals, particularly in metabolites associated with lipid metabolism and phospholipid pathways.

One of the most notable findings was the reduction in primary fatty acid amides such as oleamide, and 2-arachidonoylglycerol in BD patients. Oleamide, in particular, has been linked to sleep regulation, neurochemical

signaling, and endocannabinoid system activity.[3] Reduced levels of oleamide may therefore reflect dysregulation of sleep-wake cycles and neural signaling processes that are frequently disturbed in bipolar disorder.

Another important finding involved alterations in phosphatidylcholine derivatives. Phosphatidylcholines are major components of cell membranes and play critical roles in membrane fluidity, synaptic function, and neurotransmitter release.[4] Changes in phospholipid metabolism may therefore influence neuronal communication and brain function in individuals with BD.

The observed increase in decanoyl-L-carnitine during manic episodes suggests that energy metabolism may also be altered during acute mood episodes. Carnitine derivatives are involved in mitochondrial fatty acid transport and energy production.[5] Elevated levels may reflect changes in mitochondrial activity or energy metabolism during manic states.

Urine metabolomic findings were not consistent with serum results, indicating increased levels of several lipid-related metabolites in manic patients. This inconsistency observed among biological matrices strengthens the likelihood that the metabolic changes in question are a local rather than a systemic phenomenon.

The results also highlight the potential of metabolomics as a promising tool for biomarker discovery in psychiatric disorders. Identifying metabolic signatures associated with BD may contribute to improved diagnostic accuracy and may eventually facilitate the development of objective laboratory-based diagnostic tests.

Despite these promising findings, several limitations should be considered. First, the sample size was relatively limited, which may affect the generalizability of the results. Second, metabolic profiles may be influenced by medication use, dietary habits, and other environmental factors. Third, the cross-sectional design of the study limits the ability to establish causal relationships between metabolic alterations and mood states. Future research should involve larger, multicenter studies and longitudinal designs to better understand the temporal dynamics of metabolic changes in bipolar disorder. Additionally, integrating metabolomic data with genetic, proteomic, and clinical data may help develop comprehensive models of BD pathophysiology.

In conclusion, this study identified significant metabolic alterations in bipolar disorder patients, particularly in lipid-related metabolites and phospholipid pathways. These findings contribute to the growing body of evidence suggesting that metabolic dysregulation plays an important role in BD pathophysiology. Further studies are needed to validate these candidate biomarkers and explore their potential clinical applications in diagnosis, prognosis, and treatment monitoring.

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0214 - Investigation of the Relationship Between Interoception and Eating Behaviors in Children and Adolescents With Attention-Deficit/Hyperactivity Disorder

Abstract

Background: Attention-deflcit/hyperactivity disorder (ADHD) is increasingly conceptualized as a disorder of self-regulation involving behavioral, emotional, and physiological domains (1,2). Although interoception has been proposed as a transdiagnostic mechanism linking these domains, evidence in pediatric ADHD remains limited (3,4).

Objective: The aim of this study was to evaluate interoceptive awareness and interoceptive accuracy in individuals diagnosed with ADHD and, in addition, to examine the reciprocal interactions and interrelationships of these constructs with eating behaviors, chronotype, alexithymia, and impulsivity within a holistic framework.

Methods: A total of 212 participants aged 11-18 years with a diagnosis of ADHD were included in the study. Participants with psychiatric comorbidity (except oppositional defiant disorder), chronic illness, psychotropic medication use, or psychostimulant use within the previous 48 hours were excluded. Standardized scales and the Heartbeat Counting Task were used in the assessment.

Results: Interoceptive awareness was found to be significantly associated with ADHD severity, impulsivity, alexithymia, and eating behaviors. Interoceptive attention regulation partially mediated the relationship between impulsivity and restrained eating, whereas alexithymia mediated the relationship between ADHD severity and external eating. Interoceptive accuracy, in contrast, showed only limited associations.

Conclusion: Interoceptive awareness appears to be a central mechanism in ADHD-related dysregulation and may represent a clinically meaningful target for intervention.

Introduction

Attention-deficit/hyperactivity disorder (ADHD) is a common neurodevelopmental disorder characterized by persistent patterns of inattention, hyperactivity, and impulsivity that impair functioning (5). Epidemiological studies indicate that the worldwide prevalence of ADHD in children and adolescents is approximately 5.29% (6).

Theoretical frameworks increasingly conceptualize ADHD as a disorder of self-regulation involving cognitive, emotional, and physiological systems (1,2). This broader perspective is supported by evidence showing that individuals with ADHD frequently exhibit emotional dysregulation, altered reward processing, and deficits in adaptive behavioral control (2,7). These impairments are not limited to the core symptoms of ADHD but extend to different domains of functioning (8).

In particular, disturbances in eating behaviors and changes in sleep-circadian rhythm patterns have been reported as clinically relevant associated features in children and adolescents with ADHD (9-11). Increased emotional eating, eating in response to external food cues, and

disordered eating patterns have been reported in children and adolescents with ADHD (12,13). These behaviors are thought to be related not only to impulsivity and deficits in inhibitory control but also to differences in reward processing (14). In addition, ADHD has been associated with evening chronotype and delayed circadian phase, suggesting possible dysregulation in biological timing systems in ADHD (10,15).

Taken together, these findings indicate that ADHD involves impairments in both behavioral and physiological regulatory processes. Interoception, defined as the perception, interpretation, and integration of internal bodily signals, has emerged as a key construct linking physiological states with emotional and behavioral processes (3). Interoceptive processing plays a critical role in maintaining homeostasis, guiding adaptive behavior, and supporting emotional awareness (16). Contemporary models conceptualize interoception as a multidimensional construct comprising interoceptive accuracy, sensibility, and awareness (17).

Interoceptive dysfunction has been implicated in many psychiatric disorders characterized by emotional dysregulation, particularly anxiety disorders, depression, and eating disorders (18-20). In these conditions, impairments in the perception and interpretation of bodily signals are thought to contribute to maladaptive emotional responses and behaviors (21). In ADHD, however, interoception has only recently begun to receive attention. Existing studies suggest that individuals with ADHD may have impairments in interoceptive awareness, although findings regarding interoceptive accuracy remain inconsistent (4).

Importantly, interoception may provide a holistic framework linking impulsivity, emotional dysregulation, maladaptive eating behaviors, and circadian rhythm disturbances. Alexithymia and impulsivity are closely related constructs that may interact with interoception. Alexithymia is characterized by difficulty identifying and describing emotions and is thought to reflect impairment in the integration of interoceptive signals into conscious awareness (22,23). Impulsivity, one of the core features of ADHD, has been associated with maladaptive behaviors, including disordered eating and poor emotion regulation (24,25).

Despite these theoretical links, very few studies in pediatric ADHD samples have examined interoception, eating behaviors, chronotype, alexithymia, and impulsivity simultaneously. In addition, the distinction between subjective interoceptive awareness and objective interoceptive accuracy has not been sufficiently investigated (4,26,27).

This study aimed to address this gap within a holistic framework. Accordingly, the relationships among interoceptive awareness, interoceptive accuracy, ADHD symptom severity, eating behaviors, chronotype, alexithymia, and impulsivity were examined, and potential mediation pathways among these variables were also explored.

Methods

This study was designed as a cross-sectional clinical investigation conducted in a tertiary child and adolescent psychiatry outpatient clinic. Ethical approval was obtained, and written informed consent was obtained from both participants and their parents.

The initial sample consisted of 230 children and adolescents aged 11-18 years who presented with suspected ADHD. Diagnosis was confirmed using a DSM-5-based semi-structured clinical interview. After the inclusion and exclusion criteria were applied, 212 participants were included in the final sample.

The inclusion criteria were as follows: being between 11 and 18 years of age, meeting DSM-5 criteria for ADHD, having sufficient cognitive capacity to complete the assessments, absence of intellectual disability, absence of chronic medical illness, absence of sensory impairment, no psychotropic medication use, and no psychostimulant use within the previous 48 hours.

The exclusion criteria included psychiatric comorbidity (except oppositional defiant disorder), chronic illness, developmental delay, and medication use.

The assessment instruments included SNAP-IV, the Childhood Chronotype Questionnaire, the Dutch Eating Behavior Questionnaire, the Multidimensional Assessment of Interoceptive Awareness in Youth (MAIA-Y), the Barratt Impulsiveness Scale-Short Form, the Toronto Alexithymia Scale (TAS-20), and the Heartbeat Counting Task.

Statistical analyses included correlation analyses, hierarchical regression models controlling for covariates, and mediation analyses using the bootstrap method.

Results

A total of 212 children and adolescents diagnosed with ADHD were included in the analyses. Overall, self-reported interoceptive awareness showed stronger and more consistent associations with clinical variables than performance-based cardiac interoceptive accuracy.

With regard to ADHD symptom dimensions, inattention scores were weakly positively associated with the Not-Distracting subscale of interoceptive awareness ($r=0.15$, $p=0.03$). Hyperactivity/impulsivity scores were negatively associated with Noticing ($r=-0.15$, $p=0.03$) and Attention Regulation ($r=-0.17$, $p=0.01$). Total ADHD severity was also negatively correlated with Attention Regulation ($r=-0.17$, $p=0.02$). In hierarchical regression analyses, both

inattention (β=-0.21, p=0.041) and hyperactivity/impulsivity (β=-0.19, p=0.028) significantly and negatively predicted Attention Regulation.

Regarding eating behaviors, restrained eating was positively associated with multiple subdimensions of interoceptive awareness, with the strongest association observed for Attention Regulation (r=0.25, p<0.001). External eating was weakly positively associated only with Not-Worrying (r=0.15, p=0.03). No significant bivariate associations were found between emotional eating and the subdimensions of interoceptive awareness. In contrast, impulsivity was negatively associated with multiple dimensions of interoceptive awareness, particularly Attention Regulation (r=-0.42, p<0.001) and Self-Regulation (r=-0.41, p<0.001).

The associations between alexithymia and interoceptive awareness displayed a selective pattern. Difficulty Identifying Feelings was negatively associated with Attention Regulation (r=-0.14, p=0.04), Self-Regulation (r=-0.24, p<0.001), and Trusting (r=-0.21, p=0.002). Difficulty Describing Feelings was positively associated with Not-Worrying (r=0.22, p=0.001), whereas Externally Oriented Thinking showed positive associations with Noticing,

Not-Distracting, Not-Worrying, and Emotional Awareness (all p<0.05). Total alexithymia score was positively associated with Not-Worrying (r=0.18, p=0.01) and negatively associated with Self-Regulation (r=-0.18, p=0.008).

Cardiac interoceptive accuracy showed only limited associations. Weak positive associations were found between Noticing and accuracy scores at 25 seconds (r=0.17, p=0.02) and 35 seconds (r=0.15, p=0.03), between Attention Regulation and the 100-second accuracy score (r=0.19, p=0.01), and between total accuracy and Noticing (r=0.15, p=0.03). Beyond these, no significant associations were found between cardiac interoceptive accuracy and ADHD symptom severity, eating behaviors, or impulsivity. A weak positive association was observed between total chronotype score and 100-second cardiac interoceptive accuracy (r=0.17, p=0.01).

The relationships between eating behaviors and clinical variables showed a differentiated pattern. External eating was positively associated with inattention (r=0.25, p<0.001), hyperactivity/impulsivity (r=0.19, p=0.007), and total ADHD severity (r=0.25, p<0.001). Impulsivity was negatively associated with restrained eating (r=-0.16, p=0.02) and positively associated with external eating (r=0.23, p=0.001). Emotional eating was positively associated with Difficulty Identifying Feelings (r=0.27, p<0.001), Difficulty Describing Feelings (r=0.15, p=0.03), and total alexithymia (r=0.23, p=0.001). External eating was also positively associated with both alexithymia subdimensions and total alexithymia.

Significant positive associations were also found between ADHD severity and alexithymia. Both inattention and hyperactivity/impulsivity were positively associated with Difficulty Identifying Feelings, Difficulty Describing Feelings, and total alexithymia scores (all p<0.01). Impulsivity was likewise positively associated with Difficulty Identifying Feelings (r=0.41, p<0.001), Difficulty Describing Feelings (r=0.23, p=0.001), and total alexithymia (r=0.36, p<0.001). Total chronotype score was positively associated with inattention (r=0.27, p<0.001), hyperactivity/impulsivity (r=0.20, p=0.005), and total ADHD severity (r=0.26, p<0.001).

Hierarchical regression analyses supported this pattern. Attention Regulation was negatively predicted by both inattention and hyperactivity/impulsivity. Self-Regulation was negatively predicted by impulsivity. Restrained eating was positively predicted by BMI and impulsivity, and negatively predicted by Attention Regulation. External eating was positively predicted by BMI, Difficulty Identifying Feelings, and impulsivity, and negatively predicted by Not-Worrying. Emotional eating was positively predicted by female sex, BMI, impulsivity, and Difficulty Identifying Feelings. Chronotype was positively predicted by age, Trusting, and 100-second cardiac interoceptive accuracy, and negatively predicted by screen use and total ADHD severity. Difficulty Identifying Feelings was positively predicted by hyperactivity/impulsivity and negatively predicted by Self-Regulation. Impulsivity, in turn, was positively predicted by Difficulty Identifying Feelings and negatively predicted by Self-Regulation.

Three mediation models were statistically significant. Attention Regulation partially mediated the relationship between impulsivity and restrained eating (indirect effect=0.039, 95% bootstrap CI=0.008-0.076). Self-Regulation partially mediated the relationship between Difficulty Identifying Feelings and impulsivity (indirect effect=0.054, 95% bootstrap CI=0.018-0.097). Finally, total alexithymia partially mediated the relationship between total ADHD severity and external eating (indirect effect=0.44, 95% bootstrap CI=0.20-0.74).

Discussion

This study showed that interoceptive processes are meaningfully associated with emotional and behavioral dysregulation in children and adolescents with ADHD, although these associations did not emerge equally across all dimensions of interoception. The most consistent findings were observed for self-reported interoceptive awareness, particularly the dimensions of Attention Regulation and Self-Regulation, whereas cardiac interoceptive accuracy showed only limited and weak associations. This pattern suggests that subjective and objective interoceptive processes may be partially dissociable constructs in pediatric ADHD.

One of the main findings of the study was the negative association between ADHD symptom severity and interoceptive attention regulation. Both inattention and hyperactivity/impulsivity were associated with lower levels of Attention Regulation. This suggests that self-regulation difficulties in ADHD may not be limited to the control of overt behavior, but may also involve the capacity to direct and sustain attention toward internal bodily signals. These findings are consistent with contemporary models conceptualizing ADHD as a broader disorder of self-regulation involving cognitive, emotional, and physiological systems.

The findings regarding eating behaviors further support this framework. External eating was associated with ADHD severity, impulsivity, and alexithymia, whereas restrained eating appeared to be linked to both impulsivity and interoceptive attention regulation. In particular, the partial mediating role of Attention Regulation in the relationship between impulsivity and restrained eating suggests that difficulties in regulating attention to bodily signals may contribute to the transformation of impulsive tendencies into problematic eating patterns. Similarly, the fact that alexithymia was associated with both external and emotional eating, and mediated the relationship between ADHD severity and external eating, highlights the importance of deficits in emotional awareness in eating-related behaviors. Taken together, these findings suggest that maladaptive eating behaviors in ADHD may be related not only to behavioral disinhibition but also to difficulties in emotional awareness and interoceptive regulation.

The associations among alexithymia, impulsivity, and interoceptive awareness are also noteworthy. Difficulty Identifying Feelings was associated with lower Attention Regulation, lower Self-Regulation, and lower Trusting, and was also a strong predictor of impulsivity. The partial mediating role of Self-Regulation in the relationship between Difficulty Identifying Feelings and impulsivity suggests that the capacity to regulate internal states may be one mechanism explaining the link between impaired emotional awareness and impulsive behavior. From this perspective, emotional and bodily awareness processes may be important for understanding the heterogeneous clinical presentation of ADHD.

By contrast, cardiac interoceptive accuracy was minimally associated with ADHD symptoms, eating behaviors, and impulsivity. Significant associations were largely limited to certain interoceptive awareness subdimensions, chronotype, and externally oriented thinking. These findings suggest that, at least when assessed with the Heartbeat Counting Task, performance-based interoceptive accuracy may not be as clinically informative in this population as self-reported interoceptive awareness. Therefore, preserving the distinction between subjective and objective interoception appears important in pediatric ADHD research.

The findings regarding chronotype appear more limited and secondary. Chronotype was associated with ADHD severity and showed only restricted associations with certain interoceptive indices. This may indicate that chronotype is a more distal regulatory feature, or that its role may be better understood through longitudinal studies.

Several limitations of this study should be acknowledged. The cross-sectional design limits causal interpretations. The absence of a control group makes it difficult to evaluate the ADHD-specificity of the observed associations. The heavy reliance on self-report measures may also have increased shared method variance. In addition, cardiac interoceptive accuracy was assessed only with the Heartbeat Counting Task. Nevertheless, the study's strengths include the simultaneous examination of interoceptive awareness, interoceptive accuracy, eating behaviors, chronotype, alexithymia, and impulsivity in a relatively large clinical sample.

In conclusion, the findings suggest that self-reported interoceptive awareness, particularly through the dimensions of Attention Regulation and Self-Regulation, may be clinically meaningful for understanding emotional and behavioral dysregulation in ADHD. Interoceptive awareness appears to occupy a central position within a network

linking ADHD severity, impulsivity, alexithymia, and maladaptive eating behaviors, whereas cardiac interoceptive accuracy appears to play a more limited role. Longitudinal and intervention-based studies are needed to clarify the direction of these relationships and the clinical value of approaches targeting interoceptive processes.

Conclusion

This study showed that interoceptive processes are meaningfully associated with emotional and behavioral dysregulation in children and adolescents with ADHD, although these associations did not emerge uniformly across all dimensions of interoception. The most consistent findings were obtained for self-reported interoceptive awareness, particularly the subdimensions related to attention regulation and self-regulation. In contrast, performance-based cardiac interoceptive accuracy showed much more limited associations.

Taken together, the findings suggest that interoceptive awareness occupies a clinically meaningful position within the network of relationships among ADHD symptom severity, eating behaviors, alexithymia, and impulsivity. Future longitudinal and intervention-based studies are needed to clarify the direction of these relationships and more clearly establish the clinical value of interoception-focused approaches.

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0219 – The Relationship of Odor Perception and Facial Expression Recognition Skills With Aggression and Impulsivity in Patients With Schizophrenia

Introduction

The neurobiological difference of smell from other senses, its direct connection with the central nervous system, and its relation with lower parts of the brain such as limbic regions make it interesting in psychiatric research. The sense of smell, unlike other senses, reaches the relevant parts of the brain directly without going through the thalamus. It is transported to the central nervous system with only two neurons. This makes it easier to be affected by the central nervous system changes. The way neurodegeneration seen in schizophrenia affects the sense of smell has been an area of research interest. Studies have shown that schizophrenia patients have an impaired sense of smell (1). Recognition of facial expressions is an important skill for nonverbal communication, contributing to social cohesion (2). It is known that the ability to recognize facial expressions is negatively affected in patients with schizophrenia (3). Studies show that in schizophrenia, there is an inability to recognize identity and emotion and to distinguish emotions (4, 5). Common limbic system regions such as the orbitofrontal cortex and temporal lobe, amygdala, and entorhinal cortex are involved in odor perception and recognition of facial expressions (6). There is ample evidence that the temporal and limbic regions of the brain are affected in schizophrenia. In these studies, it has been shown that odor perception and facial expression recognition skills are affected in schizophrenia (1). Impulsivity is a significant factor for many psychiatric diseases (7). It has been shown that aggressive and violent behaviors are associated with impulsivity (8). Studies in the literature indicate that aggression and impulsivity may be related to the frontal and temporal lobes. In addition, some studies conducted in patients with schizophrenia and obsessive-compulsive disorder have shown a relationship between the sense of smell and impulsivity (9).

Considering all these, it was thought that the changes in odor perception and facial expression recognition in patients with schizophrenia and changes in impulsivity and aggression were related to neuroanatomically similar regions, and disorders in these areas might be related to each other. No study has been found in the literature examining the relationship between odor detection, face recognition, and impulsivity and aggression.

In our study, it was aimed to determine whether there is a difference in odor perception, facial expression recognition, impulsivity and aggression in patients with schizophrenia compared to healthy controls, and whether there is a relationship between odor perception and facial expression recognition skills and impulsivity and aggression in the patient group.

Our study hypothesizes that patients with schizophrenia are more unsuccessful in odor perception and facial expression recognition skills than healthy controls, and this failure is more pronounced in patients in the exacerbation period. There is a positive relationship between odor perception and facial expression recognition skills in patients with schizophrenia, and there is a negative relationship between odor perception and facial expression recognition skills and impulsivity and aggression in patients with schizophrenia.

Materials - Method

The study was approved by Ankara City Hospital No. 1 Clinical Research Ethics Committee on 16/01/2020.

22 patients with schizophrenia who were hospitalized for exacerbation at Ankara City Hospital and Gulhane Training and Research Hospital and 19 patients with schizophrenia in remission who received outpatient treatment at Ankara City Hospital were included in the study. The healthy control group, which included 20 healthy volunteers, was formed by the hospital staff and their relatives. Participants who were aged between 18-65 years, had no history of disease or respiratory surgery that could affect the sense of smell, had no concomitant medical condition or neurological disease, no other psychiatric disease, and were willing to complete the requirements of the study, were included in the study. All participants were informed about the study and signed an "Informed Voluntary Consent Form". In addition, the inclusion criteria for patients included in the exacerbation group were determined as being inpatient for exacerbation and having a PANSS total score above 70. Patients in the remission group were required to have a PANSS total score of less than 70 and to take their treatment regularly.

Data Collection Tools

Sociodemographic Data Form

It is an information form filled by the researcher, in which some sociodemographic information of the participants included in the study to be used for the research is clarified. It includes information about participants' age, gender, height, weight, education level, marital status, employment status, with whom they live, monthly income level, smoking, other diseases, forensic history, presence of psychiatric and chronic diseases in the family. Inpatient groups also include information about the disorder's duration, number of hospitalizations, and treatment.

Positive and Negative Syndrome Scale (PANSS)

In order to measure the disorder severity, symptoms and functionality of the patient group participating in the study, PANSS was applied with a psychiatric interview conducted by the researcher. The PANSS scale was developed by Kay et al. in 1987 (10), and a Turkish validity and reliability study was conducted by Kostakoglu et al. in 1999 (11).

Sniffin Sticks Smell Test

In order to measure the odor perception skills of all participants participating in the study, the Sniffin Sticks odor test with a total of 112 scent pens was applied. The test was developed by Hummel et al. in 1997 (12), and it was shown that it could be applied to Turkish society by Tekeli et al. in 2013 (13).

There are three subtests: the odor threshold test, the odor discrimination test, and the odor identification test, and a maximum of 48 points can be obtained, 16 from each subtest. Evaluation is made on the total score. A total score of 48-31 is considered "Normosmia", 30-16 is considered "Hyposmia", and scores of 15 and below are considered "Functional Anosmia".

The test applied to a participant lasted between 30-40 minutes on average, and the threshold test, discrimination test and identification test were performed, respectively. The test was conducted in a quiet and well-ventilated room, and the participants did not eat or drink anything other than water for at least 15 minutes before the test. Participants were tested with their eyes closed not to see the scent pen colors. There was a break of 5 minutes between subtests. During the test, the participants were not given any feedback on their answers or any clues.

Facial Expressions Test

In our study, 56 photographs in a set consisting of happy, sad, angry, scared, surprised, disgusted and neutral expressions prepared by P. Ekman and WV Friesen in 1976 were used to measure the facial expression recognition skills of the participants (14). The first seven of these photos of 4 male and four female models were introduced to the participants, and they were asked to guess the expression of emotion in the following 49 photos. The photographs were printed in black and white on A4 (210x297 mm) white paper and had no time limit for completing the test. No feedback was given on the answers given throughout the test.

Barratt Impulsivity Scale-11

It is a self-report scale applied to all participants in the study to assess impulsivity. There are 30 questions in total, and one of the answers is "rarely/never", "sometimes", "often", and "almost always/always". The scale has three subscales: attention, motor and lack of planning. According to the answers given, questions are given between 1-4 points, and a minimum of 30 and a maximum of 120 points are obtained in total. The higher the total score obtained, the higher the impulsivity. The scale was first developed by Patton et al. in 1995 (15), and its Turkish validity and reliability study was performed by Gule9 et al. in 2008 (16).

Buss Perry Aggression Scale

It is a self-report scale applied to all participants in the study to assess aggression. There are 29 questions in total, and participants were asked to rate each item using a 5-point Likert-type scale (1 = uncharacteristic of me, five = very characteristic of me).

The scale has four subscales: verbal aggression, physical aggression, anger and hostility. According to the answers given, points are given to the questions between 15 and a minimum of 29 and a maximum of 145 points are

obtained in total. The higher the total score obtained, the higher the aggression. Buss and Perry first developed the scale in 1992 (17), and its Turkish validity and reliability study was conducted by Madran in 2012 (18).

Statistical analysis

Study data was uploaded to the computer environment and evaluated using "SPSS for Windows 21.0 (SPSS Inc, Chicago, IL)". The conformity of the variables to the normal distribution was examined using visual (histogram and probability graphs) and analytical methods (Kolmogorov-Smirnov/Shapiro-Wilk Tests). Descriptive statistics were presented as mean (±) standard deviation for parametric assessments, median (minimum-maximum), frequency distribution, and percentage for non-parametric assessments. Chi-square and Mann-Whitney-U tests were used to compare sociodemographic and scale data. Spearmen correlation analysis was used for correlation analysis. A value of $P<0.05$ was considered significant.

Results

The comparison of the sociodemographic characteristics of the participants is shown in Table 1.

When schizophrenia patients and the control group were compared in terms of odor tests, it was observed that the control group had statistically significantly higher scores in odor threshold, discrimination, identification and total tests (p.0.002, p:0.003, p:0.001, p:0.001, respectively). When the odor test scores were compared between the groups, it was observed that there was a significant difference between the groups in all subtests; the exacerbation group had a significantly lower score at the threshold subtest. The remission group had a significantly lower score in the discrimination subtest. In the identification subtest, the control group had the highest score, the remission group had lower scores, and the exacerbation group had the lowest scores. The control group was significantly higher than the remission and exacerbation groups in total odor test scores. When the correlation between disorder duration and odor tests in the schizophrenia group was examined, it was observed that there was a significant negative correlation between disorder duration and odor identification score and total odor score (p:0.005, p:0.032, respectively). When PANSS scores and odor tests were examined, it was observed that there was no significant correlation between PANSS scores and odor tests. Table 2 shows the comparison of odor test scores among the participant groups.

When facial expression recognition tests were compared between the groups, the control group recognized sad, furious, frightened and disgusted faces statistically significantly better (p:0.07, p:0.021, p: 0.004, p: 0.002, respectively). In posthoc tests, onyly difference between exacerbation and remission group was observed in recognizing frightened facial expression. The exacerbation group recognized the frightening facial expression at a significantly higher rate than the remission group. When the relationship between facial expression recognition test scores and PANSS scores in the schizophrenia patient group was examined, there was a significant negative correlation between PANSS general psychopathology scores and the ability to recognize frightened faces (r:-0.373, p:0.016). Facial expression recognition scores between groups are shown in Table 3.

When the correlation between facial expression test scores and odor test scores in the schizophrenia patient group was examined; statistically significant positive correlation between the odor threshold test and the ability to recognize sad, furious, frightened, surprised and disgusted facial expressions (p:0.001, p:0.011, p:0.022, p:0.001, p:0.040, respectively), statistically significant positive correlation between the odor discrimination test and the ability to recognize sad, furious, frightened and surprised facial expressions (p:0.004, p:0.001, p:0.001, p:0.003, respectively), statistically significant positive correlation between the odor identification test and the ability to recognize sad, furious, frightened and surprised facial expressions (p:0.001, p:0.001, p:0.001, 0.005, respectively), statistically significant positive relation between the total odor test scores and the ability to recognize sad, furious, frightened, surprised and disgusted facial expressions (p:0.001, p:0.001, p:0.001, p:0.001, p:0.035, respectively) were observed. Table 4 shows the relationship between odor test scores and facial expression test scores.

When the relationship between odor tests and Barratt impulsivity scores in the schizophrenia group was examined, a statistically significant negative correlation was observed between odor identification and motor impulsivity and total impulsivity scores (p:0.013, p:0.047 respectively).

When the relationship between odor tests and Buss-Perry aggression scale scores in the schizophrenia group was examined; significantly negative correlation between odor threshold test and Buss Perry hostility subscale, Buss

Perry verbal aggression subscale and Buss Perry total scores (p:0.036, p:0.001, p:0.042, respectively), significantly negative correlation between odor identification test and Buss Perry hostility subscale (p:0.043), significantly negative correlation between total odor test score and Buss Perry hostility subcale and Buss Perry verbal subscale (p:0.001, p:0.048, respectively). Table 5 shows the relationship between odor test scores and Buss Perry Aggresion Scale scores.

When the relationship between facial expression test scores and Barratt impulsivity scores in the schizophrenia group was examined, a significant negative correlation was observed between the ability to recognize sad facial expressions and the inattention subscale (p.0.010).

When the relationship between facial expression test scores and Buss Perry scale was examined, a statistically significant negative correlation was observed between the ability to recognize angry facial expression and physical aggression, hostility, anger and total Buss Perry Aggression Scale score (p:0.019, p:0.002, p:0.039, p:0.003, respectively). The relationship between the Buss Perry aggression scale and facial expression recognition tests is shown in Table 6.

Discussion

Our study aimed to determine whether there is a relationship between odor perception and facial expression recognition skills and impulsivity and aggression in patients with schizophrenia. No other studies investigating this relationship could be found in the literature. As a result of our study, it was determined that schizophrenia patients might have impairments in odor perception and facial expression recognition skills compared to healthy controls and that there may be a relationship between these impairments and impulsivity and aggression.

Studies have shown that odor perception and facial expression recognition are affected in patients with schizophrenia. In 2014, Moberg et al. evaluated 67 studies on odor perception in a meta-analysis, and it was stated that patients with schizophrenia may have moderate to severe odor perception problems (19). In a study published in 2005, in which the Sniffin Sticks odor test was used to test odor detection, it was observed that patients with schizophrenia were more unsuccessful in all three subtests, namely odor threshold value, odor discrimination and odor identification (20). In this study, Rupp et al. stated that the odor perception problems in schizophrenia patients might be related to the disorder in the orbitofrontal cortex (20). Other studies are claiming that the close relationship of the olfactory brain regions with the frontotemporal and limbic regions and the fact that these regions are the regions that may be effective in the pathophysiology of schizophrenia is the cause of the odor perception problems seen in patients with schizophrenia (21). In a review published by Mandal et al. in 1998, the studies on whether facial expression recognition skills are affected in schizophrenia patients were conducted up to that time examined; it was concluded that there may be deterioration in the ability to recognize negative emotions such as fear and anger in schizophrenic patients (22). It is stated that the emotion with the most difficulty among negative emotions is the expression of fear in most of the studies (23). The brain regions responsible for the recognition of facial expressions are occipitotemporal cortices, amygdala, orbitofrontal cortex, basal ganglia and right parietal lobe (24). It is thought that the reason for the defects in recognition of facial expressions in patients with schizophrenia is the involvement of the amygdala, fusiform gyrus and superior temporal sulcus (25). Many studies claim that amygdala involvement may be associated with negative emotion recognition problems seen in patients with schizophrenia (26-28). According to the results of our study, it can be said that the ability to recognize facial expressions in schizophrenic patients may have decreased compared to healthy controls due to the damage caused by the effect of the disorder on the brain regions related to facial expression recognition skills and although the disorder is in clinical remission with the given treatment, the problem in this area may continue and there may be problems in recognizing mostly negative emotional expressions.

Although there are many studies on odor perception and facial expression recognition skills in patients with schizophrenia, the number of studies examining the relationship between the two is very few. Since it is known that the orbitofrontal cortex, anterior cingulate cortex, amygdala, and some regions of the temporal lobe are involved in both odor perception and emotion processing and that the same regions are affected in schizophrenia (29), there is an expectation that there may be a relationship between odor perception and facial expression recognition skills in patients with schizophrenia. The results of our study, confirming this expectation, show that in the schizophrenia patient group, there is a relationship between all three components of odor perception, threshold value, discrimination and identification, and recognizing facial expressions of negative emotions that are shown to be

affected in schizophrenia. One of the studies on this subject was published by Kohler et al. in 2007. In the study in which 19 schizophrenia patients and 14 healthy controls were included, there was a defect in the ability to recognize facial expressions in the patient group, but no difference was found between healthy controls in terms of odor perception; a significant correlation was found between odor detection and the ability to recognize only sad faces (6). In a study published by Neumann et al. in 2012, the relationship between odor perception and facial expression recognition after traumatic brain injury was examined, and it was found that facial expression recognition skills were impaired in dysosmic patients who were affected by trauma in and around the olfactory center compared to normosmics (30). Wilkinson et al., in 2016, showed a relationship between odor perception from the left nose and recognizing happy and frightened facial expressions in their study conducted in healthy individuals (31). As can be seen, although there are few studies conducted in other patient groups between odor detection and facial expression recognition skills, no other study has been found showing a relationship between negative facial expression recognition skills and odor detection, as determined in the results of our study in patients with schizophrenia. The neurobiological effects of schizophrenia are related to the regions related to odor perception, and facial expression recognition skills can be considered the main reason for the relationship in these two areas.

The main question for which we sought an answer in our study is whether there is a relationship between odor perception and facial expression recognition skills and impulsivity and aggression in patients with schizophrenia. It is known that impulsivity and aggression are associated with orbitofrontal cortex dysfunction and deterioration in medial prefrontal structures and amygdala functions (32). The fact that similar anatomical regions are involved in odor perception suggested that there may be a relationship between odor perception and impulsivity and aggression. In a study conducted in healthy volunteers in 2018, in which the Sniffin Sticks odor test was used, they showed that there was a negative correlation between odor discrimination and the Barratt impulsivity scale's inability to plan subtest, and there was a negative correlation between odor discrimination and odor threshold and motor impulsivity (9). In another study conducted in retired soldiers followed up with a diagnosis of posttraumatic stress disorder, the relationship between odor perception and impulsivity and aggression was examined, and it was stated that odor perception was impaired in the patient group, did not improve with treatment, and odor perception disorder could be an indicator of impulsivity and aggression (33). In an animal experiment measuring the relationship between odor perception and social behavior and aggression, it was reported that a male mouse whose olfactory bulb was removed exhibits aggressive behavior towards other mice, has problems in social communication, and the sense of smell plays an important role in social behavior (34). In a study conducted in schizophrenic patients, although the relationship between odor perception and impulsivity and aggression was not directly examined, it is claimed that there is a relationship between odor perception problems and problems in social interactions and behavior patterns in patients with schizophrenia (35). Considering the results of our study, statistically, significant relationships were found between odor perception and impulsivity and aggression in the schizophrenia patient group, in accordance with our hypothesis. The negative correlation between the scores obtained in the smell tests and the scores obtained in the impulsivity and aggression tests may indicate that the neurobiological effects of schizophrenia may affect the odor perception and may increase the impulsivity and aggression tendencies and these effects may be related to each other. Although clinical improvement may be observed with treatment, the effects in these areas may continue in the patients with schizophrenia. Some researchers have studied the relationship between facial expression recognition skills and impulsivity and aggression. In a study in 2006, it was found that there is a relationship between aggression evaluated by self-report test and evaluating non-angry faces as angry (36). A study conducted on university students in 2014 showed that the rate of error in recognizing non-angry faces as angry was high in the group with high physical aggression characteristics (37). In a study conducted by Demirbuga et al. in Turkey in 2013, 41 patients with a history of violent behavior and referred for forensic examination were compared in terms of facial expression recognition skills with 35 patients with a diagnosis of schizophrenia without a history of violence, and no difference was found between the two groups (38). In a study conducted in 2006, Weiss et al. showed that patients diagnosed with schizophrenia and schizoaffective disorder with a history of violence were more unsuccessful in recognizing angry and frightened facial expressions than patients without a history of violence (39). Antonious et al. also compared the patients with schizophrenia in their study in terms of aggression, which they evaluated with the self-report test, in two groups as high-aggressive and low-aggressiveness, and they found more problems in recognizing the implicit fear in the facial expressions in the group of high-aggressive schizophrenia patients (40). Similarly,

Frommann et al. reported in 2013 that patients with schizophrenia with a history of violence were more unsuccessful in recognizing neutral and frightened facial expressions. The deterioration in recognizing facial expressions may contribute to violent behavior in patients with schizophrenia (41). In the results of our study, it can be said that the ability to recognize facial expressions in schizophrenia patients may give an idea about impulsive and violent behaviors, since there was a significant relationship between recognizing sad facial expressions and the inattention dimension of impulsivity, and between recognizing angry facial expressions and the Buss Perry scale's physical aggression, hostility, anger and total score. It can be thought that the neurobiological damage caused by schizophrenia may cause multi-faceted effects, it may cause adaptation problems in the social life of the patients by reducing their ability to recognize facial expressions, make it difficult for them to communicate with the other person and pave the way for impulsive and aggressive behaviors.

Our study concluded that schizophrenia patients might have problems in odor perception and facial expression recognition skills, that these problems will continue in different periods of the disorder, and that these problems may be related to impulsivity and aggression in the schizophrenia patient group. Although studies have been conducted on odor perception and facial expression recognition skills in patients with schizophrenia, it can be said that the number of studies examining the relationship of these impairments with other characteristics of the patient group is low and more comprehensive studies in this area may be beneficial.

Although our study identified some valuable findings in accordance with its hypothesis, it also has some limitations. The first limitation of our study is that the sample size was not large enough to allow advanced statistical analysis. Another limitation of our study is that the exacerbation and remission patient groups in the schizophrenia patient group consisted of cross-sectionally different patients. A follow- up study in which the same patient group is evaluated in both exacerbation and remission periods will allow a clearer evaluation of the research parameters. One of the limitations of our study can be said to be that impulsivity and aggression were evaluated with self-report scales, and performance-based laboratory tests were not applied. In addition, the fact that insight was not evaluated in the schizophrenia patient groups whose data were collected with self-report scales can be considered a limitation.

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Table 1. Sociodemographic Characteristics of the Participants

Working parameter		Exacerbation n: 22	Remission n: 19	Control n: 20	Statistics	p
Age		ββ (20-63)	38 (28-60)	29 (23-55)	KW:7.525	**0.023**
Gender	Male	%63.6 (n:14)	%84.2 (n:16)	%65.0 (n:16)	χ2:2.506	0.286
	Female	%36.4 (n:8)	%15.8 (n:3)	%35.0 (n:7)		
Education	Primary Education	%31.8(n:7)	%63.2 (n:12)	%5.0 (n:1)	χ2:22.580	**0.001**
	High School	%27.3(n:6)	%26.3 (n:5)	%25.0 (n:5)		
	Associate and Undergraduate Degree	%36.4 (n:8)	%10.5 (n:2)	%40.0 (n:8)		
	Degree	%4.5 (n:1)	%0.0 (n:0)	%30.0 (n:6)		
Marital status	Married	%36.4 (n:8)	%26.3 (n:5)	%70.0 (n:14)	χ2:11.804	**0.019**
	Single	%54.5 (n:12)	%73.7 (n:14)	%30.0 (n:6)		
	Divorced	%9.1 (n:2)	%0.0 (n:0)	%0.0 (n:0)		
Employment	Working	%36.4 (n:8)	%42.1 (n:8)	%90.0 (n:18)	χ2:20.503	**0.002**
	Not Working	%50.0 (n:11)	%36.8 (n:7)	%5.0 (n:1)		
	Student	%9.1 (n:2)	%0.0 (n:0)	%0.0 (n:0)		
	Retired	%4.5 (n:1)	%21.1 (n:4)	%5.0 (n:1)		
living person	Alone	%4.5 (n:1)	%5.3 (n:1)	%5.0 (n:1)	X2:11.117	0.085
	Spouse/Children	%36.4 (n:8)	%26.3 (n:5)	%70.0 (n:14)		
	Mom Dad	%54.5 (n:12)	%52.6 (n:10)	%25.0 (n:5)		
	Friend	%4.5 (n:1)	% 15.8 (n:3)	%0.0 (n:0)		
Smoking		%50.0 (n:11)	%73.7 (n:14)	%60.0 (n:12)	X2:2.402	0.301

Note: KW: Kruskal-Wallis, χ^2: Chi-Square Test

Table 2. Comparison of Odor Test Scores Among Participant Groups

Working parameter	Exacerbation (1) n: 22	Remission (2) n: 19	Control (3) n: 20	Statistics	p	post hoc*
Odor Treshold	4.8 (1-8)	6.0 (1-12)	6.7 (2-9)	KW: 10,897	**0.004**	3=2>1
Odor	11.0 (6-15)	9.0 (4-15)	12.0 (8-15)	KW:9,647	**0.008**	3=1>2
Discrimination Odor	11.0 (4-15)	12.0 (4-16)	13.5 (10-15)	KW:20.924	**0.001**	3>2>1
Identification Odor Test Score	25.5 (11-37)	25.7 (12-37)	32.0 (24-37)	KW: 15.196	**0.001**	3>1=2

Note: KW: Kruskal-Wallis * Mann-Whitney-U Test with Bonferonni correction was used.

Table 3. Comparison of Participants' Facial Expression Recognition Skills

Working parameter	Exacerbation (1) n: 22	Remission (2) n: 19	Control (3) n: 20	Statistics	p	post hoc*
Happy	7.0 (5-7)	7.0 (6-7)	7.0 (7-7)	kW:2.072	0.355	-
Sad	5.0 (2-7)	5.0 (0-7)	7.0 (2-7)	kW:9.844	**0.007**	3>2=1
Angry	5.0 (2-7)	5.0 (1-7)	6.0 (4-7)	KW:7,771	**0.021**	3>2=1
Scared	3.5 (0-7)	2.0 (0-6)	4.0 (0-7)	KW:11.304	**0.004**	3>1>2
Surprised	6.0 (2-7)	6.0 (0-6)	6.0 (5-7)	kW: 4.500	0.105	-
Disgusted	5.0 (3-7)	4.0 (0-7)	7.0 (2-7)	KW:12,286	**0.002**	3>2=1
Neutral	7.0 (2-7)	6.0 (0-7)	7.0 (1-7)	KW: 1.962	0.375	-

Note: KW: Kruskal-Wallis, *Mann-Whitney-U Test with Bonferonni correction was used.

Table 4. Facial Expression Recognition Skills with Odor Tests in the Schizophrenia Group

		Happy	Sad	Angry	Scared	Surprised	Disgusted	Neutral	Odor Treshold	Odor Discri-mination	Odor Identi-fication	Odor Total Test
Happy	r	1,000										
Sad	r	0,084	1,000									
Angry	r	0,088	0,099	1,000								
Scared	r	0,153	0,388**	0,260*	1,000							
Surprised	r	0,322*	0,294*	0,373**	0,293*	1,000						
Disgusted	r	0,361**	0,266*	0,201	0,305*	0,196	1,000					
Neutral	r	0,386**	0,059	0,241	0,263*	0,206	0,139	1,000				
Odor Threshold	r	0,220	0,441**	0,323*	0,292	0,474**	0,264*	0,233	1,000			
Odor Discrimination	r	0,206	0,363**	0,475**	0,472**	0,374**	0,147	0,334**	0,468*** *	1,000		
Odor Identification	r	0,220	0,437**	0,446**	0,413**	0,352**	0,220	0,230	*0,594**	0,646**	1,000	
Odor Total Test	r	0,232	0,490**	0,469**	0,466**	0,412**	0,270*	0,350**	0,764**	0,845**	0,877**	1,000

Note: Spearman correlation analysis was used. *:<0.05, **:<0.01

Table 5. The Relationship between Odor Tests and Buss Perry Aggression Tests in the Schizophrenia Group

		Physical Aggression	Hostility	Anger	Verbal Aggression	Total Aggression	Odor Threshold	Odor Discrimination	Odor Identification	Odor Total Test
Physical Aggression	r	1,000								
Hostility	r	0,620**	1,000							
Anger	r	0,655**	0,699**	1,000						
Verbal Aggression	r	0,464**	0,346**	0,490**	1,000					
Total Aggression	r	0,857**	0,851**	0,885**	0,607**	1,000				
Odor Threshold Test	r	-0,167	-0,270*	-0,198	-0,277*	-0,262*	1,000			
Odor Discrimination	r	0,077	-0,227	-0,116	-0,133	-0,107	0,468**	1,000		
Odor Identification	r	-0,082	-0,260*	-0,180	-0,216	-0,196	0,594**	0,646**	1,000	
Odor Total Test	r	-0,039	-0,257*	-0,174	-0,254*	-0,194	0,764**	0,845**	0,877**	1,000

Note: Spearman correlation analysis was used. *:<0.05, **:<0.01

Table 6. The Relationship between Facial Expression Recognition Skills and Buss Perry Aggression Tests in the Schizophrenia Group

		Physical Aggression	Hostility	Anger	Verbal Aggression	Total Aggressio	Happy	Sad	Angry	Scared	Surprised	Disgusted	Neutral
Physical Aggression	r	1,000											
Hostility	r	0,620**	1,000										
Anger	r	0,655**	0,699**	1,000									
Verbal Aggression	r	0,464**	0,346**	0,490**	1,000								
Total Aggression	r	0,857**	0,851**	0,885***	0,607**	1,000							
Happy	r	-0,045	-0,004	0,045	-0,119	0,000	1,000						
Sad	r	0,083	0,046	0,123	-0,088	0,096	0,084	1,000					
Angry	r	-0,300*	-0,382**	-0,265*	-0,131	-0,378**	0,088	0,099	1,000				
Scared	r	-0,005	0,093	-0,031	-0,081	0,004	0,153	0,388*	0,260*	1,000			
Surprised	r	-0,110	-0,221	-0,230	-0,066	-0,203	0,322*	0,294*	0,373**	0,293*	1,000		
Disgusted	r	-0,249	-0,244	-0,113	-0,186	-0,241	0,361**	0,266*	0,201	0,305*	0,196	1,000	
Neutral	r	-0,097	-0,062	-0,160	-0,200	-0,148	0,386**	0,059	0,241	0,263*	0,206	0,139	1,000

Note: Spearman correlation analysis was used. *:<0.05, **:<0.01

0220 - WISC-IV Cognitive Profile, Subindices, and Index Discrepancy in Children Presenting With Attention and/or Learning Difficulties: Associations With Clinical Variables

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Objective

This study aimed to examine the WISC-IV cognitive profile in children and adolescents presenting with attention and/or learning difficulties and to elucidate the clinical significance of index discrepancy as an indicator of imbalance across cognitive domains. Additionally, the associations of WISC-IV indices and index discrepancy with age, sex, clinical symptoms assessed by the Conners' Teacher Rating Scale, ADHD diagnosis, and the clinical decision regarding the appropriateness of pharmacological treatment were evaluated.

Methods

A total of ninety-five children who were referred to a child and adolescent psychiatry outpatient clinic due to attention and/or learning concerns and who had undergone Wechsler Intelligence Scale for Children-Fourth Edition assessment as part of their clinical evaluation were included in this cross- sectional, retrospective study. Cognitive assessment included Verbal Comprehension Index, Perceptual Reasoning Index, Working Memory Index, Processing Speed Index, and Full Scale Intelligence Quotient scores. Index discrepancy was calculated using the formula (Verbal Comprehension Index plus Perceptual Reasoning Index divided by two) minus (Working Memory Index plus Processing Speed Index divided by two). Behavioral symptoms were assessed using the Conners Teacher Rating Scale, and attention-deficit/hyperactivity disorder diagnosis was determined based on Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition criteria derived from clinical records. The clinical appropriateness of pharmacological treatment was defined as a comprehensive clinical decision based on psychiatric evaluation, clinical observation, and parent and teacher reports. Statistical analyses included independent samples t-tests for group comparisons, Spearman correlation analyses for associations between variables, and logistic regression analyses to identify predictors of behavioral problems and clinical decision-making, with statistical significance set at a probability level of less than zero point zero five.

Results

A total of 95 children and adolescents presenting with attention and/or learning difficulties to a child and adolescent psychiatry outpatient clinic were included in the study. Of the participants, 59 (62.1%) were male and 36 (37.9%) were female. All participants underwent WISC-IV assessment prior to the initiation of pharmacological treatment. Clinical symptoms were evaluated using the Conners' Teacher Rating Scale.

When cognitive and clinical characteristics were examined by sex, no significant differences were found between males and females in Verbal Comprehension, Perceptual Reasoning, Working Memory, or Full Scale IQ scores (all $p > 0.05$). However, Processing Speed Index scores were significantly higher in females (97.61 ± 14.18) compared to males (90.71 ± 14.77) ($t = -2.24$, $p = 0.027$). Although index discrepancy scores were higher in males (3.19 ± 13.53) than in females (-1.76 ± 13.31), this difference did not reach statistical significance ($p = 0.085$). In terms of clinical variables, no significant sex differences were observed in Conners attention and hyperactivity subscales ($p > 0.05$), whereas conduct problems scores were significantly higher in males ($t = 2.19$, $p = 0.032$). Logistic regression analysis further identified male sex as a strong risk factor for conduct problems ($\chi^2 = 5.49$, $p = 0.019$; OR = 27.8, $p = 0.001$).

Associations between WISC-IV index scores and clinical symptoms were examined using Spearman correlation analysis in 33 participants with complete data. A moderate negative correlation was found between the Working Memory Index and Conners total score ($r = -0.439$, $p = 0.011$). Working memory also showed a significant negative correlation with the Conners conduct problems subscale ($r = -0.378$, $p = 0.030$), while its association with the attention subscale was at a trend level ($r = - 0.334$, $p = 0.058$). No significant correlations were observed

between Processing Speed, Verbal Comprehension, or Perceptual Reasoning indices and Conners attention, hyperactivity, conduct problems, or total scores (all $p > 0.05$). As expected, Conners subscales demonstrated strong intercorrelations (e.g., total score with attention $r = 0.716$, $p < 0.001$; with conduct problems $r = 0.854$, $p < 0.001$).

Index discrepancy showed a moderate negative correlation with the Working Memory Index ($r = -0.387$, $p < 0.001$) and a stronger negative correlation with the Processing Speed Index ($r = -0.463$, $p < 0.001$). In contrast, no significant associations were found between index discrepancy and Verbal Comprehension or Perceptual Reasoning indices ($p > 0.05$).

Age was negatively correlated with index discrepancy ($r = -0.263$, $p = 0.010$) and with the Perceptual Reasoning Index ($r = -0.361$, $p < 0.001$). No significant relationships were found between age and Verbal Comprehension, Working Memory, Processing Speed, or Full Scale IQ scores ($p > 0.05$).

When participants with ADHD ($n = 54$) were compared to those without ADHD ($n = 33$), a significant difference was observed only in the Verbal Comprehension Index (ADHD: 95.33 ± 12.25; non-ADHD: 103.70 ± 13.11; $p = 0.003$). No significant group differences were found for Perceptual Reasoning, Working Memory, Processing Speed, Full Scale IQ, composite indices, or index discrepancy (all $p > 0.05$).

Participants for whom pharmacological treatment was considered clinically appropriate ($n = 54$) were identified based on a comprehensive clinical evaluation integrating clinical observation, examination findings, and parent- and teacher-reported measures; WISC-IV results were not used as a direct determinant in this decision. Compared to those not considered suitable for pharmacological treatment ($n = 32$), this group demonstrated significantly lower Working Memory Index scores ($p = 0.007$), as well as lower Processing Speed Index scores ($p = 0.027$). Verbal Comprehension Index ($p = 0.012$) and Full Scale IQ ($p = 0.011$) scores were also significantly lower in this group. In contrast, no significant differences were observed in Perceptual Reasoning Index or in the higher-order reasoning composite index (Verbal Comprehension + Perceptual Reasoning) ($p > 0.05$). Derived indices revealed that the combined Working Memory + Processing Speed scores were significantly lower in the pharmacologically indicated group ($p = 0.002$). Moreover, index discrepancy was significantly higher in this group (3.06 ± 13.17) compared to those not considered for pharmacological treatment (-2.45 ± 13.75) ($t = 3.25$, $p = 0.002$).

Discussion

In this study, the cognitive profile of children and adolescents presenting with attention and/or learning difficulties was examined in relation to clinical symptoms, sex, and the clinical appropriateness of pharmacological treatment. The findings suggest that cognitive heterogeneity in ADHD is shaped not merely by diagnostic categories but rather by specific vulnerabilities across distinct cognitive domains, which meaningfully align with clinical decision-making processes (1). One of the key findings was that, although no significant sex differences were observed in overall cognitive capacity, females demonstrated significantly higher processing speed scores. This indicates that sex differences in ADHD may emerge in specific domains such as cognitive speed and information processing efficiency rather than in general intellectual ability. Lower processing speed scores observed in males may be associated with the more pronounced academic difficulties and functional impairments frequently encountered in clinical practice. In terms of clinical symptoms, while no sex differences were found in attention and hyperactivity domains, conduct problems were significantly more prevalent in males, and male sex was identified as a strong risk factor for conduct-related difficulties. This supports the notion that sex differences in ADHD are more prominently reflected in patterns of behavioral expression, which may partly explain the earlier and more frequent clinical referrals observed in boys. Regarding the cognitive meaning of index discrepancy, its negative associations with working memory and particularly with processing speed indicate a profile characterized by relatively preserved higher-order reasoning abilities alongside weaknesses in executive functions and cognitive speed, which appear to drive the observed cognitive asymmetry (2). This pattern is consistent with previous findings indicating that ADHD is characterized not by a generalized intellectual deficit but by a specific cognitive profile marked by relatively preserved verbal and perceptual reasoning abilities alongside impairments in working memory and processing speed, supporting the clinical relevance of cognitive profiling in ADHD(3).Furthermore, the negative correlation between age and index discrepancy suggests that although cognitive imbalance does not fully resolve over time, it may partially diminish with development. The absence of significant differences between ADHD and non-ADHD groups in cognitive variables such as working memory, processing speed, and index discrepancy may initially appear inconsistent with findings from studies comparing ADHD with neurotypical controls; however, this

discrepancy can be explained by the characteristics of the sample. The non-ADHD group in this study consisted of clinically referred individuals with attention and/or learning complaints rather than a neurotypical population, which may have increased the overlap in cognitive profiles between groups. In this study, the clinical appropriateness of pharmacological treatment reflected a multidimensional clinical judgment based on integrated evaluation of clinical observations, examination findings, and parent- and teacher- reported measures, with WISC-IV results not serving as a direct determinant in this decision-making process. Nevertheless, the finding that individuals for whom pharmacological treatment was considered appropriate had lower working memory and processing speed scores, as well as higher index discrepancy, indicates a notable concordance between clinical decision-making and cognitive profile. This suggests that pharmacological treatment decisions are not random or based solely on a single symptom domain but are instead shaped by a broader clinical picture characterized by impairments in executive functioning and cognitive speed that impact overall functioning (4,5). Importantly, these findings do not evaluate the cognitive effects of medication; rather, they demonstrate that clinical decisions correspond meaningfully to underlying cognitive profiles, thereby supporting the ecological validity of multidimensional clinical assessment.

Keywords: Attention Deficit Disorder with Hyperactivity, Executive Function, Neuropsychological Tests, Processing Speed, Wechsler Scales

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0224 - Orcadian Gene Interactions (*CLOCK* and *PER3*) Differentially Predict Lithium and Valproate Response in Bipolar I Disorder

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Background and Objective:

Bipolar disorder (BD) 7s a chronic psychiatric illness characterized by recurrent manic and depressive episodes, exhibiting marked heterogeneity in both clinical course and treatment response. Increasing evidence indicates that disruptions in circadian rhythm regulation play a central role in the pathophysiology and clinical presentation of BD, with alterations in biological timing being associated with mood instability, increased relapse risk, and variability in response to mood stabilizers [1].

At the genetic level, core components of the circadian clock system, particularly the CLOCK and PER3 genes, are critical for maintaining the stability of biological rhythms. Genetic variations in these clock genes are thought to contribute to interindividual differences in circadian functioning and behavioral regulation [1].

Lithium is considered a key agent in the maintenance treatment of BD and there is evidence suggesting that its therapeutic effects may be linked to its regulatory influence on biological rhythms. Across a range of organisms, including humans, lithium has been shown to lengthen the circadian period.Furthermore, experimental models of BD have demonstrated that mutations in the CLOCK gene can lead to mania-like behavioral alterations that are responsive to lithium [2]. However, studies examining the relationship between lithium's clinical efficacy and polymorphisms in circadian rhythm-related genes remain limited.

Accumulating evidence suggests that valproate may also influence circadian rhythms. Animal and human studies have demonstrated that valproate can modulate circadian rhythmicity. This effect is thought to be mediated, at least in part, by epigenetic mechanisms that may underlie its chronotherapeutic properties. Moreover, interindividual variability in treatment response to valproate may be influenced not only by differences in circadian rhythm characteristics but also by genetic polymorphisms in circadian clock-related genes [3]. Although mood stabilizers such as lithium and valproate are known to modulate circadian- related molecular pathways, the extent to which polymorphisms in circadian clock genes influence long-term treatment response in patients with BD-I remains insufficiently understood.

The present study aimed to investigate the associations between CLOCK rs1801260 and PER3 VNTR polymorphisms and differential response to lithium and valproate in patients with BD-I.

Methods:

Study Sample

This cross-sectional study was conducted at the Psychiatry Department of Ankara Bilkent City Hospital. The sample consisted of 103 patients diagnosed with bipolar disorder type I (BD-I) according to DSM-5-TR criteria. All participants were in sustained remission for at least three months, which was confirmed using the Hamilton Depression Rating Scale (HDRS < 7) and the Young Mania Rating Scale (YMRS < 7).

Inclusion criteria were: being between 18 and 65 years of age, having at least 5 years of formal education, providing written informed consent, being in sustained remission for at least three months, and having sufficient proficiency in Turkish.

Exclusion criteria included severe neurological disorders; autoimmune or chronic inflammatory diseases; metabolic or endocrine disorders; a diagnosis of cancer; pregnancy; current use of anti-inflammatory or immunosuppressive medications; current alcohol or substance use disorder; receipt of electroconvulsive therapy within the past six months; and language difficulties that could interfere with a diagnostic psychiatric interview. Written informed

consent was obtained from all participants. This study was approved by the Institutional Ethics Committee of Ankara Bilkent City Hospital, and all procedures were conducted in accordance with the Declaration of Helsinki.

Psychometric Measures

A Sociodemographic and Clinical Information Form developed by the researchers was used to assess participants sociodemographic characteristics, current treatments and medical history. Long-term prophylactic treatment response was evaluated using the ALDA scale, in which the A score rates treatment response to mood stabilizers on a scale from 0 to 10, while the B score includes factors that may influence treatment response, such as treatment adherence, concomitant treatments, and illness characteristics; the final A-B score is obtained by subtracting the B score from the A score [4]. Personal and social functioning was assessed using the Personal and Social Performance Scale across four domains: socially useful activities, interpersonal relationships, self-care, and disturbing and aggressive behaviors [5].

Genetic Analysis

Genomic DNA was isolated from peripheral venous blood using the Roche® High Pure PCR Template Preparation Kit. The CLOCK gene (rs1801260) polymorphism was analyzed using SNP real-time polymerase chain reaction based on the TaqMan® probe principle on a Bio-Rad CFX96™ system.

The PER3 VNTR polymorphism was amplified using conventional PCR with Q5 High- Fidelity DNA Polymerase, and amplification products were separated by 2% agarose gel electrophoresis. Genotypes were determined based on fragment lengths, with the 4-repeat allele corresponding to 347 bp and the 5-repeat allele to 401 bp.

Statistical Analysis

Sociodemographic and clinical characteristics of the participants were presented as number and percentage [n (%)], and descriptive statistics were calculated.

General linear models were used to examine gene-gene and gene-drug interaction effects on treatment response, adjusting for relevant clinical covariates. In addition, multiple linear regression analyses were conducted to identify independent predictors of treatment response.

All statistical analyses were performed using IBM SPSS Statistics (version 21), and the level of statistical significance was set at $p < 0.05$.

Results:

The mean age of the sample (n=103) was 37.01±10.49 years, with 46.6% (n=48) females and 53.4% (n=55) males. The mean duration of education was 13.65±2.86 years. Of the participants, 53.4% were single, 32.0% were married, and 14.6% were divorced. Regarding employment status, 62.1% were employed, 27.2% were unemployed, 7.8% were students, and 2.9% were retired. Most participants lived with their families (81.6%) and resided in urban areas (80.6%).

Smoking was reported by 64.1% of participants. While 69.9% reported no alcohol use, 28.2% were social drinkers and 14.6% had a history of substance use. Mean height, weight, and BMI were 170.52±10.93 cm, 84.19±19.53 kg, and 28.85±5.66 kg/m^2, respectively.
The mean age at onset of illness among patients with bipolar disorder was 24.99 ± 7.02 years, and the mean duration of illness was 136.33 ± 105.17 months. Patients had a mean of 2.55 ± 2.47 hospitalizations, 3.04 ± 2.30 manic episodes, and 1.79 ± 2.43 depressive episodes.

Regarding treatment, the most commonly used regimens were valproate plus antipsychotics (43.7%; n = 45) and lithium plus antipsychotics (31.1%; n = 32). These were followed by antipsychotic monotherapy (13.6%; n = 14), valproate monotherapy (6.8%; n = 7), and lithium monotherapy (3.9%; n = 4). A combination of lithium, valproate, and antipsychotics was observed in 1.0% of patients (n = 1).

General linear model analysis demonstrated a significant three-way interaction between CLOCK genotype, PER3 genotype, and mood stabilizer type (lithium vs. valproate) on ALDA A-B scores ($p = 0.002$, partial $n2 = 0.124$), indicating that treatment response varied according to the combined effects of circadian gene variants and pharmacological context (Table-1).

Simple effects analyses revealed that this interaction was specific to individuals with the CLOCK heterozygous genotype (AG). Within this subgroup, patients carrying PER3 4/45/5 genotypes showed significantly higher ALDA scores under lithium treatment ($F = 4.415$, $p = 0.039$), whereas those with the PER3 4/5 genotype demonstrated significantly higher ALDA scores under valproate treatment ($F = 6.021$, $p = 0.017$). No significant treatment-related differences were observed among CLOCK homozygotes.

In multiple linear regression analysis, the CLOCK heterozygous genotype (AG) emerged as the only independent predictor of the ALDA A score ($\beta = 0.208$, $p = 0.039$), whereas clinical variables were not statistically significant predictors (Table-2).

Table-1 General Linear Model results for the ALDA A-B score

Effect	**F**	**p value**	**partial η^2**
Medication	0,01	0.905	<0,001
CLOCK	1,20	0.278	0,017
PER3	0,01	0.906	<0,001
Medication x CLOCK	<0,01	0.970	<0,001
Medication x PER3	3,50	0.066	0,047
CLOCK x PER3	0,77	0.384	0,011
Medication x CLOCK x PER3	10,09	0.002*	0,124
Age	3,85	0.054	0,052
BMI	0,84	0.362	0,012
Smoking Status	0,07	0.799	0,001

Age, body mass index, and smoking status were included as covariates.

CLOCK genotypes were grouped as AA+GG vs. AG; PER3 genotypes as 4/4+5/5 vs. 4/5; medication as lithium vs. valproate. * $p < 0.05$.

Table-2 Multiple Linear Regression Analysis of Clinical, Circadian, and Genetic Predictors of ALDA A Score

Variable	**B**	**Std. Error**	**β**	**P**
Number of manic episodes	-0,045	0,052	-0,118	0,392
Number of depressive episodes	-0,028	0,046	-0,071	0,546
Duration of illness (months)	-0,002	0,001	-0,207	0,137
CLOCK	0,357	0,170	0,208	0,039*
PER3	-0,251	0,164	-0,150	0,130

CLOCK genotypes were grouped as AA+GG vs. AG; PER3 genotypes as 4/4+5/5 vs. 4/5. * $p < 0.05$.

Discussion:

The present study provides evidence that treatment response in BD-I is modulated by interactions between circadian clock gene polymorphisms and mood stabilizer type. A significant three-way interaction between CLOCK rs1801260, PER3 VNTR polymorphisms, and lithium versus valproate treatment indicates that genetic variation in the circadian system differentially influences treatment outcomes depending on the pharmacological context. Notably, this effect was confined to individuals with the CLOCK heterozygous genotype, suggesting a potential heterozygote advantage in modulating treatment response.

The CLOCK rs1801260 polymorphism has been widely associated with circadian- related phenotypes in BD, including age at onset, episode frequency, and sleep-wake regulation. Studies employing the ALDA scale suggest that CLOCK variants do not independently predict lithium efficacy, but rather may act as modulators of circadian phenotypes that indirectly influence clinical outcomes [6]. Similarly, the CLOCK 3111 T/C polymorphism has not

been shown to distinguish between full and partial response to valproate maintenance treatment, further supporting the notion that these variants are more closely linked to circadian and clinical characteristics than to treatment response [3].

Our findings extend this framework by demonstrating that circadian gene variants may influence treatment outcomes through gene-gene and gene-drug interactions rather than as independent predictors. The observed involvement of PER3 polymorphisms further supports the role of circadian regulation in shaping treatment response. Existing literature indicates that PER3 is associated with circadian phenotypes such as susceptibility to BD, age at onset, and seasonality; however, it has not been shown to reliably predict prophylactic lithium response at the SNP or haplotype level. In this context, PER3 may be better conceptualized as a susceptibility gene contributing to the circadian biological substrate of the disorder rather than as a direct determinant of treatment response [7,8]. The absence of a significant main effect of PER3 genotypes in our study is consistent with this view, although prior studies have not specifically examined the PER3 VNTR polymorphism, highlighting a potential source of divergence in our findings.

The limited explanatory power of single-gene effects on treatment response may reflect the inherently network-based and interactive nature of the circadian clock system. The observed differential response to lithium and valproate among patients heterozygous for both CLOCK and PER3 polymorphisms underscores the modulatory role of circadian genetic architecture in long-term outcomes. These results support the potential value of circadian genetic markers in guiding individualized treatment strategies within a precision psychiatry framework and provide a foundation for future longitudinal studies examining circadian mechanisms of treatment response.

Keywords: Bipolar Disorder, Circadian Rhythm, Genetic Polymorphism, Pharmacogenetics

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0225 - Memory Functions in the Schizophrenia Spectrum: First-Episode Psychosis and Chronic Schizophrenia Compared with Healthy Controls

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Introduction: Schizophrenia is a pervasive psychiatric syndrome characterized by positive symptoms, such as hallucinations, delusions, and disorganized speech; negative symptoms, including avolition, affective flattening, and social withdrawal; and widespread cognitive impairments affecting multiple domains such as attention, working memory, episodic memory, and processing speed. Affecting approximately one percent of the global population, this disorder is associated with persistent functional loss and a high burden of disability, arising from the complex interaction of genetic predisposition, environmental factors, neurodevelopmental processes, and dysregulations in dopaminergic and glutamatergic systems. First-episode psychosis (FEP) is a clinical term used to describe the first time an individual experiences a threshold level of psychotic symptoms, representing a critical period where the biological processes of the illness can be observed in their earliest and clearest form. Although FEP is not a distinct diagnostic category in DSM-5, it is evaluated within the framework of "recent-onset psychosis," typically covering the first 2 to 5 years following the manifestation of symptoms. Cognitive impairments, including memory functions, are considered core components of schizophrenia independent of positive and negative symptoms, manifesting significantly in both the first episode and chronic stages. Literature indicates that cognitive performance levels in individuals experiencing FEP largely mirror those of patients with chronic schizophrenia (CS), suggesting that cognitive decline is an intrinsic pathophysiological feature of the illness rather than a secondary consequence of its progression. Cognitive functions constitute a holistic process of acquiring and understanding information through thought, experience, and the senses, encompassing high-level mental processes such as attention, memory, and executive functions. These impairments, observed in up to 75% of patients, are of paramount clinical importance as they directly impact general functionality and social adaptability. Specifically, verbal memory deficit is recognized as one of the strongest neuropsychological predictors of functional outcomes. Standardized measures such as the Auditory Verbal Learning Test (AVLT) play a critical role in identifying deficits in the encoding, storage, and retrieval of verbal material. Similarly, visual memory and organizational skills evaluated via the Rey-Osterrieth Complex Figure Test (RCFT-R) objectify impairments in the immediate and delayed recall of visual information. The objective of this study is to determine neurocognitive impairments in remitted FEP and CS patients using the AVLT, RCFT-R, Stroop, and Verbal Fluency tests, and to compare them with healthy controls to provide a holistic framework for the progression of cognitive deficits across illness stages.

Method: The sample of this case-control study consisted of 39 patients with remitted FEP, 51 patients with CS meeting the DSM-5 diagnostic criteria, and 55 healthy controls (HC). The psychiatric health of the HC group was confirmed via the Structured Clinical Interview for DSM-5 (SCID-5). The HC group was matched with the CS patients in terms of age, sex, and educational background. The CS group included patients with a history of multiple episodes who had been free of active symptoms for at least the last six months and were in remission with their current treatment. The FEP group comprised individuals experiencing their first psychotic episode who were in symptomatic remission. All participants were between 18 and 65 years of age and were literate. To verify clinical stability, all patients were required to have a score of 3 or less on the specific PANSS subscale items (P1, P2, P3, N1, N4, N6, G5, G9) related to symptomatic remission criteria. History of neurological disease, intellectual disability, substance use disorder, and electroconvulsive therapy (ECT) within the last six months were established as primary exclusion criteria. Neuropsychological assessments were administered in the morning hours in a quiet environment following a standardized sequence. Verbal episodic memory was assessed using the AVLT, which evaluates the learning curve, short- and long-term free recall, and recognition components. Visual memory and visuospatial organizational abilities were evaluated through the RCFT-R consisting of copy, immediate/delayed recall, and recognition stages. Cognitive flexibility, focused attention, and generative capacity were assessed using the Stroop Test (TBAG form) and Verbal Fluency Tests. The Verbal Fluency tasks consisted of semantic (animal category) and phonemic (letters K, A, S) tasks, with a duration of one minute provided for each category to examine strategic information retrieval processes. Statistical analyses involved group comparisons using ANCOVA

or the Kruskal-Wallis H test, depending on the normality of data distribution, with Bonferroni or Dunn's tests applied for multiple comparisons.

Results: The study evaluated 39 patients with FEP, 51 patients with CS, and 55 healthy controls (HC). Analysis of sociodemographic data revealed that median age scores differed significantly across the three groups ($p < 0.001$). Post-hoc comparisons indicated that while the CS and HC groups were statistically similar in terms of age (medians of 40 and 35, respectively; $p > 0.05$), the FEP group (median 31) was significantly younger than both the CS and HC groups. No significant differences were observed between any of the groups in terms of sex ($p = 0.196$) or years of education ($p = 0.148$). Significant associations were found regarding marital and employment status; the HC group had significantly higher rates of being married (58.2%) and employed (98.2%) compared to the patient groups ($p < 0.001$). Regarding clinical variables, the CS group demonstrated a significantly longer illness duration (median 144 months) and higher hospitalization rates compared to the FEP group ($p < 0.001$). Antipsychotic dosages, calculated as olanzapine equivalents, were significantly higher in the CS group (median 23.5 mg) than in the FEP group (median 15.03 mg; $p < 0.001$). Although all patients were in symptomatic remission, the CS group exhibited significantly higher PANSS-N (negative symptoms) and PANSS-T (total) scores compared to the FEP group ($p < 0.05$). Verbal episodic memory was markedly impaired in patient groups compared to HCs. Patients demonstrated significantly lower scores in total learning (A1-A5), learning slope (A5-A1), and immediate recall (A6) ($p < 0.001$). To account for demographic differences, an ANCOVA was performed using age, sex, education, and attention (Stroop B5) as covariates. After adjusting for these factors, total recognition (AVLT A+B) remained significantly different across all groups ($p < 0.001$), with the CS group showing the most pronounced deficits. When further controlled for illness-specific variables (duration, dose, and anticholinergic use), no significant differences were found between FEP and CS groups, indicating a stabilized level of verbal memory impairment across illness stages. Regarding the RCFT-R, the CS group demonstrated significantly lower copy scores than HCs ($p = 0.007$). Significant impairments were observed in both patient groups for immediate and delayed recall ($p < 0.005$). An ANCOVA was conducted with age, education, and attention as covariates; the results confirmed that delayed recall scores remained significantly lower in both FEP and CS groups compared to HCs ($p = 0.003$), suggesting that visual memory consolidation deficits are independent of demographic and attentional factors. Attentional control and cognitive flexibility were significantly reduced in patients. The CS group exhibited significantly prolonged completion times in the interference trial (B5) compared to both FEP and HC groups ($p < 0.001$). Similarly, generative capacity was impaired; both patient groups produced fewer words in semantic and phonemic fluency tasks than HCs ($p < 0.001$). After adjusting for the effects of age and education, phonemic fluency remained significantly lower in the CS group compared to HCs ($p = 0.031$), and both patient groups showed significantly higher perseveration errors ($p < 0.001$).

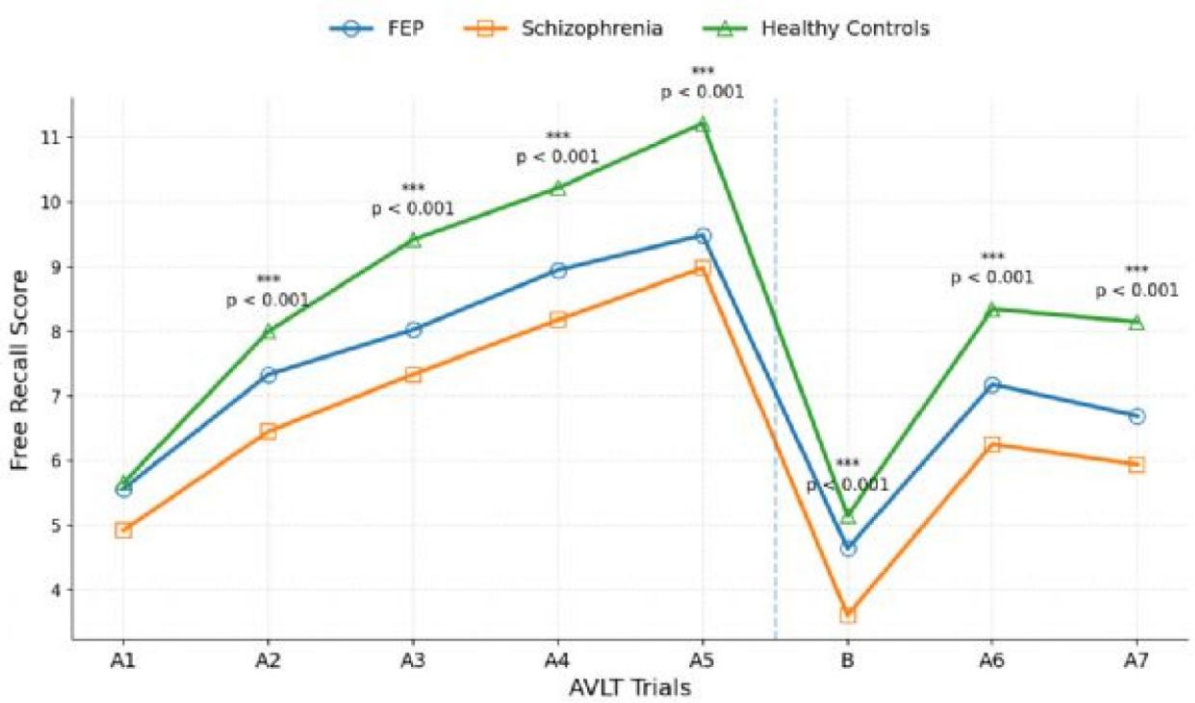
Discussion: This study, which analyzes the stages of cognitive impairment in the schizophrenia spectrum by comparing patients with remitted FEP and CS to HC at the level of neuropsychological subtest parameters, confirms that cognitive decline is not merely a consequence but a foundational component present in both stages of the illness. The AVLT results, examining verbal memory processes, align with the "encoding and retrieval deficit" pattern reported in the literature. The lack of significant differences between groups in AVLT-A1 (Immediate Registration) scores suggests that basic information entry mechanisms are relatively preserved. However, the marked decline in AVLT A1-A5 Total (Learning Capacity) and AVLT A5-A1 (Learning Slope) scores following repeated presentations indicates that the ability of patients to organize and enrich new information in working memory (encoding) is limited in both stages. The most striking finding is the profound performance loss observed in the AVLT-A7 (Delayed Recall) parameter. This stage, reflecting the transfer of information into long-term memory (consolidation), significantly differentiates the FEP and CS groups from HCs, proving that episodic memory deficit is a core feature independent of illness duration. The fact that AVLT Recognition performance is better than free recall but still lags behind the healthy group suggests that the problem lies not only in retrieval strategies but also in the weak formation of memory traces (engrams). In the RCFT-R test measuring visual memory capacity, the CS group's low performance even in the Copy phase reflects an erosion in visuospatial organization and planning skills during the chronic stage. Conversely, the decline in both Immediate and Delayed Recall scores in both FEP and CS groups indicates a widespread deficiency in the mental manipulation and long-term storage of visual material. The fact that these differences remained significant even when attention (Stroop B5) was controlled suggests that visual memory deficit is a primary consolidation disorder rather than a secondary effect of attentional impairment. Results from the Stroop Test assessing executive functions showed that the CS

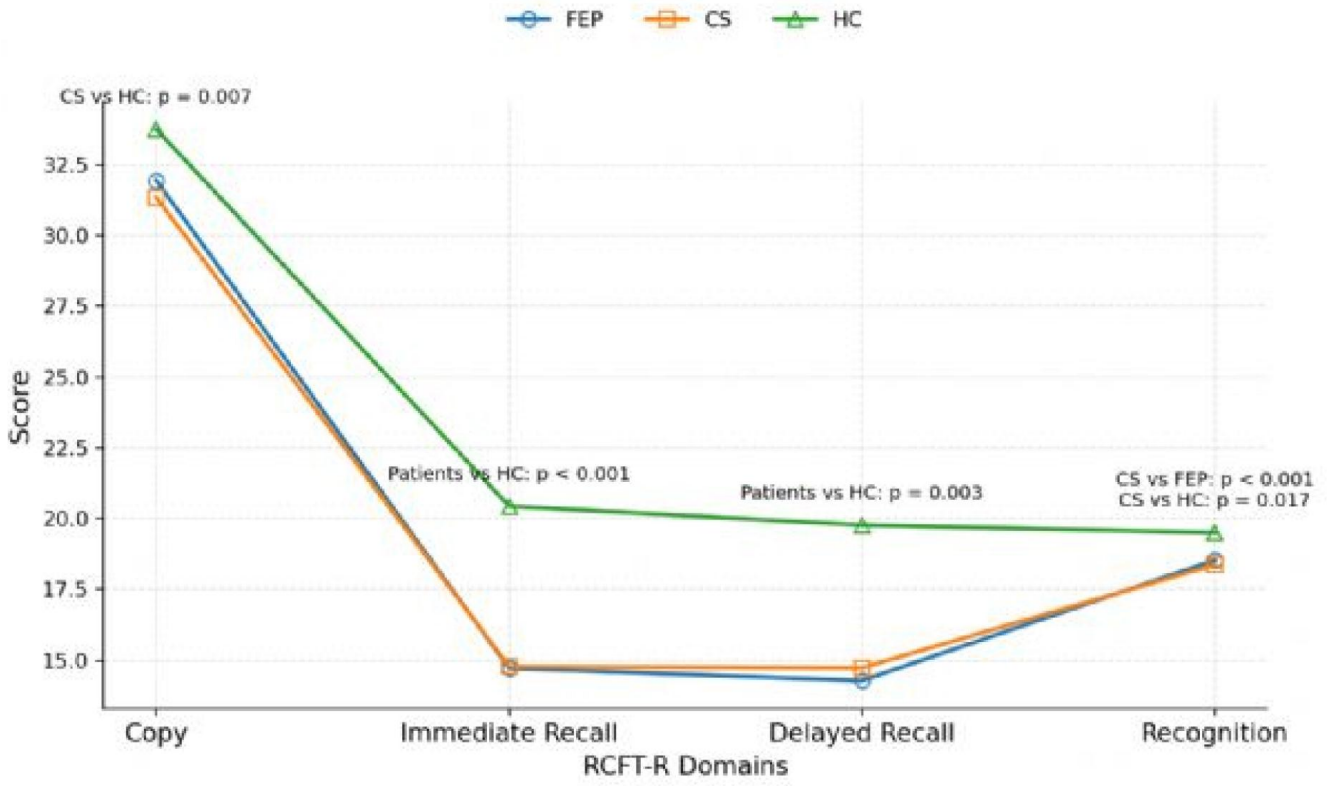
group demonstrated the lowest performance, particularly in the B5 (Interference) stage. This indicates that the ability to suppress an automatic response and maintain a goal-directed one (inhibition) becomes increasingly challenged as the illness progresses. Lower word production in Verbal Fluency tests and high Perseveration errors in the CS group reveal an impairment in strategic searching within semantic memory networks and the ability to discard inappropriate data (generative capacity). AVLT and RCFT-R subtests show that memory deficits in schizophrenia are concentrated in learning rates and long-term storage rather than immediate registration, while the similarity of memory profiles between FEP and CS groups points toward cognitive stability.

Conclusion: Our research demonstrates that deficiencies in memory and cognitive functions begin in the very early stages of schizophrenia and persist even during periods of remission. The data obtained through AVLT and RCFT-R parameters support the view that episodic memory impairments are a fundamental neurocognitive component. The performance declines in Stroop and verbal fluency tests indicate that executive functions undergo deeper deterioration, particularly in the chronic stage. These findings emphasize the critical importance of integrating comprehensive neurocognitive rehabilitation programs into the treatment process from the earliest stages of diagnosis to preserve patients' social and occupational functionality.

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FEP
CS
HC
CS vs HC: p = 0.007
Patients vs HC: p < 0.001
Patients vs HC: p = 0.003
CS vs FEP: p < 0.001
CS vs HC: p = 0.017
Score
32.5
30.0
27.5
25.0
22.5
20.0
17.5
15.0
Copy
Immediate Recall
Delayed Recall
Recognition
RCFT-R Domains

0228 - Clinical and Sociodemographic Comparison of Bipolar Depression and Major Depressive Disorder

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Ankara Bilkent §ehir Hastanesi

Background and Objectives:

Major Depressive Disorder (MDD) and bipolar depression (BD) share identical diagnostic criteria for a major depressive episode across diagnostic systems. Bipolar disorder often initially presents with a depressive episode, and prior hypomanic episodes are frequently unrecognized or underreported by patients. This situation complicates the differential diagnosis during depressive presentations and contributes to delays in the diagnosis of bipolar disorder. Indeed, previous studies have reported that the interval between the onset of symptoms and the establishment of a bipolar disorder diagnosis is approximately 7-10 years.

Misdiagnosis and delays in establishing the correct diagnosis may lead to inappropriate treatment approaches and delayed interventions. Consequently, this may result in adverse outcomes such as manic switching, rapid cycling, recurrent episodes, functional impairment, increased illness burden, and an elevated risk of suicide, ultimately worsening the course of the disorder.

Previous studies have identified non-pathognomonic differences between bipolar depression and MDD in terms of illness course, family history, and symptom patterns; however, these differences are often not sufficiently reliable for accurate differentiation in routine clinical practice, particularly during depressive episodes. The aim of this study was to compare the clinical and sociodemographic characteristics of bipolar depression and MDD to contribute to the differential diagnosis(1,2).

Methods:

This study was conducted at the Psychiatry Clinic of Ankara Bilkent City Hospital. A total of 45 patients diagnosed with BD and 54 patients diagnosed with MDD, who were determined to be in a depressive episode according to DSM-5 diagnostic criteria, were included. The participants consisted of patients aged 18-65 years, who provided informed consent and were either admitted for inpatient treatment or presented to the outpatient clinic. Sociodemographic and clinical data of the participants were recorded. Depression severity was assessed using the Hamilton Depression Rating Scale (HDRS), suicidal ideation was evaluated with the Beck Scale for Suicidal Ideation (BSSI), and hopelessness was measured using the Beck Hopelessness Scale (BHS). Current psychotropic medication use was compared between the groups.

In statistical analyses, the independent samples t-test or the Mann-Whitney U test was used for continuous variables, while the chi-square test or Fisher's exact test with Monte Carlo correction was applied for categorical variables. For item-level comparisons of the HDRS, Bonferroni correction was applied to control for multiple comparisons, and the adjusted significance level was set at ($p<0.003$).

Results:

When sociodemographic characteristics were evaluated, no significant differences were found between the BD and MDD groups in terms of age, sex, marital status, educational status, years of education, or employment status (all $p>0.05$). Similarly, no significant differences were observed between the groups in terms of smoking status or BMI (all $p>0.05$).

No significant difference was found between the groups in terms of the presence of a family history of psychiatric disorders (62.2% vs. 50%, $p=0.223$). However, the distribution of psychiatric disorders in family history differed significantly between the groups ($p=0.002$), with bipolar disorder (42.9%) and schizophrenia (25%) being more frequent in the BD group, and Major Depressive Disorder (50%) and anxiety disorders (34.6%) being more frequent in the MDD group.

When clinical characteristics were evaluated, no significant difference was found between the groups in terms of age at onset (median: 22 vs. 28.5 years, p=0.299). The duration of illness was significantly longer in the BD group than in the MDD group (median: 168 vs. 48 months, p=0.001). The duration of depressive episodes was significantly longer in the MDD group than in the BD group (median: 6 vs. 3 months, p=0.003). The number of hospitalizations and depressive episodes was significantly higher in the BD group than in the MDD group (median: 2 vs. 0 and 5 vs. 2, respectively; both p<0.001). Seasonal features were more frequent in the BD group than in the MDD group (26.7% vs. 5.6%, p=0.004). No significant differences were found between the groups in terms of psychotic features, dysthymia, hospitalization during the study period, or a history of suicide attempt prior to hospitalization (all p>0.05).

No significant difference was found between the groups in terms of the presence of a history of suicide attempts (40% vs. 27.8%, p=0.199). However, the number of suicide attempts was significantly higher in the BD group than in the MDD group (median: 3.5 vs. 1, p=0.001). No significant difference was observed between the groups in terms of the distribution of suicide attempt methods (p=0.365). In the BD group, the most common methods were drug overdose/poisoning and jumping from height (each 38.9%), followed by hanging (16.7%) and other methods (5.6%), whereas in the MDD group, drug overdose/poisoning was the most common method (60%), with jumping from height and hanging reported less frequently (each 13.3%).

The distribution of psychotropic medication use differed significantly between the groups (80% vs. 48.1%, p<0.001), with higher use in the BD group. Mood stabilizer use was markedly higher in the BD group (64.4% vs. 1.9%, p<0.001), whereas antidepressant use was higher in the MDD group (48.1% vs. 17.8%, p=0.002). Antipsychotic use was also more frequent in the BD group (62.2% vs. 13%, p<0.001). In addition, the olanzapine-equivalent dose was significantly higher in the BD group (median: 12 vs. 3.35 mg, p=0.001).

Total HDRS, BSSI, and BHS scores were similar between the groups (all p>0.05). Item-level analyses of the HDRS initially suggested differences between groups; however, these findings did not remain significant after Bonferroni correction for multiple comparisons.

Conclusions:

This study contributes to the challenge of differentiating bipolar depression from MDD by identifying clinically meaningful differences in illness course and symptom patterns.

The higher prevalence of a family history of bipolar disorder in the BD group is consistent with the literature indicating a strong genetic contribution to bipolar disorder. This finding suggests that family history may serve as an important clinical clue in the differential diagnosis, particularly in patients presenting with a first depressive episode. Similarly, previous studies have reported that a family history of bipolar disorder represents a distinguishing clinical feature between BD and MDD.

The longer total duration of illness, higher number of depressive episodes, and greater number of hospitalizations observed in the BD group suggest that bipolar disorder follows a more chronic and recurrent course, with increased illness burden and clinical severity, a greater need for hospitalization, and a poorer prognosis(3).

The shorter duration of depressive episodes observed in the BD group compared to the MDD group may be related to the episodic and fluctuating course of bipolar disorder, characterized by abrupt onset and relatively rapid resolution. Consistent with this finding, numerous studies have reported that BD is associated with shorter but more frequently recurring depressive episodes compared to MDD. In addition, regular clinical follow-up and engagement in psychoeducation among patients with BD may contribute to earlier recognition of depressive episodes and more prompt treatment-seeking behavior(4).

The higher prevalence of seasonal features in the BD group compared to the MDD group is consistent with the existing literature. This finding suggests that the presence of seasonal features may serve as a useful clinical clue in the differential diagnosis.

Although the presence of a history of suicide attempts appeared to be slightly higher in the BD group compared to the MDD group, this difference did not reach statistical significance. However, the number of past suicide attempts was significantly higher in the BD group than in the MDD group.

Although no statistically significant difference was found between the groups in terms of the distribution of suicide attempt methods, a closer examination revealed that more lethal methods, such as jumping from height and hanging, were more frequently used in the BD group, whereas less lethal methods, such as drug overdose/poisoning, were more predominant in the MDD group(5).

Taken together, these findings suggest that BD may be characterized not only by the presence of suicidal behavior but also by a more recurrent pattern and a tendency toward more severe and potentially lethal suicide attempts.

When total HDRS scores were evaluated, no significant difference was found between the BD and MDD groups, indicating comparable levels of depression severity. Although BD is generally associated with more severe depressive presentations than MDD, the lack of difference observed in our study may be attributed to the limitations of the HDRS. While the HDRS is a valid and reliable instrument for MDD, it may not adequately capture atypical features and may therefore fail to fully reflect the distinctive clinical characteristics of depression in BD.

In the initial item-level analysis of the HDRS, psychomotor retardation, reduced interest in work and activities, genital symptoms, and impaired insight were more prominent in the BD group, whereas difficulty initiating sleep and somatic anxiety were more pronounced in the

MDD group. However, these differences did not remain significant after Bonferroni correction for multiple comparisons. This suggests that individual depressive symptoms may not represent robust distinguishing features between BD and MDD. The loss of significance may also reflect the conservative nature of the correction method, which can increase the risk of type II error (false negative results). Therefore, further studies with larger sample sizes are warranted.

Overall, these findings highlight the importance of integrating longitudinal clinical course, family history, and symptom patterns in the differential diagnosis of BD and MDD, rather than relying solely on cross-sectional symptom assessments.

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Table-1 Family History

	BD	MDD	Total	Test Statistic	P
Family history of psychiatric disorders[a] Number (%)					
Present	28 (%62,2)	27 (%50)	55 (%55,6)	1,485	0,223
Absent	17 (%37,8)	27(%50)	44 (%44,4)		
Distribution of psychiatric disorders in family history[b] Number (%)					
Bipolar Disorder	12 (%42,9)	2 (%7,7)	14(%25,9)	16,026	**0,002**
Major Depressive Disorder	7 (%25)	13 (%50)	20 (%37)		
Anxiety Disorders	2 (%7,1)	9 (%34,6)	11(%20,4)		
Schizophrenia	7 (%25)	2 (%7,7)	9 (%16,7)		

[a] Chi-square test. [b] Fisher's exact test with Monte Carlo correction

Table-2 Comparison of Clinical Characteristics

Clinical Characteristics	BD	MDD	Test Statistic	P
Age at Illness Onset[a] Median (Min-Maks)	22 (10-58)	28,5 (1460)	1067,500	0,299
Duration of Illness (months)[a] Median (Min-Maks)	168 (12324)	48 (2-396)	727,500	**0,001**
Duration of Depression(months)[a] Median (Min-Maks)	3 (1-24)	6 (1-24)	798,000	**0,003**
Number of Hospitalizations'[1] Median (Min-Maks)	2 (0-13)	0 (0-4)	592,500	**<0.001**
Number of Depressive Episodes [a] Median (Min-Maks)	5 (1-17)	2 (1-12)	595,500	**<0.001**
Seasonal Pattern[b] Number (%)	12 (%26,7)	3 (%5,6)	8,509	**0,004**
Psychotic Features[c] Number (%)	4 (%8,9)	1 (%1,9)	2,535	0,174
Dysthymia[b] Number (%)	8 (%17,8)	7 (%13)	0,443	0,58
History of Suicide Attempts'[3] Number (%)	18 (%40)	15 (%27,8)	1,650	0,199
Number of Suicide Attempts[a] Median (Min-Maks)	3,5 (1-8)	1 (1-3)	44,500	**0,001**
Hospitalization During The Study Period[b] Number (%)	13 (%28,9)	14 (%25,9)	0,109	0,822
Suicide Attempt Before Hospitalization Number (%)	4 (%30,8)	3 (%20)	0,431	0,670

[a]Mann-Whitney U test; [b]Chi- square test; Fisher's exact test with Monte Carlo correction

Table-3 Comparison of the HDRS-17 Total Score and Item Scores

HDRS- 17 Item Scores[a] Median (Min-Max)	BD	MDD	Test Statistic	p
Depressed Mod	3 (2-4)	3 (2-4)	998,000	0,098
Feelings of Guilt	2 (0-4)	2 (0-3)	1012,500	0,120
Suicide	2 (0-4)	1,5 (0-4)	1100,500	0,406
Early Insomnia	1 (0-2)	1 (0-2)	870,000	0,008
Middle Insomnia	1 (0-2)	1 (0-2)	1187,000	0,834
Late Insomnia	0 (0-2)	0 (0-2)	1112,500	0,412
Work and Activities	3 (1-4)	2 (1-4)	825,000	0,003
Retardation	1 (0-3)	1 (0-3)	846,000	0,006
Agitation	0 (0-2)	0 (0-2)	11,610	0,654
Psychic Anxiety	3 (0-4)	2 (0-4)	1202,000	0,922
Somatic Anxiety	1 (0-3)	2 (0-4)	803,000	0,003
Gastrointestinal Symptoms	1 (0-2)	1 (0-2)	1180,500	0,789
General Somatic Symptoms	2 (0-2)	2 (1-2)	1183,000	0,793
Genital Symptoms	2 (0-3)	1,5 (0-3)	902,500	0,014
Hypochondriasis	0 (0-2)	0 (0-4)	1094,000	0,325
Loss of Weight	0 (0-2)	0 (0-2)	1187,500	0,832
Insight	0 (0-2)	0 (0-1)	1014,500	0,043
HDRS-17 Total[b] Mean ± SD	21,89 ± 6,43	21,11 ± 5,95	0,624	0,534

[a]Mann-Whitney U test; [b] Independent samples t-test

For item-level comparisons of the HDRS, Bonferroni correction was applied to control for multiple comparisons, and the adjusted significance level was set at $p<0.003$.

0234 -The Relationship Between Home Internet Use, Familial Sociodemographic Characteristics, and Clinical Severity in Children and Adolescents with ADHD

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders in childhood and adolescence, characterized by inattention, impulsivity, and hyperactivity that impair academic and social functioning. In recent years, increasing exposure to digital technologies has emerged as an important environmental factor potentially influencing the clinical presentation of ADHD.

Children with ADHD may be particularly vulnerable to excessive digital media use due to difficulties in impulse control and time management. Previous studies have linked increased screen exposure to greater ADHD symptom severity, sleep problems, and impaired functioning. However, most research has focused primarily on the child's individual screen time, while the broader familial digital environment and parental technology use have received less attention.

Parental digital behaviors may influence children both through modeling and through the structuring of the home digital environment. Sociodemographic factors such as parental education, family structure, and socioeconomic status may also shape digital media exposure and behavioral outcomes.

The present study aimed to examine the relationship between home internet use habits, familial sociodemographic characteristics, and clinical severity in children and adolescents diagnosed with ADHD. Understanding how family-level digital behaviors relate to ADHD severity may contribute to more comprehensive clinical management strategies.

Methods

Participants and Study Design

This retrospective descriptive study included children and adolescents diagnosed with ADHD who were followed at the Child and Adolescent Psychiatry Outpatient Clinic of Balikesir Ataturk City Hospital between 2023 and 2025. Participants were aged 7-17 years and met DSM-5 diagnostic criteria for ADHD. Patients with incomplete medical records or comorbid neurodevelopmental disorders such as autism spectrum disorder, intellectual disability, or psychotic disorders were excluded. Clinical, sociodemographic, and digital use data were obtained from electronic medical records and transferred to a standardized data collection form.

Measures

Clinical severity was evaluated using the Clinical Global Impression-Severity (CGI-S) scale. The CGI-S is a clinician-rated 7-point scale ranging from 1 (not ill) to 7 (extremely ill) and reflects overall clinical severity. Sociodemographic variables included age, sex, parental age, parental education levels, family structure, and family socioeconomic status.

Digital life variables included age at first internet use, daily internet use duration, daily digital gaming time, parental internet use, digital device ownership, and the presence of household rules regarding internet use.

Statistical Analysis

Statistical analyses were conducted using IBM SPSS Statistics. Continuous variables were expressed as mean ± standard deviation, and categorical variables were presented as frequencies and percentages.

Pearson correlation analyses were performed to examine associations between CGI-S scores and familial and digital variables. Hierarchical multiple linear regression analyses were conducted to identify independent predictors of clinical severity. Statistical significance was set at $p < 0.05$.

Results

A total of 81 children and adolescents with ADHD were included in the study. The mean age was 9.77 ± 3.00 years, and 70.4% of the participants were male. The mean CGI-S score was 3.42 ± 1.19, indicating moderate clinical severity.

The mean age at first internet use was 5.97 ± 2.64 years. Participants reported a mean daily internet use of 3.73 ± 1.96 hours and daily digital gaming time of 3.13 ± 2.04 hours.

Correlation analyses showed significant associations between CGI-S scores and several familial and digital variables. Paternal age demonstrated a moderate positive correlation with CGI-S scores ($r = 0.513$, $p < 0.001$). Age at tablet acquisition showed a negative correlation with CGI-S ($r = -0.358$, $p = 0.037$), indicating that earlier device acquisition was associated with greater clinical severity. Maternal daily internet use ($r = 0.228$, $p = 0.042$) and paternal daily internet use ($r = 0.357$, $p = 0.001$) were also positively correlated with CGI-S scores.

Hierarchical regression analysis revealed that the inclusion of digital life variables significantly increased the explanatory power of the model, accounting for 59.5% of the variance in CGI-S scores. In the final model, paternal daily internet use emerged as the strongest predictor of clinical severity ($\beta = 0.572$, $p = 0.001$). Child age also showed a significant association with CGI-S scores ($\beta = 0.439$, $p = 0.047$). Earlier tablet acquisition showed a borderline negative association with severity ($\beta = -0.414$, $p = 0.053$).

Discussion

This study demonstrated that children and adolescents with ADHD were exposed to digital technology at relatively early ages and exhibited high levels of daily internet use. The findings indicate that digital life characteristics are associated with clinical severity in ADHD.

Notably, paternal daily internet use emerged as the strongest predictor of clinical severity, highlighting the potential influence of parental digital behaviors on the home digital environment and child outcomes. These results suggest that parental modeling of digital media use may play an important role in shaping children's behavioral patterns.

Earlier digital device acquisition was also associated with greater clinical severity, supporting previous research suggesting that early screen exposure may affect attention development. However, the borderline statistical significance indicates that digital exposure likely interacts with broader familial and environmental factors.

Overall, the findings emphasize the importance of considering family digital habits during clinical assessment and management of ADHD. Family-based strategies focusing on parental digital behavior and structured media use may contribute to improved clinical outcomes.

Conclusion

Home digital environment and parental internet use appear to be associated with ADHD clinical severity. Evaluating and regulating family-level digital media habits may represent an important component of comprehensive ADHD management.

0242 - Dose-Dependent Fluoxetine-Associated Amenorrhea Without Hyperprolactinemia: A Case Report

Abstract

Background: Menstrual disturbances during selective serotonin reuptake inhibitor (SSRI) treatment may be underrecognized in psychiatric follow-up. Although SSRI- related menstrual effects are often discussed via hyperprolactinemia, clinically meaningful cycle disruption can occur despite prolactin within the reference range.

Case report: A 32-year-old woman (G1P1) who developed dose-dependent menstrual disruption during fluoxetine treatment for a first episode of major depressive disorder. After initiating fluoxetine 20 mg/day, she experienced delayed menses and hypomenorrhea; following dose escalation to 40 mg/day, she developed secondary amenorrhea with three missed cycles. Work-up excluded pregnancy and revealed normal prolactin and unremarkable endocrine, gynecologic, and pituitary evaluation. Bleeding resumed as spotting 24 days after discontinuation.

Conclusion: This brief report highlights a dose-related pattern of fluoxetine- associated menstrual suppression progressing to amenorrhea without hyperprolactinemia. Clinicians should proactively inquire about menstrual changes after SSRI initiation or dose escalation and consider medication contribution while completing a standard secondary amenorrhea work-up.

Keywords: Fluoxetine, Menstrual disorders, Secondary amenorrhea, Selective serotonin reuptake inhibitors (SSRIs), Prolactin

Intoduction

Selective serotonin reuptake inhibitors (SSRIs) are widely prescribed as first-line pharmacotherapy for major depressive disorder, yet menstrual disturbances such as delayed menses, hypomenorrhea, oligomenorrhea, and amenorrhea may remain underrecognized unless systematically assessed. In routine clinical settings, antidepressant exposure has been associated with menstruation disorders, suggesting that these symptoms are clinically relevant beyond isolated anecdotes and may influence adherence, distress, and treatment decisions [1]. When secondary amenorrhea emerges, a structured diagnostic approach is recommended—beginning with pregnancy exclusion and followed by targeted endocrine and gynecologic evaluation guided by history and examination [2]. Within this framework, documenting the temporal relationship between menstrual changes and antidepressant initiation or dose escalation is essential for identifying potentially iatrogenic contributors. Recent

case-based literature also draws attention to unexpected menstrual cycle effects associated with fluoxetine, including improvement after medication changes [3]. Here, we describe a dose-dependent case of fluoxetine-associated menstrual suppression progressing to amenorrhea without hyperprolactinemia.

Case Presentation

A 32-year-old woman (G1P1), depressive symptoms began at approximately postpartum month 20 and persisted for about 8 weeks prior to psychiatric examination; during the 3 weeks immediately preceding presentation, symptoms met DSM-5 criteria for a moderate depressive episode, including anhedonia, low energy, avolition, irritability/low frustration tolerance, impaired concentration, insomnia, and decreased appetite. She had no past psychiatric history, no medical comorbidities, and used no medications or supplements. She did not use tobacco or alcohol, consumed approximately one cup of coffee daily, and was not breastfeeding. Menstrual cycles had been regular (28-30 days) since postpartum month 6 and remained regular until antidepressant initiation. Contraception consisted of condom use.

Fluoxetine 20 mg/day was initiated. After approximately 3 weeks on 20 mg/day, due to insufficient response, the dose was increased to 40 mg/day. The next expected menses (anticipated approximately 3 weeks later) did not occur, and ongoing missing three consecutive cycles, and it was accepted she developed secondary amenorrhea. On questioned her history, she reported that menstrual delay and hypomenorrhea had already developed during the first cycle after initiating fluoxetine at 20 mg/day.

A structured work-up for secondary amenorrhea was performed in line with standard diagnostic approaches [2]. Pregnancy was excluded (P-hCG 0.00 mlU/mL). Prolactin was within the laboratory reference range (16.39 ng/mL; reference 3.34-26.72), and galactorrhea was absent. Thyroid function was within reference limits (TSH 2.227 mlU/mL; free T4 0.69 ng/dL). Gonadotropins were not suggestive of ovarian insufficiency (FSH 4.10; LH 5.72). Estradiol was 52 pg/mL (cycle day indepented). Pelvic ultrasonography was unremarkable, and pituitary MRI showed no abnormalities. Weight was stable (170 cm, 62 kg; BMI ~21.5; ~1 kg loss overall). She reported no excessive exercise and no symptoms suggestive of an eating disorder.

Because of these complaint, the patient was re-evaluated approximately 4 months after starting fluoxetine treatment, and improvement in depressive symptoms was observed (HAM-D 9). Fluoxetine was discontinued and psychotherapy continued. Twenty-four days after discontinuation, vaginal bleeding resumed as spotting. At the time of reporting (~1 month after stopping fluoxetine), a full menstrual period had not yet occurred, but the next expected cycle was not yet overdue and follow-up was ongoing.

This case is notable for a dose-related pattern and a clinically meaningful temporal association: partial menstrual disruption at 20 mg/day followed by amenorrhea after escalation to 40 mg/day, with early bleeding resumption after discontinuation. Such a graded sequence strengthens the plausibility of a medication-associated effect and reinforces the practical point that menstrual symptoms may be overlooked unless specifically queried—an issue supported by real-world clinical data linking antidepressant use to menstruation disorders [1]. The case also illustrates an important clinical nuance: SSRI-related menstrual effects are often discussed via hyperprolactinemia, yet this patient's prolactin remained within the reference range, and no structural pituitary pathology was identified. Fluoxetine-related menstrual cycle disruption has been highlighted in recent case-based literature, suggesting that clinically meaningful cycle changes can occur during treatment and may improve following discontinuation or treatment modification [3]. Earlier case-based discussions have proposed neuroendocrine mechanisms and heterogeneity in phenotype, implying that reproductive effects may arise through pathways not invariably captured by elevated prolactin alone [4,5]. Although this brief report cannot fully delineate the underlying pathophysiology, prior reports and reviews suggest that SSRI-related menstrual changes may occur even in the absence of hyperprolactinemia, supporting the plausibility of a normoprolactinemic presentation [6].

From a clinical standpoint, the work-up was consistent with recommended approaches to secondary amenorrhea [2]. Pregnancy was excluded, thyroid and gonadal indices were evaluated, and imaging was used to assess pelvic and pituitary pathology, all of which were unremarkable. Functional hypothalamic amenorrhea can be considered in the context of stress and mood symptoms; however, the patient's stable BMI, lack of significant weight loss, denial of excessive exercise/eating- disorder symptoms, and the dose-linked temporal sequence weigh in favor of a medication contribution. The principal limitation is the short follow-up after discontinuation (~1 month): spotting at 24 days represents an early dechallenge signal, but full normalization of bleeding volume and cycle regularity has not yet been documented. Longer follow-up would strengthen the clinical narrative by confirming return to baseline cycle pattern.

Conclusion

This brief report describes a dose-dependent pattern of fluoxetine-associated menstrual disruption progressing from delayed menses/hypomenorrhea at 20 mg/day

to secondary amenorrhea at 40 mg/day, with early bleeding resumption after discontinuation, despite normal prolactin and negative endocrine/gynecologic and pituitary evaluation. Clinicians should proactively inquire about menstrual changes after SSRI initiation or dose escalation and consider medication contribution while completing a standard secondary amenorrhea work-up [2]. Continued follow-up is warranted to confirm full normalization of cycle regularity and bleeding pattern.

Consent / Ethics

Written informed consent was obtained from the patient for presentation/publication of this case in a scientific meeting. Ethics approval was not required for a single case report according to local regulations and institutional policy.

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0248

Objective

Biotinidase deficiency (BD) is a rare but treatable metabolic disorder that may present with neurological and neuropsychiatric symptoms. In contrast, epilepsy is a chronic neurological condition frequently associated with high rates of psychiatric comorbidity. Although both conditions may involve cognitive and behavioral disturbances, comparative data regarding their psychiatric profiles and psychopharmacological treatment patterns are limited.

Given the reversible nature of BD with early treatment and the chronic course of epilepsy, differences in psychiatric burden and medication use may have important clinical implications. This study aimed to compare sociodemographic characteristics, psychiatric diagnoses, and psychotropic medication use between children with BD and those with epilepsy.

Methods

This retrospective study included children and adolescents aged 6-18 years who were followed in a child and adolescent psychiatry outpatient clinic of a state hospital over three years. Patients were classified into two groups based on diagnoses documented in official medical records: biotinidase deficiency (BD) and epilepsy. All diagnoses were previously established by pediatric specialists during routine clinical follow-up. Psychiatric diagnoses were extracted from medical records and had been established by a child and adolescent psychiatrist based on clinical evaluation in accordance with DSM-5 criteria.

Sociodemographic variables (age, sex), psychiatric diagnoses, and psychotropic medication use were extracted from medical records.

Psychotropic medications were categorized as antipsychotics, selective serotonin reuptake inhibitors (SSRIs), non-SSRI antidepressants, and stimulants. The presence of any psychotropic treatment and polypharmacy were also recorded.

Statistical analyses were performed using appropriate non-parametric methods due to the distribution characteristics of the data. Continuous variables were compared using the Mann- Whitney U test, while categorical variables were analyzed using Pearson chi-square or Fisher's exact test. Fisher's exact test results were prioritized due to small expected cell counts. A p- value < 0.05 was considered statistically significant.

Results

A total of 61 patients were included in the study, comprising 18 patients with BD and 43 with epilepsy. The median age was significantly lower in the BD group compared to the epilepsy group. There was no significant difference between groups in terms of sex distribution.

The overall prevalence of psychiatric diagnoses was high in both groups, with no statistically significant difference between BD and epilepsy. Similarly, the rates of attention- deficit/hyperactivity disorder (ADHD) and major depressive disorder did not differ significantly.

However, intellectual disability was significantly more prevalent in the epilepsy group compared to the BD group.

In terms of psychopharmacological treatment, antipsychotic use was significantly higher in the epilepsy group, whereas no patients in the BD group were receiving antipsychotics. Although polypharmacy was observed only in the epilepsy group, this difference did not reach statistical significance.

There were no significant differences between groups in terms of overall psychotropic medication use, SSRI treatment, or stimulant use.

In secondary analyses, stimulant use was found to be strongly associated with an ADHD diagnosis, supporting the internal consistency of the clinical data.

Discussion

This study demonstrates that while psychiatric comorbidity is highly prevalent in both BD and epilepsy, the patterns of neurodevelopmental burden and psychopharmacological treatment differ between the two conditions.

The significantly higher rate of intellectual disability observed in the epilepsy group is consistent with previous literature indicating that epilepsy, particularly when early-onset or severe, is associated with greater neurodevelopmental impairment (Reilly et al., 2014). In contrast, the lower rate of intellectual disability in the BD group may reflect the beneficial effects of early diagnosis and biotin treatment, which can prevent or mitigate long-term neurological sequelae (Wolf, 2017).

From a psychopharmacological perspective, the higher rate of antipsychotic use in children with epilepsy is noteworthy. This finding likely reflects a greater need to manage behavioral dysregulation, irritability, or comorbid neurodevelopmental conditions in this population. However, given that certain psychotropic medications, including antipsychotics, may lower seizure threshold, their use in epilepsy requires careful clinical consideration (Alper et al., 2007; Kanner, 2016).

In contrast, the absence of antipsychotic use in the BD group suggests a relatively lower severity of behavioral disturbances requiring such interventions. The similar rates of stimulant and antidepressant use across groups indicate that common psychiatric conditions such as ADHD and mood disorders are present in both populations and are managed in line with standard clinical practice. Additionally, the strong association between stimulant use and ADHD diagnosis observed in our study is consistent with previous literature highlighting the high comorbidity of ADHD in epilepsy and supporting the clinical relevance of stimulant treatment in this population (Dunn et al., 2016).

These findings highlight important clinical implications. First, children with epilepsy may require more intensive psychiatric and psychopharmacological management, particularly for neurodevelopmental and behavioral complications. Second, early diagnosis and treatment in BD may play a protective role in reducing long-term psychiatric morbidity.

Several limitations should be considered. The retrospective design limits causal inferences, and the relatively small sample size may reduce statistical power. Additionally, the study was conducted in a single center, which may limit generalizability.

Despite these limitations, this study provides novel comparative data on psychiatric comorbidity and psychotropic treatment patterns in BD and epilepsy, contributing to a better understanding of their clinical management.

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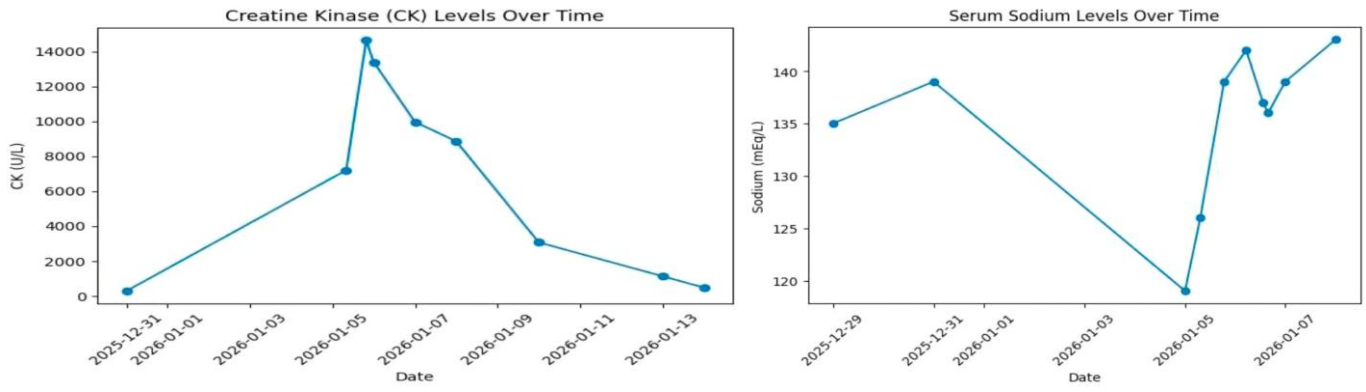
0250 - Neuroleptic Malignant Syndrome Masked By Hyponatremıa: A Schizophrenia Case With Diagnostic Challenges

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Introduction

Neuroleptic malignant syndrome (NMS) is a rare but potentially life-threatening emergency associated with antipsychotic use. Hyponatremia, on the other hand, is frequently observed in patients with schizophrenia and is associated with psychotropic medications, syndrome of inappropriate antidiuretic hormone secretion (SIADH), and low body mass index. It may lead to severe complications such as seizures and altered consciousness. In this case report, we present a patient with a long-standing diagnosis of schizophrenia who developed NMS in the presence of severe hyponatremia, which initially masked the clinical presentation.



Case

A 50-year-old male with a 30-year history of schizophrenia was followed by a Community Mental Health Center. Although family members reported regular medication use, adherence was partial, with antipsychotics administered approximately 5-6 days per week. His maintenance regimen included oral paliperidone 6 mg/day, olanzapine 20 mg/day, and monthly paliperidone palmitate depot injection (150 mg).

He was admitted with acute psychotic exacerbation characterized by persecutory delusions, aggressive behavior, and homicidal ideation. Significant psychosocial stressors included witnessing his father's fatal fall in May 2025 and experiencing romantic rejection one week prior to admission. Baseline laboratory findings revealed normal serum sodium levels (Na 139 mEq/L), iron deficiency (ferritin 14 ng/mL), anemia (Hb 10-11 g/dL), and low body mass index (17.6 kg/m^2). Oral paliperidone was increased to 12 mg/day during hospitalization, while the scheduled depot injection was withheld.

On hospital day six, the patient developed a generalized tonic—clonic seizure lasting approximately two minutes, followed by fluctuating consciousness. Emergency laboratory evaluation demonstrated severe hyponatremia (Na 119 mEq/L), and hypertonic saline therapy was initiated. Shortly thereafter, episodic fever exceeding 38°C, marked jaw and limb rigidity, tremor, and altered mental status were observed. Concurrently, CK levels increased to approximately 7000 U/L. Based on high cumulative antipsychotic exposure, hyperthermia, rigidity, altered consciousness, and CK elevation, NMS was diagnosed, and the patient was transferred to the intensive care unit. During intensive care follow-up, CK levels rose to 14,000 U/L, accompanied by rhabdomyolysis, hyperkalemia (6.8 mEq/L), hypoglycemia (32 mg/dL), elevated myoglobin (>3000 ng/mL), and autonomic instability. All antipsychotic medications were discontinued, and intensive supportive treatment was initiated. Metabolic abnormalities and muscle enzyme levels gradually resolved.

Following stabilization, the patient was transferred back to the psychiatric ward, where he was alert, cooperative, and oriented, with marked improvement in psychotic symptoms.

Conclusion

This case underscores the critical importance of monitoring electrolyte balance and cumulative antipsychotic exposure in patients with schizophrenia. Low body mass index, iron deficiency, antipsychotic polypharmacy, and acute psychosocial stressors may substantially increase vulnerability to NMS.

The temporal and causal relationship between severe hyponatremia and NMS in this case remains unclear. Hyponatremia may have precipitated NMS through its neurological effects, while NMS itself may have contributed to worsening electrolyte imbalance. Alternatively, both conditions may have developed concurrently due to shared risk factors. Severe hyponatremia may both precipitate and obscure NMS, delaying diagnosis and increasing morbidity. Early recognition and prompt multidisciplinary management are essential to improving outcomes in patients presenting with overlapping psychiatric and medical emergencies.

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0251 - Unbroken Dyad: One Self, Two Bodies - A Shared Suicide Attempt in Dizygotic Twins and a Review of the Literature

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Introduction: Shared suicidal behavior in twins is an exceptionally rare and clinically compelling phenomenon. While suicidality has a well-established familial and partly heritable basis, twin relationships also constitute a distinctive interpersonal environment in which emotional contagion, enmeshment, and impaired separation-individuation may intensify risk. Although concordance for suicidal behavior is classically higher in monozygotic than dizygotic twins, dizygotic twins may nevertheless be highly vulnerable when intense emotional interdependence and mutually reinforcing cognitive patterns are present. In adolescence, suicidal crises are often shaped by relational stress, perceived abandonment, and disruption of core attachment bonds. In certain twin dyads, the co-twin may function as a central regulator of affect and identity, such that separation is experienced not merely as distressing, but as profoundly destabilizing. This report presents a shared suicide attempt in 14-year-old dizygotic twin adolescents following classroom separation and discusses the case within a developmental and relational framework. Written informed consent for publication of anonymized clinical information was obtained from the parents/legal guardians, and assent was obtained from both adolescents.

Case Presentation: The patients were 14-year-old dizygotic twin adolescents who presented on October 10, 2025, with severe exam anxiety. During the clinical interview, it became evident that a major precipitating stressor had occurred approximately one week earlier: the twins had been assigned to separate classrooms. This transition was experienced by both adolescents as a profound emotional rupture associated with fear, helplessness, and inability to function apart. Shortly thereafter, both ingested nine tablets of bilastine together in a joint self-poisoning episode and were brought to the emergency department. At psychiatric presentation, both continued to express suicidal ideation to their mother, indicating that the act was not simply an impulsive ingestion of low lethality, but part of an active and ongoing suicidal process. Sertraline 25 mg/day was initiated at the first evaluation because of persistent anxiety, marked emotional distress, and continuing suicidal ideation. At follow-up, suicidal ideation remained active, and both twins described intrusive self-harm thoughts involving needle insertion, swallowing foreign objects such as beads, and compressing arteries. Given the persistence and severity of suicide risk, psychiatric hospitalization was planned, and the patients were referred to an external center, where they were hospitalized for two days. During this period, risperidone was initiated but discontinued adverse effects including dizziness and diplopia. In January, aripiprazole 2.5 mg/day was started and later increased to 5 mg/day, and the twins have continued to be followed in an outpatient setting.

A structured cognitive behavioral therapy (CBT) program was adapted to the specific relational characteristics of the twin dyad. The intervention included psychoeducation on anxiety, suicidality, and the cognitive model, with particular emphasis on emotional recognition, bodily awareness, and early detection of escalating distress. Shared maladaptive cognitions related to separation, abandonment, and academic pressure were identified and systematically addressed through guided questioning and the development of alternative appraisals. Core beliefs such as "we cannot cope apart" and "being separated means being unsafe" were directly targeted. Beyond symptom reduction, a central therapeutic goal was to reduce pathological overdependence by strengthening individual identity, emphasizing personal differences, and fostering separate self-representations. To address separation-related anxiety, imagery-based desensitization and graded exposure to separate settings were implemented, including behavioral tasks in which the twins engaged in different activities simultaneously. Grounding techniques and individualized coping strategies were also introduced. Given the history of a shared suicide attempt, structured safety planning and close parental involvement were integrated throughout treatment. Ongoing follow-up focuses on monitoring suicidal ideation, enhancing affect regulation, reducing enmeshment, and supporting healthier separation-individuation.

Conclusion: This case underscores that suicidality in dizygotic twins may arise not only from individual psychopathology, but also from dyadic mechanisms such as emotional contagion, cognitive synchronization, and

identity fusion. In this pair, classroom separation appears to have functioned as a powerful developmental and relational stressor that destabilized both adolescents and precipitated a joint suicidal act. The case highlights the importance of extending suicide risk assessment in twins beyond conventional individual-level factors to include the structure of the twin bond itself, particularly overdependence, shared catastrophic beliefs, and vulnerability to separation. From a clinical perspective, individually focused treatment alone may be insufficient when distress is mutually amplified within the dyad. Interventions that combine active risk management with CBT adapted to twin-specific relational dynamics may be especially valuable in reducing suicidal risk while promoting healthier developmental differentiation. Recognition of twin-specific vulnerabilities may improve prevention, formulation, and treatment planning in rare but clinically high-risk presentations of shared suicidality.

Keywords: Dizygotic twins, shared suicide attempt, shared suicidality, adolescence, cognitive behavioral therapy

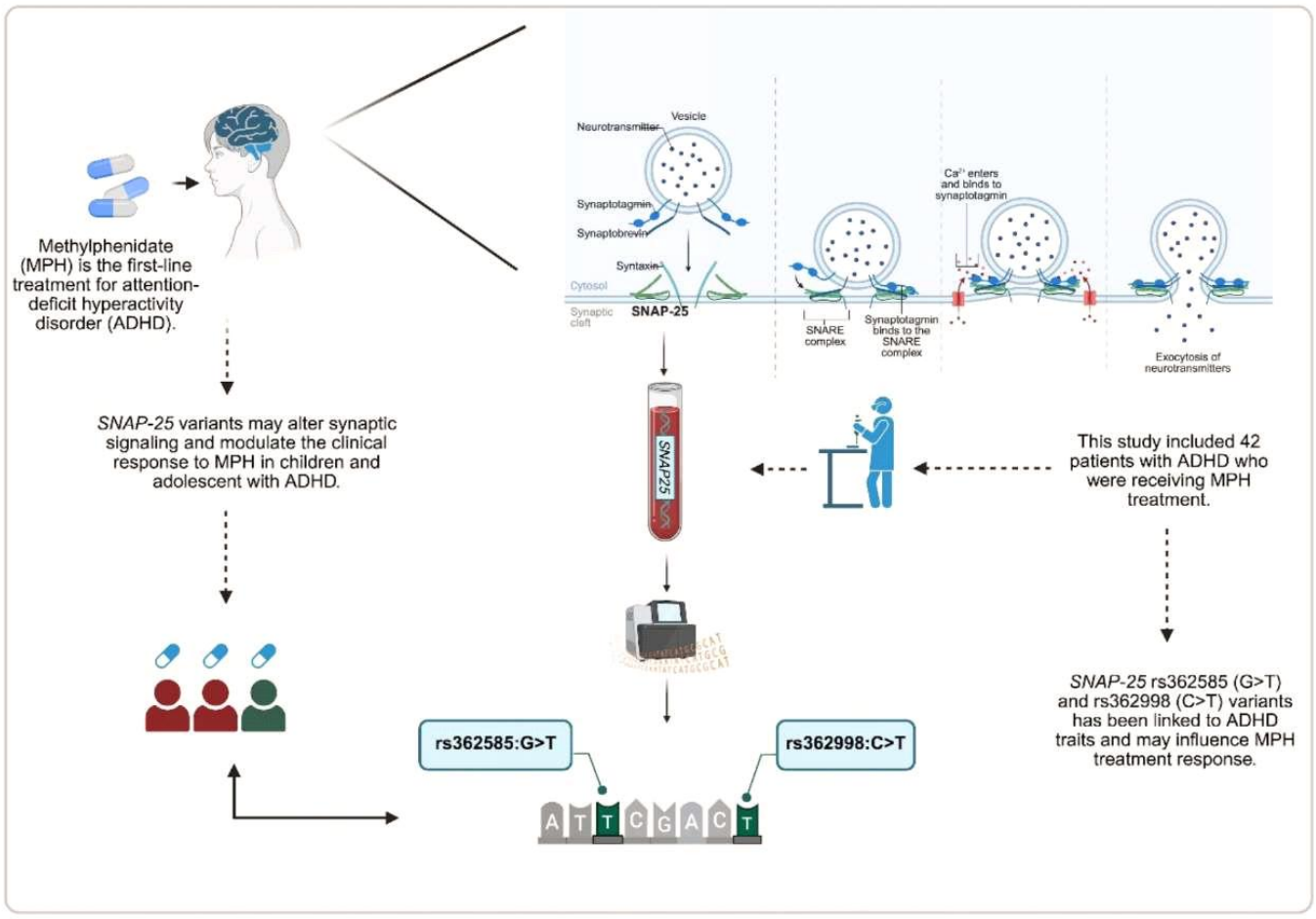
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0253 - Potential Association Between *SNAP-25* rs362998:C>T and Methylphenidate Treatment Response in ADHD: A Next-Generation Sequencing Study

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Graphical abstract. This graphic summary highlights the role of SNAP-25 gene polymorphisms in methylphenidate (MPH) treatment response and its potential impact on attention regulation in attention-deficit/hyperactivity disorder (ADHD), outlining a study investigating this association.

Objectives

Methylphenidate (MPH) is a first-line treatment for attention-deficit/hyperactivity disorder (ADHD). MPH blocks dopamine (DA) and norepinephrine (NE) transporters, increasing synaptic catecholamine levels. Neurotransmitter

release depends on presynaptic vesicle docking and fusion mediated by Synaptosomal-Associated Protein, 25 kDa (SNAP-25), a Target Soluble NSF Attachment Protein Receptor (SNARE) protein encoded by the SNAP-25 gene. This study aimed to investigate whether genetic variations in the SNAP-25 gene contribute to individual variability in the clinical response to MPH treatment among pediatric patients with ADHD.

Methods

Forty-two children and adolescents diagnosed with ADHD according to DSM-5-TR criteria [1] were prospectively treated with MPH for two months. Clinical response was assessed using the Conners'

Parent Rating Scale (CPRS) and the Turgay DSM-Based Disruptive Behavior Disorders Rating Scale (T-DBDRS). Targeted next generation sequencing (tNGS) was used to sequence the exons of the SNAP- 25 gene in the study cohort.

Results

Three common variants in SNAP-25 (rs363006, rs362585, rs362998) were analyzed. According to the T-DBDRS scale, no statistically significant association between SNAP-25 variants and treatment response was observed. Based on CPRS-defined outcomes, a statistically significant association was observed between the rs362998:C>T polymorphism and the response to MPH treatment in patients with ADHD (Fig .1.). In the genotypic model, rs362998:C>T polymorphism showed a significant association with treatment response ($p = 0.026$, OR = 9.6, 95% CI: 1.388-111.5, Fig.1a.), with the CT genotype being more frequent among responders (34.78%) than non-responders (5.26%). In the dominant model, CT+TT genotypes were significantly more frequent among the responsive group than the non- responsive group ($p = 0.027$; OR = 9.60; 95% CI: 1.39-111.5, Fig.1b.). The allelic model showed a higher frequency of the T allele in the responsive group ($p = 0.036$; OR = 7.79; 95% CI: 1.05-88.5, Fig. 1c.). In addition, a novel 3'UTR variant (c.*54T>C; chr20:10306251 T>C) was identified (Table 1).

Discussion

ADHD is a chronic neurodevelopmental condition for which MPH remains the prescribed first-line pharmacological treatment [2]. Despite its well-established efficacy, a substantial proportion of patients exhibit variable therapeutic response and tolerability, highlighting the need to better understand the biological factors underlying this heterogeneity. The SNARE complex, consisting of SNAP-25, Syntaxin-1A, and VAMP, mediates neurotransmitter release by bringing vesicle and plasma membranes together. Calcium influx activates synaptotagmin, triggering membrane fusion [3]. Given its central role, SNAP-25 has been widely studied in psychiatric disorders, with variants proposed to affect synaptic efficiency and neuronal signaling. In ADHD, SNAP-25 is a key candidate gene due to its involvement in dopaminergic and glutamatergic pathways. Several studies have reported associations between SNAP- 25 polymorphisms and ADHD [4-5]. In this study, we evaluated the association of common SNAP-25 variants with MPH treatment response, defined by improvements in CPRS and T-DBDRS scores. Therefore, the present study evaluated the association between common SNAP-25 gene variants and MPH treatment outcomes, with treatment response defined as improvement in CPRS and T-DBDRS scores.

Previous studies have shown associations between SNAP-25 variants and ADHD, but common variants such as rs3746544 and rs1051312 were rare in our population. We evaluated three SNAP-25 variants (rs363006, rs362585, rs362998) for their association with MPH treatment response. The rs362998:C>T variant was associated with improved response in CPRS-defined analysis, with T allele carriers more frequent among responders. Previous studies have reported associations between SNAP-25 variants, including rs363006 and rs362998, and ADHD. Over-transmission of the C allele of rs362998 has been observed in ADHD families [6], while rs363006 has been linked to ADHD in an independent cohort [7]. In the present study, however, no significant association between these variants and treatment response was detected based on T-DBDRS scores.

In conclusion, this exploratory study found that the SNAP-25 rs362998:C>T variant, with the T allele more frequent among responders, was associated with improved MPH response in ADHD, suggesting that genetic variation in this synaptic regulator may influence treatment outcomes and warrants further investigation in larger cohorts.

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Figure 1. Association between the SNAP25 rs362998:C>T and rs362585:G>T polymorphisms and methylphenidate (MPH) treatment response in ADHD patients assessed using the CPRS.

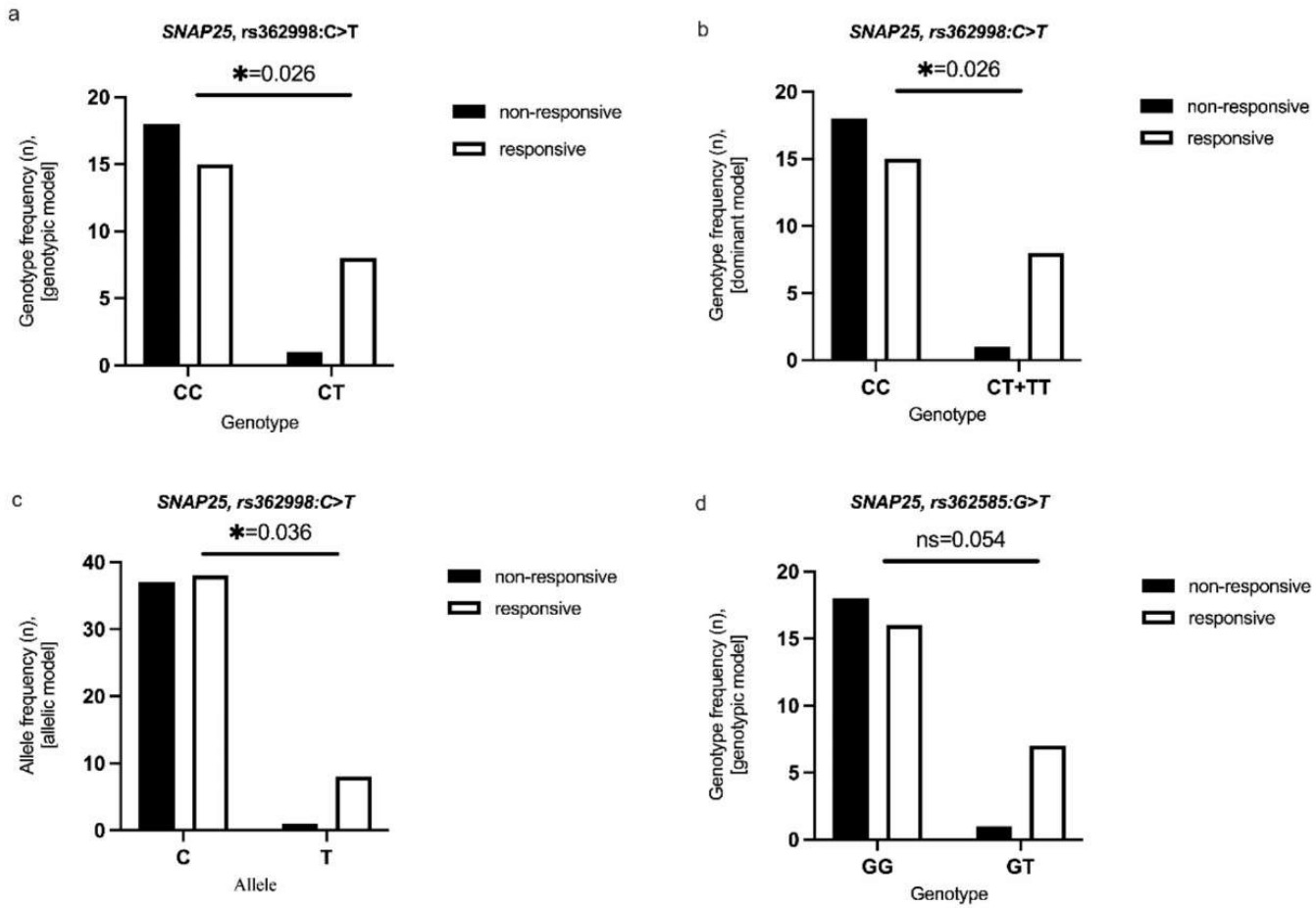


Table 1. Rare and common SNAP-25 gene variants detected in children with ADHD

ADHD Children							
No	**Variant (HGVS cDNA)**	**dbSNP ID**	**Genomic Position (GRCh38)**	**Variant Type**	**ACMG Classification**	**Amino Acid Change**	**Highest population MAF**
1	SNAP25:c.-34C>A	rs11547872	chr20:10275458 C>A	5' prime UTR variant	Benign	-	0.03
2	SNAP25:c.72+41G>T	rs362585	chr20:10275604 G>T	intron variant	Benign	-	0.23
3	SNAP25:c.330C>T	rs362998	chr20:10296973 C>T	synonymous variant	Benign	p.Asp110=	0.25
4	SNAP25:c.552+23G>A	rs363006	chr20:10299435 G>A	intron variant	Benign	-	0.34
5	SNAP25:c.*54T>C	novel	chr20:10306251 T>C	3' prime UTR variant	Not classified	-	NA

ADHD, attention-deficit/hyperactivity disorder; ACMG classification was performed according to the 2015 ACMG/AMP guidelines

MAF: Minor Allele Frequency. The highest minor allele frequency observed in any population, including 1000 Genomes Phase 3, ESP, and gnomAD.

Gene variant, position, ACMG classification, amino acid change, and Pathogenic Criteria taken from Franklin Bioinformatics.

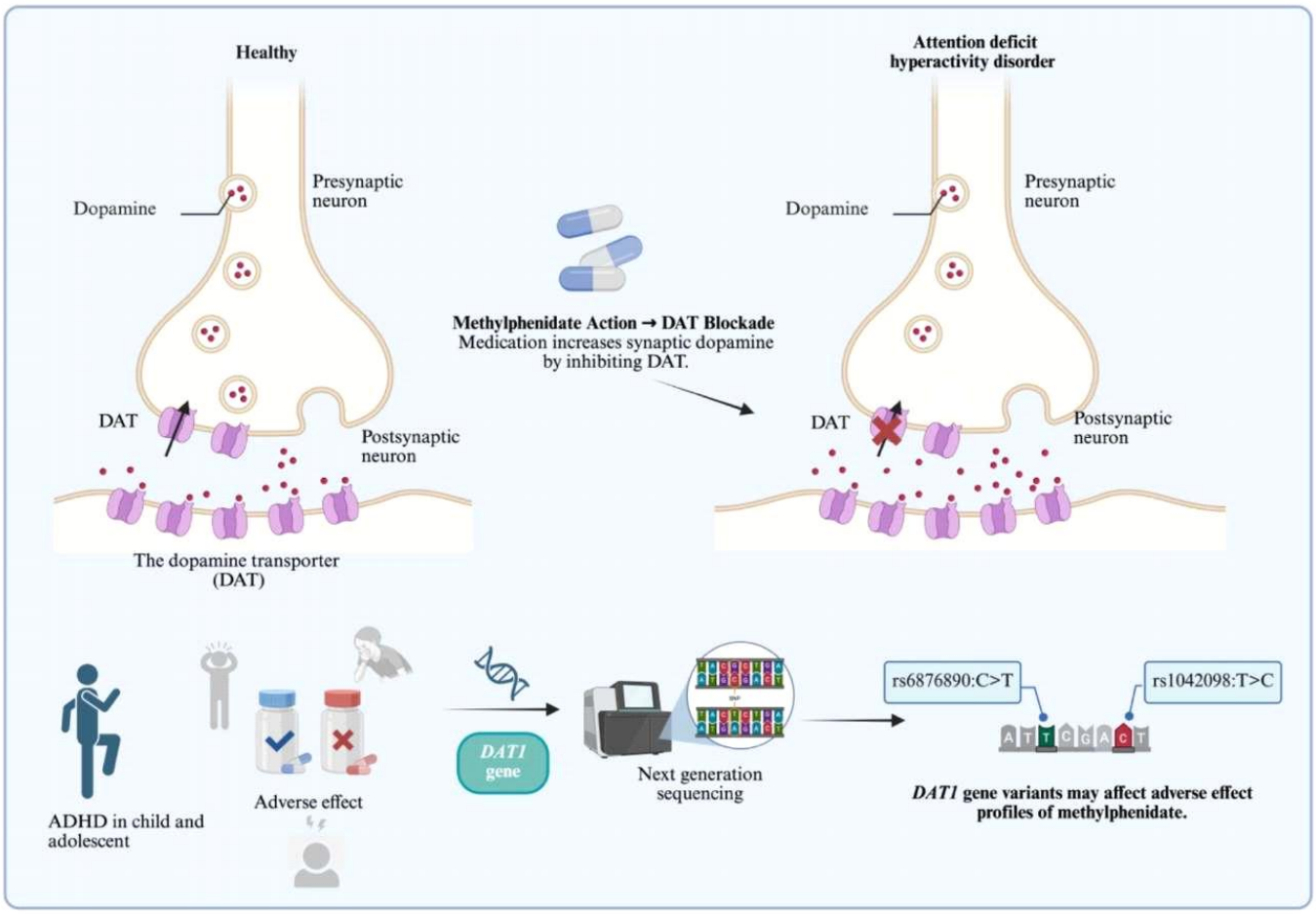
Variation type and the highest population MAF are taken from Ensembl.

NA: data not available.

0255 – Evaluation of Dopamine Transporter Gene Variations in Turkish Children with Attention Deficit Hyperactivity Disorder Treated with Methylphenidate Adverse Effects

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Graphical Abstract: This graphic illustrates that methylphenidate (MPH) increases synaptic dopamine by inhibiting the dopamine transporter (DAT) and highlights that DAT1 gene variants (rs6876890:C>T and rs1042098:T>C) may affect the development of adverse effects in children and adolescents with Attention-deficit hyperactivity disorder (ADHD).

Objective

Attention-deficit hyperactivity disorder (ADHD) is one of the most diagnosed and treated psychiatric disorders in childhood. Methylphenidate (MPH) is the first-line pharmacological treatment for ADHD. MPH exerts its pharmacological effects primarily by inhibiting the dopamine transporter (DAT). Therefore, genetic variability in the DAT1 (SLC6A3) gene may affect individual susceptibility to MPH-related adverse effects. This study aimed to investigate whether genetic variations in the DAT1 gene contribute to individual variability in the MPH-related adverse effects among pediatric patients with ADHD.

Methods

This study included 47 children and adolescents diagnosed with ADHD according to DSM-5-TR criteria [1] who were receiving MPH treatment. MPH-related adverse effects were assessed using the Barkley Stimulants Side Effects Rating Scale [2]. Participants were categorized into adverse effect positive and adverse effect negative groups. The exons of the DAT1 gene were sequenced using targeted next-generation sequencing.

Results

A total of 16 variants were identified in the DAT1 gene, including intronic, untranslated region, synonymous, splice-region, and splice polypyrimidine tract variants (Table 1). One rare intronic deletion variant c.1032-44_1032-42del, was classified as a variant of uncertain significance. Genetic association analyses identified a significant difference in genotype distribution for rs1042098:T>C among participants in adverse effects groups under both the genotypic ($p = 0.003$, OR: 10.18, 95% CI: 2.027-49.43) and recessive ($p = 0.037$, OR: 4.36, %95 CI: 1.165-14.68) models (Figure 1a and 1b, respectively). Similarly, rs6876890:C>T showed a significant association under the genotypic model ($p = 0.008$, OR: 8.67, 95% CI: 1.726-42.24) (Figure 1c).

Discussion

ADHD is one of the most common neurodevelopmental disorders worldwide, with an estimated global prevalence of approximately 8% in children and adolescents [3]. The dopamine transporter mediates dopamine uptake into neurons and is a major target for various psychostimulants, such as MPH [4]. DAT1 gene encodes the dopamine transporter [5]. In the present preliminary study, we investigated genetic variations within the DAT1 and their association with MPH-related adverse effects in Turkish children and adolescents diagnosed with ADHD. Using a tNGS approach, we identified predominantly benign and likely benign variants, with no pathogenic coding alterations. Our genetics models analysis revealed significant associations between MPH-related adverse effects and two DAT1 3' untranslated region polymorphisms, rs1042098 and rs6876890, under the genotypic model. Furthermore, rs1042098 was also found to be statistically significant in the dominant model. These findings are consistent with accumulating evidence that regulatory variation within the DAT1 3' untranslated region polymorphisms can influence gene expression, mRNA stability, or post- transcriptional regulation, thereby affecting dopamine transporter availability and stimulant tolerability [4, 6]

In the present study, a significant association was found between the rs1042098 polymorphism of the DAT1 gene and MPH-related adverse effects. In the literature, rs1042098 has primarily been investigated in relation to ADHD risk and developmental factors. Waldie et al. showed that gene- environment interactions between being small for gestational age (SGA) and DAT1 variants, reporting that rs1042098 interacted with SGA in predicting ADHD symptoms during early childhood [7]. These findings suggest that rs1042098 may contribute to clinical heterogeneity through dopaminergic mechanisms. Our results extend this evidence by indicating that this polymorphism may be associated not only with the ADHD phenotype but also with MPH-related adverse effect profiles, supporting a potential role of DAT1 variation in treatment tolerability. Nevertheless, our findings emphasize the potential importance of non-coding regulatory polymorphisms in modulating individual susceptibility to psychostimulant-related adverse effects. Our findings underscore the potential importance of non-coding and regulatory genetic variation in modulating dopamine transporter regulation and medication response. These results align with models of ADHD pharmacogenetics, which prioritize regulatory variation over rare coding mutations in explaining interindividual variability. This study provides novel, population-specific insights into the Turkish ADHD population and supports the integration of DAT1 genotyping into precision medicine approaches to optimize stimulant treatment and improve patient tolerability.

Conclusion

Our findings suggest that specific DAT1 gene polymorphisms, particularly rs1042098 and rs6876890, may be associated with MPH-related adverse effects in children and adolescents with ADHD.

Figure 1. Distribution of DAT1 gene variants (rs1042098 and rs6876890) among ADHD patients grouped as adverse effect-positive and adverse effect-negative.

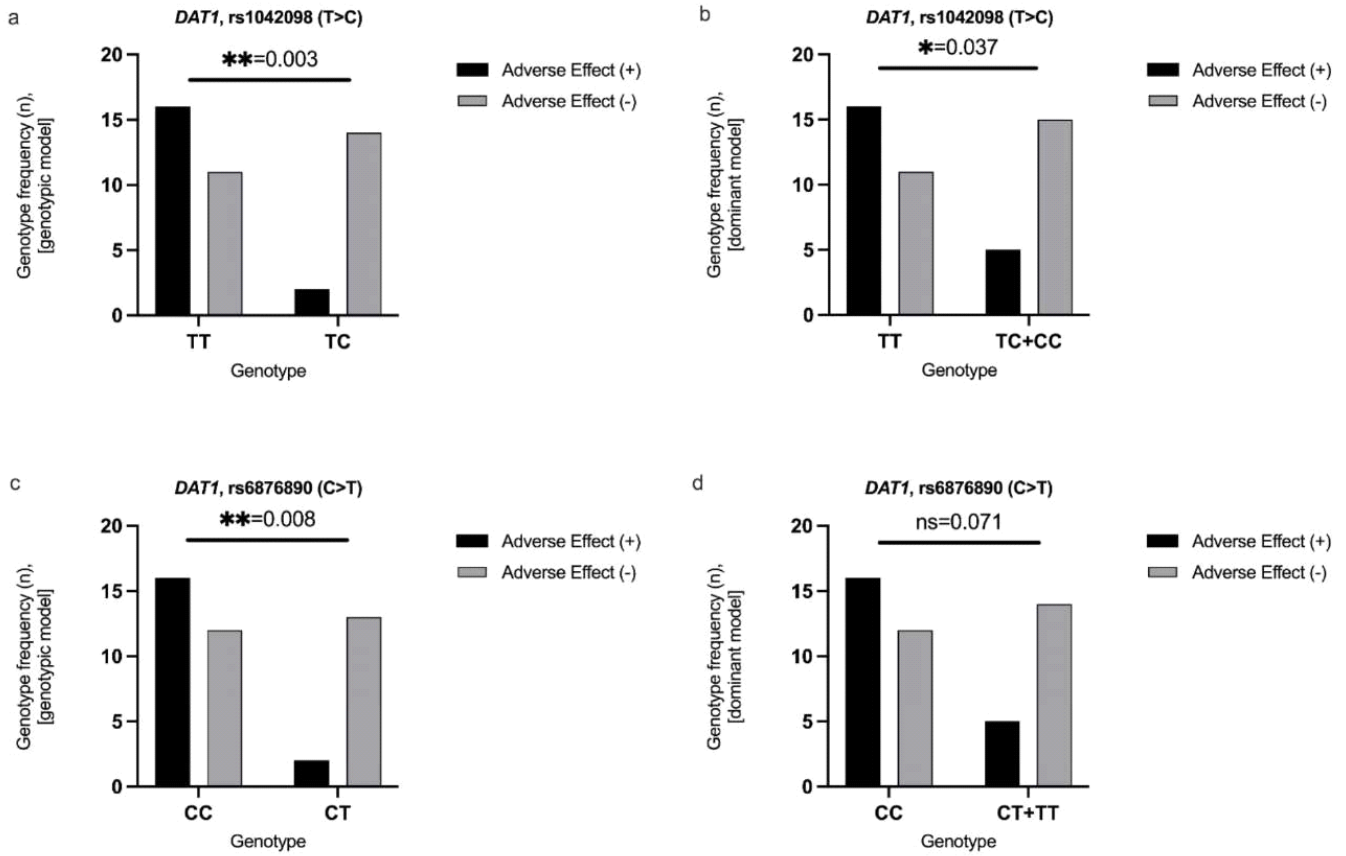


Table 1. Rare and common SLC6A3 (DAT1) gene variants detected in children with ADHD

No	Variant (HGVS cDNA)	dbSNP ID	Genomic Position (GRCh38)	Variant Type	ACMG Classification	Amino Acid Change	Highest population MAF	Pathogenic Criteria
1	SLC6A3:c.-199T>A	rs2975226	chr5-1445501 A>T	5 prime UTR variant	Benign		0.49	
2	SLC6A3:c.1031+71G>T	rs40358	chr5-1416027 C>A	intron variant	Benign		0.48	
3	SLC6A3:c.1215A>G	rs6347	chr5-1411297 T>C	synonymous variant	Benign	p.Ser405=	0.50	
4	SLC6A3:c.1270-21G>A	rs8179029	chr5-1409870 C>T	intron variant	Benign		0.27	
5	SLC6A3:c.1498+14A>G	rs429699	chr5-1409012 T>C	intron variant	Benign		0.29	
6	SLC6A3:c.*35T>C	rs1042098	chr5-1394700 A>G	3 prime UTR variant	Benign		0.50	
7	SLC6A3:c.*972C>T	rs6876890	chr5-1393763 G>A	3 prime UTR variant	Benign		0.40	
8	SLC6A3:c.1599+12C>G	rs150170183	chr5-1406176 G>C	intron variant	Likely Benign		0.01	PM2
9	SLC6A3:c.419-12C>A	rs460000	chr5-1432710 G>T	splice polypyrimidine tract variant	Benign		0.50	
10	SLC6A3:c.*1058G>A	rs535680803	chr5-1393677 C>T	3 prime UTR variant	Benign			
11	SLC6A3:c.114C>T	rs6350	chr5-1443084 G>A	synonymous variant	Benign	p.Asn38=	0.10	
12	SLC6A3:c.1032-44_1032-42del	rs1308673087	chr5-1414856 TGCA>T	intron variant	VUS		< 0.01	PM2
13	SLC6A3:c.419-68G>A	rs465989	chr5-1432766 C>T	intron variant	Benign		0.49	
14	SLC6A3:c.419-63C>T	rs465130	chr5-1432761 G>A	intron variant	Benign		0.49	
15	SLC6A3:c.162C>T	rs6351	chr5-1443036 G>A	synonymous variant	Benign	p.Pro54=	0.02	
16	SLC6A3:c.1155C>T	rs145114326	chr5-1414692 G>A	splice region variant	Benign	p.Asp385=	0.01	

ADHD, attention-deficit/hyperactivity disorder; ACMG classification was performed according to the 2015 ACMG/AMP guidelines

VUS, variant of uncertain significance. MAF: Minor Allele Frequency. The highest minor allele frequency observed in any population, including 1000 Genomes Phase 3, ESP, and gnomAD. Gene variant, position, ACMG classification, amino acid change, and Pathogenic Criteria taken from Franklin Bioinformatics. Variation type and the highest population MAF are taken from Ensembl.

Pathogenicity was evaluated in accordance with the ACMG guidelines. PM2 (Moderate): Absent or very rare in population databases (gnomAD, etc).

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0259 - A Life-Threatening Complication: Pulmonary Embolism Following Zuclopenthixol Acetate

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Introduction

Pulmonary embolism (PE) is a red-flag condition associated with high mortality and is frequently encountered in clinical practice. Venous thromboembolism (VTE), which clinically manifests as deep vein thrombosis (DVT) or its most dangerous complication, acute pulmonary embolism, is the third most common acute cardiovascular syndrome following myocardial infarction and stroke(1). Pulmonary embolism is a multifactorial pathology; however, it may also occur in patients without any identified risk factors. Compared to the general population, the risk of thromboembolism is higher in patients with psychiatric disorders, with an incidence reaching up to 11.6% after three months of treatment.(2)

Recent epidemiological studies have demonstrated that the use of antipsychotic medications may be associated with an increased risk of venous thromboembolism (VTE) and pulmonary embolism (PE). An increased incidence of VTE and PE has been reported with both first-generation and second-generation antipsychotics. This risk has been shown to be more pronounced particularly during the early phase of treatment, at higher doses, and in patients receiving depot (long-acting injectable) antipsychotics(3-4). Despite numerous epidemiological studies and meta-analyses demonstrating the association between antipsychotic use and thromboembolic events, data regarding thromboembolism related to zuclopenthixol acetate remain extremely limited.

Aim

In this poster presentation, we aimed to report a case of early-onset pulmonary embolism following the administration of zuclopenthixol acetate, in order to draw attention to this rare but life-threatening complication and to contribute to the limited data available in the literature.

Case Presentation

A 49-year-old divorced male patient with two children, retired and not actively working, had been diagnosed with bipolar disorder in 2020 and had a history of two previous psychiatric hospitalizations.

The patient presented to the emergency department on 07.10.2025 with complaints of elevated mood, affective elevation, increased speech tone and rate, marked reduction in sleep requirement (3-4 hours per day], grandiose delusions, increased religious preoccupations, uncontrolled spending, behavioral disinhibition (painting his fingernails blue because his car was blue], and decreased self-care. The current clinical presentation was evaluated as compatible with a manic episode, and the patient was admitted to the psychiatric ward.

During the first 6 days of hospitalization, the patient was started on haloperidol 5 mg IM twice daily, biperiden IM twice daily, valproic acid 500 mg twice daily, and quetiapine 100 mg once daily. On day 4 of hospitalization, due to persistence of clinical symptoms, paliperidone 6 mg once daily was added to the treatment regimen. However, as no marked improvement in manic symptoms was observed by day 6, zuclopenthixol acetate was administered.

Bilateral lower extremity venous Doppler ultrasonography revealed no evidence of acute deep vein thrombosis in the examined segments. Transthoracic Approximately 18-24 hours after zuclopenthixol acetate administration, deterioration in vital signs was observed, and oxygen saturation was found to be 81%. Subsequently, D-dimer levels were measured and found to be elevated at 1272.39. A pulmonary medicine consultation was requested. Computed tomography pulmonary angiography reported that evaluation was limited due to suboptimal slice thickness and contrast phase, and that repeat imaging would be appropriate in the presence of clinical suspicion.

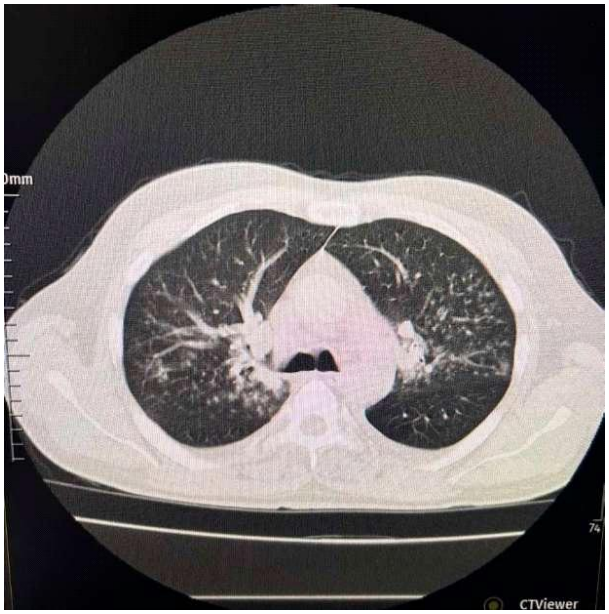
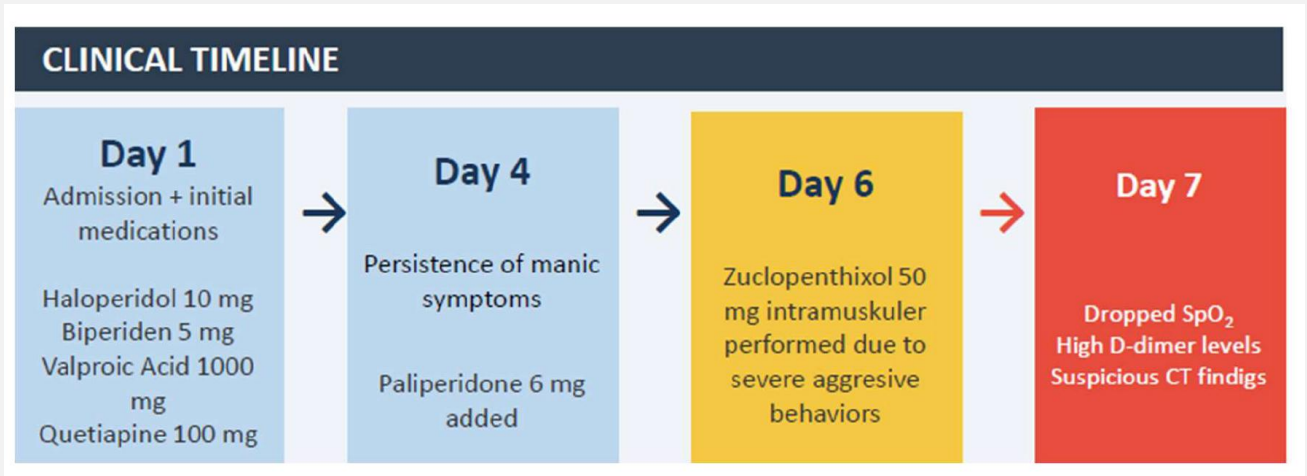


Figure 1: Pulmonary angiography shows suspicious findings for pulmonary embolism

Echocardiography demonstrated an ejection fraction of 60%, with no echocardiographic findings suggestive of massive pulmonary embolism. Given the ongoing clinical suspicion, ventilation/perfusion PET imaging was performed, and enoxaparin sodium 4000 units subcutaneously was initiated. PET imaging demonstrated segmental and subsegmental perfusion defects in both lungs, and the findings were interpreted as highly suggestive of pulmonary thromboembolism. Based on the PET results, the patient was transferred to the pulmonary intensive care unit.



Discussion

The risk of pulmonary embolism and the mechanisms associated with exposure to antipsychotic drugs (APDs) remain unclear. Both psychotic disorders and APDs may contribute to the development of pulmonary embolism. Proposed mechanisms include immobilization, metabolic syndrome, increased antiphospholipid antibodies, and hyperprolactinemia. Many APDs are reported to cause sedation as a side effect, which may lead to immobilization (5). The intramuscular administration of zuclopenthixol acetate may result in sudden and high plasma concentrations after injection; this pharmacological property, potentially leading to sedation and immobilization, may theoretically increase the risk of thromboembolism.

In this case, the absence of known major thromboembolic risk factors and the early onset of pulmonary embolism following zuclopenthixol acetate administration suggest a possible contribution of the drug. Nevertheless, a causal relationship cannot be established. However, such rarely reported cases indicate that short- acting depot antipsychotics should be carefully evaluated with respect to thromboembolic complications.

Clinicians should remain vigilant for thromboembolic complications following depot antipsychotic administration.

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0260 - Evaluation of Psycholeptic and Psychoanaleptic Drug Use in Pregnant Women

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Objective: The use of medications is often necessary in pregnant women with chronic illnesses or in cases of pathological conditions arising from pregnancy. Discontinuing treatment for pre-existing psychiatric disorders can lead to a relapse of the illness, which may negatively affect both maternal health and fetal development. Therefore, it is recommended that psychiatric treatment be carefully continued throughout pregnancy (1). Considering that the illness itself imposes an additional burden on both the mother and the fetus; failure to adjust the dose due to the absence of a safe medication during pregnancy—given pregnancy-related physiological changes (such as alterations in the activity of drug-metabolizing enzymes in the liver, differences in gastric pH and motility, increased tidal volume, decreased albumin concentration, increased glomerular filtration rate, increased maternal blood volume, increased maternal adipose tissue, and total body water, etc.), failure to adjust the dose, or discontinuing the medication without medical supervision, among other factors, can lead to the use of multiple medications during pregnancy. The use of multiple medications carries the risk of drug-drug interactions, and the likelihood of such interactions increases as the number of medications used rises. Psycholeptic and psychoanaleptic medications, which are commonly used in the treatment of psychiatric disorders, are among the most frequently used drug classes during pregnancy (2). The aim of this study was to investigate the use of psycholeptic and psychoanaleptic medications during pregnancy and to identify their adverse effects.

Methods: In this study, the data of a total of sixty pregnant women aged 18-42 who presented to the Medical Pharmacology Outpatient Clinic at Mugla Sitki Ko9man University between October 16, 2024, and January 31, 2026, were retrospectively analyzed. Thirteen pregnant women were using psycholeptic and psychoanaleptic medications. Demographic data of the pregnant women, as well as the types and number of medications they used, were determined. Drug-induced adverse events (oiiE) were examined in pregnant women using two or more medications. oiiE were identified online via the UpToDate database; those not listed in UpToDate were identified via the DrugBank database and classified according to their clinical significance (Tables 1 and 2). Descriptive statistics were used in the analysis of the data.

Results: It was determined that the thirteen pregnant women included in the study used a total of forty-three types of medications. The medications were classified according to the first three digits of their ATC (Anatomical Therapeutic Chemical Classification System) codes, as detailed in Table 1, and were found to fall into sixteen different ATC subgroups (3, Table 3). Within this distribution, it was determined that fifteen medications belonged to the psychoanaleptics (N06) subgroup, while four belonged to the psycholeptics (N05) subgroup. When examined by the number of medications used, it was observed that eleven out of thirteen pregnant women (84.6%) used two or more medications (polypharmacy), while two pregnant women (15.4%) used a single medication. The median age of the eleven women on polypharmacy was 33 (range 24-41 years). Among these women, nine (81.8%) were in their second or subsequent pregnancy, while one (9.1%) was in her first pregnancy; data for one woman were unavailable. When drug exposure was evaluated by trimester, exposure occurred in the first trimester in 10 pregnant women (90.9%) with polypharmacy, while it occurred in the second trimester in one (9.1%). Pregnant women with polypharmacy had exposure to a total of 41 different medications and 51 different active ingredients. The average number of medications used by these pregnant women was 3.73 ± 1.73 (2-8), while the average number of active ingredients was 4.64 ± 2.37 (2-10) (Table 4). A total of 29 drug-drug interactions (DDIs) were identified, 24 using the UpToDate database and 5 using the DrugBank database (Tables 1, 2, and 4). The average number of DDIs was determined to be 2.64 ± 2.50 (Table 4). The clinical significance levels of the DDIs were determined. Upon examining the oiiEs identified using the UpToDate database, it was found that 20 of them (83.3%) fell into the clinically significant D (n=4, 16.7%) and C (n=16, 66.7%) categories; there were no ottEs in the X category. It was observed that 4 oiiEs (16.7%) fell into the clinically insignificant B category; there were no interactions in the A category (Table 1). Of the 5 drug-drug interactions evaluated via the DrugBank database, 2 (40%) were moderate and 3 (60%) were minor (Table 2).

Table 1. Drug interaction categories, their descriptions according to the UpToDate database, and the number of potential drug-drug interactions (pDDIs) identified in pregnant women with polypharmacy

Drug Interaction Categories		Number (%)	
Clinically significant	**X:** Risks outweigh potential benefits; concomitant use should be avoided.	0	20 (%83,3)
	D: Consider modification of therapy; risk–benefit ratio should be evaluated.	4 (%16,7)	
	C: Monitor therapy; dose adjustment may be required.	16 (%66,7)	
Clinically insignificant	**B:** No action needed; no routine therapy modification recommended.	4 (%16,7)	4 (%16,7)
	A: No known interaction.	0	
Total		24 (%100)	

Table 2. Drug interaction categories, their descriptions according to the DrugBank database, and the number of potential drug-drug interactions (pDDIs) identified in pregnant women with polypharmacy

Drug Interaction Categories	Number (%)
Severe (Major): Risks outweigh potential benefits; concomitant use should be avoided.	0
Moderate: Concomitant use should generally be avoided; may be used only under special circumstances.	2 (%40)
Mild (Minor): Risk–benefit should be considered; therapy modification may be required if necessary.	3 (%60)
Total	5 (%100)

Table 3. Anatomical Therapeutic Chemical (ATC) Classification System and Its Levels

Level	Definition	Example (Olanzapine – N05AH03)
Level 1	Main anatomical or pharmacological group (n = 14)	N: Nervous system
Level 2	Pharmacological or therapeutic subgroup	05: Pyscholeptics
Level 3	Chemical, pharmacological, or therapeutic subgroup	A: Antpyschotics
Level 4	Chemical, pharmacological, or therapeutic subgroup	H: Diazepines, oxazepines and thiazepines
Level 5	Chemical substance	03: Olanzapine

Table 4. Total Number of Drugs, Active Substances, and Potential Drug-Drug Interactions (pDDIs) in Pregnant Women with Polypharmacy Using Psycholeptic and Psychoanaleptic Drugs

Parameter	Value
Total number of drugs	41
Mean number of drugs per patient	3,73 ± 1,73 (2-8)
Total number of active substances	51
Mean number of active substances per patient	4,64 ± 2,37 (2-10)
Total number of pDDIs	29
Mean number of pDDIs per patient	2,64 ± 2,50

Discussion: The literature reports that drug exposure during pregnancy is more common in the first trimester, and that this may be largely due to the fact that this period coincides with a time when women are often not yet aware of their pregnancy (6,7). It was observed that 90.9% of pregnant women who presented during the time period included in our study's inclusion criteria were exposed to medication during the first trimester, which is consistent with the literature. Another finding from our study indicates that the use of psycholeptic and psychoanaleptic medications during pregnancy carries a risk of drug-drug interactions, highlighting the need to consider polypharmacy in pregnancy from the perspective of drug-drug interactions. Studies evaluating drug interactions during pregnancy are limited in number. In one such study, it was reported that the use of two or more medications during pregnancy is common, with 30% of pregnant women using five or more medications (7). In our study, the proportion of cases using two or more medications was found to be 84.6%. It is thought that the significant difference between these rates may stem from the differing threshold values (two or more; five or more) used in the definition of polypharmacy. In addition, the fact that the rate of polypharmacy in our study group was as high as 84.6% suggests that the use of multiple medications may be common among patients receiving psychiatric

treatment during pregnancy. It is known that the risk of psychiatric illness exacerbation increases during pregnancy, and untreated psychiatric conditions can negatively affect both maternal and fetal health. Consequently, many pregnant women require pharmacological treatment, which often involves the concurrent use of multiple medications (8). The fact that the vast majority (83.3%) of the drug-drug interactions identified in our study fall into clinically significant categories (D and C) demonstrates that these interactions are not merely a theoretical risk but a situation that must be considered in clinical management. One of the clinically significant contributions of this study is that it highlights the need for a more careful and systematic approach in clinical practice by revealing the frequency of adverse drug interactions associated with the use of psychiatric medications during pregnancy. Our study highlights the need for a systematic review of medications prescribed to pregnant women not only for teratogenicity but also for adverse drug reactions. However, our study has some limitations. For example, while a higher number of AEs was observed in patients with polypharmacy in our study, the descriptive nature of the study design does not establish a causal relationship. Additionally, the limited sample size restricts the generalizability of the results. Future prospective studies with larger sample sizes could more clearly elucidate the clinical outcomes of drug-drug interactions during pregnancy and contribute to the identification of risky drug combinations.

In conclusion, based on the findings of our study, the use of psycholeptic and psychoanaleptic medications during pregnancy is most often associated with polypharmacy. Therefore, it should not be overlooked that psycholeptic and psychoanaleptic medications (oiiE) occurring alongside polypharmacy in pregnant women may have clinically significant consequences.

Note: While compliance with the reference limit specified in the ICP 2026 Brief Report criteria has been ensured, links to the UpToDate, Drug Interactions Checker, and WHO ATC/DDD Index databases have been included in the text to provide supporting information. These links may be removed from the text if deemed necessary during the review process.

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0265 - A Metabolic Trap Mimicking Antipsychotic-Induced Motor Side Effects: 22q11.2 Deletion Syndrome and Hypocalcemia Management

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Introduction: In syndromic neuropsychiatric care, motor symptoms emerging during antipsychotic treatment are often interpreted within the narrow framework of extrapyramidal side effects (EPS), thereby increasing the risk of diagnostic overshadowing. However, in conditions characterized by multisystem vulnerability, such as 22q11.2 deletion syndrome, coexisting metabolic disturbances—notably hypoparathyroidism and vitamin D deficiency—may lower the threshold for neuromuscular excitability, thereby mimicking antipsychotic-induced motor adverse effects or substantially amplifying an existing susceptibility to them. In such cases, the clinical hazard extends beyond simple misattribution of side effects; it also includes failure to recognize a progressive metabolic derangement and the consequent risk of unnecessary medication changes that may compromise psychiatric stability. Accordingly, evaluation of motor symptoms arising during antipsychotic treatment in syndromic patients requires an approach that moves beyond psychopharmacological attribution alone and integrates neuromuscular physiology, calcium homeostasis, and underlying medical vulnerability within a unified clinical framework. This case is instructive in demonstrating how hypocalcemia in the context of 22q11.2 deletion syndrome may constitute a metabolic trap that mimics antipsychotic-induced motor side effects and how accurate differential diagnosis can preserve both medical safety and continuity of psychiatric treatment.

Case Presentation: A 16-year-old male with 22q11.2 deletion syndrome, followed for psychotic symptoms under multiple psychotropic medications, initially presented with motor manifestations that were considered antipsychotic-related extrapyramidal symptoms (EPS). At the time these symptoms became clinically prominent, he was receiving olanzapine 10 mg twice daily, valproic acid 500 mg twice daily, biperiden 2 mg twice daily, lorazepam 2.5 mg/day, and quetiapine 100 mg twice daily. In the weeks following quetiapine augmentation, he developed impaired weight-bearing in the feet, marked gait difficulty, episodes of upward gaze deviation, prolonged leg contractions, and dystonia-like motor phenomena. The presentation was initially managed within a conventional EPS (extrapyramidal symptoms) framework. Accordingly, treatment was simplified, beginning with the agent being considered to contribute additional extrapyramidal burden; quetiapine was discontinued, and biperiden was intensified by adding an extra midday dose. Despite optimization of anticholinergic treatment and reduction of antipsychotic burden, however, motor symptoms failed to show complete and sustained remission. Although transient partial improvement in gait and ocular symptoms was observed, prolonged contractions and upward gaze episodes recurred, prompting reconsideration of the clinical formulation beyond a purely psychopharmacological adverse-effect model.

At the point when additional pathology was systematically considered, the effective psychiatric treatment backbone consisted of olanzapine, valproic acid, lorazepam, and biperiden, while quetiapine had already been discontinued. Given the known association of 22q11.2 deletion syndrome with hypoparathyroidism and calcium- phosphate imbalance, and the central role of calcium homeostasis in neuromuscular excitability, a broader metabolic workup was pursued. Following biochemical and endocrine evaluation consistent with hypocalcemia, active calcium-focused treatment was initiated. Calcitriol 0.5 mcg/day was started first, followed shortly by a calcium/vitamin D combination preparation and cholecalciferol supplementation. At subsequent follow-up, the patient was receiving calcium carbonate 1000 mg plus vitamin D 880 IU, calcitriol 0.5 mcg/day, and cholecalciferol. During this period, psychiatric and endocrine follow-up proceeded in parallel, and the psychiatric regimen was intentionally preserved in order to determine whether motor symptoms would improve with metabolic correction alone.

