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[Abstract: 0771] [Dependencies]

Understanding relapse concept

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ABSTRACT

Being considered as a social disorder, addiction has become a major problem in many countries over the past years. Despite long-term and recurrent suffering of patients, families and community caused by drug dependence, curing the disorder has not yet been possible. Although patients can stop using alcohol/drugs for a while, it is always possible that they may return to alcohol/drug use. It has long been known that addictive disorders are chronic and relapsing in nature. Specifically, relapse is viewed as a return to the disease state. Recently a shift in focus has been observed towards including minor "slips" or "lapses" with a possibility of resuming abstinence or "health" instead of considering them full-blown relapse or "disease". Stress, marital problems, financial issues, adverse life events, psychiatric comorbidity such as depression or anxiety, positive mood, social pressure, family dysfunction, and a lower level of social support are among factors that cause or precipitate relapse. Relapse cannot be counted an isolated event, but it is more of a process of becoming unable to cope with life in sobriety. However, the process may cause returning to alcohol/drug use, emotional disturbances, or even suicide. The relapse process is marked by predictable and identifiable warning signs that begin long before a return to use. Recent estimates from clinical treatment studies suggest that more than two thirds of individuals relapse within weeks to months of initiating treatment [1,2]. For 1-year outcomes across alcohol, nicotine, weight, and illicit drug abuse, studies show that more than 85% of individuals relapse and return to drug use within 1 year of treatment [3].

KEYWORDS

Addiction; dependency; relapse; relapse prevention

References

[1] Paliwal P, Hyman SM, Sinha R. Craving predicts time to cocaine relapse: further validation of the now and brief versions of the cocaine craving questionnaire. Drug Alcohol Depend. 2008;93:252–259. [PubMed: 18063320]

[2] Sinha R, Garcia M, Paliwal P, et al. Stress-induced cocaine craving and hypothalamic-pituitary adrenal responses are predictive of cocaine relapse outcomes. Arch Gen Psychiatr. 2006;63:324–331. [PubMed: 16520439]

[3] Brandon TH, Vidrine JI, Litvin EB. Relapse and relapse prevention. Annu Rev Clin Psychol. 2007;3:257–284. [PubMed: 17716056]

[Abstract: 0755] [Addictions]

Prevention of relapse in addiction and new approaches

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ABSTRACT

Relapse, lapse or slip is the term used for the situation in which people starts to use substance again after a period of abstinence. And, relapse could be a very informative process for both the patient and therapist, even it is an obstacle for the treatment process. It is an active and continuing process.

KEYWORDS

Relaps prevention; addiction; psychoeducation; moodist relapse index; feedback

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Relapse prevention and classifying the factors underlying relapse is prerequisite for any attempt to success and change in the addiction related behaviors in long-term. Relapse prevention is a clinical intervention program which especially intervenes in psychosocial context. It begins with the identification of problematic behaviors, ideas, people, places, situations, and so on.

Relapse should not be a failure or fatal ending, instead, it should be a crossroad. It is a treatment process which is individual-focused, and not like the traditional models applicable for everyone. The reason why relapse prevention program is individual-focused is the fact that high-risk situations leading to relapse are dependent on the substance used, personality features, and experiences of individuals.

The basic strategies in relapse preventions could be sorted as assessing high-risk situations, dealing with craving, resisting insistence, dealing with the thoughts related to substances and alcohol, dealing with the emergency situations, and responding to emotional crisis.

Through the consideration of risk factors for relapse given in literature, 33-items questionnaire was prepared to assess personal factors leading to relapse and give patients feedback about the prevention of using again. The questionnaire was given to the inpatients in Moodist Psychiatry and Neurology Hospital as a pilot study. The questions which could not be understood by the patients were removed, and a new 24-items questionnaire was prepared. This form was named as Moodist Relapse Index (MORI). MORI was given and asked to be fulfilled by 37 inpatients in the same hospital days between April to July 2017.

Cronbach alpha's for MORI was 0.86. The results of factor analysis showed that 7 factors could explain the total variance of 47.3%. These factors were named as dealing with stress, seeking-reward, friend environment, environment where the substance used, negative feelings, free time, and excessive self-confidence.

MORI has many advantages in terms of culture-fairness while working with patients, the assessment of personal risk factors, time-efficiency as a self-report questionnaire, providing a chance for comparison since the data were saved in a database, making easy to work with the patients by enabling the therapist to work upon concrete demonstrations, and providing systematic data.

References

[1] Marlatt GA, Gordon JR. Relapse prevention: maintenance strategies in the treatment of addictive behaviors. New York: Guilford Press; 2007.

[2] Hendershot CS, Witkiewitz K, George WH, et al. Relapse prevention for addictive behaviors. Subst Abuse Treat Prev Policy. 2011;6:17.

[3] Michael S. Listening to our clients: the prevention of relapse. J Psychoactive Drugs. 2008;40(2):167–172.

[4] Ögel K, Guliyev C, Tekin K, et al. Development of the moodist relapse index scale. 11. National Alcohol Substance Addiction Congress; 2017.

[Abstract: 0760] [Dependencies]

A model of psychoeducation for relapse prevention

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ABSTRACT

Psychoeducation is the main component of addiction treatment. Psychoeducation seems to have a wide place especially in studies conducted in relapse prevention. Psychoeducation is a cognitive intervention that reduces the likelihood of relapse; it is based on beneficial psychological principles that support behavior change. Therefore, it is considered that determining individual needs and developing specific psychoeducation models for each patient are important. In this context, three psychoeducation models have been developed: Addiction Information (BILGE), Cognitive Education for Addiction (BABIL) and Life Style Regulation Model.

In the process of addiction, the individual may have mistaken thoughts and information about alcohol/substance use. BILGE is a psychoeducation model developed to evaluate the dependency information of the dependent individual and to determine the false information and thoughts. In this model of psychoeducation, the nature and developmental stages of addiction, the steps that need to be taken for the environment, friend or lifestyle to recover and not begin again are explained from a wide perspective to the dependent individual. In this context, this model plays an important role in increasing the knowledge of addiction and realizing their misinformation about alcohol and substance use. It is also a protective psychoeducation approach for patients who are at risk of using alcohol / substance due to lack of information and misconceptions. Dependence has a negative effect on cognitive functions such as attention, memory and response inhibition. BABIL is a psychoeducation model designed to determine the cognitive processes affected by addiction and to improve the cognitive processes. It's explained to the individual that the dependence damages attention, memory and control mechanism by affecting

KEYWORDS

Addiction information; cognitive education; lifestyle regulation; psychoeducation approach; relapse prevention

some brain areas, in the perspective of biopsychosocial approach. This educational model emphasizes that even if the people don't want to, they cannot control their brain and use it again. In this context, BABIL is aimed at developing cognitive functions; it also provides detailed information on relapse prevention.

Activities such as personal care, nutrition and physical exercise decrease or even disappear due to dependence. Life Style Regulation Model is a psychoeducation model prepared to determine healthy lifestyle behaviors in dependence process and to regulate and improve the lifestyle of the individual. With this psychoeducation model, physical exercise and movement, taking care of nutrition, self-care, being interested in health issues, taking responsibility information is given to the individual. These informing suggest that the beginning of neglect for lifestyle is a premonitory of relapse. Also, it's encouraged that the dependent individual changes their lifestyle during the healing process.

The developed psychoeducation models have many advantages such as the suitability of our culture about working with the dependent individual, presenting the patient specific model, informing on different subjects related to addiction and emphasizing the relapse prevention.

References

[1] Gould TJ. Addiction and cognition. Addict Sci Clin Pract. 2010;5(2):4–14.
[2] Marlatt GA, Gordon JR. Relapse prevention: maintenance strategies in the treatment of addictive behaviors. New York: Guilford Press; 2007.
[3] Walsh R. Lifestyle and mental health. Am Psychol. 2011;66(7):579–592. doi:10.1037/a0021769

[Abstract: 0701] [Psychopharmacology]

Side effects of antipsychotic agents and treatments

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ABSTRACT
Patients with schizophrenia suffer from increased rates of multiple medical problems, this is likely due to their unhealthy lifestyle, high smoking rates, high-fat diet and obesity, poor compliance, neglect of personal care and physical activity. In additional, patients treated with conventional antipsychotics suffer from significant sexual dysfunctions, extrapyramidal and psychic side effects, sedation or vegetative side effects.

Since the introduction of atypical antipsychotics, these agents have been widely prescribed for the management of patients with schizophrenia, bipolar disorders and other psychotic disorders or conditions with severe behavioral disturbance. The increasing use of atypical antipsychotics is in part due to their lower propensity to induce extrapyramidal symptoms and tardive dyskinesia compared to typical antipsychotics. However, psychiatrists realized that while extrapyramidal symptoms and tardive dyskinesia occur less frequently with atypical agents, they also may present a different set of adverse effects. The recent findings concern weight gain, diabetes mellitus, hyperlipidemia, QTc interval prolongation, myocarditis, extrapyramidal side effects, sexual side effects, and cataract. Some recommendations about how to prevent and manage these side effects are provided by some treatment guidelines. It is suggested that atypical antipsychotics do not represent a homogeneous class and that differences in side effects should be considered by clinicians when choosing antipsychotic for an individual patient.

KEYWORDS
Schizophrenia; antipsychotics; side effects; treatment

[Abstract: 0666] [Others]

What we might lose without even knowing: health implementation directive in the practice of child and adolescent psychiatric training

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ABSTRACT
The number of children and adolescent psychiatry specialists in our country is increasing. The vast majority of the working experts are working in hospitals affiliated to the ministry of health.

KEYWORDS
Child; adolescent; Psychiatry

The services performed by the doctors during the examination are scored according to the health practice instructions. The performance payments to be made to the doctors are calculated after examining the services of the doctors for a month by the commissions formed in each hospital. These commissions may decide that the scoring is not appropriate, and the implementation of these commissions may be different for each hospital. Due to the nature of psychiatric interviews, the duration of the evaluation lasts longer than the other branches and a separate interview with the child and the family is required. Although the evaluation period is longer, there is no significant difference in the evaluation score according to the other branches in health practice instruction and this situation is reflected negatively on the performance score for child and adolescent psychiatrists.

In this presentation; the problems experienced by the children and adolescent psychiatrists regarding the health practice instruction and performance payments will be reviewed and possible solutions will be discussed together with the participants.

[Abstract: 0773] [Schizophrenia and other psychotic disorders]

AI and schizophrenia - current and prospects

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ABSTRACT

Despite the advances in schizophrenia research over the last decades, many questions have remained unanswered in terms of diagnosis and treatment. The understanding of biological mechanisms underlying schizophrenia has grown, but clinical translation of biomarkers for identifying a disorder or predicting treatment outcome has not been as expected yet. Current diagnostic classifications of psychiatric disorders solely rely on presenting signs and symptoms, and heterogeneity in clinical presentation and longitudinal instability of clinical diagnoses can be problematic for many patients [1]. Computational approaches may assist the field of schizophrenia research in several ways. Recently, machine learning approaches have been successfully applied for analysis of multimodal data including clinical, neuroimaging and genetic data to distinguish between patients with schizophrenia and healthy controls [2]. Efforts are being made towards making more accurate predictions in terms of not only diagnosis but also prognosis and response to treatment. Recent technological breakthroughs in artificial intelligence, such as unsupervised learning and deep learning are promising for improving differential diagnosis [3] and they can help moving towards a more patient-tailored treatment program.

KEYWORDS

Artificial intelligence; machine learning; schizophrenia; psychosis

References

[1] Stephan KE, Bach DR, Fletcher PC, Flint J, Frank MJ, Friston KJ, Heinz A, Huys QJ, Owen MJ, Binder EB, Dayan P. Charting the landscape of priority problems in psychiatry, part 1: classification and diagnosis. The Lancet Psychiatry. 2016;3(1):77–83.

[2] Veronese E, Castellani U, Peruzzo D, Bellani M, Brambilla P. Machine learning approaches: from theory to application in schizophrenia. Computational and mathematical methods in medicine. 2013.

[3] Gheiratmand M, Rish I, Cecchi GA, Brown MR, Greiner R, Polosecki PI, Bashivan P, Greenshaw AJ, Ramasubbu R, Dursun SM. Learning stable and predictive network-based patterns of schizophrenia and its clinical symptoms. NPJ Schizophrenia. 2017;3(1):22.

[Abstract: 0563] [Mood disorders]

AI solutions for bipolar disorder: natural language processing and face recognition

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ABSTRACT

Bipolar disorder (BD) is conventionally characterized by depressive and manic episodes although interepisodic affective instability is increasingly recognized. Automatic monitoring of bipolar disorder is aimed with a variety of new approaches, emotion recognition from audio- visual

KEYWORDS

Artificial intelligence; bipolar disorder; face recognition; natural language processing

features and text analysis with techniques of natural language processing. Turkish database for bipolar patients is collected, patients with the diagnosis of bipolar mania disorder are video-recorded and scored with clinical evaluation scales from the first day of hospitalization until the control after discharge [1]. Signal processing and machine learning (ML) methods for recognition of BD from short video clips of subjects performing a small battery of affective tasks are applied. From each face, we extract geometric features as suggested by [2] for video-based emotion recognition and extract appearance descriptors from registered faces, using a pre-trained Deep Convolutional Neural Network (DCNN) fine-tuned on a face-based emotion corpus. Under AVEC 2018 Bipolar disorder subchallange [3], a variety of techniques are also applied both for facial recognition and text analysis. Facial Action Units (FAUs) which describe human facial expression and can judge whether the subject suddenly laughs or cries without reason and Eyesight features which show whether the subject is in hostility or dull state are measured by ML techniques. Automatic analysis of verbal and nonverbal interactions' content will be helpful for the development of personalized treatment by providing the detection of rich feedbacks between patients and doctor. Text-based features have been proved effective so text features using the suite of Linguistic Analysis Tools (SALAT), Natural Language Processing Tool (siNLP) and Sentiment Analysis and Cognition Engine (SEANCE) text analysis were performed automatically on the transcripts of BD interview. The accurate prediction of symptoms in patients with bipolar disorder could facilitate timely clinical interventions enhancing patient's quality of life, altering the course of disorder and could have economic benefits for the national health service.

References

[1] Çiftçi E, Kaya H, Güleç H, et al. The Turkish audio-visual bipolar disorder corpus. Proceedings of the 1st Asian Conference on Affective Computing and Intelligent Interaction (ACII Asia), AAAC; Beijing, China; 2018. p. 6.

[2] Kaya H, Gürpinar F, Afshar S, et al. Contrasting and combining least squares based learners for emotion recognition in the wild. Proceeding ACM ICMI; 2015. p. 459–466.

[3] Ringeval F, Schuller B, Valstar M, et al. AVEC 2018 workshop and challenge: bipolar disorder and cross-cultural affect recognition. 2018 Audio/Visual Emotion Challenge and Workshop (AVEC'18); 2018 Oct 22; Seoul (Republic of Korea). New York, NY (USA): ACM. p. 11.

[Abstract: 0035] [PTSD]

What we don't touch when we touch trauma?

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ABSTRACT

Exposure to potentially traumatic experiences can lead to several difficulties from only fatigue and sleep problems to severe posttraumatic stress disorder (PTSD). Active efforts to avoid or escape from trauma-related thoughts, feelings, and situations are key features of the disorder delineated in the diagnostic criteria of PTSD. Acceptance and Commitment Therapy model suggests that experiential avoidance or escape, strategies designed to alter one's experience of private events, is a process that underlies many forms of psychopathology. When we look more closely to PTSD from ACT perspective; fusion with especially negative evaluations of self ('I am damaged', 'I am defective', 'I am weak'), self-conceptualizations only related with trauma, blurred valued areas and retiring from valued living because of experiential avoidance seems to be central of the disorder. Fusion with the literal content of unwanted thoughts and feelings (e.g., "I am damaged" and anxiety symptoms), and spending an inordinate amount of time avoiding these thoughts and feelings hinders the ability to pursue long-term values and goals. They begin to see themselves at a level where they cannot reach their values and they develop a big gap between themselves and valued domains/valued people they care about And two related concepts often overlooked in trauma therapy, guilt and shame, seems to have a role on maintenance of PTSD. Working with trauma is difficult for the victims of traumatic events as well as for the therapists and can cause emotional difficulties like anxiety and depressive symptoms as a result of helping or wanting to help a traumatized or suffering person, named as 'secondary traumatic stress', in therapists. And this can end up with therapists' avoidance behaviors which can influence the effectiveness of therapy. Interventions on guilt and shame found to be useful to overcome PTSD related difficulties and self-compassion (SC) interventions provide an effective way for this. Interventions, which show the person that they are close to the values and the small committed actions steps can serve a big source of motivation for therapeutic wellness. Relatively remained processes in the background like 'values, self-conceptualizations, shame and secondary traumatization' will be discussed from ACT perspective in this presentation.

KEYWORDS

Acceptance and Commitment Therapy; Self-Compassion; Valued actions

[Abstract: 0762] [Psychotherapy]

Loss of contact with meaning after traumatic experiences

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ABSTRACT
Victims of trauma often have a dramatical change in their judgment about self and the meaning of the world. They start to see themselves like "damaged", "impaired", "unreliable" or "disgusting" person after the trauma. Because they lose the important aspects of their lives with the trauma; they lose some functions of their body, they lose friends, relatives, maybe they lose their jobs, may be some abilities and may be lose their pudicity. When they fused with these negative self-evaluations such as damaged, disgusting or unreliable, their self-perception will be changed with that fused cognitive material and two different persons appear in the eyes of trauma victim; one is named "before the trauma" and the other one "after the trauma". Regardless of how the individual's world is *structured*, rigid fusion with such negative self-evaluations would, for all practical purposes, change that world to one that is consistent with those evaluations. Describing oneself as disgusting and unreliable, for example, would result in the undesirable aversive properties of things that are more objectively disgusting and unreliable to become attached to oneself. Such unfortunate functional transformations would also be expected to participate in maladaptive rule-governed behavior. One who is "disgusting", for example, should keep hidden from view, and should not pursue meaningful goals due to unworthiness. And one who is "unreliable" cannot enter meaningful relationships with others because they will inevitably be let down. Believing the content of such evaluations makes the world so, regardless of what the world is. They begin to see themselves at a level where they cannot reach their values and they develop a big gap between themselves and valued domains/valued people they care about. By the way victim of the trauma start to lose the contact with his values and move away from the important areas for him. With the establishment of this distancing attitude from the values makes the clinical appearance more rigid and problematic. In the direction of therapy goals in acceptance and commitment therapy we try to make contact again with the important things in his life and move the clients towards to these valued domains with openness and willingness to the private experience that contains different forms of pain associated with traumatic memory. Existing models of trauma indicate that survivors are most likely to recover when trauma related cues are met with awareness, openness, and the continuation of behaviors directed toward the pursuit of valued life aims (rather than mere regulation of emotions).

KEYWORDS
Acceptance and Commitment Therapy; Values; Trauma; Psychological Flexibility

References

[1] Hayes SC, Louma JB, Bond FW, et al. Acceptance and commitment therapy: model, processes and outcomes. Behav Res Ther. 2006;44(1):1–25.
[2] Orsillo SM, Batten, SV. Acceptance and commitment therapy in the treatment of posttraumatic stress disorder. Behav Modif. 2005;29(1):95–129.
[3] Kashdan, Todd B, Kane JQ. Post-traumatic distress and the presence of post-traumatic growth and meaning in life: experiential avoidance as a moderator. Pers Indiv Differ. 2011;50(1):84–89.

[Abstract: 0686] [PTSD]

'Am I weak or guilty?' trauma related shame and self-conceptualizations from ACT perspective

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ABSTRACT
Approximately 50–75% of the population experiences a traumatic event throughout their lives, but PTSD symptoms develop in a much lower percentage range from 8% to 15%. This exposure to potentially traumatic experiences can lead to not only severe posttraumatic stress disorder (PTSD) but also several difficulties like fatigue, sleep problems and difficulty in relationships [1]. Active efforts to avoid or escape from trauma-related thoughts, feelings, and situations are key features of the disorder delineated in the diagnostic criteria of PTSD. ACT model suggests that experiential avoidance or escape, strategies designed to alter one's experience

KEYWORDS
ACT; Posttraumatic stress disorder; Self; Shame; Self-compassion

of private events, is a process that underlies many forms of psychopathology and narrows the behavior repertoire [2]. Cognitive fusion, one another key feature of ACT model about psychopathology, is seen in the form of negative self-evaluations in PTSD. Self-evaluations related with trauma (e.g., "I am damaged", "I am defective", "I am weak") becomes an identity for the client and shaping the behaviors according to these identities put the client away from valued areas [3,4]. Another self-conceptualization related behavior, self-criticism, also plays a central role in PTSD symptoms especially in relationship difficulties. Self-compassion (SC), as an alternative approach to self-criticism, could provide an effective way for these difficulties especially focusing on a trauma related emotion 'shame'. Researches support the effectiveness of SC interventions on trauma-exposed samples [5,6]. While SC can be seen as implicitly involved in all ACT work, making it explicit in in therapy, especially when working with highly self-critical and shame-prone clients as in PTSD, may improve the outcomes.

References

[1] Shalev AY, Freedman S, Peri T, et al. Prospective study of posttraumatic stress disorder and depression following trauma. Am J Psychiatr. 1998;155(5):630–637.

[2] Hayes SC, et al. Acceptance and commitment therapy: model, processes and outcomes. Behav Res Ther. 2006;44(1):1–25.

[3] Orsillo SM., Batten SV. Acceptance and commitment therapy in the treatment of posttraumatic stress disorder. Behav Modif. 2005;29(1):95–129.

[4] Follette VM, Ruzek JI, editors. Cognitive behavioral therapies for trauma. Ch 7. Guilford Press; 2007.

[5] Kearney DJ, Malte CA, McManus C, et al. Loving-kindness meditation for posttraumatic stress disorder: a pilot study. J Trauma Stress. 2013;26(4):426–434.

[6] Au, TM, Sauer-Zavala S, King MW, et al. Compassion-based therapy for trauma-related shame and posttraumatic stress: initial evaluation using a multiple baseline design. Behav ther. 2017;48(2):207–221.

[Abstract: 0772] [Psychotherapy]

Listening to trauma: perspective from acceptance and commitment therapy to secondary traumatization

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ABSTRACT

Psychotherapists who work with trauma survivors face with a set of difficulties. Working with trauma is difficult for the victims of traumatic events as well as for the therapists. Secondary traumatic stress defined as: 'The natural consequent behaviors and emotions resulting from knowing about a traumatizing event experienced by a significant other – the stress resulting from helping or wanting to help a traumatized or suffering person. Basically, secondary traumatic stress is the presence of post-traumatic stress disorder symptoms in caregivers, which are probably connected to the patient's experience rather than the caregivers' by Figley (1995). For the therapists it could cause emotional difficulties like anxiety and depressive symptoms because of helping or wanting to help a traumatized or suffering person and this can end up with therapists' avoidance behaviors which can influence the effectiveness of therapy. From ACT (Acceptance and Commitment Therapy) perspective, the purpose of therapy is to focus on acceptance of internal events rather than reduce or control them and to help individuals act more functional and flexible way toward their values. ACT approach is based on the view that control is the problem. While working with trauma survivors, trying to avoid inner experiences related to a traumatic event and "try not to affect by trauma" is one of the main causes of the burnout. Accordingly, the ACT perspective suggests therapists to approach not only the clients' disturbing feelings and thoughts but also their own internal events with openness and non-judgmental, compassionate way. Relatively remained processes in the background like 'values, self-conceptualizations, shame and secondary traumatization' will be discussed from ACT perspective in this presentation

KEYWORDS

Secondary traumatic stress; acceptance; compassion

References

[1] Bride BE. Prevalence of secondary traumatic stress among social workers. Social work. 2007;52(1):63–70.

[2] Harris R. ACT made simple: an easy-to-read primer on acceptance and commitment therapy. New Harbinger Publications; 2009.

[3] Yavuz KF. Kabul ve Kararlılık Terapisi (ACT): Genel Bir Bakış. Turkiye Klinikleri Journal of Psychiatry Special Topics. 2015;8(2):21–27.

[4] Figley CR. Compassion fatigue as secondary traumatic stress disorder: an overview. In: CR Figley, editor. Compassion fatigue: coping with secondary traumatic stress disorder in those who treat the traumatised. New York: Brunner/Mazel Publishers; 1995. p. 1–20.

[Abstract: 0575] [ADHD]

fMRI and alcohol use disorder

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ABSTRACT

Alcohol use disorder (AUD) is a chronic relapsing disease, characterized by recurrent compulsive alcohol abuse despite of significant alcohol-related physiological, cognitive, behavioural, and social problems. AUD can be considered as the world's biggest addiction problem. According to the World Health Organization, harmful use of alcohol is estimated to lead to 6% of all deaths, 5% of the global burden of disease, and has been demonstrated to have a causal relationship with over 200 adverse health complications. This makes AUD one of the most damaging preventable causes of illness in the world. When considering the total harm of AUD including its societal costs, alcohol can be ranked as the most harmful drug in the western world by a wide margin. Neurobiological mechanisms driving adaptive changes during alcohol abuse and subsequent recovery are not fully understood and continue to be of scientific and clinical interest. This is especially relevant due to the large human suffering associated with AUD and only limited effective treatment options. Patients suffering from AUD continue to experience high relapse rates and many families and communities continue to be directly affected by AUD through multiple generations, with 40-60% heritability as well as strong environmental factors. A growing body of evidence from human functional magnetic resonance imaging (fMRI) studies is gradually helping to elucidate the differences as well as progression of functional brain changes associated with acute alcohol intoxication, alcohol addiction, as well as AUD recovery. Recent fMRI findings suggest adaptive brain plasticity in both bottom-up appetitive and top-down executive functional brain networks in both active and recovered AUD. Active AUD is associated with wide-spread functional connectivity dysfunction and maladaptive brain circuit reinforcements. The neuroimaging evidence suggests that the "addicted" brain experiences excessive activation tone in the appetitive reward networks (often centred around nucleus accumbens) and decreased tone in the executive inhibition control networks (often centred around anterior cingulate cortex). These changes appear to be exaggerated in the opposite direction (rather than recovered back to the healthy baseline) in recovered AUD patients who have achieved successful prolonged abstinence. Successful clinical recovery from AUD thus results in a new, over-compensatory adaptive state rather than reversal of the maladaptive changes caused by the chronic alcohol abuse. Limited prospective data, furthermore, suggests that wide-spread functional dysfunction and deficits in the over-compensatory adaptive changes might be associated with early relapse. These findings have implications for patient guilt, stigma, as well as future research and experimental treatment options. Pilot studies of emerging brain modulation techniques (transcranial direct current stimulation, repetitive transcranial magnetic stimulation, and electroencephalogram neurofeedback) in addictions show promising potential, but very limited evidence. This presentation will provide an overview of (1) fMRI and its use in mental health research, (2) emerging evidence of functional brain changes in AUD and AUD recovery, and (3) the implications of these findings on predicting treatment outcome as well as accelerating experimental treatments. The audience will not only learn about brain plasticity in AUD, but also gain understanding of the strengths, limitations, and potential of neuroimaging in mental health.

KEYWORDS

Addiction; alcohol use disorder; functional magnetic resonance imaging; neuroimaging; neuroplasticity

[Abstract: 0782] [Others]

Repetitive transcranial magnetic stimulation in psychiatry practice

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ABSTRACT

Repetitive transcranial magnetic stimulation (rTMS) is used in treatment of major depressive disorder and a series of neuropsychiatric diseases as per approved by FDA. rTMS is used in clinical pictures such as obsessive-compulsive disorder, auditory hallucinations of schizophrenia, posttraumatic stress disorder, panic disorder, tinnitus, dystonia, stroke, and

KEYWORDS

Depression; obsessive-compulsive disorder; psychiatry practice; rTMS; treatment resistant

migraine. However, it is most commonly accepted to be used in depression and obsessive-compulsive disorder. The treatment is recommended to be used in depression and obsessive-compulsive disorder for which no benefit has been obtained from a series of treatment modalities as advised by both FDA and companies which sale the device. Since the treatment has been accepted as an affective and reliable modality by FDA and NICE, its capability of being offered as a primary care has also been brought into question. Although rTMS is a modality preferred in cases which are resistant to pharmacologic treatment, evaluation of response to rTMS in those patients is considered biased by some researchers.