Once hypocalcemia had been identified, the child and adolescent psychiatry team explicitly adopted a strategy of maintaining the ongoing psychotropic regimen and reassessing the motor picture after calcium replacement. Subsequent follow-up demonstrated clear regression of EPS-like motor findings, including improvement in ocular symptoms and involuntary contractions, after calcium supplementation. During this improvement phase, the psychiatric regimen was not altered; olanzapine, valproic acid, and lorazepam were continued at the same doses,

while only biperiden was gradually tapered and planned for discontinuation. In other words, improvement in the motor syndrome was achieved not through further reduction of antipsychotics, but through correction of calcium homeostasis. This strategy allowed the team to avoid unnecessary antipsychotic changes that might otherwise have increased the risk of psychotic destabilization.

In the following weeks, biochemical improvement was also documented, with rising calcium levels accompanying the clinical response. The core psychiatric regimen of olanzapine, valproic acid, and lorazepam was maintained, while biperiden was gradually reduced from the full twice-daily schedule. Continued endocrine follow-up supported the interpretation that the motor findings were more consistent with hypocalcemia-related neuromuscular irritability than with classic drug-induced parkinsonism. Thus, while metabolic correction was sustained, psychiatric treatment continuity was preserved, and motor symptoms controlled without precipitating psychotic relapse. In later follow-up, benzodiazepine treatment was revised because of availability and tolerability issues; a brief trial of alprazolam was associated with behavioral worsening and was replaced with clonazepam. Nevertheless, the antipsychotic and mood stabilizer backbone remained unchanged. Taken together, the clinical course strongly suggested that the motor findings initially labeled as EPS in fact reflected a neuromuscular-metabolic phenotype related to hypocalcemia and hypoparathyroidism on the background of 22q11.2 deletion syndrome and that the most appropriate strategy was to preserve psychiatric stability while correcting the metabolic axis.

Conclusion: This case demonstrates that, in syndromic neuropsychiatric patients, motor symptoms emerging during antipsychotic treatment cannot always be adequately interpreted within a purely extrapyramidal adverse-effect framework and, if misattributed, may lead to clinically significant consequences. In patients with severe agitation, behavioral dysregulation, and persistent hallucinations or delusions, incorrectly construing such motor findings as simple drug-induced EPS may trigger unnecessary antipsychotic dose reductions, repeated medication switches, additional pharmacological burden, increased anticholinergic exposure, repeated emergency assessments, and potentially avoidable hospitalizations. Such interventions not only complicate the existing clinical picture but may also precipitate psychotic exacerbation, compromise adherence, increase caregiver burden, and ultimately further impair long-term functioning.

In this case, the persistence of motor symptoms despite anticholinergic treatment and antipsychotic adjustments aimed at reducing extrapyramidal burden necessitated a broader differential diagnostic approach grounded in neuromuscular physiology and metabolic homeostasis. Resolution of the EPS-like syndrome through correction of hypocalcemia- and hypoparathyroidism-related metabolic abnormalities, while preserving the ongoing psychiatric regimen, illustrates that accurate diagnostic framing is critical not only for symptom control but also for the maintenance of psychiatric stability. This approach is particularly important in treatment-resistant, psychotic patients receiving multiple medications, in whom avoiding unnecessary medication changes may prevent additional sedation, paradoxical behavioral worsening, cumulative adverse-effect burden, and unnecessary escalation of treatment complexity.

In child and adolescent psychiatry, the significance of such an approach extends well beyond acute symptom management. In patients with severe psychotic symptoms, agitation, and the need for long-term psychopharmacological follow-up, each inappropriate medication change may affect not only immediate clinical control but also educational continuity, family functioning, social adaptation, trust in treatment, and the broader trajectory of lifelong recovery. For this reason, evaluation of psychopharmacological adverse effects—particularly in multisystem and syndromic conditions—should be guided not by medication-centered reasoning alone, but by a holistic clinical perspective that integrates physiology, pathophysiology, coexisting medical vulnerabilities, and multidisciplinary collaboration. Such an approach is essential for minimizing unnecessary treatment burden while preserving patient safety, psychiatric stability, and long-term functional outcomes.

Keywords: 22q11.2 deletion syndrome, diagnostic overshadowing, extrapyramidal symptoms, hypocalcemia, hypoparathyroidism

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0270 - Social-Cognitive Vulnerability in Neurofibromatosis Type 1: Theory of Mind, Autism-Like Traits, Executive Functions, and MRI-Based Brain Volumetric Correlates

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Objective

Neurofibromatosis type 1 (NF1) is a common autosomal dominant neurogenetic disorder associated with significant cognitive and behavioral morbidity, including executive dysfunction and social impairments. Although social-cognitive difficulties are well documented, the underlying mechanisms remain poorly understood. Previous studies have rarely examined theory of mind (ToM), executive functions, and autism-like traits jointly in relation to brain structure. This study aimed to investigate social-cognitive vulnerability in children with NF1 by integrating behavioral assessments (ToM, executive functions, and autism-like traits) with MRI-based volumetric and cortical thickness measures of brain regions implicated in social cognition.

Methods

This two-center, cross-sectional study included 41 children aged 8-18 years: 21 diagnosed with NF1 according to revised Legius criteria and 20 age- and sex-matched typically developing controls (TDC). The control group consisted of children who underwent brain MRI due to headache but had no neurological, psychiatric, or developmental diagnosis. All participants had clinically normal intellectual functioning.

Parents completed the Social Responsiveness Scale (SRS) for autism-like traits and the Executive Function and Activity Routine Scale (EFORTS) for daily executive functioning. Children were individually administered ToM tasks, including the Reading the Mind in the Eyes Test (RMET) and second-order false belief tasks (FBTs).

Structural MRI data were analyzed using the volBrain automated online system (https://volbrain.upv.es/). Volumetric and cortical thickness measures were obtained for brain regions implicated in social cognition and executive functioning, including total cerebrum, cerebellum, brainstem, and selected cortical and subcortical structures.

Due to non-normal distributions and sample size considerations, non-parametric statistical methods were used. Continuous variables were compared using the Mann-Whitney U test, and categorical variables using chi-square or Fisher's exact test. All tests were two-tailed, and statistical significance was set at $p < 0.05$, with nominal p-values reported given the exploratory nature of the study.

Results

Children with NF1 demonstrated significantly higher autism-like traits (SRS total score; $p = 0.039$) and poorer executive functioning in daily routines (EFORTS total score; $p = 0.012$) compared to controls. Specifically, significant impairments were observed in play/leisure and social routines ($p = 0.001$ and $p = 0.003$, respectively). Regarding social cognition, the NF1 group showed reduced performance on advanced ToM measures, particularly in the RMET ($p = 0.013$). (Table 1)

Neuroanatomically, the NF1 group exhibited increased total brain volume ($p = 0.019$), largely driven by widespread white matter (WM) expansion (WM volume %; $p < 0.001$). Cerebellar and brainstem volumes were also significantly increased (all $p < 0.001$). In contrast, relative cortical gray matter (GM) proportion ($p = 0.003$)

and cortical thickness were reduced. Region-specific analyses revealed significant thickness and volume reductions in areas associated with higher-order cognition, including the frontal pole, fusiform gyrus, superior parietal lobule, and cingulate cortex.Additionally, several subcortical structures, including the amygdala ($p = 0.003$), hippocampus ($p = 0.007$), and thalamus ($p < 0.001$), showed volumetric enlargement in the NF1 group. This pattern indicates a clear neuroanatomical dissociation between cortical maturation and subcortical/white matter expansion (Table 2, Table 3, Figure 1).

Discussion

This study demonstrates that social-cognitive vulnerability in NF1 is multidimensional and closely linked to a distinct neuroanatomical profile. The findings support a model in which social difficulties arise not from a single deficit but from a broader imbalance between cortical and subcortical systems.

Consistent with previous literature, increased global brain volume in NF1 appears to be primarily driven by white matter expansion rather than ventricular enlargement. At the same time, reduced cortical gray matter proportion and cortical thickness suggest atypical cortical maturation, particularly in regions critical for social cognition and executive functioning such as the frontal pole and cingulate cortex. Subcortical volumetric enlargement, especially in limbic structures like the amygdala and hippocampus, further supports the presence of altered neurodevelopmental trajectories. This cortical-subcortical imbalance may impair the integration of higher-order regulatory processes with more basic emotional and salience-driven systems, contributing to observed social-cognitive difficulties. Importantly, unidentified bright objects (UBOs/FASI lesions), which commonly involve basal ganglia and deep white matter in NF1, may have contributed to the observed increase in white matter volume. These lesions, along with possible surrounding microstructural alterations or edema-like changes, could partially influence volumetric measurements and should be considered when interpreting MRI findings. From a clinical perspective, these findings highlight the importance of integrated neurodevelopmental assessment in NF1. Combining behavioral and neuroimaging markers may improve early identification of children at risk for social-cognitive difficulties and guide targeted interventions.

However, several limitations should be acknowledged. The control group consisted of children presenting with headache rather than population-based controls, which may introduce selection bias. The sample size was relatively small, and multiple comparisons were not adjusted due to the exploratory design. Despite these limitations, this study provides a comprehensive framework linking behavioral and neuroanatomical alterations in NF1 and supports the conceptualization of social-cognitive vulnerability as a network-level developmental imbalance.

In conclusion, this study reveals a multidimensional vulnerability pattern in the social- cognitive profile of children with NF1. Our findings demonstrate that social-cognitive difficulties arise from a developmental imbalance between cortical maturation and subcortical/white matter expansion rather than a singular core deficit. Clinically, these results suggest that the assessment of specific mechanisms, such as Theory of Mind and executive functions within social contexts, can facilitate earlier identification of at-risk children and help in planning targeted intervention strategies to improve their daily social adaptive functioning.

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Table 1. Comparison of NF1 and TDC groups in SRS, EFORTS, RMET and FBTs scores.

Variables median (25th–75th) or n (%)	NF1 n = 21	TDC n = 20	*P* value
SRS			
Total Score	51.0 (34.5-71.5)	40.0 (31.0-47.0)	**0.039**
EFORTS			
Morning and evening routines	3.7 (2.7-4.3)	3.9 (3.4-4.7)	0.124
Play and leisure routines	3.7 (2.7-4.3)	4.9 (3.9-5.0)	**0.001**
Social routines	3.6 (2.7-4.0)	4.6 (3.9-4.7)	**0.003**
Total Score	3.6 (2.8-4.3)	4.2 (3.7-4.8)	**0.012**
RMET			
Total Score	15.0 (11.5-19.0)	18.5 (17.0-21.0)	**0.013**
The Second Order FBTs			
Chocolate Bar Task	12 (57.1)	17 (85.0)	**0.050**
Ice Cream Truck Task	12 (57.1)	12 (60.0)	0.853

Note: n indicates number of the participants. Mann-Whitney U or Pearson's chi-square were used as appropriate. Statistical significance was defined as $p < 0.05$, significant results are shown in bold. Abbreviations: NF1, neurofibromatozis tip 1; TDC, typically developing children; SRS, Social Responsiveness Scale; EFORTS, The Executive Functions and Occupational Routines Scale; RMET, Reading the Mind in the Eyes Test; FBTs, False Belief Tasks.

Table 2. Comparison of brain volumetric measures between NF1 and TDC groups

Variables median (25th-75th)	NF group n = 21	TDC n = 20	*P* value
Brain volume (WM + GM) cm^3	1327.4 (1265.7-1517.5)	1244.9 (1201.4-1342.2)	0.019
Brain volume (GM + WM) %	91.2 (88.5-92.1)	90.6 (87.7-92.6)	0.835
Brainstem volume cm3	21.4 (20.0-24.8)	18.5 (17.1-18.7)	<0.001
Brainstem volume %	1.5 (1.4-1.6)	1.3 (1.2-1.4)	<0.001
Cerebellum total volume cm3	150.2 (136.5-165.4)	128.8 (120.1-139.6)	<0.001
Cerebellum total volume %	10.0 (9.2-10.8)	9.3 (8.8-9.6)	0.003
WM volume cm^3	532.8 (460.0-604.1)	451.6 (440.8-475.3)	0.003
WM volume %	34.8 (33.3-36.5)	32.5 (31.3-33.5)	<0.001
Cerebrum WM total volume cm^3	504.1 (432.1-564.1)	425.7 (415.0-450.4)	0.003
Cerebrum WM total volume %	32.9 (31.4-34.4)	30.7 (29.5-31.8)	0.001
Cerebellum WM total volume cm^3	30.5 (28.2-36.2)	25.6 (24.3-27.3)	<0.001
Cerebellum WM total volume %	2.1 (1.9-2.4)	1.8 (1.7-1.9)	<0.001
GM volume cm^3	843.8 (776.5-918.5)	804.3 (743.0-857.9)	0.124
GM volume %	55.4 (54.1-57.2)	57.4 (55.7-59.9)	0.027
Subcortical GM volume cm^3	52.6 (49.0-56.4)	46.2 (44.7-49.1)	0.003
Subcortical GM volume %	3.4 (3.3-3.6)	3.4 (3.2-3.4)	0.106
Cortical GM volume cm^3	669.1 (608.4-720.3)	646.1 (599.9-691.6)	0.375
Cortical GM volume %	43.9 (42.0-44.7)	46.4 (43.5-48.2)	0.003
Cerebrum GM total volume cm^3	727.0 (659.9-777.0)	692.0 (646.1-739.7)	0.335
Cerebrum GM total volume %	47.2 (45.5-48.1)	49.7 (46.9-51.5)	0.004
Cerebellum GM total volume cm^3	130.0 (116.6-140.8)	112.3 (104.6-120.7)	<0.001
Cerebellum GM total volume %	8.5 (8.1-9.3)	8.1 (7.7-8.5)	0.030

Note: n indicates number of the participants. Mann-Whitney U test was used. Statistical significance was defined as $p < 0.05$; significant results are shown in bold.
Abbreviations: NF1, neurofibromatozis tip 1; TDC, typically developing children; GM, grey matter.

Table 3. Comparing of Brain Regions Related to Social Cognition, Executive Function, and Emotion Regulation in NF1 and TDC groups

Variables median (25th–75th)	NF group n = 21	TDC n = 20	*P* value
Frontal Brain Regions			
Frontal total volume cm^3	230.4 (201.3-252.2)	223.8 (203.8-235.0)	0.389
Frontal total volume %	15.0 (14.3-15.5)	15.8 (14.7-16.9)	**0.020**
Frontal total thickness mm	3.4 (3.3-3.6)	3.6 (3.3-3.7)	0.095
Middle Frontal Gyrus total volume cm^3	51.8 (44.1-56.4)	49.1 (44.8-54.5)	0.549
Middle Frontal Gyrus total volume %	3.4 (3.2-3.5)	3.6 (3.2-3.9)	0.103
Middle Frontal Gyrus total thickness mm	3.4 (3.2-3.5)	3.8 (3.5-3.8)	**0.027**
Frontal Pole total volume cm^3	9.4 (8.1-10.6)	10.1 (8.6-11.0)	0.241
Frontal Pole total volume %	0.6 (0.6-0.7)	0.7 (0.6-0.8)	**0.003**
Frontal Pole total thickness mm	3.6 (3.4-3.8)	3.8 (3.4-4.1)	**0.035**
Temporal Brain Regions			
Fusiform gyrus total volume cm^3	17.5 (15.3-21.1)	18.4 (17.5-18.9)	0.449
Fusiform gyrus total volume %	1.2 (1.1-1.3)	1.3 (1.3-1.4)	**0.004**
Fusiform gyrus total thickness mm	4.1 (3.9-4.2)	4.2 (4.0-4.2)	**<0.001**
Inferior Temporal Gyrus total volume cm^3	28.6 (27.3-32.4)	29.6 (27.1-31.1)	0.794
Inferior Temporal Gyrus total volume %	1.9 (1.8-2.0)	2.0 (2.0-2.2)	**<0.001**
Inferior Temporal Gyrus total thickness mm	4.1 (3.9-4.2)	4.1 (4.0-4.2)	0.715
Middle Temporal Gyrus total volume cm^3	37.2 (35.5-41.9)	33.6 (31.6-37.0)	**0.022**
Middle Temporal Gyrus total volume %	2.4 (2.4-2.6)	2.4 (2.3-2.5)	0.434
Middle Temporal Gyrus total thickness mm	3.9 (3.7-4.0)	3.8 (3.7-4.0)	0.794
Parietal Brain Regions			
Supramarginal Gyrus total volume cm^3	21.0 (18.2-22.9)	20.7 (18.9-23.3)	0.514
Supramarginal Gyrus total volume %	1.4 (1.2-1.5)	1.5 (1.3-1.7)	**0.012**
Supramarginal Gyrus total thickness mm	3.1 (3.0-3.2)	3.2 (2.9-3.3)	0.322
Superior Parietal Lobule total volume cm^3	24.8 (22.4-27.9)	24.7 (22.6-27.0)	0.855
Superior Parietal Lobule total volume %	1.6 (1.5-1.7)	1.8 (1.7-1.9)	**0.007**
Superior Parietal Lobule total thickness mm	2.3 (2.2-2.6)	2.4 (2.2-2.6)	0.876
Cingulate Gyrus			
Anterior Cingulate Gyrus total volume cm^3	13.4 (13.0-14.8)	13.1 (12.3-13.9)	0.192
Anterior Cingulate Gyrus total volume %	0.9 (0.8-1.0)	0.9 (0.8-1.0)	0.419
Anterior Cingulate Gyrus total thickness mm	4.3 (4.2-4.3)	4.3 (4.3-4.4)	**0.022**
Middle Cingulate Gyrus total volume cm^3	12.3 (11.6-13.2)	12.3 (11.8-12.7)	0.938
Middle Cingulate Gyrus total volume %	0.8 (0.8-0.9)	0.9 (0.8-0.9)	**0.045**
Middle Cingulate Gyrus total thickness mm	3.8 (3.6-3.9)	4.0 (3.7-4.0)	**0.054**
Posterior Cingulate Gyrus total volume cm^3	11.8 (10.0-12.7)	12.0 (11.5-13.5)	0.183
Posterior Cingulate Gyrus total volume %	0.8 (0.7-0.8)	0.9 (0.8-0.9)	**<0.001**
Posterior Cingulate Gyrus total thickness mm	4.0 (3.9-4.0)	4.1 (3.9-4.2)	**0.010**
Limbic System			
Limbic total volume cm^3	49.3 (45.3-51.8)	47.4 (45.1-51.3)	0.481
Limbic total volume %	3.2 (3.1-3.5)	3.4 (3.3-3.6)	**0.027**
Limbic total thickness mm	3.9 (3.8-3.9)	4.0 (3.9-4.1)	**0.006**
Amygdala total volume cm^3	2.2 (1.9-2.4)	1.9 (1.8-2.0)	**0.003**
Amygdala total volume %	0.1 (0.1-0.1)	0.1 (0.1-0.1)	0.481
Hippocampus total volume cm^3	9.4 (8.2-10.7)	8.1 (7.7-8.6)	**0.007**

Hippocampus total volume %	0.6 (0.5-0.7)	0.6 (0.5-0.6)	0.361
Entorhinal area total volume cm^3	4.1 (3.9-4.4)	3.9 (3.7-4.3)	0.230
Entorhinal area total volume %	0.3 (0.3-0.3)	0.3 (0.3-0.3)	0.498
Parahippocampal Gyrus total volume cm^3	7.1 (6.2-7.6)	5.9 (5.6-6.6)	**0.003**
Parahippocampal Gyrus total volume %	0.5 (0.4-0.5)	0.4 (0.4-0.5)	0.081
Parahippocampal Gyrus total thickness mm	3.3 (3.0-3.4)	3.4 (3.3-3.5)	**0.037**
Insula total volume cm^3	35.5 (31.6-39.8)	34.5 (32.9-37.2)	0.814
Insula total volume %	2.3 (2.2-2.4)	2.5 (2.4-2.7)	**0.003**
Insula total thickness mm	3.5 (3.5-3.6)	3.6 (3.5-3.7)	0.220
Anterior Insula total volume cm^3	9.9 (9.2-10.9)	9.7 (9.1-10.5)	0.348
Anterior Insula total volume %	0.7 (0.6-0.7)	0.7 (0.6-0.7)	0.106
Anterior Insula total thickness mm	4.0 (3.9-4.1)	4.1 (4.0-4.1)	0.241
Posterior Insula total volume cm^3	5.5 (5.1-5.8)	5.4 (4.9-5.8)	0.774
Posterior Insula total volume %	0.3 (0.3-0.4)	0.4 (0.4-0.4)	**0.004**
Posterior Insula total thickness mm	3.7 (3.5-3.8)	3.7 (3.5-3.8)	0.855
Basal Ganglia			
Caudate total volume cm^3	9.4 (8.5-10.4)	8.4 (7.9-9.7)	0.076
Caudate total volume %	0.6 (0.6-0.7)	0.6 (0.6-0.7)	0.958
Putamen total volume cm^3	10.3 (9.7-10.9)	10.0 (9.5-10.5)	0.297
Putamen total volume %	0.7 (0.6-0.7)	0.7 (0.7-0.7)	**0.011**
Pallidum total volume cm^3	3.3 (3.1-3.5)	2.9 (2.9-3.1)	**0.004**
Pallidum total volume %	0.2 (0.2-0.2)	0.2 (0.2-0.2)	0.285
Other brain regions			
Thalamus total volume cm^3	15.6 (13.7-17.8)	13.5 (12.5-14.5)	**<0.001**
Thalamus total volume %	1.0 (1.0-1.0)	1.0 (0.9-1.0)	**0.002**
Cerebellar Vermal Lobules VI-VII volume cm^3	2.5 (2.3-2.6)	2.1 (1.9-2.3)	**<0.001**
Cerebellar Vermal Lobules VI-VII volume %	0.2 (0.1-0.2)	0.2 (0.1-0.2)	**0.027**
Cerebellar Vermal Lobules VIII-IX volume cm^3	3.3 (2.9-3.6)	2.8 (2.4-3.0)	**0.003**
Cerebellar Vermal Lobules VIII-IX volume %	0.2 (0.2-0.2)	0.2 (0.2-0.2)	**0.047**

Note: n indicates number of the participants. Mann- Whitney U test was used. Statistical significance was defined $p < 0.05$; significant results are shown in bold.
Abbreviations: NF1, neurofibromatozis tip 1; TDC, typically developing children; GM, grey matter.

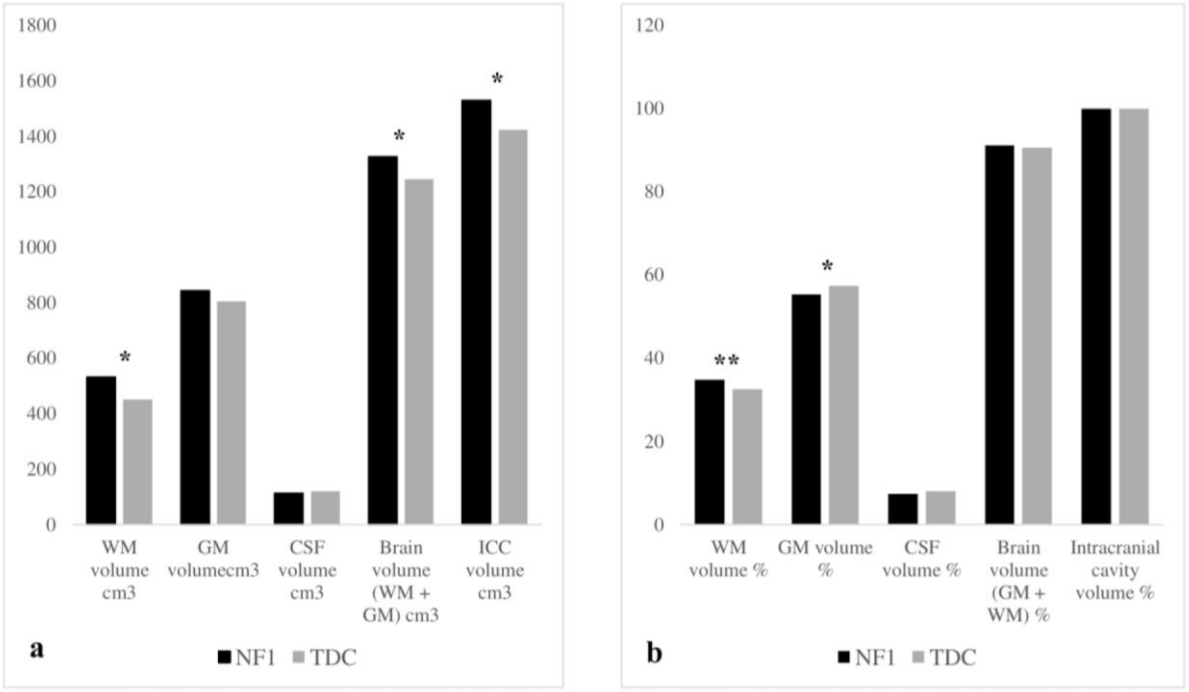


Figure 1. Comparison of global brain volumetric measures between children with NF1 and TDC.

Panel (a) illustrates median absolute volumes (cm^3) of white matter, gray matter, cerebrospinal fluid, total brain volume (WM + GM), and intracranial cavity volume in the NF1 and TDC groups.

Panel (b) presents the same measures expressed as percentages relative to intracranial cavity volume.

Bars represent median values. Detailed statistical comparisons are provided in Supplementary Table S3. NF1, neurofibromatosis type 1; TDC, typically developing children; WM, white matter; GM, gray matter; CSF, cerebrospinal fluid; ICC, intracranial cavity. * $p < 0.005$, ** $p < 0.001$.

0271 - Longitudinal Links Between Intolerance of Uncertainty, Changes in Emotion Regulation, and Non-Suicidal Self-Injury in Adolescents with a History of NSSI

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Objective

Non-suicidal self-injury (NSSI) is a prevalent and often persistent behavior in adolescence, strongly associated with emotion regulation difficulties and intolerance of uncertainty (IU) in cross-sectional studies. However, longitudinal evidence is scarce, particularly among adolescents already engaging in NSSI. The present study aimed to examine the prospective relationships between baseline IU, longitudinal changes in emotion regulation strategies, and subsequent NSSI outcomes (severity and behavioral persistence) over one year.

Three primary questions guided the investigation:

1. Does higher baseline IU predict greater NSSI severity or increased likelihood of continued NSSI at one-year follow-up, after controlling for baseline NSSI severity?

2. Do reductions in dysfunctional emotion regulation strategies (particularly internal ones) independently predict improvement in NSSI severity?

3. Do changes in emotion regulation mediate any prospective effect of baseline IU on follow-up NSSI outcomes?

By focusing on within-person change and controlling for initial symptom levels in a high- risk clinical sample, the study sought to clarify the relative roles of a stable cognitive vulnerability (IU) and dynamic regulatory processes in the maintenance of NSSI.

Methods

Participants were 42 adolescents (M age at baseline = 15.9 years, SD = 1.19, range 12.817.9; 90.5% female) with a documented lifetime history of NSSI who were reassessed after a mean interval of 12.1 months (SD = 1.4). All participants completed both baseline (T1) and follow-up (T2) assessments with no missing data on the primary variables.

NSSI severity and functions were assessed using the NSSI Behavioral Assessment Inventory (NSSI BAI), yielding autonomous function, social function, and total severity scores. Intolerance of uncertainty was measured with the Intolerance of Uncertainty Index for Children and Adolescents (IUI-C), providing total, inhibitory, and prospective subscale scores. Emotion regulation strategies were evaluated using the Emotion Regulation Scale for Adolescents (ERSA), which distinguishes internal dysfunctional, external dysfunctional, and internal functional strategies.

Paired-samples t-tests with Cohen's d evaluated within-person change from T1 to T2. Pearson correlations examined associations among change scores (Δ = T2 - T1). Multiple linear regression models predicted T2 NSSI severity, entering age, baseline NSSI severity, baseline IU, and emotion regulation change scores simultaneously. Binomial logistic regression predicted the presence/absence of NSSI during the last three months at T2. Change-based mediation was tested using bias-corrected bootstrapping (5,000 resamples) via the PROCESS macro (model 4), with baseline NSSI severity included as a covariate. All analyses were conducted in SPSS version 27 and PROCESS v4; significance was set at $p < .05$ (two-tailed).

Results

Significant within-person reductions were observed across multiple domains. NSSI total severity decreased markedly ($t(41) = -4.88$, $p < .001$, $d = -0.75$), as did both autonomous ($d = -0.81$) and social ($d = -0.56$) functions.

Total IU scores declined moderately ($t(41) = -2.98$, $p = .005$, $d = -0.46$), with similar reductions in both inhibitory and prospective subscales. Among emotion regulation strategies, internal dysfunctional regulation showed the largest improvement ($t(41) = -4.17$, $p < .001$, $d = -0.64$), followed by external dysfunctional regulation ($t(41) = -2.30$, $p = .026$, $d = -0.36$). Internal functional regulation remained stable ($p = .725$).

Change-score correlations revealed strong positive associations between reductions in NSSI severity and reductions in both IU (ΔIUI total with ΔNSSI total: $r = .92$, $p < .001$) and internal dysfunctional regulation ($r = .68$, $p < .001$). Smaller but significant associations emerged for external dysfunctional regulation ($r = .53$, $p < .001$). Age was unrelated to change in any primary variable.

In the primary multiple linear regression predicting NSSI total severity at T2, the model accounted for substantial variance (adjusted $R^2 = .35$, $F(5,36) = 5.35$, $p < .001$). Baseline NSSI severity ($\beta = .41$, $p = .003$) and greater reductions in internal dysfunctional emotion regulation ($\beta = .37$, $p = .035$) emerged as independent predictors. Baseline total IU ($p = .989$), external regulation change ($p = .092$), and age ($p = .331$) did not contribute significantly.

When predicting change in NSSI functional domains separately, reductions in internal dysfunctional regulation significantly predicted improvement in autonomous functions ($\beta = .38$, $p = .016$), whereas reductions in external dysfunctional regulation predicted improvement in social functions ($\beta = .39$, $p = .018$).

Binomial logistic regression examining predictors of NSSI presence during the last three months at T2 yielded a significant overall model ($X^2(5) = 15.10$, $p = .010$, McFadden $R^2 = .26$). Higher baseline prospective IU was the only significant predictor of continued NSSI (OR = 1.29, 95% CI [1.04, 1.61], $p = .019$). Baseline NSSI severity ($p = .603$), inhibitory IU ($p = .665$), and both emotion regulation dimensions showed nonsignificant or trend-level associations only (internal dysfunctional OR = 1.35, $p = .080$; external dysfunctional OR = 0.83, $p = .071$).

Change-based mediation analyses tested whether reductions in internal dysfunctional emotion regulation mediated the prospective effect of baseline IU on T2 NSSI severity (controlling for baseline NSSI). The indirect effect was not significant (indirect effect = -0.129, SE = 0.089, 95% BCa CI [-0.385, 0.029], $p = .148$). Path b (Δ internal dysfunctional regulation → T2 NSSI severity) was robust ($\beta = .494$, $p < .001$), whereas path a (baseline IU →Δ regulation) was nonsignificant ($\beta = -.234$, $p = .119$). Baseline IU showed no direct effect on follow-up severity ($\beta = .008$, $p = .953$), while baseline NSSI severity remained a strong direct predictor ($\beta = .374$, $p = .003$).

Discussion

The present longitudinal study provides several key insights into the maintenance of NSSI among adolescents with an established history of behavior. First, the sample exhibited clinically meaningful reductions in NSSI severity, IU, and dysfunctional emotion regulation over one year. These downward trends are consistent with prior longitudinal observations that NSSI can follow an episodic or declining course in some adolescents, particularly those who come to clinical attention or naturally develop more adaptive coping over time.

Second, and most robustly, reductions in internal dysfunctional emotion regulation strategies emerged as the strongest and most consistent predictor of decreased NSSI severity at follow-up — an effect that held after accounting for baseline severity, age, IU, and external regulation change. This finding strongly supports theoretical models (e.g., experiential avoidance model, emotional cascade model) that conceptualize NSSI primarily as a maladaptive intrapersonal affect-regulation strategy. Adolescents who showed the greatest decreases in rumination, emotional suppression, self-criticism, and other internal maladaptive responses experienced the most pronounced NSSI improvement, suggesting that targeting these processes should remain a central focus of intervention.

In contrast, baseline IU did not predict NSSI severity once baseline symptoms and regulatory change were controlled. However, higher prospective IU at baseline was associated with increased odds of any NSSI occurring during the final three months of follow-up. This dissociation implies that IU may operate primarily as a distal vulnerability factor that increases the probability of behavioral recurrence rather than determining the intensity of self-injury when it does occur. Adolescents high in prospective IU may be more likely to revert to overlearned, certainty-providing behaviors (such as NSSI) when confronted with ambiguous or unpredictable emotional states, thereby elevating risk of relapse even after periods of relative improvement.

Contrary to hypothesis, changes in emotion regulation did not mediate the prospective relationship between IU and NSSI outcomes. The absence of significant indirect effects suggests that IU does not exert its longitudinal influence primarily through short-term shifts in regulatory capacity. Instead, IU may contribute to NSSI persistence via alternative pathways — for example, by sustaining chronic anticipatory anxiety, promoting behavioral rigidity, or reinforcing avoidance repertoires that are relatively independent of moment-to-moment emotion regulation skill.

From a clinical perspective, these findings highlight two complementary intervention targets. Emotion regulation training (e.g., DBT skills modules, mindfulness-based approaches, cognitive reappraisal practice) appears particularly promising for reducing NSSI severity among adolescents already engaging in the behavior. At the same time, strategies aimed at increasing tolerance for uncertainty — such as gradual exposure to ambiguity, uncertainty acceptance exercises, and behavioral experiments that challenge the need for absolute certainty — may be especially valuable for preventing recurrence and supporting longer-term behavioral stability.

Several limitations should be noted. The modest sample size constrained power to detect smaller effects, particularly in mediation models; replication in larger samples is essential. The predominantly female composition limits generalizability to male adolescents. Reliance on self-report measures introduces potential shared method variance and recall/social desirability biases. Finally, information on psychological or pharmacological treatment received during the follow-up interval was not systematically collected, so the observed improvements cannot be unambiguously attributed to natural developmental processes versus intervention effects.

Despite these constraints, the study advances the literature by demonstrating differential longitudinal roles for emotion regulation change (proximal predictor of severity) and intolerance of uncertainty (distal predictor of behavioral persistence) in a high-risk adolescent sample. Future research should replicate these patterns in more diverse and larger cohorts, incorporate multi-informant and behavioral measures, examine longer follow-up periods, and test whether IU-targeted interventions can reduce NSSI recurrence above and beyond regulation-focused treatments. Such work will be critical for developing more precise, mechanism-informed prevention and intervention strategies for adolescent self-injury.

Table 1. Paired-Samples t-Tests Comparing T1 and T2 Scores

Variable	T1 Mean (SD)	T2 Mean (SD)	*t*(41)	*p*	Cohen's *d*
NSSI Autonomous Function	19.24 (7.15)	11.74 (8.40)	−5.26	< .001	−0.81
NSSI Social Function	12.21 (8.15)	7.38 (7.32)	−3.62	< .001	−0.56
NSSI Total	31.45 (13.84)	19.12 (14.66)	−4.88	< .001	−0.75
IUI Inhibitory	33.86 (8.39)	28.62 (8.74)	−2.91	.006	−0.45
IUI Prospective	23.12 (5.93)	19.90 (6.37)	−2.63	.012	−0.41
IUI Total	56.98 (13.15)	48.52 (14.34)	−2.98	.005	−0.46
ERSA Internal Dysfunctional	20.43 (3.12)	17.02 (5.01)	−4.17	< .001	−0.64
ERSA External Dysfunctional	14.81 (4.65)	12.83 (4.29)	−2.30	.026	−0.36
ERSA Internal Functional	14.07 (3.46)	14.29 (3.24)	0.36	.725	0.05

Table 2. Multiple Linear Regression Predicting NSSI Total at T2 Predictor B SE t p

Predictor	B	SE	*t*	*p*
Age	-1.56	1.59	-0.99	.331
NSSI Total T1	0.44	0.14	3.21	.003
IUI Total T1	0.00	0.15	0.01	.989
ΔERSA International Dysfunctional	0.93	0.42	2.19	.035
ΔERSA External Dysfunctional	0.69	0.40	1.73	.092

Model fit: $R^2 = .43$, Adjusted $R^2 = .35$, $F(5, 36) = 5.35$, $p < .001$.

Table 3. Binomial Logistic Regression Predicting NSSI Presence During the Last Three Months at Follow-up (T2)

Predictor	B	SE	z	p	Odds Ratio	95% CI for OR
Intercept	-9.23	4.21	-2.20	.028	0.0001	[0.00, 0.37]
IUI-C Inhibitory (T1)	-0.03	0.07	-0.43	.665	0.97	[0.86, 1.10]
IUI-C Prospective (T1)	**0.26**	**0.11**	**2.34**	**.019**	**1.29**	**[1.04, 1.61]**
ERSA Internal Dysfunctional (T1)	0.30	0.17	1.75	.080	1.35	[0.97, 1.89]
ERSA External Dysfunctional (T1)	-0.19	0.10	-1.80	.071	0.83	[0.68, 1.02]
NSSI BAI Total (T1)	0.02	0.03	0.52	.603	1.02	[0.96, 1.08]

Note. Outcome variable coded as 0 = NSSI present, 1 = NSSI absent during the last three months at T2. Estimates represent log odds of NSSI absence. OR = odds ratio; CI = confidence interval.

Table 4. GLM Mediation Model Predicting NSSI Severity at Follow-Up

Effect Type	Path	Estimate	SE	95% CI Lower	95% CI Upper	β	Z	p
Indirect	IUI-C Total T1 → Δ ERSA Internal Dysf. → NSSI BAI Total T2	−0.129	0.089	−0.385	0.029	−0.116	−1.45	.148
Indirect	NSSI BAI Total T1 → Δ ERSA Internal Dysf. → NSSI BAI Total T2	−0.024	0.079	−0.267	0.136	−0.023	−0.31	.758
Path a	IUI-C Total T1 → Δ ERSA Internal Dysf.	−0.094	0.061	−0.225	0.035	−0.234	−1.56	.119
Path a	NSSI BAI Total T1 → Δ ERSA Internal Dysf.	−0.018	0.058	−0.151	0.118	−0.046	−0.31	.758
Path b	Δ ERSA Internal Dysf. → NSSI BAI Total T2	1.369	0.353	0.519	2.028	0.494	3.87	< .001
Direct	IUI-C Total T1 → NSSI BAI Total T2	0.008	0.143	−0.291	0.358	0.008	0.06	.953
Direct	NSSI BAI Total T1 → NSSI BAI Total T2	0.396	0.132	0.038	0.710	0.374	3.01	.003
Total	IUI-C Total T1 → NSSI BAI Total T2	−0.121	0.164	−0.434	0.306	−0.108	−0.74	.460
Total	NSSI BAI Total T1 → NSSI BAI Total T2	0.372	0.155	−0.005	0.750	0.351	2.40	.017

Note. Confidence intervals were estimated using bias-corrected bootstrap resampling. β values represent completely standardized effect sizes.

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0282 - Self-Acceptance, Emotion Dysregulation, and Emotional Eating in Adolescents with Major Depressive Disorder: A Path-Analytic Investigation

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Objective

Major Depressive Disorder (MDD) during adolescence is associated with impairments in emotional processing and regulation, which may contribute to maladaptive coping behaviors such as emotional eating. Emotion dysregulation has been widely implicated in the development and maintenance of depressive symptoms and eating-related difficulties in youth [1]. However, the psychological mechanisms linking emotion regulation deficits to eating behaviors remain insufficiently understood.

Self-acceptance, a core component of mindfulness and psychological well-being, refers to a non-judgmental and accepting attitude toward one's internal experiences and personal characteristics. Reduced self-acceptance has been associated with depression, shame, and maladaptive coping behaviors in adolescents [2]. Self- acceptance may function as a protective factor by facilitating more adaptive emotion regulation processes [3].

Limited research has examined the role of self-acceptance in the relationship between emotion regulation and emotional eating among adolescents with MDD. Understanding this relationship may help identify psychological processes that contribute to maladaptive eating behaviors in depressed youth and inform targeted interventions.

Therefore, the present study aimed to investigate the relationships among self- acceptance, emotion regulation strategies, depressive symptoms, and emotional eating in adolescents with MDD compared with healthy controls. Specifically, we hypothesized that adolescents with MDD would demonstrate lower levels of self- acceptance and greater emotion dysregulation compared with healthy peers. Furthermore, we examined the potential mediating and moderating roles of self- acceptance in the relationship between dysfunctional emotion regulation and emotional eating.

Methods

Participants and Study Design

This study employed a cross-sectional case-control design with a total of 88 adolescents aged 12-18 years, including 44 adolescents diagnosed with Major Depressive Disorder (MDD) and 44 healthy control participants. Clinical participants were recruited from the outpatient child and adolescent psychiatry clinic at Dokuz Eylul University Faculty of Medicine, while healthy controls were recruited from non- psychiatric pediatric settings, including general pediatrics, surgical outpatient clinics, and well-child clinics. All participants underwent a comprehensive psychiatric assessment using the Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children - Present and Lifetime Version (K-SADS-PL-DSM-5), administered by trained child and adolescent psychiatrists, to confirm MDD diagnoses and ensure the absence of psychiatric disorders in healthy controls. Adolescents in the clinical group were non-medicated at the time of assessment, minimizing potential confounding effects of psychotropic medications. Exclusion criteria included neurological disorders, intellectual disability, or chronic medical illnesses. Depressive symptom severity was assessed in all participants using the Children's Depression Rating Scale-Revised (CDRS-R).

Measures

Sociodemographic Data Form. Developed by the researchers, this form collected data on participants' sociodemographic characteristics and medical history.

Kiddie Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version, DSM-5 (K-SADS-PL-DSM-5). This semi- structured interview (Kaufman et al., 1997; Ünal et al., 2019) confirmed MDD diagnoses in the clinical group and ensured the control group was psychiatrically healthy.

Children's Depression Rating Scale-Revised (CDRS-R). A 17-item clinician-rated scale measuring depressive severity (Poznanski & Mokros, 1996; Güney et al., 2018).

Adolescent and Adult Mindfulness Scale (AAMS). This self-report instrument assesses dispositional mindfulness (Droutman et al., 2018). The Turkish adaptation (Sarıçam & Çelik, 2018) includes four subscales: Attention/Awareness, Non- Judgment, Non-Reactivity, and Self-Acceptance.

Regulation of Emotions Questionnaire for Adolescents (REQ). This measure assesses internal and external functional and dysfunctional emotion regulation strategies (Phillips & Power, 2007; Yıldız & Duy, 2014).

Emotional Eating Scale for Children and Adolescents (EES-C). A 25-item scale measuring the urge to eat in response to anxiety, anger, or depression (Tanofsky- Kraff et al., 2007; Bektaş et al., 2016).

Eating Attitudes Test (EAT-26). This 26-item questionnaire assesses dieting, bulimia, and oral control (Garner et al., 1982; Savaşır & Erol, 1989).

Statistical Analysis

All analyses were conducted using SPSS. Group differences were assessed with independent samples t-tests or Mann-Whitney U tests for continuous variables and chi-square tests for categorical variables. Pearson correlations examined associations among study variables within the MDD group. Mediation and moderation analyses were performed to evaluate the role of self-acceptance in predicting emotional eating. Significance was set at $p < .05$.

Results

Demographic and Clinical Characteristics

The MDD and control groups did not differ significantly in terms of mean age. However, the proportion of females was significantly higher in the MDD group compared with the control group ($x2 = 9.03$, $p = .003$). No significant differences were observed in adolescent education level, parental education level, or family structure.
A family history of psychiatric illness was significantly more frequent in the MDD group (29.5%) compared with controls (9.1%) ($x2 = 5.43$, $p = .020$). As expected, adolescents in the MDD group had significantly higher depressive symptom severity scores on the CDRS-R compared with healthy controls ($M = 67.4$, $SD = 8.5$, $t = 16.34$, $p < .001$).

Group Comparisons on Psychological Measures

Adolescents with MDD demonstrated significantly lower self-acceptance scores on the AAMS compared with the control group ($t = 3.38$, $p = .001$). No significant differences were observed between groups on other mindfulness subscales, including attention/awareness, non-reactivity, and non-judgment.
Regarding emotion regulation strategies, adolescents with MDD reported significantly greater use of internal dysfunctional regulation strategies ($t = 7.31$, $p < .001$) and external dysfunctional strategies ($t = 3.48$, $p = .001$) compared with healthy controls. Conversely, the control group reported significantly greater use of external functional regulation strategies ($t = -3.10$, $p = .003$).
Eating-related behaviors revealed that adolescents with MDD scored significantly higher on the oral control subscale of the EAT-26 ($U = 713$, $p = .047$). However, no significant group differences were observed in total EAT-26 scores or other subscales.

Correlational Analyses

Within the MDD group, depressive symptom severity was negatively correlated with the non-judgment dimension of mindfulness ($r = -.406$, $p = .006$). Additionally, depressive severity showed strong positive correlations with internal dysfunctional emotion regulation strategies ($r = .637$, $p < .001$) and moderate positive correlations with external dysfunctional strategies ($r = .355$, $p = .001$).

Unexpectedly, depressive severity was negatively associated with emotional eating behaviors.

Moderation and Mediation Analyses

Path analysis results conducted on MDD (n = 44) and control (n = 44) groups revealed that the relationship between internal dysfunctional emotion regulation and emotional eating is specific to the MDD group and is driven by a significant direct effect (c' = -1.46, p = .040) rather than a mediated pathway. While internal dysfunctional emotion regulation predicted lower self-acceptance in the MDD group (a = -0.33, p = .014), the indirect effect through self-acceptance was non-significant (p = .929), a finding further supported by the Sobel test. In the control group, no significant paths were observed. Additionally, moderation analysis confirmed that self-acceptance did not moderate the relationship between emotion regulation and emotional eating (B = 20.341, p > .05), with simple slope comparisons and interaction plots indicating that the impact of dysfunctional regulation remains consistent regardless of self-acceptance levels.

Discussion

Adolescents with MDD in this study demonstrated significantly lower mindfulness, with the most pronounced deficit observed in self-acceptance. They also reported a higher reliance on maladaptive emotion regulation strategies and increased oral control compared to healthy peers. While self-acceptance was hypothesized to function as a bridge between emotional processing and eating behaviors, path and moderation analyses indicated that it did not mediate or moderate the link between emotion regulation and emotional eating. Instead, a significant direct effect of internal dysfunctional emotion regulation on emotional eating was observed exclusively in the MDD group. This suggests that for depressed adolescents, the urge to eat in response to emotions is a proximal, reactive byproduct of regulatory failure rather than a process filtered through self-evaluative schemas [4,5].

The observed dissociation between cognitive vulnerabilities (low self-acceptance) and behavioral outcomes (emotional eating) highlights the complexity of depressive symptoms in youth. Although the MDD group had lower overall emotional eating scores—potentially due to melancholic features like anhedonia and reduced reward sensitivity [6] —the direct association found only in this group underscores a clinical "tight coupling." In non-clinical populations, emotional eating may serve as a common, albeit suboptimal, coping mechanism. In contrast, within the MDD context, it appears to be a specific manifestation of an inability to manage internal affective states [7].

Furthermore, the negative relationship between depressive severity and emotional eating in this sample differs from some earlier literature but aligns with models emphasizing symptom heterogeneity. In more severe clinical cases, psychomotor slowing and appetite loss may naturally attenuate emotional eating behaviors. Clinically, these results suggest that while fostering self-acceptance remains vital for overall psychological well-being [8], interventions specifically targeting maladaptive emotion regulation may be more effective for addressing disordered eating patterns in depressed adolescents [9].

Keywords: Major Depressive Disorder, Adolescents, Emotion Regulation, Emotional Eating, Self-Acceptance

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Table 1. Demographic and Clinical Characteristics of Participants

Characteristic	MDD Group (n=44)	Control Group (n=44)	Test Statistic	p-value
Age (years), M (SD)	14.9 (1.5)	14.5 (1.6)	t = 1.05	.297
Gender (Female, %)	84.1%	54.5%	X^2 = 9.03	.003
Family Income (Low, %)	36.3%	52.2%	X^2 = 7.61	.024
Family History of Psychiatric Illness (%)	29.5%	9.1%	X2 = 5.43	.020
Family History of Chronic Illness (%)	40.9%	36.3%	X2 = 0.14	.708
Adolescent History of Surgery (%)	18.1%	50.0%	X2 = 9.91	.002
Maternal Age, M (SD)	44.4 (5.1)	40.4 (6.3)	U = 460.0	<.001
Paternal Age, M (SD)	48.1 (6.1)	45.4 (7.5)	U = 592.0	.032
CDRS-R Total Score, M (SD)	67.4 (8.5)	41.0 (6.8)	t = 16.34	<.001

Note: Independent samples t-tests were used for normally distributed variables (Age, CDRS-R); Mann-Whitney U tests for non-normally distributed variables (Maternal/Paternal Age); and Chi-square (χ2) for categorical variables.

CDRS-R: Children's Depression Rating Scale-Revised. Bold values indicate p < .05.

MDD: Major Depressive Disorder

Table 2. Comparison of Mindfulness, Emotion Regulation and Emotional Eating Attitudes Between MDD and Control Groups

Variable / Scale	MDD Group (M ± SD)	Control Group (M ± SD)	Test Statistic (t/U/χ^2)	p-value
Mindfulness (AAMS)				
Attention/Awareness	32.3 ± 6.7	31.0 ± 8.7	t = 0.82	**.414**
Non-Reactivity	8.4 ± 3.3	9.7 ± 3.2	t = 1.80	.076
Non-Judgement	10.4 ± 3.9	10.9 ± 3.6	t = 0.64	.522
Self-Acceptance	8.0 ± 3.4	10.4 ± 3.1	t = 3.38	**.001**
Total	59.0 ± 9.3	61.8 ± 8.3	t = 1.49	.139
Emotion Regulation (REQ)				
Internal Functional	14.7 ± 3.5	13.7 ± 3.3	t = 1.31	.194
External Functional	9.9 ± 2.9	12.0 ± 3.3	t = -3.10	**.003**
Internal Dysfunctional	17.7 ± 3.8	11.4 ± 4.2	t = 7.31	**<.001**
External Dysfunctional	11.5 ± 4.4	8.4 ± 3.5	t = 3.48	**.001**
Emotional Eating (EES)				
Feeling Unsettled	11.8 ± 4.6	14.6 ± 4.8	t = 2.66	**.009**
Anxiety/Anger/Frustration	23.4 ± 10.4	29.3 ± 9.4	t = 2.74	**.007**
Depressive Symptoms	14.9 ± 3.8	16.3 ± 4.7	t = 1.45	.151
Total	50.4 ± 16.8	60.0 ± 16.1	t = 2.70	**.008**
Eating Attitudes (EAT-26)				
Oral Control	5.2 ± 4.2	3.6 ± 3.9	U = 713	**.047**
Bulimia	3.0 ± 3.6	2.0 ± 2.1	U = 817	.262
Diet	7.6 ± 8.5	6.0 ± 5.1	U = 882	.580
Total	16.0 ± 11.5	11.6 ± 7.9	U = 722	.057
Eating Behavior				
Abnormal Eating Behavior, n (%)	12 (27%)	9 (20%)	χ2= 0.66	.417
Normal Eating Behavior, n (%)	31 (70%)	35 (79%)		

Note: T-tests used for normally distributed (Mindfulness, Emotion Regulation, Emotional Eating) and Mann-Whitney U for non-normally distributed (EAT-26 subscales/total) variables. Chi-square for categorical data. Bold: p < .05.

MDD: Major Depressive Disorder

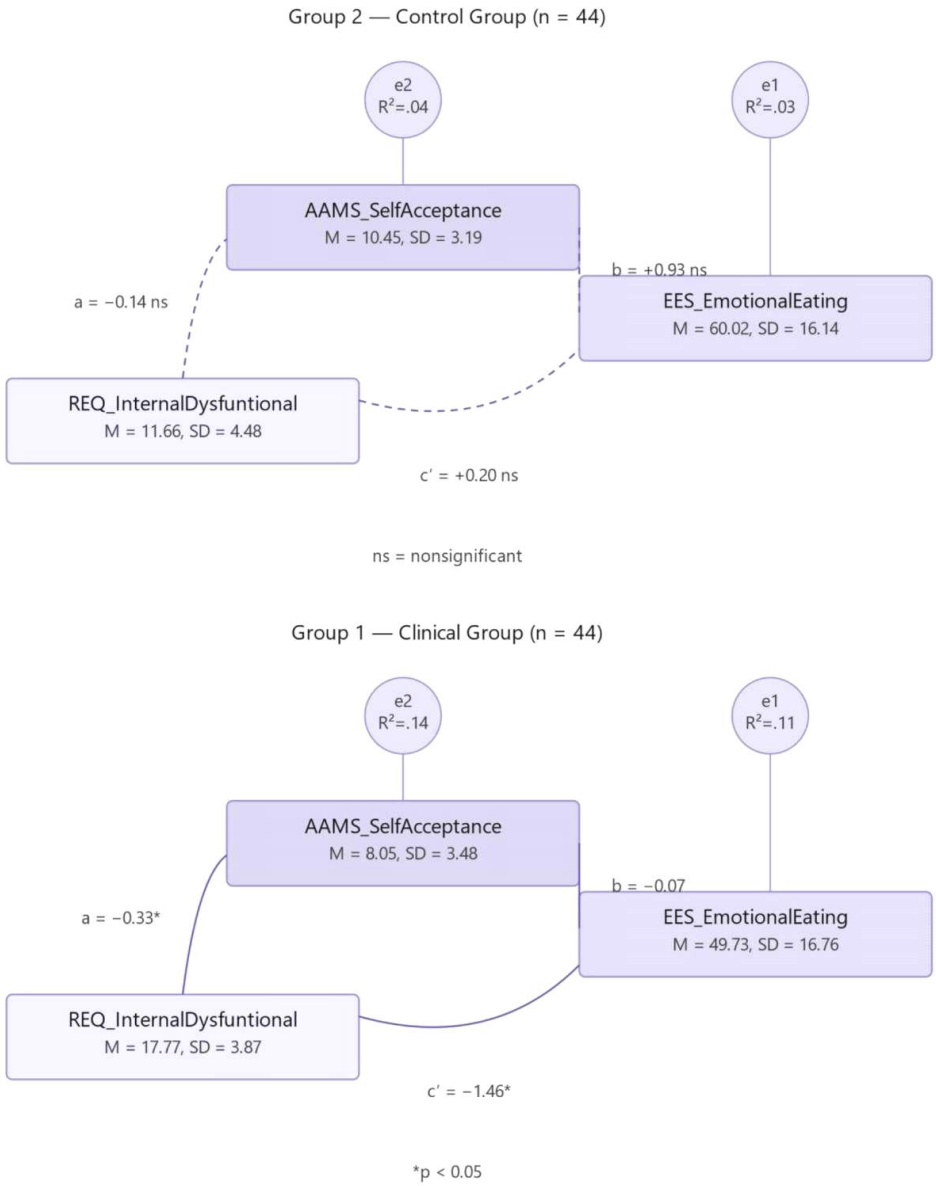
Table 3. Correlations Among Key Variables in the MDD Group (n=44)

Variable	CDRS-R Score
AAMS - Non-Judgement	-.406**
REQ - Internal Dysfunctional	.637***
REQ - External Dysfunctional	.355**
EES - Total Score	-.290*

Note: MDD: Major Depressive Disorder, CDRS-R: Children's Depression Rating Scale-Revised, AAMS: Adolescent and Adult Mindfulness Scale, REQ: Regulation of Emotions Questionnaire, EES: Emotional Eating Scale.

*ρ < .05, **ρ < .01, ***ρ < .001

Figure 1. Mediation pathways of internal dysfunctional emotion regulation predicting emotional eating via self-acceptance in MDD and control groups

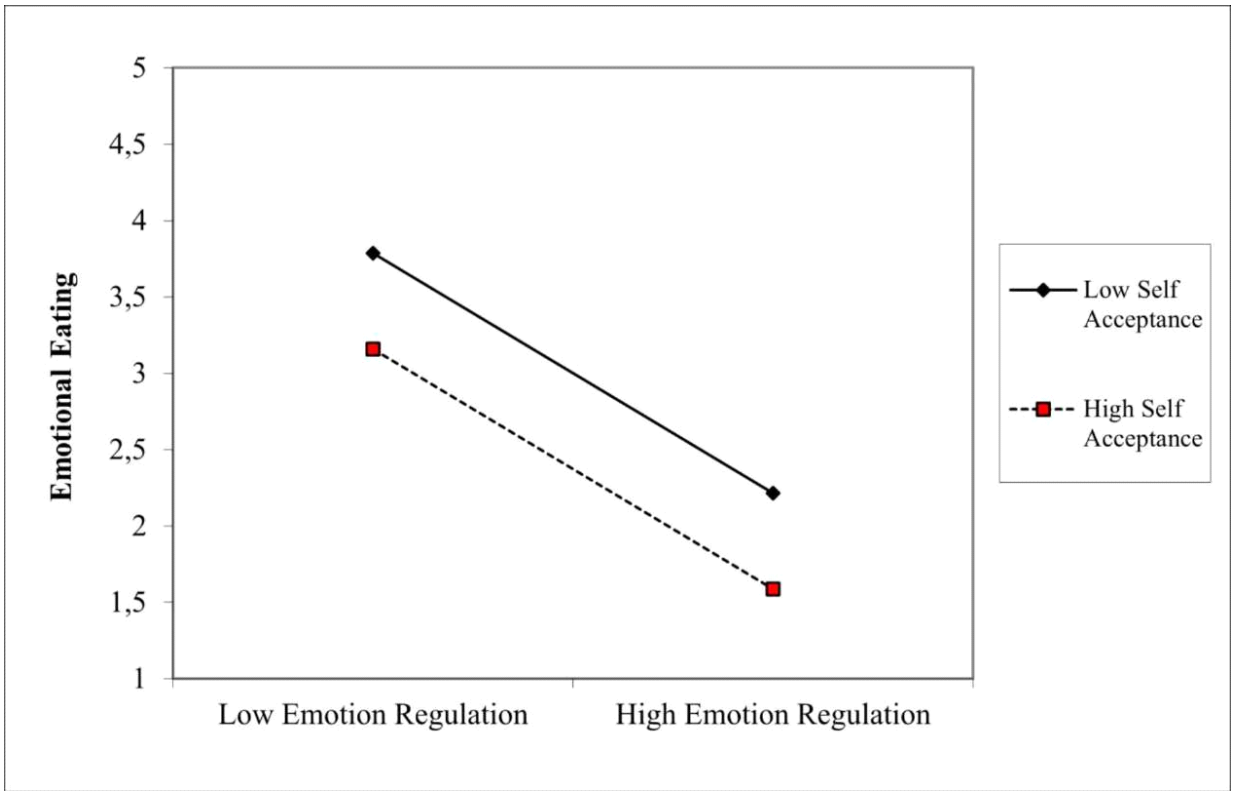


Note: AAMS: Adolescent and Adult Mindfulness Scale, REQ: Regulation of Emotions Questionnaire, EES: Emotional Eating Scale. *p<0.05

Clinical group: Indirect effect = 0.02 — Mediation not supported (path b nonsignificant, p = .930)

Control Group: Indirect effect = -0.13 — All paths non-significant; model not supported in this group

Figure 2. Relationship Between Emotion Regulation Emotional Eating and Self Acceptance in Terms of Regression Weights



0286 - Mirtazapine Induced Visual Hallucinations in an Adolescent

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Objective

Mirtazapine is an atypical antidepressant frequently used in the treatment of depressive disorders, anxiety disorders and insomnia. Its pharmacological profile includes antagonism of presynaptic a2-adrenergic receptors and postsynaptic serotonergic receptors, resulting in increased noradrenergic and serotonergic neurotransmission. In addition, its potent antagonistic effect on histamine H1 receptors contributes to its sedative properties and its frequent use in patients with insomnia and decreased appetite. Due to these characteristics, mirtazapine is often considered a well tolerated and relatively safe antidepressant in both adult and adolescent populations. Despite its favorable safety profile, rare neuropsychiatric adverse reactions have been described in the literature. These reactions include agitation, delirium, hallucinations and psychotic symptoms. Such paradoxical effects may occur in vulnerable individuals and can pose diagnostic challenges, particularly in patients with complex psychiatric presentations.

In adolescent populations, where psychotropic medications are often used in combination and where neurodevelopmental factors may influence pharmacodynamic responses, recognition of medication induced neuropsychiatric symptoms especially important.

Visual hallucinations represent an uncommon but clinically significant adverse effect associated with several psychotropic medications. When hallucinations occur shortly after medication initiation, clinicians must differentiate between primary psychotic disorders, substance related effects, neurological conditions and medication induced reactions. Early recognition of drug related causes is essential in order to prevent unnecessary diagnostic investigations and inappropriate treatment interventions.

The aim of the present brief report is to describe a case of acute visual hallucinations emerging shortly after the initiation of low-dose mirtazapine in an adolescent patient with multiple psychiatric diagnoses. The report also aims to highlight the importance of considering paradoxical neuropsychiatric reactions during pharmacological treatment in vulnerable youth.

Methods

The patient was a 14-year-old female followed in a child and adolescent psychiatry outpatient clinic. Psychiatric evaluation was conducted through comprehensive clinical interviews and mental status examinations in accordance with DSM-5 diagnostic criteria. The patient had previously been diagnosed with attention-deficit/hyperactivity disorder (ADHD), conduct disorder, recurrent mild depressive episodes, and borderline personality traits. The patient had a history of multiple pharmacological treatment changes due to recurrent and refractory behavioral and emotional symptoms. Previous medications included methylphenidate, lamotrigine, risperidone, haloperidol and fluoxetine. These treatments had been discontinued because of insufficient therapeutic response or limited clinical benefit.

At the time of presentation, the patient was receiving aripiprazole at a dose of 7.5 mg/day and sertraline at a dose of 50 mg/day. In this treatment combination, the patient experienced partial improvement in mood symptoms but reported no benefit for sleep onset difficulties and loss of appetite. These symptoms negatively affected her daily functioning and overall well-being. To reduce these complaints, mirtazapine 7.5 mg was added to the treatment regimen before bedtime.

Following the initiation of mirtazapine, the patient developed increased psychomotor activity, restlessness, and reported visual hallucinations. For this reason, the patient was placed under close clinical monitoring. A comprehensive clinical evaluation was performed, including detailed psychiatric assessment, physical examination and neurological examination. Laboratory investigations were performed to exclude potential metabolic, neurological or infectious causes that might contribute to the emergence of new neuropsychiatric symptoms. Information regarding symptom onset, duration and progression was obtained from both the patient and her

caregivers. Particular attention was paid to the temporal association between medication exposure and the onset of hallucinations.

Results

Within approximately 24 hours following the first administration of mirtazapine, the patient began to experience acute behavioral and perceptual changes. She developed increased agitation and reported the presence of vivid visual hallucinations. During clinical assessment, the patient described seeing "moving figures and distorted shapes" in her environment. These perceptual experiences were distressing for the patient and represented a marked change from her previous psychiatric symptom profile.

The patient had no prior history of psychotic symptoms, hallucinations, or perceptual disturbances. There was also no history of substance use or exposure to psychoactive substances that might account for these symptoms.

Family members confirmed that similar symptoms had never been observed before the initiation of mirtazapine. Physical examination and neurological assessment revealed no abnormalities. The patient was alert and oriented, and no focal neurological deficits were detected. Routine laboratory tests did not demonstrate any metabolic abnormalities or evidence of infection that could explain the emergence of hallucinations. Considering the close temporal relationship between the initiation of mirtazapine and the onset of visual hallucinations, a medication-induced adverse reaction was suspected.

Consequently, mirtazapine was discontinued. Following discontinuation, the patient's hallucinations resolved completely within a short period of time. No further hallucinations or psychotic symptoms were reported during subsequent follow-up visits. The patient continued her existing treatment regimen of aripiprazole and sertraline without recurrence of perceptual disturbances. This clinical course supported the interpretation that the hallucinations were likely associated with the initiation of mirtazapine rather than with an underlying primary psychotic disorder.

Discussion

Mirtazapine is widely considered a relatively safe antidepressant with a favorable side-effect profile. However, rare neuropsychiatric adverse reactions have been documented in the literature. Hallucinations and psychotic symptoms associated with mirtazapine remain uncommon but clinically relevant phenomena that may occur in susceptible individuals. Previous reports have described cases of hallucinations emerging during mirtazapine treatment, particularly in elderly patients. For example, multimodal hallucinations have been reported following the initiation of mirtazapine in older individuals, suggesting that certain biological vulnerabilities may predispose patients to paradoxical neuropsychiatric reactions [1]. Similar reactions have also been described in adolescents under specific vulnerability conditions, including severe malnutrition and complex psychiatric presentations [2].

The neurobiological mechanisms underlying mirtazapine-induced hallucinations are not fully understood. Mirtazapine exerts complex effects on multiple neurotransmitter systems, particularly serotonergic and noradrenergic pathways. Antagonism at 5-HT2 and 5-HT3 receptors combined with enhanced noradrenergic transmission may contribute to changes in sensory processing and cortical activation patterns. In certain individuals, these pharmacodynamic effects may lead to paradoxical perceptual disturbances. Interestingly, the literature also contains reports suggesting that mirtazapine may reduce hallucinations in some neurological disorders. For instance, improvement of visual hallucinations has been observed in patients with Parkinson's disease treated with mirtazapine [3]. These contrasting observations suggest that the drug's effects on perceptual processes may be highly context dependent and influenced by individual neurobiological characteristics.

Another rare neuropsychiatric adverse effect reported in association with mirtazapine is the emergence of motor tics in adolescents [4]. Such findings further support the notion that mirtazapine may produce unexpected neuropsychiatric reactions in certain vulnerable individuals, particularly during neurodevelopmental periods. Several factors may have contributed to the adverse reaction observed in the present case. First, the patient had a complex psychiatric profile characterized by multiple comorbid diagnoses. Second, she had a history of exposure to several psychotropic medications, which may have influenced neurochemical sensitivity. Third, adolescence represents a developmental period during which neurobiological systems involved in emotional regulation and sensory processing continue to mature.

The rapid onset of hallucinations following the first dose of mirtazapine and the complete resolution of symptoms after discontinuation strongly suggest a causal relationship between the medication and the observed adverse effect. The absence of alternative medical or neurological explanations further supports this interpretation. From a clinical perspective, this case highlights the importance of careful monitoring when initiating psychotropic medications in adolescents, even when low doses are used. Clinicians should remain vigilant for the emergence of unusual neuropsychiatric symptoms and consider medication-related causes when such symptoms appear shortly after treatment initiation. Early identification of medication-induced hallucinations can prevent unnecessary diagnostic procedures and reduce the risk of misdiagnosing patients with primary psychotic disorders. Prompt discontinuation of the causative drug typically results in rapid resolution of symptoms, as demonstrated in the present case.

In conclusion, the present report underscores that mirtazapine may rarely induce acute visual hallucinations in adolescents, even at low doses. Awareness of this potential adverse reaction is important for clinicians working with pediatric psychiatric populations. Further research is needed to better understand the mechanisms underlying such paradoxical responses and to identify potential risk factors associated with medication-induced hallucinations in vulnerable individuals.

Keywords: Adolescent psychiatry, Mirtazapine, Side effects, Visual hallucinations

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0291 - The Investigation of Attention Deficit Hyperactivity Disorder Comorbidity and Risk Factors in Children Diagnosed with Congenital Heart Diseae

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Background and Objective: Attention Deficit Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder characterized by persistent inattention, hyperactivity, and impulsivity inconsistent with developmental level, emerging in early childhood and often continuing into adulthood, impairing psychosocial functioning. Congenital heart disease (CHD) and its surgical treatment are established risk factors for ADHD, with neurodevelopmental disorder frequency and severity increasing with CHD complexity. This study aims to evaluate ADHD comorbidity prevalence in CHD patients and identify contributing risk factors, including sociodemographic and surgical characteristics.

Methods: The study included 112 children and adolescents (6-18 years) with CHD and a healthy control group with no cardiac pathology. Participants were categorized into: cyanotic CHD (n=37), operated acyanotic CHD (n=37), non-operated acyanotic CHD (n=38), and healthy controls (n=37). ADHD diagnosis was established using the K-SADS- PL for DSM-5. Symptom severity was assessed via the Conners' Parent (CPRS) and Teacher (CTRS) Rating Scales. The Screen for Child Anxiety Related Emotional Disorders (SCARED) was used to evaluate anxiety's confounding effect. Data on heart disease type, surgeries, and clinical features were analyzed using SPSS 20.0. Ethics Committee Approval: March 13, 2025; Kocaeli University Faculty of Medicine Non-Interventional Clinical Research Ethics Committee (GOKAEK), approval number: 2025/07/04, decision no: 2025/136.

Results: Among participants diagnosed with CHD, 34.8% met diagnostic criteria for ADHD, whereas 65.2% did not receive an ADHD diagnosis. In contrast, only 10.8% of participants in the healthy control group met criteria for ADHD, while 89.2% did not. The prevalence of ADHD was significantly higher in children with CHD compared with the healthy control group (p=0.005).

When ADHD diagnosis status was compared across these groups, a statistically significant difference was observed ($\chi^2(3)=11.146$; p=0.011). The highest ADHD prevalence was observed in the operated acyanotic CHD group (45.9%). This was followed by the cyanotic group (29.7%) and the non-operated acyanotic group (28.9%). The healthy control group demonstrated a substantially lower ADHD prevalence (10.8%).

Clinical evaluations conducted using the K-SADS-PL structured interview revealed significant differences in the median number of inattentive symptoms across the study groups (p=0.007). Inattentive symptoms were particularly prominent in the operated acyanotic CHD group. Additionally, both cyanotic and non-operated acyanotic CHD groups also showed higher inattentive symptom levels compared with the control group.

Similarly, the total number of ADHD symptoms differed significantly across groups (p=0.007). Total symptoms were particularly prominent in the operated acyanotic In contrast, the median number of hyperactivity-impulsivity symptoms did not significantly differ between groups (p=0.101).

When ADHD subtype distributions were examined, no statistically significant differences were observed across groups (p=0.052).

Because anxiety symptoms may influence attentional functioning, additional analyses were conducted controlling for anxiety severity using the Screen for Child Anxiety Related Emotional Disorders (SCARED). After controlling for anxiety scores, significant differences between groups remained for inattentive symptom counts (p=0.004) and total ADHD symptom counts (p=0.015). Bonferroni-corrected pairwise comparisons indicated that these

differences were primarily driven by the higher symptom levels observed in the operated acyanotic CHD group compared with the control group. No significant differences were observed between groups in hyperactivity-impulsivity symptom counts after controlling for anxiety.

Parent-reported Conners' Parent Rating Scale-Revised Short Form (CPRS-RS) scores revealed significant differences in the Cognitive Problems/Inattention subscale (p=0.007) and total scores (p=0.032). Post-hoc comparisons showed that children in all CHD groups had higher cognitive problems and inattention scores than those in the healthy control group.

Teacher-reported Conners' Teacher Rating Scale-Revised Short Form (CTRS-RS) results showed significant differences in the Hyperactivity subscale (p=0.015) and total scores (p=0.044). Post-hoc analyses indicated that children with CHD had higher hyperactivity and overall behavioral problem scores compared with controls.

However, no significant differences were found between groups regarding total SCARED anxiety scores (p=0.944).

When ADHD diagnosis was examined in participants with congenital heart disease (CHD) according to sociodemographic characteristics, no statistically significant differences were found between participants with and without ADHD in terms of sex, birth timing, birth weight, place of residence, family structure, number of siblings, parental consanguinity, family income level, or parental education levels (p>0.05). Similarly, the presence of a physical illness in the father was not significantly associated with ADHD diagnosis (p>0.05).

However, significant differences were observed between participants with and without ADHD regarding school performance and the presence of a physical illness in the mother (p<0.05).

For continuous variables, including age, body mass index (BMI), maternal age, paternal age, duration of hospitalization in the cardiology service, age at first reconstructive surgery, age at first cardiac surgery, age at first cardiopulmonary bypass, and duration of intensive care unit stay, no statistically significant differences were found between participants with and without ADHD (p>0.05).

A multivariable logistic regression analysis was performed to evaluate factors associated with ADHD diagnosis. Age, sex, birth weight, congenital heart disease (CHD) groups, and anxiety level (SCARED score) were included in the model. The overall model was statistically significant (Omnibus test: $\chi^2(8)=25.94$; p=0.001). The explanatory power of the model was modest (Cox & Snell $R^2=0.160$; Nagelkerke $R^2=0.228$). The overall classification accuracy of the model was 77.9%, with a higher accuracy in correctly classifying individuals without ADHD (95.3%).

According to the multivariable analysis, individuals with high anxiety risk (SCARED >25) had a significantly higher likelihood of receiving an ADHD diagnosis (OR=4.06; 95% CI: 1.71-9.68; p=0.002). When CHD groups were examined, the likelihood of ADHD diagnosis was approximately nine times higher in the operated acyanotic CHD group compared with the healthy control group (OR=9.31; 95% CI: 2.48-34.90; p=0.001). Similarly, significant associations with ADHD diagnosis were observed for the non-operated acyanotic CHD group (OR=5.14; 95% CI: 1.32-20.01; p=0.018) and the cyanotic CHD group (OR=5.08; 95% CI: 1.32-19.54; p=0.018). In contrast, age, sex, and birth weight were not significantly associated with ADHD diagnosis (p>0.05).

Conclusions: The findings of the present study demonstrate that children and adolescents with congenital heart disease show a markedly higher prevalence of ADHD compared with healthy controls. In particular, the operated acyanotic CHD group demonstrated the highest prevalence of ADHD diagnosis among all study groups. This finding suggests that both disease-related factors and treatment-related processes may contribute to neurodevelopmental vulnerability in children with congenital heart disease.

Across different assessment methods, including structured diagnostic interviews and parent and teacher rating scales, attentional difficulties emerged as the most prominent symptom domain. The predominance of inattentive symptoms rather than hyperactivity- impulsivity symptoms observed in this study is consistent with previous literature suggesting that cognitive and executive functioning difficulties may represent the primary neurobehavioral profile in children with congenital heart disease.

Another important finding of this study is the independent association between anxiety risk and ADHD diagnosis. The multivariable logistic regression analysis demonstrated that children with elevated anxiety levels had a

significantly higher likelihood of meeting diagnostic criteria for ADHD. This relationship highlights the potential interaction between emotional regulation difficulties and attentional functioning in this population.

Furthermore, the substantially increased odds of ADHD diagnosis observed in the operated acyanotic CHD group suggest that surgical treatment history and perioperative factors may play a role in shaping long-term neurodevelopmental outcomes. However, the presence of increased ADHD risk across multiple CHD subgroups also indicates that congenital heart disease itself may represent an important neurodevelopmental risk factor independent of surgical intervention.

Taken together, these findings emphasize the importance of routine neurodevelopmental and psychiatric monitoring in children with congenital heart disease. Early identification of ADHD symptoms may enable timely intervention and support, potentially improving academic, behavioral, and psychosocial outcomes in this population. To the best of our knowledge, this study represents one of the first investigations in Turkiye examining ADHD in children and adolescents with congenital heart disease. Furthermore, studies in the international literature that comprehensively evaluate ADHD using both structured clinical interviews and multi-informant (parent and teacher) rating scales remain scarce, highlighting the contribution of our findings to the existing body of knowledge.

Keywords: Attention Deficit Hyperactivity Disorder, Cyanosis, Congenital Heart Defects, Pediatric Cardiology, Neurodevelopment

0299 - Combined Use of Clozapine and Monthly Long-Acting Injectable Paliperidone Palmitate: A Case Report

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Objective

Treatment-resistant psychosis remains a major clinical challenge, particularly in patients with persistent symptoms and poor treatment adherence. Although clozapine is considered the gold standard for treatment-resistant schizophrenia [1,2], a substantial proportion of patients continue to experience residual psychotic symptoms and behavioral dysregulation. In such cases, augmentation strategies are frequently required [3,4].

Long-acting injectable (LAI) antipsychotics have been proposed as a strategy to improve adherence and reduce relapse risk [5]. However, evidence regarding the combined use of clozapine and LAI antipsychotics remains limited.

This report aims to present the clinical course, treatment response, and tolerability of combined clozapine and monthly paliperidone palmitate in a patient with late-onset psychosis characterized by prominent religious and grandiose delusions.

Methods

This study is a single-case report conducted in an inpatient psychiatric unit of a tertiary care university hospital in Turkiye.

A 50-year-old married woman with five children, living in a rural area, was admitted involuntarily due to severe psychotic symptoms and risk of harm to others. She had no prior psychiatric history or occupational experience. Premorbid personality traits were described as calm, cooperative, and compassionate.

Clinical assessment was performed using standardized psychiatric rating scales, including the Brief Psychiatric Rating Scale (BPRS), Positive Symptom Rating Scale (PSRS), and Negative Symptom Rating Scale (NSRS). A comprehensive medical evaluation, including laboratory tests, electroencephalography, brain magnetic resonance imaging, and dementia biomarkers, was conducted to exclude organic causes.

The treatment intervention consisted of initiating clozapine with gradual titration up to 300 mg/day. Monthly paliperidone palmitate injections were continued concurrently as part of a combination treatment strategy. The patient was monitored for clinical response and adverse effects throughout hospitalization.

Written informed consent was obtained from the patient and her relatives.

Results

The patient developed depressive symptoms following psychosocial stressors, including her children leaving home in 2022. After the February 6, 2023 Kahramanmara§ earthquake, she exhibited progressive psychotic symptoms, including religious-mystical, grandiose, and referential delusions. She reported receiving divine revelations and believed she had a mission to communicate them.

She had previously been treated with monthly paliperidone palmitate for approximately one year, with partial response. However, treatment was discontinued due to adverse effects, including extrapyramidal symptoms, akathisia, fatigue, and weight gain. After five months without treatment, her symptoms worsened significantly, with disorganized behavior and threats toward family members.

At admission, baseline scores were BPRS: 37, PSRS: 88, and NSRS: 16. No abnormalities were detected in laboratory or neuroimaging evaluations.

Following initiation of clozapine and continuation of monthly paliperidone palmitate, significant clinical improvement was observed. Auditory and visual hallucinations markedly decreased, and behavioral organization improved. The patient became more cooperative and better adapted to the inpatient setting.

At discharge, scores decreased to BPRS: 21 and PSRS: 34, indicating a substantial reduction in positive symptoms, while NSRS increased to 27, suggesting persistence of negative symptoms. Religious and grandiose delusions persisted but were less intense and less behaviorally disruptive.

In terms of tolerability, hypersalivation associated with clozapine was observed. No serious adverse events, including agranulocytosis, myocarditis, or major metabolic complications, were detected.

Discussion

This case demonstrates that the combination of clozapine and monthly long-acting injectable paliperidone palmitate may be an effective and well-tolerated treatment strategy in patients with treatment-resistant psychosis, particularly in the presence of adherence difficulties.

Clozapine is the most effective treatment for treatment-resistant schizophrenia; however, incomplete response is frequently observed [1,2]. Augmentation strategies, including the addition of a second antipsychotic, are commonly used despite limited high-quality evidence [3,4]. LAI antipsychotics may provide advantages by ensuring treatment continuity and maintaining stable plasma levels [5].

In this case, reintroduction of paliperidone palmitate in combination with clozapine resulted in significant improvement in hallucinations, behavioral regulation, and overall functioning, suggesting a potential synergistic effect. However, persistent delusional beliefs indicate that full remission was not achieved, which is consistent with existing literature on treatment- resistant psychosis.

The tolerability profile was favorable, with hypersalivation being the only notable adverse effect. No serious adverse events were observed, supporting the relative safety of this combination, although longer-term follow-up is necessary.

This case underscores the potential clinical utility of combining clozapine with long-acting injectable antipsychotics in complex and treatment-resistant presentations, particularly when adherence and behavioral stabilization are primary concerns. Nevertheless, the lack of controlled studies highlights the need for further research to clarify the efficacy, safety, and optimal patient selection for this combination strategy [6].

Keywords: Combination therapy; Clozapine; Long-acting injectable antipsychotics; Paliperidone palmitate; Religious delusions; Treatment-resistant psychosis

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0301 - Clinical Course and Treatment Response to the Two-Injection Start Regimen of Aripiprazole Long-Acting Injectable in Bipolar Mania with Psychotic Features: A Case Report

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Abstract

Background: The two-injection start (TIS) regimen of aripiprazole monohydrate long-acting injectable (AOM) allows rapid attainment of therapeutic plasma concentrations. Evidence regarding its clinical effectiveness in bipolar disorder remains limited.

Case Presentation: A 37-year-old woman with bipolar disorder presented with a manic episode accompanied by psychotic features. Because of poor treatment adherence and recurrent hospitalizations. a TIS regimen of aripiprazole LAI (two intramuscular injections of 400 mg administered on the same day) was initiated during hospitalization. Despite initiation of the TIS regimen. manic and psychotic symptoms persisted and adjunctive pharmacotherapy including divalproex and haloperidol was required. Over a 4-week follow-up period gradual clinical improvement was observed. The Young Mania Rating Scale score decreased from 47 at admission to 11 at discharge. and the Positive and Negative Syndrome Scale total score decreased from 88 to 54.

Conclusion: Although the TIS regimen provides pharmacokinetic advantages. rapid achievement of therapeutic plasma concentrations may not always result in immediate

clinical remission in severe manic episodes with psychotic features. Adjunctive treatments may still be required in complex clinical presentations.

Keywords: bipolar disorder; aripiprazole; long-acting injectable; two-injection start; mania

Introduction

Long-acting injectable antipsychotics are widely used to improve treatment adherence in severe psychiatric disorders.[1] Poor adherence to oral antipsychotic treatment is associated with relapse, hospitalization, and increased suicide risk.[2] Consequently, LAI formulations are increasingly recommended for patients with recurrent episodes or poor adherence.

Although LAI antipsychotics have been extensively investigated in schizophrenia, fewer studies have evaluated their effectiveness in bipolar disorder.[3,4] Available evidence suggests that LAIs may reduce relapse rates, emergency department visits, and hospitalization duration in bipolar disorder.[4,5]

Aripiprazole is a third-generation antipsychotic characterized by partial agonism at dopamine D2 receptors and serotonergic receptors.[6] Because of its favorable tolerability profile and lower metabolic risk compared with many second-generation antipsychotics, it has become an important therapeutic option in bipolar disorder.

The recently introduced two-injection start regimen allows rapid achievement of therapeutic plasma concentrations without the need for prolonged oral supplementation.[7,8] However, clinical data regarding its effectiveness during acute manic episodes remain limited.

Case Presentation

A 37-year-old married woman with one child was admitted with severe manic symptoms accompanied by psychotic features. During the preceding 20 days she developed persecutory and grandiose delusions, increased speech, irritability, reduced need for sleep, and behavioral disturbances.

The patient had been diagnosed with bipolar disorder at the age of 17 and had experienced multiple manic episodes requiring hospitalization. Previous treatments included lithium. divalproex. olanzapine. paliperidone. risperidone. and haloperidol. Several medications were discontinued due to adverse effects including weight gain and hyperprolactinemia.

Mental status examination revealed elevated mood. pressured speech. increased psychomotor activity. flight of ideas. persecutory and grandiose delusions. and absence of insight.

After the admission. treatment was initiated with oral aripiprazole 10 mg/day. divalproex 500 mg/day. and clonazepam 2 mg/day. The aripiprazole dose was increased gradually to 20 mg/day. On the fourth day of hospitalization two intramuscular injections of aripiprazole monohydrate 400 mg were administered as part of the TIS regimen.

Despite treatment. manic and psychotic symptoms persisted and adjunctive haloperidol therapy was introduced. Divalproex was titrated to 1500 mg/day and serum valproate levels reached 85 |ig/mL. Clinical rating scales demonstrated progressive improvement after the treatment adjustments. YMRS scores decreased from 47 at admission to 11 at discharge. and PANSS total scores decreased from 88 to 54.

The patient was discharged after 43 days of hospitalization with partial remission of manic and psychotic symptoms and scheduled outpatient follow-up.

Discussion

This case illustrates the clinical course of bipolar mania treated with the TIS regimen of aripiprazole long-acting injectable. Although the regimen offers pharmacokinetic advantages by rapidly achieving therapeutic plasma concentrations. clinical remission may still require additional pharmacological interventions.

Previous studies have reported rapid symptom improvement with the TIS regimen.8'9 However. the persistence of symptoms in our patient suggests that illness severity and psychotic features may influence treatment response. Adjunctive medications such as mood stabilizers or typical antipsychotics are frequently used in clinical practice.

Another important issue is the difference between pharmacokinetic efficacy and clinical effectiveness. While plasma concentrations may reach therapeutic levels rapidly, symptom improvement may depend on multiple factors including illness severity, neurobiological mechanisms, and individual pharmacodynamic responses.

The present case highlights the importance of individualized treatment strategies in bipolar disorder. The use of LAI antipsychotics may improve adherence and long-term outcomes, but clinicians should remain prepared to combine pharmacological approaches when managing complex manic episodes with psychotic features.

Limitations of this report include the single-case design and the concurrent use of additional medications, which makes it difficult to isolate the specific contribution of the TIS regimen. Larger prospective studies are needed to better evaluate the effectiveness of this strategy in bipolar disorder.

Data Availability Statement

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethics Statement

Written informed consent was obtained from the patient for publication of this case report.

Conflict of Interest

The authors declare no conflict of interest.

Funding

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0304 - Sleep Problems and Behavioral Symptoms in Preschool Children with Autism Spectrum Disorder: A Cross-sectional Study

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Objective:

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by deficits in social communication and repetitive behaviors. Sleep problems are common in children with ASD, may be overlooked in clinical practice, and can affect physical, behavioral, and emotional functioning (3). The relationship between sleep and behavioral problems may be bidirectional. This study aimed to examine sleep characteristics and their relationship with behavioral symptoms in preschool-aged children with ASD compared with typically developing peers.

Methods:

This cross-sectional study included 64 children aged 36-72 months diagnosed with ASD according to DSM-5 criteria and 58 healthy controls. Sleep characteristics were evaluated with the Children's Sleep Habits Questionnaire (CSHQ), and behavioral problems were assessed using the Aberrant Behavior Checklist (ABC) and the Child Behavior Checklist for Ages 1.5-5 (CBCL 1.5-5). Children with genetic, neurological, or metabolic disorders, psychiatric comorbidities, or psychotropic medication use within the past three months were excluded.

Results:

The median age in the ASD group was 54 months (IQR: 47.75-61.25), while in the control group it was 61 months (IQR: 52.0-65.0), with no significant age difference between the groups ($Z= -1.8251$, $p=0.0682$). A statistically significant difference was found between the groups in terms of gender distribution ($\chi2=7.1763$, $p=0.0074$)(Table 1).

As all sleep variables were non-normally distributed (Shapiro-Wilk, $p<0.05$), group comparisons were performed using the Mann-Whitney U test. There were no statistically significant differences between the ASD and control groups in terms of bedtime resistance, sleep onset delay, sleep duration, sleep anxiety, night wakening, parasomnias and sleep disordered breathing. However, the control group had significantly higher daytime sleepiness scores and total sleep scores compared to the ASD group. Effect sizes were generally small, except for daytime sleepiness, which showed a moderate effect size (Table 2).

When behavioral scales were compared, the ASD group scored higher on many subscales. Large effect sizes were found for attention problems ($d=1.1201$), externalizing problems ($d=1.0946$), aggressive behavior ($d=1.0143$), and hyperactivity ($d=0.9076$). Moderate-to-large effects were also observed for ABC total score and the social withdrawal/lethargy, anxiety/depression, and withdrawn subscales. No significant differences were found for inappropriate speech or CBCL sleep problems, and effect sizes were low (Table 3).

The relationship between sleep problems and behavioral problems in the ASD group was examined using Spearman correlation analysis. A strong positive correlation was found between the total sleep score and the CBCL sleep problem subscale ($\rho=0.5942$, $p<0.001$). In addition, moderate positive correlations were found between total sleep score and total CBCL score ($\rho=0.4583$, $p=0.0001$), internalizing problems ($\rho=0.3849$, $p=0.0017$), anxiety/depression ($\rho=0.3885$, $p=0.0015$), aggressive behavior ($\rho=0.3658$, $p=0.0030$), total ABC score ($\rho=0.3540$, $p=0.0041$), and externalizing problems ($\rho=0.3535$, $p=0.0042$). Weak but significant associations were also found with irritability, lethargy/social withdrawal, inappropriate speech, and hyperactivity. No significant association was found between total sleep score and the subscales of stereotypic behavior, withdrawn, and attention problems (Table 4).

In the regression analysis for CBCL total score, bedtime resistance was a significant predictor (B=5.86, t=5.00, p<0.001). Higher bedtime resistance scores were associated with higher CBCL total scores (Table 5).

Multiple linear regression analysis was conducted to examine the effect of sleep parameters on behavioral problems. The model created for the total ABC score was found to be significant (R^2=0.300, F=13.084, p<0.001). Bedtime resistance and sleep disordered breathing were identified as significant predictors of behavioral problems (Table 6).

Table 1: Sociodemographic characteristics of ASD and healthy control (HC)

Variable	ASD (n=64)	HC (n=58)	Statistic	p
Age (months)	54.0000 (47.7500-61.2500)	61.0000 (52.000065.0000)	Z = - 1.8251	0.0682
Sex			χ2 = 7.1763	0.0074
Boy	43 (67.1875%)	24 (41.3793%)		
Girl	21 (32.8125%)	34 (58.6207%)		

Table 2: Comparison of CSHQ scores between ASD and HC

Variable	ASD mean	HC mean	p	Cohen's d	Effect size (r)
Bedtime resistance	10.9219	10.6724	>0.0500	0.0961	Small
Sleep onset delay	1.3281	1.3103	>0.0500	0.0306	Small
Sleep duration	3.5469	3.8103	>0.0500	-0.2450	Small
Sleep anxiety	6.7344	7.1552	>0.0500	-0.2187	Small
Night wakening	4.4375	4.2414	>0.0500	0.1510	Small
Parasomnias	8.5938	8.3276	>0.0500	0.1507	Small
Sleep disordered breathing	3.6250	3.5345	>0.0500	0.0717	Small
Daytime sleepiness	8.9688	11.7759	<0.0001	-1.1154	Moderate
Total sleep score	44.1406	46.6897	0.0119	-0.3990	Small

Table 3: Comparison of behavioral problem scale scores between the ASD and HC

Scale	ASD mean	HC mean	Z	p	Cohen's d
ABC scale					
Irritability	8.6094	4.8103	3.5271	0.0004	0.5610
Hyperactivity	14.1562	6.1034	5.6060	<0.0001	0.9076
Stereotypic behavior	2.3281	0.8621	3.3246	0.0001	0.4705
Lethargy / Social withdrawal	5.8750	2.1207	5.1472	<0.0001	0.6999
Inappropriate speech	2.1250	1.7759	0.2640	0.7882	0.1542
ABC total score	34.3750	15.6724	5.5188	<0.0001	0.7989
CBCL scale					
Emotionally Reactive	1.8281	1.5517	1.1766	0.2393	0.1125
Anxiety/Depression	4.1719	2.0517	4.0629	<0.0001	0.6553
Somatic complaints	2.3125	1.7069	2.0942	0.0316	0.2141
Withdrawn	3.7969	1.3793	4.7934	<0.0001	0.7970
Sleep Problems	2.6719	2.4138	0.9125	0.3561	0.1017
Attention problems	4.2031	1.8448	5.6829	<0.0001	1.1201
Aggressive behavior	12.0469	4.9655	5.8905	<0.0001	1.0143
Internalizing problems	12.1093	6.6896	4.1680	<0.0001	0.5554
Externalizing problems	16.2500	6.8103	6.0956	<0.0001	1.0946
CBCL Total Problems	44.0781	22.3103	5.2164	<0.0001	0.7767

Table 4: Correlations between total sleep score and behavioral problem scales in the ASD

Behavior Scale	Spearman p	p
Emotionally Reactive	0.4709	<0.0001
Anxiety/depression	0.3885	0.0015
Somatic complaints	0.3390	0.0061
Withdrawn	0.1813	0.1518
CBCL Sleep problems	0.5942	<0.0001
Attention problems	0.1723	0.1735
Aggressive behavior	0.3658	0.0030
Internalizing problems	0.3849	0.0017
Externalizing problems	0.3535	0.0042
CBCL Total problems	0.4583	0.0001
ABC total score	0.3540	0.0041
Irritability	0.3228	0.0093
Hyperactivity	0.2637	0.0353
Stereotypic behavior	0.2107	0.0947
Lethargy / Social withdrawal	0.2979	0.0168
Inappropriate speech	0.2662	0.0335

Table 5: Linear regression model for CBCL total problems score in the ASD

Variable	B	t	p
Bedtime resistance	5.86	5.00	<0.001

Table 6: Linear regression model for ABC total score in the ASD

Variable	B	t	p
Bedtime resistance	4.45	4.29	0.0001
Sleep disordered breathing	3.81	2.07	0.042

Discussion:

The main finding of this study is that preschool children with ASD had a clearly higher level of behavioral problems than healthy controls, and that sleep problems were significantly related to emotional and behavioral difficulties within the ASD group. The large effect sizes found for attention problems, hyperactivity, aggressive behavior, and externalizing problems suggest that a range of problem behaviors often accompanies the disorder.

The most striking sleep-related finding was that, contrary to expectations, daytime sleepiness and total sleep scores were higher in the control group. However, large preschool studies have generally reported higher total CSHQ scores in children with ASD (1). A large early childhood study also showed that whether a child's sleep is rated as problematic by a caregiver depends not only on the child's sleep pattern itself, but also on which sleep behaviors the caregiver sees as problematic (2). Although this finding is not specific to ASD, it suggests that the higher CSHQ scores in our control group compared to the community sample may have been influenced not only by actual sleep difficulties but also by differences in how parents perceive and report sleep problems (1,2). A review highlighted that while sleep problems are very common in ASD, they are often underrecognized and underreported in clinical practice (3). This pattern is also important in terms of the limits of the measurement tool used in our study. Although CSHQ is a widely used parent report scale, it was not originally developed for ASD, and one study reported that the standard CSHQ cut-off point may have low specificity in ASD, while CSHQ-Autism performed better (4).

Although there was no predicted group difference in sleep scores, in the ASD group, higher total sleep scores were associated with total behavioral problems, internalizing problems, anxiety/depression, aggressive behavior, externalizing problems, and total ABC scores. This suggests that sleep may be a clinically meaningful marker in this age group (5-7). In the regression analyses, bedtime resistance predicted both the CBCL total problems score and the ABC total score, while sleep-disordered breathing predicted the ABC total score. These findings suggest that difficulties at bedtime and breathing-related sleep problems may be clinically important areas in behavioral dysregulation. Therefore, in preschool children with ASD, not only difficulty falling asleep and nighttime awakenings should be investigated, but also breathing-related sleep symptoms such as snoring, sleeping with the mouth open, observed apnea and restless breathing (7).

From a clinical point of view, our findings suggest that, in preschool ASD, sleep may be less a categorical group marker and more a modifiable clinical area related to behavioral burden. Therefore, routine sleep screening from the time of diagnosis is important, especially for bedtime routines and breathing-related sleep symptoms. These findings also suggest that early sleep-focused screening and targeted interventions may improve not only nighttime sleep but also daytime behavioral regulation.

The main limitations of this study are its cross-sectional design, the use of parent-report measures, and the difference in sex distribution between the groups. In conclusion, sleep problems in preschool children with ASD may not always be clearly different from those seen in healthy controls, and families may sometimes report them less often. However, because sleep disturbance within the ASD group is closely related to behavioral problems,

sleep assessment should remain a routine part of psychiatric evaluation in ASD, especially when bedtime resistance or sleep-disordered breathing is present.

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0305 - Evaluation of the Post-Discharge Effectiveness of the SAMBA Program Implemented During Compulsory and Voluntary Hospitalization of Patients with Alcohol and Substance Use Disorders

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Introduction

The use of addictive substances has been increasing both in Turkiye and worldwide. emerging as a significant public health problem.

Remission and relapse are fundamental processes that determine the course of substance use disorders. Relapse is defined as a return to previous levels of substance use and related habits following a period of remission and is considered one of the most significant challenges in the treatment process.

In the treatment of alcohol and substance use disorders. in addition to pharmacotherapy. the development of relapse coping skills and the combined application of motivational interviewing with behavioral and group-based psychosocial interventions have been shown to prolong remission and improve treatment outcomes. The Smoking. Alcohol and Substance Addiction Treatment Program (SAMBA) is a structured group psychoeducation program aimed at increasing awareness. enhancing motivation for change. and equipping individuals with the skills necessary to prevent relapse in individuals with alcohol and substance use disorders.

In the treatment of substance use disorders. some patients seek treatment voluntarily. whereas others are admitted compulsorily through family referral or legal procedures. In Turkiye. compulsory treatment practices are carried out as outpatient follow-up within the framework of probation under Article 191/2 of the Turkish Penal Code and as compulsory hospitalization under Article 432 of the Turkish Civil Code. Although compulsory hospitalization is ethically controversial. it is implemented within legal frameworks in many countries on the grounds that it may be protective and life-saving in severe cases (1).

The literature reports that there are significant differences in demographic characteristics and clinical course between patients with alcohol and substance use disorders who undergo compulsory versus voluntary hospitalization. and these differences may influence treatment planning and outcomes (2).

The aim of this study was to investigate whether relapse rates after discharge and time to relapse differ between compulsory and voluntary hospitalization groups among patients with alcohol and substance use disorders who participated in the SAMBA program.

Methods

This study was conducted at Bolu izzet Baysal Mental Health and Diseases Hospital between January 2023 and July 2025. A total of 133 patients were initially included in the study. comprising 69 patients who were hospitalized compulsorily under Article 432 of the Turkish

Civil Code in the Forensic Psychiatry unit and 64 patients who were hospitalized voluntarily in the AMATEM unit.

A total of 35 patients from the Forensic Psychiatry unit and 16 patients from the AMATEM unit who could not be followed up after discharge were excluded. The final sample consisted of 82 patients, including 34 in the compulsory hospitalization group and 48 in the voluntary hospitalization group.

All patients were diagnosed with alcohol or substance use disorder according to DSM-5-TR criteria, were between 18 and 70 years of age, and had at least three months elapsed since discharge. Written or verbal informed consent was obtained from all participants.

Patients attended the SAMBA program sessions three times per week for four weeks. Patients with insufficient medical records, those who refused to participate, those who did not regularly attend SAMBA sessions, and those

with psychiatric comorbidities such as schizophrenia, bipolar disorder, or intellectual disability were excluded from the study.

Sociodemographic characteristics, clinical data, and type of hospitalization were obtained retrospectively through file review. The effectiveness of the SAMBA program in the post- discharge period was evaluated through AMATEM outpatient follow-ups and/or telephone interviews conducted by a psychiatrist at least three months after discharge. A data collection form developed by the researchers, including remission duration and substance use status, was used for this evaluation.

The study was designed as a retrospective cohort study. Ethical approval was obtained from the relevant ethics committee.

Statistical Analysis

Statistical analyses were performed using SPSS 22.0 for Windows (SPSS Inc., Chicago, USA). Categorical variables were expressed as number (n) and percentage (%). The chi-square test or Fisher's exact test was used to compare categorical variables. Time to relapse was analyzed using the Kaplan-Meier method, and survival curves between groups were compared using the log-rank test. A p-value of <0.05 was considered statistically significant in all analyses.

Results

The relationships between relapse status and clinical variables among the patients included in the study are presented in Table 1. A statistically significant difference was found in relapse rates according to admission diagnosis ($p = 0.023$). Patients with a history of rehospitalization after discharge had significantly higher relapse rates compared to those without such a history ($p < 0.001$). In addition, a statistically significant association was found between age at first substance use and relapse status in the compulsory treatment group ($p = 0.038$). In contrast, no statistically significant associations were found between relapse status and the following variables: post-discharge AMATEM outpatient follow-up, medication use after discharge, number of inpatient treatments at AMATEM, family history of substance use disorder, history of probation, history of suicide attempt, and type of hospitalization (compulsory vs. voluntary) (all $p > 0.05$) (Table 1).

The mean length of hospital stay was similar in both groups (mean = 28.1 ± 4.6 days in patients with relapse and 28.3 ±3.4 days in patients without relapse; $p = 0.857$). with no statistically significant difference observed.

During the one-year follow-up period. relapse was observed in 53 cases (67.9%). while 25 cases were censored (32.1%). The Kaplan-Meier curve demonstrated that relapses were clustered in the early period (Figure 1).

The median relapse-free survival time was 90 days (95% CI: 60-180 days; estimated using the Brookmeyer-Crowley method based on Kaplan-Meier confidence intervals).

The relapse-free survival probabilities at days 30. 90. 180. and 365 were estimated as 0.641 (95% CI: 0.524-0.736). 0.449 (95% CI: 0.336-0.555). 0.354 (95% CI: 0.249-0.460). and 0.257 (95% CI: 0.144-0.386). respectively (Table 2).

Table 1. Clinical characteristics of the patients

Variable (Category)	No Relapse (n=0), n (%)	Relapse (n=1), n (%)	Total (n)	p-value (two-tailed)
Admission diagnosis			82	0.023
Multiple psychoactive substance use disorder (F19.2)	18 (66.7)	25 (45.5)	43	
Alcohol use disorder (F10.2)	5 (18.5)	27 (49.1)	32	
Opioid use disorder (F11.2)	3(11.1)	2 (3.6)	5	
Alcohol + substance use disorder	1 (3.7)	1 (1.8)	2	
Post-discharge AMATEM outpatient follow-up			82	0.093
No	7(25.9)	26 (47.3)	33	
Yes	20 (74.1)	29 (52.7)	49	
History of rehospitalization after discharge			82	<0.001
No	25 (92.6)	30 (54.5)	55	
Yes	2 (7.4)	25 (45.5)	27	
Post-discharge medication use			82	0.413
No	6 (22.2)	10 (18.2)	16	
Yes	21 (77.8)	45 (81.8)	65	
Age at onset of substance use (compulsory treatment group)			34	0.038
None	1 (8.3)	6 (27.2)	7	
<14 years	4(33.3)	1 (4.5)	5	
15-18 ya§ arasi	6 (50.0)	7 (31.8)	13	
>19 years	1 (8.3)	8 (36.3)	9	
Number of inpatient treatments at AMATEM			82	0.977
None	15 (55.6)	32 (58.2)	47	
Once	5 (18.5)	8 (14.5)	13	
Twice	1 (3.7)	3 (5.5)	4	
>3 times	6 (22.2)	12 (21.8)	18	
Family history of substance use disorder (voluntary group)			48	1.000
No	10 (66.7)	22 (66.7)	32	
Yes	5 (33.3)	11 (33.3)	16	
History of probation			82	0.165
None	11 (40.7)	30 (54.5)	41	
Currently under probation	3(11.1)	10 (18.2)	13	
Past probation history	13 (48.1)	15 (27.3)	28	
History of suicide attempt			82	0.578
No	20 (74.1)	44 (80.0)	64	
Yes	7(25.9)	11 (20.0)	18	
Type of hospitalization			82	1.000
Voluntary	12 (44.4)	21 (38.2)	48	
Compulsory	15 (55.6)	34 (61.8)	34	

Table 2. One-year relapse-free survival in patients undergoing the SAMBA program

Time (days)	Number at risk (n)	Cumulative relapse, n(%)	Relapse-free survival, S(t)	%95 CI (log–log)
1	78	12 (15,4)	0,846	0,745 – 0,910
30	66	28 (35,9)	0,641	0,524 – 0,736
60	50	35 (44,9)	0,551	0,435 – 0,653
90	43	43 (55,1)	0,449	0,336 – 0,555
120	35	46 (59,0)	0,410	0,301 – 0,516
180	28	50 (64,1)	0,354	0,249 – 0,460
365	9	53 (67,9)	0,257	0,144 – 0,386

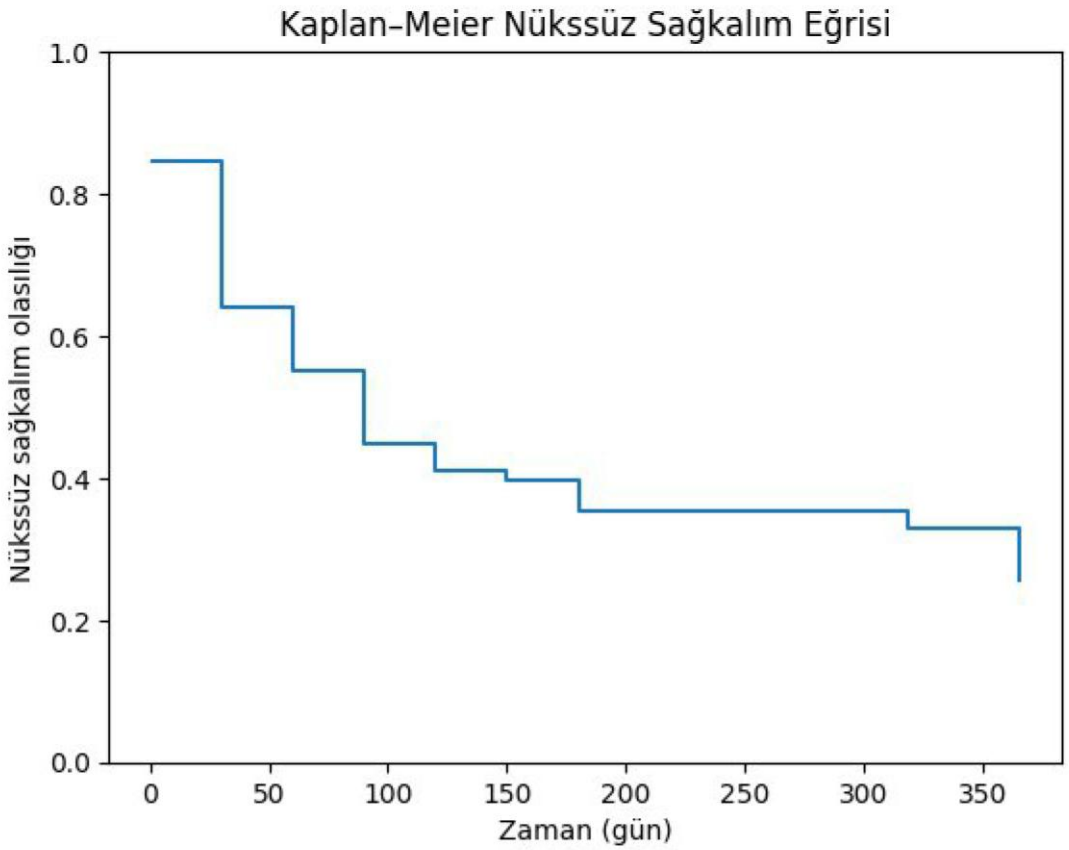


Figure 1. Kaplan-Meier relapse-free survival curve

Relapse-free survival was compared according to the reason for hospitalization (compulsory vs. voluntary) using the Kaplan-Meier method (Figure 2). During the follow-up period. relapse was observed in 20 of 32 patients (62.5%) in the compulsory hospitalization group and in 33 of 46 patients (71.7%) in the voluntary hospitalization group.

The median relapse-free survival time was 90 days in both groups. Survival curves between the groups did not differ significantly according to the log-rank test ($\chi 2(1) = 0.48$. $p = 0.487$) (Table 3).

Table 3. Relapse-free survival probabilities (S(t)) at specific time points

Time (days)	S(t).Compulsory hospitalization	S(t). Voluntary hospitalization
30	0.625	0.652
90	0.469	0.435
180	0.406	0.313
365	0.339	0.188

Relapse-free survival was compared according to the reason for hospitalization (compulsory vs. voluntary) using the Kaplan-Meier method. During the follow-up period. relapse was observed in 20 of 32 patients (62.5%) in the compulsory hospitalization group and in 33 of 46 patients (71.7%) in the voluntary hospitalization group.

The median relapse-free survival time was 90 days in both groups. The survival curves did not differ significantly between the groups according to the log-rank test ($\chi^2(1) = 0.48$, $p = 0.487$) (Table 3).

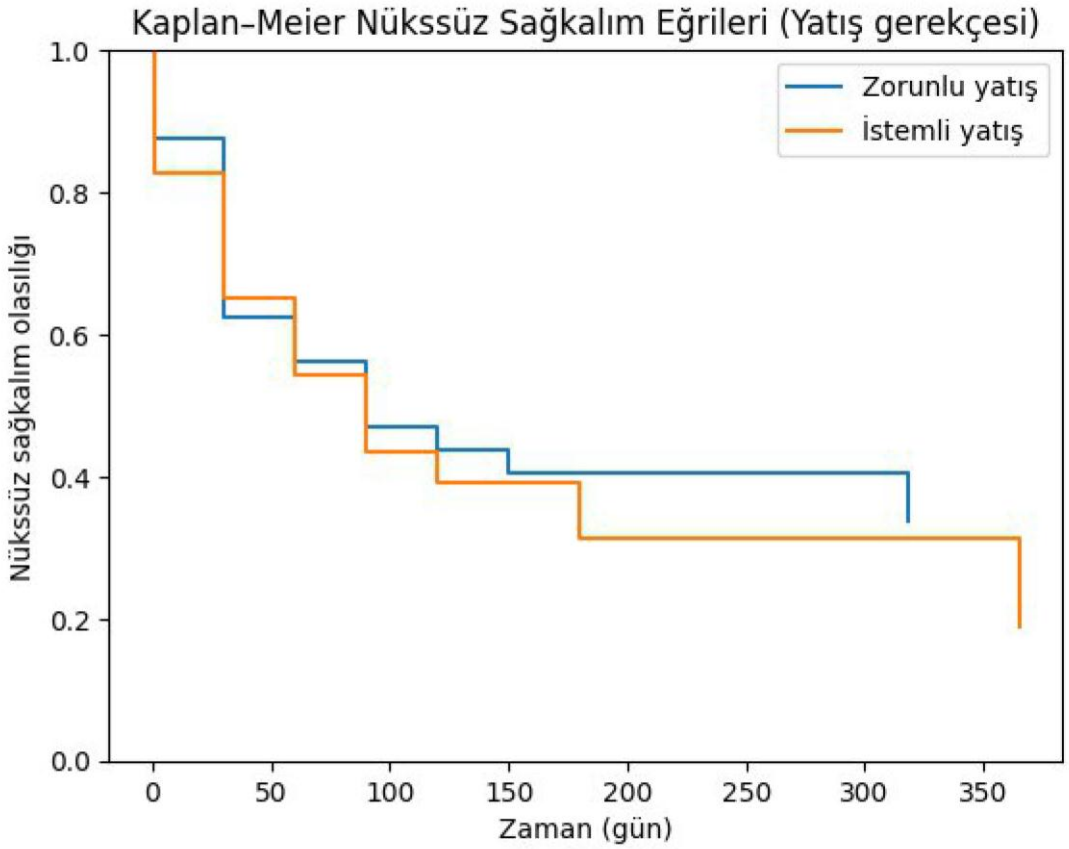


Figure 2. Kaplan-Meier relapse-free survival curve according to the reason for hospitalization

Discussion

In this study, it as found that relapses in patients ith alcohol and substance use disorders predominantly occurred in the early period following discharge, and relapse-free survival decreased markedly over time during the one-year follow-up. The relapse rate during the one- year follow-up was 67.9%o, and the median relapse-free survival time was calculated as 90 days. Kaplan-Meier analysis showed that relapses were particularly concentrated within the first three months. This finding is consistent with the literature reporting that relapses following inpatient addiction treatment are most common within the first 30-90 days and reach high rates during the 6-12 month follow-up period (3).

International studies have reported that structured psychoeducation and skill-based interventions applied after inpatient treatment do not completely eliminate relapse; however, they provide a partial reduction in relapse rates and prolong the time to relapse compared to standard follow-up (4). Similarly, studies conducted in Turkiye among patients under probation have demonstrated that SAMBA programs, when supported by post-discharge follow-up, can reduce relapse rates and positively influence the clinical course (5).

In our study, no significant difference was found in relapse-free survival between compulsory and voluntary hospitalization groups receiving the SAMBA program. This finding suggests that the type of hospitalization alone does not determine the clinical course after discharge. Although the literature reports mixed findings regarding compulsory treatment, some studies indicate that compulsory treatment may improve access to care but does not confer superiority over voluntary treatment in long-term outcomes. Our findings similarly suggest that relapse risk may be more closely related to individual and clinical characteristics rather than the type of hospitalization.

We found that a history of rehospitalization after discharge was strongly associated with relapse. The significantly higher relapse rates among patients with a history of rehospitalization indicate a more severe clinical course and a higher risk profile in this group. This finding supports the chronic and relapsing nature of substance use disorders and highlights the need for more intensive and continuous treatment approaches for high-risk patients.

The significant association between age at onset of substance use and relapse in the compulsory treatment group suggests that individuals who begin substance use at an earlier age are at higher risk of relapse. The negative effects of early onset on neurodevelopmental processes and the establishment of more persistent behavioral patterns may increase the severity of addiction and adversely affect treatment response. Therefore, developing preventive strategies targeting early initiation of substance use is of great importance.

No significant association was found between relapse and variables such as AMATEM outpatient follow-up, post-discharge medication use, family history, and probation status. This finding reflects the multifactorial nature of relapse and suggests that the impact of individual variables may be limited.

Taken together, these findings indicate that the SAMBA program is an important psychoeducational component in addiction treatment; however, its effectiveness becomes more pronounced when supported by regular post-discharge follow-up, supportive interventions, and individualized continuing care models. In particular, the implementation of more intensive monitoring and intervention strategies in the early post-discharge period is critical for preventing relapse.

This study has several limitations. The relatively small sample size, retrospective design, and partial reliance on telephone interviews for relapse assessment may limit the generalizability of the findings. Additionally, the use of self-reported data during follow-up may introduce bias.

In conclusion, relapse in alcohol and substance use disorders is most common in the early period following treatment and is closely associated with individual clinical characteristics. Therefore, strengthening early post-treatment follow-up and intervention strategies is of critical importance.

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0319 - Depression, Anxiety, Hopelessness, and Alexithymia in Parents of Children Undergoing Postnatal Meningomyelocele Surgery: A Multicenter Cross-Sectional Study

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Ethics approval: Istinye University Ethics Committee — Protocol No: 24-157U, June 2024 Keywords: Alexithymia, Anxiety, Depression, Hopelessness, Myelomeningocele, Parents

Abstract

Objective: Postnatal myelomeningocele (MMC) surgery improves neonatal outcomes but imposes a sustained psychological burden on parents. This study evaluates depression, anxiety, hopelessness, and alexithymia in parental dyads across the post-operative adaptation phase (3-24 months post- surgery), with particular attention to dyadic cross-partner associations.

Methods: Cross-sectional dyadic study of 145 parental couples. Paired-samples t-tests compared mothers and fathers within each dyad. Pearson correlations and multiple linear regression identified predictors of depression. Instruments: Beck Depression Inventory (BDI), Beck Anxiety Inventory (BAI), Beck Hopelessness Scale (BHS), Toronto Alexithymia Scale (TAS-20). Effect sizes (Cohen's d) were calculated throughout.

Results: Mothers reported significantly higher depression ($d=0.34$, $p=0.002$) and anxiety ($d=0.41$, $p=0.006$) than fathers. Moderate-to-severe depression prevalence: 32.5% in mothers vs. 27.7% in fathers. No significant between-group differences emerged for hopelessness or alexithymia (both $p>0.50$). Critically, maternal hopelessness correlated strongly with paternal depression ($r=0.61$, $p<0.001$), supporting a dyadic vulnerability model.

Conclusion: Psychiatric symptoms persist well beyond the acute surgical period. Dyadic psychosocial screening, targeting hopelessness and alexithymia in both parents from three months post-delivery, is recommended as a standard component of postnatal MMC follow-up programmes.

1. Introduction

Myelomeningocele (MMC) is a severe congenital neural tube defect associated with motor paralysis, bladder and bowel dysfunction, and hydrocephalus, requiring lifelong multidisciplinary management. The landmark MOMS Trial demonstrated that in-utero MMC repair significantly improves neonatal neurological outcomes compared with postnatal repair, including reduced shunting requirements and enhanced lower extremity motor function. [1]

Despite these clinical advances, the psychological trajectory of parents who navigate postnatal surgery decisions and their long-term sequelae has received comparatively limited systematic attention. The perioperative period constitutes an acute emotional crisis involving uncertainty, grief, and anticipatory anxiety. However, the 3-to-24-month window following surgery — which we term the "adaptation phase" — may be equally or more psychologically demanding. As the initial optimism of a successful surgical intervention recedes, parents must accommodate the chronic reality of disability management, ongoing rehabilitation, and uncertain developmental prognosis. This transitional phase represents a clinically underserved period where psychiatric morbidity may consolidate into chronic illness.

Alexithymia — the difficulty identifying and describing one's own emotional states — has been consistently associated with depression and maladaptive coping in caregiving populations. [2] Parents with alexithymic traits may not recognise or report psychological distress, making proactive clinical screening especially important. This study employs a dyadic analytical framework, acknowledging that partners within a couple do not constitute independent observations. We hypothesise that the psychiatric status of one parent is directly linked to that of the other, and that cross-partner associations in hopelessness and alexithymia may explain a meaningful proportion of individual vulnerability to depression.

Materials and Methods

Participants

This cross-sectional study recruited 145 parental dyads from a tertiary university hospital; all participants' children had undergone successful postnatal MMC repair. Data were collected between 3 and 24 months post-operatively (mean 13.4 ± 5.7 months). Inclusion criteria: parents over 18 years with no pre-existing psychiatric diagnosis prior to pregnancy. Exclusion: cognitive impairment, incomplete data, or history of major psychiatric illness. Due to differential instrument completion, complete pairs ranged from n=20 (BHS, TAS-20) to n=86 (BDI). Ethics approval: Istinye University Ethics Committee — Protocol No: 24-157U, June 2024.

Instruments

Beck Depression Inventory (BDI): depressive severity; BDI≥17 = moderate depression. Beck Anxiety Inventory (BAI): somatic and cognitive anxiety. Beck Hopelessness Scale (BHS): negative future expectation. Toronto Alexithymia Scale (TAS-20): emotional identification difficulty; TAS-20≥61 = clinically significant alexithymia. Internal consistency was acceptable for all scales in the present sample (Cronbach's α: BDI=0.87, BAI=0.89, BHS=0.82, TAS-20=0.84).

Statistical Analysis

Paired-samples t-tests compared mean scores within each couple, appropriate given data non- independence. Effect sizes were quantified using Cohen's d. Pearson correlations examined within- and cross-parent associations. Multiple linear regression (BDI dependent; BHS, BAI, TAS-20 as predictors) was conducted on cases with complete data across all four scales (n=29). Variance inflation factors (VIF<2.5) confirmed no multicollinearity. SPSS v.26 and Jamovi v.2.3 were used.

Results

Descriptive Statistics and Group Comparisons

Table 1 presents mean scores and standard deviations. Mothers scored significantly higher than fathers on depression (M=14.27 vs. 11.11; t=3.19, p=0.002, d=0.34) and anxiety (M=11.58 vs. 10.44; t=2.87, p=0.006, d=0.41), both yielding small-to-medium effect sizes. No statistically significant differences emerged for hopelessness (t=0.58, p=0.571, d=0.13) or alexithymia (t=-0.24, p=0.817, d=-0.05); these comparisons were based on 20 complete dyads and are exploratory (Table 2). Moderate-to-severe depression was present in 32.5% of mothers (n=37/114) and 27.7% of fathers (n=26/94).

Table 1. Descriptive Statistics by Parent Group (Mean ± SD)

Variable	Mothers Mean	Mothers SD	Fathers Mean	Fathers SD	n
BDI (Depression)	14.27	8.70	11.11	9.03	94-114
BAI (Anxiety)	11.58	11.79	10.44	11.40	52-114
BHS (Hopelessness)	5.94	4.33	5.28	3.31	20-62
TAS-20 (Alexithymia)	48.48	15.81	49.35	15.04	20-31

BDI=Beck Depression Inventory; BAI=Beck Anxiety Inventory; BHS=Beck Hopelessness Scale; TAS-20=Toronto Alexithymia Scale.

Table 2. Paired-Samples T-Test Results: Mothers vs. Fathers

Scale	Mothers M±SD	Fathers M±SD	n pairs	t	p	Cohen's d
BDI (Depression)	14.27 ± 8.70	11.11 ± 9.03	86	3.19	**0.002***	**0.34**
BAI (Anxiety)	11.58 ± 11.79	10.44 ± 11.40	49	2.87	**0.006***	**0.41**
BHS (Hopelessness)†	5.94 ± 4.33	5.28 ± 3.31	20	0.58	0.571	0.13
TAS-20 (Alexithymia)†	48.48 ± 15.81	49.35 ± 15.04	20	-0.24	0.817	-0.05

*p<0.05. † Exploratory: based on n=20 complete dyads. Paired t-test on complete dyads only.

Correlations and Predictors of Depression

Within-group correlations confirmed significant associations between depression, anxiety, hopelessness, and alexithymia in both mothers (r=0.38-0.49) and fathers (r=0.39-0.56), all p<0.05. Cross-parent analyses identified two clinically important associations: maternal hopelessness with paternal depression (r=0.61, p<0.001) and maternal depression with paternal hopelessness (r=0.52, p<0.001), supporting a bidirectional dyadic vulnerability model. Regression analysis on the complete sub-sample (n=29) explained 37% of variance in maternal BDI scores (R^2=0.37, p=0.006), with BHS (β=0.51, p<0.001) and BAI (β=0.26, p=0.03) as significant predictors.

Discussion

Gender Differences: Smaller Effects Than Previously Assumed

Mothers showed significantly elevated depression and anxiety relative to fathers, with effect sizes in the small-to-medium range (d=0.34 and d=0.41 respectively). These values are considerably smaller than those commonly cited or assumed in the perinatal psychiatry literature (d>0.80), emphasising the importance of reporting data-derived rather than presumed estimates. The pattern nonetheless has a plausible clinical basis: mothers typically assume primary responsibility for post-operative medical follow-up, physical therapy appointments, and daily care management, while fathers more commonly resume occupational roles relatively quickly following the child's birth. Our data confirm that this gendered caregiving asymmetry and its psychiatric correlate persist across the full 24-month adaptation window — it is not merely an acute postpartum phenomenon.

Hopelessness and Alexithymia: The Dyadic Dimension

The absence of statistically significant between-group differences for hopelessness and alexithymia was unexpected given our hypotheses, but is at least partially attributable to the small complete-pair sample sizes (n=20) that substantially limited statistical power for these comparisons. These findings should not be interpreted as clinical irrelevance. On the contrary, the cross-parent correlation analyses represent the most clinically salient contribution of this study: maternal hopelessness and paternal depression were strongly correlated (r=0.61), as were maternal depression and paternal hopelessness (r=0.52). These bidirectional associations demonstrate that one parent's sense of futility directly amplifies the other's depressive symptoms — a dynamic invisible to individual-level analyses and with direct implications for intervention design. Couple-based therapeutic approaches, rather than programmes focused exclusively on the mother, are indicated by these data. [3]

Alexithymic parents — who struggle to identify or articulate emotional distress — represent a clinically important subgroup in this population. [4] Standard referral pathways depend on self- identified distress or help-seeking behaviour; parents with alexithymic traits are unlikely to use these pathways despite significant internal burden. Systematic screening using the TAS-20 at structured follow-up intervals, rather than reliance on patient-initiated consultation, is therefore essential.

Regression Model and Predictors

The regression model (R^2=0.37, n=29) identified hopelessness and anxiety as the strongest modifiable predictors of depression. Although the small sample renders these findings preliminary, they are consistent with theoretical frameworks linking negative future cognition and somatic hyperarousal to depressive consolidation. Targeted interventions addressing hopelessness — such as cognitive reframing and problem-focused coping strategies — and anxiety management may be particularly effective in preventing the progression from acute distress to chronic psychiatric disorder in this population.

Limitations

The cross-sectional design precludes causal inference. High missing-data rates for BHS and TAS-20 (complete pairs n=20) substantially limit reliability for these scales; findings must be considered exploratory. Single-centre recruitment limits generalisability. All instruments are self-reported, and the exclusion of parents with pre-existing psychiatric diagnoses may underestimate population-level burden. The regression sample (n=29) is insufficient for stable coefficient estimation. Future longitudinal, multicentre studies employing the Actor-Partner Interdependence Model (APIM) [5] are warranted to formally model cross-partner effects and identify optimal intervention timing.

Conclusion

Postnatal MMC surgery imposes a significant and sustained psychological burden on parental dyads, with mothers carrying a disproportionate load in depression and anxiety throughout the adaptation phase. The cross-parent correlations between hopelessness and depression represent the most clinically actionable finding of this study, supporting a dyadic rather than individual model of parental distress. Alexithymia warrants proactive clinical attention, as affected parents are unlikely to self-refer despite high internal burden. We recommend integrating structured, couple-centred psychosocial screening — including BDI, BHS, and TAS-20 — at 3, 6, 12, and 24 months post-delivery as a standard component of postnatal MMC surgical follow-up programmes. This approach may meaningfully improve both parental mental health and long-term child developmental outcomes.

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0327 - The Relationship Between Metacognition and Childhood Adverse Experiences in Patients with Bipolar Disorder and a History of Suicide Attempts

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Introduction

Bipolar disorder (BD) is a chronic mood disorder characterized by recurrent depressive. manic. or hypomanic episodes. often accompanied by significant impairment in social and occupational functioning and high rates of morbidity and mortality . It is estimated to affect approximately 40 million individuals worldwide and represents a serious public health concern . The disorder typically begins in adolescence or early adulthood and persists throughout life. with a multifactorial etiology involving genetic. biological. and environmental factors .

Bipolar disorder is frequently associated with diagnostic delays and misdiagnosis. Studies indicate that approximately 69% of patients receive an incorrect diagnosis at their first outpatient visit. and there may be an average delay of nearly nine years between symptom onset and accurate diagnosis . Clinically. manic episodes are characterized by increased energy. decreased need for sleep. elevated self-esteem. and excessive involvement in risky activities. whereas depressive episodes involve low mood. loss of energy. and suicidal ideation .

Beyond mood symptoms. bipolar disorder is notable for its markedly increased risk of suicide. Individuals with BD have been reported to have a suicide risk approximately 15 times higher than the general population. and 25-50% attempt suicide at least once during their lifetime . This high rate of suicidal behavior underscores the importance of identifying underlying psychological mechanisms contributing to suicide risk.

Recent studies suggest that psychosocial factors such as adverse childhood experiences (ACEs) and metacognitive processes may play a significant role in this risk. Adverse childhood experiences—including emotional. physical. and sexual abuse. neglect. and exposure to domestic violence—have been shown to negatively affect the clinical course of bipolar disorder and increase suicide risk . The accumulation of such experiences has been associated with a higher likelihood of suicide attempts. rapid cycling. and more severe affective episodes . Furthermore. exposure to childhood trauma has been linked to earlier onset of the disorder and more severe clinical presentations.

Metacognition. first defined by Flavell in 1979. refers to the capacity to recognize. monitor. and regulate one's own cognitive processes. often described as "thinking about thinking" . Within this framework. dysfunctional metacognitive beliefs—such as the perception that thoughts must be controlled or that cognitive processes are unreliable—have been associated with various forms of psychopathology. including suicidal behavior. Studies in individuals with major depressive disorder and a history of suicide attempts have shown that such individuals tend to perceive a lack of control over their thoughts as a weakness and exhibit increased self-blame . Similar findings have been reported in individuals with bipolar disorder, where maladaptive metacognitive beliefs are linked to suicidal behavior .

Despite increasing interest in these factors, studies examining the combined role of adverse childhood experiences and metacognitive processes in bipolar disorder remain limited. In particular, the extent to which metacognitive beliefs mediate the relationship between early- life adversity and suicidal behavior has not been sufficiently explored.

In this context, the present study aimed to investigate the relationship between adverse childhood experiences and metacognitive beliefs in individuals with bipolar disorder, and to examine the role of these beliefs in suicidal behavior.

Methods

This study was conducted with participants recruited from the outpatient psychiatry clinic of Van Yuzuncu Yil University Medical Faculty. The sample consisted of individuals diagnosed with bipolar disorder and a healthy

control group. The bipolar disorder group was divided into two subgroups based on the presence or absence of a history of suicide attempt.

A total of 159 participants were included in the study: 52 individuals with bipolar disorder and a history of suicide attempt, 54 individuals with bipolar disorder without a history of suicide attempt, and 53 healthy control participants.

Data were collected using standardized assessment tools. Adverse childhood experiences were assessed using the Adverse Childhood Experiences Questionnaire (ACE-Q), which evaluates exposure to various forms of abuse, neglect, and household dysfunction during childhood. Metacognitive beliefs were measured using the Metacognitions Questionnaire-30 (MCQ-30), which includes multiple subdimensions such as cognitive confidence, positive beliefs about worry, need to control thoughts, and cognitive self-consciousness. Suicidal behavior was evaluated using the Suicide Behaviors Questionnaire (SBQ).

All statistical analyses were performed using SPSS software. Group comparisons were conducted to evaluate differences in scale scores among participants. Correlation analyses were used to examine the relationships between ACE, MCQ-30, and SBQ scores. Logistic regression analyses were performed to identify predictors of suicide attempts. In addition, mediation analyses were conducted to investigate whether metacognitive beliefs mediated the relationship between adverse childhood experiences and suicidal behavior.

Results

Participants with bipolar disorder and a history of suicide attempt had significantly higher scores on the ACE-Q, MCQ-30, and SBQ compared to both bipolar participants without suicide attempts and healthy controls. These findings indicate that both childhood adversity and dysfunctional metacognitive beliefs are more prominent in individuals with suicidal behavior.

Correlation analyses revealed significant positive relationships between ACE scores, MCQ-30 scores, and SBQ scores. Higher levels of childhood adversity were associated with more dysfunctional metacognitive beliefs and greater severity of suicidal behavior. Among the

MCQ-30 subdimensions, the need to control thoughts demonstrated particularly strong correlations with both ACE and SBQ scores.

Mediation analyses showed that metacognitive beliefs partially mediated the relationship between adverse childhood experiences and suicidal behavior. Specifically, ACE total scores exerted an indirect effect on suicidal behavior through metacognitive processes. In particular, neglect-related ACE scores were found to influence suicidal behavior indirectly via increased need to control thoughts.

Logistic regression analyses further demonstrated that both ACE abuse scores and MCQ-30 total scores were significant predictors of suicide attempts. Each one-point increase in these variables was associated with an increased likelihood of having a history of suicide attempt. These findings suggest that both early-life adversity and maladaptive metacognitive beliefs independently contribute to suicide risk in bipolar disorder.

In addition, subgroup analyses within the bipolar disorder sample indicated that individuals with a history of suicide attempt had consistently higher metacognitive dysfunction across multiple MCQ-30 subdomains. Similarly, ACE subscale analyses showed that both abuse and neglect dimensions were elevated in this group. These patterns support the notion that different forms of childhood adversity may contribute to suicidal behavior through overlapping cognitive mechanisms.

Discussion

The present study demonstrates that adverse childhood experiences and dysfunctional metacognitive beliefs are significantly associated with suicidal behavior in individuals with bipolar disorder. Importantly, the findings suggest that metacognitive processes may play a mediating role in the relationship between early-life adversity and suicidality.

Consistent with previous literature, individuals with a history of suicide attempts reported higher levels of childhood adversity. Exposure to abuse and neglect during early developmental periods may lead to long-term

alterations in cognitive and emotional functioning, increasing vulnerability to maladaptive coping strategies and suicidal behavior. The association between ACEs and more severe clinical features of bipolar disorder has been well documented, including earlier onset, more frequent episodes, and increased comorbidity.

The results of this study extend these findings by highlighting the role of metacognitive beliefs. In particular, the need to control thoughts emerged as a key factor linking childhood adversity to suicidal behavior. Individuals who experience neglect may develop rigid beliefs about the necessity of controlling their thoughts, which may contribute to increased psychological distress. Attempts to suppress or control intrusive thoughts may paradoxically intensify these thoughts, thereby increasing emotional dysregulation and suicide risk.

Furthermore, reduced cognitive confidence, another component of metacognitive dysfunction, may contribute to increased vulnerability by impairing individuals' ability to trust their own cognitive processes. This may lead to heightened uncertainty, rumination, and difficulty in problem-solving, all of which are associated with suicidal ideation. In this respect, the relationship between childhood adversity and suicidality in bipolar disorder may operate not only through the emotional burden of trauma, but also through the maladaptive cognitive styles shaped by these early experiences.

Another important finding is that different components of adverse childhood experiences may contribute to suicidality through partially distinct pathways. While abuse-related ACE scores emerged as significant predictors of suicide attempts, neglect appeared to be particularly relevant in its indirect association with suicidal behavior through metacognitive processes, especially the need to control thoughts. This pattern suggests that the impact of early-life adversity in bipolar disorder extends beyond trauma exposure itself and involves the way individuals interpret and regulate their internal experiences.

From a clinical perspective, these findings suggest that interventions targeting maladaptive metacognitive beliefs may be beneficial in reducing suicide risk among individuals with bipolar disorder. Therapeutic approaches focusing on modifying dysfunctional beliefs about thinking processes and improving cognitive flexibility may help individuals develop more adaptive coping strategies.

However, several limitations should be considered. The cross-sectional design of the study limits the ability to draw causal conclusions regarding the relationships between variables. Additionally, the use of self-report measures may introduce recall bias, particularly in the assessment of childhood experiences. Future longitudinal studies are needed to better understand the temporal dynamics of these relationships and to evaluate the effectiveness of metacognition-focused interventions.

In conclusion, adverse childhood experiences contribute to suicidal behavior in individuals with bipolar disorder not only directly but also indirectly through maladaptive metacognitive beliefs. These findings highlight the importance of considering both early-life adversity and cognitive processes in understanding and preventing suicidal behavior in bipolar disorder.

Keywords: Adverse childhood experiences, bipolar disorder, metacognition, suicide

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0334 - Association of Metabolic Control with Diabetes-Related Distress and Health-Related Quality of Life in Adolescents with Type 1 Diabetes Mellitus: A Cross-Sectional Study

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Objective

Type 1 Diabetes Mellitus (T1DM) is not only a chronic disease requiring metabolic monitoring; it also represents a multidimensional life burden that requires continuous self-care, insulin administration, blood glucose monitoring, dietary regulation, and constant vigilance against glycemic fluctuations. This ongoing treatment burden may affect not only glycemic control but also diabetes-specific distress and health-related quality of life in adolescents (1,2). Current guidelines recommend that psychosocial domains should be evaluated as part of routine diabetes follow-up in children and adolescents with T1DM (1). Diabetes distress has emerged as a clinically important construct associated with treatment adherence, self-care behaviors, and glycemic outcomes (2,3,7).

While the literature indicates that diabetes distress is common in adolescents and may be associated with poorer metabolic control, the relationship between generic health- related quality of life and HbA1c appears to be more variable (2,3). In contrast, the relationship between diabetes-specific distress and quality of life appears to be more consistent, particularly in the domains of emotional well-being and daily functioning (2). This study aimed to investigate the relationship between HbAlc, as an indicator of metabolic control, and diabetes-specific distress and health-related quality of life in adolescents with T1DM; it also aimed to examine the relationship between diabetes-specific distress and quality-of-life domains.

Methods

This cross-sectional study included 60 adolescents aged 12-18 years who were being followed with a diagnosis of T1DM. Sociodemographic and clinical data were recorded, and current HbA1c level was used as an indicator of metabolic control.

Diabetes-specific distress was assessed using the Problem Areas in Diabetes-Teen (PAID-T), a diabetes-specific self-report scale developed for adolescents. Generic health-related quality of life was assessed using the adolescent self-report version of the KIDSCREEN-52 (4-6). The relationships between HbA1c and PAID-T total and subscale scores, as well as KIDSCREEN-52 domain scores, were examined, and the relationships between PAID-T and KIDSCREEN-52 domains were evaluated. Statistical significance was set at $p<0.05$.

Results

The mean age of the 60 adolescents included in the study was 15.05±2.00 years; 34 were female (56.7%) and 26 were male (43.3%). The mean HbA1c level was 8.33±1.51. The mean PAID-T total score was 38.43±15.89; mean subscale scores were 13.07±5.87 for emotional burden, 14.53±6.88 for family/friend-related stress, and 10.93±5.73 for treatment-related stress.

As HbA1c levels increased, emotional burden subscale scores also increased significantly ($r=0.344$; $p=0.007$). Similarly, a significant positive correlation was found between HbA1c and the family/friend-related stress subscale ($r=0.300$; $p=0.020$). The relationship between total PAID-T score and HbA1c was also significant and positive

(r=0.423; p<0.001). Although the treatment-related stress subscale showed a positive association, this relationship did not reach statistical significance (r=0.193; p=0.140).

When the relationships between KIDSCREEN-52 domains and HbAlc were examined, most correlation coefficients were weak and generally negative in direction; however, none of these relationships reached statistical significance.

In contrast, marked negative associations were observed between PAID-T and KIDSCREEN-52 adolescent domains. As PAID-T total scores increased, significant decreases were found in psychological well-being (r=-0.482; p<0.001), self-perception (r=-0.485; p<0.001), moods and emotions (r=-0.436; p<0.001), school environment (r=- 0.479; p<0.001), physical well-being (r=-0.377; p=0.003), parent relations (r=-0.298; p=0.021), and social acceptance (r=-0.258; p=0.047). In contrast, no significant relationships were found between PAID-T total scores and the domains of autonomy, financial resources, or social support.

When PAID-T subscales were examined, higher emotional burden scores were associated with lower psychological well-being (r=-0.553; p<0.001), self-perception (r=-0.531; p<0.001), moods and emotions (r=-0.428; p<0.001), physical well-being (r=- 0.449; p<0.001), parent relations (r=-0.396; p=0.002), school environment (r=-0.375; p=0.003), and social acceptance (r=-0.264; p=0.042). As family/friend-related stress scores increased, significant decreases were found in physical well-being (r=-0.354; p=0.006), psychological well-being (r=-0.467; p<0.001), moods and emotions (r=- 0.440; p<0.001), self-perception (r=-0.525; p<0.001), parent relations (r=-0.306; p=0.017), and school environment (r=-0.428; p<0.001). The treatment-related stress subscale showed a significant negative correlation only with school environment (r=- 0.414; p<0.001).

Discussion

The main finding of this study is that HbA1c appears to be more strongly related to diabetes-specific distress than to generic health-related quality of life in adolescents with T1DM. The significant positive correlations between HbA1c and PAID-T emotional burden, family/friend-related stress, and total scores suggest that the daily burden of diabetes management and the accompanying emotional strain may be closely related to metabolic outcomes. Diabetes distress may show a more sensitive relationship with HbA1c because, unlike general psychological symptoms, it reflects the burden arising directly from living with and managing diabetes (2,3,7). The literature also indicates that diabetes distress is common in adolescents and may be associated with poorer glycemic control (2,3).

In contrast, the lack of significant associations between HbA1c and KIDSCREEN-52 domains may be explained by the multidimensional nature of quality of life. Health- related quality of life is influenced not only by metabolic indicators but also by developmental characteristics, family relationships, peer experiences, school functioning, and the broader psychosocial context (5,6). Therefore, the effect of HbA1c on quality of life may appear weaker or more indirect when assessed using a generic scale.

The second important finding of this study is that the relationship between diabetes- specific distress and quality of life was more pronounced. In particular, the moderate negative correlations between PAID-T total scores and psychological well-being, moods and emotions, self-perception, and school environment suggest that diabetes distress is not limited to emotional strain alone, but is also closely related to adolescents' psychological well-being, self-concept, and daily functioning. The stronger associations of the emotional burden subscale with psychological well-being and self-perception support the impact of diabetes-related emotional pressure on self-concept and well-being. The fact that the family/friend-related stress subscale was also associated with similar domains suggests that diabetes-related burden is experienced not only individually but also within relational and social contexts. Therefore, diabetes-specific distress should be considered not merely as an accompanying finding in clinical assessment, but also as a psychosocial target that requires monitoring (1,7).

This pattern suggests that there may be a clinically meaningful interaction among "metabolic control - diabetes-specific distress - quality of life" in adolescents with T1DM. Although metabolic control did not show a direct and strong relationship with quality of life, diabetes-specific distress was associated both with HbAlc and with multiple quality-of-life domains. Although these findings do not demonstrate causality, they suggest that diabetes distress may constitute a central clinical axis linking metabolic control and psychosocial functioning.

From a clinical perspective, these findings indicate that focusing solely on HbAlc is not sufficient in the follow-up of adolescents with T1DM. Routine screening for diabetes- specific distress may help identify adolescents experiencing difficulties particularly in the emotional and school-functioning domains of quality of life at an earlier stage (1,7). It is important that psychosocial follow-up and appropriate interventions be considered as a standard component of diabetes care.

Table 1. Relationship Between HbAlc and KIDSCREEN-52 and PAID-T

	HbA1c	
	r value	**p value**
KIDSCREEN-52		
Physical well-being	-0.080	0.544
Psychological well-being	-0.009	0.945
Moods and emotions	-0.097	0.459
Self-perception	0.003	0.984
Autonomy	-0.124	0.347
Parent relations	-0.037	0.779
Financial resources	-0.192	0.142
Social support	0.118	0.368
School environment	-0.217	0.096
Social acceptance	-0.011	0.933
PAID-T		
PAID-T Emotional burden	0.344	**0.007**
PAID-T Family/friend-related stress	0.300	**0.020**
PAID-T Treatment-related stress	0.193	0.140
PAID-T Total	0.423	**<0.001**

"SPSS: Pearson correlation test"

Table 2. Relationship Between KIDSCREEN-52 and PAID-T

KIDSCREEN-52 Adolescent	PAID-T Emotional burden		PAID-T Family/friend- related stress		PAID-T Treatment-related stress		PAID-T Total	
	r value	p value	r value	p value	r value	p value	r value	p value
Physical well-being	-0.449	**<0.001**	-0.354	**0.006**	-0.142	0.281	-0.377	**0.003**
Psychological well-being	-0.553	**<0.001**	-0.467	**<0.001**	-0.198	0.129	-0.482	**<0.001**
Moods and emotions	-0.428	**<0.001**	-0.440	**<0.001**	-0.243	0.061	-0.436	**<0.001**
Self-perception	-0.531	**<0.001**	-0.525	**<0.001**	-0.162	0.215	-0.485	**<0.001**
Autonomy	-0.080	0.543	0.050	0.705	-0.029	0.824	-0.026	0.843
Parent relations	-0.396	**0.002**	-0.306	**0.017**	-0.051	0.701	-0.298	**0.021**
Financial resources	-0.231	0.075	-0.152	0.247	-0.200	0.125	-0.230	0.077
Social support	-0.156	0.235	-0.089	0.499	-0.178	0.173	-0.166	0.204
School environment	-0.375	**0.003**	-0.428	**<0.001**	-0.414	**<0.001**	-0.479	**<0.001**
Social acceptance	-0.264	**0.042**	-0.228	0.079	-0.175	0.181	-0.258	**0.047**

SPSS: Pearson correlation test

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0344 - Comparison of Telomere Length in Patients with Schizophrenia Using Clozapine

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Brief report

Objective: Research on aging and telomere length in mental illnesses has increased in recent years. Telomeres are repetitive nucleotides that protect chromosomal DNA from degradation. They are regarded as a biological marker of cellular aging because they shorten with each cell division. Many factors such as oxidative stress, inflammation, and lifestyle habits can accelerate telomere shortening.

Studies examining telomere length in schizophrenia have produced inconsistent findings. While many studies reported shorter telomeres in patients, others found no difference or even longer telomeres. These inconsistencies may be related to methodological differences, clinical heterogeneity, and the effects of treatment. Furthermore, antipsychotic medications, including clozapine, may influence biological aging processes, however studies investigating their relationship with telomeres are limited.

This study aimed to compare telomere length between patients with schizophrenia and healthy controls and to investigate the association between clozapine use and telomere length.

Methods: This is a cross-sectional study including 54 patients diagnosed with schizophrenia according to DSM-5 criteria and 26 healthy controls matched for age and sex. Patients were recruited from a specialized outpatient clinic and were divided into two groups: clozapine users (n=27) and non-clozapine users (n=27).

Inclusion criteria required participants to be between 18 and 65 years of age and to provide informed consent. Patients with comorbid psychiatric disorders, substance use disorders (except smoking), neurological conditions, or significant systemic illnesses were excluded. Clozapine users had been receiving regular treatment for at least one year.

Clinical assessments were performed using standardized instruments, including the Positive and Negative Syndrome Scale (PANSS), Clinical Global Impression (CGI), Calgary Depression Scale for Schizophrenia, Brief Negative Symptom Scale,

Extrapyramidal Symptom Rating Scale, and Personal and Social Performance Scale. Sociodemographic data and body mass index were also recorded.

Genomic DNA was extracted, and telomere length was measured using real-time quantitative polymerase chain reaction (RT-qPCR).

Assumptions of normality were tested and statistical analyses were conducted using appropriate parametric and non-parametric tests depending on data distribution. Group comparisons were performed using Student's t-test, Mann-Whitney U test, and ANOVA/Kruskal-Wallis tests. Analysis of covariance (ANCOVA) was used to control for potential confounders such as age, sex, and body mass index. Correlation and regression analyses were conducted to identify factors associated with telomere length. Statistical significance was set at $p < 0.05$.

Results: There were no significant differences between patient and healthy control groups in terms of age or sex distribution. Telomere length was significantly longer in patients with schizophrenia compared to healthy controls ($p = 0.01$). Subgroup analyses revealed that clozapine users had significantly longer telomeres than non-clozapine patients ($p = 0.003$). Additionally, clozapine users had significantly longer telomeres than healthy controls ($p = 0.001$). In contrast, no significant difference in telomere length was observed between non-clozapine patients and healthy controls ($p = 0.302$).

When all three groups were analyzed together, a significant overall difference was found ($p = 0.001$), with post hoc analyses indicating that the difference was primarily driven by the clozapine group. These findings remained significant after adjusting for potential confounding variables, including age, sex, body mass index, and metabolic parameters.

Correlation analyses showed a negative association between LDL cholesterol levels and telomere length.

Discussion: The present study found that telomere length was longer in patients with schizophrenia compared to healthy controls, which is not consistent with the studies supporting the hypothesis of accelerated aging in schizophrenia. Importantly, this finding appeared to be mainly due to the patients receiving clozapine treatment.

The absence of a significant difference between non-clozapine patients and healthy controls suggests that telomere length alterations may not be directly related to schizophrenia itself. However, this finding might be influenced by treatment-related factors.

The potential effects of antipsychotics on telomere biology can be explained by several mechanisms, including antioxidant properties, activation of the Wnt/p-catenin pathway via GSK3P inhibition, increased telomerase expression, and BDNF-mediated neuroprotective effects. However, studies revealed inconsistent findings. Some in vitro studies have suggested that clozapine does not protect telomeres against oxidative damage or may even reduce telomerase expression. A study evaluating olanzapine and clozapine together has reported that telomere shortening effects may be primarily associated with olanzapine rather than clozapine. Therefore, current evidence is heterogeneous.

Negative relationship between LDL cholesterol levels and telomere length suggests that higher concentrations of LDL may contribute to accelerated telomere shortening. This finding is consistent with the fact that elevated LDL is associated with increased oxidative stress and telomeres are sensitive to oxidative damage.

This study has several limitations. Its cross-sectional design prevents conclusions about causality. The sample size is relatively small. Also, the absence of drug-naive patients and some patients receiving combination antipsychotic treatments limit the ability to investigate the treatment effects on telomere length.

Despite these limitations, the findings contribute to the growing body of literature suggesting that biological aging markers in schizophrenia may be influenced by treatment factors.

In conclusion, telomere length in schizophrenia may not always reflect accelerated biological aging and may be influenced by antipsychotic treatment. Clozapine use appears to be associated with longer telomere length. Longitudinal studies, particularly those including drug-naive patients, are needed to clarify the direction and mechanisms of this relationship in the future.

Keywords: Schizophrenia, Telomere Length, Clozapine, Biological Aging

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0346 - Evaluation of Drug-Related Suicide Attempts in Terms of Age, Gender, and Time

Background

Suicide attempts represent a major public health concern worldwide and remain one of the leading causes of morbidity among young populations. Although completed suicide is more frequent in males, suicide attempts are reported more commonly among females and frequently involve less lethal methods such as drug overdose. Deliberate self- poisoning constitutes a substantial proportion of emergency department admissions and intensive care consultations related to suicidal behavior. Understanding the clinical and sociodemographic characteristics of these cases is essential for identifying high-risk groups and developing preventive strategies. (1, 2)

Drug intoxication is among the most frequently used methods in suicide attempts, particularly among adolescents and young adults. Previous studies have shown that pharmaceutical agents such as antidepressants, analgesics, and other commonly accessible medications are often involved in self-poisoning cases. In addition to demographic factors, psychiatric disorders, psychosocial stressors, and medical conditions have been associated with increased risk of suicidal behavior. Identifying these factors may help clinicians improve early recognition and management of patients presenting with suicidal intoxication. (2,3)

Methods

This retrospective study included all individuals admitted to Qorum Erol Olgok Training and Research Hospital between 2014 and 2024 with a diagnosis of intentional drug overdose. Demographic variables (age and gender) and temporal variables (month, season, and year) were obtained from hospital electronic records. Descriptive statistics (mean ± SD, frequency, and percentage) were calculated, and distributions were analyzed using chi-square and t-tests or non-parametric equivalents depending on normality. Temporal clustering was evaluated by month and season to determine potential seasonal variation. Data were analyzed using SPSS version 28.0. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Results

A total of 1,226 patients admitted due to intentional drug overdose between 2014 and 2024 were included in the analysis. The mean age of the patients was 31.8 ± 12.8 years (median: 29; range: 12-85 years). The highest proportion of cases was observed in the 2130 age group (34.2%), followed by the 31-40 age group (23.3%). Detailed demographic characteristics are presented in Table 1.

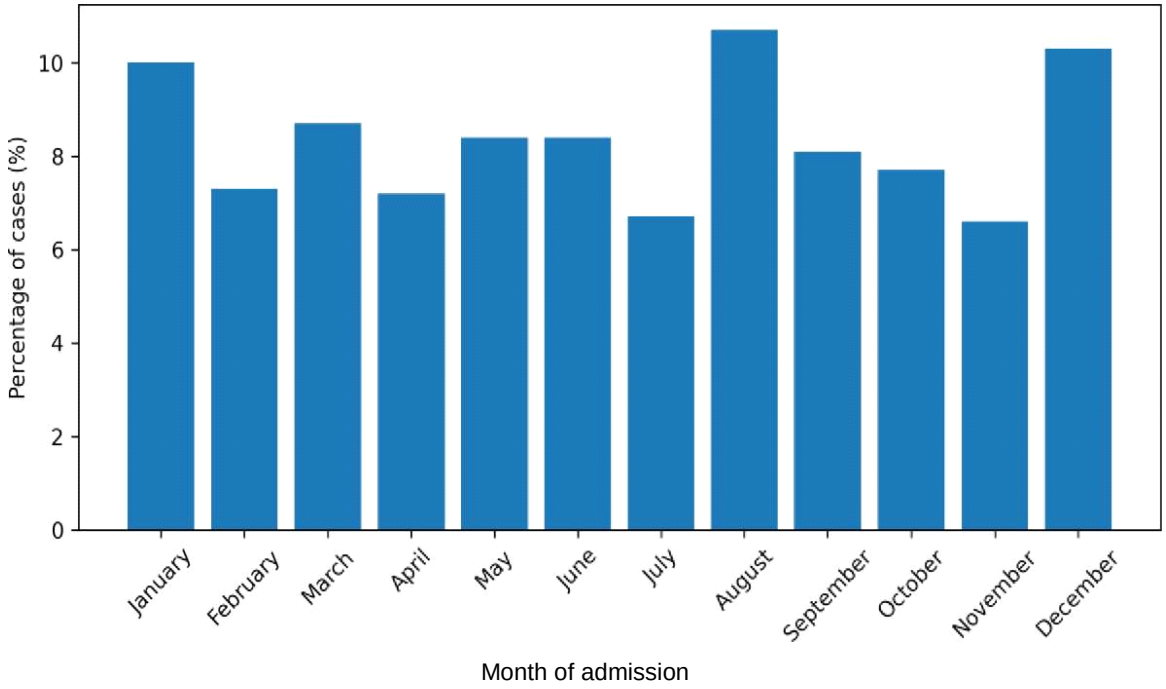
Table 1. Demographic characteristics of patients

Variable	n (%)
Age (mean ± SD)	31.8 ± 12.8
Median age	29
Range	12-85
Age groups	
12-20	257 (21.0%)
21-30	419 (34.2%)
31-40	286 (23.3%)
Female	727 (59.3%)
Male	484 (39.5%)
Not specified	15 (1.2%)

Female patients constituted the majority of the sample (n = 727, 59.3%), while males accounted for 39.5% (n = 484). Gender information was unavailable for 15 cases (1.2%).

Regarding temporal distribution, the highest number of admissions occurred in August (n = 131, 10.7%), December (n = 126, 10.3%), and January (n = 122, 10.0%). The monthly distribution of cases is shown in Figure 1. Overall, a modest bimodal pattern was observed, with increases in late winter and late summer.

Figure 1. Monthly distribution of drug-related suicide attempts (2014-2024)



Birth-month analysis showed the highest frequency among individuals born in January (n = 194, 15.8%). Across the ten-year study period, the highest annual incidence occurred in 2016 (n = 243, 19.8%), whereas the lowest frequencies were observed in 2023 (3.7%) and 2024 (3.8%).

Discussion

This study investigated the demographic and temporal characteristics of drug-related suicide attempts over a ten-year period in a regional hospital in Turkiye. The findings showed that suicide attempts by drug overdose occurred predominantly among young adults, particularly in the 21-30 age group. Previous studies have similarly reported that intentional self-poisoning is more common among adolescents and young adults, highlighting the vulnerability of this population to psychosocial stressors and impulsive behaviors (4, 5).

In the present study, females constituted the majority of the cases. This finding is consistent with the well-known "gender paradox" in suicidal behavior, in which women attempt suicide more frequently while men more often die by suicide using more violent methods (2, 6).

Temporal analysis demonstrated modest seasonal variation, with relative increases during late winter and late summer months. Seasonal fluctuations in suicide attempts and self-poisoning have been reported in several epidemiological studies, suggesting that environmental factors, psychosocial stressors, and seasonal changes may influence suicidal behavior patterns (1, 3).

Overall, the findings contribute to the limited regional epidemiological data on drug- related suicide attempts in Turkiye and support previous international observations regarding demographic and temporal trends in suicidal behavior (1, 7, 8, 9).

Conclusion

In conclusion, this ten-year regional analysis showed that drug-related suicide attempts were most frequently observed among young adult females and demonstrated modest temporal variation throughout the year. These findings highlight the importance of identifying demographic risk groups and temporal patterns in suicidal behavior. Recognizing high-risk populations and periods may help guide targeted prevention strategies and improve mental health interventions.

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0347 - *MTHFR* A1298C Polymorphism in Psychosis with Neurodevelopmental Red Flags: A Case Report

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Introduction

Psychotic disorders are influenced by neurodevelopmental, genetic, epigenetic, and metabolic factors. One-carbon metabolism has been proposed as one of the metabolic pathways relevant to psychotic disorders. Disturbances in one-carbon metabolism may contribute to psychotic symptoms through effects on DNA methylation, neurotransmitter synthesis, and mitochondrial function. [1] Methylenetetrahydrofolate reductase *(MTHFR)* is one of the principal enzymes in one-carbon metabolism and plays a central role in maintaining folate-homocysteine balance and supporting methylation processes essential for epigenetic regulation. Reduced *MTHFR* activity may result in decreased methylation capacity and elevated homocysteine levels, thereby affecting monoaminergic transmission and glutamatergic signaling. [1,2] Disrupted monoamine synthesis and methylation processes may impair dopaminergic and serotonergic function, while accumulation of homocysteine may contribute to NMDA receptor dysfunction, oxidative stress, and glutamatergic neurotoxicity, all of which have been implicated in psychotic symptomatology. [2] Common *MTHFR* polymorphisms, including C677T and A1298C, may reduce enzyme activity and thereby alter homocysteine metabolism. Previous studies have more consistently linked the C677T polymorphism with schizophrenia and psychotic symptoms, whereas the A1298C variant has shown more variable associations, including reported links with bipolar disorder and attention-deficit/hyperactivity disorder. [3] In this report, we present a 21-year-old female patient with psychotic symptoms, neurodevelopmental abnormalities, heterozygous *MTHFR* A1298C polymorphism, and elevated homocysteine levels.

Case

A-21-year-old woman was admitted to the psychiatry unit because of behavioral and psychotic symptoms. At admission, she exhibited irritability, hostility toward family members, poor self-care, persecutory and paranoid ideation. Her symptoms had led to functional impairment by one month before admission. Her mental status examination revealed poor self-care, affective lability with occasional irritability, loosened associations, paranoid and persecutory delusions on her thought process and also increased psychomotor activity. Her Positive and Negative Syndrome Scale (PANSS) total score was 115 with a predominance of the positive scale.

Physical examination revealed notable findings for neurodevelopmental disorder. These findings included microcephaly, partially corrected strabismus and dysmorphic features such as simian crease. She had a history of delayed walking, delayed dentition, intermittent urinary incontinence, and hyperactive behaviors during development. Family history revealed that the patient's mother had experienced seizures in childhood and also has strabismus. Our patient had previously required two hospitalizations because of behavioral dysregulation, enuresis, encopresis. There was no clear history in these hospitalization histories suggestive of mood disorder like bipolar affective disorder.

Treatment was initiated with paliperidone and olanzapine, which gradually titrated to 9 mg/day and 20 mg/day, respectively. During the course of hospitalization, neurological evaluation was performed because of her neurodevelopmental history, atypical clinical features and also urinary

symptoms. Cranial Magnetic Resonance Imaging (MRI) revealed brachycephaly and mild cerebellar tonsillar ectopia. Electroencephalography (EEG) demonstrated epileptiform abnormalities in the left temporal and frontocentral regions. Because of these EEG findings, valproate was added to treatment and titrated to 1000 mg/day. During hospitalization, the patient showed marked improvement in psychotic symptoms and behavioral disorganization. Her irritability decreased, self-care improved and urinary incontinence resolved. At discharge, her PANSS score had decreased from 115 to 75 with greater improvement in positive symptoms, although residual symptoms remained.

Because of the combination of psychosis, neurodevelopmental abnormalities and the patient's dysmorphic features; genetic and metabolic workup had been initiated. The evaluation showed elevated homocysteine levels and a heterozygous *MTHFR* A1298C polymorphism. On the basis of these findings the patient was referred to the Metabolism Department for further assessment and possible replacement therapy. During outpatient follow-up, she remained in remission and her paliperidone dose was gradually reduced and stopped.

Conclusion

Although previous studies have more commonly reported stronger relation between schizophrenia and the *MTHFR* C677T polymorphism with male predominance, this case illustrates a clinical appearance including psychotic symptoms in a female patient with *MTHFR* A1298C polymorphism. [2,3,4] The presence of neurodevelopmental abnormalities, atypical clinical features, physical findings, and partial treatment response supports a possible contribution of congenital disease or epigenetic changes. [5] This case is also important in highlighting the need to consider genetic and epigenetic changes, given the possible contribution of changes in one-carbon metabolism to psychosis. Some studies have shown that homocysteine levels are also related with psychotic symptoms and their severity. [6] Evaluating homocysteine levels in patients with psychosis may be valuable as part of a comprehensive clinical approach. Also assessment of *MTHFR* polymorphisms and related metabolic parameters may be clinically meaningful in selected patients with atypical features or treatment-resistant psychosis. Besides, folate and B-vitamin supplementation may represent a promising strategy in selected cases, particularly when abnormalities in folate-pathway metabolism are identified. [7] Our case emphasizes psychosis may arise from different etiological factors. One-carbon metabolism, genetic variations, and epigenetic mechanisms, underline the importance of integrating metabolic and genetic evaluations into the diagnostic and therapeutic process, particularly in patients with atypical clinical features.

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0353 - Cervical Dystonia Following Dental Extraction in a 13-year-old Girl with Autism Spectrum Disorder: A Case Report

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Introduction

Cervical dystonia is a focal movement disorder characterized by sustained or intermittent involuntary contractions of the cervical muscles, leading to abnormal postures and repetitive movements of the neck (1). It is most commonly idiopathic (primary); however, in some cases, it may develop secondary to peripheral injury (2). Peripherally induced movement disorders (PIMD) were first defined in 1899 (3), and several cases have subsequently been reported (4,5). Dental procedures have been identified as potential precipitating factors in peripherally induced dystonia (6,7). In previously reported cases, dystonia developing after dental interventions has most frequently manifested as oromandibular dystonia (2,6,7). In this report, we present a rare case of cervical dystonia that developed following dental extraction in a 13-year-old child with autism spectrum disorder.

Case Presentation

A 13-year-old nonverbal girl with a diagnosis of autism spectrum disorder was referred to our clinic due to the acute onset of abnormal neck posture and pain. Her medical history revealed ongoing treatment with methylphenidate 27 mg/day and aripiprazole 5 mg/day for behavioral symptoms. There was no prior history of movement disorders, neurological disease, or recent infection.

The patient had undergone extraction of five teeth under general anesthesia. The anesthetic course was reported as uneventful, and no perioperative complications were documented. Shortly after recovery from anesthesia, she developed painful forward flexion of the neck. The abnormal posture was sustained and exacerbated by attempts at voluntary movement.

Due to concern for possible cervical spine pathology or intracranial involvement, the patient was evaluated by neurology and neurosurgery services. Physical and neurological examinations revealed no focal deficits apart from the abnormal neck posture. Laboratory investigations and neuroimaging studies, including cervical and cranial imaging, were unremarkable.

Upon evaluation in our clinic, an acute cervical dystonic reaction was suspected based on the clinical presentation. Intramuscular biperiden (2 mg) was administered as anticholinergic treatment. A rapid clinical response was observed, with significant improvement in neck posture and pain within a short period, followed by complete resolution of symptoms. No recurrence was noted during follow-up. The patient was discharged with recommendations for medication review and outpatient monitoring.

Conclusion

This case report suggests that dental procedures may act as a triggering factor for cervical dystonia in pediatric patients, particularly in those with neurodevelopmental disorders. Increased clinical

awareness of this rare presentation may help clinicians avoid unnecessary diagnostic procedures, reduce patient distress, and ensure timely and effective management.

Keywords: Autism, cervical, child, dystonia

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There are no conflicts of interest related to this case report

0356 - Investigation of Plasma Amino Acid Profiles in Children Diagnosed with Obsessive- Compulsive Disorder Using LC-MS/MS Analysis

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Objective: Obsessive-compulsive disorder (OCD) is a psychiatric disorder characterized by obsessions and compulsions, typically following a chronic course and significantly impairing functioning. The lifetime prevalence in the pediatric population ranges between 0.25% and 4%. Its etiology remains unclear, with genetic, biological, and environmental factors acting together. Current evidence indicates that, in addition to dopaminergic, serotonergic, and glutamatergic dysfunction, neuroinflammation and oxidative imbalance contribute to OCD pathophysiology.

Amino acids play a key role in the biochemical basis of neuropsychiatric disorders as precursors of neurotransmitters and regulators of brain development and neurotransmission. Although research in this area has increased, data on OCD remain limited and are mostly derived from adult populations using limited panels and less sensitive methods.

This study aimed to evaluate plasma amino acid levels in pediatric OCD using liquid chromatography-tandem mass spectrometry (LC-MS/MS), compare them with healthy controls, and examine their association with symptom severity to identify potential biochemical mechanisms and candidate biomarkers.

Methods:

Study Design

This cross-sectional case-control study was approved by the Ataturk University Non- Interventional Clinical Research Ethics Committee (Approval No: B.30.2.ATA.0.0A.00/204). All participants and their families were informed about the study, and written informed consent was obtained.

Participants

The study included 39 children and adolescents aged 8-17 years diagnosed with OCD according to DSM-5 criteria at the Child and Adolescent Psychiatry Outpatient Clinic of Ataturk University Research Hospital, along with 39 age- and sex-matched healthy controls. Participants had not received prior pharmacological or psychotherapeutic treatment or had been treatment-free for at least 6 months. Individuals with metabolic, genetic, endocrine, neurological, or chronic diseases, active infection, special dietary habits, or use of vitamin/amino acid supplements within the previous two months that could affect amino acid metabolism were excluded. Sample size was calculated a priori using G*Power software (a=0.05; power=80%; Cohen's d=0.66), and at least 37 participants per group were considered sufficient.

Measurements

The Schedule for Affective Disorders and Schizophrenia for School-Age Children - Present and Lifetime Version, DSM-5 Turkish Adaptation (K-SADS-PL-DSM-5-T) and the Revised Child Anxiety and Depression Scale (RCADS) were administered to assess comorbidities. OCD symptom severity was evaluated using the Children's Yale-Brown Obsessive Compulsive Scale (CY-BOCS).

Venous blood samples were collected between 09:00 and 10:00 after at least 8 hours of fasting. Routine biochemical parameters were measured using standard autoanalyzers. Plasma samples were separated by centrifugation and stored at -80°C until analysis. Plasma levels of 43 amino acids and related metabolites were

measured using liquid chromatography-tandem mass spectrometry (LC-MS/MS; Agilent 6460 Triple Quadrupole) with the JASEM amino acid kit.

Statistical Analysis

Categorical variables were expressed as numbers and percentages, and continuous variables as mean ± standard deviation. Group comparisons were performed using Student's t-test or Mann- Whitney U test according to data distribution. Amino acid levels were analyzed using analysis of covariance (ANCOVA) controlling for age, sex, and body mass index (BMI), and effect sizes were reported as partial eta-squared (η^2p). For multiple comparisons, False Discovery Rate (FDR) correction was applied using the Benjamini-Hochberg method. The discriminative performance of significant metabolites was evaluated by receiver operating characteristic (ROC) analysis. Associations between significant amino acids and CY-BOCS scores were examined using correlation analyses. Statistical analyses were performed using SPSS 24.0, and ROC analyses were conducted using R 3.6.1, with $p<0.05$ considered statistically significant.

Results: The study included 78 children and adolescents. The OCD group comprised 39 participants (18 males, 21 females), and the control group included 39 participants (14 males, 25 females). There were no significant differences between the groups in terms of age, sex, body mass index, educational level, or sociodemographic characteristics ($p>0.05$).

No significant differences were observed between the groups in WBC, neutrophil percentage, lymphocyte percentage, platelet count, AST, ALT, BUN, creatinine, CRP, or vitamin B12 levels ($p>0.05$). However, folate levels were significantly lower in the OCD group compared to controls ($p=0.002$).

Plasma amino acid levels were analyzed using ANCOVA controlling for age, sex, and BMI. Glycine, aspartic acid, carnosine, and ethanolamine levels were higher in the OCD group, whereas phenylalanine levels were higher in controls. In contrast, cystine levels were markedly lower in the OCD group and showed the highest effect size ($\eta^2p=0.255$).

After Benjamini-Hochberg FDR correction, only cystine levels remained significant between groups ($q<0.05$).

ROC analysis demonstrated that cystine levels discriminated OCD patients from controls (AUC=0.764; 95% CI: 0.656-0.872; $p<0.001$). The optimal cut-off value was 34.5 nmol/mL, with 64% sensitivity and 82% specificity.

No significant associations were found between altered amino acids and CY-BOCS total score or folate levels ($p>0.05$).

Discussion: In this study, significant differences in plasma amino acid profiles were identified in the pediatric OCD group; after FDR correction for multiple comparisons, only cystine levels remained significant. This finding indicates that decreased plasma cystine represents the most robust and reliable biochemical result of the study. ROC analysis for cystine demonstrated a moderate discriminative performance (AUC=0.764). These results suggest that cystine is not only a statistically significant variable but may also serve as a potential biomarker candidate with clinical discriminative utility.

The biological significance of decreased cystine levels is noteworthy in relation to glutamatergic neurotransmission and cellular redox balance. Cystine regulates extracellular glutamate levels via the cystine-glutamate antiporter system, which establishes a feedback mechanism inhibiting synaptic glutamate release through mGluR2/3 activation (1). Decreased cystine levels may weaken this inhibitory mechanism and contribute to glutamatergic hyperactivity. Additionally, cystine serves as a source of cysteine required for glutathione synthesis and is an essential component of the cellular antioxidant defense system. Therefore, low cystine levels may be associated with oxidative stress. Although oxidative stress markers were not measured, this finding suggests a potential link between glutamatergic dysregulation and redox imbalance. The literature on the therapeutic effects of N-acetylcysteine on OCD symptoms is also consistent with this biochemical pathway (2).

The increased levels of glycine and aspartic acid observed in our study indicate alterations in excitatory amino acid metabolism. Glycine functions as a co-agonist at NMDA receptors and modulates glutamatergic transmission. Similarly, aspartate, together with glutamate, is among the primary excitatory ligands of NMDA receptors (3). The increase in these molecules suggests a possible elevation in excitatory tone within the glutamatergic system.

However, the loss of statistical significance after FDR correction indicates that these changes should be interpreted as hypothesis-generating. Furthermore, as the measurements were obtained from peripheral blood, these amino acid levels may not directly reflect central neurotransmission.

The lack of statistically significant findings for glutamate levels suggests that dysregulation in glutamate homeostasis, rather than its absolute concentration, may be more relevant in OCD. Glutamate is tightly regulated in the central nervous system, and peripheral levels have limited capacity to reflect central alterations. Therefore, our findings support that the glutamatergic system in OCD should be evaluated not solely based on glutamate levels but within a broader biochemical network including receptor activity, transporter systems, and co-agonist dynamics.

The increases in ethanolamine and carnosine levels represent other notable findings. Ethanolamine, a precursor of anandamide, has been implicated in neuropsychiatric disorders and may influence neurotransmitter systems, including serotonergic pathways, and be related to alterations in phospholipid metabolism and associated signaling processes (4) and is also associated with phospholipid metabolism and mitochondrial membrane integrity. Carnosine, a dipeptide with antioxidant and anti-inflammatory properties, can reduce reactive oxygen species and exert regulatory effects on the glutamatergic system (5). These findings suggest that, in pediatric OCD, not only neurotransmission but also cellular stress responses and energy metabolism may play a role.

The decreased phenylalanine levels may be related to phenylalanine-tyrosine-catecholamine metabolic pathways. However, it is not possible to directly infer central neurotransmitter levels from peripheral amino acid concentrations. Moreover, since phenylalanine is an essential amino acid, dietary factors should be considered as a potential influence on this finding.

Lower folate levels may be associated with neurotransmitter synthesis and methylation processes (6); however, the absence of a significant association between folate and the significant amino acids suggests that these alterations may be too complex to be explained by a single biochemical pathway.

To the best of our knowledge, this study is among the first to comprehensively examine cystine levels within a broad amino acid profiling approach in pediatric OCD and to demonstrate its clinical discriminative potential. The persistence of cystine after FDR correction and its ROC performance support its role not only as a statistical finding but also as a clinically applicable biomarker candidate. The findings indicate a biochemical dysregulation in pediatric OCD, particularly involving glutamatergic regulation and oxidative stress processes through cystine, and suggest that this molecule may also be considered a potential therapeutic target. In this respect, the study demonstrates that OCD pathophysiology is not limited to monoaminergic systems but should be addressed within a broader biochemical network, offering a perspective that may contribute to biomarker-based diagnosis and targeted treatment approaches. However, further studies with larger samples, longitudinal designs, and multimodal approaches are required to validate these findings.

Keywords: Obsessive-Compulsive Disorder; Child; Cystine; Biomarkers; Tandem Mass Spectrometry; Amino Acids

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Table 1. Demographic characteristics of participants

Variable	**Category**	**OCD**	**Control**	**p**
		n(%) / Mean ± SD	**n(%) / Mean ± SD**	
Age (years)		12,74 ± 2,49	11,97 ± 2,31	0,162[c]
Gender	Male	18(46,2)	14(35,9)	0,357[a]
	Female	21(53,8)	25(64,1)	
Education (years)		7,46 ± 2,37	7,15 ± 2,20	0,555 [c]
BMI		18,07 ± 2,91	17,76 ± 2,00	0,583[c]
Maternal psychiatric history	Yes No	5(12,8) 34(87,2)	4(10,3) 35(89,7)	1,000[b]
Maternal education (years)		9,10 ± 4,38	9,30 ± 4,65	0,842[c]
Paternal psychiatric history	Yes No	3(7,7) 36(92,3)	2(5,1) 37(94,9)	1,000[b]
Paternal education (years)		10,58 ± 4,03	10,12 ± 3,88	0,609 [c]
Family income	Low	14 (35,9)	11 (28,2)	0,877[b]
	Middle	22 (56,4)	25 (64,1)	
	High	3 (7,7)	3 (7,7)	

a.Pearson Chi-Square b.Fisher's Exact Test c.Student's T-Test

Table 2. Hematological and non-amino acid biochemical parameters

Parameter	**OCD (n=39)**	**Control (n=39)**	**P**
	Mean ± SD	**Mean ± SD**	
WBC (10^3	7,52 ± 2,01	8,19 ± 1,99	0,144[a]
Neutrophil (%)	52,23 ± 9,74	54,24 ± 8,69	0,339[a]
Lymphocyte (%)	37,84 ± 9,17	36,03 ± 8,57	0,372[a]
Platelet (10^3	321,51 ± 66,96	308,38 ± 56,98	0,352[a]
AST (U/L)	21,14 ± 5,64	21,89 ± 5,96	0,573[a]
ALT (U/L)	14,24 ± 3,47	14,28 ± 5,08	0,966[a]
BUN (mg/dl)	9,83 ± 2,43	10,14 ± 2,71	0,598[a]
Creatinine (mg/dl)	0,54 ± 0,11	0,54 ± 0,08	0,859[a]
CRP (mg/l)	2,49 ± 1,22	2,66 ± 3,75	0,252[b]
Vitamin B12 (pg/ml)	240,84 ± 92,08	263,25 ± 117,77	0,555[b]
Folate (ng/ml)	7,88 ± 3,30	10,48 ± 3,72	0,002[a]

a.Student's T-Testi b. Mann-Whitney U

Table 3. Plasma amino acids

Amino Acid (nmol/ml)	OCB (n=39)	Control (n=39)	ANCOVA		
	Mean SD	Mean SD	F(1,72)	p	η^2p
Glycine	253,642 11,873	213,538 12,400	5,37	0,023	0,069
Cystine	28,947 2,110	44,210 2,204	24,62	<0,001	0,255
Log-Aspartic Acid	1,919 0,097	1,502 0,102	8,634	0,004	0,107
Log-Carnosine	0,081 0,025	-0,009 0,026	6,057	0,016	0,078
Log-Ethanolamine	1,574 0,051	1,367 0,054	7,678	0,007	0,096
Log-Phenylalanine	3,968 0,032	4,077 0,034	5,369	0,023	0,069
Log-Cystathionine	-0,724 0,077	-0,947 0,081	3,939	0,051	0,052
Tryptophan	68,581 1,933	73,306 2,018	2,813	0,098	0,038
Tyrosine	77,222 3,107	81,942 3,245	1,086	0,301	0,015
Isoleucine	51,200 1,89	50,041 1,974	0,177	0,675	0,002
GABA	0,073 0,002	0,072 0,002	0,336	0,564	0,005
Norvaline	10,746 0,254	10,881 0,266	0,133	0,717	0,002
Valine	279,984 8,654	291,914 9,038	0,894	0,347	0,012
Alanine	464,965 15,377	436,553 16,060	1,607	0,209	0,022
Asparagine	62,857 2,416	66,231 2,523	0,918	0,341	0,013
Proline	274,726 12,962	278,612 13,537	0,042	0,838	0,001
Glutamine	732,804 10,304	720,153 10,762	0,709	0,402	0,010
Arginine	67,841 3,601	64,654 3,761	0,369	0,546	0,005
Log-Taurine	6,086 0,064	5,975 0,066	1,425	0,236	0,019
Log-Leucine	4,662 0,032	4,617 0,034	0,950	0,333	0,013
Log-Methionine	3,485 0,037	3,425 0,038	1,262	0,265	0,017
Log-2-Aminoadipic Acid	-0,123 0,044	-0,086 0,046	0,320	0,573	0,004
Log-Glutamic Acid	3,671 0,055	3,550 0,057	2,270	0,136	0,031
Log-Beta Alanine	1,199 0,094	1,157 0,098	0,095	0,758	0,001
Log-2-Aminobutyric Acid	0,793 0,057	0,669 0,059	2,274	0,136	0,031

Amino Acid (nmol/ml)	OCB (n=39)	Control (n=39)	ANCOVA		
	Mean SD	Mean SD	F(1,72)	p	η^2p
Log-Threonine	5,065 0,046	5,083 0,048	0,071	0,790	0,001
Log-Serine	5,040 0,037	5,048 0,038	0,023	0,880	<0,001
Log-Trans-4-Hydroxyproline	1,813 0,040	1,855 0,042	0,486	0,488	0,007
Log-Sarcosine	3,681 0,110	3,992 0,115	3,737	0,057	0,049
Log-Citrulline	3,347 0,046	3,436 0,048	1,733	0,192	0,024
Log-O-Phosphoethanolamine	0,911 0,190	0,583 0,199	1,405	0,240	0,020
Log-O-Phosphoserine	-0,174 0,042	-0,226 0,043	0,713	0,401	0,010
Log-Argininosuccinic Acid	-0,636 0,054	-0,526 0,056	1,975	0,164	0,027
Log-Histidine	4,584 0,072	4,670 0,075	0,687	0,410	0,009
Log-Ornithine	4,640 0,048	4,674 0,050	0,247	0,621	0,003
Log-Lysine	2,198 0,014	2,192 0,015	0,083	0,775	0,001
Log-Anserine	0,180 0,004	0,185 0,005	0,698	0,406	0,010
Log-1-Methylhistidine	0,366 0,013	0,366 0,013	0,089	0,766	0,001

An analysis of covariance (ANCOVA) was performed to compare the two groups while controlling for age, sex, and BMI.

Table 4. FDR-Corrected results

Amino Acid (nmol/ml)	OCd (n=39)	Control (n=39)	F(1,72)	p	FDR q	η^2p
	Mean SD	Mean SD				
Cystine	28,95 2,11	44,21 2,20	24,62	<0,001	0,019	0,255

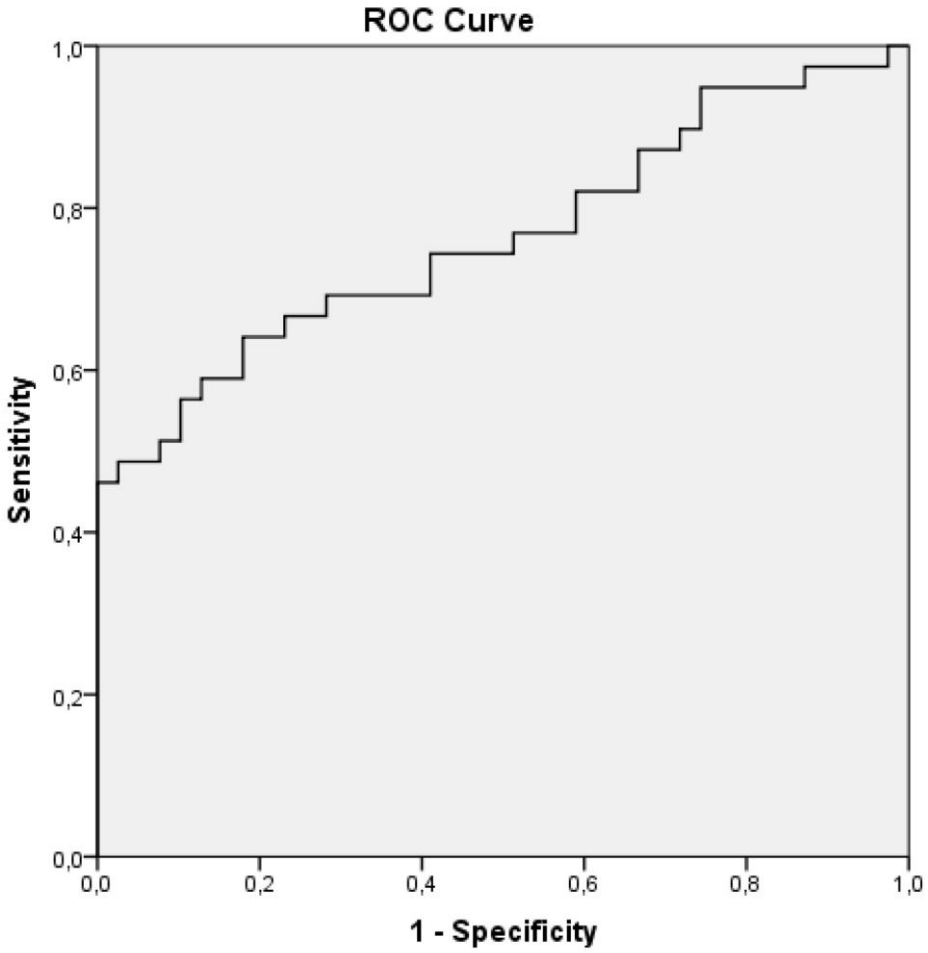


Figure 1. Receiver operating characteristic (ROC) curve for cystine

0359 - Substance/Medication-Induced Psychotic Disorder Following Beta-Blocker Use: A Pediatric Case Report

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Introduction

Beta-adrenergic receptor antagonists (beta-blockers) are widely used agents, particularly in the treatment of cardiovascular diseases. Lipophilic beta-blockers are known to cross the blood- brain barrier more readily and are therefore more prone to cause central nervous system (CNS) adverse effects.

Reported CNS side effects include hallucinations, vivid dreams, sleep disturbances, delirium, depression, and psychotic symptoms. These effects typically emerge within the first week following initiation of treatment and are often associated with higher doses. However, neuropsychiatry adverse effects have also been reported at low doses. Data in the pediatric population remain limited.

In this report, we present the case of an 8-year-old child who developed acute-onset psychotic symptoms following treatment with propranolol, a lipophilic beta-blocker.

Case Presentation

An 8-year-old male patient with a history of congenital biliary atresia, who had undergone Kasai portoenterostomy at 3 months of age, was being followed with diagnoses of cirrhosis and pulmonary hypertension and was in the evaluation process for liver transplantation. He was referred to the child and adolescent psychiatry clinic due to hospital phobia and behavioral problems occurring prior to invasive procedures.

During the psychiatric evaluation, the patient reported feelings of hopelessness related to previous unsuccessful surgeries, intense fear of surgical interventions, and a desire not to die. Family history revealed symptoms of hyperactivity, impatience, and attention difficulties since early childhood, which were also supported by teacher reports. An increase in irritability and restlessness had been noted over the past month. Mental status examination demonstrated increased psychomotor activity, impatience, interrupting behavior, and excessive speech, findings consistent with attention-deficit/hyperactivity disorder (ADHD). Based on the clinical evaluation, diagnoses of ADHD and adjustment disorder with anxiety symptoms were established.

Treatment was initiated with sertraline 25 mg/day (with planned titration), methylphenidate 10 mg/day, and low-dose haloperidol (3 drops twice daily) to manage behavioral agitation prior to medical procedures. At the one-month follow-up, a marked reduction in anxiety and irritability was observed, and the treatment regimen was maintained.

Approximately one month later, propranolol 40 mg/day was initiated for pulmonary hypertension. Beginning on the second day of treatment, the patient developed severe aggression, destructive behavior toward the environment, threatening behavior with a knife, paranoid thoughts of being harmed, and visual hallucinations described as seeing "jinn." In addition, he attempted to set the house on fire, made attempts to run away from home, exhibited shouting and talking during sleep, and developed secondary enuresis.

On mental status examination, consciousness was clear, orientation to person, place, and time was intact, and attention and concentration were preserved, with no evidence of fluctuation in consciousness. No findings suggestive of delirium were observed.

Considering the temporal relationship between the initiation of propranolol and the onset of symptoms, the absence of a prior history of psychosis, the lack of clinical and laboratory findings consistent with hepatic encephalopathy, and the rapid resolution of symptoms following discontinuation of propranolol, the clinical picture was evaluated as substance/medication-induced psychotic disorder according to DSM-5-TR criteria. The Naranjo Adverse Drug Reaction Probability Scale score was 5, supporting a probable drug-related association.

Discussion

Propranolol, due to its high lipophilicity, has greater penetration into the CNS, which may increase the risk of neuropsychiatric adverse effects.

One proposed mechanism involves suppression of noradrenergic neurotransmission through postsynaptic beta-adrenergic and serotonergic receptor blockade. However, the exact pathophysiology of propranolol-induced psychosis remains unclear.

Psychosis and visual hallucinations associated with metoprolol have also been reported [1,2]. More recent case reports have documented neuropsychiatric symptoms related to propranolol use. In a case reported in 2021, visual and auditory hallucinations developed after initiation of propranolol; although initially considered hepatic encephalopathy, organic causes were excluded, and symptoms resolved completely following discontinuation of propranolol [3].

Similarly, delayed-onset hypnopompic visual hallucinations have been reported after long-term propranolol use, with significant improvement following switching to a less lipophilic beta- blocker such as carvedilol or atenolol. These findings suggest that CNS penetration of propranolol may have clinical relevance not only for acute-onset but also delayed neuropsychiatric symptoms.

Recent systematic reviews have also demonstrated an association between lipophilic beta- blockers and neuropsychiatric adverse effects such as hallucinations and confusion [4]. These findings suggest that propranolol may induce psychotic symptoms across different clinical contexts and age groups [5]. The present case supports this literature by demonstrating acute- onset psychotic symptoms with marked behavioral dysregulation in a pediatric patient.

Key words: Adrenergic beta-Antagonists, Child, Hallucinations, Psychotic Disorders, Substance-Related Disorders

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0361 - Examining the Relationship Between Death Literacy and Death Anxiety

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Background and Objective: Death anxiety is a multidimensional construct encompassing emotional, cognitive, somatic, and behavioral responses to the awareness of one's own mortality. It plays a significant role in psychological distress and various mental health disorders. Death literacy, defined as the knowledge and skills required to understand and navigate end-of-life issues, has recently emerged as an important concept in public health and palliative care. Although growing evidence suggests that death literacy may enhance psychological resilience and facilitate healthier engagement with death-related topics, research examining its relationship with death anxiety remains limited, particularly in Turkey. This cross-sectional online study aimed to investigate the relationship between death literacy and death anxiety in a community sample.

Methods: This study was conducted as an online cross-sectional survey among individuals aged 18 years and older living in Turkey who were able to read and understand Turkish and provided informed consent. Participants were recruited via social media platforms and email. Data were collected using a Demographic Information Form, the Turkish version of the Death Literacy Index (DLI), and the Templer Death Anxiety Scale (DAS). Ethical approval for the study was obtained from the Non-Interventional Research Ethics Committee of Cumhuriyet University (Date of approval: 09.10.2025, Decision No: 2025-10/33).

Results: The mean age of participants was 27.39 ± 12.06 years; 54.4% were female, 77.6% were single, and 77.2% were university graduates. The mean death anxiety score was 8.33 ± 3.16 (range: 1-15), indicating generally elevated levels of death anxiety. The mean total death literacy score was 80.17 ± 16.24 (range: 29-116). Correlation analysis revealed a significant moderate negative relationship between death literacy and death anxiety ($r = -.30$, $p < .001$), suggesting that higher levels of death literacy were associated with lower levels of death anxiety. Age was positively correlated with death anxiety ($r = .16$, $p < .001$), while no significant relationship was found between age and death literacy. Multiple linear regression analysis demonstrated that the overall model was significant ($F(4, 521) = 24.73$, $p < .001$) and explained 16% of the variance in death anxiety ($R^2 = .16$). Death literacy emerged as a significant negative predictor of death anxiety ($B = -0.047$, $p < .001$). Age was a positive predictor ($B = 0.040$, $p < .001$), whereas male gender ($B = -1.222$, $p < .001$) and higher educational level ($B = -0.621$, $p = .011$) were significant negative predictors of death anxiety.

Conclusions: The findings indicate that death literacy is significantly and inversely associated with death anxiety in a community sample. Individuals with greater knowledge, skills, and communicative competence regarding death and end-of-life issues appear to experience lower levels of anxiety related to mortality. These results suggest that death literacy may function as a protective and modifiable factor in reducing death anxiety. Public health initiatives, educational programs, and community-based interventions aimed at improving death literacy could contribute not only to enhanced end-of-life preparedness but also to improved psychological well-being. Future longitudinal and interventional studies are recommended to further clarify the causal mechanisms underlying this relationship and to develop targeted strategies for vulnerable populations.

Keywords: Death literacy, Death anxiety, End-of-life awareness, Psychological resilience

0363 - The Effect of Child-Centered Play Therapy on Self-Compassion and Emotion Regulation Skills in Children Diagnosed with Cancer

Introduction and Aim

Childhood cancer is a traumatic process that affects not only physical health but also the psychological and emotional development of children. The diagnosis and treatment process leads to intense emotional responses such as anxiety, fear, and stress, and may negatively affect children's emotion regulation skills [1]. In addition, low levels of self-compassion make it more difficult for children to cope with the challenges they experience and may negatively affect their psychological adjustment [2][3].

Emotion regulation is defined as the ability of an individual to recognize, control, and express emotions appropriately, whereas self-compassion refers to the individual developing an understanding, accepting, and supportive attitude toward themselves [4]. These two constructs are among the fundamental internal resources that increase psychological resilience, especially in stressful life conditions such as chronic illness [3][5].

Play therapy is an effective psychosocial intervention method that enables children to express their emotions in natural ways. In particular, the child-centered play therapy approach has been demonstrated in many studies to be effective in resolving children's emotional and behavioral problems and in supporting their psychological well-being [6][7].

In this context, the aim of this study is to examine the effects of child-centered play therapy on the emotion regulation and self-compassion levels of children diagnosed with cancer.

Method

The study was conducted using a pretest-posttest control group experimental design. The study was carried out at Necmettin Erbakan University Faculty of Medicine, Department of Pediatrics, and during the implementation process, a dedicated play therapy room was established within the hospital. Initially, 40 children were included in the study, and at the end of the process, the research was completed with 34 children (17 experimental, 17 control). Child-centered play therapy was applied to the experimental group for 12 weeks, with 1-2 sessions per week, each lasting approximately 20 minutes. The entire process lasted approximately 5 months (June-October 2024). No therapy was applied to the control group during the research process, and only routine medical care was maintained. However, after the study was completed, 12 sessions of child-centered play therapy were also applied to this group in accordance with ethical principles.

Results

At the beginning of the study, it was observed that the emotion regulation levels of the experimental and control groups were quite close to each other. The mean of the experimental group was determined as 2.40, while the mean of the control group was determined as 2.38. The analyses conducted showed that there was no statistically significant difference between the groups ($U = 173.50$, $p = .84$).

A similar situation was also present in terms of self-compassion levels at the beginning. The mean of the experimental group was found to be 32.53, while the mean of the control group was found to be 33.94, and it was determined that this difference was not statistically significant ($U = 115.50$, $p = .31$).

After the implementation, a clear change was observed in terms of emotion regulation skills. While the mean of the experimental group increased to 2.93, the mean of the control group remained at 2.60. The analyses showed that there was a statistically significant difference in favor of the experimental group ($U = 82.50$, $p < .05$).

A significant increase was also observed in self-compassion levels. While the mean of the experimental group increased to 40.88, the mean of the control group remained at 33.94. It was determined that this difference was quite strong and statistically significant ($U = 26.50$, $p < .001$).

These findings show that child-centered play therapy is effective in both improving emotion regulation skills and increasing self-compassion.

Conclusion and Contribution

This study reveals that child-centered play therapy is an effective intervention method in supporting the psychological well-being of children diagnosed with cancer. The fact that children can regulate their emotions in a healthier way and develop a more understanding attitude toward themselves are among the main gains of this therapy. In addition, the establishment of a dedicated space for play therapy within the hospital provides an important model in terms of integrating psychosocial interventions into clinical settings. In this respect, the study provides a strong contribution to the field of pediatric oncology both scientifically and in practice.

This study not only presents statistically significant results but also puts forward a concrete and applicable model that touches the emotional world of children diagnosed with cancer. It is seen that a structured play therapy application within a hospital environment strengthens children's psychological resilience and contributes to the development of a more compassionate inner attitude toward themselves. In this respect, the study makes a strong contribution to a holistic care approach in the field of pediatric oncology and emphasizes that not only the physical but also the emotional well-being of children should be supported.

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0366 - Melanopsin-Mediated Pupillary Responses in Remitted Bipolar Disorder Using Chromatic Pupillometry: A Preliminary Report

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Introduction

Visible light, particularly in the short-wavelength blue spectrum, plays a crucial role in regulating circadian rhythms, mood, and sleep-wake cycles. These effects are mediated primarily through melanopsin-containing intrinsically photosensitive retinal ganglion cells (ipRGCs), a specialized class of retinal neurons that transmit non-image-forming photic signals to several brain regions involved in circadian regulation (1). IpRGCs project to structures such as the suprachiasmatic nucleus (SCN), the central circadian pacemaker, as well as other brain areas involved in alertness, mood regulation, and hormonal rhythms (2). Through these pathways, light exposure modulates physiological processes including melatonin secretion, sleep timing, alertness, and emotional regulation. Blue light is particularly effective in activating ipRGCs because melanopsin has peak sensitivity in the short-wavelength range of the visible spectrum (3).

Bipolar disorder (BD) is strongly associated with disturbances in circadian rhythms and abnormal sensitivity to environmental light cues. Clinical and epidemiological evidence suggests that disruptions in biological rhythms—such as sleep deprivation, seasonal changes, or altered light exposure—can trigger affective episodes including depression and mania. These observations support the hypothesis that abnormalities in light processing and circadian entrainment may play a role in the pathophysiology of BD (4).

Early research proposed the concept of retinal supersensitivity to light in BD, based on findings showing enhanced melatonin suppression following light exposure in some patients and their offspring (5). This led to the suggestion that altered light sensitivity could represent a biological vulnerability or endophenotype associated with the disorder. However, subsequent studies have produced inconsistent results, indicating that the relationship between light sensitivity and BD remains incompletely understood.

One method for evaluating the functional integrity of the ipRGC system is chromatic pupillometry, a non-invasive technique that measures pupillary responses to specific wavelengths of light (6). When ipRGCs are stimulated by blue light, they produce a sustained pupillary constriction that persists even after the light stimulus is removed. This phenomenon is known as the post-illumination pupil response (PIPR) and is considered a physiological marker of melanopsin-driven activity. Because PIPR reflects ipRGC function, chromatic pupillometry has emerged as a useful tool for investigating non-visual light responses and circadian phototransduction in both healthy individuals and clinical populations.

Despite increasing interest in circadian mechanisms in mood disorders, relatively few studies have investigated melanopsin-mediated pupillary responses in BD. Therefore, the present preliminary study aimed to evaluate blue light-induced pupillary responses using chromatic pupillometry in patients with BD in remission compared with healthy control participants, thereby exploring potential alterations in melanopsin-driven photic signaling associated with the disorder.

Methods

This preliminary cross-sectional study included patients with BD (n = 5) and healthy control participants (n = 10). The mean age of the total sample was 30.5 ± 5.72 years, with 31.5% male and 62.5% female participants. All BD participants were in strict remission at the time of assessment, defined as scores below 7 on both the Hamilton Depression Rating Scale (HDRS) and the Young Mania Rating Scale (YMRS).

All participants underwent a comprehensive ophthalmologic examination prior to pupillometric testing in order to exclude ocular pathology that might influence pupillary responses. This examination included assessment of visual acuity and optical coherence tomography (OCT) imaging to rule out retinal or optic nerve abnormalities. Participants with refractive errors exceeding ±3 diopters were excluded to minimize potential optical confounds affecting retinal illumination and pupillary measurements.

Chromatic pupillometry was conducted in accordance with established methodological standards for pupillography (7). Pupillary responses were recorded using the multiomodal chromatic pupillometer (MonCV One, Metrovision, France), an infrared video-based pupillography device designed for assessment of pupil light reflexes. The system allows continuous recording of pupil diameter under controlled light stimulation conditions using integrated chromatic LED light sources. Prior to stimulation, participants underwent a 10- minute dark adaptation period in a dimly lit environment to standardize retinal adaptation and stabilize baseline pupil diameter. Following dark adaptation, participants were exposed to a 1- second pulse of short-wavelength blue light, which preferentially stimulates ipRGCs. Pupil diameter was recorded continuously during and after light stimulation. Continuous recording enables precise quantification of the post-illumination pupil response (PIPR), a sustained pupillary constriction occurring after stimulus offset that reflects melanopsin-driven ipRGC activity. Baseline pupil diameter was determined from the pre-stimulus recording period. All pupillary response parameters were normalized relative to baseline pupil diameter to control for interindividual differences in resting pupil size.

Primary outcome measures included: early area under the curve (AUC) of the pupil response following stimulus offset; late AUC reflecting sustained pupil constriction; PIPR at 6 seconds and 25 seconds after light offset and melanopsin-mediated PIPR amplitude, representing the magnitude of sustained pupillary constriction relative to baseline.

Results

The analysis revealed differences in ipRGC-mediated pupillary responses between patients with BD and healthy control participants. Specifically, the melanopsin-mediated PIPR amplitude, calculated using a 1-second time window beginning 1.8 seconds after stimulus offset, was significantly reduced in the BD group compared with the control group (0.33 ± 0.10 vs. 0.48 ± 0.08; $p = 0.019$). This finding indicates a lower sustained melanopsin-driven pupillary constriction in individuals with BD.

Additional pupillometric metrics showed a similar trend. Early AUC values, calculated for the 2-10 seconds post-illumination interval, were lower in the BD group compared with controls (14.96 ± 8.73 vs. 23.58 ± 4.26), although this difference did not reach statistical significance ($p = 0.075$). Likewise, late AUC values, representing the sustained pupil response during the 10-25 seconds post-illumination window, were also reduced in the BD group relative to controls (14.41 ± 10.74 vs. 22.54 ± 5.91), but the difference was not statistically significant ($p = 0.129$).

Similarly, the PIPR at 6 seconds showed a trend toward lower values in the BD group compared with controls ($p = 0.055$), while PIPR measured at 25 seconds did not differ between groups ($p = 0.440$).

Finally, exploratory analyses indicated that pupillometric parameters were not significantly associated with demographic variables, including age and sex ($p > 0.05$ for all comparisons), suggesting that these factors did not account for the observed group differences in melanopsin-mediated responses.

Conclusion

The present study demonstrated that melanopsin-mediated pupillary responses to blue light were reduced in patients with BD during remission compared with healthy controls. This finding supports the hypothesis that alterations in ipRGC-mediated photic signaling may persist even during euthymic phases of BD.

The results of the current study are partially consistent with the findings reported by Madsen et al., who conducted a chromatic pupillometry study investigating ipRGC- mediated pupillary responses in BD (8). In their cross-sectional investigation including patients with BD, unaffected relatives, and healthy controls, ipRGC-mediated pupillary responses were not significantly different across the entire groups. However, subgroup analysis revealed that patients in remission exhibited reduced PIPR responses compared with healthy controls. This pattern closely resembles the observations in the present study, where remitted BD patients showed a reduction in melanopsin-

mediated PIPR amplitude relative to controls. The convergence of findings across these studies suggests that attenuated ipRGC responsiveness may represent a persistent physiological characteristic during euthymic phases.

Additionally, the Danish study included unaffected first-degree relatives, allowing the authors to explore whether alterations in ipRGC-mediated photic signaling represent a trait vulnerability marker or a state-related phenomenon in BD. Their findings indicated that ipRGC-mediated pupillary responses were not altered in unaffected relatives, suggesting that these abnormalities may not constitute an inherited endophenotype of BD. However, several methodological aspects of that study should be considered when interpreting the findings. The BD sample included both BD type I and BD type II patients, and the subgroup of patients in remission consisted of only nine individuals (8). Therefore, the results of both the Danish study and the present investigation should be interpreted cautiously.

In addition to BD, reduced melanopsin-driven pupillary responses have been reported in several affective conditions, including seasonal affective disorder and major depressive disorder, suggesting that ipRGC dysfunction may represent a broader biomarker of mood- related circadian dysregulation (9, 10). Because ipRGCs play a central role in transmitting environmental light signals to circadian and mood-regulating brain regions—including the suprachiasmatic nucleus and other hypothalamic structures—altered ipRGC signaling may contribute to the well-documented circadian disturbances observed in BD.

Importantly, the present study represents the first chromatic pupillometry investigation assessing melanopsin-mediated pupillary responses in BD conducted in Turkiye. This contributes to the emerging literature on retinal and circadian biomarkers of mood disorders and provides initial evidence suggesting that melanopsin-driven pupil responses may serve as a potential physiological indicator of circadian dysregulation in BD.

However, several limitations should be considered when interpreting the findings. Most notably, the sample size in the present study was small, which may have limited the statistical power to detect differences across all pupillometric parameters. In addition, psychotropic medications, circadian timing of assessments, and environmental light exposure may influence pupillary dynamics and should be carefully controlled in future studies. Larger studies using standardized pupillometry protocols and longitudinal designs will be necessary to clarify the role of ipRGC function across different illness phases of BD.

In conclusion, the present preliminary findings suggest that melanopsin-mediated pupillary responses are reduced in remitted BD, supporting the hypothesis that abnormalities in ipRGC-mediated photic signaling may contribute to circadian dysregulation in mood disorders.

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0374 - Meaning in Life Profiles and Mental Health Outcomes Among Medical Students: A Latent Profile Analysis

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Objective

Medical students are at increased risk for depression, anxiety, burnout, and suicidal behavior due to sustained academic pressure, competitive environments, and identity-related challenges inherent to medical training. Identifying protective psychological constructs that may mitigate these risks is therefore of considerable clinical and educational importance. Meaning in life has emerged as a key resilience factor and has been associated with improved well-being and reduced suicidality [1-3].

However, most existing studies have adopted variable-centered approaches, examining the presence of meaning and the search for meaning as independent predictors. Such approaches assume population homogeneity and may overlook meaningful within-person configurations. Person-centered approaches, such as latent profile analysis, allow for the identification of naturally occurring subgroups based on how individuals simultaneously experience meaning presence and search.

The present study aimed to identify distinct meaning-in-life profiles among medical students and to examine their associations with depression, anxiety, burnout, and suicidal behavior. It was hypothesized that profiles characterized by high meaning presence would demonstrate more favorable mental health outcomes, whereas profiles characterized by low meaning presence—particularly when combined with high meaning search—would exhibit elevated psychological distress and suicidal vulnerability.

Methods

This cross-sectional study included 568 medical students (mean age = 21.9 years, SD = 1.9; 59.5% female) recruited from multiple universities across Turkey. Participants were eligible if they were currently enrolled in medical school and aged 18 years or older. Data were collected anonymously through an online survey platform following institutional ethical approval and informed consent procedures.

Meaning in life was assessed using the Meaning in Life Questionnaire, which evaluates two dimensions: presence of meaning and search for meaning [1]. Depression and anxiety symptoms were measured using the Hospital Anxiety and Depression Scale. Burnout was assessed with the Maslach Burnout Inventory-Student Survey, including emotional exhaustion, cynicism, and academic efficacy dimensions. Suicidal behavior was evaluated using the Suicidal Behaviors Questionnaire, assessing lifetime ideation and attempts, recent ideation, and perceived future risk.

Latent Profile Analysis (LPA) was conducted using meaning presence and search scores as indicator variables. Model selection was based on multiple statistical criteria, including the Akaike Information Criterion, Bayesian Information Criterion, adjusted Bayesian Information

Criterion, entropy values, and likelihood-ratio tests [4]. Solutions were evaluated in terms of statistical fit, parsimony, interpretability, and class size adequacy.

Differences across latent profiles in depression, anxiety, burnout, and suicidal behavior were examined using the Bolck-Croon-Hagenaars method, which accounts for classification uncertainty and preserves the integrity of latent class formation. Statistical significance was set at $p < .05$.

Results

A four-profile solution demonstrated the best fit to the data based on statistical and theoretical considerations. The identified profiles were labeled as Low Meaning-Low Search (4%), High Meaning-High Search (61%), High

Meaning-Low Search (14%), and Low Meaning-High Search (22%). Entropy values indicated acceptable classification accuracy.

Significant differences were observed across all mental health outcomes ($p < .001$). The High Meaning-Low Search profile consistently exhibited the most adaptive psychological pattern, with the lowest levels of depression, anxiety, emotional exhaustion, cynicism, and suicidal behavior, and the highest academic efficacy scores. This profile reflects a stable and internalized sense of meaning.

In contrast, the Low Meaning-High Search profile demonstrated the highest levels of psychological distress and suicidal behavior. Individuals in this group reported significantly elevated depression, anxiety, burnout, and suicidal behavior compared to all other profiles. These findings suggest that high levels of meaning search in the absence of established meaning presence may reflect existential instability and increased vulnerability.

The Low Meaning-Low Search profile also exhibited elevated psychological distress, although generally lower than the Low Meaning-High Search group. This pattern may indicate disengagement rather than active distress. Across all comparisons, meaning presence emerged as the primary differentiating factor associated with better mental health outcomes.

Importantly, high levels of meaning search were not associated with adverse outcomes when meaning presence was also high, indicating that meaning search may function adaptively only when supported by an established sense of meaning.

Discussion

The present findings highlight the importance of adopting a person-centered approach in understanding the role of meaning in life among medical students. Consistent with prior literature, meaning presence emerged as a central protective factor associated with lower levels of depression, anxiety, burnout, and suicidal behavior [3,5]. Students characterized by high meaning presence and low search exhibited the most favorable psychological outcomes, suggesting that an internalized and stable sense of purpose serves as a key resilience mechanism in demanding academic environments.

Conversely, the combination of low meaning presence and high meaning search was associated with the highest levels of psychological distress and suicidal behavior. These results extend previous findings by suggesting that meaning search, often conceptualized as adaptive, may instead indicate vulnerability when it occurs in the absence of established meaning. This profile may reflect unresolved existential concerns, identity instability, and psychological disequilibrium. This finding contributes to the literature by demonstrating that meaning search is not inherently adaptive, but rather context-dependent and may become maladaptive depending on the presence of an established sense of meaning.

The identification of a high-risk profile characterized by low meaning presence and high search has important clinical implications. From a clinical perspective, screening for meaning in life profiles may provide a practical and scalable method for identifying vulnerable medical students. Interventions that focus on strengthening meaning presence—such as reflective practices, purpose-oriented counseling, and identity-focused educational programs—may be more effective than approaches that emphasize meaning search alone.

These findings may also have implications for treatment planning, as meaning in life could be considered a complementary target alongside pharmacological and psychotherapeutic interventions in improving mental health outcomes. Incorporating existential and meaning- centered components into mental health care may enhance treatment effectiveness, particularly in high-risk student populations.

The study has several limitations. The cross-sectional design limits causal inference, and longitudinal studies are needed to examine transitions between meaning profiles over time. The reliance on self-report measures may introduce bias. Additionally, the use of a convenience sampling method may limit the representativeness of the sample.

Conclusion

Meaning in life operates through distinct configurations that differentially relate to mental health outcomes. Meaning presence appears to be the primary protective factor, whereas high meaning search without established

meaning presence represents a potential marker of vulnerability. These findings underscore the importance of person-centered assessment approaches and highlight meaning presence as a key target for mental health promotion and suicide prevention among medical students.

Keywords: Meaning in Life; Depression; Anxiety; Burnout; Suicide; Medical Students

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0375 - The Role of Emotion Regulation and Sleep Quality in the Relationship Between Problematic Digital Media Use and Functioning in Adolescents With ADHD: A Cross- Sectional Study

Nadide Fettahlıoğlu

Abstract

Background: Attention-Deficit/Hyperactivity Disorder (ADHD) is a prevalent neurodevelopmental disorder characterized by functional impairment. In recent years, the relationship between problematic digital media use and ADHD has attracted increasing attention; however, studies examining its association with functioning and psychological processes remain limited.

Objective: This study aimed to investigate problematic patterns of internet, gaming, social media, and smartphone use among adolescents with ADHD, and to examine their associations with functioning, ADHD symptom severity, emotion regulation difficulties, irritability, and sleep quality.

Methods: A total of 193 adolescents aged 12-18 years with a DSM-5-based clinical diagnosis of ADHD were included. Parents completed the SNAP-IV, Affective Reactivity Index-Parent Form, and Weiss Functional Impairment Rating Scale-Parent Form (WFIRS- P). Adolescents completed the Internet Gaming Disorder Scale-Short Form, Bergen Social Media Addiction Scale, Smartphone Addiction Scale-Short Form, Young Internet Addiction Test-Short Form, Pittsburgh Sleep Quality Index (PSQI), Difficulties in Emotion Regulation Scale (DERS), and Affective Reactivity Index-Child Form.

Results: SNAP inattention scores were positively correlated with internet addiction ($r=0.30$, $p<0.001$), gaming addiction ($r=0.25$, $p=0.001$), smartphone addiction ($r=0.15$, $p=0.03$), and social media addiction ($r=0.24$, $p=0.001$). WFIRS total scores were associated with internet addiction ($r=0.33$, $p<0.001$), gaming addiction ($r=0.26$, $p<0.001$), smartphone addiction ($r=0.18$, $p=0.01$), and social media addiction ($r=0.21$, $p=0.004$). DERS total scores showed moderate positive correlations with internet addiction ($r=0.52$, $p<0.001$) and gaming addiction ($r=0.42$, $p<0.001$). Smartphone usage duration showed trend-level but nonsignificant associations with family ($r=-0.14$, $p=0.06$) and academic functioning ($r=-0.13$, $p=0.08$). Sleep quality and irritability levels were also clinically noteworthy.

Conclusions: Problematic digital media use in adolescents with ADHD is associated with functional impairment, ADHD symptom severity, and emotion regulation difficulties. These findings highlight the importance of systematically assessing digital media use patterns in clinical evaluation and intervention.

Introduction

Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders in childhood and adolescence, characterized by symptoms of inattention, hyperactivity, and impulsivity (1). ADHD extends beyond core symptoms, leading to significant impairments in academic performance, social relationships, family functioning, and daily life skills (2). Particularly during adolescence, increasing autonomy and widespread access to digital technologies have made digital media use a clinically relevant domain in individuals with ADHD (3).

With the rapid increase in internet, social media, online gaming, and smartphone use, these behaviors have been shown to evolve into problematic patterns characterized by loss of control, psychological distress, and functional impairment in some individuals (4). Contemporary approaches conceptualize this phenomenon under the broader framework of "problematic digital media use," encompassing multiple digital behaviors rather than a single construct such as "internet addiction" (5).

The association between ADHD and problematic digital media use has gained increasing research interest. Evidence suggests that children and adolescents with ADHD are more likely to exhibit problematic digital media use, particularly due to impulsivity, attentional difficulties, and executive dysfunction (6,7). Digital environments, offering rapid rewards and high stimulation, may be especially appealing to this population (8).

Importantly, the clinical significance of problematic digital media use extends beyond usage frequency. It has been associated with impairments in academic functioning, social relationships, family interactions, and quality of life (9). Therefore, functioning constitutes a key dimension in evaluating problematic digital media use.

Recent literature also highlights the role of emotion regulation difficulties, irritability, and sleep disturbances in this relationship. Digital media use may serve as a short-term coping strategy for negative emotional states, while potentially reinforcing maladaptive usage patterns over time (10). Thus, problematic digital media use should be conceptualized as a multidimensional construct involving behavioral, emotional, and biological processes.

However, several gaps remain in the literature. Most studies focus on single types of digital behavior and fail to adopt a comprehensive approach. Additionally, the simultaneous examination of functioning and mediating mechanisms remains limited, particularly in clinical samples of adolescents with ADHD.

Aim

This study aimed to examine digital media use behaviors in adolescents with ADHD within a comprehensive framework. Specifically, it sought to evaluate the frequency and severity of internet, gaming, social media, and smartphone use; to assess functioning across multiple domains including academic performance, family relationships, social interactions, self- concept, life skills, and risk-taking behaviors; to analyze the relationships between digital media use and functioning; to investigate the associations between ADHD symptom severity,

digital media use, and functioning; to test the mediating role of digital media use in the relationship between ADHD symptom severity and functioning; and to evaluate the contributions of emotion regulation difficulties, irritability, and sleep quality to these relationships.

Methods

This cross-sectional, single-center, clinical study was conducted at the Child and Adolescent Psychiatry Outpatient Clinic of Goztepe Prof. Dr. Suleyman Yal9in City Hospital following approval from the istanbul Medeniyet University Non-Interventional Research Ethics Committee.

Adolescents aged 12-18 years with a DSM-5-based clinical diagnosis of ADHD were included. Exclusion criteria were suspected autism spectrum disorder or intellectual disability, lack of personal smartphone ownership, non-attendance in formal education, illiteracy, sensory impairments, chronic medical conditions, and incomplete questionnaires. The final sample consisted of 193 participants. Written informed consent was obtained from participants and their parents.

Parents completed the SNAP-IV, Affective Reactivity Index-Parent Form, and Weiss Functional Impairment Rating Scale-Parent Form. Adolescents completed the Internet Gaming Disorder Scale-Short Form, Bergen Social Media Addiction Scale, Smartphone Addiction Scale-Short Form, Young Internet Addiction Test-Short Form, Pittsburgh Sleep Quality Index, Difficulties in Emotion Regulation Scale, and Affective Reactivity Index- Child Form.

Results

A total of 193 adolescents with ADHD were included. The mean age was 14.5±1.7 years, and the majority were male. Of the participants, 62.2% were classified as combined type and 37.3% as predominantly inattentive type.

Mean scores were as follows: SNAP-IV total 1.14±0.54, WFIRS total 0.76±0.44, and DERS total 87.64±21.32. ARI scores were 4.01±2.24 (child) and 4.19±2.25 (parent), while PSQI mean score was 7.41±2.63, indicating generally poor sleep quality.

Correlation analyses revealed that SNAP inattention scores were positively associated with internet addiction ($r=0.30$, $p<0.001$), gaming addiction ($r=0.25$, $p=0.001$), smartphone addiction ($r=0.15$, $p=0.03$), and social media addiction ($r=0.24$, $p=0.001$). SNAP hyperactivity/impulsivity was also positively correlated with internet addiction ($r=0.25$, $p<0.001$), smartphone addiction ($r=0.18$, $p=0.01$), and social media addiction ($r=0.20$, $p=0.006$), but not with gaming addiction ($r=0.12$, $p=0.09$).

WFIRS total scores were positively correlated with internet addiction (r=0.33, p<0.001), gaming addiction (r=0.26, p<0.001), smartphone addiction (r=0.18, p=0.01), and social media addiction (r=0.21, p=0.004). Among subdomains, academic functioning showed the strongest associations.

DERS total scores demonstrated moderate positive correlations with internet addiction (r=0.52, p<0.001) and gaming addiction (r=0.42, p<0.001). Most DERS subdimensions were significantly associated with these variables, except for awareness.

Smartphone usage duration showed weak and non-significant correlations with functioning and ADHD symptoms. No significant associations were found between age, age of smartphone acquisition, or usage duration and digital addiction scores.

Discussion

The findings indicate that problematic digital media use in adolescents with ADHD is significantly associated with functional impairment, symptom severity, and emotion regulation difficulties.

Inattention appears to be more strongly related to problematic digital media use than hyperactivity/impulsivity, suggesting that attentional deficits may increase vulnerability to highly stimulating digital environments.

The association between digital media use and functional impairment, particularly in academic and social domains, highlights the clinical relevance of these behaviors.

Emotion regulation difficulties showed moderate associations with internet and gaming addiction, suggesting that digital media use may serve as a maladaptive coping mechanism.

The lack of significant associations with smartphone usage duration suggests that qualitative aspects of use may be more clinically relevant than duration alone.

Conclusion

Problematic digital media use in adolescents with ADHD is associated with functional impairment, ADHD symptom severity, and emotion regulation difficulties.

These findings underscore the importance of systematically assessing digital media use patterns in clinical practice. Addressing digital behaviors alongside symptoms and psychosocial functioning may contribute to more comprehensive and effective interventions.

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0382 - Psychiatrists' Preferences for Long-Acting Injectable Antipsychotics and Their Attitudes Toward New-Generation Psychotropic Medications: How Open Are Psychiatrists to Innovation?

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Objective

Antipsychotic treatments are crucial for managing psychotic symptoms and reducing relapse risk; however, poor treatment adherence to oral antipsychotics remains a major barrier to relapse prevention. Developed in the 1960s to address this problem, long-acting injectable antipsychotics (LAIAs) support treatment continuity through controlled-release mechanisms and less frequent administration. Despite these advantages, LAIA prescribing rates for schizophrenia patients across Europe generally remain below 30% and vary across countries. This limited use may reflect negative attitudes toward this treatment method held by both psychiatrists and patients[1].

Several studies have examined factors contributing to the low prescription rates of LAI medications[1-5]. Patel et al. reported that 30.1% of psychiatrists experienced stress when offering LAIAs, mainly because of concerns about side effects and complications at treatment initiation[1]. Di Giorgio et al. found that, although 98% of psychiatrists acknowledged the long- term benefits of LAIAs, diagnostic uncertainty in first-episode psychosis, limited treatment options, restricted dose flexibility, and the risk of prolonged side effects complicated their use in routine clinical practice[2].

In recent years, the range of psychotropic medications has expanded significantly; in particular, new molecules such as cariprazine, brexpiprazole, lurasidone, and esketamine, as well as new treatment approaches like the aripiprazole dual-dose initiation protocol and the 6- month formulation of paliperidone, have begun to be incorporated into psychiatric practice. Although these innovations appear promising in terms of efficacy and safety, their integration into clinical practice may be shaped by psychiatrists' knowledge, attitudes, prescribing behaviors, and non-clinical factors.

Due to the lack of literature comprehensively addressing psychiatrists' awareness and attitudes toward new-generation psychotropic (NGP) medications and new treatment approaches in Turkey, this study was designed to investigate physicians' clinical decisionmaking processes and the dynamics of their adaptation to pharmacological innovations. In this context, by investigating the relationship between physicians' individual levels of innovativeness and their medication preferences, the study aims to identify psychiatrists' educational needs and develop strategies to support the integration of new treatment options into clinical practice.

Methods

This cross-sectional, descriptive study was initiated after approval was obtained from the Pamukkale University Ethics Committee (No. E-61016787-020-767165) and was conducted between October 2025 and January 2026. The study sample consisted of psychiatrists and psychiatry residents who voluntarily participated in an online survey. Data were collected using Google Forms. Participants were contacted by phone, through social media accounts, and via social media groups of which they were members. The data collection instruments included the "Survey on the Use of Long-Acting Injectable Antipsychotics and Attitudes Toward New

Psychotropic Medications Among Psychiatrists," developed by the researchers, and the "Individual Innovativeness Scale (IS)".

Assessment Instruments

The "Survey on the Use of Long-Acting Injectable Antipsychotics and Attitudes Toward New Psychotropic Medications Among Psychiatrists " consists of 36 questions designed to assess participants' sociodemographic characteristics, clinical approaches to the use of long- acting injectable antipsychotics, factors influencing drug

selection and administration preferences, and their knowledge and attitude levels toward NGPs. The Individual Innovativeness Scale was developed by Hurt et al. (1977) and adapted into Turkish by Kilmer and Odaba§i (2010). It is a 5-point Likert scale consisting of 20 items. The scale has four subscales: "resistance to change," "opinion leadership," "openness to experience," and "risk- taking." Scores of 81 or higher are interpreted as "innovators"; scores between 69 and 80 as "early adopters"; scores between 57 and 68 as "interrogators"; scores between 46 and 56 as "skeptics"; and scores of 45 or lower as "traditionalists." Individuals scoring above 68 are considered highly innovative, while those scoring below 64 are considered to have a low level of innovation.

Statistical Analysis

The data were analyzed using SPSS v25.0. Continuous variables are presented as mean ± standard deviation, while categorical variables are presented as counts and percentages. Descriptive statistical analysis was performed.

Results

A total of 205 individuals were included in the study; 65(31.7%) were men and 140(68.3%) were women, and all participants completed the questionnaires. The participants' mean age was 36.54±8.93 years. Among the participating psychiatrists, 76(37.1%) were residents/research fellows, 91(44.4%) were specialists, and 38(18.5%) were academics. Of the participants, 44(21.5%) worked at a state hospital, 26(12.7%) at a city hospital, 68(33.2%) at a university hospital, 40(19.5%) at a training and research hospital, 11(5.4%) at a private hospital, and 16(7.8%) worked in private practices.

Participants' LAIA usage habits and clinical application preferences are presented in Table 1, while clinical approaches, attitudes, and practice characteristics regarding NGP are presented in Table 2. The participants' total IS score mean was determined to be 65.26±10.56. When the subscales were examined, the highest mean score was found in the Resistance to Change subscale (20.12±5.60). According to the categorical classification based on IS scores, the vast majority of participants were in the "Interrogators" group (n = 82, 40.0%). The mean subscale scores and subgroup distributions for the Individual Innovativeness Scale are presented in Table 3. When examining the characteristics participants prioritized in their selection of LAIA, the most frequently cited factor was that the medication had been used in the patient's history and was beneficial (92.2%). This was followed by the following factors: The drug's efficacy(90.2%), the drug's side effect profile(86.3%), less frequent dosing(68.8%), patient preference (27.8%), price advantage(19%), and the option to administer to multiple injection sites(10.7%).

When examining the practice of switching from the monthly paliperidone LAI formulation to 3-month or 6-month formulations, 67.2% of participants reported that they had considered or implemented this switch. In contrast, the reasons cited by those who did not implement or consider the switch were, in order: lack of sufficient experience (19.6%), the need for close monitoring of the patient (12.7%), indecision (10.8%), concern about side effects (5.4%), and patient refusal (2.5%).

While 54.1% of participants preferred the aripiprazole LAIA double-dose initiation protocol, 23.6% of these individuals preferred it only for inpatients. 40.9% stated they did not have sufficient experience, 17.7% cited concerns about the side effects of administering two injections on the same day, and 1.5% indicated they did not choose it because patients refused. Additionally, 51% of participants agreed that the aripiprazole LAIA dual-dose initiation regimen is easy to administer and has a safety/tolerability profile similar to that of the single- injection initiation regimen, while 4.4% disagreed and 44.6% were undecided.

When participants were asked to rate their own level of knowledge about these NGPs on a scale of 1 to 5 (1 = I know nothing and 5 = I know very well), the scores were: Cariprazine 2.57±1.11; brexpiprazole 2.92±1.07; esketamine 2.51±1.16; aripiprazole LAIA dual-dose initiation protocol 3.43±1.26; lurasidone 3.53±1.05; and paliperidone LAIA 6-month formulation 3.20±1.24.

While 25.4% of participants found the guidance provided by clinical guidelines (APA, NICE, etc.) regarding the use of new medications to be sufficient, 22.4% did not find it sufficient, and 52.2% indicated they were undecided.

When considering prescribing a new medication, the factor that most influenced participants was a lower side effect profile (88.3%); other influencing factors were, in order: higher efficacy compared to current treatments (85.4%), contribution to clinical experience (66.3%), and professional curiosity and the desire to keep up with current practices (56.1%).

When examining the barriers to prescribing NGP medications, the primary obstacle at both the local and global levels was that the medication in question was not covered by insurance or approved for the indicated use (87.7%; 82.4%).

While psychiatrists' greatest need regarding new medications was found to be "more training, seminars, and literature" at 62.9%, an analysis of how they learn about new medications revealed that 83.9% learn about them through word of mouth in professional settings, and 79% learn about them from pharmaceutical companies.

Discussion

It has been observed that the vast majority of psychiatrists feel inadequate regarding the use of LAIA, particularly avoid prescribing it for patients over 65 years of age, and that extrapyramidal side effects are the most common reason for their hesitation. In clinical practice, it has been determined that the efficacy and side effect profiles of LAIA are the most important factors influencing prescribing decisions, and that the most common reason for not prescribing it is a lack of experience. The lack of guidelines regarding attitudes toward NGP, expectations of higher efficacy and fewer side effects compared to current medications, and the absence of insurance or indication coverage—the most common barrier to prescribing—have been identified as the most significant factors. Additionally, while information about new medications is most frequently obtained in professional settings and through pharmaceutical companies, it is noteworthy that the highest demand for these medications relates to the need for education, seminars, and literature. Furthermore, the fact that 40% of psychiatrists are "interrogators" is consistent with the study's findings. The expectation that a newly launched drug will be superior to existing ones is the most significant factor influencing the use of the new drug. Conversely, it has been suggested that the low rates of potential contribution to clinical experience and professional curiosity/desire to follow current practices may be associated with resistance to change.

In conclusion, given the resistance to change and interrogative attitudes toward LAIA and NGP, it is believed that literature and educational support are important for positive changes in attitudes and openness to innovation.

Keywords: Antipsychotic agents, JnnovatJveness, practice patterns, psychJatrJst

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Table 1: Participants' LAIA Usage Habits and Clinical Application Preferences

Variables	Options	n	%
Preference for LAIA in clinical practice	Yes	202	98.5
	No	3	1.5
The option that best suits the perspective on the use of LAIA	**Use in patients with compliance issues**	**131**	**63.9**
	Use as early as possible	55	26.8
	Use in severe symptoms/resistant cases	18	8.8
	Those hesitant due to side effect risks	1	0.5
Preference rates for LAIA in appropriate indications	<%10	15	7.3
	%10-25	50	24.4
	%25-50	**64**	**31.2**
	%50-75	45	22.0
	>%75	31	15.1
Feeling competent in using LAIA	Yes	134	65.4
	Partially	47	22.9
	No	24	11.7
Reasons for feeling inadequate*	Lack of sufficient knowledge about the drug	12	12
	Insufficient review of the literature	21	28
	Insufficient experience with its use	**57**	**76**
	Inadequate management of side effects	23	30.7
	First attack - acute phase	62	30.2
At what stage of the disease was LAIA preferred*	Maintenance treatment	192	93.7
	If there is treatment resistance	126	61.5
Reasons for avoiding LAIA application*	If the patient tolerates oral antipsychotics well	126	61.5
	Patient's refusal of LAIA	148	72.2
	Patient characteristics (age, comorbidities, etc.)	**157**	**76.6**
	Preference not to use LAIA	51	24.9
	High cost	54	26.3
Patient characteristics for which LAIA application was avoided*	Being under 18 years of age	104	50.7
	Being over 65 years of age	**126**	**61.5**
	LAIA incompatibility of the patient and/or relative	**131**	**63.9**
	Accompanying metabolic disease	66	32.2
	Accompanying neurological disease	93	45.4
	Additional diseases other than metabolic and neurological diseases	51	24.9
	Unclear diagnosis	122	59.5
Side effects feared when applying LAIA*	Problems at the injection site	15	7.3
	Allergic reactions	37	18.0
	Metabolic side effects	42	20.5
	Extrapyramidal side effects	**137**	**66.8**
	Cardiac side effects (QTC prolongation, etc.)	115	56.1
	Neuroleptic malignant syndrome and similar serious side effects	131	63.9
	Unpredictable side effects	89	43.4
Key features prioritized in the LAIA selection*	**The patient has used it in the past and benefited from it**	**189**	**92.2**
	The drug's efficacy	**185**	**90.2**
	The drug's side effect profile	**177**	**86.3**
	Less frequent dosing	141	68.8
	Patient preference	57	27.8
	Price advantage	39	19
	The option to administer it in multiple injection sites	22	10.7

* Multiple selections have been made. (LAIA: Long-acting injectable antipsychotic)

Table 2: Participants' Clinical Approach, Attitudes, and Practice Characteristics Regarding New Generation Psychotropics

Variables	Options	n	%
Consideration and implementation of switching from monthly paliperidone LAI to 3-monthly and 6-monthly formulations*	Yes	137	67.2
	No, because I have no experience	**40**	**19.6**
	No, because I want the patient to be closely monitored	26	12.7
	Undecided	22	10.8
	No, because I am concerned about the side effects	11	5.4
	No, because patients do not want it	5	2.5
The preference for and reasons for not preferring the double injection initiation protocol of monthly aripiprazole LAIA*	**I do not administer it because I do not have sufficient experience.**	**83**	**40.9**
	I administer it (to both inpatients and outpatients).	62	30.5
	I administer it (only to inpatients).	48	23.6
	I do not administer it because I am concerned about the side effects of administering two injections on the same day.	36	17.7
	I do not administer it because patients do not want it.	3	1.5
Agreement with the statement: "The double-dose initiation regimen of aripiprazole LAI is easy and has a safety/tolerability profile similar to the single-injection initiation regimen."	**Yes**	**104**	**51**
	Undecided	91	44.6
	No	9	4.4
The rates of preference for new-Generation psychotropics and protocols as the first choice in appropriate patients*	Cariprazine	89	44.9
	Brexpiprazole	101	51
	Esketamine	22	11.1
	Lurasidone	**153**	**77.3**
	Double-dose initiation of Aripiprazole	115	58.1
	Paliperidone 6-month formulation	79	39.9
The rate at which new-generation psychotropic drugs and protocols are definitely not preferred as the first choice in suitable patients*	Cariprazine	34	18.3
	Brexpiprazole	32	17.2
	Esketamine	**156**	**83.9**
	Lurasidone	14	7.5
	Double-dose initiation of Aripiprazole	39	21
	Paliperidone 6-month formulation	82	44.1
Agreement with the statement "Clinical guidelines (APA, NICE, etc.) are sufficient for guiding the use of new drugs."	**I am undecided**	**107**	**52.2**
	I agree	52	25.4
	I disagree	46	22.4
Factors influencing the decision to prescribe a newly introduced drug*	**Lower side effect profile**	**181**	**88.3**
	Higher efficacy compared to current treatments	**175**	**85.4**
	Potential contribution to clinical experience	136	66.3
	Professional curiosity and desire to keep up with current practices	115	56.1

Variables	Options	n	%
Situations that may prevent prescribing drugs that are new to Turkey but have been used elsewhere in the world*	**Not covered by insurance or indication**	**179**	**87.7**
	Inaccessibility	151	74
	Lack of sufficient personal experience	119	58.3
	Concerns about side effects and possible complications	105	51.5
	Lack of clinical guidelines or evidence	101	49.5
	Unknown long-term effects	91	44.6
	Use of drugs for habit and comfort	83	40.7
	Insufficient personal theoretical knowledge	81	39.7
Circumstances that may prevent prescribing newly introduced drugs worldwide and in Turkey*	**Not covered by insurance or indication**	**168**	**82.4**
	Lack of clinical guidelines or evidence	148	72.5
	Inaccessibility	142	69.6
	Lack of sufficient personal experience	138	67.6
	Unknown long-term effects	130	63.7
	Concerns about side effects and possible complications	130	63.7
	Insufficient personal theoretical knowledge	105	51.5
	Use of drugs for habit and comfort	82	40.2
Psychiatrists' needs regarding new drugs	**Need for more training, seminars, and literature on new drugs**	**129**	**62.9**
	Increased clinical experience of colleagues	64	31.2
	Regular and frequent company visits	12	5.9
How psychiatrists learn about new drugs*	**Perceptions in professional settings**	**172**	**83.9**
	Through drug companies	**162**	**79**
	Literature	**152**	**74.1**
	Through professional associations	105	51.2
	Social media	31	15.1
	Not being informed	1	0.5

* Multiple selections have been made. (LAIA: Long-acting injectable antipsychotic, APA: American Psychiatric Association, NICE: National Institute for Health and Care Excellence)

Table 3: Individual Innovativeness Scale Subgroup Distributions

Individual Innovativeness Scale Subgroups	**Mean Scores±SD**	
Opinion Leadership	17.77±3.76	
Openness to Experience	18.77±3.44	
Risk Taking	6.84±1.84	
Resistance to Change	20.12±5.60	
Scale Total Score	65.26±10.56	
Individual Innovativeness Scale Categories	**n**	**%**
Traditionalists	4	2.0
Skeptics	40	19.5
Interrogators	82	40.0
Early Adopters	59	28.8
Innovators	20	9.8

SD: Standard Deviation

0388 - Anticipated Stigma in Adolescents with Chronic Illness: Validation of the Turkish CIASS-A and Associations with Psychosocial Outcomes

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Objective

Chronic illnesses are known to affect not only physical health but also various aspects of psychosocial functioning, particularly during adolescence. This period is marked by increasing independence, identity development, and sensitivity to peer relationships, which may make adolescents more vulnerable to social stressors.

Stigma has been identified as an important factor influencing psychological well-being in individuals with chronic conditions and has been conceptualized as a social process involving labeling, stereotyping, and discrimination^]. Among its components, anticipated stigma refers to the expectation of being judged, rejected, or treated differently in the future. Even in the absence of direct negative experiences, such expectations may shape behavior, including avoidance of social situations or reluctance to seek help.

Most studies in this area have focused on specific illnesses, which limits the ability to compare findings across different conditions. The Chronic Illness Anticipated Stigma Scale (CIASS), developed by Earnshaw et al., offers a broader approach by assessing stigma across different social contexts [2]. However, there is still a lack of validated tools for assessing anticipated stigma among adolescents in Turkiye.

The aim of this study was to adapt the CIASS-Adolescent Form (CIASS-A) into Turkish, examine its validity and reliability, and explore its associations with psychological variables such as anxiety, depression, self-esteem, and quality of life.

Methods

This cross-sectional study was conducted in pediatric outpatient clinics of a university hospital. A total of 271 adolescents aged between 12 and 18 years participated. The clinical group consisted of 215 adolescents diagnosed with epilepsy or type 1 diabetes, while the control group included 56 adolescents with allergic rhinitis.

Ethical approval was obtained from the Kocaeli University Non-Interventional Clinical Research Ethics Committee (KU GOKAEK-2025/17/10; 19.08.2025).

The CIASS-A was translated using a forward-backward translation process, and content validity was evaluated with expert input using the Lawshe method. The scale is based on the original CIASS developed by Earnshaw et al [2].

Participants completed a sociodemographic form along with the Turkish CIASS-A, the Rosenberg Self-Esteem Scale, the Hospital Anxiety and Depression Scale (HADS), and the Pediatric Quality of Life Inventory (PedsQL).

Confirmatory factor analysis (CFA) was used to examine construct validity. Internal consistency was assessed using Cronbach's alpha, and test-retest reliability was evaluated over a three-week interval. Group differences, correlations, and regression analyses were performed using standard statistical methods.

Results

The CFA results supported the original three-factor structure of the scale, with acceptable fit indices (χ^2/df = 2.87, CFI = 0.95, RMSEA = 0.083) (Figure 1). Factor loadings ranged between 0.32 and 0.78. These findings further support the structural validity of the scale and indicate that the items adequately represent the underlying latent constructs. The consistency of factor loadings across subscales suggests that the CIASS-A captures anticipated stigma in a stable and coherent manner across different social contexts.

The overall internal consistency of the scale was good (Cronbach's alpha = 0.846), with subscale values between 0.74 and 0.80. Test-retest reliability was also high ($r = 0.824$, $p < 0.01$) (Table 2). Item-total correlation coefficients ranged between 0.325 and 0.634, indicating acceptable to good item discrimination.

Adolescents in the case group reported higher levels of anticipated stigma compared to the control group ($p < 0.001$). This difference was particularly noticeable in school and healthcare-related contexts, with a moderate-to-strong effect size (Table 2).

Higher stigma scores were associated with higher levels of anxiety and depression, as well as lower self-esteem and quality of life ($p < 0.01$). In regression analysis, quality of life appeared to be the strongest predictor of anticipated stigma (Table 3). This finding highlights the central role of perceived well-being in shaping adolescents' expectations of stigma and suggests that subjective health perception may be more influential than individual symptom levels.

Discussion

The results suggest that the Turkish version of the CIASS-A is a valid and reliable tool for assessing anticipated stigma in adolescents with chronic illness. The three-factor structure observed in this study is consistent with the original scale and supports the idea that stigma operates across multiple social domains. In addition, the confirmation of the original factor structure strengthens the cross-cultural applicability of the CIASS-A and supports its use in diverse clinical populations.

To our knowledge, this is the first study to examine anticipated stigma using a transdiagnostic approach in Turkish adolescents with chronic illness. This may be useful in allowing comparisons across different medical conditions.

The findings also indicate that anticipated stigma is linked to various aspects of psychological well-being. Adolescents who reported higher stigma also tended to report more emotional difficulties and lower quality of life [3]. These findings are in line with previous studies suggesting that stigma can act as an ongoing stressor [4]. This also suggests that interventions targeting stigma may have indirect benefits on emotional well-being and functional outcomes [5].

An interesting finding was that quality of life emerged as the strongest predictor of stigma. This may indicate that how adolescents perceive their overall well-being plays an important role in how they anticipate social evaluation.

Higher stigma levels in school and healthcare settings point to these environments as important areas for intervention. The scale appears to be brief and practical, and could potentially be used in routine clinical settings to identify adolescents who may need additional psychosocial support.

Another important implication of this study is that anticipated stigma may remain unrecognized in routine clinical settings, as it does not always manifest through overt behaviors. Therefore, systematic assessment using brief and structured tools such as the CIASS-A may help clinicians identify hidden psychosocial difficulties and improve individualized care planning.

In Turkiye, care for adolescents with chronic illness is often focused on medical management. Incorporating tools that assess stigma and psychosocial functioning may help provide more comprehensive care.

Keywords: Adolescent; chronic illness; anticipated stigma; validity; reliability; quality of life

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Tables and Figures

Table 1. Reliability Analysis of the Turkish CIASS-A

Scale	Alpha	n
Friends and Family	0,746	4
School peers and teachers	0,786	4
Healthcare Providers	0,797	4
Total Stigma	0,846	12

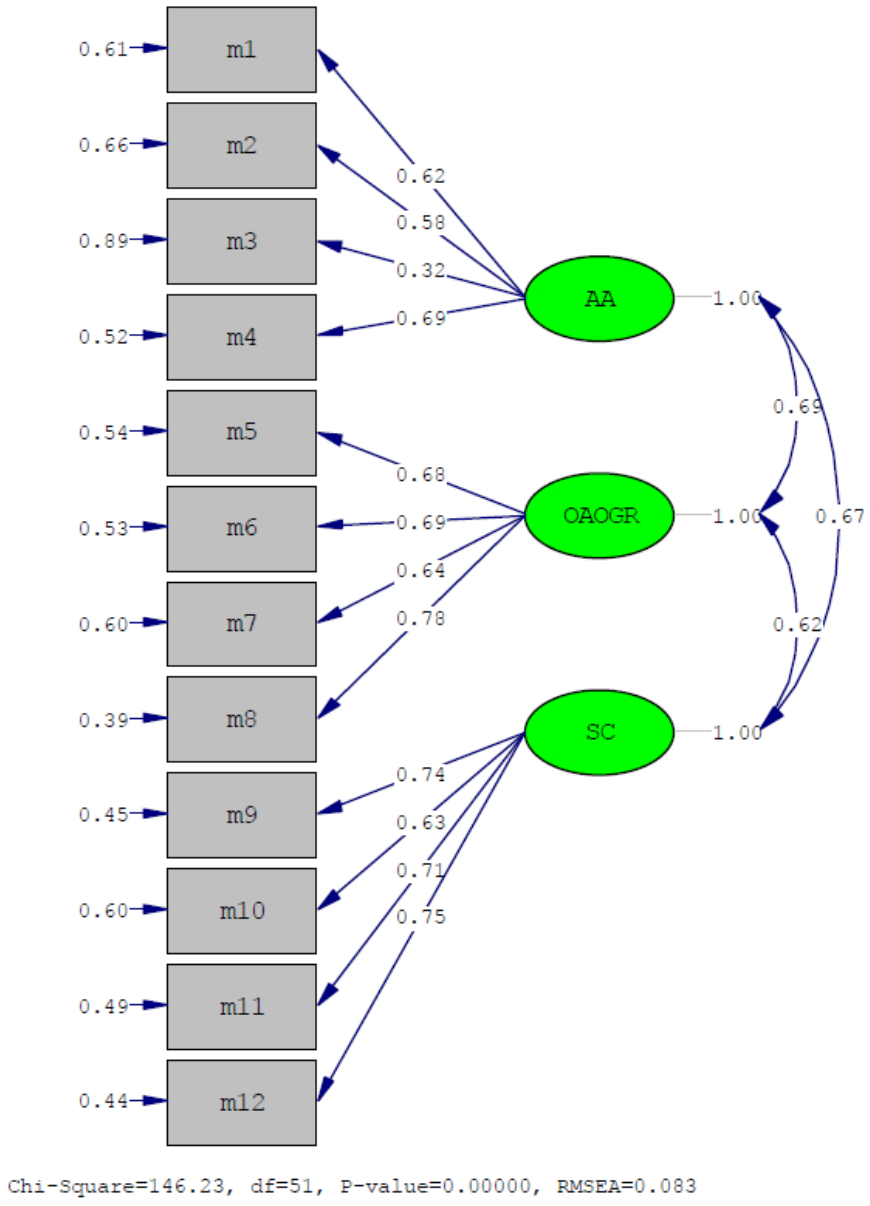
Table 2. Comparison of Anticipated Stigma Scores Between Groups

Groups	Control	Case	p
	Mean ± SD	Mean ± SD	
Friends and Family	1,28±0,48	1,43±0,54	0,059
School peers and teachers	1,36±0,64	1,72±0,76	**0,001**
Healthcare Providers	1,08±0,29	1,49±0,64	**0,001**
Total Stigma	1,24±0,41	1,55±0,52	**0,001**

Table 3. Regression Analysis

Predictor	B (control)	B (case)	p
Self-esteem	-		>0.05
Anxiety	-		>0.05
Depression	-		>0.05
Quality of Life	-0.154	-0.163	<0.01

Figure 1. Confirmatory Factor Analysis Model of the Turkish CIASS-A



0395 - Comparison of Clinical and Socio-Demographic Characteristics and Rorschach Test Findings of Offenders Diagnosed with Schizophrenia and Substance-Induced Psychosis in Turkey: A Retrospective Study

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Introduction

Psychotic disorders are severe mental health conditions characterized by impaired reality testing and disturbances in perception, thinking, and behavior. Among these disorders, schizophrenia has been associated with a higher rate of criminal behavior compared with the general population and other psychiatric conditions [1]. Violence and criminal behavior among individuals with psychiatric disorders represent an important public health concern. Previous studies have identified several factors associated with violent behavior in psychosis, including positive psychotic symptoms, hostility, substance use, childhood behavioral problems, poor impulse control, and treatment noncompliance [1-3]

Substance use is one of the most significant factors contributing to psychosis and violent behavior. Psychoactive substances may induce psychotic symptoms by altering dopaminergic and glutamatergic neurotransmission [4]. Methamphetamine increases dopamine release and blocks dopamine transporters, producing dopaminergic hyperactivity that may trigger hallucinations and delusions similar to those observed in schizophrenia [5]. Cannabis, on the other hand, affects glutamatergic pathways and NMDA receptor functioning, contributing to cognitive impairments and negative symptoms [6,7]. Dysregulation of these neurotransmitter systems is thought to play a critical role in the emergence of psychotic symptoms.

According to DSM-5-TR, substance-induced psychotic disorder is diagnosed when psychotic symptoms such as hallucinations or delusions occur in temporal association with substance intoxication or withdrawal [8]. Although symptoms may resolve after detoxification, longitudinal studies have shown that a considerable proportion of patients with substance- induced psychosis later develop chronic psychotic disorders such as schizophrenia [9]. Meta- analytic evidence suggests that approximately one-quarter of individuals with substance- induced psychosis transition to schizophrenia spectrum disorders [10].

Despite different etiological mechanisms, the clinical presentations of schizophrenia and substance-induced psychosis may become similar over time, making differential diagnosis difficult [11,12]. This diagnostic challenge becomes particularly important in forensic psychiatry settings where clinicians evaluate criminal responsibility and risk of future violence. High-Security Forensic Psychiatry (HSFP) units frequently assess offenders whose psychiatric conditions may have contributed to criminal acts.

Psychological assessment tools may assist clinicians in understanding cognitive and emotional processes underlying criminal behavior. The Rorschach Test is a projective method that evaluates perceptual organization, emotional regulation, and thought processes by analyzing how individuals interpret ambiguous inkblot stimuli [13]. Such assessments may provide valuable insights into perceptual distortions, internal stress, and threat perception.

The present study aimed to compare offenders diagnosed with schizophrenia and those diagnosed with substance-induced psychosis in a HSFP unit in Turkey with respect to their Rorschach Test findings and clinical-sociodemographic characteristics. We hypothesized that substance use increases perceptual distortions and internal stress, which may contribute to violent behavior and criminal involvement.

Methods

This retrospective study was conducted in the HSFP unit of Bursa City Hospital in Turkey between June 2024 and February 2025. Ethical approval was obtained from the institutional ethics committee (29/05/2024 – No: 2024-9/1). Patient records between November 2019 and December 2024 were retrospectively reviewed.

Among 1,017 male patients referred by judicial authorities for forensic psychiatric evaluation, individuals diagnosed with schizophrenia or substance-induced psychosis were screened for eligibility. Inclusion criteria included a confirmed diagnosis according to DSM-5 criteria, documented Rorschach Test results, accessible court records, and verified substance use through toxicology tests in the substance-induced psychosis group. Patients with additional psychiatric or neurological diagnoses or incomplete records were excluded.

Sociodemographic and clinical information was collected using a structured data form including variables such as age, education level, employment status, living conditions, substance use history, and characteristics of committed offenses.

Rorschach Test protocols were evaluated using the Exner Comprehensive System by an experienced clinical psychologist [13]. Responses were scored according to location, determinants, content, and form quality. Particular attention was given to variables related to perceptual distortions, internal stress, and threat perception.

Statistical analyses were performed using SPSS version 22. Quantitative variables were expressed as medians and ranges, while categorical variables were presented as frequencies and percentages. Group comparisons were conducted using the Mann-Whitney U test for continuous variables and the chi-square test for categorical variables. Statistical significance was set at $p < 0.050$.

Results

The final study sample consisted of 70 male forensic psychiatric patients, including 47 patients with schizophrenia and 23 with substance-induced psychosis. The mean age of the schizophrenia group was slightly higher than that of the substance-induced psychosis group; however, this difference was not statistically significant (p=0.164).

Patients in the substance-induced psychosis group had a significantly higher number of previous forensic incidents and a greater total number of offenses compared with the schizophrenia group (p=0.003 and p=0.002, respectively). In contrast, the number of psychiatric hospitalizations was significantly lower in the substance-induced psychosis group (p=0.007). Sociodemographic and clinical characteristics of the groups are presented in Tables 1 and 2.

Regarding substance use patterns, cannabis (91.3%) and methamphetamine (78.3%) were the most commonly used substances among patients diagnosed with substance-induced psychosis. The distribution of substance types used within the substance-induced psychosis group is provided in Table 3.

Analysis of Rorschach Test findings revealed significant differences between the groups. The substance-induced psychosis group demonstrated significantly higher scores in Unusual Movement Quality (Unusual:MQ), Total Y, and the Hypervigilance Index (HVI) (p=0.008, p=0.025, and p=0.037, respectively).

Unusual:MQ reflects disturbances in perceptual organization and disorganized thought processes. Elevated scores indicate distorted perception and difficulties interpreting environmental stimuli accurately. Total Y represents internal stress and feelings of situational pressure, whereas HVI reflects heightened vigilance toward potential threats. These findings suggest that individuals with substance-induced psychosis may experience more intense perceptual distortions, increased internal stress, and heightened threat perception compared with individuals diagnosed with schizophrenia. The results of the Rorschach Test findings showing statistically significant differences between the groups are presented in Table 4.

Discussion

This study compared clinical, sociodemographic, and psychological characteristics of offenders diagnosed with schizophrenia and substance-induced psychosis in a high-security forensic psychiatry setting. Our findings suggest that substance-induced psychosis is associated with greater perceptual distortions, higher internal stress levels, and increased hypervigilance as measured by the Rorschach Test.

Psychotic disorders have long been associated with violent behavior, although the relationship is complex and influenced by multiple risk factors. Substance use is one of the most important factors that increases the risk of violence in individuals with psychotic disorders [2,14]. Previous studies have shown that the combination of schizophrenia and substance use substantially increases the likelihood of criminal behaviour compared with schizophrenia alone [14]. Consistent with these findings, patients with substance-induced psychosis in our study had a significantly higher number of forensic incidents than patients with schizophrenia. Psychoactive substances

may increase impulsivity, reduce behavioral inhibition, and exacerbate psychotic symptoms, thereby increasing the likelihood of aggressive or criminal behavior.

Interestingly, patients in the substance-induced psychosis group had fewer psychiatric hospitalizations. Several explanations may account for this observation. Individuals with substance use problems may be less likely to seek treatment or may have poorer adherence to psychiatric care. In addition, psychotic symptoms may be perceived as temporary consequences of substance use rather than manifestations of a chronic psychiatric disorder, leading to reduced hospitalization.

The Rorschach Test findings provide additional insight into psychological processes underlying these behaviors. Higher Unusual:MQ scores in the substance-induced psychosis group suggest increased perceptual distortions and cognitive disorganization. Elevated Total Y scores indicate higher internal stress and impaired emotional regulation. Similarly, increased HVI scores reflect heightened threat perception and defensive vigilance. These characteristics may contribute to impulsive or aggressive behavior. Distorted perceptions and exaggerated threat sensitivity may lead individuals to misinterpret environmental cues as threatening, potentially triggering defensive or aggressive responses. Substance use, particularly cannabis and methamphetamine, may intensify these psychological processes by altering dopaminergic and glutamatergic neurotransmission. Previous studies have demonstrated that substance use can exacerbate hallucinations, delusions, and emotional instability in individuals with psychosis [15-17]. For these reasons, the substance-induced psychosis group may have committed more offenses and may have been involved in a higher number of forensic cases. Our findings are remarkable and important in the sense that these clinical features, which we frequently see in patients with substance-induced psychosis in practice, are also confirmed by Rorschach Test results.

From a forensic perspective, these findings highlight the importance of considering substance use when assessing criminal responsibility and risk of recidivism in psychotic offenders. Effective treatment strategies should address both psychotic symptoms and substance dependence simultaneously. Integrated treatment approaches that combine addiction treatment with psychiatric care may reduce relapse rates and prevent future criminal behavior. Previous studies have shown that untreated substance use disorders significantly increase relapse and crime risk among individuals with psychosis [18].

Conclusion

Substance use represents a major public health concern and a significant risk factor for violent behavior in individuals with psychotic disorders. Our findings suggest that offenders with substance-induced psychosis demonstrate higher levels of perceptual distortion, internal stress, and threat sensitivity compared with offenders diagnosed with schizophrenia.

The Rorschach Test may provide valuable insights into cognitive and emotional processes underlying violent behavior in psychotic individuals. Integrating addiction treatment with psychotic disorder management may be crucial for reducing recidivism and improving clinical outcomes in forensic psychiatric populations.

Future prospective studies with larger samples are needed to further examine the relationship between substance use, perceptual distortions, and violent behavior in psychotic offenders.

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Table 1 Sociodemographic characteristics of the groups

	Schizophrenia	Substance-induced Psychosis	p	Total
	n(%)			n(%)
Marital status				
Single	30 (63.8)	11 (47.8)		41 (58.6)
Divorced/Widow	6 (12.8)	4 (17.4)	0.440	10 (14.3)
Married	11 (23.4)	8 (34.8)		19 (27.1)
Military service				
Hasn't done it yet	1 (2.1)	0 (0.0)		1 (1.4)
Non-psychiatry exempt	1 (2.1)	0 (0.0)	-	1 (1.4)
Exempt	16 (34.1)	2 (8.7)		18 (25.8)
Made	29 (61.7)	21 (91.3)		50 (71.4)

Education status				
Literate	4 (8.5)	0 (0.0)		4 (5.7)
Primary School	14 (29.8)	7 (30.4)	-	21 (30.0)
Middle School	11 (23.4)	9 (39.2)		20 (28.6)
High School	10 (21.3)	6 (26.1)		16 (22.8)
University	8 (17.0)	1 (4.3)		9 (12.9)
Employment status				
Not working	27 (57.5)	11 (47.8)	0.638	38 (54.3)
Works irregularly	8 (17.0)	6 (26.1)		14 (20.0)
Works regularly	12 (25.5)	6 (26.1)		18 (25.7)
Place of residence				
On the street	0 (0.0)	1 (4.3)		1 (1.4)
Alone	4 (8.5)	7 (30.5)		11 (15.7)
Family	40 (85.1)	14 (60.9)	-	54 (77.1)
At the nursing home	1 (2.1)	1 (4.3)		2 (2.9)
Other relatives	2 (4.3)	0 (0.0)		2 (2.9)
Type of judicial writing				
CPC 74 - TPC 32	42 (89.4)	21 (91.4)		63 (90.0)
TPC 57	5 (10.6)	1 (4.3)	-	6 (8.6)
Other	0 (0.0)	1 (4.3)		1 (1.4)
Decision				
Social healing	5 (10.6)	2 (8.7)		7 (10.0)
TPC 32/1	37 (78.8)	19 (82.7)		56 (80.0)
TPC 32/2	5 (10.6)	1 (4.3)	-	6 (8.6)
Has criminal capacity	0 (0.0)	1 (4.3)		1 (1.4)
Taylor				
Non-violence	7 (14.9)	5 (21.7)		12 (17.1)
Minimal violence	7 (14.9)	6 (26.2)		13 (18.6)
Moderate violence	20 (42.6)	5 (21.7)	-	25 (35.7)
Moderately serious violence	13 (27.6)	7 (30.4)		20 (28.6)
Serious violence	0 (0.0)	0 (0.0)		0 (0.0)

CPC: Criminal Procedure Code; TPC: Turkish Penal Code; $p<0.050$.

Table 2 Clinical characteristics of the groups

	Schizophrenia	Substance-induced Psychosis	p[a]	Total
	n(%)			n(%)
Hospitalization in the last 1 year				
No	41 (87.2)	21 (91.3)	-	62 (88.6)
Yes	6 (12.8)	2 (8.7)		8 (11.4)
History of psychiatric treatment				
No	5 (10.6)	6 (26.1)	-	11 (15.7)
Yes	42 (89.4)	17 (73.9)		59 (84.3)
History of suicide attempt				
No	41 (87.2)	18 (78.3)	-	59 (84.3)
Yes	6 (12.8)	5 (21.7)		11 (15.7)
History of physical illness				
No	42 (89.4)	23 (100.0)	-	65 (92.9)
Yes	5 (10.6)	0 (0.0)		5 (7.1)
Family history of psychiatric disorder				
No	35 (74.5)	20 (87.0)	-	55 (78.6)
Yes	12 (25.5)	3 13.0)		15 (21.4)
Previous forensic case history				
No	26 (55.3)	4 (17.4)	0.003	30 (42.9)
Yes	21 (44.7)	19 (82.6)		40 (57.1)
Smoking				
No	14	0	-	14 (20.0)
Yes	33	23		56 (80.0)
Alcohol use				
No	30	9	0.051	39 (55.7)
Yes	17	14		31 (44.3)
	Median (Min-Max)		p[b]	Median (Min-Max)
Age	34 (20-65)	32 (23-41)	0.164	33 (20-65)
Duration of disorder	7 (1-30)	7 (2-15)	0.272	7 (1-30)
Number of hospitalizations	1 (0-5)	0 (0-3)	0.007	1 (0-5)
Number of forensic incidents	0 (0-4)	2 (0-5)	0.002	1 (0-5)

a: p value for chi-square test analysis; b: p value for Mann-Whitney U test analysis; $p<0.050$.

For the previous criminal history variable, Cramér's V = 0.355 (medium size effect) was found.

Table 3 Characteristics of patients in the substance-induced psychosis group regarding the type of substance use

		n (%)
Cannabis		
	No	2 (8.7)
	Yes	21 (91.3)
Cocaine		
	No	21 (91.3)
	Yes	2 (8.7)
Methamphetamine		
	No	5 (21.7)
	Yes	18 (78.3)
Heroin		
	No	22 (95.7)
	Yes	1 (4.3)
Cypress		
	No	16 (69.6)
	Yes	7 (30.4)
Ecstasy		
	No	15 (65.2)
	Yes	8 (34.8)
Bonzai		
	No	18 (78.3)
	Yes	5 (21.7)
Inhalant		
	No	22 (95.7)
	Yes	1 (4.3)

Table 4 Significant findings between schizophrenia and substance-induced psychosis groups in terms of Rorchach Test variables

	Schizophrenia	Substance-induced Psychosis	p	r
	Median (Min-Max)			
Unusual : MQ	0 (0-2)	0 (0-4)	0.008	0.317
Total Y	0 (0-2)	0 (0-4)	0.025	0.268
HVI	0 (0-2)	1 (0-6)	0.037	0.249

Unusual : MQ: Unusual:Movement Quality, HVI: Hypervigilance Index, $p<0.050$.

0396 - Social Skills in Specific Learning Disorder: Relationships with Executive Functions, Theory of Mind, and Emotion Regulation

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Background and Objective

Specific learning disorder (SLD) is a neurodevelopmental disorder defined by DSM-5- TR as persistent difficulties in learning and using academic skills for at least six months despite appropriate interventions [1]. Although reading, writing, and mathematics are the most commonly affected domains, the socio-cognitive dimensions of SLD tend to be overshadowed by the predominant focus on academic difficulties in both assessment and treatment. However, the impact of SLD extends well beyond the academic domain, encompassing multiple interrelated socio-cognitive areas including executive functions, theory of mind, emotion regulation, and social skills [2].

This study aimed to examine the relationships among executive functions, theory of mind, emotion regulation, and social skills in children with SLD, and to investigate whether these variables predict social skills.

Method

The study was conducted at the Department of Child and Adolescent Psychiatry, Kocaeli University Faculty of Medicine. Between February and August 2025, 35 children aged 8-12 years diagnosed with SLD according to DSM-5 criteria formed the clinical group, while 35 healthy children matched for age and sex comprised the control group. Inclusion criteria required a WISC-IV full-scale IQ or at least one subtest score of 80 or above. Children with ADHD, autism spectrum disorder, or intellectual disability were excluded.

All participants were evaluated by two clinicians using DSM-5 criteria. Children in the SLD group additionally completed the SLD Battery. Executive functions were assessed with the Behavior Rating Inventory of Executive Function (BRIEF); theory of mind with first- and second-order false belief tasks (Sally-Anne, Smarties, Chocolate, Ice Cream Van) and the Reading the Mind in the Eyes Test (RMET); emotion regulation with the Emotion Regulation Checklist (ERC); and social skills with the Social Skills Rating Scale (SSRS). Independent samples t-test and chi- square analyses were used for group comparisons; Pearson and point-biserial

correlations for relationship analyses; linear regression for predictive analyses; and ROC analysis for clinical discrimination.

Results

Executive Functions

The SLD group scored significantly higher than controls on BRIEF inhibition ($p=0.005$), emotional control ($p=0.002$), working memory ($p<0.001$), monitoring ($p=0.006$), shifting ($p=0.009$), and planning/organization ($p<0.001$) subscales. Significant impairment was also observed in the behavioral regulation index ($p=0.001$), metacognition index ($p<0.001$), and global executive composite ($p<0.001$). No significant difference was found for the initiate and organization of materials subscales.

Emotion Regulation

The SLD group scored significantly higher than controls on all ERC subscales: emotion regulation ($p<0.001$), lability/negativity ($p<0.001$), and total score ($p<0.001$). Higher scores reflect poorer emotion regulation.

Social Skills

The SLD group performed significantly lower than controls on the SSRS total score ($p<0.001$) and ten of eleven subscales, including basic social skills, conversational skills, relationship initiation and maintenance, group

cooperation, emotional skills, self-control, and cognitive skills. No significant difference was found for the accepting outcomes subscale (p=0.621).

Theory of Mind

Pass rates were significantly higher in the control group on the Sally-Anne (p=0.005), Chocolate (p=0.002), and Ice Cream Van tests (p=0.008). The Ice Cream Van test yielded a particularly striking finding: 62.9% of controls passed compared to only 31.4% in the SLD group. RMET scores were also significantly lower in the SLD group (SLD: 18.6±3.12; control: 21.71±2.16; p<0.001). Item-level analysis revealed that both groups accurately identified basic emotional expressions; however, children with SLD notably confused anger with remorse on item 26.

ROC Analyses

For the RMET, AUC=0.807 (95% CI: 0.703-0.911, p<0.001); a cutoff of 20.50 yielded 71.4% sensitivity and 74.3% specificity. For the SSRS, AUC=0.833 (95% CI: 0.740.927, p<0.001); a cutoff of 274.50 yielded 74.3% sensitivity and 77.1% specificity.

Correlation and Regression Analyses

The ERC total score showed a significant negative correlation with SSRS total score in the SLD group (r=-0.338; p<0.05) and significantly predicted social skills in regression analysis (B=-1.756; p=0.047; R^2=0.114). Passing the Chocolate test

significantly predicted relationship initiation-maintenance skills (B=6.299; p=0.048; R^2=0.114), while passing the Ice Cream Van test predicted basic conversational skills (B=4.265; p=0.049; R^2=0.112). Despite numerous significant negative correlations at the subscale level, BRIEF global scores did not significantly predict SSRS scores in regression models.

Discussion

The findings demonstrate that SLD is a multidimensional neurodevelopmental condition with profound effects on socio-cognitive functioning beyond academic difficulties. Although significant correlations were found between executive functions and specific social skill domains, the absence of direct prediction in regression models suggests that this relationship is mediated by variables such as academic self-efficacy, peer rejection experiences, and emotional regulatory capacity rather than being directly causal [3, 4].

Emotion regulation and theory of mind emerged as significant predictors of social skills, highlighting the need for intervention programs that extend beyond academic support. Early targeting of second-order theory of mind and emotion regulation capacities represents a promising therapeutic strategy [5, 6]. Item-level RMET analysis revealed a specific difficulty in distinguishing anger from remorse in children with SLD, a finding with direct clinical implications for emotion-focused interventions. To our knowledge, this study is among the first to evaluate the discriminative power of the RMET and SSRS in SLD through ROC analysis.

In Turkey, special education services for children with SLD largely focus on academic difficulties such as reading, writing, and mathematics, while socio-cognitive domains including executive functions, theory of mind, and emotion regulation remain outside the scope of intervention. Our findings suggest that integrating social skills training and emotion regulation-focused psychosocial interventions into special education curricula is of critical importance for the developmental well-being of these children. Future research should directly examine the mediating roles of language skills, emotional understanding capacity, academic self-efficacy, and peer rejection experiences in the relationships among executive functions, theory of mind, emotion regulation, and social skills; clarifying these mechanisms would provide meaningful contributions to both theoretical models and the design of targeted intervention programs.

Keywords: Specific learning disorder, executive functions, theory of mind, emotion regulation, social skills

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0403 - VR-Based Intervention for Problematic TikTok Use in University Students: Effects on Metacognition and Psychological Symptoms

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TikTok, a platform based on short-form video content, has rapidly become one of the most popular social media platforms worldwide, often associated with long screen time and highly personalized content algorithms [1,2]. The phenomenon of problematic TikTok use (PTU) has emerged as a growing behavioral concern among university students, with increasing evidence associating it with elevated levels of anxiety, depression, and stress [3,4], as well as maladaptive metacognitive beliefs that encourage repeated use [5]. Despite growing interest in interventions for problematic social media use, evidence-based treatments remain limited. Virtual reality therapy (VRT) represents a novel and immersive intervention approach that may enhance emotional regulation, attentional control, and metacognitive awareness. The present study aimed to evaluate the effectiveness of a four-week VRT intervention in reducing PTU and psychological symptoms among university students and to examine the role of metacognitive beliefs before and after the intervention.

A single-group pretest-posttest design was implemented with 24 university students (M_age = 22.2, SD = 1.66). Inclusion criteria required participants to be active TikTok users and enrolled in a higher education institution. Exclusion criteria included current psychiatric treatment, history of neurological disorders, and prior VRT experience. All participants provided informed consent prior to enrollment. Participants completed the TikTok Addiction Scale (TTAS), the Metacognitions Questionnaire-30 (MCQ-30), and the Depression Anxiety Stress Scale-21 (DASS-21) at baseline and following the intervention.After the initial assessment, participants engaged in a structured VRT program consisting of twelve 30-minute sessions delivered over four weeks via XRHealth software in combination with Pico headsets; the platform has been authorized by several international regulatory bodies, including the U.S. Food and Drug Administration (FDA), Israel's Ministry of Health (AMAR), and the Australian Register of Therapeutic Goods (ARTG) [6]. The intervention spanned four weeks and consisted of twelve sessions, each lasting about 30 minutes. Because XRHealth does not provide specific recommendations for treating problematic TikTok use, the number of sessions was determined with reference to earlier studies applying VRT in mental health contexts [7]. The intervention included relaxation exercises, guided breathing, attentional training, and mindfulness-based activities within immersive virtual environments. Assessments were administered under standardized conditions, and participants were instructed to maintain regular sleep patterns and refrain from substance use during the 24 hours preceding testing. Paired-sample statistical analyses were used to examine pre-post differences, while correlation and multiple regression analyses explored associations between PTU, metacognitive beliefs, and psychological symptoms.

Following the intervention, significant reductions were observed in TTAS tolerance (TTAS-T; $d = 0.615$, $p < .05$) and conflict (TTAS-C; $d = 0.478$, $p < .05$), as well as in anxiety symptoms measured by the DASS-21 (DASS-A; $d = 0.637$, $p < .05$), indicating medium to large effect sizes.

Although reductions were observed across nearly all subscales following the VRT intervention, including TTAS-MM, TTAS-W, TTAS-R, MCQ-PBW, MCQ-NMW, MCQ-NCT, MCQ-CSC, MCQ-CC, DASS-D, and DASS-S, these changes did not reach statistical significance. However, significant improvements were found in TTAS-T, TTAS-C, and DASS-A, indicating that the intervention effectively reduced tolerance and conflict associated with TikTok use, as well as anxiety symptoms. Regression analyses demonstrated that maladaptive metacognitive beliefs were significantly associated with higher levels of PTU at baseline.

The findings provide preliminary evidence that VR-based interventions may effectively reduce specific dimensions of problematic TikTok use, particularly tolerance and conflict, and alleviate anxiety symptoms among university students. The results also highlight the central role of metacognitive beliefs in PTU, suggesting that interventions

targeting metacognitive processes may enhance treatment efficacy. Further research using controlled designs and larger samples is recommended to confirm and extend these findings.

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0409 - Evolution of Buprenorphine/Naloxone Maintenance Therapy into Intravenous Misuse: A Case of Pharmacodynamic Destabilization, Hepatitis C Seropositivity, and Psychotic Symptoms

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Introduction

Opioid use disorder is a chronic, relapsing condition associated with significant functional impairment, social and occupational deterioration, and a high risk of serious medical complications. The buprenorphine/naloxone (BNX) combination is an effective agent for managing withdrawal symptoms and reducing craving during maintenance treatment. However, in some patients, altering the route of administration—particularly intravenous (IV) use—may reinforce the search for rapid and intense effects, thereby weakening the protective effects of treatment; injection-related infections and psychiatric comorbidities may further complicate clinical management.

This poster aims to present the clinical features of intravenous misuse developing during BNX treatment, and to discuss its impact on the treatment process in the context of hepatitis C positivity and accompanying psychotic symptoms, as well as its clinical implications.

Case Presentation

A 29-year-old male patient had a history of polysubstance experimentation beginning in adolescence; however, his clinical course was primarily characterized by opioid use disorder that began in early adulthood and progressively worsened over the years. His history included cigarette use during primary school years, cannabis use at age 13, ecstasy use at age 15, and rapid transition to heroin. He reported experiencing a marked sense of well-being and detachment from reality following ecstasy use. Heroin use began around the age of 20, occasionally reaching high daily doses, and attempts to quit were unsuccessful. Over time, his functioning deteriorated significantly, leading to social and occupational losses. A family history revealed intravenous use of opioids and BNX among siblings.

His treatment history included three inpatient admissions to the Addiction Treatment and Research Center (AMATEM) at Sakarya Training and Research Hospital in 2019, 2023, and 2025; it was understood that he continued to use illicit substances and BNX following discharge.

The patient reported that since the age of 24, he had been crushing prescribed Suboxone 8 mg tablets, dissolving them in boiled water, and injecting them intravenously three to four times per week. He described intravenous use as producing a "stronger effect, marked sedation, and faster relief; similar to heroin." His reports of tremors, sweating, restlessness, and irritability when not using the medication suggest a pattern oscillating between withdrawal-related distress and the pursuit of rapid reward.

Psychiatric evaluation revealed suspiciousness, persecutory ideation, referential and mystical delusions, as well as auditory and visual hallucinations. The patient was hospitalized in the male psychiatry ward of Sakarya Training and Research Hospital due to psychotic symptoms. Complete blood count and liver and renal function tests were within normal limits. In his medical history, hepatitis C was noted, and serological testing revealed anti-HCV positivity, while other ELISA parameters were within normal limits. Urine toxicology screening was positive for buprenorphine and negative for other substances.

Treatment and follow-up were maintained with olanzapine 20 mg/day, buprenorphine/naloxone 16 mg/day, and quetiapine 100 mg/day. During inpatient follow-up, psychotic symptoms markedly regressed and social interaction improved.

The patient was discharged on day 23 in a clinically stable condition, with a plan for close follow-up at the Addiction Treatment and Research Center outpatient clinic.

Discussion

Pattern of Misuse and Clinical Evolution: This case demonstrates that buprenorphine/naloxone treatment should be re-evaluated not only in terms of its pharmacological properties but also in terms of its pattern of use. The most striking clinical feature of this case is the evolution of BNX treatment into intravenous misuse.

Pharmacological Properties of BNX: The pharmacological properties of BNX are critical in this context. Buprenorphine is a high-affinity partial ^-opioid agonist, whereas naloxone is a potent ^-opioid antagonist. While the plasma half-life of buprenorphine is approximately 40 hours (long-acting), naloxone is eliminated much more rapidly (approximately 1 hour). This pharmacokinetic difference allows buprenorphine to remain active in plasma for a longer duration while limiting the duration of naloxone's effect, potentially contributing to pharmacological conditioning.

Deterrent Effect of Naloxone: Naloxone has minimal clinical effect when administered sublingually due to its low bioavailability; however, when administered intravenously, it has the potential to cause abrupt receptor blockade. Nevertheless, in this case, the absence of the expected deterrent effect of naloxone and the patient's report of heroin-like euphoria following IV BNX use suggest that the effect of buprenorphine predominates. This finding indicates that parenteral misuse of BNX cannot always be prevented and may be restructured through reward mechanisms in certain patients.

Reinforcement Mechanisms: The observed pattern of use reflects a cyclical process involving avoidance of withdrawal symptoms (negative reinforcement) and the pursuit of rapid and intense subjective effects following injection (positive reinforcement). This dual reinforcement loop appears to be a key mechanism sustaining the addictive behavior.

Infectious Complications: Intravenous substance use significantly increases the risk of serious complications, including HIV/HCV infections, endocarditis, and intravascular infections. The hepatitis C positivity observed in this case represents a clinical consequence of long-term injection behavior. Therefore, management of such patients requires not only addiction treatment but also regular monitoring for infectious diseases and a multidisciplinary approach.

Psychiatric Comorbidity: From a psychiatric perspective, the presence of concurrent methamphetamine and cannabis use, along with psychotic symptoms, is noteworthy. Stimulant substances may exacerbate psychotic symptoms, and the interaction between substance use patterns and psychopathology should be carefully considered in treatment planning.

Treatment Implications: From a treatment perspective, abrupt discontinuation of BNX may lead to severe withdrawal symptoms and increased relapse risk. Long-acting buprenorphine depot formulations (e.g., weekly or monthly injections) may be effective in breaking this cycle by preventing medication manipulation and maintaining stable serum levels.

Clinical Recommendation: These findings indicate that clinicians should not rely solely on dose adjustment, but should actively assess the route of administration and patterns of misuse.

Conclusion

Intravenous misuse of BNX represents a high-risk condition in which a therapeutic agent evolves into uncontrolled use. This case highlights that, in patients with clinical destabilization during BNX treatment, careful assessment of the route of administration is critical and standard treatment approaches may not always be sufficient.

Integrated and individualized treatment strategies, including harm reduction approaches, depot buprenorphine formulations, and effective management of comorbid psychiatric conditions, are recommended.

Keywords: Opioid use disorder, buprenorphine/naloxone, intravenous misuse

Ethical Statement: Written informed consent was obtained from the patient for this case report.

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0410 - Progression from Selective Mutism to Bipolar Disorder: A 10-Year Clinical Follow-Up of a Case

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Abstract

Introduction

Selective mutism (SM) is an anxiety disorder characterized by a persistent failure to speak in specific social settings during childhood. The etiology of SM has been associated with oppositional behaviors and anxiety-based behavioral inhibition. In DSM-IV-TR, it was classified among disorders first diagnosed in infancy, childhood, or adolescence, whereas in DSM-5 it is categorized under anxiety disorders. Although onset is typically before the age of five, clinical recognition often occurs after school entry. Psychiatric comorbidities are common in SM; however, progression to bipolar disorder (BD) has rarely been reported. The aim of this case report is to discuss the clinical features and diagnostic process of a patient with SM and mild intellectual disability (MID) who progressed to BD during adolescence.

Methods

All data related to the case were retrospectively evaluated through child and adolescent psychiatry outpatient clinic records, teacher reports, psychological assessment results, and family interviews. During follow-up, the patient's academic progress, level of social functioning, behavioral changes, and response to treatment were recorded in detail.

Case

The patient, diagnosed with mild intellectual disability at the age of eight, presented with complaints of not speaking with peers and teachers, social withdrawal, negativistic and irritable behavior, and academic difficulties. Treatment with risperidone was initiated (0.5 mg/day, later increased to 1 mg/day). During risperidone treatment, improvement in social communication and partial reduction in negativism and irritability were observed. Although there was no history of seizures, an EEG was performed following reports of hypnagogic visual hallucinations, which revealed bilateral frontotemporal paroxysmal activity. The patient was referred to pediatric neurology; however, no diagnosis of epilepsy was established. During this period, the family discontinued psychiatric follow-up. Two years later, due to an increase in SM symptoms, the patient was re- evaluated and fluoxetine (20 mg/day) monotherapy was initiated; however, no improvement in SM symptoms was observed. Partial symptom improvement was achieved after adding risperidone (0.5 mg/day) to the treatment. At the age of 12, the clinical picture included a marked decline in academic performance, persistent social withdrawal, repetitive questioning, ...a marked decline in academic performance, persistence of social withdrawal, and the emergence of obsessive symptoms characterized by repetitive questioning. During follow-up, within one month of reducing risperidone, there was an increase in speech output and psychomotor activity, heightened sexual drive, decreased need for sleep, and increased irritability. The patient was diagnosed with a manic episode of bipolar disorder. Remission was achieved with risperidone and valproate treatment.

Discussion

This case represents a rare but clinically significant longitudinal progression from selective mutism to bipolar disorder, particularly notable due to its course in the presence of mild intellectual disability. Although selective mutism is typically defined as an anxiety-based disorder specific to childhood, its long- term clinical course—especially in the presence of neurodevelopmental comorbidities—has not been sufficiently elucidated.

In this case, early symptoms such as mutism, irritability, and negativism initially appeared consistent with selective mutism; however, the fluctuating course, limited treatment response, and the later emergence of atypical features (including visual hallucinations and obsessive symptoms) suggested a more complex underlying psychopathology.

Notably, the worsening of symptoms following fluoxetine treatment and the subsequent emergence of manic features—such as increased psychomotor activity, reduced need for sleep, increased speech, and irritability—are consistent with the literature indicating that antidepressants may precipitate activation and manic switch in individuals with bipolar vulnerability. This finding highlights the importance of careful treatment planning in children diagnosed with selective mutism.

The presence of mild intellectual disability may further complicate the diagnostic process by altering the expression of clinical symptoms and masking the typical presentation of mood disorders. Additionally, neurodevelopmental comorbidities may contribute to a heterogeneous clinical picture and make early recognition of affective symptoms more challenging. During follow-up, the development of a manic episode according to DSM-5 criteria and a significant response to mood stabilizer treatment (valproate) support the classification of the clinical presentation within the bipolar disorder spectrum. The observed improvement in both mood stabilization and behavioral symptoms with valproate further strengthens this interpretation.

This case suggests that, particularly in subgroups of selective mutism characterized by prominent negativism and co-occurring neurodevelopmental features and hallucinations, the condition may represent a prodromal phase of bipolar disorder. It also underscores the need to consider this possibility during treatment planning.

The presence of negativism and hallucinations alongside selective mutism and mild intellectual disability, as well as the lack of response to fluoxetine and the marked improvement with risperidone, indicate an atypical clinical course.

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0416 - Meaningful Gains with Small Data: Poisson-Port, a Novel Neural Network Architecture—Validated on Two Challenging Psychiatric Datasets (Depression Severity and Suicide-Risk Classification)

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Introduction

Artificial intelligence has advanced rapidly with deep learning, but its success largely depends on massive datasets and substantial computational power. In fields such as medical diagnostics, psychiatry, and rare event analysis, labeled data are often expensive, limited, or ethically difficult to obtain, making classical deep learning models prone to overfitting and poor generalizability in small-sample settings. In contrast, biological neural systems can learn from limited and noisy inputs, adapt to changing environments, and make functional decisions under uncertainty. Inspired by these properties, this study proposes the Poisson Port architecture, which preserves the practical usability of classical ANNs while selectively incorporating biologically inspired firing principles within a statistical framework. The model is designed for data-constrained settings and is evaluated on both synthetic and challenging clinical datasets.

Conceptual Background

Limitations of Classical Artificial Neural Networks (ANNs)

Traditional ANNs are highly capable of modeling complex, non-linear relationships due to their multi-layered structures. However, these architectures are characterized by "data hunger," requiring high processing power, dense memory usage, and significant energy consumption [1]. In heterogeneous and noisy clinical datasets, a limited sample size often leads classical ANNs to memorize random noise (overfitting) rather than capturing underlying patterns. Consequently, their performance and reliability are not consistent across all data types, particularly in small- sample regimes.

Biological Information Processing: Firing Dynamics and Selective Abstraction

Biological nervous systems remain functional despite incomplete and noisy inputs, partly through firing-based and energy-efficient information processing. However, directly reproducing all biological mechanisms in artificial systems increases both theoretical and computational complexity [1]. A more practical strategy is to selectively abstract useful principles, such as stochastic firing and threshold-based processing, into computational models. Poisson Port follows this approach.

Spiking Neural Networks (SNNs) and Current Technical Obstacles

Spiking Neural Networks provide a more biologically inspired framework by encoding information through spike timing. Although promising for energy-efficient and event-based processing, their training is more difficult than that of conventional networks because the discrete nature of spikes limits direct gradient-based optimization and often requires surrogate methods. In addition, limited software infrastructure and lack of standardization continue to restrict their widespread practical use.

Methodology

Technical Structure of the Poisson Port Architecture

The Poisson Port architecture models action potentials using a Poisson distribution, creating a hybrid framework that blends the input layers of classical ANNs with the firing logic of SNNs. The primary components of the model include:

- **Input and Weighting:** The input section resembles classical ANN structures in its linear product of lambda (A) variables and weight combinations.
- **Firing Modeling:** The output layer is re-modeled as a Poisson distribution, excluding the time variable while maintaining the probabilistic nature of the distribution. This preserves the "firing rate" logic while reducing computational complexity along the time axis.
- **Gating (Port) Mechanism:** A specific threshold (k) is established at the synaptic junction. Only probabilities exceeding this threshold are transmitted to the next neuron. This "all-or-nothing" principle ensures the damping of noise and the preservation of meaningful signals.

Mathematical Foundations and Probabilistic Gating

The computational flow of the model is based on the following mathematical foundations:

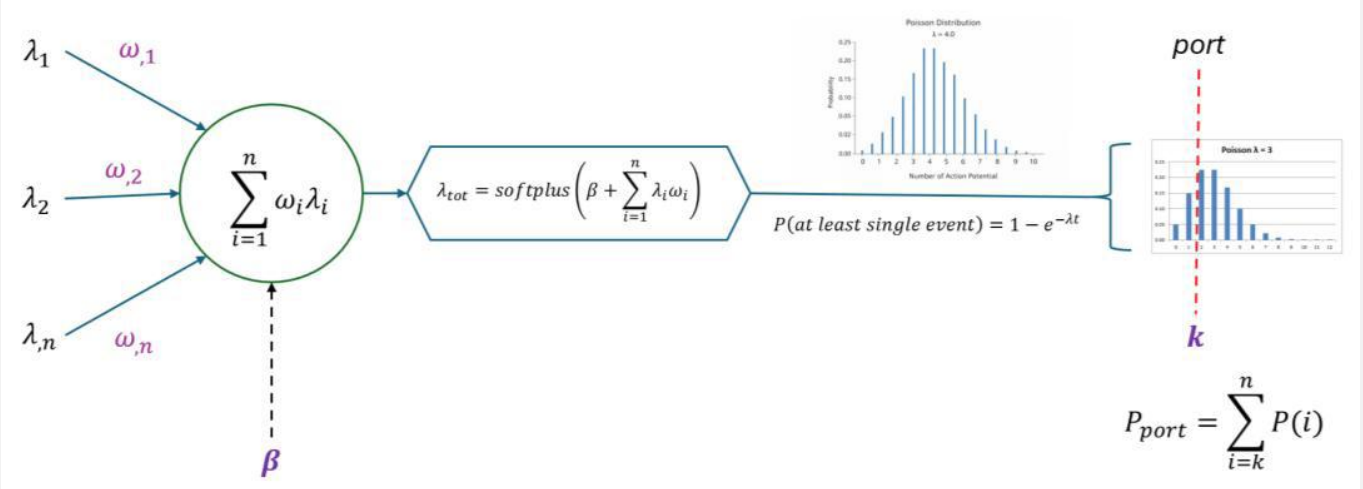
1. Total Lambda Calculation: The sum of inputs (λ_i) and weights (ω_i) is combined with a bias (β) and passed through a softplus activation function:

$$\lambda_{tot} = softplus\ (\beta + \sum_{i=1}^{n} \omega_i \lambda_i$$

2. **Probability Distribution:** The probability of at least one firing event occurring is expressed as:

$$P(at\ least\ one\ event) = 1 - e^{-\lambda t}$$

3. **Port (Threshold) Function:** The gating mechanism applied over the cumulative probability value is defined as: The k parameter is a critical threshold that determines the selectivity of the model, acting as a natural regulation boundary that prevents overfitting



Experimental Design and Validation Stages

To evaluate the performance and adaptability of the model, a three-stage testing strategy with increasing complexity was followed:

- **Stage I: Basic Validation with Virtual Datasets:** Five synthetic (virtual) datasets were used to measure statistical consistency and fundamental learning capabilities. This provided a controlled environment to determine convergence speed and hyperparameter sensitivity.

- **Stage II: Challenging Clinical Datasets and Heterogeneity Analysis:**

  o **Suicide Dataset (Small Sample):** Consisting of only 75 samples, this dataset tested the model's ability to generalize when data is critically scarce.

  o **Depression Dataset (Large and Heterogeneous):** With 807 samples, this dataset is quantitatively larger but highly heterogeneous, with a low sample count for "severe depression" cases. This measured the model's success in identifying minority classes (rare cases) within unbalanced distributions.

- **Stage III: Deep Learning Benchmarking and Visual Feature Analysis:** The model's capacity for feature hierarchy construction and processing complex structural information was tested using MNIST and raw Parkinson's drawing data. This stage compared Poisson Port against classical deep learning architectures (ANN, CNN).

Comparison Models and Evaluation Metrics

A standard protocol was followed for comparing AI algorithms. Cross-validation was used to ensure reliability in small datasets. Performance was measured using Accuracy, Recall (Sensitivity), Precision, F1-Score, and Specificity. Comparison models included XGBoost and Logistic Regression (strong in low-data scenarios), as well as classical ANN and Convolutional Neural Networks (CNN).

Results

Performance on Virtual (Generic) Datasets

Initial tests on five virtual datasets confirmed the stability and statistical significance of the Poisson Port architecture:

Table 1: Accuracy on Virtual Datasets

Dataset	Accuracy (%)
Dataset 1 (Generic)	97.5%
Dataset 2 (Generic)	86.9%
Dataset 3 (COVID-19)	89.2%
Dataset 4 (Database Final)	86.5%
Dataset 5 (MNIST - Virtual Test)	95.7%

Analysis of Challenging Clinical Datasets: Depression and Suicide

Clinical data was used to measure success in heterogeneous and unbalanced structures.

A. Depression Dataset (4-Class, 807 Samples) Despite the rarity of severe depression cases, Poisson Port achieved 95.8% accuracy, performing on par with Logistic Regression (95.9%) and outperforming classical ANN (90.0%) and XGBoost (88.2%) [1].

Table 2: Depression Dataset Performance Comparison

Model	Accuracy (%)	Correct Predictions / Total
Poisson Port	95.8%	773 / 807
Logistic Regression	95.9%	774 / 807
Classical ANN	90.0%	726 / 807
XGBoost	88.2%	712/807

B. Suicide Dataset (2-Class, 75 Samples) In this critically low-sample case, Poisson Port achieved 76.0% accuracy. While classical ANN performed poorly (61.3%), Poisson Port successfully competed with statistical models like XGBoost (78.7%).

Deep Learning Benchmarking: MNIST Performance

The scalability of Poisson Port to deeper structures requiring complex feature extraction was tested on the MNIST dataset (1438 test samples).

Table 3: MNIST Benchmarking Results

Model	Accuracy (%)	Correct Predictions / Total
Poisson Port	92.2%	1326/1438
Classical ANN	89.2%	1283/1438

Feature Analysis in Data-Constrained Visual Tasks: Parkinson's Data

The model's most significant advantage emerged in diagnosing Parkinson's disease from patient drawings (50 training, 30 test samples). This task requires visual feature extraction but is severely limited by data volume.

Table 4: Parkinson's Dataset Comparison: Poisson Port vs. Classical CNN [2]

Metric	Poisson Port	Classical CNN
Accuracy	76.67%	66.67%
Precision	78.57%	77.78%
Recall	73.33%	46.67%
F1-Score	75.86%	58.33%

Classical CNNs suffered significantly in recall (46.67%) due to data scarcity, whereas Poisson Port maintained a balanced performance (73.33% recall) through its probabilistic gating mechanism.

Discussion

Learning and Generalization in Small Data Scenarios

The most fundamental finding of this study is the Poisson Port architecture's ability to maintain high performance without succumbing to overfitting in small sample sizes. While classical ANNs tend to optimize weights toward random noise when data is limited, Poisson Port's probabilistic approach and synaptic "port" threshold ensure a more balanced learning process [1]. This mechanism ensures only meaningful signals propagate, thereby enhancing generalization.

Balancing Statistical Stability and Deep Feature Learning

Traditional machine learning algorithms (XGBoost, Logistic Regression) excel at structured small data but face architectural limitations in "deepening" or learning complex deep features. Poisson Port preserves the stability of these statistical models in small clinical datasets while maintaining functionality in deep network structures, as evidenced by MNIST and Parkinson's tests. The model remains mathematically traceable like logistic regression while offering the flexibility to capture complex patterns through multi-layered architectures.

Beyond SNNs: From Biological Inspiration to Engineering Solutions

Spiking Neural Networks (SNNs) are difficult to train on standard hardware due to their time- dependence and non-differentiable nature. By abstracting the time variable and utilizing the stochastic nature of the Poisson distribution, the Poisson Port architecture combines the firing logic of SNNs with the computational ease of classical ANNs [1].

This strategy validates that the abstraction of functional principles (selective information processing, stochastic thresholding) is more critical for engineering success than the direct replication of biological processes.

Conclusion

The Poisson Port architecture supports AI use in clinical diagnosis and psychiatry, where data are scarce and costly, by achieving high accuracy and recall with small datasets and overcoming key limitations of classical deep learning in tasks such as Parkinson's diagnosis and suicide risk analysis. Its three-stage validation showed stability on general data, robustness on limited clinical data, and scalability in deep learning tasks, while its hardware flexibility and lower dependence on large data centers improve accessibility for both clinical practice and academic research.

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0471 - Carbon Footprint of Antipsychotic Treatment in Schizophrenia: A Comparative Analysis of Oral and Long-Acting Injectable Antipsychotics

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Introduction: Climate change is one of the most significant global health threats, and healthcare systems contribute substantially to greenhouse gas emissions. The carbon footprint, defined as the total amount of greenhouse gas emissions generated by an activity, is widely used to assess environmental impact [1]. Pharmaceuticals are an important contributor to healthcare-related emissions due to their production, transportation, and disposal processes. In psychiatry, antipsychotic medications are widely used in long-term treatment of schizophrenia and are administered either as daily oral formulations or long-acting injectable (LAI) formulations. Previous studies suggest that LAI antipsychotics may improve adherence and reduce relapse and hospitalization rates compared to oral treatments [2,3]. Since these factors influence healthcare utilization, they may also affect the environmental burden of care. However, direct comparisons of carbon footprint between oral and LAI antipsychotic treatments are lacking. Therefore, this study aimed to compare the carbon footprint of oral and LAI antipsychotic treatments and to identify factors associated with environmental impact in patients with schizophrenia.

Methods: In this retrospective cross-sectional study, 59 patients with schizophrenia attending the Psychiatry Outpatient Clinic of Van Yuzuncu Yil University Faculty of Medicine were included; analyses were completed with 54 patients due to missing data. Participants were categorized into three groups: oral, long-acting injectable (LAI), and combination therapy. Carbon footprint was estimated based on healthcare utilization and environmental indicators, including frequency of healthcare visits, transportation type, travel distance to healthcare facilities, medication packaging disposal practices, hospitalization, and environmental awareness and sustainability attitudes. Descriptive statistics, distribution analyses, and hierarchical regression models were applied.

1. Data Collection

Data were obtained from electronic medical records and structured questionnaires, including:

- Sociodemographic characteristics
- Medication adherence (Morisky Medication Adherence Scale)
- Functional status (Brief Functioning Assessment Scale)
- Healthcare utilization variables (visit frequency, transportation, hospitalization)

2. Carbon Footprint Calculation

Carbon footprint (kg CO_2/year) was calculated based on:

- Transportation-related emissions
- Medication packaging waste
- Hospitalization-related emissions

3. Statistical Analysis

Statistical analyses were performed using SPSS version 28.0 and Python 3.9. Descriptive statistics, distribution analyses, and hierarchical regression models were used. Log transformation was applied due to skewed distribution. Statistical significance was set at $p < 0.05$.

Results: Analyses were conducted on a complete-case sample of 54 participants. Descriptive statistics of the continuous variables are presented in Table 1. The mean annual carbon footprint (TOTAL_CO_2) was 793.65 kg CO_2 (SD = 1034.12), with a median of 379.3 kg CO_2 and a range of 39.0-5192.0 kg CO_2, indicating substantial variability across participants. The mean log-transformed carbon footprint (LOG_TOTAL_CO_2) was 5.966 (SD = 1.258). The mean medication adherence score was 4.019 (SD = 1.596), functionality score was 31.722 (SD = 20.137), treatment duration was 10.13 years (SD = 7.69), and mean age was 37.259 years (SD = 11.649) (Table 1). The distribution of TOTAL_CO_2 showed right skewness; therefore, LOG_TOTAL_CO_2 was used in regression analyses. Hierarchical regression model comparisons are presented in Table 2. The model including only treatment modality explained a minimal proportion of variance (R^2 = 0.016, adjusted R^2 = -0.022), indicating that treatment type alone was not a significant predictor of carbon footprint. When clinical variables were added, explanatory power increased (R^2 = 0.113, adjusted R^2 = 0.02). The inclusion of contextual variables further increased the explained variance (R^2 = 0.185), although adjusted R^2 remained low (-0.005), suggesting limited model efficiency. The optimized parsimonious model yielded R^2 = 0.161 and the highest adjusted R^2 (0.034), indicating a modest improvement in model performance. Standardized regression coefficients from the optimized model are presented in Table 3. Medication adherence showed the strongest positive association with carbon footprint (β = 0.279), followed by age (β = 0.216), while treatment duration showed a negative association (β = -0.243). Overall, these findings indicate that carbon footprint variability was only weakly explained by the models and was more strongly associated with clinical characteristics than treatment modality.

Discussion: This study found that antipsychotic treatment type (oral vs. LAI) was not significantly associated with carbon footprint. Although LAI treatments are known to improve adherence and reduce relapse [4], these advantages did not translate into lower carbon emissions. Instead, medication adherence emerged as the strongest predictor. Higher adherence was associated with increased carbon footprint, likely due to increased healthcare interactions such as outpatient visits and transportation. These findings suggest that environmental impact is more strongly influenced by healthcare utilization patterns rather than treatment modality alone. This is consistent with previous literature emphasizing the role of healthcare systems and service organization in carbon emissions [5,6]. The study has limitations, including small sample size, single-center design, and indirect estimation of carbon footprint. Future studies should include larger samples and life-cycle-based analyses.

Keywords: Schizophrenia, carbon footprint, antipsychotics, environmental sustainability, healthcare utilization

Funding: Scientific Research Projects Coordination Unit of Van Yuzuncu Yil University

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Table 1. Descriptive Statistics of Continuous Variables (Complete-Case Sample, n = 54)

Variable	count	mean	std	min	25%	50%	75%	max
TOTAL_CO2	54.0	793.646	1034.119	39.0	169.0	379.3	1177.0	5192.0
LOG_TOTAL_CO2	54.0	5.966	1.258	3.689	5.135	5.94	7.072	8.555
Medication_Adherence	54.0	4.019	1.596	0.0	3.0	4.0	5.0	6.0
Functionality_Score	54.0	31.722	20.137	0.0	15.25	29.0	46.75	72.0
Treatment_Duration_Years	54.0	10.13	7.69	1.0	4.0	9.5	13.75	35.0
Age	54.0	37.259	11.649	19.0	29.25	34.0	45.0	64.0

Table 2 Hierarchical Model Comparison

Model	R^2	Adjusted R2
Model 1 (Treatment)	0.016	-0.022
Model 2 (Clinical)	0.113	0.02
Model 3 (Context)	0.185	-0.005
Model 4 (Optimized)	0.161	0.034

Table 3 Standardized Regression Coefficients (Optimized Model)

Predictor	Standardized Beta
Medication_Adherence	0.279
Treatment_Duration_Years	-0.243
Age	0.216

Invited Speech Texts

17th International Congress on Psychopharmacology & Child and Adolescent Psychopharmacology / Psychotherapy

0131 - Antidepressants and the Placebo Effect

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Background and Objective: Antidepressants are widely used pharmacological agents for the treatment of various psychiatric disorders, particularly major depressive disorder, anxiety disorders, obsessive-compulsive disorder, and post-traumatic stress disorder. However, some studies have shown that the effectiveness of these medications—especially in mild to moderate cases of depression—can be difficult to distinguish from the placebo effect. A placebo is a substance or intervention with no pharmacological effect. Nonetheless, due to a patient's positive expectations about the treatment, physiological or psychological improvement can be observed. This improvement is known as the placebo effect. In disorders characterized by subjective symptoms, such as depression, this effect can be remarkably strong. Many studies have demonstrated that antidepressants are statistically superior to placebo. However, this superiority is particularly evident in cases of severe depression. In a large-scale network meta-analysis published by Cipriani et al. in The Lancet (2018), antidepressants were found to be more effective than placebo, but the effect sizes were generally small to moderate (SMD ≈ 0.3–0.5). Similarly, Fournier et al. (2010, JAMA) emphasized that this effect becomes more pronounced in severe depression cases.Comparable findings have been observed in studies involving children and adolescents. Among SSRIs, fluoxetine has shown significant superiority over placebo in treating adolescent depression and has been approved by the FDA. However, the placebo response in child and adolescent populations remains high, typically ranging from 40% to 60%. In disorders that affect internal states—such as depression and anxiety—the placebo effect can account for 30–50% of symptom improvement. Factors such as treatment expectation, the doctor–patient relationship, and the perceived meaning of the treatment can amplify this effect. Therefore, randomized, double-blind, placebo-controlled trials are essential to differentiate true pharmacological effects from placebo-induced changes.

Despite their relatively modest effect sizes, antidepressants retain an essential role in treatment planning due to benefits such as reducing relapse rates and suicide risk. Nevertheless, in cases of mild to moderate depression, psychoeducation and psychotherapeutic interventions should be considered as first-line treatment options.

Conclusions: Although antidepressants remain vital tools in the treatment of depression, the contribution of the placebo effect—particularly in milder cases—should not be overlooked. In the future, personalized treatment approaches and the use of biological markers may help identify which patients are most likely to benefit from pharmacological interventions.

Keywords: Psychopharmacology, antidepressants, placebo

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AuthorToEditor: Sayın bilim kurulu, Placebo effect in psychiatry konulu oturum önerimizi kabul ettiğiniz için teşekkür ederiz. Bu bildiri bu oturumun antidepressants and the placebo effect başlıklı konuşmasının özetidir. Saygılarımla Prof. Dr. Burcu Akın Sarı ORCID: 0000-0002-9730-7206

0134 - The Intergenerational Transmission and Healing of Anxiety: Epigenetic Mechanisms, Therapeutic Approaches and Prevention

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Anxiety disorders, including separation anxiety disorder, are increasingly conceptualized as lifespan conditions rooted in the interaction between attachment systems, stress regulation, and early environmental exposures. While familial clustering of anxiety has long suggested inherited vulnerability, contemporary models emphasize that anxiety risk is not solely genetically determined. Instead, it emerges from dynamic interactions between biological susceptibility, early relational experiences, and cumulative psychosocial stress. Within this developmental framework, epigenetic mechanisms provide a critical link between early adversity and long-term emotional and behavioral outcomes.

Epigenetics refers to environmentally sensitive regulatory processes—such as DNA methylation, histone modification, and non-coding RNA activity—that influence gene expression without altering the underlying DNA sequence. These mechanisms enable early life stress and caregiving experiences to leave lasting effects on brain systems involved in emotion regulation. Animal and human studies indicate that prenatal and early-life stress is associated with epigenetic alterations in limbic and cortical regions—particularly the amygdala and cingulate cortex—as well as in stress-related pathways within the hypothalamic–pituitary–adrenal (HPA) axis. Such changes have been associated with heightened stress reactivity and difficulties in affect regulation, which may increase vulnerability to anxiety-related psychopathology across development. The prenatal period represents a particularly sensitive window for epigenetic programming. Maternal anxiety, depression, and psychosocial stress during pregnancy have been associated with DNA methylation changes in placental and cord blood epigenetic markers related to neurodevelopment and stress regulation, including increased methylation of the glucocorticoid receptor gene (NR3C1), suggesting altered stress responsivity early in life. Importantly, parental anxiety may influence offspring through multiple, interacting pathways, including prenatal stress exposure, postnatal caregiving behaviors, and shared environmental contexts. Attachment insecurity may function as an important psychological pathway within this process. Anxious caregiving styles, such as overprotection or heightened threat signaling, can reinforce children's fear responses and may be associated with alterations in stress-related epigenetic regulation. Similarly, anxiety-prone parental behaviors may influence children through vicarious learning and epigenetic priming of stress-related circuits, with oxytocin- and corticotropin-related pathways discussed as possible biological mechanisms.

Crucially, epigenetic mechanisms are increasingly viewed as modifiable rather than fixed markers of risk. Accumulating evidence indicates that psychosocial interventions targeting stress regulation, attachment-related processes, and emotional processing can be accompanied by measurable changes in stress-related epigenetic markers, alongside clinical improvement. These changes have been observed in epigenetic markers related to neuroendocrine stress regulation, suggesting partial normalization of stress-responsive systems. Together with emerging pharmacological approaches that influence epigenetic regulation, non-pharmacological interventions such as psychotherapy and enriched environments support the concept of sustained biological and psychological plasticity. From a preventive perspective, the most effective opportunities to interrupt intergenerational anxiety cycles appear early in development. Perinatal mental health interventions, early identification and treatment of parental anxiety, and attachment-focused parent–infant therapies represent critical points of intervention. Integrating epigenetic insights with attachment-informed and evidence-based treatments may ultimately promote resilience and reduce the long-term burden of anxiety across generations.

Keywords: Anxiety, Attachment, Early Environmental Adversity, Epigenetics, Intergenerational Transmission

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0135 - Safety and Efficacy of Atomoxetine in Children and Adolescents with Autism Spectrum Disorder

Nevin Kılınç

Mersin İl Sağlık Müdürlüğü Anamur Devlet Hastanesi

Autism Spectrum Disorder (ASD) is a severe, lifelong neurodevelopmental condition characterized by early-onset deficits in social communication and the presence of repetitive sensory–motor behaviors. It has a strong genetic basis, along with other contributing factors, and imposes substantial social costs and a significant burden on the quality of life of affected individuals and their families. (1,2) Attention Deficit Hyperactivity Disorder (ADHD) and ASD are increasingly prevalent neurodevelopmental conditions, with symptoms and associated impairments frequently persisting into adulthood. Both disorders impose substantial functional limitations and societal costs, which are further exacerbated in individuals with comorbid ASD and ADHD. Collectively, these conditions account for a significant proportion of children receiving school-based services, underscoring their considerable public health relevance. (3) Atomoxetine is effective in reducing hyperactivity, inattention, and impulsivity in individuals with comorbid ASD and ADHD, although its efficacy is lower and adverse effects are more frequent than in idiopathic ADHD. Atomoxetine, a selective dopamine and norepinephrine reuptake inhibitor approved for ADHD across the lifespan, represents a viable alternative treatment and may confer partial benefits on certain core ASD symptoms and executive function deficits; however, its effects on overall social functioning appear limited. (1)

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Keywords: Autism, Psychopharmacology, Atomoxetine
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0158 - Efficacy and Safety of Alpha-2 Agonists in Autism Spectrum Disorder

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Autism spectrum disorder (ASD) is a neurodevelopmental condition characterized by persistent deficits in social communication and interaction, along with restricted and repetitive behaviors and interests, with onset in early childhood. 1Pharmacological treatment in ASD primarily targets comorbid symptoms rather than core features, as co-occurring conditions such as ADHD (attention deficit hyperactivity disorder), anxiety, irritability, and sleep disturbances are common.2–4

Alpha-2 adrenergic receptor agonists, may be considered particularly in patients with ASD who also experience hyperactivity, irritability, impulsivity, aggression, sleep disturbances, and anxiety. 2–4While stimulant medications are typically first-line treatments for ADHD, treatment selection in children with ASD requires careful consideration of comorbidity profiles and tolerability.2

Clinical studies suggest that guanfacine is associated with reductions in hyperactivity, impulsivity, and ADHD symptoms.5 Clonidine has been associated with improvements in hyperactivity, impulsivity, hyperarousal, and social functioning; moreover, some studies have reported signals of benefit for sleep, attentional difficulties and aggression.6 However, close monitoring is required due to potential adverse effects such as sedation, hypotension, and bradycardia.2,3

In clinical practice, alpha-2 agonists are initiated at low doses and titrated gradually based on symptom response and tolerability. 2 When anxiety significantly interferes with functioning, behavioral and educational interventions should be considered as first-line approaches.2 If these strategies are insufficient or not feasible, pharmacological treatment may be selected based on target symptoms and tolerability. Given the variable evidence regarding efficacy and side-effect burden of selective serotonin reuptake inhibitors (SSRIs) in ASD, treatment decisions should be made with caution. In this context, guanfacine may be considered as a second-line option for anxiety associated with ASD, particularly when accompanied by hyperarousal or sleep disturbances. 2 For sleep onset difficulties, alpha-2 agonists are initiated at low doses and titrated slowly at intervals of 3–7 days, with adjustments based on efficacy and adverse effects.2 In cases of irritability associated with ASD, behavioral and educational strategies should be implemented first. If symptoms are moderate to severe or refractory to non-pharmacological interventions, pharmacological treatment may be considered.2

Although it has been demonstrated that alpha-2 receptor agonists affect various symptoms in patients diagnosed with ASD and many studies have found that side effects are tolerable, the sample size and level of evidence in these studies are limited.5

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Keywords: Alpha-2 agonist, autism spectrum disorder, efficacy, safety
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0166 - Intervention strategies for Antipsychotic-Induced Weight Gain: Lifestyle, Switching and Pharmacologic Augmentation

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Managing antipsychotic-induced weight gain (AIWG) is a clinical priority to reduce cardiometabolic risk and support long-term adherence, particularly in severe mental illness. While lifestyle interventions are the standard first-line approach, their real-world effectiveness is often insufficient due to iatrogenic sedation, cognitive deficits, and motivational challenges inherent to this population. Accordingly, the literature increasingly emphasizes practical, individualized delivery—"small-steps" goal setting, brief structured follow-up, and supportive tools—ideally initiated early in treatment. When the metabolic burden outweighs expected benefit and clinical stability allows, switching to agents with lower weight-gain liability (e.g., aripiprazole, ziprasidone, lurasidone) may be considered, but decisions must balance metabolic safety against relapse risk, and switching is frequently constrained in clozapine-treated or treatment-resistant patients. For pharmacologic augmentation, metformin remains the best-established option, with consistent evidence for modest mitigation of weight gain and metabolic risk. GLP-1 receptor agonists (e.g., liraglutide and semaglutide) are increasingly discussed as higher-efficacy options for selected patients with AIWG or metabolic complications, with emerging data in antipsychotic-treated populations and feasibility considerations (tolerability, monitoring, access) informing real-world use. Alternative strategies (e.g., topiramate, zonisamide, or agent-specific combinations such as olanzapine/samidorphan) may be reserved for specific situations given heterogeneity of evidence and tolerability constraints. Ultimately, a proactive and multidisciplinary approach that integrates behavioral and pharmacological strategies is essential to curtail cardiometabolic risk in this vulnerable population. By addressing AIWG early as a core clinical target, clinicians can apply an early, stepped-care sequencing framework that improves metabolic health while safeguarding long-term psychiatric stability.

Keywords: Antipsychotic-induced weight gain, Antipsychotic switching, Cardiometabolic risk, GLP-1 receptor agonists, Metformin

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0172 - The Role of SSRIs in the Management of Obsessive-Compulsive Disorder: Where Do We Stand?

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Obsessive-compulsive disorder (OCD) is a psychiatric condition with early onset in childhood and adolescence, characterized by a heterogeneous clinical course and significant functional impairment. Treatment planning is guided by multiple factors, including symptom severity, level of insight, comorbid psychiatric conditions, and familial factors. In clinical practice, cognitive behavioral therapy with an exposure and response prevention (ERP) component is considered the core psychotherapeutic approach, while selective serotonin reuptake inhibitors (SSRIs) represent one of the most commonly preferred pharmacological treatments in moderate to severe cases (Geller & March, 2012; Steele et al., 2024).

Available evidence indicates that SSRIs are associated with a statistically significant reduction in obsessive and compulsive symptoms in children and adolescents with OCD compared to placebo; however, the magnitude of this effect may be clinically modest (Mills et al., 2020). Treatment response appears to vary across individuals, and factors such as baseline symptom severity, comorbid anxiety disorders, the presence of tic disorders, and family involvement have been reported to influence clinical outcomes (McGuire et al., 2015). These findings suggest that SSRI treatment should not be viewed as a universal standalone solution but rather as a component of an individualized treatment approach.

Studies comparing SSRI monotherapy with combined treatment strategies suggest that the combination of SSRIs and ERP may be associated with higher response rates, particularly in patients with marked symptom burden and functional impairment (McGuire et al., 2015; Song et al., 2022). By directly targeting avoidance behaviors and compulsive rituals, ERP appears to complement the effects of pharmacotherapy. Although SSRIs provide an effective treatment option when access to psychotherapy is limited, combined treatment approaches are generally considered to offer potential advantages in terms of long-term clinical outcomes (Steele et al., 2024). From a safety perspective, SSRIs are generally reported to have an acceptable tolerability profile in children and adolescents. The most commonly observed adverse effects include gastrointestinal symptoms, headache, sleep disturbances, and behavioral activation (Mills et al., 2020). As activation symptoms may emerge particularly during the early phases of treatment, initiating treatment at low doses with gradual titration is recommended. Careful clinical monitoring for suicidal ideation and behavior, along with appropriate family psychoeducation, remains an essential component of the treatment process (Geller & March, 2012).

In conclusion, SSRIs continue to represent a widely used and evidence-supported pharmacological option in the treatment of pediatric OCD. Optimal clinical outcomes are most likely to be achieved through an integrated treatment approach that combines ERP-based psychotherapy with individualized risk–benefit assessment and regular clinical monitoring (Steele et al., 2024).

Keywords: Children, Adolescent, Obsessive-Compulsive Disorder, Treatment

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0180 - Overview of Glucagon-Like Peptide-1 Receptor Agonists (GLP-1 RAs): Pharmacokinetic/Pharmacodynamic considerations, feasibility, and choice

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Introduction

Glucagon-like peptide-1 (GLP-1) is an incretin hormone released from intestinal L cells in response to nutrient intake and exerts its effects via activation of the GLP-1 receptor. GLP-1 enhances glucose-dependent insulin secretion in pancreatic β cells, promotes β-cell preservation by supporting regeneration and inhibiting apoptosis, suppresses glucagon release from α cells, delays gastric emptying, and strengthens central satiety signaling, thereby reducing food intake. As a result, GLP-1 exerts favorable metabolic effects across multiple tissues, including adipose tissue, skeletal muscle, and the cardiovascular system (Drucker, 2018; Hinnen, 2017; Lim & Brubaker, 2006).

Endogenous GLP-1 is rapidly degraded within minutes by the enzyme dipeptidyl peptidase-4 (DPP-4). Structural modifications applied to GLP-1 receptor agonists (GLP-1RAs) markedly prolong their half-lives. Pharmacodynamic differences between short-acting and long-acting GLP-1RAs directly influence their clinical effects on postprandial glucose control, appetite regulation, and weight loss (Nauck et al., 2021).

Beyond their established role in the treatment of type 2 diabetes and obesity, GLP-1RAs have demonstrated clinical benefits in several obesity-related conditions, including cardiovascular disease and heart failure, chronic kidney disease, obstructive sleep apnea, osteoarthritis, and metabolic dysfunction–associated steatohepatitis. (Kosiborod et al., 2024; Lincoff et al., 2023; Newsome et al., 2024; Perkovic et al., 2024) An increasing body of evidence suggests that GLP-1RAs may exert potential neuropsychiatric effects in addition to their metabolic actions. Preclinical and translational studies have indicated possible antidepressant and anxiolytic properties, while early clinical findings have reported promising outcomes in alcohol use disorder, substance use disorders, and binge-eating behavior (Brunchmann et al., 2019; Da Porto et al., 2020; Pozzi, 2019). In contrast, analyses of real-world data derived from the FDA Adverse Event Reporting System (FAERS) have shown disproportionate reporting of depressive symptoms and suicidal ideation associated with liraglutide and semaglutide compared with metformin and insulin, whereas no comparable signal has been identified for dulaglutide, exenatide, lixisenatide, or tirzepatide (McIntyre et al., 2024). Accordingly, the FDA's recommendation for a cautious approach appears justified. Until more definitive evidence becomes available, prescribing GLP-1 receptor agonists in patients with a history of depression or suicide attempts should be guided by a careful and individualized risk–benefit assessment. GLP-1RAs have also attracted increasing interest as adjunctive therapeutic options for managing second-generation antipsychotic–associated weight gain, insulin resistance, and metabolic disturbances. In psychiatric populations, agent selection should involve a comprehensive evaluation of pharmacokinetic properties, dosing frequency, gastrointestinal tolerability, potential suicidality risk, drug–drug interactions, and patient adherence.

Conclusion

In conclusion, integrating GLP-1 receptor agonists into the management of antipsychotic-induced metabolic adverse effects may substantially improve long-term physical health outcomes in individuals with severe mental illness. A pharmacokinetic- and pharmacodynamic-informed approach to GLP-1RA selection may further facilitate the development of personalized treatment strategies across both metabolic and psychiatric patient populations.

Keywords: antipsychotic-induced weight gain, GLP-1 receptor agonists, pharmacodynamics, pharmacokinetics, psychiatry

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0237 - Heart Rate Variability and Psychiatric Disorders: Diagnostic Categories and Clinical Correlates of HRV

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Background and Objective:

Heart Rate Variability (HRV) is a fundamental physiological indicator reflecting autonomic nervous system balance and vagal tone. In child and adolescent psychopathology, HRV has gained increasing attention due to its associations with stress sensitivity, emotion regulation processes, and neurodevelopmental mechanisms. This presentation aims to provide a comprehensive synthesis of HRV profiles across pediatric psychiatric diagnostic groups based on current clinical studies and meta-analyses.

Methods:
Systematic reviews, meta-analyses, and large-sample clinical studies published between 2000 and 2024 in PubMed, Scopus, and Web of Science databases were examined. Findings were categorized under anxiety disorders, major depressive disorder, attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD), externalizing disorders, post-traumatic stress disorder (PTSD), schizophrenia, and bipolar disorder.

Results:
In children and adolescents with anxiety disorders, marked reductions in resting HRV (particularly HF-HRV and RMSSD) indicate heightened sympathetic activation. Meta-analyses in major depressive disorder show moderate reductions in resting HRV and a negative correlation between HRV and depression severity. Although resting HRV findings in ADHD are heterogeneous, vagally mediated HRV consistently decreases during cognitive and emotional tasks.

The relationship between HRV and neurophysiological markers of executive functioning in youth is noteworthy. Meta-analytic evidence demonstrates that reduced vagal tone is associated with poorer cognitive flexibility, inhibition, and working memory performance, suggesting HRV as a biological correlate of executive functions. This association is more prominent in children with ADHD, ASD, and emotion regulation difficulties. In ASD, significant reductions in HRV are observed both at rest and under social stress, with pronounced impairments in RSA reactivity. Longitudinal data in externalizing disorders show that low resting vagal tone is linked to aggression, impulsivity, and emotion regulation deficits. PTSD exhibits one of the most pronounced HRV reductions, with hyperarousal, intrusion, and dissociative symptoms strongly related to diminished vagal tone. In schizophrenia, autonomic dysregulation is associated with negative symptoms, cognitive slowing, and social withdrawal; meta-analyses consistently show reduced vagally mediated HRV and sympathetic–parasympathetic imbalance. In bipolar disorder, both depressive and manic episodes demonstrate significant decreases in HF-HRV, increased sympathetic activity, and impaired autonomic flexibility. Some studies also indicate persistent HRV abnormalities during euthymia, suggesting a possible trait-level biological vulnerability.

Conclusions:
Reduced HRV emerges as a widespread and transdiagnostic biological pattern across child and adolescent psychopathology. Although not a stand-alone diagnostic marker, HRV holds substantial potential as a neurophysiological indicator for assessing emotion regulation capacity, stress reactivity, and treatment response monitoring.

Keywords: Heart rate variability, adolescent psychopathology, neurodevelopmental

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0313 - Digital Behaviors and Mental Health: Clinical Assessment and Novel Approaches

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The rapid integration of digital technologies into daily life has led to marked changes in the clinical presentation of mental health problems across developmental stages. Although digitalization has emerged as a defining feature of the developmental environment of Generation Z and Generation Alpha, shaping cognitive, emotional, and social processes from early childhood, clinical observations and recent evidence suggest that digital behavior–related difficulties are increasingly encountered across all age groups, including adults from Generation Y and older generations. From a clinical perspective, digital behaviors have increasingly become an essential component of psychiatric assessment rather than a peripheral contextual factor, particularly in individuals presenting with attentional difficulties, emotional dysregulation, sleep problems, impulsivity, and functional impairment. In this context, digital behaviors may interact with underlying vulnerabilities and modify symptom expression, thereby complicating clinical evaluation and differential diagnosis. This presentation focuses on the clinical assessment of digital behaviors and discusses emerging approaches to clinical management and treatment in the context of digital behavior–related mental health problems. Digital behaviors are conceptualized as clinically meaningful patterns that can shape symptom expression and functional impairment, underscoring the importance of accurate assessment as a foundation for appropriate and individualized therapeutic strategies. By integrating current evidence with clinical experience, this presentation aims to provide a practical, assessment-oriented framework that supports both clinical evaluation and evidence-based treatment approaches across developmental stages.

Keywords: Children and adolescents, Clinical assessment, Digital behaviors, Mental health, Treatment approaches

AuthorToEditor: 17.04.2026 / 13:45-15:15 Mental Health of Generation Z and Generation Alpha Children panelinin Digital Behaviors and Mental Health: Clinical Assessment and Novel Approaches sunumun özeti ektedir. Dr. Mehmet Can Erata ORCHID-0000-0002-6394-8988

0321 - Clinical Applications and Interventions of Heart Rate Variability in Child and Adolescent Psychiatry: From Biomarker to Biofeedback

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Background: Heart rate variability (HRV) reflects the dynamic balance between sympathetic and parasympathetic activity of the autonomic nervous system and has recently gained attention as a psychophysiological marker in psychiatry. In child and adolescent mental health, HRV is considered a promising tool for assessing stress responses, monitoring treatment effects, and guiding psychophysiological interventions.

Methods: This presentation synthesizes recent systematic reviews and empirical studies (2014–2024) exploring HRV in child and adolescent psychiatric populations. The clinical use of HRV in stress assessment, pre–post treatment changes with pharmacotherapy, psychotherapy, and mindfulness-based interventions, as well as methodological issues such as device variability and measurement standards, are discussed. Practical applications including HRV biofeedback, breathing training, and lifestyle-related approaches are also reviewed.

Findings: Decreased HRV has been consistently associated with elevated stress, anxiety, and depressive symptoms. HRV tends to increase following pharmacological or psychotherapeutic interventions, reflecting improved autonomic flexibility. HRV biofeedback and resonance frequency breathing (~0.1 Hz) have shown efficacy in enhancing emotion regulation and reducing anxiety. Mindfulness, yoga, adequate sleep, nutrition, and moderate physical activity further contribute to HRV improvement. However, measurement variability, developmental differences, and device heterogeneity limit clinical standardization in pediatric settings.

Conclusion: HRV represents a sensitive, non-invasive biomarker for emotional regulation and treatment response monitoring in child and adolescent psychiatry. HRV biofeedback and lifestyle-based approaches are promising adjunctive interventions, supporting the integration of physiological self-regulation into everyday clinical practice. Future studies should focus on establishing age- and sex-specific normative databases and long-term efficacy across clinical populations.

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Keywords: Autonomic Nervous System, Biofeedback, Biomarker, Child and Adolescent Psychiatry, Emotion Regulation, Heart Rate Variability

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0339 - Reconsideration of Diagnosis in Treatment Resistance: What does the current literature say about the diagnostic confusion?

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Diagnostic differentiation between neurodevelopmental disorders and major psychiatric conditions remains a persistent challenge in adult psychiatry. Attention-deficit/hyperactivity disorder (ADHD) is frequently confused with bipolar disorder due to overlapping features such as distractibility, impulsivity, emotional lability, and increased activity, while adults with autism spectrum disorder (ASD) are sometimes misdiagnosed with psychotic disorders because of atypical communication styles, sensory experiences, or social misunderstanding. These diagnostic errors may result in inappropriate pharmacological treatment, delayed interventions, and poorer long-term outcomes.

In adults, differential diagnosis between ADHD and bipolar disorder, and ASD and psychosis, should focus on key distinguishing principles such as episodic and persistent symptom patterns, longitudinal course, age of onset, developmental history, sleep and circadian changes, judgment, and functional decline. It is crucial to differentiate mood elevation from emotional reactivity and psychotic symptoms from sensory or cognitive phenomena associated with autism.

A clinically focused and longitudinal approach aims to improve diagnostic accuracy in adult psychiatric practice, promote nuanced differential diagnosis, and support evidence-based decision-making when evaluating complex cases such as ADHD, bipolar disorder, autism, and psychosis.

Keywords: ADHD, Autism Spectrum Disorder, Bipolar Disorder, Differential Diagnosis, Psychosis

0415 - Hypersalivation and Constipation; GI Side Effects

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Clozapine is considered the gold standard treatment for treatment-resistant schizophrenia; however, its use is limited by a broad side effect profile, including potentially serious gastrointestinal complications. Clozapine-induced gastrointestinal hypomotility (CIGH) is an increasingly recognized but often underdiagnosed clinical condition. Reduced gastrointestinal motility associated with clozapine is common and may lead to delayed gastric emptying, gastrointestinal dysmotility, and constipation; however, it may also progress to serious and potentially life-threatening events such as paralytic ileus, bowel obstruction, gastrointestinal infarction, gastrointestinal necrosis, and gastrointestinal perforation. Clozapine is estimated to be approximately three times more likely to cause constipation compared to other antipsychotics. Although the exact mechanism has not been fully elucidated, it is primarily attributed to clozapine's anticholinergic and antiserotonergic effects [1]. Clozapine antagonizes cholinergic M1 and M3 receptors, thereby interfering with the autonomic regulation of the intestines, inhibiting smooth muscle contraction, and slowing intestinal transit. Antagonism of serotonin 5-HT2, 5-HT3, 5-HT6, and 5-HT7 receptors also plays a role in gastrointestinal motor and sensory functions. Antiadrenergic effects may additionally contribute. Identified risk factors include advanced age, male sex, and higher clozapine dose [2]. Clozapine-induced gastrointestinal hypomotility is a preventable cause of serious morbidity and mortality. It is thought to remain insufficiently recognized in clinical practice.

Hypersalivation, another anticholinergic adverse effect, is also one of the most frequently encountered side effects of clozapine [3]. Nocturnal hypersalivation has been reported to occur more frequently than daytime hypersalivation. Excessive salivation at night is more prominent and is referred to as the "wet pillow sign." Hypersalivation may cause choking sensations during sleep, nighttime awakenings, hoarseness or voice disturbances, and chronic cough. Clozapine-induced sialorrhea may contribute to and increase the risk of potentially life-threatening aspiration pneumonia. The pathophysiology of clozapine-induced excessive salivation has not been fully understood; however, antagonism of muscarinic M1, M2, M3, and M5 receptors, stimulation of M4 muscarinic receptors, and alpha-2 receptor antagonism are thought to play a role in the development of this clinical problem [4]. Vasoactive intestinal peptide may potentiate the effects of clozapine and lead to excessive salivation. Additionally, alterations in the swallowing reflex and reduced laryngeal peristalsis are believed to contribute to this condition. Rapid titration is known to be a potential risk factor. Both non-pharmacological interventions and pharmacological treatment options are available. Clozapine-induced hypersalivation may result in reduced quality of life and treatment non-adherence.

Both CIGH and hypersalivation have significant clinical and psychosocial consequences. While gastrointestinal hypomotility may lead to severe conditions requiring emergency medical intervention, hypersalivation markedly reduces quality of life, causes social embarrassment, and decreases treatment adherence. Since discontinuation of clozapine due to adverse effects may result in psychiatric destabilization, early recognition and proactive management of these adverse effects are of critical importance. Pharmacological and non-pharmacological treatment options are available, and structured monitoring protocols should be integrated into routine care for patients receiving clozapine in order to reduce morbidity, mortality, and treatment discontinuation rates.

Keywords: Clozapine, Constipation, Hypersalivation

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0417 - Growing Up in The Digital World: Parenting, Privacy and Social Media Behaviors in Children and Adolescant and Impact on Adult Psychiatry, The Impact of Social Media Use in Childhood and Adolescence on Adult Psychiatry

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This panel examines the multidimensional impact of social media use during childhood and adolescence on adult psychiatric morbidity from both neurodevelopmental and clinical perspectives. It is posited that intensive early-life engagement with social media may contribute to long-term neuropsychiatric alterations by influencing prefrontal cortex maturation, reward sensitivity, social feedback processing, and emotional regulation mechanisms. According to recent statistics, in 2022 individuals spent an average of 151 minutes per day on social media, clearly underscoring the global significance and pervasive impact of these platforms (1).

Problematic social media use during childhood and adolescence is associated with an increased risk of anxiety disorders in adulthood (2). In particular, mechanisms such as social comparison, fear of online evaluation, and digital social feedback loops appear to intensify symptoms of social anxiety and generalized anxiety disorder. With regard to depressive symptomatology, chronic online exposure to social media during adolescence and early adulthood may increase vulnerability to adult depression through pathways involving loneliness, low self-esteem, sleep disturbances, and alterations in reward-processing sensitivity. Moreover, the current literature suggests that social media may increase suicide risk among youth through mediating factors such as emotional numbing, social isolation, and cyberbullying.

The association between early social media use and adult symptoms of Attention-Deficit/Hyperactivity Disorder (ADHD) will be addressed through the lenses of attentional control, digital multitasking habits, and models of immediate reward expectancy. In particular, studies demonstrating a relationship between high screen time and continuous exposure to digital stimuli during childhood and the severity of adult ADHD symptoms will be presented in this session. Moreover, the coding architecture of numerous online platforms and applications—including social media—is deliberately structured to activate the limbic system and variable reward sensitivity (3). These mechanisms resemble the neurobiological systems implicated in substance-related addictions and other behavioral addictions, thereby reinforcing repetitive and prolonged patterns of use (4).

The potential impact of early digital experiences on mood disorders in adulthood, particularly bipolar disorder, has increasingly been recognized. Disruptions in the sleep–wake cycle, heightened levels of online stimulation, irregularities in social rhythms, and increased emotional reactivity may exert significant effects on bipolar vulnerability.

Finally, the relationship between social media use and psychotic disorders will be examined, particularly in the context of the reinforcement of paranoia-related cognitive patterns through online content, digital isolation, cyberbullying, and traumatic online experiences. In at-risk youth populations, when social media use converges with the social withdrawal and functional decline commonly observed during the prodromal phase, online avoidance and isolation may contribute to the exacerbation of symptoms—or, conversely, emerging symptoms may further intensify maladaptive patterns of social media engagement—thereby creating a reciprocal cycle. This comprehensive session aims to propose an integrative model elucidating the impact of early digital experiences on adult psychiatry, to identify both risk and protective factors, and to develop evidence-informed recommendations for clinical practice and preventive mental health policies.

Keywords: Social media use, anxiety, bipolar disorder, ADHD, digital stimuli, psychosis

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0418 - Digital Parenting and Sharenting: A New Space Between Childhood and Parenting

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The term “sharenting”, a combination of “sharing” and “parenting”, refers to the practice of parents publicly sharing information about their children on social media. This is not merely a “sharing behavior”. It represents the transposition of parenting into the digital environment. Sharenting often emerges from positive intentions, driven by various parental motivations. Parents often want to express love, pride, and attachment toward their children in online spaces. In addition, the pursuit of social support and validation, the desire to demonstrate parenting competence, to share experiences with other parents, and to belong to social communities—sometimes even economic gains—constitute common motivations behind this behavior. Nevertheless, the potential effects of such shared content on the child’s future self-perception, self-esteem, and social relationships require careful consideration. Sharenting is a process in which the parent speaks on behalf of the child, potentially limiting the child’s right to their own narrative. The circulation of images or private moments belonging to the child in a public digital sphere, and their control by the parent, may reduce the child’s subjectivity to an externally defined image. The permanence of digital content can have long-term effects on how the child will be remembered and represented in the future. Particularly during adolescence, such exposure may create vulnerabilities in identity formation, self-esteem, and peer relationships. The child may face a digital identity that no longer feels like their own. Encountering previously shared content can lead to emotions such as shame, anxiety, withdrawal, or difficulties in social relationships. Moreover, adolescents often wish to maintain control over their own images and information, and they may feel discomfort regarding parental sharing. All these considerations indicate that sharenting is not merely a behavior related to social media use, but a phenomenon closely tied to the boundaries of the parent–child relationship and the perception of autonomy. This presentation will explore the definition and developmental process of the concept of "sharenting," the reasons parents engage in this behavior, and the associated psychosocial implications.

Keywords: Adolescent, children, digital world, sharenting

0419 - Beyond the Scale: Neuropsychiatric Weight of Obesity in Severe Mental Illness

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Dalhousie University

Obesity is the fifth leading cause of death globally and one of the leading causes of disability. While the general medical impacts of obesity, including cerebrovascular complications, are relatively well-recognized, the less obvious effects on brain health are often overlooked. Obesity is frequently linked with brain cortical thinning, subcortical atrophy, accelerated brain aging, cognitive impairments, and an increased risk of dementia, even in absence of additional cardiovascular risk factors. This is particularly concerning in people with severe mental illness (SMI), where the rates of obesity are high and the brain effects of obesity and psychiatric disorders may interact. Indeed, we and others have demonstrated that obesity adds to brain gray and white matter alterations in SMIs. Variations in extent of obesity contribute to variations in extent of brain alterations in people with bipolar disorders or schizophrenia. Importantly, obesity related brain changes explain part of the cognitive impairment already in people with first episode of psychosis. Moreover, baseline weight or weight gain predict future acceleration of brain aging and hippocampal atrophy across SMIs. These brain effects could explain why obesity in SMI is associated with worse psychiatric outcomes, including greater psychiatric morbidity, chronicity, disability, functional decline, and worse responses to psychiatric medications. Monitoring weight and body composition thus becomes relevant for managing psychiatric, cognitive and brain health. Future research should investigate if prevention or treatment of obesity, i.e. with GLP1 agonists, could prevent or improve neurostructural changes and related psychiatric outcomes, including cognitive impairment.

Keywords: Obesity, bipolar disorders, brain, MRI

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0420 - Neuroprotective Effects of Lithium Food for Thought

Tomas Hajek

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Lithium is the gold standard for treatment of bipolar disorders. It is one of the most bioactive elements and among psychiatric medications it has the strongest and most replicated evidence for neuroprotective effects in both pre-clinical and clinical studies. Among 28 psychiatric medications, including antipsychotics, anticonvulsants, antidepressants, Li demonstrates the most replicated evidence for neuroprotection in the widest range of models. Here, I will review studies documenting neuroprotective effects of Li in human participants, including brain imaging studies performed by us and others. There is in fact level I evidence for positive association between Li treatment and brain gray matter volume, which has been repeatedly demonstrated in cross sectional studies, meta-analyses, mega-analyses, replicated longitudinal studies including a randomized controlled trial. Next, I will summarize epidemiological/treatment studies of association between Li treatment and risk of dementias. Separately, I will review evidence linking environmental exposure to Li with lower risk of dementia, as the basis for suggesting that lower Li levels, but longer exposure are needed for neuroprotective effects. This is supported also by a recent preclinical study demonstrating association between Li defficiency and risk of dementia and suggesting that Li has a physiological role at very low doses. This study was critical as it renewed interest in Li, whereas traditionally research into the neuroprotective effects of Li has been limited by the fact that as a natural element, Li cannot be patented. There are currently no registered studies of Li for treatment of Alzheimer dementia. This creates emphasis on testing Li also in other neurodegenerative disorders. We recently studied effects of Li in glaucoma, which is a neurodegenerative loss of retinal ganglion cells. In a pharmacoepidemiological study, we demonstrated that relative to a single prescription, higher cumulative exposures to Li might be protective against glaucoma. While Li has shown highly replicated evidence for neuroprotection at different levels of study, there is a real danger that this research will stop and never be translated into clinical application. By reviewing this body of evidence, I hope to stimulate more interest in neuroprotective effects of Li.

Keywords: Lithium, brain, dementia, neuroprotection

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0421 - Arketamine: The Overlooked Driver of Ketamine's Rapid and Sustained Antidepressant Effects

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Chiba University

Depression is a leading cause of disability worldwide, underscoring the urgent need for rapid-acting and durable therapeutic strategies. Over the past decade, the dissociative anesthetic ketamine has attracted considerable attention because a single subanesthetic dose can produce antidepressant effects within hours that persist for one to two weeks despite its short plasma half-life. Clinically, however, maintenance of these benefits typically requires repeated administration, which increases the risk of dissociative and psychotomimetic symptoms, abuse liability, cognitive disturbance, and other adverse outcomes. Elucidating the biological mechanisms responsible for ketamine's sustained antidepressant effects is therefore essential for the development of safer and more effective next-generation treatments.

Accumulating clinical evidence indicates that dissociative symptoms resulting from N-methyl-D-aspartate receptor (NMDAR) blockade are not necessary for the antidepressant actions of ketamine or esketamine. Consistent with this observation, preclinical studies demonstrate that arketamine, the (R)-enantiomer with substantially lower affinity for NMDARs, produces more robust and longer-lasting antidepressant-like effects than esketamine while inducing fewer behavioral abnormalities and neurotoxic changes. These findings challenge the traditional NMDAR-centric framework and instead implicate alternative molecular mechanisms—particularly activation of the brain-derived neurotrophic factor (BDNF)–TrkB signaling cascade and downstream extracellular signal–regulated kinase (ERK) pathways—as critical mediators of sustained synaptic plasticity and antidepressant efficacy.

Following regulatory approval, intranasal esketamine has been widely implemented for treatment-resistant depression across multiple regions. Nevertheless, pharmacovigilance analyses derived from international safety databases, including those of the World Health Organization and the U.S. Food and Drug Administration Adverse Event Reporting System, have raised concerns regarding adverse psychiatric outcomes such as suicidal ideation, as well as unresolved questions about long-term safety and tolerability. These real-world observations highlight the importance of refining ketamine-based therapeutics to maximize benefit while minimizing harm.

Our recent preclinical investigations further demonstrate that the antidepressant efficacy of esketamine is comparable to that of its principal metabolite, (S)-norketamine, whereas the metabolite exhibits a more favorable side-effect profile. This evidence suggests that downstream metabolites—and more broadly, enantiomer-specific pharmacology—may play decisive roles in determining both therapeutic response and adverse effects. Such findings reinforce the concept that ketamine's clinical actions cannot be fully explained by NMDAR inhibition alone.

Taken together, converging clinical, pharmacological, and mechanistic data support a revised conceptual framework in which arketamine functions as a principal driver of ketamine's rapid and sustained antidepressant effects. This perspective emphasizes the necessity of disentangling the distinct biological contributions of ketamine enantiomers and their metabolites rather than relying solely on racemic formulations or receptor-blockade models. Future progress will require rigorously designed, double-blind clinical trials directly comparing arketamine with esketamine, as well as studies evaluating esketamine versus (S)-norketamine, to determine optimal efficacy, durability, and safety. Such investigations may ultimately enable the development of next-generation rapid-acting antidepressants that retain therapeutic potency while substantially reducing adverse outcomes.
In this keynote lecture, I will synthesize current evidence regarding enantiomer-specific and metabolite-dependent mechanisms underlying ketamine's antidepressant actions and discuss future translational strategies aimed at improving the treatment of major depressive disorder.

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Keywords: Depression, Ketamine, Esketamine, Arketamine, S-norketamine, NMDA receptor

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0422 - Gut–Brain Axis Mechanisms Underlying MDMA-Induced Stress Resilience

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3,4-Methylenedioxymethamphetamine (MDMA), widely known as ecstasy or molly, is commonly used in recreational settings because it enhances sensory perception, euphoria, empathy, and social bonding. Beyond recreational use, recent clinical studies have investigated MDMA-assisted psychotherapy for post-traumatic stress disorder (PTSD), based on the hypothesis that MDMA-induced release of prosocial neurohormones—particularly serotonin and oxytocin—facilitates trauma processing and reduces long-term psychological distress. However, in August 2024, the U.S. Food and Drug Administration declined approval of MDMA-assisted therapy for PTSD, citing methodological limitations, uncertain durability of benefit, and safety concerns, including cardiovascular risk and abuse potential.

Emerging observational evidence in humans nevertheless suggests that MDMA may enhance stress resilience. Among survivors of the October 7, 2023 Supernova music festival attack, individuals who had taken unadulterated MDMA reported lower psychological distress, better sleep quality, and greater post-event social engagement than substance-naïve survivors or those who used cannabis and/or alcohol. Population-level survey analyses similarly associate MDMA or psilocybin exposure with reduced odds of suicidal ideation, psychological distress, and major depressive episodes, although causal inference remains limited. Collectively, these findings raise the possibility that MDMA contributes to stress resilience in humans.

Preclinical studies provide mechanistic insight into this phenomenon. MDMA increases extracellular serotonin by promoting its release and inhibiting reuptake, thereby improving mood, reducing anxiety, and enhancing emotional processing. Because the majority of systemic serotonin originates in the gastrointestinal tract, gut–brain communication is likely central to these effects. In murine models, MDMA pretreatment attenuated chronic stress–induced splenomegaly, anhedonia-like behavior, and elevated interleukin-6 levels, while reshaping gut microbiota composition and circulating metabolites. Importantly, subdiaphragmatic vagotomy abolished these protective effects, demonstrating that MDMA-induced resilience depends on vagus nerve–mediated gut–brain signaling. Additional studies show that intermittent MDMA exposure mitigates cuprizone-induced demyelination and ovariectomy-induced bone loss, further supporting systemic brain–body resilience mechanisms.

Oxytocin signaling represents another critical pathway. MDMA stimulates oxytocin release from hypothalamic nuclei, promoting social bonding, trust, and stress reduction. Notably, intestinal microbiota—particularly Limosilactobacillus reuteri—can enhance host oxytocin secretion via enteroendocrine signaling, revealing a microbiota-driven mechanism linking the gut to social and emotional regulation. Consistently, vagotomy markedly blunts both basal and MDMA-induced oxytocin elevations, underscoring the central role of the vagus nerve in MDMA’s oxytocinergic and resilience-enhancing actions.

Despite these promising findings, MDMA carries substantial long-term risks, including neurotoxicity, hyperthermia, hyponatremia, cardiovascular and psychiatric complications, cognitive impairment, and addiction liability. Elucidation of vagus-dependent gut–brain mechanisms may therefore enable the development of safer therapeutic strategies—such as microbiota-targeted interventions or vagus nerve stimulation—that reproduce resilience-enhancing effects without MDMA-associated adverse outcomes.

In summary, converging clinical, epidemiological, and preclinical evidence indicates that MDMA enhances stress resilience through coordinated modulation of serotonin, oxytocin, and vagus-mediated gut–brain signaling. Clarifying these mechanisms may facilitate the development of novel and safer treatments for trauma-related disorders, including PTSD. This symposium will review current evidence supporting a vagus nerve–dependent gut–brain axis underlying MDMA-induced resilience.

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Keywords: MDMA, Stress resilience, Gut microbiota, Gut-brain axis, Vagus nerve
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0426 - Callous–Unemotional Traits and Impulsivity in Digital Interaction,Risky Challenges, Cyberbullying, and Clinical Findings

Hilal Aydemir

Hilal Aydemir, Serbest Hekim, Muayenehane

The rapid expansion of digital platforms has significantly transformed behavioral patterns among children and adolescents. This shift is particularly evident in youth displaying callous–unemotional (CU) traits combined with high impulsivity. CU traits—characterized by reduced empathy, shallow affect, and diminished guilt—play a substantial role in shaping online interactions and risk-taking behaviors in digital environments.

This presentation examines the risk-oriented digital behaviors observed in children and adolescents with CU traits. A primary focus is the increased participation in risky social media challenges, driven by motivations such as online visibility, attention seeking, and the reinforcing nature of digital feedback. When impulsivity co-occurs with CU traits, these individuals tend to make rapid decisions, exhibit poor risk evaluation, and engage more frequently in high-risk online activities.

Cyberbullying is one of the most prominent antisocial digital behaviors associated with CU traits. Reduced sensitivity to punishment, limited emotional responsiveness, and diminished social norm awareness contribute to the frequency and intensity of online aggression. This presentation will review current literature linking CU traits with cyberbullying behaviors and will illustrate these findings through clinical examples and observed behavior patterns.

Finally, the session highlights clinical implications, focusing on strategies for early identification and intervention. Screening tools, clinical assessment considerations, and interventions targeting emotional awareness and behavioral regulation will be discussed. Understanding this emerging digital risk profile is essential for mental health professionals, educators, and families aiming to promote safer online engagement among youth.