Response to treatment is 95% in patients with untreated first attack depression, while 63% is in remission [1]. Only 20 to 40 % of the patients with depression who receive proper pharmacotherapy and psychotherapy are reported to be in complete remission [2,3]. This ratio is found to be 28 to 33% in STAR*D study [4]. When response to treatment is evaluated from this perspective, rTMS seems not to be less effective than other pharmacologic agents. In STAR*D study, the remission ratios reached after drug changes and empowerment strategies in the tertiary care, are reported to be 13 to 14% [5]. Regarding response to treatment in rTMS 36.6% remission after one to two failed medication therapies and; 28.9% remission after 3-4 failed medication therapies are reported [6]. All these facts encourage researchers that this treatment should be considered one of the primary care. The term "treatment resistant depression" is used in a certain way for rTMS treatment by some researchers. Its efficiency is tried to be revealed through clinical studies which failed in 1 to 4 pharmacologic treatments. This idea suggests that rTMS should not be one of the primary care options unless a medical requisite is in place such as pregnancy or breastfeeding. General opinion is psychiatry practice is observed to be in this direction. Age is the prominent factor in the predictors of response to rTMS treatment. The reason for this can be cerebral plasticity and excitability of the cerebral tissue which change with the age. Particularly BDNF studies are supportive of this hypothesis. According to some researchers, cognitive-affective symptoms in patients with depression demonstrate a better recovery than somatic symptoms. However, it seems contradictory to those findings that somatic symptoms in some patients with some diseases other than depression, such as panic disorder, psychotic disorder and bipolar disorder, shows recovery with rTMS. Depressive mood, guilt, sleep disorders and less severe depression are assessed to be important response predictors [7,8]. When both, debates on treatment priority of rTMS and debates on predictors of response to treatment, are considered as a whole, two results can be concluded on the priority of rTMS in psychiatric diseases; one is the patients who cannot receive pharmacologic treatment, and the other one is the patients' treatment resistant symptoms, such as somatic symptoms.

References

[1] Wang YM, Li N, Yang LL, et al. Randomized controlled trial of repetitive transcranial magnetic stimulation combined with paroxetine for the treatment of patients with first-episode major depressive disorder. Psych Res. 2017;254:18–23.

[2] Bauer M, Whybrow PC, Angst J, et al. World federation of societies of biological psychiatry (WFSBP) guidelines for biological treatment of unipolar depressive disorders, part 1: acute and continuation treatment of major depressive disorder. World J Biol Psychiat. 2002;3:5–43.

[3] Rush AJ, Kraemer HC, Sackeim HA, et al. Report by the ACNP Task force on response and remission in major depressive disorder. Neuropsychopharmacology. 2006;31:1841–1853.

[4] Rush AJ, Trivedi MH, Wisniewski SR, et al. Acute and longer-term outcomes in depressed outpatients requiring one or several treatment steps: a STAR*D report. Am J Psychiat. 2006;163:1905–1917.

[5] Wisniewski SR, Rush AJ, Nierenberg AA, et al. Can Phase III trial results of antidepressant medications be generalized to clinical practice? A STAR*D report. Am J Psychiat. 2009;166:599–607.

[6] Lisanby S, Husain MM, Rosenquist PB, et al. Daily left prefrontal repetitive transcranial magnetic stimulation in the acute treatment of major depression: clinical predictors of outcome in a multisite, randomized controlled clinical trial. Neuropsychopharmacology. 2009;34:522–534.

[7] Brakemeier E-L, Luborzewski A, Danker-Hopfe H, et al. Positive predictors for antidepressive response to prefrontal repetitive transcranial magnetic stimulation (rTMS). J Psychiat Res. 2007;41:395–403.

[8] Fitzgerald PB, Hoy KE, Anderson RJ, et al. A study of the pattern of response to rTMS treatment in depression. Depress Anxiety. 2016;33:746–753.

[Abstract: 0574] [Anxiety disorders]

Childhood anxiety disorders: into adolescence and adulthood, diagnosis and difficulties in treatment

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ABSTRACT
This sessions primary objective is to review the knowledge of Childhood Anxiety Disorders in terms of diagnosis and treatment. In addition changes that occur into adolescence and adulthood and their results will be discussed. A Literature review for childhood anxiety into adolescence and adulthood, treatment strategies and assessment problems was conducted. Anxiety Disorder is among the most common problems observed in children but there are difficulties in diagnosis. Difficulty of children to express anxiety, definition of anxiety by families and the importance they place on the diagnosis makes it challenging to diagnose . Types of Anxiety Disorders, evaluated under the Anxiety Disorders topic, must be differentiated. Furthermore differential diagnosis of other diseases presenting with similar symptoms is essential. Starting age of Anxiety Disorders is critical as it determines the reflection of the disorder in adolescence and adulthood. DSM 5 has changed the classification of Anxiety Disorders for children, adolescents and adults. It is crucial that treatment suitable for the problem and the person is selected. Anxiety disorders are common in children and adolescents but they are also commonly overlooked. Data clearly demonstrates that treatment can significantly lower distress and improve functioning. In addition it plays a crucial role for preventing adulthood disorders

KEYWORDS
Childhood; Adolescent; Adulthood; assessment; treatment

References

[1] Rynn MA, Walkup JT, Compton SN, et al. Child/adolescent anxiety multimodal study: evaluating safety. J Am Acad Child Psy. 2015;54(3):180–190.
[2] Ginsburg G, Becker-Haimes E, Keeton C, Kendall PC. Results from the child/adolescent anxiety multimodal extended long-term study (CAMELS): primary anxiety outcomes. J Am Acad Child Psy. 2018;57(7):471–480.
[3] Zehgeer A, Ginsburg GS, Lee P, et al. Pharmacotherapy adherence for pediatric anxiety disorders: predictors and relation to child outcomes. Child Youth Care For. 2018;47(5):633–644.

[Abstract: 0708] [Psychopharmacology]

Is hypothalamic dysregulation a main cause of olanzapine-induced weight change?

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ABSTRACT
Olanzapine; It is an atypical antipsychotic agent which is used frequently in the psychiatric clinic, where patients are most adaptable and used for the longest period. Although it has been shown to cause a lower level of extrapyramidal symptoms than typical antipsychotics, it has other side effects that significantly affect the quality of life of the patient. The most important of these side effects is weight gain. Olanzapine is one of the antipsychotics that causes the most weight gain. As is known, control of body weight in humans is carried out by the hypothalamus in the brain. The Ventro Medial Nucleus, Lateral Hypothalamic Area, Arcuat Nucleus, Para Ventricular Nucleus, Dorso Medial Nucleus, Suprachiasmatic Nucleus and Orbito Frontal Cortex in the hypothalamus are the main areas that play a role in the regulation of food intake. Especially arcuate nucleus of the hypothalamus that are synthesized and released neuropeptides from POMC, CART, agrp and NPY; human habits, body weight and energy metabolism are known to regulate. Various serotonergic receptors were found on these neurons in the arcuate nucleus of the hypothalamus. It is known to have 5HT2c receptors on POMC neurons in ARC. It is known that 5HT1b receptors are present on the cell body of NPY and AgRP neurons. There are also 5HT1b receptors on the axons with GABA ergic synapses in which the NPY and AgRP neurons interfere with POMC neurons [2]. With serotonergic receptors affected by olanzapine and serotonergic receptors which are effective in regulating food intake, important researches have been made on serotonergic systems in order to elucidate the mechanism of olanzapine induced weight gain [1]. The physiological mechanisms underlying the weight gain of olanzapine are not yet fully elucidated. However, in recent years, the main role in olanzapine-related weight gain is thought to be the central stimulation of appetite-enhancing mechanisms. With antagonist effects of olanzapine on serotonin receptors in central sites, the normal functioning of the ARC in patients is impaired and the mechanism begins to reverse. In particular, it is thought that weight gain occurs due to the change in the balances of serotonin 5HT2c and 5HT1b receptors on the antagonist effect and the release of the neuropeptide. Further studies have been deemed necessary to evaluate the changes in the plant in order to clarify the process that started with weight gain and led to degradation in many metabolic areas. Fully elucidating the mechanisms that underlie

KEYWORDS
Olanzapine; neuropeptide; weight gain; hypothalamus

drug-induced weight gain is of great importance in terms of combating the weight gain side effects that can cause serious consequences. Detecting the receptors, we will target in the fight against weight gain will be a guide in the development of new drugs that do not have this side effect.

References

[1] Ak M, Sezlev D, Sutcigil L, Akarsu S, Ozgen F, Yanik T. Psychoneuroendocrinology. The investigation of leptin and hypothalamic neuropeptides role in first attack psychotic male patients: olanzapine monotherapy. 2013;38 (3):341–347.

[2] Reynolds GP, Kirk SL. Metabolic side effects of antipsychotic drug treatment–pharmacological mechanisms. Pharmacol Ther. 2010;125(1):169–179.

[3] Parsons B, Allison DB, Loebel A, et al. Weight effects associated with antipsychotics: a comprehensive database analysis. Schizophr Res. 2009;110:103–110.

[Abstract: 0720] [Psychopharmacology]

Overview of the effect of SSRIs on central eating regulation from the essitolapram model

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ABSTRACT

Psychiatric patients have higher morbidity and mortality and lower life expectancy than the general population. The physical health of these patients is impaired due to drug side effects, lack of physical activity, poor nutrition, smoking, alcohol and substance use. Obesity and cardiovascular diseases are among the most important physical health problems that reduce life expectancy. A significant number of these patients dies from cardiovascular or metabolic complications. Although obesity may occur in psychiatric patients due to mental illness, it may also occur due to psychotropic use. Psychotropics such as antipsychotics, mood stabilizers and antidepressants are widely known to cause weight gain. Weight gain may lead to medical comorbidity and relapse of mental illness, which complicates patient compliance. Although it is known that antidepressants cause less weight gain than antipsychotics and mood stabilizers, they may cause more problems due to the more widespread use of these drugs. There are not enough studies about weight gain related to antidepressant use. At the present time, antidepressants are widely prescribed. However, clinical success is not at this rate. One of the most important reasons for this condition is the non-compliance of the patients. Some of the factors that impair treatment compliance are sedation and weight gain. However, the mechanism underlying antidepressants causing weight gain has not yet been fully elucidated. Body weight is affected by various factors in many ways in the light of our current data, it can be said that body weight is determined by the interaction of environmental factors and biological systems regulating the balance of appetite and energy. Psychotropic drugs seem to influence neurobiological mechanisms that regulate appetite and energy balance as an environmental factor. In this study, the effects of escitalopram, which is commonly used today, on weight gain and metabolic risk factors such as BMI, waist circumference, hip circumference, body measurements, lipid panel and glucose-insulin system were investigated. In order to investigate the underlying mechanism of these metabolic side effects, the levels of some of the central and peripheral neuropeptides that regulate the food intake and energy balance system were measured and their relationship with these metabolic parameters was investigated. For this purpose, 45 patients who were admitted to Necmettin Erbakan University Meram School of Medicine Psychiatry Clinic and decided to receive escitalopram treatment were included in the study and completed with 30 patients. At the beginning of the treatment and at the 12th week controls, the necessary measurements were made, and blood samples were taken, and biochemical tests were performed. The data were entered into the IBM SPSS 23.00 program and analyzed statistically. As a result of the study, it was observed that weight and waist and hip circumferences were significantly increased in patients using escitalopram. Lipid panel and fasting blood glucose levels were not significantly different. There was a low but significant change in neuropeptide levels. As a result, it can be said that escitalopram causes significant weight gain. This seems to be through appetite-related central and peripheral neuropeptides. The results of this study are largely consistent with previous studies. This study has limitations such as low number of patients and lack of control group. Thus, large scale studies are needed in this field.

KEYWORDS

Escitalopram; weight gain; neuropeptides

[Abstract: 0680] [Neuroscience: Neuroimaging-Genetic Biomarkers]

Normal physiology of eating behavior

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ABSTRACT

In human physiology, there are many factors that regulate food intake.

Hypothalamus Regions Involved in Food Intake

VentroMedial Nukleus (VMN). The ventromedial nucleus neurons known as the center of toughness are sensitive to glucose and have the task of regulating glucagon release. It inhibits the nutrition center by activating the VMN following eating behavior.

Lateral Hypothalamic Area (LHA). The lateral hypothalamic field acts primarily as a center of hunger. LHA and VMN are mutually correlated in the regulation of food intake and energy metabolism.

Arcuat Nukleus (ARC). NPY, AgRP neurons are orexigenic, POMC and CART neurons are anorexigenic.

Stimulation of POMC neurons reduces food intake and increases energy consumption. The stimulation of NPY, AgRP neurons increases food intake.

Paraventricular Nucleus (PVN). NPY, AgRP and POMC, sourcing from ARC; MCH sourcing from CART neurons and LHA takes the nutrient signals from the orexin neurons. The effect of leptin increases the release of MSH (POMC derivative) in anorexigenic neurons. The extensions of these neurons reach PVN in the hypothalamus. By stimulating the MSH receptor, melanocortin-4 receptor (MC4R), energy expenditure is increased while decreasing nutrient intake.

Dorsomedial Nucleus (DMN). DMN is important in the regulation of food intake with circadian rhythm.

Suprachiasmatic Nucleus (SCN). It is important in the rhythm of daily food intake in SCN. SCN inhibits LHA activity by stimulating VMN leading to suppression of light stimulation.

Orbitofrontal Cortex (OFC). It is the area related to the detection of nutrients and the control of impulses.