Keywords: Callous–Unemotional Traits, Impulsivity, Digital Interaction

0427 - Psychopharmacological Management of Autism Spectrum Disorder: General Principles

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Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by persistent impairments in social communication and restricted, repetitive behaviors across the lifespan [1]. Beyond core features, individuals with ASD frequently present with irritability, aggression, anxiety, depression, attention-deficit/hyperactivity disorder (ADHD) symptoms, sleep disturbances, self-injurious behaviors, and epilepsy. These co-occurring difficulties substantially impair functioning and quality of life for patients and families. No medication directly treats the core social-communication deficits or restricted behaviors that define ASD. Psychopharmacological treatment is therefore symptom-focused and adjunctive rather than disease-modifying [2].

Given the limited and heterogeneous evidence base, safe prescribing in ASD requires a structured clinical framework. Four core principles optimize the risk–benefit ratio: Start Low and Go Slow, emphasizing cautious titration due to increased sensitivity to adverse effects; Monitoring Matters, requiring systematic assessment of efficacy, metabolic parameters, and neurological side effects; One Size Does Not Fit All, reflecting phenotypic and pharmacodynamic variability; and Back to the Basics, highlighting the importance of evaluating underlying medical contributors such as pain, gastrointestinal problems, sleep deprivation, or seizure activity before escalating medication. These principles are particularly relevant because individuals with ASD may have difficulty reporting adverse effects and may present atypical responses [3].

Behavioral and psychosocial interventions remain first-line treatment, with medication reserved for clearly defined, impairing symptoms. Risperidone and aripiprazole are the only medications approved by the U.S. Food and Drug Administration for severe irritability and aggression in children and adolescents with ASD [4]. These agents demonstrate short-term efficacy for tantrums, aggression, and self-injury; however, weight gain, metabolic disturbances, hyperprolactinemia, and extrapyramidal symptoms require careful monitoring.

For ADHD-like symptoms, stimulants may be effective, though response rates are lower and adverse effects more frequent than in non-autistic populations. Atomoxetine and α2-adrenergic agonists can be considered when stimulants are poorly tolerated or emotional dysregulation predominates. Selective serotonin reuptake inhibitors show mixed evidence for anxiety and repetitive behaviors and may induce behavioral activation in pediatric patients. Anticonvulsants remain essential for seizure management but demonstrate inconsistent behavioral benefits.

Emerging pharmacological strategies targeting glutamatergic transmission, oxidative stress, neuroinflammation, and oxytocinergic pathways reflect growing interest in mechanism-based interventions [2]. However, these approaches remain investigational and require further validation. Polypharmacy is common in ASD and increases the risk of drug–drug interactions and cumulative metabolic burden. International guidelines therefore emphasize linking each medication to a defined target symptom, introducing agents sequentially, and conducting periodic reassessment [1]. Future directions increasingly emphasize biomarker-informed, precision-based prescribing models integrated within multimodal care frameworks [2].

In summary, psychopharmacological management in ASD requires individualized decision-making, cautious titration, systematic monitoring, and integration within multidisciplinary care. The goal is to reduce functional impairment and improve overall quality of life rather than to treat autism itself.

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Keywords: ADHD, Antipsychotics, Autism spectrum disorder, Irritability, Precision medicine, Psychopharmacology
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0428 - Clinical Evidence on GLP-1 Receptor Agonists: Effects on Glucose Dysregulation and Antipsychotic-Induced Weight Gain

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Second-generation antipsychotics, particularly clozapine and olanzapine, are highly effective in the treatment of schizophrenia but frequently cause clinically significant weight gain and metabolic dysregulation, including impaired glucose tolerance and type 2 diabetes. These adverse outcomes increase cardiometabolic morbidity and may compromise adherence. Glucagon-like peptide-1 receptor agonists (GLP-1 RAs) promote weight loss and improve glycemic control in the general population, making them attractive candidates to mitigate antipsychotic-induced weight gain (AIWG) and related glucose dysregulation in schizophrenia.[1]

In a systematic review and meta-analysis pooling seven randomized controlled trials (n=398) in schizophrenia spectrum disorders treated with antipsychotics, GLP-1 RA therapy was superior to placebo for reducing body weight (mean difference [MD] -4,68 kg), body mass index (MD -1,09 kg/m²), and waist circumference (MD -3,66 cm). Glycemic control also improved, with a significant reduction in HbA1c (MD -0,26). Adverse events (predominantly gastrointestinal [e.g., nausea, vomiting, diarrhea]) were more frequent with GLP-1 RAs (risk ratio [RR] 2,3), whereas serious adverse events were less frequent (RR 0,44).[2]

Real-world data are scarce; however, available reports indicate that GLP-1 RAs can be implemented in routine care for patients with severe mental illness. A published case report described once-weekly semaglutide (0,5 mg) in a 50-year-old woman with schizophrenia, obesity, and type 2 diabetes, showing substantial weight loss from 81,2 to 69,3 kg over 6 months with a marked reduction in fat mass and without loss of skeletal muscle index, alongside maintained glycemic control.[3]

Preclinical studies further support a mechanistic rationale that may be relevant to early metabolic risk beyond gradual weight change. In mice, pharmacologic GLP-1R agonism with liraglutide or exendin-4 completely prevented acute olanzapine-induced hyperglycemia; liraglutide increased insulin and prevented the olanzapine-associated rise in glucagon, thereby normalizing the glucagon-to-insulin ratio.[4]

Overall, current clinical and translational evidence supports GLP-1 RAs as a promising strategy for addressing AIWG and glucose dysregulation in schizophrenia, with gastrointestinal tolerability as the main limitation.[2] Future work should define optimal patient selection, dosing, and long-term cardiometabolic outcomes in routine psychiatric practice.

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Keywords: Antipsychotic, GLP-1 Receptor Agonist, Glucose Dysregulation, Weight Gain

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0429 - Attention Deficit Hyperactivity Disorder: Treatment From Childhood To Adulthood Cognitive Behavioral Therapy for Attention Deficit Hyperactivity Disorder

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Objective: While pharmacotherapy remains the primary biological intervention for Adult ADHD, approximately 50% of patients fail to achieve full functional normalization. This panel conceptualizes ADHD not as a deficit of knowledge, but as a "disorder of implementation" where the core challenge lies in the inability to apply skills in the "point of performance." Our objective is to present a modular Cognitive Behavioral Therapy (CBT) framework designed to bridge the "executive gap" by integrating neurobiological psychoeducation with strategic behavioral contingencies.

Phase I: Neurocognitive Psychoeducation & Therapeutic Reframing

The therapeutic alliance is predicated on a neurobiological reframe: ADHD is presented not as a failure of volition, but as a neuropsychiatric deficit in behavioral inhibition rooted in prefrontal-striatal circuit dysfunction. We address the secondary emotional dysregulation arising from chronic failure—specifically the "Incompetence" and "Self-distrust" schemas. By externalizing the disorder, we reduce patient shame and increase engagement with compensatory strategies.

Phase II: Strategic Modular Interventions

The panel details the four pillars of the "Mastering Your Adult ADHD" protocol, grounded in neuropsychological principles:

Organization & Planning: Implementation of the "Single-System Principle" and the A-B-C prioritization matrix to externalize executive demands and reduce cognitive load.

Distractibility Management: Reducing amygdala-driven stress through "Environmental Engineering" and the "Mental Parking Lot" technique to defer impulsive cognitions and manage digital intrusions.

Adaptive Thinking (Cognitive Restructuring): Training patients to replace dysfunctional internal dialogues (e.g., "The Perfectionist Critic") with a rational, task-oriented metacognitive framework.

Overcoming Procrastination: Utilizing the "5-Minute Rule" to lower the activation threshold, thereby triggering dopaminergic reward pathways via task initiation rather than task completion.

Phase III: Analysis of Compensatory Avoidance Patterns

We will analyze subtle maladaptive patterns such as "Pseudo-productivity" (prioritizing low-stakes tasks to avoid high-stakes goals) and "Project Juggling" (novelty-seeking as an escape from sustained attention requirements). We emphasize that for systems to be sustainable, they must remain visible, simple, and physically salient.

Conclusion: By incorporating standardized monitoring via the ASRS (Symptom Tracking) and the WFIRS-P (Functional Impairment Scale), CBT transforms clinical gains into "second nature" functionality. The integration of a support person (optional) further reinforces the external scaffolding necessary for long-term success.

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Keywords: Adult ADHD, Cognitive Behavioral Therapy, Therapist Guide
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0430 - Riding the Wave: The Impact of Menstrual Cycles on Cognition and Emotion in Girls with ADHD

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Attention-Deficit/Hyperactivity Disorder (ADHD) has long been conceptualized through a male-centric lens and historically viewed as a static neurodevelopmental trait [1]. However, emerging evidence—ranging from recent systematic reviews to qualitative reports of patients' lived experiences—highlights that symptom severity and medication efficacy fluctuate significantly alongside *the waves* of ovarian steroid hormones, estradiol (E2) and progesterone (P4) [2, 3].

During the follicular phase, specifically peaking at the pre-ovulatory stage, circulating 17β-estradiol levels are at their highest. Estrogen functions as a neuroprotective agent that enhances dopaminergic transmission in critical brain regions, such as the prefrontal cortex and striatum. Mechanisms underlying this effect include the stimulation of dopamine synthesis and the inhibition of dopamine reuptake, which effectively increases synaptic dopamine availability. Consequently, this high-estrogen environment is associated with improved executive functioning, working memory, and processing speed, potentially masking or ameliorating ADHD symptoms during this window. Following ovulation, the luteal phase is characterized by a rise in progesterone. Progesterone often exerts inhibitory effects via the GABAergic system, which can antagonize the excitatory and cognitive-enhancing benefits of estrogen [4].

Conversely, the late luteal and premenstrual phases are marked by a rapid withdrawal of both estradiol and progesterone. For females with ADHD, who inherently present with baseline catecholaminergic dysfunction, this abrupt withdrawal of estradiol acts as a critical second hit [4]. This transient state of reduced dopaminergic tone may destabilize frontostriatal networks, potentially impairing top-down inhibitory control. Simultaneously, the rapid fluctuation of progesterone and its neuroactive metabolites alters $GABA_A$ receptor conformation and sensitivity. These neurosteroid-mediated changes diminish the capacity to regulate environmental stressors, amplifying emotional reactivity and internalizing symptoms. In the context of a vulnerable ADHD brain, this dual mechanism—dopaminergic withdrawal compounded by neurosteroid-induced dysregulation—disrupts the balance between excitatory and inhibitory signaling [2].

These neurobiological shifts are particularly impactful during adolescence, as menarche introduces dramatic hormonal fluctuations while top-down cognitive control networks are still maturing. This elevates the risk for maladaptive coping and functional impairment in academic and social settings during these critical years. Recognizing the menstrual cycle is essential for ADHD management in females. Clinicians should incorporate cycle tracking into assessments to support diagnostic accuracy and improve long-term outcomes. Furthermore, treatment strategies should consider titrating interventions to specific cycle phases, particularly during periovulatory and luteal windows of risk.

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Keywords: Adolescent Psychiatry, Attention Deficit Disorder with Hyperactivity, Estradiol, Female, Menstrual Cycle, Progesterone

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0431 - Investigation of Psychosocial Challenges and Coping Methods Experienced By Children Diagnosed With ADHD

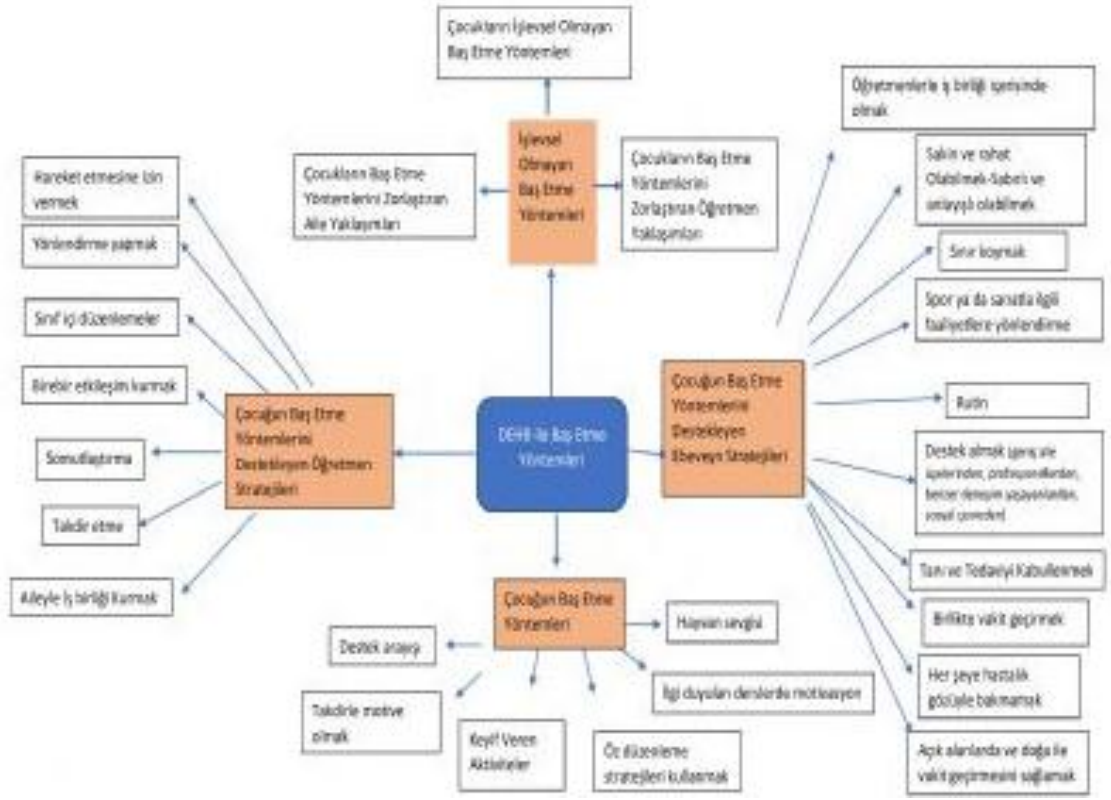
Merve Gülçin Tutlu Temizyürek, Saziye Senem Başgül

Psychology, Hasan Kalyoncu University, Gaziantep/Türkiye

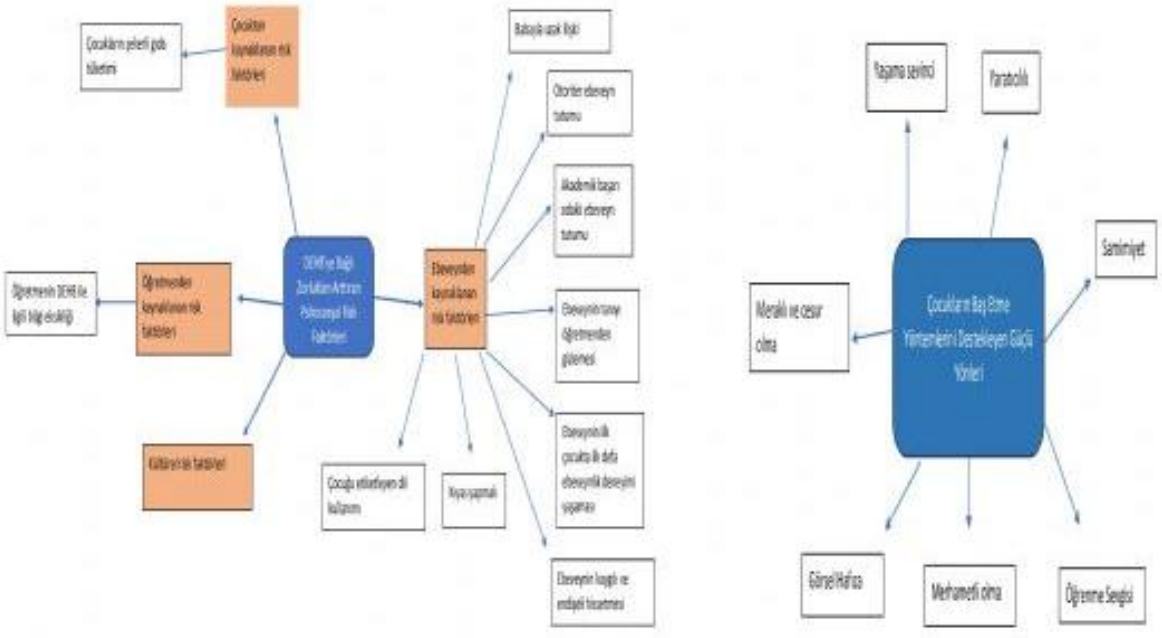
This study examined the psychosocial challenges and coping strategies experienced by children aged 7-11 diagnosed with ADHD from the perspectives of children, parents, and teachers. The study was conducted using a phenomenological design, one of the qualitative research methods, with semi-structured in-depth interviews conducted with a total of 21 participants diagnosed with ADHD: 6 children, 5 teachers who have these students, and 10 parents of these children. During the data collection process, semi-structured interview forms were used for parents and teachers, while drawing and storytelling techniques were employed alongside interviews for children. MAXQDA Analytics Pro (24.4.1) program was used for data analysis. Through thematic analysis, 4 themes and a total of 23 categories belonging to these themes were identified. The theme "Challenges Caused by ADHD" encompasses academic, emotional, cognitive, behavioral and social difficulties, challenges related to the diagnosis-treatment process, and difficulties related to the education system. The theme "Coping Methods with ADHD" includes children's coping methods, parent and teacher strategies supporting children's coping methods, and dysfunctional coping methods. The theme "Children's Strengths Supporting Their Coping Methods" contains categories of joy of living, visual memory, being curious and brave, being compassionate, sincerity, creativity, and love of learning. The theme "Psychosocial Risk Factors Increasing ADHD-Related Difficulties" encompasses risk factors stemming from children, parents, teachers, as well as environmental and cultural risk factors. The study revealed that children with ADHD face challenges not only with attention deficit and hyperactivity, but across a wide spectrum from behavioral control to academic achievement, from social relationships to emotional regulation. It was observed that families encountered misguidance during the diagnosis process, experienced side effects in medication treatment, and that the current education system created additional difficulties for these children. However, it was found that children developed their own unique coping strategies such as drawing, spending time in nature, doing sports, and love for animals. Family support, spending time together, establishing routines, and discovering children's strengths are important. These findings indicate that children with ADHD require not only symptom management but multifaceted psychosocial support. The research reveals that each child has unique abilities and that when these strengths are recognized, children can better cope with difficulties. Collaborative work among families, teachers, and specialists can significantly improve children's quality of life. These results are important for developing support programs for children with ADHD, reconsidering educational policies, and increasing societal understanding.

Keywords: Attention Deficit Hyperactivity Disorder, Phenomenological Design, Challenge, Coping

DEHB ile Baş Etme Yöntemleri



Psikososyal Risk Faktörleri-Baş Etme Yöntemlerini Destekleyen Güçlü Yönler



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0432 - Social Media Use in Children and Adolescents: Emotional, Behavioral, and Clinical Reflections

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Social media has become increasingly central to the developmental experiences of children and adolescents and now constitutes a significant determinant of mental health. Although digital platforms offer opportunities for social connection, peer communication, and self-expression, they also introduce emotional and behavioral risks that are being observed with growing frequency in clinical practice. This presentation aims to provide a comprehensive overview of how social media use influences affect regulation, behavioral patterns, and academic functioning in young people, integrating current scientific evidence with clinical observations.

Recent research highlights that not only the quantity but also the quality of social media use plays a critical role in psychological outcomes. Excessive, unregulated, or problematic use has been associated with depressive symptoms, anxiety, emotional instability, and reduced self-esteem. Mechanisms such as social comparison, fear of missing out (FOMO), and exposure to idealized digital identities contribute to these patterns. Young people with pre-existing vulnerabilities—such as ADHD, emotional dysregulation, or interpersonal sensitivity—may be particularly at risk, as social media interactions can intensify symptom expression and functional impairment.

Beyond emotional consequences, social media use also affects cognitive and behavioral functioning. Nighttime screen use, blue light exposure, and anticipatory engagement disrupt sleep quality, leading to attention difficulties, memory problems, and poorer academic performance. Rapid content cycles, reward-based algorithms, and multitasking behaviors may further exacerbate symptoms of inattention and impulsivity, particularly in youth with ADHD. Clinical examples will illustrate how these patterns manifest in daily functioning, including emotional outbursts following screen restriction, anxiety driven by online peer interactions, and fluctuations in attention associated with continuous digital stimulation.

Despite these risks, social media use is not inherently pathological. Protective factors—including parental monitoring, open communication, strong emotional regulation skills, and supportive peer relationships—can buffer negative outcomes. Evidence-based guidelines underscore the importance of structured screen routines, balanced offline activities, and fostering digital literacy within families. The presentation will offer practical recommendations for incorporating digital behavior into psychiatric evaluations, focusing on screen habits, content exposure, peer dynamics, and sleep–wake patterns.

Overall, this presentation aims to provide a holistic, current, and clinically applicable framework for understanding the impact of social media use on child and adolescent mental health. The goal is not to pathologize digital engagement but to guide clinicians and families toward safe, adaptive, and developmentally appropriate media practices.

Keywords: Affect Regulation, ADHD and Vulnerability, Child and Adolescent Psychiatry, Emotional and Behavioral Outcomes, Sleep and Cognitive Function, Social Media Use

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0434 - Mechanisms of Antipsychotic-Induced Weight Gain (AIWG)

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Antipsychotic-induced weight gain (AIWG) represents a significant clinical complication in the pharmacological management of psychiatric disorders, frequently associated with reduced treatment adherence and increased long-term cardiometabolic morbidity. Although the metabolic risk profile varies substantially among second-generation antipsychotics—with clozapine and olanzapine demonstrating the highest propensity for weight gain—the underlying physiological architecture involves a complex integration of multiple biological systems. Current evidence indicates that AIWG is not solely a product of increased caloric intake but results from a coordinated disruption of central appetite regulation, peripheral metabolic signaling, genetic vulnerability, and alterations in the gut microbiome. The primary mechanism of AIWG involves the localized disruption of neurotransmitter signaling within the hypothalamus. High-affinity antagonism of histamine H1 and serotonin 5-HT2C receptors serves as a robust pharmacological predictor of weight gain. These interactions activate the AMP-activated protein kinase (AMPK) and neuropeptide Y (NPY) pathways, which collectively enhance orexigenic signaling, promote hyperphagia, and diminish postprandial satiety. Furthermore, dopamine D2 receptor blockade modulates the mesolimbic reward circuitry, which may increase hedonic feeding behavior and influence resting energy expenditure. Additionally, muscarinic M3 receptor antagonism has been identified as a factor in the disruption of cholinergic-mediated insulin and glucose regulation, potentially increasing the risk of type 2 diabetes mellitus independently of changes in total adiposity. Beyond receptor-mediated central effects, antipsychotics exert significant influence on the systemic endocrine profile. Treatment is often associated with a rapid elevation in circulating leptin levels, subsequently leading to a state of leptin resistance that compromises the hormone's anorexigenic function. Concurrently, dysregulation in the secretion of ghrelin, adiponectin, and glucagon-like peptide-1 (GLP-1) contributes to the development of metabolic syndrome. Transcriptomic analyses further suggest that these agents upregulate essential genes involved in lipid biosynthesis, particularly through the activation of sterol regulatory element-binding transcription factors (SREBF). Clinical observations confirm that dyslipidemia and dysglycemia can manifest through direct peripheral actions on hepatic and adipose tissues, occasionally preceding measurable changes in body mass index. Individual susceptibility to AIWG is significantly moderated by polygenic factors. Single nucleotide polymorphisms (SNPs) in candidate genes, including HTR2C, DRD2, MC4R, and ADRA2A, have been identified as primary risk markers, with increased vulnerability observed in antipsychotic-naive and pediatric populations. Moreover, emerging research delineates the role of the gut microbiota in metabolic outcomes. Antipsychotic administration can induce significant dysbiosis, altering microbial metabolites that regulate energy harvesting, intestinal permeability, and systemic low-grade inflammation. In conclusion, AIWG results from a convergence of receptor-mediated hypothalamic alterations, endocrine dysregulation, and direct cellular effects on lipid metabolism. A comprehensive understanding of these integrated pathways is critical for the development of targeted pharmacological interventions and the mitigation of the metabolic burden associated with antipsychotic therapy. Continued research into genetic markers and microbiome interactions is necessary to facilitate personalized monitoring and improved clinical outcomes in patients requiring long-term treatment.

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Keywords: Antipsychotics, weight gain, histamine H1 receptor, serotonin 5-HT2C receptor, metabolic syndrome, leptin resistance
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0435 - The Role of GluN3A Subunit in Regulation of Stress-coping Strategies

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Background and Objective: The hippocampus is a highly complex structure, defined by profound structural, functional, and molecular heterogeneity. The non-classical N-methyl-D-aspartate receptor subunit GluN3A, is enriched in the hippocampal CA1 subregion and has a poorly understood functional role, especially in stress-related behavioral regulation.

Methods: Here, we employed chronic social defeat stress (CSDS) and GluN3A knockout models, alongside behavioral, molecular, genetic, pharmacological, and circuit-level analyses to investigate the role of hippocampal GluN3A in stress coping.

Results: We found that CSDS selectively reduced GluN3A expression in the intermediate CA1 (CA1i), and GluN3A loss, either stress-induced or genetic, promoted passive coping, whereas CA1i-specific GluN3A overexpression reversed this phenotype. Consistent with its enrichment in pyramidal neurons, c-Fos staining and fiber photometry revealed that GluN3A is required for CA1i pyramidal neuronal activation during coping. Further chemogenetic manipulations demonstrated that activating CA1i neurons facilitated active coping, while inhibition induced passivity. Moreover, local D-serine administration rapidly shifted coping from passive to active via GluN3A-dependent activation of CA1i neurons. Circuit tracing showed that CA1i neurons project broadly, and GluN3A overexpression selectively enhanced downstream activity in CA3 and the infralimbic cortex. Conclusions: Together, our findings identify GluN3A as a key molecule in the stress-coping of CA1i, and reveal a GluN3A-dependent mechanism underlying D-serine-induced stress resilience.

Keywords: NMDA receptor, GluN3A subunit, Hippocampus, Stress Coping, D-serine

0436 - Stigma in Mental Health: Insights from MENA

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Stigma towards mental illness is a global phenomenon. Various definitions of stigma are used, all reflecting ignorance (poor knowledge), prejudice (poor attitude) and discrimination (poor behavior) associated with mental illness and people who have it. Often driven by cultural and religious misconceptions, it's a major barrier to seeking help, resulting in underutilization of mental health services, treatment gaps and poor outcomes. A study across 16 Arab countries revealed that one third of respondents had poor knowledge about mental health and a similar percentage had negative help-seeking attitude. Stigma isn't among the general public only; literature evidence shows misconceptions and prejudices occur among healthcare providers as well as people with lived experience, referred to as internalized stigma. A group of Turkish authors argue mental health literacy may be a neglected competency in medical training, where knowledge is earned through years of experience. The implications for patients are significant and expand beyond mental health to inequity in their physical health management with diagnostic overshadowing.

The Middle East, despite a historical background of compassionate care towards people with mental illness and establishing some of the first mental health facilities in the world, now has deeply rooted stigmatizing and stereotypical attitudes towards mental illness. Concepts such as shame, weakness, religious non-adherence and divine punishment prevail, and concerns about confidentiality, employment and marriage prospects deter people from accessing mental health services when needed. Beliefs in the supernatural, evil eye and witchcraft make traditional healers often the first choice for help. Half of university students in Qatar believe mental illness is a punishment from God.

Initiatives to raise public awareness have gained momentum over the years with global activities aimed at mental health education and prevention. Anti-stigma campaigns provide better information, call for equity in mental health, enhancing access to care, inclusion and recovery, among others. However, evidence suggests such campaigns may enhance knowledge but have little effect on changing behaviors. An online training program in mental health for family physicians in Türkiye resulted in significant enhancement in mental health knowledge, but a modest effect on beliefs and attitudes. Contact with people with lived experience seems to have the most robust evidence across major domains of better knowledge, attitude and behavior. Celebrities speaking openly about their own mental health experience utilize their wide platforms to fight stigma, normalize conversations and encourage help-seeking behavior. Several Arab and Turkish celebrities were forthcoming with their experiences; interestingly, mostly female. Successful campaigns need to endorse community engagement, including service users, and address the needs of the target population, while utilizing the benefits and wider reach of technology. Enabling policies and legislation are crucial for prevention and promotion efforts. For the unique cultural platform of the Middle East, collaborating with religious healers is expected to yield more positive outcomes.

Keywords: Stigma, mental health, awareness, Middle East, Türkiye

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0437 - Agranulocytosis: Is the Risk Really Great?

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Treatment-resistant schizophrenia (TRS), affecting approximately 30% of individuals diagnosed with schizophrenia, leads to substantial functional impairment and imposes a considerable burden on both caregivers and health care systems. Clozapine remains the only treatment approved by the US Food and Drug Administration (FDA) and licensed by most health authorities for the management of TRS [1]. Despite its well-established clinical superiority—demonstrated by reductions in suicide risk, relapse, and rehospitalization—clozapine continues to be underutilized worldwide [2]. One of the principal barriers to both initiation and maintenance of clozapine treatment is considered to be the requirement for lifelong hematological monitoring. The historical origins of this concern are largely attributed to reports of agranulocytosis-related deaths in Finland in 1975. Although clozapine was reintroduced into clinical use following the landmark study by Kane et al. in 1988 demonstrating its efficacy in TRS, persistent safety concerns have continued to limit prescribing, thereby restricting patient access to effective treatment [1]. Current regulations in many countries follow European Medicines Agency (EMA) recommendations, requiring weekly monitoring of white blood cell (WBC) counts and absolute neutrophil counts (ANC) during the first 18 weeks of treatment, followed by monthly monitoring thereafter [2]. In 2015, the FDA revised clozapine monitoring criteria by lowering the treatment discontinuation threshold for ANC from 1500/mm³ to 1000/mm³ and to 500/mm³ in patients with benign ethnic neutropenia (BEN) [1]. Nevertheless, meta-analytic evidence indicates that severe neutropenia, commonly defined as clozapine-associated agranulocytosis, occurs at a rate of approximately 0.9%. This adverse effect appears acute and time-limited, supporting reduced monitoring frequency. In this context, the European Clozapine Task Force proposed in December 2024 the relaxation of hematological monitoring frequency after 12 and 24 months of treatment, lowering ANC thresholds for treatment initiation and discontinuation, adopting differentiated criteria for BEN, and revising monitoring procedures following treatment interruption, with the aim of facilitating broader implementation of clozapine treatment [2]. In Türkiye, updated clozapine monitoring recommendations were issued to physicians on January 15, 2026, by the Pharmacovigilance and Controlled Substances Department of the Turkish Medicines and Medical Devices Agency. This update eliminated the requirement for WBC monitoring and recommended ANC-based surveillance alone, harmonizing treatment initiation and continuation thresholds with definitions of mild (ANC: 1000–1500/mm³), moderate (ANC: 500–999/mm³), and severe neutropenia (ANC <500/mm³). Clozapine initiation was recommended only in patients with ANC >=1500/mm³ and in individuals with confirmed BEN when ANC >=1000/mm³. Furthermore, ANC monitoring was advised weekly during the first 18 weeks, monthly during the subsequent 34 weeks, every 12 weeks after the first year in the absence of prior neutropenia, and annually after two years without neutropenic events [3]. In conclusion, restricted access to clozapine—despite its status as a first-line treatment for patients with TRS—due to concerns regarding agranulocytosis represents a significant missed therapeutic opportunity for both patients and health care systems. Regulatory revisions implemented in Türkiye, in parallel with updated European and North American clozapine prescribing and hematological monitoring frameworks, represent an important step toward overcoming this major public health challenge.

Keywords: Agranulocytosis, clozapine, hematological monitoring, neutropenia, treatment-resistant schizophrenia

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0438 - PTSD and Complex PTSD in Children and Adolescents: Treatment Approaches

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Children and adolescents frequently encounter potentially traumatic events (PTEs), though only a small number of those exposed develop Post-Traumatic Stress Disorder (PTSD). Exposure to multiple and prolonged PTEs may result in additional symptoms including emotion dysregulation, negative self-concept, and interpersonal difficulties; these symptoms are conceptualized as Complex PTSD (CPTSD) in ICD-11. Recent research has shown that CPTSD is more prevalent than PTSD in child and adolescent psychiatric settings (Sachser et al., 2017). While a substantial body of research exists on PTSD treatment in children and adolescents, studies investigating treatment approaches specifically for CPTSD in this age group remain limited. Available evidence suggests that Trauma-Focused Cognitive Behavioral Therapy (TF-CBT) produces comparable effects for both PTSD and CPTSD. Treatment protocols for CPTSD are generally structured around two phases: a stabilization phase and a trauma processing phase. However, this sequential approach has been criticized for potentially delaying access to effective trauma-focused treatment (De Jongh et al., 2016; Van Vliet et al., 2024). Notably, insufficient assessment of trauma burden and comorbid psychopathology represents a significant methodological limitation across existing studies.

From a clinical perspective, given the high likelihood of multiple trauma exposure and co-occurring neglect histories in this population, we argue that the stabilization phase is an essential component of treatment — one in which children develop emotion regulation and interpersonal skills that are necessary not only for engaging with trauma processing but for their broader functioning in daily life. Furthermore, we suggest that children with CPTSD have a particular need for these skills, and that targeted skill-building may serve as an important scaffold for addressing the disturbances in self-organization that are central to the disorder. Existing studies have predominantly focused on symptom reduction as the primary outcome, with insufficient attention to changes in functional skills and quality of life, and have reported considerable dropout rates. In this presentation, evidence-based treatment approaches will be discussed in light of these limitations and our clinical experience.

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Keywords: Complex PTSD, PTSD, children and adolescents, treatment

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0439 - A Case of Behavioral Addiction Transformation Following Bariatric Surgery: From Binge-Eating Disorder to Severe Gambling Disorder

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Introduction

Addiction transformation refers to the emergence or escalation of a new addictive behavior following remission or restriction of a previous addiction. In the context of bariatric surgery, postoperative increases in substance use disorders have been documented; however, behavioral addictions such as Gambling Disorder are less frequently reported. Alterations in reward processing, reduced food-related reinforcement, and pre-existing impulse-control vulnerability may contribute to shifts in reinforcement-seeking behavior after surgery. We present a longitudinal case of Binge-Eating Disorder evolving into severe Gambling Disorder following sleeve gastrectomy, highlighting clinical course and psychopharmacological management.

Case Presentation

A 30-year-old married female nurse first presented to our outpatient clinic in April 2021 with compulsive eating behaviors consistent with Binge-Eating Disorder since adolescence. Treatment included structured psychoeducation, stress management interventions, boundary-setting skills, food diary monitoring, and pharmacotherapy with fluoxetine 20 mg/day and naltrexone 50 mg/day. A significant reduction in binge-eating episodes was observed during follow-up; however, medication adherence was inconsistent, and the patient discontinued follow-up in early 2022.

She underwent sleeve gastrectomy in late 2024 at an external center without psychiatric reassessment. Preoperative body mass index was 28 kg/m², which decreased to 24 kg/m² postoperatively, accompanied by a marked reduction in binge-eating symptoms.

Although she had previously engaged in occasional online slot gambling without loss of control, approximately one year after surgery her gambling behavior escalated to a severe pattern. Within two months, she reported daily online gambling and accumulated approximately 1.5 million Turkish Lira in debt through bank loans and informal borrowing. The clinical presentation met DSM-5 criteria for severe Gambling Disorder.

Psychiatric evaluation revealed impulsivity, affective lability, unstable interpersonal patterns, and concealment behaviors. Gambling escalation was temporally associated with family-related stressors. Interpersonal conflicts appeared to trigger feelings of rejection and worthlessness preceding loss-of-control behaviors. Rumination about financial losses, sleep disturbance, and depressive symptoms were also present.

Pharmacological management included sertraline 50 mg/day, naltrexone 50 mg/day, and N-acetylcysteine augmentation, combined with structured behavioral interventions. During recent follow-up, gambling frequency decreased, financial protective measures were implemented, and insight into relapse triggers improved, although vulnerability persisted.

Conclusion

This case illustrates a behavioral addiction transformation from Binge-Eating Disorder to severe Gambling Disorder following bariatric surgery in a patient with pre-existing impulse-control vulnerability. The temporal association suggests a shift in reward-seeking behavior after restriction of food-related reinforcement. Partial clinical response to opioid antagonism and glutamatergic modulation supports the potential involvement of opioid and reward-circuit pathways in behavioral addictions. Early psychiatric screening and longitudinal follow-up before and after bariatric surgery may be essential to prevent delayed recognition of post-surgical addiction transformation.

Keywords: Bariatric Surgery, Binge-Eating Disorder, Gambling Disorder, Impulse Control Disorders, Naltrexone

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0440 - Hidden in the Classroom: Spesific Learning Disorders in the Female Population

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Although Specific Learning Disorder (SLD) is one of the most frequently diagnosed neurodevelopmental conditions in childhood, a persistent gender disparity characterizes identification and referral processes. Clinical data often report male-to-female ratios ranging from 2:1 to 4:1; however, large-scale epidemiological and school-based screening studies suggest that actual prevalence rates are considerably more balanced. When universal screening measures are implemented instead of teacher-based referrals, gender differences substantially decrease. This discrepancy highlights a significant "referral bias," whereby identification systems disproportionately capture boys exhibiting disruptive, externalizing behaviors. Educational risk profiles have historically aligned with the male phenotype—hyperactivity, impulsivity, and classroom disruption—while girls with subtler presentations remain under-recognized.Current research indicates that girls with SLD (including Dyslexia, Dyscalculia, and Dysgraphia) are more likely to present with internalizing patterns. Rather than disrupting classroom order, they may appear compliant, quiet, socially adapted, or perfectionistic, often accompanied by anxiety or psychosomatic complaints. The literature increasingly describes this pattern as the "camouflage effect" or masking. Camouflaging refers to compensatory strategies that obscure academic deficits. Girls frequently rely on memorization, contextual guessing, peer support, excessive time investment in homework, and overpreparation to maintain adequate performance. Strong verbal abilities or higher general intelligence may further conceal weaknesses in phonological processing, working memory, or processing speed. The intersection of SLD and Attention-Deficit/Hyperactivity Disorder (ADHD), particularly the inattentive presentation, further complicates detection. Inattentive symptoms—such as distractibility or mental wandering—lack overt behavioral disruption and therefore attract less teacher attention. Consequently, girls with comorbid SLD and inattentive ADHD may remain unidentified until academic demands intensify during adolescence. Recent studies support the "severity threshold hypothesis," demonstrating that girls who ultimately receive an SLD diagnosis often exhibit more severe academic underachievement than diagnosed boys. In other words, diagnostic recognition frequently occurs only when compensatory mechanisms are no longer sustainable. The psychosocial consequences of delayed identification are significant. Longitudinal findings associate undiagnosed learning difficulties in girls with heightened risks of chronic anxiety, depressive symptoms, academic burnout, low self-esteem, and learned helplessness. Because many girls internalize academic struggles as personal inadequacy rather than neurodevelopmental differences, secondary emotional difficulties may become more impairing than the primary learning disorder itself. These findings underscore the necessity of gender-sensitive assessment frameworks. Sole reliance on grades or behavioral disruption is insufficient for equitable identification. Comprehensive evaluation protocols should incorporate task completion time, cognitive effort, emotional distress during learning tasks, and functional fatigue. Universal screening models and increased awareness of internalizing presentations are essential to reducing diagnostic disparities. Addressing masked difficulties in girls is not merely a refinement of clinical practice; it is a prerequisite for ensuring educational equity and protecting long-term mental health outcomes.

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Keywords: Comorbidity, diagnostic delay, gender differences, masking (camouflage), psychopathology, specific learning disorder

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0441 - ADHD Medications and the Placebo Effect

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Attention-Deficit/Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder characterized by persistent symptoms of inattention, hyperactivity, and impulsivity that significantly affect academic, occupational, and social functioning. Pharmacological treatments—particularly stimulant medications such as methylphenidate and amphetamine derivatives—are widely considered first-line interventions due to their strong evidence base for symptom reduction. Meta-analytic findings consistently demonstrate that ADHD medications produce moderate to large improvements in core symptoms across children, adolescents, and adults, supporting their clinical efficacy compared with placebo conditions (1,3). However, the presence of measurable placebo responses in ADHD clinical trials raises important questions regarding the relative contributions of pharmacological mechanisms and psychological factors.

Placebo effects refer to beneficial outcomes resulting from patients' expectations, beliefs, and the therapeutic context rather than from the pharmacologically active components of treatment. In psychiatric research, placebo responses can significantly influence trial outcomes, particularly in conditions where symptom assessment relies on subjective measures (4,6). Studies examining ADHD trials report notable placebo response rates, indicating that participants may experience symptom improvements even when receiving inactive treatments (2,5). These responses may be influenced by expectancy effects, increased clinical monitoring, and the structured environment of clinical trials.

Neurobiological research suggests that placebo responses involve measurable brain processes. Expectations of improvement can activate reward-related neural pathways, including dopaminergic systems implicated in both ADHD pathology and stimulant medication mechanisms (7). This overlap suggests that placebo-induced expectations may partially mimic neurochemical processes targeted by ADHD medications. Despite the presence of placebo responses, meta-analyses consistently show that active ADHD medications significantly outperform placebo in reducing core symptoms and improving functioning (1,3). Understanding placebo mechanisms may help refine clinical trial design and enhance treatment outcomes through improved patient communication and therapeutic context.

In conclusion, ADHD medications demonstrate strong efficacy in symptom management, although placebo effects represent a meaningful component of observed improvements. Integrating insights from placebo research may contribute to more comprehensive treatment approaches that combine pharmacological and contextual factors.

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Keywords: ADHD, clinical trials, placebo effect, stimulant medications, treatment efficacy
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0442 - Repetitive Behaviors: Obsessive–Compulsive Disorder or Autism Spectrum Disorder?

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Repetitive and restrictive behaviors (RRBs) and sensory phenomena (SP) are commonly observed across several neurodevelopmental and neuropsychiatric conditions, particularly autism spectrum disorder (ASD), obsessive–compulsive disorder (OCD), and Tourette syndrome (TS). Despite their apparent phenomenological similarity, these behaviors may arise from distinct psychological mechanisms and neurobiological processes. This panel aims to explore the phenomenology of repetitive behaviors across neurodevelopmental disorders and to discuss how clinicians can differentiate similar-looking behaviors that may reflect different underlying mechanisms. Particular emphasis will be placed on the dimensional and transdiagnostic nature of repetitive behaviors, highlighting both shared features and disorder-specific characteristics that are relevant for clinical assessment and treatment. From a developmental standpoint, ritualistic behaviors are not intrinsically pathological. Repetitive patterns are frequently observed in normal childhood development and may fulfill adaptive roles, including enhancing predictability, emotional regulation, and a sense of control. When these behaviors become rigid, persistent, and linked to considerable distress or impairment, they may signify underlying psychopathology. Neurobiological models suggest that repetitive behaviors and sensory phenomena are associated with disruptions in corticostriatal circuits and basal ganglia pathways that regulate motor control, cognitive flexibility, and goal-directed behavior. Dysfunction within these systems may contribute to difficulties in inhibiting repetitive actions, shifting behavioral sets, and regulating sensory input.

A key focus of the panel will be the phenomenological differences between repetitive behaviors observed in OCD and ASD. In OCD, compulsions are typically ego-dystonic and emerge within a characteristic obsessional sequence in which intrusive thoughts generate anxiety that is subsequently reduced through ritualistic behaviors. In contrast, repetitive behaviors in ASD are often ego-syntonic and closely linked to a need for routine, sameness, or sensory regulation. In these cases, distress usually arises when the behavior cannot be completed rather than from a preceding intrusive thought.

Sensory phenomena—including “just-right” perceptions, feelings of incompleteness, tactile discomfort, or internal tension—have been reported in a large proportion of individuals with these disorders and may function as internal triggers for repetitive behaviors. Emerging research suggests that while ASD and OCD symptoms tend to cluster into distinct domains, sensory experiences may serve as a bridge between them. For example, unusual sensory interests in ASD may evolve into behaviors that resemble compulsive checking or ordering. This overlap creates important diagnostic challenges in clinical practice, particularly when differentiating anxiety-driven compulsions from routine-based or sensory-driven behaviors.

In this panel, recent empirical findings and clinical perspectives will be integrated to provide a clearer conceptual framework for understanding repetitive behaviors across neurodevelopmental disorders. A dimensional and mechanism-focused approach may improve differential diagnosis and guide more targeted interventions for children and adolescents presenting with repetitive behaviors. In addition, a brief clinical case vignette will be presented to illustrate the diagnostic challenges encountered in distinguishing obsessive–compulsive symptoms from autism-related repetitive behaviors and to highlight key phenomenological clues that may support more accurate differential diagnosis in complex presentations.

Keywords: Autism spectrum disorder, differential diagnosis, neurodevelopmental disorders, obsessive–compulsive disorder, repetitive behaviors; sensory phenomena

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0443 - Is Early Diagnosis Possible With Digital Health Technologies in Individuals at Clinically High Risk of Developing Psychosis?

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Digital health technologies involve the unobtrusive, background collection of an individual's social interaction and movement (behavioral) data, as well as sleep and heart rate (biological) data, via smartphones and wearable devices. By capturing micro-level changes that may go unnoticed during clinical examinations, this method essentially acts as an early warning system. Early diagnosis of psychosis is quite challenging with traditional clinical methods. The primary reason for this is that prodromal symptoms are generally vague, and patients rarely seek professional help during this phase. The fundamental digital indicators facilitating early diagnosis include social isolation, psychomotor retardation, autonomic nervous system dysfunction, and natural language processing algorithms. Social isolation, indicated by sudden drops in the number of calls and messages, along with an increase in time spent at home derived from GPS data, serves as the first sign of the onset of negative symptoms. Psychomotor retardation, reflected in keyboard typing and touchscreen tapping speeds, indicates an impairment in the prefrontal cortex, the center of executive functions. Autonomic nervous system dysfunction, evidenced by a decrease in heart rate variability and sleep efficiency measured by smartwatches, proves that the brain is in a continuous state of alarm. Natural language processing algorithms detect a decrease in semantic density within social media posts or voice recordings. Thus, they can identify thought disorders before they progress to a clinical stage. Digital diagnostic methods carry significant potential. However, transition rates to psychosis in the clinical risk group appear to be on a downward trend. This situation raises the risk of false positives and the accompanying issue of stigmatization. Therefore, digital phenotyping should be positioned as a clinical decision support system, rather than a replacement for the physician. These individuals should be regularly monitored through structured interviews for up to three years, as long as symptoms persist.

Keywords: Digital Technology, Early Diagnosis, Psychotic Disorders

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0445 - Efficacy and Safety of Antiepileptic Drugs in the Management of Behavioral Issues in Autism Spectrum Disorders

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Introduction

Autism spectrum disorder (ASD) is a heterogeneous neurodevelopmental disorder characterized by persistent deficits in social communication and restricted, repetitive patterns of behavior. In addition to its core symptoms, ASD is frequently accompanied by neurological and psychiatric comorbidities. Among these conditions, epilepsy represents one of the most common and clinically significant comorbidities. Epidemiological studies suggest that approximately 20–30% of individuals with ASD develop epilepsy during their lifetime, a prevalence considerably higher than that observed in the general population (1). The co-occurrence of ASD and epilepsy has been attributed to shared neurobiological mechanisms, including disruptions in cortical connectivity, genetic vulnerabilities, and imbalances between excitatory glutamatergic and inhibitory gamma-aminobutyric acid neurotransmission (2). The management of epilepsy in individuals with ASD presents important clinical challenges. Anticonvulsants constitute the main pharmacological treatment for seizure control in this population. Several medications are commonly prescribed, including valproate, lamotrigine, levetiracetam, and topiramate. While these agents are primarily used for the treatment of epileptic seizures, their potential effects on behavioral symptoms associated with ASD have also attracted clinical interest. For example, valproate has been reported to reduce irritability and aggression in some individuals with ASD, although concerns regarding metabolic effects and teratogenicity limit its use in certain populations (3). Lamotrigine is often considered to have a relatively favorable cognitive profile, whereas levetiracetam is widely used for seizure control but may occasionally exacerbate behavioral symptoms such as irritability or agitation. Topiramate has also been investigated as an adjunctive treatment and may provide benefits for some behavioral symptoms, although cognitive adverse effects may restrict its clinical utility. Despite increasing clinical interest, evidence supporting the use of anticonvulsants for the treatment of behavioral symptoms in ASD remains limited and heterogeneous. Systematic reviews suggest that while antiepileptic medications are effective in controlling seizures in individuals with ASD and comorbid epilepsy, their benefits for core or associated behavioral symptoms of ASD are inconsistent and not well established (4). Furthermore, electroencephalographic abnormalities are frequently observed in individuals with ASD, with some studies reporting abnormal findings in up to 60% of cases. This overlap supports the hypothesis that ASD and epilepsy may share underlying neurobiological mechanisms, including synaptic dysfunction and imbalance between excitatory and inhibitory neural networks (5).

Conclusion

Anticonvulsants play a central role in the treatment of epilepsy in individuals with autism spectrum disorder. However, current evidence suggests that their use should primarily focus on seizure management rather than the treatment of core ASD symptoms. While certain antiepileptic medications may have beneficial effects on irritability or mood dysregulation in selected cases, robust evidence supporting their routine use for behavioral symptoms is still lacking. Clinicians should therefore adopt an individualized treatment approach that carefully balances seizure control, potential psychiatric effects, and adverse drug reactions. Future research is needed to clarify the neurobiological mechanisms linking ASD and epilepsy and to determine whether specific antiepileptic agents may have targeted benefits in particular ASD subgroups.

Keywords: Anticonvulsants, autism spectrum disorder, epilepsy, neurodevelopmental disorders, psychopharmacology

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0446 - Executive Dysfunction and Emotional Dysregulation: A Holistic Approach to Differential Diagnosis

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Executive dysfunction and emotional dysregulation are common clinical features encountered across multiple psychiatric conditions in children and adolescents. Difficulties in attention regulation, cognitive flexibility, planning, and inhibitory control often coexist with problems in emotional modulation, irritability, and impulsive reactions. These overlapping manifestations frequently blur diagnostic boundaries, particularly among attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD), disruptive behavior disorders, mood disorders, and trauma-related conditions.

In clinical practice, executive dysfunction and emotional dysregulation are sometimes interpreted as disorder-specific symptoms, although increasing evidence suggests that they may represent transdiagnostic processes reflecting shared neurodevelopmental and neurobiological mechanisms. Relying solely on categorical diagnostic frameworks may therefore lead to misinterpretation of symptom clusters and contribute to diagnostic uncertainty.

This presentation discusses how executive functioning and emotion regulation difficulties intersect across diagnostic categories and proposes a holistic approach to differential diagnosis. Developmental history, contextual factors, comorbidities, and functional impairment will be emphasized as key elements guiding clinical assessment. In addition, current conceptual models highlighting dimensional and transdiagnostic perspectives will be reviewed.

Recognizing the interplay between executive dysfunction and emotional dysregulation may help clinicians move beyond rigid diagnostic boundaries and support more individualized and developmentally informed clinical formulations in child and adolescent psychiatry.

Keywords: ADHD, Child and adolescent psychiatry, Differential diagnosis, Emotional dysregulation, Executive dysfunction, Transdiagnostic processes

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0447 - Challenges in Women's Mental Health Sexual Dysfunction and Management in Women

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Objective: Female sexual health represents a dynamic interaction between biological predisposition, psychological resilience, and the socio-cultural environment. This panel conceptualizes female sexual dysfunction not merely as a deficit in desire, but as a complex "disorder of implementation" where the core challenge often lies in the inability to prioritize sexual arousal amidst competing executive demands. Our objective is to prioritize the clinical approach by reconciling the diagnostic nuances between DSM-5-TR and ICD-11. While DSM-5-TR consolidates desire and arousal into Female Sexual Interest/Arousal Disorder (FSIAD) to reflect the overlapping nature of these phases in women, ICD-11 maintains Hypoactive Sexual Desire Disorder (HSDD) as a distinct entity within its new dedicated chapter on sexual health. A critical component of the diagnostic process involves ensuring that symptoms persist for at least six months and result in significant clinical distress, thereby differentiating transient fluctuations from persistent dysfunction.

Etiologically, we move beyond linear models to examine the impact of cognitive load and environmental "noise" on sexual functioning. We analyze how "Incompetence" and "Self-distrust" schemas, often reinforced by restrictive socio-cultural scripts, act as internal inhibitors that increase the activation threshold for sexual response. Within this framework, sexual dysfunction is reframed as a manifestation of "cognitive fatigue," where the excessive executive demands of daily life deplete the mental energy required to sustain sexual attention. Therapeutic interventions focus on cognitive restitution, training patients to replace the "Perfectionist Critic" internal dialogue with a rational, task-oriented metacognitive framework that facilitates presence in the "point of performance." Finally, we address the role of external environmental stressors, specifically the iatrogenic constraints imposed by psychopharmacological interventions. While secondary to the broader biopsychosocial context, the blunting of dopaminergic reward pathways by SSRIs and SNRIs is managed as a technical barrier to be navigated through strategic dose modification or the integration of pro-sexual agents like Bupropion.

Conclusion: By externalizing the disorder and utilizing standardized monitoring via the FSFI (Female Sexual Function Index) and functional impairment scales, clinicians can transform therapeutic gains into sustainable functionality. This holistic management ensures that symptomatic stabilization is never achieved at the expense of the patient's sexual quality of life, maintaining the necessary external scaffolding for long-term relational success.

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Keywords: Sexual Dysfunction, Women's Health, Women's Sexual Health

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0448 - Lithium, the First-Line Medication for Bipolar Disorder; How Should We Cope with Its Major Functional Limitation: Tremors

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Objective: Lithium remains the cornerstone of bipolar disorder management, recognized by the American Psychiatric Association (APA) and the British Association for Psychopharmacology (BAP) for its unique anti-suicidal properties and prophylactic efficacy. However, postural tremor—affecting approximately %25-50 of patients—is not merely a cosmetic concern; it represents a major functional limitation that impairs fine motor skills and leads to social withdrawal. The objective of this panel is to provide a systematic algorithm for the clinician, guided by the Maudsley Prescribing Guidelines (15th ed.) and NICE clinical standards, ranging from pharmacokinetic modifications to advanced pharmacological interventions.

Clinical Management: The first step is the precise neurocognitive differentiation between fine physiological tremor and the "coarse" tremor that serves as a vital red flag for impending lithium toxicity. According to Maudsley protocols, management should follow a hierarchical sequence. The initial phase involves optimizing the lithium regimen by titrating the dose to the lowest effective serum concentration if clinically feasible, and minimizing the peak plasma concentration by switching to sustained-release (SR) formulations consolidated into a single nighttime administration. Concomitantly, clinicians must screen for secondary aggravators, including lithium-induced thyroid dysfunction (TSH monitoring) and the impact of other tremorogenic medications (e.g., antipsychotics or SSRIs).

Simultaneously, the elimination of external factors such as dehydration, sodium depletion, excessive caffeine, nicotine, and comorbid anxiety is essential. If behavioral and kinetic adjustments prove insufficient, pharmacological "scaffolding" is initiated. Propranolol (30-120~mg/day) is the first-line agent of choice. However, Maudsley and RANZCP guidelines offer high-dose Pyridoxine (Vitamin B6, 1200~mg/day) as a robust alternative for patients with contraindications such as asthma or bradycardia. For refractory cases, the panel examines the utility of Primidone and Topiramate (25-50~mg/day) as "second-line specialist interventions".

Conclusion: Lithium-induced tremor is not a justification for treatment discontinuation but a technical barrier to be managed. When the stepwise treatment algorithm is applied—integrating metabolic screening, dose optimization, and targeted pharmacology—functional recovery is achievable for the vast majority of patients.

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Keywords: Bipolar Disorder, Lithium-Induced, Management, Tremor

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0449 - Use of Trace Elements in Psychiatric Disorders

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Trace elements are essential nutrients that are present in small amounts in all living organisms. Zinc (Zn), copper (Cu), iron (Fe), selenium (Se), magnesium (Mg), chromium (Cr), cobalt (Co), fluoride (F), iodine (I), manganese (Mn), molybdenum (Mo), and, controversially, boron (B) and vanadium (V) are considered important trace elements in humans. Trace elements play crucial roles in physiological functions [1]. These micronutrients, which are required in small amounts for normal physiological processes, have significant roles in the central nervous system by influencing enzymatic reactions, neurotransmitter synthesis, and synaptic transmission [2]. Both excess and deficiency of trace elements may contribute to the development of various diseases, including mental disorders. A growing body of research has investigated the role of trace elements in several psychiatric conditions, including major depressive disorder, schizophrenia, anxiety disorders, bipolar disorder, and autism spectrum disorder. Recent studies suggest that certain trace elements may be associated with the pathophysiology of psychiatric disorders. For instance, reduced zinc levels have been reported to be associated with depression and anxiety symptoms. Additionally, abnormalities in zinc metabolism have been suggested to contribute to the pathophysiology of autism spectrum disorder. Disturbances in iron metabolism, particularly iron deficiency and anemia, have been shown to be associated with the onset and severity of depressive symptoms [1]. Several studies have also demonstrated that zinc and iron supplementation may reduce symptoms of depression and anxiety [3]. Kim et al. reported that iron dysregulation is associated with negative symptoms in patients with schizophrenia [4]. Another trace element, selenium, has been shown to have an inverse relationship between dietary intake and depressive symptoms. Research on the effects of selenium on anxiety is relatively recent; however, most evidence suggests an inverse association between selenium levels and anxiety, indicating that selenium supplementation may have a promising effect in alleviating anxiety symptoms [3]. Magnesium, another commonly studied trace element, has been investigated in preliminary studies aiming to enhance the effectiveness of pharmacotherapy in schizophrenia through magnesium supplementation [5]. Magnesium supplementation has also been shown to alleviate depressive symptoms, and different subtypes of depression have been associated with magnesium deficiency [1]. Copper, which is known to be associated with neurodegenerative processes, has also been implicated in psychiatric disorders. Alterations in copper metabolism may be related to depression and cognitive functioning. Some studies have reported that elevated serum copper levels are associated with major depressive disorder and neuronal metabolic changes. Furthermore, copper may influence psychiatric symptoms through its effects on neurotrophic factors and inflammatory processes [1]. In conclusion, findings from the literature suggest that trace elements may influence psychiatric symptoms through neurobiological mechanisms as well as inflammatory and neurotrophic pathways. A balanced diet that ensures adequate intake of these micronutrients may contribute to the alleviation of psychiatric disorders [1]. In this presentation, the role of trace elements in psychiatric disorders will be discussed in light of the current literature, with particular emphasis on the neurobiological effects, clinical findings, and potential therapeutic applications of commonly studied elements.

Keywords: Neurobiological mechanisms, psychiatric disorders, trace elements

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0450 - Self-Compassion and Trauma: The Role of Self-Compassion in the Healing Process

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Most evidence-based treatments for post-traumatic stress disorder (PTSD) in adolescents are grounded in the cognitive-behavioral model. Trauma-focused approaches such as Trauma-Focused Cognitive Behavioral Therapy (TF-CBT) and Cognitive Processing Therapy facilitate cognitive reappraisal and behavioral engagement through trauma discussion and exposure, thereby reducing avoidance and maladaptive cognitions (Banks et al., 2015; de Arellano et al., 2014). While these interventions demonstrate strong efficacy, emerging perspectives highlight the potential contribution of third-wave psychotherapeutic approaches, including mindfulness and self-compassion, as complementary components in trauma treatment.

PTSD is often characterized by persistent negative emotional states directed toward the self, including shame, guilt, anger, and self-criticism. These processes may be particularly relevant during adolescence, a developmental period marked by heightened self-focus and vulnerability to self-evaluative distress. Self-compassion, defined as a stance of kindness toward oneself in moments of suffering, has been associated with reduced shame sensitivity and lower levels of post-traumatic stress symptoms (Kangaslampi & Peltonen, 2019). Compassion-based approaches may therefore offer a promising pathway for addressing self-critical cognitions that frequently accompany trauma. Importantly, compassion-based interventions do not necessarily require direct exposure to trauma memories, which may reduce treatment barriers related to avoidance or emotional overwhelm. In addition, these approaches have demonstrated favorable levels of engagement and acceptability in clinical settings (Kirby, 2017). Recent reviews suggest that compassion-focused interventions may represent a novel and developmentally appropriate avenue for adolescent PTSD treatment (Brinckmann et al., 2024).

This presentation reviews current evidence on the role of self-compassion in trauma recovery and discusses potential clinical mechanisms through which compassion-based approaches may support healing processes in trauma-exposed individuals.

Keywords: Compassion-focused therapy, Self-compassion, Shame and self-criticism, Post-traumatic stress disorder, Trauma

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0451 - Clinical Management of Digital Addictions and Modern Psychiatric Approaches

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Introduction
The rapid expansion of digital technologies has led to a significant increase in problematic use of the internet, social media, online gaming, and smartphones. These behaviors are commonly described in the literature as digital addiction or problematic internet use. Digital addictions are particularly prevalent among adolescents and young adults and have been associated with impaired academic and occupational functioning, sleep disturbances, and social withdrawal. Increasing evidence suggests that these behaviors share core characteristics with behavioral addictions, including impaired control, craving, tolerance-like features, and continued use despite negative consequences. Moreover, neuropsychological research has highlighted the role of reward system sensitivity, impulsivity, and deficits in executive control processes. Consequently, the clinical assessment and management of digital addictions require an integrative psychiatric perspective that considers both psychological and neurobiological mechanisms.

Objective
This study aims to review current psychiatric approaches to the clinical assessment and management of digital addictions. In particular, it focuses on cognitive behavioral therapy–based interventions, digital detox strategies, and the emerging role of digital mental health applications in treatment.

Discussion
Comprehensive clinical evaluation of digital addictions requires detailed assessment of an individual's digital behavior patterns, including duration and frequency of use, primary purposes of digital engagement, and its impact on daily functioning. Several standardized instruments have been developed to support clinical assessment, such as the Internet Addiction Test (IAT), the Smartphone Addiction Scale (SAS), and the Internet Gaming Disorder Scale. From a theoretical perspective, the Interaction of Person–Affect–Cognition–Execution (I-PACE) model provides a comprehensive framework for understanding the development and maintenance of behavioral addictions by emphasizing the interaction between individual predispositions, affective responses, cognitive biases, and executive control mechanisms. Neuroimaging and behavioral studies suggest that dysregulation in reward-related neural circuits and impaired inhibitory control may contribute to the persistence of problematic digital behaviors. Among current treatment approaches, cognitive behavioral therapy (CBT) currently has the strongest empirical support. CBT-based interventions typically focus on identifying maladaptive cognitive processes related to digital use, enhancing self-regulation and time-management skills, and promoting alternative offline activities. Digital detox strategies—defined as structured periods of reduced or restricted digital media use—may also be integrated into therapeutic interventions as part of behavioral modification programs. Additionally, digital mental health applications and online therapeutic platforms have emerged as promising tools to improve access to care. However, concerns regarding data privacy, ethical standards, and clinical validity remain important considerations.

Conclusion
Digital addiction represents an increasingly important behavioral health problem with significant psychological and functional consequences. Effective clinical management requires comprehensive assessment, attention to comorbid psychiatric conditions, and the application of evidence-based psychotherapeutic interventions. Cognitive behavioral therapy and structured behavioral interventions currently represent the most promising treatment approaches. Future research should further examine long-term treatment outcomes, the clinical effectiveness of digital interventions, and ethical frameworks guiding the use of digital mental health technologies.

Keywords: Digital addiction, problematic internet use, cognitive behavioral therapy, behavioral addictions, digital mental health

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0452 - Recognizing Depression With Mixed Features

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1. The Diagnostic Challenge

Recognizing mixed features is notoriously difficult because the dominant depressive mood often masks the underlying "activated" symptoms. The DSM-5 Paradox: To qualify for the specifier, a patient must show three or more manic symptoms (e.g., grandiosity, pressured speech, or decreased need for sleep). Excluded Symptoms: Paradoxically, the DSM-5 excludes irritability, distractibility, and psychomotor agitation from the formal "mixed features" count because they occur in both depression and mania. However, many clinicians argue these "overlapping" symptoms are actually the hallmarks of a mixed state. It is recommended to use Koukopoulos' diagnostic criteria for mixed depression (1).

Misdiagnosis: These patients are frequently misdiagnosed with standard Unipolar Depression, Anxiety Disorders, or Borderline Personality Disorder.

2. Clinical Presentation: "Agitated Depression"

Patients often describe feeling "tired but wired." Key clinical markers include (2): Racing Thoughts: A subjective sense of "crowded" or "pressured" thinking. Physical Restlessness: An inability to sit still (akathisia-like) or purposeless pacing. Decreased Need for Sleep: Distinct from insomnia; the patient may sleep only 3–4 hours but feel strangely energized or "driven" the next day.

3. High Clinical Stakes

Research indicates that mixed features are associated with: Increased Suicide Risk: The combination of profound hopelessness (from depression) and high energy/impulsivity (from mania) creates a dangerous "volatile" state. Poor Treatment Response: Patients with mixed features typically do not respond well to traditional antidepressant monotherapy.

Iatrogenic Risk: Standard antidepressants (SSRIs/SNRIs) can worsen mixed symptoms, potentially triggering a full manic switch or increasing "rapid cycle disease".

4. Management and Treatment

Treatment shifts away from the standard "depression protocol" toward a mood-stabilizing framework: Atypical Antipsychotics: Drugs like lurasidone, quetiapine, and asenapine have shown the strongest evidence for treating depressive episodes with mixed features (3). Mood Stabilizers: Lithium or Valproate are often utilized to address the "activation" before or alongside other treatments.

Antidepressant Caution: If used, antidepressants should be introduced slowly and usually only in combination with a mood stabilizer or antipsychotic to "buffer" the risk of over-activation.

Keywords: Antidepressant, bipolar disorder, depression, diagnosis, mixed depression

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0453 - The Relationship Between Substance Use Disorder, ADHD, and Bipolar Disorder

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The relationship between Substance Use Disorder (SUD), ADHD, and Bipolar Disorder (BD) is defined by a high rate of comorbidity driven by shared neurobiological pathways. Both ADHD and BD are independently associated with an increased risk of developing SUD, as individuals often use substances to "self-medicate" symptoms like impulsivity, emotional dysregulation, and mood instability (1). This creates a "triple threat" where the presence of one disorder significantly complicates the clinical course and severity of the others, often leading to earlier onset of symptoms and higher rates of hospitalization.

The relationship between ADHD and Bipolar Disorder (BD) is characterized by a high rate of clinical comorbidity, estimated between 10% and 20% in adults, driven by shared genetic vulnerabilities and neurobiological dysfunction within the dopaminergic and noradrenergic systems (2). This overlap creates a significant diagnostic challenge, as the chronic executive dysfunction and impulsivity of ADHD often mimic the episodic mania or hypomania seen in BD, requiring clinicians to distinguish between lifelong traits and paroxysmal mood shifts. Effective management necessitates an integrated, sequential treatment strategy that prioritizes mood stabilization through agents like lithium or anticonvulsants before addressing ADHD symptoms, as the introduction of stimulants without a stabilized mood base carries a high risk of triggering manic episodes or accelerating cycle frequency.

The relationship between ADHD and Substance Use Disorder (SUD) is a complex bidirectional association where approximately 23% of adults seeking treatment for addiction also meet the criteria for ADHD, a prevalence rate significantly higher than that of the general population. This comorbidity is driven by shared neurobiological deficits in the dopaminergic reward system—which increase impulsivity and sensation-seeking—as well as the "self-medication" hypothesis, where individuals use substances like nicotine, alcohol, or illegal stimulants to quiet racing thoughts or temporarily improve executive function. Clinical outcomes for this dual diagnosis are often more severe, involving an earlier onset of substance use, higher rates of hospitalization, and a more chronic course of illness; however, recent 2026 clinical guidelines suggest that integrated treatment—including the use of long-acting stimulants or non-stimulants alongside behavioral therapy—can significantly reduce mortality and improve long-term recovery prospects (3).

Keywords: Addiction, ADHD, Bipolar Disorder, Substance Use Disorder

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0454 - The Monthly Storm: Premenstrual Syndrome and Emotional Dysregulation in Adolescence

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Adolescence is a critical window where neurodevelopmental processes intersect with hormonal maturation and emotional sensitivity reaches its peak. Recent studies conducted on adolescent girls in Turkey indicate that the prevalence of Premenstrual Syndrome (PMS) is as high as 61.2 percent, with more than half of this group (50.6 percent) experiencing severe symptoms and significant emotional dysregulation. This "monthly storm" is not merely a physical discomfort but a systemic condition associated with alterations in neural networks within the brain.

Current neuroimaging data demonstrate that this condition is rooted in changes within brain network structures. An observed increase in connectivity within the Salience Network during the luteal phase leads individuals to become hypersensitive to negative emotional stimuli and impairs their ability to filter these stimuli, resulting in dysregulation. Furthermore, fluctuations in estrogen and progesterone have been found to weaken the top-down control between the amygdala and the prefrontal cortex (PFC). This mechanism provides the biological basis for the sudden and impulsive emotional outbursts observed in adolescents.

Emotion regulation difficulties manifest through failures in cognitive strategy selection during this process. Systematic reviews in the literature emphasize that the tendency toward rumination is the strongest predictor of PMS severity. Adolescents with poor emotion regulation skills perceive menstrual pain and emotional symptoms as much more catastrophic. The reduction in emotional clarity during the luteal phase triggers maladaptive coping mechanisms, thereby exacerbating the clinical presentation.

"The Monthly Storm" is a phenomenon that directly threatens academic success, social relationships, and general mental health in adolescent girls. Turkish data show that the majority of this group attempts to manage this process without medical support. It is essential for clinicians to monitor the menstrual cycle from an emotion regulation-focused perspective and prioritize interventions targeting maladaptive strategies such as rumination in order to develop treatment protocols specific to the female phenotype.

Keywords: Adolescence, Premenstrual Syndrome (PMS), Emotional Dysregulation, Salience Network, Rumination

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0455 - The Neurobiology of Social Dysfunction: a Transdiagnostic and Translational Approach

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Social dysfunction is one of the most pervasive and disabling features across psychiatric disorders, yet it remains insufficiently captured in current diagnostic frameworks. Traditional categorical systems group biologically heterogeneous populations under broad syndromic labels. While this approach has improved diagnostic reliability, it has limited progress in identifying underlying mechanisms and developing targeted treatments. Converging evidence from neuroscience, genetics, and behavioral research suggests that core functional domains - rather than diagnostic categories - may provide a more biologically meaningful framework for understanding psychopathology. Among these domains, social dysfunction represents a particularly powerful transdiagnostic phenotype, as impairments in social engagement, affiliation, and social cognition are observed across conditions including major depression, schizophrenia, and autism spectrum conditions.

This keynote lecture will present a transdiagnostic and translational perspective on the neurobiology of social dysfunction, focusing on the role of large-scale brain networks - particularly the Default Mode Network (DMN) - in shaping social behavior and its disturbances. The DMN plays a central role in internally oriented cognition, including self-referential processing, mentalizing, and the representation of others' intentions. These processes form a neural foundation for social functioning. Converging neuroimaging findings indicate that alterations in DMN connectivity and network dynamics are consistently associated with social impairments across diagnostic categories, suggesting that dysfunction within this network may represent a shared neural substrate underlying social dysfunction.

Drawing on insights from large-scale translational initiatives such as the PRISM Project, the lecture will illustrate how dimensional measures of social functioning can be linked to distributed brain circuits rather than diagnostic labels. Studies integrating behavioral phenotyping, neuroimaging, and computational modeling demonstrate that variability in social motivation and social cognitive processing is associated with altered interactions between the DMN and networks involved in reward processing, salience detection, and cognitive control. These findings cut across diagnostic boundaries, reinforcing the view that social dysfunction reflects disturbances in core neural systems rather than disorder-specific pathology.

A central theme will be the integration of human neuroscience with backtranslation to animal models to establish causal mechanisms. Human neuroimaging studies can identify network-level signatures of social dysfunction, such as altered DMN connectivity. These observations can then be translated into experimentally tractable animal paradigms targeting homologous circuits involved in social approach, affiliation, and social learning. Using tools such as optogenetics, chemogenetics, and circuit-specific pharmacology, animal models enable causal testing of how specific neural pathways regulate social behavior and provide mechanistic validation of hypotheses derived from human imaging.

Because key components of social behavior - such as social approach, avoidance, and affiliative motivation - are evolutionarily conserved, aligning cross-species constructs allows the mapping of interacting neural circuits across humans and animal models. Within a broader transdiagnostic framework for precision psychiatry, social dysfunction thus provides a powerful paradigm for linking dimensional behavioral phenotypes to neural circuitry and experimentally testable mechanisms. By integrating network neuroscience, multimodal phenotyping, and cross-species causal approaches, this framework aims to move beyond syndromic classification toward a biologically grounded model of psychiatric illness and ultimately enable mechanism-based interventions that improve social functioning across disorders.

Keywords: Default Mode Network, precision psychiatry, translational neuroscience, neural networks, cross-species models, social behavior
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0456 - Technology and Attention Economy: Designing Behavior

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In contemporary digital ecosystems, behavioral outcomes are not merely incidental byproducts of technology use but are systematically shaped through deliberately engineered environmental architectures. This theoretical review examines the intersection of the attention economy, persuasive technology design, and psychiatric vulnerability, arguing that problematic digital engagement emerges from the interaction between individual psychiatric vulnerabilities and engineered digital environments.

Herbert Simon's concept of bounded rationality and Daniel Kahneman's dual-process model of cognition establish that human attentional capacity constitutes a finite and exploitable resource. As the abundance of information increases cognitive load, System 1 processing becomes increasingly dominant, rendering individuals more susceptible to environmentally structured behavioral patterns.

Within the framework proposed by Davenport and Beck, the primary objective of digital platforms extends beyond content production toward the construction of engagement-optimized architectures in which user attention functions as the principal commodity. Zuboff's concept of behavioral futures markets further extends this perspective, demonstrating that platform-based economic models are built upon the large-scale analysis of behavioral data to predict and shape future actions. In this configuration, users simultaneously function as both raw material and product within data-driven behavioral markets.

According to Fogg's Behavior Model, any behavior requires the simultaneous convergence of motivation, ability, and a prompt. Digital platforms systematically optimize these three variables through interface design, friction-reducing interaction patterns, and algorithmically mediated feedback mechanisms. Eyal's Hook Model further elaborates the cyclical reinforcement structure underlying habit formation in digital environments, illustrating how variable reward schedules sustain repeated engagement.

The neurobiological correlates of these design strategies are increasingly well documented. Sherman and colleagues demonstrated through functional neuroimaging that social media feedback activates mesolimbic reward circuitry, including the ventral striatum and ventral tegmental area, findings consistent with Schultz's dopaminergic reward prediction error model. Micro-design features such as infinite scroll, autoplay, and one-tap interaction operationalize Skinner's variable ratio reinforcement paradigm within commercial digital architectures, systematically lowering the behavioral threshold required for engagement.

Importantly, engineered digital environments do not exert uniform effects across individuals. Brand and colleagues' Interaction of Person-Affect-Cognition-Execution (I-PACE) model posits that individual differences in reward sensitivity, inhibitory control, emotional regulation, and executive functioning constitute key determinants of susceptibility to problematic digital engagement. Individuals with attention-deficit/hyperactivity disorder (ADHD), characterized by heightened reward sensitivity and reduced inhibitory control, may be particularly vulnerable to the reinforcing dynamics of hook-cycle architectures. Similarly, affective dysregulation and negative urgency, commonly observed across depressive and anxiety-spectrum conditions, may amplify the reinforcing salience of variable digital reward structures.

Conceptualizing digital addiction solely as an individual-level phenomenon risks overlooking the systemic and design-level determinants of maladaptive engagement. A framework that integrates environmental design factors with individual psychiatric vulnerabilities may enable clinicians and researchers to move beyond generic screen-time reduction recommendations toward mechanism-informed, targeted interventions.

The ethical implications arising from the interface between persuasive technology design and psychiatric vulnerability warrant increasing attention within both clinical training and public health policy, particularly as the prevalence of screen-based behavioral disorders continues to rise globally.

Keywords: Attention economy, persuasive design, digital addiction, behavioral architecture, psychiatric vulnerability, dopaminergic reward
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0457 - Should Emotion Regulation Difficulties Be Included Among the Diagnostic Criteria for Attention Deficit Hyperactivity Disorder?

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Emotional dysregulation (ED), including emotional lability and related affective symptoms, is frequently discussed as a core or co-occurring feature of ADHD across age groups. Overall, ED is a frequent and clinically significant dimension in both childhood and adulthood ADHD, though exact prevalence figures depend on methodology and sample, with a consensus that ED affects a substantial portion of individuals with ADHD and substantially contributes to impairment and functional outcomes.

ED is widely observed in people with ADHD and is clinically meaningful, but it is not listed as a core DSM-5 diagnostic criterion for ADHD. This reflects historical nosology, dimensional versus categorical reasoning, concerns about specificity versus comorbidity, and considerations about diagnostic utility and validity within DSM-5 criteria. Several reviews and empirical studies argue that ED is an associated feature or transdiagnostic risk factor that modulates impairment, rather than a discrete, standalone diagnostic symptom bundle used to define ADHD in DSM-5. Nevertheless, contemporary literature increasingly treats ED as a central, clinically relevant dimension within ADHD, with pharmacological and psychosocial treatments showing measurable effects on ED symptoms.
DSM-5 requires a discrete set of observable symptoms that cluster into core domains (inattention and/or hyperactivity-impulsivity) with clear diagnostic thresholds. Emotional dysregulation, while pervasive in many ADHD cases, is not uniquely specific to ADHD and frequently co-occurs with internalizing/externalizing disorders. DSM-5 aims to balance sensitivity and specificity; including ED as a core criterion could reduce specificity due to high comorbidity with mood, anxiety, and disruptive behavior disorders. This concern is discussed in reviews critiquing the DSM-5 structure and its ability to capture heterogeneity without inflating false positives.

ADHD is conceptualized as a neurodevelopmental disorder with dimensional variation in symptom severity and impairment. Several scholars argue that ED reflects a dimensional trait (emotional reactivity, lability, irritability) that traverses disorders, supporting transdiagnostic approaches (HiTOP/RDoC) rather than a single DSM-5 category. This stance is reflected in critiques of DSM-5's categorical criteria and calls for dimensional characterization of emotional processes within ADHD (and beyond).

ED is common across multiple psychiatric conditions (bipolar spectrum, ODD/CD, anxiety and depressive disorders). If ED were a core diagnostic criterion for ADHD, it would complicate differential diagnosis and potentially blur boundaries between ADHD and comorbid conditions. Some studies emphasize that ED in ADHD is partly attributable to comorbidity or overlapping symptomatology, whereas others find ED to be more tightly linked to core ADHD symptoms, suggesting a nuanced relationship rather than a simple one-to-one diagnostic criterion.

ED is a prevalent, impairing, and clinically meaningful dimension in ADHD, but DSM-5 favors discrete, ADHD-specific core symptoms to maintain diagnostic specificity. Findings from studies suggest that transdiagnostic and longitudinal aspects should be considered in the diagnosis of mental disorders. This approach is based on the integration of molecular and genetic data, various environmental factors, imaging findings, clinical observations, and pharmacological research data collected longitudinally.

Keywords: ADHD, Emotional Dysregulation, DSM-5

0458 - Clinical Aspects and Neurobiological Basis of Digital Addictions

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The rapid expansion of digital technologies has fundamentally reshaped human behavior, communication, and social interaction. While digital platforms provide numerous benefits, increasing evidence suggests that excessive and uncontrolled engagement with digital environments may lead to behavioral patterns resembling addictive behaviors. In recent years, the concept of digital addiction has emerged within the broader framework of behavioral addictions, highlighting the potential clinical and neurobiological mechanisms underlying problematic digital technology use.

From a clinical standpoint, digital addiction is characterized by persistent and compulsive engagement with digital platforms such as social media, online gaming, and smartphone-based applications despite significant impairment in psychological, social, or occupational functioning. Commonly reported clinical features include loss of control over digital use, compulsive repetition of digital behaviors, tolerance-like increases in usage duration, withdrawal-like psychological discomfort when access is restricted, and functional impairment in daily life. Although digital addiction itself is not yet universally recognized as an independent diagnostic entity in major psychiatric classification systems, related conditions—most notably gaming disorder—have been included in the ICD-11, reflecting growing recognition of technology-related behavioral addictions.

A growing body of neuroscientific research indicates that problematic digital technology use is closely associated with neural mechanisms involved in reward processing and reinforcement learning. In particular, the mesolimbic dopamine system—comprising the ventral tegmental area, nucleus accumbens, and prefrontal cortex—plays a central role in encoding reward prediction, motivation, and habit formation. Digital platforms frequently utilize design strategies that interact with these neural processes, including variable reward schedules, algorithm-driven content recommendations, personalized feedback systems, and continuous notification mechanisms. These design features may activate dopaminergic pathways and reinforce repeated engagement through mechanisms similar to those observed in other behavioral addictions.

Beyond neurobiological mechanisms, psychological vulnerability factors also contribute significantly to the development and maintenance of digital addictive behaviors. The Interaction of Person–Affect–Cognition–Execution (I-PACE) model provides a comprehensive theoretical framework suggesting that digital addiction arises from the dynamic interaction between individual predispositions, affective states, cognitive biases, and executive control processes. Individuals with higher impulsivity, depressive symptoms, social anxiety, or loneliness may exhibit increased susceptibility to maladaptive digital engagement.

Overall, digital addiction should be conceptualized as a multifactorial behavioral phenomenon resulting from the interaction between neurobiological reward systems, individual psychological vulnerabilities, and the behavioral architecture of digital platforms. A deeper understanding of these mechanisms may improve clinical awareness, support preventive mental health strategies, and guide future research on technology-related behavioral addictions.

Keywords: Digital addiction, Behavioral addiction, Dopamine, Reward learning, I-PACE model, Attention economy

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0459 - Aggression and Impulsivity in Youth: ADHD, Bipolar Spectrum, or Disruptive Mood Dysregulation Disorder?

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Aggression and impulsivity are among the most frequent and clinically impairing reasons for referral in child and adolescent psychiatry. Yet these phenomena are diagnostically nonspecific and can emerge across neurodevelopmental, mood, and disruptive behavior disorders. In daily practice, the key challenge is not merely recognizing aggression or impulsivity, but determining whether they primarily reflect the inhibitory control deficits and emotional impulsiveness of attention-deficit/hyperactivity disorder (ADHD), the episodic mood pathology of bipolar disorder, or the chronic, non-episodic irritability that characterizes disruptive mood dysregulation disorder (DMDD). Recent integrative reviews emphasize that irritability and reactive aggression are transdiagnostic constructs associated with impairment, suicidality, and adverse developmental outcomes, while also showing meaningful differences in longitudinal course, neurobiology, and treatment response.

In ADHD, impulsivity is a core dimension and commonly coexists with emotional dysregulation, frustration intolerance, and reactive aggression. Current evidence suggests that a substantial subgroup of youth with ADHD show clinically significant irritability, and available reviews indicate that optimized stimulant treatment and psychosocial interventions may reduce associated irritability and aggressive outbursts, although the evidence base remains more limited for irritability than for core ADHD symptoms.

By contrast, bipolar disorder requires careful attention to episodicity. The most reliable discriminator remains the presence of distinct mood episodes with accompanying changes in energy, activity, sleep need, and broader manic phenomenology, rather than chronic trait-like irritability alone. Recent reviews on pediatric bipolar misdiagnosis underline that overlap with ADHD remains one of the major sources of diagnostic error, often leading either to overdiagnosis of bipolarity in chronically dysregulated youth or to under-recognition of true episodic mood disorder.

DMDD occupies an intermediate but distinct position in this differential diagnosis. It was introduced to capture severe, persistent irritability with recurrent temper outbursts in youth who do not show the episodic manic pattern required for bipolar disorder. Contemporary reviews highlight that DMDD is highly comorbid with ADHD and other disorders, and that treatment evidence is still limited and heterogeneous; psychosocial approaches are generally considered first-line, with pharmacologic strategies often guided by comorbidity, especially ADHD. This presentation will review aggression and impulsivity through a clinically pragmatic, transdiagnostic lens, focusing on chronic versus episodic irritability, the phenomenology of impulsive aggression, common diagnostic traps, and treatment implications. The aim is to help clinicians move beyond surface symptom overlap and toward a more developmentally informed differential diagnosis of ADHD, bipolar disorder, and DMDD.

Keywords: Attention deficit hyperactivity disorder, aggression, bipolar disorder, disruptive mood dysregulation disorder, impulsivity, irritability

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0460 - Kristin Neff's Self-Compassion Model and Self-Compassion in Attention-Deficit/Hyperactivity Disorder

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Background

Self-compassion has gained increasing attention in psychological research as an important construct associated with emotional resilience, adaptive coping, and psychological well-being. According to Kristin Neff's theoretical framework, self-compassion consists of three interconnected components: self-kindness versus self-judgment, common humanity versus isolation, and mindfulness versus over-identification (Neff, 2003). These components represent an adaptive way of relating to oneself during experiences of difficulty, failure, or emotional distress. Individuals with higher levels of self-compassion tend to approach challenges with understanding and balanced emotional awareness rather than harsh self-criticism.

Attention-Deficit/Hyperactivity Disorder (ADHD) is a common neurodevelopmental disorder characterized by persistent patterns of inattention, hyperactivity, and impulsivity that impair functioning. Beyond core symptoms, children and adolescents with ADHD frequently experience academic and social difficulties and emotional dysregulation, which may contribute to negative self-perceptions and reduced psychological well-being. Self-compassion may represent a protective psychological resource by promoting a supportive, non-judgmental internal attitude. Evidence suggests that self-compassion difficulties may persist into adulthood in individuals with ADHD, with studies reporting lower self-compassion and reduced well-being among adults with ADHD compared to controls (Beaton et al., 2022). However, relatively few studies have examined self-compassion in children and adolescents with ADHD, highlighting the importance of investigating this construct earlier in development.

Methods

This cross-sectional study examined the relationships between self-compassion, self-esteem, and psychological well-being in children and adolescents with ADHD compared with age- and sex-matched healthy controls. The study was conducted at a child and adolescent psychiatry outpatient clinic in Türkiye.

The sample included 100 participants with ADHD and 100 healthy controls aged 10–17 years. Diagnoses were established using structured DSM-5–based clinical interviews. Psychometric measures included the Self-Compassion Scale for Youth (SCS-Y), the Piers-Harris Children's Self-Concept Scale (PHCSCS) for self-esteem, and the Stirling Children's Well-Being Scale (SCWBS) for psychological well-being.

Group comparisons were performed between ADHD and control groups. Within the ADHD group, Spearman correlation analyses examined associations among study variables, and multiple linear regression analyses assessed whether self-compassion and self-esteem independently predicted psychological well-being.

Results

Children and adolescents with ADHD demonstrated significantly lower levels of self-compassion, self-esteem, and psychological well-being compared with the healthy control group. Within the ADHD group, self-compassion showed a moderate positive correlation with psychological well-being ($r = 0.55$, $p < 0.001$), while self-esteem demonstrated a stronger positive association ($r = 0.66$, $p < 0.001$).

Multiple linear regression analysis indicated that both self-compassion and self-esteem were significant independent predictors of psychological well-being. The overall regression model explained 49.6% of the variance in psychological well-being ($R^2 = 0.496$). Self-esteem showed a stronger predictive effect ($\beta = 0.35$, $p < 0.001$), while self-compassion also made a significant independent contribution ($\beta = 0.19$, $p = 0.026$).

Conclusion

The findings highlight the importance of self-related psychological processes in youth with ADHD. Both self-compassion and self-esteem appear to contribute independently to psychological well-being, suggesting that these

constructs play complementary roles in emotional functioning. Kristin Neff's self-compassion model provides a useful theoretical framework for understanding how children and adolescents with ADHD relate to themselves in the context of challenges and perceived failures.

Keywords: ADHD, Adolescents, Psychological well-being, Self-compassion, Self-esteem

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0461 - Clinically-Based Critical Issues in the Use of Psychedelics in Treatment-Resistant Depression

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Background and Objective: The recent widespread interest in the use of psychedelics as potential therapeutic agents for various mental disorders, and particularly depression and treatment-resistant depression (TRD), raises a series of clinical and ethical questions that must be carefully considered.

Methods: First, it is important to limit this discussion to the exclusive medium- and long-term use of psychedelics, because their use in drug-assisted psychotherapy involves too many clinical variables to be considered in patient case studies and doesn't provide adequate scientific evidence. Analysis of the results of reviews on the clinical efficacy of psychedelics in TRD provides significant elements that should not be overlooked when assessing both the real appropriateness and the potential critical issues of their use. It is essential to consider the clinical aspects of the use of the various molecules considered (psilocybin, MDMA, LSD, etc.) individually, not those of psychedelics as a group. The neurobiological mechanisms of action of individual substances must be compared with the neurobiologicy of each disorder. The relationship between these two factors is currently completely unclear.

Results: Some critical issues that remain unresolved can be deduced from the literature, such as: a) the need for clinical acquisitions based on statistical evidence in large clinical populations of patients with specific depressive disorders; b) the need for evidence from controlled clinical trials with other active drugs, rather than placebo; c) the need to evaluate the effect on mood not only in healthy subjects but also in clinically depressed subjects. Comparing the results of different studies is seriously difficult due to differences in patient composition and the type, modality, and dosage of the substances administered. Significant clinical studies are still very few, and the number of patients treated is vastly smaller than that typically required in registration studies for other drugs, and the results are still inconclusive. The impractical blinding of studies represents a serious obstacle to objective research, which should, however, be carried out in populations with any previous use of psychedelics. There are no clear indications regarding the effective duration of treatment or the duration of any clinical remission. The role of patient and therapist expectations is certainly crucial, but not adequately considered. Ethical considerations are important with respect to clinical risk. There is a risk of treating seriously ill patients with drugs of unproven efficacy, of inducing acute psychotic states with clinical worsening, and of inducing unpredictable long-term psychosis. The relationship between drug-induced alterations in consciousness and their potential therapeutic effect is still largely unclear and must be clinically clarified.

Conclusions: The use of psychedelics in depression certainly appears promising, but many significant clinical and ethical issues remain unclear, and their resolution is crucial to the truly useful use of these substances.

Keywords: Psychedelics, treatment of depression, clinical critical issues, ethical critical issues

0463 - Clinical Applications and Interventions of Heart Rate Variability in Child and Adolescent Psychiatry: From Biomarker to Biofeedback

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Heart rate variability (HRV) reflects the dynamic balance between sympathetic and parasympathetic activity of the autonomic nervous system and has recently gained attention as a psychophysiological marker in psychiatry. In child and adolescent mental health, HRV is considered a promising tool for assessing stress responses, monitoring treatment effects, and guiding psychophysiological interventions.

Methods:

This presentation synthesizes recent systematic reviews and empirical studies (2014–2024) exploring HRV in child and adolescent psychiatric populations. The clinical use of HRV in stress assessment, pre–post treatment changes with pharmacotherapy, psychotherapy, and mindfulness-based interventions, as well as methodological issues such as device variability and measurement standards, are discussed. Practical applications including HRV biofeedback, breathing training, and lifestyle-related approaches are also reviewed.

Findings:

Decreased HRV has been consistently associated with elevated stress, anxiety, and depressive symptoms. HRV tends to increase following pharmacological or psychotherapeutic interventions, reflecting improved autonomic flexibility. HRV biofeedback and resonance frequency breathing (~0.1 Hz) have shown efficacy in enhancing emotion regulation and reducing anxiety. Mindfulness, yoga, adequate sleep, nutrition, and moderate physical activity further contribute to HRV improvement. However, measurement variability, developmental differences, and device heterogeneity limit clinical standardization in pediatric settings.

Conclusion:

HRV represents a sensitive, non-invasive biomarker for emotional regulation and treatment response monitoring in child and adolescent psychiatry. HRV biofeedback and lifestyle-based approaches are promising adjunctive interventions, supporting the integration of physiological self-regulation into everyday clinical practice. Future studies should focus on establishing age- and sex-specific normative databases and long-term efficacy across clinical populations.

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Keywords: Heart Rate Variability, Biomarker, Biofeedback

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0464 - Psychiatry and Spiritualism

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In the Turkish Language Association (TDK) dictionary, the word “maneviyat” (spirituality) is defined as “non-material, spiritual things” or “strength of heart, morale.” Etymologically, maneviyat is the plural form of the Arabic word manevi. The word manevi is defined as “related to meaning; especially related to hidden and deep meanings.” In the Kubbealtı Dictionary, the concept is explained in a little more detail: it is described as “matters that are not related to matter or physical objects but are connected with spirit and meaning, spiritual things. Its opposite is materiality.”

At first glance, the definition of spirituality seems to have a meaning close to that of culture. However, upon closer examination, we see that it is not so much a similarity in meaning as a resemblance. Both spirituality and culture share a similarity in that they attempt to define what is uniquely human among all other beings. Because the concept of spirituality has multiple dimensions of meaning and different disciplines emphasize different aspects of it, there is currently no definition that is universally accepted by everyone. The journey of the concept in our language has become even more complicated in recent years because it has begun to be used as the equivalent of the Western terms “spirituality” or “spiritualité.”

The words spirituality and its root spirit, which are also translated into Turkish as “ruhsallık” or “tinsellik,” have a long historical background. In earlier studies, we discussed in detail the use of the word spirit in Islamic thought as ruh (soul) and nefs (self). According to the Oxford Collocation Dictionary, when used as an adjective (spiritual), the word refers to a person’s deep thoughts, emotions, and feelings and carries meanings related to religion and the Church. As a noun, spiritualism refers to the belief that messages can be received from the spirits of the dead, while a spiritualist is someone who receives messages from spirits.

We can say that the close relationship between spirituality and religion is valid not only for the Islamic culture we live in but also for all religious cultures of the traditional world. Certainly, spirituality was not exactly the same as religion, but they were close enough that distinguishing between them was often difficult. We emphasize this point because, until recently, a similar closeness and overlap in meaning between religion and spirituality existed in Western languages as well. However, a new development has emerged in recent times. As interest in Eastern religions and New Age beliefs has increased among people living in Western societies, an interesting situation has arisen. The term spirituality has begun to occupy a semantic space in opposition to traditional religion (Christianity). Some researchers have even suggested that the growing interest in spirituality is a result of distancing from traditional religion.

We believe that a consensus can be reached around the universal definition of spirituality as “feeling life and, as a result of the knowledge gained throughout life, understanding oneself, one’s relationships with others, the meaning of life, and one’s place in the universe.”

Keywords: Culture, Islam, self, soul, spirituality

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0465 - Dying While Living: Where Life Touches Death

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Death, as a concept, is ambiguous, relative, and complicated. Especially in recent times, as a result of developments in modern medicine, the process of death has almost been divided into tiny parts. The loss of consciousness, the cessation of the heart and breathing, and brain death have begun to replace the main death as small deaths, making the confusion even more difficult to untangle. “Clinical death,” “brain death,” “biological death,” and “cell death” correspond to different conditions. Each of them is death, indeed, but they are all different kinds of death from one another. These definitions of death are not limited to these. The severe withdrawal of people with serious mental illnesses—so intense that they do not speak to anyone—is sometimes called “psychological death.” In this talk, however, we would first like to address and discuss the concept of “psychological death” as we defined it in our book Hoşçakal.

Psychological death is related to a person’s awareness of death, and its first visible layer is a mode of our being as “being-toward-the-world.” Being human condemns us to what we may call the concerns of life. In the world, we are compelled to deal with things with certain purposes in mind—practically, with the help of tools and instruments, and with a certain care. While living in the world, we are always involved with something; we cannot escape the burdens of life. Most of the time, within the worries of daily livelihood, we do not even feel this flow of life.

But when our engagement with the world calms down, when we no longer have something to occupy ourselves with—for example, when we sit idly, rest, exercise our right to be lazy, idle away time, or take a deep “ohh” breath after finishing a task—anxiety emerges from the corners where it had been hiding. We look at ourselves for a moment and perceive the world as something that stands there independently of our goals, tools, rules, and efforts: “there it is, just standing there.” The world stands there; and we are here as a bundle of desires. Seeing the gap between us and the world is frightening. When our desire is activated, we merge with the world; but if our desire cannot act, cannot turn into engagement, cannot attach itself to a concern, we notice the separation and the gap grows larger and larger before us. This gap is a threat of “worldlessness.” The world may be completely taken away from us. Worldlessness means not being able to take part in the game of life, not living, becoming nothing—that is, “death.” Therefore, in every unlived experience, in every piece of life our desire cannot reach, what we experience is “psychological death.”

Human beings are not entities that drift endlessly within infinity, merely enjoying the world or clinging entirely to it and playing with its possibilities. Human existence is finite; it is of a kind that comes to an end.

Keywords: Being towards the world, being towards death, death, Heidegger, psychological death

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0466 - Responses to the Theses of Dostoevsky's The Gambler

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One of the most important writers of world literature, Fyodor Mikhailovich Dostoevsky's novel The Gambler offers a great opportunity to examine gambling behavior and the psychology of a gambling addict. The connection between his personal life and his novels has been the subject of many studies and theses, but The Gambler is considered to be almost entirely inspired by Dostoevsky's own life. Some even describe the work as "an artistic expression of Dostoevsky's love affair with Polina Suslova in Europe and the author's passion for gambling." The events in The Gambler strongly resemble Dostoevsky's real life: Dostoevsky himself resembles the character Aleksey in the novel, and Polina Suslova closely resembles the character Polina Aleksandrovna. The book is both a self-criticism and a defense. In this talk, starting from the perspective presented in our book "The Game of Life," we will attempt to respond to the defense offered by Dostoevsky's gambler.

In our view, the main factor that distinguishes a gambling addict—the gambler—from those who occasionally play games of chance or those who never play is the belief that they will inevitably win. Certainly, the belief that one will win, that this time one will finally break the cycle of bad luck, that fate will eventually smile upon them, exists in all of us. Many people occasionally try their luck in games of chance just for entertainment. But what is the difference between standing behind our choices, believing that what we do is right, and sometimes trying our luck—and the gambling addict's absolute belief that they will certainly win? The answer is: a difference as great as mountains.

We say "as great as mountains" because we do not fall into the mistake of modern psychological sciences and psychiatry by trying to explain the difference in terms of quantity or measurable phenomena. We do not say that the gambling addict is different simply because they play for longer periods or take greater risks. We say "as great as mountains" because we, in one way or another, take responsibility for our actions and choose virtues as our guiding principle in life. Even though we often make mistakes and feel regret, we still choose to follow the path of virtues, set our guides for life accordingly, and accept the consequences of our choices.

The gambling addict, however, does not take the possibility of being wrong into account at all. In their world, ambiguity, contingency, and other possibilities have no place. They believe with certainty that they will win, and when they lose, they attribute the responsibility not to themselves but to other factors. In terms of the belief in certainty, the gambler is, in a sense, more modern than all other modern individuals; their belief in certainty becomes incurable. Moreover, their guiding principle is no longer to live a virtuous life. Gradually, they abandon the aim of avoiding evil as much as possible, standing on the side of goodness and good deeds, and earning a lawful livelihood as the reward for their effort.

Keywords: Dostoevsky, gambler, gambling addict, game of chance, game of life

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0467 - Digital Addictions and the Distortion of Time in The Modern Age

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The rapid development of digital technologies over the past three decades has led to the emergence of a novel phenomenon known as digital addiction. This umbrella term encompasses internet addiction, social media addiction, smartphone addiction, and gaming addiction. Global prevalence estimates of digital addiction subtypes range from 6.04% to 26.99%, highlighting its growing importance as a public health problem. From a neurobiological perspective, digital addiction is characterized by a set of underlying pathophysiological processes that significantly overlap with the clinical manifestations observed in substance use disorders. Particularly among adolescents and young adults in the risk group, the coexistence of an immature prefrontal cortex and increased sensitivity of the reward system constitutes a developmentally vulnerable state (1).

Time perception is a cognitive process that involves the evaluation of both the duration and the sequential order of events. The discrepancy between objective time and perceived time is referred to as distortion of time. Alterations in time perception, occurring as either downward bias (underestimation) or upward bias (overestimation), constitute a significant phenomenon in addictive behaviours. In abstinence states, upward bias can render delays in reward difficult to tolerate, thereby increasing vulnerability to the repetition of addictive behaviours. Particularly in digital addictions, downward bias observed during addictive behaviour may lead to prolonged use, tolerance development, and behavioural reinforcement (2). Consequently, addiction and time perception distortion may form a cycle in which addiction alters time perception, while altered time perception further reinforces addictive behaviour. Time perception distortions observed during withdrawal in digital addiction hold a distinctive and important role. Subjective feelings of inner discomfort and tension can lead to a homeostatic imbalance in the flow of time perception. Alongside these experiences, impairments in time perception may also contribute to impulsivity, which is frequently associated with addiction. Perceived delay significantly influences the tendency to favour immediate rewards rather than delayed rewards. This homeostatic disturbance is likely related to alterations in the interoceptive awareness system. Dysfunction in the insular cortex not only disrupts the internal perception of time but also leads to difficulties in impulse regulation. In this context, the insular cortex, together with the prefrontal cortex, supports the persistence of the addiction cycle (2,3).

Distortions in time perception should not be considered merely as a consequence of digital addiction; it also represents a mechanism that contributes to the exacerbation and maintenance of digital addictive behaviours. In the treatment and prevention of addictions, a comprehensive approach combining pharmacological methods with cognitive behavioural and family and community-based interventions is required. Within this holistic framework, time perception may represent a potential target for intervention in digital addiction. Future research focusing on time perception will play a critical role in elucidating the pathophysiology of digital addiction, which is becoming increasingly prevalent in society, and may contribute to the development of time perception–targeted interventions.

Keywords: Digital addiction, Time estimation, Time distortion, Time perception

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0468 - Clinical Interventions for Impulsive and Compulsive Symptoms: From CBT to Pharmacotherapy

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Impulsivity and compulsivity are key transdiagnostic dimensions observed across a wide range of psychiatric disorders, including obsessive–compulsive disorder (OCD), attention-deficit/hyperactivity disorder (ADHD), substance use disorders, behavioral addictions, and certain personality disorders. Traditionally conceptualized as opposing constructs, impulsivity refers to premature or poorly considered actions driven by reward sensitivity, whereas compulsivity involves repetitive behaviors performed to reduce distress or anxiety. Contemporary research, however, increasingly conceptualizes these phenomena as part of a shared neurobehavioral spectrum involving dysfunction in cortico-striato-thalamo-cortical circuits and abnormalities in serotonergic, dopaminergic, and glutamatergic neurotransmission. These overlapping mechanisms contribute to deficits in inhibitory control, reward processing, and habit learning, thereby influencing both impulsive and compulsive behaviors.

From a therapeutic perspective, psychological interventions remain a cornerstone in the management of impulsive and compulsive symptoms. Cognitive behavioral therapy (CBT) has demonstrated substantial efficacy across several disorders characterized by these behavioral dimensions. In obsessive-compulsive disorder, exposure and response prevention (ERP), a specific CBT-based technique, is considered the first-line psychotherapeutic treatment and has consistently shown significant reductions in compulsive rituals and avoidance behaviors. ERP facilitates inhibitory learning and reduces pathological anxiety by encouraging patients to confront feared stimuli while refraining from compulsive responses. Beyond OCD, CBT-based interventions targeting cognitive distortions, emotional regulation, and maladaptive decision-making have also shown effectiveness in behavioral addictions, compulsive sexual behavior, and problematic internet use. These approaches emphasize restructuring dysfunctional beliefs and improving impulse control through structured behavioral strategies.

Pharmacotherapy represents another fundamental component of treatment, particularly in moderate to severe cases or when psychotherapy alone is insufficient. Selective serotonin reuptake inhibitors (SSRIs) remain the first-line pharmacological treatment for OCD and related disorders, highlighting the critical role of serotonergic dysregulation in compulsivity. For patients with treatment-resistant symptoms, augmentation strategies with atypical antipsychotics such as aripiprazole or risperidone may enhance treatment response. In impulsivity-dominant disorders, medications targeting dopaminergic and noradrenergic systems—such as stimulants and atomoxetine—have been shown to improve inhibitory control and reduce impulsive behaviors. More recently, glutamatergic modulators including N-acetylcysteine and memantine have gained increasing research interest as potential adjunctive treatments for obsessive-compulsive and related disorders. Emerging evidence from clinical trials suggests that modulation of glutamatergic signaling may represent a promising strategy for treatment-resistant compulsive symptoms.

Recent developments in neuroscience have further expanded treatment possibilities through neuromodulation techniques such as repetitive transcranial magnetic stimulation (rTMS) and transcranial direct current stimulation (tDCS), which aim to directly modulate dysfunctional frontostriatal circuits implicated in compulsive and impulsive behaviors. Preliminary studies suggest that combining neuromodulation with psychotherapeutic approaches such as ERP may enhance treatment outcomes and improve long-term symptom control in refractory cases.

In clinical practice, optimal management of impulsive and compulsive symptoms requires an integrative and individualized treatment strategy. Combining evidence-based psychotherapeutic interventions with targeted pharmacotherapy allows clinicians to address both behavioral patterns and underlying neurobiological mechanisms. A dimensional and transdiagnostic perspective may therefore improve treatment personalization and enhance functional outcomes across a wide spectrum of psychiatric disorders characterized by impulsivity and compulsivity.

Keywords: Impulsivity, Compulsivity, Cognitive Behavioral Therapy, Pharmacotherapy, Obsessive-Compulsive Disorder, Neuromodulation

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0469 - Statistics on Children Drawn to Crime

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According to UNICEF’s 2020 and 2024 data, nearly 260,000 children worldwide are held within the justice system[1]. The term 'deprivation of liberty' covers their entire journey: from initial police detention and pre-trial custody to post-verdict rehabilitation or imprisonment.

Statistically, South Asia has the highest total number of detained children, at 111,000. When looking at the frequency of detention per 100,000 children, the top three regions are North America (34), Latin America (26), and South Asia (24). On a positive note, estimates suggest there are 67,000 fewer children in detention today than in 2018. Significant drops in both detention rates and total numbers have been recorded in North America, East Asia, the Pacific, and Latin America.

However, global data remains highly fragmented. Many countries face serious data gaps especially in rural areas. While basic details like age, gender, and the type of offense are usually tracked, deeper information about the root causes of delinquency is often missing, making it difficult to conduct a truly profound analysis. In Türkiye, juvenile justice is based on the principle that "imprisonment should be a last resort." However, current data shows significant pressure on the system. According to the Ministry of Justice and TÜİK, there were 3,532 children in correctional facilities in 2024. Notably, three-quarters of these children are in "pre-trial detention," which raises questions about the speed of trials and the use of alternatives to custody.

The number of children brought to security units due to delinquency increased by 9.8% over the previous year, reaching 202,785. The most common offenses are assault (40.4%) and theft (16.6%)[2]. Demographically, 95.7% of these individuals are male, and most are between the ages of 15 and 18. Furthermore, statistics show that nearly half of all crimes in Türkiye are committed by people under the age of 25, and 90% of adult offenders first encountered the justice system during their youth[3].

A child’s path toward crime is too complex to be linked to a single reason. Bronfenbrenner’s Ecological Systems Theory suggests that a child is not an isolated individual but is shaped by their environment. Like the layers of an onion, a child is affected by every level of their surroundings[3]:

• Individual Factors: Biological changes during adolescence, being male, risk-taking behavior, ADHD, oppositional defiant disorder, and substance abuse can increase the risk of delinquency.

• Family and Environment: Poverty is the most critical factor. Broken homes, domestic violence, low education levels, and the challenges of migration also play major roles.

• Education and the Street: Children who drop out of school or lack future plans—especially those working unsupervised on the streets—become much more vulnerable to exploitation by criminal groups.

The fact that criminal involvement often begins at a young age shows how vital the justice system's first contact with a child can be. While fragmented data makes it hard to track each child's journey, analyzing these statistics is essential to understanding the environmental factors that lead to a child being drawn to crime.

Keywords: Juvenile deliquency, statistics and numerical data, risk factors

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0470 - Current ECT Application Protocols and Management of Side Effects

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ECT is performed under general anesthesia with muscle relaxants, typically 2-3 times weekly for 6-12 sessions, following a 6-8 hour fast. Pre-treatment involves clarifying indications, educating patient and family, and establishing baseline psychiatric and cognitive status. A physical exam must review medications and comorbidities, particularly cardiopulmonary or neurological issues and prior anesthesia history. Dental health and handedness should be noted. Recommended tests include serum potassium, ECG, and beta-hCG for women of childbearing age.

Most psychotropics, including antidepressants and antipsychotics, are continued during ECT for synergistic efficacy[1]. Morning doses are given after post-procedure awakening. Conversely, anticonvulsants and benzodiazepines should be tapered as they reduce seizure duration and effectiveness. High-dose clozapine and low-potency first-generation antipsychotics require caution due to risks like prolonged seizures, hypotension, and QTc prolongation. While lithium is generally safe, withholding one or two doses prior to ECT is suggested to minimize post-ictal delirium and succinylcholine interactions.

In accordance with the Turkish Ministry of Health Patient Rights Regulation and the 2006 ECT Application Directive, informed consent must be obtained from the patient, guardian, or first-degree relatives. In urgent medical cases where relatives cannot be reached, ECT may be administered based on the documented decision of two psychiatric specialists.

While there is no rigid standard for the number of sessions, studies indicate an average of 7 sessions, with 90% of patients remitting within 9 or fewer treatments. To prevent relapse, sessions should be tapered gradually once a clinical plateau is reached. Monitoring should involve clinician-administered scales rather than self-reports—such as the Hamilton Rating Scale for Depression (HAM-D) or MoCA for cognitive assessment—to avoid inaccuracies caused by potential confusion or amnesia.

ECT-related mortality is exceptionally rare, with post-2001 data showing fewer than 1 death per 100,000 sessions, typically due to cardiopulmonary events. Physical side effects include headache, nausea, muscle pain, and dental injuries[2]. Cognitive side effects remain the most significant concern. The severity of cognitive impairment following ECT is determined by factors such as electrode configuration, the dosage and waveform of the stimulus, and the specific anesthesia protocol used[3]. Although 50-80% of patients report some memory loss acutely, objective tests often show that the benefits outweigh the risks, particularly in patients with high cognitive reserve. Higher rates of subjective memory impairment are noted in females, younger patients, lithium users, and non-responders.

Continuation and Maintenance ECT are two-stage processes aimed at sustaining recovery following acute treatment. Continuation ECT (C-ECT) is administered during the first six months after a successful course, with the primary goal of preventing a relapse of the current episode. Maintenance ECT (M-ECT) covers the period beyond the initial six months and aims to protect the patient from the recurrence of a new illness episode. Typically conducted as outpatient procedures, these treatments target patients with high relapse risk or treatment resistance, starting weekly and gradually tapering to monthly intervals. Ultimately, this structured approach—from rigorous pre-ECT assessment to personalized maintenance protocols—ensures that acute clinical gains are translated into sustained, long-term recovery.

Keywords: Electroconvulsive Therapies, Adverse Effects, Standards

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0472 - Juvenile Offenders: From Concept to Reality — A Global and Turkish Perspective

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Juvenile offending is a complex phenomenon shaped by developmental, psychological, social, and legal factors. Contemporary research increasingly emphasizes that delinquent behavior during adolescence should not be interpreted solely through a criminal justice lens. Instead, it should be understood within a developmental framework that considers the biological and psychosocial changes characteristic of adolescence. Neurodevelopmental studies show that brain regions responsible for impulse control, emotional regulation, and decision-making continue to mature during this period. As a result, adolescents may demonstrate greater impulsivity, increased risk-taking behavior, and heightened susceptibility to peer influence.

Globally, juvenile justice systems have gradually shifted from punitive responses toward rehabilitative and restorative justice approaches that emphasize protection, accountability, and social reintegration. International frameworks such as the United Nations Convention on the Rights of the Child and the Beijing Rules highlight that children in conflict with the law should be treated within systems that prioritize rehabilitation and reintegration. The United Nations Committee on the Rights of the Child recommends that the minimum age of criminal responsibility should not be set below 14 years.

The age of criminal responsibility varies across jurisdictions. In several European countries, including Germany, Italy, and Spain, it begins at 14 years, while some countries adopt higher thresholds. In Türkiye, criminal responsibility is regulated within a developmentally sensitive framework. The Child Protection Law enacted in 2005 aligned national practices with international child-rights standards and strengthened a rights-based approach to children in conflict with the law. Children under 12 are not criminally responsible, while responsibility for those aged 12–15 depends on their capacity to understand the legal consequences of their actions.

Recent statistics illustrate the scale of juvenile justice involvement in Türkiye. According to the Turkish Statistical Institute (TÜİK), the number of children brought to security units due to alleged involvement in crime increased from 178,834 to 202,785 in 2024. The most common alleged offenses include injury, theft, and drug-related crimes. In the same year, judicial decisions were issued for 211,946 children, including 63,712 penal decisions, of which 43,128 resulted in imprisonment sentences. Preventive measures such as counseling orders were applied to 51,386 children.

These findings indicate that approaches based solely on punishment are insufficient within juvenile justice systems and highlight the need for developmentally sensitive and rehabilitation-oriented policies. In this context, forensic psychiatric assessment plays an important role in identifying the developmental characteristics, mental health conditions, and psychosocial risk factors of juveniles in conflict with the law. Such evaluations may contribute to the development of more appropriate and individualized judicial responses that take into account the specific needs of the child and emphasize rehabilitation.

Keywords: child and adolescent mental health, criminal responsibility, forensic psychiatry, juvenile offenders

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0474 - Criminal Responsibility and Forensic Psychiatric Evaluation in Juvenile Offenders

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Criminal responsibility in juveniles should be examined not only from a legal perspective but also within developmental, psychological, and social contexts. A crime is generally defined as an act punishable by law. According to the Child Protection Law in Türkiye, a child in conflict with the law is defined as a child who is alleged to have committed an act defined as an offense, who is subject to investigation or prosecution for that act, or against whom a security measure has been imposed due to such an act. This definition reflects a fundamental principle of juvenile justice systems: they emphasize protection, rehabilitation, and social reintegration rather than punishment alone.

Criminal responsibility depends on an individual's capacity to understand the nature and consequences of their actions and to regulate their behavior accordingly. Individuals who lack the capacity to control their behavior or comprehend the consequences of their actions cannot be held criminally responsible. Because children and adolescents are still undergoing biological, cognitive, and moral development, their responsibility for criminal acts must be evaluated with careful consideration of developmental factors.

In Türkiye, the legal framework governing criminal responsibility is primarily defined in Article 31 of the Turkish Penal Code. Children under the age of twelve have no criminal responsibility. For children aged twelve to fifteen, criminal responsibility depends on whether the child has the capacity to perceive the legal meaning and consequences of the act and to direct their behavior accordingly. This determination requires expert evaluation. Adolescents aged fifteen to eighteen are considered criminally responsible, although the law provides reduced penalties due to their developmental status. Other legal provisions, such as mental illness and temporary conditions, may also influence the assessment of criminal responsibility.

Forensic psychiatric evaluation therefore plays a crucial role in this process. During these assessments, clinicians examine the child's biopsychosocial development, including cognitive capacity, reasoning ability, judgment, impulse control, and moral understanding. Psychiatric, neurological, and environmental factors may also influence the child's ability to regulate behavior and appreciate the consequences of their actions.
In conclusion, determining criminal responsibility in juveniles is not merely a legal process but a multidisciplinary assessment integrating developmental, psychiatric, and social dimensions. The ultimate goal of juvenile justice is not punishment alone but the protection, rehabilitation, and reintegration of the child into society.

Keywords: Adolescence, criminal responsibility, forensic psychiatry, juvenile justice, juvenile offenders

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0475- Childhood Trauma, High Trauma Load, and the Roadblocks to PTSD Recovery

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Childhood trauma refers to adverse experiences that occur during development, especially repeated or interpersonal forms of harm such as physical abuse, sexual abuse, emotional abuse, neglect, attachment disruption, domestic violence exposure, and chronic invalidation. These experiences are clinically important not only because they increase the risk of later post-traumatic stress disorder (PTSD), but also because they shape the developing self. When trauma occurs early, and particularly when it is inflicted by caregivers or trusted others, its effects often extend beyond fear-based memories. It may alter affect regulation, identity formation, expectations of safety, and the capacity to trust relationships. Current conceptual work on PTSD emphasizes that trauma responses involve disturbances not only in memory and arousal, but also in identity and self-organization, helping explain why many patients with childhood trauma present with complex, layered clinical pictures rather than a "pure" PTSD syndrome.

High trauma load refers to the cumulative burden of traumatic exposure across the lifespan. It includes not just the number of traumatic events, but also their chronicity, interpersonal nature, developmental timing, and recurrence. A person with high trauma load may have experienced multiple distinct traumas or prolonged trauma across years. Clinically, this matters because trauma load is associated with a dose-response pattern: as the number and accumulation of traumatic experiences rise, the likelihood of PTSD and the overall severity of symptoms increase. High trauma load also helps explain why two patients with the same diagnosis may look very different in practice. One may recover after focused trauma treatment, while another remains highly symptomatic because trauma has become embedded in personality organization, coping style, and social functioning.

The roadblocks to PTSD recovery in such patients are therefore multidimensional. First, childhood trauma often contributes to dissociation, shame, negative self-beliefs, and chronic affect dysregulation. Second, repeated trauma frequently disrupts attachment and interpersonal trust, making disclosure, alliance formation, and treatment engagement more difficult. Third, cumulative trauma is commonly accompanied by depression, substance misuse, somatic distress, anxiety, and personality-related difficulties, all of which may obscure the PTSD picture and slow recovery. A recent systematic review identified key pathways linking childhood complex trauma to complex PTSD, including dissociative processes, relationship with self, emotional developmental processes, social developmental processes, and broader systemic or contextual factors. These mechanisms help explain why some patients improve only partially, relapse under stress, or continue to experience substantial impairment despite symptom reduction.

Importantly, trauma-focused therapies remain effective, and patients with extensive trauma histories can show meaningful improvement. However, evidence suggests that individuals with higher trauma load often begin treatment with greater severity and are more likely to have residual symptoms after therapy, even when they benefit overall. This means recovery should be understood not simply as fear extinction, but as a broader developmental process involving affect regulation, self-repair, relational safety, and restoration of functioning. For psychiatric practice, the key implication is clear: when PTSD is rooted in childhood trauma, cumulative adversity, assessment and treatment must be formulation-based, longitudinal, attentive to both symptoms and the damaged structures of self that sustain them

Keywords: Childhood trauma, high trauma load, PTSD, roadblocks

0476 - AI standardized patients: Transforming psychiatry training through simulation

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Background and Objective: Precision psychiatric diagnosis and advanced medical education urgently require high-fidelity Standardized Patients (SPs). However, current Large Language Model (LLM)-based virtual patients suffer from the "alignment paradox" and "semantic gap," exhibiting shallow pathological cognition, over-compliance, and a lack of disease-specific multimodal representations (e.g., psychomotor retardation, blunted affect). This project aims to propose a novel Generative AI Standardized Patient (AISP) paradigm based on "pathological core inverse mapping and multimodal mirror empowerment" to overcome these translation bottlenecks and transform psychiatric training and AI evaluation.

Methods: The research employs a tripartite, highly coupled architecture. First, a dynamic evolutionary model of pathological cognition is constructed using Inverse Reinforcement Learning (IRL) to infer latent value functions from real consultation trajectories, enabling the natural emergence of psychological defense mechanisms (e.g., clinical resistance, agitation). Second, a forward-detection-guided inverse multimodal reconstruction algorithm utilizes differentiable clinical discriminators to constrain generative networks, synthesizing diagnostically meaningful acoustic and facial features while establishing a cross-modal semantic consistency mechanism. Finally, a "Doctor-Patient-AI" quaternary closed-loop adversarial ecosystem is built to facilitate automated Adversarial Clinical Turing Tests (ACTT).

Results: The proposed AISP is expected to achieve a logic consistency of over 90% during multi-turn inductive pathological interactions. The generated multimodal biomarkers will attain an 85% feature match rate when evaluated by professional psychiatric diagnostic models, significantly outperforming generic virtual avatars. Furthermore, the project will establish the field's first high-fidelity multimodal psychiatric synthetic case benchmark dataset (Synthetic-Psych-500). Within the adversarial ecosystem, the AISP will serve as a pressure-testing platform, projected to improve the diagnostic robustness of target AI systems by over 30% on edge and extreme cases.

Conclusions: The AISP paradigm successfully transforms virtual patients from superficially compliant chatbots into authentic digital twins with deep pathological fidelity. By bridging the gap between generative AI and clinical psychiatry, this project provides a scalable, zero-risk "high-pressure simulator" for medical education and a robust "touchstone" for validating intelligent diagnostic systems, thereby driving the methodological advancement of computational psychiatry.

Keywords: Large Language Models, Computational Psychiatry, Standardized Patient

0477 - AI-Assisted Psychopharmacology: Personalized Medication Selection in Treatment-Resistant Cases

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Treatment-resistant psychiatric disorders remain a major challenge in psychopharmacology because sequential trial-and-error prescribing may prolong symptom burden, increase adverse effects, and delay functional recovery. Recent advances in pharmacogenomics, artificial intelligence (AI), and multimodal biomarker research have renewed interest in personalized medication selection, particularly in depressive disorders. This presentation reviews the current state of AI-assisted psychopharmacology with emphasis on treatment-resistant depression, where the evidence base is currently most developed, and briefly considers emerging implications for child and adolescent psychiatry.

The presentation first addresses clinically actionable precision prescribing. Current CPIC guidance supports the use of CYP2D6, CYP2C19, and CYP2B6 genotypes to inform serotonin reuptake inhibitor prescribing, whereas evidence for SLC6A4 and HTR2A remains insufficient for routine clinical use [1]. This translational approach is supported by a recent systematic review and meta-analysis showing that pharmacogenetically informed antidepressant treatment is associated with improved remission and fewer adverse drug events compared with usual care [2], as well as by a randomized clinical trial demonstrating superior remission and response outcomes and lower adverse drug reaction rates with pharmacogenomic-guided treatment [3].

The talk then examines emerging AI-based clinical decision support systems designed to move beyond single-marker approaches. Recent adult studies suggest that deep-learning models can generate individualized remission probabilities across multiple antidepressant options [4], while early prospective evidence from the AID-ME trial supports the feasibility of AI-enabled decision support in real-world depression care [5]. Multimodal work integrating electrophysiological, neuroimaging, immune, and metabolic data further suggests a transition from simple outcome prediction toward treatment selection [6]. At the same time, treatment-resistant depression is biologically heterogeneous, and inconsistent phenotyping likely constrains reproducibility, external validity, and clinical applicability [7].

The final section addresses child and adolescent psychiatry. In this area, the strongest evidence for difficult-to-treat depression remains strategy-based rather than algorithmic, as shown in TORDIA, where medication switch combined with cognitive behavioral therapy outperformed medication switch alone [8]. A central take-home message of this presentation is that AI-assisted psychopharmacology should currently be viewed not as a replacement for clinician judgment, but as a structured decision-support approach that may refine treatment selection in complex and treatment-resistant cases. At present, the most clinically mature personalization tools remain careful phenotyping and pharmacogenomics, whereas AI-based models are best understood as emerging tools for treatment ranking, response prediction, and stratification. Their future value will depend on robust validation, interpretability, fairness, and integration with real-world clinical decision making. The future of psychopharmacology is likely to be neither purely algorithmic nor purely intuitive, but a clinically grounded partnership between expert judgment and data-informed personalization

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Keywords: Artificial intelligence, child and adolescent psychiatry, depression, mental health, psychiatry, treatment resistance
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0478 - Neurobiological Foundations of Antisocial Behavior in Juvenile Offenders

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Antisocial behavior in children and adolescents should be understood within a developmental framework that extends beyond social adversity alone. Recent literature increasingly supports the view that juvenile offending emerges from the interaction of environmental exposures, neurodevelopmental processes, and biologically mediated vulnerabilities that shape emotional regulation, reinforcement learning, empathy, and behavioral control. Rather than reflecting a single causal pathway, antisocial behavior appears to arise from multiple developmental trajectories in which neural systems remain continuously influenced by family context, trauma, stress, and broader ecological conditions. This "embedded brain" perspective is particularly useful in explaining why some youths exposed to adversity develop persistent antisocial patterns while others do not.

At the neurobiological level, growing evidence implicates alterations in frontolimbic and paralimbic circuits involved in impulse regulation, affective responsivity, moral learning, and reward–punishment processing. Dysfunction involving the prefrontal cortex, orbitofrontal cortex, amygdala, hippocampus, and thalamic networks may contribute to deficits in inhibitory control, reduced sensitivity to distress cues, impaired empathic processing, and maladaptive decision-making. Importantly, a large 2024 coordinated mega-analysis from the ENIGMA Antisocial Behavior working group reported widespread cortical and subcortical structural differences in youths with conduct disorder, including lower cortical surface area and reduced subcortical volumes in regions such as the amygdala, hippocampus, and thalamus. These findings reinforce the idea that antisocial behavior in youth is associated with distributed brain-system alterations rather than isolated regional abnormalities.

This neurodevelopmental model also aligns with clinical evidence showing that conduct disorder and related antisocial phenotypes are highly heterogeneous. Variability in age at onset, limited prosocial emotions/callous-unemotional traits, emotional dysregulation, and psychiatric comorbidity suggests that juvenile offenders do not constitute a biologically uniform group. Instead, neurobiological vulnerability likely interacts with temperament, social learning, trauma exposure, and developmental timing to produce distinct antisocial profiles.

Among environmental contributors, childhood maltreatment deserves particular emphasis because of its association with altered neurodevelopment and severe affective-interpersonal traits. A 2023 meta-analysis demonstrated a significant positive association between childhood maltreatment and callous-unemotional traits in youth, supporting the view that early adversity may shape neurocognitive and emotional pathways relevant to persistent antisocial behavior. Taken together, current evidence supports a multidimensional neuropsychiatric understanding of juvenile offending that integrates brain development, stress-related adaptation, and gene–environment interplay. Importantly, such a framework should not be interpreted deterministically; rather, it may help refine early identification, prevention, and individualized intervention strategies in at-risk youth.

Keywords: Antisocial behavior, Conduct disorder, Juvenile delinquency

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0479 - The Tired Patient: Understanding and Differentiating Sleep Complaints in Psychiatry Clinic

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Sleep-related complaints represent a ubiquitous yet diagnostically challenging presentation in psychiatric practice, with patients frequently describing themselves as "tired" without precise phenomenological characterization. This symposium addresses the critical clinical imperative to systematically differentiate fatigue, excessive daytime sleepiness (EDS), and lethargy in patients with major depressive disorder (MDD), bipolar disorder, and anxiety disorders, while delineating primary sleep pathology from secondary manifestations and iatrogenic medication effects. Accurate phenotypic classification of these overlapping yet neurobiologically distinct constructs is essential for targeted therapeutic intervention and improved functional outcomes. Supporting this need for precision, contemporary evidence demonstrates that fatigue, EDS, and lethargy constitute phenomenologically and pathophysiologically discrete entities that are frequently conflated in clinical assessment. Fatigue manifests as subjective exhaustion and diminished energy without obligate sleep propensity, whereas EDS represents objective sleep tendency during wakefulness, and lethargy encompasses psychomotor retardation with cognitive slowing [1]. In MDD and bipolar depression, residual fatigue persists in 40-60% of patients despite mood symptom remission, representing a distinct treatment target independent of sleep architecture disturbances [1]. Furthermore, hypersomnolence complaints in mood disorders frequently reflect circadian dysregulation, sleep inertia, or medication-induced sedation rather than true hypersomnia, necessitating objective polysomnographic and multiple sleep latency testing for diagnostic precision [2]. Anxiety disorders present unique challenges, with hyperarousal-driven insomnia coexisting with subjective fatigue secondary to chronic sleep deprivation and autonomic dysregulation. Primary sleep disorders—including obstructive sleep apnea, restless legs syndrome, and circadian rhythm disorders—demonstrate elevated prevalence in psychiatric populations yet remain systematically underdiagnosed, confounding treatment response and perpetuating disability. Additionally, psychotropic medications exert complex effects on sleep architecture and daytime alertness: selective serotonin reuptake inhibitors may induce or exacerbate REM sleep behavior disorder and periodic limb movements; atypical antipsychotics frequently cause sedation through histaminergic antagonism; and benzodiazepines suppress slow-wave sleep while paradoxically inducing daytime fatigue [3]. Given these complexities, the clinical differentiation of sleep complaints requires structured assessment incorporating subjective scales, objective actigraphy, and when indicated, formal sleep laboratory evaluation. Clinicians must systematically evaluate temporal patterns, precipitating factors, and functional consequences to distinguish primary psychiatric symptoms from comorbid sleep disorders and medication adverse effects. The bidirectional relationship between sleep disturbances and psychiatric illness necessitates integrated treatment approaches that address both domains concurrently. Failure to recognize primary sleep pathology in psychiatric patients results in treatment resistance, polypharmacy escalation, and persistent functional impairment. Conversely, attributing all sleep complaints to underlying psychiatric illness may delay the diagnosis of life-threatening conditions such as severe obstructive sleep apnea. Therefore, medication selection must balance therapeutic efficacy against sleep-wake cycle disruption, with consideration of timing, dosing strategies, and adjunctive sleep-promoting interventions. Ultimately, the "tired patient" in psychiatry represents a complex diagnostic challenge requiring nuanced differentiation of fatigue, EDS, and lethargy across MDD, bipolar disorder, and anxiety disorders. Systematic assessment protocols that distinguish primary sleep disorders from secondary manifestations and iatrogenic effects are essential for precision psychiatry. Clinicians must integrate subjective phenomenology with objective sleep metrics and maintain vigilance for comorbid sleep pathology to optimize treatment outcomes and functional recovery.

Keywords: Fatigue, excessive daytime sleepiness, major depressive disorder, bipolar disorder, sleep disorders, psychopharmacology

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0480 - Psychopharmacological Management of Impulsivity, Aggression, and Comorbidities in Juvenile Offenders: Evidence-Based Approaches

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Psychiatric comorbidity is highly prevalent among juvenile offenders and plays a central role in the emergence, persistence, and clinical severity of impulsivity, aggression, and behavioral dyscontrol. Conditions such as Attention-Deficit/Hyperactivity Disorder (ADHD), Conduct Disorder, substance use disorders, trauma-related psychopathology, and mood dysregulation frequently accompany antisocial and delinquent behaviors, often complicating both assessment and intervention. In this context, psychopharmacology should not be viewed as a treatment for delinquency itself, but rather as a clinically targeted approach to the psychiatric symptom domains and neurobehavioral disturbances that contribute to functional impairment, high-risk behavior, and resistance to rehabilitation.

From a neurobiological perspective, impulsive aggression and behavioral dysregulation in this population are closely linked to abnormalities in dopaminergic and serotonergic neurotransmission, disrupted reward processing, deficits in executive control, and impaired affect regulation. These mechanisms are also shaped by developmental adversity, trauma exposure, and ongoing environmental stressors, creating complex clinical phenotypes in which irritability, emotional lability, impulsivity, and aggression frequently overlap. Such heterogeneity makes treatment selection particularly challenging and underscores the need for individualized pharmacological decision-making based on symptom profile, comorbidity pattern, developmental stage, and contextual risk factors.

Within current clinical practice, stimulants occupy an important place in the management of youths with ADHD-related impulsivity, inattention, and behavioral disinhibition, particularly when executive dysfunction contributes to aggression or poor behavioral control. Atypical antipsychotics are more often considered in the short-term management of severe aggression, irritability, and affective dysregulation, especially in cases where behavioral symptoms are intense, persistent, and insufficiently responsive to psychosocial interventions alone. Mood stabilizers may also be used in selected patients with marked emotional lability, recurrent aggression, or co-occurring mood symptoms, although their place in treatment is often more individualized and clinically nuanced.

At the same time, psychopharmacological treatment in juvenile offenders remains clinically and ethically complex. The evidence base is uneven across diagnostic groups, some prescribing practices rely on limited or off-label data, and the risks of adverse effects, polypharmacy, and poor adherence are particularly salient in this vulnerable population. These challenges may be further amplified by substance misuse, diagnostic overlap, unstable family or institutional environments, and the danger of using medication primarily as a tool of behavioral suppression rather than as part of a clearly formulated therapeutic plan.

A rational approach therefore requires that medication use be anchored in careful diagnostic assessment, explicit target symptoms, ongoing safety monitoring, and integration with psychosocial, family-based, trauma-informed, and rehabilitative interventions. Seen in this light, psychopharmacology has a meaningful but circumscribed role in juvenile forensic mental health: not as a standalone solution, but as one component of a broader, developmentally sensitive, and ethically grounded treatment framework.

Keywords: Aggression, Juvenile offenders, Psychiatric comorbidity, Psychopharmacology

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0481 - Psychotherapy and the placebo effect

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Psychotherapy refers to therapeutic interventions grounded in psychological theories and delivered by trained professionals, and it is currently considered an effective and widely accepted treatment for many psychiatric disorders.Placebo refers to interventions that are physically and pharmacologically inert and do not exert a direct therapeutic effect on the body(1).However, the placebo effect describes improvements that arise not from the physical properties of a treatment but from factors such as the patient's expectations, the therapeutic context, and the relationship established with the clinician (2).

Psychotherapy operates primarily through mechanisms derived from psychological theories rather than pharmacological or physiological processes.For this reason, the concept of placebo used in drug trials is difficult to apply to psychotherapy.If placebo effects are defined as outcomes produced by factors other than the physical properties of a treatment, the effects of psychotherapy—being inherently psychological—could theoretically be classified as placebo effects.This paradox has led to debates about whether the outcomes of psychotherapy should be interpreted as the result of specific therapeutic mechanisms or as reflections of nonspecific factors such as expectations, therapeutic alliance, and treatment context.Attempts to address this issue have largely involved the development of "sham psychotherapies" or psychological placebo interventions within randomized controlled trials; however, these efforts have been challenged by conceptual ambiguities and methodological limitations (3).

In psychotherapy research, distinguishing specific factors from nonspecific factors in psychotherapy is particularly difficult.Many elements that are considered nonspecific in pharmacological studies actually represent core components of psychotherapy.These factors are shared across different therapeutic approaches and may substantially contribute to treatment outcomes(4).For this reason, placebo-controlled designs that are effective in pharmacological research cannot be easily implemented in psychotherapy studies.

In conclusion, designing a placebo in psychotherapy is highly challenging. Several factors contribute to this difficulty, including the impossibility of implementing double-blind procedures in psychotherapy research, the challenge of reliably mimicking the structure of psychotherapeutic interventions as placebo conditions, and the difficulty of clearly separating specific factors from nonspecific factors(4,5).Psychotherapy has been shown to be superior to placebo; however, the potential contribution of placebo effects should also be considered.Evidence from studies using different placebo methodologies suggests that the effects of well-designed placebo interventions may approach those of active treatments (5).

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0482 - Placebo Overview in Psychiatry

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In psychiatry, placebo has long been considered a pharmacologically inert control intervention used in randomized controlled trials to compare active treatments. However, growing evidence suggests that placebo responses can produce meaningful psychobiological effects across different psychiatric disorders (1,2). Therefore, the magnitude of the placebo response has become an important factor in interpreting treatment efficacy and in the design of clinical trials (3).

Recent studies suggest that the placebo effect is not uniform across psychiatric disorders. Placebo responses are generally higher in mood disorders such as major depressive disorder and bipolar disorder than in schizophrenia (3, 4). In addition, placebo responses tend to be stronger in children and adolescents compared with adults. In depression trials, this strong placebo response can reduce the difference between drug and placebo, which may complicate the interpretation of clinical results (5).

Several meta-analyses have examined factors that influence placebo response. These studies show that lower baseline symptom severity, more recent study periods, and unbalanced randomization favoring the active drug are associated with higher placebo responses. However, reliable individual predictors of placebo response are still limited (3, 4).

Experimental and neuroimaging studies also suggest that placebo effects have a biological basis. Placebo responses appear to be related to treatment expectations, learning processes, and emotional regulation. These processes are associated with changes in several neurotransmitter systems, particularly dopaminergic reward pathways and endogenous opioid systems (1, 2).

In conclusion, the placebo response can make it more difficult to accurately evaluate treatment efficacy in psychiatric research. At the same time, it provides important insights into therapeutic processes in clinical practice. Understanding placebo mechanisms may help improve treatment outcomes by optimizing the patient–physician relationship, managing expectations, and improving the therapeutic context. Future research clarifying the biological mechanisms and individual factors underlying placebo response may help improve the design of clinical trials and support the development of more personalized treatment approaches (1–3).

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Keywords: Placebo, placebo effect, psychiatry

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0483 - Neurobiological and Genetic Underpinnings of Ultra-High Risk for Psychosis

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Ultra-high risk for psychosis (UHR) describes a state in which individuals do not yet meet the diagnostic criteria for a psychotic disorder but have a markedly elevated risk of transitioning to psychosis. The UHR state is typically defined based on three main clinical criteria groups. The first includes Attenuated Psychotic Symptoms, characterized by paranoid or referential thoughts and perceptual abnormalities that occur with lower intensity than full-blown psychotic symptoms. The second group consists of Brief Limited Intermittent Psychotic Symptoms, in which psychotic-level symptoms emerge but remit spontaneously within a few days. The third group involves individuals with either a first-degree family history of psychosis or schizotypal personality disorder, accompanied by a significant decline in social or academic functioning within the past year. Although rates vary across samples, approximately 30% of individuals meeting UHR criteria develop a psychotic disorder over time (1). Identification of this risk state has therefore become a central focus of early detection and intervention programs. Accordingly, extensive efforts have been directed toward identifying neurobiological and genetic differences between UHR individuals and healthy controls, and between UHR individuals who later convert to psychosis and those who do not.

Given that one of the UHR subgroups is partly defined by familial risk (a history of psychosis in first-degree relatives combined with functional decline), investigating the genetic liability associated with UHR is particularly important. Accumulating evidence suggests that vulnerability to psychosis is shaped by an interaction between genetic predisposition and neurodevelopmental processes. Genome-wide association studies have demonstrated that psychosis has a polygenic architecture involving numerous susceptibility genes affecting synaptic plasticity, dopaminergic signaling, and neurodevelopmental pathways. Studies also show that the polygenic risk score for schizophrenia is higher in UHR individuals than in healthy controls, and even higher in those who later convert to psychosis (2). Beyond polygenic risk, candidate gene studies suggest that variations in NRG1 (3) and DAOA (4) may be associated with the transition from UHR to psychosis.

Neuroimaging studies have also investigated the neurobiological correlates of UHR. A systematic review reported increased volumes in the bilateral median cingulate cortex, right fusiform gyrus, left superior temporal gyrus, and right thalamus, whereas reduced gray matter volumes were observed in the right gyrus rectus and bilateral superior frontal gyrus (5). In a large structural neuroimaging study, individuals at UHR who later converted to psychosis showed reduced cortical thickness in the bilateral paracentral, right superior temporal, and left fusiform regions compared with both healthy controls and non-converting UHR individuals (6). Studies examining white matter integrity revealed microstructural abnormalities in major association tracts, particularly the superior longitudinal fasciculus, inferior longitudinal fasciculus, and inferior fronto-occipital fasciculus (7). Electrophysiological research has shown that early stages of schizophrenia and UHR are associated with increased delta and gamma band power, sensory gating deficits measured by P50 and N100, and reduced mismatch negativity and P300 amplitudes (8).

Taken together, genetic and neurobiological findings indicate that the UHR state reflects heightened vulnerability to psychotic disorders and represents a critical window for early detection and preventive interventions.

Keywords: Ultra high risk for psychosis, genetic, neuroimaging

0485 - PTSD and Complex PTSD in Children and Adolescents: Assessment and Measurement Tools

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Traumatic experiences during childhood and adolescence are common worldwide and represent a major public health concern. Epidemiological studies indicate that a substantial proportion of children are exposed to at least one potentially traumatic event during development, and such exposures may lead to a range of psychological outcomes, including Post-Traumatic Stress Disorder (PTSD) and, in some cases, Complex Post-Traumatic Stress Disorder (CPTSD) (Alisic et al., 2014). Diagnostic conceptualizations of PTSD differ between classification systems. While DSM-5 defines PTSD across four symptom clusters including re-experiencing, avoidance, negative alterations in cognition and mood, and hyperarousal (American Psychiatric Association, 2013), ICD-11 adopts a more parsimonious model consisting of three core symptom clusters: re-experiencing, avoidance, and a persistent sense of current threat (World Health Organization, 2019).

Importantly, ICD-11 also introduces Complex Post-Traumatic Stress Disorder (CPTSD) as a distinct diagnostic entity characterized by the core PTSD symptoms together with disturbances in self-organization, including affect dysregulation, negative self-concept, and relational disturbances (World Health Organization, 2019; Brewin et al., 2017). These conceptual developments have stimulated research examining the latent structure of PTSD symptoms, with factor analytic studies investigating whether trauma-related symptoms are better represented by alternative diagnostic models such as the ICD-11 three-factor structure or broader multidimensional frameworks (Hyland et al., 2017).

In parallel, advances in trauma assessment research have highlighted the importance of operationalizing diagnostic constructs through structured measurement instruments that capture both core PTSD symptoms and disturbances associated with complex trauma exposure. These instruments aim to assess trauma-related symptom patterns in a developmentally sensitive manner and to reflect the diagnostic frameworks proposed by both DSM-5 and ICD-11. Another aspect of trauma assessment concerns the use of dimensional versus categorical approaches in the evaluation of trauma-related symptoms. While diagnostic systems rely on categorical thresholds for PTSD diagnoses, many contemporary measurement instruments adopt dimensional scoring strategies that allow for a more nuanced evaluation of symptom severity.

This presentation will focus on assessment and measurement approaches used to evaluate PTSD and CPTSD in children and adolescents. Particular attention will be given to how different measurement strategies operationalize DSM-5 and ICD-11 diagnostic constructs and how these approaches contribute to the characterization of trauma-related symptom patterns in developmental populations.

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Keywords: Trauma, traumatic event, pstd, complex ptsd

0486 - AI-Driven Self-Management and Monitoring Systems in Daily Life

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Health systems worldwide are under pressure internationally from the increasing chronic disease burden, shortages in the workforce, and longstanding disparities in access to care. These converging challenges have made the introduction of artificial intelligence (AI)-based solutions and digital health tools into the healthcare sector more and more unavoidable (1). Psychiatric care is one of the areas where change could be particularly dramatic. Conventional psychiatric evaluation largely provides a cross-sectional overview observed at the time of the clinical visit. Therefore, it is naturally constrained as a tool for identifying symptom alterations, behavioral triggers, and harbingers as they occur in the daily environment. AI-powered digital platforms are being proposed as potential technologies to close this crucial gap. Their realistic potential impact on general psychiatric practice can be grouped under a few salient headings: decreasing the clinical load by automating the routine documentation, screening instruments, and scheduling of appointments; early warning systems for proactive detection of symptom worsening; systematic monitoring of treatment compliance; and provision of tailored psychoeducation according to the needs of individual patients. A recently published systematic review on AI in mental health showed that machine learning-based monitoring tools can predict treatment response and monitor an ongoing prognosis in several psychiatric disorders (2). Supporting this, a feasibility study conducted in an adolescent population found that smartphone-based digital phenotyping for mental health risk prediction is feasible and acceptable to young users, with completion and engagement rates approaching encouraging levels (3). In the case of mood disorders, applications allowing the user to record daily mood changes, sleep time, medication use, and some specific symptoms, in addition to wearable devices monitoring sleep quality, recovery indices, and body temperature variability, could provide both clinicians and patients with the unique chance of performing real time and continuous psychiatric follow-up in the inter-visit period. This ecological methodology for symptom monitoring has great potential to positively impact clinical decision-making and ultimately improve time to treatment. These tools appear to have a positive effect based on the current evidence; though, their correct application needs to be determined by care needs of the individuals and the availability of suitable clinical and other services. Toward this end, and to facilitate that AI-based self-management solutions can be enabled, interdisciplinary cooperation between healthcare providers, technology developers, and policy makers is needed, and these technologies are to be developed and implemented in an ethics-informed manner and in alignment with existing healthcare system infrastructures.

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Keywords: Artificial intelligence, mental health monitoring, digital health, psychiatric monitoring, mood disorders

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0487 - PTSD and CPTSD in Children and Adolescents: Emotional Difficulties

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Children and adolescents represent a particularly vulnerable population when it comes to exposure to potentially traumatic events (PTEs) (1). Adverse childhood experiences — ranging from abuse, neglect, and domestic violence to natural disasters, and loss — are far more common than is often recognized, and their consequences for psychological development can be profound and enduring. The responses that emerge following traumatic exposure vary considerably across individuals; while some children and adolescents demonstrate resilience or experience only mild, transient symptoms, others go on to develop severe, persistent, and functionally impairing psychopathology.

Despite this clinical reality, trauma-related disorders in children and adolescents remain significantly underidentified. Although trauma- and stressor-related disorders as defined in the DSM have gained considerable traction in clinical practice — likely reflecting greater familiarity with DSM nosology in many training contexts —, the ICD-11 (2) diagnoses of post traumatic stress disorder (PTSD) and, in particular, complex post traumatic stress disorder (CPTSD) remain comparatively underrecognized in pediatric and adolescent populations. Increasing diagnostic awareness and clinical literacy around these disorders is therefore not merely an academic concern, but a pressing public health imperative.

The diagnosis of CPTSD becomes relevant in children and adolescents who have been exposed to chronic, repeated, and inescapable PTEs — circumstances that are, by nature, more likely to occur within caregiving or attachment relationships, such as ongoing abuse or neglect by a primary caregiver. In these cases, clinicians may observe not only the core PTSD symptom clusters of re-experiencing, avoidance, and hyperarousal, but also a distinct constellation of disturbances in self-organization (DSO): persistent negative self-perception, pronounced difficulties in emotion regulation, and significant challenges in initiating and sustaining meaningful interpersonal relationships.

It is precisely this broader pattern of disruption — affecting not just trauma-related symptoms but the very architecture of the developing self — which serves as the defining differentiator between CPTSD and PTSD, making it imperative for clinicians to recognize the distinction. In younger populations, findings suggest that emotion dysregulation is present in up to 60% of community-based youth with histories of interpersonal trauma. While direct pediatric meta-analytic data remains limited, evidence from trauma-exposed adult populations has consistently demonstrated that post-traumatic stress symptoms are substantially associated with broader difficulties in emotion regulation (3). Emotion dysregulation should not be conceptualized as an isolated symptom, but rather as a pervasive phenomenon that impacts all domains of functioning in children and adolescents—ranging from peer relationships and academic achievement to family dynamics—ultimately compounding long-term mental health risks.

This panel aims to bridge the gap between research and clinical practice by focusing on the differential diagnosis of PTSD and CPTSD in children and adolescents, with particular attention to the emotion regulation difficulties that characterize both conditions. By fostering greater diagnostic clarity and a deeper understanding of the developmental dimensions of trauma, we hope to contribute meaningfully to ensuring that children and adolescents receive the accurate diagnoses — and the evidence-based, developmentally sensitive interventions they need and deserve.

Keywords: Trauma, child and adolescent, post traumatic stress disorder, complex post traumatic stress disorder, emotional difficulties

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0488 - Reconsideration of Diagnosis in Treatment Resistance Do we call Adults With Autism Psychosis?

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Autism spectrum disorder (ASD) and psychotic disorders are distinct diagnostic entities, yet differentiating between them in clinical practice can be highly challenging. This difficulty arises from both symptom overlap and the possibility that the two conditions may co-occur in the same individual. For this reason, clinicians increasingly emphasize the importance of using clear terminology such as “autism and psychosis” or “ASD with comorbid psychosis,” rather than combining them into a single label. Such wording reflects the fact that ASD and psychosis are separate conditions with different developmental trajectories, clinical characteristics, and treatment implications.

One of the main sources of diagnostic confusion is the overlap in social and behavioral presentation. Individuals with ASD may show poor eye contact, limited emotional reciprocity, social withdrawal, unusual speech patterns, and rigid or repetitive behaviors. Similar features may also be observed in schizophrenia spectrum disorders, particularly in the form of negative symptoms such as affective flattening, reduced speech, and diminished social engagement. As a result, long-standing autistic traits may be misinterpreted as psychotic symptoms, while early psychosis may be overlooked in autistic individuals.

Developmental history is therefore one of the most important elements in differential diagnosis. ASD is a neurodevelopmental condition with onset in early childhood, characterized by persistent differences in social communication and restricted or repetitive patterns of behavior. In contrast, psychosis typically emerges later, often during adolescence or early adulthood, and usually involves a clear deterioration or change from previous baseline functioning. Identifying whether a symptom represents a lifelong pattern or a new clinical change is essential for accurate diagnosis.

Another diagnostic challenge concerns unusual beliefs, sensory experiences, and self-talk. In ASD, sensory sensitivities, intense preoccupations, imaginative experiences, or literal interpretations of reality may resemble psychotic phenomena. However, psychosis is more likely to involve persistent hallucinations, fixed delusional beliefs, disorganized thought processes, and impaired reality testing. Careful exploration of the phenomenology, context, and meaning of these experiences is necessary.

A thorough psychiatric assessment should include developmental history, collateral information from family members, clinical observation, and, when needed, structured tools for both ASD and psychosis. Rather than relying only on overlapping social deficits, clinicians should focus on disorder-specific features and longitudinal course. Distinguishing psychosis from autism, and recognizing their possible co-occurrence, is crucial for correct diagnosis, individualized treatment, and improved long-term outcomes.

Keywords: Autism spectrum disorder, Psychosis, Differential diagnosis

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0489 - Artificial Intelligence and the Future of Addiction

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The rapid integration of artificial intelligence (AI) technologies into digital ecosystems is playing an increasingly central role in monitoring, guiding, and optimizing human behavior. In particular, recommendation systems and reinforcement learning algorithms can reinforce problematic use and addiction-like behavioral patterns through reward-based mechanisms by creating feedback loops aimed at maximizing user interaction. Parallel to this, AI-based chatterboxes and virtual companionship systems are fostering new forms of digital intimacy and attachment by establishing emotional interactions with individuals; while this can provide supportive effects, especially for individuals experiencing loneliness and social isolation, it also carries the risk of over-attachment and digital addiction. On the other hand, deepfake technologies pose serious ethical and psychosocial threats such as identity manipulation, privacy violations, and non-consensual content production; while algorithmic gambling and gamified reward systems increase the risks associated with addiction processes by manipulating user behavior through behavioral economics-based design strategies. In contrast, the digital phenotyping approach; Smartphone sensor data, movement patterns, and big data derived from online interactions offer promising biomarkers for the early detection and monitoring of depression and other psychiatric conditions. This panel aims to discuss the bidirectional impact of AI-powered digital systems on addictive behaviors across four key axes: (1) behavior optimization through recommendation systems and reinforcement learning, (2) AI companion relationships and digital proximity, (3) deepfake and algorithmic behavior manipulation, and (4) digital phenotyping and mental health prediction. Consequently, AI technologies, while generating novel environmental determinants that increase addiction risks, also offer innovative opportunities for psychiatric assessment, monitoring, and early intervention. Understanding this complex interaction requires critical interdisciplinary approaches, ethical guidelines, and clinical integration strategies that integrate psychiatry, behavioral sciences, and AI research.

Keywords: Artificial Intelligence, Behavioral Addiction, Digital Phenotyping

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0490 - Postpartum Paternal Depression: An Overlooked Public Health Concern

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The transition to parenthood represents a significant life event that can profoundly affect the mental health of both mothers and fathers. While postpartum maternal depression has been extensively studied, postpartum paternal depression (PPPD) remains underrecognized despite its substantial impact on family dynamics and child development. The postpartum period, defined as the first 12 months following childbirth, is associated with increased psychological vulnerability in fathers due to biological, psychological, and social changes. Epidemiological studies suggest that the prevalence of PPPD ranges between 3% and 5%. Interestingly, some studies have suggested that the risk of depression among fathers in the postpartum period may not differ significantly from that of non-fathers, indicating that additional contextual and relational factors may play a critical role. A wide range of risk factors has been identified for PPPD. These include a personal or maternal history of depression, unplanned pregnancy, prenatal anxiety or depressive symptoms in the father, poor physical health, unemployment, substance use, and exposure to adverse life events. Psychosocial factors such as low perceived social support, marital dissatisfaction, high perceived stress, and difficulties in father–infant bonding are also strongly associated with increased risk. Additionally, factors related to the infant, such as sleep disturbances and preterm birth, as well as contextual stressors like economic hardship, further contribute to vulnerability. The postpartum period between 3 to 6 months appears to be particularly critical for the emergence of paternal depressive symptoms. From a neurobiological perspective, hormonal changes may play a role in PPPD. Studies have indicated that decreases in testosterone levels following childbirth may be associated with increased depressive symptoms in fathers. These biological changes, combined with psychosocial stressors, may contribute to the development of depressive symptomatology. Clinically, PPPD presents with symptoms similar to those observed in depression among men in the general population. These include low mood, irritability, anxiety, anhedonia, impaired concentration, feelings of worthlessness or excessive guilt, fatigue, and sleep disturbances. However, depressive symptoms in men may sometimes manifest as irritability, anger, or behavioral withdrawal, which can lead to underdiagnosis. Screening for PPPD is increasingly recommended. Early identification is essential for timely intervention and prevention of adverse outcomes. Management strategies for PPPD are generally similar to those used in major depressive disorder. However, interventions tailored specifically for fathers may provide additional benefits. Psychosocial interventions, including parenting skills training, and group-based support programs, have demonstrated promising results. Recent trials have shown that such interventions can significantly reduce depressive symptoms in fathers while also improving child emotional and social development. Furthermore, psychoeducation programs, co-parenting support, and interventions targeting infant sleep regulation may enhance paternal mental health outcomes. Paternal depression can negatively affect father-infant bonding, parenting quality, and marital relationships. Importantly, it has also been associated with adverse developmental outcomes in children, including cognitive delays, language difficulties, and increased risk of psychopathology. In conclusion, postpartum paternal depression is an important yet often neglected mental health issue with significant implications for families and society. Increasing awareness, improving screening practices, and developing father-specific interventions are essential steps toward addressing this condition effectively.

Keywords: Father–infant bonding, Fatherhood transition, Perinatal mental health, Postpartum paternal depression

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0491 - Intolerance of Uncertainty as a Transdiagnostic Factor and Intolerance of Uncertainty in Children with Anxiety Disorders

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In today's rapidly changing and increasingly complex world, uncertainty has become an unavoidable aspect of everyday life. Individuals are frequently required to cope with ambiguous, unpredictable, and uncontrollable situations across academic, social, familial, and personal domains. While uncertainty may be distressing for many, for some it constitutes a particularly aversive psychological state associated with marked discomfort, heightened vigilance, and maladaptive emotional and behavioral responses. In this context, intolerance of uncertainty (IU) has emerged as a highly relevant construct in contemporary psychopathology research. Since gaining prominence in the mid 1990s, IU has been broadly defined as a dispositional inability to endure the aversive response triggered by the perceived lack of sufficient information and the prospect of uncertain future events. Over time, IU has come to be recognized not only as a feature of specific psychiatric disorders, but also as a transdiagnostic construct implicated in the development and maintenance of a wide range of psychopathological conditions.

This transdiagnostic perspective is particularly important in child and adolescent psychiatry, where emotional and behavioral symptoms often overlap and where early vulnerability markers may inform prevention and intervention efforts. Developmental characteristics of childhood and adolescence render uncertainty especially salient during these periods. Children and adolescents with elevated IU are more likely to perceive uncertain or ambiguous situations as threatening, even in the absence of objective danger. This tendency may manifest as excessive worry, reassurance seeking, avoidance behaviors, indecisiveness, and difficulty tolerating everyday unpredictability. Such responses may impair adaptive functioning, disrupt peer and family relationships, and reinforce anxiety related cognitions over time.

Notably, IU appears to play a central role in both the emergence and maintenance of anxiety disorders in children and adolescents. Higher levels of IU have been associated with a greater tendency to overestimate threat, underestimate coping abilities, and experience persistent distress in situations involving novelty, ambiguity, or incomplete information. In addition, IU may interact with other vulnerability factors, such as negative affectivity, cognitive biases, and emotion regulation difficulties, thereby increasing susceptibility to anxiety symptoms and related functional impairment. From a clinical perspective, recognition of the role of IU in childhood anxiety disorders has important implications for both assessment and treatment planning. Evaluating IU may contribute to a more comprehensive understanding of the cognitive mechanisms underlying anxiety symptoms and may help identify children at risk for persistent presentations. Moreover, interventions that directly target IU, such as cognitive restructuring of maladaptive threat related beliefs, graduated exposure to uncertainty, enhancement of emotional tolerance, and the development of flexible coping strategies, may improve treatment outcomes beyond symptom focused approaches alone. Integrating IU focused strategies into evidence based psychotherapeutic interventions may therefore strengthen treatment efficacy and promote long term resilience.
In conclusion, IU is a clinically meaningful and developmentally relevant transdiagnostic factor in child and adolescent mental health. Its prominent role in childhood anxiety disorders underscores the need for greater attention to uncertainty related processes in both research and clinical practice.

Keywords: Intolerance of Uncertainty, Transdiagnostic Factor, Children, Anxiety Disorders

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0493 - Withdrawal Symptoms: Psychotic Prodrome, Depression, or Anxiety?

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Background: Social withdrawal is a common yet diagnostically complex presentation in child and adolescent psychiatry. Adolescents may disengage from peers, family interactions, and previously enjoyed activities for a variety of psychological reasons. Although withdrawal is frequently observed in mood and anxiety disorders, it may also represent an early indicator of emerging psychotic disorders. Because these conditions share overlapping clinical features during early stages, differentiating between them may be challenging for clinicians. Accurate identification of the underlying psychopathology is particularly important in adolescence, as early recognition and appropriate intervention may significantly influence long-term functional and clinical outcomes. In recent years, increasing attention has been given to transdiagnostic symptoms and the "gray zones" between psychiatric disorders. Withdrawal symptoms represent one such transdiagnostic feature, appearing across depressive disorders, anxiety disorders, and clinical high-risk states for psychosis. This presentation aims to review the clinical characteristics of withdrawal symptoms in adolescents and to discuss key clinical clues that may help differentiate psychotic prodrome from depressive and anxiety-related withdrawal.

Methods: This presentation is based on a narrative review of the current literature on social withdrawal in adolescents, with a focus on studies examining early psychosis, depressive disorders, and anxiety disorders. Relevant findings from clinical high-risk for psychosis research, developmental psychopathology, and adolescent mental health literature were examined. In addition, common clinical observations from child and adolescent psychiatric practice were integrated with existing evidence to highlight practical considerations in differential diagnosis.

Results: Withdrawal associated with psychotic prodrome is typically characterized by progressive social disengagement accompanied by functional decline, emerging negative symptoms, and subtle cognitive or perceptual changes. Adolescents at clinical high risk for psychosis may exhibit decreased motivation, diminished emotional expression, suspiciousness, or attenuated psychotic experiences. In contrast, withdrawal in depressive disorders is commonly linked to anhedonia, low energy, persistent sadness or irritability, and feelings of hopelessness. Adolescents with depression often withdraw because previously rewarding activities no longer produce pleasure. Withdrawal related to anxiety disorders, particularly social anxiety disorder, tends to be driven by fear of negative evaluation and avoidance of social situations rather than loss of interest. In these cases, adolescents may continue to experience enjoyment in activities when anxiety is reduced. Several clinical features may help differentiate these presentations, including the temporal course of symptoms, the presence of affective symptoms, cognitive or perceptual disturbances, and whether withdrawal reflects reduced motivation or avoidance of feared situations.

Conclusion: Withdrawal in adolescents represents a transdiagnostic symptom that may signal different underlying psychiatric conditions. Careful assessment of accompanying emotional, cognitive, and behavioral features is essential for accurate differential diagnosis. Recognizing whether withdrawal reflects psychotic risk, depressive pathology, or anxiety-related avoidance has important implications for early intervention strategies and treatment planning in child and adolescent psychiatry.

Keywords: Anxiety Disorders, Child and Adolescent Psychiatry, Depression, Psychotic Prodrome, Social Withdrawal

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0494 - Life Coherence and Mental Health

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Recent research on the impact of stress on mental health emphasizes the importance of focusing not only on psychological disorders but also on resilience. Resilience is defined as the process of successful adaptation and the capacity to emerge stronger when faced with adversity, trauma, threats, or significant stressors—such as family issues, serious health problems, or financial hardships. It is suggested that resilience factors contribute to the preservation or rapid recovery of mental health during or after exposure to stressors. Consequently, these factors represent potential targets for interventions aimed at enhancing resilience. One of the most significant evidence-based resilience factors is the Sense of Coherence (SOC).

Sense of Coherence, or "Life Coherence," is a psychological concept that explains the extent to which an individual perceives the world as meaningful, manageable, and comprehensible. SOC is the core component of the salutogenesis model developed by Aaron Antonovsky (1979, 1987), which aims to shift the focus from "risk and disease" to "health and well-being." In other words, unlike the traditional medical approach (pathogenesis) that focuses on the causes of illness, salutogenesis focuses on the origins of health and how it is sustained. Antonovsky defined the three dimensions that constitute the Sense of Coherence as follows:

1. Comprehensibility: The extent to which an individual cognitively perceives encountered situations as clear, ordered, and predictable.

2. Manageability: The degree to which an individual perceives their internal and external resources (personal skills, social support, etc.) as sufficient to meet demands and cope with challenges.

3. Meaningfulness: The extent to which an individual feels that life is emotionally meaningful and that certain problems are challenges worthy of investment rather than merely burdensome obstacles. According to Antonovsky, this is the most critical component; without meaning, the others cannot be sustained. These three sub-dimensions are hypothesized to form the foundation for successful coping with stressors. In essence, life coherence acts as a mental compass that enables individuals to focus on the question "How can I cope with this and what can I learn?" rather than "Why me?" when facing trauma or daily stress.

Consistent with theoretical expectations, various studies have demonstrated that SOC has strong correlations with health and health-related behaviors, serving as a predictor for psychological well-being, depression, and anxiety. In individuals with chronic illnesses or mental disorders, an increased sense of coherence can positively influence quality of life and treatment adherence. While numerous systematic reviews have shown a significant negative correlation between SOC and mental health issues in middle-aged and older adult populations, research focusing on younger age groups—where SOC development is presumed to be crucial—remains relatively scarce. Nonetheless, emerging evidence supports a negative relationship between SOC and mental health problems in youth as well. Furthermore, while SOC is generally accepted to develop from childhood through young adulthood and stabilize around age 30, it can still be strengthened throughout life through lifelong learning and therapy

Keywords: Comprehensibility, manageability, meaningfulness, resilience, sense of coherence

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0495 - Assessment Sleep Complaints Under Time Constraints: Selection and Application of Clinical Scales

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Sleep disturbances are among the most common complaints in psychiatric practice, yet their assessment is often inadequate in routine clinical encounters because of time constraints. In brief consultations, clinicians must adopt a practical and targeted approach focusing on core domains such as sleep onset latency, sleep maintenance, early morning awakening, total sleep duration, circadian pattern, and daytime consequences including fatigue and excessive sleepiness. However, unstructured clinical questioning may fail to capture the multidimensional nature of sleep disturbances.

Objective methods provide more detailed physiological information in sleep assessment. Polysomnography (PSG) remains the gold standard for evaluating sleep architecture and related physiological parameters. In addition, the Multiple Sleep Latency Test (MSLT), a daytime polysomnographic procedure, is particularly important in the diagnosis of disorders such as narcolepsy. Despite their diagnostic value, these methods are not widely applicable in routine psychiatric practice because of limited accessibility, high cost, and the impracticality of administering them to every patient presenting with sleep-related complaints.

In this context, validated clinical scales serve as a practical bridge between subjective clinical evaluation and objective measurement. Among the most commonly used instruments, the Pittsburgh Sleep Quality Index (PSQI) provides a multidimensional assessment of sleep quality, the Insomnia Severity Index (ISI) measures the severity and clinical impact of insomnia symptoms, and the Epworth Sleepiness Scale (ESS) evaluates daytime sleepiness. Owing to their ease of administration and strong psychometric properties, these scales are particularly useful in time-limited settings. Sleep diaries also remain valuable in routine clinical practice, especially for monitoring sleep patterns over time.

Beyond these commonly used measures, several additional instruments may enhance assessment in specific clinical contexts. The Athens Insomnia Scale (AIS) and the Sleep Condition Indicator (SCI) help evaluate insomnia symptoms within a diagnostic framework, while the PROMIS Sleep Disturbance Scale offers a more flexible and contemporary approach. In addition, the Sleep Hygiene Index (SHI) and the Pre-Sleep Arousal Scale (PSAS) are useful for assessing behavioral insomnia and anxiety-related sleep difficulties.

Sleep assessment should also be tailored to specific patient populations. In patients with depression and anxiety, sleep disturbances often occur as part of broader symptom clusters, whereas in older adults, sleep fragmentation and circadian rhythm changes require careful interpretation of standard measures. In chronic medical conditions, the Medical Outcomes Study (MOS) Sleep Scale helps evaluate the relationship between sleep and general health. In attention-deficit/hyperactivity disorder (ADHD), circadian preference, delayed sleep phase, and social jetlag may be assessed using the Morningness–Eveningness Questionnaire and the Munich Chronotype Questionnaire (MCTQ).

Recent advances in digital health technologies have expanded the possibilities for sleep assessment. Wearable devices and actigraphy-based systems provide longitudinal data through continuous monitoring of sleep patterns in real-life settings. However, these methods should be considered complementary rather than replacements for gold-standard assessments.

In conclusion, structured assessment of sleep complaints is essential in time-limited psychiatric practice. Appropriate selection and effective use of clinical scales can improve diagnostic accuracy, guide treatment planning, support research standardization, and ultimately enhance patient care.

Keywords: Sleep assessment, insomnia, Pittsburgh Sleep Quality Index, sleep scales

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0496 - An Integrated AI Ecosystem for Treatment-Resistant Cases: Collaboration Between Clinicians, Patients, and Technology

Yusuf Selman Çelik

Treatment-resistant psychiatric conditions—including treatment-resistant depression (TRD), refractory anxiety disorders, and persistent psychotic symptoms—affect approximately 30% of patients and remain a major challenge in contemporary psychiatry. Despite advances in pharmacotherapy and psychotherapy, many individuals do not achieve adequate remission, highlighting the need for more adaptive and personalized care models. Artificial intelligence (AI) offers an opportunity to move beyond fragmented approaches toward integrated, data-driven systems that combine clinical expertise with continuous patient data.

This panel explores the concept of an integrated AI ecosystem—a collaborative framework that brings together clinical decision support systems (CDSS), patient-facing digital tools, and clinician–AI partnerships—to improve outcomes in treatment-resistant psychiatric conditions.

The proposed ecosystem consists of three interconnected components. First, AI-powered clinical decision support systems use machine learning algorithms trained on multimodal data, including electronic health records, neuroimaging, and genomic information. These systems can help identify patterns of treatment resistance, predict individual response trajectories, and support more targeted treatment selection, reducing reliance on trial-and-error approaches. Second, patient-facing digital tools—such as AI-based conversational agents, digital therapeutics, and mobile applications—extend care beyond the clinic. Evidence suggests that these tools can significantly reduce depressive symptoms and psychological distress, particularly when they are multimodal, interactive, and embedded in daily digital environments. They also enhance accessibility and promote active patient engagement in treatment. Third, digital phenotyping enables continuous, passive monitoring of behavioral and physiological data through smartphones and wearable devices. This approach allows for real-time detection of symptom changes, early identification of relapse risk, and delivery of just-in-time adaptive interventions. When integrated with clinical assessments, these data support the development of dynamic and longitudinal patient profiles.

The integration of AI into psychiatric care raises important ethical challenges, including algorithmic bias, data privacy, and the risk of over-reliance on automated systems. Current regulatory frameworks remain insufficient to fully address these concerns, particularly in the context of generative AI. It is essential that the clinician–patient relationship remains central, with AI functioning as a supportive tool. Transparent reporting, informed consent, and clinician oversight are critical to ensuring safe and equitable implementation.

An integrated AI ecosystem has the potential to transform the care of treatment-resistant psychiatric conditions by enabling precision psychiatry, continuous monitoring, and scalable interventions. Future efforts should focus on rigorous clinical validation, interdisciplinary collaboration, and the development of clear ethical guidelines to ensure that AI enhances—rather than replaces—the human elements of psychiatric care.

Keywords: Artificial Intelligence, Clinical Decision Support Systems, Treatment-Resistant Depressive Disorder

AuthorToEditor: This panel addresses a highly relevant and evolving area in psychiatry by proposing an integrated AI ecosystem for treatment-resistant conditions. The topic is of significant clinical and academic importance, particularly in light of increasing interest in precision psychiatry and digital mental health interventions. The content is based on current literature and aims to provide a conceptual and practical framework for future research and clinical implementation. No conflict of interest is declared. ORCID: 0000-0002-1131-2814

0497 - ADHD Mothers: Management of ADHD During Pregnancy and Lactation

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As one of the most prevalent neurodevelopmental disorders, Attention-Deficit/Hyperactivity Disorder (ADHD) is characterized by core symptoms of inattention and/or hyperactivity-impulsivity that reach clinically impairing levels. Initially regarded as a childhood disorder predominantly affecting males, ADHD is now well-recognized in both adult men and women; however, women remain more likely to be undiagnosed (1). Despite its increasing recognition in women of reproductive age, managing ADHD during pregnancy and lactation remains a significant clinical challenge, especially as the prevalence of medication use has risen substantially in recent decades without a corresponding increase in clear, evidence-based guidelines (2).

Managing ADHD during pregnancy involves a complex risk-benefit analysis, as untreated symptoms are linked to adverse obstetric outcomes like pre-eclampsia and gestational hypertension, alongside significantly higher risks of comorbid postpartum depression and anxiety (3). Regarding pharmacological safety, while most ADHD medications are generally considered safe, some studies indicate a small but significant increase in the risk of specific malformations depending on first-trimester exposure (4). Crucially, while longitudinal data provide some reassurance by showing no significant adverse impact on long-term neurodevelopmental or growth outcomes in offspring (4), a substantial need for further high-quality research remains due to the overall scarcity of comprehensive evidence in the field. This highlights the importance of cautious and individualized clinical monitoring.

During the lactation period, clinical decisions must prioritize both maternal stability and neonatal safety by evaluating the transfer of medications into breast milk. While many ADHD medications are considered compatible with breastfeeding due to low relative infant doses, clinicians should maintain individualized monitoring for potential adverse effects such as irritability or sleep disturbances (5).

Focusing on the clinical dilemmas, pharmacological safety data, and multi-modal treatment approaches essential for supporting ADHD mothers during the perinatal and lactation periods, this presentation examines the critical balance between the risks of intrauterine and neonatal medication exposure and the clinical consequences of untreated maternal ADHD, which can profoundly impact obstetric outcomes, breastfeeding stability, and the fundamental quality of early parenting.

Keywords: Attention-deficit/hyperactivity disorder, lactation, pregnancy

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0498 - Neuroinflammatory Responses to Childhood and Adolescent Trauma

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Traumatic experiences are prevalent during childhood and adolescence. Such experiences have been associated with numerous mental health issues, most notably post-traumatic stress disorder (PTSD). In recent years, research has increasingly concentrated on the relationship between PTSD and inflammation. However, these investigations have predominantly focused on adult populations and have yielded inconsistent evidence. Additionally, there is an ongoing debate about the causality and direction of the relationship between PTSD and inflammatory processes. The purpose of this presentation is to examine the relationship between PTSD and inflammation in children and adolescents.

Keywords: Adolescence, childhood, inflammation, PTSD

0501 - Intolerance of Uncertainty and Post-Traumatic Stress Disorder in Children

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Introduction and Theoretical Framework

Post-Traumatic Stress Disorder (PTSD) in children is a complex condition that severely disrupts the developmental trajectory. While traditional models focus primarily on traumatic memory, recent research highlights Intolerance of Uncertainty (IU) as a fundamental catalyst for chronic symptomatology. IU is defined as a cognitive bias where a lack of information about the future is perceived as an inherent threat. In the aftermath of trauma, a child's once-stable world transforms into a landscape of unpredictable danger. This forces the child into a state of perpetual "preparedness" for non-existent threats, keeping the stress response active long after the traumatic event has concluded (Carleton, 2016).

Pathophysiological Mechanisms and Neural Circuitry

The intersection of IU and PTSD is rooted in the dysfunction of the brain's fear-processing architecture. In traumatized children, the distinction between "known threats" and "uncertainty" collapses due to the following neurobiological disruptions:

Amygdala-vPFC Disconnection: High IU is associated with a hyper-responsive amygdala. Under normal conditions, the ventromedial Prefrontal Cortex (vmPFC) provides "top-down" inhibition to soothe the amygdala. In pediatric PTSD, this functional connectivity is weakened; the vmPFC fails to regulate the amygdala, leaving the child in a state of emotional flooding. This deficit prevents the child from distinguishing between neutral ambiguity and genuine danger (Morriss & McSorley, 2019).

Impaired Prediction Error Processing: The human brain functions as a "prediction machine." Children with high IU struggle to learn that an uncertain situation has ended safely (fear extinction). This places an immense cognitive load on the Anterior Cingulate Cortex (ACC), manifesting clinically as hypervigilance and constant environmental scanning (Tanovic et al., 2018).

HPA Axis and Hippocampal Atrophy: Chronic uncertainty acts as a continuous stressor, keeping the Hypothalamic-Pituitary-Adrenal (HPA) axis in a state of overactivation. Prolonged exposure to glucocorticoids can be neurotoxic to the developing hippocampus. Consequently, the child becomes unable to distinguish between the "then and there" of the trauma and the "here and now" of a safe but uncertain present (Logue et al., 2018).

Therapeutic Strategies and Clinical Implications

Effective treatment for pediatric PTSD must transcend the traumatic narrative and address the underlying mechanism of IU:

1. IU-Enhanced Trauma-Focused CBT: Beyond traditional desensitization, "uncertainty exposures" are integrated. The goal is to shift the child's belief from "I must know everything to be safe" to "I can handle not knowing."

2. Acceptance and Commitment Therapy (ACT): ACT targets the experiential avoidance associated with IU. Children learn to observe their anxiety about the future without being paralyzed by it, pursuing valued actions despite inherent uncertainties (Sarıkoç & Uğur, 2024).

3. Parental Co-Regulation: Success is highly dependent on parental behavior. Parents are trained to resist providing "excessive reassurance," which reinforces the belief that uncertainty is unbearable, and instead model calm acceptance of the unknown.

Conclusion

Intolerance of Uncertainty serves as the "engine" that sustains PTSD symptoms. By targeting pathophysiological

pathways—specifically amygdala-PFC connectivity and prediction error processing—clinicians can provide children with the tools to navigate an inherently uncertain future with psychological resilience.

Keywords: Intolerance of Uncertainty, Post-Traumatic Stress Disorder, Children

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0502 - AI-Driven Prediction of Drug-Drug and Drug-Gene Interactions in Psychopharmacology: A New Era of Safety

Abas Haşimoğlu

Başakşehir Çam ve Sakura Şehir Hastanesi, Çocuk ve Ergen Ruh Sağlığı ve Hastalıkları Anabilim Dalı

Subject: The application of artificial intelligence (AI) and machine learning (ML) frameworks in predicting drug-drug interactions (DDIs) and drug-gene interactions (DGIs), with a specialized focus on psychiatric pharmacotherapy and precision medicine.

Background: Adverse drug reactions (ADRs) triggered by DDIs and DGIs represent a formidable clinical challenge, particularly in the context of polypharmacy and complex psychiatric treatments. Traditional in-vivo and in-vitro screening methods are highly resource-intensive, time-consuming, and frequently fail to capture the multi-dimensional nature of adverse effects in human bodies. AI and computational ML algorithms offer cost-effective, high-throughput alternatives to identify potential interactions early in the treatment pipeline. While DDI prediction is well-studied, modeling the specific impact of genetic variants on drug metabolism—crucial for personalized psychiatry—remains a critical frontier.

Methods: This review critically evaluates and synthesizes current computational paradigms, encompassing supervised, semi-supervised, self-supervised, and graph-based network learning models. Traditional ML algorithms, including Random Forest, Support Vector Machines (SVM), and Gradient Boosting, are assessed for structured biomedical data classification. Deep learning and transformer-based natural language processing models, specifically BioBERT, are examined for their ability to extract complex interactions from unstructured medical literature. Crucially, we investigate the novel "VarDrug" multimodal machine learning framework, which integrates self-supervised variant encoders pre-trained on 100,000 GRCh38 variants, molecular chemical fingerprints, and gene co-expression profiles to predict how genetic mutations alter drug efficacy and toxicity.

Results: Computational models demonstrate unprecedented predictive accuracy in pharmacology. NLP models such as BioBERT achieve robust F1-scores up to 0.99 in text-based DDI extraction tasks. In the realm of pharmacogenomics, the VarDrug framework successfully predicts variant-drug interactions, yielding a top-2 accuracy of 0.93 utilizing a Random Forest classifier. When applied to complex psychiatric conditions like Borderline Personality Disorder (BPD), these network models successfully identify pivotal genetic hubs—most notably CYP2D6, ABCB1, and HTR2A. These specific genes significantly alter the pharmacokinetics, metabolism, and central nervous system penetration of widely prescribed antipsychotics and antidepressants. Furthermore, AI-generated networks can map overlapping mechanisms of action (MOAs) to guide synergistic, combination treatments for drug-resistant cases.

Conclusion: AI and ML algorithms substantially enhance our capacity to predict both DDIs and DGIs, establishing a robust foundation for personalized, genotype-guided psychiatric treatments. Despite these monumental strides, routine clinical implementation is currently hindered by pervasive challenges, including severe data imbalance (where non-interacting pairs skew models), the "cold start" problem for novel medications lacking clinical history, and the problematic "black box" nature of deep learning systems. Future pharmacological research must prioritize the development of Explainable AI (XAI) frameworks and the comprehensive integration of multi-omics data to ensure algorithmic transparency, mitigate bias, and safely embed these predictive tools into everyday clinical decision-making

Keywords: Drug-Drug Interactions (DDI), Machine Learning, Pharmacogenomics, Precision Medicine

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0503 - Intolerance of Uncertainty in Children with Eating Disorders

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Eating disorders are complex psychiatric conditions that arise from the interaction of biological vulnerabilities, individual psychological characteristics, and sociocultural influences, and they can significantly affect both physical and mental health. These disorders are associated with substantial morbidity and may negatively influence psychological functioning, social relationships, and quality of life. In recent years, increasing attention has been directed toward cognitive mechanisms that may contribute to the development and maintenance of eating disorders. One construct that has gained particular interest in this context is intolerance of uncertainty, which refers to a dispositional tendency to perceive ambiguous or unpredictable situations as threatening and distressing. Individuals with high levels of intolerance of uncertainty often respond to such situations with heightened cognitive, emotional, and behavioral reactions.

Although intolerance of uncertainty was initially conceptualized mainly in relation to anxiety disorders, recent research suggests that it may also play an important role in the psychopathology of eating disorders. Several characteristics frequently observed in individuals with eating disorders—including a strong need for control, perfectionistic tendencies, rigid cognitive styles, and difficulties in cognitive flexibility—may be associated with a reduced tolerance for uncertainty. These traits may contribute to the use of maladaptive strategies aimed at managing distress triggered by unpredictable or ambiguous situations.

In this context, individuals may attempt to restore a sense of control through behaviors related to eating and body weight. For example, strict calorie monitoring, rigid adherence to dietary rules, ritualized eating behaviors, and excessive monitoring of body weight may function as strategies intended to reduce the discomfort associated with uncertainty. Although such behaviors may temporarily reduce anxiety, they may also contribute to the persistence of eating disorder symptoms.

This panel aims to examine the relationship between eating disorders and intolerance of uncertainty in light of current literature. Previous studies indicate that individuals with eating disorders tend to exhibit higher levels of intolerance of uncertainty compared with healthy populations, and that this tendency may be associated with rigid dietary rules, control behaviors, and maladaptive cognitive processes related to eating and body image. In addition, individuals characterized by both high perfectionism and elevated intolerance of uncertainty may be more likely to interpret the possibility of failing to meet expectations or coping with unpredictable situations as threatening. Intolerance of uncertainty has also been described as a transdiagnostic cognitive process that may contribute to the maintenance of various psychiatric disorders, including eating disorders. Therefore, therapeutic approaches that aim to enhance tolerance of uncertainty may represent a valuable target within psychological interventions. In this context, improving individuals' ability to cope with ambiguity and unpredictability may help reduce reliance on rigid control behaviors related to eating. Overall, intolerance of uncertainty appears to represent an important cognitive dimension in understanding the psychopathology of eating disorders and may provide useful insights for clinical assessment and therapeutic intervention strategies.

Keywords: Adolescents, cognitive control, eating disorders, intolerance of uncertainty, perfectionism

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0504 - Post-SSRI Sexual Dysfunction (PSSD): Diagnostic Challenges, Management Strategies and Future Directions

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Post-SSRI Sexual Dysfunction (PSSD) is a condition characterized by persistent sexual dysfunction that continues after discontinuation of serotonin reuptake inhibiting (SRI) antidepressants. Although sexual side effects during antidepressant treatment are well known, the persistence of these symptoms after drug cessation has only recently begun to receive broader scientific and clinical attention. Patients with PSSD frequently report reduced genital sensitivity, diminished libido, erectile dysfunction, anorgasmia, decreased orgasmic intensity, and emotional blunting. These symptoms may persist for months or even years after stopping the medication and can significantly impair quality of life, intimate relationships, and psychological well-being (1).
One of the major challenges surrounding PSSD is diagnostic uncertainty. Currently, PSSD is not formally recognized as a distinct disorder in major diagnostic classification systems such as DSM-5 or ICD-11. As a result, clinicians may attribute persistent symptoms to depression relapse, anxiety, relationship difficulties, or psychological factors rather than considering a medication-related etiology. Furthermore, patients often report that their concerns are dismissed or minimized in clinical encounters, which may delay recognition and appropriate support. Another diagnostic difficulty arises from the overlap between sexual dysfunction related to psychiatric disorders themselves and dysfunction potentially induced by pharmacological treatments. Careful clinical history taking, including the temporal relationship between antidepressant use and symptom onset, is therefore crucial (2).
Management of PSSD is also challenging due to the lack of evidence-based treatment guidelines. Various pharmacological and non-pharmacological interventions have been explored, though results remain inconsistent. Some case reports and small studies have suggested potential benefits from medications such as bupropion, vortioxetine, or phosphodiesterase-5 inhibitors like sildenafil or tadalafil. Other proposed approaches include dopaminergic agents, hormonal interventions, nutraceuticals, and experimental therapies. Psychological interventions, particularly cognitive-behavioural therapy, may also play a role in helping patients cope with the distress associated with persistent sexual symptoms and their impact on relationships and self-esteem. Nevertheless, none of these strategies can currently be considered a definitive treatment (3).
Given these uncertainties, further research is urgently needed to clarify the prevalence, risk factors, and course of PSSD. Prospective longitudinal and neurobiological studies may help identify underlying mechanisms and potential therapeutic targets. At the same time, greater awareness among clinicians is essential to improve recognition, patient validation, and informed consent regarding possible long-term sexual side effects of antidepressants. Overall, PSSD represents an emerging clinical concern at the intersection of psychiatry, sexual medicine, and pharmacology, and addressing its diagnostic challenges while developing effective management strategies will be crucial for improving patient care and guiding clinical practice.

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Keywords: Post-SSRI Sexual Dysfunction (PSSD), Antidepressant-induced sexual dysfunction, Diagnostic challenges, Treatment strategies, Long-term adverse effects
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0505 - Postpartum Paternal Depression and Maternal Mental Health: Clinical Implications of a Dyadic Risk Process

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Postpartum paternal depression (PPPD) has increasingly been recognized as a clinically significant condition with substantial implications for maternal mental health and family functioning. Recent studies demonstrate a moderate positive association between paternal and maternal depression, indicating that when one parent experiences depression, the risk for the other parent increases significantly. Longitudinal data from early postpartum cohorts further suggest that paternal depression predicts maternal depression, while maternal depressive symptoms reciprocally predict paternal outcomes. Additionally, factors such as low social support and reduced marital satisfaction reinforce this bidirectional interaction. These findings underscore the importance of assessing both parents' mental health during the postpartum period. Meta-analytic evidence supports a moderate correlation between paternal and maternal depression. In particular, maternal prenatal or postpartum depression predicts paternal depression, and vice versa. When considering marital satisfaction, social support, and family functioning, low marital satisfaction and high levels of family conflict are associated with increased depressive symptoms in both mothers and fathers. Similarly, decreased social support exacerbates the emotional burden on both parents, with higher rates of depressive symptoms observed in families with limited support networks. Multivariate analyses indicate that insomnia, a history of depression, anxiety, stress, and an adverse family environment predict the persistence of moderate to severe depressive symptoms in both parents. Moreover, longitudinal findings suggest that elevated depressive symptoms in one parent during the first six months postpartum tend to intensify in the other parent over time. Paternal depression is also associated with impaired dyadic functioning and reduced marital satisfaction, contributing to declines in relationship quality, disrupted communication, and increased interpersonal conflict. Increasing paternal depressive symptoms may initiate a cyclical process that amplifies maternal emotional burden and diminishes perceived spousal support. When both parents exhibit depressive symptoms, there is a marked deterioration in family functioning, including disrupted communication patterns and elevated levels of conflict. PPPD also indirectly affects maternal well-being through increased caregiving burden and child-related stressors. Co-occurring parental depression is associated with higher levels of maternal stress, loneliness, and exhaustion, alongside reduced perceived support. Furthermore, impaired parental mental health may negatively affect child outcomes and caregiving quality, thereby intensifying maternal feelings of inadequacy and guilt. Consequently, given this bidirectional interaction, it is essential to monitor the psychological well-being of both parents during the postpartum period. Interventions aimed at improving family communication and strengthening social support systems are strongly recommended. The father's psychological state during the postpartum period plays a critical role in maternal mental health; therefore, holistic, family-centered approaches are essential.

Keywords: Maternal mental health, Paternal postpartum depression, Perinatal mental health

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0506 - Clinical Evaluation and Screening in the Early Diagnosis of Dementia

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Early diagnosis of dementia has become a critical priority in clinical practice, as it enables timely intervention, care planning, and potential slowing of disease progression. Dementia often develops insidiously, with subtle cognitive and behavioral changes preceding overt functional decline. Therefore, systematic clinical evaluation combined with appropriate screening tools is essential for early detection, particularly at the stage of mild cognitive impairment (MCI).

Clinical evaluation begins with a comprehensive history, including cognitive symptoms (e.g., memory loss, executive dysfunction), behavioral changes, and functional impairment. Collateral information from caregivers is crucial, as patients may lack insight. Assessment should also include medical, psychiatric, and medication histories to identify reversible causes such as depression, metabolic disorders, or drug effects. A focused neurological and mental status examination helps identify patterns suggestive of neurodegenerative processes. Screening tools play a central role in early detection. Commonly used instruments include the Mini-Mental State Examination (MMSE), Montreal Cognitive Assessment (MoCA), and Addenbrooke's Cognitive Examination (ACE). Among these, MoCA is particularly sensitive for detecting MCI, with reported sensitivity ranging from 81% to 97%, although specificity is somewhat lower (60–86%). In contrast, ACE demonstrates high sensitivity (79–100%) and specificity (around 86%) for dementia detection.

Brief tools such as Mini-Cog and informant-based questionnaires (e.g., AD8) are especially useful in primary care due to their rapid administration and feasibility. Screening approaches can be categorized into clinician-administered tests, self-administered tools, and informant-based assessments, each contributing complementary information.

Despite their utility, screening tools are not diagnostic. Their performance varies depending on education level, cultural context, and disease stage. Therefore, abnormal screening results should prompt further evaluation, including neuropsychological testing, neuroimaging, and laboratory investigations.

Recent advances highlight the growing role of digital biomarkers and machine learning approaches, such as analysis of drawing behavior or eye-tracking patterns, which may improve early detection accuracy in prodromal stages. However, these methods require further validation before routine clinical implementation.

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Keywords: Cognitive screening, demantia, early diagnosis

0507 - Attention Deficit Hyperactivity Disorder: Treatment in Childhood

Akın Tahıllıoğlu

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Attention-deficit/hyperactivity disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood and remains a major cause of functional impairment across home, school, and peer settings. Treatment in childhood is therefore not simply a matter of reducing symptoms, but of improving daily functioning, supporting developmental trajectories, and preventing secondary emotional, academic, and family-related difficulties. In clinical practice, effective treatment requires a multimodal and individualized approach rather than a one-size-fits-all model.

This panel will provide a practical overview of current treatment strategies for ADHD in childhood, with particular emphasis on how evidence can be translated into real-world care. The talk will begin with the core principles of treatment planning, including comprehensive assessment, identification of comorbid psychiatric and developmental conditions, psychoeducation for parents and teachers, and the definition of realistic treatment targets. Rather than focusing only on symptom counts, clinicians should aim to improve classroom functioning, family routines, emotional regulation, and the child's self-esteem.

A major focus of the session will be pharmacological treatment. Stimulants remain the first-line option for many school-age children because of their robust and consistent efficacy. The discussion will address the clinical use of methylphenidate and amphetamine-based preparations, the choice between short-acting and long-acting formulations, common adverse effects, and principles of dose titration and monitoring. Non-stimulant options, including atomoxetine and alpha-2 adrenergic agonists, will also be reviewed, especially in cases involving intolerance, partial response, sleep problems, tics, anxiety, or specific comorbidity profiles. Particular attention will be paid to the need for regular follow-up, growth and cardiovascular monitoring, assessment of adherence, and the importance of shared decision-making with families.

The panel will also highlight the indispensable role of non-pharmacological interventions. Parent training, behavioral classroom strategies, school collaboration, and environmental modifications remain central components of care, particularly in younger children and in those with significant functional difficulties. These approaches are often most effective when implemented consistently across settings and when caregivers are actively supported throughout the treatment process. The presentation will emphasize that medication and psychosocial interventions should not be viewed as competing alternatives, but as complementary tools whose relative weight may change according to age, severity, family context, and treatment response.

Finally, the session will address several ongoing clinical challenges: treatment adherence, misconceptions and stigma surrounding medication, management of comorbidities, and the need for long-term follow-up as children move into adolescence. Overall, the panel argues that successful treatment of childhood ADHD depends on combining evidence-based interventions with careful clinical judgment, developmental sensitivity, and sustained collaboration with families and schools.

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Keywords: ADHD, childhood, pharmacological treatment, behavioral interventions, multimodal treatment, child psychiatry

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0508 - The Silent Ripple: Effects of Paternal Postpartum Depression on Child Development

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Postpartum depression in fathers is a clinically significant yet systematically underrecognized condition, affecting approximately 10% of new fathers, nearly double the rate observed in the general male population. While maternal postpartum depression has received substantial research and clinical attention, the paternal counterpart remains a largely overlooked public health concern. This presentation examines the downstream effects of paternal postpartum depression (PPD) on child development, synthesizing epidemiological, neurobiological, and longitudinal evidence to support its routine clinical recognition.

A narrative review of the literature was conducted to synthesize current evidence on the association between paternal PPD and adverse child outcomes, to delineate the primary mechanistic pathways underlying this relationship, and to identify implications for psychiatric practice. The presentation focuses on child behavioral, psychiatric, and neurodevelopmental outcomes.

Evidence consistently demonstrates that paternal PPD is associated with increased internalizing problems—including anxiety, depression, and emotional dysregulation—as well as externalizing problems such as aggression, behavioral dysregulation, and attention deficits in children. Large cohort studies report a 17% higher total externalizing behavior score at age 5, independent of maternal depression, while children of depressed fathers show a 33–70% increased risk of emotional or behavioral disorders. Three primary mechanistic pathways are identified: (1) impaired father–infant bonding and attachment; (2) family-level processes, including co-occurring maternal depression and interparental conflict, which may account for up to two-thirds of the association; and (3) disrupted paternal parenting behaviors, characterized by reduced sensitivity and increased hostility. Notably, paternal depressive symptoms often follow a chronic and stable trajectory across the perinatal period, underscoring the need for longitudinal rather than single time-point assessment.

Paternal PPD exerts a measurable and clinically meaningful impact on child development through multiple converging pathways. These findings highlight the family system—rather than the individual parent—as the appropriate unit of clinical focus. Psychiatric practice should move toward systematic screening of fathers during the perinatal period and the development of gender-sensitive diagnostic approaches.

Keywords: Paternal postpartum depression, child development, perinatal psychiatry, intergenerational transmission, parenting behavior

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0509 - Enhancing the Therapeutic Process Through Chatbots and AI Assistants

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Treatment resistance in psychiatry is a major clinical and economic challenges in mental health care. It is defined as the persistence of significant symptoms despite an accurate diagnosis and adequate pharmacological treatment. Treatment resistance affects an estimated 20–60% of patients across major psychiatric disorders. It is associated with healthcare costs up to ten times higher than those for treatment-responsive populations (Howes et al., 2021). Some authors argue that "treatment resistance" is not a discrete diagnostic category. Instead, it is seen as an imprecise term covering treatment failures with varied causes. Despite this, the concept remains widely used in clinical practice to describe difficult-to-treat cases and guide stepped-care decisions (Mcintyre et al., n.d.; McIntyre et al., 2023). The mechanisms behind treatment resistance are multifactorial. They include pharmacokinetic variability, medication non-adherence, psychiatric comorbidity, diagnostic ambiguity, and neurobiological heterogeneity. In routine clinical practice, these challenges are further compounded by episodic symptom monitoring, which can delay the recognition of early non-response or impending relapse. Conversational AI and chatbot-based systems are emerging as promising supports for psychiatric care, especially for those with treatment-resistant presentations. Instead of replacing the clinician, these technologies can help maintain therapeutic continuity between appointments. They facilitate symptom assessment, provide psychoeducation, and support engagement with treatment (Li et al., 2023). Recent evidence shows that AI-assisted interventions can improve some clinical outcomes (Rony et al., 2025). Digital phenotyping platforms adopt smartphone-derived data to detect relapse risk earlier (Liu et al., 2024). Passive sensing applications such as mindLAMP aggregate multimodal data streams, including cognitive task performance, GPS coordinates, and physical activity metrics, to model an individual's mental health trajectory longitudinally; while BiAffect employs machine learning algorithms applied to keyboard interaction metadata, such as typing variability and inter-keystroke pauses, to identify behavioral signatures predictive of manic and depressive episodes (Allen, 2020; Pham et al., 2022). These tools are especially relevant in treatment-resistant cases, where early recognition of deterioration is critical. Specialized digital approaches such as avatar-based therapies for persistent psychotic symptoms also show the promise of technology-assisted care for symptoms unresponsive to established approaches (Garety et al., 2021). The emerging evidence is encouraging, but moving from controlled research settings to everyday clinical practice demands more than efficacy data alone.

Significant limitations nevertheless persist, encompassing concerns related to empathic responsiveness, algorithmic transparency, data privacy, and medico-legal accountability. When integrated within structured clinical pathways, however, AI-assisted tools hold considerable potential to provide a scalable and clinically meaningful extension of care for patients with treatment-resistant psychiatric disorders.

Keywords: Treatment resistance, artificial intelligence, chatbot, digital phenotyping, psychiatric care, conversational agents

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0510 - Adaptive Support or Reinforced Rigidity? Family Accommodation in Autism Spectrum Disorder

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Family accommodation refers to changes in caregiver behavior intended to reduce or prevent a child's distress associated with psychiatric symptoms. These behaviors include modifying routines, providing reassurance, assisting avoidance of anxiety-provoking situations, or participating in symptom-related behaviors. While such responses aim to alleviate immediate distress, growing evidence suggests that accommodation may contribute to the maintenance or exacerbation of symptoms by reinforcing avoidance and limiting opportunities for children to develop independent coping strategies (1).

In autism spectrum disorder (ASD), family accommodation has emerged as a systemic factor influencing both symptom expression and functional outcomes. Anxiety is a common co-occurring condition in ASD, affecting approximately 40% of autistic children and significantly impacting daily functioning and quality of life (2). Families frequently modify routines, provide reassurance, or help children avoid anxiety-provoking situations to manage distress. Community studies show that accommodation behaviors are highly prevalent, with most parents reporting at least one such behavior (2).

Importantly, family accommodation is associated not only with anxiety severity but also with its broader impact on functioning. Higher levels of accommodation are linked to greater interference in daily life, including family routines, social participation, and parental well-being. Even after controlling for child and parent anxiety, accommodation remains significantly associated with functional disruption, suggesting caregiver responses independently shape the impact of anxiety (3). Beyond anxiety, accommodation also occurs in response to core autism features, particularly restricted and repetitive behaviors (RRBs). Parents adjust activities, provide preferred objects, or avoid triggers to prevent distress. RRB severity is strongly associated with accommodation (4), which is also linked to increased disruptive behaviors, suggesting reinforcement of maladaptive patterns over time (5). Collectively, these findings highlight family accommodation as a key family-level process influencing symptom maintenance in ASD. While it may provide short-term relief, it may contribute to long-term impairment by maintaining avoidance and limiting skill development.

This panel presentation will review current evidence on family accommodation in ASD, focusing on anxiety, RRBs, and functioning. It will discuss underlying mechanisms and clinical implications, emphasizing family-based interventions and the role of caregivers as key partners in treatment.

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Keywords: Family accommodation, Autism spectrum disorder, Anxiety, Restricted and repetitive behaviors, Child functioning

AuthorToEditor: Symposia Oral Session-4 15.04.2026 / 16:30-18:00 Family Accommodation As a Transdiagnostic Mechanism in Child and Adolescent Psychopathology Moderatörler: Dr. Vahdet Görmez Dr. Alperen Bıkmazer Adaptive Support or Reinforced Rigidity? Family Accommodation in ASD Dr. Büşra Nur Dambasan Gündüz ORCID:0000-0003-2474-1287

0511 - Exploring Sleep Problems From a Psychiatrist's Perspective: What Do Sleep Complaints Reveal About Underlying Psychiatric Disorders?

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Sleep complaints in psychiatry are not merely accompanying symptoms; rather, they constitute a clinical window that can reveal the nature, stage, and risk profile of the underlying disorder. Longitudinal meta-analyses indicate that insomnia disorder is not simply a symptom of depression; it may represent a risk process for the development of depression and has transdiagnostic significance across a broader range of psychopathology (1). Disruptions in sleep quality, continuity, and timing interact bidirectionally with mood symptoms, anxiety, psychotic symptoms, and substance use. Therefore, sleep-related complaints may function as an early warning domain within psychiatric assessment (2).

The differential diagnostic evaluation usually begins with the following question: Is the sleep complaint a component of the psychiatric condition, does it reflect a primary sleep disorder, or is it related to medication or substance effects? Obstructive sleep apnea, restless legs syndrome/periodic limb movement disorder, narcolepsy, and circadian rhythm sleep–wake disorders should be systematically screened. In bipolar disorder, a decreased need for sleep, irregular sleep timing, and sleep changes preceding mood episodes—particularly before mania or hypomania—are clinically informative. In high-risk populations, reduced sleep duration, difficulties initiating sleep, and general sleep disturbances have been reported as potential prodromal indicators of bipolar disorder. In anxiety disorders, difficulties initiating sleep and cognitive–emotional hyperarousal are prominent, whereas in post-traumatic stress disorder, nightmares frequently co-occur with insomnia; a recent meta-analysis reported the prevalence of insomnia in PTSD to be approximately 60%. In psychotic disorders, sleep and circadian rhythm disruptions may exacerbate symptom severity; targeted sleep interventions in ultra-high-risk populations have been shown to produce sustained reductions in insomnia severity (3). In ADHD, objective disturbances in sleep parameters and increased arousal have been reported, and sleep problems have been suggested to contribute to clinical presentation. In substance use disorders, sleep disturbances may persist even after detoxification; a recent study reported reduced total sleep duration in alcohol, nicotine, and opioid use disorders, as well as decreased slow-wave sleep in alcohol and cocaine use disorders. In older adults, insomnia, short or long sleep duration, excessive daytime sleepiness, and sleep-related breathing disorders have been associated with a significantly increased risk of cognitive decline and dementia (4).

Clinically, obtaining a structured sleep history—systematically assessing sleep timing, sleep onset latency, nocturnal awakenings, nightmares, excessive daytime sleepiness, and medication or substance use—can strengthen diagnostic reasoning. The use of brief screening scales and, when indicated, sleep diaries, actigraphy, or polysomnography in selected cases may further support the evaluation. For chronic insomnia disorder, the first-line treatment is Cognitive Behavioral Therapy for Insomnia (CBT-I), whereas sleep hygiene recommendations alone are generally insufficient. However, sleep restriction interventions should be applied cautiously in individuals vulnerable to mania or hypomania, with individualized planning and close monitoring of mood symptoms (5).

From a practical psychiatric perspective, sleep complaints should therefore be interpreted not as a single symptom but as a pattern that may signal different psychiatric or primary sleep disorders. Such an approach may both accelerate differential diagnosis and reduce the risk of overlooking comorbid primary sleep disorders.

Keywords: Circadian rhythm, Differential diagnosis, Psychiatric disorders, Sleep disturbances

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0512 - Meaning *in* life or meaning *of* life?

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The meaning of life has been examined through diverse perspectives; however, a consensus and precise definitions remain elusive. An approach to address this ambiguity is to differentiate between *the meaning of life and meaning in life (MIL)*. The former chiefly pertains to existential and philosophical inquiries, whereas the latter is regarded as a subjective experience or mental state.

While defining MIL is challenging, it is widely agreed that it involves three key components: *comprehension, purpose, and existential mattering*. Comprehension involves recognizing patterns and maintaining a consistent understanding of life events. Purpose serves as the motivation behind a person's goals and actions. Additionally, existential mattering pertains to the feeling of value and worth that one assigns to one's own life. In summary, perceiving life as meaningful depends on one's sense of having a clear purpose, value, or significance.

Keywords: Comprehension, existential mattering, meaning in life, purpose

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0514 - Psychopharmacological Treatment of Emotion Regulation Difficulties in Attention Deficit Hyperactivity Disorder

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Emotion dysregulation (ED), encompassing emotional lability, irritability, and poor frustration tolerance, is increasingly recognized as a core dimensional feature of attention deficit hyperactivity disorder (ADHD) rather than a mere epiphenomenon. Neurobiological evidence from functional connectivity studies demonstrates that ED in ADHD arises from disrupted prefrontal-limbic circuitry, particularly involving reduced dorsolateral prefrontal cortex (DLPFC) top-down control over the amygdala and ventral striatum, alongside abnormal orbitofrontal cortex connectivity. These circuit-level impairments are underpinned by deficient catecholaminergic signaling, where both dopaminergic and noradrenergic transmission in the prefrontal cortex play crucial roles in modulating emotional reactivity, salience processing, and cognitive-emotional integration. Imaging-genetic studies further support that monoaminergic gene variants affecting dopamine and norepinephrine transporter function influence prefrontal-amygdala connectivity and emotional lability severity. This neurobiological framework provides a strong rationale for psychopharmacological interventions targeting these specific neural circuits.

Stimulant medications, including methylphenidate and lisdexamfetamine, constitute the first-line pharmacological treatment for ED in ADHD. These agents enhance prefrontal norepinephrine and dopamine levels at therapeutic doses, thereby strengthening the signal-to-noise ratio of prefrontal neurons and improving top-down regulatory control over limbic structures. Clinical evidence indicates that methylphenidate produces moderate-to-large effect sizes (Cohen's d approximately 0.6–0.8) on irritability and emotional lability measures in children and adolescents over three to six months of treatment. Longitudinal cohort data further demonstrate that medicated adolescents show significant ED score reductions approaching normative levels compared to unmedicated ADHD peers, suggesting a normalizing class effect of ADHD pharmacotherapy on emotional outcomes.

Non-stimulant agents offer important alternative and adjunctive options for managing ED. Atomoxetine, a selective norepinephrine reuptake inhibitor that also increases prefrontal dopamine through norepinephrine transporter-mediated mechanisms, demonstrates small-to-moderate effects on emotional symptoms, though with less consistency than stimulants. Alpha-2 adrenergic agonists, particularly extended-release guanfacine, enhance prefrontal network stability through postsynaptic alpha-2A receptor activation, showing particular promise in reducing emotional outbursts and irritability in patients with comorbid oppositional defiant disorder or those who remain stimulant-refractory.

For complex presentations with severe ED and comorbid oppositional defiant disorder or disruptive mood dysregulation, a stepwise pharmacological algorithm is recommended. The initial step involves optimized long-acting stimulant monotherapy with adequate dose titration and systematic monitoring. When partial response persists, augmentation with an alpha-2 agonist or addition of atomoxetine is indicated as the second step. For treatment-resistant severe aggression and mood instability, judicious adjunctive use of atypical antipsychotics, primarily risperidone or aripiprazole, may be considered with careful metabolic and extrapyramidal monitoring. Mood stabilizers such as divalproex or lithium remain reserved for the most refractory cases under specialist supervision.

Importantly, residual ED frequently persists even with optimized pharmacotherapy, underscoring that psychopharmacological treatment should be embedded within a comprehensive multimodal framework incorporating behavioral parent training, cognitive-behavioral strategies, and school-based supports. Future research should prioritize ED as a primary outcome measure in ADHD pharmacotherapy trials and explore novel circuit-targeted neuromodulation approaches to address this clinically significant yet insufficiently studied dimension of the disorder.

Keywords: Emotion dysregulation, ADHD, psychopharmacology

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0515 - Neural Signatures of Emotional Biases in Major Depressive Disorder and Their Potential Clinical Application

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The neurocognitive mechanisms underlying variability in depression outcomes and recurrence risk are poorly understood, yet are essential to develop better psychological, neuromodulation and pharmacological treatments, as well as for stratified approaches to improve long-term outcomes.

Major depressive disorder (MDD) is characterized by a proneness to experience excessive self-blaming emotions, and overgeneral self-critical thinking. Co-morbidity with trauma-related and other anxiety disorders is high and transdiagnostic more general negative perceptual biases have been described.

As part of the Antidepressant Advisor Study (ADeSS), we probed the potential of neural signatures of emotional biases in predicting clinical outcomes in a sample of difficult-to-treat MDD after four months of primary care (n=45). More specifically, we investigated self-blaming biases, negative perceptual biases and dysfunction of task-independent subgenual networks as captured by fMRI at baseline, i.e. self-blame-selective right anterior temporal-subgenual connectivity, amygdala activation in response to emotional faces, and resting-state posterior subgenual cortex connectivity, respectively. Taken together, these three neural measures explained a meaningful proportion of the variance (32%) in clinical outcomes [1].

Emotional biases have also been identified as vulnerability factors in developing a recurring depressive episode. As part of the Neurocognitive Signatures Predicting Risk of Recurrent Depression (NESPRED) project (OSF: https://osf.io/625th/overview), we sought to replicate work identifying self-blame-selective right anterior temporal lobe hyper-connectivity with the posterior subgenual cortex (Brodmann Area 25) as a risk factor [2]. The prospective cohort study was completed by 129 psychotropic medication-free patients with remitted MDD and no significant current co-morbidity, who were clinically followed up over a 14-month period. As expected, self-blame-related right anterior temporal lobe-posterior subgenual cortex (BA25) connectivity predicted risk of subsequent recurrence. However, we observed hypo-connectivity – the opposite to what was observed in previous event-related task design studies, likely resulting from higher task-related subgenual fMRI deactivation in the current study, which employed a block design.

The findings from the ADeSS and NESPRED studies confirm the reproducible pathophysiological relevance of self-blame-related anterior temporal-subgenual connectivity independently of previously described MDD signatures, thereby informing future personalised neuromodulation treatments designed to target anterior temporal-subgenual circuits.

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Keywords: Depression, difficult-to-treat, fMRI, self-blame, emotions, biomarkers

AuthorToEditor: This abstract is for one of the talks which forms part of the symposium "Cognitive neuropsychopharmacology: recent advances". Both studies that I will present on have ethical approval in place, but I think the "Has the consent form been received?" question refers to case studies specifically, hence "no" as this is not applicable. My ORCID is: https://orcid.org/0000-0002-1470-4063

0516 - Psychotic Symptoms and Autism Spectrum: Risk and Protective Factors

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Autism is characterized by persistent difficulties in social communication, restricted interests, and sensory sensitivities, whereas psychosis involves symptoms such as delusions, hallucinations, disorganized behaviour, and thought disorder. Despite these differences, there are important overlaps in clinical presentation. Social withdrawal, atypical perceptual experiences, and mentalization difficulties can be observed in both conditions. Moving beyond categorical diagnoses, recent research supports a dimensional approach. Both autistic traits and psychotic experiences are not confined to clinical populations but are continuously distributed across the general population. Many individuals show subclinical levels of autistic traits, and psychotic experiences, such as mild hallucinations or unusual beliefs, are relatively common and often transient. This perspective provides an understanding of how these traits relate to mental health across development, particularly during adolescence and early adulthood. Increasing evidence suggests that individuals with higher levels of autistic traits may be more vulnerable to psychotic experiences.

Epidemiological and meta-analytic findings support this association. Around 9.4% of individuals with autism develop clinically significant psychosis, while subthreshold psychotic symptoms may be present in up to 24% [1, 2]. Certain clinical characteristics, including older age, male sex, and lower cognitive functioning, have been linked to a higher likelihood of psychosis within the autism spectrum. These findings indicate that autism may increase vulnerability to psychosis, although this relationship is not deterministic.

Genetic factors play an important role in the relationship between autism and psychosis, with shared vulnerabilities in neurodevelopmental and synaptic pathways. Environmental factors also play a key role in shaping this risk. Childhood trauma, bullying, obstetric complications, and cannabis use have consistently been associated with psychotic experiences. However, studies specifically examining how these exposures interact with autistic traits remain limited. Recent findings suggest that childhood adversity may strengthen the relationship between autistic traits and psychotic experiences, acting as a moderating factor and highlighting the role of environmental sensitivity [3].

At the same time, autistic traits do not inevitably lead to negative outcomes. Protective factors are increasingly recognized as essential in shaping individual trajectories. Structural factors such as social inclusion, access to healthcare, and supportive environments can reduce vulnerability. In addition, individual psychosocial resources, including adaptive coping strategies, resilience, and certain personality traits, may help reduce the severity or persistence of psychotic symptoms.

Overall, these findings support a dimensional and transdiagnostic understanding of autism and psychosis. While shared genetic, environmental, and cognitive mechanisms may increase vulnerability, protective factors play a crucial role in shaping outcomes. A balanced approach that considers both risk and resilience is essential for improving early identification, prevention strategies, and long-term mental health outcomes across the autism spectrum.

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Keywords: Autism, psychosis, risk factors, protective factors, prevention

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0517 - Psychotherapeutic Interventions for Emotion Regulation Difficulties in Attention Deficit Hyperactivity Disorder

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Emotion dysregulation, encompassing emotional lability, heightened reactivity, and deficient regulation strategies, is a core feature of ADHD across the lifespan. While pharmacological interventions primarily target catecholaminergic circuits, psychotherapeutic approaches offer complementary mechanisms by directly training adaptive emotion regulation skills. This presentation reviews systematic reviews, meta-analyses, and randomized controlled trials published between 2019 and 2026, examining the comparative efficacy of cognitive behavioral therapy (CBT), dialectical behavior therapy (DBT)-based programs, and mindfulness-based cognitive therapy (MBCT) in addressing ED in both adult and pediatric ADHD populations.

Among psychotherapeutic modalities, structured CBT programs have accumulated the strongest evidence for reducing ED in adult ADHD. Meta-analytic data demonstrate small-to-moderate effect sizes on emotional dysregulation measures, with improvements maintained at follow-up. A recent meta-analysis has confirmed that structured CBT significantly improves both core symptoms and emotional and functional outcomes in adults. Randomized controlled trials further show that CBT modules incorporating cognitive restructuring, emotion monitoring, trigger identification, and alternative response development produce clinically meaningful reductions in emotional symptoms alongside improvements in executive functioning and quality of life persisting up to 24 weeks.

DBT-derived interventions target ED through mindfulness, distress tolerance, emotional modulation, and interpersonal effectiveness modules. Meta-analytic findings indicate small-to-moderate improvements in emotional functioning and quality of life, though effects on core ADHD symptoms appear more limited compared to CBT. These programs show particular utility for patients presenting with prominent anger outbursts, low frustration tolerance, and relational difficulties, especially when borderline personality features co-occur. Current consensus supports integrating DBT components with structured CBT to address both cognitive-executive and emotion regulation deficits.

Mindfulness-based approaches improve emotional self-regulation through enhanced prefrontal-amygdala network functioning, with neuroimaging evidence supporting functional changes in regions critical for emotion regulation. While effect sizes for ED reduction are slightly smaller than CBT, mindfulness-based interventions show advantages for anxiety, depression, rumination, and self-compassion deficits frequently accompanying ADHD. In the pediatric domain, structured group-based programs combining executive function training with emotion regulation skill building have demonstrated robust outcomes. A large randomized controlled trial of executive and inhibitory control training in children with ADHD aged 8–13 years reported large effect sizes for ED reduction on parent reports (Cohen's d = 1.25 post-treatment) sustained at follow-up (d = 1.22 at 2–4 months). School-based programs incorporating social-emotional components show meaningful improvements in classroom behavior, emotional outbursts, and social functioning. Multi-component programs integrating psychoeducation, role-playing and parent-teacher involvement yield the most significant gains when delivered at high intensity with multi-context implementation.

Current evidence supports a stepped, multimodal psychotherapeutic framework in which structured CBT serves as the foundation, supplemented by DBT modules for patients with prominent emotional reactivity and mindfulness components for those with significant internalizing comorbidity. Schema therapy provides a transdiagnostic framework targeting the developmental origins of emotion dysregulation through schema modes, complementing skill-based approaches such as CBT and DBT. For children and adolescents, group-based programs combining executive function and emotion regulation training with family and school involvement represent the most effective model. These strategies should be integrated with pharmacotherapy to achieve optimal functional outcomes across the lifespan.

Keywords: Adhd, emotion dysregulation, psychotherapy

AuthorToEditor: This abstract presents a narrative review comparing the efficacy of CBT, DBT-based, and mindfulness-based psychotherapeutic interventions for emotion regulation difficulties in ADHD across the lifespan. It synthesizes meta-analytic evidence, highlights domain-specific therapeutic mechanisms, and proposes a stepped multimodal psychotherapy framework. ORCID: 0000-0001-7557-0778

0518 - Clinical Spectrum and Phenomenology of Post-SSRI Sexual Dysfunction

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Post-SSRI sexual dysfunction (PSSD) is an iatrogenic and increasingly recognized condition characterized by the persistence of sexual side effects following discontinuation of serotonergic antidepressants. Despite growing clinical awareness, its phenomenological boundaries remain insufficiently delineated, and its epidemiology remains difficult to quantify due to underrecognition, diagnostic ambiguity, and methodological limitations in existing studies (Healy & Mangin, 2024).

Phenomenologically, PSSD extends beyond conventional antidepressant-induced sexual dysfunction by its chronicity and qualitative distinctiveness. Core symptoms include genital anesthesia, markedly reduced libido, erectile dysfunction, and pleasureless or absent orgasm (anorgasmia). These symptoms are often described as qualitatively distinct from depression-related sexual impairment, with patients reporting a dissociation between subjective desire and physiological sexual response (Reisman, 2021). Notably, genital sensory changes—frequently described as numbness or anesthesia—may represent a distinguishing clinical feature that supports differential diagnosis.

The clinical spectrum extends beyond sexual symptoms alone. Emerging evidence suggests a broader constellation including emotional blunting, anhedonia, apathy, and cognitive dulling, pointing toward a more generalized disturbance in reward and affective processing (Reisman, 2021). This expanded phenotype complicates diagnostic clarity, as symptom overlap with residual depressive states or withdrawal phenomena may lead to misattribution.

Regarding clinical course, symptom onset may occur during treatment, upon dose reduction, or after discontinuation, with persistence ranging from months to years. The trajectory is heterogeneous: some individuals report partial improvement, whereas others experience enduring symptoms with significant functional and relational consequences (Healy et al., 2022).

A major challenge lies in the lack of universally accepted diagnostic criteria. Recent consensus proposals emphasize persistence after drug cessation, absence of pre-morbid sexual dysfunction, and the presence of characteristic features such as genital hypoesthesia and orgasmic impairment (Healy et al., 2022). Nevertheless, underdiagnosis remains common, with symptoms frequently misinterpreted as psychiatric relapse or psychosocial dysfunction (Healy & Mangin, 2024).

In summary, PSSD represents a multidimensional syndrome encompassing sexual, sensory, and affective domains. Refining its phenomenological characterization is essential for accurate diagnosis, improved patient validation, and the advancement of mechanistic and therapeutic research.

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Keywords: Anhedonia, Emotional blunting, Genital anesthesia, Post-SSRI sexual dysfunction, Sexual dysfunction

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0519 - Psychosocial Interventions for Ultra-High Risk for Psychosis: Early Intervention Strategies, Psychotherapy, and Community-Based Models

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Individuals at ultra-high risk (UHR) for psychosis often present with subthreshold psychotic symptoms together with social, emotional, and cognitive impairments. Early intervention is considered essential to reduce symptom severity and to prevent or delay transition to full psychosis. Current treatment guidelines primarily recommend psychosocial interventions such as cognitive behavioral therapy (CBT), family-focused therapy, integrated psychological interventions (IPI), and needs-based intervention (NBI). These approaches are generally preferred over pharmacological treatments because they are well tolerated and have a low risk of adverse effects. CBT is one of the most extensively studied interventions for individuals at UHR. A meta-analysis including studies with follow-up periods of 12, 24, and 48 months reported that CBT significantly reduced the risk of transition to psychosis, with a 54% reduction at 12 months and a 37% reduction at longer follow-up periods. Randomized controlled trials have also shown that CBT results in faster improvement in attenuated positive symptoms compared with supportive therapy. Structured CBT protocols, such as the van der Gaag protocol, have demonstrated reductions in subclinical psychotic symptoms and lower transition rates. Although some debate continues regarding effect size and methodological limitations, there is growing consensus that CBT should be considered a first-line preventive intervention because of its effectiveness and minimal side effects. Since many UHR individuals are adolescents or young adults living with their families, family involvement is an important component of early intervention. Family-focused therapy aims to improve communication, strengthen problem-solving skills, and teach coping strategies for stress. Studies have shown that both long-term family psychotherapy and structured family-focused therapy can improve negative symptoms and attenuated positive symptoms. Comparative research indicates that family-focused therapy may be more effective than standard long-term care in improving interpersonal communication and problem-solving abilities. IPI is a multimodal approach that combines individualized CBT, cognitive remediation, and social skills training. This intervention targets cognitive deficits, distorted beliefs, and perceptual disturbances while also improving social functioning. Research comparing IPI with needs-based intervention suggests that IPI may delay the onset of psychosis for up to 24 months, indicating that comprehensive interventions may provide additional benefit for some individuals. Needs-based intervention is frequently used because of the heterogeneity of symptoms in the UHR population. NBI typically includes supportive psychotherapy, case management, family psychoeducation, clinical monitoring, crisis intervention, and when necessary, non-antipsychotic medication. Some studies have found lower transition rates when CBT is combined with low-dose risperidone compared with NBI alone, whereas other findings suggest that supportive techniques such as non-directive reflective listening may reduce distress related to psychotic symptoms, emphasizing the importance of individualized care. Community-based prevention programs also play a significant role. The Australian Headspace program, a national youth mental health service for individuals aged 12–25 years, has shown that accessible and youth-oriented early intervention services can significantly improve symptoms, functioning, and overall well-being, supporting their importance in preventing progression to psychosis.

Keywords: Early psychosis intervention, psychosis transition prevention, ultra-high risk for psychosis

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0520 - Self-Compassion and Its Emotion-Regulatory Role in Mood, Anxiety, and Eating Disorders

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Self-compassion is defined as a kind, non-judgmental, and mindful way of relating to one's own suffering, encompassing self-kindness, common humanity, and mindfulness. A growing body of empirical research indicates that self-compassion plays a critical role in emotion regulation, thereby influencing the development, maintenance, and treatment outcomes of mood disorders, anxiety disorders, and eating disorders. Emotion regulation (ER) refers to processes that influence the intensity, duration, and expression of emotional responses and is a core mechanism underlying many psychiatric disorders. Deficits in ER are associated with increased vulnerability to psychopathology, including anxiety and mood disorders. Emerging evidence suggests that self-compassion contributes to mental health primarily through its role in facilitating adaptive ER processes. Systematic review findings indicate that ER significantly mediates the relationship between self-compassion and mental health outcomes across different populations.

Self-compassion is associated with increased use of adaptive ER strategies such as cognitive reappraisal and acceptance. At the same time, it is linked to reduced reliance on maladaptive strategies such as emotional suppression and experiential avoidance. As an adaptive ER strategy, self-compassion enables individuals to approach negative experiences with openness, tolerance, and contextual reframing. Furthermore, self-compassion attenuates the relationship between negative affect and psychopathology, thereby functioning as a resilience mechanism.

In mood disorders, deficits in adaptive ER are central to the development and maintenance of depressive symptoms. Experimental evidence demonstrates that self-compassion is effective in reducing depressed mood and performs comparably to cognitive reappraisal and acceptance strategies. Moreover, self-compassion may be particularly beneficial for individuals with higher baseline levels of depression.

In anxiety disorders, self-compassion predicts lower levels of social anxiety both directly and indirectly through ER mechanisms. Specifically, higher self-compassion is associated with reduced expressive suppression and increased cognitive reappraisal. These ER processes are key pathways through which self-compassion influences anxiety symptoms.

In eating disorders, self-compassion plays a crucial role in regulating shame and body image dissatisfaction. Lower self-compassion is associated with higher external shame, body dissatisfaction, and disordered eating behaviors. Studies indicate that individuals with eating disorders often exhibit high levels of self-criticism and low self-compassion, contributing to the persistence of pathological eating patterns. Enhancing self-compassion has been shown to decrease binge eating, emotional eating, and body image disturbances, while improving overall psychological well-being.

Overall, the current evidence positions self-compassion as a central transdiagnostic mechanism that enhances adaptive emotion regulation and buffers against the development and maintenance of psychopathology. Beyond its protective role, self-compassion appears to actively transform maladaptive emotional processes by reducing self-criticism, shame, and avoidance while promoting acceptance and psychological flexibility. These findings highlight the clinical relevance of targeting self-compassion within therapeutic interventions, particularly in disorders characterized by emotion dysregulation. Integrating self-compassion–based approaches into existing treatment models may not only improve symptom reduction but also foster long-term resilience and relapse prevention across mood, anxiety, and eating disorders.

Keywords: Anxiety disorders, eating disorders, emotion regulation, mood disorders, self-compassion

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0521 - The Psychiatric Spectrum of Impulsivity and Compulsivity: A Theoretical and Neurobiological Framework

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Impulsivity and compulsivity are often described as opposing clinical tendencies, yet in psychiatric practice they rarely function as simple opposites. More often, they overlap, interact, and shift across the course of illness. Impulsivity broadly refers to rapid, poorly planned, reward-driven behavior, whereas compulsivity describes repetitive, rigid, and often relief-driven acts that persist despite adverse consequences. A dimensional framework is therefore more informative than a purely categorical one, as it captures the interplay between disinhibition, habit formation, reward sensitivity, and cognitive inflexibility across diagnoses.

The neurobiological substrates of impulsivity and compulsivity, together with their clinical manifestations across psychiatric disorders, are central to this framework. At the neural level, impulsivity is strongly linked to dysfunction within cortico-striatal circuitry, particularly an imbalance between weakened top-down control from prefrontal regions and excessive bottom-up influence from reward-related limbic and ventral striatal systems. Reduced regulatory input from the dorsolateral prefrontal cortex, medial prefrontal cortex, orbitofrontal cortex, inferior frontal gyrus, pre-supplementary motor area, and anterior cingulate cortex may contribute to impaired inhibitory control, premature responding, and difficulty delaying gratification. Dopaminergic signaling appears central to reward salience and action invigoration, whereas serotonergic systems are more closely related to behavioral restraint and inhibitory control; noradrenergic and glutamatergic pathways further modulate response inhibition and corticostriatal plasticity.

Compulsivity, by contrast, is more closely associated with dysfunction in cortico-striato-thalamo-cortical loops, especially hyperactivity within orbitofrontal-striatal pathways and impaired top-down regulation from prefrontal and cingulate regions. In this context, behavior gradually shifts from goal-directed choice to habitual responding, such that repetitive acts may continue even when they no longer produce reward and instead serve only to reduce tension, uncertainty, or distress. This transition model is clinically important because it explains why many disorders do not remain fixed at one pole: what begins as reward-seeking may, over time, become rigid repetition. The ventral-to-dorsal striatal shift frequently described in addiction models provides a useful neurobiological account of this progression.

Impulsivity and compulsivity are increasingly recognized as transdiagnostic dimensions extending across traditional diagnostic categories in psychiatry. ADHD, disruptive behavior disorders, borderline and antisocial personality pathology, and certain phases of bipolar disorder are more often characterized by prominent impulsive features, whereas obsessive-compulsive disorder and related conditions predominantly reflect compulsive mechanisms. At the same time, substance use disorders, gambling disorder, autism spectrum disorder, eating disorders, and bipolar disorder may show mixed phenotypes in which impulsive and compulsive processes coexist or shift in relative prominence over time. The clinical value of a dimensional framework therefore lies in its ability to define behavior in terms of dominant psychopathological mechanisms rather than categorical diagnosis alone.

The measurement and treatment implications of this framework are clinically substantial. A dimensional and neurobiologically informed perspective may strengthen clinical formulation, support mechanism-oriented intervention strategies, and provide a more accurate account of the dynamic psychopathology that cuts across psychiatric disorders.

Keywords: Compulsivity, cortico-striatal circuits, dimensional approach, impulsivity, transdiagnostic psychiatry

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0522 - Standard Treatments in ASD and Defining Resistance

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The cornerstone of contemporary Autism Spectrum Disorder (ASD) management relies on a dual-track approach: intensive behavioral-educational interventions and targeted pharmacological management. Standard psychosocial treatments, such as Applied Behavior Analysis (ABA), Early Start Denver Model (ESDM), and Social Skills Training (SST), aim to enhance functional communication and social reciprocity. Pharmacologically, current FDA-approved treatments are limited to atypical antipsychotics, specifically risperidone and aripiprazole, indicated solely for the management of irritability and associated agitated behaviors. However, these interventions primarily address peripheral symptoms rather than the core socio-communicative deficits of ASD. Despite strict adherence to these gold-standard protocols, clinical practice frequently encounters a significant subset of patients who do not achieve expected developmental milestones or behavioral stabilization, leading to the complex and often debated phenomenon of treatment resistance.

Defining "resistance" in ASD is significantly more challenging than in episodic psychiatric disorders like major depression or schizophrenia. In ASD, resistance is not merely the absence of symptom remission, as ASD is a lifelong neurodevelopmental condition, but rather a persistent failure to respond to at least two evidence-based interventions of adequate intensity, duration, and adherence. This definition must be multidimensional, encompassing several critical clinical domains. First, "Core Symptom Resistance" refers to the lack of progress in social communication despite high-intensity behavioral therapy. Second, "Maladaptive Behavior Resistance" involves persistent aggression, self-injury, or profound irritability that remains refractory to first-line atypical antipsychotics. Third, "Comorbidity-Driven Resistance" highlights how unrecognized ADHD, anxiety, or sleep disorders can mask treatment efficacy, creating a "pseudo-resistance" that complicates the clinical picture and leads to unnecessary polypharmacy.

Furthermore, the emergence of resistance is increasingly viewed through a neurobiological lens. Atypical neurodevelopmental trajectories, characterized by an functional imbalance between cortical regulatory systems and subcortical emotional salience networks, may underpin why certain individuals remain non-responsive to conventional sensory and behavioral modifications. Identifying these social-cognitive vulnerabilities early is essential for shifting the treatment paradigm from a trial-and-error approach to a more precision-based model. This requires a comprehensive diagnostic reevaluation, including a systematic search for underlying medical conditions such as epilepsy, gastrointestinal issues, or genetic syndromes that may contribute to the resistant phenotype. In conclusion, this presentation argues that breaking treatment resistance requires a paradigm shift from simple "symptom-matching" to a sophisticated "functional-behavioral and neurobiological" analysis. By standardizing the definition of resistance and distinguishing it from pseudo-resistance caused by untreated comorbidities, clinicians can better identify when to escalate care toward integrated multidisciplinary models. This foundational session sets the stage for the ÇÖZGEM model, which emphasizes a holistic synergy between psychiatric, neurological, and specialized educational perspectives to address the most complex and treatment-refractory ASD cases in a structured clinical setting.

Keywords: Autism spectrum disorder, Treatment resistance, Pseudo-resistance, Neurodevelopmental outcomes, Multidisciplinary approach, ÇÖZGEM model

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0523 - Current Evidence on Pharmacotherapy and Neuromodulation in the Treatment of Gambling Disorder

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Gambling disorder (GD) is a severe behavioral addiction characterized by impaired impulse control, reward dysregulation, and persistent gambling despite negative psychosocial consequences. Despite its significant public health impact, there are currently no FDA-approved medications for GD, prompting researchers to investigate both off-label pharmacotherapies and novel, circuit-level neuromodulation techniques.

Current evidence indicates that opioid receptor antagonists are the most clinically validated medications for GD. Recent network meta-analyses demonstrate that nalmefene and naltrexone yield the highest efficacy in reducing gambling symptom severity and improving quality of life compared to placebo, although they are also associated with lower tolerability and higher dropout rates due to adverse side effects. Serotonergic agents, including selective serotonin reuptake inhibitors (SSRIs) such as paroxetine and fluvoxamine, have shown effectiveness in reducing gambling frequency and compulsivity, particularly when administered at higher doses or in patients without severe comorbid depression. Glutamatergic agents (e.g., N-acetylcysteine, topiramate) show preliminary promise for patients exhibiting high impulsivity, while mood stabilizers like lithium are highly beneficial for individuals with co-occurring bipolar affective disorder. Conversely, dopamine modulators and atypical antipsychotics, such as olanzapine, have generally failed to show significant superiority over placebo.

Non-invasive brain stimulation techniques offer a safe and well-tolerated alternative by directly targeting the prefrontal networks responsible for executive control, decision-making, and craving regulation. High-frequency repetitive transcranial magnetic stimulation (rTMS), most frequently targeting the left dorsolateral prefrontal cortex (DLPFC), has consistently demonstrated success in substantially reducing cue-induced gambling cravings and severity. Recent clinical adaptations suggest that combining rTMS with simultaneous symptom provocation (e.g., exposing the patient to gambling-related cues during stimulation) may further enhance therapeutic outcomes by activating maladaptive neural circuits during the intervention. Similarly, transcranial direct current stimulation (tDCS) applied to the DLPFC or ventromedial prefrontal cortex (vmPFC) has been shown to decrease risk-taking, improve cognitive flexibility, and reduce delay aversion.

While nalmefene and naltrexone currently possess the most robust supportive evidence among pharmacological options, tolerability issues highlight a critical need for alternative and adjunctive treatments. Neuromodulation, particularly high-frequency rTMS over the left DLPFC, provides a promising, effective non-pharmacological intervention, especially for patients who refuse or do not respond well to medications. Future large-scale, double-blind randomized controlled trials are essential to standardize stimulation parameters, optimize drug dosing, and evaluate the long-term efficacy of these emerging treatments.

Keywords: Gambling, Drug Therapy, Transcranial Magnetic Stimulation

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0524 - Wearable Technologies and Digital Phenotyping: Behavioral Biomarkers in Depression

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Major depressive disorder is a highly prevalent, heterogeneous, and disabling condition, and this heterogeneity continues to challenge both diagnosis and longitudinal monitoring. Conventional assessment in depression still depends heavily on episodic clinical interviews and self-report scales, which are valuable but limited by recall bias, sparse sampling, and reduced sensitivity to day-to-day fluctuations. Digital phenotyping offers a complementary approach by enabling moment-by-moment quantification of behavior and physiology in naturalistic settings through smartphones and wearable devices. This approach aligns with the broader goal of measurement-based and more personalized psychiatry.

Wearable technologies offer a promising framework for converting raw sensor-derived information into clinically interpretable behavioral biomarkers in depression. Wearable systems can assess multiple domains relevant to depressive psychopathology, including psychomotor activity, sleep and circadian regulation, cardiovascular indices such as heart rate and heart rate variability, and broader patterns of mobility, routine, and social engagement. The clinical relevance of these signals lies in their passive and continuous acquisition under real-world conditions, which may reduce patient burden, minimize recall bias, and provide a more ecologically grounded account of temporal symptom fluctuations.

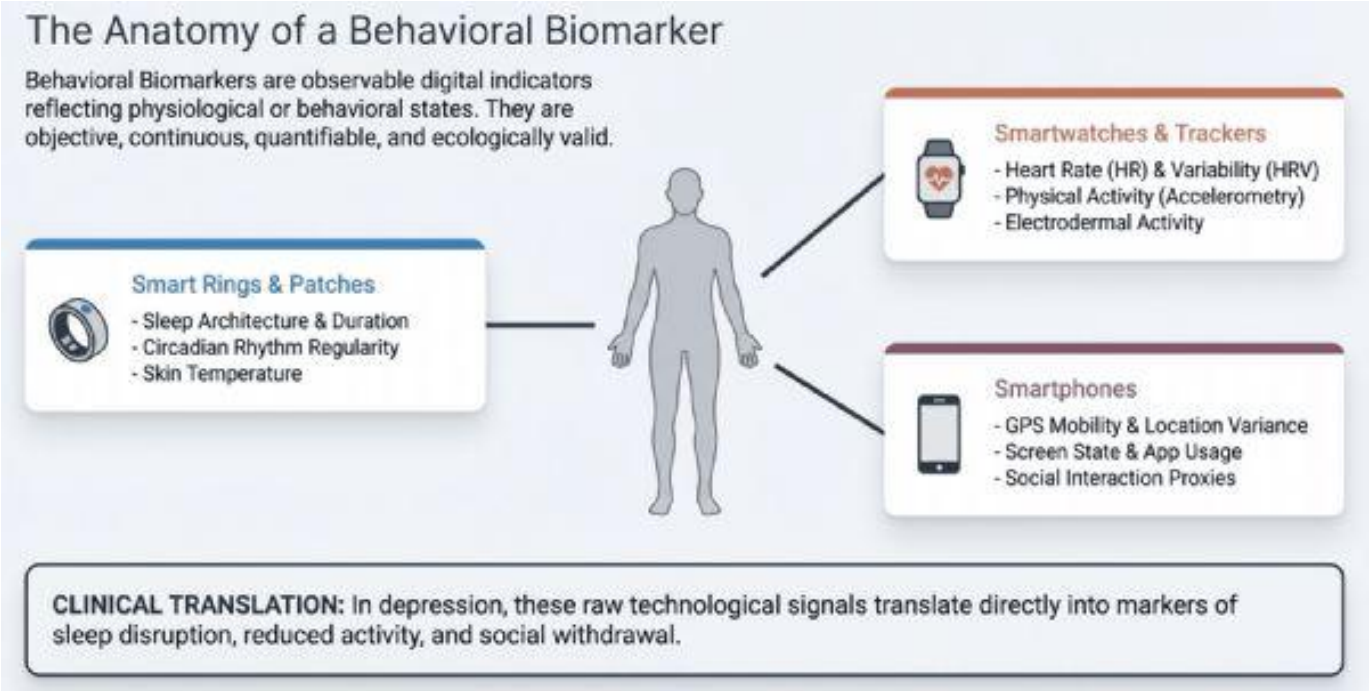
Accumulating evidence indicates that multimodal digital biomarkers may distinguish depressive from non-depressive states, while also contributing to symptom severity estimation and the detection of temporal clinical change. Longitudinal studies integrating smartphone and wearable data have identified meaningful differences across sleep, physical activity, phone use, and GPS-derived mobility, with machine learning models demonstrating moderate discriminatory performance. Emerging findings also suggest that personalized models may outperform generalized approaches, underscoring the substantial interindividual variability of depressive digital phenotypes. In adults with major depressive disorder, passive sensing data further indicate that profiles characterized by sleep dysregulation, persistently reduced heart rate variability, and diminished social engagement may represent clinically meaningful subgroups associated with poorer functioning, even when symptom severity alone does not clearly differentiate them.

Despite this promise, the current evidence base remains limited by small or selective samples, heterogeneity in devices and analytic algorithms, missing data, variable adherence, and persistent uncertainty regarding which digital features are sufficiently robust, scalable, and disorder-specific for clinical application. Even seemingly simple indicators, such as wearable wear time, may provide clinically relevant information, although their interpretability is likely to depend on integration with broader behavioral, sleep-related, and physiological measures. In addition, translating digital phenotyping into psychiatric practice requires careful consideration of privacy, informed consent, data governance, patient trust, and the ethical boundaries of continuous monitoring.

Wearable technologies and digital phenotyping should be understood not as substitutes for clinical assessment, but as complementary tools that can enhance its continuity and ecological depth. Their promise lies in improving the early detection of clinical worsening, the monitoring of treatment response and relapse risk, and the personalization of depression care.

Keywords: Behavioral biomarkers, depression, digital phenotyping, passive sensing, wearable technologies

Behavioral Biomarkers in Digital Phenotyping of Depression



Conceptual model showing how smartphone- and wearable-derived signals may translate into clinically meaningful behavioral markers in depression.

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0525 - Self-Compassion as A Transdiagnostic Construct: Shared Mechanisms and Psychological Flexibility

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Self-compassion has emerged as an important concept in emotional regulation and resilience in psychiatric disorders. Defined by self-kindness, common humanity, and mindfulness, self-compassion reflects how individuals relate to themselves in the face of distress and failure [1]. Rather than being specific to a disorder, self-compassion may function as a transdiagnostic process.

Self-criticism and shame are maladaptive self-related processes linked to an overactivated threat system. Self-compassion appears to counter these processes within a framework in which emotional functioning can be understood in terms of three interacting systems—threat, drive, and soothing systems—with self-compassion primarily associated with activation of the soothing system [2]. Research has revealed strong associations between self-compassion and psychological well-being, and negative relationships with anxiety, depression, and stress [3]. In ADHD, emotion dysregulation is a major source of impairment [4], and lower self-compassion and higher levels of self-criticism are also observed. Similar processes are present in mood and trauma-related disorders, where emotion dysregulation and maladaptive self-evaluative processes are core features. Self-compassion may address these shared vulnerability mechanisms across diagnostic categories.

Psychological flexibility refers to the ability to adapt to situational demands, shift cognitive stance and behavioral responses, and engage with internal experiences in a flexible and value-aligned manner [5]. From this perspective, self-compassion may be conceptualized as a self-related process supporting psychological flexibility through its effects on shared vulnerability mechanisms.

Self-compassion may serve as a transdiagnostic construct by reducing self-criticism and enhancing psychological flexibility, providing a framework for understanding vulnerability and resilience. This perspective may help bridge symptom-focused and process-based approaches in psychopathology research and treatment.

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Keywords: Emotion regulation, Psychological flexibility, Self-compassion, Self-criticism, Transdiagnostic processes

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0526 - Post-SSRI Sexual Dysfunction (PSSD): Neurobiological Mechanisms and Pathophysiology

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Post–selective serotonin reuptake inhibitor sexual dysfunction (PSSD) is an enduring iatrogenic condition in which sexual dysfunction that arises during treatment with selective serotonin reuptake inhibitors (SSRIs) or serotonin–norepinephrine reuptake inhibitors (SNRIs) persists for >=6 months after drug discontinuation. Emerging regulatory recognition and the introduction of a MedDRA code underscore that PSSD is a distinct clinical entity rather than a variant of depression or transient withdrawal, yet the underlying mechanisms remain incompletely characterized and incidence and prevalence are still unknown because of major methodological and contextual barriers, including patient underreporting and limited clinician awareness.

A multidomain symptom cluster (sexual, sensory, autonomic, and cognitive) was linked to persistent alterations in serotonergic signaling, with a central role proposed for 5 HT receptor downregulation and desensitization and potential downstream disturbance of neurosteroid and oxytocin systems by Klaas et al., which may contribute to reduced sexual desire, anorgasmia, and "emotional blunting."[1] Conceptual models of PSSD posit that chronic SSRI/SNRI exposure induces long lasting changes in serotonergic (and possibly noradrenergic) neurotransmission within corticolimbic and spinal sexual circuits, with secondary effects on dopaminergic reward pathways and neuroendocrine modulators of arousal and bonding.[1]

Objective clinical data support additional peripheral neurovascular and sensory pathology. In the largest clinical series to date, a cohort of 43 men meeting syndromic criteria for PSSD (mean age 31 years) showed high rates of severe erectile dysfunction on standardized measures, frequent hypoactive sexual desire, orgasmic dysfunction, and decreased genital sensation. Doppler ultrasonography demonstrated erectile tissue inhomogeneity consistent with cavernosal fibrosis and reduced erectile tissue expandability in most evaluated patients, while quantitative sensory testing revealed abnormal vibration and thermal thresholds in nearly 90% of those with orgasmic dysfunction and/or genital numbness, suggesting small fiber or peripheral neuropathic involvement.[2] At the population level, commentary on incidence highlights formidable obstacles to epidemiologic quantification, including challenges in designing suitable longitudinal studies, difficulty disentangling persistent dysfunction from withdrawal phenomena, withdrawal related inability to discontinue therapy, embarrassment and non disclosure of sexual symptoms, and failure to recognize the link between enduring dysfunction and prior SSRI/SNRI exposure.[3]

Taken together, current evidence supports a multidimensional pathophysiologic model in which enduring post treatment alterations in serotonergic and possibly noradrenergic systems interact with neurosteroid and oxytocin signaling and with peripheral penile vascular and sensory nerve changes to produce persistent sexual dysfunction in susceptible individuals. However, data remain largely inferential, with few mechanistic biomarkers and no established disease modifying treatments. Future research priorities include standardized diagnostic criteria, prospective longitudinal cohorts, receptor imaging, detailed sensory phenotyping, and pharmacogenetic studies to delineate risk factors, elucidate mechanisms, and guide preventive and therapeutic strategies.

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Keywords: PSSD, serotonergic dysregulation, sexual dysfunction

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0528 - Artificial Intelligence Applications in Psychopharmacology: Current Status from Diagnosis to Treatment

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Psychiatric disorders present longstanding diagnostic and therapeutic challenges, largely due to the absence of objective biomarkers and the subjective nature of clinical assessment. Artificial intelligence (AI) technologies are increasingly being explored as tools to support clinical decision-making in psychopharmacology, though their large-scale adoption in routine practice remains limited. This presentation aims to provide a realistic overview of the current state of AI applications in psychopharmacology, from diagnostic support to treatment optimization, alongside an honest appraisal of existing limitations.

In the diagnostic domain, machine learning models trained on neuroimaging, electronic health records, and behavioral data have demonstrated potential in supporting the identification of psychiatric disorders such as major depressive disorder, bipolar disorder, and schizophrenia. Natural language processing techniques can extract clinically relevant information from unstructured physician notes in electronic health records and convert it into structured diagnostic cues, offering a supplementary layer of assessment alongside clinical judgment. However, most studies remain confined to single-center cohorts, and external validation across diverse populations is still limited.

Regarding pharmacological treatment, machine learning algorithms have shown promise in predicting individual patient responses to psychiatric medications by analyzing genetic, metabolic, and clinical data, with the potential to reduce trial-and-error prescribing. In the area of drug-drug interaction management, models such as DrugBERT and Graph Neural Networks have improved the accuracy of interaction prediction by processing large-scale biomedical literature and molecular data. Decision support systems integrating pharmacogenomic data and patient-specific clinical parameters have been proposed to assist in the selection of antidepressants, antipsychotics, and mood stabilizers, though their clinical implementation remains in early validation phases.

Despite these developments, significant barriers to real-world deployment persist. Concerns around data privacy, algorithmic bias, lack of model interpretability, and underrepresentation of marginalized groups in training datasets continue to undermine the reliability and equity of AI tools in psychiatric settings. Furthermore, the absence of standardized validation frameworks and limited clinician preparedness for AI integration pose practical challenges that must be addressed before these tools can be responsibly embedded into clinical workflows.
In conclusion, AI holds genuine but carefully qualified promise in psychopharmacology. Current evidence supports its potential as a supplementary decision-support tool rather than a replacement for clinical expertise. This presentation will review findings from studies published between 2023 and 2025, discuss methodological strengths and limitations, and outline realistic pathways for the responsible integration of AI into psychopharmacological practice.

Keywords: Artificial intelligence, machine learning, psychiatric diagnosis, psychopharmacotherapy

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0529 - Is the Monoamine Hypothesis of Depression Still Valid?

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Major depressive disorder is a significant mental disorder that is highly prevalent in both children and adults, leading to a decline in quality of life and a deterioration in social and family life (1). However, due to its multidimensional etiology and complex phenomenological characteristics, current theories aimed at explaining the pathophysiology of depression have not yet reached an ultimate level. In the 1950s, the role of monoamines in depression was emphasized, a hypothesis termed the biogenic amine or monoamine hypothesis was proposed, suggesting that depression could be a functional deficiency of monoamines in the synaptic cleft, and it was thought that this might be involved in the mechanisms of action of drugs used in the treatment of depression, selective serotonin reuptake inhibitors, monoamine oxidase inhibitors, tricyclic antidepressants (2). However, the fact that clinical response emerges weeks later despite drugs increasing synaptic monoamine levels within hours, and the observation of treatment resistance in a significant portion of cases, has pointed to the existence of different mechanisms in the pathophysiology. In line with increasing scientific evidence, the potential interactions of genetic, epigenetic, and environmental effects have been more clearly established; evidence regarding the role of the hypothalamo-pituitary-adrenal axis and the influence of various immunologic and endocrinologic factors has increased, and the potential effects of neuroinflammation have been examined in various studies. It has been revealed that there may be certain differences in both the functional and structural characteristics of the brain, especially in the prefrontal cortex, amygdala, and hippocampus (3). In this presentation, the limitations of the monoaminergic system in explaining the pathophysiology of major depressive disorder alone will be reviewed, and next-generation biomarkers and neuroplasticity-based treatment strategies will be discussed in the light of current evidence.

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Keywords: Major Depressive Disorder, Monoamine Hypothesis, Antidepressants

0530 - Multidisciplinary Approaches to Treatment Resistance in Autism Spectrum Disorder and the ÇÖZGEM Model

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Treatment resistance in Autism Spectrum Disorder (ASD) represents a significant clinical challenge, particularly in cases where core symptoms and associated behavioral, emotional, and functional impairments persist despite evidence-based interventions. While pharmacological and behavioral treatments constitute the backbone of standard care, a considerable subgroup of children continue to demonstrate partial or minimal response. This highlights the necessity of adopting comprehensive, individualized, and multidisciplinary frameworks that extend beyond conventional approaches.

This presentation aims to conceptualize treatment resistance in ASD from a developmental and systems-oriented perspective and to introduce the ÇÖZGEM (Child Special Needs Center) model as an integrative service delivery framework. Treatment resistance in ASD is not a unitary construct but rather emerges from the interaction of multiple factors, including neurodevelopmental heterogeneity, comorbid psychiatric conditions such as ADHD and anxiety disorders, sensory processing differences, family dynamics, environmental stressors, and variability in treatment adherence and access. Therefore, a fragmented or single-modality intervention approach is often insufficient.

The ÇÖZGEM model represents a structured, multidisciplinary system designed to address these complexities through coordinated care. It integrates child and adolescent psychiatry, clinical psychology, speech and language therapy, occupational therapy with a focus on sensory integration, special education, and family counseling within a unified clinical framework. A core principle of the model is the formulation of individualized treatment plans based on comprehensive biopsychosocial assessment, including symptom severity, functional impairments, developmental profile, sensory reactivity patterns, and family context.

A key strength of the ÇÖZGEM approach lies in its emphasis on dynamic treatment planning and continuous outcome monitoring. Regular interdisciplinary case meetings enable the adjustment of interventions based on the child's evolving needs and response patterns. Persistent behavioral dysregulation, for example, may be conceptualized not solely as a pharmacological target but also as a manifestation of unmet sensory needs or communication deficits, prompting coordinated modifications across therapeutic domains.

Family involvement constitutes another central component of the model. Caregivers are actively engaged in psychoeducation, skill-building, and treatment implementation, which enhances generalization of therapeutic gains and reduces the risk of perceived treatment failure. Additionally, the model prioritizes early identification of resistance patterns, allowing for timely intensification or diversification of interventions.

Preliminary clinical observations suggest that such integrative and flexible care models may improve treatment responsiveness, functional outcomes, and caregiver satisfaction, particularly in complex and treatment-refractory cases. However, systematic research is needed to evaluate the efficacy and scalability of the ÇÖZGEM model across different clinical settings.

In conclusion, overcoming treatment resistance in ASD requires a paradigm shift from linear, modality-specific interventions to coordinated, multidisciplinary care systems. The ÇÖZGEM model offers a clinically grounded and adaptable framework that addresses the multifactorial nature of resistance and supports more effective, personalized treatment trajectories.

Keywords: Autism spectrum disorder, Treatment resistance, Pseudo-resistance, Neurodevelopmental outcomes, Multidisciplinary approach, ÇÖZGEM model

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0531 - The Concept of Children Drawn to Crime and Victims of Crime

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Ongoing changes in social order and family structure have been accompanied by a gradual increase in the number of children drawn to crime, making this issue an important area of concern for law, child and adolescent psychiatry, social work, education, and forensic sciences. At the same time, victims of crime in childhood constitute another vulnerable group whose needs often extend beyond legal assessment alone. Children's contact with the justice system should therefore not be regarded solely as a legal matter, but as a complex process shaped by developmental, psychological, familial, and social factors. A sound conceptual framework is essential for understanding both offending behavior and victimization, as well as the broader contexts in which these experiences arise.

In the Turkish legal system, the term child drawn to crime is preferred over stigmatizing expressions such as criminal child, reflecting a child centered and rights based approach. Under Law No. 5395 on Child Protection, a child drawn to crime is defined as a child under investigation or prosecution for an act defined as an offence by law, or a child for whom a security measure has been imposed because of such an act. Within the same legal framework, victims of crime are evaluated within the broader category of children in need of protection. This perspective indicates that children should not be reduced to judicial labels such as offender or victim, but should instead be regarded as individuals requiring protection, support, and developmentally appropriate intervention.

The core principles of the child justice system include safeguarding the best interests of the child, ensuring procedures appropriate to age and developmental level, preventing discrimination, enabling the participation of both the child and family in relevant processes, and preserving a rights based approach throughout judicial and psychosocial interventions. Accordingly, the child justice system extends beyond court proceedings and requires coordination among law enforcement, prosecutors, courts, social services, healthcare providers, and mental health professionals.

From the perspective of child and adolescent psychiatry, this issue is particularly significant. Children who come into contact with the justice system frequently present with trauma histories, behavioral problems, attention deficit hyperactivity disorder, substance use, mood symptoms, and multiple psychosocial adversities. Many children drawn to crime have also been exposed to neglect, abuse, poverty, exclusion, and chronic instability, whereas victims of crime may experience substantial emotional and developmental consequences if timely support is not provided. Accordingly, the relationship between children and crime cannot be adequately addressed through punitive or security oriented approaches alone; it also requires protective, restorative, and trauma informed perspectives.

This panel aims to discuss the core concepts related to children drawn to crime and victims of crime within a contemporary legal and psychosocial framework, and to emphasize why child justice should be approached not only through judicial definitions, but also through developmental, psychiatric, and social perspectives.

Keywords: Child and adolescent psychiatry, child justice system, child protection, children drawn to crime, juvenile justice, victims of crime

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0532 - The Role of SSRIs in the Management of Post-Traumatic Stress Disorder: Where Do We Stand?

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Post-traumatic stress disorder (PTSD) is a debilitating psychiatric condition that may develop following exposure to traumatic events such as abuse, violence, natural disasters, or severe accidents (1). In children and adolescents, PTSD can lead to significant impairments in emotional regulation, academic functioning, interpersonal relationships, and long-term mental health outcomes (2). Psychotherapeutic interventions, particularly trauma-focused cognitive behavioral therapy (TF-CBT), are considered first-line treatment in pediatric populations (4). However, pharmacological treatments are often considered in moderate to severe cases or when psychotherapy alone does not provide sufficient clinical improvement (2).

Among pharmacological options, selective serotonin reuptake inhibitors (SSRIs) are the most widely studied and prescribed medications for PTSD (3). SSRIs are believed to exert their therapeutic effects through modulation of serotonergic pathways involved in fear processing, emotional regulation, and stress response systems (3). Evidence from adult populations consistently demonstrates that SSRIs such as sertraline, paroxetine, and fluoxetine are effective in reducing core PTSD symptoms, including re-experiencing, avoidance, and hyperarousal (5). In contrast, evidence in children and adolescents remains limited and somewhat inconsistent (2). Several randomized controlled trials and open-label studies suggest that SSRIs may provide modest benefits, particularly for comorbid anxiety and depressive symptoms (2,3). However, findings across pediatric studies are heterogeneous, and some trials have failed to demonstrate clear superiority over placebo (3).

Concerns regarding medication side effects, developmental considerations, and the relatively strong efficacy of psychotherapy further complicate pharmacological decision-making in this population (2). Current clinical guidelines generally recommend SSRIs as a second-line or adjunctive treatment in pediatric PTSD (2,5). Their use is particularly considered when trauma-focused psychotherapy is insufficient or not accessible (4). Recent research has also highlighted the potential benefits of combined treatment approaches integrating pharmacotherapy with evidence-based psychotherapies (5). Nevertheless, significant gaps remain regarding optimal dosing strategies, long-term outcomes, and predictors of treatment response in youth (2).

In conclusion, SSRIs remain an important pharmacological option in the treatment of PTSD, with well-established efficacy in adult populations. However, evidence in children and adolescents is limited and inconsistent, and SSRIs are therefore generally recommended as second-line or adjunctive treatments. Clinical decision-making should take into account developmental factors, side effect profiles, and the priority of psychotherapy. Future research is needed to provide stronger evidence in pediatric populations and to inform more precise and individualized treatment approaches.

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Keywords: PTSD, SSRIs, trauma-focused cognitive behavioral therapy, combined treatment approach

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0533 - The Adult Face of Attachment: Lifelong Clinical and Relational Manifestations of Separation Anxiety

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Attachment theory provides an important theoretical framework for understanding the relational dimension of separation anxiety. Bowlby proposed that early interactions with primary caregivers lead to the development of internal working models, which subsequently shape expectations, emotional responses, and behavioral patterns in later interpersonal relationships. Building on this theoretical framework, Ainsworth classified attachment patterns as secure and insecure, identifying anxious/ambivalent and avoidant attachment patterns within the insecure category. Later, Main and Solomon expanded this classification by introducing disorganized attachment patterns. These attachment patterns are thought to play an important role in the development of emotional regulation capacities and interpersonal functioning.

Longitudinal studies indicate that attachment patterns formed during childhood show a certain degree of continuity into adulthood. The adult attachment framework proposed by Hazan and Shaver, and later expanded by Bartholomew and Horowitz, conceptualizes attachment as relatively stable relational orientations observed in close relationships. Based on internal working models, Bartholomew and Horowitz identified four primary adult attachment styles: secure, preoccupied, dismissing, and fearful. Insecure attachment patterns, particularly anxious or ambivalent attachment styles, have been shown to be strongly associated with separation anxiety symptoms in adulthood.

For many years, separation anxiety was primarily conceptualized as a disorder specific to childhood. However, accumulating scientific evidence and epidemiological studies over the past two decades have demonstrated that adult separation anxiety may represent the continuation of separation anxiety beginning in childhood, while in some individuals symptoms may emerge for the first time during adulthood. In a large-scale epidemiological study, Shear and colleagues reported that approximately 77.5% of individuals diagnosed with Adult Separation Anxiety Disorder (ASAD) experienced symptom onset during adulthood. In the same study, the lifetime prevalence of ASAD was estimated to be approximately 6.6%, while the one-year prevalence was around 1.9%. The conceptualization of Adult Separation Anxiety Disorder and its inclusion in DSM-5 indicate that attachment-related psychopathology may extend across the lifespan.

From a clinical perspective, adult separation anxiety is characterized by excessive and developmentally inappropriate distress related to real or anticipated separation from significant attachment figures. According to DSM-5 diagnostic criteria, the condition is characterized by symptoms such as intense anxiety related to separation from attachment figures, persistent worries about harm befalling them, avoidance of being alone, and marked distress in situations involving separation, and it must lead to clinically significant impairment in functioning. In this context, manifestations such as fear of abandonment and difficulty tolerating being alone may emerge as important clinical features that also affect interpersonal functioning.

This presentation aims to review the current literature on adult separation anxiety from epidemiological, clinical, and relational perspectives, and to discuss the lifelong manifestations of attachment-based separation anxiety and their implications for psychiatric practice.

Keywords: Adult separation anxiety disorder, attachment styles, attachment theory

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0534 - Working With Attention Deficit and Hyperactivity Through Schema Therapy: A Practical Mode-Based Framework

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Attention-Deficit/Hyperactivity Disorder (ADHD) is a complex neurodevelopmental condition that extends beyond attentional difficulties and impulsivity, significantly impacting emotional regulation, interpersonal functioning, and self-concept across the lifespan. In clinical practice, focusing solely on behavioral symptoms often leads to limited and short-term improvements. This course proposes a practical, schema therapy-based, mode-focused framework that enables clinicians to understand and intervene at a deeper emotional level.

The workshop begins with a brief overview of ADHD, covering its neurobiological basis, core symptom clusters, and common clinical presentations. This is followed by an introduction to the schema therapy model, including core emotional needs, early maladaptive schemas, and schema modes. Particular emphasis is placed on how unmet needs and repeated relational patterns shape the emergence of maladaptive coping responses in individuals with ADHD.

Building on this foundation, the course explores the schema and mode profiles frequently observed in individuals with ADHD. Modes such as the Impulsive Child, Angry Child, and Inattentive/Disorganized modes are discussed in relation to the commonly observed schema of insufficient self-control/self-discipline. Participants will learn how these modes manifest in everyday functioning, including emotional outbursts, low frustration tolerance, and difficulties in sustaining attention and goal-directed behavior.

A central focus of the workshop is strengthening the "Healthy Adult" (Wise/Smart Mode) through resource-oriented interventions. Techniques aimed at enhancing positive and functional modes—referred to as the "Smart Team"—are introduced, with an emphasis on building self-regulation, flexibility, and internal guidance systems. Participants are encouraged to conceptualize treatment not only in terms of reducing maladaptive modes but also actively cultivating adaptive ones.

Practical tools for assessment and intervention are demonstrated throughout the course. These include creating a "Mode Sketch" to visually map the individual's internal system, beginning with positive modes to increase engagement. The "Mode Interview" technique is introduced to facilitate direct dialogue with different parts, helping individuals understand the function and impact of their behaviors through guided questioning.

Experiential techniques are then presented to address common ADHD-related challenges. Chair work is used to manage impulsive and self-critical modes by introducing compassionate limits and internal structure. Imagery-based interventions, including inner house work, are utilized to address interpersonal difficulties and strengthen internal resources. These techniques are adapted to be developmentally appropriate and engaging, incorporating visual and symbolic elements when needed. Experiential techniques will be briefly presented. Finally, especially for children with ADHD, the role of caregivers and significant others is emphasized through a schema-informed coaching approach. Participants will learn how relational patterns and significant others' schemas and modes interact with ADHD-related difficulties, and how involving the broader relational system can support the generalization of therapeutic gains into daily life.

This course offers a concise, experience-oriented framework designed for clinicians working with individuals with ADHD, aiming to enhance their ability to address attention and self-regulation difficulties through a mode-based, integrative schema therapy perspective.

Keywords: ADHD, Schema Therapy, Schema Modes, Psychotherapy

AuthorToEditor: This workshop focuses on how ADHD can be conceptualized and treated using a schema therapy and mode-based framework. It aims to provide clinicians with practical tools to understand and work with ADHD through schema modes and experiential techniques in clinical practice. ORCID: 0000-0001-7557-0778

0535 - Emotion Regulation Difficulties in Adult Attention Deficit Hyperactivity Disorder: A Mediating Mechanism of Functional Impairment?

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Emotion dysregulation (ED), characterized by emotional lability, heightened emotional reactivity, and deficient emotion regulation strategies, is increasingly conceptualized as a core dimensional feature of adult attention deficit hyperactivity disorder (ADHD) rather than a secondary consequence. Accumulating evidence suggests that ED may function as a primary mediating mechanism through which core ADHD symptoms translate into global functional impairment across occupational, social, and interpersonal domains. This presentation examines recent findings from mediation analyses and structural equation modeling studies investigating this mechanistic pathway, evaluates the domain-specific functional consequences of ED in adult ADHD, and discusses the therapeutic implications of directly targeting emotion regulation difficulties.

Studies employing mediation analysis and structural equation modeling consistently demonstrate that ED serves as a significant partial mediator between core ADHD symptoms and functional outcomes. Research utilizing the Comprehensive Emotion Regulation Inventory reveals that specific ED dimensions, including emotional impulsivity and strategy application deficits, are associated with large effect sizes (Cohen's d approximately 0.8–1.0) across work, academic, and relationship functioning domains. Structural equation modeling studies report that approximately 20–40% of the total effect of ADHD symptoms on functional impairment is transmitted through ED and related cognitive-emotional strategies, with indirect pathways consistently reaching statistical significance. Additional mediation analyses demonstrate that ED mediates the relationship between ADHD symptoms and depressive outcomes, further expanding the functional impact of this mechanism. These findings collectively position ED not merely as an additional symptom but as a substantial explanatory pathway for the functional burden of adult ADHD.

Domain-specific analyses reveal that while ED significantly impairs occupational functioning through increased workplace conflict, job instability, and performance fluctuations, the most pronounced and consistent effects emerge in romantic and interpersonal relationships. Emotional lability, low frustration tolerance, and reactive anger contribute to hostile conflict patterns, lower dyadic adjustment, and elevated relationship dissolution rates. The relational domain demonstrates the largest effect sizes in studies comparing ED-related functional impairment across life areas, suggesting that intimate relationships represent the most vulnerable functional target of ADHD-associated emotion dysregulation.

From a therapeutic perspective, systematic reviews of pharmacological interventions indicate that stimulants and atomoxetine reduce ED symptoms with small-to-moderate effect sizes, with emotional behavior improvements ranging from 36–129% relative to non-emotional domains. Notably, emerging evidence suggests that changes in ED may predict functional improvement more robustly than changes in core ADHD symptoms alone. Psychotherapeutic approaches specifically targeting ED, including blended digital-group interventions and programs incorporating dialectical behavior therapy and mindfulness-based components, demonstrate large pre-post reductions in ED scores with parallel improvements in quality of life and social functioning. These ED-targeted programs appear to provide additional functional gains beyond standard symptom-focused cognitive behavioral therapy.

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Keywords: Adult ADHD, emotion dysregulation, functional impairment, mediation analysis, treatment outcomes, emotional lability

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0536 - The Challenge of Treating the Clinically High-Risk Group for Psychosis

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Clinical High Risk for Psychosis (CHR-P) represents a pre-psychotic clinical state and is a key target within the early intervention paradigm. CHR-P individuals are defined by the presence of attenuated psychotic symptoms (APS), brief limited intermittent psychotic symptoms (BLIPS), or genetic risk accompanied by functional decline. This group is associated with a high burden of disability and significant psychiatric morbidity, highlighting its clinical importance.

The risk of transition to psychosis in CHR-P individuals is dynamic, with an estimated rate of approximately 15–25% within 2–3 years. However, the majority of individuals do not develop psychosis, creating substantial uncertainty in clinical decision-making. Furthermore, CHR-P is a heterogeneous condition, with varying trajectories. Comorbid depression and anxiety are common, and functional impairment is often prominent.

The role of psychopharmacology in preventing psychosis remains controversial. Current evidence indicates that antipsychotic medications do not provide robust or consistent prevention of transition to psychosis. The 2025 WPA meta-analysis, including 24 randomized controlled trials and approximately 3,000 CHR-P individuals, demonstrated no superior intervention. Antipsychotics show only small effects on symptoms and are associated with significant side effects, including metabolic complications, extrapyramidal symptoms, and prolactin elevation.

Accordingly, current guidelines recommend psychosocial interventions as the first-line approach, while antipsychotics should be reserved as a last option. Clinical management should prioritize the treatment of existing symptoms rather than attempting to prevent psychosis directly. Interventions targeting depression, anxiety, sleep disturbances, and substance use are essential components of care.

The management of CHR-P individuals also involves significant ethical challenges, including the risk of labeling and stigma, diagnostic uncertainty, lack of reliable biomarkers, and the complexity of a “watchful waiting” approach. A key clinical question remains whether treatment should focus on reducing the risk of psychosis or alleviating current distress.Emerging directions in this field include personalized care, a focus on functional recovery, and the integration of digital and virtual reality–based interventions. In conclusion, there is currently no pharmacological strategy with proven efficacy in preventing psychosis in CHR-P populations. Therefore, individualized care, family involvement, and symptom-oriented treatment should be prioritized. Not all CHR-P individuals require medication, and functional outcomes should be considered more important than transition alone.

Keywords: Clinical high risk for psychosis (CHR-P);, Attenuated psychotic symptoms (APS);, Early intervention;, Psychosis prevention

0537 - Theoretical and Clinical Perspectives on Resistance in the Therapeutic Process in Sex Therapy

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Resistance encountered in sex therapy should not be conceptualized merely as non-compliance or a lack of motivation. Instead, it must be understood as a multifaceted clinical phenomenon arising from the complex interplay of individual, relational, and sociocultural dynamics. Resistance manifests in various forms, such as irregular attendance, non-adherence to between-session assignments, avoidant communication patterns, externalization of symptoms, and partner-oriented blame. Essentially, these behaviors often express unconscious fears of change, shame, performance anxiety, and underlying relational conflicts.

Psychotherapy research consistently demonstrates that resistance and therapeutic alliance ruptures are prevalent throughout treatment and significantly impact outcomes. In the context of sexual dysfunction, factors such as avoidance, shame, and negative outcome expectancies may substantially limit both help-seeking behavior and treatment adherence. Specifically, cognitive avoidance and performance-focused self-monitoring are known to maintain sexual dysfunctions during therapy (1). Furthermore, fear, avoidance, and the perception of bodily threat are closely associated with treatment resistance in cases of vaginismus and dyspareunia (2).

Rather than an obstacle to be eliminated, treatment resistance should be viewed as a clinically significant indicator of the symptom's function. The therapist's primary task is to decode what the resistance—and the symptom it reinforces—signifies for the individual. Symptoms often serve a regulatory function, shielding the patient from fears of rejection, inadequacy, or intimacy. Therefore, the therapist must recognize the protective meaning underlying the resistance, strengthen the therapeutic alliance, and foster a safe environment to explore the patient's ambivalence toward change.

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Keywords: Cognitive avoidance, sexual dysfunction, therapeutic alliance, treatment resistance

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0539 - Lateral Habenular Tet2 Mediates Social Deficits

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Background and Objective: The lateral habenula (LHb) is a critical brain region involved in regulating social behaviors, and its dysfunction has been implicated in social deficits such as those seen in autism spectrum disorder. However, the molecular mechanisms by which the LHb modulates social behavior remain largely unknown. Ten-Eleven Translocation 2 (Tet2), a DNA hydroxymethylase involved in epigenetic regulation, has been linked to depressive-like behaviors, but its role in the LHb and in social behavior is unclear.

Methods: To investigate the role of Tet2 in the LHb in social behavior, we generated *Tet2* conditional knockout (cKO) mice and performed behavioral assays including the three-chamber sociability test (TCT), open field test, and marble-burying test. Using viral-mediated gene knockdown and rescue strategies, we manipulated Tet2 expression specifically in the LHb and in glutamatergic neurons. We assessed neuronal activity via miniature two-photon calcium imaging, and performed genome-wide 5hmC sequencing to identify downstream molecular targets. Results: We found that Tet2 is highly expressed in the LHb. *Tet2* cKO mice exhibited impaired sociability without changes in locomotion, stereotyped behaviors, or anxiety-like behaviors. Conditional knockdown of Tet2 in the LHb recapitulated this social deficit, while restoring Tet2 expression in the LHb of *Tet2* cKO mice rescued sociability. Tet2 deficiency increased neuronal activity in the LHb, and inhibition of glutamatergic neuron excitability restored social behavior. Mechanistically, Tet2 deficiency reduced 5hmC modifications on the promoter of the autism-related gene *Sh3rf2*, leading to decreased Sh3rf2 expression. Overexpression of Sh3rf2 in the LHb of *Tet2* cKO mice rescued sociability.

Conclusions: Our findings identify Tet2 in the LHb as a key epigenetic regulator of social behavior, acting by modulating Sh3rf2 expression and neuronal activity. These results suggest that Tet2 in the LHb represents a potential therapeutic target for social behavior deficits in disorders such as autism.

Keywords: Ten-eleven translocation 2, lateral habenula, sociability

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0540 - The Impact of Emotion Regulation Skills on Parenting in Adults With Attention Deficit Hyperactivity Disorder

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Attention deficit hyperactivity disorder (ADHD) in adults is increasingly recognized as a condition that extends beyond core symptoms of inattention and impulsivity, with emotion dysregulation (ED) emerging as a clinically significant dimensional feature. Adults with ADHD frequently exhibit heightened emotional reactivity, poor frustration tolerance, and difficulty modulating anger, which profoundly affect interpersonal functioning, particularly in the parenting domain. This presentation reviews recent evidence on how emotion regulation deficits in adults with ADHD influence parenting behaviors, parent-child interactions, and family cohesion, and discusses the broader clinical implications for assessment and intervention.

Longitudinal and cross-sectional studies demonstrate that higher levels of ADHD-related ED in parents, especially anger reactivity and low frustration tolerance, are associated with harsher, more inconsistent disciplinary practices and reduced supportive parenting behaviors. Mediation analyses reveal that emotion regulation difficulties represent a specific pathway through which parental ADHD symptoms translate into negative parenting outcomes, even after controlling for parental depression and child behavioral problems. Research indicates that parents with ADHD who struggle with emotion regulation tend to respond to child noncompliance or emotional distress with disproportionate anger, punitive reactions, or complete withdrawal, creating coercive interaction cycles that reinforce child externalizing and internalizing symptoms.

The bidirectional nature of these dynamics is well established. Children's ADHD symptoms and oppositional behaviors increase parental stress and negative affect, which in turn exacerbate overreactive parenting and emotional exhaustion, forming a self-perpetuating cycle. Twin and sibling studies indicate that while the intergenerational transmission of ADHD behaviors is largely genetically mediated, parental ED contributes to the emotional climate of the family through environmental pathways. Recent longitudinal evidence demonstrates that specific maternal emotion regulation difficulties, such as limited emotional awareness and impulsive responding, differentially predict emotion-focused parenting behaviors, which subsequently shape children's own emotion regulation capacities and developmental trajectories.

Beyond parenting, ED in adults with ADHD significantly impacts marital satisfaction and intimate partner relationships. Studies report that emotional lability, irritability, and poor conflict resolution skills in the ADHD partner are associated with higher marital conflict, lower dyadic adjustment, and increased risk of intimate partner violence. Emotion regulation difficulties account for a substantial proportion of variance in relationship satisfaction, highlighting ED as a central mechanism linking adult ADHD to relational distress. Additionally, differential diagnostic considerations are essential, as affective lability in adult ADHD overlaps with borderline personality disorder and bipolar disorder, yet differs in duration, triggers, and associated features. Emerging evidence also identifies ED as a key mediating variable in the relationship between adult ADHD and substance use disorders, where maladaptive emotion regulation strategies serve as a pathway to self-medication behaviors. Pharmacological treatment of ADHD with stimulants and non-stimulants has been shown to reduce emotional reactivity and improve overall functional outcomes, potentially creating a foundation for more effective parenting and relational functioning when integrated with targeted psychosocial interventions including emotion regulation skills training and mindful parenting programs. A comprehensive multimodal approach addressing both core ADHD symptoms and emotion regulation deficits in affected parents holds significant promise for improving outcomes across the entire family system.

Keywords: Adult ADHD, Emotion dysregulation, Family functioning, Interpersonal relationships, Parenting behaviors

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0541 - Personality Disorders and PTSD: Cracking the Code of Treatment Resistance

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Complex Traumatic Stress Disorder (CTSD) has emerged as a distinct clinical entity within the ICD-11 framework, characterized by its development from chronic and repeated exposure to traumatic events, often beginning in childhood. Unlike traditional Post-Traumatic Stress Disorder (PTSD), which typically follows a single or limited traumatic incident, CTSD is linked to prolonged and multifaceted trauma, particularly within interpersonal relationships. This extended exposure not only predisposes individuals to CTSD but also results in a broader and more complex symptom profile. In addition to the core PTSD symptoms of re-experiencing, avoidance, and hyperarousal, CTSD is marked by significant impairments in emotion regulation, negative self-perception, and persistent interpersonal difficulties. Patients frequently report overwhelming feelings of shame or guilt, alongside increased risks for depression, anxiety, personality disorders, and suicide. These additional psychopathological features underscore the clinical severity of CTSD compared to PTSD. The developmental nature of CTSD, stemming from early and chronic exposure to trauma, complicates the diagnostic process, particularly given its overlap with other psychiatric disorders such as borderline personality disorder. The absence of a separate classification in the DSM-5 further hinders the establishment of standardized diagnostic criteria and consistent clinical evaluations, thereby challenging both research efforts and therapeutic interventions. Treatment for CTSD necessitates an integrated, multi-modal approach. Initial management focuses on establishing stabilization and a secure therapeutic alliance, which is critical for addressing the complex needs of these patients. Once safety and stabilization are achieved, trauma-focused psychotherapeutic interventions become essential. These therapies are tailored to mitigate not only the core symptoms of PTSD but also the broader spectrum of disturbances in emotion regulation, self-esteem, and interpersonal functioning. Pharmacological treatments serve as adjuncts, targeting specific symptom clusters and comorbid conditions to enhance overall therapeutic outcomes. Complementary holistic approaches may also be incorporated to provide a comprehensive treatment strategy that addresses the physical, emotional, and relational dimensions of CTSD. In summary, CTSD represents a challenging and multifaceted disorder that significantly differs from classical PTSD in both its etiology and clinical presentation. Improving diagnostic clarity, raising awareness, and refining treatment protocols are imperative to better serve individuals who have experienced prolonged and intense trauma. Continued research into the underlying mechanisms and tailored therapeutic interventions for CTSD is crucial to enhance patient outcomes and quality of life.

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Keywords: PTSD, Complex PTSD, Personality Disorder
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0542 - Neuropsychological Challenges in Children and Adolescents Diagnosed with PTSD and Complex PTSD

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The impact of trauma on cognitive functioning in children and adolescents diagnosed with Post-Traumatic Stress Disorder (PTSD) and Complex PTSD is notably evident, particularly in the domains of working memory and sustained attention. Working memory is a critical cognitive function for the temporary storage and processing of information. Research indicates that individuals with PTSD and Complex PTSD face significant difficulties, especially during complex tasks. Chronic stress resulting from traumatic experiences adversely affects the functioning of the prefrontal cortex, thereby diminishing working memory capacity. This reduction in capacity directly impacts both academic performance and social functioning.

Moreover, children with PTSD and Complex PTSD frequently exhibit difficulties in sustaining attention. These individuals demonstrate heightened sensitivity to external stimuli. The hypervigilance stemming from trauma redirects a child's attention continually towards perceived threats, making it challenging to concentrate on lessons and maintain engagement in group interactions within classroom settings. Consequently, both academic performance and the development of social skills are negatively affected, further exacerbating the psychological well-being of these children. Weaknesses in attention functioning pave the way for social conflicts, feelings of loneliness, and social exclusion, and are associated with secondary issues such as anxiety and low self-esteem. All of these findings suggest that difficulties in working memory and sustained attention create interrelated and compounding barriers in the daily lives of children and adolescents diagnosed with PTSD and Complex PTSD. Therefore, it is crucial that educational and therapeutic interventions are designed to focus not only on emotional regulation but also on cognitive functioning deficits. The integration of neuropsychological assessment findings into school environments and therapeutic processes should be regarded as a critical step towards trauma recovery and healthy development.

Keywords: Ptsd, Complex Ptsd, Neuropsychological Challenges

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0543 - What is the Prevalence and Impact of Comorbid Mental Disorders in Individuals at Clinically High Risk for Psychosis?

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Among individuals at clinical high risk for psychosis (CHR-P-UHR), non-psychotic comorbid mental disorders are highly prevalent, with approximately 60-80% of individuals meeting criteria for at least one additional psychiatric disorder. Mood and anxiety disorders are the most common comorbidities, although trauma-related conditions, obsessive-compulsive symptoms, substance use problems, and personality pathology are also frequently observed at clinically meaningful rates. These comorbid conditions contribute substantially to the clinical burden of CHR-P states, being associated with more severe attenuated psychotic symptoms, greater psychological distress, increased suicidality and self-harm risk, more prominent negative symptoms, apathy, and disorganized behavior. They are also consistently linked to poorer global and social functioning, reduced quality of life, and, in some cases, particularly poor functioning in relation to alcohol use disorder and schizotypal personality disorder. Longitudinal findings further indicate that non-psychotic comorbid disorders often persist or recur over time, while new disorders may emerge during follow-up, with persistent comorbidity representing a strong predictor of unfavorable long-term functional outcomes. At the same time, some evidence suggests that individuals with more complex comorbidity profiles may still achieve meaningful functional improvement with appropriate treatment.

In contrast, the relationship between comorbidity and transition to psychosis remains inconsistent. Evidence generally indicates that the presence of additional non-psychotic disorders does not substantially increase transition risk overall, and some mood and anxiety disorders may even be associated with lower transition rates in certain subgroups. However, longitudinal evidence also suggests that specific clinical characteristics, such as a history of depressive episodes, may be linked to a less favorable symptom trajectory and an elevated risk of transition. Cannabis misuse appears to be the most consistent exception, showing a more robust association with conversion risk across studies. Taken together, the current evidence does not support a single, uniform model in which comorbidity directly drives psychosis onset in all CHR-P individuals. Rather, comorbid disorders should be understood as core determinants of distress, symptom persistence, and functional impairment, underscoring the importance of a transdiagnostic clinical approach that addresses current psychopathology and functioning in addition to psychosis prevention.

Keywords: Clinical high risk for psychosis, Mood disorders, Psychiatric comorbidity, Transition to psychosis

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0544 - Complementary and Alternative Treatments for Resistant Symptoms in ASD - Current Evidence

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Autism Spectrum Disorder (ASD) is a heterogeneous neurodevelopmental condition characterized by persistent deficits in social communication and restricted, repetitive patterns of behavior. Despite advances in behavioral and pharmacological interventions, many individuals with ASD continue to exhibit treatment-resistant symptoms, including irritability, sleep disturbances, gastrointestinal dysfunction, and sensory dysregulation. These unmet clinical needs have increased interest in complementary and alternative medicine (CAM) as adjunctive therapeutic strategies.

The prevalence of CAM use in ASD is high, ranging from 28% to 95%, reflecting parental concerns regarding limited efficacy and potential adverse effects of conventional treatments, as well as a preference for more "natural" approaches. CAM modalities include biologically based treatments (e.g., dietary modifications, vitamin and mineral supplementation, omega-3 fatty acids, probiotics, melatonin), mind–body approaches (e.g., music therapy, yoga, massage), and sensory-based therapies (e.g., hippotherapy, sensory integration). These interventions are used to address both core symptoms and associated comorbidities such as sleep disturbances, gastrointestinal symptoms, and behavioral dysregulation.

However, current high-level evidence highlights important limitations. A recent umbrella review evaluating 248 meta-analyses across 19 CAM modalities found that no intervention demonstrated high-quality evidence supporting efficacy for either core or associated ASD symptoms. Although some interventions, including melatonin, music therapy, and animal-assisted therapies, showed statistically significant and sometimes large effect sizes, these findings were supported by low or very low levels of evidence.

More specifically, melatonin demonstrates relatively strong evidence for improving sleep disturbances, while music therapy shows moderate benefits in enhancing social communication and interaction. In contrast, widely used interventions such as gluten-free/casein-free diets, omega-3 supplementation, and probiotics yield inconsistent findings, and randomized controlled trials generally do not support their efficacy for core ASD symptoms. Additionally, chelation therapy and hyperbaric oxygen therapy are not recommended due to insufficient evidence and potential safety concerns.

From a pathophysiological perspective, the gut–brain axis has emerged as a key conceptual framework linking microbiota, immune regulation, and neurodevelopment. Although microbiota-targeted interventions may exert indirect neurobehavioral effects, current evidence is insufficient for routine clinical use.

Clinically, integrating CAM into ASD management requires a cautious, evidence-based, and patient-centered approach. Clinicians should actively inquire about CAM use, maintain open and non-judgmental communication, and align interventions with measurable treatment goals. CAM should be considered adjunctive rather than alternative to standard treatments. Within multidisciplinary care models such as ÇÖZGEM, CAM approaches can be incorporated into individualized treatment plans. The ÇÖZGEM model emphasizes coordinated care involving multiple professionals, enabling comprehensive and personalized interventions. This framework is particularly valuable for addressing treatment-resistant symptoms through multimodal strategies.

In conclusion, CAM interventions are widely used in ASD and may provide benefits for selected symptoms; however, current evidence supports their role primarily as adjunctive treatments within a multidisciplinary framework. Future research should prioritize well-designed randomized controlled trials to identify patient subgroups most likely to benefit.

Keywords: Autism spectrum disorder, Complementary and alternative medicine, Multidisciplinary Approach, Treatment resistance, ÇÖZGEM model

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0545 - Solution Proposals for the Relationship Between Crime and Children

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Suç ve çocuk ilişkisi, gelişimsel psikoloji, çocuk ve ergen psikiyatrisi, kriminoloji ve sosyal politika alanlarını kapsayan çok boyutlu bir konudur. Bu panelde, çocukların suça maruz kalma biçimleri (mağdur, tanık veya fail olarak) ve bu sürecin gelişimsel etkileri ele alınarak, önleme ve müdahaleye yönelik çözüm önerileri sunulacaktır.

Erken dönem olumsuz yaşantılar; aile içi işlev bozuklukları, ihmal, istismar ve toplumsal şiddete maruz kalma gibi faktörler, çocuklarda davranışsal sorunlar ve ilerleyen dönemde suç davranışı açısından önemli riskler oluşturmaktadır. Nörobiyolojik kırılganlıkların da bu süreçte rol oynayabileceği bilinmektedir. Bununla birlikte, güvenli bağlanma, okul bağlılığı ve erken ruh sağlığı müdahaleleri gibi koruyucu faktörler, risklerin etkisini azaltabilmektedir.

Sunumda çözüm önerileri çok düzeyli bir çerçevede ele alınacaktır: bireysel düzeyde erken tanılama ve ruh sağlığı hizmetlerine erişim, aile düzeyinde ebeveynlik becerilerinin güçlendirilmesi ve çevresel stresörlerin azaltılması, toplumsal düzeyde ise sosyoekonomik eşitsizliklerin azaltılması ve çocuk koruma sistemlerinin güçlendirilmesi. Ayrıca, sağlık, eğitim ve hukuk sistemleri arasındaki iş birliği ile travma odaklı yaklaşımların önemi vurgulanacaktır.

Sonuç olarak, suç ve çocuk ilişkisine yönelik etkili müdahaleler, bütüncül ve kanıta dayalı yaklaşımlar gerektirmektedir.

Keywords: Çocuk, çocuk ve ergen ruh sağlığı, erken müdahale, gelişimsel psikopatoloji, koruyucu faktörler, suç

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0546 - Family-Driven Meal Modifications and Accommodation in Eating Disorders

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Eating disorders are complex psychiatric conditions associated with significant medical, psychological, and social consequences. These disorders not only affect individuals but also profoundly impact family functioning. In children and adolescents, parents and caregivers play a central role in meal preparation, structuring eating routines, and monitoring eating behaviors. Therefore, family responses to eating disorder symptoms can significantly influence the course, severity, and maintenance of the illness (1).

In recent years, the concept of family accommodation has gained increasing attention in the literature. Family accommodation refers to changes in caregivers' behaviors aimed at reducing distress associated with the patient's symptoms. In the context of eating disorders, these behaviors are most commonly observed around mealtimes. Families may prepare "safe" foods preferred by the patient, avoid feared foods, modify portion sizes, allow meal skipping, or provide frequent reassurance during meals. Although these behaviors are typically driven by parental concern and attempts to reduce conflict, they may inadvertently contribute to the persistence of disordered eating patterns by reinforcing avoidance and limiting opportunities for adaptive coping (2).

Accumulating evidence suggests that higher levels of family accommodation are associated with increased symptom severity, greater caregiver burden, and poorer treatment outcomes. While accommodating behaviors may reduce anxiety for both the patient and family members in the short term, they may reinforce maladaptive patterns and hinder recovery in the long term. This process can be understood within a negative reinforcement framework, in which accommodating behaviors reduce distress in the short term but contribute to the maintenance of the disorder over time (3). Furthermore, emerging findings indicate that family accommodation may independently contribute to functional impairment beyond symptom severity alone. In this context, accommodation may also interfere with exposure to feared foods and disrupt the development of flexible eating behaviors, which are essential components of recovery (4).

In pediatric populations, the impact of family accommodation may be particularly pronounced, as parents typically have substantial control over meal preparation and eating routines. Consequently, family involvement has become a central component of evidence-based interventions. Family-Based Treatment (FBT), which has strong empirical support for adolescent anorexia nervosa, conceptualizes parents as a key resource in treatment and encourages them to take an active role in restoring healthy eating behaviors while gradually reducing accommodating responses (5). Addressing family accommodation within treatment may therefore be essential for improving both clinical and functional outcomes. Integrating caregivers into treatment planning may also enhance adherence and support the generalization of therapeutic gains to the home environment.

This presentation will review the current literature on family accommodation and family-driven meal modifications in eating disorders and discuss their clinical implications, particularly in children and adolescents. In addition, practical approaches to identifying and addressing accommodating behaviors in clinical settings will be presented. A better understanding of family accommodation may contribute to more effective, family-centered interventions and improved long-term outcomes.

Keywords: Adolescents, Caregiver Behavior, Eating Disorders, Family Accommodation, Family-Based Treatment, Meal Modifications

AuthorToEditor: Family Accommodation As a Transdiagnostic Mechanism in Child and Adolescent Psychopathology Family-Driven Meal Modifications and Accommodation in Eating Disorders Dr. Melike Ayşegül Kara Karaman Orcid id:0009-0003-9820-8423

0547 - Strategies to Overcome Resistance: Creative and Innovative Interventions in Sex Therapy

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Strategies to Overcome Resistance: Creative and Innovative Interventions in Sex Therapy

Resistance represents a frequent and clinically significant phenomenon in sex therapy. Nevertheless, it should not be reduced to noncompliance, insufficient motivation, or reluctance to change. Rather, resistance often reflects a protective and, at times, adaptive response shaped by shame, performance anxiety, fear of failure, bodily hyperarousal, relational vulnerability, and avoidance. Given that sexual difficulties are embedded in highly sensitive domains such as intimacy, self-image, control, and exposure, resistance in sex therapy commonly emerges across cognitive, affective, somatic, and interpersonal dimensions.

This presentation examines resistance in sex therapy from a clinically informed perspective and discusses creative and innovative interventions that may enhance therapeutic engagement when conventional verbal approaches remain limited. From this standpoint, the clinical task is not to confront resistance in a corrective or oppositional manner, but to explore its function and to utilize it as a meaningful point of entry into the therapeutic process. Such an approach requires a therapeutic stance characterized by curiosity, validation, pacing, and collaboration. Within this framework, several intervention strategies merit consideration. Reframing resistance as a form of self-protection may reduce defensiveness and strengthen the therapeutic alliance. Similarly, the flexible restructuring of homework assignments and therapeutic tasks may be particularly valuable when such interventions are experienced by individuals or couples as tests of adequacy, performance, or relational success. In addition, individualized adaptations of sensate focus exercises and graded exposure procedures may reduce avoidance by shifting the emphasis from performance-based outcomes to bodily awareness, tolerance, and shared experiential engagement. The presentation further addresses the role of symbolic and indirect methods in the management of treatment resistance. Metaphor, externalization, guided imagery, and selected experiential techniques may facilitate access to material that is otherwise experienced as overly threatening, shame-laden, or difficult to verbalize. In a similar vein, bibliotherapeutic resources and media-assisted reflective practices may serve as adjunctive tools by creating sufficient emotional distance while simultaneously supporting exploration, mentalization, and insight. Particular attention is also given to the relationship between body image, self-attunement, and sexual avoidance. In some patients, resistance may be closely associated with bodily alienation, self-critical attitudes, and negative sexual self-schemas. In such cases, body-focused self-care rituals, mindfulness-based interventions, and self-compassion-oriented practices may function as supportive adjuncts that facilitate reconnection with the body and reduce avoidance.

Overall, creative and innovative interventions in sex therapy should not be regarded as peripheral or eclectic additions, but as clinically meaningful strategies that complement established therapeutic approaches. When implemented within a secure therapeutic alliance and tailored to the needs of the individual or couple, such interventions may contribute to the reduction of resistance, the strengthening of therapeutic engagement, and the deepening of the therapeutic process.

Keywords: Sex therapy, treatment resistance, creative interventions, sensate focus, sexual avoidance

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0548 - Management of Psychiatric Comorbidities in Attention Deficit Hyperactivity Disorder

Fadime Bayraktar

Çiğli Eğitim ve Araştırma Hastanesi

Attention-Deficit/Hyperactivity Disorder (ADHD) is frequently characterized by a high rate of psychiatric co-occurrence, with longitudinal studies indicating that approximately 60-80% of affected children and adolescents meet the criteria for at least one comorbid condition. The presence of comorbidities, such as Anxiety Disorders, Oppositional Defiant Disorder (ODD), Tic Disorders, and Autism Spectrum Disorder (ASD), complicates the diagnostic process, worsens the long-term prognosis, and necessitates a more nuanced pharmacological approach.

This presentation aims to provide a comprehensive framework for the evidence-based management of these complex clinical profiles, focusing on treatment prioritization and potential drug-drug interactions. The primary challenge in managing comorbid ADHD lies in the "therapeutic hierarchy." For patients presenting with severe mood dysregulation or acute suicidality, stabilization of the mood disorder often precedes ADHD-specific interventions. In the context of Internalizing Disorders, such as Generalized Anxiety Disorder, clinicians must weigh the risk of stimulant- induced anxiety against the potential reduction in anxiety following improved executive functioning. Evidence suggests that Selective Serotonin Reuptake Inhibitors (SSRIs) can be safely combined with stimulants, or alternatively, Atomoxetine may serve as an effective monotherapy for both domains. Regarding Externalizing Disorders, ODD and Conduct Disorder require optimized stimulant dosing to mitigate aggression, often necessitating augmentation with alpha-2 agonists or low-dose atypical antipsychotics in refractory cases. The management of ADHD in the context of Tic Disorders has undergone a paradigm shift; contemporary meta-analyses debunk the myth that stimulants inevitably worsen tics, although alpha-2 agonists remain a first-line consideration due to their dual efficacy. Furthermore, treating ADHD in children with ASD requires heightened clinical vigilance due to increased sensitivity to adverse effects and lower overall response rates compared to neurotypical peers.

Pharmacokinetic considerations, particularly involving the Cytochrome P450 system (e.g., the inhibition of CYP2D6 by fluoxetine affecting atomoxetine metabolism), are vital for ensuring safety and preventing toxicity. In conclusion, the successful management of comorbid ADHD requires a personalized, rational polypharmacy approach that balances symptomatic relief with the minimization of the cumulative side-effect burden.

Keywords: ADHD, Comorbidity, Pharmacotherapy, Stimulants, Polypharmacy, Child

0550 - Current Pharmacological Treatment Approaches in Ultra-High Risk for Psychosis

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Ultra-High Risk for Psychosis (UHR-P) is a clinical condition emerging in adolescence or young adulthood through the interplay of genetic vulnerability, biological sensitivity, and environmental stressors. While individuals do not yet meet the diagnostic criteria for a full-threshold psychotic disorder, their risk of transitioning to psychosis is substantially higher than that of the general population. UHR-P should be conceptualized not merely as a 'pre-psychotic' stage, but as a critical window for early intervention, during which cognitive difficulties, negative symptoms, and functional decline may already be manifest.

Pharmacological interventions for UHR-P have been studied for three decades. Early research was driven by the objective to 'halt the illness' through antipsychotic treatment. However, since the mid-2000s, the field has increasingly questioned the efficacy, risk–benefit ratio, and long-term outcomes of antipsychotics. Currently, the aim of antipsychotic use in UHR-P is not to prevent the transition to psychosis, but to achieve clinical stabilization in carefully selected cases. Although short-term improvements in positive symptoms may occur, data suggesting potential adverse effects on cognitive domains—particularly in adolescents—underscore the need for caution.

A consistent theme across nine contemporary international guidelines is that pharmacological agents are not recommended as first-line treatment. While the NICE guideline advises against antipsychotic use for UHR-P, eight other guidelines reserve low-dose, time-limited antipsychotic treatment for cases where psychosocial interventions have proven insufficient and a rigorous risk–benefit evaluation supports their use. Furthermore, all guidelines strongly recommend treating comorbid conditions—such as depression, anxiety, and sleep disturbances—with appropriate medications, including antidepressants and anxiolytics.

Current discussions on UHR-P pharmacotherapy revolve around three broad biological axes:

1. Dopaminergic Modulation: Agents with a high affinity for D3 receptors, such as cariprazine, may offer advantages for negative symptoms and cognitive functioning—crucial given the prevalence of social withdrawal in UHR-P.

2. Non-dopaminergic Systems: Cannabidiol (CBD), acting via the endocannabinoid system, may exert antipsychotic-like effects without direct D2 receptor blockade, potentially enhancing clinical acceptability in younger populations due to its favorable tolerability.

3. Oxidative Stress and Neuroinflammation: Compounds like N-acetylcysteine (NAC) may provide a neuroprotective framework by increasing glutathione levels and modulating glutamatergic balance.

Overall, contemporary approaches emphasize preserving functioning by modulating underlying biological mechanisms rather than preventing psychosis per se. UHR-P is a heterogeneous syndrome rather than a uniform pre-illness state; therefore, interventions should be tailored to individual symptom profiles within a risk-benefit-based framework.

Keywords: Antipsychotics, cannabidiol (CBD), cariprazine, N-acetylcysteine (NAC), ultra-high risk for psychosis (UHR-P), UHR-P pharmacotherapy

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0551 - Family Accommodation as a Transdiagnostic Mechanism in Child and Adolescent Psychopathology Interrupting the Trauma Loop: Familial Accommodation in PTSD

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When a child cries out in fear at night, what does a good parent do? They go. They comfort. They protect. But what happens when protection becomes the problem? This question lies at the heart of family accommodation research—a growing body of literature examining how well-intentioned caregiver responses to a child's distress may inadvertently maintain and exacerbate psychopathology.

Family accommodation refers to the adjustments caregivers make in their daily routines and interactions to help a child avoid distress associated with psychological symptoms. These behaviors—facilitating avoidance, providing excessive reassurance, modifying household routines, participating in ritualistic behaviors, or removing potential triggers—are almost always well-intentioned. And they almost always make things worse. By negatively reinforcing avoidance and ritualistic behaviors, and by insulating the child from the natural consequences of their symptoms, accommodation creates a self-perpetuating cycle: short-term relief at the cost of long-term recovery, increasing symptom severity, and growing dependence on the very behaviors that maintain the disorder. Although early empirical work on family accommodation focused almost exclusively on obsessive–compulsive disorder (OCD), research has since expanded rapidly across diagnostic boundaries. A synthesized review across multiple diagnostic categories — including anxiety disorders, eating disorders, autism spectrum disorder (ASD), tic disorders, and post-traumatic stress disorder (PTSD) — concluded that accommodation is not only highly prevalent across these conditions, but consistently associated with more severe symptoms, greater functional impairment, poorer treatment outcomes, and increased caregiver burden.

In PTSD, family accommodation manifests across all four DSM-5 symptom clusters. A caregiver may redirect family conversations away from trauma-related content, modify sleeping arrangements to help the child avoid nightmares, avoid crowded or triggering environments, or—perhaps most distinctively—suppress their own emotional needs out of fear of provoking an angry outburst. Initial work systematically mapping these behaviors onto PTSD's unique diagnostic profile identified clinically distinct drivers—among them, avoidance organized around a specific traumatic event, emotional numbing, and dysregulated anger. Family members do not simply accommodate fear—they accommodate rage, silence, and secrecy.

The empirical picture is developing rapidly. A scoping review found that accommodation was consistently associated with elevated PTSD severity, poorer relationship outcomes, and worse response to individual trauma-focused therapies — findings largely derived from adult samples. Extending this literature to younger populations, evidence from a pediatric sample demonstrated that accommodation partially mediated the association between PTSD symptom severity and functional impairment in trauma-exposed children, underscoring that the family system is not merely a backdrop to recovery — it is an active mechanism within it. Nonetheless, the evidence base remains relatively nascent, and longitudinal studies are needed to clarify the causal mechanisms through which accommodation shapes PTSD trajectories in children and adolescents.

Taken together, the available evidence points to a variable that is present across virtually every disorder we treat, that meaningfully shapes symptom trajectories and treatment outcomes, and that we consistently fail to act upon. Family accommodation is perhaps the most clinically significant factor we overlook—commonly present in our formulations, yet rarely translated into systematic assessment or targeted intervention in everyday clinical practice.

Keywords: Child and adolescent psychopathology, family accommodation, post-traumatic stress disorder, transdiagnostic mechanisms

AuthorToEditor: 0000-0002-6616-1283 Symposia Oral Session-4 15.04.2026 / 16:30-18:00 Family Accommodation As A Transdiagnostic Mechanism in Child And Adolescent Psychopathology

0552 - Psychiatric Conditions in Children Drawn to Crime and Victims of Crime

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Kirikkale Yuksek Ihtisas Hastanesi, Çocuk ve Ergen Psikiyatrisi, Kırıkkale

Children drawn to crime (CDC) is a highly vulnerable population with elevated rates of psychiatric disorders, neurodevelopmental difficulties, and adverse childhood experiences that frequently intersect. The prevalence of psychiatric disorders in CDC is higher than in the general adolescent population. Previous studies suggest that approximately 50–70% of these youths meet criteria for at least one psychiatric disorder, with some variability depending on sample characteristics and assessment methods.

Externalizing disorders are the most frequent diagnostic group. Attention-deficit and hyperactivity disorder and conduct disorder are the most frequently diagnosed, and they commonly co-occur. These diagnoses are typically associated with impulsivity, difficulties in behavior regulation and aggression. Over time, this may be accompanied by academic failure, school withdrawal, and a persistent pattern of antisocial behavior such as harming others, damaging property, and engaging in behaviors that violate societal norms.

Neurodevelopmental disorders, particularly intellectual disability, are not uncommon in this population. Insufficient cognitive capacity may affect the ability to understand the consequences of actions and judgement. As a result, these children may be more likely to become involved in illicit activities.

Depression, anxiety, and trauma-related disorders are internalization disorders that are reported less consistently in clinical samples of children drawn to crime; however, they are more common than in the general population. The symptoms of these internalization disorders are often less visible, especially when externalizing behaviors are predominant in the clinical presentation. Still, emotional distress in children with a history of neglect or abuse can lead to internalization disorders and impact both victimization and delinquency. A key consideration is that externalizing disorders are directly linked to delinquent behavior, internalizing disorders have more complex, bidirectional relationships, explained by models such as acting-out, failure, reactive anxiety, and buffering frameworks.

While a clinical evaluation is crucial to determine the conditions such as attention-deficit hyperactivity disorder, intellectual disability, mood disorders, post-traumatic stress disorder, substance use, psychotic disorders and autism spectrum disorder psychiatric diagnoses alone seldom provide a comprehensive understanding of these children's situations. Delinquent behavior often results from a complex interaction of personal vulnerabilities and environmental risk factors, including family instability and breakdown, insufficient social support, disrupted education, exposure to violence, parental involvement in criminal activities, substance use, and school dropout, all of which are strongly linked to both initial delinquent acts and repeat offences. In many cases, understanding these contextual influences provides more meaningful insights into future risks than diagnosis alone.
In summary, children drawn to crime and those exposed to victimisation display complex and interconnected difficulties. Recognising their needs involves moving beyond diagnostic labels to consider developmental pathways, family environment, and social pressures. Early detection and various multidisciplinary, coordinated interventions—especially those adopting a trauma-informed approach—are crucial for fostering better long-term outcomes and decreasing the likelihood of ongoing delinquency.

Keywords: Attention-deficit and hyperactivity disorder, children drawn to crime, conduct disorder, externalizing disorders, juvenile delinquency

AuthorToEditor: I am honoured to submit this panel summary and would like to express my appreciation for the Scientific Committee’s dedication to advancing the field through this distinguished meeting. Sincerely, Seda KAFALI ORCID: 0000-0002-4696-9664

0553 - History and Neurobiological Mechanisms of ECT

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Electroconvulsive therapy (ECT) stands as one of the most effective yet paradoxical treatments in psychiatry. Its origins trace back to early convulsive therapies, reflecting a broader historical attempt to modulate brain function through induced physiological "shock." The conceptual roots of ECT can be found in the 16th century, when seizures were first hypothesized to alleviate psychiatric symptoms. This idea gained scientific grounding in the early 20th century with Ladislas von Meduna's chemically induced seizures, based on the proposed antagonism between epilepsy and schizophrenia (1).

The transition to electrically induced seizures in 1938 by Ugo Cerletti and Lucio Bini marked a pivotal innovation, offering a more controllable and reproducible method (2). ECT rapidly disseminated worldwide and became a cornerstone of biological psychiatry during the 1940s and 1950s. However, its historical trajectory is complex and ethically fraught. Notably, in the past, ECT technology was misused in isolated instances as a means of harm, highlighting the vulnerability of medical innovations to sociopolitical abuse (3). Following the advent of antipsychotic and antidepressant medications in the mid-20th century, ECT usage declined, further compounded by negative portrayals in media and public discourse. Nevertheless, in recent decades, ECT has undergone a resurgence as a highly effective, evidence-based intervention for severe and treatment-resistant psychiatric disorders, particularly major depression.

Despite its long clinical history, the precise neurobiological mechanisms underlying ECT remain incompletely understood. Contemporary models emphasize a multifactorial process involving neurophysiological, neurochemical, and neuroplastic changes. One prominent framework is the anticonvulsant hypothesis, suggesting that repeated ECT sessions enhance inhibitory neurotransmission while reducing excitatory activity, leading to a functional "reset" of dysregulated neural circuits. This is supported by observed increases in seizure threshold and postictal EEG suppression over the course of treatment.

Neuroimaging studies further demonstrate dynamic alterations in cerebral blood flow and metabolism, characterized by an initial hypermetabolic state during the seizure followed by postictal functional suppression. These changes are thought to contribute to symptom improvement by modulating overactive limbic and prefrontal networks. Additionally, ECT exerts profound neurotrophic effects. Preclinical and clinical evidence indicates increased hippocampal neurogenesis and upregulation of brain-derived neurotrophic factor (BDNF), potentially reversing stress-related neural atrophy associated with mood disorders.

Endocrine responses also play a role, with acute elevations in hormones such as cortisol and prolactin following ECT, alongside longer-term normalization of hypothalamic–pituitary–adrenal (HPA) axis dysregulation. Collectively, these findings suggest that ECT operates not through a single mechanism, but via coordinated modulation of neural networks, synaptic plasticity, and systemic physiological processes.

In summary, ECT exemplifies the evolution of psychiatric therapeutics from empirically driven origins to a modern, neurobiologically informed intervention. Understanding both its historical context and its complex mechanisms is essential for optimizing its use in the era of personalized and AI-assisted psychiatry.

Keywords: Electroconvulsive therapy (ECT), history, neurobiology

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0554 - Temporal Disintegration in Psychotic Disorders

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Time is not merely a linear progression of objective moments; rather, it is the fundamental structure of human consciousness. As William James famously described, the present is not a knife-edge but a "saddleback" looking in two directions simultaneously. Phenomenologically, Husserl's concept of the "extended now" illustrates that for a melody to be perceived as music, rather than a series of disconnected beeps, the mind must employ retention (holding the immediate past) and protansion (anticipating the immediate future). In schizophrenia, this tripartite temporal architecture collapses, leading to temporal disintegration. This breakdown prevents the synthesis of a cohesive specious present, causing the fluid stream of consciousness to fragment into isolated, meaningless snapshots. This review explores how these phenomenological disruptions are anchored in specific neurobiological and psychophysical timing deficits.

At the neurobiological level, the Prefrontal Cortex (PFC) acts as a bridge for cross-temporal contingencies, integrating working memory with preparatory sets to organize behavior over time. Francisco Varela's neurodynamic model posits that the now emerges from the rapid, large-scale synchronization of neuronal assemblies, akin to an orchestra achieving a perfect harmony. In psychosis, this synchronization fails, resulting in "chronotaraxis," where micro-disconnections between the PFC, thalamus, and cerebellum disrupt the timing of information transfer. Psychophysical evidence highlights a widened Temporal Binding Window (TBW) in patients, which impairs the ability to merge multisensory inputs into a unitary experience. This delayed broadcast of reality means the brain cannot keep pace with physical time, leading to a loss of autonoetic awareness. When the gap between an internal intention and its perceived sensory consequence widens, the patient may experience passivity phenomena, perceiving their own thoughts as external inserted voices or their movements as being controlled by outside forces.

Research using the Internal Clock Model indicates that while accuracy (absolute time estimation) may be preserved, precision (consistency) is severely compromised in schizophrenia. While patients may maintain relative accuracy in mean temporal estimations, they exhibit significantly increased intra-individual variability, indicating a profound deficit in temporal precision rather than a systematic bias in central tendency. This cognitive dysmetria reflects a failure in the cortical-thalamic-cerebellar-thalamic-cortical (CCTCC) circuit to coordinate the precise timing of cognitive and sensory outputs. Electrophysiological markers, such as reduced Mismatch Negativity (MMN) amplitude, further confirm that the psychotic brain cannot pre-attentively respond to rhythmic deviations. Notably, these timing deficits often precede overt psychosis, suggesting they serve as a trait marker or endophenotype that remains relatively independent of dopamine-targeting medications.

Temporal disintegration represents a fundamental shift from a symphony of neural integration to a cacophony of fragmented moments. The fracture of the "extended now" disrupts the very foundation of the self, transforming a continuous existence into a series of alien, disconnected events. Understanding these deficits through the lens of timing of perception offers a path toward new digital therapeutic interventions. Future rehabilitation protocols should prioritize sensory-level temporal training, such as auditory recalibration, to help patients bridge the temporal gaps in their experience, ultimately aiming to reintegrate their fractured reality and restore a stable sense of agency.

Keywords: Schizophrenia, Time Perception, Temporal Disintegration, Prefrontal Cortex, Cognitive Dysmetria, Phenomenonology

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0555 - Anxiety Disorders in Schizophrenia: Mechanisms of Resistance, Clinical Burden and Management

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Schizophrenia is a chronic and disabling psychiatric disorder that is frequently accompanied by comorbid anxiety disorders. Epidemiological studies indicate that approximately 40% of individuals with schizophrenia meet diagnostic criteria for at least one anxiety disorder, while nearly half report clinically significant levels of anxiety symptoms. Anxiety symptoms are frequently observed across all stages of schizophrenia. These include the prodromal phase, early psychosis, and chronic stages (1).

The frequent co-occurrence of schizophrenia and anxiety disorders may reflect shared genetic and environmental vulnerabilities. Heightened anxiety can exacerbate stress sensitivity in individuals predisposed to psychosis, thereby facilitating the emergence or exacerbation of psychotic symptoms. Conversely, core features of schizophrenia, such as perceptual disturbances and paranoid ideation, may give rise to secondary anxiety. Current evidence, though limited, suggests that anxiety may not merely be comorbid, but an integral component of the schizophrenia spectrum (2).

Anxiety, although highly prevalent in schizophrenia, remains underrecognized and insufficiently studied, and is associated with numerous adverse outcomes. It contributes to reduced quality of life, impaired social functioning, increased feelings of shame, and diminished self-esteem. Clinically, it is also linked to more frequent hospitalizations and higher rates of substance and alcohol abuse. Importantly, anxiety is associated with an elevated risk of suicide attempts and greater lethality of such behaviors. These outcomes underscore the importance of systematic assessment of anxiety symptoms in patients with schizophrenia, as early identification and management may mitigate these adverse effects (1).

Management of anxiety symptoms in schizophrenia requires careful consideration of the underlying etiology, including whether anxiety arises from undertreated positive symptoms or serves as an early warning sign of psychotic relapse. A thorough evaluation of symptom development, severity, and their contribution to the overall clinical presentation is essential for informed treatment planning (2). A variety of pharmacological and psychosocial interventions have shown promise in alleviating anxiety in schizophrenia. Antipsychotic medications may variably reduce or, in some cases, exacerbate anxiety, and there is evidence that adjunctive treatments such as anxiolytics, antidepressants, and pregabalin can have beneficial effects on anxiety symptoms. Structured psychosocial interventions, particularly cognitive-behavioral therapy for psychosis (CBTp), have been shown to effectively reduce anxiety-related distress, highlighting the importance of careful assessment and appropriate management of anxiety symptoms in individuals with schizophrenia (3).

Anxiety in schizophrenia represents a significant and multifaceted clinical challenge. Its high prevalence, complex bidirectional relationship with psychotic symptoms, and association with poor functional and clinical outcomes highlight the importance of thorough assessment and targeted management of anxiety symptoms. Evidence-based pharmacological and psychosocial interventions, particularly CBTp programs and judicious use of adjunctive medications, can mitigate anxiety-related distress and improve overall patient outcomes. Further research is warranted to refine these strategies, optimize treatment selection, and develop novel interventions tailored to the specific needs of patients with schizophrenia.

Keywords: Anxiety, comorbidity, psychosis, schizophrenia, treatment resistance

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0556 - The Origins of Attachment: The Biopsychosocial Roots of Separation Anxiety in the Perinatal Period

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The perinatal period, spanning pregnancy through the first year after birth, represents a critical window in which the foundations of the mother–infant relationship are established. During this time, complex biological, psychological, and social processes interact to shape early attachment. One of the key phenomena emerging within this developmental context is separation anxiety, which reflects the activation of the attachment system and plays an adaptive role in maintaining proximity between caregiver and infant. Understanding the biopsychosocial mechanisms underlying perinatal separation anxiety is essential for elucidating the early development of attachment and its implications for later mental health outcomes.

From a neurobiological perspective, attachment is supported by a network of brain regions involved in emotional processing, reward, and stress regulation. Functional neuroimaging studies have demonstrated that maternal responses to infant cues activate areas such as the amygdala, prefrontal cortex, hypothalamus, and nucleus accumbens. These neural circuits are closely linked with neurochemical systems that facilitate bonding and caregiving behavior. Among these, oxytocin plays a central role in promoting social bonding, trust, and maternal sensitivity, while dopaminergic reward pathways reinforce caregiving behaviors and positive parent–infant interactions. At the same time, the hypothalamic–pituitary–adrenal (HPA) axis contributes to the regulation of stress responses during early caregiving experiences, influencing both maternal emotional regulation and infant stress reactivity (Feldman, 2017; Swain et al., 2014).

Psychological factors also significantly influence the development of attachment and separation anxiety during the perinatal period. Maternal attachment representations, early life experiences, and preexisting mental health conditions may shape the way mothers perceive and respond to their infants' signals. For instance, insecure maternal attachment styles and unresolved childhood trauma have been associated with heightened maternal anxiety and altered caregiving patterns. These psychological processes may contribute to increased sensitivity to separation and heightened vigilance toward infant well-being.

In addition to biological and psychological influences, social and environmental factors play a crucial role in shaping early attachment processes. Social support, partner relationships, cultural expectations surrounding motherhood, and socioeconomic stressors can all influence maternal emotional adjustment during pregnancy and the postpartum period. Insufficient social support and perinatal mood disorders, particularly postpartum depression and anxiety, have been shown to negatively affect maternal responsiveness and infant bonding.

Taken together, attachment and separation anxiety during the perinatal period should be understood within a biopsychosocial framework in which neurobiological systems, psychological processes, and social environments interact dynamically. Early identification of heightened separation anxiety and disturbances in attachment processes may provide important opportunities for preventive interventions. Supporting maternal mental health and strengthening early caregiving relationships may ultimately promote secure attachment and healthier developmental trajectories for children.

Keywords: Attachment behavior, hypothalamic-pituitary-adrenal axis perinatal period, separation anxiety, oxytocin

0557 - From Neurobiological Foundations to Psychodynamic Patterns in Attention-Deficit/Hyperactivity

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Attention-Deficit and Hyperactivity Disorder (ADHD) is predominantly conceptualized within a neurobiological framework, emphasizing dopaminergic dysregulation, frontostriatal circuitry dysfunction, and executive impairments. While these models have significantly advanced our understanding of the disorder, they remain largely descriptive and insufficient in capturing the subjective and relational dimensions of ADHD. Notably, current diagnostic systems define ADHD based on behavioral criteria rather than etiological mechanisms, underscoring the need for complementary perspectives that address the individual's inner experience (1). This presentation aims to build a conceptual bridge between neurobiological foundations and psychodynamic patterns in ADHD, arguing that neurobiological vulnerabilities are translated into disturbances in intrapsychic organization and relational functioning. From this perspective, core symptoms such as inattention, impulsivity, and hyperactivity can be understood not merely as deficits in cognitive control, but as manifestations of impaired ego integration and disrupted self-regulatory capacities. Psychoanalytic formulations suggest that ADHD reflects a disturbance in the synthetic and organizing functions of the ego, affecting the individual's ability to modulate affect, sustain attention, and maintain coherent internal experience (2).

Psychodynamic theories further expand this understanding by highlighting the role of early relational experiences and object representations. Children with ADHD often demonstrate difficulties in forming stable internal representations of self and others, leading to impairments in affect regulation, mentalization, and interpersonal functioning. These relational disruptions may manifest as frustration, aggression, or withdrawal in social contexts, all of which reflect deeper disturbances in the development of the self (3) In this sense, ADHD can be understood as a disorder of internal organization as much as of attention.

Importantly, psychodynamic and neurobiological approcahes should be viewed as complementary explanatory systems. Historically, ADHD has been shaped by multiple "research narratives," with neurological and psychodynamic perspectives offering different but intersecting accounts of the same clinical phenomena (4). An integrative approach allows for a more comprehensive understanding, wherein neurobiological predispositions interact with developmental and relational contexts to produce the observable symptomatology. Despite the dominance of pharmacological and behavioral treatments, their long-term efficacy remains inconsistent, suggesting that symptom reduction alone may not address the underlying disturbances in self-regulation and internal organization (5). Psychodynamic approaches, particularly those integrating ego psychology and object relations, offer valuable insights into these deeper processes and highlight the importance of empathy, relational attunement, and the development of self-regulatory capacities in treatment.

In conclusion, ADHD is not solely as a neurodevelopmental disorder but a complex condition at the intersection of brain, mind, and relational experience. Bridging neurobiological and psychodynamic perspectives enables a more nuanced understanding of ADHD.

Keywords: Attention-Deficit and Hyperactivity Disorder (ADHD);, Psychodynamic psychiatry;, Neurobiology

AuthorToEditor: SYMPOSIA ORAL SESSION-48 17.04.2026 / 17:30-19:00 The Overlooked Psychodynamic Dimension Of Attention-Deficit/Hyperactivity Disorder adlı panelde yer alan konuşma için hazırlanmış özet metindir. Özette yer alan referanslar: References: 1. Salomonsson B. Interpreting the inner world of ADHD children: psychoanalytic perspectives. International Journal of Qualitative Studies on Health and Well-being [Internet]. 2017 Jun 7;12(sup1):1298269. Available from: https://doi.org/10.1080/17482631.2017.1298269 2. Gilmore K. A Psychoanalytic Perspective On Attention-Deficit/Hyperactivity Disorder. Journal of the American Psychoanalytic Association [Internet]. 2000 Aug 1;48(4):1259–93. Available from: https://doi.org/10.1177/00030651000480040901 3. Conway F. Current Research and Future Directions in Psychodynamic Treatment of ADHD: Is Empathy the Missing Link? Journal of Infant, Child, and Adolescent

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0558 - Inflammatory and Neurotrophic Biomarkers in Depression: CRP, IL-6, BDNF, and Beyond

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Major depressive disorder (MDD) involves complex biological mechanisms that go far beyond the classical monoamine hypothesis. In recent years, immune dysregulation and impaired neuroplasticity have emerged as central paradigms in understanding the pathophysiology of the disease. Elevated inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) define clinically significant patient subgroups characterized by more severe symptomatology, cognitive impairment, and poor treatment response. The role of IL-6 is not limited to being merely an indicator of disease presence. A significant association has been found between high IL-6 and CRP levels and a better response to ECT (electroconvulsive therapy); high pre-treatment IL-6 levels have been associated with a greater reduction in depressive symptoms. On the neurotrophic level, BDNF (brain-derived neurotrophic factor) stands out as an indispensable molecule for neuronal survival and synaptic plasticity. It is thought that BDNF levels are decreased in patients with depression, leading to structural and functional changes, particularly in the hippocampus and prefrontal cortex. BDNF levels show significant differences between patients with depression and MDD with and without suicidal attempts; increased BDNF levels reduce both depression and suicide risk. However, the clinical usefulness of peripheral BDNF measurement is debatable: although it is accepted that BDNF in the central nervous system plays a role in antidepressant efficacy and MDD pathophysiology, the reflection of this process in serum BDNF levels has limited diagnostic or prognostic value. The BDNF Val66Met polymorphism continues to attract the attention of clinicians. Recent meta-analyses highlight inconsistencies in the findings. While no significant association has been found between the polymorphism and antidepressant treatment response in the general population, when contradictory studies are excluded, the Met allele appears to predict a positive treatment response, particularly in patients of East Asian origin using SSRIs. The importance of multi-biomarker approaches is also increasing. A comprehensive analysis encompassing thirty observational studies, two randomized controlled trials, and twelve reviews consistently confirmed the association between high inflammatory marker levels and depressive symptoms in various MDD subtypes, including melancholia, atypical depression, and anxious depression, and in different remission states. The importance of the inflammation-neuroplasticity axis becomes even more apparent when it comes to rapidly acting antidepressants. Ketamine/esketamine stands out as the first agent to demonstrate potent and rapid effects that go beyond monoaminergic modulation through NMDA receptor antagonism, glutamate enhancement, and the re-regulation of BDNF/TrkB signaling. Consequently, biomarkers such as CRP, IL-6, and BDNF offer valuable tools in elucidating the biological heterogeneity of MDD. While the prognostic and predictive potential of these markers is becoming increasingly clear, broader, multicenter, and longitudinal studies are needed to identify immunometabolic subtypes and to incorporate biomarker-guided treatment selection into routine practice.

Keywords: Brain-derived neurotrophic factor, C-reactive protein, Depression, Inflammation, Interleukin-6

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0559 - Catatonia and Autism Spectrum: Risk Factors and Early Intervention

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Catatonia is an increasingly recognized yet underdiagnosed neuropsychiatric syndrome in individuals with autism spectrum disorder (ASD), characterized by disturbances in motor behavior, affect, and cognition. Although traditionally linked to schizophrenia, current evidence highlights its transdiagnostic nature and relevance in neurodevelopmental conditions. In ASD, catatonia typically emerges during adolescence or early adulthood and is often preceded by functional decline, including deterioration in communication, social engagement, and adaptive functioning.

Epidemiological studies indicate that catatonic features in ASD are more common than previously assumed, with prevalence estimates ranging from 6% to 20% depending on methodology [1]. More recent data suggest rates around 10.7% in adolescent and young adult samples [2]. Catatonia in ASD rarely occurs in isolation and is better conceptualized as a multifactorial syndrome arising from the interaction between neurobiological vulnerability and environmental stressors.

Key risk factors include psychosocial stressors such as loss, life transitions, and increased environmental demands. Emotional dysregulation, anxiety, and depressive symptoms are also strongly associated. Neurobiological mechanisms involving GABAergic, dopaminergic, and glutamatergic dysregulation have been implicated, alongside possible genetic contributions [3]. Late regression—defined as the loss of previously acquired social and communicative skills during adolescence—represents a critical clinical feature, with growing evidence linking it closely to catatonia [2].

Differentiating catatonia from core ASD features remains challenging due to overlapping symptoms such as mutism, stereotypies, negativism, and motor slowing. A key distinction is a marked change from baseline functioning rather than symptom presence alone. Clinicians should be alert to new-onset motor abnormalities, increased passivity, and decline in daily functioning.

Early recognition is crucial, as untreated catatonia is associated with significant morbidity, including functional deterioration, reduced oral intake, medical complications, and mortality. Delayed diagnosis may result in inappropriate treatments, particularly excessive antipsychotic use. In contrast, benzodiazepines and electroconvulsive therapy have demonstrated strong efficacy, often producing rapid improvement [3]. Lorazepam response is frequently both diagnostic and therapeutic.

Routine screening for catatonic features should be integrated into ASD assessments, particularly in the presence of regression or unexplained motor changes. Multidisciplinary management is essential, and future research should prioritize longitudinal studies, biomarkers, and ASD-specific diagnostic frameworks.

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Keywords: Autism, catatonia, regression, risk factors

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0560 - Sedation and Seizures; What Should We Do?

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Clozapine remains the most effective pharmacological treatment for treatment-resistant schizophrenia; however, its use is frequently limited by adverse effects that compromise adherence. Among these, sedation and seizure risk are particularly critical due to their impact on early discontinuation and patient safety. This presentation focuses on these adverse effects and their clinical management.

Sedation is the most common adverse effect of clozapine and a leading cause of treatment discontinuation. Prevalence estimates vary widely between 16% and 67%, reflecting heterogeneity in definitions such as somnolence, fatigue, and drowsiness [1]. Sedation is primarily mediated through histamine H1 and muscarinic receptor antagonism and typically emerges early during dose titration. Although partial tolerance may develop, a significant proportion of patients experience persistent sedation with negative effects on quality of life and functioning. Clinically, sedation may also contribute to complications such as aspiration pneumonia and venous thromboembolism. Management of clozapine-induced sedation requires a structured and individualized approach. Initial strategies include reducing or discontinuing concomitant sedative medications, slowing dose titration, and administering the majority or entirety of the dose at night. Therapeutic drug monitoring is increasingly recommended to identify the minimum effective dose. In stable patients, cautious dose reduction may be considered. Adjunctive pharmacological strategies, particularly aripiprazole augmentation, may reduce sedation, although evidence remains limited [1, 2]

Seizures are a recognized but often overestimated adverse effect of clozapine. A 1-year cumulative risk of approximately 5% has been reported, although incidence varies widely depending on study duration and methodology [3]. Generalized tonic–clonic seizures are the most common presentation, but myoclonic, atonic, partial, and absence seizures may also occur. Importantly, seizure risk is multifactorial and cannot be attributed to a single parameter such as dose or serum concentration. Although higher doses (e.g., >600 mg/day) and plasma levels (>1000 ng/mL) are associated with increased risk, seizures may occur at lower doses and therapeutic levels. Clinical management focuses on identifying precipitating factors, including rapid dose changes, pharmacokinetic interactions (e.g., CYP1A2 inhibitors), and smoking cessation. Following a seizure, clozapine should not be automatically discontinued; instead, dose reduction, slower retitration, and evaluation of contributing factors are recommended. When anticonvulsant treatment is required, valproate remains the most supported option, although alternatives such as lamotrigine, gabapentin, and topiramate may be considered in selected cases [2, 3]. A key clinical principle is that both sedation and seizures are generally manageable and rarely constitute absolute contraindications to continued treatment. Given the lack of equally effective alternatives in treatment-resistant schizophrenia, premature discontinuation of clozapine may result in substantial clinical deterioration. Therefore, proactive monitoring, patient education, and individualized dose optimization are essential to ensure treatment continuity and maximize therapeutic benefit, particularly during early treatment phases when discontinuation risk is highest and clinical stability has not yet been achieved.

Keywords: Clozapine, sedation, seizures, treatment-resistant schizophrenia, adverse effects

AuthorToEditor: Beyza Erdogan Akturk ORCİD:0000-0002-5072-9241 İlgili panel oturumu bilgileri: Symposıa Oral Sessıon-42 17.04.2026 / 15:30-17:00 Side Effects and Management of Clozapine, The Revolutionary Drug in Psychiatry Moderatör: Dr. Çiçek Hocaoğlu Agranulocytosis; Is the Risk Really Great? Dr. Cansu Ünsal Mavi Hypersalivation and Constipation; GI Side Effects Dr. Esra Yalım Sedation and Seizures; What Should We Do? Dr. Beyza Erdoğan Aktürk

0561 - Trauma and Autism Spectrum: Risk Factors and Protective Interventions

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Autism spectrum disorder (ASD) is not limited to neurodevelopmental differences; it is a condition characterized by increased sensitivity to both biological and environmental stressors throughout an individual's life. Individuals with ASD are known to be exposed to adverse life events, such as childhood abuse, peer bullying, and social isolation, to a greater extent than their typically developing peers. This vulnerability is associated with both ASD-specific individual characteristics, such as difficulties in social communication, sensory sensitivity, heightened stress responses, and low tolerance for changes in routine, as well as exclusionary social attitudes and inadequate support systems.

The impact of trauma on individuals with ASD may manifest as behavioral changes, newly emerging problem behaviors, increased stereotypies, regression in social skills, and sleep and eating disorders, deviating from classic post-traumatic stress disorder symptoms, and may be misinterpreted as part of the ASD itself. Additionally, the limited expression of emotions in these individuals may lead to the overlooking of their trauma history.

Although recognizing and addressing trauma in individuals with ASD is challenging, the literature in this area appears to be limited. Current data highlight the necessity of adapting trauma-focused psychoeducation and psychotherapy approaches to an individual's needs; they also underscore the importance of protective interventions, such as early detection and screening, parent education programs supporting secure attachment, collaboration with education and health systems, and social skills training.

In this presentation, the risk factors leading to vulnerability to traumatic experiences in individuals with ASD as well as protective interventions that support mental health will be addressed within both clinical and societal contexts. Findings in this area not only help reduce psychopathology but also shed light on strategies aimed at improving the quality of life and social participation of individuals with ASD.

Keywords: Autism Spectrum Disorder, Neurodevelopmental Disorders, Trauma and Stressor Related Disorders

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0563 - Inflammatory Mechanisms in Cognitive Disengagement Syndrome

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Cognitive Disengagement Syndrome (CDS), formerly referred to as Sluggish Cognitive Tempo (SCT), is a neuropsychological phenotype characterized by excessive daydreaming, mental fogginess, low energy levels, and slowed information processing. Although it is frequently confused with Attention-Deficit/Hyperactivity Disorder (ADHD), CDS is considered to have a distinct symptom profile and unique etiological underpinnings. Recent research suggests that the symptomatology of CDS is not only psychological but also closely associated with biological processes such as systemic inflammation and oxidative stress.[1]

The effects of inflammation on the central nervous system are mediated through cytokines and other immune mediators. Pro-inflammatory cytokines (particularly interleukin-6 and C-reactive protein (CRP) are hypothesized to influence arousal and attentional networks in the brain, thereby contributing to the "brain fog" and hypoactive behavioral patterns observed in CDS.[2] Peripheral inflammation may cross the blood–brain barrier, leading to microglial activation, disruption of neurotransmitter balance, and consequent slowing of cognitive processing. A study conducted by Kılıçoğlu et al. (2023) demonstrated that inflammatory markers in children and adolescents diagnosed with CDS were significantly elevated compared to healthy control groups. In this study, most inflammatory markers—except for tumor necrosis factor-alpha (TNF-α)—were found to be increased in the CDS group, and this elevation paralleled markers of oxidative stress.[1] Furthermore, the higher prevalence of CDS symptoms in individuals with chronic inflammatory conditions (e.g., inflammatory bowel disease or asthma) supports the presence of an "immune–cognitive" interaction.[3]

This relationship between CDS and inflammation opens new avenues for therapeutic approaches. In addition to standard stimulant medications, anti-inflammatory strategies and lifestyle interventions (such as diet and sleep regulation) may be effective in improving cognitive processing speed. Future studies should focus on longitudinal designs to clarify the causal effects of inflammation on CDS.

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Keywords: Cognitive Disengagement Syndrome, Sluggish Cognitive Tempo, inflammation

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0565 - The First Traces of Attachment: Developmental Continuity in Childhood Attachment and Separation Anxiety

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Attachment relationships begin as regulatory systems of proximity, protection, and emotional co-regulation, but they soon become developmental templates that shape how children interpret absence, distress, and reunion. This panel proposes that separation anxiety is best understood not as an isolated symptom cluster, but as one of the earliest clinically visible expressions of the child's attachment system under strain. Drawing on developmental psychopathology, attachment theory, and contemporary child mental health research, the panel will examine how early caregiver responsiveness, attachment security, and family emotion-regulation patterns contribute to both adaptive separation distress and maladaptive trajectories that may culminate in separation anxiety disorder (1).

The central argument of the panel is that developmental continuity matters. Attachment is neither fully fixed nor infinitely malleable; rather, it shows patterned continuity across development while remaining sensitive to caregiving change. This makes childhood separation anxiety a particularly informative clinical phenomenon: it emerges at the intersection of normative dependency, neurodevelopmental maturation, environmental stress, and internal working models of safety and availability. In this framework, secure attachment supports the gradual toleration of distance, school entry, exploration, and autonomous emotion regulation, whereas insecure attachment may be associated with heightened separation-related threat appraisals and greater reliance on external soothing. The panel will further argue that separation anxiety should be considered along both homotypic and heterotypic developmental pathways—persisting as separation-focused distress in some children, while in others evolving into broader internalizing patterns such as generalized anxiety, social anxiety, panic-related vulnerability, and depressive symptoms across adolescence and early adulthood (2).

A second focus of the panel is the family context in which separation anxiety is maintained or transformed. We will discuss how parental accommodation, overprotection, inconsistent availability, and difficulty scaffolding autonomy can unintentionally reinforce avoidance and amplify attachment-related fears. Rather than framing caregivers as simple risk factors, the panel will emphasize dyadic and transactional processes: the anxious child shapes the caregiving environment, and the caregiving environment, in turn, shapes the child's expectations of safety, reassurance, and separation. This perspective has direct clinical implications for assessment and intervention. Developmentally informed evaluation should distinguish normative separation protest from clinically impairing anxiety, attend to attachment representations and caregiving patterns, and consider family-based interventions that strengthen secure-base functioning while reducing accommodation and promoting graduated autonomy (3). Overall, the panel aims to reposition childhood separation anxiety as a key developmental signal—one that links the earliest traces of attachment to later emotional continuity, clinical risk, and opportunities for timely preventive intervention.

Keywords: Attachment, separation anxiety, developmental continuity, caregiving sensitivity, family accommodation, child psychopathology

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0566 - Neuroinflammatory Pathways in Attention-Deficit/Hyperactivity Disorder

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Attention-Deficit/Hyperactivity Disorder (ADHD) is characterized by pervasive and persistent behavioral symptoms of inattention, hyperactivity, and/or extreme for age that interfere with social or academic functioning. Diagnosis requires the emergence of symptoms before the age of twelve and that symptoms present in two or more settings. ADHD is common, affecting about 5% of children worldwide, with boys being overrepresented, on average, approximately 2:1. ADHD is a complex neurodevelopmental condition arising from the interplay between genetic and environmental factors. Traditionally explained by imbalances in dopaminergic and noradrenergic systems, recent literature within the last five years has brought "neuroinflammation" and "immune system activation" to the forefront of ADHD etiology.[1]

Recent preclinical and clinical research indicates that microglial cells, the resident immune cells of the central nervous system, are directly linked to ADHD-like behaviors. Chronic activation of microglia leads to an excessive release of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α. This release disrupts the process of synaptic pruning and weakens the connectivity between the prefrontal cortex and the basal ganglia. Increasing evidence supports the idea that high levels of pro-inflammatory cytokines may have neurotoxic effects on the neural circuits responsible for attention processing. This, in turn, may contribute to the development and persistence of ADHD-related behavioral symptoms.

Oxidative stress, another pillar of neuroinflammatory processes, causes a disruption in homeostatic balance in individuals with ADHD. Modern meta-analyses report that ADHD patients exhibit lower total antioxidant capacity and significantly higher markers of oxidative stress. This environment shifts tryptophan metabolism toward the "Kynurenine pathway."[2] The conversion of tryptophan into kynurenine instead of serotonin leads to the accumulation of quinolinic acid, a neurotoxic substance that increases excitotoxicity via the glutamatergic system. Evidence published since 2020 strongly suggests that the gut microbiota influences ADHD symptomatology through systemic inflammation. Increased intestinal permeability (leaky gut) allows circulating lipopolysaccharides to cross the blood-brain barrier, initiating low-grade neuroinflammation. This triggers the migration of peripheral immune cells, such as T-lymphocytes and monocytes, into the brain, correlating with the severity of hyperactivity and impulsivity in ADHD.

In conclusion, ADHD is increasingly recognized not merely as a neurotransmitter deficiency but as a chronic low-grade inflammatory state. These findings have opened discussions regarding the potential supplementary roles of anti-inflammatory agents, omega-3 supplementation, and antioxidant strategies alongside traditional stimulants.[3] Clarifying these neuroinflammatory pathways is critical for developing biomarkers and personalized treatment protocols in ADHD management.

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Keywords: ADHD, Cytokines, Microglia, Neuroinflammation, Oxidative Stress

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0567 - Psychopharmacological Approaches to Resistant Symptoms in ASD

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Autism spectrum disorder (ASD) is a complex and heterogeneous neurodevelopmental condition in which treatment resistance frequently emerges, particularly in clinically challenging symptom domains. Psychopharmacological interventions in ASD can be conceptualized within two broad frameworks: (1) treatments targeting core symptoms, and (2) treatments addressing associated or comorbid symptom clusters. This distinction is critical for both clinical decision-making and the rational management of treatment resistance.

To date, no pharmacological agent has received regulatory approval specifically for the core symptoms of ASD, including deficits in social communication and restricted, repetitive behaviors. However, a growing body of research has explored potential pharmacological targets, including oxytocinergic, glutamatergic, and serotonergic pathways. Although findings to date have been inconsistent and have not yet translated into routine clinical use, these studies have significantly contributed to a better understanding of the neurobiological mechanisms underlying ASD. As these pathways become more clearly elucidated, they may offer promising avenues for the development of more targeted and effective treatments in the future. Therefore, while pharmacological interventions are not currently considered first-line for core symptoms, ongoing research continues to hold potential for future therapeutic advances.

In contrast, pharmacotherapy plays a more established role in the management of associated symptoms, which often drive functional impairment and clinical urgency. These include irritability, aggression, self-injurious behavior, attention dysregulation, anxiety, and sleep disturbances. Among these, second-generation antipsychotics—particularly risperidone and aripiprazole—have the strongest evidence base and remain the only agents approved for irritability in ASD. Other agents, including stimulants, atomoxetine, alpha-2 agonists, and selective serotonin reuptake inhibitors, are frequently used in a symptom-targeted manner, although treatment response is variable and adverse effects are more common compared to typically developing populations.

Within the ÇÖZGEM model, the management of treatment-resistant symptoms is embedded in a structured, multidisciplinary framework. Target symptoms are first defined through comprehensive assessment by a coordinated team, including child psychiatrists, psychologists, child development specialists, occupational therapists, and speech and language therapists, alongside other relevant professionals. Initial interventions prioritize behavioral and psychosocial strategies. However, in cases where these approaches are insufficient, a team-based decision is made to initiate pharmacological treatment.

A key feature of this model is the integration of detailed psychoeducation prior to medication initiation, ensuring that families are actively involved in shared decision-making. Pharmacological treatment is then individualized based on symptom profile, developmental level, and comorbidities. Medications are introduced using a “start low, go slow” approach, with careful titration to the minimum effective dose. Adverse effects are systematically monitored, and treatment response is evaluated using objective and longitudinal clinical records.

This integrative and measurement-based approach reduces inappropriate polypharmacy, enhances treatment adherence, and supports more precise targeting of resistant symptoms. Ultimately, psychopharmacology in ASD should not be viewed as a standalone intervention but as one component of a broader, individualized, and multidisciplinary treatment strategy.

Future directions should focus on mechanism-based treatments and biomarker-guided interventions to improve outcomes in treatment-resistant ASD populations.

Keywords: Autism spectrum disorder, treatment resistance, psychopharmacology, multidisciplinary approach, ÇÖZGEM model

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0568 - Future-Oriented Anxiety, Social Media, and Activism in Adolescents: Clinical Features and Interventions

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Adolescence is a developmental period in which sensitivity to uncertainty, peer evaluation, and identity formation converges with growing awareness of large-scale societal threats. In this context, future-oriented anxiety may be understood as a clinically useful formulation for distress organized around anticipated instability, loss of control, and uncertainty rather than immediate danger alone [1]. Contemporary adolescents increasingly encounter concerns related to climate change, political instability, social fragmentation, academic pressure, and economic insecurity within a digital environment in which such threats are repeatedly amplified, personalized, and socially negotiated [1,2]. Social media should therefore not be conceptualized as a single or uniform exposure. Rather, different online activities may be associated with emotional outcomes through distinct psychosocial pathways, including social comparison, approval anxiety, perceived support, and repeated exposure to idealized or catastrophic content [2].

This panel proposes that adolescent activism occupies a clinically important position at the intersection of future-oriented anxiety and digital life, particularly when anxiety is organized around collective and future-focused threats such as climate change [3]. Emerging literature suggests that anxiety related to large-scale threats may, under some conditions, mobilize engagement, meaning-making, and action, while under other conditions it may also contribute to helplessness, interpersonal strain, emotional exhaustion, or hopelessness [3]. Accordingly, activism should be conceptualized neither as inherently protective nor as inherently pathological, but as a developmental response whose psychological meaning depends on context, perceived efficacy, emotional containment, and the adolescent's broader social ecology [1,3]. A similar interpretive caution is necessary for social media use, where some forms of online communication may support connection and expression, whereas others appear more strongly associated with anxiety through comparison-based self-evaluation and approval-seeking processes [2]. The panel therefore argues for an integrative framework linking intolerance of uncertainty, digitally mediated threat exposure, and action-oriented coping in order to better understand how contemporary adolescents experience and manage future-directed fears [1-3].

From a clinical perspective, assessment should move beyond generic questions about "screen time" or activism involvement and instead examine the content of future fears, perceived controllability, repetitive threat monitoring online, reassurance seeking, comparison-based distress, sleep disruption, family responses, and whether activism functions primarily as agency, belonging, avoidance, or emotional overinvestment [1-3]. Intervention planning should likewise be formulation-based and developmentally informed, combining evidence-based anxiety care with attention to the adolescent's digital ecology and civic context [1,2]. Particularly relevant components may include psychoeducation about uncertainty, cognitive-behavioral work targeting catastrophic forecasting and rumination, strategies to reduce maladaptive comparison and approval dependence online, emotion regulation skills, family guidance around media practices, and support for bounded, values-consistent forms of participation that preserve agency without intensifying burden [1-3]. By integrating developmental psychopathology, digital mental health, and youth engagement with collective threats, this panel aims to provide clinicians with a nuanced framework for distinguishing impairment from engagement and for supporting adolescents whose anxieties are increasingly organized around the future [1-3].