Neurochemical Peptides Regulating Food Intake

Orexigenic Peptides

Neuropeptid Y (NPY). In general, known NPY1, NPY5 receptors by increasing appetite NPY2, NPY4 receptors is related to reducing appetite. However, the orexigenic effect is known to be significant because the NPH5 receptor amount is more intense than other receptors in LHA.

Agouti-Related Peptide (AgRP). AgroP, an orxigenic peptide, increases food intake and search behavior. Leptin reduces the release of AgRP. Increased AgRP was associated with obesity.

Orexin A-B (Hypocretin 1-2). In the case of fasting, the release of Orexin A-B is increasing. Orexigenic neurons are stimulated when blood glucose falls, and the stomach is emptied, resulting in a nutrient-enhancing effect.

Melanin Concentrator Hormone (MCH). The MCH-containing neuron bodies emerge from the LHA known as the feeding center. By administration of leptin, MCH is reduced and food intake is reduced.

Ghrelin. It is mainly an orecsiogenic peptide released from the stomach. It produces the orexigenic effect via NPY and AgRP secreted from ARC.

Anorexigenic Peptides

Leptin. It is mainly secreted from adipose tissue. The main role in the body is to regulate food intake and energy metabolism with negative feedback on the brain and to prevent the development of obesity.

Proopiomelanocortin (POMC). POMC is the pioneer of MSH, the main regulator of energy balance.

Cocaine Amphetamine Associated Peptide (CART)

It was determined that food intake decreased by issuing CART.

Cholecystokinin (CCK). In food intake, endogenous CCK reduces the amount of food.

Glucagon-Like Peptide (GLP-1). GLP-1 is another gut hormone released in response to food intake. While delaying gastric emptying with cholecystokinin-like effect, it provides stimulation of glucose-induced insulin synthesis and secretion and suppression of glucagon secretion.

Mediators in Food Intake. Dopamine, serotonin, noradrenaline, acetylcholine and histamine are also mediators of food intake.

KEYWORDS

Eating Behavior; Normal Physiology; Food Intake; Neurochemical Peptides

[Abstract: 0729] [Forensic Psychiatry]

Considerations in drug use in children and adolescents: forensic psychiatric approach

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ABSTRACT

Nowadays, the sensitivity to medical malpractice and lawsuits against physicians have increased, and it has also become frequently mentioned in written and visual media. A lawsuit against a physician for occupational responsibility, even if unsuccessful; can result in social and occupational stigma, emotional stress and loss of performance. For these reasons, it is inevitable for physicians to review possible legal consequences during medical practice. Social prejudices and populist rhetoric about drug treatment in child and adolescent psychiatry cause problems in professional practice, and yet they are at risk, although the frequency of the case is small. The use of psychotropic drugs in children and adolescents is limited and FDA approval is not available for many agents. At the same time, it is seen that the use of psychotropic drugs for off-indication is quite frequent. Contraindications and adverse effects that may occur after the use of the drug should be considered.

Families are included in the treatment process in children and adolescents. Therefore, both the child and his family should be informed about the effects of treatment, adverse effects and possible alternative therapies, informed consent should be obtained from both the child and his family according to age and developmental level. It should be kept in mind that the recording of the findings of the examination and the information shared with the family will be protective in the possible legal process.

KEYWORDS

Child; drug use; malpractice; forensic; protection

[Abstract: 0753] [Psychopharmacology]

Managing of common adverse effects of antipsychotics

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ABSTRACT

Antipsychotics are the first-line treatment for psychotic disorders and some are also approved for treatment of bipolar disorders, generalized nonpsychotic anxiety, adjunctive therapy for major depressive disorder, hyperactivity, severe childhood behavioral problems, irritability associated with autism spectrum disorder and Tourette syndrome. In addition, these are prescribed off label for other conditions. The utility of these drugs is hampered by their adverse effects. Each antipsychotic medication has a unique adverse effect profile, which affects individuals differently and the adverse effects of antipsychotics range from minor tolerability issues to life threatening situation.

Neurological effects

Dystonias are involuntary contractions of antagonistic muscle groups. The best management strategy of dystonias is prevention with anticholinergic medication. Benztropine or biperiden is effective in dystonias prophylaxis and intramuscular anticholinergics or antihistaminics (diphenhydramine) are indicated in acute dystonic reactions. Benzodiazepines and aripiprazole are also thought to be effective in treating dystonias

Akathisia refers to a feeling of restlessness and tension that usually compels the sufferer to near-constant motion. Centrally-acting beta-adrenergic antagonists (propranolol), benzodiazepines and anticholinergics have been used for akathisia. The antidepressant mirtazapine has shown propranolol equivalency in several trials. 5-HT2A/C antagonists mianserin and ritanserin, 5-HT1B/1D agonist zolmitriptan and cyproheptadine which has 5-HT2 antagonism have shown efficacy in small studies.

Parkinsonism consists of a number of drug-induced symptoms resembling Parkinson's disease, such as mask-like facies, resting tremor, cogwheel rigidity, shuffling gait, and psychomotor retardation (bradykinesia). Anticholinergic medications have been used for parkinsonism and amantadine may use in patients who don't tolerate anticholinergic medications.

Tardive dyskinesia consists of choreoathetoid movements of the tongue, lower face and extremities. First treatment strategy is reduction in the dose of antipsychotic and switching to an antipsychotic with a low risk, such as clozapine, aripiprazole or quetiapine. Inhibitors of VMAT2

KEYWORDS

Antipsychotics; adverse effects; akathisia; parkinsonism; metabolic effects; obesity

valbenazine and tetrabenazine, benzodiazepines and botulinum toxin injections have been used to control symptoms of tardive dyskinesia.

Metabolic effects

Many antipsychotics are associated, to variable degrees, with weight gain, hypertension, and adverse effects on lipid and glucose metabolism. If these effects occur, lifestyle modifications and switching to an antipsychotic with lower risk are recommended for the first treatment strategy. Statins are used to treat dyslipidemias, and antihypertensive medications are used to treat hypertension. Medications including metformin, topiramate, and adjunctive aripiprazole have shown efficacy for reducing antipsychotic-induced weight gain. Recently approved weight loss drugs including lorcaserin, bupropion, naltrexone and liraglutide have not been tested specifically for antipsychotic-induced weight gain. Clinical trials of atomoxetine, dextroamphetamine, famotidine, and nizatidine have failed to show benefit.

Anticholinergic effects

Anticholinergic advers effects of antipsychotics include dry mouth, constipation, urinary hesitancy, visual disturbance, tachycardia and cognitive impairment. Decreasing the antipsychotic dose is the first treatment strategy, because anticholinergic effects are dose-related. Switching to a less anticholinergic medication is another option.

References

[1] Goldberg, JF, Ernst CL. Managing the side effects of psychotropic medications. American Psychiatric Pub, 2018.
[2] Cooper SJ, Reynolds GP, Barnes T, et al. BAP guidelines on the management of weight gain, metabolic disturbances and cardiovascular risk associated with psychosis and antipsychotic drug treatment. J Psychopharmacol. 2016;30(8):717–748.
[3] Stroup, T. S., & Gray, N. Management of common adverse effects of antipsychotic medications. World Psychiatry, 2018;17(3):341–356.

[Abstract: 0690] [Psychopharmacology]

Approach to management of common side effects associated with antidepressants

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ABSTRACT

Antidepressant drugs are widely used to treat not only psychiatric disorders but also other medical problems in children and adolescents. Side effects are defined as undesirable symptoms caused by medical treatment. Side effects are common with antidepressant drugs and pose substantial challenges to treatment. They frequently precipitate early discontinuation or the usage of subtherapeutic doses of antidepressants, therefore, generate obstacles to reaching remission from depression and anxiety, in addition to preventing relapse. Moreover, they may hinder future treatment attempts and result in untreated long-term psychiatric disorders. First of all, caregivers and the children must be informed about the potential risks and side effects, as well as the benefits of the pharmacotherapy. Consent and assent for initiation of medication should be given by them. Secondly, starting with low doses and slow pacing for increasing doses are essential while treating children with psychotropic medication. Additionally, appropriate management of side effects, which may prevent early drop out of pharmacotherapy comprises considerate drug choice, the anticipation of frequent and/or infrequent though severe side effects, as well as adding of suitable adjunctive treatments to deal with emergent symptoms. After black box warning issued by the US Food and Drug Administration about usage of antidepressant drugs in youngsters, epidemiological data and clinical trials did not show enhanced risk of suicidality with antidepressant treatment. However, black box warning is not displaced from the prospectus of the drugs. Consequently, clinicians should inform the parents about suicidality risk associated with depression and observe patients closely for any signs of suicidality across the duration of the therapy. Another important issue of antidepressant treatment in children is antidepressant induced mania (AIM) and antidepressant induced activation (AIA). AIM can be foreseen by taking a watchful history for previous AIM, age of onset of depressive symptoms, psychosis, and hereditary background for mood disorders. Clinician should also monitor closely 'red flags' in response to antidepressant therapy, for example changes in sleep, irritability, and psychosis. Thus, in children with rapid onset, psychotic features, psychomotor retardation, a family history of mood disorders, and a history of AIM, clinician may prefer another treatment option or adding a mood stabilizer as a prophylaxis. On the other hand, AIA is mostly seen among preadolescent children in the first weeks of the treatment. Children with intellectual disability and autism spectrum disorders are more susceptible for AIA and decreasing dose of the antidepressant may be useful. Other common side effects are somnolence, insomnia and nausea. Somnolence and insomnia may be managed by assessment

KEYWORDS

Antidepressant; side effect; children; adolescent; suicidality

of sleep habits, counseling on sleep hygiene and changes in dosing schedule. Management of nausea includes taking medications with food or the use of divided dosing. In this presentation, management of important side effects of antidepressant treatment in children and adolescents will be discussed.

References

[1] Kelly K, Posternak M, Jonathan EA. Toward achieving optimal response: understanding and managing antidepressant side effects. Dialogues Clin Neurosci. 2008;10(4):409–418.

[2] Safer DJ, Zito JM. Treatment-emergent adverse events from selective serotonin reuptake inhibitors by age group: children versus adolescents. J Child Adolesc Psychopharmacol. 2006;16(1–2):159–169.

[3] Isacsson G, Rich CL. Antidepressant drugs and the risk of suicide in children and adolescents. Pediatric Drugs. 2014:16(2);115–122.

[Abstract: 0667] [Psychopharmacology]

Approach to management of common side effects associated with psychostimulants

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ABSTRACT

Attention-deficit/hyperactivity disorder (ADHD) is the most common neuropsychiatric disorder in school-age in children and adolescents. Methylphenidate is the most commonly used drug prescribed for ADHD; it has been used in practice for more than 50 years [1]. Methylphenidate appears to have positive effect on reducing the core symptoms of hyperactivity, impulsivity, and inattention in children and adolescents with ADHD. It is licensed for use in children aged six years and older[1]. Methylphenidate dose varies from patient to patient. The dose needs to be titrated individually in order to maximize benefits and minimize potential adverse events. ADHD medication appears to be discontinued in 13% to 64% of patients from all age groups [2]. The most commonly reported adverse events associated with methylphenidate are headache, sleep problems, fatigue, and decreased appetite. Methylphenidate can also impair both children's height and weight. Serious adverse events, such as psychosis and mood disorders, are reported to affect some of children treated with methylphenidate[1]. Although an association between the use of stimulants and sudden unexplained death among children and adolescents has been described in some studies further research is needed to determine whether these deaths are related to methylphenidate. Tremor, tics and dyskinetic disorders are also described in the literature after methylphenidate usage [3]. Dyskinesias recently started to attract attention with the increased use of methylphenidate. In this presentation, the most common side effects associated with methylphenidate use and the intervention to these effects will be reviewed.

KEYWORDS

Methylphenidate; side effect; Attention-deficit/ hyperactivity disorder

References

[1] Storebø OJ, Pedersen N, Ramstad E. et al. Methylphenidate for Attention-deficit/ hyperactivity disorder (ADHD) in children and adolescents - assessment of adverse events in non-randomized studies. Cochrane Database of Systematic Reviews. 2018;5:Art. No.: CD012069

[2] Adler LD, Nierenberg AA. Review of medication adherence in children and adults with ADHD. Postgraduate Medicine. 2010;122 (1):184–191.

[3] Selcen Yaroglu Kazanci, Mahmut Cem Tarakcioglu, Lida Bulbul, Neslihan Ozkul Saglam, Sami Hatipoglu. Should We Continue Methylphenidate Treatment Despite Orofacial or Extremity Dyskinesias? Klinik Psikofarmakoloji Bülteni-Bulletin of Clinical Psychopharmacology. 2015;25(4):399–402.

[Abstract: 0742] [Non-biological treatments]

Definition of neurofeedback and use in child and adolescent psychiatry

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ABSTRACT
It was learned that it was possible to rearrange brain wave patterns in the late 1960s and 1970s. This brain wave training is called EEG-biofeedback. NF is based on the hypothesis that the central nervous system can regulate its own function, such as the regulation of many physiological reactions. The aim is to consciously regulate brain wave activity. The idea that NF can be used in clinical practice has been shown that cats conditioned to produce a specific EEG frequency (SMR) have a high seizure threshold when exposed to the convulsant agent methylhydrazine. Although the use of NF in children began in the 1970s, it has been observed that it has become more common in clinical practice in the last decade. Especially, Attention-deficit/ hyperactivity disorder is the main disorder in which NF is used as a treatment method in children. Several studies have shown slow wave activity (4–8 Hz) in ADHD, especially in the posterior regions of the brain and / or a decrease in alpha and / or beta activity during attention watch as well as resting EEG. This neurophysiological deviation typically provides a foresight for θ/β education in children with ADHD. Thus, direct training and reconfiguration of the electrical activity patterns in the brain through neurofeedback; provides us with additional options for treatment in diagnoses such as ADHD, learning disability, stroke, head injury, deficits after brain surgery operations, uncontrolled epilepsy, cognitive decline with age, depression, anxiety disorders, OCB, and autism. Neurofeedback can also be used to achieve higher performance in normal individuals, athletes and managers. These training sessions help the person to gradually change and reconstruct brain wave patterns. For example, in some people it is necessary to increase the speed and amplitude of waves in some regions of the brain, while others may need to learn to reduce speed and amplitude. There are also different forms of neurofeedback. These; Slow Cortical Potentials Training, the low-energy neurofeedback system, Hemoencephalography, Live Z-Score Neurofeedback Training, LORETA Neurofeedback Training. Mild side effects may occur during neurofeedback. For example, sometimes you may feel tired or anxious, may feel headaches; or may feel restless. Most of these side effects passes shortly after the training session. It can be assumed that the positive results from neurofeedback originate from a combination of the effects of placebo and neurofeedback's own positive effect. Although it has been shown to be effective in some studies, controlled research is needed to apply Neurofeedback to various diagnoses in many other areas. Although early studies report positive results for EEG-biofeedback effectiveness but the results from the current studies and meta-analyzes that have been good designed bring suspicion to this. In this presentation, it is aimed to make definition of NF and to review the usage areas in child and adolescent psychiatry.