Keywords: Adolescents, future-oriented anxiety, social media, activism, uncertainty, clinical intervention

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0569 - Purpose in Life and Mental Health in Adolescence

Fatma Yıldırım

Antalya Şehir Hastanesi

Purpose in life refers to having a sense of direction, future-oriented goals, and motivation toward meaningful activities. In recent years, it has gained increasing attention in psychiatry as an important protective factor for mental health. Understanding purpose in life during adolescence is particularly critical, as this developmental period is characterized by identity formation, emotional vulnerability, and the construction of future expectations.

Adolescence represents a key stage in which individuals begin to explore values, establish long-term goals, and develop a sense of direction. Disruptions in this process may contribute to the development and persistence of psychopathology. A growing body of evidence indicates that higher levels of purpose in life are associated with lower levels of depression, anxiety, and suicidal ideation, as well as greater psychological well-being and life satisfaction.

In clinical practice, adolescents presenting with depressive symptoms frequently describe a loss of direction, lack of motivation, and a diminished sense of purpose. These experiences often manifest as behavioral withdrawal, academic decline, and disengagement from previously rewarding activities. Importantly, loss of purpose should not be conceptualized solely as a consequence of depression but also as a contributing and maintaining factor.

Purpose in life may also be indirectly affected in various clinical conditions. In attention-deficit/hyperactivity disorder, difficulties in executive functioning, planning, and delayed reward processing may interfere with the ability to establish and sustain long-term goals. Similarly, adolescents with chronic medical conditions or adjustment difficulties may experience disruptions in daily functioning and future expectations, which can hinder the development of a stable sense of purpose.

From a therapeutic perspective, purpose in life is a modifiable construct. Interventions such as behavioral activation, values-based approaches, acceptance and commitment therapy, and narrative techniques can support adolescents in identifying meaningful goals and rebuilding a sense of direction. Addressing purpose in life in clinical settings may not only reduce symptoms but also enhance long-term resilience.

In conclusion, purpose in life is a core dimension of adolescent mental health that deserves greater attention in both assessment and intervention. Beyond symptom reduction, supporting adolescents in developing a sense of direction may provide a more comprehensive and developmentally sensitive approach to psychiatric care.

Keywords: Adolescence, Depression, Meaning in life, Mental health, Psychological well-being, Purpose in life

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0570 - Ethics, Data Security and Clinical Integration – Boundaries of AI Use in Child and Adolescent Psychiatry

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Background:
Artificial intelligence (AI) is increasingly being used in child and adolescent psychiatry, offering significant opportunities for early diagnosis, monitoring, and personalized interventions. However, the use of these technologies in a sensitive population such as children and adolescents also brings important ethical, legal, and clinical challenges that must be carefully addressed.

Objective:
This presentation aims to examine the boundaries of AI use in child and adolescent psychiatry, particularly from the perspectives of ethics, data security, and clinical integration.

Methods:
Within this scope, a comprehensive literature review was conducted using predefined keywords related to artificial intelligence, child and adolescent psychiatry, and ethical, legal, and clinical integration concepts. Controlled terms and free-text keywords were combined using Boolean operators to ensure a broad and inclusive search. The search strategy was designed to include key concepts such as data privacy, regulatory frameworks (e.g., GDPR and HIPAA), algorithmic transparency, clinical governance, and informed consent. The search was performed across PubMed, Scopus, and Web of Science databases, with database-specific adaptations applied while preserving the core conceptual framework. All retrieved records were imported into Zotero, where duplicate entries were identified and removed. Given the heterogeneity of the included literature, a formal single risk-of-bias tool was not suitable for all studies; therefore, study quality was evaluated descriptively based on design, relevance, and methodological transparency. In addition, international guidelines from organizations such as WHO, UNICEF, and OECD were reviewed to contextualize ethical and governance frameworks, while noting the current lack of a comprehensive national guideline in Türkiye.

Results:
The findings indicate that while AI-based tools have the potential to improve access to care and support personalized interventions, they also raise significant concerns regarding data privacy, informed consent, algorithmic bias, and transparency. Ethical challenges are particularly pronounced in minors due to their developmental vulnerability and dependence on caregivers. Furthermore, issues related to clinical integration, including clinician oversight, training requirements, and workflow adaptation, remain insufficiently addressed in the current literature.

Conclusion:
AI applications in child and adolescent psychiatry must be implemented within clearly defined ethical and clinical boundaries. Strengthening data security, ensuring transparency, and developing context-specific governance frameworks are essential for safe and responsible integration into clinical practice.

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Keywords: Artificial intelligence, child and adolescent psychiatry, clinical integration, data security, ethics, privacy

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0571 - Substance Use Disorders in Schizophrenia: Resistance Pathways, Relapse Risks and Management

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Substance use disorders (SUDs) are among the most clinically consequential comorbidities in schizophrenia, substantially influencing illness onset, symptom burden, treatment response, relapse liability, and long-term functional outcome. In the context of an increasingly accessible and high-potency global drug market, this comorbidity has become even more clinically relevant. Rather than representing a coincidental co-occurrence, SUD in schizophrenia is best understood as a major determinant of poor prognosis, associated with impaired adherence, recurrent hospitalization, suicidality, and excess mortality. Large cohort and review data suggest that nearly half of patients with schizophrenia meet criteria for at least one SUD, with tobacco, alcohol, cannabis, and cocaine among the most commonly implicated substances (1, 2).

Schizophrenia and addiction appear to share, at least in part, a common neurobiological substrate centred on dysregulation of mesolimbic and mesocortical dopaminergic pathways. Psychoactive substances acutely enhance striatal dopamine transmission, whereas chronic exposure induces neuroadaptations that may increase psychosis vulnerability and sustain compulsive substance use (1). In parallel, hypodopaminergic states associated with chronic substance exposure may aggravate anhedonia and negative affect, thereby reinforcing repeated substance seeking. Within schizophrenia, impairments in reward processing, executive dysfunction, and hypofrontality may further promote self-medication and reduced control over use. At the behavioural level, recurrent intoxication, withdrawal, and non-adherence establish a cycle that increases relapse risk and may diminish antipsychotic responsiveness over time (1-3)

Clinical management should begin with systematic screening for substance use, careful distinction between primary psychosis and substance-induced phenomena, and an individualized relapse formulation incorporating current use patterns, withdrawal states, and psychosocial stressors. Treatment is most effective when delivered through an integrated model combining pharmacotherapy, motivational interviewing, cognitive-behavioural interventions, case management, and social rehabilitation. Compared with sequential or parallel models of care, integrated approaches appear more effective in sustaining engagement and reducing relapse (1, 2).

Antipsychotic selection is particularly important in this population. Available evidence suggests that the choice of antipsychotics may influence both the risk of developing comorbid SUD and subsequent relapse patterns. Clozapine appears to confer the strongest protection against relapse and may also reduce the risk or persistence of substance use (2, 3). Long-acting injectable antipsychotics may provide additional benefit, particularly in patients with poor adherence, by improving treatment continuity and reducing the likelihood of relapse. While second-generation antipsychotics remain the usual first-line option overall, partial dopamine agonists such as aripiprazole, cariprazine, and brexpiprazole also appear promising in selected patients, although long-term comparative data remain limited. Given the heightened burden of metabolic, infectious, and social morbidity in dual-diagnosis populations, ongoing physical health surveillance should be considered an integral component of care (1, 2). In sum, SUD in schizophrenia is a major driver of treatment resistance and relapse. Early detection, individualized antipsychotic treatment, and integrated multimodal care remain the most defensible clinical framework for improving outcomes in this highly vulnerable population (1-3).

Keywords: Schizophrenia, substance use disorders, treatment resistance, relapse, integrated treatment

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0572 - The Chemical Biology of Psychoactive Substances

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Despite major advances in basic brain science over the past decade, the discovery of truly new drugs for neuropsychiatric disorders has lagged, exposing a central paradox: why has our mechanistic understanding of the brain accelerated so rapidly while therapeutic innovation has largely stalled? This talk addresses that gap by positioning psychoactive compounds—especially classic psychedelics and addiction-relevant substances—not as "mere substances," but as precise chemical tools that can be engineered, calibrated, and deployed to interrogate brain function across scales. Building on the premise that many clinically relevant states are emergent properties of distributed circuits rather than single molecular targets, the program presented here uses multi-target (polypharmacological) perturbations to dissect how defined receptor-level events propagate through intracellular signaling to reshape synaptic plasticity, circuit computations, and brain-wide network connectivity. The speaker will describe a systematic chemical-biology framework for turning psychedelics into mechanistic probes that reveal how transient changes in neuromodulatory signaling can relax rigid network states, open windows of metaplasticity, and bias learning toward durable behavioral change—features that may help explain why limited dosing can sometimes yield long-lasting clinical benefits. A core theme is that psychedelics provide a unique bridge between consciousness science and disease pharmacology: by aligning subjective shifts in perception, self-processing, and affect with objective readouts of neural dynamics, the work aims to translate "experience-level" phenomena into tractable biomarkers of target engagement and therapeutic mechanism. Beyond neurons, the talk will emphasize that neuroimmune modulation is not peripheral, but often integral to brain-state transitions, influencing glial activity, inflammatory tone, synapse remodeling, and stress-resilience programs that intersect with depression, trauma-related disorders, and substance use disorders. By integrating neuroimmune biology with circuit mapping, the program seeks a more complete and causally grounded account of both therapeutic opportunity and safety liability. The presentation will further outline how this chemistry-enabled approach can support anti-addiction therapy development by identifying convergent nodes where maladaptive learning, stress signaling, and immune dysregulation meet, and by using psychoactive probes to map and normalize the circuits underlying craving, habit, and relapse vulnerability. Finally, the speaker will discuss translational strategies that connect discovery to clinical breakthroughs, including designing next-generation probes and candidate therapeutics with controllable kinetics and improved dissociation between beneficial plasticity and unwanted acute psychoactivity, and using mechanism-linked biomarkers to guide patient stratification, dosing, and combination interventions. Overall, the talk argues that neurochemical biology—deploying psychoactive molecules as rigorously defined perturbations—can convert rapid progress in brain science into an actionable pipeline for new treatments.

Keywords: Psychedelics, Neurochemical biology, Polypharmacology, Brain network plasticity

0573 - Intolerance of Uncertainty in Children with Hoarding Disorder

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Hoarding disorder (HD) is increasingly recognized as a distinct psychiatric condition characterized by persistent difficulty discarding possessions and excessive acquisition, resulting in significant clutter and functional impairment. While saving behaviors may be adaptive under certain conditions, such as resource scarcity, they may become maladaptive when they interfere with daily functioning. Individuals with HD often attribute emotional or instrumental value to their possessions and experience distress when faced with decisions about discarding them. Clinical observations suggest that decision-making difficulties are a central feature of the disorder, particularly in situations involving uncertainty about the potential future usefulness of items.

Cognitive-behavioral models propose that hoarding symptoms are maintained by information-processing deficits, dysfunctional beliefs, and avoidance-based coping strategies. Individuals may overestimate the future usefulness of objects and perceive discarding decisions as consequential. These beliefs are associated with increased distress and may contribute to delays or avoidance in decision-making. Avoidance behaviors, such as postponing or refraining from discarding possessions, may reduce distress in the short term while maintaining hoarding-related difficulties over time. Difficulties in attention, categorization, and memory processes may further complicate decision-making and contribute to the persistence of the disorder.

Within this framework, intolerance of uncertainty (IU) has been proposed as a relevant cognitive factor in hoarding disorder. IU refers to a tendency to perceive uncertain situations as aversive and to experience discomfort when faced with ambiguity or incomplete information. Individuals with higher levels of IU are more likely to interpret uncertain situations as threatening and may engage in strategies aimed at reducing uncertainty. In the context of hoarding, uncertainty regarding the future usefulness of possessions or the consequences of discarding them may be particularly salient.

Empirical findings indicate that IU is associated with hoarding symptoms. Studies in both non-clinical and clinical samples report that higher IU levels are related to greater hoarding severity, even after accounting for anxiety and depression. IU appears to be more closely related to difficulty discarding and excessive acquisition, whereas clutter may reflect the cumulative outcome of these processes. These findings suggest that IU may be linked to decision-making difficulties and avoidance tendencies observed in hoarding.

Given the overlap between hoarding disorder and anxiety-related conditions, IU has been conceptualized as a potential transdiagnostic factor. However, existing studies remain limited, and much of the available evidence is based on cross-sectional designs. Therefore, the extent to which IU contributes uniquely to hoarding symptoms requires further investigation, particularly in clinically diagnosed samples.

From a clinical perspective, examining IU may provide additional insight into the cognitive processes underlying hoarding behaviors. Interventions targeting IU, such as cognitive restructuring and gradual exposure to uncertainty, have shown benefit in anxiety-related conditions and may be relevant in hoarding disorder. However, further research is needed to clarify the role of IU in treatment response.

In conclusion, intolerance of uncertainty appears to be a relevant cognitive factor in hoarding disorder, particularly in relation to decision-making difficulties and avoidance processes. A clearer understanding of this relationship may contribute to refining theoretical models and informing future research and clinical practice.

Keywords: Avoidance, Hoarding Disorder, Intolerance of Uncertainty, Uncertainty

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0574 - Artificial Intelligence in Psychopharmacology: Ethical Issues, Challenges and Future Trends

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Artificial intelligence (AI) is increasingly integrated into psychopharmacology as a tool for prediction, optimization, and clinical decision support. While current discussions largely frame AI in instrumental terms, this perspective risks overlooking its broader impact on clinical practice. This presentation examines AI within a historical and conceptual trajectory, arguing that contemporary digital systems may not only assist clinical decision-making but also reshape the relational, epistemic, and ethical dimensions of care. Early analyses of computer use in clinical encounters suggested that the presence of a computer transforms the doctor–patient dyad into a triadic interaction involving doctor, patient, and machine. Within such configurations, clinical interaction can no longer be understood as purely dyadic, as each element mediates and reshapes the relationship between the others. More recent philosophical approaches have further emphasized that digital technologies can actively mediate perception, knowledge, and responsibility within clinical settings. Building on these perspectives, this presentation argues that the integration of AI represents an intensification of this triadic structure. AI does not merely function as an auxiliary tool but operates as an active mediating element that participates in structuring what counts as clinically relevant information and how decisions are justified. In doing so, it tends to privilege statistical and algorithmic forms of reasoning over interpretive judgment.

This shift has important ethical implications. While AI may enhance clinical accuracy and support beneficent decision-making, it also reframes what counts as “benefit,” often privileging population-level optimization over individual, context-sensitive outcomes. The principle of non-maleficence becomes more complex, as harms may emerge not only from incorrect decisions but from systemic biases, over-reliance on algorithmic outputs, and opacity in decision-making processes. Autonomy may be constrained as clinical decision-making becomes increasingly structured by data-driven systems, potentially narrowing the space for meaningful deliberation. At the same time, issues of justice arise as AI systems risk reproducing and amplifying existing inequalities embedded in training data. Taken together, these developments suggest that AI does not invalidate classical bioethical principles but alters the conditions under which they are interpreted and applied in clinical practice.

In addition, emerging literature points to the risk of dehumanization, as increasing reliance on AI may weaken empathic engagement and shift clinical encounters toward more transactional interactions. Among the key issues addressed in this presentation is the transformation of trust. AI appears to redistribute trust from interpersonal relations toward system-based reliability, creating a “trust gap” between confidence in algorithmic outputs and trust in clinical judgment. Furthermore, the opacity of many AI systems raises questions of accountability. When decisions are informed by non-transparent models, responsibility for adverse outcomes becomes difficult to attribute, challenging traditional notions of professional responsibility and informed consent.

These issues are particularly salient in child and adolescent psychiatry, where clinical care depends heavily on trust, developmental understanding, and contextual interpretation. This presentation concludes that ethical evaluation of AI in psychopharmacology should extend beyond accuracy and efficiency. Future developments should aim to integrate AI in ways that support, rather than undermine, the interpretive and relational foundations of psychiatric practice.

Keywords: Artificial Intelligence, Bioethics, Psychopharmacology

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0575 - Reopening Critical Periods: Psychedelics, Sigma-1 Receptor Signaling, and Late-Life Neuroplasticity

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This talk explores how psychedelics can reopen critical periods of plasticity in the adult and aging brain. Critical periods are developmental windows during which neural circuits exhibit heightened malleability, enabling efficient learning and circuit refinement; their closure in adulthood contributes to reduced adaptability and increased vulnerability to neuropsychiatric and neurodegenerative disorders. Psychedelics such as psilocybin, lysergic acid diethylamide, and related psychoplastogens have emerged as powerful tools to transiently restore juvenile-like plasticity states, yet the molecular logic underlying this phenomenon remains incompletely understood. Focusing on the concerted roles of the 5-HT2A receptor and the sigma-1 receptor as central signaling hubs, this lecture will discuss how psychedelics engage multi-target pharmacology to modulate cortical excitability, intracellular calcium dynamics, mitochondrial function, lipid microdomains, and neuroimmune interactions, thereby reshaping synaptic and network-level plasticity beyond classical single-receptor serotonin frameworks. Particular emphasis will be placed on the role of 5-HT2A receptors in amplifying cortical gain, prediction error, and network flexibility, alongside the role of sigma-1 receptors as stress-adaptive chaperones at mitochondria–endoplasmic reticulum contact sites, where they integrate neuromodulatory signals with cellular energy metabolism and proteostasis. Building on electrophysiological, molecular, and systems-level evidence, the talk will outline how psychedelic-induced signaling cascades can lower plasticity thresholds, promote structural and functional synaptic remodeling, and bias neural circuits toward a more flexible, exploratory operating regime even in late life. These mechanisms will be discussed in the context of prediction error processing, network criticality, and the balance between stability and adaptability in cortical and subcortical systems. Finally, the translational implications of reopening critical periods will be considered, including opportunities and risks for treating depression, addiction, neurodegeneration, and age-related cognitive decline. By framing psychedelics as chemical probes rather than single-target drugs, this lecture aims to integrate 5-HT2A and sigma-1 receptor biology into a broader theory of late-life neuroplasticity, offering a mechanistic roadmap for harnessing critical-period reopening as a strategy for brain repair and functional recovery.

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Keywords: Psychedelics, Critical-period plasticity, 5-HT2A receptor, Sigma-1 receptor

0576- Understanding Generations Z and Alpha: Definitions and Developmental Context

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The concepts of Generation Z and Generation Alpha have become increasingly prominent in academic, clinical, and educational discourse. Yet, these categories are often used descriptively rather than analytically, and their boundaries remain conceptually imprecise. Recent scholarship suggests that the label Generation Z is widely used but variably defined, while Generation Alpha is already well established in the literature despite still limited evidence that it represents a clearly distinct developmental or psychological category [1,2]. Accordingly, generational labels may be most useful not as fixed explanatory constructs, but as heuristic frames for examining how historical, technological, and socioecological conditions shape development.

From a developmental perspective, what distinguishes these cohorts is less their nominal generational identity than the environments in which they mature. Generation Z has largely come of age within an always-connected digital ecosystem shaped by mobile media, continuous connectivity, and intensified online peer culture [1]. Generation Alpha, in turn, is growing up in contexts in which digital technologies are present from very early childhood and increasingly embedded in learning, communication, and everyday family life [2]. These conditions have important implications for identity formation, emotion regulation, peer relationships, and mental health.

Recent evidence also suggests that digital life should not be conceptualized in uniformly negative terms. A systematic review of social media use and adolescent identity development found that the quality of online engagement appears more consequential than the mere quantity of use: active participation was associated with greater identity exploration, authentic self-presentation with higher self-concept clarity, and comparison-oriented engagement with greater identity distress [3]. Such findings support a more nuanced developmental interpretation in which digital environments may function both as opportunities for self-development and as sources of psychological vulnerability.

This presentation argues that clinicians and researchers should move beyond reductive stereotypes of contemporary youth as uniformly “screen-addicted,” “fragile,” or simply “digitally native.” Instead, Generations Z and Alpha should be understood through a developmental psychopathology framework that situates mental health within the interaction of developmental tasks, family and peer ecologies, digital environments, and broader sociocultural change. Such a framework may improve conceptual clarity, help distinguish normative adaptation from psychopathology, and inform developmentally sensitive, digitally literate, and context-responsive preventive and clinical approaches.

Keywords: Generation Z, generation alpha, developmental context, adolescence, digital environments, youth mental health

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0577 - The Role of ECT in Current Clinical Guidelines and Treatment-Resistant Conditions

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Electroconvulsive therapy (ECT), within the framework of current clinical guidelines, continues to maintain its position as one of the treatment options with a high level of efficacy, particularly in psychiatric conditions requiring rapid intervention and those resistant to treatment. Leading authorities such as the American Psychiatric Association, the National Institute for Health and Care Excellence, and the World Federation of Societies of Biological Psychiatry emphasize the high clinical value of ECT in critical conditions including severe mood disorders, psychotic states, and catatonia (1–4). Therefore, ECT is being repositioned not merely as a "last resort," but as a fundamental intervention that provides rapid clinical stabilization (5).

In clinical practice, ECT is most commonly used in major depressive episodes and is strongly recommended, particularly for patients with treatment-resistant depression, psychotic features, or a high risk of suicide (2,4,5). In addition, ECT is a guideline-supported and effective option for treatment-resistant mania and schizophrenia—especially in clozapine-resistant cases (1). Catatonia is among the conditions in which the fastest response to ECT is observed; severe agitation, acute psychotic exacerbations, and other refractory states are also areas where the effectiveness of ECT is particularly evident (1,5). High response rates and rapid onset of effect reported in agitation and aggression associated with dementia further indicate that ECT is an effective and acceptable option in selected patients (6).

In recent years, artificial intelligence- and neuroimaging-based studies have provided promising results in predicting ECT response, particularly through findings related to limbic structures; however, these approaches are still far from providing reliable clinical decision support at the individual level (7).

In conclusion, ECT continues to play a central role in the management of severe psychiatric conditions, and in the future, it may be implemented earlier and in a more personalized manner with the integration of artificial intelligence–supported and biomarker-based approaches.

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Keywords: Electroconvulsive Therapy, Depressive Disorder, Catatonia, Schizophrenia, Artificial Intelligence

0579 - Artificial Intelligence-Supported Psychotherapy and Digital Phenotyping in Children and Adolescents: A Systematic Review

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Background: Artificial intelligence (AI) is increasingly transforming child and adolescent mental health care by supporting the delivery, personalization, and optimization of psychotherapeutic interventions. AI-enabled approaches, including chatbot-based interventions, relational agents, app-based programs, gamified tools, and immersive technologies, may improve scalability, accessibility, and user engagement. In parallel, digital phenotyping, defined as the passive and continuous collection of behavioral and physiological data through smartphones, wearable devices, and related technologies, offers an ecologically valid framework for real-time monitoring of mental states. This may be particularly valuable in pediatric populations, where conventional self-report measures are often limited by developmental and contextual factors.

Objective: This systematic review aimed to synthesize the evidence on the clinical effectiveness, methodological quality, user acceptability, and ethical considerations of AI-supported psychotherapeutic interventions in individuals aged 0–18 years, with particular attention to digital phenotyping components where available.

Methods: This review was conducted in accordance with PRISMA 2020. A systematic search of PubMed/MEDLINE, Scopus, Web of Science, PsycINFO, and IEEE Xplore was performed for studies published between January 2005 and December 2025. Search terms included controlled vocabulary and free-text keywords related to artificial intelligence, psychotherapy, digital phenotyping, behavioral sensing, and digital biomarkers. A total of 1,489 records were identified. After title and abstract screening, 19 studies were retained for full-text assessment. Three studies were excluded for not meeting the predefined eligibility criteria. Reference screening of 29 systematic reviews yielded 9 additional eligible studies. Overall, 25 studies were included in the final synthesis. Data were extracted using a structured framework covering study design, participant characteristics, intervention modality, digital phenotyping features, outcomes, and methodological quality. Owing to substantial heterogeneity across studies, findings were synthesized narratively.

Results: Included studies were grouped into six primary intervention modalities: chatbot-based interventions, virtual therapist or relational agent systems, app-based structured psychotherapy, gamified interventions, VR/AR-based interventions, and hybrid digital interventions. Across studies, the most frequently reported outcomes were anxiety symptoms, depressive symptoms, treatment engagement, and accessibility of care. The evidence base suggested promising effects on symptom reduction and user engagement in several intervention formats; however, most studies were small-scale, methodologically heterogeneous, and varied considerably in intervention design, outcome measurement, and follow-up duration. Digital phenotyping components were incorporated in a subset of studies, primarily to support real-time monitoring, personalization, and early identification of treatment response or relapse risk. Recurrent ethical concerns included privacy, data security, algorithmic bias, transparency, and the need for clinician oversight.

Conclusions: AI-supported psychotherapy and digital phenotyping represent promising but still methodologically immature areas within child and adolescent mental health. Future research should prioritize clearer operational definitions of AI-supported care, standardized outcome reporting, robust ethical governance, and hybrid care models that integrate digital innovation with clinician-led practice.

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Keywords: Artificial intelligence, psychotherapy, digital phenotyping, child and adolescent psychiatry, precision psychiatry, chatbot

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0580 - Future Directions and Innovations in ECT

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Electroconvulsive therapy (ECT), with a history spanning over eighty years, remains one of the most effective biological treatments for severe clinical conditions such as treatment-resistant depression, mania, catatonia, and clozapine-resistant schizophrenia. With the technological opportunities, like artificial intelligence (AI), this treatment method is moving beyond being merely a "last resort" and is redefining its place in psychiatry as a personalized neuromodulation technique integrated with AI, advanced imaging, and precision medicine tools. AI technologies modernize the decision-making and application processes of ECT, making it safer and more effective. Machine learning models, trained on data from electronic health records, can predict patients' need for ECT with high accuracy by analyzing critical factors such as suicide risk assessments and violence scores. Furthermore, deep learning algorithms have the potential to determine the individualized electrical dosage required for successful seizure induction with over 90% precision by evaluating a patient’s pre-ictal EEG data.

In the field of predicting treatment response, machine learning algorithms can classify whether a patient will respond to ECT with nearly 80% accuracy using pre-treatment structural and functional MRI data. This allows patients who are unlikely to respond to be protected from unnecessary risks and costs. Neuroimaging studies have also proven that ECT does not cause brain damage; instead, it stimulates neuroplasticity and neurogenesis in the medial temporal region, creating a "brain-enabling" effect that helps reduce the social stigma surrounding the treatment.

In the near future, technical optimizations such as the use of ultrabrief pulse width and low current amplitude are expected to become widespread to minimize cognitive side effects during ECT applications. Research continues on innovative approaches like Focal Electrically Administered Seizure Therapy (FEAST), which focuses the seizure on the right prefrontal cortex, and Magnetic Seizure Therapy (MST), which induces seizures using magnetic fields as alternatives to traditional methods. Within the framework of precision medicine, the use of large-scale genomic data (such as the Gen-ECT-ic consortium) and biomarkers such as Brain-Derived Neurotrophic Factor and Vascular Endothelial Growth Factor will enable the development of treatment protocols tailored to each patient's biological profile.

By combining with AI and advanced biotechnology tools, ECT will continue to fulfill its life-saving role at the center of modern psychiatry as a more predictable and personalized treatment

Keywords: Artificial Intelligence, Electroconvulsive Therapy, Treatment Resistance

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0581 - Existential Significance and Mental Health

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The sense of meaning in life (MIL: Meaning in Life) has been discussed for thousands of years in religion, literature, and philosophy, and is widely regarded as a fundamental human concern. Contemporary psychology has also shown increasing interest in this concept, and meaning in life has become the subject of a growing number of studies.More recently, an academic consensus has emerged regarding the definition of meaning in life (MIL), reflecting the common features emphasized across the literature. Meaning in life refers to the overall set of subjective judgments through which individuals perceive their lives as valuable and significant, comprehensible and coherent, and defined by the adoption or maintenance of one or more highly valued overarching purposes or missions. This definition encompasses three dimensions of meaning: comprehension, purpose, and mattering/significance. (1)

Psychological approaches addressing existential anxiety also discuss the human struggle to feel that one's life is significant despite the absence of objective proof for such significance. Mattering refers to the extent to which individuals believe that their existence carries importance, value, and meaning in the world. It involves experiencing one's life and actions as consequential, effective, and meaningful. Individuals with a low sense of mattering tend to feel that their existence carries little importance and that their absence would make little difference in the world. (2)

It is important to consider the adaptive role played by a sense of mattering. Maintaining the view that one's own existence and the important aspects of one's life possess deep and objective value in the world may be adaptive. In individuals with a high level of mattering, existence carries greater meaning, significance, and deep value; death-related and existential anxiety are buffered, and according to Terror Management Theory, mattering may reduce the impact of death anxiety. In addition, greater inner balance is maintained in the face of existential threats, and a greater sense of calm is preserved when self-worth is threatened. In general, feeling significant within one's life and interpersonal relationships is positively associated with belongingness and self-esteem, and negatively associated with depression, loneliness, and suicidal ideation. (3,4)

This panel will discuss the psychological effects of perceiving one's own existence as important and valuable (mattering) within the framework of meaning in life and existential processes.

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Keywords: Existential significance, mental health, mattering

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0582 - PTSD with Comorbidities: Untangling the Factors That Fuel Treatment Resistance

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Objective: The objective of this presentation is to examine, in light of current evidence, the impact of psychiatric comorbidities frequently accompanying post-traumatic stress disorder (PTSD) on clinical phenomenology, therapeutic response, and treatment resistance. Within this framework, a clinical decision-making model for the prioritization and integration of comorbid conditions into the treatment plan is proposed, with particular emphasis on the necessity of reassessing co-occurring disorders when treatment resistance is encountered.

Content: Post-traumatic stress disorder is a clinical entity that frequently co-occurs with a wide range of psychiatric conditions—most notably major depressive disorder, anxiety disorders, substance use disorders, bipolar disorder, psychotic disorders, obsessive-compulsive symptoms, and somatization—thereby substantially complicating both the diagnostic and therapeutic processes. The first section of this presentation addresses the high prevalence of comorbidity in PTSD and its implications for clinical decision-making. It will be discussed that comorbidity not only increases symptom burden but also contributes to diagnostic masking, misattribution of illness severity, and the perpetuation of treatment resistance.

The second section examines the pathophysiological and psychopathological mechanisms through which comorbid disorders sustain treatment resistance. Particular attention will be directed toward the association between severe depressive symptomatology and motivational deficits, hopelessness, and diminished treatment engagement; the reinforcing effect of anxiety disorders on avoidance behaviors; the adverse impact of substance use disorders on treatment adherence and pharmacokinetic response; and the manner in which bipolar disorder and psychotic features constrain the feasibility of trauma-focused psychotherapeutic interventions. In this context, it will be underscored that a PTSD-exclusive treatment approach may prove insufficient in treatment-resistant cases, necessitating a comprehensive psychiatric reassessment.

The third section focuses on clinical assessment strategies and treatment prioritization. A thorough psychiatric history, chronological timeline-based symptom mapping, systematic family history inquiry, and the differential diagnosis between primary and comorbid conditions will be presented as the fundamental components of treatment planning. A stepped treatment model will be proposed, wherein acute clinical emergencies—such as acute psychotic episodes, high suicidal risk, or severe withdrawal syndromes—are stabilized first, followed by the management of severe depressive episodes and active substance use prior to initiating trauma-focused psychotherapy. Furthermore, the adaptation of evidence-based psychotherapeutic approaches in cases with comorbid depression, anxiety disorders, and substance use disorders, as well as the individualization of pharmacotherapeutic strategies according to co-occurring symptom clusters, will be discussed.

Conclusion: Treatment resistance observed in PTSD cannot, in the majority of cases, be attributed solely to the severity of trauma-related symptoms. The failure to recognize co-occurring psychiatric disorders, the inadequacy of their stabilization, or the absence of their holistic integration into the treatment plan may play a determining role in the persistence of resistance. Accordingly, the clinical approach should be founded upon an integrative model that does not exclude comorbidities but rather systematically identifies, monitors, and incorporates them into the treatment algorithm. When treatment resistance is encountered, the fundamental question should not merely be “Is the therapeutic response adequate?” but concurrently “Have the co-occurring psychiatric conditions been sufficiently evaluated and appropriately integrated into the treatment plan?”

Keywords: PTSD, psychiatric comorbidity, treatment resistance, integrated treatment

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0583 - ADHD in Elder Women: Premenopausal and Postmenopausal Period

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Attention-Deficit/Hyperactivity Disorder (ADHD) is a neurodevelopmental disorder that is not limited to childhood but can persist throughout life, and it exhibits a clinical course closely associated with hormonal changes in women (1,2). In particular, fluctuations in estrogen and progesterone levels during the premenopausal and postmenopausal periods have a significant impact on cognitive function and mood, thereby altering the severity of ADHD symptoms (3,4). The fact that ADHD is often diagnosed late in women and tends to present with a predominantly inattentive phenotype leads to confusion with cognitive complaints that emerge during menopause (2).

When evaluated in terms of hormonal effects, estrogen's regulatory role on dopaminergic and noradrenergic systems is noteworthy. These neurotransmitter systems, which have significant effects on prefrontal cortex functions, are critical in attention, executive functions, and motivational processes (5). Therefore, an increase in ADHD symptoms is expected during periods of declining estrogen levels. Indeed, systematic reviews indicate that low estradiol levels are associated with worsening ADHD symptoms (3).

The perimenopausal and menopausal transition is a critical period regarding ADHD. During this process, the irregular decline in estrogen levels leads to significant impairments in attention, working memory, and processing speed (1,6). Studies indicate that women diagnosed with ADHD experience menopausal symptoms more severely and exhibit more pronounced cognitive complaints (7). Additionally, comorbid conditions such as anxiety, depression, and sleep disorders can further exacerbate ADHD symptoms during this period (8).

In the postmenopausal period, persistently low estrogen levels create more pronounced effects on neurocognitive functions. It has been reported that in some women during this period, existing ADHD symptoms worsen, while in others, attention and executive function deficits emerge for the first time (9). This situation leads to an overlap between menopause-related cognitive changes and ADHD, resulting in diagnostic challenges.

From a neurobiological perspective, estrogen's effects on increasing dopamine release and supporting synaptic plasticity are directly related to the pathophysiology of ADHD (5,10). The decline in these neuroprotective effects during menopause negatively impacts not only ADHD symptoms but also overall cognitive performance (11).

Therefore, attributing cognitive complaints during menopause solely to aging is insufficient. In conclusion, the clinical course of ADHD in women is closely linked to hormonal life stages, and the menopausal period is a phase in which symptoms become more pronounced. Therefore, ADHD must be considered in the differential diagnosis when evaluating complaints of attention and cognitive function in women of middle age and older. Additionally, the integration of hormonal changes into treatment approaches represents an important area for future clinical research.

Keywords: Attention-Deficit/Hyperactivity Disorder, menopause, premenopause, postmenopause, women's health

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0584 - The Flow and Freezing of Time in Mood Disorders

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The subjective experience of time undergoes profound alterations in mood disorders. In major depressive disorder (MDD), time is perceived as "slowed down" or "frozen," whereas manic states are accompanied by a sense of time "speeding up" or "flying by." Despite the clinical ubiquity of these complaints, the neurobiological mechanisms underlying temporal distortions and their diagnostic potential remain insufficiently characterized. Converging evidence from phenomenology, cognitive neuroscience, and chronobiology now indicates that altered time processing is not a mere epiphenomenon but a core pathophysiological feature of mood disorders with substantial diagnostic and prognostic implications.

From a phenomenological perspective, depression produces a fundamental desynchronization between internal and external time. As Fuchs has articulated, depressed individuals become "overgrown by the past," losing their capacity for future-directed temporal orientation. Conversely, mania accelerates the internal clock, generating a mismatch in which the external world appears "too slow"—a phenomenon central to Northoff's "Too Fast or Too Slow" framework. Notably, the subjective experience of temporal slowing constitutes a significant and independent predictor of suicidal ideation severity, even after controlling for overall depression severity, thereby elevating this phenomenological observation to the status of a critical clinical warning signal.

At the neurobiological level, the Striatal Beat Frequency (SBF) model provides a robust framework for understanding temporal processing deficits in mood disorders. Dopaminergic dysregulation within cortico-striatal-thalamic circuits alters the oscillatory patterns of medium spiny neurons in the striatum, thereby distorting the neural encoding of time intervals. Complementing this circuit-level account, Wittmann's "Embodied Time" theory links interoceptive processing in the insular cortex to the subjective sense of temporal flow, explaining how the somatic heaviness characteristic of depression manifests as a perceived deceleration of time.

A particularly promising translational finding concerns the differential utility of temporal processing tasks as diagnostic biomarkers. Perceptual timing deficits, as measured by the temporal bisection task, are specific to unipolar depression and normalize upon remission, functioning as a "state marker". In contrast, motor timing deficits, assessed through paced finger-tapping paradigms, persist in euthymic bipolar patients, reflecting permanent cerebellar network dysfunction and thus constituting a "trait marker". This state-versus-trait dissociation offers a clinically accessible tool for resolving the persistent diagnostic ambiguity between unipolar and bipolar depression.

Within a chronopsychiatric framework, mood disorders represent a fundamental disruption of circadian architecture. Sleep-wake disturbances precede manic episodes by approximately 112 days and depressive episodes by 210 days, defining a prodromal "Chronos Syndrome" with significant early intervention potential. Current pharmacological agents operate, in part, through chronobiotic mechanisms: lithium and valproate stabilize molecular clock gene expression via GSK-3β inhibition, while ketamine rapidly restores disrupted temporal processing. Emerging therapeutic strategies—including Interpersonal and Social Rhythm Therapy, chronotype-informed interventions, and closed-loop neuromodulation—further underscore the centrality of temporal regulation in mood disorder management and point toward a future of temporally precise, personalized psychiatry.

Keywords: Bipolar disorder, chronopsychiatry, circadian rhythm, depression, mood disorders, time perception

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0585 - Machine Learning–Based Prediction of Treatment Response in Child and Adolescent Psychiatry

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Background:
Treatment response in child and adolescent psychiatry is highly heterogeneous, and clinical decision-making often relies on trial-and-error approaches. This variability can delay improvement, increase exposure to ineffective interventions, and reduce adherence. Machine learning (ML) offers a promising framework to model complex, non-linear interactions among clinical, behavioral, and biological variables, enabling more individualized, data-driven treatment strategies. However, the reliability and clinical applicability of ML-based prediction models in pediatric populations remain unclear.

Objective:
This systematic review aimed to synthesize current evidence on ML-based models for predicting treatment response in children and adolescents with psychiatric disorders, focusing on predictive performance, data modalities, methodological features, and clinical utility.

Methods:
This review followed PRISMA guidelines and a PROSPERO-registered protocol. Eligible studies included participants aged 0–18 years and applied ML techniques to predict treatment response, remission, or differential treatment benefit. Studies were required to report at least one performance metric (e.g., accuracy, AUC, sensitivity, specificity). Database searches identified 655 records; 19 full-text articles were assessed, and 10 studies met inclusion criteria. Due to substantial heterogeneity in diagnoses, predictors, outcomes, and validation strategies, a narrative synthesis was conducted. Given the heterogeneity of the included literature, a formal single risk-of-bias tool was not suitable for all studies. Therefore, study quality was appraised descriptively according to design, relevance, and methodological transparency.

Results:
Included studies addressed a range of psychiatric conditions, most commonly attention-deficit/hyperactivity disorder, anxiety and depression. Sample sizes varied widely, from small neurobiological cohorts (<30 participants) to large clinical datasets (>2,000 cases). Findings were grouped into three domains: (1) clinical and early-response models, (2) neurobiological models, and (3) digital or multimodal models.

Clinical models consistently identified early symptom improvement as the strongest predictor of later outcomes, with moderate performance. Models incorporating early-treatment dynamics outperformed those based on baseline variables. Neurobiological models, including neuroimaging and electrophysiological measures, occasionally showed higher performance but were limited by small samples, restricted generalizability, and lack of external validation. Digital and multimodal approaches integrating clinical, behavioral, and passive data demonstrated promising results, suggesting added value from combining data sources. ML models performed comparably to or better than conventional statistical methods, although direct comparisons were limited.

Conclusions:
ML-based approaches show promising but preliminary potential for predicting treatment response in pediatric psychiatry. Early symptom change appears to be the most robust predictor, while multimodal models may offer the greatest future value for precision psychiatry. The field remains constrained by methodological heterogeneity, limited external validation, and reporting. Future research should prioritize larger pediatric cohorts, standardized methodologies, and independent validation to improve reproducibility, generalizability, and clinical applicability.

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Keywords: Child and adolescent psychiatry, early treatment response, machine learning, multimodal modeling, precision psychiatry, treatment response prediction

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0586 - The Role of Neuroinflammation in Autism Spectrum Disorder: From Pathophysiology to Clinical Implications

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Autism spectrum disorder (ASD) is a neurodevelopmental condition characterized by persistent deficits in social communication and restricted or repetitive patterns of behavior [1]. The prevalence of ASD has increased substantially in recent decades. According to data from the Centers for Disease Control and Prevention (CDC), the prevalence of ASD has risen from approximately 1 in 152 children in 2002 to about 1 in 31 children in 2022. Current estimates further suggest that ASD affects approximately 1% of the global population. This increasing prevalence highlights the need to better understand the biological and pathophysiological mechanisms underlying ASD.

Accumulating evidence suggests that neuroimmune mechanisms and chronic neuroinflammatory processes may contribute to the pathophysiology of ASD [2]. In particular, postmortem studies have provided important insights into inflammatory alterations within the autistic brain. A systematic review of postmortem studies reported neuroinflammatory changes across multiple brain regions, including the prefrontal cortex, anterior cingulate cortex, cerebellum, and amygdala [1]. These investigations demonstrated increased activation of microglia and astrocytes, together with elevated levels of pro-inflammatory cytokines and chemokines, suggesting the presence of persistent neuroinflammatory responses in individuals with ASD [1].

At the cellular level, microglial activation appears to play a central role in ASD-related neuroinflammation. Microglia are the resident immune cells of the central nervous system and are involved in synaptic pruning, regulation of neuronal connectivity, and the release of inflammatory mediators [2]. Dysregulation of microglial activity during critical periods of brain development may disrupt synaptic organization and contribute to atypical neural circuit formation. Such mechanisms have been proposed as potential contributors to the altered brain connectivity frequently observed in individuals with ASD [2].

In addition to glial activation, alterations in immune-related gene expression have also been reported in the autistic brain. Molecular analyses of postmortem anterior cingulate cortex tissue revealed significant differences in the expression of genes associated with inflammatory signaling pathways and immune regulation [3]. These findings suggest that immune-related abnormalities in ASD occur not only at the cellular level but also at the molecular level.

Overall, converging evidence from postmortem neuropathological and molecular studies indicates that neuroinflammatory mechanisms may play an important role in the neurobiology of ASD. Increased glial activation, altered cytokine signaling, and immune-related gene expression changes collectively support the hypothesis that immune dysregulation contributes to atypical brain development in ASD [2,3]. A better understanding of these neuroinflammatory mechanisms may therefore facilitate the identification of biological biomarkers and the development of novel therapeutic strategies for ASD.

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Keywords: Autism Spectrum Disorder, Neuroinflammation, Neuroimmune Mechanisms, Biomarkers

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0587 - Pharmacogenetics and Artificial Intelligence: Personalized Medication Selection in Treatment-Resistant Cases

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Background: Treatment resistance in child and adolescent psychiatry remains a major challenge, particularly in depressive, psychotic disorders where medication selection relies on trial-and-error. Pharmacogenetics has emerged as a strategy to reduce uncertainty by identifying genetic variants associated with drug metabolism, tolerability, and response. CYP2D6 and CYP2C19 are the most studied and clinically actionable pharmacogenes, while SLC6A4 remains a potential moderator of antidepressant response. In parallel, artificial intelligence (AI) and machine learning (ML) approaches enable integration of genetic, demographic, clinical data into prediction models, supporting individualized prescribing in complex cases.

Objectives: This systematic review aimed to evaluate the accuracy and clinical relevance of AI- and ML-based pharmacogenetic models for predicting antidepressant and antipsychotic treatment response in children and adolescents. Specific objectives were to examine studies involving CYP2D6, CYP2C19, and SLC6A4 variants; compare AI-supported pharmacogenetic models with clinical assessment or genotype-guided approaches; and assess outcomes related to treatment response and adverse effects.

Methods: A systematic review was conducted using PubMed, Scopus, and Web of Science for studies published between 2015 and 2025. The search combined controlled vocabulary and free-text terms related to pharmacogenetics, artificial intelligence, machine learning, antidepressants, antipsychotics, treatment response, adverse effects, and pediatric psychiatry. Eligibility criteria followed PICO: children and adolescents with psychiatric disorders; AI/ML-based pharmacogenetic models; comparison with clinical evaluation or genetic testing; and outcomes including treatment response and side-effect burden. Reference screening and citation tracking were performed. A total of 1,057 records were identified, 505 remained after duplicate removal and screening, 123 underwent full-text assessment, and 8 studies were included. Methodological quality was evaluated using the Q-Genie tool.

Results: CYP2D6 and CYP2C19 emerged as the strongest pharmacogenetic candidates for individualized psychotropic prescribing, particularly regarding antidepressant metabolism and adverse-effect risk. Multigene pharmacogenetic approaches demonstrated greater predictive value than single-gene models. AI/ML models performed best when genetic data were integrated with biodemographic and clinical variables. For antipsychotics, genotype-informed dose adjustment appeared most relevant for selected CYP2D6-related agents, including risperidone and aripiprazole, although pretreatment genotyping was not supported. Across pediatric studies, sample sizes were small, replication limited, and methodological heterogeneity high. Overall study quality was fair to moderate.

Conclusion: AI-supported pharmacogenetic modeling represents a promising precision psychiatry approach for treatment-resistant youth, particularly when centered on CYP2D6 and CYP2C19 within multivariable frameworks. However, current pediatric evidence remains insufficient for routine implementation. These models should be considered adjunctive decision-support tools, while future research should prioritize larger prospective cohorts, external validation, standardized outcomes, and improved methodological quality. Their greatest utility lies in complex cases characterized by nonresponse, intolerance, or polypharmacy.

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Keywords: Artificial intelligence, CYP2C19, CYP2D6, machine learning, pharmacogenetics, treatment resistance

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0588 - Digital Natives and Mental Health: The Use of Artificial Intelligence in Young People's Psychiatric Treatment Processes

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Background: The mental health needs of "digital natives," a generation socialized within continuously connected digital environments, are reshaping child and adolescent psychiatric practice. Rising rates of depression, anxiety, stress-related symptoms, and psychosocial dysregulation among young people have coincided with intensified smartphone use, social media immersion, and digitally mediated peer interaction. In parallel, artificial intelligence (AI) has begun to enter psychiatric care through tools designed for risk detection, symptom monitoring, decision support, and digitally delivered intervention. Recent reviews suggest that AI is already being applied across adolescent mental health care in diagnosis, treatment planning, symptom monitoring, and prognosis, although the field remains uneven and diagnostically weighted [1].

Current Evidence and Clinical Applications:

Emerging work in digital phenotyping suggests that adolescents' everyday digital traces may provide clinically meaningful markers of mental state. In one 2024 study, passive data from smartphones and tablets were combined across 15 categories of daily digital activity, generating 1,523 candidate features as potential digital biomarkers for adolescent mental health assessment; the proposed machine learning framework improved predictive performance relative to conventional modeling strategies [2]. Such findings are clinically important because they point toward earlier recognition of distress before symptom escalation becomes visible in routine consultation.

At the intervention level, AI-driven digital therapeutics are extending the reach of youth psychiatric care. These include conversational agents, adaptive cognitive behavioral therapy platforms, and multimodal systems capable of tailoring responses to emotional state, engagement pattern, and inferred risk. A 2024 prototype for adolescent mental health management reported 85% accuracy for gaslighting detection and 87% accuracy for verbal abuse detection, while its disaster-recovery support component was associated with improved stress reduction in simulated settings [3]. More broadly, a 2025 systematic review of digital interventions for adolescent anxiety and depressive symptoms identified 20 studies and 17 distinct interventions; although only a minority were rated "quite promising," the review concluded that several digital approaches show meaningful potential for accessibility, engagement, and symptom reduction [4].

Taken together, the literature indicates a shift from episodic, reactive care toward proactive and longitudinal models of psychiatric support. AI may help clinicians identify subtle changes in functioning, personalize treatment intensity, and extend monitoring beyond the clinic. Recent evidence also emphasizes that adolescent AI research remains concentrated in assessment, with comparatively less mature evidence for treatment implementation and long-term outcome improvement [1].

Conclusion:
AI has substantial potential to augment youth psychiatric treatment processes by improving early identification, personalization, and continuity of care. Its most appropriate role is not to replace clinicians, but to function as a carefully governed decision-support and therapeutic extension within ethically grounded, developmentally sensitive, and clinically supervised care systems. Future research should prioritize prospective validation, equity, explainability, and real-world effectiveness in child and adolescent populations.

Keywords: Artificial Intelligence, Digital Health, Mental Health

AuthorToEditor: Dear Scientific Committee, I would like to kindly note that this abstract has been prepared as part of the panel titled "Artificial Intelligence in Psychopharmacology: Applications, Experiences and Future Perspectives", which is scheduled to take place in Hall 6, on 16:45–18:15, within Symposia Oral Session-20. The panel will be held with the following session chairs and speakers: Session Chairs: Selma Tural Hesapçıoğlu Muhammed Tayyib Kadak Presentations: • Artificial Intelligence Applications in Psychopharmacology: Current

Status from Diagnosis to Treatment – Yasin Çalışkan • Digital Natives and Mental Health: The Use of Artificial Intelligence in Young People's Psychiatric Treatment Processes – Yavuz Meral • Artificial Intelligence in Psychopharmacology: Ethical Issues, Challenges and Future Trends – Tuncay Sandıkçı • AI-Driven Prediction of Drug-Drug Interactions in Psychopharmacology: A New Era of Safety – Abas Haşimoğlu Accordingly, the submitted abstract should be considered as a panel presentation abstract belonging to this symposium session. ORCID iD: https://orcid.org/0000-0002-4767-6126 Kind regards, Yavuz Meral, MD

0589 - Parental Support or Symptom Alignment? Accommodation Dynamics in ADHD

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Family accommodation (FA) refers to the ways in which family members—primarily parents—change their own behaviors and daily routines to help a relative alleviate or avoid the distress caused by a psychological disorder. Current clinical perspectives define Attention-Deficit/Hyperactivity Disorder (ADHD) not merely as an attentional deficit but primarily as a Self-Regulation Disorder. A critical clinical resistance factor emerging in treatment literature is "Family Accommodation" (FA), wherein caregivers or partners modify their behaviors and routines to alleviate the patient's immediate distress or functional deficits. This presentation aims to evaluate the neurobiological costs of FA and its role in maintaining functional impairment across the lifespan. Current research suggests that FA in ADHD functions as a "secondary brain" phenomenon, where parents provide external organization to compensate for a child's executive function (EF) deficits in planning, organization, and self-regulation.

Research indicates that family accommodation is endorsed by approximately 97% of caregivers in ADHD cases, representing a profound transdiagnostic similarity to prevalence rates seen in anxiety disorders. Although these behaviors are well-intentioned expressions of empathy, they paradoxically reinforce child dysfunction. From a neurobiological perspective, FA serves as an external compensatory mechanism for executive dysfunction. It is discussed in the literature that such chronic external regulation may impede prefrontal cortex (PFC) maturation and negatively affect the development of functional connectivity within internal self-regulation networks. Furthermore, this "artificial stability" provided by the family environment is anticipated to limit neural plasticity by decreasing the individual's requirement to activate internal neural circuitries. Over time, high levels of FA are significantly associated with increased symptom severity, functional impairment, and profound caregiver burden, often manifesting as parental burnout and marital conflict.

In adult ADHD populations, accommodation dynamics frequently transfer from parents to romantic partners. This partner accommodation is defined not merely as a relational discord but as a major barrier to treatment adherence and the acquisition of adult life skills. Such role shifts in relationships often lead to a "parent-child" dynamic, resulting in a significant functional disability that precludes independent living and diminishes emotional intimacy. Family accommodation is a significant variable that negatively impacts the prognosis of ADHD by masking executive deficits and maintaining maladaptive behaviors. Interventions must move beyond simple symptom control to include a systematic reduction of accommodation. Clinicians are encouraged to employ Planned Autonomy or Dose-controlled Challenges, allowing patients to experience natural consequences in low-stakes environments.

The primary clinical recommendation is the integration of the "scaffolding" method into treatment protocols. This involves the gradual, systematic withdrawal of external supports, thereby necessitating the active engagement of the patient's prefrontal capacity and facilitating neurobiological maturation.

Keywords: Family-accommodation, neuroplasticity, scaffolding, self-regulation

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0590 - ADHD in Women From The Perspective of Hormonal Cycles: Neurobiology, Clinic and Treatment Options

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The Menstrual Cycle and ADHD in the Reproductive Period: PMS, Comorbidities and Treatment Approaches

Attention-deficit/hyperactivity disorder (ADHD) in women has long been underrecognized, partly due to its variable clinical presentation across the reproductive lifespan. Hormonal fluctuations during the menstrual cycle are increasingly understood to influence the severity of ADHD symptoms. In particular, changes in estrogen levels may affect cognitive functions, attention, and emotional regulation, leading to cyclical variations in symptom expression.

Premenstrual syndrome (PMS) and premenstrual dysphoric disorder (PMDD) commonly co-occur with ADHD and may contribute to increased functional impairment. Women with ADHD often report worsening of attention difficulties, irritability, and emotional dysregulation during the premenstrual phase. This overlap suggests shared underlying mechanisms and highlights the importance of considering hormonal influences in clinical assessment.

Psychiatric comorbidities such as anxiety and depressive disorders are frequently observed in women with ADHD and may fluctuate in parallel with hormonal changes. These interactions can complicate both diagnosis and treatment, requiring a more nuanced and individualized clinical approach.

Treatment strategies should take into account the potential impact of menstrual cycle phases on symptom severity. While stimulant medications remain the mainstay of ADHD treatment, some patients may benefit from flexible or adjusted treatment approaches across the cycle. In addition, interventions targeting premenstrual symptoms, including pharmacological and non-pharmacological options, may improve overall functioning. Psychoeducation, symptom tracking, and behavioral strategies focused on emotional regulation are important components of care.

In conclusion, the interaction between hormonal changes and ADHD symptoms plays a significant role in the clinical presentation of women during the reproductive period. A personalized and phase-informed approach may enhance treatment outcomes and improve quality of life.

Keywords: ADHD, Menstrual Cycle, Premenstrual Syndrome, Premenstrual Dysphoric Disorder

0591 - Use of Anti-Amyloid Therapies in the Treatment of Mild Cognitive Impairment

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For decades, the pharmacological management of Alzheimer's disease (AD) has been limited to symptomatic treatments like cholinesterase inhibitors and memantine. While providing temporary cognitive benefits, they do not alter the underlying neuropathological trajectory. Particularly in Mild Cognitive Impairment (MCI) due to AD, symptomatic treatments have failed to delay progression to dementia, leaving a profound therapeutic gap. The recent clinical validation of the amyloid cascade hypothesis has catalyzed a historic paradigm shift. The introduction of disease-modifying therapies (DMTs), specifically anti-amyloid monoclonal antibodies such as lecanemab and donanemab, represents the first intervention capable of altering the biological course of the disease.

These novel agents target distinct conformations of amyloid-beta (Aβ) peptides to clear toxic amyloid plaques. In the CLARITY AD trial, lecanemab demonstrated a 27% slowing of clinical decline on the CDR-SB over 18 months. Recent 48-month data presented at AD/PD 2026 confirmed that continuous lecanemab treatment sustains cognitive benefits. Similarly, the TRAILBLAZER-ALZ 2 trial revealed that donanemab slowed clinical progression by 36% in patients with low/medium tau pathology, utilizing a finite-dosing protocol where treatment is discontinued upon achieving amyloid clearance. Long-term data from early 2026 indicated that clinical benefits are maintained for up to three years post-discontinuation.

Despite these achievements, integrating anti-amyloid therapies into clinical practice introduces substantial challenges. The most critical safety concern is Amyloid-Related Imaging Abnormalities (ARIA), encompassing edema (ARIA-E) and hemorrhage (ARIA-H). ARIA incidence is highly correlated with the APOE ε4 genotype, reaching up to 40-50% in homozygous patients, necessitating mandatory pre-treatment genetic testing and rigorous MRI monitoring. Consequently, the European Medicines Agency (EMA) restricted these drugs to non-homozygous individuals. Furthermore, appropriate patient selection mandates biological confirmation of amyloid pathology via PET imaging or emerging blood-based biomarkers, such as plasma p-tau217, recently validated as an industry standard.

In Turkey, while these therapies are not yet licensed by the regulatory authority, their inclusion as a "considerable" option in the Ministry of Health's 2025 Clinical Protocol signals an imminent transition. Navigating this new era requires a multidisciplinary approach emphasizing biomarker-supported early diagnosis, mandatory APOE genotyping, risk-benefit analysis tailored to individual genetic profiles, and shared decision-making with patients and caregivers. Subcutaneous formulations such as remternetug, and emerging combination strategies promise to make this transformative era more accessible and effective for patients with MCI.

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Keywords: Alzheimer, Anti-Amyloid Therapies, ARIA, Donanemab, Mild Cognitive Impairment, Lecanemab

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0593 - Phenomenology at the Intersection: Case-Based Exploration of Schizo-Obsessive Disorder with Expert Insight

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Case-Based Exploration of Schizo-Obsessive Disorder with Expert Insight

The co-occurrence of schizophrenia spectrum disorders and obsessive-compulsive symptoms represents a complex clinical entity often referred to as schizo-obsessive disorder. This intersection challenges traditional diagnostic boundaries, as patients may present with overlapping phenomenological features, including intrusive thoughts, compulsive behaviors, and psychotic symptoms such as delusions and hallucinations.

A phenomenological perspective is essential in differentiating obsessive thoughts from delusional beliefs. While obsessions are typically experienced as intrusive and ego-dystonic, delusions tend to be held with stronger conviction and reduced insight. However, in clinical practice, this distinction is not always clear-cut, particularly in patients with impaired insight or fluctuating symptom profiles.

Case-based approaches provide valuable insight into this overlap by illustrating how obsessive-compulsive symptoms may precede, accompany, or emerge during the course of psychotic disorders. These cases highlight the heterogeneity of presentations and the importance of careful longitudinal assessment.

Comorbidity in this population is associated with greater functional impairment, increased symptom severity, and more complex treatment needs. Pharmacological management may be challenging, as certain antipsychotic medications have been associated with the emergence or exacerbation of obsessive-compulsive symptoms. At the same time, serotonergic treatments may alleviate obsessive symptoms but require cautious use in the context of psychosis.

An integrated treatment approach combining pharmacological strategies with psychotherapeutic interventions is recommended. Cognitive-behavioral techniques adapted to the level of insight and reality testing may be beneficial. Expert-informed, individualized care is crucial in addressing both symptom domains and improving overall functioning.

In conclusion, schizo-obsessive presentations require a nuanced understanding of phenomenology and a flexible, patient-centered approach to diagnosis and treatment.

Keywords: Chizo-obsessive disorder, phenomenology, comorbidity, psychosis

0594 - PTSD and Complex PTSD: Unpacking the Roots of Treatment Resistance

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Post-Traumatic Stress Disorder (PTSD) has long been understood in clinical psychiatry not merely as a reaction to a singular terrifying event, but as a state where the nervous system remains functionally locked in a survival mode. However, for individuals subjected to prolonged, repeated, or early-childhood interpersonal violence, the clinical picture often transcends the traditional boundaries of PTSD. The formal recognition of Complex PTSD (CPTSD) in the ICD-11 has shifted the focus toward "Disturbances in Self-Organization" (DSO), which include chronic affect dysregulation, persistent negative self-concept, and profound relational difficulties. While standard trauma-focused protocols such as EMDR or TF-CBT yield highly effective results in PTSD cases, this process may encounter various clinical barriers arising from disturbances in self-organization in individuals diagnosed with CPTSD. This resistance is often rooted in epistemic mistrust, a phenomenon where the patient perceives incoming social and therapeutic information as inherently threatening, thereby sabotaging the therapeutic alliance before it can even begin. Furthermore, recent neurobiological insights from 2025 and 2026 suggest that chronic trauma may induce a cellular brake on neuroplasticity, where astrocytic GABA levels in the prefrontal cortex physically inhibit the brain's capacity for fear extinction and the integration of new, safe memories. Consequently, the failure of standard treatments is not necessarily a failure of the patient or the technique, but rather a mismatch between the intervention and the patient's narrowed window of affective tolerance. Overcoming this resistance requires a shift from merely processing traumatic memories to first stabilizing the fragmented self and addressing the biological rigidity that prevents cognitive restructuring. Current literature now emphasizes that the path to recovery for complex cases lies in synchronizing intensive, frequent therapeutic interventions with biological modulators to bypass these ingrained neural and psychological barriers. In this session, we will discuss the clinical differentiation between PTSD and CPTSD, the specific neurobiological mechanisms that foster treatment resistance, and the emerging evidence-based strategies designed to break through these therapeutic impasses.

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Keywords: PTSD, Complex PTSD, Treatment Resistance

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0595 - Ultra-High Risk for Psychosis: Diagnostic Criteria and Clinical Assessment Methods

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Psychotic disorders, with schizophrenia as a prototypical example, are a group of mental health conditions that are predominantly chronic and impose major personal, social, and economic burdens on individuals and society. These disorders are generally understood to emerge after a prodromal phase marked by subtle psychological and behavioral changes. Within this spectrum, the concept of ultra-high risk (UHR), now increasingly referred to as clinical high risk for psychosis (CHR-P), is used to identify individuals who do not currently meet full diagnostic criteria for a psychotic disorder but who are at increased likelihood of developing psychosis in the near future. The main goal of identifying this group is to detect psychotic disorders early, enable timely intervention, and reduce later functional impairment. At the same time, high risk should not be understood only as a predictor of psychosis transition, but also as a broader transdiagnostic condition associated with other serious outcomes such as depression, mania, anxiety, and personality disorders.

In the diagnosis of UHR/CHR-P, three classical groups of criteria are used. The first is Attenuated Psychotic Symptoms (APS), referring to subthreshold positive symptoms that emerged within the past year but did not reach the level of a full psychotic episode. The second is Brief Limited Intermittent Psychotic Symptoms (BLIPS/BIPS), which are distinct psychotic episodes lasting less than one week and resolving spontaneously without treatment. The third is Genetic Risk and Deterioration (GRD), defined by a significant decline in functioning within the past year together with either psychosis in a first-degree relative or schizotypal personality disorder in the individual. There has been debate about whether these criteria should be included as a distinct DSM-5 diagnosis to improve intervention and resource allocation. However, critics argue that basing diagnosis on vague or subthreshold symptoms may increase stigma, unnecessary antipsychotic use, and false positives. These concerns make structured assessment essential. For this reason, semi-structured tools such as the Structured Interview for Psychosis-Risk Syndromes (SIPS) and the Comprehensive Assessment of At-Risk Mental States (CAARMS) are recommended. However, the two systems differ in symptom thresholds, duration, and frequency criteria. More recently, the PSYCHS interview has been developed to improve conceptual and operational standardization between them. Clinical evaluation should not rely only on positive symptoms. Research shows that negative symptoms, impaired functioning, cognitive deficits, and neurocognitive difficulties, especially in verbal memory, are important predictors of transition to psychosis. Certain electrophysiological measures, such as mismatch negativity, may also contribute to risk assessment. However, biomarkers are not yet standardized enough for routine clinical use. Therefore, the most reliable approach remains the combined use of structured interviews and functional evaluation. Current evidence also shows that risk cannot be explained by diagnostic labels alone and must be considered with individual and environmental factors, comorbidity, suicide risk, substance use, family history, and social or occupational functioning. Overall, UHR/CHR-P should be viewed not only as a precursor to psychosis but as a broad-spectrum mental health risk syndrome requiring comprehensive and multidimensional assessment.

Keywords: Clinical High Risk for Psychosis (CHR-P), Early Detection of Psychosis, Psychosis-Risk Assessment, Ultra-High Risk (UHR)

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0596 - Biomarker Studies in Individuals at Clinically High Risk for Psychosis and First Psychotic Episode

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Schizophrenia spectrum disorders are severe neuropsychiatric conditions that typically emerge during late adolescence and early adulthood, causing profound cognitive, emotional, and socioeconomic impairments. The traditional psychiatric paradigm, which relies on diagnosing the illness during the first episode of psychosis (FEP), has fundamentally shifted over the past two decades. Current neurodevelopmental models conceptualize psychosis not as a sudden brain injury, but as a progressive "dysmaturation" process resulting from the complex interplay of genetic vulnerabilities, environmental stressors, and epigenetic modifications. This paradigm shift has redirected clinical attention toward the prodromal phase, known as the clinically high-risk (CHR) state. While approximately 15% to 35% of CHR individuals transition to overt psychosis within three years, the remaining majority either stabilize or remit. This clinical uncertainty creates a critical dilemma: false positives risk exposing young patients to unnecessary antipsychotic treatments with severe metabolic side effects, whereas false negatives leave a deteriorating brain unprotected. Overcoming this challenge requires transitioning from symptom-based diagnostic systems to a "Precision Psychiatry" framework, which redefines psychiatric illnesses into biologically distinct biotypes using objective biomarkers.

Neuroimaging has been the flagship of biomarker research. Structural magnetic resonance imaging (sMRI) studies have consistently demonstrated accelerated gray matter loss, particularly in the hippocampus and superior temporal gyrus, as a robust predictor of transition to psychosis. Furthermore, magnetic resonance spectroscopy (MRS) has revealed that the core neurochemical pathology is a severe glutamatergic hyperactivity rather than purely dopaminergic dysfunction. Elevated striatal glutamate levels, exhibiting massive effect sizes (Cohen's d = 1.39), induce excitotoxicity that drives the observed structural atrophy.

Functional MRI and diffusion tensor imaging further highlight the collapse of thalamocortical connectivity and white matter integrity, reflecting a profound failure in sensory gating and neural network synchronization. Electrophysiological markers, such as mismatch negativity (MMN) and P300, offer highly translational and cost-effective tools. MMN deficits, reflecting N-methyl-D-aspartate receptor hypofunction and impaired predictive coding, serve as early indicators of the impending psychotic storm. Concurrently, peripheral blood biomarkers have proven that psychosis is not merely a brain disorder but a systemic pathology. Elevated pro-inflammatory cytokines (e.g., IL-6, TNF-) cross the blood-brain barrier, triggering microglial hyperactivation and aggressiveα synaptic pruning. This inflammatory cascade is accompanied by severe oxidative stress, characterized by depleted glutathione levels and increased lipid peroxidation. Recent longitudinal studies have further demonstrated that epigenetic aging clocks accelerate dramatically at the point of conversion, with biological age advancing approximately 2.8 years per calendar year.

Despite these monumental discoveries, recent comprehensive umbrella reviews indicate that no single biomarker has yet achieved the reliability required for routine clinical implementation. The future of high-risk psychosis research lies in multimodal machine learning approaches. By integrating neuroimaging data, electrophysiological signals, peripheral inflammatory markers, polygenic risk scores, and novel digital phenotyping metrics, algorithms can now predict conversion and treatment response with accuracies exceeding 85%. Adhering to the Precision Psychiatry Roadmap, the ultimate goal is to detect the biological sparks of psychosis and extinguish them with targeted neuromodulatory or anti-inflammatory interventions before the clinical fire consumes the brain.

Keywords: Biomarkers, Clinically high risk, First episode psychosis, Neuroimaging, Neuroinflammation, Precision psychiatry

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0597 - Under-Discussed Clinical Phenomena in ADHD: Time Blindness, Hyperfocus, Social Anxiety, Masking, and Rumination

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Ankara Etlik Şehir Hastanesi

Abstract

Objective: Attention-Deficit/Hyperactivity Disorder (ADHD) is commonly defined in terms of observable symptoms such as inattention, hyperactivity, and impulsivity. However, clinical observations suggest that certain internal cognitive and emotional processes that influence individuals' daily functioning are only limitedly addressed within standard diagnostic frameworks. Phenomena such as time blindness and difficulties in time organization, hyperfocus, mind wandering, rumination, social anxiety, and masking provide an important context for understanding the functional outcomes of ADHD. The aim of this presentation is to examine these processes within the framework of attention regulation and to discuss their potential interactions in a clinical context.

Findings: Although hyperfocus, rumination, and mind wandering present with different phenomenological characteristics, they may be considered as distinct manifestations of difficulties in attention regulation. Hyperfocus is characterized by an intense and prolonged fixation of attention on a specific stimulus; rumination involves repetitive and sustained attention directed toward internal, often negatively valenced thoughts; whereas mind wandering refers to a shift of attention away from the ongoing task toward internally generated thoughts. These patterns may reflect difficulties in directing, maintaining, and flexibly shifting attention in accordance with contextual demands. Within this framework, time blindness may be conceptualized not only as a disturbance in time perception, but also as a temporal manifestation of impaired attention regulation. The inability to organize attention across time may affect the capacity to monitor the passage of time, anticipate durations, and structure behavior temporally. During hyperfocus, attention may become excessively fixed within a particular time segment, whereas during mind wandering and rumination, temporal continuity may be disrupted. These patterns may be associated with difficulties in time organization. Fluctuations in attention regulation may become particularly evident in social contexts. Difficulties in sustaining attention during conversations, missing social cues, or challenges in following the flow of interactions may contribute to social experiences becoming more demanding for some individuals. Repeated experiences of this nature may be associated with increased sensitivity and anxiety in social settings. In turn, symptoms of social anxiety may further influence attentional processes, suggesting a bidirectional interaction. In response to these challenges, some individuals may develop compensatory strategies commonly described as masking. These strategies may include deliberate efforts to appear attentive and engaged, mentally rehearsing social interactions in advance, overpreparing, or avoiding demanding social situations. While such approaches may support functioning in the short term, they may also be associated with increased cognitive load and fatigue over time.

Conclusion: The difficulties observed in ADHD may not be limited to externally observable symptoms; internal cognitive and emotional processes related to attention regulation may also play a significant role in functional outcomes. Considering the relationships between time blindness and difficulties in time organization, as well as hyperfocus, rumination, and mind wandering, may contribute to a more comprehensive understanding of social experiences, emotional responses, and coping strategies in ADHD. This approach may facilitate the development of more context-sensitive and individualized strategies in clinical assessment and intervention.

Keywords: ADHD, attention regulation, hyperfocus, mind wandering, social anxiety, masking

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0598 - Biomarker Use in Clinical Practice: Is Targeted Antidepressant Treatment Possible?

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Major depressive disorder is a clinically heterogeneous condition, yet treatment selection is still largely based on trial and error. For this reason, the real clinical value of biomarker research in depression lies beyond biologically defining the disorder. Its main promise is the ability to predict which treatment is more likely to benefit a given patient. [1,2]

Although classical subtypes help describe the clinical diversity of depression, the current literature increasingly views depression as a syndrome made up of biologically distinct subgroups. These subgroups are difficult to explain through a single biomarker. This perspective supports a shift from symptom-based classification toward a precision psychiatry approach grounded in mechanisms. [2,3] Clinically, the problem is clear: remission rates after the first treatment attempt remain limited, ineffective initial treatments delay recovery, and there is often no strong scientific basis to guide the next treatment choice when the first treatment fails. [1,3]

In this context, biomarkers in depression should be considered as more than a research tool for understanding the biological basis of the disorder. They may also serve as a clinical instrument to improve treatment decisions. The key question is not simply whether a biological signal can be found. More importantly, it is whether that signal can guide treatment choice, predict response, and improve clinical decision making. [1,2] In that sense, biomarkers provide an important conceptual basis for moving toward more targeted and patient specific treatment strategies in depression. [1,3]

This presentation addresses the clinical use of biomarkers in depression through the question: is targeted antidepressant treatment possible? First, it discusses why a biomarker-based approach is needed in depression. It also examines how current treatment practice remains insufficient. It then outlines the clinical meaning of biomarkers, their potential contribution to treatment selection, and why blood-based indicators have attracted particular attention.

Thereafter, it discusses how the field has moved from the search for single markers toward more integrated models. It also examines the promising aspects of current biomarker research. In addition, it considers why these findings have not yet become part of routine clinical practice. Finally, the presentation addresses which basic features future biomarkers should have in order to achieve real clinical relevance. In this way, it aims to discuss both the current limits and the future potential of targeted antidepressant treatment.

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Keywords: Antidepressant treatment, biomarker, major depressive disorder, precision psychiatry, targeted-treatment

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0599 - Hidden Behavioral Addictions in Youth: Compulsive Buying, Exercise, Hypersexual Behavior, and Binge Eating

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Behavioral addictions, historically described as impulse control disorders, are increasingly recognized as a significant mental health concern, particularly among youth. These conditions include patterns such as compulsive shopping, binge eating, exercise addiction, and hypersexual behavior. Unlike substance addictions, they involve compulsive engagement in rewarding behaviors that provide psychological relief or pleasure despite harmful consequences.

At the neurobiological level, behavioral addictions share core mechanisms with substance use disorders. Central to this process is the mesolimbic reward system, an evolutionarily conserved neural circuit that reinforces survival-related behaviors. Evidence suggests that this system can be altered not only by substances but also by repeated behaviors, leading to similar patterns of reinforcement and dependency. Through neuroplasticity, repeated high-reward behaviors can reshape neural circuits in ways comparable to pharmacological effects.

Learning and memory processes play a critical role in both substance and behavioral addictions. Functional neuroimaging studies further demonstrate that activities such as eating, exercising, or shopping activate brain regions similar to those implicated in drug addiction, particularly within the mesocorticolimbic system. Despite growing evidence supporting these similarities, significant debate remains regarding the classification of behavioral addictions. Critics argue that expanding diagnostic categories risks pathologizing everyday behaviors and blurring the boundary between normal and pathological functioning. In addition, high comorbidity rates complicate diagnosis. Many behavioral addictions co-occur with mood disorders, anxiety disorders, ADHD, and personality disorders, raising the question of whether these behaviors represent independent conditions or secondary manifestations of underlying psychopathology.

These conceptual challenges become clearer when examining specific behavioral addictions. Compulsive buying disorder (CBD) is characterized by uncontrollable urges to purchase unnecessary items despite negative consequences. Individuals typically experience preoccupation with shopping, temporary relief during purchasing, and subsequent feelings of guilt or distress. The addictive component appears to lie in the act of spending itself, which activates reward and decision-making systems. However, CBD is not formally recognized in DSM-5 or ICD-11, partly due to its high comorbidity with mood and personality disorders.

Compulsive sexual behavior involves persistent and intense sexual urges that are often used as a coping mechanism for negative emotional states. Its classification remains controversial, as it is frequently associated with underlying psychiatric conditions. While DSM-5 does not include it as a distinct disorder, ICD-11 classifies it under impulse control disorders.

Binge eating disorder, in contrast, has stronger neurobiological support. Evidence links it to dopaminergic and serotonergic dysregulation, as well as impaired inhibitory control, resulting in compulsive consumption patterns similar to substance addiction. Exercise addiction is defined by excessive and uncontrollable physical activity despite harmful consequences. It may function either as a primary addiction or as a secondary behavior associated with body image concerns or eating disorders, highlighting the importance of differential diagnosis. Overall, the shared neurobiological and behavioral mechanisms between substance and behavioral addictions underscore the importance of early recognition and intervention, particularly in youth populations. Nevertheless, ongoing debates regarding classification, comorbidity, and diagnostic boundaries highlight the need for further empirical research to establish clearer clinical frameworks and more effective treatment strategies.

Keywords: Behavioral addictions, binge eating, compulsive buying, exercise addiction, hypersexual behavior, youth

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0600 - Game or Addiction? Assessing Gaming and Gambling Disorders in Children and Adolescents

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The rapid expansion of digital gaming environments has introduced new challenges in distinguishing normative engagement from clinically significant behavioral addiction in children and adolescents. While diagnostic frameworks such as Gaming Disorder and Internet Gaming Disorder provide structured criteria, emerging evidence emphasizes that functional impairment rather than duration or frequency of use remains the most reliable clinical discriminator. In this context, problematic gaming is increasingly conceptualized through its displacement effect, wherein excessive engagement replaces essential developmental activities such as sleep, academic performance, and peer interaction.

Recent literature highlights that the evolution of game design has significantly altered the risk landscape. In particular, loot boxes have been identified as a critical intersection between gaming and gambling. These systems operate on variable ratio reinforcement schedules, a well-established mechanism associated with high behavioral persistence, and incorporate psychological features such as near-miss outcomes and salient audiovisual feedback. Empirical studies suggest that exposure to loot boxes is associated with increased risk of problem gambling behaviors, especially in adolescents, whose neurodevelopmental stage is characterized by heightened reward sensitivity and immature cognitive control.

Beyond isolated mechanics, the broader convergence of gaming and gambling ecosystems has become increasingly evident. Virtual in-game items, including cosmetic skins, can acquire real world monetary value and be used within third-party platforms for gambling-like activities, a phenomenon commonly referred to as skin gambling. Similarly, the growing accessibility of esports betting introduces additional pathways for early exposure to gambling behaviors among minors. This convergence supports the “gateway hypothesis,” proposing that early engagement with gambling-like features in games may increase vulnerability to subsequent gambling disorders. From a clinical perspective, assessment requires a multidimensional approach that integrates behavioral, functional, and financial indicators. Key features include loss of control, persistence despite negative consequences, and the extent to which gaming or gambling-related activities interfere with daily functioning. Importantly, financial behaviors such as unplanned or concealed spending serve as critical markers in differentiating gaming disorder from gambling-related pathology. Differential diagnosis remains essential, as conditions such as ADHD, depressive disorders, and anxiety disorders may present with overlapping behavioral patterns, including excessive screen use or avoidance-based engagement.

Taken together, current evidence underscores the need for a practice-oriented framework that moves beyond categorical definitions and incorporates emerging digital risk structures into clinical assessment. These domains will be addressed within an integrated model emphasizing functional impairment, gambling-like game mechanics, and developmental vulnerability.

Keywords: Gaming Disorder, Loot Boxes, Adolescent Behavioral Addiction, Gambling Convergence, Digital Gaming

0601 - Shared Neurobiology, Neurodevelopmental Windows, and Pediatric Vulnerability in Behavioral Addictions

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Behavioral addictions, including problematic internet use, gaming disorder, and excessive social media engagement, have increasingly been recognized as significant concerns in child and adolescent mental health. Although these conditions do not involve exogenous substances, converging evidence indicates that they share core neurobiological mechanisms with substance use disorders. At the same time, developmental characteristics unique to childhood and adolescence critically shape vulnerability to these behaviors. The rapid expansion of digital environments has resulted in early and sustained exposure of children to highly stimulating, reward-driven systems, making it essential to understand these behaviors within an integrated neurodevelopmental framework.

This paper provides a comprehensive overview of the shared neurobiological substrates underlying behavioral addictions, outlines key neurodevelopmental windows, and examines factors contributing to heightened vulnerability in pediatric populations. Particular emphasis is placed on the role of the mesolimbic dopamine system, including projections from the ventral tegmental area to the nucleus accumbens, which mediate reward processing, motivation, and reinforcement learning. Repeated engagement with rewarding digital stimuli may lead to neuroadaptations such as increased salience attribution to reward-related cues and diminished responsiveness to natural reinforcers. In parallel, alterations in prefrontal cortical regions associated with executive control, impulse regulation, and decision-making contribute to reduced top-down regulation of behavior.

The paper also highlights the importance of neurodevelopmental timing. During childhood and adolescence, ongoing processes such as synaptic pruning and myelination shape neural circuits in an experience-dependent manner. A developmental imbalance between early-maturing limbic systems and the later-developing prefrontal cortex results in heightened reward sensitivity combined with limited inhibitory control. This imbalance creates a neurobiological context in which individuals are particularly responsive to immediate rewards and less capable of regulating behavior in the presence of highly reinforcing stimuli.

In addition, pediatric vulnerability is examined as a multifactorial construct involving biological, psychological, and environmental influences. Immature executive functions, heightened dopaminergic sensitivity, and ongoing emotional development interact with external factors such as continuous access to digital technologies, reinforcement-based platform design, and social feedback mechanisms. These interacting influences contribute to patterns of repeated engagement and increased susceptibility to behavioral dysregulation.

Overall, this integrative perspective underscores that behavioral addictions in youth are best understood as the result of dynamic interactions between neurobiological systems and developmental processes. Recognizing these mechanisms provides a framework for conceptualizing behavioral addictions within pediatric populations and highlights the importance of considering both brain-based and developmental factors in future research.

Keywords: Adolescence, Behavioral addiction, Dopaminergic reward system, Neurodevelopment, Pediatric vulnerability, Prefrontal cortex

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0602 - Theoretical Controversies and Conceptual–Diagnostic Boundaries in Behavioral Addictions

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Behavioral addictions occupy a contested space at the intersection of addiction psychiatry, impulse-control disorders, and compulsivity-related conditions. The central question is not only whether repetitive, reward-seeking behaviors can become genuinely addictive, but also how such conditions should be classified when they share features with substance use disorders, obsessive-compulsive phenomena, and problems of self-regulation. This issue is particularly complex in child and adolescent populations, where high engagement, novelty seeking, and fluctuating self-control are often developmentally normative.

Current diagnostic frameworks reflect this ambiguity. While gambling disorder is formally recognized as a behavioral addiction, other conditions such as gaming have been more cautiously incorporated, and several repetitive behaviors remain outside formal classification systems. These inconsistencies highlight the ongoing tension between phenomenological similarity, neurobiological evidence, and clinical utility in defining what constitutes an addiction.

A key conceptual concern in this field is the risk of overpathologizing everyday behaviors. Not all frequent or highly valued activities should be labeled as addictive. To qualify as a disorder, behaviors should be persistent, associated with impaired control, and result in significant functional impairment. This distinction is especially critical in youth, where behaviors such as prolonged gaming, intensive exercise, or online engagement may reflect normative developmental processes or transient coping strategies rather than psychopathology.

Beyond categorical distinctions, a dimensional perspective may offer a more nuanced understanding. Behavioral addictions can be conceptualized along an impulsivity–compulsivity continuum. Early stages are often characterized by reward sensitivity and impulsive engagement, whereas more persistent patterns may involve habitual, negatively reinforced, and compulsive behaviors. Emerging evidence suggests that compulsivity-related traits may be present even in early phases, supporting transdiagnostic models that cut across traditional diagnostic boundaries.

From a developmental perspective, the challenge is not to expand diagnostic categories indiscriminately, but to clarify the mechanisms underlying these behaviors and their clinical significance at different stages of maturation. Some presentations may align more closely with addictive processes, while others may be better understood within impulse-control or obsessive-compulsive frameworks. In children and adolescents, this distinction requires particular caution, with emphasis on functional impairment, persistence, and context rather than intensity alone. Ultimately, advancing the field depends not only on refining diagnostic systems, but also on sharpening conceptual boundaries and maintaining a developmentally informed, clinically grounded approach.

Keywords: Adolescence, behavioral addictions, compulsivity, impulsivity, nosology, overpathologizing

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603 Abstract: The Performance Paradox in ADHD

Title: The Performance Paradox in ADHD: How the Motivation–Anxiety Cycle Sabotages Treatment and How CBT Helps

Introduction: Attention-Deficit/Hyperactivity Disorder (ADHD) is increasingly recognized not merely as a deficit of attention, but as a disorder of self-regulation and executive function. The "Performance Paradox" refers to the jarring discrepancy between an individual's cognitive potential and their actualized life outcomes. While patients often possess the requisite skills to succeed, the neurobiological inability to consistently engage these skills leads to a debilitating cycle of "performance-by-crisis." This panel explores how the interplay between impaired motivation and compensatory anxiety creates a feedback loop that often undermines standard pharmacological and psychological interventions.

The Motivation–Anxiety Cycle: At the heart of the paradox lies the "Procrastination–Scurry" cycle. Due to dopamine-related deficits in the reward system, individuals with ADHD struggle to initiate tasks that lack immediate salience. To compensate, they unconsciously utilize anxiety as a "neurobiological starter motor." By delaying tasks until the eleventh hour, the resulting surge of cortisol and adrenaline provides the focus that the dopaminergic system fails to supply. Over time, this makes anxiety a functional necessity for performance, leading to a "maladaptive symbiosis" where the patient fears that treating their anxiety or relaxing will result in total loss of productivity.

Sabotage of Treatment: This cycle poses a significant challenge to clinical management. When anxiety acts as a primary driver for performance, standard treatments can be perceived as threats. For instance, stimulant medication may improve focus but might also heighten physiological arousal, potentially exacerbating the underlying anxiety. Conversely, if anxiolytics are prescribed without addressing the ADHD-driven executive dysfunction, the patient may lose their only "functional" motivator, leading to a collapse in productivity. This "Catch-22" often results in poor treatment compliance or partial remission of symptoms.

The Role of CBT: Cognitive Behavioral Therapy (CBT) specifically tailored for ADHD offers a vital intervention by deconstructing this cycle. Instead of merely managing time, CBT targets the "All-or-Nothing" cognitive distortions and the emotional dysregulation that fuels the paradox. Through cognitive restructuring, patients learn to decouple performance from panic. Behavioral activation and "chunking" techniques help lower the threshold for task initiation, reducing the need for anxiety-driven adrenaline spikes. By building robust executive function bridges—such as externalizing time and utilizing environmental cues—CBT transitions the patient from "survival-based performance" to "regulated performance."

Conclusion: Addressing the Performance Paradox requires moving beyond symptom checklists. Clinicians must recognize anxiety not just as a comorbidity, but often as a dysfunctional coping mechanism for ADHD. A multimodal approach that integrates pharmacological support with ADHD-specific CBT allows patients to break the motivation-anxiety loop, ultimately aligning their performance with their true potential.

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Keywords: ADHD, Cognitive Behavioral Therapy, Executive Function, Performance Paradox, Anxiety, Motivation

604 - The Camouflage Effect: Understanding Autism Spectrum Disorder in Girls

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Autism Spectrum Disorder (ASD) has traditionally been conceptualized based on predominantly male presentations, which has contributed to the underrecognition of ASD in girls. Emerging evidence indicates that girls with ASD may present with a distinct phenotype, particularly in social behavior and coping strategies, which can complicate timely identification. One important factor contributing to this underdiagnosis is the phenomenon of "camouflaging."

Camouflaging refers to the conscious or unconscious use of strategies to mask social communication difficulties and adapt to neurotypical expectations. These strategies may include imitation of peers, use of rehearsed social scripts, suppression of autistic traits, and increased effort to maintain socially appropriate interactions. While such strategies may facilitate short-term social adaptation, they often require significant cognitive and emotional effort.

Girls with ASD are more likely to demonstrate relatively preserved superficial social reciprocity and may develop interests that appear socially typical, which can further obscure underlying difficulties. As a result, their clinical presentation is frequently misinterpreted as anxiety, shyness, or mood-related problems rather than ASD. Previous studies have consistently shown that camouflaging behaviors are more prevalent in females and are associated with delayed diagnosis (1,2).

In clinical practice, many adolescent girls present primarily with internalizing symptoms such as anxiety and depression rather than overt social communication difficulties, which may obscure the underlying autistic profile. This discrepancy between external functioning and internal distress represents a key challenge for clinicians. Notably, prolonged camouflaging has been linked to increased psychological burden, including higher levels of stress, depressive symptoms, and suicidal ideation (3).

The camouflaging effect has important implications for both assessment and intervention. Current diagnostic tools may lack sensitivity to detect subtle or compensated autistic traits in girls, highlighting the need for a more nuanced and gender-informed clinical approach. Comprehensive evaluation should include developmental history, subjective experiences, and context-dependent functioning rather than relying solely on observable behaviors.

In conclusion, camouflaging plays a central role in the underrecognition of ASD in girls and contributes to delayed diagnosis and increased mental health burden. Greater awareness of gender differences in ASD and refinement of assessment strategies are essential for improving early identification and providing appropriate support for this population.

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Keywords: Autism Spectrum Disorder; Camouflaging; Female Autism Phenotype; Adolescence; Internalizing Symptoms; Diagnostic Delay

605 - Clozapine-Induced Myocarditis: Importance of Early Screening and Management

Abstract:
Clozapine remains the gold standard for treatment-resistant schizophrenia, demonstrating superior efficacy compared to other antipsychotics. While effective, its use is limited by potentially serious adverse effects, including agranulocytosis, seizures, gastric hypomotility syndrome, and myocarditis. Although relatively rare, Clozapine-associated myocarditis (CAM) carries substantial morbidity and mortality risk, necessitating increased clinical awareness and systematic monitoring strategies.

The incidence of clozapine-induced myocarditis varies widely, ranging from 0.03% to 8.5%, while cardiomyopathy is less common, occurring in approximately 0.02% to 1% of patients. In a large meta-analysis including 258,961 individuals, myocarditis was reported in approximately 0.07% of patients, with an associated mortality rate of 0.04%. Myocarditis usually occurs early, with a mean onset of 17 days and 82% of cases within three weeks; fever often appears between days 10 and 16 as a prodromal sign. Cardiomyopathy develops later, on average after 14.4 months, typically presenting with heart failure symptoms such as dyspnea and palpitations.

Risk factors for clozapine-associated myocarditis (CAM) include rapid dose titration, concomitant use of sodium valproate, older age, and the initial four weeks of treatment, with potential contributions from genetic and environmental predispositions. Recent infections or mRNA vaccination may complicate early diagnosis, and current recommendations suggest delaying clozapine initiation following such conditions to reduce risk. Clinical presentation is often nonspecific and may include fever, chest pain, dyspnea, tachycardia, fatigue, myalgias, palpitations, dizziness, syncope, or flu-like symptoms.

Structured screening protocols are essential for early detection. Recommended monitoring includes baseline and serial measurements of high-sensitivity troponin and C-reactive protein (CRP), along with electrocardiography. Elevations in cardiac biomarkers (troponin above the 99th percentile or NT-proBNP ≥125 pg/mL) should prompt further evaluation, including echocardiography or cardiac magnetic resonance imaging using the modified Lake Louise criteria. Supportive imaging findings may include left ventricular dysfunction or abnormal global longitudinal strain.

Early recognition of CAM is critical, as prompt discontinuation of clozapine typically leads to symptom resolution and reversibility of myocardial injury. Patients require hospitalization and multidisciplinary management, including cardiology consultation. Supportive treatment may involve beta-blockers, angiotensin-converting enzyme inhibitors, diuretics, and, in selected cases, corticosteroids. Physical activity restriction during the acute phase is advised due to increased risk of arrhythmias.

Rechallenge with clozapine after CAM remains controversial. Case series suggest that careful patient selection, consideration of CRP levels, baseline left ventricular function, and slow up-titration may improve the likelihood of a successful rechallenge. Recommended titration protocols start with 6.25 mg on day 1, 25 mg on day 7, 50 mg on day 14, and 100 mg by week 4, providing gradual desensitization to clozapine's hypothesized IgE-mediated hypersensitivity reaction. Approximately 65% of patients in reported cases successfully resumed therapy without recurrent myocarditis, highlighting the importance of individualized risk assessment and close monitoring.

In conclusion, awareness and early detection of CAM are critical to reduce morbidity and mortality. Routine screening during the initial weeks of clozapine therapy, combined with careful dose titration and prompt intervention upon symptom onset, can minimize adverse outcomes. Multidisciplinary management, including psychiatric and cardiology collaboration, remains essential for optimizing patient safety and therapeutic success.

Keywords: Clozapine, Myocarditis, Cardiotoxicity, Screening, Treatment-Resistant Schizophrenia, Risk Factors

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606 - Title: The Chemical Biology of Psychoactive Substances

Abstract

Despite major advances in basic brain science over the past decade, the discovery of truly new drugs for neuropsychiatric disorders has lagged, exposing a central paradox: why has our mechanistic understanding of the brain accelerated so rapidly while therapeutic innovation has largely stalled? This talk addresses that gap by positioning psychoactive compounds—especially classic psychedelics and addiction-relevant substances—not as "mere substances," but as precise chemical tools that can be engineered, calibrated, and deployed to interrogate brain function across scales. Building on the premise that many clinically relevant states are emergent properties of distributed circuits rather than single molecular targets, the program presented here uses multi-target (polypharmacological) perturbations to dissect how defined receptor-level events propagate through intracellular signaling to reshape synaptic plasticity, circuit computations, and brain-wide network connectivity. The speaker will describe a systematic chemical-biology framework for turning psychedelics into mechanistic probes that reveal how transient changes in neuromodulatory signaling can relax rigid network states, open windows of metaplasticity, and bias learning toward durable behavioral change—features that may help explain why limited dosing can sometimes yield long-lasting clinical benefits. A core theme is that psychedelics provide a unique bridge between consciousness science and disease pharmacology: by aligning subjective shifts in perception, self-processing, and affect with objective readouts of neural dynamics, the work aims to translate "experience-level" phenomena into tractable biomarkers of target engagement and therapeutic mechanism. Beyond neurons, the talk will emphasize that neuroimmune modulation is not peripheral, but often integral to brain-state transitions, influencing glial activity, inflammatory tone, synapse remodeling, and stress-resilience programs that intersect with depression, trauma-related disorders, and substance use disorders. By integrating neuroimmune biology with circuit mapping, the program seeks a more complete and causally grounded account of both therapeutic opportunity and safety liability. The presentation will further outline how this chemistry-enabled approach can support anti-addiction therapy development by identifying convergent nodes where maladaptive learning, stress signaling, and immune dysregulation meet, and by using psychoactive probes to map and normalize the circuits underlying craving, habit, and relapse vulnerability. Finally, the speaker will discuss translational strategies that connect discovery to clinical breakthroughs, including designing next-generation probes and candidate therapeutics with controllable kinetics and improved dissociation between beneficial plasticity and unwanted acute psychoactivity, and using mechanism-linked biomarkers to guide patient stratification, dosing, and combination interventions. Overall, the talk argues that neurochemical biology—deploying psychoactive molecules as rigorously defined perturbations—can convert rapid progress in brain science into an actionable pipeline for new treatments.

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607 - Reopening Critical Periods: Psychedelics, Sigma-1 Receptor Signaling, and Late-Life Neuroplasticity

Abstract

This talk explores how psychedelics can reopen critical periods of plasticity in the adult and aging brain. Critical periods are developmental windows during which neural circuits exhibit heightened malleability, enabling efficient learning and circuit refinement; their closure in adulthood contributes to reduced adaptability and increased vulnerability to neuropsychiatric and neurodegenerative disorders. Psychedelics such as psilocybin, lysergic acid diethylamide, and related psychoplastogens have emerged as powerful tools to transiently restore juvenile-like plasticity states, yet the molecular logic underlying this phenomenon remains incompletely understood. Focusing on the concerted roles of the 5-HT2A receptor and the sigma-1 receptor as central signaling hubs, this lecture will discuss how psychedelics engage multi-target pharmacology to modulate cortical excitability, intracellular calcium dynamics, mitochondrial function, lipid microdomains, and neuroimmune interactions, thereby reshaping synaptic and network-level plasticity beyond classical single-receptor serotonin frameworks. Particular emphasis will be placed on the role of 5-HT2A receptors in amplifying cortical gain, prediction error, and network flexibility, alongside the role of sigma-1 receptors as stress-adaptive chaperones at mitochondria–endoplasmic reticulum contact sites, where they integrate neuromodulatory signals with cellular energy metabolism and proteostasis. Building on electrophysiological, molecular, and systems-level evidence, the talk will outline how psychedelic-induced signaling cascades can lower plasticity thresholds, promote structural and functional synaptic remodeling, and bias neural circuits toward a more flexible, exploratory operating regime even in late life. These mechanisms will be discussed in the context of prediction error processing, network criticality, and the balance between stability and adaptability in cortical and subcortical systems. Finally, the translational implications of reopening critical periods will be considered, including opportunities and risks for treating depression, addiction, neurodegeneration, and age-related cognitive decline. By framing psychedelics as chemical probes rather than single-target drugs, this lecture aims to integrate 5-HT2A and sigma-1 receptor biology into a broader theory of late-life neuroplasticity, offering a mechanistic roadmap for harnessing critical-period reopening as a strategy for brain repair and functional recovery.

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608 - Mind the Gap: Rethinking Lithium Monitoring for the 21st Century

Rebecca Strawbridge

Despite lithium's established role as a first-line treatment for bipolar disorder and an augmentation strategy in treatment-resistant depression, monitoring in routine practice remains suboptimal, with only around half of patients receiving guideline-concordant testing (Nikolova et al., 2018). This gap continues to pose challenges for patient safety, treatment continuity, and service efficiency. This presentation will describe a randomised feasibility trial evaluating the integration of point-of-care (POC) monitoring into routine lithium care for individuals with unipolar and bipolar affective disorders receiving maintenance treatment (Kerr-Gaffney et al., 2025).

The trial evaluates a POC device that is already validated, clinically accurate, and certified for use in the Netherlands, capable of measuring both lithium and creatinine from capillary blood samples. The primary aim is to determine the feasibility of testing effectiveness in a future definitive trial, focusing on recruitment, retention, adherence to monitoring standards, and the risk of contamination between trial arms. Acceptability to patients and clinicians is examined alongside practical and service-level considerations relevant to secondary care and shared-care models.

Secondary outcomes include changes in health-related quality of life, depressive and manic symptoms, healthcare utilisation, and health economic measures, providing preliminary data to inform the design of a larger multi-site randomised controlled trial. Eighty participants are randomised 1:1 to POC monitoring or standard laboratory-based testing and followed over 30 weeks. The proportion of participants meeting recommended monitoring guidelines is the primary outcome, complemented by patient-reported and service-use measures.

This work explores whether validated POC testing can offer a scalable approach to improving safety, patient experience, and monitoring adherence in long-term lithium treatment.

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609 - Difficult to Treat Depression

Christopher Aiken

One in three patients with major depressive disorder do not achieve meaningful recoveries with antidepressants. Raising the dose, switching antidepressants, and augmenting with other antidepressants are common in practice but failed in large trials. This talk will review effective strategies, emphasizing treatments with long-term benefits in what is often a chronic disorder. Lithium and pramipexole are supported by long-term trials, but antipsychotics failed in most maintenance trials. Psychotherapy has evidence in treatment-resistant depression, and may offer better preventative effects than antidepressants. Some treatments address common deficits in depression, such as light therapy, l-methylfolate, vitamin D, omega-3 fatty acids, and probiotics. In other cases, we can identify causes of treatment resistance that point the way to specific treatments, such as bipolar disorder, psychotic depression, vascular depression, and inflammation. When pharmacotherapy is ineffective, neuromodulation brings higher rates of remission, including ECT, TMS, and accelerated (SNT) TMS.

Top Research Updates of Past Year

This talk will review clinical trials with the potential to change practice, focusing on novel findings and large, placebo-controlled trials. We will consider the quality of the evidence as we examine trials that tested pramipexole, saffron, quetiapine, lithium, psychodynamic therapy, and the ketogenic diet for depression; GLP-1 agonists for alcohol use disorder; mentalization therapy for antisocial personality disorder; and a new lithium interaction. We review recent neuropsychiatric approvals and rejections from the US Food and Drug Administration (FDA).

Oral Research Presentations

[Abstract:0095]

Abuse Of Unexpected Drugs: A Case Series

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Samsun Mental And Nervous Diseases Hospital

Introduction: Prescription drugs with clinically relevant sedative, analgesic, anxiolytic, anesthetic, or stimulant effects have the potential for abuse. Oxybutynin, baclofen, and carbamazepine are drugs that can be abused in this regard. This case series examines unanticipated drug abuse through three cases in which oxybutynin, baclofen, and carbamazepine were misused solely for their sedative or anxiolytic effects rather than their medicinal properties. İnformed consent has been obtained from all participants.

Case presentation:

Case 1:

A 25-year-old unemployed male patient applied to an inpatient clinic for drug addiction treatment. He reported that he had been using oxybutynin for 5 years to get rid of other substances he was abusing (cannabis and methamphetamine). Oxybutynin tablets were readily available from pharmacies without a prescription. The number he used gradually increased, and he had recently reached 250–300 pills per day (1250–1500 mg/day). After taking oxybutynin, he felt more energetic and relaxed, and his anxiety improved. While under the influence of the drug, he described some psychotic symptoms, including visual and auditory hallucinations and persecutory delusions. He experienced cravings, but no withdrawal symptoms. He was treated with carbamazepine 400 mg/day and olanzapine 20 mg/day. After treatment, his symptoms resolved completely within five weeks.

Case 2:

A 34-year-old unemployed male was referred to the inpatient clinic. He reported using baclofen for eight years to relax and feel better. He had easy access to the medication without a prescription. Initially, he took baclofen twice daily, but gradually increased the number of pills over time. Currently, he was taking 70-80 baclofen tablets daily (700-800 mg/day), for the past two years (700-800 mg/day). He reported that baclofen made him feel better, relaxed, and free of negative thoughts. However, his family also described anger, aggression, and psychotic symptoms. This was his second hospitalization to get off this drug. During inpatient treatment, he experienced withdrawal delirium. After his delirium improved, he was admitted to addiction treatment and discharged after six weeks.

Case 3:

A 28-year-old male prisoner insisted on a prescription for carbamazepine during an outpatient visit to the prison. However, no psychiatric diagnosis was found during the examination or in his past medical records that would require him to use this medication. His medical history revealed that the patient had fabricated various psychiatric symptoms that had led doctors to prescribe carbamazepine. He was crushing and snorting 600-800 mg of carbamazepine daily to feel better. Addiction treatment was started by discontinuing the carbamazepine. Four weeks later, the craving had significantly reduced.

Conclusion: Physicians often face the dilemma of avoiding contributing to the non-medical use of prescription medications while attempting to treat legitimate symptoms. Physicians are very cautious when prescribing commonly abused drugs, such as benzodiazepines or opioids. However, any medication that may have sedative or relaxing effects should be considered to have the potential for abuse. Strategies should be developed to prevent the over-the-counter availability of drugs that may have such effects and to implement preventative measures, such as monitoring the duration and dosage of use.

Keywords: Drug abuse, Oxybutynin, Baclofen, Carbamazepine

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[Abstract:0108]

Healthcare Workers' Knowledge, Attitudes, and Stigma About ADHD

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Background and Objective: Stigma refers to the negative labeling of individuals or groups based on characteristics such as mental illness, ethnicity, or disability. Attention-deficit/hyperactivity disorder (ADHD) is a neurodevelopmental disorder characterized by symptoms of inattention, hyperactivity, and impulsivity, and is among the most frequently diagnosed psychiatric disorders in childhood. Healthcare workers increasingly encounter children with ADHD in clinical practice. Insufficient knowledge and awareness may influence attitudes and approaches toward affected children and hinder appropriate access to treatment. This study aimed to evaluate healthcare workers' attitudes, stigma, and knowledge levels regarding ADHD in the province of Samsun and to examine the relationship between their beliefs toward mental illness. Additionally, ADHD-related attitudes and stigma levels were compared across sociodemographic characteristics and ADHD-related clinical experiences.

Methods: Healthcare workers from various departments of several hospitals in Samsun, Turkey were reached using the snowball sampling method and invited to complete an online survey. Participants (n=166) completed the Healthcare Worker Form, the ADHD Assessment Questionnaire, and the Beliefs Toward Mental Illness Scale (BTMIS). This study was approved by the University Clinical Research Ethics Committee with the decision number GOKAEK 2025/5/17.

Results: A total of 166 healthcare professionals participated in the study. The mean age of the participants was 39.76 ± 8.68 years, and their mean duration of professional experience was 16.90 ± 9.83 years. Most of them were female. Approximately 70% of participants identified ADHD as a brain- or genetics-based condition, and most endorsed behavioral therapy, medication, and psychotherapy as primary treatment approaches. Across the entire sample, ADHD-related stigma scores showed a significant positive correlation with the BTMIS total score ($r=0.307$, $p<0.001$) as well as with its subscales of shame ($r=0.187$, $p=0.016$), helplessness ($r=0.299$, $p<0.001$), and dangerousness ($r=0.248$, $p=0.001$). More than half of the healthcare workers believed that all children with ADHD are highly hyperactive, have behavioral problems, experience side effects from medication, that ADHD does not fully improve with treatment, and that children with ADHD should be placed in separate classrooms. Stigma scores were significantly higher among women ($p=0.029$) and parents ($p=0.023$), while misinterpretation scores were higher in those with a history of psychiatric evaluation ($p=0.040$). No significant difference was found in all other variables (such as receiving education about ADHD, closeness to the individual with ADHD, providing health care, experiencing difficulties, and use of psychiatric medication) in terms of both misinterpretation and stigmatization.

Conclusions: This study revealed multidimensional patterns in healthcare workers' knowledge, misinterpretation tendencies, and stigmatizing attitudes toward ADHD. Despite high awareness of ADHD, misconceptions and stigma persist. The most notable finding was the positive correlation between beliefs about mental illness and stigma toward individuals with ADHD. Elevated perceptions of helplessness and dangerousness were associated with increased stigmatizing attitudes. These results suggest that healthcare workers' general beliefs about mental illness strongly shape their attitudes toward ADHD. Therefore, interventions aimed at reducing stigma should not be limited solely to ADHD-focused education but should also address negative beliefs and perceptions surrounding mental illness more broadly.

Keywords: Adhd, mental illness, misinterpretation, stigmatization

Table 1. Participants' Knowledge and Experience About ADHD

Questions		N (%)
Have you ever heard of the diagnosis of ADHD? (n, %)	Yes	161 (97.0)
	No	5 (3.0)
Where did you hear about ADHD? (n, %)	High school/University courses	66 (39.8)
	From a physician	37 (22.3)
	From a relative/friend	19 (11.4)
	Internet	19 (11.4)
	In-service trainings	18 (10.8)
	Television	5 (3.0)
	I have not heard of it	2 (1.2)
What is the most appropriate definition of ADHD? (n, %)	A problem originating from the brain/genetics	115 (69.3)
	A medical term for "naughtiness"	27 (16.3)
	A problem caused by "high intelligence"	15 (9.0)
	A problem caused by environmental factors	8 (4.8)
	I do not know	1 (0.6)
What is the most appropriate treatment for ADHD? (n, %)	Behavioral therapy	122 (73.5)
	Medication	113 (68.1)
	Psychotherapy	102 (61.4)
	Special education	85 (51.2)
	Family therapy	94 (56.6)
Have you ever suspected a child might have ADHD? (n, %)	Yes	129 (77.7)
	No	37 (22.3)
What did you do when you suspected ADHD? (n, %)	Referred to child psychiatry	90 (54.2)
	Gave advice to the family	40 (24.1)
	I have not encountered such a case	24 (14.5)
	Referred to a pediatrician	12 (7.2)

Note: ADHD = Attention-deficit/hyperactivity disorder.

Table 2. Inter-Scale Correlation Analysis Findings

		1	2	3	4	5
Misinterpretation (1)	r	-				
	p					
Stigmatization (2)	r	0.120	-			
	p	0.122				
Shame (3)	r	**-0.175***	**0.187***	-		
	p	**0.024**	**0.016**			
Helplessness (4)	r	-0.125	**0.299****	**0.444****	-	
	p	0.109	**0.000**	**0.000**		
Dangerousness (5)	r	-0.094	**0.248****	**0.233****	**0.700****	
	p	0.229	**0.001**	**0.003**	**0.000**	.
Total (6)	r	-0.140	**0.307****	**0.469****	**0.941****	**0.879****
	p	0.072	**0.000**	**0.000**	**0.000**	**0.000**

Note: Spearman correlations, *p < .05, **p < .01

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[Abstract:0111]

The Attitudes, Beliefs and Knowledge of Parents About Methylphenidate Treatment

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Background and Objective: Attention-Deficit/Hyperactivity Disorder (ADHD) is one of the most common neurodevelopmental disorders of childhood, affecting approximately 5–7% of school-aged children. In the treatment of ADHD, methylphenidate (MPH) is frequently recommended as the first-line pharmacological intervention. However, it is well recognized that misconceptions—such as beliefs that methylphenidate causes addiction, alters personality, or impairs growth—are widespread in the general population and even among some healthcare professionals. Previous research has shown that parental lack of knowledge and concerns regarding medication treatment may result in premature discontinuation, dose omission, or refusal of pharmacotherapy. In the limited number of studies conducted in Turkey, it has been reported that both physicians and parents have insufficient knowledge about methylphenidate use, and that some families describe the medication as a "sedative" or resort to inappropriate patterns of use. The aim of this study is to examine the level of knowledge and feedback of parents of children diagnosed with ADHD regarding the use of methylphenidate.

Methods: Parents of 53 children aged 6 to 17 years who had been receiving methylphenidate treatment for at least one month and had a diagnosis of ADHD were included in the study. Parents were asked to complete a 14-item questionnaire developed by a specialist in child and adolescent psychiatry, which assessed their level of knowledge regarding methylphenidate and their observations of the medication's effects on their children.

Results: Among the participants, 45.3% (n = 24) had been receiving methylphenidate treatment for more than one year. No statistically significant differences were observed in responses between the group treated for less than one year and the group treated for more than one year. A total of 83.0% (n = 44) of parents reported that they had been sufficiently informed about methylphenidate use prior to initiating treatment. In addition, 32.1% (n = 17) of parents expressed concerns that the medication might be addictive. Furthermore, 60.4% (n = 32) reported concerns regarding the visibility of methylphenidate use in official records or issues related to confidentiality, while 35.8% (n = 19) indicated concerns that methylphenidate might cause long-term harm to their child. Overall, 83.0% (n = 44) of parents reported general satisfaction with methylphenidate treatment. In the analyses conducted, no statistically significant association was found between parental education level and responses to any of the 14 items ($p > 0.05$). All questions and responses are presented in Table 1.

Conclusions: A systematic assessment of parents' levels of knowledge regarding methylphenidate, the identification of common misconceptions, and the subsequent planning of targeted educational programs are of great importance. In this regard, the present study aims to both improve medication adherence in clinical practice and reduce misconceptions about methylphenidate within the community. The findings of this study are expected to contribute to the development of informational materials and psychoeducational programs designed for parents.

Keywords: Attention-Deficit/Hyperactivity Disorder, children, methylphenidate, parents

Questions Regarding Methylphenidate

Questions	Yes n (%)	No n (%)	I am not sure n (%)
I think that methylphenidate hasincreased my child's attention span	44 (83,0)	2 (3,8)	7 (13,2)
I have observed that the medication has had a positive effect on my child's academic performance	42 (79,2)	4 (7,5)	7 (13,2)
I received adequate information before starting methylphenidate treatment	44 (83,0)	4 (7,5)	5 (9,4)
I am informed about the possible side effects of methylphenidate	43 (81,1)	5 (9,4)	5 (9,4)
I have observed noticeable side effects in my child during medication use	15 (28,3)	35 (66,0)	3 (5,7)
I am concerned about the potential for methylphenidate to cause dependence in my child	17 (32,1)	19 (35,8)	17 (32,1)
I think that methylphenidate has had a positive effect on my child's social relationships	35 (66,0)	10 (18,9)	8 (15,1)
My child's teacher reported positive changes following the initiation of medication	43 (81,1)	4 (7,5)	6 (11,3)
We have difficulty using the medication regularly	4 (7,5)	49 (92,5)	0 (0,0)
I am generally satisfied with methylphenidate treatment	44 (83,0)	0 (0,0)	9 (17,0)
I think that methylphenidate has changed my child's personality	18 (34,0)	24 (45,3)	11 (20,8)
I am concerned about issues related to confidentiality or the medication's appearance in official records associated with methylphenidate use	32 (60,4)	15 (28,3)	6 (11,3)
I sometimes feel guilty about giving medication to my child	26 (49,1)	20 (37,7)	7 (13,2)
I am concerned that methylphenidate may cause harm to my child in the long term	19 (35,8)	17 (32,1)	17 (32,1)

AuthorToEditor: I would appreciate your consideration of my oral presentation abstract. Thank you Hilal Yazıcı Kopuz, Department of Child and Adolescent Psychiatry, Gaziantep City Hospital, Gaziantep, Turkey. hilal.yazici.ctf@gmail.com. https://orcid.org/0009-0001-2244-2980 Seda Erbaş, Department of Child and Adolescent Psychiatry, Gaziantep City Hospital, Gaziantep, Turkey. sedaerbas@yahoo.com. https://orcid.org/0000-0001-9591-6000

[Abstract:0113]

Psychological Correlates of Pain Severity in Adolescents with Non-Cardiac Chest Pain: A Multivariate Analysis

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Background and Objective:

Non-cardiac chest pain (NCCP) is a common reason for pediatric cardiology referrals during adolescence, typically in the absence of an identifiable organic pathology. Previous studies have highlighted the role of psychological factors such as anxiety sensitivity, alexithymia, somatosensory amplification, and pain catastrophizing in the perception and persistence of chest pain. Although these constructs have been individually associated with pain outcomes in various somatic conditions, their collective predictive power for pain intensity in NCCP remains uncertain. The present study aimed to evaluate the extent to which these psychological variables predict pain severity in adolescents with NCCP through a multivariate regression analysis.

Methods:

This naturalistic, cross-sectional study included all adolescents who presented with chest pain to the Pediatric Cardiology Clinic of Tepecik Training and Research Hospital over a 1.5-year period, between March 2024 and September 2025. After comprehensive anamnesis, physical examination, laboratory, and imaging evaluations, those without an identifiable organic cause were included. Sociodemographic and clinical data were collected using an Individual Assessment Form, which captured pain frequency, intensity, and qualitative characteristics. Psychological variables were assessed using the Childhood Anxiety Sensitivity Index (CASI), Toronto Alexithymia Scale (TAS-20), Pain Catastrophizing Scale for Children (PCS-C), and Somatosensory Amplification Scale (SSAS). A multivariate linear regression analysis was performed using the enter method to identify predictors of pain severity. All statistical analyses were performed using SPSS 25.0, with $p < .05$ considered significant.

Results:

Thirty-one adolescents (77.4% female; mean age = 14.9 ± 1.6 years) who met inclusion criteria during the study period were analyzed. The most common pain characteristic was compressive pain localized predominantly to the left chest region. The mean pain severity was 5.29 ± 2.28, and the mean weekly pain frequency was 3.87 ± 2.41. The multivariate regression model did not significantly predict pain severity, $F(7, 23) = 0.588$, $p = .759$ (Adjusted $R^2 = .106$). None of the psychological variables—including anxiety sensitivity, alexithymia, somatosensory amplification, or pain catastrophizing—showed a significant individual contribution to pain intensity.

Conclusions:

Although no significant predictors of pain severity were identified, these findings emphasize the complex, multifactorial nature of pain perception in adolescents with NCCP. The results indicate that psychological factors may contribute to pain through indirect or interacting mechanisms rather than direct predictive effects. Considering the limited sample size derived from the naturalistic 1.5-year cohort and the cross-sectional design, future studies with larger and longitudinal samples are warranted to clarify the psychological and developmental pathways underlying NCCP in youth. Identifying these mechanisms may ultimately inform tailored psychotherapeutic interventions, including cognitive-behavioral and emotion-focused approaches, to mitigate functional impairment and recurrent healthcare utilization in affected adolescents. Nevertheless, these results underscore the importance of adopting integrative biopsychosocial frameworks when evaluating idiopathic pain in pediatric cardiology settings.

Keywords: Adolescents, alexithymia, anxiety sensitivity, non-cardiac chest pain pain catastrophizing

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[Abstract:0120]

Perceived Competence to Initiate Clozapine Treatment Among Child and Adolescent Psychiatrists: The Role of Institutional Support and Professional Experience

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Background and Objective: Clozapine is the most effective antipsychotic for treatment-resistant psychotic disorders, yet its use in child and adolescent psychiatry remains limited. In addition to clinical considerations, clinicians' perceived competence and institutional context may influence decisions to initiate clozapine. This study assessed child and adolescent psychiatrists' perceived competence in initiating clozapine treatment and examined associated individual and institutional factors.

Methods: This cross-sectional study included 77 child and adolescent psychiatrists who completed a structured online survey. Perceived competence was assessed using a single self-report item rated on a five-point Likert scale and dichotomized as competent ("often/always") versus not competent ("sometimes/rarely/never") for binary logistic regression analysis. The survey also assessed professional experience, outpatient workload, difficulty managing medication side effects, and difficulty referring patients for inpatient psychiatric care. Perceived adequacy of institutional and physical support was additionally assessed using a yes/no item covering access to inpatient services, experienced support staff, and pharmacological resources for acute intervention.

Results: Overall, 43 clinicians (55.8%) were classified as competent, whereas 34 (44.2%) were classified as not competent. The regression model was statistically significant ($\chi^2(5) = 31.61$, $p < .001$), explained 51.5% of the variance in perceived competence (Nagelkerke $R^2 = 0.52$), and correctly classified 77.9% of cases. Longer professional experience was significantly associated with higher perceived competence (OR = 1.26, 95% CI = 1.01–1.57, p =.041), whereas outpatient workload was not (OR = 1.24, 95% CI = 0.36–4.28, p =.727). Perceived adequacy of institutional and physical support showed the strongest independent association with perceived competence; clinicians reporting inadequate institutional support were significantly less likely to feel competent (OR = 0.05, 95% CI = 0.01–0.52, p =.011). Difficulty managing medication side effects was not independently associated with perceived competence (OR = 3.06, 95% CI = 0.56–16.62, p =.199). Difficulty referring patients to inpatient psychiatric care showed a trend-level association but did not reach statistical significance (OR = 4.35, 95% CI = 0.95–19.94, p =.058).

Conclusions: Perceived competence to initiate clozapine treatment among child and adolescent psychiatrists appears to be associated with both professional experience and institutional conditions, with inadequate institutional support showing a particularly strong association with lower self-reported competence. These findings should be interpreted cautiously because of the cross-sectional design, the use of a single-item self-report measure, and the possibility of conceptual overlap among some institutional variables. In addition, perceived competence may not directly reflect actual clozapine prescribing behavior. Despite these limitations, improving clinicians' perceived competence in initiating clozapine may require not only individual education but also institutional structures that support timely assessment, monitoring, and access to inpatient care when needed. Future research should examine institutional support in greater detail, including access to inpatient care, the availability of experienced support staff, and pharmacological resources for acute intervention. Efforts to improve clozapine use in child and adolescent psychiatry may therefore need to address organizational and systemic barriers in addition to individual-level training.

Keywords: Child and adolescent psychiatry, clozapine, institutional support, perceived competence, professional experience

Binary Logistic Regression Analysis of Predictors of Perceived Competence to Initiate Clozapine Treatment

Predictor	B	SE	Wald χ^2	p	Odds Ratio	95% CI for OR
Years of professional experience (continuous)	0.23	0.11	4.17	0.041	1.26	1.01-1.57
Outpatient workload	0.22	0.63	0.12	0.727	1.24	0.36-4.28
Perceived adequacy of institutional support	-2.95	1.16	6.47	0.011	0.05	0.01-0.52
Difficulty managing medication side effects	1.12	0.87	1.65	0.199	3.06	0.56-16.62
Difficulty referring to inpatient care	1.47	0.78	3.59	0.058	4.35	0.95-19.94

Note. Outpatient workload was dichotomized as <=100 versus >100 patients per week. Perceived adequacy of institutional support was coded as 0 = adequate and 1 = inadequate. Difficulty managing medication side effects and difficulty referring patients to inpatient psychiatric care were coded as 0 = no and 1 = yes.

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[Abstract:0122]

Parental Burnout is Related to Attention-Deficit/Hyperactivity Disorder Symptoms in a Large Sample of Turkish Mothers

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Background and Objective: Parental burnout is a growing concern in modern life. Maternal ADHD symptoms may cause difficulties in managing daily parenting demands, leading to burnout in parental role. We examined whether ADHD symptoms in mothers predict parental burnout beyond psychological distress and family-related factors.

Methods: We applied an online survey including sociodemographic characteristics and relevant measurement tools for ADHD symptoms, parental burnout, and psychological distress among Turkish mothers from community without ADHD diagnosis (N = 674).

Results: Parental burnout showed significant correlations with maternal ADHD symptoms (r = .42, p < .001) and psychological distress (r = .66, p < .001). ADHD symptoms were also correlated with psychological distress (r = .45, p < .001). A partial Spearman correlation controlling for all sociodemographic, maternal health, psychological distress, and child-related factors indicated that ADHD symptoms remained significantly associated with parental burnout, although attenuated (r = .19, p < .001). A multiple regression model explained 47.0% of the variance in burnout (Adjusted R^2 = .46), with psychological distress emerging as the strongest predictor (β = .60, p < .001). Maternal ADHD symptoms remained an independent predictor (β = .09, p = .006), alongside the number of children (β = .13, p < .001) and having a child with a neurodevelopmental condition (β = .10, p = .002). Bootstrap confidence intervals supported the robustness of effects.

Conclusions: Findings highlight that parental burnout in Turkish mothers is related to ADHD symptoms independently from psychological distress and family-related factors. Psychological well-being of mothers is essential for families, while undiagnosed and untreated ADHD in women may be important to consider.

Keywords: Attention-deficit/hyperactivity disorder, family, maternal health, parenting, parental burnout

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[Abstract:0129]

Caffeine Use Disorder and Attention-Deficit/Hyperactivity Disorder Symptoms in Medical Students: A Cross-Sectional Study

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Background and Objective: Attention Deficit Hyperactivity Disorder (ADHD) is increasingly recognized among university and medical students, influencing academic performance and lifestyle. Caffeine, the most consumed psychoactive substance globally, is often used to enhance alertness and concentration. However, its relationship with ADHD symptoms remains controversial. While some studies suggest caffeine may improve attention and executive functions by modulating dopaminergic activity others report no significant benefit and even a potential link to caffeine use disorder in individuals with ADHD. This study aimed to investigate the association between coffee consumption and ADHD symptoms in medical students, and to examine whether caffeine use disorder (CUD) contributes independently to ADHD symptom severity.

Methods: This cross-sectional study included 210 medical students aged 17–32 years. Participants were grouped according to ADHD presence based on the Adult ADHD Self-Report Scale (ASRS v1.1). Data on coffee, tea, energy drink, and caffeinated soft drink consumption were collected. The Caffeine Use Disorder Scale (CUDS) assessed caffeine-related behavioral dependence. Descriptive statistics, correlation analyses, and multivariable linear regression were conducted controlling for age, sex, study hours, and sleep duration. Ethical approval was obtained, and informed consent was collected.

Results: Students with ADHD reported significantly higher consumption of coffee ($p = 0.029$), energy drinks ($p = 0.031$), and caffeinated carbonated beverages ($p = 0.017$) compared to those without ADHD. Mean daily study duration was shorter in the ADHD group ($p = 0.007$) (Table 1). CUDS and ASRS total scores showed a positive moderate correlation ($r = 0.338$, $p < 0.001$). Coffee consumption correlated weakly but significantly with total ASRS ($r = 0.139$, $p = 0.045$) and inattention subscale scores ($r = 0.166$, $p = 0.016$). In regression analysis, CUDS remained an independent predictor of ASRS total score ($\beta = 0.206$, $p = 0.011$), after adjusting for demographic and behavioral variables, while ADHD diagnosis was the strongest predictor ($\beta = 1.477$, $p < 0.001$). The model showed strong explanatory power (Adjusted $R^2 = 0.476$) (Table 2).

Conclusions: Medical students with ADHD symptoms exhibit greater caffeine intake and higher risk of caffeine use disorder than their non-ADHD peers. Although mild caffeine consumption may transiently improve attention, excessive or dependency-level use appears linked with elevated ADHD symptom burden, consistent with previous reports among adult. These findings underscore the importance of monitoring caffeine use patterns in students with attentional difficulties and raising awareness about potential behavioral dependence. Further longitudinal studies are warranted to clarify causal mechanisms and optimal caffeine intake thresholds in ADHD populations.

Keywords: Attention-Deficit/Hyperactivity Disorder, Caffeine Consumption, Caffeine Use Disorder, Medical Students

Table 1: Sociodemographic and clinical variables

	Control	ADHD	p value
Number	165	45	
Age (mean±SD)	21.6 ± 2.41 min-max: 17-32	21.8 ± 2.26 min-max: 18-28	0.679
Gender (f/m)	109 / 56 (66.1% / 33.9%)	32 / 13 (71.1% / 28.9%)	0.523
Class (median; IQR)	4.00; 4.00 min-max: 1-6	4.00; 4.00 min-max: 1-6	0.648
Study hours (median; IQR)	2.00; 1.00 min-max: 1-5	2.00; 1.00 min-max: 1-4	0.007*
Sleep duration (median; IQR)	4.00; 2.00 min-max: 1-7	4.00; 2.00 min-max: 2-7	0.841
Coffee consumption (median; IQR)	1.00; 2.00 min-max: 0-5	2.00; 2.00 min-max: 0-4	0.029*
Tea consumption (median; IQR)	1.00; 2.00 min-max: 0-4	1.00; 3.00 min-max: 0-3	0.300
Energy drink consumption (median; IQR)	1.00; 0.00 min-max: 1-3	1.00; 1.00 min-max: 1-3	0.031
Caffeinated soft drink consumption (median; IQR)	2.00; 2.00 min-max: 1-4	2.00; 2.00 min-max: 1-4	0.017*
Caffeine related insomnia (median; IQR)	2.00; 2.00 min-max: 1-5	2.00; 2.00 min-max: 1-5	0.966
CUDS (median; IQR)	3.00; 8.00 min-max: 0-23	9.00; 11.00 min-max: 0-23	<0.001*
ASRS Total (mean± SD)	27.2 ± 7.69 min-max: 3-47	44.8 ±8.98 min-max: 29-68	<0.001*
ASRS Inattentiveness (mean ± SD)	13.9 ±4.38 min-max: 1-26	23.3 ± 5.22 min-max: 16-36	<0.001*
ASRS Hyperactivity/impulsivity (mean ±SD)	13.4 ±4.89 min-max: 0-31	21.5 ±5.38 min-max: 11-32	<0.001*

*SD: standard deviation, IQR: interquartile range, ADHD: Attention Deficit Hyperactivity Disorder, f/m: female/male, CUDS: Caffeine Use Disorder Scale, ASRS: Adult ADHD Self-Report Scale For variables following a parametric distribution, the independent samples t-test was used, and mean and SD values were given. For variables not following a parametric distribution and ordinal variables, the Mann Whitney U test was used, and median and IQR values were given. For categorical variables, the chi-square test was used, and percentiles were given. Ordinal variables were coded as ordered categories where higher values indicate higher frequency/amount. * $p < 0.05$ is considered statistically significant.*

Table 2: Multiple linear regression analysis predicting ASRS total scores

Predictor	**B(Estimate)**	**95% CI**	**SE**	**β (Beta)**	**p**
(Intercept)	35.220	-	0.845	-	<0.001
Male gender	-1.772	[-4.168, 0.623]	1.303	-0.165	0.175
Age	-0.485	[-0.950, -0.019]	0.260	-0.107	0.063
Study hours	-0.329	[-1.646, 0.989]	0.691	-0.026	0.635
Sleep duration	0.141	[-0.799, 1.018]	0.532	0.015	0.791
Coffee	-0.801	[-1.938, 0.336]	0.563	-0.094	0.156
Tea	-0.778	[-1.687, 0.131]	0.474	-0.087	0.102
Energy drink	-0.579	[-2.999, 1.840]	1.269	-0.025	0.648
Caffeinated soft drink	0.640	[-0.520, 1.799]	0.579	0.059	0.270
Caffeine related insomnia	-0.153	[-1.203, 0.898]	0.610	-0.015	0.803
CUDS	0.361	[0.112, 0.611]	0.140	0.206	0.011*
ADHD group	15.885	[13.085, 18.684]	1.589	1.477	<0.001*

*Model fit: $F(11, 198) = 18.2$, $p < 0.001$, Adjusted $R^2 = .476$. No multicollinearity (VIF = 1.05–2.07); homoscedasticity confirmed (Breusch–Pagan p =.892); normality of residuals confirmed (Kolmogorov-Smirnov p = 0.981). CI: Confidence interval, ADHD: Attention Deficit Hyperactivity Disorder, CUDS: Caffeine Use Disorder Scale, ASRS: Adult ADHD Self-Report Scale * p < 0.05 is considered statistically significant.*

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[Abstract:0140]

Gender-Based Assessment of Academic Procrastination, Diagnosis and Cognitive Performance in Children

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Background and Objective:

Academic procrastination behavior has been shown to be common in children and adolescents, as in all age groups, and to be associated with various negative outcomes. Academic procrastination includes difficulties related to preparing term papers, studying for examinations, and completing homework on time. In this study, hypotheses were formulated based on the assumptions that psychiatric problems would be more frequent among children and adolescents with academic procrastination behavior; that differences might be observed in groups exhibiting symptoms of attention-deficit/hyperactivity disorder (ADHD) and depression/anxiety; and that neuropsychological test performance might be impaired in both groups.

Methods:

The sample consisted of patients aged 10–18 years who presented for the first time to the Child and Adolescent Psychiatry Outpatient Clinic of Antalya Training and Research Hospital and were evaluated using a semi-structured clinical interview. The primary aim was to assess the frequency of academic procrastination behavior and neuropsychological test performance in children and adolescents without diagnoses of intellectual disability or specific learning disorder. Ethical approval for the study was obtained from the Ethics Committee of Antalya Training and Research Hospital (Approval No: 2025-446).

Results:

A total of 98 children were included in the study, comprising 54 girls (55.1%) and 44 boys (44.9%). When compared by gender, girls had significantly higher age and duration of education than boys. No difference was found between genders when comparing academic procrastination scale scores. Analysis of the Conners Rating Scale subscale scores revealed that cognitive problems, oppositional behavior, and hyperactivity scores were significantly higher in boys. When the Child and Adolescent Depression Inventory subscale scores were compared, panic disorder subscale scores were significantly higher in girls. In terms of Stroop Test sub-results, the color-naming factor score was significantly higher in boys compared to girls. A significant positive correlation was found between Stroop Test Part 3 completion times and the oppositional behavior, hyperactivity, and cognitive problems subscales of the Conners Rating Scale. Additionally, a significant positive correlation was observed between the total depression scale score and both age and years of education. Linear regression analysis revealed that academic procrastination scale scores were significantly predicted by cognitive problem scores, generalized anxiety, and major depression subscale scores. Gender was not found to be a significant predictor.

Conclusions:

The findings of this study demonstrate the effects of gender on cognitive problems, oppositional behaviors, and hyperactivity symptoms. In addition to the higher frequency of these symptoms in boys, the elevated color-naming factor scores on the Stroop Test indicate poorer neurocognitive performance in male participants. These results suggest that gender may be associated with certain subdomains of cognitive symptoms. Further investigation with larger sample sizes is warranted to clarify these associations.

Keywords: Cognitive impairment, child and adolescent, conduct disorder, attention deficit hyperactivity disorder, depression

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[Abstract:0142]

Mental Health Literacy, Depression, Perceived Stress, and Psychological Help-Seeking During Pregnancy

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Objective

Depressive symptoms experienced during pregnancy may lead to adverse outcomes for maternal mental health and child development and are associated with various psychological and behavioral factors. Mental health literacy, perceived stress, and attitudes toward seeking psychological help may play an important role in shaping pregnant women's help-seeking behaviors. Previous studies have reported that lower levels of mental health literacy and higher levels of depressive symptoms are associated with more negative attitudes toward seeking psychological help. The aim of this study was to examine the relationships between mental health literacy, depressive symptoms, perceived stress, and attitudes toward seeking psychological help among pregnant women.

Methods

This cross-sectional study was conducted with 114 pregnant women aged between 18 and 40 years (M = 29.48 ± 4.75) who attended the Obstetrics and Gynecology Outpatient Clinic of Rize State Hospital. Data were collected using the Mental Health Literacy Questionnaire–Short Form for Adults (MHL), the Perceived Stress Scale (PSS), the Edinburgh Postnatal Depression Scale (EPDS), the Mental Help-Seeking Attitudes Scale (MHSAS), and a personal information form. Descriptive statistics, Pearson correlation analysis, and multiple linear regression analysis were used to analyze the data.

Results

The mean scores were 69.53 (SD = 9.85) for mental health literacy, 35.57 (SD = 7.58) for perceived stress, 7.25 (SD = 5.00) for depressive symptoms, and 50.42 (SD = 7.33) for attitudes toward seeking psychological help. Mental health literacy was positively and significantly correlated with attitudes toward seeking psychological help ($r = .34$, $p < .001$), whereas no significant relationship was found between mental health literacy and depressive symptoms or perceived stress. Depressive symptoms were positively correlated with perceived stress ($r = .67$, $p < .001$) and negatively correlated with attitudes toward seeking psychological help ($r = -.39$, $p < .001$). Perceived stress was also negatively associated with attitudes toward seeking psychological help ($r = -.21$, $p = .022$). Multiple linear regression analysis indicated that mental health literacy ($\beta = .30$, $p < .001$) and depressive symptoms ($\beta = -.41$, $p < .001$) were significant predictors of attitudes toward seeking psychological help, explaining 24.5% of the variance in the model. Perceived stress was not found to be a significant predictor ($\beta = .08$, $p = .476$).

Conclusions

The findings of this study demonstrate that attitudes toward seeking psychological help among pregnant women are significantly associated with mental health literacy and depressive symptoms. Higher levels of depressive symptoms were associated with more negative attitudes toward help-seeking, whereas higher mental health literacy was associated with more positive attitudes. These results suggest that interventions aimed at improving mental health literacy and identifying and addressing depressive symptoms at an early stage during pregnancy may strengthen positive attitudes toward seeking professional psychological help.

Keywords: Mental health literacy, depression, perceived stress, psychological help-seeking, pregnancy

[Abstract:0148]

Theory of Mind, Alexithymia, and Perceived Loneliness in Adolescents with Attention-Deficit/Hyperactivity Disorder

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Background and Objective: Adolescence is a critical developmental period for social cognition and emotional processing. Difficulties in theory of mind, alexithymia, and perceived loneliness have been associated with various psychiatric conditions. In adolescents with Attention-Deficit/Hyperactivity Disorder (ADHD), difficulties in social-cognitive functioning, emotional awareness, and peer relationships are commonly reported and may contribute to psychosocial impairment. This study aimed to examine theory of mind abilities, alexithymia, and perceived loneliness in adolescents with ADHD and to compare these domains with those of healthy peers, as well as to explore associations among these variables within each group.

Methods: The sample consisted of 49 adolescents diagnosed with ADHD according to the DSM-V diagnostic criteria and age- and sex-matched 34 healthy controls, all aged between 12 and 18 years. Socioeconomic status was assessed using the Hollingshead–Redich Scale, which evaluates parental education and occupational status. All participants were assessed using the Kiddie-Schedule for Affective Disorders and Schizophrenia for School Age Children-Present and Lifetime Version DSM-5 (K-SADS-PL). The healthy control group had no current or lifetime psychiatric diagnoses. Theory of mind was assessed using the Reading the Mind in the Eyes Test (RMET), alexithymia with the Toronto Alexithymia Scale (TAS-20), and perceived loneliness with the UCLA Loneliness Scale. All statistical analyses were performed using SPSS, with the level of statistical significance set at $p < .05$.

Results: Adolescents with ADHD demonstrated significantly lower RMET scores compared to healthy controls ($Z = -2.232$, $p = .026$), indicating poorer theory of mind abilities. The ADHD group showed significantly higher scores on all TAS-20 subscales (all $p < .001$), reflecting elevated levels of alexithymia. Perceived loneliness was also markedly higher in the ADHD group ($Z = 6.544$, $p < .001$). Correlation analyses revealed no significant associations between TAS-20, UCLA Loneliness, and RMET scores within the ADHD group (all $p > .05$). In contrast, in the healthy control group, TAS-20 total scores were significantly correlated with UCLA total scores ($r = .495$, $p = .002$), whereas RMET scores were not significantly associated with either TAS-20 or UCLA scores.

Conclusions: The findings indicate that adolescents with ADHD exhibit impairments in theory of mind, elevated levels of alexithymia, and increased perceived loneliness compared to healthy peers. Notably, the absence of significant associations between theory of mind, alexithymia, and loneliness within the ADHD group may indicate a disruption in the typical interplay between emotional awareness, social-cognitive processing, and subjective social experiences. In contrast, the significant association between alexithymia and loneliness observed in healthy adolescents suggests that, under typical developmental conditions, difficulties in emotional awareness are closely linked to feelings of social isolation. The lack of such relationships in adolescents with ADHD may reflect a more generalized pattern of social-emotional dysfunction, where multiple impairments coexist independently rather than interacting in a coherent manner. Integrating social-cognitive and emotional processing targets into clinical assessment and intervention programs may enhance psychosocial outcomes for adolescents with ADHD.

Keywords: Alexithymia, Attention-Deficit/Hyperactivity Disorder, Loneliness, Theory of mind

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[Abstract:0167]

Astrocytic Extracellular Matrix Proteins in ADHD: The Role of Thrombospondin-1, Sparc, and Sparcl-1

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Background and Objective:

Attention deficit hyperactivity disorder (ADHD) is a prevalent neurodevelopmental disorder characterized by persistent inattention, hyperactivity, and impulsivity, frequently accompanied by executive dysfunction and social impairment. Beyond classical dopaminergic models, accumulating genetic, neuroimaging, and experimental evidence indicates that altered synaptic development, disrupted glutamatergic signaling, and excitation–inhibition imbalance contribute to ADHD pathophysiology. These findings suggest that abnormalities in circuit maturation and synaptic organization may play an important role in the disorder.

Astrocytes regulate synapse formation, stabilization, pruning, and plasticity through the secretion of extracellular matrix (ECM) proteins. Thrombospondin-1 (Tsp-1), Sparc, and Sparcl-1 are key astrocyte-derived ECM proteins involved in excitatory synaptogenesis, dendritic spine maturation, and glutamatergic network refinement. Although these proteins have been increasingly investigated in neurodevelopmental disorders, their relevance to ADHD remains insufficiently understood, representing an important mechanistic gap in the field.

This study aims to evaluate serum levels of Tsp-1, Sparc, and Sparcl-1 in children with ADHD and to examine their associations with behavioral symptoms, executive dysfunction, and social impairments. By integrating molecular biomarker data with detailed behavioral phenotyping, the study seeks to provide further insight into glial contributions to ADHD pathophysiology and the relationship between astrocyte-derived ECM signaling and glutamatergic function.

Methods:
Eighty-six children aged 6–12 years were enrolled, including 46 medication-naïve children diagnosed with ADHD according to DSM-5 criteria and 40 age- and sex-matched neurotypical controls. The Kiddie Schedule for Affective Disorders and Schizophrenia–Present and Lifetime Version (K-SADS-PL) was administered to confirm diagnoses and exclude comorbid psychiatric disorders. Behavioral symptoms were assessed using the Conners' Parent Rating Scale–Revised: Long Form (CPRS-R:L), executive functioning with the Behavior Rating Inventory of Executive Function (BRIEF) Parent Form, and social functioning with the Social Responsiveness Scale (SRS). Fasting morning venous blood samples were collected, and serum concentrations of Tsp-1, Sparc, and Sparcl-1 were quantified using enzyme-linked immunosorbent assay (ELISA). Group comparisons and correlations were analyzed using appropriate statistical methods.

Results:
Serum levels of Sparc ($p = 0.020$), Sparcl-1 ($p = 0.049$), and Thrombospondin-1 ($p < 0.001$) were significantly lower in the ADHD group compared with controls. Within the ADHD group, Tsp-1 levels showed significant positive associations with CPRS-R:L subscales, including Oppositional Behavior, Psychosomatic Symptoms, and the ADHD Index (all $p < 0.05$). Sparc levels demonstrated significant negative associations with executive function domains, including inhibition, planning, and self-regulation (all $p < 0.05$). Additionally, children with Social Responsiveness Scale scores above the clinical cutoff exhibited significantly lower Sparc levels than those below the cutoff ($p = 0.019$).

Conclusions:
This study provides the first evidence of reduced serum astrocyte-derived ECM proteins in children with ADHD, implicating glia-mediated synaptic dysregulation in the disorder's pathophysiology. These findings suggest a novel glia-centered mechanism and support the potential of ECM proteins as biomarkers for ADHD subtypes and as targets for future therapeutic interventions.

Keywords: ADHD, astrocytes, ECM, Thrombospondin-1, Sparc, Sparcl-1

AuthorToEditor: Nilüfer YILDIZ KISMET: ORCID: 0009-0000-6664-6486 Selen SEZEN GÖKÇEARSLAN: ORCID: 0009-0008-2919-9347 Serenay ELGÜN ÜLKAR: ORCID: 0000-0002-1751-090X Sadettin Burak AÇIKEL: ORCID: 0000-0002-8964-9513 This study received approval from the Ethics Committee of Ankara University Faculty of Medicine (Ethics approval number: i11-778-23) and was supported by the Ankara University Scientific Research Projects Coordination Unit (Grant No. TTU-2024-3526). The protocol adhered to the principles of the Helsinki Declaration.

[Abstract:0171]

Psychometric Properties of the Turkish Version of the Bipolar Self-Efficacy Scale (BPSES)

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Background and Objective: Self-efficacy refers to individuals' beliefs about their capacity to organize and execute behaviors required to achieve specific goals. In bipolar disorder, effective illness management necessitates sustained engagement in self-management tasks such as maintaining medication routines, detecting early warning signs of mood episodes, implementing behavioral and lifestyle adjustments, and seeking appropriate psychiatric support. Preliminary research suggests that individuals with bipolar disorder may exhibit reduced confidence in performing these tasks, reflecting lower self-efficacy. However, instruments commonly used in clinical and research settings assess general self-efficacy and do not capture disorder-specific domains of illness management. This measurement gap limits the capacity to evaluate how individuals perceive their ability to influence disorder-related trajectories. To address this need, the Bipolar Self-Efficacy Scale (BPSES) was developed as a disorder-specific measure to assess perceived efficacy in managing bipolar illness. The present study examined the psychometric properties of the Turkish version of the BPSES in individuals with bipolar disorder in clinical remission.

Methods: A total of 141 patients diagnosed with bipolar disorder, under psychiatric follow-up at Silifke State Hospital, and in clinical remission for at least eight weeks were included. Alongside sociodemographic data, participants completed the Bipolar Self-Efficacy Scale (BPSES), the Altman Self-Rating Mania Scale, the Adult Depression Severity Scale, and the General Self-Efficacy Scale – Turkish Form. Psychometric analyses included item–total correlations to assess item discrimination, exploratory factor analysis (EFA) and confirmatory factor analysis (CFA) to examine dimensionality and construct validity, and internal consistency analyses (Cronbach's alpha) to evaluate reliability. Convergent validity was assessed through correlations between BPSES total scores and scores on the General Self-Efficacy Scale, the Altman Self-Rating Mania Scale, and the Adult Depression Severity Scale. Ethical approval was obtained from the Toros University Health Sciences Ethics Committee (01.12.2023/139). The study represents a pilot validation of the Turkish BPSES.

Results: Participants had a mean age of 33.87 years (SD = 9.64), and 70.2% were female. Item–total correlations ranged between 0.595 and 0.873, indicating adequate item discrimination. Exploratory factor analysis supported a unidimensional structure accounting for 65.51% of the variance, with factor loadings ranging from 0.601 to 0.889 (Bartlett's test of sphericity: $\chi^2 = 1339.55$, df = 136, $p < 0.001$). Internal consistency was excellent (Cronbach's $\alpha = 0.969$). Convergent validity analyses showed that BPSES total scores were positively correlated with general self-efficacy ($r = 0.287$, $p = 0.015$) and negatively correlated with depression severity ($r = -0.249$, $p = 0.035$). Correlation with mania severity did not reach statistical significance ($r = 0.082$, $p = 0.495$).

Conclusions: The Turkish version of the BPSES demonstrated strong psychometric properties and appears suitable for assessing disorder-specific self-efficacy in bipolar disorder. Findings highlight the relevance of evaluating illness-management capacities beyond general self-efficacy frameworks and support the potential use of the BPSES in clinical and research contexts to inform comprehensive care, psychoeducation, and treatment outcome assessment. It may facilitate disorder-specific clinical assessment. Further research with larger and more diverse samples is warranted to refine psychometric estimates and evaluate longitudinal and treatment-related sensitivity.

Keywords: Bipolar disorder, self-efficacy, psychometrics, validity, reliability

Figure 1. Scree Plot

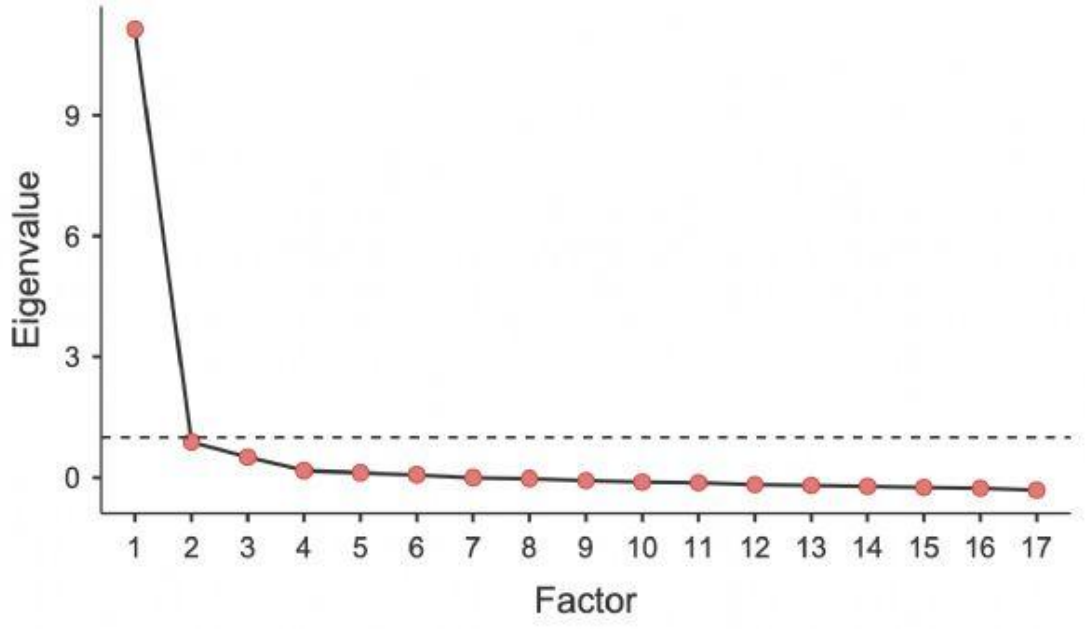


Table 1. Factor Loadings

	Factor 1	Uniqueness
item 1	0.823	0.322
item 2	0.829	0.312
item 3	0.802	0.356
item 4	0.782	0.389
item 5	0.818	0.330
item 6	0.717	0.485
item 7	0.854	0.270
item 8	0.831	0.310
item 9	0.889	0.210
item 10	0.851	0.276
item 11	0.746	0.444
item 12	0.793	0.371
item 13	0.818	0.330
item 14	0.794	0.369
item 15	0.876	0.233
item 16	0.886	0.215
item 17	0.601	0.639

Note. The minimum residual extraction method was used in conjunction with oblimin rotation.

Table 2. Item Reliability Statistics

	Mean	Standard Deviation	Item - Total Correlation	If Item Dropped Cronbach's α
item 1	7.66	3.14	0.812	0.967
item 2	6.58	3.18	0.818	0.967
item 3	7.20	3.30	0.792	0.967
item 4	7.31	3.28	0.772	0.967
item 5	6.90	3.37	0.809	0.967
item 6	6.11	3.36	0.708	0.968
item 7	6.44	3.39	0.838	0.967
item 8	6.36	3.31	0.813	0.967
item 9	5.83	3.43	0.873	0.966
item 10	6.02	3.41	0.832	0.967
item 11	4.62	3.29	0.735	0.968
item 12	5.16	3.29	0.780	0.967
item 13	5.31	3.21	0.800	0.967
item 14	5.62	3.28	0.784	0.967
item 15	6.11	3.07	0.860	0.966
item 16	5.91	3.12	0.868	0.966
item 17	6.11	3.46	0.595	0.970

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[Abstract:0173]

Eating Problems in Children with ASD and Maternal Mental Health: The Mediating Role of Maternal Quality of Life

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Background: Childhood eating problems affect 20–30% of all children and up to 70–90% of those with neurodevelopmental disorders [1]. In autism spectrum disorder (ASD), feeding difficulties are typically more complex and persistent, with prevalence estimates up to 80% [2]. Even mild problems can distress children and caregivers, disrupt family routines, and increase stress, while severe difficulties may cause growth retardation and other health complications [3]. Yet comprehensive ASD-specific tools spanning multiple feeding domains, and studies testing how parental well-being relates to feeding difficulties remain limited in clinical contexts, including Türkiye. Validating a multidimensional measure while examining family-centered pathways may provide novel, clinically actionable evidence for assessment and intervention planning.

Objectives: This study aimed (1) to examine the psychometric properties of the Aut-Eat Questionnaire (AEQ)[4] in a clinical sample of children with ASD in Türkiye and (2) to investigate associations between children's eating problems, maternal quality of life, and maternal depression, including a mediation model.

Methods: The sample comprised 352 children with ASD (2–12 years) and their mothers, recruited from a tertiary child psychiatry outpatient clinic. Parents completed the AEQ, Brief Autism Mealtime Behavior Inventory (BAMBI), WHOQOL-BREF, and Hospital Anxiety and Depression Scale (HADS). A subsample of 18 parents completed the AEQ again after 2–3 weeks. Internal consistency, test–retest reliability, and confirmatory factor analysis (CFA) were used to evaluate the AEQ. Convergent validity was examined via correlations with BAMBI scores and weight percentiles. Mediation analysis (PROCESS, Model 4) tested whether maternal depression mediates the association between maternal QoL and children's eating problems, controlling for child gender, maternal age, maternal education, and ASD severity.

Results: The AEQ showed high internal consistency and excellent test–retest reliability. CFA supported the six-factor structure with acceptable fit indices. AEQ subscales correlated significantly with BAMBI scores, supporting convergent validity. Children's eating problems were positively associated with maternal anxiety and depression and negatively associated with maternal quality of life. Notably, maternal depression partially mediated the link between maternal quality of life and children's eating problems, indicating both direct and indirect effects.

Conclusions: The Turkish AEQ is a reliable and valid tool for assessing eating problems in children with ASD. By combining large-sample cross-cultural validation with a family-centered mediation model, this study offers a novel contribution to the literature and underscores the need for holistic interventions that address both child feeding difficulties and maternal mental health in psychiatric settings.

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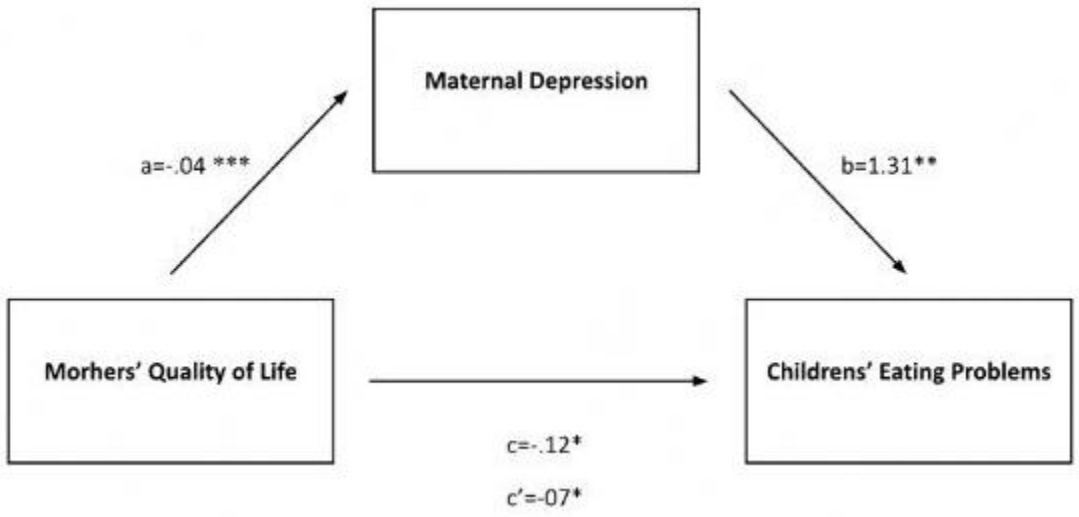
Keywords: Autism Spectrum Disorder, Aut-Eat Questionnaire (AEQ), Eating Problems, Maternal Depression Parental Quality of Life, Psychometric Validation

Results for Indirect Effects in the Mediation Model

	Coefficient of Path	%95 Confidence Interval Level	
		Lower limit of confidence interval	*Upper limit of confidence interval*
Direct Effects			
MQL→MD	-.04	-.045	-.036
MD→ CEP	1.31	.340	2.278
Indirect Effects			
MQL→MD→ CEP	-.05	-.091	-.015

MQL: Maternal Quality of Life, MD: Maternal Depression, CEP: Children's Eating Problem

The Model for Children's Eating Problems



AuthorToEditor: Uzmanlık tezi kapsamında, Prof. Dr. A. Cahid Örengül danışmanlığında yürütülen bu çalışmada, Otizm Spektrum Bozukluğu'nda (OSB) yeme problemlerini değerlendirmeye yönelik güncel ve kapsamlı bir ölçek olan AutEat Questionnaire (AEQ) Türkçeye uyarlanmış ve psikometrik özellikleri klinik örneklemde değerlendirilmiştir. Bu uyarlama ile Türkiye'de OSB'de yeme problemleri alanında yapılacak araştırma ve klinik değerlendirmelerde kullanılabilecek nitelikli bir ölçme aracı literatüre kazandırılmıştır. Ayrıca çalışma, OSB'de sık görülen yeme problemlerinin klinik önemine dikkat çekerek, tanı ve tedavi sürecinde çocuk ve ebeveyni birlikte ele alan bütüncül yaklaşımın gerekliliğini vurgulamaktadır. Büşra Nur Dambasan Gündüz https://orcid.org/0000-0003-2474-1287 Abdurrahman Cahid Orengul https://orcid.org/0000-0002-4720-6167

[Abstract:0175]

Investigation of Cognitive Function, Activities of Daily Living, and Depression in Adults Aged 65 Years and Older

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Background and Objective: Aging is a natural process that involves irreversible biological, psychological, and social changes accompanied by declining physiological reserve and adaptive capacity. Among individuals aged 65 years and older, cognitive impairment, depressive symptoms, and functional dependence are common. Previous studies have shown that lower Mini-Mental State Examination (MMSE) scores are associated with greater functional dependency, as measured by the Barthel Index, and with higher depression scores on the Geriatric Depression Scale (GDS)[1]. Depression and cognitive impairment frequently co-occur in late life and may mutually influence each other, with depressive symptoms potentially accelerating cognitive decline and cognitive impairment increasing vulnerability to depression through reduced autonomy and social withdrawal[2]. Research has also indicated that both cognitive impairment and depression negatively affect quality of life, partly through functional limitations[1]. However, their combined impact on functional independence remains unclear. This study aims to examine the relationships among cognitive status, depression level, and daily living activities in individuals aged 65 years and older using standardized assessment tools.

Methods: This descriptive, cross-sectional study was conducted over a three-month period with adults aged 65 years and older who applied to the Psychiatry Outpatient Clinic of Göztepe Prof. Dr. Süleyman Yalçın City Hospital. After providing informed consent, participants completed a sociodemographic form, the MMSE to assess cognitive functioning, the GDS to assess depressive symptoms, and the Barthel Index to evaluate activities of daily living. Participants with severe cognitive, psychiatric, or sensory impairments preventing assessment were excluded.Data were analyzed using descriptive statistics and correlation analyses to examine the associations among age, cognitive functioning, depressive symptoms, and functional status.

Results: The study sample included 92 older adults (mean age 74.6±6.73 years). The majority of participants were female (73.9%,n=68). Mean MMSE score was 28.0 (SD=2.93), indicating preserved cognition, mean GDS score was 6.5 (SD=5.86), indicating low to mild depressive symptom levels, mean Barthel Index score was 97.6 (SD=6.27), indicating high functional independence.

Correlation analyses showed that age was significantly and negatively associated with MMSE scores (ρ=-0.26, p=.012) and Barthel Index scores (ρ=-0.31, p=.002). Cognitive functioning was positively associated with functional independence (ρ=0.24, p=.021). Depressive symptoms were not significantly associated with age or functional status; however, a trend-level negative association was observed between GDS and MMSE scores (ρ=-0.20, p=.054). These results represent preliminary findings, as data collection is ongoing. Conclusions: The preliminary findings indicate that increasing age is associated with lower cognitive performance and reduced functional independence among older adults. The positive association between MMSE and Barthel Index scores suggests a close relationship between cognitive functioning and functional capacity, indicating that declines in daily functioning may be accompanied by poorer cognitive performance. These findings emphasize the importance of maintaining functional independence to support cognitive health. Although depressive symptoms were not significantly associated with functional status, the trend-level negative relationship with cognitive functioning is consistent with previous literature, suggests potential clinical relevance of subclinical symptoms, highlighting the need to consider depressive symptoms even below diagnostic thresholds[3]. Overall, these findings support the value of a multidimensional assessment approach integrating cognitive, emotional, and functional domains, suggesting that preserving functional activity and early identification of depressive symptoms may support healthy aging.

Keywords: Aging, Cognitive impairment, Depressive symptoms, Functional independence, Geriatric psychiatry

[Abstract:0184]

Examining The Mediating Role of Sleep Quality in The Relationship Between Impulsivity and Emotion Regulation Among Adolescents Referred to Child and Adolescent Psychiatry Clinics

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Background and Objective: Good-quality sleep is a fundamental component of a healthy life. While the relationship between sleep and overall health has long been recognized, its links with psychological functioning have received growing attention in recent years. Adolescence is a critical developmental period during which emotion regulation continues to mature and impulse control is challenged. Sleep disruptions during this stage may therefore adversely affect not only physiological well-being but also emotional and behavioral regulation. Increasing evidence indicates that poor sleep quality is associated with greater impulsivity and difficulties in emotion regulation (1–3). Accordingly, this study aims to examine the mediating role of sleep quality in the association between impulsivity and emotion regulation difficulties in adolescents referred to child and adolescent psychiatry clinics.

Methods: This cross-sectional study was conducted between November 10, 2025, and January 10, 2026, and included 119 adolescents aged 12–18 years who presented to the Child and Adolescent Psychiatry Outpatient Clinics of Uşak Training and Research Hospital, Kastamonu Training and Research Hospital, and Kızıltepe State Hospital. Participants completed the Brief Symptom Inventory (BSI), the Difficulties in Emotion Regulation Scale–Short Form (DERS-SF), the Pittsburgh Sleep Quality Index (PSQI), and the Barratt Impulsivity Scale–11 (BIS-11). Mediation analyses using structural equation modeling (SEM) were performed to examine whether sleep quality mediated the association between impulsivity and emotion regulation difficulties.

Results: The sample included 119 adolescents (M age = 15.3, SD = 1.6; 79.0% female). Impulsivity was associated with poorer sleep quality ($\beta = 0.22$, SE = 0.08, $p = .006$), and poorer sleep quality was associated with greater emotion regulation difficulties ($\beta = 0.28$, SE = 0.07, $p < .001$). Impulsivity also showed a significant direct association with emotion regulation difficulties ($\beta = 0.43$, SE = 0.07, $p < .001$). The indirect effect via sleep quality was small but significant ($\beta = 0.08$, SE = 0.04, $p = .050$), yielding a total effect of $\beta = 0.65$ (SE = 0.10, $p < .001$) (Figure 1, Table 1).

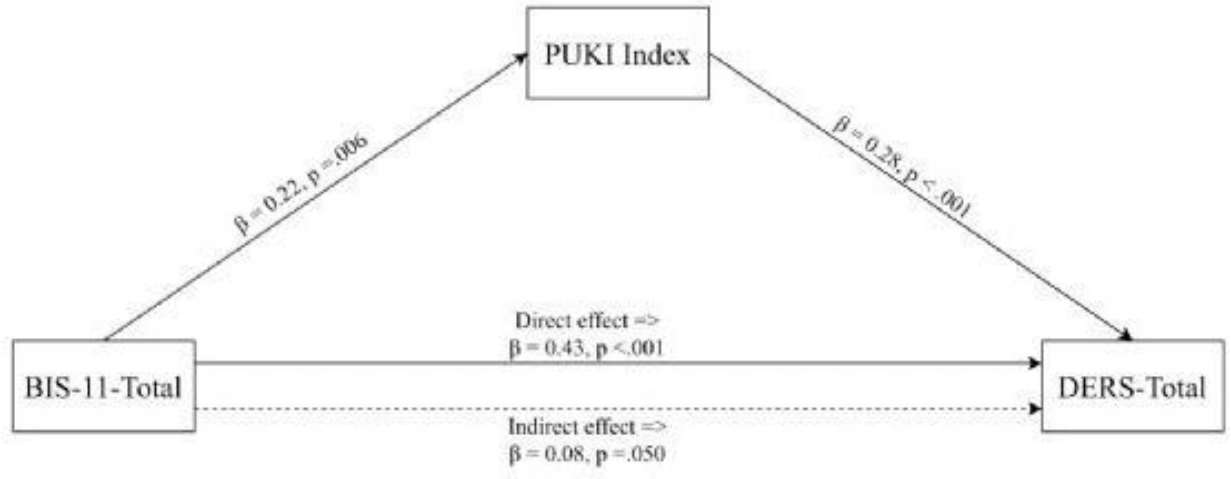
Conclusions: The findings suggest that sleep quality is partially associated with the relationship between impulsivity and emotion regulation difficulties. While impulsivity shows a strong association with emotion regulation, sleep disturbances may represent an additional factor linked to this relationship. Given the observed associations among sleep, impulsivity, and emotion regulation, sleep patterns should be considered in clinical assessments during this critical developmental period.

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Keywords: Adolescence, emotion regulation difficulties, impulsivity, sleep quality

Mediation model illustrating the direct and indirect associations between impulsivity, sleep quality, and emotion regulation difficulties



Direct, Indirect, and Total Effects of Impulsivity on Emotion Regulation Difficulties via Sleep Quality

Path	Standardized β	SE	z	P
Direct Effects				
BIS-11 → PSQI	0.22	0.08	2.73	.006
PSQI → DERS	0.28	0.07	3.73	<.001
BIS-11 → DERS	0.43	0.07	6.56	<.001
Indirect effect				
BIS-11 → PSQI → DERS	0.08	0.04	1.96	.050
Total effect				
BIS-11 → DERS	0.65	0.10	6.47	<.001

Note. Standardized path coefficients (β) are reported. Age and sex were included as covariates in all models. Mediation analyses were conducted using structural equation modeling (SEM) with robust standard errors, estimated via maximum likelihood with missing values (MLMV). Indirect, direct, and total effects were evaluated using nonparametric bootstrap procedures with 5,000 resamples. BIS-11 = Barratt Impulsiveness Scale–11; PSQI = Pittsburgh Sleep Quality Index (global score); DERS = Difficulties in Emotion Regulation Scale (total score).

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[Abstract:0191]

The Effect of Comorbid Autism Traits on Perceived Social Support, Loneliness and Suicide Risk in Adolescents Diagnosed with Social Anxiety Disorder

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Background and Objective: This study aimed to examine the effects of social anxiety severity on perceived social support, loneliness, and suicide risk in adolescents diagnosed with Social Anxiety Disorder (SAD), and to investigate these variables in the presence of co-occurring autism traits. By evaluating the impact of symptom severity in adolescents with SAD, the study emphasizes the importance of differential diagnosis due to overlapping features with autism traits.

Methods: 80 adolescents diagnosed with SAD according to DSM-5 criteria were included in the study. All participants completed the Liebowitz Social Anxiety Scale (LSAS). The adolescents also completed the Multidimensional Scale of Perceived Social Support (MSPSS), the UCLA Loneliness Scale–Short Form (UCLA-SF), and the Suicide Probability Scale (SPS). Associations between social anxiety and avoidance severity and these variables were analyzed. Parents of the participants completed the Autism Spectrum Quotient–Adolescent Version (AQ-A), and participants were divided into two groups based on the scale's cutoff score as those with and without autism traits.

Results: A significant positive correlation was found between LSAS scores and UCLA-SF scores, whereas no significant association was observed between LSAS scores and MSPSS total or subscale scores. LSAS scores were positively correlated with the hopelessness, suicidal ideation, and hostility subscales, as well as the total score of the SPS, while a significant negative correlation was observed with the negative self-evaluation subscale of the SPS. AQ-A total scores were negatively correlated with the significant other, friend, and total MSPSS scores. Additionally, the communication subscale and total scores of the AQ-A were positively correlated with loneliness. No significant associations were found between AQ-A subscales and SPS subscales or total scores. Adolescents with autism traits (n = 36) demonstrated significantly lower perceived social support from friends and significantly higher loneliness scores compared to those without autism traits.

Conclusions: The findings indicate a direct association between social anxiety symptom severity and loneliness, while perceived social support appears to play a more indirect role, potentially acting as a mediating factor in suicide risk. Autism traits were found to be relatively common among adolescents with SAD and were associated with perceived social support and loneliness, but not with suicide risk. Further studies with larger sample sizes are needed to better understand autism traits and related factors in adolescents with SAD.

Keywords: Social anxiety disorder, autism traits, loneliness, suicide, perceived social support

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[Abstract:0192]

Predictors of Smartphone Distraction: The Contributions of Emotion Regulation Difficulties, Anticipatory Pleasure, and ADHD Inattentive Symptoms

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Background and Objective: Smartphones are central to young adults' daily communication, academic work, and social interaction; however, frequent checking and persistent mental preoccupation may disrupt sustained attention. "Smartphone-distraction" can be conceptualized as difficulty maintaining task focus due to repeated attentional shifts and cognitive intrusions. Beyond the duration of use, individual psychological processes may contribute to this pattern. This study examined the incremental contributions of emotion regulation difficulties, pleasure dimensions, and ADHD-related symptoms to smartphone-related distraction after controlling for sociodemographic and clinical factors. We also explored whether observed gender differences would remain significant after accounting for these psychological variables.

Methods: A cross-sectional, online survey was conducted among university students at Uşak University between March and June 2025. Participants were recruited via official departmental social media groups and completed self-report measures through Google Forms. The final sample comprised 316 students aged 18–30 who provided informed consent. Ethics approval was obtained from the Usak University Non-Interventional Clinical Research Ethics Committee (decision no:590-590-03). Smartphone distraction was assessed with the Smartphone Distraction Scale(SDS). Emotion regulation difficulties were measured with the Difficulties in Emotion Regulation Scale(DERS); hedonic functioning with the Temporal Experience of Pleasure Scale(TEPS;anticipatory and consummatory subscales); and ADHD symptoms with the Adult ADHD Self-Report Scale (ASRS;inattentive and hyperactivity/impulsivity subscales). Hierarchical multiple regression tested predictors of SDS scores in four steps: (1)age and gender; (2)income and employment; (3)regular smoking and alcohol use; and (4)DERS, TEPS subscales, and ASRS subscales.

Results: Women reported higher SDS and DERS scores than men, and higher TEPS anticipatory and consummatory scores; ASRS scores did not differ by gender. In regression analyses, age and gender explained 1.7% of the variance in SDS (R^2=.017; model p=.070), with gender emerging as a significant predictor at this step. Adding income and employment did not significantly improve the model (ΔR^2=.007; p=.335), nor did adding smoking and alcohol use (ΔR^2=.012; p=.139). The addition of psychological variables produced a substantial and significant increase in explained variance (ΔR^2=.303; p<.001), yielding a final model explaining 33.9% of SDS variance (R^2=.339). In the final model, higher DERS, higher TEPS anticipatory pleasure, and higher ASRS inattentive symptoms were independently associated with higher smartphone-related distraction. In contrast, TEPS consummatory pleasure and ASRS hyperactivity/impulsivity were not significant predictors. Notably, the effect of gender became non-significant after psychological variables were included, suggesting that initial gender differences were largely accounted for by underlying emotion regulation, reward expectancy, and inattentive symptom dimensions.

Conclusions: This study indicates that, among university students, smartphone-related distractibility can be accounted for only to a limited extent after controlling for sociodemographic variables; by contrast, the phenomenon appears to be meaningfully linked to underlying psychological processes. In the fully adjusted model, difficulties in emotion regulation, elevated anticipatory pleasure, and symptoms reflecting the inattentive dimension of ADHD independently predicted smartphone-related distractibility. In contrast, consummatory pleasure and hyperactivity/impulsivity symptoms did not provide additional, specific explanatory value. Accordingly, preventive and intervention programs for university populations should prioritize strengthening emotion regulation skills,

managing impulsive reward-seeking tendencies related to reward anticipation, and supporting sustained attentional control.

Keywords: Anticipatory pleasure, Emotion regulation difficulties, Inattentive symptoms, Smartphone distraction, University students

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[Abstract:0198]

Neuroepigenetic-Regulating Long Non-Coding RNA Polymorphisms in Newly Diagnosed Psychosis

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Background and Objective

Schizophrenia is a multifactorial neuropsychiatric condition shaped by genetic vulnerability, immune imbalance, and epigenetic dysregulation. Recent studies highlight the importance of neuroinflammation and disrupted gene-expression control in early disease mechanisms. Long non-coding RNAs (lncRNAs) act as key regulators of transcription through chromatin remodeling, enhancer modulation, and post-transcriptional processes, influencing synaptic plasticity, neurodevelopment, and inflammatory signaling. Genetic variants within these regulatory regions may therefore alter neuronal and immune functions, contributing to psychosis susceptibility. However, the role of neuroepigenetic lncRNA polymorphisms in early-stage psychosis remains poorly defined. Investigating newly diagnosed, first-episode patients prior to prolonged medication exposure may better reflect primary genetic effects. This study aimed to evaluate the distribution of neuroepigenetic-regulating lncRNA polymorphisms in newly diagnosed psychosis and to explore their potential functional and clinical relevance.