KEYWORDS
Neurofeedback; child and adolescent; EEG-biofeedback

[Abstract: 0723] [ADHD]

Neurofeedback and attention-deficit/ hyperactivity disorder

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ABSTRACT
Attention-deficit/ hyperactivity disorder (ADHD) with symptoms of attention deficit, hyperactivity and impulsivity is one of the most common psychiatric disorders in youth. The most effective treatment of this neurobiological disorder is medication. Currently, the first line of treatment for children and adolescents diagnosed with ADHD from age 6 years onward consists of psychostimulant medication, which is primarily methylphenidate (MPH). There is substantial evidence for MPH improving functioning on symptom domains of attention, impulsivity and social behavior, with high effect sizes ranging from 0.63 to 0.85. Some limitations of psychostimulants, however, are the short duration of treatment effects, achieving no or only partial response in some patients, the lack of achieving long-term remission and short-term adverse effects such as fatigue, nausea and loss of appetite. Further issues include the significant portion of parents who have nondrug treatment preferences and the compliance of stimulants. Long-term adverse effects are yet to be identified, but there is increased awareness about their potential for adverse cardiovascular effects and suppression of growth in children. Also other treatment strategies are suggested with addition of medication to improve symptoms and overall functioning. Neurofeedback (NF) is referred to as a "possibly efficacious" treatment in the current evidence-based reviews; therefore, more research is needed to determine its effects especially in combination with other treatments. NF has been one of nonpharmacological ADHD treatment strategies since early 1970s. NF, or electroencephalogram (EEG) biofeedback, is a relatively new, noninvasive approach for treating multiple brain-related conditions. Epilepsy has been one of the first

KEYWORDS
Neurofeedback; adhd; randomized trials; efficacy; cortical activation

therapeutic applications of NF that has been subject to extensive NF research. More conditions in which NF is being used include ADHD, learning disabilities, strokes, head injury, insomnia, depression, obsessive–compulsive disorder and drug addiction. NF attempts to normalize the disrupted brain waves that are associated with these conditions by means of repeated training based largely on operant conditioning. Although the overall working mechanisms of NF are partially explained by operant conditioning principles, the implications of how such training may influence biological processes at the hormonal or cellular level remains not fully understood. The assumption is that brain waves reflect neural functions, and that training in brain waves may improve neural functions, subsequently leading to improvements in ADHD symptom domains and behavior. NF is thus a method that assists subjects to control their brain waves consciously. The most frequently used type of NF used to treat ADHD is frequency/power NF, which is used to change the amplitude or speed of specific brain waves in particular brain locations, such as the frontal or parietal lobes. Another type of NF that is sometimes used to treat ADHD is slow cortical potential (SCP)-NF, which improves the direction of SCPs. Other types of NF include hemoencephalographic NF, functional MRI NF, low-resolution electromagnetic tomography and near-infrared spectroscopy NF, which are used for the treatment of various disorders. Although there have been preliminary studies which the findings show that neurofeedback is effective, current studies well-designed and meta-analyzes reported the efficacy of NFB is unclear.

References

[1] Razoki B. Neurofeedback versus psychostimulants in the treatment of children and adolescents with attention-deficit/hyperactivity disorder: a systematic review. Neuropsychiatric disease and treatment. 2018;14:2905–2913.

[2] Yan L, Zhang J, Yuan Y, Cortese S. Effects of neurofeedback versus methylphenidate for the treatment of attention-deficit/hyperactivity disorder protocol for a systematic review and meta-analysis of head-to-head trials. Medicine. 2018;97(39):e12623.

[3] Hudak J, Rosenbaum D, Barth B, Fallgatter AJ, Ehlis AC. Functionally disconnected: A look at how study design influences neurofeedback data and mechanisms in attention-deficit/hyperactivity disorder. PloS one. 2018;13(8):e0200931.

[Abstract: 0670] [Autism]

Neurofeedback and autism spectrum disorders

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ABSTRACT

Autism Spectrum Disorder (ASD) is a neurodevelopmental disorder that occurs in the early developmental period of life and is characterized by deficits in social communication and interaction and limited, repetitive behaviors or interests [1]. ASD is a common disorder and affecting 1 in 59 children [2], with a significant impact on quality of life of child and family. Unfortunately, there is no definitive cure for ASD. Although no evidence-based cure exists for ASD, psychosocial and pharmacologic interventions can improve the quality of life of children with ASD and their families. Therefore parents of children with ASD may seek out complementary and alternative therapies in the treatment of ASD. However, most of these interventions lack scientific evidence, or have been proven to be ineffective [3]. Among alternative treatment approaches, there is increasing interest in the use of non-invasive treatments such as neurofeedback in recent years. Neurofeedback, also called EEG biofeedback, is a computerized method based on tracking electrical activity of the brain and giving feedback about it. It represents a learning process that enables the person to observe, control and change his/her own brain activity. The goal of neurofeedback is to effect brain wave activity to reorganize electrical activity during desired behaviors and improve behavioral or cognitive process. In some studies, it has been investigated the usage of neurofeedback in children with ASD and have shown its positive efficiency on attention and behavior. Although some investigators have stated positive effect of neurofeedback in autism, such as social interaction problems and communication deficits, these studies have significant methodological limitations including small sample size and short duration. Another important limitation to keep in mind that neurofeedback can be used children with ASD whose IQ above 70 (high- functioning ASD). These restriction does not allow for the generalization of the findings of studies. The existing evidence does not support to use neurofeedback protocols that inhibit theta and reward beta activity or sensorimotor rhythm, as a treatment that can be recommended for ASD. Findings from studies suggest that neurofeedback may hold promise for the treatment of ADHD - like symptoms rather than improvement in core symptoms of ASD. Further clinical researches should focus on possible effect of neurofeedback on autism and other neurodevelopmental disorders.

KEYWORDS

Neurofeedback; Autism spectrum disorders; treatment; children

References

[1] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. V ed. Washington (DC): American Psychiatric Association; 2013.

[2] Baio J, Wiggins L, Christensen DL, et al. Prevalence of Autism Spectrum Disorder Among Children Aged 8 Years - Autism and Developmental Disabilities Monitoring Network, 11 Sites, United States, 2014. Morbidity and mortality weekly report. Surveillance summaries. Washington, (DC: 2002). 2018;67(6):1–23.

[3] Rossignol DA. Novel and emerging treatments for autism spectrum disorders: a systematic review. Ann Clin Psychiatry. 2009;21:213–236.

[Abstract: 0712] [Others]

Neurofeedback and other psychopathologies

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ABSTRACT

Through the late 1960s, it was learned that brain wave patterns could be consciously controlled. Such training of brain waves has been identified as neurofeedback (NF). Main purpose of NF is to regulate brain wave activity, in a conscious manner. Because, it has long been observed that brain activity was different in certain conditions than normally expected, and it has been suggested that this might be linked to certain clinical symptoms. Research regarding clinical use of NF have initially focused on animals. Clinical NF was defined following the observation of higher seizure threshold among cats that had been conditioned to generate a specific EEG frequency, in the face of being exposed to a convulsant agent, such as methylhydrazine. Later on, this procedure was tested on humans, with the aim of improving clinical response and outcome in treatment-resistant epilepsy. Although started in 1970s, use of NF for clinical purposes in children has become much more common within last 10 years. Attention-deficit/ hyperactivity disorder (ADHD) remains as the primary disorder where NF has been mainly used as a treatment option in children. A review of literature on this subject has revealed the first case report addressing the efficacy of NF treatment in ADHD, published in 1976. Many controlled studies and metanalysis following the aforementioned paper have reported discrepancies regarding the outcome of treatment with NF in ADHD, that yet still remains as a questionable entity. Efficiency of NF as a treatment option has also been evaluated in Autism Spectrum Disorder (ASD). Other than ADHD and ASD, NF has also been used to treat a number of other psychiatric conditions such as Specific Learning Disorder (SLD), Intellectual Disability, Enuresis Nocturna, Abuse and Neglect of Childhood. Observed increases in theta activity along with decreased alpha activity among children diagnosed with SLD has raised the possibility that NF might be effective as a treatment option for these children. Preliminary studies on the subject have been carried out by Fernandez and colleagues (2003, 2006, 2007). However the lack of randomization, no group comparison being available, unspecified nature of blindness, and small sample size have been listed as significant methodological concerns. Inadequacy of controlled studies addressing efficacy of NF in the treatment of NF and small sample sizes suggest a lack of enough evidence to recommend use of NF as a treatment option in children with SLD. Therefore, NF should not be used as an alternative treatment approach for children with SLD and should not replace evidence based educational interventions. Even though results collected from the studies that assess efficiency of NF treatment in children with ADHD and SLD suggest an increase in posttreatment IQ scores, limited number of studies that evaluate efficiency of NF in intellectual disability makes it even harder to recommend NF treatment in this specific subpopulation. With this presentation, we have aimed to review other psychiatric conditions in child and adolescent psychiatry, where NF has been used so far.

KEYWORDS

Abuse and neglect; enuresis nocturna; intellectual disability; learning disorder; neurofeedback

References

[1] Lubar JF, Shouse MN. EEG and behavioral changes in a hyperkinetic child concurrent with training of the sensorimotor rhythm (SMR): a preliminary report. Biofeedback Self Regul. 1976; 1:293–306.

[2] Fernandez T, Herrera W, Harmony T, et al. EEG and behavioral changes following neurofeedback treatment in learning disabled children. Clin Electroencephalogr. 2003;34:145–152.

[3] Fernandez T, Harmony T, Fernandez-Bouzas A, et al. Changes in EEG current sources induced by neurofeedback in learning disabled children. An exploratory study. Appl Psychophysiol Biofeedback. 2007;32:169–183.

[Abstract: 0789] [Others]

COURSE 5

An update on the scope, challenges and controversies in the forensic psychiatry practice

Moderator: Mustafa Solmaz (TAP)

Forensic psychiatric expert witnessing in criminal and civil law

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ABSTRACT

Identifying the boundaries and context of the interaction between psychiatry and the law is a subject of a long-standing puzzlement for the members of both professions. Nevertheless, it is well known that there are certain paths in which both civil and criminal legal systems rely on psychiatric input. Psychiatrists have been increasingly aware of the need for expertise in legal aspects of psychiatric practice and in satisfying the legal systems' needs for psychiatric participation in adjudicating matters involving mental health. Indeed, such a necessity has led to the fact that forensic psychiatry has become one of the most acknowledged and respected psychiatric subspecialties in particular countries in recent decades. Forensic psychiatry primarily covers the field of expert witnessing; it also deals with the patients' clinical needs. Mens rea is the mental element of an offense, and psychiatric disorders have the potential to influence the competency or capacity to form any particular intention or behavior that can lead to a crime. Therefore, psychiatrists are frequently asked to evaluate a defendant's mental state at the time of the offense to determine the required mens rea that is related to the crime. In different countries, psychiatrists are involved in various stages in law systems. For instance, assessment for insanity defense (or competency assessment for criminal responsibility) is one of the vital parts of forensic work in Mainland Europe countries, including Turkey, while very few cases of insanity come to the courts in Anglo-American law. On the other hand, in the United Kingdom, if there is a suspicion of a presented mental disorder of the offender that is thought unfair to proceed with the trial, psychiatrists are invited to assess an individual's fitness to plead (competence to stand trial in the United States). Clinicians are needed to indicate whether a defendant has sufficient understanding and cognition to comprehend the purpose of trial proceedings or to defend him/herself in front of the court. Although forensic psychiatry usually deals with the assessment and management of mentally disordered offenders and other patients with mental disorders who are, or have been potentially or actually violent, civil legislations also occasionally require psychiatric testimony. Civil law which relies heavily upon common law is the term used for the law dealing with disputes between individuals or organizations. Psychiatrists become involved in civil law on an occasional basis which usually requires a detailed clinical evaluation for judgment and decision-making abilities. Psychiatrists may be asked to comment on the mental capacity or state of mind of a patient or individual in relation to a contract or statement, to consider whether a particular act or omission committed by a defendant has caused a psychiatric disorder, or to comment a patient's requisite for authorization of a legal representation or a legal supervision in order to employ official proceedings. The civil law system used in most parts of the world is quite different. In Turkey, the Turkish Civil Code regulates the issues mentioned above that become subjects of psychiatric expert witnessing. In this course, major principles of forensic psychiatric evaluation in the context of criminal and civil law will be presented with demonstrative case examples.

KEYWORDS

Civil law; criminal law; criminal responsibility; expert witnessing; forensic psychiatry

References

[1] Stone JH, Roberts M, O'Grady J, et al. Faulk's basic forensic psychiatry. 3rd ed. Oxford: Blackwell Science; 1999.

[2] Gunn J, Taylor PJ. Forensic psychiatry: clinical, legal and ethical issues. 2nd ed. New York: CRC Press; 2014.

[3] Simon RI, Gold LH. The American psychiatric publishing textbook of forensic psychiatry. Washington (DC): American Psychiatric Publishing; 2004.

[4] Rogers R, Bagby RM, Chow MMK. Psychiatrists and the parameters of expert testimony. Int J Law Psychiatry. 1992;15(4):387–396.

[5] Binder RL. Liability for the psychiatrist expert witness. Am J Psychiatry, 2002;159(11):1819–1825.

[Abstract: 0780] [Others]

COURSE 5

An update on the scope, challenges and controversies in the forensic psychiatry practice

Moderator: Mustafa Solmaz (TAP)

Involuntary and coercive treatment in mental health care: current legal issues

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ABSTRACT

The relevance of coercive treatment for psychiatry has been underestimated for a long period in the history of this discipline. It is only within the last few decades that it has been viewed as an increasingly important area for clinical and research initiatives. This topic is both complex and sensitive. It is complex as it involves clinical, public health, ethical, and legal issues. Furthermore, it is sensitive as it deals with delicate aspects of human experience and interpersonal relations. Involuntary or coercive treatment of the mentally ill is an essential matter in the context of civil law. It is among the most controversial issues in mental health care and is the subject of ongoing debate among patients, mental health professionals, and a wider public due to its both ethical and legal amorphous characteristics. In Europe and other developed countries, independent mental health laws are in force and regulate involuntary commitment of psychiatric patients that mainly possess a danger to him/herself or the public due to their mental disorder. Mental health laws authorize the psychiatrists to determine a patient's need for involuntary treatment and hospitalization; however, for instance, clinicians' decisions would be challenged and frequently need to be backed by a second opinion or an independent tribunal according to the Mental Health Act in the United Kingdom. In some countries including Turkey, responsible psychiatrists should apply to the civil court for involuntary psychiatric treatment for non-criminal psychiatric patients. This course aimed to provide practical insight in the field of coercive psychiatric treatment with several case illustrations that may help psychiatrists regarding evaluation and treatment processes that are encountered in clinical settings.

KEYWORDS

Coercion; compulsive treatment; dangerousness; forensic order; involuntary treatment; legislation

References

[1] Stone JH, Roberts M, O'Grady J, et al. Faulk's basic forensic psychiatry. 3rd ed. Oxford: Blackwell Science; 1999.

[2] Gunn J, Taylor PJ. Forensic psychiatry: clinical, legal and ethical issues. 2nd ed. New York: CRC Press; 2014.

[3] Salize HJ, Dreßing H, Peitz M. Compulsory admission and involuntary treatment of mentally ill patients-legislation and practice in EU-member states. Cent Inst Ment Heal Res Proj Final Report, Mannheim, Ger. 2002;15.

[4] Kallert TW. Coercion in psychiatry. Current opinion in psychiatry. 2008;21(5):485–489.

[5] Coercive Treatment in Psychiatry: Clinical, Legal and Ethical Aspects. In Kallert TW, Mezzich JE, Monahan J, editors. Sussex: Wiley-Blackwell; 2011.

[6] Uygur N. Forensic psychiatry practical guide. 2nd ed. Psychiatric Association of Turkey [in Turkish]. Ankara: Pelin; 2014. p. 40–72.

[Abstract: 0795] [Others]

Eating disorders in adolescence: the maudsley treatment model

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Speaker: Hakan Öğütlü

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ABSTRACT

Eating disorders are serious disorders that affect adolescents with increasing frequency. Two major subgroups of the disorders are recognized; anorexia nervosa, and bulimia nervosa. They can be associated with significant morbidity and mortality. Anorexia Nervosa (AN) in eating disorders is one of the most serious disorders that seriously threaten life. The incidence of female adolescents is 0.3–0.7%. Mortality rate is reported as 7–18%. In anorexia nervosa, children or adolescents restrict food intake and this restriction causes a significant reduction in body weight. The patient is very scared of gaining weight or getting fat; behaves in a way that makes weight gain difficult, even though he is significantly low in body weight. These behaviors may be self-vomiting, laxative use or excessive exercise. There is a problem with how the patient perceives the body weight or shape. Bulimia Nervosa (BN) is a disorder with recurrent binge eating episodes. Many people can

KEYWORDS

Maudsley; Family Based Therapy; FBT; Anorexia Nervosa; Bulimia Nervosa

eat more food than they can eat. The control over eating is abolished. They do inappropriate behaviors like vomiting, laxative using, exercising to avoid gaining weight after eating. Bulimia patients may not be aware of their condition, because they may be normal weight or overweight, as opposed to anorexic patients. Family-Based Treatment (FBT) is a new treatment modality for eating disorders that was created by combining appropriate methods of family therapy approaches. FBT or Maudsley approach that was developed in England for anorexia nervosa (AN). Eventually spread all over the world. FBT is considered to be the first choice of adolescent AN treatment as evidence-based, although few studies showed that adapted FBT for adolescent bulimia nervosa is an acceptable treatment modality. Treatment success rates of FBT are around 70%. Adolescent is a part of the family in FBT; for this reason interest of parents is very important for treatment success. Parents should respect their children during the treatment, they should be able to look from adolescent's perspective. All problems and disputes in the family should be postponed. The treatment should be the main objective of the family. Parents are temporarily authorized on adolescent to reduce severity of the disease. Once success is achieved, parents may become able to control adolescent's eating behavior. FBT consists of three phases. At phase 1, therapist just focuses on eating and gaining weight of patient. Parents are given responsibility of adolescent eating. When the patient reaches 90 % of calculated target weight, treatment is passed to phase 2. Previously postponed problems begin to be raised at this phase. Control over eating passes from parents to adolescent with supervision of parents, slowly. At phase 3, adolescent should increase personal autonomy and set appropriate family boundaries. Although FBT is first-line modality for adolescent AN, only a few therapists apply this therapy in our country. In this workshop, family-based treatment applications for eating disorders in adolescence will be taught.

[Abstract: 0687] [Mood disorders]

Development of multiple proteomic marker panel and predictive model for the onset and progress of mood disorders

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ABSTRACT

The authors tried to develop a predictive model using multiple proteomic marker panel and ultra-high speed multiple proteomic marker analysis platform to differentiate major psychiatric disorders and to predict the onset and progression of high-risk group of major psychiatric disorders. The study subjects consisted of age- and sex- matched 40 subjects with BD, 38 subjects with MDD, 40 subjects with SPR, and 40 healthy controls, recruited from 8 psychiatric centres in South Korea from June 2017. The objective severity of symptoms was assessed using BPRS, HAMD, and YMRS. Protein marker candidates were selected by data mining and extracted from blood samples of the subjects by quantitative proteomics applying in-depth proteome profiling using Q-exactive Orbitrap Mass Spectrometer and a Multiple Reaction Monitoring (MRM) technique using a Triple Quadrupole Mass Spectrometer. Proteins with different expression level between groups were selected by pair-wise analysis, and protein marker candidates that could distinguish each group were selected by ANOVA and Pearson correlation-based hierarchical clustering. Multivariate analysis using machine learning was performed on the amount of quantified proteins among groups, and feature selection using Support Vector Machine algorithm and leave-one-out cross validation was performed. A 56 high-risk group was also recruited until now, and 6-month and 12-month follow-up of clinical evaluation and proteome was accomplished. 63-proteomic marker candidates were extracted, and each group was clustered with a considerably high correlation by Pearson correlation-based hierarchical clustering. A panel consisting of 13 protein marker candidates showed 82.5% to 96% of the sensitivity, specificity, and accuracy in distinguishing each diagnostic group from the control group. A prospective study to predict progression of high-risk group will be presented and discussed. A blood proteomic marker panel was a plausible biomarker which could contribute to differentiate major psychiatric disorders. Future development integrating clinical evaluation and proteomics of high-risk group will enable to predict progress of major psychiatric disorders.

KEYWORDS

Bipolar disorder; proteomics; precision medicine; high-risk

References

[1] Comes et al. Transl Psychiatry. 2018;8:160.

[2] Ren et al. Psychoneuroendocrinology. 2017;86:17–24.

[Abstract: 0787] [Schizophrenia and other psychotic disorders]

Psychosis spectrum disorder: from the genome to the exposome and to the phenome

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ABSTRACT

Psychosis spectrum disorder is a complex phenotype with a multifactorial etiology that remains poorly understood. Although genome-wide association studies now explain up to 30-40% of the genetic variation by single nucleotide polymorphisms, these heritability estimates of schizophrenia (the proportion attributable to genetics) are still much lower than those based on twin studies. These findings indicate the role of non-genetic factors, such as obstetric complications, childhood trauma, and cannabis use. The pathoetiology appears to involve genetic underpinnings that act by sensitizing individuals to effects of environmental exposures: gene-environment interaction. We have recently discussed the challenges of environmental research in psychiatry and how the exposome (totality of the environmental exposures) framework, an agnostic exposure-wide analytic approach akin to genome-wide analysis, might help us with embracing the complexity of the environment in the context of multiplicity and correlations of exposures. Genome-wide analyses consistently show pleiotropic mechanisms across mental disorders, and mental health outcomes exhibit a dynamic network structure, with an interplay between exposures and symptom domains. The presentation discusses these challenges and provides our recent findings showing the independent and joint effects of exposome and genome across psychosis spectrum.

KEYWORDS

Schizophrenia; psychosis; genetics; environment; epidemiology

[Abstract: 0696] [Schizophrenia and other psychotic disorders]

Schizophrenia and sleep disorders

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ABSTRACT

Sleep disorders are extremely prevalent among diagnosed schizophrenics, affecting almost 30–80% of patients' complaint of insufficient sleep, difficulties getting to sleep, waking up during the sleep period, and/or early waking and being unable to go back to sleep, decreased sleep efficiency, and short/long total sleep time [1–3]. Studies implicate either the exogenous effects of psychoactive agents such as psychotropic medications, alcohol, illegal substances or the endogenous effects of disease-related pathophysiological processes on sleep continuity and architecture. Schizophrenia and sleep disorders have been linked to dysfunction in specific neural circuits including dopaminergic and serotoninergic pathways. Melatonin may play a role in the neurodevelopmental origins of schizophrenia. Melatonin deficiency may be involved in impaired axogenesis reported in schizophrenia [4]. Schizophrenic patients report various patterns of disordered sleep, mainly insomnia, restless legs syndrome, obstructive sleep apnea syndrome (OSAS), narcolepsy [1,3]. Insomnia has traditionally been thought of as a consequence of psychotic symptoms, however recent research indicates that insomnia itself contributes to the development of psychotic experiences [2]. Insomnia related to limb movement have a relationship with long-term neuroleptic therapy [1,5]. OSAS may also be important in schizophrenia that estimates of prevalence among persons with schizophrenia. Advanced age and excess weight, psychotropic medication exposure increases the risk of severe OSAS [6,7]. There are several case reports of patients with narcolepsy and schizophrenia. The occurrence of psychotic symptoms and hallucinatory episodes in narcolepsy has been reported as responsible for delayed diagnosis due to the misdiagnosis of schizophrenia. Thus, up to 7% of all patients with a diagnosis of schizophrenia may have a psychotic variant of narcolepsy [5]. Although have not enough research about both diseases co-occurrence, sleep difficulties constitute a source of clinical concern since they are associated with greater symptom severity, increased relapse rates, worse prognosis, and a diminished quality of life.

KEYWORDS

Schizophrenia; sleep; insomnia

References

[1] Assimakopoulos K, Karaivazoglou K, Skokou M, Kalogeropoulou M, Kolios P, Gourzis P, et al. Genetic Variations Associated with Sleep Disorders in Patients with Schizophrenia: A Systematic Review. Medicines. 2018;5:27.

[2] Chiua VW, Reec M, Jancab A, Iyyalold R, Dragovic M, et al. Sleep profiles and CBT-I response in schizophrenia and related psychoses. Psychiatry Research. 2018;268:279–287.

[3] Van den Noort M, Bosch P. Schizophrenia and Sleep Disorders: An Introduction. Medicines. 2018;5:94.

[4] Sahbaz C, Özer OF, Kurtulmus A, Kırpınar I, Sahin F, et al. Evidence for an association of serum melatonin concentrations with recognition and circadian preferences in patients with schizophrenia. Metabolic Brain Disease. 2019. doi:10.1007/s11011-019-00395-3

[5] Kaskie R, Graziano B, Ferrarelli F. Schizophrenia and sleep disorders: links, risks, and management challenges. Nature and Science of Sleep. 2017;9:227–239.

[6] Copps B, Yu C, Sylvestre JD, Segal M, Looper K, et al. Obstructive sleep apnea: underdiagnosed in older adults with severe mental illness? Sleep Breath. 2015;19:1407–1408.

[7] Alam A, Chengappa R, Ghinassi F. Screening for obstructive sleep apnea among individuals with severe mental illness at a primary care clinic. General Hospital Psychiatry. 2012;34:660–664.

[Abstract: 0207] [Schizophrenia and other psychotic disorders]

Schizophrenia and addiction

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ABSTRACT

Schizophrenia, a complex neuropsychiatric syndrome that affects nearly 1% of the population and includes positive, negative, and cognitive symptoms, is associated with a variety of co-occurring substance use disorders. The Epidemiologic Catchment Area survey showed that individuals with schizophrenia have a high rate of substance (e.g., alcohol, cannabis, and cocaine) use disorders – nearly 5 times the rate seen in the general population. Moreover, compared to declining rates of tobacco smoking in the general US population, the rate of smoking in those with schizophrenia remains disturbingly high (between 70 and 90%). The use of substances in people with schizophrenia is associated with significantly increased morbidity and mortality and use of most of them worsens the overall course of schizophrenia, as evidenced by increased rates of hospitalization, more treatment non-adherence, brain volume loss, increased violence and suicide, and overall increased societal costs. Unfortunately, the current options for treating substance use disorder in patients with schizophrenia are limited, and the underlying basis of substance use in these patients is not well understood . The comorbidity of schizophrenia and substance abuse has attracted increasing attention in the past years, with multiple potential links, including genetic vulnerability, neurobiological aspects, side effects of medications, and psychosocial factors being under discussion. The link between the use of substances and the development of psychoses is demonstrated by the high prevalence of substance abuse in schizophrenia. Apart from alcohol misuse, substances commonly abused in this patient group include nicotine, cocaine, and cannabis. In particular, heavy cannabis abuse has been reported to be a stressor eliciting relapse in schizophrenic patients. In general, substance use in psychosis is associated with poorer outcomes, including increased psychotic symptoms and poorer treatment compliance. Since both disorders have been observed to be closely interdependent, a particular treatment for schizophrenic patients with comorbidity of substance abuse is needed in order to provide more effective care. In this article, we discuss various potential modes of interaction and interdependence, and the possibility of embarking on new therapeutic paths for treating this particular population. Epidemiological research in this field focuses on the identification of risk factors, the temporal relationship of the onsets of the disorders, and on specific symptoms. The proportion of schizophrenic patients with comorbidity of substance abuse varies in published studies from 10% to 70%, depending on how patients are diagnosed with schizophrenia, the types of populations studied, and the different ways of defining drug and alcohol disorders. However, an increasing number of publications demonstrate a high prevalence of substance abuse in schizophrenia. Up to 50% of patients with schizophrenia exhibit either alcohol or illicit drug dependence, and more than 70% are nicotine-dependent. In particular, heavy cannabis abuse has been reported to be a stressor, eliciting relapse in patients with schizophrenia and related disorders. Consistent findings concerning demographic characteristics and gender aspects suggest that male persons of younger age and lower educational level are associated with a greater risk for substance abuse. However, it seems important to mention that substance abuse difficulties among women with schizophrenia are often insufficiently identified, and that women with comorbidity of substance abuse are less likely to obtain substance abuse treatment.