Methods

Peripheral blood samples were obtained from newly diagnosed, first-episode psychosis/schizophrenia patients and demographically matched healthy controls recruited at Aydın Adnan Menderes University. A total of 150 individuals were included, comprising 70 newly diagnosed psychosis/schizophrenia patients and 80 healthy controls. Genomic DNA was isolated using standard extraction methods. Genotyping was performed using the Fluidigm SNPtype™ platform, a microfluidic real-time PCR–based system that enables accurate and high-throughput allele discrimination.

The following lncRNA polymorphisms related to neuroepigenetic and transcriptional regulation were analyzed: CCAT2 (rs6983267), CCAT1 (rs67085638), CASC8 (rs7014346), PVT1 (rs13281615), HULC (rs7763881), and LINC01430 (rs6017342). Genotypic and allelic frequencies were compared between groups. Dominant, recessive, and additive inheritance models were tested to identify risk patterns. Variants with significant associations were further examined using in-silico functional annotation and expression quantitative trait loci (eQTL) databases to assess their potential regulatory and transcriptional effects.

Results

The groups were comparable in age, sex, education, and marital status ($p > 0.05$).

Significant differences in genotype distributions between patients and controls were observed for rs13281615, rs67085638, rs6983267, rs7014346, and rs7763881 (all $p <= 0.05$), whereas rs6017342 showed no significant association. Inheritance model analyses demonstrated enrichment of recessive risk, particularly at rs13281615, rs7014346, and rs6983267. Functional annotation indicated that most associated variants were located within regulatory regions and were predicted to exert transcriptional or eQTL-like effects.

Conclusions

Neuroepigenetic-regulating lncRNA polymorphisms appear to constitute a coordinated regulatory signature associated with susceptibility to psychosis in newly diagnosed individuals. These findings support the involvement of gene-expression dysregulation and epigenetic mechanisms in early disease pathogenesis. Regulatory lncRNA variants may serve as promising biomarkers and potential targets for early diagnosis and precision psychiatry approaches.

Keywords: Epigenetic, lncRNA Polymorphisms, inflammation, schizophrenia

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[Abstract:0199]

Relationship Between Maternal Emotion Regulation Difficulties, Feeding Practices, and Preschool Malnutrition: A Cross-Sectional Comparison

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Background and Objective: Malnutrition remains a major public health problem worldwide, particularly among preschool-aged children, and is associated with increased morbidity, mortality, and long-term neurodevelopmental consequences. Beyond biological factors, early childhood malnutrition is closely influenced by psychosocial determinants, including parental feeding practices, caregiver mental health, and parent–child interaction patterns. Feeding in early childhood represents a dynamic relational process shaped by children's developmental characteristics and caregivers' emotional and behavioral responses. However, studies simultaneously examining children's eating behaviors together with maternal emotion regulation abilities and parental acceptance–rejection patterns in malnourished preschool children remain limited.This study aimed to investigate the relationship between maternal emotion regulation difficulties, parental acceptance–rejection behaviors, and eating behavior problems in malnourished preschool children compared with healthy peers.

Methods: This cross-sectional, comparative study included 118 children aged 36–72 months. The malnutrition group consisted of 60 children diagnosed with malnutrition by a pediatric endocrinologist at the Department of Pediatric Endocrinology, Ankara Bilkent City Hospital. Fifty-eight age- and sex-matched healthy children without medical or psychiatric diagnoses constituted the control group. Children with chronic medical conditions, autism spectrum disorder, or developmental delay were excluded.

Psychiatric evaluations were conducted by the researcher using DSM-5–based clinical interviews. Mothers completed the Early Childhood Adaptive Eating Behavior Scale (ECAEBS), Maternal Attitudes Toward the Feeding Process Scale (MATFPS), Difficulties in Emotion Regulation Scale–Short Form (DERS-16), Parental Acceptance–Rejection Questionnaire (PARQ), Brief Symptom Inventory (BSI), Strengths and Difficulties Questionnaire (SDQ), and Spousal Support Scale. Anthropometric measurements were recorded for all participants. Statistical analyses included group comparisons, correlation analyses, and multiple linear regression models.

Ethical approval was obtained prior to the initiation of the study. All participants and their parents were fully informed about the study, and written informed consent was obtained before enrollment.

Results: Children with malnutrition had significantly lower birth weights and shorter breastfeeding durations compared with controls. Eating behavior problems—including food refusal, sensory-based selectivity, and aggressive or resistant behaviors during meals—were significantly more frequent in the malnutrition group. Emotional and behavioral problems and attention-deficit/hyperactivity symptoms were also more prevalent; 63.3% of malnourished children met criteria for at least one psychiatric diagnosis, with Avoidant/Restrictive Food Intake Disorder being the most common.

Mothers of malnourished children demonstrated significantly higher levels of anxiety, depression, somatization, and emotion regulation difficulties. While no group difference was observed in the warmth/affection subscale of the PARQ, hostility, neglect, and rejection scores were significantly higher in the malnutrition group. Perceived spousal support was significantly lower among these mothers. Regression analyses revealed that negative maternal feeding practices were the strongest predictor of children's eating behavior problems.

Conclusions: Preschool malnutrition is closely associated with maternal emotion regulation difficulties, adverse parental attitudes, and problematic feeding interactions. These findings emphasize that malnutrition in early childhood should be evaluated not only as a nutritional condition but also within a psychosocial and relational

framework. Integrating parental mental health assessment, feeding guidance, and family-based interventions into malnutrition management may improve both nutritional outcomes and child mental health. Longitudinal studies with larger samples are warranted to clarify causal pathways.

Keywords: Eating Behavior, Emotion Regulation, Malnutrition, Parental Acceptance–Rejection, Preschool Children

AuthorToEditor: This study was conducted as part of the author's medical specialty thesis. Ethical approval and written informed consent were obtained prior to participation. Corresponding author (first author) ORCID ID: 0000-0002-7828-1194

[Abstract:0200]

The Structural Framework of Anhedonia: A Transdiagnostic Analysis of Striato-Cortical Circuits

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Objective: Anhedonia is a core symptom shared across various psychiatric diagnoses, including Major Depressive Disorder, Bipolar Disorder, and Schizophrenia. Given the limitations of traditional categorical boundaries in capturing shared underlying mechanisms, this study seeks to identify structural biomarkers that contribute to pleasure-processing deficits. In alignment with the NIMH-RDoC framework, this study aims to investigate the structural correlates of anhedonia severity within striato-cortical reward circuits through a dimensional approach, utilizing an open-access dataset.

Methods: Data from 195 participants (including various psychiatric diagnoses and healthy controls) from the "Transdiagnostic Connectome Project" (TCP) available on the OpenNeuro database were analyzed. Anhedonia severity was assessed using the Temporal Experience of Pleasure Scale (TEPS; measuring both anticipatory and consummatory pleasure). High-resolution T1-weighted MRI images were processed via the volBrain (v1.0) automated segmentation pipeline. To eliminate variations in individual head size, all volumetric data were normalized to Total Intracranial Volume (TIV ratios). Outlier analysis was conducted to minimize biological noise. Statistical analyses, including Ranked Partial Correlation and ANCOVA, were performed while controlling for age, sex, and TIV.

Results: Rank-order correlation analyses, independent of data distribution, revealed a highly significant negative correlation between the total orbitofrontal cortex (OFC) volume ratio and total anhedonia severity ($p=0.0005$). Notably, a significant association was identified between the right medial prefrontal cortex (mPFC) volume ratio and anticipatory pleasure (TEPS-AP) ($p=0.0022$). Extreme group analysis (EGA; comparing the healthiest 25% vs. the most severely anhedonic 25%) demonstrated statistically significant differences in total caudate volume ratios ($p=0.0044$), highlighting the critical role of striatal structures in severe pleasure loss. Furthermore, when the sample was categorized by median split, a trend toward significance was observed for total OFC volume between high and low anhedonia groups ($p=0.06$).

Conclusion: Our findings suggest that anhedonia is not merely a transient functional disturbance but is associated with concrete structural reductions in the anterior and medial regions of the brain. Specifically, the volumetric integrity of the right mPFC and OFC appears to be a determining factor in an individual's ability to anticipate and imagine future rewarding events. This structural mapping provides evidence for the trait-like nature of anhedonia pathophysiology and establishes a foundation for the development of more precise, personalized therapeutic interventions.

Keywords: Anhedonia, MRI, RDoC, mPFC, OFC, Transdiagnostic

Table 1: Demographic and Clinical Characteristics of Participants

Variable	Patient Group (n=104)	Healthy Control (n=91)	Statistic (t/Chi-sq)	p-value
Age (Mean +/- SD)	35.86 +/- 13.77	32.66 +/- 12.77	t=1.67	0.095
Sex (M/F)	48 / 56	41 / 50	Chi-sq=0.01	0.992
TEPS-AP (Anticipatory Pleasure)	41.30 +/- 9.79	44.84 +/- 7.58	t=-2.79	0.006*
TEPS-CP (Consummatory Pleasure)	37.10 +/- 7.33	38.71 +/- 5.94	t=-1.67	0.095
Total Anhedonia Score	78.39 +/- 15.03	83.56 +/- 11.41	t=-2.67	0.008*

Table 2: Comparison of Brain Volume Ratios Across Groups

Brain Region (Ratio)	Comparison Type	Group 1 (Mean ± SD)	Group 2 (Mean ± SD)	Statistic (F/t)	p-value
Total Hippocampus	Diagnosis-Based (Patient vs HC)	3.52 ± 0.35 (Pts)	3.61 ± 0.32 (HC)	F=4.08	0.0490*
Total Caudate	Symptom-Based (EGA: Low vs High)	6.24 ± 0.62 (Low)	5.88 ± 0.68 (High)	t=2.91	0.0044*
Total OFC	Symptom-Based (Median Split)	16.45 ± 1.55 (Low)	15.98 ± 1.72 (High)	F=3.57	0.0600

All analyses were conducted using ANCOVA, controlling for Age, Sex, and Total Intracranial Volume (TIV). EGA (Extreme Group Analysis) represents the comparison between the top 25% and bottom 25% of anhedonia severity scores.

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[Abstract:0211]

Examining The Relations of Mindfulness and Metacognitive Processes in Adolescents With Obsessive–Compulsive Disorder

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Background and Objective: Obsessive–compulsive disorder (OCD) in adolescence is characterized by intrusive thoughts and compulsive behaviors that affect functioning. Maladaptive metacognitions about the meaning and controllability of thought were suggested as a mechanism underlying OCD symptoms. Mindfulness is defined as awareness of the present moment and is investigated as a construct that might change these metacognitive processes and enhance cognitive flexibility. Studies showed that low mindfulness was associated with symptom severity and poorer treatment outcomes in OCD. However, the role of mindfulness and its relations in adolescent OCD samples are poorly investigated. In this study, we aimed to investigate the associations between mindfulness, metacognition, OCD symptom severity, and other clinical variables in adolescents with OCD. Additionally, we tried to explore how mindfulness may be linked to maladaptive metacognition, considering the relations of internalizing symptoms and OCD symptom severity.

Methods: In this cross-sectional study, 30 adolescents aged 12–18 years who had an OCD diagnosis were assessed according to DSM-5 criteria with semi-structured clinical interviews. Children's Yale-Brown Obsessive Compulsive Scale (CYBOCS) was conducted to confirm OCD diagnosis and to determine OCD symptom severity. Participants were asked to complete Mindful Attention Awareness Scale (MAAS), Metacognition Questionnaire for Children and Adolescents (MCQ-C), Thought-Action Fusion Inventory for Children (TAFSIC), and Revised Child Anxiety and Depression Scale (RCADS). In addition to descriptive findings, correlation analyses were conducted to examine the associations between mindfulness, metacognition, TAF, OCD, and internalizing symptoms. This study was approved by the Institutional Review Board (Registration number: 2025-192).

Results: The mean age of adolescents was 14.61 (±1.91), and 60% (n=18) of them were boys. The mean age of initial OCD diagnosis was 13.20 (±2.45), with the mean duration of 3.66 (±3.89) years. Additionally, 10 (33.3%) adolescents had psychiatric comorbidities. Consistent with our expectations, MAAS scores were significantly correlated with MCQC-Total (r=-0.417), as well as cognitive monitoring (r=-0.406), negative meta-worry (r=-0.376), punishment/responsibility (r=-0.364), and TAF-Likelihood Self (r=-0.399), which indicates substantial associations among low mindfulness and maladaptive metacognition. MAAS scores were also significantly associated with panic symptoms (r=-0.471), separation anxiety (r=-0.375), depression (r=-0.734), total anxiety (r=-0.416), and total internalization (r=-0.544) subscales of RCADS. Unexpectedly, there was no significant relationship between MAAS and OCD severity. However, the MCQC Negative-Meta-worry subscale was correlated with obsession severity (r=0.453).

Conclusions: In this study, the association between low mindfulness and maladaptive metacognition, particularly negative meta-worry and cognitive monitoring, supports the idea that low mindfulness may contribute to maladaptive beliefs in OCD adolescents. Despite the nonexistence of direct relationships with OCD symptoms, mindfulness had substantial correlations with internalizing symptomatology, which may suggest the important effects of low mindfulness on emotional concepts in this vulnerable group of adolescents. Conversely, metacognition showed a relationship with obsessions, underlying their relation to core OCD symptomatology. Together, the results of this study emphasize that working with mindfulness and metacognition may provide transdiagnostic treatment strategies for adolescents.

Keywords: Anxiety, depression, metacognition, mindfulness, obsessive-compulsive disorder, thought-action fusion

Correlations between mindfulness, metacognition, OCD severity and internalizing symptoms in children with OCD

	Age	MAAS	MCQC Cognitive Monitoring	MCQC Positive Meta-Worry	MCQC Negative Meta-worry	MCQC Punishment Responsibility	MCQC Total
Age	-	-.356	.316	.167	.314	.284	.356
MAAS	-.356	-	-.406*	-.104	-.376*	-.364*	-.417*
YBOCS-C Obsession Compulsion Total	.012 -.247 -.134	-.060 .162 .059	.317 .041 .201	-.008 -.187 -.112	.453* .007 .258	.204 .071 .155	.330 -.010 .180
RCADS Gen. Anxiety Panic Disorder Social Anxiety Separation Anx. Depression Total Anxiety Total Internalizing	.204 .284 .075 .123 .232 .256 .269	-.264 -.471** -.176 -.375* -.734*** -.416* -.544**	.464* .429* .100 .097 .347 .453* .457*	.076 .027 -.150 -.192 -.148 -.065 -.095	.689*** .473** .282 .469** .400* .651*** .627***	.453* .424* .100 .396* .354 .504** .499**	.575** .461* .130 .298 .338 .539** .521**

*Pearson correlation analysis (r values are reported) *p<0.05, **p<0.01, ***p<0.001; MAAS: Mindful Attention Awareness Scale, MCQC: Metacognition Questionnaire for Children and Adolescents, YBOCS-C: Yale-Brown Obsessive Compulsive Scale for Children, RCADS: Revised Children Anxiety and Depression Scale*

Correlations between mindfulness, TAF, OCD severity and internalizing symptoms in children with OCD

	TAFIC Likelihood Others Negative	TAFIC Likelihood Others Positive	TAFIC Likelihood Self	TAF Harm Avoidance	TAF Total
Age	.187	-.061	.119	-.120	.056
MAAS	-.256	-.272	-.399*	.021	-.298
YBOCS-C Obsession Compulsion Total	.302 .159 .261	-.009 -.091 -.057	.201 .122 .183	.351 .095 .252	.286 .107 .222
RCADS Gen. Anxiety Panic Disorder Social Anxiety Separation Anx. Depression Total Anxiety Total Internalizing	.513** .439* .146 .320 .217 .454* .418*	.323 .340 .012 .104 .227 .257 .268	.446* .481** .110 .292 .263 .457* .435*	.351 .402* .028 -.058 .247 .302 .310	.534** .541** .103 .228 .306 .486** .470**

*Pearson correlation analysis (r values are reported) *p<0.05, **p<0.01, ***p<0.001; MAAS: Mindful Attention Awareness Scale, TAFIC: Thought Action Fusion Inventory for Children, YBOCS-C: Yale-Brown Obsessive Compulsive Scale for Children, RCADS: Revised Children Anxiety and Depression Scale*

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[Abstract:0238]

Emotional Problems, Alexithymia, Body Image and Interoception in Adolescent Girls With Anorexia Nervosa

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Background and Objective: The aim of this study was to examine the relationships between anxiety, depression, alexithymia, body image, and interoception in adolescent girls with current and remitted anorexia nervosa and to compare these findings with healthy controls.

Methods: The study sample consisted of three groups of adolescent girls aged 14–17 years: 24 individuals with current anorexia nervosa (cAN), 20 individuals with anorexia nervosa in remission (rAN), and 33 healthy controls. Participants were assessed using the Eating Disorder Examination Questionnaire (EDE-Q), Revised Child Anxiety and Depression Scale-Child Version (RCADS-CV), Toronto Alexithymia Scale (TAS-20), Body Shape Questionnaire (BSQ), Multidimensional Assessment of Interoceptive Awareness in Youth (MAIA-Y), and Heartbeat Tracking Task. $p<0.05$ was accepted as the significance level.

Results: In our study, significant differences were found between the groups in terms of EDE-Q global and subscale scores, BSQ total score, and MAIA-Y trusting subscale score. In the cAN group, a significant positive correlation was found between the EDE-Q global score and depression and anxiety scores of RCADS-CV, BSQ total score, TAS-20 total score, and MAIA-Y not worrying subscale score; a significant negative correlation was found between the EDE-Q global score and the MAIA-Y trusting subscale score. In the rAN group, a significant positive correlation was found between the EDE-Q global score depression and anxiety scores of RCADS-CV, BSQ total, and TAS-20 total scores; a significant negative correlation was found between the EDE-Q global score and the MAIA-Y self-regulation and trusting subscale scores.

Conclusions: These findings suggest that eating disorder severity in adolescent girls with current and remitted anorexia nervosa is associated with anxiety, depression, alexithymia, and body image disturbance, alongside alterations in interoceptive sensibility. Among the interoceptive dimensions, trusting in particular appears to remain relevant during remission and may represent a potential target for intervention.

Keywords: Adolescents, Alexithymia, Anorexia Nervosa, Body Image, Interoception

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[Abstract:0243]

Intergenerational Transmission of Post-Migration Stress and Peer Exclusion in Second-Generation Syrian Refugee Children in a Shared Low-Resource District: A Longitudinal Mixed-Methods Study

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Background: Second-generation refugee children born in host countries may show elevated psychosocial risk despite no direct exposure to premigration trauma. Intergenerational mechanisms linking caregiver post-migration stress to early peer exclusion remain underexplored in low-resource primary-school contexts.

Methods: This prospective mixed-methods cohort study included 400 children aged 5-7 years (200 Syrian-origin refugee-background children born in Türkiye; 200 Turkish peers matched on age/sex) recruited from the same low-resource districts and schools in Nizip. Teacher- and parent-reported Strengths and Difficulties Questionnaire (SDQ) outcomes were collected at baseline (T1) and 6-month follow-up (T2). Refugee-background caregivers completed the Refugee Post-Migration Stress Scale (RPMS) and a caregiver-reported child acculturative stress measure T1. We examined group differences in SDQ outcomes. Within the refugee-background sample, we tested a moderated mediation model (PROCESS Model 14) in which RPMS predicted teacher-rated T2 Peer Problems indirectly via child acculturative stress, with parental Turkish proficiency moderating the acculturative stress → peer problems path, adjusting for baseline Peer Problems, age, sex, maternal education, and number of siblings. Semi-structured teacher interviews (n=50) were thematically analyzed and integrated with quantitative findings.

Results: Refugee-background children showed higher Peer Problems and Total Difficulties across informants and timepoints, with larger differences in teacher ratings. Within the refugee-background sample, higher caregiver post-migration stress was associated with higher child acculturative stress, which prospectively predicted higher teacher-rated T2 Peer Problems. Indirect effects were strongest when parental Turkish proficiency was lowest. Teacher interviews contextualized mechanisms including language-mediated gatekeeping of play, rapid peer categorization, and spillover of family stress into classroom emotion regulation.

Conclusions: In a shared low-resource setting, refugee-specific post-migration stressors and language access barriers plausibly contribute to early peer exclusion. Findings motivate combined school-based language access/inclusion strategies and scalable family stress supports.

Keywords: Acculturative stress, peer exclusion, post-migration stress, second-generation refugees, language access, SDQ

[Abstract:0244]

Maternal Smartphone Addiction, Parenting Stress, and Children's Perceived Emotional Neglect and Attachment Insecurity

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Background and Objective: Problematic smartphone use among caregivers may interrupt parent–child interaction (technoference) and reduce sensitive responding. Repeated disruptions in caregiver availability may contribute to children's perceptions of emotional neglect and insecure attachment.

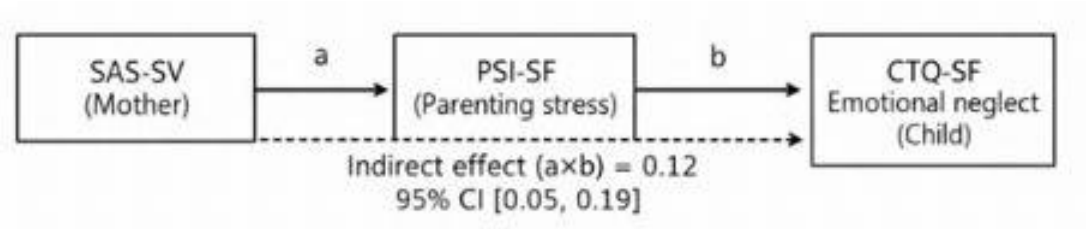
Methods: This cross-sectional study included 120 mother–child dyads (children aged 8–12 years) recruited from an outpatient child and adolescent mental health clinic in Turkey (June–November 2025). Mothers completed the Smartphone Addiction Scale–Short Version (SAS-SV) and the Parenting Stress Index–Short Form (PSI-SF). Children completed the Childhood Trauma Questionnaire–Short Form (CTQ-SF; emotional neglect) and the Attachment Style Classification Questionnaire (ASCQ). Pearson correlations and multiple linear regression were used, and mediation analysis tested whether parenting stress mediated the association between maternal smartphone addiction and child emotional neglect (bootstrapped 95% confidence interval).

Results: Maternal smartphone addiction correlated with children's perceived emotional neglect ($r = 0.46$, $p<0.001$) and insecure attachment ($r = 0.39$, $p < 0.01$). Parenting stress correlated with emotional neglect ($r = 0.41$, $p<0.01$). In regression, SAS-SV and PSI-SF jointly explained 31% of the variance in emotional neglect ($R^2 = 0.31$; $F(2,117) = 26.28$, $p < 0.001$), and both predictors were independently associated with higher emotional neglect (standardized $\beta = 0.28$ and $\beta = 0.25$, respectively). The indirect effect of maternal smartphone addiction on emotional neglect via parenting stress was 0.12 (95% bootstrap CI [0.05, 0.19]).

Conclusions: Problematic maternal smartphone use and elevated parenting stress were associated with higher child-reported emotional neglect and insecure attachment. Clinical assessment and interventions may benefit from addressing caregiver smartphone use patterns together with parenting stress management.

Keywords: Smartphone Addiction, Parenting Stress, Emotional Neglect, Attachment İnsecurity, Mediation

Figure 1. Mediation model (conceptual)



Parenting stress (PSI-SF) mediates the association between maternal smartphone addiction (SAS-SV) and child-reported emotional neglect (CTQ-SF).
Indirect effect (a×b) = 0.12; 95% bootstrap CI [0.05, 0.19].

[Abstract:0247]

Polypharmacy and Comorbidity Burden in Adolescents With Autism Spectrum Disorder in an Inpatient Psychiatric Unit

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Background and Objective: The aim of this study was to examine the sociodemographic and clinical characteristics, comorbidity profiles, pharmacological treatment patterns and relationships among clinical variables in adolescents hospitalized in a child and adolescent psychiatry inpatient unit who were diagnosed with autism spectrum disorder (ASD) or had ASD as a comorbid condition.

Methods: This retrospective cross-sectional study included 31 adolescents hospitalized in a child and adolescent psychiatry inpatient unit with a diagnosis of ASD or comorbid ASD. Sociodemographic characteristics, reasons for hospitalization, comorbid diagnoses, the number of medications used at admission and discharge and Clinical Global Impression–Severity (CGI-S) and Clinical Global Impression–Improvement (CGI-I) scores were obtained from medical records. Descriptive statistics were used for data analysis. Relationships among clinical variables were evaluated using Spearman correlation analysis. The numbers of medications used at admission and discharge were compared using the Wilcoxon signed-rank test, and differences in medication use according to the presence of behavioral problems were analyzed using the Mann–Whitney U test. Statistical significance was set at $p < 0.05$.

Results: The mean age of the participants was 14.4 ± 1.8 years, and 74.2% were male. Special education support was received by 61.3% of the patients, and at least one comorbid psychiatric diagnosis was present in 93.5%. When comorbidities were examined, attention-deficit/hyperactivity disorder was observed in 44.8% of the patients, psychotic disorders in 20.7%, intellectual disability in 22.6% and depressive disorders in 17.2%. Behavioral problems were present in the majority of the cases (90.3%). The mean length of hospitalization was 33.9 ± 17.8 days. The mean number of daily medications at admission was 2.13 ± 1.48, increasing to 3.19 ± 1.40 at discharge; this increase was statistically significant ($p = 0.001$). Pharmacological assessment revealed a polypharmacy rate of 74.2% at discharge. Antipsychotics were the most frequently prescribed medications and were used in all patients. Following antipsychotics, the most commonly used medication groups were antidepressants (32.3%), guanfacine (29.0%) and mood stabilizers (25.8%). The most common discharge treatment was antipsychotic monotherapy (25.8%), while the most frequent combination therapy consisted of antipsychotics and antidepressants (22.5%). Correlation analyses demonstrated significant positive associations between the number of daily medications at discharge and both the length of hospitalization and the number of daily medications used at admission. In addition, CGI-S scores were positively correlated with the number of daily medications used at both admission and discharge. A significant positive correlation was also found between CGI-S and CGI-I scores. Although patients with behavioral problems tended to use a higher number of daily medications, this difference did not reach statistical significance.

Conclusions: This study demonstrates that adolescents with ASD or comorbid ASD treated in an inpatient psychiatric unit have a high comorbidity burden, marked clinical complexity and polypharmacy. The increase in medication use with greater illness severity and longer hospitalization suggests that more intensive pharmacological approaches are preferred in the management of behavioral and psychiatric symptoms accompanying ASD. These findings highlight the importance of comprehensive and individualized treatment planning for adolescents with ASD during inpatient care.

Keywords: Autism spectrum disorder, comorbidity, inpatient care, polypharmacy, psychopharmacological treatment

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[Abstract:0285]

Evaluation of the Relationship Between Social Appearance Anxiety and Body Image with Obesity and Lipid Profile in Patients with Obsessive-Compulsive Disorder

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Objective: Obsessive-compulsive disorder (OCD) is a disorder characterized by obsessions and compulsions that typically begin in adolescence or early adulthood. It has been reported that many OCD patients are affected by obesity and metabolic syndrome or are at risk of developing metabolic syndrome, and that these patients should not neglect their medical care in addition to their psychiatric treatment. Negative body image attitudes are associated with low self-esteem, general mental health problems, and obesity. In our study, we planned to investigate body image in patients diagnosed with OCD and the relationship between these characteristics and serum lipid levels, obesity, self-esteem, and sociodemographic and clinical data.

Methods: The study included 52 patients with OCD diagnosed according to DSM-5 criteria, aged between 18 and 50 years, and 52 healthy volunteers. The study was approved by the Atatürk University Research Hospital Ethics Committee on October 31, 2025, under number B.30.2.ATA.0.01.00/451. Written informed consent was obtained from all patients and healthy volunteers participating in the study. Participants completed a sociodemographic-clinical data form, the Yale-Brown Obsessive-Compulsive Disorder Rating Scale (YBOCS), the Social Appearance Anxiety Scale, the Body Image Scale, and the Rosenberg Self-Esteem Scale respectively. Serum HDL, LDL, triglyceride, and total cholesterol levels were determined using routine laboratory methods.

Results: Patients and healthy controls were significantly similar in terms of age, gender, and years of education ($p>0.05$). While no statistically significant difference was found between the patient and control groups in body mass index, waist circumference, and HDL levels; statistically significant differences were found in LDL levels ($p=0.034$), triglyceride levels ($p=0.011$), and total cholesterol levels ($p=0.001$). Social Appearance Anxiety Scale, Rosenberg Self-Esteem Scale, and Body Image Scale scores were higher in the patient group; the difference was statistically highly significant ($p<0.001$). In the patient group, no significant p-value was found in the correlation test between serum lipids and the Social Appearance Anxiety Scale, Rosenberg Self-Esteem Scale, Y-BOCS and Body Image Scale. In the control group, there was a positive correlation between the Social Appearance scale and Body Mass Index ($r=0.354$, $p=0.010$) and Total Cholesterol ($r=0.283$, $p=0.042$). Due to the insufficient sample size, sub-analysis regarding the drugs used could not be performed.

Conclusions: Our study found higher serum LDL, triglyceride, and total cholesterol levels in OCD patients, suggesting that a large number of OCD patients are affected by or at risk of developing metabolic syndrome. It can be concluded that medical care should not be neglected alongside psychiatric treatment for these patients, that a holistic approach to addressing negative body image and low self-esteem could be aimed at in the treatment of OCD patients, and that more comprehensive studies are needed in this area.

Keywords: Body image, obesity, OCD, self-esteem, serum lipids

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[Abstract:0287]

Short-Form Video Platforms in Child and Adolescent Psychiatry: A Bibliometric Analysis of Research Trends and the Shift Toward Psychopathological Focus

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Background and Objective: The emergence of short-form video platforms such as TikTok, Instagram Reels, and YouTube Shorts has transformed the digital environment for youth. While these platforms offer novel modes of engagement, their impact on neurodevelopmental processes and mental health symptoms has become a significant clinical concern in child psychiatry. To guide future research and clinical priorities, the present study evaluated the scientific literature on short-form videos through a comprehensive bibliometric approach.

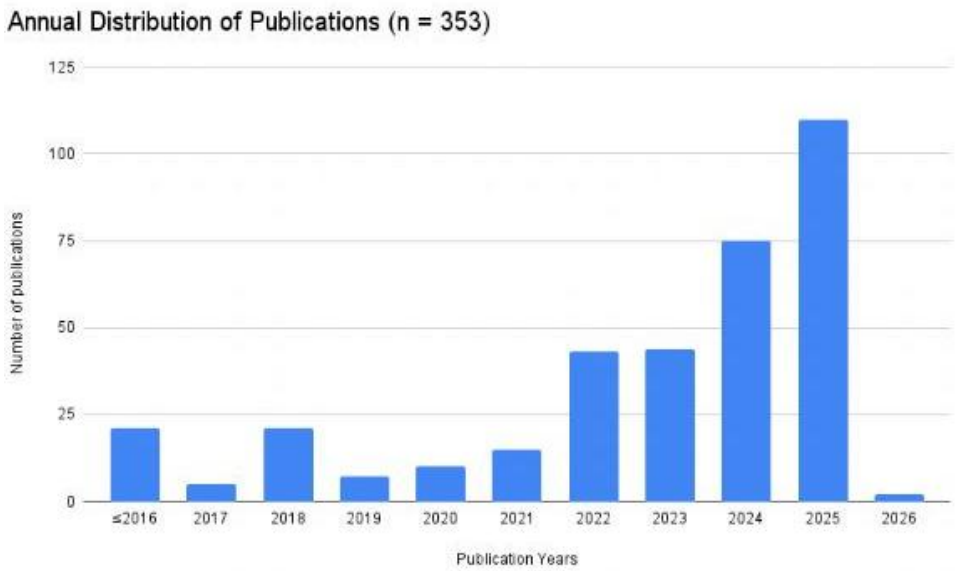
Methods: The data used in this bibliometric citation analysis were obtained from the Clarivate Analytics Web of Science (WoS) Core Collection database. The global literature on short-form videos was scanned without date limitation for publications containing predefined terms related to TikTok, Instagram Reels, and YouTube Shorts in the title, abstract, or keywords, along with mental health–related outcomes (e.g., ADHD, impulsivity, and digital addiction). Only original research articles and review papers were included. A total of 353 publications were identified and included in the analysis. Article information, including the year of publication, journal, title, author, country, affiliation, keywords, and citation counts, was recorded and exported in a tab-delimited format. To improve field-level interpretability, psychology-related categories were aggregated under a unified domain. Descriptive statistics, co-authorship, and keyword co-occurrence analyses were conducted to clarify research trends and collaborative networks. In addition, citation analysis was performed to assess the scientific impact of highly cited contributors in the field. VOSviewer (version 1.6.16) was used as the primary software tool for constructing and visualizing bibliometric networks.

Results: Research on short-form videos demonstrated a marked acceleration after 2021. Annual publication output increased sharply, rising from fewer than 20 papers in 2020 to 75 in 2024 and peaking at 110 publications in 2025. Keyword co-occurrence analysis identified a significant paradigm shift; earlier research focused predominantly on "prevention," "parenting," and "attachment", whereas the 2024–2025 period is heavily concentrated on "short video addiction," "TikTok," "ADHD-like symptoms," and "impulsivity". Geographical analysis showed that while 18 countries contributed, international collaboration was limited, with 13 countries forming the main connected group. Despite 126 contributing institutions, institutional collaboration was notably restricted, with only 8 institutions forming co-authorship links, primarily centered around Sun Yat-sen University. Among 219 contributing authors, only 11 were connected within the core collaboration network, which consisted predominantly of authors affiliated with Chinese institutions.

Conclusions: The findings demonstrate a significant shift in research focus from protective frameworks to the investigation of direct psychopathological consequences. The dominance of addiction-related terms in the most recent literature suggests that short-form video use has evolved into an established clinical problem requiring formal identification and treatment. Although intervention approaches are emerging, bibliometric mapping reveals that these efforts still lag behind the rapid expansion of addiction-oriented publications. The limited level of international and institutional collaboration highlights an urgent need for multicenter studies to understand the global impact of algorithmic platforms on the developing brain. Ultimately, this bibliometric transformation suggests that short-form video consumption is increasingly being conceptualized as a specific clinical phenomenon linked to attention, impulsivity, and emotional regulation.

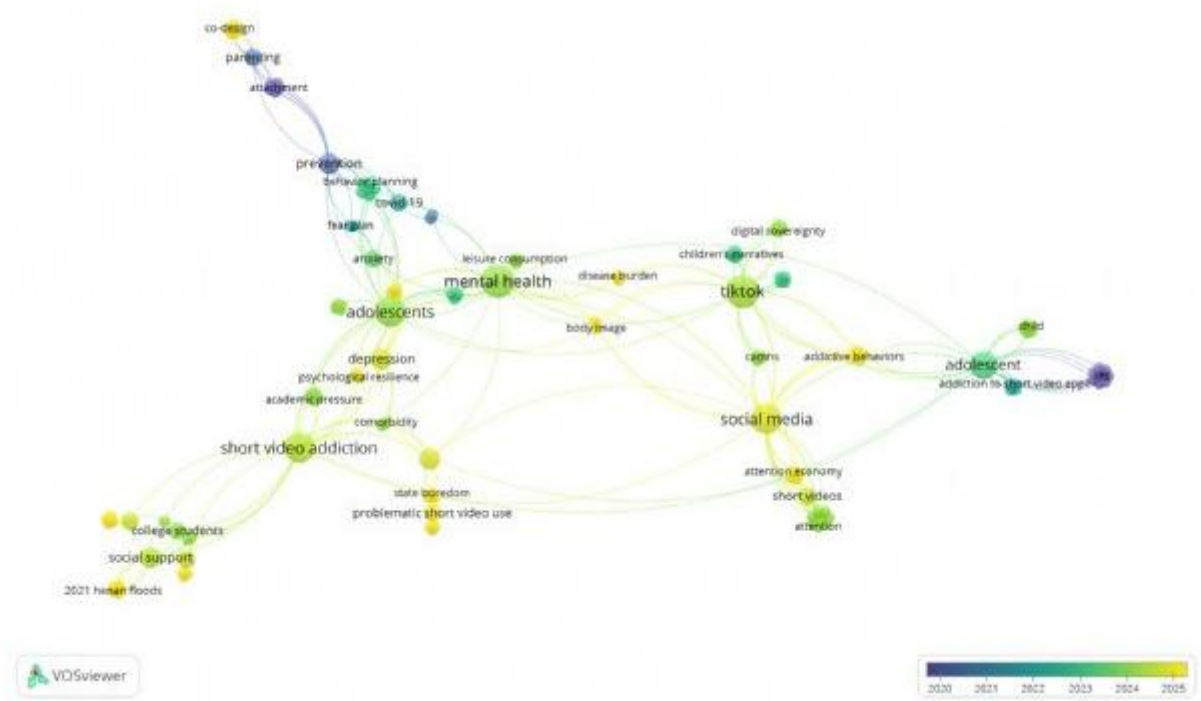
Keywords: Attention deficit hyperactivity disorder, bibliometric analysis, child and adolescent psychiatry, digital addiction, impulsivity, short-form videos

Annual Distribution of Publications (2017–2026)



The figure presents the chronological progression of scientific production concerning short-form videos. An exponential growth trend is observed starting from 2021, reaching a peak of 110 publications in 2025, which underscores the rapidly increasing academic significance of this topic in child and adolescent psychiatry.

Keyword Co-occurrence and Overlay Visualization Analysis



This figure illustrates the conceptual clustering and chronological evolution of keywords within the short-form video literature. Purple-colored nodes (e.g., prevention, parenting, attachment) represent protective and developmental frameworks dominant in the earlier stages of research. In contrast, the bright yellow nodes (e.g., TikTok, short video addiction, ADHD-like symptoms, impulsivity) demonstrate the current surge of psychopathology-oriented research during the 2024–2025 period.

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[Abstract:0288]

Associations Between Sharenting Behavior, Parenting Attitudes, and Adolescents' Internet Addiction

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Background and Objective: The pervasive integration of social media and mobile technologies into daily life has led to increased internet use, particularly amonig individuals who feel a strong need to remain constantly connected. With the widespread use of smartphones, face-to-face interactions have increasingly been replaced by digital engagement, giving rise to phubbing, defined as ignoring others in social settings due to smartphone use. This behavior has become normalized n contemporary communication patterns. These digital changeshave also reshaped parenting practices, making it increasingly common for parents to share images and videos of their children on social media platforms, a phenomenon known as sharenting. This behavior is often motivated by the desire for social support, maintaning family connections and expressing parental identity. Beyond digital pratices, parentig attitudes and the emotinal climate of the family play a crucial role in adolescents' psychosocial adjustment and problematic internet use. The combined role of sharenting, parenting attitudes in adolescents internet addiction remains underexplored. In this context, the study aims to explore how sharenting behaviors and parenting attitudes together relate to adolescents' internet addiction.

Methods: Participants were 93 adolescents aged 12–17 years who attended a child and adolescent psychiatry outpatient clinic. Sharenting behavior was assessed using the Sharenting Scale, and parental attitudes were evaluated using the Parental Attitude Research Instrument (PARI), which was completed by the parents. Adolescents completed the Young's Internet Addiction Test–Short Form to assess internet addiction and the General Phubbing Scale to measure phubbing behavior. Data were analyzed using SPSS 25.0 with appropriate parametric and non-parametric tests and correlation analyses. Statistical significance was set at $p<.05$.

Results: Correlation analyses revealed that sharenting behaviors were positively associated with adolescents' internet addiction and phubbing. Higher levels of sharenting were also significantly related to more negative parenting attitudes, particularly rejection of the housewife role and higher overall parenting attitude scores. Phubbing demonstrated a strong positive association with adolescents' internet addiction, indicating that increased parental phone-related disengagement in social contexts was linked to higher levels of problematic internet use among adolescents. In contrast, democratic parenting showed no significant relationships with sharenting, phubbing, or adolescents' internet addiction. Although overprotective, authoritarian parenting attitudes and marital discord were strongly interrelated with each other, these parenting variables were not directly associated with adolescents' internet addiction. Overall, the findings suggest that adolescents' internet addiction is more closely related to parental digital behaviors than to specific parenting styles.

Conclusions: The findings suggest that sharenting and phubbing behaviors are important digital risk factors associated with adolescents' internet addiction. While specific parenting styles were not directly linked to internet addiction, negative parenting attitudes may contribute indirectly by shaping the digital and emotional context of the family. These results highlight the need to consider parental digital behaviors alongside traditional parenting attitudes when addressing adolescents' problematic internet use.

Keywords: Adolescents, Internet addiction, Parental attitudes, Sharenting

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[Abstract:0302]

The Relationship Between Psychosocial Risk, Psychiatric Symptoms, and Post-Traumatic Growth With Quality of Life in Organ Transplant Recipients: Preliminary Findings From a Longitudinal Study

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Background and Objective: Organ transplantation is one of the most effective treatment approaches for individuals with end-stage organ failure, significantly prolonging survival and improving quality of life. However, the transplantation process entails not only biological challenges but also substantial psychological, social, and behavioral burdens. In the post-transplant period, treatment adherence, quality of life, and psychiatric symptoms play a critical role in the sustainability of clinical outcomes (Bailey et al., 2021; DiMartini & Dew, 2021). Numerous studies have demonstrated that pre-transplant psychosocial factors are associated with post-transplant outcomes such as adherence, rejection, and mortality (Dobbels et al., 2009; Maldonado, 2019).

Methods: This study is a prospective, observational cohort study including adult organ transplant recipients followed at the Organ Transplant Unit of İzmir City Hospital. The study population consists of patients aged 18 years and older who were monitored in the transplant outpatient clinic, underwent planned pre-transplant psychiatric evaluation, and continued post-transplant clinical follow-up. Sample size was calculated using G*Power, and a total of 140 participants were planned to be enrolled. The analyses presented here reflect preliminary findings from the ongoing longitudinal study at the post-transplant 3-month time point (T3), based on data obtained from 21 participants. Assessments were conducted at five time points: pre-transplant (T1), post-transplant week 1 (T2), post-transplant month 3 (T3), month 6 (T4), and month 12 (T5). Psychosocial risk was assessed using the Stanford Integrated Psychosocial Assessment for Transplantation at T1 only. The Immunosuppressive Therapy Adherence Scale and the DSM-5 Posttraumatic Stress Disorder Checklist were administered at T3, T4, and T5. Depression and anxiety were assessed using the Hospital Anxiety and Depression Scale and Beck Depression Inventory; psychological vulnerability with the Psychological Vulnerability Scale; psychological resilience with the Adult Resilience Scale; and post-traumatic growth with the Posttraumatic Growth Inventory. Quality of life was evaluated at all time points using the World Health Organization Quality of Life Scale–Brief Form and the Short Form Health Survey. Ethical approval for the study was obtained from the İzmir City Hospital Ethics Committee under decision number 2025/363.

Results: Participants with lower pre-transplant psychosocial risk demonstrated significantly higher levels of immunosuppressive treatment adherence at the 3-month post-transplant assessment ($p = .021$). The same group also reported significantly higher quality of life scores ($p = .018$). In addition, participants with higher pre-transplant psychological resilience exhibited significantly greater post-traumatic growth at T3 ($p = .030$). No statistically significant findings were observed for the remaining scales or time points.

Conclusions: Preliminary findings from the 3-month follow-up suggest that lower pre-transplant psychosocial risk and greater individual psychological resources may be associated with improved treatment adherence and quality of life in the early post-transplant period. These results are consistent with previous studies highlighting the role of psychosocial assessment in transplant outcomes (Dobbels et al., 2009; Bailey et al., 2021). The observed association between resilience and post-traumatic growth aligns with the theoretical framework proposed by Tedeschi and Calhoun (2004). As these findings are based on preliminary analyses, final conclusions will be drawn after completion of all planned follow-up assessments.

Keywords: Organ transplantation, Post-traumatic growth, Psychosocial risk, Quality of life

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[Abstract:0312]

Metabolic Syndrome and Cognitive Function in Bipolar I Disorder: A Pilot Cross-Sectional Study Accounting for Illness Burden and Treatment

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Background and Objective: Metabolic syndrome (MetS), defined by central obesity, hypertension, dyslipidemia, and impaired glucose regulation, is overrepresented in bipolar disorder in clinical samples and contributes to excess cardiovascular morbidity and premature mortality. Beyond cardiometabolic risk, MetS may affect the brain through insulin resistance, inflammation, endothelial dysfunction, and adipokine alterations, mechanisms linked to slowed processing speed and executive dysfunction. In Bipolar I Disorder (BD-I), cognitive deficits often persist during euthymia, yet reports on the MetS cognition relationship remain inconsistent. Variation in age, cumulative illness burden (episode recurrence and depressive predominance), and exposure to psychotropics with metabolic liability may obscure associations and introduce confounding by indication. Accounting for these factors is essential when interpreting neuropsychological outcomes in BD-I samples. This study aimed to compare cognitive performance between Bipolar I Disorder (BD-I) patients with and without MetS and to explore whether selected cognitive indices are associated with MetS status.

Methods: In this pilot cross-sectional study, 45 euthymic outpatients with BD-I (MetS+: n=18; MetS-: n=27) were assessed using the Stroop Color–Word Test (Cards 3–5: completion time and errors), Trail Making Test (TMT-A/B), and Wisconsin-type indices derived from the Wisconsin Card Sorting Test (WIS1–WIS13). Clinical and demographic variables included age, years of education, mood ratings (HAM-D, YMRS), illness course characteristics, and current medications. Group comparisons were performed using t-tests or Mann–Whitney U tests as appropriate, with Benjamini–Hochberg false discovery rate correction across cognitive outcomes. Derived cognitive measures included TMT B-A and a Stroop interference proxy (time ratio Card 5/3). Multivariable logistic regression examined whether age, education, TMT ratio, and Stroop ratio were associated with MetS.

Results: Patients with MetS were older than those without MetS (39.2 ± 8.2 vs. 34.1 ± 8.2 years; p=0.049) and exhibited greater illness burden, including higher total number of episodes (p=0.022) and depressive episodes (p=0.026). Valproate exposure was significantly more frequent in the MetS+ group (p<0.001), while no differences were observed for other psychotropic agents. Cognitive performance differences between groups were small and did not remain significant after correction for multiple testing. In logistic regression analysis (n=41), the model showed modest discrimination (AUC=0.69; R^2=0.097). Age demonstrated a trend-level association with MetS (OR=2.15 per SD increase; p=0.069), whereas TMT and Stroop interference ratios were not significantly associated with MetS status.

Conclusions: In this pilot sample of BD-I patients, MetS clustered with older age and greater illness burden rather than robust cognitive impairment. The higher prevalence of valproate exposure among MetS+ patients suggests that treatment-related confounding cannot be excluded. Larger longitudinal studies employing composite cognitive domains and careful adjustment for medication effects are needed to determine whether MetS contributes to neurocognitive vulnerability beyond age, illness burden, and treatment effects in bipolar disorder.

Keywords: Bipolar disorder, cognition, executive function, metabolic syndrome

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[Abstract:0325]

Aggression and Impulsivity in Methamphetamine and Alcohol Use Disorders: A Comparative Preliminary Study

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Background and Objective: In recent years, increasing attention has been directed toward the role of impulsivity and aggression in substance use disorders (SUDs) (1). Impulsivity not only increases the risk of initiating substance use but also facilitates relapse and the maintenance of SUDs. Aggression has been defined as overt behavior intended to cause harm to another individual. As a personality trait, aggression refers to recurrent and persistent aggressive attitudes across various contexts. It has been suggested that individuals with alcohol, heroin, cocaine, and methamphetamine dependence exhibit higher levels of aggression compared to nonsubstance using individuals. The primary aim of this study was to compare levels of impulsivity and aggression in individuals diagnosed with Methamphetamine Use Disorder (MUD) and Alcohol Use Disorder (AUD) with those of a healthy control group. The secondary aims were: To examine whether impulsivity and aggression subdimensions differ according to substance type and to evaluate the relationship between impulsivity and aggression levels.

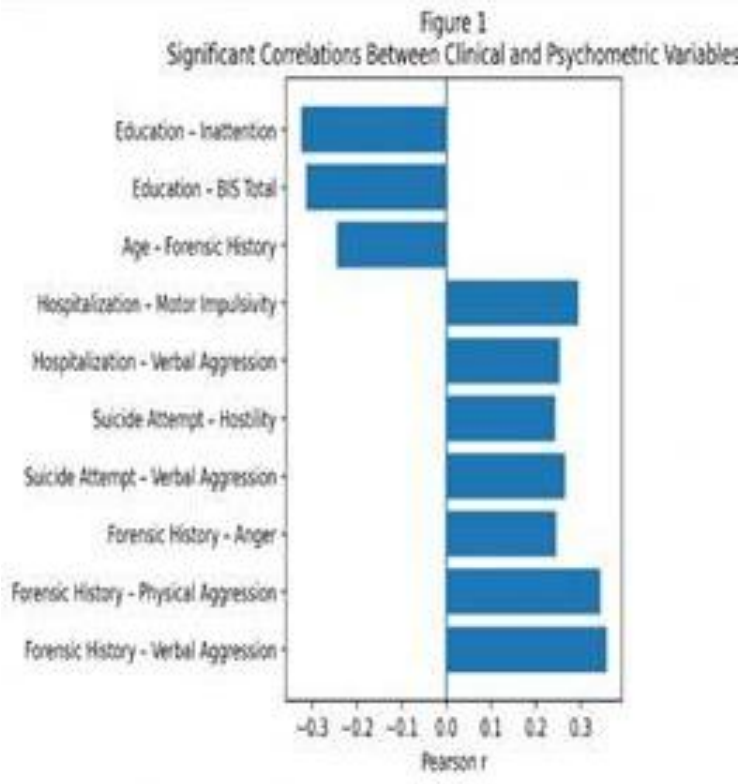
Methods: The study included patients who presented to the Samsun Mental Health and Psychiatric Hospital AMATEM unit and psychiatry outpatient clinics, as well as patients admitted for inpatient treatment in the AMATEM service. Additionally, patients who presented to the Erzurum City Hospital Department of Mental Health and Psychiatry outpatient clinic and were diagnosed with Methamphetamine Use Disorder (MUD) or Alcohol Use Disorder (AUD) according to the Structured Clinical Interview for DSM-5 Disorders – Clinician Version (SCID-5-CV) were enrolled in the study. Ethical approval for the study was obtained (Approval date: December 31, 2025; Decision No: 2025/13/5). Statistical analyses were performed using SPSS version 22.0.

Results: Sociodemographic and clinical characteristics are shown in Table 1, while selected clinical variables and between-group comparisons of scale scores are presented in Table 2. Both alcohol and methamphetamine groups demonstrated significantly higher total aggression and impulsivity scores compared to healthy controls; however, no significant differences were found between the alcohol and methamphetamine groups. Non-planning impulsivity was the only subdimension that did not show a significant difference across groups. Forensic history, suicide attempts, and number of hospitalizations were positively associated with aggression and impulsivity subdimensions. Age and education level showed significant negative correlations with forensic history and impulsivity measures. (*p <.05, **p <.01)

Conclusions: The findings indicate that aggression and impulsivity represent important constructs to be addressed in the treatment of both methamphetamine and alcohol use disorders. However, different subdimensions may constitute priority therapeutic targets depending on the type of substance dependence, and these distinctions should be carefully considered when developing and implementing therapeutic interventions.

Keywords: Alcohol, methampetamine, agression, impulsivity

Significant correlations between clinical and psychometric variables



Comparison of sociodemographic and clinical variables between groups

	Alcohol (n=36)	**Methampetamine (n=32)**	**Healty Control (n=50)**	**Total (n=118)**	
Gender					
Male	33 (%91.7)	29 (%90.6)	37 (%74.0)	99 (%83.9)	$\chi^2(2, N = 118) = 6.31, p < 0.05$
Female	3 (%8.3)	3 (%8.3)	13 (%26.0)	19 (%16.1)	
Child					
Yes	25 (%69.4)	17 (%53,1)	18 (%36.0)	60 (%50.8)	$\chi^2(2, N = 118) = 9,46\ p < 0.05$
No	11 (%30.6)	15 (%46.9)	32 (%64)	58 (%49.2)	
Employment status					
With employment	24 (%66.7)	14 (%43.8)	44 (%88.0)	82 (%96.5)	$\chi^2(2, N = 118) = 24.29\ p < 0.05$
Part-time	4 (%11.1)	13 (%40.6)	4 (%8.0)	21 (%17.8)	
Without employment	8 (%22.2)	5 (%15.6)	2 (%4.0)	15 (%12.7)	
Monthly income					
<20K	11 (%30.6)	15 (%45.9)	3 (%6.0)	29 (%24.6)	$\chi^2(2, N = 118) = 27.08\ p < 0.05$
20-30k	8 (%22.2)	3 (%9.4)	3 (%6.0)	14 (%11.9)	
>30k	17 (%47.2)	14 (%43.8)	44 (%88.0)	75 (%63.6)	
Residential status					
City center	22 (%61.1)	25 (%78.1)	44 (%88.0)	91 (%77.1)	$\chi^2(2, N = 118) = 8.60\ p < 0.05$
District	14 (%38.9)	7 (%21.9)	6 (%12.0)	27 (%22.9)	
Living situation					
Living with familiy	19 (%52.8)	12 (%37.5)	27 (%54.0)	58 (%49.2)	$\chi^2(2, N = 118) = 6.83\ p > 0.05$
Living with parents	12 (%33.3)	17 %53.1)	13 (%26.0)	42 (%35.6)	
Alone	5 (%13.9)	3 (%9.4)	10 (%20.0))	18 (%15.3)	
Smoking					
Yes	33 (%91.7)	30 (%93.8)	24 (%48.0)	87 (%73.7)	$\chi^2(2, N = 118) = 29.70\ p < 0.05$
No	3 (%8.3)	2 (%6.3)	26 (%52.0)	31 (%26.3)	
Forensic history					
Yes	13 (%36.1)	22 (%68.8)	-	35 (%51.5)	$\chi^2(1, N = 68) = 7.22\ p < 0.05$
No	23(%63.9)	10 (%31.3)	-	33 (%48.5)	
Suicide Attempt					
Yes	8 (%22.2)	5(%15.6)	-	13 (%19.1)	$\chi^2(1, N = 68) = 0.48\ p > 0.05$
No	28 (%77.8)	27 (%84.4)	-	55 (%80.9)	
Hospitalization					
Yes	18 (%50)	25 (%78.1)	-	43 (%63.2)	$\chi^2(1, N = 68) = 5.76\ p < 0.05$
No	18 (%50)	7 (%21.9)	-	25 (36.8)	
Medical comorbidity					
Yes	15 (%41.7)	5 (%15.6)	6 (%12.0)	26 (%22.0)	$\chi^2(2, N = 118) = 11.78\ p < 0.05$
No	21 (%58.3)	27 (%84.4)	44 (%88.0)	92 (%78.0)	

Comparison of scale scores and clinical variables between groups

	Alcohol (n=36)	Methampetamine (n=32)	Healty Control (n=50)	Total (N=118)	
Age	36 ± 8.6	30.4 ± 7.5	32.5 ± 7.1	33 ± 8.0	F(2,115) = 4.81, p < 0.05
Education	10.1 ± 3	10.4 ± 2.5	12 ±2.9	11 ±3	F(2,115) = 5.76, p < 0.05
BIS-11Total	29.8±7.7	30.5±7.8	26.1±7.4	28.4±7.8	F(2,115) = 4.16, p < 0.05
Inattention	9.6 ±4.2	9.6 ±2.7	8 ±2.6	8.9 ±3.3	F(2,115) = 3.62, p < 0.05
Motor Impulsivity	9.7 ±2.7	11 ±3.4	8.6 ±3	8.9 ±3.3	F(2,115) = 6.34, p < 0.05
Non-planning	10.6±4	9.9±3.5	9.6±3.8	9.9±3.7	F(2,115) = 0.74, p > 0.05
BPASTotal	69±17.7	83±18.9	66.6±14.5	72±17.8	F(2,115) =10.12, p < 0.05
Physical Agression	19.8±6	24.2±7.1	18.7±4.7	20.5±6.2	F(2,115) = 9.22, p < 0.05
Anger	17.1±5.9	20.2±5.3	16±7.1	17.5±6.5	F(2,115) = 4.56, p < 0.05
Hostility	19.6±6	22.1±5.6	18.4±4.8	19.8±5.6	F(2,115) = 4.71, p < 0.05
Verbal Agression	13.4±3.6	16.4±3.3	13.5±3.3	14.2±3.6	F(2,115) = 8.64, p < 0.05

BIS-11; Barratt Impulsiveness Scale BPAS; Buss-Perry Aggression Scale

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[Abstract:0332]

Transition to Long-Acting Injectable Aripiprazole in Chronic Psychiatric Disorders: A Retrospective Analysis of Clinical Functioning and Caregiver Burden

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Background and Objective: Long-acting injectable (LAI) antipsychotics improve treatment adherence and reduce relapse in chronic psychiatric disorders. Aripiprazole once monthly, through its partial D2 agonist mechanism, has demonstrated clinical efficacy in schizophrenia cohorts. However, real-world data across diagnostic groups remain limited, and the relationship between functional improvement and reduction in caregiver burden has not been sufficiently investigated. This study aimed to evaluate the effects of switching to LAI aripiprazole on clinical severity, overall functioning, and caregiver burden in a sample of patients with chronic psychiatric disorders, and to examine the association between functional gains and caregiver burden.

Methods: In this retrospective, single-center, observational study, medical records of consecutive patients with chronic psychiatric disorders who were switched from oral antipsychotics to LAI aripiprazole 400 mg/month between January and November 2025 at Erzurum City Hospital Psychiatry Outpatient Clinic and Community Mental Health Center were reviewed. Patients with complete baseline (month 0) and 6-month assessments were included. The Global Assessment of Functioning (GAF), Clinical Global Impression–Severity (CGI-S), Clinical Global Impression–Improvement (CGI-I), Clinical Global Impression–Efficacy Index (CGI-E), and Zarit Burden Interview (ZBI) were administered. The Wilcoxon signed-rank test was used for non-normally distributed variables. Effect sizes (Cohen's d, Wilcoxon r) and Spearman correlations were calculated.

Results: Thirty-nine patients were included (mean age 40.9±9.8 years; 64.1% male; mean illness duration 15.9±6.8 years). Diagnoses were schizophrenia (17.9%), bipolar disorder (20.5%), schizoaffective disorder (28.2%), and other psychotic disorders (33.3%). At month 6, significant improvements were observed across all outcome measures. GAF scores increased from 53.15±14.81 to 66.44±17.58 (25.0% increase; $p<0.001$, $d=0.789$). CGI-S decreased from 5.13 to 4.62 ($p<0.001$, $d=0.678$), and 53.8% of patients achieved a CGI-I response (<=2). CGI-E scores showed significant improvement ($p=0.002$, $d=0.553$), with deterioration observed in only 2.6% of patients. ZBI scores decreased from 23.82±7.71 to 20.79±6.53 ($p<0.001$, $d=0.880$); improvement was observed in 66.7% of caregivers. A strong correlation was found between functional improvement and reduction in caregiver burden ($\rho=-0.681$, $p<0.001$), which remained significant after controlling for baseline GAF, illness duration, and age (partial $r=-0.659$ to -0.747, all $p<0.001$). No significant differences in treatment response were observed among diagnostic groups (Kruskal–Wallis $p=0.363$).

Conclusions: Switching to LAI aripiprazole was associated with clinically meaningful improvements in functioning, illness severity, and caregiver burden, with moderate-to-large effect sizes, in a chronic psychopathological sample. The strong correlation between functional improvement and reduction in caregiver burden extends beyond schizophrenia-specific data in the literature. These findings support the clinical relevance of caregiver burden as an important outcome in LAI antipsychotic treatment.

Keywords: Caregiver burden, Chronic psychiatric disorders, Clinical functioning, Long-acting injectable aripiprazole

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[Abstract:0333]

The Invisible Barrier to Mandatory Treatment: The Relationship Between Internalized Stigma and Treatment Motivation in Probationers

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Background and Objective: Individuals with Substance Use Disorders (SUD) under probation face a risk of "double stigma" arising from both their clinical diagnosis and their involvement in the criminal justice system. This intersectional stigma can precipitate the "why try" effect, severely eroding self-efficacy and diminishing the willingness to engage in recovery. This study aims to cross-sectionally evaluate internalized stigma levels in individuals under probation and investigate the predictive impact of these stigma constructs on treatment motivation.

Methods: The study was approved by the Ege University Ethics Committee (Decision No: 25-11T/38, Date: 06.11.2025). The sample included 152 volunteers diagnosed with SUD who were attending the Probation Outpatient Clinic of Izmir City Hospital. Data were collected using a Sociodemographic Data Form, the Internalized Stigma of Mental Illness (ISMI) Scale, and the Treatment Motivation Questionnaire (TMQ). Statistical analyses were performed using SPSS; Pearson correlation analysis was utilized to assess the relationship between stigma sub-dimensions and treatment motivation scores.

Results: The mean age of participants was 28.7 ± 5.3 years, with a male predominance. Comparative analyses revealed that female participants and individuals with a history of incarceration exhibited significantly higher scores in the "Alienation" and "Stereotype Endorsement" sub-dimensions ($p<0.05$). A primary finding was the statistically significant negative correlation between total internalized stigma levels and treatment motivation ($p<0.05$); as internalized stigma increased, the motivation for treatment notably declined. Furthermore, the "Social Withdrawal" sub-dimension was identified as a significant factor associated with reduced engagement in the treatment process.

Conclusions: Our findings suggest that internalized stigma acts as a substantial psychological barrier to treatment adherence in the probation context, particularly for women and those with a history of incarceration. Forensic psychiatry protocols must go beyond substance monitoring and integrate psychosocial interventions targeting stigma to improve outcomes. Crucially, low treatment motivation in this population should be viewed not merely as resistance to therapy, but as a consequence of internalized societal labels. Unless the "criminal-addict" identity is deconstructed to restore self-worth, mandatory treatment risks remaining a mechanical obligation rather than a transformative rehabilitative process.

Keywords: Probation, Internalized Stigma, Treatment Motivation, Substance Use Disorder

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[Abstract:0341]

Irritability in Generalized Anxiety Disorder: Preliminary Cross-Sectional Findings on its Associations With Theory of Mind and Psychological Symptoms

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Background and Objective: Generalized Anxiety Disorder (GAD) is a chronic psychiatric condition with a reported lifetime prevalence of approximately 3.7% and is associated with significant functional impairment across multiple life domains. Irritability is a common clinical feature of GAD and is included among the diagnostic criteria in DSM-5. Previous findings suggest that elevated irritability in GAD may be associated with poorer treatment response; however, studies examining the severity of irritability and its psychosocial correlates in GAD remain limited. Theory of mind (ToM), defined as the capacity to understand and predict others' behavior based on mental states such as beliefs, desires, emotions, and intentions, represents an important social–cognitive skill. Higher irritability has been linked to poorer ToM abilities in different clinical conditions. A recent systematic review and meta-analysis reported impaired ToM performance in social anxiety disorder, but highlighted the scarcity and inconsistency of findings in GAD populations. This study aimed to investigate irritability levels, ToM abilities, and factors associated with irritability in individuals with GAD compared with healthy controls.

Methods: The sample consisted of 37 participants: 19 individuals diagnosed with GAD and 18 healthy controls. All participants completed the Depression Anxiety Stress Scale-21 (DASS-21), the Reading the Mind in the Eyes Test (RMET), and Brief Irritability Test. Sociodemographic variables included age, gender, education level, and income. Group differences were examined using independent samples t-tests and chi-square analyses as appropriate. Hierarchical multiple linear regression analysis was conducted to determine predictors of irritability. In Step 1, demographic variables were entered; in Step 2, diagnostic group was added; in Step 3, DASS-21 depression, anxiety, and stress subscale scores were included; and in Step 4, RMET scores were entered.

Results: The GAD and control groups did not differ significantly in age (34.9±12.4 vs 39.7±9.1 years; p=0.19), however the GAD group had a higher proportion of women (84.2% vs 50.0%; $\chi^2(1)=4.94$, p=0.026), lower educational attainment ($\chi^2(2)=11.27$, p=0.004), and lower income levels ($\chi^2(3)=12.26$, p=0.007). Participants with GAD had significantly higher irritability scores than controls (17.0±7.0 vs 11.8±4.7; t(35)=2.62, p=0.013). They also showed higher stress (12.05±6.36 vs 5.44±4.91; p=0.001), anxiety (11.21±6.73 vs 2.83±3.38; p<0.001), and depression (11.21±6.25 vs 5.56±5.58; p=0.006). RMET performance did not differ between groups (22.68±3.35 vs 23.83±3.09; p=0.29). In regression analyses, demographic variables were not significant predictors of irritability. Diagnostic group predicted irritability initially, but lost significance after emotional symptom variables were entered. Among DASS-21 subscales, stress was the strongest predictor ($\beta\approx0.85$, p<0.01), whereas anxiety and depression were not significant. RMET scores were not associated with irritability. The final model explained approximately 62% of the variance.

Conclusions: Although irritability is elevated in individuals with GAD, its association with diagnostic status appears to be largely explained by concurrent stress symptoms rather than diagnosis itself. These findings suggest that irritability may represent heightened stress reactivity rather than a disorder-specific feature. Social–cognitive performance, as measured by RMET, did not contribute to irritability levels in the current study. Clinically, assessing and targeting stress burden may be more relevant than focusing on diagnosis alone when addressing irritability.

Keywords: Anxiety, depression, generalized anxiety disorder, irritability, stress, theory of mind

AuthorToEditor: Sayın Bilim Kurulu, Çalışmamız özgün olup daha önce başka bir kongrede sunulmamış ve yayımlanmamıştır. Yazarların herhangi bir çıkar çatışması bulunmamaktadır. Sunum türü olarak sözlü sunum tercihimiz bulunmaktadır. Saygılarımla, Hakan Yılmaz ORCID:0000-0002-1478-3937

[Abstract:0357]

Evaluation of the Treatment Efficacy and Tolerability of Caripiprazine in Schizophrenia and Bipolar Disorder: Case Series

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Background and Objective: Cariprazine is a piperazine-based atypical antipsychotic approved by the U.S. Food and Drug Administration (FDA) in 2015 for the treatment of schizophrenia and manic or mixed episodes associated with bipolar disorder in adults. Its pharmacological profile differs from that of other antipsychotics, as it acts as a partial agonist at dopamine D2/D3 and serotonin 5-HT1A receptors, while exhibiting antagonistic activity at 5-HT2A, 5-HT2B, and histamine H1 receptors.1 In 2022, cariprazine was also approved by the FDA as adjunctive therapy for major depressive disorder. Cariprazine is associated with a lower risk of weight gain, dyslipidemia, and hyperglycemia.2,3 Given its unique receptor profile and its relatively recent introduction in our country, this study presents case reports to determine the clinical efficacy and tolerability of cariprazine

Methods: Our study included a total of four patients, three with schizophrenia and one with bipolar disorder, depressive episode, who were initiated on cariprazine treatment at a university hospital psychiatry clinic. Written and verbal informed consent was obtained from the patients. Scales were administered before and 8 weeks after cariprazine use. The Scale for the Assessment of Positive Symptoms(SAPS) and Scale for the Assessment of Negative Symptoms (SANS) were administered to the patients with schizophrenia; the Hamilton Depression Rating Scale(HAM-D) was administered to the patient with bipolar disorder. In addition, all patients were administered the Clinical Global Impressions (CGI) and the UKU side-effect rating scales.

Results: The mean age of the cases was 45.5±13.52 years. All patients were male. The mean cariprazine dose was 4.87±0.75 mg/day. Cariprazine was initially started at 3 mg/day for all patients; due to partial benefit, it was increased to 6 mg/day in one case and to 4.5 mg/day in the other cases at the follow-up appointment 4 weeks later. No patient discontinued the medication due to side effects. A drug-related mild side effect was observed in one case; this included increased dreaming, dry mouth, and burning in the eyes. The clinical characteristics and the scale scores of the cases are shown in Table 1.

Conclusions: Recent studies have shown that cariprazine has demonstrated promising results in the treatment of bipolar disorder, especially depressive episodes, as well as schizophrenia.4,5 Cariprazine may provide high therapeutic efficacy and tolerability in the treatment of schizophrenia and bipolar disorder. These characteristics may indicate that cariprazine is a promising pharmacological option for clinicians in contemporary psychiatric practice.

Keywords: Antipsychotic, Bipolar Disorder, Cariprazine, Schizophrenia

The clinical characteristics and the scale scores of the cases

Case	Age	Diagnosis	Cariprazine Dosage(mg/day)	CGI B/A	HAM-D B/A	SAPS B/A	SANS B/A
1	30	Schizophrenia	6	9/6	-	39/3	52/1
2	63	Schizophrenia	4.5	7/7	-	12/3	33/10
3	45	Schizophrenia	4.5	9/5	-	58/17	87/57
4	44	Bipolar Disorder Depressive Episode	4.5	13/5	49/27	-	-

(CGI: Clinic Global Impressions, HAM-D: Hamilton Depression Rating Scale, SAPS: Scale for the Assessment of Positive Symptoms, SANS: Scale for the Assessment of Negative Symptoms, B/A: Before/After)

[Abstract:0360]

Cross-Validated Bipolar Disorder Classification and Risk Stratification Using Penalized Logistic Regression and Machine Learning in a Single-Center Pilot Case–Control Study

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Background and Objective: Bipolar disorder (BD) is clinically heterogeneous, and diagnostic/risk judgments often rely on longitudinal history. We aimed to quantify covariate-adjusted case–control associations and to develop internally validated predictive models—including a transportable, closed-form risk formula—to distinguish BD patients from healthy controls using easily obtainable personal/clinical features.

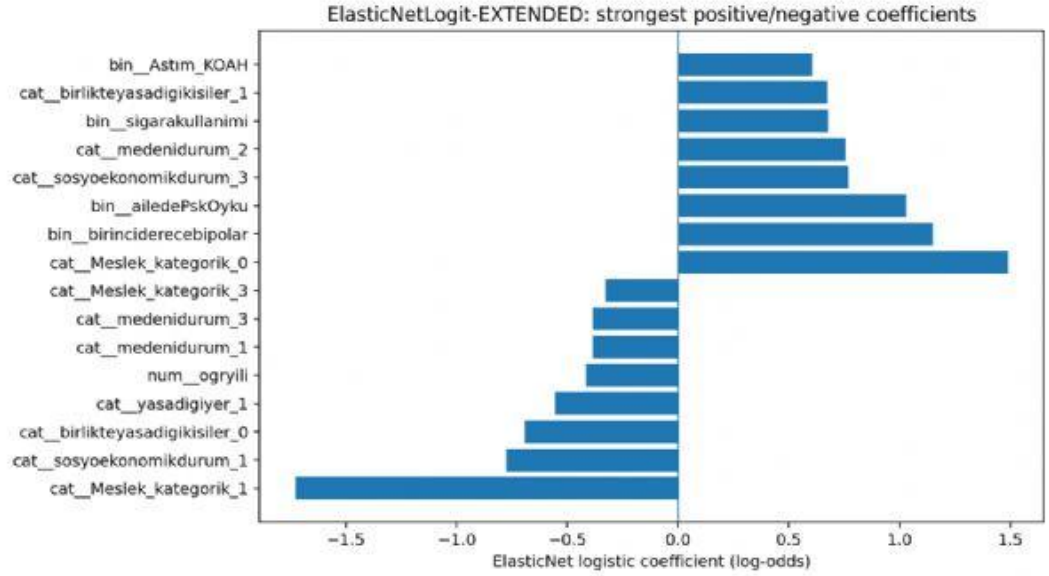
Methods: We analyzed a single-center, cross-sectional pilot case–control dataset (N=105; BD n=54; controls n=51). Candidate predictors included demographics (age, sex, years of education), lifestyle exposures, family history variables, medical comorbidities, and socio-functional indicators. To avoid leakage or trivial separation, BD-specific disease-course variables and medication dose metrics were excluded from case–control prediction. Group contrasts used Mann–Whitney U tests (continuous variables summarized as median [IQR]) and chi-square or Fisher's exact tests (categorical variables). Covariate-adjusted associations were estimated with logistic regression adjusted for age, sex, and education, with sparse-cell/separation diagnostics used to prioritize stable, parsimonious models. Predictive modeling used stratified 5-fold cross-validation with out-of-fold predicted probabilities and within-cross-validation hyperparameter tuning. We compared an interpretable elastic-net logistic regression with tree-based classifiers (Random Forest, HistGradientBoosting, Extreme Gradient Boosting), reporting out-of-fold AUC and Brier score. To derive a transportable risk score, redundancy among candidate predictors was reduced by hierarchical clustering of mixed-type feature associations; representative predictors were then fit in a ridge-penalized logistic regression yielding a closed-form probability. Rule-out and rule-in thresholds were derived from out-of-fold probabilities to target sensitivity >=0.95 and specificity >=0.95, defining low/medium/high risk bands.

Results: BD cases had fewer education years (12 [5–15.75] vs 16 [14–18]; p<0.001). Family psychiatric history was more frequent in BD (50.0% vs 9.8%; p<0.001), as was a first-degree BD relative (33.3% vs 5.9%; p=0.001); occupation also differed markedly between groups ($p<1\times10^{-10}$). In covariate-adjusted single-exposure models (age, sex, education), family psychiatric history was strongly associated with BD (OR 13.12, 95% CI 3.83–44.86; p<0.001) and first-degree BD family history showed a similarly large effect (OR 13.35, 95% CI 3.09–57.69; p<0.001). In the combined family-history model, family psychiatric history remained significant (OR 7.14, 95% CI 1.50–33.88; p=0.013) while the first-degree term attenuated (OR 2.96; p=0.269); model AUC was 0.873. For prediction, elastic-net logistic regression (extended features) achieved OOF AUC 0.899 (Brier 0.129) and Random Forest achieved OOF AUC 0.923 (Brier 0.105); the ridge-based transportable formula achieved OOF AUC 0.928 (Brier 0.103). Using thresholds P<0.306 (LOW) and P>=0.800 (HIGH), 80.4% of controls were classified as LOW risk and 66.7% of BD cases as HIGH risk (observed BD rates: 4.7% in LOW and 94.7% in HIGH), with rule-out sensitivity 0.963 and rule-in specificity 0.961.

Conclusions: In this single-center pilot case–control study, familial psychiatric history was the most robust covariate-adjusted signal for BD, consistent with BD's high heritability in family/genetic studies. Across models, routinely obtainable variables showed strong internally validated discrimination, and the closed-form ridge score yielded interpretable risk strata with rule-out/rule-in thresholds. However, socioeconomic/functional predictors (e.g., occupation) may reflect illness consequences or sampling and reduce transportability. External validation, prevalence calibration, and sensitivity analyses excluding potentially downstream variables are required before clinical use.

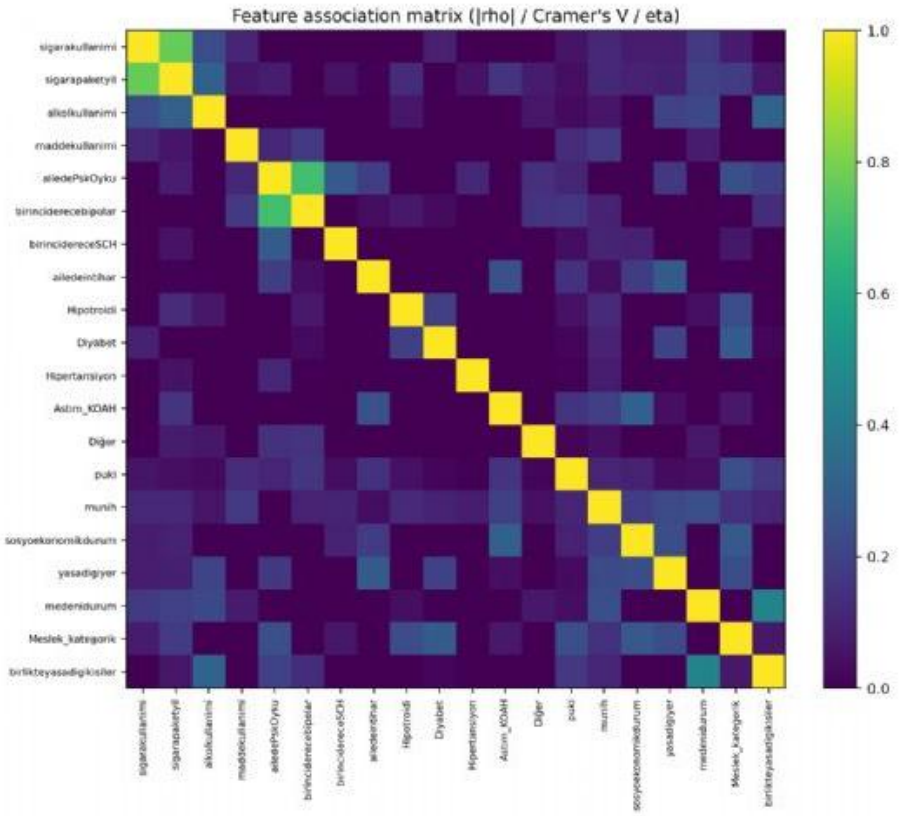
Keywords: Bipolar disorder, Case–control study, Cross-validation, Elastic net logistic regression, Machine learning

ElasticNetLogit-EXTENDED coefficients (log-odds)



Positive coefficients increase predicted probability of bipolar status; negative coefficients decrease it. Coefficients reflect the fitted model on full data after internal tuning and should be interpreted cautiously.

Feature association heatmap (mixed-type measures)



Values represent association strength (0-1) using the appropriate metric per feature

Core adjusted models (age + sex + education adjusted)

Model	Term	OR (95% CI)	p-value
M1_familyPsych	Age	1.05 (0.99-1.10)	0.1059
M1_familyPsych	Years of Education	0.78 (0.67-0.91)	0.001828
M1_familyPsych	Male (sex)	1.37 (0.48-3.93)	0.5531
M1_familyPsych	Family History of Psychiatric Disorder	13.12 (3.83-44.86)	4.098e-05
M2_firstDegreeBipolar	Age	1.02 (0.98-1.07)	0.3374
M2_firstDegreeBipolar	Years of Education	0.75 (0.64-0.87)	0.0001991
M2_firstDegreeBipolar	Male (Sex)	1.11 (0.40-3.09)	0.845
M2_firstDegreeBipolar	First-Degree Family History of Bipolar Disorder	13.35 (3.09-57.69)	0.0005212

Core adjusted logistic regression models. Each model includes age, sex (male), and education years. Family psychiatric history and first-degree bipolar history show strong adjusted associations when modeled separately.

Shows the combined adjusted model including both family-history predictors

Term	OR (95% CI)	p-value
Years of Education	0.77 (0.66-0.90)	0.001098
Family History of Psychiatric Disorder	7.14 (1.50-33.88)	0.01339
Age	1.04 (0.99-1.10)	0.1255
First-Degree Family History of Bipolar Disorder	2.96 (0.43-20.17)	0.2687
Male(sex)	1.27 (0.44-3.70)	0.6623

Combined adjusted logistic regression (age + sex + education + family psychiatric history + first-degree bipolar history). When both family variables enter together, the broader family psychiatric history retains significance while first-degree bipolar history attenuates, consistent with overlap/collinearity and limited sample size.

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[Abstract:0365]

Drug-Related Suicide Deaths: Medicolegal Analysis of sociodemographic and clinical characteristics

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Background and Objective: Suicide is a major global public health problem, causing an estimated 727,000 deaths annually and prevention efforts emphasize multisectoral, evidence-based strategies, including restriction of access to lethal means such as medications. Drug ingestion is a common suicide method, yet comprehensive evidence on the sociodemographic and clinical profiles of people who die by drug-related suicide remains limited in Türkiye. This study aimed to examine the sociodemographic and clinical characteristics of individuals who died by suicide via drug overdose in Türkiye, and to analyze the types of drugs used. The aim of this study is to analyze the drug type, associated sociodemographic and clinical factors and to inform public health policies and risk-focused preventive interventions in clinical practice.

Methods: This retrospective study was conducted at the Council of Forensic Medicine. Sociodemographic and clinical characteristics, the type of drug(s) involved, and history of psychiatric illness were obtained through comprehensive review of case files available in the UYAP system. A total of 34 cases who were determined in 2024 to have died by suicide due to drug ingestion were included. Statistical analyses were performed using IBM Statistical Package for Social Sciences version 20.0.

Results: The mean age was 42.0 ± 17.5 years, and 55.9% were female. Most individuals were not regularly employed (64.7%); education level was primary or middle school in 50.0%, high school in 35.3%, and university in 14.7%. A known psychiatric disorder was documented in 52.9%, while 11.8% had a prior suicide attempt. A suicide note was documented in 2.9%, and a pre-suicide argument in 11.8%. Single-agent ingestion occurred in 52.9%, two agents in 26.5%, three agents in 14.7%, and four agents in 5.9%. The distribution of drug groups were cardiovascular/metabolic agents (29.4%) and anesthetic/neuromuscular agents (29.4%), analgesic/opioids (17.6%), antidepressants (17.6%), mood stabilizers/antiepileptics (14.7%,), and benzodiazepines/sedatives and antipsychotics (each 8.8%). Psychotropic drugs were involved in 47.1% overall and were more common among those with a known psychiatric history (72.2% vs. 18.8%). Notably, 20.6% were healthcare workers; 85.7% of these cases involved anesthetic/neuromuscular agents compared with 14.8% among non–healthcare workers. Compared with non–healthcare workers, healthcare workers were all employed and had higher educational attainment, and they tended to be younger with a lower female-to-male ratio.

Conclusions: In this medicolegal series of drug-related suicide deaths, over half of the cases had a documented psychiatric history and nearly half involved psychotropic medications, underscoring the clinical importance of systematic risk assessment and effective treatment of mental disorders as core clinical prevention strategies. The prominent involvement of anesthetic/neuromuscular agents among healthcare workers, aligns with evidence that health professionals may show elevated vulnerability, supporting targeted prevention strategies like secure storage, inventory controls/audits, and proactive staff mental health support. Limiting access to large quantities of high-risk medicines is important to prevent drug-related suicides and should be considered as a practical public health measure. Overall, integrating clinical risk management with targeted access-control measures is essential to strengthen suicide prevention efforts for drug-related deaths.

Keywords: Drug-related suicide, forensic psychiatry, healthcare workers, medicolegal, psychotropic drugs

AuthorToEditor: We respectfully request consideration for an oral presentation. This study presents a medicolegal analysis of drug-related suicide deaths using UYAP-based forensic records, with a specific focus on drug class patterns and occupational access (healthcare workers). The findings may inform both clinical risk management and means-restriction strategies.

[Abstract:0372]

Intrusive Verbal Thought and Intrusive Visual Imagery in Depressive and Anxiety Disorders: The Role of Serotonergic Medication

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Background and Objective: Involuntary cognitive processes, particularly intrusive visual imagery (IVI) and intrusive verbal thoughts (IVT), are key features of depressive and anxiety disorders (1,2). These phenomena are partially independent and linked to distinct cognitive systems (3). In depression, intrusive negative autobiographical memories are associated with symptom severity, and sensory–perceptual aspects of depressive cognitions relate to greater psychopathology (1,4). Repetitive negative thinking is a transdiagnostic mechanism maintained by interpretation biases (5).

This study compared verbal–visual differentiation between IVT and IVI across depressive disorder, anxiety disorder, and healthy controls, and examined whether it varies with serotonergic medication use.

Methods: A total of 252 participants were included: 84 healthy controls, 84 with depressive disorder, and 84 with anxiety disorder. Diagnoses were established by a psychiatrist according to DSM-5 criteria. Participants completed a Sociodemographic Form, the IVI and IVT questionnaires, and the Beck Depression (BDI) and Anxiety (BAI) Inventories. The IVT–IVI difference score was calculated by subtracting IVI from IVT scores. Analyses were stratified by serotonergic medication use. Medication users were compared with the Mann–Whitney U test; non-users with the Kruskal–Wallis test followed by Dunn–Bonferroni post hoc tests. Significance was set at $p < 0.05$.

Results: Of the sample, 64.8% were female (n = 163) and 35.2% male (n = 89); mean age was 33.91 ± 10.12 years (18–62). BDI scores differed significantly across groups ($p < 0.001$), with the depressive group scoring highest, followed by the anxiety group, and controls scoring lowest. Similarly, BAI scores differed ($p < 0.001$), with the anxiety group scoring highest, followed by the depressive group and controls. Among serotonergic medication users, IVT–IVI difference scores did not differ between anxiety and depressive groups ($p = 0.310$). In non-users, differences were significant ($p < 0.001$), with higher scores in the depressive group than in controls ($p < 0.001$) and the anxiety group ($p = 0.040$); no difference was found between the latter two ($p = 0.174$).

Conclusions: Verbal–visual differentiation was most pronounced in unmedicated patients with depressive disorder, indicating a predominance of verbally mediated intrusions in this group. The absence of differences among serotonergic medication users suggests a modulatory effect of medication. These findings support the clinical relevance of modality-based cognitive profiling and modality-specific interventions.

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Keywords: Anxiety disorders, depressive disorder, mental imagery, rumination

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[Abstract:0385]

An Investigation of the Relationship Between Reading-Writing Performance and Executive Functions, Neurocognitive Profile, and Functionality in Children with Specific Learning Disorder and Comorbid AttentionDeficit/Hyperactivity Disorder

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Background and Objective: This study examined the relationship between reading–writing performance and mental health problems, executive dysfunctions, neurocognitive deficits, and functional impairment in children diagnosed with specific learning disorder (SLD) with comorbid attention-deficit/hyperactivity disorder (ADHD), and compared them with children diagnosed with ADHD only. In addition, risk factors predicting reading–writing performance and functional impairment were investigated.

Methods: SLD was diagnosed through DSM-5 and supported by the Reading–Writing Assessment Battery (OYAB), grade-appropriate calculation performance tests, and the Bender–Gestalt Test. ADHD diagnosis was determined using DSM-5, Conners' Parent Rating Scales, and teacher report forms. The study sample consisted of 106 children, including 71 diagnosed with SLD with comorbid ADHD and 35 diagnosed with ADHD only. Based on OYAB composite scores, the SLD with comorbid ADHD group was divided into "very low performance" (n = 36) and "low performance" (n = 35) subgroups. Psychiatric disorders were assessed using K-SADS-PL; reading–writing performance was assessed using OYAB; and cognitive profiles were evaluated with WISC-IV. Parent-report measures included the Conners Parent Rating Scale–Revised Short Form (CPRS-R:S), the Revised Child Anxiety and Depression Scale–Parent Version (RCADS-P), the Behavior Rating Inventory of Executive Function–Parent Form (BRIEF-P), and the Weiss Functional Impairment Rating Scale–Parent Report (WFIRS-P).

Results: According to K-SADS-PL, threshold and subthreshold anxiety symptoms were more frequently observed in the very low performance subgroup. On OYAB reading, writing, comprehension, and total composite scores, the ADHD-only group performed significantly better than the SLD with comorbid ADHD group. Overall performance was highest in the ADHD-only group, followed by the low-performance SLD with comorbid ADHD subgroup, and lowest in the very low-performance SLD with comorbid ADHD subgroup. For comprehension scores, no significant difference was observed between the ADHD-only group and the low-performance SLD with comorbid ADHD subgroup; both groups performed significantly better than the very low-performance subgroup. WISC-IV results demonstrated that perceptual reasoning, working memory, processing speed, and full-scale IQ scores were significantly lower in the SLD with comorbid ADHD group compared with the ADHD-only group; however, these scores were not significantly associated with reading–writing performance levels. No significant group differences were observed in executive function behaviors. Children in the very low performance subgroup exhibited greater functional impairment, particularly in school and learning domains. Functional impairment was significantly predicted by behavioral regulation (an executive function indicator), inattention symptoms, and total anxiety and depression scores. Regression analyses indicated that impairments in working memory and processing speed constitute significant risk factors for learning difficulties and poor reading–writing performance in the comorbid presentation of SLD and ADHD; however, longitudinal studies are required to clarify the direction of these associations.

Conclusions: In conclusion, among children with SLD and comorbid ADHD, low reading–writing performance is associated with increased psychiatric symptoms and more pronounced functional impairment, especially in academic domains. Executive dysfunctions, inattention symptoms, and internalizing symptoms emerge as key risk factors exacerbating functional difficulties. These findings emphasize that early diagnosis and intervention should target not only academic skills but also psychiatric and cognitive processes to support long-term adaptation and quality of life.

Keywords: Attention-deficit/hyperactivity disorder, executive functions, functionality, neurocognitive profile, reading–writing performance, specific learning disorder

AuthorToEditor: ORCID iD: 0009-0002-2828-0609

[Abstract:0389]

Admission Augmentation and Polypharmacy Patterns as Pragmatic Indicators of Treatment Resistance in Patients Hospitalized for a Depressive Episode: Associations With Electroconvulsive Therapy Use

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Selcuk University Faculty of Medicine

Background and Objective: Treatment-resistant depression (TRD) is common among psychiatric inpatients, and neuromodulation (electroconvulsive therapy [ECT], repetitive transcranial magnetic stimulation [rTMS]) is recommended in evidence-based treatment algorithms. In routine inpatient practice, determinants of selecting ECT versus alternative pathways are rarely quantified. We examined whether admission augmentation and polypharmacy markers—pragmatic proxies consistent with possible treatment resistance—were associated with ECT use.

Methods: We conducted a single-center retrospective cohort study of adults (>=18 years) hospitalized for a DSM-5-TR depressive disorder between March 1, 2023 and January 1, 2026. For descriptive reporting, patients were categorized as no neuromodulation, ECT, or rTMS. For inferential analyses, we modeled ECT (yes/no) using rTMS and no neuromodulation as the comparison (non-ECT) to improve model stability given limited ECT events. The primary proxy exposure was antidepressant plus antipsychotic treatment at admission (AD+AP); sensitivity proxies were >=2 psychotropic classes at admission (across antidepressant/antipsychotic/mood stabilizer) and a composite probable-TRD proxy (any antidepressant plus dual antidepressants or augmentation with antipsychotic/mood stabilizer). Continuous variables were summarized as median [IQR] and compared using Kruskal–Wallis tests; within-patient change in Hamilton Depression Rating Scale (HAMD) scores was tested with the Wilcoxon signed-rank test. Categorical variables were compared using χ^2 or Fisher's exact tests. Separate multivariable logistic regression models estimated adjusted odds ratios (aOR, 95% CI) for ECT for each proxy. Because augmentation and polypharmacy may reflect psychotic features or acute suicidal risk, models were additionally adjusted for psychotic features and active suicidal ideation at admission; we also performed a sensitivity analysis excluding psychotic-featured cases. All tests were two-sided (α=0.05).

Results: Eighty-three patients were included (median age 33 [24–47] years; 50.6% female). Neuromodulation was delivered in 36.1% (ECT 20.5% [n=17], rTMS 15.7% [n=13]); 63.9% received no neuromodulation (n=53). Admission HAMD was 23 [17.5–27] and discharge HAMD was 8 [5.5–12] (Wilcoxon p<0.001). Admission HAMD did not differ across groups (Kruskal–Wallis p=0.245), whereas length of stay did (12 [7–16] vs 20 [17–25] vs 14 [9–21] days; p=0.002). At admission, 39.8% received AD+AP; AD+AP was more frequent in the ECT group than in non-ECT patients (64.7% vs 33.3%; Fisher's exact p=0.026; OR=3.67). In models adjusted for age, sex, admission HAMD, psychotic features, and suicidal ideation, AD+AP remained associated with ECT (aOR=4.31, 95% CI 1.28–14.50; p=0.018). Sensitivity proxies showed similar associations (>=2 classes: aOR=3.97, 95% CI 1.19–13.19; p=0.024; probable-TRD proxy: aOR=3.79, 95% CI 1.11–12.93; p=0.034). Excluding patients with psychotic features yielded a comparable estimate for AD+AP (aOR=3.93, 95% CI 1.11–13.90; p=0.034). Conclusions: Among depressive inpatients, ECT use was more strongly associated with admission augmentation and polypharmacy markers than with symptom severity at admission, and these associations persisted after accounting for psychotic features and active suicidal ideation. Pragmatic proxies derived from routine medication reconciliation may help quantify real-world neuromodulation decision pathways and improve transparency of referrals. As an observational, single-center study, causality cannot be inferred and residual confounding is likely. Prospective, multi-center studies with standardized TRD staging (dose, duration, adherence, and failed trials) are needed to validate these markers and to optimize neuromodulation selection in hospitalized depression.

Keywords: Depressive disorders, Neuromodulation, Electroconvulsive therapy (ECT), Repetitive transcranial magnetic stimulation (rTMS)

AuthorToEditor: ORCİD ID: 0009-0007-9285-9195 This study was approved by the Selçuk University Faculty of Medicine Local Ethics Committee (Decision No: 2026/98, Meeting No: 2026/4, Date: 24 February 2026).

[Abstract:0392]

Investigation of Factors Associated with Suicide Attempts in Patients with Major Depressive Disorder

Fatma Nur Meral Altınay, Muhammed Can Altınay, Fatih Ekici

Selcuk University Faculty of Medicine

Background and Objective: Although active suicidal ideation during hospitalization and lifetime suicide attempts are common among psychiatric inpatients, clinical risk formulations may underemphasize illness-course indicators (e.g., episode burden) relative to acute symptom severity. We aimed to identify sociodemographic, familial, and clinical factors associated with active suicidal ideation during the index admission and determine correlates of lifetime history of suicide attempt, and to test independent predictors using multivariable models. Accordingly, we evaluated both outcomes within the inpatient cohort using standardized measures.

Methods: At admission, age, sex, cigarette smoking, alcohol use, illicit substance use, comorbid medical illness, family history of psychiatric illness and suicide attempt, psychiatric comorbidity, presence of psychotic symptoms, number of prior episodes, and Hamilton Depression Rating Scale (HAMD) score were recorded. Outcomes were (i) active suicidal ideation during the index hospitalization (present/absent) and (ii) lifetime history of suicide attempt (present/absent). Bivariate analyses used chi-square tests and independent-samples t tests. Binary logistic regression models were fitted for each outcome; both models included age, sex, psychotic symptoms, admission HAMD score, number of episodes, and alcohol use. B coefficients and p values were reported (two-sided, $p<0.05$). Results: In this observational study, 94 inpatients (47 women, 50.0%; mean age 36.56±15.13 years) were evaluated. Active suicidal ideation was present in 71.3% (n=67), and lifetime suicide attempt history in 44.7% (n=42). Psychiatric comorbidity was observed in 59.5% (n=56), and psychotic symptoms in 6.4% (n=6). Family history of psychiatric illness was 51.1% (n=48), and family history of suicide attempt 11.7% (n=11). In bivariate analyses, lifetime suicide attempt history was not associated with sex, age, family history of psychiatric illness, illicit substance use, or admission HAMD score. In contrast, it was significantly associated with family history of suicide attempt ($\chi^2=6.951$; $p=0.011$), cigarette smoking ($\chi^2=4.952$; $p=0.021$), alcohol use ($\chi^2=6.661$; $p=0.037$), psychiatric comorbidity ($\chi^2=8.566$; $p=0.036$), and a higher number of prior episodes ($t=3.158$; $p=0.002$). Active suicidal ideation was not associated with sex, family history of psychiatric illness, comorbid medical illness, or psychotic symptoms; however, it was associated with alcohol use ($\chi^2=7.684$; $p=0.021$) and showed a borderline association with illicit substance use ($\chi^2=5.718$; $p=0.057$). It was also more frequent among patients with higher admission HAMD scores and more episodes. In multivariable analyses, lifetime suicide attempt history remained independently associated only with number of episodes ($B=0.485$; $p=0.007$; OR≈1.62 per episode). Active suicidal ideation was independently associated with admission HAMD score ($B=0.162$; $p=0.002$; OR≈1.18 per HAMD point); number of episodes showed borderline significance ($B=0.462$; $p=0.051$; OR≈1.59 per episode). Age, sex, psychotic symptoms, and alcohol use were not significant in adjusted models (all $p>0.05$). Conclusions: In this psychiatric inpatient sample, suicidality phenotypes appear to diverge across clinical markers: lifetime suicide attempt history is most strongly related to illness course/episode burden, whereas active suicidal ideation during hospitalization is primarily predicted by acute depressive severity (HAMD). These findings suggest that incorporating longitudinal episode history together with standardized assessment of admission severity—rather than focusing solely on momentary symptom intensity—may support more sensitive risk stratification, closer monitoring, and more targeted intervention planning in inpatient settings.

Keywords: Major depressive disorder, suicidal ideation, suicide attempt, clinical predictors, number of previous episodes

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[Abstract:0401]

The Protective Role of Self-Compassion Against Psychiatric Morbidity in Dermatological Diseases: A Cross-Sectional Pilot Study

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Background and Objective: Chronic dermatological conditions frequently exert a profound psychosocial burden, impairing patients' overall well-being. While the link between skin disease severity and psychiatric morbidity is well-established, the role of protective cognitive constructs remains under-investigated in the field of psychodermatology.

To evaluate the relationships among dermatological quality of life, anxiety, depression, and psychological resilience factors—specifically self-compassion and flourishing—in an outpatient dermatology cohort.

Methods: This cross-sectional pilot study included 23 adult patients (11 males, 12 females; mean age: 39.4) diagnosed with chronic skin diseases including psoriasis, atopic dermatitis, and chronic pruritus. Participants completed a battery of validated psychometric instruments: the Dermatology Life Quality Index (DLQI), Hospital Anxiety and Depression Scale (HADS), Self-Compassion Scale-Short Form (SCS-SF), Brief Resilience Scale (BRS), and Flourishing Scale. Due to the sample size, data were analyzed using non-parametric statistical methods, specifically Spearman's rank correlation and the Mann-Whitney U test.

Results: A significant positive correlation was observed between dermatological life quality impairment (DLQI) and anxiety levels (HADS-A) ($r = 0.448$, $p = 0.032$). Notably, self-compassion exhibited a strong negative correlation with both depression (HADS-D: $r = -0.575$, $p = 0.004$) and anxiety (HADS-A: $r = -0.516$, $p = 0.012$). Furthermore, higher self-compassion was robustly associated with increased flourishing ($r = 0.905$, $p < 0.001$) and psychological resilience ($r = 0.534$, $p = 0.009$). Gender analysis revealed that male patients demonstrated significantly higher baseline psychological resilience compared to females (mean BRS scores: 24.18 vs. 18.17, $p = 0.007$).

Conclusions: Preliminary findings indicate that while dermatological burden directly exacerbates clinical anxiety, self-compassion acts as a potent cognitive buffer, significantly mitigating psychiatric comorbidities. Female patients may be particularly vulnerable to the psychological impact of chronic skin diseases, demonstrating lower baseline resilience. These results underscore the necessity of integrating self-compassion interventions and multidisciplinary approaches into routine psychodermatology practice. Further large-scale, adequately powered studies are warranted to confirm these findings.

Keywords: Anxiety, Depression, Psychodermatology, Quality of Life, Resilience, Self-Compassion

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[Abstract:0408]

Early Changes in Inflammatory Indices Obtained from Hemogram After Electroconvulsive Therapy

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Objective: Electroconvulsive therapy (ECT) is an effective treatment for severe psychiatric disorders, yet its biological mechanisms remain incompletely understood. Neuroimmune and inflammatory pathways may be involved in ECT-related effects, and peripheral blood markers may help characterize this biology in routine clinical settings. Hemogram-derived indices—neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), monocyte-to-lymphocyte ratio (MLR), systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), and aggregate index of systemic inflammation (AISI)—are accessible markers of systemic inflammation. We retrospectively evaluated early pre–post changes in these indices across an ECT course and explored diagnostic heterogeneity.

Methods: We included 130 inpatients (18–65 years) who received ECT at Adnan Menderes University Hospital between March 1, 2021 and December 1, 2025. DSM-5 diagnoses comprised major depressive disorder, bipolar disorder (depressive or manic episode), schizophrenia, or other psychiatric disorders. Complete blood counts were obtained >=24 h before the first ECT session and within 24 h after the final session using standardized morning sampling. NLR, PLR, MLR, SII, SIRI, and AISI were computed from raw hematologic values. Pre–post comparisons were tested with the Wilcoxon signed-rank test in the overall sample and within diagnostic subgroups (depression, bipolar depression, bipolar mania, schizophrenia, and other diagnoses). Significance was set at $p<0.05$. The study was approved by the local Ethics Committee (Approval No: 2026/27), and written informed consent was waived due to the retrospective design.

Results: Mean age was 43.9 ± 14.8 years and 60% were female. The mean number of ECT sessions was 9.35 ± 3.59. In the overall sample, none of the indices changed significantly from pre- to post-ECT (all $p>0.05$), with small effect sizes ($r=0.01$–0.13), suggesting minimal early group-level differences within 24 h after completion of ECT. Subgroup analyses suggested heterogeneity: in the bipolar mania subgroup, NLR decreased significantly after ECT ($p=0.047$). In the “other diagnoses” subgroup, both SIRI and AISI decreased significantly ($p=0.048$ for each). No other within-subgroup pre–post differences reached statistical significance.

Discussion: Hemogram-derived systemic inflammatory indices showed no significant overall early pre–post differences after completion of ECT, although selected indices decreased in specific diagnostic categories. Clinical response–biomarker associations could not be examined because standardized symptom severity scales (e.g., HAM-D, YMRS, PANSS) were not systematically available in retrospective records. Limitations include the retrospective single-center design, potential residual confounding (e.g., medical comorbidities or intercurrent conditions), and reliance on a single early post-ECT sampling time point. Prospective longitudinal studies incorporating validated clinical scales, repeated sampling, and a broader inflammatory biomarker panel (e.g., CRP, IL-6, TNF-α) are warranted to clarify whether inflammatory dynamics track symptom change and to better characterize immunomodulatory patterns associated with ECT.

Keywords: Electroconvulsive therapy, systemic inflammation, hemogram, NLR, bipolar disorder, schizophrenia

AuthorToEditor: This study is a retrospective, ethics committee–approved cohort study (Approval No: 2026/27). The requirement for written informed consent was waived by the ethics committee due to the retrospective design. Orcid: 0009-0004-8742-6238

[Abstract:0413]

Depression Severity as the Primary Predictor of Functional Outcomes: The Role of Coping and Metacognitive Beliefs in Adjustment Disorder

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Background and Objective: Adjustment disorder (AD) is characterized by emotional and behavioral responses to identifiable stressors and is frequently associated with clinically significant functional impairment. Although depressive symptoms are common in AD, the psychological mechanisms that shape functional outcomes remain insufficiently understood. In particular, the relative contributions of coping strategies and metacognitive beliefs to functioning have not been established. This study aimed to investigate the effects of coping strategies and metacognitive beliefs on psychosocial functioning in patients with AD and to examine whether these variables mediate the relationship between depression severity and functional impairment.

Methods: This cross-sectional study included 83 male patients aged 19–30 years diagnosed with AD according to DSM-5 criteria in a psychiatric outpatient setting. The sample exhibited high rates of substance use (63.9%), self-mutilation (71.1%), and suicide attempt history (27.7%). Participants completed the Beck Depression Inventory (BDI), Coping Strategies Questionnaire (CSQ), Metacognitions Questionnaire-30 (MCQ-30), and Functioning Assessment Short Test (FAST). Pearson correlation analyses, multiple linear regression, and bootstrap-based mediation analyses (5000 resamples, 95% BCa CI) were performed. Statistical significance was set at $p < 0.05$.

Results: Depression severity was the strongest predictor of functional impairment ($\beta = 0.42$, $p < 0.001$). Higher coping skills were significantly associated with better functioning ($\beta = –0.28$, $p = 0.010$), while metacognitive beliefs did not independently predict functional outcomes ($p = 0.635$). Among coping subscales, biochemical avoidance showed a strong positive association with dysfunction ($\beta = 0.31$). Neither coping strategies nor metacognitive beliefs significantly mediated the depression–functioning relationship (indirect effects: 95% BCa CI crossing zero). The overall model explained 31.9% of variance in functioning.

Conclusions: Functional impairment in AD is primarily driven by depression severity rather than metacognitive processes. While adaptive coping strategies show protective associations, they do not significantly mediate the effect of depressive symptoms on functioning. Notably, coping through substance use emerged as a significant contributor to dysfunction in this high-risk sample. These findings underscore the need for depression-targeted interventions and coping skills training—particularly addressing biochemical avoidance—in the management of AD. Future longitudinal studies with larger, mixed-gender samples are needed to clarify causal relationships.

Keywords: Adjustment disorder, coping, metacognitive beliefs, depression, functioning, substance use

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[Abstract:0444]

Multidimensional Assessment of Acne Vulgaris: Links Between Psychiatric Symptoms, Disordered Eating, and Dermatologic Severity

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Background and Objective: Acne vulgaris is a common chronic inflammatory skin disease that extends beyond dermatologic symptoms to include psychosocial and behavioral consequences. Although the psychological impact of acne has been acknowledged, the interplay between psychiatric symptoms, eating behaviors, and quality of life remains underexplored.

Objective: To evaluate the associations between psychiatric symptoms, eating behavior patterns, and quality of life among patients with acne vulgaris compared to healthy controls.

Methods: This case-control study included 120 patients with acne vulgaris and 100 sex-matched controls, age-adjusted in the analyses. Dermatologic assessment included the Global Acne Grading System (GAGS), acne scar characteristics, and the Turkish Acne Quality of Life (TAQoL) questionnaire. Psychiatric and behavioral evaluations included the Brief Psychiatric Rating Scale (BPRS), the Eating Attitudes Test (EAT), the Beck Depression and Anxiety Inventories, Night Eating Syndrome parameters, and the Eating Disorder Examination Questionnaire Turkish Short Form-13 item (EDE-Q-13). Statistical analyses were performed using SPSS 25.0. applying Mann-Whitney U, Pearson chi-square, logistic regression, and partial correlation tests with Benjamini-Hochberg correction. Significance was set at $p<0.05$

Results: Patients with acne were predominantly female (80.0%) and significantly younger than controls (median age, 20 vs. 24 years, $p < 0.001$). A family history of acne was the strongest predictor of disease (OR = 5.5, 95% CI: 2.4-13.1, $p < 0.001$), with several familial cases increasing the risk up to 23-fold. Compared with controls, acne patients had significantly higher scores for psychiatric symptoms (BPRS, $p<0.001$), depressive ($p<0.001$) and anxiety symptoms ($p<0.001$), and disordered eating (EAT, $p=0.001$). Logistic regression identified binge-eating behavior (as measured by the EDE-Q-13 subscale) as an independent predictor of acne (OR = 2.4, $p < 0.001$). Partial correlation analyses revealed that depression ($r =0.25$, $p = 0.008$), anxiety ($r = 0.29$, $p = 0.002$), binge eating ($r = 0.23$, $p = 0.013$), and body dissatisfaction ($r = 0.25$, $p = 0.009$) were all positively correlated with poorer acne quality of life—night eating duration associated with the extent of trunk involvement ($r=0.20$, $p=0.038$).

Conclusion: Acne vulgaris is linked to psychiatric distress and impaired quality of life, supporting multidisciplinary management approaches.

Keywords: Acne Vulgaris, Dermatologic severity, Disordered eating, psychiatric symptoms, Quality of Life

AuthorToEditor: 0000-0003-0412-365X

Oral Presentation Award

[Abstract:0093]

Guanfacine-Associated Stuttering in Autism Spectrum Disorder: The First Reported Case in the Literature

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Background: Guanfacine is a selective alpha-2 adrenergic agonist frequently prescribed for attention-deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD) in children and adolescents. Commonly reported adverse effects include somnolence, headache, appetite changes, and constipation. However, speech fluency disturbances have not been previously reported.

Case Presentation: We report the case of a 14-year-old boy diagnosed with ASD at the age of three. He was followed in the child psychiatry clinic for behavioral problems, hyperactivity, sleep disturbances, and impulsivity. He had previously received atomoxetine, methylphenidate, and aripiprazole, all of which were discontinued due to adverse effects. While on risperidone (3 mg/day) and valproic acid (500 mg/day), worsening symptoms prompted initiation of guanfacine at 1 mg/day. On the second day of guanfacine therapy, the patient developed prominent stuttering. Neurological examination, electroencephalography, and brain magnetic resonance imaging were normal, and no environmental triggers were identified. The stuttering completely resolved within a few days after discontinuation of guanfacine.

Discussion: Guanfacine is known to exert regulatory effects on attention and impulsivity via the prefrontal cortex. However, its potential influence on speech fluency has not been documented. The temporal relationship between guanfacine initiation and the onset of stuttering, followed by complete resolution after drug discontinuation, suggests a strong causal association. To the best of our knowledge, no previous reports have described stuttering as an adverse effect of guanfacine, rendering this case unique and clinically significant.

Conclusion: Clinicians should be aware that guanfacine may rarely induce stuttering in children with ASD and/or ADHD. Newly emerging speech disturbances in patients receiving guanfacine should prompt consideration of a drug-related adverse effect. Reporting such cases to pharmacovigilance systems is essential to improve understanding of this potential association.

Keywords: Guanfacine, Stuttering, Autism Spectrum Disorder, Adverse Effect

Table 1. Medication Timeline

Medication	Dose	Duration	Adverse Effect	Outcome
Atomoxetine	25 mg/day	2 months	Irritability	Discontinued
Methylphenidate	20 mg/day	1 month	Appetite loss	Discontinued
Aripiprazole	5 mg/day	3 months	Sedation	Discontinued
Risperidone	3 mg/day	Ongoing	None	Continued
Valproic acid	500 mg/day	Ongoing	None	Continued
Guanfacine (XR)	1 mg/day	2 days	Stuttering	Resolved after discontinuation

Summary of the patient's previous and current pharmacological treatments, including medication names, dosages, treatment durations, observed adverse effects, and clinical outcomes. The table highlights that stuttering emerged only after the initiation of guanfacine and resolved promptly following its discontinuation.

Table 2. Mechanistic Hypothesis: Neurotransmitter Systems Potentially Involved

System	Effect of Guanfacine	Potential Contribution to Stuttering
Noradrenergic	α2A receptor agonism → ↓ norepinephrine release	Reduced cortical inhibition may affect fluency control
Dopaminergic	Indirect modulation via prefrontal cortex	Altered basal ganglia output → speech motor dysfluency
GABAergic	Secondary interaction possible	Disinhibition of motor circuits

Proposed neurochemical mechanisms that may underlie guanfacine-associated stuttering. The table outlines the possible roles of noradrenergic, dopaminergic, and GABAergic systems in modulating speech fluency through alterations in cortical and subcortical pathways.

AuthorToEditor: Burak Kamış ORCID ID: 0000-0003-2140-5448 Presentation: This case report was presented as an oral presentation at the 10th National Social Pediatrics Symposium, held on October 9–11, 2025.

[Abstract:0096]

Social and Psychiatric Risk Profiles of Juveniles Involved in Sexual Offenses: Analysis of 82 Forensic Cases

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Background and Objective: Juvenile sexual offending is influenced by social, familial, and psychiatric factors, yet systematic data from non-Western forensic settings are limited. This study aimed to describe the sociodemographic, psychosocial, and psychiatric characteristics of juveniles referred for forensic psychiatric evaluation in Turkey and to examine variables associated with the nature of sexual abuse (act type, penetration status, and frequency) and additional non-sexual offending.

Methods: This retrospective cross-sectional study included 82 male juveniles evaluated between January 2022 and July 2023 by the Sixth Specialization Board of the Council of Forensic Medicine in Istanbul, Turkey, following allegations of sexual offenses. Data were collected using a standardized sociodemographic and clinical extraction form, integrating information from official forensic files and routine forensic psychiatric interviews. Sexual acts were categorized as penetrative (oral, vaginal, anal, or digital) or non-penetrative, and cases were classified as intrafamilial (relatives or cohabiting) or extrafamilial. Descriptive and comparative analyses were conducted using chi-square and Fisher's exact tests.

Ethics approval was obtained from the Education and Scientific Research Commission of the Council of Forensic Medicine (Ref. No: 1162; October 4, 2021).

Results: The mean age of the juveniles was 15.6 ± 1.2 years (range: 13–17.9). Most lived in nuclear families (69.5%) and low- to middle-income households (59.7%); parental separation was noted in 30.5%. Psychiatric diagnoses were present in 9.8%, predominantly attention-deficit/hyperactivity disorder and conduct disorder. Cigarette use was reported in 30.5%, while no illicit drug use was detected. Penetration occurred in 72% of cases and did not differ significantly between intrafamilial (64.5%) and extrafamilial (76.5%) contexts ($p = 0.312$). The most frequent act types were rubbing (37.8%), touching (34.1%), and anal and vaginal contact (45.1% and 24.4%, respectively). Repeated abuse (>=2 episodes) was observed in 58.5% and was significantly higher in intrafamilial than extrafamilial cases (74.2% vs. 49.0%, $p = 0.037$). Victims were female in 56.1% and male in 42.7% of cases; one case involved two victims. Penetration was more frequent among male victims ($p = 0.006$). Juveniles with a family history of crime were more likely to commit additional non-sexual offenses ($p = 0.035$, OR = 4.83).

Conclusions: The findings delineate a distinct psychosocial-forensic profile of juvenile sexual offenders in Turkey. High rates of penetrative acts and significantly greater repetition in intrafamilial cases emphasize the influence of family-system risks on offense persistence. The higher penetration rates among male victims highlight the need for gender-sensitive prevention and assessment approaches. Moreover, the link between familial criminality and broader offending patterns underscores the importance of integrated interventions that address both individual vulnerabilities and intergenerational risk transmission.

Keywords: Family violence, Forensic psychiatry, Juvenile delinquency, Risk factors, Sexual offenses, Turkey

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[Abstract:0100]

The Effectiveness of Nonpharmacological vs. Pharmacological Interventions in Major Depression, Bipolar Disorder, and Schizophrenia

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Arabian Gulf University

Background and Objective: Major depressive disorder (MDD), bipolar disorder (BD), and schizophrenia (SCZ) are severe mental illnesses affecting 25% of adults annually, causing productivity losses and high costs. While pharmacological treatments dominate for these disorders, nonpharmacological approaches are gaining attention but remain under researched in Bahrain. This study aimed to evaluate and compare the prevalence and outcomes of pharmacological versus nonpharmacological interventions in Bahrain.

Methods: This study employed an observational cross-sectional facility-based design. It was conducted at the governmental psychiatric hospital in Salmaniya, Bahrain, from June 2024 to July 2024. The study involved 166 adult patients diagnosed with MDD, BD, or SCZ who were selected through simple random sampling. Data, which included demographic data, disease history, pharmacological treatment, nonpharmacological management, and patient outcomes, were collected via the Global Assessment of Functioning (GAF-Clinical) and the World Health Organization Quality of Life (WHOQOL) questionnaires.

Results: Pharmacological-only treatment was used by 68% of the participants, 23% received combined treatments, and 3% received nonpharmacological treatment alone. There were significant differences in the GAF scores, with the combined treatment group ($M = 61.05$) scoring higher than the pharmacological-only group ($M = 55.96$, $p = 0.016$). Patients on fewer medications (3 or fewer) generally had higher GAF scores, indicating better functioning. The WHOQOL scores were not significantly different across the treatment groups.

Conclusions: Compared with pharmacological treatment alone, the combination of pharmacological and nonpharmacological treatments significantly improved psychological, social, and occupational functioning, as indicated by higher Global Assessment of Functioning (GAF) scores. Notably, the number 3 of medications prescribed did not correlate with the GAF score, suggesting that the type of therapy influences treatment efficacy more than the quantity of medication used alone does.

Keywords: Mental Disorders, Pharmacological Interventions, Nonpharmacological Interventions, Treatment Outcomes, Bahrain

[Abstract:0109]

Lamotrigine Co-Administration May Reduce Effectiveness of Intravenous Ketamine in the Treatment of Major Depressive Episodes

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Background and Objective:

Intravenous (IV) ketamine has a therapeutic window. Thus, coadministration with a drug that may share a similar mechanism of action may alter the effective dose. Both ketamine and lamotrigine inhibit the N-methyl-D-aspartate (NMDA)-associated sodium channel in an activity-dependent manner so that co-administration of the two agents may alter the apparent effective dose of ketamine in treatment for depression. There are data suggesting that coadministration of ketamine and lamotrigine may reduce the effectiveness of the ketamine treatment. This was investigated in a new cohort of IV ketamine treated patients.

Methods:
We retrospectively evaluated Montgomery Åsperg Depression Rating Scale (MADRS) scores in patients receiving IV ketamine with (n = 7) and without (n = 49) co-administration with lamotrigine. The extent of improvement from baseline to 24 hours after the third round of IV ketamine was compared with an unpaired, two-tailed t-test.

Results:

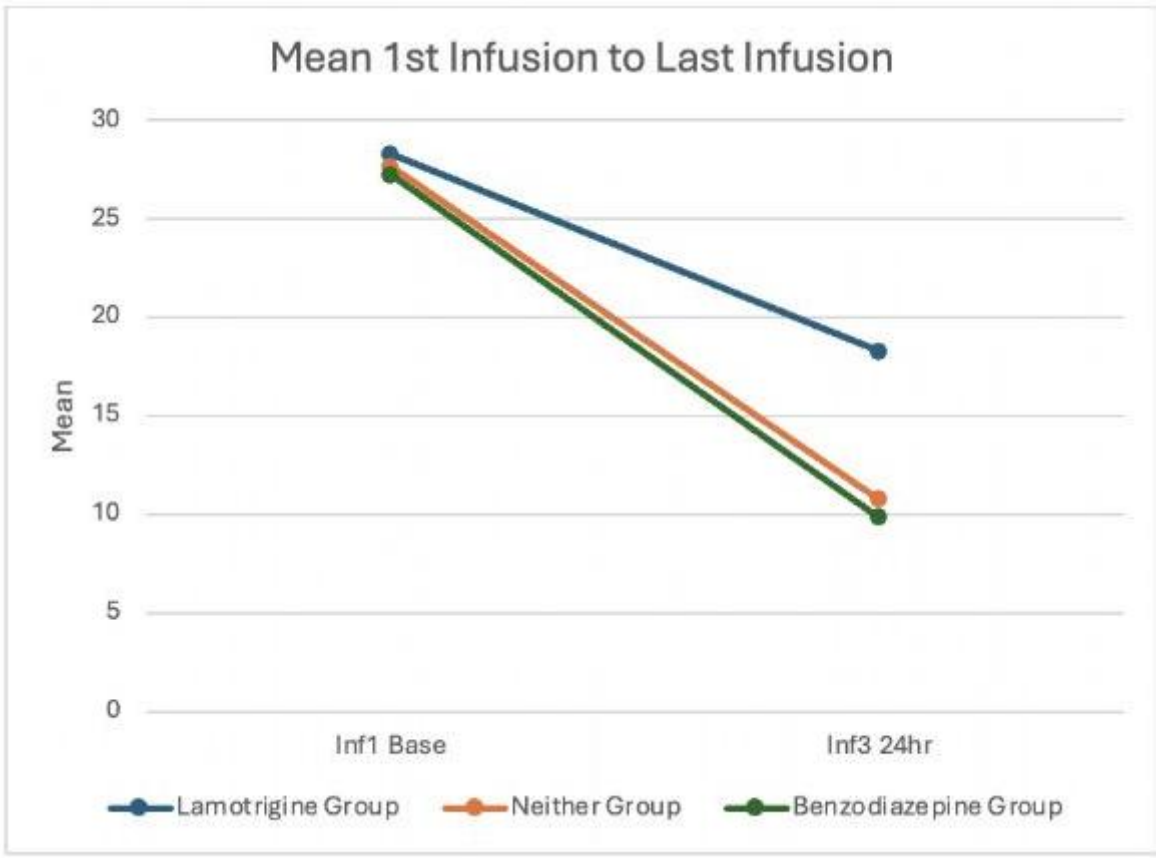
Patients receiving lamotrigine (baseline 28.3 ± SD 11.4; after 3rd treatment 18.3 ± 31.4; ns) did not improved to the same extent as patients not receiving lamotrigine (baseline 27.8 ± SD 6.2; after 3rd treatment 10.8 ± 9.0; $P < 0.0001$). The extent of improvement was non-significantly greater in patients not receiving concomitant lamotrigine (total improvement 16.9 ± 9.0 vs. 10.0 ± 1.38, $P = 0.07$, $t = 2.01$).

Conclusions:

While the data are limited by the small sample of lamotrigine -co-treated patients, they support the concept of a therapeutic window for ketamine, and possibly also for lamotrigine. Co-administration of the two medications should be avoided or minimized.

Keywords: Ketamine, Lamotrigine, Major Depression, Treatment response

Difference between baseline and final evaluation in patients treated with IV ketamine (0.5 mg/kg) and cotreated with lamotrigine (LAM, n = 7), a benzodiazepine (BNZ, n = 18), and neither (n = 49).



AuthorToEditor: Irem Hacisalihoglu Aydin Orcid No: 0000-0001-5768-8553 Yazarların tam listesini ve 1500 kelimelik brief reportu içeren döküman burkon turizm elif.gogdas@burkon.com mailine iletilmiştir. Saygılarımla

[Abstract:0118]

Reducing Peer Bullying in Children Who Stutter Through an ACT-Informed Group Intervention: Preliminary Findings

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[1]Dr Funda Öztürk Child and Adult Psychiatry, Private Clinic; Konya Karatay University
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Background and Objective: Peer bullying is common among children who stutter and is associated with increased psychosocial risk. Although psychosocial interventions are increasingly incorporated into stuttering therapy, limited evidence exists regarding interventions that explicitly target peer bullying in young children who stutter (CWS). The present pilot study aimed to examine the feasibility and preliminary outcomes of an Acceptance and Commitment Therapy (ACT)–based group intervention designed to reduce peer bullying experiences and enhance psychosocial functioning in early school-age CWS.

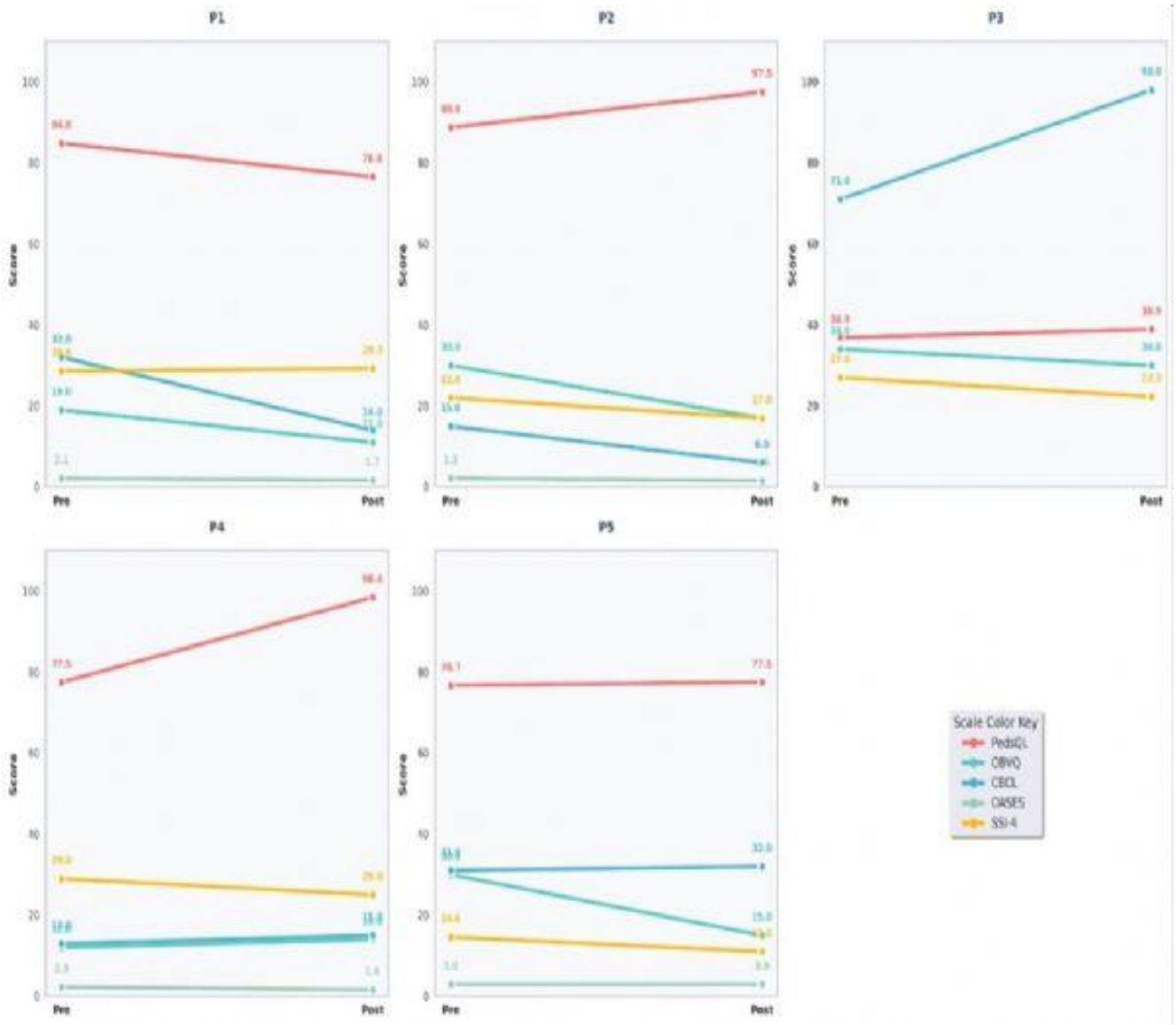
Methods: Five first- and second-grade children aged 7–8 years who stutter participated in an eight-week group intervention. The program integrated bullying-focused psychoeducation with structured coping and assertiveness strategies and core ACT components, including present-moment awareness, acceptance, cognitive defusion, self-as-context, values clarification, and committed action. Pre- and post-intervention assessments included measures of stuttering severity (SSI-4), the impact of stuttering on daily life (OASES-S), peer bullying experiences (OBVQ), emotional and behavioral functioning (CBCL), and health-related quality of life (PedsQL).

Results: Most participants demonstrated reductions in stuttering severity and in the overall impact of stuttering on daily life following the intervention. Peer bullying scores decreased for the majority of children, and improvements in health-related quality of life were observed across the sample. Individual variability in treatment response was evident, which is consistent with the exploratory nature and small sample size of pilot studies.

Conclusions: The findings provide preliminary support for the feasibility and potential utility of integrating ACT principles with bullying-focused strategies in group interventions for young CWS. Emphasizing psychological flexibility, mindfulness, and adaptive coping skills within a group format may represent a promising approach for addressing both fluency-related challenges and broader psychosocial difficulties associated with peer bullying.

Keywords: ACT, bullying, group intervention, mindfulness, psychological flexibility, stuttering

Figure 1. Individual pre- and post-intervention scores across outcome measures for P1-P5



AuthorToEditor: 0000-0002-6628-6633 This presentation reports findings from a pilot ACT-based group intervention targeting peer bullying and psychosocial functioning in young children who stutter. The program was developed and implemented through close collaboration between speech and language therapy and child and adolescent psychiatry, ensuring an integrated focus on both fluency-related and psychosocial processes. By combining bullying-focused psychoeducation with core ACT principles, the study addresses an important clinical gap and offers preliminary evidence for a feasible, multidisciplinary group-based intervention.

[Abstract:0123]

Clinical and Inflammatory Determinants of Length of Hospital Stay in Patients with Bipolar Disorder Manic Episodes

Pelin Göksel

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Background and Objective: This study aimed to evaluate the clinical and inflammatory predictors of hospitalization duration in patients under 65 years old diagnosed with Bipolar Disorder- Manic Episode, with a particular focus on the association between the neutrophil-to-lymphocyte ratio (NLR) and length of stay.

Methods: Patients hospitalized with a diagnosis of manic episod and younger than 65 years were included. Missing treatment variables (lithium, antipsychotic, antidepressant, mood stabilizer, combined therapy) were completed based on medication records. Hematological parameters were used to calculate NLR, PLR, and the Systemic Immune-Inflammation Index (SII). Outliers were removed using the IQR method. Inflammatory markers were log-transformed. A multivariable linear regression model was constructed to identify predictors of hospitalization duration. Regression model assumptions, including normality and multicollinearity, were assessed. Independent variables included log-NLR, log-PLR, log-SII, age, sex, combined treatment, and lithium use. Hematological parameters were obtained at hospital admission before treatment initiation. Approval was obtained from the Ondokuz Mayıs University Clinical Research Ethics Committee (Date: 18.12.2025. No: 2025/618)

Results: Seventy-five patients were included in the final analysis. The model demonstrated that log-NLR was a significant predictor of hospitalization duration ($\beta = 31.75$, $p = 0.027$). Other variables, including log-PLR, log-SII, age, sex, combined treatment, and lithium use, did not show statistically significant associations with length of stay ($p > 0.05$). Lithium showed a trend toward shorter hospitalization, while combined therapy showed a trend toward longer stay, though neither reached significance.

Conclusion: This study identifies NLR as a significant inflammatory marker associated with prolonged hospitalization in patients under 65 with manic episod patients. Higher inflammatory burden, reflected by elevated NLR, is linked to longer inpatient treatment duration. In contrast, SII and PLR did not make a meaningful contribution to predicting the clinical course. These findings suggest that NLR can be used as a practical and accessible biomarker to predict hospitalization outcomes in patients with manic episodes. Limitations include the retrospective design, modest sample size, and lack of standardized symptom severity measures.

Keywords: Manic attack, bipolar disorder, length of hospital, inflammatory determinants

Multivariable Linear Regression Analysis of Predictors of Length of Hospital Stay

Variable	Coefficient (β)	p- value	Interpretation
log-NLR	+31.75	0.027	Significant — Higher NLR is associated with longer hospitalization
log-SII	+3.61	0.74	Not significant
log-PLR	-4.59	0.55	Not significant
Age	+0.26	0.15	Trend toward significance, but not statistically significant
Gender	+2.70	0.58	Not significant
Combined treatment	+3.70	0.40	Not significant
Lithium Use	-8.00	0.19	Trend toward shorter hospitalization, but not significant

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[Abstract:0136]

Psychotropic Recommendations Following Child and Adolescent Psychiatry Consultation in a Pediatric Burn Unit: A Five-Year Retrospective Review

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Background and Objective: Pediatric burns represent a multidimensional crisis that extends beyond tissue injury. Owing to their traumatic nature, repeated procedures, pain, and prolonged hospitalization, they impose a substantial psychosocial burden on both children and families. Consequently, mental health needs frequently emerge in pediatric burn care due to emotional, cognitive, and behavioral difficulties. This study aimed to describe the five-year pattern of child and adolescent psychiatry consultations requested from a pediatric burn unit, focusing on consultation indications, clinical impressions/preliminary diagnoses, recommended interventions, and reconsultation

Methods: A single-center, retrospective descriptive design was used. Electronic medical records of inpatients hospitalized in the pediatric burn unit between November 2020 and November 2025 who received a child and adolescent psychiatry consultation were reviewed. We extracted sociodemographic data, burn-related characteristics, surgical information, length of stay, the hospital day on which consultation was requested, consultation indications, clinical impressions/preliminary diagnoses, recommended psychosocial and pharmacological interventions, and the number of reconsultations. Descriptive statistics were reported.

Results: A total of 174 consultation requests were identified for 106 hospitalizations. Mean age was 105.32±60.42 months (min–max: 13–216 months). The most common burn etiologies were scalds (46.7%, n=50) and flame burns (43.9%, n=47). Burns were extensive in 78.5% (n=84) of cases, and the most frequently reported burn depth was second-degree (70.1%, n=75). Facial involvement was present in 44.3% (n=47/106) and genital involvement in 25.5% (n=27/106).

The most frequent consultation indication was agitation and nonadherence to treatment (48.6%, n=52). Regarding clinical impressions/preliminary diagnoses, adjustment disorder (41.1%, n=44) and general psychiatric evaluation (39.3%, n=42) were the two most common categories. Acute stress disorder + PTSD co-occurred in 11.2% (n=12); anxiety + depression in 2.8% (n=3); substance use in 3.7% (n=4); and delirium in 0.9% (n=1).

Among recommended interventions, nonpharmacological approaches were most common (51.4%, n=55). Pharmacological recommendations included antipsychotics (27.3%, n=29), antihistamines (10.3%, n=11), benzodiazepines (6.5%, n=7), and SSRIs (3.7%, n=4). Among cases receiving pharmacological treatment/recommendations, the most frequently used agents were risperidone (54.9%, n=28/51) and hydroxyzine (21.5%, n=11/51).

A need for reconsultation was observed in 30.8% (n=33) of cases. The most common pattern was one reconsultation (22.4%, n=24); fewer cases had two (n=4), three (n=3), or seven (n=1) reconsultations, and one case had 19. The number of reconsultations was positively associated with length of stay (Spearman $\rho=0.393$, $p<0.001$). Reconsultations also correlated positively with number of surgeries ($\rho=0.439$, $p<0.001$) and burn percentage ($\rho=0.347$, $p<0.001$). In addition, length of stay showed strong correlations with number of surgeries ($\rho=0.787$, $p<0.001$) and burn percentage ($\rho=0.683$, $p<0.001$).

Conclusions: This five-year review indicates a substantial demand for psychiatric consultation among pediatric burn inpatients, with requests largely centered on behavioral regulation and treatment adherence. Clinical impressions most commonly involved adjustment difficulties/adjustment disorder, acute stress reactions, and less frequently delirium. Reconsultation in approximately one-third of cases suggests that a single consultation may often be insufficient in this setting. These findings support integrating mental health care into pediatric burn

management through an early, longitudinal, and follow-up–oriented consultation–liaison model, rather than relying solely on ad hoc referrals.

Keywords: Child and adolescent psychiatry, Pediatric burn care, Psychiatric consultation, Reconcultation

Demographic and burn-related characteristics of hospitalizations (N=106)

Variable	
Age (months), mean ± SD (min–max)	105.32 ± 60.42 (13–216)
Sex (Male/Female), n (%)	63 (59.4) / 43 (40.6)
Burn etiology, n (%)	
– Scald	50 (46.7)
– Flame	47 (43.9)
– Contact	4 (3.7)
– Electrical	3 (2.8)
– Chemical	2 (1.9)
Extent, n (%)	
– Extensive	84 (78.5)
– Localized	22 (20.6)
Burn depth, n (%)	
– Second-degree	75 (70.1)
– Mixed second- and third-degree	28 (26.2)
– Third-degree	3 (2.8)
Burn location	
– Face involvement, n (%)	47/106 (44.3)
– Genital involvement, n (%)	27/106 (25.5)
– Trunk involvement, n (%)	58/106 (54.0)
– Upper extremity involvement, n (%)	70/106 (66.0)
– Lower extremity involvement, n (%)	64/106 (60.0)
Underwent surgery, n (%)	65 (60.7)
Length of hospital stay (days), mean ± SD (min–max)	29.88 ± 25.23 (2–139)
Consultation request day, mean ± SD (min–max)	9.84 ± 12.40 (1–97)
Discharge status, n (%)	Alive 104 (97.2) / Deceased 2 (1.9)
Functional impairment, n (%)	26 (24.3)

Spearman's rank correlations

Association	**ρ**	**p**
Number of reconsultations ↔ Length of hospital stay	0.393	<0.001
Number of reconsultations ↔ Number of surgeries	0.439	<0.001
Number of reconsultations ↔ Burn percentage (TBSA)	0.347	<0.001
Length of hospital stay ↔ Number of surgeries	0.787	<0.001
Length of hospital stay ↔ Burn percentage (TBSA)	0.683	<0.001

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[Abstract:0149]

"Use of Propranolol in Non-Akathisic Pediatric Psychiatric Patients: A Retrospective Evaluation"

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Objective: Propranolol, a non-selective beta-adrenergic antagonist, is traditionally used in pediatric psychiatry for performance anxiety and antipsychotic-induced akathisia. This study explores the therapeutic potential of propranolol in child and adolescent patients exhibiting emotional dysregulation, impulsive aggression, and autonomic hyperarousal in the absence of akathisia.

Methods: Medical records of pediatric patients prescribed propranolol (10–40 mg/day) for various psychiatric indications were retrospectively reviewed. Demographic characteristics, primary diagnoses, treatment durations, and clinical outcomes were analyzed, specifically excluding patients with drug-induced akathisia.

Results: Preliminary findings indicate that propranolol contributes significantly to symptom regulation, particularly in cases involving severe emotional reactivity and physiological anxiety.

Case 1: A 15-year-old male diagnosed with ADHD, Oppositional Defiant Disorder (ODD), and Conduct Disorder. Despite primary treatments, persistent impulsive aggression remained a challenge. The addition of 40 mg/day propranolol led to a marked improvement in anger management and a reduction in behavioral outbursts.

Case 2: A 16-year-old male with Bipolar Disorder, followed for severe anger control problems and frequent hospital admissions. Following the initiation of 40 mg/day propranolol as an adjunctive therapy, a significant decrease in the frequency of explosive outbursts (anger attacks) was observed, resulting in improved clinical stability and fewer emergency admissions.

Case 3: A 16-year-old female with Major Depressive Disorder and ADHD. She presented with prominent physical symptoms of anxiety (tachycardia, tremors) that exacerbated her depressive state. Adding 20 mg/day propranolol effectively targeted these autonomic symptoms, leading to a substantial reduction in overall anxiolytic distress and improved functional recovery.

Overall, the treatment was well-tolerated across all cases with minimal adverse effects. The most pronounced clinical responses were observed in patients whose behavioral dysregulation was driven by peripheral physiological arousal.

Conclusions: These findings suggest that propranolol acts as a potent stabilizing agent beyond its conventional indications. Neureobiologically, by moderating peripheral adrenergic activity, propranolol may decrease interoceptive feedback to the insular cortex, subsequently attenuating amygdala hyperactivation. This mechanism helps inhibit the "fight-or-flight" response and promotes physiological calmness, facilitating better emotional regulation in pediatric populations. Further prospective, controlled studies are warranted to evaluate long-term efficacy and clarify these underlying neurobiological pathways.

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Keywords: Propranolol, Child and Adolescent Psychiatry, Emotional dysregulation

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[Abstract:0160]

Clinical Display and Comorbid Diseases of the Posttraumatic Embitterment Disorder in Anxiety Outpatient Clinic

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Background and Objective: If there is a life, then there is a stressor. When the numbers and the kinds of stressors increase, both our perception and our reactions depending on this state will change. Feeling in a situation of desperate, helpless and irrevocable, we might lose our hopes. If this situation is perceived as weakness, injustice, the emotions of anger and heartsickness will appear. If this case continues too long, it will turn into the posttraumatic embitterment disorder that causes the loss of physical and mental functionality, which ruins work, family and social life of the person.

Methods: The patients who have come to the anxiety outpatient clinic have all been evaluated by considering the items of standardazing the posttraumatic embitterment disorder. Some self evaluation measures have been carried out at a sample that consists of 344 people afterward

Results: Posttraumatic embitterment disorder has been determined as the proportions of %47,3 and %4,6 for diagnosed and undiagnosed DSM-5 symptomatic people who have come to the anxiety clinic. This rate of %51,9 is quite high in total. Apart from this, %0,8 of distressed emotion which is not pathological has been determined for some healthy volunteers. Some significant connections of embitterment disorder have been detected between the women who are housewives and the men who work in private companies.There is a positive correlation between the symptoms and the intensity of the posttraumatic embitterment disorder.In this study, the posttraumatic embitterment disorder has been determined as quite common among the people who was examined in the anxiety outpatient clinic. The identical diagnosis of higher DSM-5 are similar to the works done in other countries. This is significant as the first work in Turkey which studies the posttraumatic embitterment disorder in terms of psychiatric experience. There are some differences and similarities due to cultural diversity between literature and demographic features. It is understood that when the variety and number of stressors increase, the posttraumatic embitterment disorder will increase. And also the change in being perceived of stressor according to gender has come into prominence because of cultural diversity. The improvement of posttraumatic embitterment disorder might be risky for women who are housewives and men who work in private companies. The positive correlation of the posttraumatic embitterment disorder with disease duration explains the significance of chronicity in functionality. The rate of higher posttraumatic embitterment disorder on symptomatic people who are not diagnosed as DSM-5 might indicate the population whose treatment is inadequate in the clinic.

Conclusions: The posttraumatic embitterment disorder is common in our clinical experience. It should be considered as comorbid or a single diagnosis for this disorder and treatment of psychopathology. There are some spesific therapeutic interventions for the posttraumatic embitterment disorder, which are thought to increase the success of treatment by being added to the therapy plan.

Keywords: Anxiety, comorbidity, disorder, psychiatric outpatient

[Abstract:0209]

Evaluation of the Relationship Between Repetitive Negative Thinking and Quality of Life in Women Diagnosed with Endometriosis

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Background and Objective: Endometriosis is a chronic condition that markedly impairs women's quality of life and daily functioning, mainly due to pelvic pain and related psychological factors. Beyond physical symptoms, anxiety, psychological inflexibility, and repetitive negative thinking may contribute to disease-related disability. This study aimed to examine the relationships between quality of life and functioning and pelvic pain severity, anxiety, psychological inflexibility, and repetitive negative thinking in women with endometriosis.

Methods: This single-center, observational, cross-sectional study included women aged >=18 years diagnosed with endometriosis who attended an endometriosis and pelvic pain outpatient clinic. Data were collected via same-day face-to-face interviews and routine gynecological examinations. Sociodemographic and clinical data were recorded using a structured form. Quality of life and functioning were assessed with the SF-36 and EHP-30. Anxiety and depression were measured using HADS. Repetitive negative thinking was evaluated with the Penn State Worry Questionnaire and Ruminative Responses Scale, and psychological inflexibility with the AAQ-II. Pelvic pain severity was assessed using the Psychological Inflexibility in Pain Scale.

Results: The study sample consisted of 111 participants with a mean age of 34.7±7.4 years Most participants were married (59.5%), 33.3% were single, and 7.2% were divorced. More than half of the sample had a university degree or higher education (54.1%), followed by high school (27.9%), primary school (10.8%), and middle school education (7.2%).Non-parametric correlation analyses demonstrated that disease-related functioning and quality of life measured by the EHP were strongly associated with pelvic pain severity ($r = 0.67$, $p < .001$), psychological inflexibility (AAQ-II) ($r = 0.41$, $p < .001$), rumination ($r = 0.31$, $p < .001$), overall anxiety and depression (HADS total score) ($r = 0.51$, $p < .001$), and self-criticism ($r = 0.28$, $p = .003$).General health perception (GH) showed weaker associations. It was significantly related to pain severity ($r = -0.22$, $p = .018$) and overall anxiety and depression ($r = -0.24$, $p = .010$), whereas its associations with psychological inflexibility (AAQ-II) ($r = -0.18$, $p = .058$) and self-criticism ($r = -0.03$, $p = .729$) were not statistically significant. In a multiple regression model including pain severity, rumination, anxiety, depression, and psychological inflexibility (AAQ-II), none of the variables independently predicted general health perception ($p > .05$). In contrast, a multiple linear regression model predicting disease-related functioning (EHP) was significant and explained 53.3% of the variance ($R = 0.73$, $R^2 = 0.533$). Higher pain severity ($\beta = 0.75$, $p < .001$), greater psychological distress ($\beta = 1.54$, $p < .001$), increased rumination ($\beta = 0.21$, $p = .032$), and higher self-criticism ($\beta = 0.42$, $p = .034$) independently predicted poorer functioning, while psychological inflexibility was not a significant independent predictor.

Conclusions: These findings indicate that disease-related functioning and quality of life in women with endometriosis are closely associated with pelvic pain severity and psychological factors, particularly anxiety, depression, rumination, self-criticism, and psychological inflexibility. General health perception showed weaker and less consistent relationships. Addressing both pain and maladaptive cognitive-emotional processes may be essential for improving functional outcomes and quality of life in women with endometriosis.

Keywords: Anxiety, depression, endometriosis, pelvic pain, psychological inflexibility

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[Abstract:0215]

Emotional Blunting Associated With Antidepressant Treatment: A Cross-Sectional Study

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Emotional blunting is defined as a common adverse effect reported by patients during antidepressant treatment and may negatively affect treatment adherence, functioning, and quality of life. The aim of this study was to evaluate the level of emotional blunting in patients using antidepressants and to examine its association with antidepressant class, sex, and medication dose.

Methods

This cross-sectional study included a total of 97 patients receiving antidepressant treatment. Emotional blunting was assessed using a 6-item emotional blunting scale. Internal consistency of the scale was evaluated using Cronbach's alpha coefficient. Participants were categorized according to antidepressant class as SSRI (n=83) and SNRI (n=14) users. The SSRI group was additionally compared across its subgroups. Medication doses were converted into equivalent doses, and correlation analyses were performed between dose and emotional blunting subscale scores. The study was approved by the relevant ethics committee (TBAEK-80, dated 02.01.2025). All statistical analyses were conducted using appropriate methods.

Results

Mean (SD) age was 43.2 (17.6) years, 79 patients were female (77.5%). Majority of the patients were diagnosed with major depressive disorder (56.7%), whereas other major diagnoses included anxiety disorders (35%) and obsessive compulsive disorder (5.1%). Most used SSRI was escitalopram (39.7%), whereas other SSRIs included sertraline (33.7%), fluoxetine (15.6%), paroxetine (9.7%) and vortioxetine (1.3%). Reliability analysis of the emotional blunting scale showed item-level Cronbach's alpha values ranging between 0.72 and 0.74, with a total Cronbach's alpha coefficient of 0.74. No statistically significant difference was found between SSRI and SNRI groups in terms of total emotional blunting scores. The durations of antidepressant use were similar in both groups (SSRI subgroup 29.3 (3.1); SNRI subgroup 33.7 (8.7) months, p=0.66). Sex-based comparisons revealed that male patients had significantly higher scores than female patients in the indifference related to medication subscale (3.04 vs. 2.26; p=0.004), the perceived emotional change induced by medication subscale (1.96 vs. 1.41; p=0.01), and total emotional blunting scores (12.83 vs. 10.62; p=0.01). No significant differences were observed when SSRIs were compared across subgroups (vortioxetine group was excluded due to small sample size). ANOVA results revealed no significant difference in terms of duration of SSRI use across SSRI subgroups (p=0.31). Correlation analysis based on equivalent medication doses demonstrated that higher doses were significantly associated with increased scores on emotional experiencing (r=0.200; p=0.04).

Conclusion

This study demonstrates that emotional blunting associated with antidepressant treatment may occur independently of antidepressant class and appears to be more pronounced in male patients. Additionally, the findings suggest a potential association between emotional experiences and medication dose. Systematic assessment of emotional blunting during antidepressant treatment may be clinically important for improving treatment adherence and guiding individualized treatment strategies.

Keywords: Antidepressants, Emotional blunting, Side effects, SNRI, SSRI

AuthorToEditor: This study aims to examine the often overlooked adverse effect of emotional blunting during antidepressant treatment in relation to clinical and demographic variables. The findings are expected to contribute to improving treatment adherence and supporting individualized treatment approaches in clinical practice. Thank you for your consideration. ORCID: 0000-0001-5024-0385

[Abstract:0249]

Differences and Similarities Between Mothers and Fathers in Perceptions of Child Sleep and Psychosocial Functioning

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Background and Objective: Sleep difficulties are common in early childhood and constitute a major source of stress for families. Disturbed child sleep has been consistently associated with impaired parental sleep quality, increased emotional distress, and elevated symptoms of anxiety and depression. From a psychiatric and family systems perspective, parental responses to child sleep problems are shaped not only by the child's sleep characteristics but also by caregiving roles, perceived partner support, and broader family dynamics.

Mothers and fathers may differ in how they perceive and report child sleep disturbances and the associated psychosocial burden. Such differences may influence parental well-being, caregiving practices, and overall family functioning, yet paternal perspectives remain relatively underrepresented in research on early childhood sleep.

The present study aimed to compare mothers' and fathers' reports of child sleep characteristics, perceived partner support, parental sleep quality, mood-related symptoms (anxiety and depression), parental knowledge and self-efficacy, and overall parenting strain, in order to identify potential parent-specific patterns relevant to family-centered mental health interventions.

Methods: This cross-sectional study included 25 mothers and 25 fathers of young children. Data were collected using a structured, self-report questionnaire completed independently by each parent. The questionnaire assessed child sleep characteristics (nighttime awakenings, total nighttime sleep duration, and sleep difficulty indicators) and parental psychosocial variables, including perceived partner support, sleep quality, mood-related symptoms, parental knowledge and self-efficacy, and parenting strain.

Group comparisons between mothers and fathers were performed using appropriate parametric or non-parametric tests. A two-tailed p value < 0.05 was considered statistically significant.

Results: Mothers reported a significantly higher frequency of child nighttime awakenings compared with fathers ($p = 0.025$), indicating greater maternal awareness of nocturnal sleep disruptions. In contrast, fathers reported significantly higher levels of perceived total partner support ($p = 0.002$).

No significant differences were observed between mothers and fathers with respect to total child nighttime sleep duration, overall child sleep difficulty indicators, parental sleep quality, anxiety-related symptoms, depressive symptoms, parental knowledge or self-efficacy, or overall parenting strain (all $p > 0.05$).

Overall, these findings suggest that although mothers and fathers differ in their perceptions of specific sleep-related and support-related domains, their emotional well-being, sleep quality, and perceived parenting burden are largely comparable.

Conclusions: The findings indicate that the psychosocial impact of early childhood sleep difficulties is experienced in broadly similar ways by mothers and fathers, with notable differences in nighttime caregiving awareness and perceived partner support. Mothers appear to be more attentive to nighttime awakenings, whereas fathers report higher perceived support within the caregiving context.

From a psychiatric and family systems perspective, these results highlight the importance of incorporating both maternal and paternal viewpoints when evaluating child sleep problems and associated parental distress. Interventions targeting early childhood sleep and parental mental health may benefit from addressing caregiving roles, shared responsibilities, and partner support dynamics. Recognizing subtle yet meaningful parent-specific perceptions may enhance the effectiveness of family-centered prevention and intervention strategies.

Keywords: Early childhood sleep, Parental mental health, Maternal and paternal differences, Nighttime awakenings, Partner support

Comparison of Child Sleep and Parental Psychosocial Variables Between Mothers and Fathers

Variable	Mothers (n = 25)	Fathers (n = 25)	*p* value
Child nighttime awakenings (frequency)	2.72 ± 1.243 (2–4)	1.92 ± 1.152 (1–3)	**0.025**[a]
Total nighttime sleep duration (hours)	8.72 ± 0.799 (8–9)	8.56 ± 0.718 (8–9)	0.487[a]
Child sleep difficulty score	6.44 ± 2.007 (6–8)	6.76 ± 2.658 (7–8)	0.117[a]
Total perceived partner support	14.12 ± 3.7914 (12–16)	17.68 ± 4.0818 (15–20)	**0.002**[b]
Parental knowledge & self-efficacy score	3.84 ± 0.944 (3–5)	3.40 ± 0.823 (3–4)	0.095[a]
Perceived social support score	2.32 ± 1.602 (1–3)	3.04 ± 1.813 (1–5)	0.176[a]
Parental total sleep duration (hours)	5.60 ± 0.715 (5–6)	5.92 ± 0.706 (5–6)	0.102[a]
Parental sleep quality score	1.56 ± 0.582 (1–2)	1.76 ± 0.662 (1–2)	0.253[a]
Depressive symptoms score	1.40 ± 1.041 (1–2)	1.08 ± 0.761 (1–1)	0.304[a]
Anxiety symptoms score	1.52 ± 0.872 (1–2)	1.20 ± 0.761 (1–1)	0.127[a]
Total parenting strain score	17.24 ± 6.5719 (11–23)	15.76 ± 5.4115 (12–20)	0.389[b]

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[Abstract:0252]

Evaluation of Comorbid Psychiatric Symptoms in Patients Presenting With Sleep Disorders During The COVID-19 Pandemic

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Background and Objective: The COVID-19 pandemic has been associated with substantial psychological effects just as physical health consequences. Individuals who have experienced COVID-19 may be at increased risk for anxiety, depressive symptoms, sleep disturbances, and trauma-related stress due to uncertainty regarding disease progression, social isolation, fear of transmission, and concerns about long-term health outcomes. Although numerous studies have investigated the psychological impact of the pandemic in the general population, research focusing specifically on individuals who have personally experienced COVID-19 remains limited. The aim of this study was to examine the relationship between having experienced COVID-19 and psychological outcomes, including sleep quality, anxiety, depression and traumatic stress symptoms.

Methods: This cross-sectional study included 78 adult participants. Sociodemographic characteristics and clinical symptoms were assessed using a structured data collection form. Sleep quality was evaluated using the Pittsburgh Sleep Quality Index (PSQI), anxiety and depression symptoms were assessed with the Hospital Anxiety and Depression Scale (HADS), and traumatic stress symptoms were measured using the Impact of Event Scale (IES). Participants were divided into two groups based on self-reported COVID-19 infection status (experienced COVID-19 vs. not experienced COVID-19). Continuous variables were analyzed using the Mann–Whitney U test due to non-normal data distribution. Categorical variables were examined using chi-square tests. Statistical significance was set at $p < 0.05$.

Results: Of the participants, 46.2% (n = 36) reported having experienced COVID-19, while 53.8% (n = 42) reported no history of COVID-19. Individuals who had experienced COVID-19 demonstrated significantly higher IES scores compared with those who had not experienced COVID-19 ($p = 0.040$), indicating greater levels of traumatic stress symptoms.

No statistically significant differences were observed between the two groups in terms of PSQI total scores, HADS-Anxiety scores, or HADS-Depression scores ($p > 0.05$), although rank averages for these measures were higher among participants who had experienced COVID-19.

Crosstab analyses revealed a significant association between COVID-19 infection status and anxiety symptoms ($p < 0.001$). Anxiety was reported by 94.4% of participants who had experienced COVID-19, compared with 54.8% of those who had not. In addition, a significant relationship was found between COVID-19 infection and appetite changes ($p = 0.016$), with appetite changes being more frequent among individuals who had experienced COVID-19. No significant associations were identified between COVID-19 infection status and gender or loss of interest or pleasure ($p > 0.05$).

Conclusions: The findings of this study suggest that individuals who have experienced COVID-19 may be particularly vulnerable to traumatic stress and anxiety symptoms. Appetite changes also appear to be more common following COVID-19 infection, reflecting possible interactions between psychological and somatic responses. Although no significant differences were observed in sleep quality, anxiety, and depression scale scores, the results indicate heterogeneity in psychological responses following COVID-19. These findings show the importance of psychological screening in individuals with a history of COVID-19 and highlight the need for further longitudinal studies with larger samples.

Keywords: Anxiety, COVID-19, sleep quality, traumatic stress

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[Abstract:0266]

Patterns of Complementary And Alternative Medicine Use Among Children And Adolescents With Autism Spectrum Disorder

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Background: This study investigates the utilization of complementary and alternative medicine (CAM) practices, the perceived benefits of these methods, and the beliefs regarding the etiology of the disorder among 136 children with Autism Spectrum Disorder (ASD) and their families.

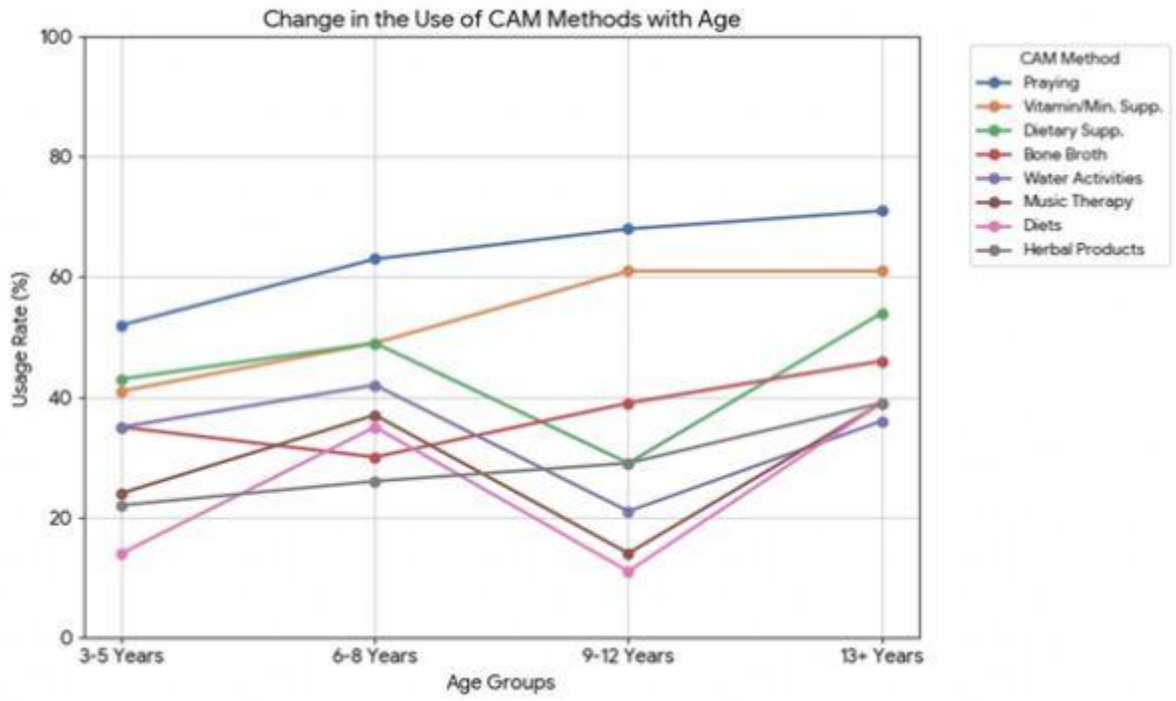
Methods: The cross-sectional research included 136 participants, predominantly male (72.8%), with a mean age of 8.9 years. Data were collected regarding the types of CAM methods used, parental beliefs about the causes of ASD, and the primary sources of information families relied upon for guidance.

Results: A high prevalence of CAM use was identified, with 91.2% of participants reporting the use of at least one method; the most common practices were prayer (62.5%), vitamin/mineral supplementation (51.5%), and dietary supplements (44.1%). CAM methods shift dynamically with age, with prayer and vitamin supplements showing a steady increase while methods like music therapy and specialized diets exhibit fluctuating patterns. Furthermore, logistic regression revealed that obtaining information from other physicians, or special educators significantly increased the likelihood of perceiving benefits from these practices, whereas sociodemographic factors had no independent effect.

Conclusion: CAM utilization is widespread among families of children with ASD and is significantly influenced by shared peer experiences, specific information sources, and professional advice. These findings emphasize the necessity for accurate professional guidance to help families navigate CAM options effectively.

Keywords: Autism Spectrum Disorder, Complementary and Alternative Medicine, Parental Perception, Perception of Etiology

Figure 1. Changes in the Usage Rates of Complementary and Alternative Medicine (CAM) Methods by Age



When the use of CAM methods was examined across age groups (Figure 1), parental preferences were found to change notably as children grew older. Praying and vitamin/mineral supplementation showed a steady age-related increase, reaching the highest usage rates in older age groups. In contrast, music therapy and specialized diets demonstrated a fluctuating pattern, with increased use during the early school-age period (6–8 years), a decline in the 9–12-year age group, and a subsequent peak during adolescence (>=13 years). Additionally, dietary supplements and water-based activities showed a marked decrease particularly in the 9–12-year age group. Overall, these findings suggest that families dynamically adjust supportive CAM interventions in response to their children's evolving developmental needs and symptom profiles.

AuthorToEditor: This abstract presents original findings on complementary and alternative medicine use among children with autism spectrum disorder, with potential clinical and psychopharmacological implications. The study was conducted with ethics committee approval and has not been previously presented or published. ORCID iDs of all authors have been provided. https://orcid.org/0000-0002-1573-3165 https://orcid.org/0000-0001-8866-6525 https://orcid.org/0009-0007-8066-5721 https://orcid.org/0000-0002-7443-9505 https://orcid.org/0009-0004-2790-0970 https://orcid.org/0000-0001-6310-6492 https://orcid.org/0000-0002-7802-6839 https://orcid.org/0000-0001-5298-0264 https://orcid.org/0000-0003-2988-4631

[Abstract:0300]

Evaluation of The Relationship Between Social Media Addiction and Emotion Regulation Difficulties, Eating Behaviors and Orthorexia Nervosa in Adolescents

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Background and Objective: With the rapid increase in social media use in recent years, adolescents' engagement with social media platforms has grown significantly. As the duration of use increases, the potentially addictive effects of social media become more pronounced. Adolescents use social media not only as a means of communication but also as a tool for emotion regulation. However, this pattern of use may contribute to changes in eating behaviors and the development of excessively health-focused attitudes. In this context, orthorexia nervosa—although not yet formally included in diagnostic classification systems—has emerged as a phenomenon of growing interest.The aim of this study was to examine the relationship between social media addiction and difficulties in emotion regulation, eating behaviors, and orthorexic tendencies among high school students aged 14–18 years.

Methods: The research was conducted in Denizli between January 2025 and May 2025. After obtaining approval from the Provincial Directorate of National Education and the ethics committee, informed consent was obtained from both participants and their parents. A total of 500 high school students were administered the Social Media Addiction Scale for Adolescents, the Difficulties in Emotion Regulation Scale–Short Form, the Dutch Eating Behavior Questionnaire, the ORTO-11 test, and a sociodemographic data form prepared by the researcher. Data from 407 students who completed all measures were included in the analyses.

Results: Of the participants, 56.5% were female, and the mean age was 15.64 years. The highest participation rate was observed among students attending Anatolian high schools. The most frequently used social media platforms were WhatsApp and Instagram, and nearly half of the participants reported using social media for 1–3 hours per day. Our findings indicated that as social media addiction levels increased, difficulties in emotion regulation and disordered eating behaviors also increased. Female participants had higher scores on social media addiction, emotion regulation difficulties, and eating behaviors, and they also demonstrated more pronounced orthorexic tendencies. Although no direct association was found between social media addiction and orthorexic tendencies, a significant indirect relationship was identified through emotion regulation difficulties and eating behaviors. Another noteworthy finding was the moderating role of socioeconomic status. At lower socioeconomic levels, social media addiction was associated with decreased orthorexic tendencies, whereas at higher socioeconomic levels, it was associated with increased orthorexic tendencies.

Conclusions: In conclusion, social media addiction appears to be associated with orthorexic tendencies indirectly through emotion regulation processes and eating behaviors. In a period when social media use is highly prevalent among adolescents, identifying risk factors and vulnerable groups is of critical importance. Preventive interventions targeting social media addiction may also have indirect protective effects on emotion regulation difficulties and problematic eating behaviors.

Keywords: Eating behavior, Emotion regulation difficulties, Orthorexia nervosa, Social media addiction

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[Abstract:0334]

Association of Metabolic Control with Diabetes-Related Distress and Health-Related Quality of Life in Adolescents with Type 1 Diabetes Mellitus: A Cross-Sectional Study

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Background and Objective: Type 1 diabetes mellitus (T1DM) is a chronic disease characterized by autoimmune-mediated pancreatic beta-cell loss and absolute/relative insulin deficiency. During adolescence, multidimensional self-management demands—such as daily insulin administration, blood glucose monitoring, dietary adjustments including carbohydrate counting, and management of hypo-/hyperglycemia risks—make diabetes care challenging not only in terms of metabolic control but also psychosocial burden. This study aimed to jointly evaluate the relationship between HbA1c, diabetes-related distress, and health-related quality of life (HRQoL) in adolescents with T1DM.

Methods: This cross-sectional study included 60 adolescents aged 12–18 years who were followed with a diagnosis of T1DM. Sociodemographic data and clinical characteristics (diabetes duration, outpatient follow-up frequency, history of hospitalization/intensive care unit admission, and most recent HbA1c value) were recorded. Diabetes-related distress was assessed using the Problem Areas in Diabetes scale (PAID-T), and HRQoL was assessed with the KIDSCREEN-52 (adolescent self-report). Participants with comorbid physical or psychiatric disorders were excluded.

Results: The mean most recent HbA1c was 8.33±1.51% (min–max: 5.6–13.0). PAID-T scores were 13.07±5.87 for emotional distress, 14.53±6.88 for family/friend-related stress, 10.93±5.73 for treatment-related stress, and 38.43±15.89 for total diabetes-related distress. HbA1c was significantly associated with PAID-T emotional distress, family/friend-related stress, and total score ($p=0.007$; $p=0.020$; and $p<0.001$, respectively). Although KIDSCREEN-52 scores tended to decrease as HbA1c increased, no statistically significant association was observed in any domain (all $p>0.05$).

Conclusions: Poorer metabolic control in adolescents with T1DM was associated with higher diabetes-related distress; however, no significant association was found between metabolic control and HRQoL. The cross-sectional design, the multidimensional nature of HRQoL, and potential mediating/confounding factors may have limited the detectability of this relationship. Distress may adversely affect glycemic outcomes not only through biological pathways but also via disruptions in self-care behaviors, sleep and eating patterns, and treatment adherence. Therefore, routine screening of diabetes-related distress alongside HbA1c in clinical follow-up and planning psychoeducation and targeted psychosocial interventions for adolescents with elevated distress are important. Larger, preferably longitudinal and/or multicenter studies are needed to clarify directionality and potential mediation mechanisms.

Keywords: Adolescents, Diabetes-related distress, Health-related quality of life, Type 1 diabetes mellitus

Correlations Between HbA1c and KIDSCREEN-52 Subscale Scores and PAID-T Scores

Measure	**r**	**p**
KIDSCREEN-52		
Physical Well-Being	-0.080	0.544
Psychological Well-Being	-0.009	0.945
Moods & Emotions	-0.097	0.459
Self-Perception	0.003	0.984
Autonomy	-0.124	0.347
Parent Relations & Home Life	-0.037	0.779
Financial Resources	-0.192	0.142
Social Support & Peers	0.118	0.368
School Environment	-0.217	0.096
Social Acceptance (Bullying)	-0.011	0.933
PAID-T	r	p
Emotional Burden	0.344	0.007
Family/Friend-Related Stress	0.300	0.020
Treatment-Related Stress	0.193	0.140
Total Score	0.423	<0.001

"SPSS: Pearson correlation test"

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[Abstract:0335]

Knowledge, Clinical Expectations, and Ethical Attitudes of Child and Adolescent Psychiatrists in Türkiye toward Xanomeline–Trospium (KarXT): Preliminary Findings

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Background/Objectives: Xanomeline–trospium (KarXT), a novel antipsychotic combining central muscarinic M1/M4 receptor agonism with peripheral antimuscarinic modulation, represents a paradigm shift by offering a dopamine-independent (non-D2) mechanism with a potentially more favorable metabolic profile, distinct from traditional dopamine-sparing or D2-antagonizing agents. Although KarXT has been approved for adult schizophrenia, its relevance, acceptability, and ethical implications in child and adolescent psychiatry remain unexplored. This study aimed to assess knowledge, awareness, clinical expectations, and ethical attitudes toward KarXT among child and adolescent psychiatrists in Türkiye and to present preliminary findings from this national survey.

Methods: In this cross-sectional study, child and adolescent psychiatry residents, specialists, and academics, practicing in Türkiye completed an author-developed online questionnaire assessing knowledge, clinical expectations, safety perceptions, and ethical attitudes toward KarXT. Group comparisons were performed using chi-square tests.

Results: A total of 259 professionals participated (82 residents, 140 specialists, and 37 academics) in this study. Residents demonstrated higher awareness of KarXT and its mechanism than specialists, with academics falling between these groups ($p=0.002$ and $p=0.001$); conversely, specialists were more likely to avoid prescribing without U.S. Food and Drug Administration (FDA) approval ($p=0.013$). No statistically significant differences were observed across years of professional experience for knowledge, perceived clinical benefit, safety expectations, or prescribing attitudes toward KarXT (all $p>0.05$). Clinicians working in centers with inpatient units demonstrated greater knowledge of KarXT and its mechanism of action (both $p<0.001$), were more likely to endorse metabolic advantages and lower rates of treatment discontinuation due to side effects ($p=0.032$; $p=0.008$), and were less likely to avoid prescribing in the absence of FDA approval ($p=0.004$). Anticipated therapeutic benefit was most frequently reported for psychosis/schizophrenia (95.8%), followed by autism spectrum disorder–related irritability/aggression (66.0%) and bipolar disorder (47.1%).

Conclusions: These findings indicate considerable clinical interest among child and adolescent psychiatrists in Türkiye toward KarXT, particularly in relation to its dopamine-independent mechanism and potential metabolic advantages, while also acknowledging limited formal knowledge. Together, these observations highlight the importance of pediatric-specific evidence, professional guidance, and clearly defined ethical frameworks before any broader consideration of off-label use of KarXT in child and adolescent psychiatry.

Keywords: KarXT, xanomeline–trospium, child and adolescent psychiatry, muscarinic agonist, dopamine-independent antipsychotic, prescribing attitudes

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[Abstract:0337]

Early Symptom Onset and Clinical Course in Pediatric Obsessive-Compulsive Disorder: Gender Differences and Severity Predictors

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Background: Obsessive–compulsive disorder (OCD) is a chronic and impairing psychiatric condition that frequently emerges during childhood and adolescence. Pediatric OCD has been associated with greater symptom chronicity, functional impairment, and developmental burden. Although demographic and temporal clinical variables such as age at symptom onset, duration of illness, and treatment history are routinely documented in clinical practice, their interrelationships and potential influence on symptom severity remain incompletely understood in pediatric samples. Furthermore, gender-related differences in clinical presentation and developmental trajectory continue to be inconsistently reported across studies.

Objective: This study aimed to examine the clinical characteristics of a pediatric OCD sample, to investigate gender differences in age at onset and illness course variables, and to evaluate whether demographic and temporal clinical factors predict OCD severity.