KEYWORDS

Schizophrenia; addiction; psychopharmacology

[Abstract: 0806] [Others]

Schizophrenia and sexual dysfunctions

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ABSTRACT
Sexual dysfunctions have been described as being common in schizophrenia patients. The pathophysiology behind their development remains unclear. They can be secondary to the disease itself or an adverse event of antipsychotic medication. The relationship between schizophrenia and sexual dysfunction is variable and different between men and women. There are variety of serious problems in sexual functionality due to both natural course and applied pharmacotherapy. Antipsychotic medications are known to be commonly associated with sexual dysfunction. There are many factors increasing the frequency of sexual dysfunction, and thus decreasing the quality of life and causing incompliance to drug usage. The evaluation of antipsychotics is often restricted to prolactin measurement. Clinician should keep these problems in mind and talk to their patients on this topic to pinpoint the source of the problem and to minimize them, to enhance the quality of life and treatment compliance.

KEYWORDS
Sexual dysfunction; schizophrenia; sexual life; sexual side effects; antipsychotic; medications

References

[1] Malik P. Sexual dysfunction in schizophrenia. Curr Opin Psychiatry. 2007;20(2):138–142.
[2] Hocaoglu C, Celik FH, Kandemir G, Guveli H, Bahceci B. Sexual dysfunction in outpatients with schizophrenia in Turkey: a cross-sectional study. Shanghai Arch Psychiatry. 2014;26(6):347–356.

[Abstract: 0105] [Schizophrenia and other psychotic disorders]

Schizophrenia and suicide

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ABSTRACT
The high incidence of suicidal behavior in schizophrenia is well documented. Suicide is the chief cause of premature death among individuals with schizophrenia. The lifetime rate of completed suicide is estimated to be around 10% [1]. Estimates of non-fatal suicide attempts range from 18% to 55% [2]. Further, 40% to 54% of individuals with schizophrenia either try or think about ending their lives [1]. There is substantial evidence demonstrating a relationship between these different suicidal expressions. Completed suicide in psychosis has been linked to past suicide attempts, and to past and recent suicidal ideation. Suicidal ideation is also associated with increased risk of suicide attempts. While the absence of suicidal ideation should not be assumed to indicate the absence of risk, the presence of suicidal ideation is a potential early warning for more severe suicidal behavior. As reflected in existing suicide prevention strategy, it is an important phenomenon to identify in vulnerable individuals, and a potential target for intervention [3]. Depression, hopelessness, and few reasons for living have been reliably identified as risk factors; however, there is a need for consideration of other affect related processes, such as self-esteem and schematic (core evaluative) beliefs. These have been identified as etiological and maintaining factors in studies of depression and may play a similar role in depressed mood and suicidal thinking in schizophrenia. Suicidality might equally be driven by the positive symptoms of schizophrenia. There are several reasons to expect hallucinations and delusions to be specific risk factors, including a tendency for irrational thinking and behavior when positive symptoms are pronounced, and intentional acts of suicide taken for psychotic reasons (e.g., to escape delusional persecution). Findings concerning the suicide risk associated with hallucinations and delusions have, however, been varied [4]. This may in part be explained by evidence that individual positive symptom types relate differently to suicidal behavior, for example, paranoid delusions have been shown to be associated with an increased risk of suicide but somatic delusions with a decreased risk [5]. Further, the experience of psychotic symptoms can be seen as multidimensional, for instance, delusional experience differs in levels of belief conviction, preoccupation, and distress. It may be that aspects such as delusional distress relates more

KEYWORDS
Schizophrenia; risk factors; suicidal behavior

closely to suicidal ideation; however, existing studies have not looked at suicidal ideation in relation to the multidimensional nature of psychosis. Suicide intent is frequent among patients with schizophrenia and studies show that this is associated with distress in the patients, more hospitalizations and medical costs, and premature mortality. In this presentation, I aimed to comprehensively evaluate psychosocial risk factors associated with suicidality in patients with schizophrenia.

References

[1] Cassidy RM, Yang F, Kapczinski F, Passos IC. Risk Factors for Suicidality in Patients with Schizophrenia: A Systematic Review, Meta-analysis, and Meta-regression of 96 Studies. Schizophr Bull. 2018;44 (4):787–797. doi:10.1093/schbul/sbx131

[2] Hocaoglu C, Babuc ZT. Suicidal ideation in patients with schizophrenia. Isr J Psychiatry Relat Sci. 2009;46 (3):195–203.

[3] Fang X, Chen Y, Wang Y, Zhang C. Identification of risk factors for suicidal ideation in patients with schizophrenia. Psychiatry Res. 2018;271:195–199. doi:10.1016/j.psychres.2018.11.051

[4] Kim SW, Kim SJ, Mun JW, Bae KY, Kim JM, Kim SY, et al. Psychosocial factors contributing to suicidal ideation in hospitalized schizophrenia patients in Korea. Psychiatry Investig. 2010;7(2):79–85. doi:10.4306/pi.2010.7.2.79

[5] Marunkevych YY· Peculiarities of suicidal behavior of patients with paranoid schizophrenia with various clinical symptoms.Wiad Lek. 2018;71(7):1392–1397.

[Abstract: 0647] [Others]

An endophenotype in children and adolescents: irritability and current approachesIrritability: definition, clinical presentation and its place in child psychiatry

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ABSTRACT

Irritability is defined as a low threshold to experience anger in response to frustration. Stringalis described irritability as a mood of easy annoyance and touchiness, characterized by anger outbursts and anger. It is one of the most common symptoms in youth and is part of the clinical presentation of several disorders. Prevalence estimates of severe irritability in community samples of children and adolescents range from 0.12% to 5%. Irritability has been surrounded by controversy in child and adolescent psychiatry. First, the debate was driven by the boundaries between chronic irritability and bipolar diagnosis. Between the mid-1990s and early 2000s in the United States, there was a dramatic increase in the rate of diagnosis of Bipolar Disorder (BD) in children and adolescents. Than a series of longitudinal, family, behavioral, and pathophysiological studies differentiate classically defined episodic pediatric bipolar disorder from chronic irritability without distinct manic or hypomanic episodes (operationalized as severe mood dysregulation). Once the boundaries with BD were clarified, the debate turned to the proposal of a new diagnosis category, whose main symptom is emotional dysregulation. Firstly, Severe Mood Dysregulation (SMD) was proposed as a phenotypic differentiation from BD. Secondly, the DSM-5 working group excluded the hyperarousal criterion from SMD, since it overlapped with attention and hyperactivity disorder (ADHD) symptoms, and proposed a new diagnostic category called Disruptive Mood Dysregulation Disorder (DMDD). Although evidence suggests a dimensional distribution across the pediatric population, the DSM-5 indicates the adoption of DMDD as a new category entity. Thirdly, Irritable dimension within the Oppositional Defiance Disorder (ODD) diagnosis is characterized by temper outbursts, annoyance, and touchiness. Similar to SMD, the Irritable dimension shows strong association with emotionality. However, even though both share the same core features, the assumption of SMD/DMDD as a specifier of ODD (Irritable dimension) was considered insufficient to describe the phenotype clinically. If ODD and DMDD co-occur, DMDD should be the diagnosis given. Fourthly, irritability as a dimension phenotype cutting across diagnoses. The DSM-5 indicates the adoption of DMDD as a new category entity. Chronic, severe irritability is one of the most common symptoms in childhood and adolescence and is very impairing. Furthermore, childhood irritability predicts suicidality, social impairment, and internalizing problems such as depression and anxiety disorders in adulthood. Irritability is also seen in externalizing problems such as ADHD, conduct disorders, ODD. Even it is not a main symptom in many disorders such as social phobia, it may occur in all psychiatric disorders in child and adolescents. Focusing on both normative and pathologic development, we will review the construct of irritability from its origins in aggression and disruptive behavior research to its contemporary relevance for affective psychopathology in child and adolescent psychiatry. We want to discuss the symptom of irritability and review the literature up to date.

KEYWORDS

Irritability; disruptive mood; child and Adolescent

[Abstract: 0648] [Disruptive behavior disorders]

An endophenotype in children and adolescents: irritability and current approachesIrritability: as a symptom in infancy and early childhood

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ABSTRACT

Determining behavioral difficulties in preschool children is very complicated due to the fact that aggression can be seen in the course of normal development. In developmental perspective, negativism, opposing behavior, and 'the desire to act on its own' are necessary to achieve independence, as it relates to separation from attachment figures. It is hard to regard these behaviors as abnormal since they do not impair the functioning of the child. On the other hand, it should be considered as a symptom when it starts to cause developmental and relational difficulties. However, it is very difficult to make normal and abnormal differentiation of anger in early childhood. Besides, anger is a normal emotion response, present from an early stage of life that serves to survive from an evolutionary point of view. Irritability is described as predisposition to anger and anger outbursts. However, the concepts of irritability, anger, frustration and aggression can be used interchangeably in psychiatric terminology and daily language. Although there is a need for irritability to survive at the very beginning of life, this requirement decreases with success in self-regulation as the infant grows. Studies report that the problematic expression of aggression is related to disinhibition and inadequate self-regulation and that regulation and inhibition develop in the first years of life. This difficulty in self-regulation has a clinical appearance as over-arousal, low frustration capacity and touchiness, and may cause serious deterioration in daily functioning. In longitudinal studies, it has been shown that irritability in children causes serious effect on functioning even in the absence of psychiatric disease. It is stated that chronic irritability in pre-school period is related to internalization and externalization disorders in the later period of life. In this respect, it may be the precursor of many early childhood mental difficulties. Nevertheless, it is not easy to predict whether irritability will evolve into ADHD (attention deficit and hyperactivity disorder)/ODD (oppositional defiant disorder), where behavioral problems are at the forefront, or MDD (major depressive disorder)/DMDD (disruptive mood dysregulation disorder) in which difficulties in emotion regulation are significant. Irritability as a symptom cannot be limited only in relation to these diagnoses. In the autism spectrum disorder (ASD), intense irritability can be seen, which may sometimes mask diagnostic symptoms, complicate the daily life of the child and exacerbate the existing sensory sensitivities. In addition, irritability can be seen in the course of organic diseases, secondary to brain damage or as side effects of some drugs. In this case, it is important to carefully address the early childhood irritability in clinical practice of child and adolescent psychiatry. The aim of this presentation is to discuss the clinical and etiologic features of irritability in early childhood and to discuss the current data about its course.

KEYWORDS

Irritability; anger; early childhood; preschool psychopathology; infancy

[Abstract: 0634] [Others]

An endophenotype in children and adolescents: irritability and current approachesIrritability: as a symptom in childhood

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ABSTRACT

Irritability can be defined as an increase in the tendency of anger to be compared to peers. Recently, this clinical problem has become the center of attention of important research in the fields of child psychiatry and clinical neuroscience [1]. This interest is due to the recognition of the clinical significance of irritability as one of the most common causes of mental health for children [2]. Considering this situation, irritability is the primary feature of Disruptive Mood Dysregulation Disorder (DMDD), a new diagnosis in DSM-5 [3]. Also, irritability is seen in other childhood psychiatric disorders such as contrast disorder, anxiety disorders, Attention-deficit/ hyperactivity disorder, post-traumatic stress disorder, behavior disorder, major depressive disorder, bipolar disorder and autism spectrum disorder. Preschool children and school-age children are the most troublesome age group and often

KEYWORDS

Irritability; childhood; neurodevelopmental disorder

experience tantrums when they experience frustration. The question of whether irritability is typical or atypical is still not fully answered. Although symptoms of irritability have been present in children for a long time, research into the evaluation, etiopathology and treatment of these children has been ongoing [4]. Some studies have shown that these symptoms are present in very young children, may be permanent and are associated with impairment when they are permanent [5]. The relationship between neurodevelopmental disorders and irritability is considered important by many researchers. In a review on irritability, it was stated that irritability was a stable dimension and that it was associated with psychological disorders in the later period [6]. Researchers' interest in ADHD is increasingly directed towards irritability, irritable mood and irritable behavior in individuals with ADHD [7]. In one study, a neurobiologically different and stable ADHD subtype characterized by extreme negative emotion levels was defined. In another study, it was shown that irritability was mediated by ADHD and alcohol use problems in adolescents with ADHD [8].The explanation of the mechanisms by which these symptoms persist in childhood and how to change these trajectories is a critical goal for new research. In this presentation, we wanted to discuss irritability symptom in children by the help of current debates.

References

[1] Brotman MA, Kircanski K, Stringaris A, Pine DS, Leibenluft E. Irritability in youths: a translational model. American Journal of Psychiatry. 2017;174 (6):520–532.

[2] Peterson BS, Zhang H, Santa Lucia R, King RA, Lewis M. Risk factors for presenting problems in child psychiatric emergencies. Journal of the American Academy of Child & Adolescent Psychiatry. 1996;35 (9):1162–1173.