Methods: This retrospective study included 30 pediatric patients diagnosed with OCD who were evaluated at a tertiary child and adolescent psychiatry clinic. Ethics Committee approval was obtained from the Institutional Review Board (Decision No: TBAEK-157). Clinical and demographic data were extracted from archived medical records. The Clinical Global Impression Scale (CGI) was used to evaluate patients' illness severity, treatment response, and treatment-related adverse effects. The CGI is a brief and widely used clinician-rated instrument that assesses psychiatric symptoms based on the clinician's overall clinical judgment. The scale consists of three components: Clinical Global Impression–Severity (CGI-S), Clinical Global Impression–Improvement (CGI-I), and Side Effect Severity. Statistical analyses were performed using SPSS, with significance set at $p < .05$ (two-tailed).

Results: The sample comprised 30 patients, 15 of whom were female (50%). Females showed a predominance of contamination/cleaning symptoms, while males more frequently exhibited control- and symmetry/ordering-related obsessions and compulsions. Independent samples t-tests indicated significant gender differences in both age at presentation ($t(28) = -2.64$, $p = .013$) and age at symptom onset ($t(26) = -2.67$, $p = .013$), with females presenting earlier ($M = 12.89$ vs. 14.87 years) and exhibiting earlier symptom onset ($M = 11.47$ vs. 13.54 years) compared to males.

Pearson correlation analyses demonstrated a strong positive association between age at presentation and age at symptom onset ($r = .882$, $p < .001$). Age at symptom onset was negatively correlated with OCD duration ($r = -.389$, $p = .045$) and treatment duration ($r = -.493$, $p = .009$), while delay to presentation was positively associated with OCD duration ($r = .589$, $p = .004$).

Conclusion: Our findings provide preliminary evidence for a gender-specific phenotype in OCD presentation but underscore the complexity of factors contributing to illness severity. The primary limitation of this study, its modest sample size and consequent limited statistical power, necessitates a cautious interpretation of the regression analyses. While the observed gender difference in onset age is robust within this cohort, the non-significant predictors of severity should be viewed as hypothesis-generating rather than definitive. To advance the field, future investigations must prioritize large-scale, multi-center collaborations with longitudinal designs.

Keywords: Age at onset, gender differences, pediatric OCD, symptom severity

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[Abstract:0343]

Sociodemographic and Developmental Characteristics of Adults With Intellectual Disability, With and Without Autism, Followed in an Adult Neurodevelopmental Psychiatry Outpatient Clinic

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Background and Objective: Intellectual disability (ID) is a neurodevelopmental disorder characterized by limitations in cognitive and adaptive functioning. Autism spectrum disorder (ASD) frequently co-occurs with ID and significantly affects developmental features, functional level, and care needs. However, comparative studies of adults with ID with and without ASD are limited. This study aims to retrospectively evaluate the sociodemographic characteristics, developmental history, and medical comorbidities of adults with ID with and without ASD followed in an adult neurodevelopmental psychiatry outpatient clinic.

Methods: This retrospective, observational, cross-sectional study included adults with intellectual disability (ID) with and without autism spectrum disorder (ASD) who presented to an adult neurodevelopmental psychiatry outpatient clinic between January 2017 and December 2025. Medical records and electronic data were reviewed retrospectively. Sociodemographic characteristics, developmental history, and accompanying medical comorbidities, including epilepsy and other chronic conditions, were evaluated.

Results: A total of 329 participants were included in the study. The mean age of the participants was 25.66 ± 7.28 years. majority were male (n = 210, 63.8%), single (n = 323, 98.2%), living with their families (n = 316, 96.0%), and had a high school level of education (n = 140, 42.0%). Most participants had received special education (n = 295, 89.7%) and were not employed (n = 298, 90.6%). Regarding intellectual disability, 137 individuals (41.6%) had mild and 126 (38.3%) had moderate intellectual disability. Epilepsy was present in 84 participants (25.5%), allergic conditions in 42 (12.8%), and other medical comorbidities in 103 (31.3%).When participants were compared according to the presence of autism spectrum disorder (ASD), significant differences were observed between groups. Male sex was significantly more frequent in the ASD group ($p = 0.01$), and individuals with ASD were more likely to have received special education ($p = 0.013$) and to have severe intellectual disability ($p = 0.022$). In contrast, participants without ASD had significantly higher rates of employment ($p = 0.007$), epilepsy ($p = 0.040$), and medical comorbidities ($p < 0.001$).

Conclusions: This study highlights the distinct sociodemographic and clinical profile of individuals with intellectual disability, who were predominantly male, single, living with their families, unemployed, and largely dependent on special education services. Within this population, the presence of ASD was associated with male predominance, higher rates of severe intellectual disability, and greater need for special education, whereas epilepsy and medical comorbidities were more frequent among individuals without ASD. These findings emphasize the importance of considering both developmental and medical characteristics when planning multidisciplinary care and support strategies for individuals with intellectual disability.

Keywords: Intellectual Disability, Autism Spectrum Disorder, Neurodevelopmental Disorders, Medical Comorbidities

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[Abstract:0394]

Clinicians' Compliance with Turkish Psychiatric Association Guideline Recommendations in the Treatment of Bipolar Depression: a Retrospective Study From Turkey, Ankara

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Background and Objective: Bipolar disorder(BD) is a chronic mental disorder with periodic recurrence of manic, hypomanic and depressive episodes. Depressive episodes are among the most common and devastating periods in the course of the BD. Judd et al. showed that individuals with BD spend 40% to 60% of their lifetime with depressive symptoms which characterized not only by acute manifestations but also by prolonged recovery periods. The Turkish Psychiatric Association (TPA), has proposed treatment guideline to clinicians for BD published in 2022. Altınbaş et al. stated that the rate of compliance with TPA guidelines is insufficient; they suggested that compliance varies according to the severity of depression. In this study, we aimed to examine the compliance of clinicians in Turkey with the TPA guideline and its effect on remission rates

Methods: In our retrospective study, the treatment records of 131 patients admitted to the psychiatry clinic of our hospital and followed up with the diagnosis of Bipolar Depression were analyzed and the treatment according to the severity of the episode was compared with the recommendations of the TPA guideline. In this study, remission was defined based on the clinical judgment of the treating physician and structured follow-up records, considering a significant reduction in depressive symptoms and overall improvement in psychosocial functioning. (Ethics Approval Number: 25.12.2024-TABED 2-24-706)

Results: Among 131 patients, 58 (43.94%) were male, 92 (69.7%) were married.The mean age of the patients was 42.71 years.75.56% (n=99) of the patients were followed up with bipolar I. Of the all patients, 72.86% (n=102) were in mild-moderate episodes. The rate of adherence to guideline in the 1st line was 67.46% for mild-moderate episodes and 65.51% for severe episodes, while the rate of adherence in the 2nd line was 70.37% for mild-moderate episodes and 67.85% for severe episodes.6 patients who needed 3rd line treatment were in severe episode, compliance in the third-line was 83.33%, and 4 patients who needed 3rd line treatment in mild-moderate episode, treatment protocol was organized in full compliance (n=4). The remission rate of treatments that did not adhere to guideline was 66.6%, while the remission rate of complianced with guideline was 75.34%

Conclusions: We observed that adherence to the guideline increased as the treatment step progressed, regardless of the severity of depression.It is interesting that the compliance with treatment guideline in the treatment of BD is lower in severe episodes at each step. It is thought that multicenter studies with larger samples are needed to clearly understand the extent to which the current treatment guidelines for the treatment of BD are adopted by Turkish psychiatrists.
Keywords: Adherence, bipolar disorder, depressive episode, treatment guideline

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[Abstract:0405]

Investigating Nomophobia in Adolescents: Examining its Relationship With Intolerance of Uncertainty and Emotion Regulation Difficulties – Preliminary Findings

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Background and Objective:

Nomophobia, defined as the fear or anxiety of being without access to a mobile phone, has become increasingly prevalent among adolescents. Developmental characteristics of adolescence, including heightened emotional reactivity and ongoing maturation of regulatory capacities, may increase vulnerability to smartphone-related anxiety. Psychological constructs such as intolerance of uncertainty and difficulties in emotion regulation may contribute to excessive reliance on smartphones as a coping strategy. This study aimed to examine the prevalence of nomophobia among high school adolescents and to investigate its association with intolerance of uncertainty, difficulties in emotion regulation, and perceived social support. The findings presented here represent preliminary results from an ongoing study with a planned total sample of 700 adolescents.

Methods:

This cross-sectional preliminary analysis included 396 high school students who voluntarily agreed to participate in the study. Permission was obtained from the Ministry of National Education prior to data collection, and ethical approval was granted by the Institutional Ethics Committee (Approval No: 2025-02/53). Data were collected using the Nomophobia Questionnaire (NMP-Q), the Intolerance of Uncertainty Scale, the Difficulties in Emotion Regulation Scale, the Perceived Social Support Scale, and a sociodemographic information form. Participation was voluntary, and all procedures were conducted in accordance with ethical standards.

Results:

Of the 396 participants, 251 (63.4%) were female and 145 (36.6%) were male, with a mean age of 14.75 ± 0.93 years. A total of 355 adolescents (89.6%) reported owning a personal smartphone. The mean daily smartphone use was 141.63 ± 84.77 minutes, and the mean daily social media use was 89.92 ± 71.37 minutes. Regarding nomophobia severity, 196 participants demonstrated moderate levels, 161 (40.7%) mild levels, 31 (7.8%) severe levels, and 8 (2.0%) showed no nomophobia symptoms. Female participants reported significantly higher nomophobia scores (68.22 ± 22.22) compared to males (62.31 ± 24.86) ($p = 0.015$). Nomophobia scores were positively correlated with difficulties in emotion regulation ($r = 0.347$, $p < 0.001$) and intolerance of uncertainty ($r = 0.245$, $p < 0.001$). No statistically significant association was found between nomophobia and perceived social support ($p = 0.833$).

Conclusions:

These preliminary findings from an ongoing larger-scale study suggest that nomophobia is highly prevalent among high school adolescents and is significantly associated with intrapersonal regulatory vulnerabilities. The moderate association with emotion regulation difficulties indicates that adolescents who struggle to manage emotional responses may be more prone to smartphone-related anxiety. Similarly, intolerance of uncertainty may increase reliance on smartphones as a source of reassurance. The absence of a significant relationship with perceived social support suggests that nomophobia may be more closely linked to internal regulatory processes than to interpersonal protective factors. As recruitment continues toward the planned sample of 700 participants, further analyses are expected to clarify predictive pathways and strengthen the robustness of these findings.

Keywords: Adolescents, Emotion Regulation, Mobile Phone Use, Nomophobia, Social Support, Uncertainty

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[Abstract:0411]

The Role of Maintenance ECT in Treatment-Resistant Schizophrenia: A Case Series

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Introduction: Schizophrenia remains a profound global public health challenge, with approximately one-third of diagnosed patients exhibiting significant resistance to standard first-line antipsychotic regimens [1]. Although clozapine is the gold standard for managing treatment-resistant schizophrenia (TRS), clinical options for patients with clozapine intolerance or inadequate response are notably limited. Electroconvulsive therapy (ECT) serves as a highly effective intervention for controlling acute psychotic exacerbations [2]. However, high relapse rates following acute ECT have necessitated the strategic use of maintenance ECT (M-ECT) to sustain long-term clinical stability. Emerging literature suggests that M-ECT effectively reduces re-hospitalization rates and enhances functional outcomes in complex TRS cases [3]. This retrospective case series, conducted with anonymized data, evaluates the longitudinal impact of M-ECT across six distinct clinical presentations.

Case presentation: The clinical characteristics and treatment trajectories of the six patients are summarized below:

•Case 1: A 43-year-old female with a 24-year history of schizoaffective disorder. Following her March 2025 hospitalization, she responded to 9 acute ECT sessions. Her maintenance phase involved 11 M-ECT sessions alongside clozapine (400 mg) and lamotrigine (300 mg). She has successfully maintained clinical remission as of April 2025.

•Case 2: A 29-year-old male with a 9-year history of schizophrenia. Hospitalized in June 2025 for a severe relapse, he underwent 11 acute ECT sessions. Maintenance therapy consisted of 8 M-ECT sessions combined with clozapine (400 mg). He continues to exhibit stable remission during follow-up.

•Case 3: A 37-year-old male diagnosed with schizophrenia 9 years ago. After 8 acute ECT sessions during his June 2025 hospitalization, he transitioned to 6 M-ECT sessions. His pharmacological regimen includes paliperidone palmitate and chlorpromazine (400 mg), effectively sustaining his clinical remission.

•Case 4: A 63-year-old female with a 35-year history of schizophrenia. During her August 2024 hospitalization, she received 8 acute ECT sessions. Her maintenance phase comprised 19 M-ECT sessions integrated with clozapine (350 mg), clonazepam (2 mg), mirtazapine (15 mg), and quetiapine (700 mg total daily dose). She remains in stable remission.

•Case 5: A 21-year-old male with a 6-year history of schizophrenia. Following 15 acute ECT sessions in May 2024, he received 16 M-ECT sessions. Augmented with clozapine (450 mg) and amisulpride (200 mg), this combined strategy has successfully sustained long-term clinical remission.

•Case 6: A 42-year-old male with 17 years of schizophrenia and six prior hospitalizations. His April 2025 hospitalization involved 12 acute ECT sessions. Subsequent maintenance included 17 M-ECT sessions and clozapine (400 mg), resulting in sustained remission and preventing further relapses.

Conclusion: This case series reinforces the critical therapeutic role of M-ECT in maintaining remission for TRS patients with histories of frequent hospitalizations. M-ECT provides a safe and highly effective augmentation strategy for individuals failing multiple antipsychotics or suffering from severe side effects. Our clinical observations demonstrate that systematic M-ECT application ensures long-term stability and prevents the cycle of re-hospitalization. Integrating M-ECT earlier into standard TRS management protocols could significantly improve long-term prognosis and patient quality of life.

Keywords: Electroconvulsive therapy, maintenance, schizophrenia, treatment resistant

AuthorToEditor: 0009-0005-0498-3555

Psychometric Properties of the Abridged Version of the Autism-Spectrum Quotient (AQ-28) in a Turkish Non-Clinical Sample

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Background and Objective

The Autism Spectrum Quotient – Short Form (AQ-28) is a widely used instrument for assessing autistic traits in non-clinical populations. The scale was derived from the original 50-item Autism Spectrum Quotient and reflects five domains of autistic characteristics within a hierarchical structure in which social-domain factors load on a higher-order Social Behaviour construct. Although the AQ-28 has been validated in several languages, no comprehensive psychometric evaluation has been conducted for the Turkish version. The present study aimed to evaluate the reliability, factorial validity, measurement invariance, and item-level psychometric properties of the Turkish AQ-28 in an undergraduate sample.

Methods

A cross-sectional online survey was conducted among undergraduate students aged 18–30 years using a convenience sampling procedure, resulting in a final sample of 683 participants. The AQ-28 and the short form of the Empathy Quotient (EQ-Short) were administered. Confirmatory factor analyses (CFA) were conducted using robust weighted least squares estimation for ordinal data. Reliability was assessed using Cronbach's alpha and McDonald's omega coefficients. Convergent and discriminant validity were evaluated using standardized factor loadings, composite reliability, average variance extracted, Fornell–Larcker criteria, and HTMT ratios. Item properties were examined using corrected item–total correlations and graded response models. Measurement invariance across gender was tested using multi-group CFA. Differential item functioning (DIF) was evaluated using both MIMIC modeling and ordinal logistic regression likelihood-ratio χ^2 tests.

Results

The correlated five-factor model demonstrated acceptable fit (CFI = .91, TLI = .90, RMSEA = .07). The higher-order model, consistent with the original structure, provided comparable fit and was retained as the primary solution. After allowing a single residual covariance, model fit improved ($\chi^2(344) = 1433.33$, CFI = .914, TLI = .905, RMSEA = .068, SRMR = .077). Internal consistency was acceptable for the total score ($\alpha = .73$, $\omega = .77$) and ranged from .60 to .76 across subscales. Multi-group CFA supported configural, metric, and scalar invariance across gender. Small gender differences were observed for Routine, Switching, and Numbers and Patterns subscales. DIF analyses identified six items with statistically significant but small uniform DIF effects across gender. Convergent validity was externally supported by moderate-to-strong negative correlations between AQ-28 scores and empathy ($r = -.55$ for total score; $r = -.60$ for the higher-order Social Behaviour factor). Convergent validity indices indicated acceptable construct representation despite AVE values below .50, as supported by moderate standardized factor loadings, adequate composite reliability coefficients ($CR \geq .60$), and satisfactory McDonald's omega values. Discriminant validity was supported by both Fornell–Larcker and HTMT criteria. Item response theory analyses (GRM) indicated generally adequate item discrimination and acceptable item fit.

Conclusions

The Turkish AQ-28 demonstrates satisfactory reliability and validity in undergraduate students. The higher-order structure of the original scale was supported, and measurement properties were stable across gender. Findings indicate that the Turkish AQ-28 is a reliable and structurally valid instrument for assessing autistic traits in non-clinical populations.

Poster Research Presentations

[Abstract:0101]

Catatonia as a Prelude to Mania: A Bipolar Case Report

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Introduction: Catatonia is a neuropsychiatric syndrome characterized by motor, behavioral, and autonomic abnormalities that can accompany a wide range of psychiatric or medical conditions (1). Although historically associated with schizophrenia, recent studies have shown that catatonia most frequently occurs in mood disorders, particularly bipolar disorder (2). Recognition of catatonia in bipolar patients is crucial since delayed diagnosis may result in prolonged morbidity or life-threatening complications. This case presents a bipolar affective disorder patient who developed acute catatonia mimicking an organic condition and showed rapid improvement after benzodiazepine treatment.

Case presentation: A 50-year-old single male lawyer with a 10-year history of bipolar affective disorder was admitted to the emergency department due to sudden unresponsiveness and lack of reaction to verbal or painful stimuli. Physical examination revealed pinpoint pupils, and naloxone administration produced only transient improvement. Toxicological screening and cranial MRI excluded substance intoxication and acute neurological pathology. The patient was transferred to the intensive care unit for observation. During the initial three days, neurological evaluation showed no catatonic signs, but nystagmus was noted. On the fourth day, psychiatric reassessment revealed waxy flexibility, suggestive of catatonia, and intravenous diazepam therapy was initiated. Following benzodiazepine administration, the patient's consciousness improved, and manic symptoms such as flight of ideas, pressured speech, and grandiosity emerged. He was transferred to the psychiatry inpatient unit, where treatment with valproic acid (1000 mg/day) and diazepam (20 mg/day) was continued. As his manic symptoms decreased, diazepam was gradually tapered. However, the patient demonstrated poor medication compliance and concealed valproic acid tablets without swallowing them; therefore, olanzapine 5 mg/day was added. On the ninth day of hospitalization, he showed marked clinical improvement and was discharged in partial remission.

This case highlights the diagnostic challenge between organic and psychiatric etiologies of altered consciousness, especially when initial presentations resemble intoxication or neurological dysfunction (3).

Conclusion: This case underlines the importance of considering catatonia in the differential diagnosis of acute altered mental status in patients with a history of mood disorders. Benzodiazepine responsiveness is not only a key diagnostic indicator but also an effective treatment strategy for catatonia secondary to bipolar disorder. Early recognition and prompt pharmacological intervention can prevent unnecessary invasive procedures and improve prognosis.

Keywords: Benzodiazepine response, bipolar disorder, catatonia

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[Abstract:0106]

Cyclic Vomiting Syndrome in an Adolescent With Autism Spectrum Disorder: A Case Report Highlighting Behavioral Management Challenges

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Introduction: Cyclic vomiting syndrome (CVS) is a functional neurogastroenterological disorder defined by recurrent, stereotypical episodes of intense vomiting with complete remission between attacks. Although its pathophysiology remains unclear, growing evidence implicates mitochondrial dysfunction, migraine-spectrum physiology, and ion-channel abnormalities. Genetic studies have identified variants in genes related to energy metabolism and ion transport, suggesting that some CVS subtypes may represent channelopathies or mitochondrial disorders. Mitochondrial vulnerabilities are also frequently reported in autism spectrum disorder (ASD), along with gastrointestinal dysfunction mediated through microbiome and energy-metabolism disruptions. This overlap highlights the importance of considering neurodevelopmental comorbidities in early-onset or severe CVS cases. Case presentation: This report describes a 15-year-old male with ASD and mild intellectual disability followed for more than a decade in child psychiatry, who presented with worsening vomiting episodes consistent with CVS. His developmental history included delays from age two, a diagnosis of childhood autism at age four, and long-standing aggression, hyperactivity, and stereotypies requiring multiple psychotropic trials. Vomiting episodes began at age seven, occurring in prolonged cyclical patterns with symptom-free intervals. Initially induced through inserting fingers or objects into his mouth, vomiting later evolved into purposeful initiation via gag reflex stimulation.

Across several years, he was treated with risperidone, methylphenidate, atomoxetine, fluoxetine, and levetiracetam for comorbid behavioral and neurological symptoms, with variable tolerability. In 2021, following examinations and two recorded endoscopies that revealed no organic problem, and after recurring episodes of 5-6 vomiting attacks per day lasting several weeks, the diagnosis of CVS was officially made in pediatrics. At presentation in March 2025, he exhibited increased hyperactivity, aggression toward his mother, and both spontaneous and intentional vomiting, accompanied by significant weight loss.

Fluoxetine initially decreased vomiting frequency and behavioral dysregulation; however, symptoms worsened after two months, prompting discontinuation. Alprazolam was similarly ineffective. The treatment approach then shifted toward behavioral strategies. Daily routines were restructured, behavioral plans were coordinated with special education teachers, and a reward–response system was implemented to target intentional vomiting. Over subsequent weeks, vomiting frequency markedly declined, ultimately occurring only once every two weeks. Behavioral functioning and participation in special education improved, underscoring the value of psychosocial interventions in complex CVS–ASD interactions.

Conclusion: This case contributes to growing evidence that mitochondrial and neurodevelopmental vulnerabilities may coexist in CVS and ASD. Pharmacological interventions often yield limited benefit in such patients, whereas structured behavioral and environmental strategies may provide substantial clinical improvement. The findings highlight the need for multidisciplinary collaboration among gastroenterology, genetics, and child psychiatry, as well as careful assessment of behavioral components—such as intentional vomiting—that may mimic or exacerbate CVS episodes. Further research is needed to clarify the biological mechanisms underlying the CVS–ASD overlap and to develop individualized, multimodal treatment approaches.

Keywords: Cyclic Vomiting Syndrome, Autism Spectrum Disorder, neurodevelopment

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[Abstract:0130]

A Case of Rapid Nocturnal Enuresis Resolution After Fluoxetine Initiation

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Introduction: Primary monosymptomatic nocturnal enuresis (PMNE) is a common pediatric condition with multifactorial etiology. Standard treatments include desmopressin and anticholinergics, while tricyclic antidepressants are third-line options. Selective serotonin reuptake inhibitors (SSRIs) are more often associated with urinary incontinence than improvement. We report a case of rapid remission of treatment-resistant PMNE following fluoxetine initiation for anxiety.

Case presentation: A 7-year-old boy diagnosed with Social Anxiety Disorder (DSM-5-TR) was started on fluoxetine 10 mg/day. He had lifelong nocturnal enuresis occurring 1–2 times nightly, with no six-month dry period, normal urologic evaluation, and no daytime urinary symptoms, consistent with Primary Monosymptomatic Nocturnal Enuresis(PMNE). Behavioral interventions and prior oxybutynin treatment were ineffective. Family history was positive for childhood enuresis.Complete cessation of enuresis occurred within 48 hours of fluoxetine initiation and was confirmed by clinical follow-up using the Nocturnal Enuresis Symptom Score and Clinical Global Impressions scale. No additional enuresis-directed interventions were introduced. Enuresis did not recur during 12 weeks of follow-up, and social functioning improved. Mild transient abdominal pain was the only adverse effect.

Conclusion: The rapid response suggests a mechanism independent of the delayed anxiolytic effects of SSRIs. Fluoxetine-related modulation of sleep–arousal mechanisms and serotonergic control of lower urinary tract function may contribute to early symptom resolution. Although causality cannot be established, this case highlights a potential role for fluoxetine in select patients with treatment-resistant PMNE and comorbid anxiety, warranting further controlled investigation.

Written informed consent was obtained from the patient’s parents for presentation and publication of this case.

Keywords: Anxiety, enuresis, fluoxetine, serotonin, sleep

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[Abstract:0145]

Delusional Parasitosis Presenting with the Matchbox Sign in an Elderly Patient: A Case Report

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Introduction
Delusional Parasitosis (DP) is a somatic-type delusional disorder characterized by the persistent but false belief of being infested by small organisms such as insects or parasites. It is frequently observed in elderly populations and often accompanied by the "matchbox sign," where patients provide non-biological samples as evidence of infestation. This case aims to discuss the clinical management of late-onset DP and the significance of ruling out organic etiologies.

Case
A 77-year-old housewife, who is illiterate, widowed, and mother of seven, living in a rural area with her daughter, was brought to the psychiatric outpatient clinic by her relatives. She had no prior history of psychiatric illness. Her medical history included hypertension and a total thyroidectomy. Her complaints began approximately four years ago, following familial issues and feelings of loneliness. She reported that small, black, motile, lice-like insects were crawling on her body, biting her, and causing pruritus. The itching was found to increase especially at night, disrupting her sleep patterns.

Driven by her delusional perceptions, the patient had reportedly packaged household items to prevent "infestation." She also claimed to observe insects crawling on her daughter. The daughter's exhibition of similar avoidance behaviors during this process was evaluated as a possible *folie à deux* (shared psychotic disorder). During the clinical interview, the patient presented fabric lint and skin debris, which she identified as insects, inside a chewing gum box; this phenomenon is defined in the literature as the "Matchbox Sign."

Over the past four years, the patient was initially evaluated in dermatology departments, and upon finding no evidence of parasitic infestation, she was referred to psychiatry. In 2023, she was diagnosed with Delusional Disorder at an external center and prescribed Escitalopram 10 mg/day and Olanzapine 2.5 mg/day; however, she discontinued the medication after one month, believing it was ineffective. Physical examination revealed no visible skin lesions despite the complaint of severe pruritus. Vital signs and neurological examination were unremarkable. Routine blood tests and cranial magnetic resonance imaging (MRI) were within normal limits.

During the mental state examination, her appearance was compatible with her age, her self-care was good, and she was cooperative and fully oriented. The patient, whose mood was euthymic and affect was congruent, experienced significant distress regarding "infestation." Her thought content revealed systematized somatic delusions about insects crawling on her body. It was determined that her reality testing and judgment were partially impaired within the delusional axis, and her insight was inadequate. Her social relations and activities of daily living were preserved. Her Standardized Mini-Mental State Examination (SMMSE) score was 26/30. The patient had recently experienced an involuntary weight loss of 7 kg (from 94 kg to 87 kg).

The patient was diagnosed with Delusional Parasitosis. Considering her previous treatment non-compliance and symptom profile, treatment was initiated with Sertraline 50 mg/day and Sulpiride 50 mg/day, which is effective for somatic delusions.

Conclusion
This case emphasizes the importance of comprehensive organic screening and a multidisciplinary approach in late-onset delusional parasitosis.

Keywords: Antipsychotic Agents, Delusional Parasitosis, Geriatrics

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[Abstract:0151]

Clozapine-Associated Stuttering With EEG Abnormalities: Resolution With Valproate and Long-Term Stability — A Case Report

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Introduction: Clozapine remains the most effective antipsychotic for treatment-resistant schizophrenia; however, it is associated with a range of adverse effects, including rare but clinically significant speech disturbances. Among these, stuttering has been reported with an estimated prevalence of approximately 0.9% and typically emerges in a dose-dependent manner during rapid titration or dose escalation. In most published cases, symptoms improve following dose reduction.

The underlying pathophysiology of clozapine-induced stuttering (CIS) appears heterogeneous. Some reports suggest an association with epileptiform activity or seizures, while others describe cases occurring in the absence of electroencephalographic (EEG) abnormalities, implicating dysfunction of motor speech circuits. Importantly, documentation of CIS accompanied by a transition from a previously normal EEG to abnormal findings at symptom onset, followed by sustained remission under antiepileptic coverage with successful re-titration to higher clozapine doses, remains scarce. This case aims to address these gaps by presenting a well-characterized instance of CIS with objective clinical and neurophysiological data.

Case presentation: A 45-year-old unmarried man with a 13-year history of schizophrenia was admitted with persecutory delusions, auditory hallucinations, and agitation. Previous trials of risperidone, paliperidone, olanzapine, amisulpride, and haloperidol had been ineffective. He had been medication-free for three months prior to admission. The patient smoked 1.5 packs per day and had no relevant medical comorbidities. Baseline laboratory investigations and neurological examination were unremarkable. A routine EEG performed six months earlier had been normal.

On admission, the Positive and Negative Syndrome Scale (PANSS) total score was 141. Clozapine was initiated at 50 mg/day and titrated to 300 mg/day by day 13, resulting in marked improvement in agitation and psychotic symptoms. On day 15, while receiving 300 mg/day of clozapine, the patient developed sudden-onset stuttering characterized predominantly by repetition phenomena. EEG revealed generalized paroxysmal abnormalities, in contrast to the previously normal EEG. No clinical seizures were observed. At that time, the PANSS total score had improved to 91.

Clozapine was reduced to 200 mg/day, and valproic acid 1000 mg/day was initiated. The stuttering resolved completely within 48 hours. Clozapine was subsequently re-titrated under valproate coverage to 500 mg/day without recurrence of stuttering. PANSS scores continued to improve, reaching 78 by discharge. During 1.5 years of follow-up on clozapine 500 mg/day and valproic acid 1000 mg/day, the patient remained clinically stable with no recurrence of stuttering or psychotic symptoms.

Conclusion: This case supports the hypothesis that clozapine-induced stuttering may, in some patients, be mediated by subclinical epileptiform activity. The emergence of EEG abnormalities concurrent with symptom onset, rapid resolution following valproate initiation, and successful long-term re-titration of high-dose clozapine highlight the importance of EEG assessment and consideration of adjunctive antiepileptic therapy. Early recognition may allow continuation of clozapine without compromising safety or efficacy.

Keywords: Clozapine, Electroencephalography, Stuttering

Longitudinal Course of Treatment, PANSS Scores, and Clinical Features

Day	Clozapine (mg/day)	Valproate (mg/day)	PANSS Total	Clinical Notes
Day 1	50	-	141	Severe psychosis
Day 13	300	-	-	Hallucinations regressed
Day 15	300	-	91	Stuttering onset, EEG abnormality
Day 16	200	1000	-	Dose reduction + valproate added
Day 18	200	1000	-	Stuttering resolved
Day 20	250	1000	-	Re-titration
Day 24	300	1000	-	No recurrence
Day 30	350	1000	-	Stable
Day 34	400	1000	84	Improvement, no stuttering
Day 40	500	1000	78	Stable, discharged
1.5-year follow-up	500	1000	-	No recurrence for 1.5 years

AuthorToEditor: Bu olgu sunumu, klozapinle ilişkili nadir bir yan etki olan kekemeliğin (clozapine-induced stuttering) ortaya çıkışı sırasında daha önce normal olan EEG'nin patolojik hale gelmesini ve antiepileptik tedaviye hızlı yanıtını belgelemektedir. Olgu, valproik asit eşliğinde klozapin dozunun güvenli şekilde yeniden artırılabildiğini ve uzun dönemli remisyonun sürdürülebildiğini göstermesi açısından klinik açıdan öğreticidir. Ayrıca klinik seyir PANSS ile nicel olarak izlenmiştir. Bu yönleriyle, klozapinle ilişkili konuşma bozukluklarının olası epileptiform mekanizmasına ve pratik klinik yönetime katkı sağlamayı amaçlamaktadır. ORCID Turceun İleri Akdoğan: 0000-0001-9123-3798 Bedirhan Şenol: 0000-0002-0611-1858 Hasan Kaya: 0000-0002-9289-6013

[Abstract:0162]

Manic Episode Triggered By Discontinuation of Aripiprazole in an Adolescent Diagnosed With Kleine–Levin Syndrome: Kleine–Levin Syndrome or Mood Disorder?

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Introduction: Kleine-Levin Syndrome (KLS) is a rare, episodic disorder that typically begins during adolescence and follows a relapsing-remitting course, with hypersomnia as the predominant symptom.It occurs more frequently in males and the average age of onset is 15 years. Hypersomnia is accompanied by cognitive and behavioral symptoms, while patients are completely asymptomatic between episodes.During episodes, which are characterized by sleeping for most of the day, some patients exhibit hypersexuality and compulsive eating behaviors, whereas others present with psychiatric symptoms such as derealization and apathy.The abrupt onset, severe intensity, and recurrent pattern of hypersomnia episodes are the core features that distinguish KLS from other psychiatric conditions. KLS has been reported to show comorbidity with mood disorders.

Case Presentation: A female adolescent whose hypersomnia episodes began at the age of 15 years and who was followed up for three years with a diagnosis of KLS due to the sudden onset and episodic pattern of her symptoms is reported. The patient's hypersomnia episodes consistently began abruptly after completely asymptomatic periods and lasted exactly one week.At the end of each episode, she would awaken in the morning and resume her normal daily activities, including attending school, without residual symptoms.During episodes, she hardly woke up only for physiological needs and slept more than 21 hours per day. Derealization and apathy were the predominant accompanying symptoms. Comprehensive viral and autoimmune screenings yielded normal results. Her first EEG obtained during episode revealed frontal intermittent rhythmic delta activity (FIRDA) during wakefulness. Polysomnography performed during an episode was normal.However, multiple sleep latency test (MSLT) revealed three sleep-onset REM periods (SOREMP).The patient was treated with aripiprazole at doses ranging from 10 to 15 mg for approximately three years. While hypersomnia episodes recurred every three weeks during medication-free periods, the interval between episodes was prolonged to 4-7 months under aripiprazole treatment.While the patient was under adult psychiatry follow-up, her KLS episodes started to occur once a month again despite aripiprazole, and she was hospitalized with a manic episode after the cessation of aripiprazole.Three months after discharge she was not free of KLS episodes despite lithium 900 mg and olanzapine 20 mg treatment.

Conclusion: Although rare, cases of KLS comorbid with mood disorders have been reported. In the present case, the diagnosis of KLS was considered definitive due to the highly characteristic nature of the hypersomnia episodes. However, the prolongation of intervals between episodes with aripiprazole treatment and the emergence of a manic episode following medication discontinuation raise the possibility of a shared etiological mechanism between KLS and bipolar disorder, both of which have episodic course. In patients whose episodes are characterized by hypersomnia, apathy, derealization, and depressed mood, the clinical presentation may closely resemble depressive episodes, often leading to diagnostic delays. The reported benefit of lithium in KLS cases and the significant reduction in attack frequency observed with aripiprazole in the present case further support the hypothesis of overlapping etiopathogenetic mechanisms between KLS and bipolar disorder. Indeed, it can be suggested that KLS may represent a subtype within the bipolar spectrum.

Keywords: Mood disorder, Klein-Levin syndrome, aripiprazole

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[Abstract:0165]

When Imaging is Silent, Clinical Clues Speak: A Case of Poretti–Boltshauser Syndrome with Normal Brain MRI

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Introduction: Poretti–Boltshauser syndrome is rare genetic disorder of brain malformation with ocular findings due to mutation in the LAMA1 gene. It is typically characterized by cerebellar cystic dysplasia, developmental delay, and ocular findings. However, in some cases, brain magnetic resonance imaging (MRI) may be normal or nonspecific, which can complicate the diagnostic process. This case report presents the diagnostic process of a patient who initially presented with strabismus, as well as the intervention planning undertaken due to developmental delay.

Case presentation: We report a 1-year-1-month-old male who initially presented with strabismus and was later evaluated for developmental delay. Developmental assessment revealed absent independent walking, toe walking with support, preserved unsupported sitting, and lack of observable expressive speech. Ophthalmological examination showed esotropia, searching nystagmus, bilateral optic disc pallor, and macular atrophy. Neurological examination demonstrated increased muscle tone with spasticity predominantly affecting the upper extremities and hyperreflexia.

Brain MRI showed normal cerebellar and posterior fossa structures, with no evidence of cystic dysplasia or hypoplasia. Orbital MRI was unremarkable. Despite normal neuroimaging, genetic analysis (NGS/WES) identified a homozygous pathogenic variant in the LAMA1 gene, confirming the diagnosis of Poretti–Boltshauser syndrome. Given the developmental delay compared to age-matched peers, a structured program of physical therapy and developmental rehabilitation was initiated, along with regular ophthalmological and neurological follow-up. Conclusion: This case illustrates that Poretti–Boltshauser syndrome is not limited to the presence of typical cerebellar anomalies and may be diagnosed even in the absence of characteristic radiological findings. In early childhood, brain MRI may appear normal, potentially creating a false sense of diagnostic reassurance and leading to overlooked genetic etiologies or delayed diagnosis. Our findings demonstrate that, despite the lack of a radiological phenotype, careful clinical evaluation based on key clinical clues—particularly developmental delay and associated ocular abnormalities—can enable timely diagnosis. Therefore, rare syndromes such as Poretti–Boltshauser syndrome should be considered in the differential diagnosis of children presenting with developmental delay and ocular findings, and genetic analyses should be planned at an early phase of evaluation.

Keywords: Poretti–Boltshauser syndrome, intellectual disability, ocular findings, magnetic resonance imaging

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[Abstract:0168]

Cotard Syndrome in a 50-Year-Old Female

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The Cotard delusion (CD) is identified as a specific subtype of monothematic delusion in which patients exhibit profound nihilistic belief involving negation of bodily existence and denial of life (1). The condition is frequently associated with severe depressive episodes, psychotic disorders, and certain neurological abnormalities. Comprehensive understanding of the sociodemographic profile, phenomenology, and psychiatric history of affected individuals is essential for accurate diagnosis and effective management.

The patient is a 50-year-old married woman, presented in April 2025 with complaints of persistent sensations of cardiac disruption, accompanied by nihilistic delusions expressed as herself was dead.

Clinical evaluation revealed the patient experienced recurrent visual hallucinations of a girl resembling her childhood self, for years. According to collateral history from her spouse, the patient had a longstanding pattern of nihilistic ideation, including statements such as “I am bone” and “I am gone, bury me,” which had resurfaced over the preceding 1–2 months. Episodes of psychomotor retardation, including refusal to leave bed, food intake reduction, and diminished responsiveness, were documented. The patient attributed these behavioral manifestations to the perceived disappearance of herself in an alternate dimension, leaving her in a skeletal state, thereby inhibiting verbal or motor responses.

The patient’s psychiatric history indicates onset of nihilistic delusions approximately 20 years prior. About 20 years ago, she experienced severe functional decline, including prolonged bed rest and refusal of oral intake, necessitating psychiatric hospitalization, during which she received electroconvulsive therapy. Post-discharge, the intensity of nihilistic delusions diminished, with intermittent recurrences lasting 1–2 months, after which she resumed routine activities. Outpatient management over the years included olanzapine 10 mg/day and risperidone 2 mg/day, with sertraline 50 mg/day added within the last 3–4 years.

Upon admission to the psychiatric inpatient unit in April 2025, risperidone and sertraline were discontinued, and olanzapine was titrated to 20 mg/day. Despite one month of treatment, the patient exhibited no attenuation of her nihilistic delusions and exhibited food refusal and medication noncompliance, asserting that she was “bone.” Consequently, olanzapine was discontinued, and a transition to risperidone was initiated. The patient subsequently underwent 8 sessions of electroconvulsive therapy (ECT). Over the course of the ECT sessions, a progressive remission of her nihilistic delusions and visual hallucinations was observed. She was discharged with risperidone 4 mg/day and biperiden 2 mg/day.

The patient exhibited a chronic and fluctuating pattern of nihilistic delusions extending over two decades, characterized by intermittent periods of remission and relapse. This case contributes to the clinical understanding of Cotard delusion by illustrating prolonged course with intermittent symptom reemergence, visual hallucinations, and treatment resistance requiring ECT. This case underscores the importance of comprehensive clinical evaluation—including detailed psychiatric history, assessment of functional impairment, and longitudinal symptom patterns. The therapeutic response to ECT in this case further supports its position as an intervention in the management of severe or refractory presentations of Cotard delusion.

(1) Bott, N., Keller, C., Kuppuswamy, M., Spelber, D., & Zeier, J. (2016). Cotard Delusion in the Context of Schizophrenia: A Case Report and Review of the Literature. Frontiers in psychology,

Keywords: Cotard delusion, Nihilistic delusions, Treatment resistance, Electroconvulsive therapy

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[Abstract:0174]

A Systemic and Relapse-Prevention Approach to Alcohol Use Disorder in a Surgeon: Partner-Involved Treatment

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Introduction:
This poster presents a structured, time-limited, systemic intervention for Alcohol Use Disorder (AUD) in a high-functioning individual, emphasizing relapse prevention, partner involvement, and behavioral chain analysis. The objective was to move beyond abstinence to establish sustainable self-management, improve dyadic coping, and restructure the partner's role from "protector" to supporter of recovery.

Case presentation:

A 47-year-old married female surgeon presented with Alcohol Use Disorder. Treatment comprised 12 systemic sessions over three months, involving both the patient and her spouse. Written informed consent was obtained from both partners for the anonymous presentation of this case. The core clinical focus was partner-related triggers and the interpersonal consequences of drinking. Clinical progress and symptom severity were monitored using the Alcohol Use Disorders Identification Test (AUDIT).

The intervention was multi-faceted: 1. Individual Self-Management: The patient completed a self-management planning sheet, identifying a hierarchy of high-risk triggers. For each trigger, she mapped the behavior chain leading to drinking and developed alternative coping strategies (e.g., urge-surfing techniques, scheduled alternative activities). She practiced drink refusal skills and challenged "dangerous thoughts" about alcohol's perceived benefits. 2. Dyadic System Restructuring: The couple worked jointly to devise a practical plan for managing alcohol in the home. A critical component involved the partner listing his past "protective" behaviors (e.g., hiding bottles, making excuses) that inadvertently enabled the AUD. He then formulated new, "non-protective" responses to support accountability. The couple was trained in constructive conflict resolution and joint problem-solving to reduce relational stress as a trigger.3. Relapse Prevention Framework: The therapy focused on increasing the tangible positive rewards of sobriety. For each identified trigger situation, the patient analyzed the perceived positive consequence of drinking (e.g., tension relief) and identified an alternative activity yielding a similar benefit. Early warning signs of relapse were identified collaboratively, and concrete plans were devised for their management.

Conclusion:
This case demonstrates the efficacy of integrating cognitive-behavioral relapse prevention with systemic couple therapy for AUD. Engaging the partner was not merely supportive but instrumental in changing the dysfunctional relational "dance" that maintained the addiction. By having the partner shift from protective enabling to supportive accountability, the home environment was transformed into a recovery-facilitating space. The use of the AUDIT provided a baseline and a metric to track reductions in hazardous use. The structured use of self-management planning and behavior chain analysis empowered the patient with insight and practical tools, moving her from a passive to an active role in her recovery. The emphasis on substituting the function of alcohol with healthier alternatives addresses the core drivers of use, making sobriety more rewarding and sustainable. For high-functioning individuals like healthcare professionals, where stigma and professional consequences are significant, this discrete, skills-focused, and dyadic approach can be particularly acceptable and effective. Future directions include the use of additional standardized measures, such as the Dyadic Adjustment Scale (DAS), pre- and post-intervention to quantitatively assess improvements in relationship functioning. The case underscores that treating AUD in a relational context can build a robust, shared system for long-term recovery management.

Keywords: Alcohol Use Disorder, systemic couple therapy, relapse prevention, behavioral chain analysis, dyadic coping, high-functioning individual

[Abstract:0176]

Paliperidone-Induced Toxic Hepatitis in a Patient with Psychosis: A Case Report

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Introduction: Paliperidone is an atypical antipsychotic and the active metabolite of risperidone, predominantly eliminated via renal excretion with minimal hepatic metabolism. Therefore, clinically significant hepatotoxicity is considered uncommon. Nevertheless, rare cases of drug-induced liver injury associated with paliperidone have been reported, usually presenting as idiosyncratic reactions (Khorassani et al., 2020). We report a case of severe toxic hepatitis temporally associated with paliperidone treatment in a patient with a psychotic disorder.

Case presentation: A 50-year-old woman was referred to our psychiatric ward following corneal transplantation due to severe self-inflicted ocular injury. The injury resulted from a delusional belief that a foreign body and inflammation were present in her eye. For approximately one year, she repeatedly manipulated her eye and perioral area in attempts to remove the perceived object, ultimately leading to corneal melting.

The patient had been followed intermittently for many years with diagnoses of anxiety disorder, depression, and conversion disorder, without a previously established diagnosis of psychosis. Retrospective evaluation revealed longstanding functional impairment, including social withdrawal, reduced self-care, and decline in daily functioning. Mental status examination demonstrated somatic delusions and tactile hallucinations related to the perceived foreign body, with partial insight and impaired judgment.

Prior to hospitalization, treatment with amisulpride had been initiated in the outpatient setting, with a plan to titrate the dose up to 600 mg/day. However, due to poor medication adherence and persistence of symptoms, inpatient admission was planned for treatment optimization. At admission, mild elevations in liver function tests were noted. Given treatment nonadherence and the expectation of low hepatotoxic risk, paliperidone was initiated with the plan to transition to a long-acting injectable formulation.

Approximately one week after initiation, liver enzymes increased markedly. Although the patient remained asymptomatic, laboratory evaluation revealed severe hepatocellular injury, with peak values of ALT: 1918 U/L, AST: 531 U/L, and GGT: 698 U/L. Hepatobiliary ultrasonography demonstrated grade 2–3 hepatic steatosis without biliary obstruction or focal hepatic lesions. Internal medicine consultation concluded that the clinical course and temporal relationship were consistent with paliperidone-induced toxic hepatitis. All psychotropic medications were discontinued, and the patient was followed with supportive care. Liver enzyme levels gradually declined to the normal level after drug withdrawal.

During the medication-free inpatient follow-up period, no worsening of psychotic symptoms or high-risk behaviors was observed. After discharge, amisulpride was reintroduced in the outpatient setting at a low dose of 200 mg/day without recurrence of liver enzyme elevation.

Conclusion: This case illustrates a rare but severe instance of paliperidone-induced toxic hepatitis in a patient with psychotic disorder. Similar cases in the literature describe hepatocellular injury occurring shortly after paliperidone initiation and resolving following drug discontinuation, supporting an idiosyncratic mechanism. Current reports emphasize prompt cessation of paliperidone, close biochemical monitoring, and avoidance of rechallenge. When antipsychotic treatment is required, switching to an alternative agent after normalization of liver enzymes has been suggested, without a mandatory washout period beyond biochemical recovery (Slim et al., 2016).

Keywords: Drug-induced liver injury, Paliperidone, Psychosis, Toxic hepatitis

[Abstract:0195][Kabul:Poster][B. Yetişkin Psikiyatri]

Resolution of Brain Fog in A Patient With Bipolar Depression Through Shift From Fluoxetine-Olanzapine Combination to Vortioxetine and Olanzapine in 3 Days

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Introduction: Fluoxetine-olanzapine combination is supported by several large randomized trials and meta-analyses in adults with bipolar depression [1]. However vortioxetine-olanzapine combination has very limited data [2]. While vortioxetine improved cognitive function in major depressive disorder studies, its effect on brain fog in bipolar depression has not been studied. We present a patient with bipolar depression remitted on fluoxetine combined with olanzapine but having complaint of brain fog and resolution of it with switching to vortioxetine in just three days.

Case presentation: A 65-year-old female patient with a diagnosis of bipolar disorder presented with a depressive episode lasting approximately one year, accompanied by a suicide attempt, inner restlessness, weight loss, and decreased energy. She was initiated on an olanzapine/fluoxetine combination (24/50 mg/day), valproic acid (1000 mg/day), and clonazepam (2 mg/day). After several weeks, the patient complained of subjective cognitive impairment described as "brain fog." Fluoxetine was discontinued and replaced with vortioxetine (10 mg/day), while olanzapine was continued at 10 mg/day. Valproic acid (1000 mg/day) and clonazepam (2 mg/day) were maintained without change. Within three days of this antidepressant switch, the patient reported a marked improvement in cognitive clarity.

Discussion: In this case, the improvement in cognitive functions was most likely related with switch from fluoxetine to vortioxetine in patient's treatment regimen while other drugs staying same. Vortioxetine, a multimodal antidepressant, enhances serotonergic activity while modulating several other neurotransmitter systems involved in emotional blunting, anhedonia, and cognitive dysfunction [3]. Vortioxetine shows promise in improving mood symptoms in bipolar depression while its effect on cognitive dysfunction ("brain fog") remains uncharted. In a naturalistic, open-label study of 60 patients treated for 24 weeks, vortioxetine, added to mood stabilizers—including olanzapine in some cases, yielded a 73% response rate and a 52% remission rate. This study did not assess cognitive outcomes, and so the time to cognitive improvement specifically in bipolar patients remains unknown [2]. In major depressive disorder, vortioxetine showed "significant differences from placebo as early as week two at a dose of 20 mg/day" and there are "dose-response relationships for cognitive benefits" with "higher doses (20 mg/day) associated with faster and more sustained effects" by Cuomo et al., 2024, though specific timing for cognitive improvement onset is not provided [3]. Our case used vortioxetine at a dose of 10 mg/day and brain fog subsided just in three days. The current evidence base for vortioxetine's effects on cognitive dysfunction ("brain fog") comes from major depression studies, there are no studies directly assessing cognitive outcomes in bipolar depression.

Conclusion: This case highlights if a patient with bipolar depression and cognitive complaints would use an antidepressant with mood stabilizer and antipsychotics giving a chance to vortioxetine may be worthy. Future research should elucidate the cognitive dysfunction in bipolar disorder and tailor treatment plans to individual needs which will improve treatment outcomes.

Keywords: Antidepressant switch, bipolar depression, cognitive dysfunction, vortioxetine

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[Abstract:0197]

Guanfacine in a Patient with ADHD and Hypertension: A Single Solution for Two Conditions?

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Introduction: Attention-Deficit/Hyperactivity Disorder (ADHD) is a common neurodevelopmental condition in childhood and adolescence, most frequently treated with stimulant medications. However, stimulant treatment may be limited in patients with cardiovascular comorbidities such as hypertension. This case aims to present the effectiveness of guanfacine in an adolescent with comorbid ADHD and hypertension.

Case presentation: A 14-year-old male was admitted to the child psychiatry outpatient clinic with complaints of inattention, hyperactivity, and behavioral problems. He had been diagnosed with ADHD at the age of 10 and previously treated with methylphenidate, from which he derived partial benefit. During stimulant treatment, elevated blood pressure levels were detected, leading to irregular use and eventual discontinuation of the medication. Despite cessation of stimulant therapy, hypertension persisted during pediatric follow-up. The patient was subsequently evaluated by pediatric cardiology, and propranolol treatment was initiated. Although partial improvement in blood pressure was achieved, ADHD symptoms persisted, and the patient was referred to our clinic.

On physical examination, blood pressure was measured as 140/90 mmHg. ADHD diagnosis was confirmed based on DSM-5 criteria and clinical rating scales. Routine laboratory investigations were unremarkable. Considering the coexistence of ADHD and hypertension, extended-release guanfacine was initiated at 1 mg/day and titrated to 3 mg/day at two-week intervals. During follow-up, marked improvement in ADHD symptoms and normalization of blood pressure values (120/80 mmHg) were observed. Mild transient sedation occurred during the early phase of treatment and resolved spontaneously. The patient continues to be followed up with pediatric cardiology.

Conclusion: This case suggests that guanfacine may be an effective and well-tolerated treatment option for adolescents with comorbid ADHD and hypertension, particularly when stimulant medications are contraindicated or poorly tolerated. Guanfacine may provide simultaneous control of both psychiatric symptoms and blood pressure. Further studies with larger samples are needed to clarify its role in this clinical population.

Keywords: Guanfacine, attention-deficit/hyperactivity disorder, hypertension

AuthorToEditor: Ceyda BIYIKLI ORCID- 0009-0009-2011-3006 Emine AYDEMİR ORCID- 0009-0006-9511-9910 Öznur ADIGÜZEL AKMAN ORCID- 0000-0001-8196-6664 Dear Members of the Scientific Committee, This submission is a case report presenting clinical observations on the use of guanfacine in a child diagnosed with Attention-Deficit/Hyperactivity Disorder (ADHD) who had comorbid hypertension and showed insufficient response to standard ADHD treatments. In this case, guanfacine demonstrated clinically meaningful effects on both ADHD symptoms and blood pressure regulation. Through this case, we aim to highlight the potential dual therapeutic benefit of guanfacine in treatment-resistant ADHD cases accompanied by hypertension. We believe that this report may contribute to clinical awareness and support individualized treatment approaches. Respectfully submitted

[Abstract:0229]

Hyperprolactinemia and Reversible Pituitary Lesion During Early Antipsychotic Treatment in First-Episode Psychosis: A Case Report

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Introduction: Hyperprolactinemia is a common adverse effect of antipsychotic treatment due to dopamine D2 receptor antagonism. Although prolactin elevation is usually considered a drug-related phenomenon, pituitary imaging abnormalities detected during antipsychotic treatment may raise diagnostic uncertainty between true adenomas and functional, reversible pituitary changes. We present a case of first-episode psychosis with marked hyperprolactinemia detected shortly after antipsychotic initiation and a transient pituitary lesion.[1] [2]

Case presentation: A 19-year-old female presented to the emergency department with bizarre and grandiose delusions involving her adrenal glands, severe psychological distress, and a suicidal gesture involving ingestion of bleach. According to her family, she had been experiencing paranoid delusions as well as auditory and visual hallucinations for approximately 1.5 years, with marked worsening over the preceding 6–7 months. She had no history of psychiatric treatment. Physical and neurological examinations were unremarkable. Initial laboratory investigations and brain MRI revealed no acute pathology.

Risperidone was initiated for treatment of first-episode psychosis at a dose of 2 mg/day and titrated to 4 mg/day over one week. As part of routine laboratory monitoring, a serum prolactin level measured on the third day of treatment was elevated at 128 ng/mL. Over the subsequent two weeks, the patient developed symptoms consistent with hyperprolactinemia, prompting further evaluation; risperidone was discontinued, olanzapine was initiated and titrated up to 30 mg/day, and aripiprazole 5 mg/day was added.

An endocrinology consultation was obtained, and secondary causes of hyperprolactinemia—including hypothyroidism, pregnancy, renal failure, and macroprolactinemia—were excluded. Pituitary MRI demonstrated a 6 × 2 mm non-enhancing lesion in the pars intermedia, consistent with either a pituitary microadenoma or functional cystic change.

On follow-up, the prolactin level decreased to 28.6 ng/mL, and repeat pituitary MRI showed resolution of the previously identified lesion. The patient was discharged after significant clinical improvement, accompanied by reduced PANSS scores. She is currently in remission, with normalized prolactin levels and stable pituitary findings on follow-up MRI.

Conclusion: This case illustrates that marked hyperprolactinemia and pituitary imaging abnormalities may emerge early during antipsychotic treatment in first-episode psychosis. Aripiprazole has a unique pharmacological profile due to its partial agonist activity at dopamine D2 receptors, with a lower risk of extrapyramidal symptoms and minimal prolactin elevation compared to other antipsychotics. The disappearance of the pituitary lesion after normalization of prolactin levels suggests a dynamic, functional pituitary change rather than a fixed structural adenoma. Adjunctive aripiprazole was effective in reducing prolactin levels while maintaining antipsychotic efficacy, supporting its role in the management of antipsychotic-induced hyperprolactinemia.[1] [2] [3]

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Keywords: Aripiprazole, First-Episode Psychosis, Hyperprolactinemia, Pituitary Lesion, Risperidone

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[Abstract:0233]

Diagnostic Value of a Structured Mental State Examination With Sign Language Interpretation: A Case of Schizophrenia with Preschool-Onset Total Deafness and Speech Impairment

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Introduction

Psychiatric assessment of psychosis in deaf individuals is challenging due to communication barriers, limited clinician familiarity with Deaf culture, and structural limitations in healthcare systems (2,4).

This case presents a patient with total deafness and speech impairment acquired during the preschool period, followed for schizophrenia for many years.

This case illustrates diagnostic challenges and underscores the clinical importance of a structured mental status examination with professional sign language interpreter support to improve diagnostic accuracy.

Case

A 70-year-old male patient developed bilateral total hearing loss at age 5 following a traffic accident. He learned sign language through state-supported education and has documented total deafness with speech impairment, without hearing aids or cochlear implant use.

He has been diagnosed with schizophrenia since age 23 and obsessive–compulsive disorder with hoarding behavior for the past six years. There is no history of substance use, and a second-degree family member has schizophrenia. Dementia was excluded by neurology.

He has been hospitalized five times. Relapses were associated with treatment nonadherence, aggression and homicidal threats expressed through sign language.

Mental Status Examination and Methods:

A certified sign language interpreter trained in ethics and confidentiality provided, direct translation without commentary. Written informed consent was obtained from the patient and legal guardian for the examination and case publication.

Written question cards were prepared. During written communication, the patient failed to understand abstract concepts such as “suicide,” “hearing,” “to command,” and “divorce.” When the same questions were asked using sign language, meaningful responses were obtained. In writing, the patient denied persecution and visual hallucinations; however, through the interpreter, he described foreign warplane attacks, police surveillance, and threats by devils.

The patient was asked whether he experienced voices speaking to him, heard sounds, perceived communication through sign language, or received messages as written text, images, or lip movements in his mind (6). He reported only long-standing nonverbal auditory experiences described as “cık cık,” “dın dın,” and “zın zın,” which caused distress. Attention was limited, associations were loose, and insight was absent.

Differential Diagnosis: Early sign language training, episodic psychosis, absence of insight, and poverty of thought exclude language deprivation syndrome. (7).

Conclusion

Prelingual hearing loss typically associated with visual, somatic and simple vocal experiences, and postlingual hearing loss with more complex, speech-like hallucinations (1). In this case, hearing loss occurred during the preschool period, after initial language development but before literacy and abstract thinking were fully established (5). Terminology such as peri-lingual or early post-lingual deafness, used in the otorhinolaryngology literature, is proposed for consideration in psychiatric practice (5).

This case demonstrates that reliance on written communication alone can yield false-negative findings, with delusions and hallucinations identified only through sign language interpreter–assisted assessment.

This examination was the first interpreter-assisted assessment conducted during the long course of the patient's illness and it highlights ongoing structural limitations in access to interpreter-supported psychiatric assessment (2, 3). A structured mental status examination with the support of a qualified mental health sign language interpreter who is familiar with deaf culture is required (4).

Keywords: Deafness, Schizophrenia, Sign Language

AuthorToEditor: https://orcid.org/0009-0005-9772-3786 Dear Members of the Scientific Committee, During my first year of residency, I attended the 14th ICP as an observer, an experience that left a strong and lasting impression on me. I am therefore highly motivated to be part of the congress again. This year, I am honored to submit my first case report as a poster presentation. Participation in the congress would be extremely valuable for my academic development; however, financial support is required for my attendance, and I have therefore applied for a scholarship. I would be grateful for the opportunity to join the congress and share my work, and I sincerely thank you for your efforts and consideration. Yours sincerely, Bengisu Nermin Yedievli

[Abstract:0268]

A Rare but Reversible Adverse Effect of Escitalopram: Drug-Induced Alopecia

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Introduction

Selective serotonin reuptake inhibitors (SSRIs) are widely used for depressive and anxiety disorders because they are effective and generally well tolerated. While their adverse effects are most often discussed in terms of gastrointestinal complaints, sleep changes, or sexual dysfunction, dermatologic reactions are less frequently recognized in routine practice. Hair loss is a rare adverse effect reported with SSRIs, typically described through case reports and small series.

Escitalopram, the S-enantiomer of citalopram, is commonly preferred due to its high selectivity for the serotonin transporter and a relatively favorable drug–drug interaction profile, particularly in older adults. Despite its overall safety, a small number of reports have linked escitalopram to diffuse hair loss that emerged weeks to months after initiation and improved after discontinuation or switching to another antidepressant. These observations suggest a reversible and possibly drug-specific phenomenon in susceptible individuals.

The mechanism of SSRI-related hair loss is not fully established. However, the diffuse, non-scarring pattern described in most reports is consistent with telogen effluvium. It has been proposed that changes in local neuroendocrine signaling at the hair follicle—potentially involving serotonin and melatonin pathways—may disrupt the hair growth cycle and trigger premature transition from anagen to telogen. Recognizing this uncommon adverse effect is clinically important, as hair loss may substantially affect quality of life and treatment adherence.

Case Presentation

A 71-year-old woman with no known chronic medical conditions presented with anxiety, low mood, reduced motivation, pessimistic thoughts, and difficulty initiating sleep. Over recent months, she had been providing full-time, unassisted care for her 102-year-old mother, which markedly increased psychosocial stress. She was evaluated at an external center and started on escitalopram 10 mg/day.

By the second week of treatment, the patient reported marked improvement in her psychological symptoms and tolerated the medication well, with no early adverse effects. A six-month treatment plan was established. During the second month of treatment, however, she developed diffuse hair loss. She consulted her primary care physician, and an evaluation was performed including complete blood count, iron studies, thyroid function tests, vitamin B12 and folate levels, fasting glucose, and HbA1c. All results were within normal limits. Review of prior medical records documented only a history of vitamin D deficiency.

Given the temporal relationship between escitalopram exposure and the onset of hair loss, drug-induced alopecia was considered. Escitalopram was discontinued and treatment was switched to sertraline 50 mg/day. During follow-up, a marked reduction in hair shedding was observed. Written informed consent for scientific use of clinical data was obtained from the patient.

Conclusion

Escitalopram-associated hair loss is a rare but clinically meaningful adverse effect that appears reversible in many cases. This report highlights the need to consider antidepressant-related alopecia in patients with new-onset diffuse, non-scarring hair loss after SSRI initiation, once common medical causes have been excluded. Switching to an alternative antidepressant may be an effective management strategy and can help maintain adherence while minimizing patient distress.

Keywords: Drug-induced alopecia, Escitalopram, Telogen effluvium

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[Abstract:0272]

Co-occurrence of Sleep Attacks and Neuropsychiatric Symptoms: Initial Experience of Kleine–Levin Syndrome in Our Clinic

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Introduction:

Kleine-Levin Syndrome (KLS) is a rare sleep disorder reported to affect 1-4 individuals per million, characterized by episodes of hypersomnolence accompanied by cognitive and behavioral disturbances such as derealization, confusion, apathy, compulsive eating, and hypersexuality. Episodes typically last from a few days to several weeks; these periods are followed by inter-episodic phases during which sleep and behavior patterns return to normal. This case report aims to present the clinical manifestations of a patient who presented to our outpatient clinic. Written informed consent was obtained.

Case presentation:

A 20-year-old male university student presented via e-mail suspecting he had Kleine-Levin Syndrome. He reported four 7–10 day episodes over four years, characterized by sleeping up to 20 hours daily. During wakeful hours, he experienced intense sleep urge, clouded consciousness, derealization, hyperphagia, and hypersexual behaviors (frequent masturbation while drowsy). Perceptual disturbances included blackouts, visual hallucinations of dogs, and auditory hallucinations of music. Post-episode phases were marked by high energy and self-confidence despite 48-hour insomnia. The first episode involved intense suicidal ideation, while others were triggered by exam stress or physical activity.

Previously, he was prescribed Olanzapine (2.5 mg/day), Moclobemide (150 mg/day), and Clomipramine (10 mg/day) without a formal diagnosis. At presentation, symptomatic Methylphenidate (10 mg/day) and Sertraline (50 mg/day) had been recommended. Our evaluation confirmed KLS, as the patient met all ICSD-3 criteria. There was no other comorbid psychiatric diagnosis. Psychoeducation was provided to both the patient and his family. Lithium and Modafinil were discussed, along with the information regarding the lack of sufficient evidence for these treatments. Although the patient was willing to undergo Lithium treatment, the family did not consent, and the patient complied with the family's recommendation. Modafinil 200 mg/day was recommended for use during symptomatic periods. The patient was specifically cautioned regarding precipitating factors and potential mood changes, as well as cognitive and behavioral symptoms during episodes.

Conclusion:

Kleine-Levin Syndrome (KLS) is accompanied by many neuropsychiatric symptoms in its clinical presentation, and there are no specific biomarkers or neuroimaging findings unique to the diagnosis. Our case, which fulfills all the criteria mentioned in the diagnostic guidelines, aims to draw the attention of clinicians to the clinical features of the disease and to protect the patient from heavy pharmacological treatments. If symptoms have a sudden onset and follow an episodic course, and if neuropsychiatric symptoms accompany the clinical picture, KLS can be considered in the differential diagnosis. Although various pharmacological agents are reported in the literature to have therapeutic responses during or between episodes, evidence-based data supporting the efficacy of these treatments are still limited. In this regard, preventive recommendations may stand out as a reliable and effective treatment strategy option.

Keywords: Hypersomnolence, Hyperphagia, Derealization, Hypersexuality, Neuropsychiatry

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[Abstract:0281]

Aripiprazole-Induced Late-Onset Visual Disturbance: A Case Report

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Introduction: Second-generation antipsychotics are widely used in the treatment of psychotic disorders due to their efficacy and relatively favorable tolerability profiles. ripiprazole is one of the frequently preferred agents due to its partial dopamine D2 receptor agonist activity, which is associated with a relatively lower risk of hyperprolactinemia. Despite its generally favorable tolerability profile, rare visual adverse effects have been reported in association with aripiprazole use (1,2). In this case report, we present a transient episode of blurred vision that developed following dose reduction after long-term aripiprazole treatment and resolved after drug discontinuation.

Case presentation: A 21-year-old female patient has been followed for approximately two years with a diagnosis of non-organic psychosis. Due to hyperprolactinemia associated with previous antipsychotic treatments, her medication was changed to aripiprazole 30 mg/day approximately one year earlier. During treatment, significant improvement in psychotic symptoms, increased functioning, and affective improvement were observed, and the patient began university education. No significant treatment-related adverse effects were observed. Given the persistence of clinical stability, the dose of aripiprazole was gradually tapered to 10 mg/day. The patient remained on this dose for approximately three months. At the end of this period, the patient reported a complaint of blurred vision. The medication was discontinued on the day symptom onset occurred; nevertheless, the visual disturbance persisted for approximately one week and then resolved spontaneously. During this period, no accompanying symptoms such as eye pain, redness, headache, nausea, or vomiting were reported. Due to the transient course of the symptoms and their spontaneous resolution, a detailed ophthalmological evaluation was not performed. The patient has been followed without pharmacological treatment for the past four months, remains in full clinical remission, and has not experienced any recurrence of visual symptoms. Written informed consent was obtained from the patient for the publication of this case report.

Conclusion: This case demonstrates that visual adverse effects associated with aripiprazole use may occur not only during treatment initiation but also after long-term stable use and during periods of dose reduction. According to the literature, the most commonly reported ocular adverse effects associated with aripiprazole include blurred vision and transient myopia, which resolve completely in most cases following discontinuation of the medication (1,2). Nevertheless, it should be taken into account that serious conditions such as acute angle-closure glaucoma, although rare, have been reported, and newly emerging visual symptoms should be carefully evaluated (3). In this case, the persistence of visual symptoms for a short period despite drug discontinuation suggests that the pharmacodynamic effects of aripiprazole may resolve gradually over time. Although the lack of a detailed ophthalmological evaluation represents a limitation, the spontaneous resolution of symptoms without permanent sequelae is consistent with the transient and reversible pattern of ocular adverse effects described in the literature (2). Moreover, recent analyses examining real-world adverse event reports support that ocular adverse effects associated with atypical antipsychotics can be encountered in clinical practice and underscore the importance of clinical awareness (4). In clinical practice, monitoring patients for visual symptoms during dose adjustments of aripiprazole treatment is of clinical importance.

Keywords: Aripiprazole, blurred vision, antipsychotic adverse effects

AuthorToEditor: Yazılı onam aldığımı önceki gönderide yazmayı unuttuğum için metin içine yazarak tekrar gönderiyorum. ayrıca orcid numaramı ve kaynaklarıda eklemeiştim. buraya ekliyorum. ORCID:0000-0002-5206-8774 Kaynaklar 1.Karadağ H, Acar M, Özdel K. Aripiprazole induced acute transient bilateral myopia: a case report. Balkan Med J. 2015 Apr;32(2):230-2. doi:10.5152/balkanmedj.2015.15292. 2.Bulgu Y, Genc S. Aripiprazole-induced acute transient bilateral myopia: a case report. Beyoglu Eye J. 2020;5(1):57–58. doi:10.14744/bej.2020.71677. 3.Vishwanath R, Soman S, Mysore Nagaraj AK, et al. Acute angle-closure glaucoma associated with aripiprazole: a case report. Indian J Psychol Med. 2024;46(3):273–274. doi:10.1177/02537176231199210. 4.Mu C, Chen L. Characteristics of eye disorders induced by atypical antipsychotics: a real-world study from 2016 to 2022 based on Food and Drug Administration Adverse Event Reporting System. Front Psychiatry. 2024;15:1322939. doi:10.3389/fpsyt.2024.1322939.

[Abstract:0292]

Guanfacine Treatment for Polypharmacy-Resistant Behavioral Problems in Autism

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Introduction: Autism spectrum disorder is a neurodevelopmental disorder characterized by deficits in social communication and interaction beginning in early childhood, along with restricted, repetitive field of interests and behaviors. In some cases, severe and treatment-resistant self-injury, aggression, and irritability accompany the clinical picture. This case presentation discusses symptom regression with guanfacine treatment in a typical autism case resistant to multiple medications.

Case presentation: A 6-year-old and 3-month-old male patient was brought to our clinic due to aggression, self-harm, and harm to others. The evaluation revealed that the patient had been diagnosed with typical autism at age 2 and had begun special education, that his 9-year-old brother was being monitored and treated for autism and attention deficit hyperactivity disorder, that the mother had a phonological disorder and intellectual disability, and that the father was in prison due to substance abuse. Medical records indicate that the patient had previously used olanzapine 10 mg/day, haloperidol 2 mg/day, methylphenidate 30 mg/day, aripiprazole 5 mg/day, and risperidone 2 mg/day, but these treatments were ineffective. It was learned that methylphenidate treatment increased irritability and aggression, and olanzapine caused sedation as a side effect. As the patient's behavioral problems persisted during follow-ups, atomoxetine 35 mg/day, risperidone 4 mg/day, and quetiapine IR and XR 50 mg/day treatments were initiated at different times, but no response was obtained. Due to the patient's significant behavioral problems and hyperactivity, as well as repeated attempts to run away from school and his mother, he was brought to appointments by his mother tied with a rope or with handcuffs on his wrist. Guanfacine was added to the ongoing and ineffective treatments of risperidone 2 mg/day and quetiapine 25 mg/day, and the dose was gradually increased to 3 mg/day. In subsequent sessions, it was learned that the patient's behavioral problems and hyperactivity had decreased, there were no escape behaviors, and he was able to adapt to special education. It was observed that the mother was able to bring the patient to appointments without using restraints and could use public transportation. Although the mother was cooperative, she had difficulty adhering to treatment due to financial problems and cognitive limitations, so a social investigation of the family was initiated. Due to the presence of different neurodevelopmental disorders in first-degree relatives, the patient was referred to the neurology unit for further genetic investigation.

Conclusion: The presented case addresses the efficacy of guanfacine treatment in managing severe behavioral problems and impulsivity resistant to multiple drug therapies in patients diagnosed with autism. Although guanfacine has FDA approval for attention deficit hyperactivity disorder, its use as monotherapy or adjuvant therapy should also be considered for the treatment of behavioral problems, aggression, hyperactivity, and impulsivity. Our case also emphasizes the importance of evaluating behavioral problems in patients diagnosed with autism not only on a neurobiological basis but also in the context of genetic predisposition, low socioeconomic status, and weak psychosocial support systems.

*Verbal and written consent was obtained from the patient’s parents.”

Keywords: Autism, guanfacine, polypharmacy

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[Abstract:0293]

Sertraline Treatment in a Patient With Anxiety Disorder Presenting With Pathological Laughter: A Case Report

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Introduction: Pathological laughter, also called inappropriate or paradoxical laughter, is characterized by involuntary, excessive, and context-incongruent episodes of laughter. It is most commonly associated with neurological conditions such as stroke, traumatic brain injury, neurodegenerative disorders, or pseudobulbar affect and is typically conceptualized as a dysfunction within cortico–limbic–subcortical neural networks. In many cases, pathological laughter coexists with pathological crying and is accompanied by identifiable structural or functional abnormalities of the central nervous system.

Reports of pathological laughter occurring in the absence of neurological pathology are rare, and its association with primary psychiatric disorders remains insufficiently described. Altered emotional regulation and serotonergic modulation have been proposed as potential mechanisms underlying such presentations. This case report describes an adolescent patient with anxiety disorder who presented with isolated pathological laughter in the absence of neurological disease while receiving sertraline treatment.

Case Presentation: An 18-year-old female patient presented with a one-year history of involuntary laughter episodes occurring in inappropriate contexts, particularly during stressful situations such as examinations or interpersonal conflicts. The laughter episodes were experienced as uncontrollable and were associated with significant social impairment, avoidance behaviors, and anticipatory anxiety. The patient also reported prominent anxiety symptoms, including excessive worry, inner tension, sleep disturbance, palpitations, irritability, fatigue, and decreased concentration. Family members confirmed emotionally labile behavior and inappropriate laughter during serious conversations. Mental status examination revealed an anxious mood with incongruent affect and frequent laughter during the interview, while thought process, perception, judgment, and insight were intact.

Psychometric assessments indicated moderate anxiety and depressive symptoms. Neurological evaluation, including cranial magnetic resonance imaging and computed tomography, revealed no structural abnormalities, and neurological consultation excluded an organic etiology. Laboratory investigations were unremarkable except for iron deficiency. Based on the clinical findings, a diagnosis of anxiety disorder accompanied by pathological laughter was established. Treatment with sertraline 50 mg/day and iron replacement therapy was initiated. The patient tolerated treatment well without adverse effects. At follow-up on day 44, pathological laughter had resolved, anxiety symptoms had markedly decreased, and functional improvement was observed. Follow-up psychometric assessments demonstrated significant reductions in anxiety and depressive symptom scores.

Conclusion: Pathological laughter is most frequently reported in association with neurological disorders and is often accompanied by pathological crying. In contrast, the present case demonstrated isolated pathological laughter without neurological deficits or pathological crying, suggesting a distinct clinical presentation. The clinical improvement observed during sertraline treatment supports the hypothesis that serotonergic mechanisms and anxiety-related emotional dysregulation may contribute to pathological laughter in the absence of organic pathology.

This case underscores the importance of considering psychopharmacological factors and psychiatric etiologies in patients presenting with pathological laughter after neurological causes have been excluded. Increased awareness of this atypical presentation may help prevent misdiagnosis, unnecessary neurological investigations, and delays in appropriate treatment.

Keywords: Anxiety disorder, Pathological laughter, Sertraline

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[Abstract:0294]

Sertraline-Associated Acute Urinary Retention Following Dose Escalation: A Case Report

Salim Çağatay Kağızman

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Introduction: Acute urinary retention (AUR) is a painful and potentially serious urological emergency that may result in urinary tract infection and acute renal failure if not promptly recognized and treated. While AUR is most frequently associated with obstructive causes, non-obstructive, drug-induced cases are increasingly recognized in clinical practice. Several psychotropic medications, particularly those with anticholinergic properties, have been implicated in the development of AUR. Selective serotonin reuptake inhibitors (SSRIs) are generally considered to have minimal anticholinergic effects; however, rare cases of SSRI-associated urinary retention have been reported. Sertraline, one of the most commonly prescribed SSRIs, is usually well tolerated, yet its effects on lower urinary tract function are not fully understood. Serotonergic pathways are known to play an important role in the central and peripheral regulation of micturition, suggesting a possible mechanism for this rare adverse effect. This report presents a case of sertraline-associated acute urinary retention occurring shortly after dose escalation, highlighting the importance of clinical awareness of this uncommon but clinically significant adverse event.

Case presentation: A 55-year-old female patient with fibromyalgia and anxiety symptoms who developed acute urinary retention following sertraline dose escalation is presented. The patient had been receiving duloxetine for fibromyalgia-related pain, which was gradually tapered and discontinued due to insufficient clinical benefit. Sertraline was initiated at 25 mg/day and titrated to 50 mg/day, resulting in partial improvement of anxiety symptoms. Due to residual symptoms, the dose was increased to 100 mg/day. Ten days after dose escalation, the patient developed dysuria, urinary frequency, decreased urinary output, and suprapubic discomfort. Laboratory investigations, including complete blood count, biochemical analysis, and urinalysis, were within normal limits. Urinary ultrasonography revealed a post-void residual volume of 280 mL. A pressure–flow study demonstrated a normal bladder–urethral pressure-flow profile, excluding obstructive pathology. Urethral catheterization was performed, leading to symptomatic relief.

Given the close temporal relationship between sertraline dose escalation and symptom onset, sertraline was tapered and discontinued. Spontaneous micturition resumed within a few days after discontinuation, and the catheter was subsequently removed without recurrence of urinary symptoms. For ongoing anxiety symptoms, escitalopram 10 mg/day was initiated, which was well tolerated and not associated with any urinary adverse effects during follow-up. The resolution of symptoms following sertraline discontinuation strongly supported a drug-induced etiology. Conclusion: Although rare, sertraline-associated acute urinary retention represents a clinically important adverse effect, particularly following dose escalation. Experimental and clinical evidence suggests that serotonergic modulation of lower urinary tract function may increase external urethral sphincter tone and inhibit the micturition reflex through central mechanisms involving the Onuf nucleus and lumbosacral autonomic pathways. Failure to recognize this association may lead to unnecessary invasive diagnostic procedures and delays in appropriate management. Clinicians should consider medication-related causes, including SSRIs, in patients presenting with new-onset urinary symptoms during antidepressant treatment. Increased awareness of this rare adverse effect may facilitate timely intervention and improve patient safety.

Keywords: Acute urinary retention, Selective serotonin reuptake inhibitors, Sertraline

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[Abstract:0308]

Pharmacological Management of Inappropriate Sexual Behaviors in a Adolescent With Autism Spectrum Disorder: Use of GnRH Agonists

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Introduction: Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by deficits in social communication, restricted interests, and repetitive behaviors. In individuals with ASD, increased sexual urges during adolescence may manifest as inappropriate sexual behaviors and irritability. In resistant cases where standard pharmacological treatments are ineffective, alternative approaches are needed. This case report presents and discusses the effectiveness of GnRH analog therapy in managing inappropriate sexual behaviors in a adolescant patient with ASD, whose symptoms were resistant to various antipsychotic and psychotropic medications.

Case presentation: A 15-year-old nonverbal male patient diagnosed with ASD presented to our clinic due to inappropriate sexual behaviors and increased irritability. The patient had a history of short-term risperidone and aripiprazole use until the age of 10 for stereotypies and self-injurious behaviors. From the age of 10, due to the escalation of inappropriate sexual behaviors and aggression, treated with risperidone, olanzapine, quetiapine, and aripiprazole at effective doses for three years; however, no clinical benefit was observed. At age 13, a combination therapy of olanzapine and guanfacine was initiated. Despite one year of treatment, there was no improvement.

Due to persistent inappropriate sexual behaviors and irritability despite multiple antipsychotic treatments, the patient was referred to pediatric endocrinology. It was considered that the patient might benefit from Gonadotropin-Releasing Hormone agonist (GnRH) therapy. After informing the family about off-label drug prescription, the treatment process, and potential side effects, and obtaining their informed consent, 3.75 mg of leuprolide acetate was initiated intramuscularly every 28 days. The patient was evaluated at four-week intervals to monitor potential side effects and assess the severity of the condition. Symptom severity was evaluated by the clinician using the Clinical Global Impression-Severity scale.

During the first dose of 3.75 mg leuprolide acetate, the patient continued his existing olanzapine and guanfacine regimen. At the evaluation four weeks after the first injection, the family reported a decrease in inappropriate sexual behaviors and irritability. The clinician-rated CGI-S score dropped from 6 to 4, and no side effects were reported. Following the second injection, as the CGI-S score decreased to 2, olanzapine treatment was gradually tapered and discontinued.

The patient's serum total testosterone level, which was 455 ng/dl prior to leuprolide acetate treatment, was measured at 7.62 ng/dl after the second injection. After the third injection, it was observed that the patient's symptoms had completely resolved, and the discontinuation of the current guanfacine treatment was planned. It has been observed that GnRH analog treatment reduced the patient’s inappropriate sexual behaviors and irritability symptoms, allowing for the tapering and eventual discontinuation of psychotropic medications. Consequently, it has been decided to continue the GnRH analog therapy, with regular follow-up appointments in the pediatric endocrinology and child psychiatry outpatient clinics throughout the treatment process.

Conclusion: GnRH analogs may be considered an alternative option for symptom control in selected resistant cases. However, due to limited data, widespread clinical use of this approach is not yet recommended.

Keywords: GnRH agonist, irritability, leuprolide acetate, sexual behaviour

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[Abstract:0315]

Escitalopram Discontinuation Related Brain Zaps in Panic Disorder: A Case Report

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[1]Kocaeli Şehir Hastanesi
[2]Başakşehir Çam ve Sakura Şehir Hastanesi

Introduction: Antidepressant discontinuation syndrome (ADS) comprises somatic, affective, and sensory symptoms that may occur after abrupt discontinuation or rapid dose reduction of serotonergic antidepressants. Meta-analytic data indicate that discontinuation symptoms occur in approximately 15% of patients, underscoring the clinical relevance of this phenomenon. “Brain zaps” are among the most distinctive symptoms and are typically described as brief electric shock-like sensations in the head, often triggered by head or sudden eye movements. Although ADS is more commonly associated with short half-life agents, similar sensory symptoms have been reported with selective serotonin reuptake inhibitors (SSRIs), including escitalopram. However, detailed clinical descriptions of escitalopram-related brain zaps in patients with panic disorder are limited. We report a case illustrating the clinical features, functional impact, and natural course of escitalopram discontinuation-related brain zaps in a patient with panic disorder.

Case presentation: A 31-year-old male with panic disorder and Hashimoto’s thyroiditis (treated with levothyroxine sodium 75 µg/day) was started on escitalopram. Baseline laboratory investigations were normal, and no psychiatric comorbidity was present. Escitalopram was initiated at 5 mg/day and increased to 10 mg/day after one week. During the first treatment week, the patient experienced decreased appetite and diarrhea, with approximately 3 kg unintentional weight loss; these symptoms resolved spontaneously. The patient achieved symptomatic stability approximately 40 days after treatment initiation and received regular follow-up with concurrent cognitive behavioral therapy.

After eight months of stable remission, escitalopram was reduced to 5 mg/day for one month, followed by alternate-day dosing for one week, and then discontinued. Within several days of discontinuation, the patient developed brief electric shock-like sensations in the head, predominantly triggered by head or sudden eye movements. Episodes lasted a few seconds and initially occurred approximately once per hour. The symptoms significantly impaired daily functioning because they were frequently triggered during movement. Mild subjective imbalance was reported, without true vertigo, visual symptoms, syncope, or focal neurological deficits. Neurological examination was normal, and the clinical presentation was clearly distinct from panic relapse. The patient declined pharmacological or symptomatic treatment and opted for reassurance and close follow-up. Over the following weeks, symptom frequency and intensity gradually decreased, with complete resolution within approximately one month. The Naranjo Adverse Drug Reaction Probability Scale score was 5, indicating a probable adverse drug reaction. According to Chouinard’s classification, the presentation was consistent with acute antidepressant withdrawal. Written informed consent was obtained.

Conclusion: This case highlights brain zaps as a distinctive and functionally impairing manifestation of antidepressant discontinuation. The consistent triggering by head and eye movements supports hypotheses implicating vestibulo-oculomotor or brainstem-related mechanisms. Although escitalopram is generally considered to have a lower risk of discontinuation symptoms, clinically significant sensory phenomena may still occur. Awareness of this presentation is important to avoid misinterpretation as panic relapse and to guide appropriate tapering and patient education strategies.

Keywords: Antidepressant discontinuation syndrome, brain zaps, escitalopram

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[Abstract:0316]

Escitalopram-Responsive Ménière's Disease with Symptom Recurrence After Discontinuation: A Case Report

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Introduction: Ménière's disease is a chronic inner ear disorder characterized by recurrent episodes of vertigo, fluctuating hearing loss, tinnitus, and aural fullness. Although its peripheral vestibular pathology is well established, accumulating evidence suggests that central mechanisms and psychosocial stress may influence attack frequency and severity. Psychiatric comorbidity and stress-related exacerbations are frequently reported in patients with Ménière's disease. Serotonergic pathways, particularly projections from the dorsal raphe nuclei to vestibular nuclei, have been implicated in vestibular processing and central sensory modulation. However, the potential role of selective serotonin reuptake inhibitors (SSRIs) in modulating Ménière's disease activity remains insufficiently described. We report a case in which escitalopram treatment was associated with sustained remission of Ménière's attacks, with symptom recurrence after discontinuation and resolution following re-initiation.

Case presentation: A 53-year-old male with no previous psychiatric history and a highly stressful professional life presented with recurrent vertigo attacks severe enough to require multiple emergency department visits. Following otolaryngological evaluation and appropriate diagnostic investigations, he was diagnosed with Ménière's disease. Standard management was initiated, including betahistine therapy and dietary sodium restriction. Despite adherence to treatment, vertigo attacks persisted and continued to significantly interfere with daily functioning and occupational performance.

Given the prominent psychosocial stress and the refractory nature of symptoms, a psychiatric consultation was requested. Although no formal depressive or anxiety disorder was diagnosed, significant work-related stress and heightened emotional distress were noted. Escitalopram was initiated at 5 mg/day and increased to 10 mg/day. Following initiation of escitalopram, the patient reported a marked reduction in vertigo attacks, with subsequent complete cessation of Ménière's episodes. He remained on escitalopram with regular follow-up and continued to be free of vertigo attacks for approximately two years, during which his occupational functioning and quality of life significantly improved.

After two years of stable remission, escitalopram was gradually reduced and discontinued. Approximately 10 days after complete discontinuation, the patient experienced recurrence of vertigo attacks consistent in severity and phenomenology with his previous Ménière's episodes. Escitalopram was subsequently re-initiated at the prior effective dose. Following re-initiation, the patient again experienced complete resolution of vertigo attacks, with no further episodes reported during follow-up. This reproducible temporal pattern suggested a relationship between escitalopram treatment and control of Ménière's disease activity.

Conclusion: This case suggests that escitalopram may play a modulatory role in Ménière's disease activity, potentially through central serotonergic mechanisms and stress-related pathways influencing vestibular processing. Although Ménière's disease is characterized by a fluctuating course and spontaneous remissions cannot be fully excluded, the clear challenge–dechallenge–rechallenge pattern observed in this case supports more than a coincidental association. Serotonergic modulation of vestibular nuclei and stress-related autonomic influences on endolymphatic homeostasis may represent plausible mechanisms. While escitalopram is not a standard treatment for Ménière's disease, this observation highlights the potential importance of central modulation in refractory vestibular disorders and warrants further systematic investigation.

Keywords: Escitalopram, Ménière's disease, Serotonergic modulation

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[Abstract:0338]

Severe Restrictive Anorexia Nervosa with Multi-Organ Failure: A Clinical Case Analysis

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Introduction: Anorexia nervosa is the psychiatric disorder with the highest mortality rate and may lead to severe medical complications in cases of extreme malnutrition. Prolonged starvation can result in life-threatening systemic consequences, including cardiac failure, coagulopathy, electrolyte imbalance, and neurological injury.

Case presentation: A 17-year-old female was admitted with profound weight loss, fatigue, and bradycardia. At presentation, her height was 159 cm, weight was 23 kg, and body mass index (BMI) was 9 kg/m². Restrictive eating behaviors had begun approximately one year earlier following peer criticism regarding her body shape. She progressively eliminated fats and carbohydrates, markedly restricted caloric intake, and denied binge eating or purging behaviors.

Due to severe cachexia and hemodynamic instability, she required intensive care unit admission. During hospitalization, she developed disseminated intravascular coagulation, acute heart failure requiring multiple vasoactive agents, persistent hypotension, and multi-organ dysfunction. Mechanical ventilation and subsequent tracheostomy were necessary. As her cardiovascular status deteriorated, she experienced sudden cardiovascular collapse culminating in cardiac arrest. Extracorporeal membrane oxygenation (ECMO) was considered; however, profound hemodynamic instability precluded safe transfer to a tertiary center for ECMO implementation. Following prolonged resuscitation efforts, return of spontaneous circulation was successfully achieved.

Refeeding was complicated by electrolyte imbalance consistent with refeeding syndrome. Magnetic resonance imaging revealed a pontine lesion compatible with central pontine myelinolysis. Additionally, superior mesenteric artery syndrome required jejunostomy placement for enteral nutrition. She was discharged from the intensive care unit in an immobilized condition with multiple pressure ulcers secondary to prolonged critical illness. With substantial family support, she underwent an intensive physical rehabilitation program and gradually regained motor function; she is currently able to ambulate independently.

With multidisciplinary medical and psychiatric management, gradual nutritional rehabilitation was achieved. Ten months after admission, her weight increased to 48 kg (BMI 19 kg/m²), and menstruation resumed following weight restoration. Psychiatric evaluation confirmed Restricting-Type Anorexia Nervosa with comorbid depressive symptoms and prominent perfectionistic traits. Pharmacological treatment with olanzapine 5 mg/day and fluoxetine 20 mg/day was initiated. Partial clinical benefit was observed; however, the patient demonstrated resistance and limited adherence to pharmacological treatment.

Conclusion: This case highlights that extreme restrictive anorexia nervosa can progress to catastrophic systemic complications, including multi-organ failure, cardiac arrest, prolonged immobilization, and severe neurological injury. The patient's course demonstrates how advanced malnutrition may lead to critical illness requiring intensive care, followed by significant functional impairment and prolonged rehabilitation. Despite the life-threatening medical course, substantial recovery was achieved through multidisciplinary management, structured nutritional rehabilitation, intensive physical therapy, and strong family support.

This case underscores the necessity of early recognition of medical instability in adolescents with severe anorexia nervosa and the importance of integrated psychiatric and medical intervention. It also illustrates the challenges of pharmacological management, as partial benefit may be observed despite limited adherence. Timely intervention and sustained family involvement remain essential in improving long-term functional and psychiatric outcomes in severe cases.

Keywords: Anorexia Nervosa, Severe Malnutrition, Multi-Organ Failure

Brain MRI (DWI): Central Pontine Myelinolysis



Magnetic resonance imaging demonstrated a 13 × 10 mm midline pontine lesion with peripheral diffusion restriction, highly suggestive of central pontine myelinolysis. Supratentorial imaging revealed mild cerebral atrophy with mild secondary enlargement of the lateral ventricles.

AuthorToEditor: Dear Members of the Scientific Committee, I submit this case of a 17-year-old patient with severe restricting-type anorexia nervosa complicated by life-threatening multi-organ dysfunction requiring intensive care management. During hospitalization, she developed acute cardiac failure, disseminated intravascular coagulation, refeeding syndrome, central pontine myelinolysis, and superior mesenteric artery syndrome. She was discharged from the ICU in an immobilized state with pressure ulcers but gradually regained mobility with strong family support and structured rehabilitation. Psychiatric treatment with olanzapine 5 mg and fluoxetine 20 mg provided partial benefit, although treatment resistance was noted. This case underscores the severe systemic consequences of anorexia nervosa and the importance of multidisciplinary management in adolescents. Sincerely, Feyza Nur Gümrükçü, MD ORCID:0000-0002-6232-5383

[Abstract:0348]

Pseudologia Fantastica: Clinical and Forensic Challenges

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Introduction: Pseudologia Fantastica(PF) is a psychopathological condition characterized by chronic, elaborate and often dramatic storytelling that is disproportionate to any identifiable external gain. Although lying may occur as part of normal psychological functioning—often associated with shame, guilt or avoidance of conflict—PF represents a more pervasive and maladaptive behavioral pattern. First described in 1891 by the German physician Anton Delbrueck, PF has also been referred to as pathological lying or mythomania. Despite its longstanding recognition in the literature, it is not classified as an independent diagnostic category in the DSM-V.

The narratives produced by individuals with PF are typically complex, colorful and at times fantastical, yet they often contain fragments of reality that make them appear plausible. These stories frequently position the individual in heroic or victimized roles and seem designed to elicit admiration, sympathy or attention. Insight tends to fluctuate between conscious deception and partial belief; however, these narratives do not reach the fixed and unshakeable quality of psychotic delusions. The absence of standardized diagnostic criteria and the difficulty in distinguishing PF from malingering, factitious disorder, personality disorders or psychotic conditions create substantial clinical and forensic challenges.

Case Presentation: A 35-year-old single, unemployed male with a middle school education was referred for forensic psychiatric evaluation and attended the assessment accompanied by family members. Psychiatric examination revealed no abnormalities in thought content or perception, and there was no evidence of psychosis. However, increased speech output and manipulative psychomotor behavior were observed during the interview. The patient provided exaggerated and inconsistent autobiographical accounts, raising doubts about the accuracy of his statements. It was unclear whether these distortions were fully intentional or partially outside his conscious control.
Collateral information obtained from family members indicated a longstanding pattern of persistent lying, unrealistic and exaggerated narratives, and dramatic behavior. During the evaluation process, when confronted with contradictory evidence, the patient withdrew some of his claims but continued to defend the central narrative by modifying minor details. Notably, some of the individuals, places, and timelines he mentioned were real; however, fictional elements were constructed around these factual components, reinforcing the impression of pseudologia fantastica.

Following a comprehensive psychiatric assessment, and in the absence of a psychotic disorder or other major mental illness that would impair reality testing, the patient was determined to have full criminal responsibility. Informed consent was obtained from the patient, and identifying details have been anonymized.

Conclusion: Pseudologia Fantastica represents a complex condition at the intersection of psychiatry and forensic evaluation. Although it involves chronic and elaborate lying, individuals with PF typically retain reality testing and do not demonstrate fixed delusional beliefs. Therefore, in the absence of a major psychiatric disorder, criminal responsibility is generally preserved.

Nevertheless, differentiating pathological lying from delusional processes may be clinically challenging, particularly in forensic contexts where legal outcomes depend heavily on psychiatric interpretation. Greater awareness of PF and its clinical characteristics is essential to improve diagnostic accuracy, support appropriate forensic decision-making, and minimize potential medical, legal, and social consequences associated with misinterpretation of this phenomenon.

Keywords: Pseudologia Fantastica, Forensic Psychiatry, Criminal Responsibility

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[Abstract:0349]

Acute Urinary Retention Following Transition from Oral to Depot Risperidone

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Introduction

Antipsychotic medications, including risperidone, are widely used in the management of schizophrenia and other psychotic disorders. While both oral and long-acting injectable (depot) formulations are generally well tolerated, antipsychotics can rarely cause acute urinary retention (AUR) due to anticholinergic and alpha-adrenergic effects. Recognizing this potential adverse effect is essential for safe pharmacological management, especially when switching formulations.

Case Presentation

A 32-year-old male with schizophrenia, previously stabilized on oral risperidone, was transitioned to depot risperidone due to adherence concerns. Within days of the first injection, he developed difficulty urinating, lower abdominal discomfort, and progressive bladder distension.

Physical examination revealed palpable suprapubic fullness, consistent with glob vesicale. Bladder ultrasound confirmed significant urinary retention. No history of urologic pathology, prostatic hypertrophy, or other medications contributing to retention was identified.

The patient required urgent catheterization, which relieved the retention. Risperidone therapy was re-evaluated, with careful dose adjustment and monitoring, and the patient was successfully switched back to oral formulation without recurrence of urinary retention.

Conclusion

Depot risperidone can rarely induce acute urinary retention, possibly due to its higher plasma peaks and alpha-adrenergic or anticholinergic effects compared with oral formulations. Clinicians should monitor for urinary symptoms, especially in patients with prior antipsychotic exposure or risk factors for retention. Early recognition and intervention, including catheterization and dose adjustment, are crucial to prevent complications such as vesical overdistension, infection, or renal impairment.

This case highlights the importance of vigilance for urinary retention as a rare but significant adverse effect of depot antipsychotics. Transitioning from oral to long-acting injectable risperidone requires careful monitoring for urologic complications. Awareness of this potential side effect can guide timely intervention and optimize patient safety.

Keywords: Long-Acting Injectable Antipsychotics, Acute Urinary Retention, Risperidone

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[Abstract:0350]

Probable Ocular Migraine–Like Visual Phenomena After Escitalopram Dose Escalation: A Case Report

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Introduction: Selective serotonin reuptake inhibitors (SSRIs) are widely prescribed and generally well tolerated; however, visual adverse events have been reported. A published case report has described a temporal association between citalopram and new-onset migraine with visual aura, suggesting that SSRIs may, in selected individuals, trigger aura-like phenomena. In contrast, the available case-report literature provides limited or no specific evidence implicating escitalopram as a trigger for visual aura or retinal migraine. Nevertheless, patient-reported pharmacovigilance data have documented a range of visual complaints—including blurred vision, photopsia, and visual field defects—across multiple agents, including escitalopram.

Retinal (ocular) migraine is a rare and distinct entity characterized by transient monocular visual disturbances, usually followed by migraine headache, and is thought to involve retinal hypoperfusion or vasospasm. Importantly, transient monocular visual loss may also result from vascular or procedure-related causes that can clinically mimic migraine, including amaurosis fugax. Therefore, when new visual symptoms emerge after SSRI initiation or dose escalation, careful differentiation between migraine-related phenomena and vascular etiologies is essential.

Case presentation: A 36-year-old woman with no known medical comorbidities presented to the psychiatric outpatient clinic with anxiety. Escitalopram was prescribed at a target dose of 10 mg/day. During the first week, she used 5 mg/day and reported no adverse effects.

On the day the dose was increased to 10 mg/day, she experienced an acute visual disturbance in her right eye. The episode lasted approximately one hour. During the first 15 minutes, she described blurred vision accompanied by positive visual phenomena resembling flashes of light. This was followed by complete loss of vision in the affected eye. The episode was painless but accompanied by mild nausea. She denied micropsia, macropsia, or prior similar events. A mild headache developed shortly after the visual symptoms resolved.

The patient initially presented to a pharmacy, where blood pressure and blood glucose measurements were within normal limits. She was subsequently evaluated by an ophthalmologist. After ocular examination, recent medication changes were reviewed, and discontinuation of escitalopram was recommended. The ophthalmologist considered the presentation to be compatible with an ocular migraine–like phenomenon.

Following cessation of escitalopram, the patient's visual symptoms resolved completely within approximately 36 hours and did not recur. Escitalopram was subsequently replaced with vortioxetine, which was well tolerated, and no recurrence of visual symptoms was observed during follow-up.

Informed consent was obtained from the patient, and identifying details have been anonymized.

Conclusion: This case describes a transient, painless, monocular visual disturbance temporally associated with escitalopram dose escalation. While SSRI-triggered migraine with aura has been reported with citalopram, case-report evidence specifically linking escitalopram to visual aura or retinal migraine remains limited. However, broader patient-reported data suggest that serotonin reuptake inhibiting antidepressants, including escitalopram, may be associated with visual complaints such as blurred vision, photopsia, and visual field disturbances. This case highlights the importance of awareness of rare but potentially distressing visual adverse effects of SSRIs among psychiatrists, and underscores the need for timely collaboration with ophthalmology when such symptoms emerge.

Keywords: Escitalopram, Visual adverse effect, SSRI-associated visual disturbances

AuthorToEditor: Ezgi Şişman: 0000-0001-9496-991X Mualla Keskinsezer: 0009-0004-8061-1211 Abstract number 0345 refers to the same case; however, it was mistakenly submitted under the category Child Psychiatry – Psychopharmacology (A.09). Therefore, we have re-submitted the abstract under the correct category, Adult Psychiatry – Psychopharmacology (B.09). 0345 numaralı bildiri aynı olguya ait olup kategorisi yanlışlıkla Çocuk Psikiyatrisi- Psikofarmakoloji (A.09) seçilerek sisteme yüklendiğinden bildirimizi tekrar doğru kategoride (Yetişkin psikiyatri Psikofarmakoloji B.09) yükledik.

[Abstract:0355]

Case Report of a Patient with Autism Spectrum Disorder Carrying a MYT1L Variant

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Introduction: MYT1 (myelin transcription factor 1), MYT1L (myelin transcription factor 1-like), and ST18 (suppression of tumorigenicity 18) are members of the MYT1 family of transcription factors. Heterozygous mutations in the MYT1L gene have been associated with intellectual disability, developmental delay, aggressive behavior, autistic features, and obesity. This case report describes a patient who presented to a child and adolescent psychiatry outpatient clinic with attention deficits, hyperactivity, sensory sensitivity to loud noises, and speech impairment, and who was found to carry a heterozygous variant in the MYT1L gene following genetic testing.

Case presentation: An 8-year-7-month-old boy was brought to a child and adolescent psychiatry clinic with complaints of attention deficits, hyperactivity, and sensory sensitivity to loud noises. He was repeating the second grade due to difficulties in acquiring reading and writing skills. His first psychiatric evaluation was conducted at the age of three because of poor eye contact, sensory sensitivity to loud noises, and hyperactivity. He was diagnosed with Autism Spectrum Disorder (ASD) and began receiving special education following the diagnosis. The patient was born via cesarean section at 38 weeks of gestation, weighing 3500 grams. He did not require neonatal intensive care after birth. He began walking at 13 months, spoke his first words at 18 months, and formed two-word sentences at 30 months. Psychiatric evaluation based on DSM-5 criteria, conducted by a child and adolescent psychiatrist, confirmed diagnoses of ASD, Attention Deficit Hyperactivity Disorder (ADHD), and mild intellectual disability. The patient was started on short-acting methylphenidate at a dose of 10 mg/day. Given the presence of obesity, he was referred to the medical genetics department. Genetic testing identified a heterozygous c.89G>A (p.Ser30Asn) variant in the MYT1L gene.

Conclusion: Deletions involving chromosome 2p25.3 are associated with intellectual disability and obesity. The smallest region of overlap defined by the detection of 2p25.3 deletions includes the MYT1L gene. MYT1L plays a critical role in the maturation of neural stem cells as well as in the differentiation and proliferation of oligodendrocyte progenitor cells, processes that are essential for central nervous system myelination. Copy number variants, indels, and single-nucleotide variants affecting MYT1L are associated with an autosomal dominant neurodevelopmental syndrome characterized by obesity, polyphagia, intellectual disability/developmental delay, and behavioral disturbances. The most frequently reported type of variation in MYT1L is microdeletion. De novo MYT1L mutations have been identified in patients with ASD. Genetic analysis of our case revealed a heterozygous c.89G>A (p.Ser30Asn) variant in the MYT1L gene. The clinical presentation was consistent with previous reports, including early-onset obesity, speech delay, intellectual disability, ASD, and ADHD. Treatment with methylphenidate at a dose of 10 mg/day resulted in a reduction in ADHD symptoms and improved school functioning, with no observed side effects. Several case series of individuals with MYT1L variants have been reported in the literature. To our knowledge, no cases have been reported from Turkey. This case report underscores the importance of considering genetic evaluation when clinical features suggestive of MYT1L-related neurodevelopmental disorders are identified in clinical practice.

Keywords: Intellectual disability, autism, obesity, MYT1L

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[Abstract:0358]

Suicide Attempt with Oleander Ingestion in an Adolescent

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Introduction: The prevalence of depression and depressive symptoms among adolescents has increased significantly in recent years. Suicidal behavior is a severe consequence of adolescent depression. Oleander contains cardiotoxic glycosides called cardenolides. Ingestion of oleander seeds can produce a clinical presentation very similar to digoxin poisoning. Symptoms may include vomiting, diarrhea, dizziness, bradycardia, sinus and atrioventricular block, and other cardiac dysrhythmias. Several cases of adult fatalities following intentional oleander ingestion have been reported. This case report describes an adolescent who attempted suicide through oleander ingestion.

Case presentation: A 16-year-9-month-old male adolescent was admitted to the pediatric intensive care unit for arrhythmia following ingestion of oleander leaves. He was subsequently referred to the child and adolescent psychiatry clinic. During the psychiatric assessment in the intensive care unit, it was learned that the patient had been feeling unhappy for about a year. He had become withdrawn, spent most of his time in his room, experienced attention problems that caused his grades to decline, and had difficulty falling asleep. During this consultation, the patient denied ingesting oleander for suicidal intent. He reported that after ingesting three oleander leaves at night and subsequently vomiting, he informed his family that he had been poisoned. The psychiatric assessment revealed depressed affect and mood, with no evidence of perceptual abnormalities or thought content disturbances, and the patient did not report any suicidal ideation or plans. Following a DSM-5–based evaluation by a child and adolescent psychiatrist, he was diagnosed with major depressive disorder and referred to the outpatient clinic. At a follow-up appointment, the patient admitted that he knew oleander leaves were poisonous, had researched the plant's toxic effects online, and had planned a suicide attempt approximately two weeks prior. Treatment with sertraline 50 mg/day was initiated.

Conclusion: Oleander is a toxic plant that, even in small amounts, can cause serious arrhythmias and life-threatening conditions due to its cardiac glycosides. After ingestion, the cardiac glycoside oleandrin is rapidly absorbed from the gastrointestinal tract and distributed to tissues including the heart, liver, and kidneys. It inhibits the Na^+/K^+-ATPase pump, leading to increased intracellular calcium levels and resulting in cardiac arrhythmias. There are case reports of oleander leaves being ingested for suicidal purposes, including leaves consumed after boiling in water. In Sri Lanka, consumption of oleander seeds for suicidal purposes reportedly became widespread after two school-aged girls ingested oleander seeds in 1980. Fatalities resulting from oleander ingestion have also been reported in the literature. In this case, the patient was monitored in the pediatric intensive care unit for gastrointestinal and cardiac symptoms following ingestion of oleander leaves. The patient initially denied a suicide attempt during the psychiatric evaluation in the pediatric intensive care unit, but a follow-up interview revealed that the attempt had been planned. Following a DSM-5–based psychiatric assessment conducted in the intensive care unit, the patient was diagnosed with major depressive disorder and referred for outpatient follow-up. This case demonstrates that adolescents may initially deny suicidal behavior, underscoring the importance of follow-up interviews for accurate clinical evaluation.

Keywords: Suicide, adolescent, oleander, depression

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[Abstract:0364][Kabul:Poster][A. Çocuk Psikiyatri]

Severe Intellectual Disability and Epilepsy Associated with a Homozygous ATP8A2 Splice-Region Variant: A Case Report from a Multiplex Family

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Introduction: Children and adolescents with severe intellectual disability are defined by profound cognitive limitations together with lifelong dependence in daily functioning and a persistent need for intensive support. Under DSM-5 and ICD-11 frameworks, diagnosis is based on deficits in intellectual functioning, impaired adaptive functioning (communication, social participation, self-care), and onset during the developmental period. Severity is determined primarily by adaptive functioning rather than IQ alone (1,2). Individuals with severe disability typically function within an IQ range of approximately 20–35. Language abilities are usually limited to a restricted vocabulary and brief phrases, with difficulty understanding complex language. Academically, the capacity to acquire literacy or basic mathematical skills is markedly constrained or absent. In daily life, these individuals require extensive assistance with dressing, feeding, and hygiene, and generally need continuous supervision because independent living is not achievable (3,4). The prevalence of intellectual disability is ~1%, whereas severe/profound forms occur in 6 per 1,000. Children with persistent developmental delays often require specialized educational settings, and increasing severity is associated with a substantially higher likelihood of identifying a specific genetic or organic etiology (5). In individuals with homozygous ATP8A2 variants, the phenotype is characterized by severe hypotonia, global motor delay, and hyperkinetic movement disorders; most patients do not achieve head control or independent ambulation. The course may also include cognitive impairment, nystagmus, and neuroimaging abnormalities, including cerebellar atrophy on brain MRI (6).

Case Presentation: A 15-year-old male is being followed with a diagnosis of disequilibrium syndrome type 4 (MIM: 615268). He is non-verbal and immobile/non-ambulatory, has no toilet training, and requires diapers. He is unable to tolerate solid foods and is limited to liquid intake; due to growth failure and malnutrition, he receives formula supplementation. Oral feeding is maintained, and his body mass index is -2 SD. He was born at term. Marked early gross motor delay was evident, with failure to achieve head control within the first 6 months of life and absence of unsupported sitting. He is followed by neurology with epilepsy; despite dual antiseizure therapy, approximately one seizure per week is reported. Physical examination reveals pronounced muscle atrophy, absent deep tendon reflexes (areflexia), scoliosis, and ptosis; height and weight are below age-matched peers. Brain MRI demonstrated bilateral posterior periventricular white matter T2/FLAIR hyperintensity. A history of parental consanguinity (first degree cousins) consistent with autosomal recessive inheritance. Genetic evaluation included chromosomal microarray analysis (CMA) from peripheral blood, which did not detect any clinically significant copy-number changes. Following a normal CMA result, whole-exome sequencing (WES) was performed and identified a homozygous ATP8A2 variant, c.1868-3C>G (intronic, near the splice region), classified as likely pathogenic according to ACMG/AMP criteria. Variant confirmation and intrafamilial segregation were conducted by Sanger sequencing: the parents and one unaffected sibling were heterozygous carriers, another unaffected sibling was variant-negative, and two siblings and a cousin with a similar phenotype were homozygous for the variant.

Keywords: Exome Sequencing, Hypotonia, Intellectual Disability

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[Abstract:0369]

Late Recognition of Autism Spectrum Disorder in a 55-Year-Old Man Followed as Schizoaffective Disorder: Diagnostic Pitfalls, Polypharmacy Burden and Suicide Risk

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Introduction: We highlight the clinical value of considering autism spectrum disorder (ASD) in adults followed for years with an atypical schizoaffective diagnosis. In this case, lifelong social-communication difficulties and rigidity were misattributed to negative symptoms, contributing to recurrent impulsive suicide attempts and high medication burden.

Case Presentation: A 55-year-old married man with two children, a university graduate and a retired person presented after a medication-induced impulsive suicide attempt. The attempt occurred amid three months of escalating anxiety, social withdrawal, and affective lability; he had retired two months earlier due to worsening complaints. The presentation favored a mixed affective exacerbation over a primary psychotic relapse, while attention and orientation were preserved. He had been treated for decades under bipolar/schizoaffective formulations and, prior to the attempt, was receiving seven psychotropic agents: clozapine 50 mg, valproate 1000 mg, lamotrigine 200 mg, ziprasidone 40 mg, lorazepam 1 mg, biperidene 1 mg, and propranolol 20 mg. He had four prior impulsive suicide attempts and five psychiatric hospitalizations. He reported a prolonged period without mood swings or psychotic symptoms while treated with clozapine and mood stabilizers; recently he complained of decreased social interaction, ruminative thoughts, and chronic anxiety. His history included adjustment difficulties following a traffic accident in 1989 and a diagnosis of bipolar disorder with manic symptoms. Medical comorbidities included diabetes and hypertension; there was no smoking, alcohol, or substance use.

A detailed developmental history, corroborated by first-degree relatives, revealed a lifelong neurodevelopmental pattern: childhood preference for solitary play and quiet environments, circumscribed interests (e.g., model aeroplanes), persistent avoidance of group interactions, difficulty intuitively understanding social rules and interpreting others' feelings/intentions, marked distress with changes in plans, and "anger/freeze" responses. Screening supported high autistic traits (Autism Spectrum Quotient-10: 9/10). To clarify diagnosis, the Ritvo Autism Diagnostic Scale–Revised (RAADS-R) score was 153/240, supporting ASD. Routine laboratory tests, EEG and ECG were unremarkable, and metabolic comorbidities were optimized during hospitalization. He was discharged in stable condition on clozapine 150 mg and valproate 1000 mg only. Psychotropic burden was reduced from seven agents to two at discharge, illustrating a rationalization opportunity once the diagnostic formulation shifted toward ASD-informed care.

Conclusion: In adults, ASD traits can overlap with social withdrawal and features often labeled as negative symptoms in psychotic disorders, creating diagnostic and pharmacotherapeutic pitfalls. Persistent, early-onset social-communication difficulties, low tolerance for change, and restricted interests supported ASD that was independent of episodic mood/psychotic symptoms in this patient. Missing ASD may drive avoidable antipsychotic polypharmacy, while unaddressed rigidity/anxiety and social maladjustment may contribute to recurrent impulsive self-harming behaviors. Although brief screening scales are not diagnostic, when combined with collateral developmental history they can improve diagnostic awareness. Recognizing co-occurring ASD in later life can rationalize psychotropic regimens and enable targeted interventions (psychoeducation, ASD-informed anxiety management, and suicide prevention planning), improving safety and functional outcomes. Systematic developmental history-taking and ASD-focused assessment should be considered in chronic psychotic-spectrum presentations marked by prominent anxiety, rigidity, and repeated suicidality.

Keywords: Autism Spectrum Disorder, Schizoaffective Disorder, Suicide, Clozapine, Polypharmacy

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[Abstract:0370]

Bright-Light Therapy in a Case of Non-Seasonal Recurrent Resistant Depression in a Geriatric Patient

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Introduction

The aim of this case presentation is to discuss the contribution of bright light therapy to clinical symptoms in a geriatric patient with long-standing depressive symptoms who did not respond adequately to multiple pharmacological treatments, and to emphasize its role as a supportive treatment option in geriatric depression.

Case Presentation

A 64-year-old married woman with three children and an elementary school education was admitted to our clinic due to complaints of anhedonia, anergy, difficulty falling asleep, and loss of appetite that had persisted for approximately two years and increased in recent months, compounded by suicidal thoughts that had emerged over the past fifteen days.

Her history indicates that she has been followed for depressive symptoms for approximately forty years, with the first symptoms beginning in her twenties. She has had three impulsive suicide attempts, has been admitted to a psychiatric ward once before, and has undergone one course of ECT. She has a history of multiple psychiatric treatments, including sertraline, venlafaxine, duloxetine, bupropion, trazodone, quetiapine, and aripiprazole. She stated that she did not benefit significantly from the medications or ECT.

Her medical history includes diabetes, hypertension, asthma, glaucoma, and a history of thyroidectomy and radiotherapy due to thyroid cancer.

During the mental status examination, decreased self-care, reduced speech rate and volume, depressive mood and affect, goal-directed associations, anergy and anhedonia, and passive suicidal thoughts were observed. The patient was admitted to our clinic with a preliminary diagnosis of major depressive disorder. The Hamilton Depression Scale score calculated at admission was 20 points. During her hospitalization, her pre-admission treatments of duloxetine 120 mg and trazodone 100 mg were continued, and lamotrigine 12.5 mg and melatonin 2 mg treatments were initiated. To enhance the treatment, ten sessions of bright light therapy were administered, and individual supportive counseling sessions were conducted. A marked reduction in ruminative thoughts and suicidal thoughts was observed during the hospitalization. The Hamilton Depression Scale score calculated prior to discharge was 7 points. The patient, whose depressive symptoms had improved, was discharged with duloxetine 120 mg, trazodone 100 mg, lamotrigine 25 mg, and melatonin 2 mg treatments.

Conclusion

Late-life depression is clinically challenging due to accompanying medical illnesses, a tendency toward chronicity, and limited response to pharmacological treatments. In the presented case, a long history of depression and persistent symptoms despite multiple medication trials suggest a treatment-resistant picture. Sleep disturbance, anhedonia, and circadian rhythm disorder are prominent in the clinical course. Bright light therapy has been reported to be effective in seasonal and non-seasonal major depression, well tolerated, and particularly beneficial in treatment-resistant cases. Studies in geriatric patients have shown this method to be safe and effective in reducing depressive symptoms. This case demonstrates that bright light therapy may be an effective adjunctive option, particularly in geriatric patients with depression accompanied by sleep disturbance and anhedonia.

Keywords: Anhedonia, bright-light therapy, circadian rhythm, depression, geriatrics, sleep disturbance

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[Abstract:0376]

"Is More Better?": Liver Enzyme Elevation Following Self-Escalated Naltrexone in Gambling Disorder

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Introduction: Gambling Disorder (GD) is characterized by persistent and recurrent problematic gambling behavior that impairs functionality. Opioid antagonists, particularly naltrexone, are frequently preferred in pharmacological treatment for their ability to reduce craving by modulating dopaminergic activity in the reward system. Although naltrexone is generally well-tolerated, dose-dependent hepatotoxicity is a known risk. This case report presents the clinical course and management of a patient with GD who developed elevated liver enzymes following the self-administration of supratherapeutic doses.

Case presentation: A 28-year-old male patient presented to our outpatient clinic with a 2-year history of online casino gambling. Following psychiatric evaluation and motivational interviewing, he was diagnosed with GD, and naltrexone 50 mg/day (1x1) was initiated. Baseline liver function tests (LFTs) were AST: 27 U/L and ALT: 32 U/L. At the first follow-up, the patient reported a partial reduction in gambling but persistent intense craving, particularly after work hours. Consequently, the dose was increased to 100 mg/day (2x1).

The patient presented earlier than the scheduled follow-up, stating his medication had run out. He reported self-increasing the dose to 200 mg/day (2 tablets in the morning, 2 in the evening) as he felt the 100 mg dose was insufficient, noting better craving control at the higher dose. Laboratory tests revealed elevated enzymes with AST: 122 U/L and ALT: 88 U/L. Other lab tests were within the normal range. The potential hepatotoxic effects were explained to the patient, the naltrexone dose was reduced back to 100 mg/day, and N-acetylcysteine (NAC) 1200 mg (2x1) was added for its hepatoprotective and glutamatergic effects. Two weeks later, LFTs showed regression to near-normal levels (AST: 38 U/L, ALT: 41 U/L).

Conclusion: While naltrexone is effective for GD, it carries a potential for hepatocellular injury, particularly at high doses, as warned by the FDA. This case highlights how "treatment non-adherence" or "impulsive dose escalation" in addiction patients can alter the drug's safety profile. In our case, the rapid normalization of enzymes following dose reduction and NAC addition supports drug-induced toxicity. Clinicians must strictly monitor naltrexone dosage and regularly assess liver function, especially in patients with impulse control disorders.

Keywords: Gambling Disorder, Naltrexone, Hepatotoxicity, Case Report, N-acetylcysteine

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[Abstract:0377]

Catatonia as the First Manifestation of Dementia with Lewy Body (DLB): A Case Report

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Introduction: Although catatonia is frequently associated with neuropsychiatric syndromes, there is little evidence for its association with dementia with Lewy bodies (DLB). DLB is a neurodegenerative disorder characterized by cognitive fluctuations, visual hallucinations, and Parkinsonism. In this case report, we will discuss a patient who manifested with catatonia syndrome symptoms such as negativism, mutism, stereotyped behavior, agitation, and grimaces, and was subsequently diagnosed with DLB.

Case presentation: A 75-year-old female patient was brought by her husband in October 2024 because of auditory and visual hallucinations accompanied by sleep disturbances characterized by reduced sleep duration, difficulty maintaining sleep, and dream-enactment behaviors suggestive of REM sleep behavior disorder (RBD), as reported by her husband. Stereotypical behaviors, negativism, mutism, grimace, and agitated behaviors were detected with a detailed examination. Catatonia syndrome was considered, and she was hospitalized to understand the underlying neuropsychiatric syndrome of catatonia. The Bush-Francis Catatonia Rating Scale score was 11. During hospitalization, it was observed that her consciousness changed during the day, and she complained of forgetfulness. It was determined that she first presented to our clinic in November 2023 with visual hallucinations added to depressive symptoms, and olanzapine 10 mg treatment was started in another center at that time. In our follow-up, it was known that olanzapine was discontinued because visual hallucinations did not regress with olanzapine treatment. In February 2024, she was admitted to the psychiatry clinic with depressive symptoms, withdrawal, visual hallucinations, and decreased sleep.

A decrease in depressive symptoms was observed with sertraline and mirtazapine treatment. Benzodiazepine treatment (lorazepam 2.5 mg twice a day) was initiated for the diagnosis of catatonia syndrome immediately after admission to the clinic—catatonic symptoms resolved with this treatment. Following treatment, the Bush-Francis Catatonia Rating Scale score decreased from 11 to 3, indicating significant clinical improvement. Since the patient had an altered state of consciousness during the day, executive dysfunction, episodic memory impairment, bilateral minimal bradykinesia, right and left-hand bradykinesia, postural instability, and short-stepping gait, dementia with DLB, Parkinsonism, and limbic encephalitis were considered as a preliminary diagnosis for neuropsychiatric syndromes causing catatonia. A neurologist consulted her. When visual hallucinations, episodic memory impairment, deterioration in cognitive functions, deterioration with antipsychotics, Parkinsonism, and neuroimaging findings were evaluated, DLB diagnosis was considered in the foreground. Memantine was added to the treatment by the neurologist. Written informed consent for publication of this case report was obtained from the patient's legal guardian.

Conclusion: Fluctuating cognitive impairment, persistent visual hallucinations, Parkinsonism, and neuroleptic sensitivity characterize DLB. Diagnosis is often difficult because of the diversity of symptoms. Acetylcholinesterase inhibitors and memantine are used to treat DLB. In the present case, the patient was hospitalized for diagnosis and treatment with symptoms of marked impairment in functionality, episodic memory impairment, visual hallucinations, Parkinsonism, negativism, stereotyped behaviors, agitation, mutism, and grimace. The clinical syndrome causing catatonia was found to be DLB. Catatonia in DLB may result from neurotransmitter imbalances, particularly in the GABA and dopamine systems. This case highlights that catatonia may represent an atypical presentation of DLB.

Keywords: Lewy Body Dementia, Catatonia Syndrome, visual hallucinations, parkinsonism

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[Abstract:0378]

Dissociative Amnesia in the Context of Prolonged Grief Disorder: A Case Report

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Introduction: Dissociative amnesia is characterized by retrieval blockade of episodic autobiographical memories arising in the context of psychological trauma (whether known or not), with no evidence of brain damage on structural imaging.Prolonged Grief Disorder is characterized by persistent and impairing grief responses lasting at least 12 months after the death of a close person. In the present case, persistent generalized retrograde amnesia emerged in the context of prolonged grief following an acute interpersonal threat.

Case presentation: An 18-year-old male living with his parents and older sister presented with sudden loss of autobiographical memory following an acute interpersonal conflict. He had no prior neurological history and was preparing for university entrance examinations.

Five days before admission, he encountered his father while riding a scooter. His father reacted angrily, shouted at him for speeding, picked up a stone, and threatened to damage the scooter. Shortly afterward, the patient developed marked autonomic arousal, including profuse sweating and agitation, followed by collapse. During transportation to the emergency department, he became unresponsive and remained minimally responsive for several hours, reportedly opening his eyes only after painful stimulation. Upon regaining consciousness, he appeared confused, failed to recognize family members, repeatedly asked where he was, and described the experience as if "life had restarted."

Neurological evaluation was unremarkable. Non-contrast cranial CT, diffusion-weighted MRI, follow-up structural MRI, and both wake and sleep EEG recordings revealed no abnormalities. There was no evidence of traumatic brain injury, seizure disorder, intoxication, metabolic disturbance, or other medical condition. The absence of focal neurological deficits, postictal confusion, or global cognitive impairment reduced the likelihood of an organic etiology. Given the acute emotional trigger and normal neurodiagnostic findings, the collapse and unresponsiveness were considered consistent with a functional (conversion/dissociative) presentation.

Following the episode, the patient demonstrated generalized retrograde autobiographical amnesia. Initially, he did not recognize close relatives and was unable to recall his home address. One week later, he became disoriented outside and required assistance to return home. Anterograde memory remained intact; he was able to encode and retain new information. However, at four-month follow-up, significant retrograde autobiographical amnesia persists.

Psychiatric assessment revealed that one year prior, he had lost his grandfather, to whom he was strongly attached. He had participated directly in burial rituals and subsequently exhibited persistent yearning, avoidance of reminders, emotional distress when the topic was raised, and functional impairment for several months, consistent with prolonged grief disorder. He remains unable to recall the grandfather's death.

There was no evidence of psychotic disorder, mood disorder with psychotic features, substance use disorder, or malingering.

Conclusion: In this adolescent presenting with persistent retrograde amnesia following an acute paternal confrontation, extensive neurological assessment did not reveal an organic or substance-related cause. Psychotic disorders, mood disorders, anxiety-related conditions, and malingering were considered in the differential diagnosis, although ongoing affective symptoms warrant continued evaluation. Preserved anterograde memory and the clinically meaningful context of prolonged grief supported a dissociative formulation. This case highlights the importance of cautious, longitudinal assessment in unexplained autobiographical memory loss.

Keywords: Prolonged grief, dissociative amnesia, functional

[Abstract:0379]

Five-Session Written Exposure Therapy in a Young Woman with PTSD, Borderline Personality Disorder, and Bulimia Nervosa: Feasibility and Short-Term Clinical Outcomes

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Introduction: Posttraumatic stress disorder (PTSD) following childhood sexual abuse is often complicated by emotion dysregulation, non-suicidal self-injury (NSSI), and eating disorder symptoms. Borderline personality disorder (BPD) may further impede engagement in trauma-focused treatments through intense affect and avoidance. Written Exposure Therapy (WET) is a brief, five-session exposure-based intervention that may be pragmatic when dropout risk is high. We report the feasibility and short-term outcomes of WET in a young woman with PTSD, comorbid BPD, and bulimia nervosa.

Case Presentation: A 21-year-old woman presented with marked impulsivity, rapid mood shifts, recurrent NSSI (wrist cutting; pouring hot wax on her body), and binge–purge behavior. She described rapid transitions between intense happiness, emptiness, and suicidal thoughts. For the preceding 6–7 months, she had been vomiting 4–5 times daily, accompanied by body image distortion and fear of weight gain. PTSD symptoms included recurrent intrusive memories and nightmares, insomnia, intense avoidance of trauma reminders, guilt and worthlessness, hypervigilance, and exaggerated startle response. Trauma-related cues resembling the perpetrator triggered intense autonomic arousal, an urge to flee, and subsequent NSSI urges. History revealed childhood familial neglect and sexual abuse by a relative at age 11. The patient was followed with diagnoses of PTSD, BPD, and bulimia nervosa. Baseline clinical monitoring included PTSD severity (PCL-5/CAPS-5), emotion dysregulation (DERS), and daily/weekly binge–purge frequency. Pharmacotherapy was initiated with fluoxetine 40 mg/day, aripiprazole 10 mg/day, and topiramate 25 mg/day (planned titration) targeting impulsive eating and vomiting. Given prominent avoidance and functional impairment during hospitalization, trauma-focused psychotherapy was commenced, and WET was administered. WET was delivered once weekly for five sessions (30–45 minutes each), with limited therapist intervention and no between-session homework. These features provide a significant advantage for patients who have difficulty regulating their emotions and who struggle to continue with long-term therapy. In each session, the patient completed structured written exposure to the same index trauma, focusing on thoughts, feelings, and bodily reactions during the event. Intense anxiety and avoidance were prominent in early sessions but attenuated over time; later sessions allowed more comprehensive processing of trauma memories. During a two-month follow-up, trauma-related nightmares decreased markedly, avoidance behaviors subsided, and trigger-related anxiety became more tolerable. No binge–purge episodes or NSSI were observed during follow-up, and the patient reported improved tolerance of previously avoided trauma cues and re-engagement in interpersonal contexts without escalation to self-harm.

Conclusion: Studies indicate that WET is effective in PTSD treatment. This case supports the feasibility of WET in a patient with complex comorbidities and a high risk of disengagement. Given minimal between-session demands, WET may be particularly suitable for inpatient or early post-discharge settings where adherence is fragile. The brief, structured format may reduce treatment burden and avoidance while enabling exposure-based processing, with potential downstream benefits on NSSI and bulimic behaviors when these are functionally linked to trauma-related affect dysregulation. Interpretation is limited by concurrent pharmacotherapy, short follow-up, and the absence of reported numeric pre/post symptom scores; nevertheless, the observed reduction in avoidance and trauma-cue reactivity suggests clinically meaningful gains.

Keywords: Posttraumatic Stress Disorder, Borderline personality disorder, written exposure therapy, sexual trauma, psychotherapy

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[Abstract:0380]

Epilepsy-Related Encephalopathy in an Advanced Age Bipolar Disorder Case

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Introduction: Epilepsy-related encephalopathy refers to clinical presentations in which epileptiform activity itself causes cognitive, behavioral, and psychiatric impairment that cannot be explained by a structural lesion. It is mostly characterized by fluctuating changes in consciousness, behavioral disorganization, and atypical neuropsychiatric symptoms. The combination of cognitive fluctuations, psychiatric symptoms, and epileptic phenomena occurring in older age creates diagnostic difficulties. This case presents an atypical neuropsychiatric picture requiring multidisciplinary evaluation.

Case Presentation: A 65-year-old male patient has been followed up for 13 years with a diagnosis of bipolar disorder and has a history of multiple psychiatric hospitalizations. Over the past month, he has developed confusion, disorientation, visual hallucinations, urinary incontinence, and disorganized behavior, which become more pronounced in the morning. These behaviors are accompanied by depressed mood and anhedonia complaints. Approximately 25 days ago, he was admitted to the neurology ward with a preliminary diagnosis of SVO. After no acute pathology was detected, he was referred to the psychiatry clinic. He was admitted to the psychiatry ward with preliminary diagnoses of catatonia, encephalopathy, and Lewy body dementia. During the interictal period, the mental status examination revealed full orientation, decreased speech rate and volume, subdepressive mood, and natural thought content. Anhedonia and anhedonia were observed, and insight was preserved.

The patient's laboratory findings were normal, with a history of hypertension and diabetes. Neurology evaluated Parkinson's plus syndromes, epilepsy, and encephalitis in the differential diagnosis. The awake EEG showed intermittent 5–6 Hz theta activity and slow wave paroxysms. CSF examination was within normal limits, and the autoimmune and paraneoplastic panel was negative. FDG-PET revealed bilateral frontal and thalamic hypometabolism; a pattern typical of dementia syndromes was not observed.

During follow-up, two episodes of confusion, visual hallucinations, and incontinence attacks were observed; these episodes were considered epileptic seizures. Levetiracetam was initiated, and zonisamide was added when seizures persisted. No seizures were observed after combined treatment. Based on the clinical response, a diagnosis of epilepsy-related encephalopathy was considered.

Discussion: This case presents a complex clinical picture involving epileptic phenomena, signs of parkinsonism, frontal lobe dysfunction accompanied by confusional states, and psychiatric symptoms. The absence of a typical dementia pattern on FDG-PET, but the presence of frontal and thalamic hypometabolism, suggests epilepsy-related network dysfunction or a prodromal neurodegenerative process.

The long-term cognitive effects of bipolar disorder may have contributed to the picture. Valproic acid, used for the treatment of bipolar disorder, may have masked the epileptic picture. The clinical picture's response to antiepileptic treatment is consistent with encephalopathy developing due to epileptic network dysfunction.

Conclusion: Confusional and psychotic symptoms that emerge in older patients with a long history of bipolar disorder should not be evaluated solely as a psychiatric relapse. Differential diagnosis should consider epilepsy-related psychiatric symptoms, cognitive and behavioral impairment due to epilepsy-related encephalopathy, atypical delirium, and neurocognitive vulnerability accompanying bipolar disorder. Epileptic processes must always be considered in the differential diagnosis of changes in consciousness and behavior that occur in advanced age.

Keywords: Epilepsy-Related Encephalopathy, Bipolar Disorder, Late-Onset Neuropsychiatric Symptoms, Epileptic Network Dysfunction, Frontal and Thalamic Hypometabolism

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[Abstract:0381]

The Healing Power of Being Seen: A Case Report Illustrating the Psychiatric Implications of Family Disclosure in Gender Dysphoria

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Introduction: Gender dysphoria is defined as a marked incongruence between an individual's experienced or expressed gender identity and their sex assigned at birth. The literature consistently indicates that adolescents experiencing gender dysphoria are at increased risk for depression, anxiety, and self-harming behaviors. However, social support—particularly family acceptance—has been identified as a crucial protective factor that significantly reduces these risks. This case report discusses the clinical course of a transgender male adolescent whose self-harming behaviors completely resolved following disclosure of his gender identity to his family.

Case Presentation: The patient, a 20-year-old transgender man assigned female at birth, reported that he first became aware of his attraction to girls during primary school and described a preference for playing games typically associated with boys, as well as choosing toys such as toy guns and cars. During adolescence, following menarche, he developed intense feelings of disgust toward his body and began to identify himself as male. At the age of 17, he was admitted to a child and adolescent psychiatry outpatient clinic due to depressive symptoms and self-harming behaviors, including cutting his arms with razor blades. He was diagnosed with major depressive disorder and generalized anxiety disorder. During follow-up, due to escalation of self-harm behaviors—such as burning himself with cigarettes and attempting self-strangulation with a cable—he required inpatient psychiatric treatment.

During hospitalization, he disclosed his history of gender dysphoria to his clinician for the first time and decided to come out to his family after discharge. He initially shared his gender identity with his mother, whose accepting and supportive response was followed by the complete cessation of self-harming behaviors. Marked improvement in psychological symptoms was observed after his mother accompanied him in purchasing male clothing, supported him in cutting his hair short, and began addressing him using a name chosen by the family.

Conclusion: This case highlights the protective role of family support against self-harming behaviors in a transgender male adolescent. Previous studies have reported lifetime prevalence rates of self-harm among transgender youth ranging between 40% and 60%. However, family acceptance, social support, and affirmation of gender identity have been shown to significantly reduce this risk.

Whittle et al. (2024) reported that transgender youth exhibit higher rates of depression and suicidal ideation compared to their cisgender peers; however, these disparities diminish substantially in supportive environments. Similarly, Bird et al. (2024) demonstrated that family and peer support, as well as affirming behaviors such as the use of identity-congruent clothing and chosen names, exert a strong protective effect on mental health outcomes. Family acceptance and social support play a critical role in the reduction of psychiatric symptoms and self-harming behaviors among adolescents experiencing gender dysphoria. In this context, obtaining a comprehensive psychosocial history during the differential diagnostic process-particularly in adolescents presenting with depression, anxiety, or self-harm-is essential, with careful consideration of gender dysphoria and sexual orientation diversity. Mental health professionals who adopt an informative, supportive, and family-inclusive approach in gender identity–related processes may significantly enhance treatment response and overall quality of life.

Keywords: Gender Dysphoria, Self-Harm, Family Support, Transgender Male

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[Abstract:0386]

Olfactory Reference Disorder Emerging After COVID-19 Related Anosmia: A Case Report

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Introduction: Olfactory Reference Disorder (ORD), also known as Olfactory Reference Syndrome (ORS), is characterized by a persistent preoccupation with the belief of emitting an offensive body odor that is not perceived, or only minimally perceived, by others and results in significant distress, repetitive behaviors, and functional impairment. In ICD-11, ORD is classified among Obsessive–Compulsive and Related Disorders, while DSM-5 conceptualizes it under Other Specified Obsessive–Compulsive and Related Disorders. Although olfactory disturbances became common during the COVID-19 pandemic, the development of persistent odor-related psychopathology following transient smell loss remains insufficiently described.

Case presentation: A 36 year old male forklift operator was referred for psychiatric evaluation after repeated medical consultations due to a persistent belief that he emitted a foul body odor. He lived alone and had no significant medical comorbidity or substance use. During acute COVID-19 infection early in the pandemic, he experienced a period consistent with near-complete anosmia. During this time, he began to believe that he emitted an unpleasant odor, initially from his feet and later from his entire body.

Over the following years, his general olfactory function gradually recovered; however, the belief that he emitted an offensive odor persisted and progressively intensified. He began to interpret ordinary reactions from others, such as opening windows or covering their noses as signs that he smelled bad. Over time, he increased his washing behaviors, changed clothes frequently, and started avoiding enclosed environments, especially public transportation. As these concerns intensified, they led to increasing social withdrawal and eventually interfered with his ability to work.

Following partial recovery of his sense of smell, he sought help from several non-psychiatric services. He first presented to internal medicine and dermatology and was later referred to neurology, otorhinolaryngology, and gastroenterology. Despite repeated examinations and laboratory investigations, no medical explanation for his complaints was identified. Because his symptoms continued and his functioning declined, he was ultimately referred for psychiatric evaluation. On mental status examination, he appeared anxious and preoccupied with the belief that he emitted a foul odor. This belief was maintained with delusional intensity and very limited insight. No additional psychotic symptoms were observed. Pharmacological treatment was recommended and prescribed; however, the patient rejected psychiatric treatment, insisting that his condition was not psychological, and continued to seek care from non-psychiatric specialties. Differential diagnosis included schizophrenia spectrum disorders and obsessive–compulsive disorder with absent insight; nevertheless, the monosymptomatic odor-focused presentation and lack of broader psychotic features supported a diagnosis of ORD.

Conclusion: This case illustrates the emergence of persistent olfactory reference symptoms following COVID-19 related anosmia despite subsequent recovery of olfactory function. The dissociation between sensory recovery and worsening psychopathology suggests that maladaptive cognitive interpretations of transient sensory disruption may contribute to the development of ORD. Early recognition of this presentation may help prevent unnecessary medical investigations, delays in appropriate treatment, and severe functional deterioration. Written informed consent was obtained from the patient for publication.

Keywords: Olfactory Reference Disorder, COVID-19, Anosmia

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[Abstract:0397]

Hyperacusis After Escitalopram Treatment: A Case Report

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Introduction: Hyperacusis (reduced sound tolerance) is a condition that can occur often in association with tinnitus/migraine/anxiety, but can also occur without peripheral ear pathology. Mechanisms such as increased central auditory "gain" and decreased inhibition have been proposed as causes of hyperacusis. Tinnitus has been reported among otological side effects associated with selective serotonin reuptake inhibitors (SSRIs), while hyperacusis is a less frequently reported complaint. This case report describes hyperacusis that developed after starting escitalopram and rapidly regressed upon discontinuation.

Case presentation: A 52-year-old, married, retired teacher with two children presented to the psychiatry outpatient clinic for the first time with insomnia, loss of appetite, malaise, restlessness, and difficulty concentrating for the past 3 months (increased in recent weeks due to economic stressors). Routine tests including TSH, B12, and folate, and the requested brain MRI were normal. The patient scored 20 on the Hamilton Depression Scale (HAM-D) and 22 on the Hamilton Anxiety Scale (HAM-A), which is considered consistent with moderate severity. The patient was monitored for a moderate depressive episode. Sertraline was started at 25 mg/day and titrated to 100 mg/day; at 2 months, the HAM-D decreased to 15, indicating a 60–70% benefit. Due to upper extremity rashes, sertraline was discontinued within 2 weeks on the recommendation of dermatology. Restlessness developed during dose reduction, so escitalopram 5 mg/day was started, increased to 10 mg/day after one week. At a one-month follow-up, the patient described hyperacusis that had been noticeable for about a week; there was no accompanying infection/trauma, headache, vertigo, or tinnitus. ENT evaluation showed normal otoscopy, pure tone audiometry, and acoustic reflexes; no primary ear pathology was detected. (ASHA sources emphasize the central role of detailed history and audiological examination in the evaluation of hyperacusis.) Organic pathology was ruled out during neurology consultation. Escitalopram was gradually discontinued; hyperacusis significantly decreased/regressed after 1 week. Since sleep disturbance was the primary issue, mirtazapine 7.5–15 mg/day was initiated and titrated to 45 mg/day; the patient was followed in remission at 3 months with a good response and no side effects. Mirtazapine's α2 antagonism and 5-HT2/5-HT3 blockade, along with its NaSSA profile and significant H1 antagonism, are highlighted in the literature as having a sedative effect. Verbal and written consent was obtained from the patient for the presentation of this case.

Conclusion: In this case, the temporal relationship between escitalopram treatment and hyperacusis was evaluated as a drug-related side effect due to the exclusion of ENT/neurological etiologies and the rapid improvement upon discontinuation of the drug. When initiating or changing SSRIs, it is important to inquire about and conduct an interdisciplinary evaluation for complaints of auditory hypersensitivity, although these are rare.

Keywords: Hyperacusis, escitalopram, SSRI, drug side effect, depression, mirtazapine

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[Abstract:0398]

Brexpiprazole-Induced Recurrent Hiccups During Antidepressant Augmentation: A Case Report

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Introduction:
Hiccups are repetitive, involuntary contractions of the diaphragm and inspiratory muscles followed by abrupt glottic closure. While usually transient and benign, persistent hiccups may reflect neurological, metabolic, structural, or medication-related causes. The reflex arc includes vagal and phrenic afferents, brainstem integration, and diaphragmatic efferent output. Dopaminergic and serotonergic systems are believed to modulate this circuitry. Brexpiprazole, a serotonin–dopamine modulator with partial agonist activity at D2 and 5-HT1A receptors and antagonism at 5-HT2A receptors, is approved for major depressive disorder augmentation and schizophrenia. Although generally well tolerated, rare adverse effects may occur. We report a case of brexpiprazole-associated hiccups in a patient with anxiety-prominent depression.

Case presentation:

A 21-year-old female presented with marked anxiety, irritability within a depressive mood state, high internal tension, affective reactivity, and persistent insomnia. She described continuous inner restlessness, heightened emotional sensitivity to minor stressors, difficulty maintaining sleep, and fluctuating dysphoria. There was no history of psychosis, neurological illness, substance use, or prior episodes of persistent hiccups. Her previous psychiatric treatment history included only fluoxetine, which had been discontinued due to insufficient response. Sertraline was initiated and titrated to 100 mg/day. Although partial improvement was observed, ruminative thoughts remained prominent. To address persistent symptoms, brexpiprazole 0.5 mg/day was added as augmentation therapy. Approximately 2–3 hours after the first dose of brexpiprazole, the patient developed continuous hiccups lasting nearly 18 hours. The following day, she did not take brexpiprazole and no hiccups occurred. Three to four days later, brexpiprazole 0.5 mg/day was reintroduced, and hiccups recurred again within a few hours, lasting approximately 18 hours. The medication was subsequently discontinued, after which hiccups did not recur. Physical examination was unremarkable. Laboratory investigations, including complete blood count, liver and renal function tests, thyroid function tests, and serum electrolytes were within normal limits. No metabolic, neurological, or structural cause explaining the hiccups was identified. Following discontinuation of brexpiprazole, trifluperazine 1 mg/day was initiated as an alternative augmentation strategy, leading to clinical remission without recurrence of hiccups. Written informed consent was obtained from the patient for publication.

Conclusion:

This case suggests that brexpiprazole, although generally associated with a favorable tolerability profile, may rarely induce hiccups. The clear temporal relationship and positive rechallenge support a probable drug-related effect. Brexpiprazole's partial agonist activity at D2 receptors may alter dopaminergic tone within brainstem circuits regulating diaphragmatic reflexes, while serotonergic modulation through 5-HT1A agonism and 5-HT2A antagonism may further influence phrenic nerve excitability. Clinicians should consider medication-related etiologies when new-onset hiccups occur following initiation of serotonin-dopamine modulators. In the absence of structural or metabolic risk factors, symptoms may be transient and self-limited; however, careful monitoring is warranted. Further research is needed to clarify underlying neurobiological mechanisms and potential vulnerability factors associated with brexpiprazole-induced hiccups.

Keywords: Brexpiprazole, Hiccups, Augmentation Therapy

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[Abstract:0400]

Misdiagnosed Parkinsonism and Dementia: A Case of Bipolar Depression Presenting with Catatonia

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Introduction: Catatonia is a neuropsychiatric syndrome characterized by motor and behavioral abnormalities, including stupor, mutism, negativism, posturing, and psychomotor retardation. Due to overlapping clinical features, catatonia may mimic Parkinsonism or early-stage dementia, leading to frequent misdiagnosis, particularly in older patients. Delayed recognition can result in inappropriate treatments, unnecessary pharmacological exposure, and significant functional deterioration. Catatonia's most commonly associated with mood disorders, especially bipolar disorder, and is highly treatable when promptly identified.[1–3] We report a case initially misdiagnosed as Parkinson's disease and dementia, later correctly diagnosed as bipolar depression with catatonic features, with full recovery following appropriate treatment.

Case presentation: A 59-year-old woman with a 40-year history of bipolar affective disorder was admitted to the psychiatry clinic. She had remained in long-term remission on lithium, which was discontinued two years earlier due to lithium intoxication and replaced with valproic acid.

Five months before admission, after sustaining a wrist fracture, the patient became distressed, tearful, and anxious about recovery. Four months before admission, she experienced dizziness, shuffling gait, imbalance, and recurrent falls, and was diagnosed with drug-induced parkinsonism at an external neurology clinic. Levodopa/benserazide was initiated without benefit. One month later, her condition worsened, with increasing rigidity, marked psychomotor slowing, gait impairment, and progressive memory deficits, leading to a second neurological evaluation and a diagnosis of early-stage amnestic dementia.

In April 2025, she was admitted to psychiatry due to refusal of food and fluids, urinary incontinence, hallucinations, disorientation, and inability to recognize familiar individuals. Mental status examination revealed tearfulness, prolonged response latency, negativism, echolalia, posturing, and impaired orientation and memory, consistent with catatonia.

Despite regular use, duloxetine, bupropion, lamotrigine, and valproic acid provided no clinical benefit and were discontinued. Clonazepam was initiated with partial response, but significant catatonic symptoms persisted. Electroconvulsive therapy (ECT) was therefore started, seven sessions were completed.

Following ECT, the patient showed rapid and marked improvement. Gait disturbances resolved completely, cognitive functions normalized, and full orientation was restored. Dopaminergic treatment was discontinued after neurological reassessment, with no subsequent deterioration.

Conclusion: This case highlights that catatonia can closely mimic both parkinsonism and dementia, creating a high risk of misdiagnosis and unnecessary treatment. In patients with mood disorders who present with prominent motor, cognitive, and functional impairments, catatonia should remain a central diagnostic consideration. Failure to recognize this condition may delay effective therapy and prolong patient suffering. Careful clinical assessment and diagnostic vigilance are essential, particularly when symptoms fail to respond to dopaminergic or neurodegenerative treatment strategies. Early identification of catatonia and prompt initiation of evidence-based interventions—especially benzodiazepines and electroconvulsive therapy—can result in dramatic clinical improvement and complete functional recovery. As demonstrated in this case, timely and accurate diagnosis may reverse severe neuropsychiatric presentations that otherwise resemble irreversible neurodegenerative disease.

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Keywords: Bipolar disorder, Catatonia, Electroconvulsive therapy

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Award-Winning Poster Presentations

[Abstract:0133]

Comparative Risk of Incident Irritable Bowel Syndrome in PTSD Patients Treated with SSRIs vs. SNRIs: A Real-World TriNetX Cohort Study

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Background and Objective:

Post-Traumatic Stress Disorder (PTSD) is increasingly recognized as a disorder with systemic physiological consequences, involving profound dysregulation of the gut-brain axis. Patients with PTSD exhibit a high prevalence of functional gastrointestinal disorders, particularly Irritable Bowel Syndrome (IBS), driven by autonomic imbalance and altered serotonergic signaling. Pharmacotherapy remains a cornerstone of PTSD management, with Selective Serotonin Reuptake Inhibitors (SSRIs) and Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs) serving as first-line interventions. SRIs may enhance motility via 5-HT3 and 5-HT4 pathways, though effects vary. Conversely, SNRIs increase noradrenergic signaling, where presynaptic α2-adrenergic activation reduces enteric acetylcholine release, potentially predisposing patients to constipation. Nevertheless, real-world evidence comparing the risk of incident IBS between these two drug classes in PTSD populations remains limited.

This study aimed to compare the incidence of newly diagnosed IBS in adult patients with PTSD who initiated treatment with SSRIs (Fluoxetine, Sertraline, Paroxetine, Citalopram, Escitalopram, Fluvoxamine) versus those initiating SNRIs (Venlafaxine, Duloxetine, Desvenlafaxine).

Methods:

We conducted a retrospective cohort study using the TriNetX global health research network, analyzing de-identified electronic health records from over 150 million patients. The study population included adults (age≧18) with diagnosis of PTSD. To define a cohort with stable medication exposure, patients were required to have at least three documented instances of the respective index medication prescription. We strictly excluded patients with schizophrenia spectrum disorders, delusional disorders, and dementia to minimize confounding by indication or other neuropathologies. Propensity Score Matching was utilized to balance cohorts on age, sex, race, and key comorbidities including Generalized Anxiety Disorder, Major Depressive Disorder, chronic pain, fibromyalgia, and antipsychotic use. The primary outcome was the risk of incident IBS, analyzed using Kaplan-Meier survival curves and Cox proportional hazards models.

Results:

After 1:1 propensity score matching (n=38,428 per group), the SNRI cohort demonstrated significantly lower 1-year IBS-free survival (98.16%) compared to the SSRI cohort (98.67%). With 594 incident cases versus 439. The Cox proportional hazards model yielded a Hazard Ratio of 1.39 (95% CI: 1.23–1.57), indicating that stable SNRI use was associated with a 38.8% higher risk of subsequent IBS diagnosis compared to stable SSRI use.

Conclusions:

This study provides evidence that in patients with PTSD, treatment with SNRIs is associated with a significantly elevated risk of developing IBS compared to SSRIs. This increased risk may be attributable to the noradrenergic inhibition of gastrointestinal motility inherent to SNRIs, or the gastrointestinal sequelae of discontinuation syndromes often seen with venlafaxine. Clinicians should be vigilant regarding the potential for SNRIs to induce de novo gastrointestinal dysfunction, particularly in PTSD patients with pre-existing somatic vulnerabilities.

Keywords: Post-Traumatic Stress Disorder, Irritable Bowel Syndrome, SSRIs, SNRI

[Abstract:0183]

Pediatric Schizoaffective Disorder: A Case Report

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Introduction: Schizoaffective Disorder (SAD) is a complex diagnosis characterized by concurrent psychotic and significant mood symptoms. It is notably rare and diagnostically challenging in children and adolescents, as presentation can disrupt developmental and academic functioning. This report details the longitudinal case of a pediatric patient exhibiting chronic psychotic symptoms, behavioral dysregulation, and a history of treatment resistance.

Case presentation: A 15-year-old girl was admitted in November 2023 due to prominent behavioral changes, perceptual disturbances, marked social withdrawal, severe mood swings, significant impairment in daily functioning, and repetitive motor behaviors such as jumping and spinning. Mental status examination revealed affective lability, irritability, borderline adaptive capacity, disorganized speech and behavior, disorientation, and clear psychotic symptoms. The mood-related symptoms were observed to have a devastating impact on her overall level of functioning. According to her family, she had no history of substance use.

Symptoms first emerged during the 2020 pandemic. Before then, she had no psychiatric history, an unremarkable birth, age-appropriate developmental milestones, good academic performance, and timely literacy acquisition. After onset, she progressively developed marked social withdrawal, self-talk, reduced sleep, running away from home, excessive masturbatory behavior, poor self-care, and significant behavioral problems. According to her family, symptoms began shortly after exposure to frightening videos with sexual content.

To exclude organic reasons, she underwent thorough evaluation by pediatric neurology, genetics, and metabolic disease specialists; no abnormalities were detected. In various external centers prior to this admission, she had received multiple psychiatric medication—including methylphenidate, risperidone, atomoxetine, fluoxetine, aripiprazole, lorazepam, quetiapine, and olanzapine—under preliminary diagnoses of attention-deficit/hyperactivity disorder (ADHD), autism spectrum disorder (ASD), schizophrenia, and bipolar disorder at different points in time. Despite these interventions, no significant or sustained clinical improvement was achieved.

During follow-up, Risperidone dose was increased(up to 6 mg/day),and Aripiprazole was added due to hyperprolactinemia. Following an insufficient response,a transition to Clozapine was planned,and patient was referred to an inpatient unit on April 2024.During her 54-day hospitalization, her diagnosis was evaluated as SAD (bipolar type). Clozapine was initiated on July 2024 and she was discharged on a medical regimen of Clozapine 300 mg/day, Valproate (Depakote)500 mg/day,and Quetiapine 200 mg/day.

In outpatient follow-ups,although disorganized speech persisted,a decrease in jumping and aimless wandering was observed.She is able to perform simple household chores,though clinical fluctuations continue periodically. During the examination on May 2025 due to an inadequate response regarding irritability, Quetiapine was discontinued,and Lurasidone was cross-titrated from 20 mg/day to 40 mg/day within one month.During the clinical follow-up,improvements such as increased speech intelligibility,a reduction in repetitive expressions,and more meaningful responses to questions were observed. Subsequently,Valproate treatment was discontinued due to hair loss and weight gain. Written informed consent was obtained from the patient's family.

Conclusion: This case highlights the diagnostic complexity and treatment resistance of pediatric SAD, with initial misdiagnoses and multiple failed trials. Psychotic symptoms, worsening since childhood and resistant to several antipsychotics, co-occurred with prominent affective features. The clozapine-lurasidone combination yielded partial symptom relief. Given limited literature on childhood SAD, early recognition, close monitoring, and tailored treatment are essential for improving prognosis.

Keywords: Pediatric SAD, psychotic disorder, lurasidone, clozapine

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[Abstract:0257]

Attitudes Toward Regular Oral Medication Use and Medication Adherence in a Community Sample

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Objective

Regular oral medication use constitutes a cornerstone in the effective management of chronic diseases; nevertheless, medication nonadherence remains a persistent and clinically significant obstacle to optimal therapeutic outcomes. Forgetting to take medication represents one of the most frequently reported manifestations of nonadherence. The present study aimed to evaluate attitudes toward regular oral medication use and levels of medication adherence among individuals using chronic oral medications in the community, and to investigate age-related differences in these variables.

Methods

This cross-sectional study comprised 340 participants who reported regular use of chronic oral medications and completed an online survey. Individuals with chronic psychiatric disorders other than depressive and anxiety disorders were excluded. Sociodemographic characteristics, attitudes toward medication use, and medication adherence behaviors were systematically assessed. The sample was stratified into two age groups: younger (<40 years) and older (40 years and above) participants. Intergroup comparisons and correlation analyses were performed using appropriate statistical procedures.

Results

The mean age of participants was 43.33±12.59 years (range: 18–78); 55.3% were male and 44.7% were female. Overall, 64.4% reported occasionally forgetting to take their medication. Regarding frequency, 49.3% reported forgetting medication one day per week, 34.2% two days, 13.2% three days, 2.3% four days, and 0.9% five days, yielding a median of 2 forgotten days per week. Furthermore, 65.6% of participants perceived daily medication intake as difficult.

The younger group (n=157) demonstrated a mean age of 32.67±6.36 years, whereas the older group (n=183) had a mean age of 52.48±8.86 years. The median number of forgotten medication days was 2 among younger and 1 among older participants. Medication forgetting was significantly more prevalent in younger individuals (77.1% vs. 53.6%; $p<0.001$), as was perceiving daily medication use as difficult (75.8% vs. 56.8%; $p<0.001$). A weak yet statistically significant negative correlation was identified between age and the number of forgotten days ($r = -0.146$; $p = 0.031$).

Conclusion

Medication nonadherence—particularly medication forgetting—appears to be highly prevalent among individuals using chronic oral medications in a community sample. Both the frequency of forgetting and the perception of daily medication use as burdensome were more pronounced in individuals younger than 40 years. The inverse association between age and the frequency of forgotten medication days underscores the potential value of age-specific interventions aimed at enhancing medication adherence, particularly within younger populations.

Keywords: Attitudes, Chronic disease, Medication adherence, Oral medication, Treatment compliance

AuthorToEditor: This study aims to evaluate attitudes toward regular oral medication use and levels of medication adherence in a community sample, with a particular focus on age-related differences. The findings are expected to contribute to a better understanding of medication adherence behaviors and to inform targeted interventions aimed at improving adherence in clinical and public health settings. Thank you for your consideration. ORCID: 0009-0008-1967-6349

[Abstract:0307]

Schizophrenia-Like Behavioral Alterations in a Prenatal Poly (I:C)-Induced Model of Rats

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Objective: Schizophrenia is a severe, chronic, and multifactorial psychiatric disorder affecting approximately 1% of the general population and is characterized by positive, negative, and cognitive symptoms [1,2]. Increasing evidence indicates that exposure to viral or bacterial infections during pregnancy induces maternal immune activation (MIA), which increases the risk of neurodevelopmental disorders, including schizophrenia, in the offspring [3,4]. Accordingly, maternal immune activation animal models are considered valuable translational tools for investigating the behavioral consequences of immune challenges during gestation.

Therefore, the aim of the present study was to evaluate the effects of prenatal Poly (I:C) exposure on schizophrenia-related behavioral domains in rats by comparing behavioral outcomes between control and Poly(I:C)-exposed groups.

Methods: Maternal immune activation was induced by a single subcutaneous injection of polyinosinic–polycytidylic acid (Poly I:C) administered at a dose of 8 mg/kg to pregnant Wistar albino rats on gestational day 15. Male offspring were assigned to either the control group or the Poly(I:C)-exposed group (n = 8 per group). In adulthood, animals were subjected to a battery of behavioral tests designed to assess behaviors relevant to the core symptom domains of schizophrenia. Locomotor activity and exploratory behavior were evaluated using the Open Field Test, while social behavior was assessed using the Social Interaction Test. Recognition memory was examined using the Novel Object Recognition Test. All behavioral assessments were conducted in accordance with standardized procedures.

Results: Prenatal exposure to Poly (I:C) was associated with behavioral alterations compared to the control group. Poly (I:C)-exposed animals exhibited reduced social interaction, impaired recognition memory, and changes in locomotor and exploratory behavior patterns. In the Social Interaction Test, rats in the Poly (I:C) group showed decreased engagement in social behaviors relative to controls. In the Novel Object Recognition Test, prenatal Poly (I:C) exposure was associated with a reduced preference for the novel object, indicating alterations in recognition memory performance. In the Open Field Test, Poly (I:C)-exposed animals displayed differences in locomotor activity and exploratory behavior compared to control animals. The observation of these behavioral changes in adulthood suggests that prenatal immune challenge may lead to long-lasting effects on schizophrenia-related behavioral domains.

Discussion: The findings of this study indicate that prenatal maternal immune activation induced by Poly(I:C) exposure is associated with persistent behavioral alterations in adult offspring. Changes observed in social interaction, recognition memory, and locomotor behavior are consistent with behavioral domains commonly associated with the core symptom clusters of schizophrenia. These results support existing literature suggesting that immune challenges occurring during critical periods of gestation can result in long-term behavioral consequences. Overall, the present study reinforces the utility of the prenatal Poly(I:C) model as a translational approach for investigating schizophrenia-like behavioral phenotypes and provides a behavioral framework for future studies aimed at exploring the neurodevelopmental effects of maternal immune activation.

Keywords: Behavioral tests, Cognitive deficits, Maternal immune activation, Poly(I:C), Schizophrenia

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[Abstract:0342]

Onset of Motor Tics Following Citicoline Use in a Child With Dopaminergic Sensitivity: A Case Report

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Introduction:

Citicoline (CDP-choline) is a widely used nutritional supplement commonly regarded as safe and used to support cognitive functions in children and adolescents. To our knowledge, no cases directly associating citicoline use with tic symptoms have been reported in the literature. However, considering its effects on dopaminergic neurotransmission, citicoline may act as a triggering factor for tic symptoms, particularly in individuals with dopaminergic sensitivity. This report presents a pediatric case of motor tic onset following citicoline use.

Case presentation:

A 12-year-old male sixth-grade student presented to a child and adolescent psychiatry outpatient clinic with newly emerging motor tics. He had no known neurological or systemic medical disorder. Approximately three weeks prior to admission, eye-blinking motor tics appeared following initiation of citicoline supplementation by his mother. Pediatric and ophthalmologic evaluations did not reveal an organic etiology. No vocal tics were observed.

Ten days after discontinuation of citicoline, tic frequency decreased by approximately 50%, and further spontaneous improvement was observed during follow-up without pharmacological intervention.

Past psychiatric history revealed attention-deficit/hyperactivity disorder, predominantly inattentive presentation. Previous treatments included methylphenidate formulations and aripiprazole. Motor tics had emerged during methylphenidate treatment and reportedly worsened with aripiprazole, suggesting dopaminergic sensitivity. At the time of evaluation, mental status examination was largely unremarkable, and tics did not cause significant academic or social impairment.

The temporal relationship between exposure and symptom onset, improvement after discontinuation, prior sensitivity to dopaminergic agents, and absence of alternative medical explanations were evaluated using the Naranjo Adverse Drug Reaction Probability Scale.

Conclusion:

Improvement of tic symptoms following discontinuation of citicoline suggests that citicoline-associated tics may be reversible in this case. The absence of functional impairment and spontaneous regression supports the possibility of a trigger-related phenomenon rather than a primary tic disorder. Over-the-counter cognitive supplements should be carefully considered in children with dopaminergic sensitivity due to potential neuropsychiatric effects. This case highlights the importance of evaluating potential triggers before initiating pharmacological treatment in newly emerging tic symptoms.

Keywords: Citicoline, Dietary supplements, Dopaminergic sensitivity, Motor tic, Pediatric psychopharmacology

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[Abstract:0373]

Low-Dose Aripiprazole Augmentation and Thermoregulation Management in Generalized Anxiety Disorder with Dissociative Features Following Pediatric Hodgkin's Lymphoma: A Case Report

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Introduction: A significant portion of patients with anxiety disorders remain symptomatic despite adequate trials of first line pharmacological agents such as SSRIs for at least 8 weeks [1]. The presence of dissociative symptoms in these cases serves as a primary indicator of poor response to standard treatment regimens, reflecting a complex psychopathology often rooted in childhood trauma and chronic environmental stress [2]. In such treatment resistant scenarios, SSRI monotherapy frequently fails to overcome dissociative barriers, necessitating augmentation strategies like aripiprazole [2]. Aripiprazole's efficacy stems from its unique neurotransmitter stabilizing mechanism as a partial agonist at D2 and 5 HT1A receptors; it has been reported that this partial agonism, along with the facilitation of cortical and hippocampal glutamatergic transmission, may serve as a key therapeutic mechanism in resolving dissociative symptoms such as depersonalization and derealization [3]. However, body temperature homeostasis is also regulated by D2 activity in the preoptic anterior hypothalamus (POAH). Antipsychotic effects in this region can disrupt the thermoregulatory threshold, leading to autonomic symptoms, particularly in individuals with high autonomic sensitivity. It is hypothesized that central sensitization resulting from chronic medical trauma, such as a pediatric oncology history, may further increase susceptibility to these responses. This report discusses the management of aripiprazole induced autonomic side effects through micro-titration in an adolescent with medical trauma.

Case Presentation: A 16-year-4-month-old female patient presented with severe dissociative symptoms, including dissociative amnesia (dissociative agraphia, confusion at bus stops, and inability to recall primary relatives' names) and depersonalization/derealization episodes. Her medical history was significant for Hodgkin's Lymphoma at age 12. Following oncology treatment, she was diagnosed with Generalized Anxiety Disorder (GAD), but failed to respond to Escitalopram and Risperidone due to poor tolerability and non-compliance. Physical examination and investigations showed no organic pathology. The patient was started on aripiprazole 5 mg/day; however, severe autonomic symptoms including hot flashes, tachycardia, and vomiting developed after the first dose. Instead of discontinuing the drug, the dose was reduced to 2.5 mg/day for one week. Subsequently, a micro-titration protocol involving alternate-day dosing (2.5 mg and 5 mg) was applied for one week to facilitate D2 receptor adaptation, successfully reaching the target dose of 5 mg/day without recurrence of side effects.

Conclusion: The partial agonistic effect of aripiprazole on thermoregulatory D2 receptors in the POAH forms the pathophysiological basis of the transient autonomic dysregulation observed in this case. This presentation should be evaluated not merely as a side effect, but as a manageable thermoregulatory threshold sensitivity. In adolescents with a history of pediatric oncology, increased somatic sensitivity due to CNS sensitization must be considered. Consequently, in anxiety cases with comorbid dissociative symptoms, clinical success should be aimed through individualized micro-titration schemes that exceed the standard start low, go slow principle.

Keywords: Aripiprazole, Thermoregulation, Micro-titration, Dissociation, Central Sensitization

Table 1: Micro-titration Protocol and Autonomic Threshold Management

Phase	Daily Dose	Clinical Strategy & Observations
Initial (Day 1)	5 mg	Acute Autonomic Dysregulation: Severe hot flashes, tachycardia, and emesis.
Stabilization (Week 1)	2.5 mg	Dose reduction to establish autonomic stability and tolerability.
Micro-titration (Week 2)	2.5 mg / 5 mg (Alternate days)	Non-linear Titration: Aiming for gradual D2 receptor adaptation and threshold adjustment.
Maintenance (Week 3+)	5 mg	Achievement of target dose with full tolerability and clinical response.

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[Abstract:0387]

Cognitive Disengagement Syndrome in Cornelia de Lange Syndrome: Expanding the Neuropsychiatric Phenotype

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Introduction: Cornelia de Lange Syndrome (CdLS) encompasses a spectrum of findings from mild to severe. CdLS is characterized by distinctive facial features, growth restriction, hypertrichosis, and upper-limb reduction defects that range from subtle phalangeal abnormalities to oligodactyly(1). Also CdLS is a multisystem genetic disorder characterized by distinctive physical features, intellectual disability (ID), and a spectrum of neurodevelopmental and psychiatric problems(2). The vast majority of CdLS patients have varying degrees of intellectual disability. Behavioral difficulties are common in individuals with CdLS, with anxiety, attention problems and self-injurious behaviors in affected individuals(3). Cognitive Disengagement Syndrome (CDS), formerly known as Sluggish Cognitive Tempo, is a distinct cluster of attentional difficulties increasingly recognized as impacting academic functioning beyond typical inattentive symptoms of ADHD(4). CDS is characterized by persistent cognitive deficits including daydreaming, mental confusion, appears lost in fog and physical activity, and challenges in emotional regulation and social functioning (5).

This report presents a detailed psychiatric evaluation of a pediatric patient with Cornelia de Lange Syndrome at the time of admission to the psychiatry clinic.

Case Presentation: A 12-year-old male patient had been followed in the genetics department with a diagnosis of CdLS. A heterozygous c.647del variant in the BRD4 gene had been identified.

The patient was receiving levothyroxine sodium treatment for comorbid hypothyroidism; current thyroid function tests were within normal limits. His facial features were noted to differ from those of his peers. Phenotypically, short stature, hypertelorism, a depressed nasal bridge, thick eyebrows, and oligodactyly were observed. He presented in January 2026 due to a decline in academic performance. His mother reported that he was generally a compliant child but exhibited slower behavior compared to his peers, often appearing daydreaming and immersed. His teacher reported that he adhered to classroom rules and didn't disrupt classroom order; however, his attention was easily distracted, and he appeared slow. During the clinical interview, he demonstrated delayed response latency but provided accurate answers.

Cognitive performance was evaluated using the WISC-R, which revealed a full-scale IQ score of 73. ID wasn't considered the primary factor explaining the clinical presentation. The patient scored 3.0 on the Child and Adolescent Behavior Inventory–Slow Cognitive Tempo (CABİ-SCT) scale.

Conclusion: This case highlights the necessity of comprehensive psychiatric evaluation in patients with CdLS. Clinical observations, CABİ-SCT results, and WISC-R findings supported the presence of CDS. The use of the CABİ scale and WISC-R testing also contributes meaningfully to clinical assessment by facilitating differentiation between intellectual disability and CDS. In the present case, although the patient demonstrated borderline intellectual functioning (IQ = 73), clinical observations revealed disproportionate cognitive slowing, delayed response latency, and daydreaming-like mental disengagement. These features were more consistent with CDS, a distinct construct characterized by excessive mental fogginess, slowed information processing, reduced alertness, and behavioral passivity.

Keywords: Cornelia de Lange Syndrome, Cognitive Disengagement Syndrome, intellectual disability

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[Abstract:0407]

Epistaxis Associated with Risperidone Dose İncrease: A Case Report

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Introduction: Risperidone is a second-generation antipsychotic commonly used in child and adolescent psychiatry to manage disruptive behaviors, including hyperactivity, irritability, and aggression. While weight gain and sedation are frequent adverse effects, hemorrhage is rarely reported. This case highlights a rare occurrence of epistaxis associated with risperidone-induced thrombocytopenia and 5-HT2A receptor antagonism.

Case presentation: A 15-year-old female patient presented to the emergency department following a drug overdose suicide attempt. Clinical evaluation revealed a one-year history of anhedonia, anergy, sleep disturbances, and irritability, accompanied by intermittent self-harming behaviors and impaired impulse control.

Based on a preliminary diagnosis of Major Depressive Disorder, risperidone was initiated at 0.5 mg/day. The patient reported no adverse effects at the initial dose; however, upon titration to 1 mg/day, she experienced daily episodes of epistaxis (nosebleed). During the treatment period, physical examination, vital signs, and laboratory investigations, including platelet counts, were within normal limits.

Otorhinolaryngology consultation revealed no local etiology for the bleeding. There was no history of intranasal substance use, bleeding disorders, or allergies. Family history was negative for coagulopathy, and the patient was not taking any other medications concurrently. Due to persistent epistaxis, risperidone was discontinued, resulting in the complete resolution of the bleeding episodes.

Conclusion: Various mechanisms may underlie risperidone-induced hemorrhage, including thrombocytopenia and 5-HT2A receptor antagonism. Although thrombocytopenia is a recognized side effect of atypical antipsychotics and has been reported with risperidone, the normal platelet count in our case suggests that the epistaxis was not a direct consequence of a quantitative platelet deficiency.Instead, it is proposed that 5-HT2A receptor antagonism leads to bleeding by inhibiting the release of vasoconstrictors from platelets and reducing platelet aggregation. Given risperidone's high affinity for 5-HT2A receptors, we hypothesize that this mechanism likely resulted in the observed epistaxis. While hemorrhage remains a rare adverse effect, clinicians must remain vigilant regarding bleeding risks, particularly in susceptible patients with a history of coagulation disorders or those concurrently using medications that impair hemostasis, such as NSAIDs, aspirin, or anticoagulants. This case underscores the importance of monitoring dose-dependent epistaxis as a rare but significant adverse effect of risperidone, likely mediated by serotonergic pathways rather than quantitative thrombocytopenia.

Keywords: Epistaxis, risperidon, thrombocytopenia

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