[3] AmericanPsychiatricAssociation. Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub; 2013.

[4] Althoff RR. Pediatric Irritability Comes of Age. Elsevier; 2018.

[5] Wiggins JL, Briggs-Gowan MJ, Estabrook R, Brotman MA, Pine DS, Leibenluft E, et al. Identifying clinically significant irritability in early childhood. Journal of the American Academy of Child & Adolescent Psychiatry. 2018;57(3):191–199. e2.

[6] Vidal-Ribas P, Brotman MA, Valdivieso I, Leibenluft E, Stringaris A. The status of irritability in psychiatry: a conceptual and quantitative review. Journal of the American Academy of Child & Adolescent Psychiatry. 2016;55(7):556–570.

[7] Karalunas SL, Fair D, Musser ED, Aykes K, Iyer SP, Nigg JT. Subtyping attention-deficit/hyperactivity disorder using temperament dimensions: toward biologically based nosologic criteria. JAMA psychiatry. 2014;71(9):1015–1024.

[8] Harty SC, Gnagy EM, Pelham Jr WE, Molina BS. Anger-irritability as a mediator of Attention-deficit/ hyperactivity disorder risk for adolescent alcohol use and the contribution of coping skills. Journal of child psychology and psychiatry. 2017;58(5):555–563.

[Abstract: 0645] [Others]

An endophenotype in children and adolescents: irritability and current approachesIrritability: as a symptom in adolescence

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ABSTRACT

Irritability can be defined as an elevated proneness to anger relative to peers. Clinically impairing irritability in adolescents began to gain more attention as interest grew in the diagnosis of pediatric bipolar disorder (BPD). Beginning in the 1990s, child psychiatry researchers suggested that while pediatric BPD can present with distinct episodes of mania or hypomania as in adults, the more typical pediatric presentation was chronic, severe irritability and hyperarousal symptoms. Indeed, more recent evidence indicates that the pathophysiological correlates of the trait of irritability itself differ between BPD and Disruptive Mood Dysregulation Disorder (DMDD). The Youths with DMDD exhibit severe and recurrent temper outbursts that are more easily elicited, longer lasting, and contextually atypical relative to those of their peers. Outbursts are characterized by motor activity, prominent displays of anger and other negative emotions, and verbal as well as sometimes physical displays of reactive aggression. Between temper outbursts, severely irritable adolescents also have a persistent angry mood, involving hostile nonverbal behaviors and reports of being annoyed over many days. Thus, DMDD includes affective and behavioral. Adolescents generally come to child and adolescent psychiatry clinic with the symptom of irritability and clinicians have to make the differentiation from DMDD and other psychiatric diseases. When we differentiate DMDD from the BD, DMDD is characterized by chronic irritability, whereas, irritability in BD is episodic, representing a change from the person's

KEYWORDS

Irritability; adolescence; mood disorders

usual state. Thus, the typical mood of DMDD is consistently irritable or angry, while that of BD varies across euthymic, depression, and mania. Intermittent explosive disorder (IED): the two disorders differ in frequency of outbursts (2/week for IED for 3 months versus 3/week for DMDD). Critically, there is no requirement of persistent irritability in IED although it may be present. Since criteria may be met for both disorders, DSM-5 stipulates that DMDD takes precedence over IED. However, IED is appropriate when the duration is below one year. Oppositional Defiant Disorder (ODD): Both DMDD and ODD criteria include irritability and temper outbursts. The two disorders differ in (1) severity: in DMDD, outbursts must occur 3 times/week, but only once a week in ODD; (2) duration: 12 months for DMDD, and 6 months for ODD; and (3) pervasiveness and impairment: DMDD must impair function in two of three settings and be severe in one setting; there is no such requirement for ODD. Thus, more children with DMDD will meet criteria for ODD, than the reverse. Youths with chronic irritability (including when it occurs in the context of oppositional defiant disorder) are at elevated risk for later depression and anxiety, but not manic episodes. Early manifestation of chronic irritability during childhood, especially when combined with depressive/anxious mood, may be associated with an elevated risk for adolescent suicidality. Also high levels of childhood irritability also predict increased risk for suicidality and functional impairment in adulthood. The type of irritability and episodic nature of irritability may help us to understand this symptom. In this panel we wanted to discuss irritability symptom in youth by the help of our studies and current debates.

[Abstract: 0766] [Psychotherapy]

Self-harming behavior: definition, prevalence and psychosocial risk factorsa

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ABSTRACT

Self-harm behavior includes behaviors such as self-cutting, scratching, and burning, done without the conscious intent to take one's life. Some studies have revealed adolescents who engage in self-harm behavior is to get relief from distress; to escape from a situation; and to show how desperate they are feeling. Some epidemiologic studies have demonstrated high rates of self-harm behavior in adolescents. In recent years, rates of self-harm behavior are quite high in contemporary populations of youth. Approximately 5–13% of school students reported self-harm behavior in United Kingdom and Australia. Over half of adolescents who have self-harmed report engaging in more than one episode in their lifetime, showing the repetitious nature of this behavior. Moreover, a previous history of self-harm is a key risk factor for suicide and so self-harm has become a growing public health concern. A high association between self-harming behavior and different psychiatric disorders, such as depression, anxiety, bipolar disorder, and borderline personality disorder, has been shown. Self-harm behavior is also associated with eating disorders, a history of abuse or trauma, awareness of self-harm in peers, family members who self-harm, drug misuse, and low self-esteem. Suicide ideation and attempts are more likely to be reported among those with repeated non-suicidal self-harm. Early interventions and prevention programs may reduce the number of serious physical injuries resulting from self-harm behavior and lower the risk of future suicide in young people. This presentation will include the definition, prevalence of self-harm behavior. And discuss about the psychosocial risk factors of self-harm behavior.

KEYWORDS

Self-harm behavior; deliberate self-harm; self-cutting; self-mutilation; para-suicidal behaviors; psychosocial risk factors

References

[1] Gratz KL. Risk factors for and functions of deliberate self-harm: an empirical and conceptual review. Clin Psychol Sci Pract. 2003;10:192–205.

[2] Klonsky ED. The functions of deliberate self-injury: a review of the evidence. Clin Psychol Rev. 2007;27:226–239.

[3] Madge N, Hewitt A, Hawton K, et al. Deliberate self-harm within an international community sample of young people: comparative findings from the Child & Adolescent Self-harm in Europe (CASE) Study. Journal of Child Psychology and Psychiatry. 2008;49:667–677.

[4] Owens D, Horrocks J, House A. Fatal and non-fatal repetition of selfharm – Systematic review. British Journal of Psychiatry. 2002;181:193–199.

[5] Tuisku V, Pelkonen M, Karlsson L et al. Suicidal ideation, deliberate self-harm behavior and suicide attempts among adolescent outpatients with depressive mood disorders and comorbid axis I disorders. Eur Child Adolesc Psychiatry. 2006;15:199–206.

[6] Nixon MK, Cloutier P, Jansson SM. Nonsuicidal self-harm in youth: a population-based survey. CMAJ. 2008;178:306–312.

[Abstract: 0709] [Others]

Self-harming behaviour: its neurobiology

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ABSTRACT

It was attempted to examine the aetiology of self-harm behaviour, which is a frequent mental health problem during adolescence, in many aspects, and it was put forth that certain environmental, social and familial factors and the circle of friends of the adolescent, in addition to the traumas experienced during childhood, affect it. However, the neurobiological and genetic aspects of self-harm behaviour have not been understood completely yet. In the examination of the cerebrospinal fluid (CSF) of individuals exhibiting self-harm behaviours, serotonin metabolite 5-hydroxyindoleaceticacid (5-HIAA) levels were found to be low, and it was stated that low serotonin levels were correlated to self-harm and violence behaviours. There are also studies showing that the CSF serotonin level and peripheric serotonin level decrease in self-harm behaviours with no intention of suicide. In the last ten years, the concept of neuroplasticity explaining the neurones in the brain and the structural properties of the synapses created by them and the changes in their functions depending on various environmental stimulants started to be emphasised in the self-harm and suicide attempt aetiology.

BDNF is a neurotrophin with a very important role in neuroplasticity. In the recent ten years, the role of BDNF in the pathophysiology of suicide behaviour has attracted the attention of researchers. In a study carried out by Kim et al. (2007), the plasma BDNF levels of patients diagnosed with depression who attempted and did not attempt suicide and healthy controls were compared, and it was found out that plasma BDNF level is related to the suicide behaviour in depression. According to the findings obtained from the results of the same study, it was put forth that BDNF level can be a biological indicator in depression with suicide thoughts.

The low level of BDNF in suicide-related patients is explained by decreasing the BDNF expression by the low serotonin function causing suicide thoughts and attempts. BDNF and serotonin are held responsible for synaptic plasticity, neurogenesis and neuronal survival. These two signals regulate each other. Whether BDNF can be a biological marker for suicide behaviour in adolescents keeps on being an issue of concern.

In a study examining the BDNF messenger ribonucleic acid (mRNA) expression in peripheral mononuclear blood cells of depression patients with and without suicide attempt, the BDNF mRNA expression in peripheral mononuclear cells of depression patients was found to be low when compared to healthy controls, and it was shown that the BDNF mRNA expression of those depression patients who attempted suicide was lower. In post-mortem studies, significantly low BDNF levels were indicated in the hippocampus and prefrontal cortexes of suicide cases, independently of the psychiatric diagnosis. In a study in the literature carried out on BDNF Val66Met polymorphism in two groups with self-harm behaviour and suicide attempts, it was indicated that the co-existence of negative environmental factors during childhood and BDNF homozygote Val allele is a possible risk factor for the formation of both suicide and self-harm behaviour.

KEYWORDS

Adolescent; neurobiology; self-harming behaviour

[Abstract: 0714] [Psychopharmacology]

Self-harming behavior: pharmacological treatment

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ABSTRACT

Self-harming behavior is an important mental health problem that has been increasing in recent years and is frequently seen in adolescence [1]. Self-harming behavior in adolescents may represent a temporary distress period, but, as recent studies have shown, there may be an important indicator of a significant increase in mental health problems in the later period of life and a stronger risk of further suicidal behavior in the future [2]. Favazza classified self-harming behavior in four groups: major, stereotypic, compulsive and impulsive self-harm [3]. There is no psychiatric disorder specific to self-harm and may accompany many disorders. In the clinical sample, self-harming behavior is accompanied by a high rate of psychiatric

KEYWORDS

Self-harming behavior; mental disorders; adolescents; pharmacotherapy

disorders. In one study, at least one psychiatric disorder was diagnosed in 92% of the patients with self-harming (including suicide attempt) over 15 years of age. Depression, anxiety disorders, posttraumatic stress disorder, dissociative disorder, substance use disorders, conduct disorder, eating disorders and personality disorders (especially borderline personality disorder) are the most common mental disorders in adolescents with self-harming. Therefore, when planning pharmacological treatment, it is necessary to consider the characteristics of the accompanying psychiatric disorder and self-harm behavior. SSRIs can be used as the first choice, especially in self-injurious behavior accompanied by depression and anxiety disorders. In cases with aggression, second generation antipsychotics, lithium, mood regulating anticonvulsants, psychostimulants SSRIs, anxiolytics, alpha adrenergic agonists, beta blockers and sedatives may be an option. The relationship between self-harming behavior and suicide is frequently emphasized by professionals in the field. Mood stabilizers, especially lithium and valproate, may be considered as an alternative to treatment in patients with self-injury and suicidal thoughts. Although there is not enough evidence in children and adolescents, some studies have shown that opioids (buprenorphine) and opioid antagonists (naltrexone) also reduce the severity and frequency of self-injurious behavior in children and adolescents. Although a treatment plan can be made according to the presence of comorbid axis I diagnoses in self-harm behavior, it is also important whether the current clinical situation creates an urgent need for intervention. Antipsychotics (haloperidol, risperidone, olanzapine, etc.) or benzodiazepines can be used in patients who referring to the emergency departments, whom have severe, self-harm behavior, agitation, or tantrum. Although pharmacotherapy is used in cases with acute and chronic self-harming behavior, it is not enough; a more comprehensive treatment plan is required. Therefore, stressful life events need to be considered accurately, coping and emotion regulation skills of adolescents with self-harming behavior need to be improved, and additionally social support should be strengthened.

References

[1] Hawton K, Hall S, Simkin S, et al. Deliberate self-harm in adolescents: a study of characteristics and trends in Oxford, 1990–2000. J Child Psychol Psychiatry. 2003;44(8):1191–1198.

[2] Portzky G, van Heeringen K. Deliberate self-harm in adolescents. Curr Opin Psychiatry. 2007;20(4):337–342.

[3] Simeon D, Favazza AR. Self-injurious Behaviors: Phenomenology and Assessment. In: Simeon D, Hollander E, Editors. Self-injurious Behaviors: Assessment and Treatment. Washington (DC): American Psychiatric Press; 2001.

[Abstract: 0739] [Impulse control disorders]

Self-harming behavior: treatment with psychotherapy

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ABSTRACT

Non-suicidal self-injury (NSSI) among adolescents is gaining increasing attention in both clinical and scientific arenas. The lifetime prevalence of NSSI is estimated to vary between 7.5% to 8% for preadolescents, increasing to between 12% and 23% for adolescents. Child and adolescent clinicians are left with little evidence-based guidance for treating this challenging population. In my speech, I will try to provide some guidance, evaluations of treatments for adolescents with NSSI and related conditions, such as deliberate self-harm and borderline personality disorder. Indeed, to date, no treatments have been specifically designed and evaluated for adolescents engaging in clinically-significant levels of NSSI. The reviews the dearth of psychotherapeutic treatments designed specifically for adolescents with NSSI. Even without the guidance of empirically-supported treatments for NSSI, clinicians must still treat adolescents with NSSI. Some national guidelines have been developed for DSH; again, NSSI and suicidal self-injury have been combined in most of these guidelines. In 2004, the National Institute for Health and Clinical Excellence (NICE; http://www.nice.org.uk) in the United Kingdom published a clinical guideline for DSH. However, the NICE guidelines only reference the need for at least 3 months of "an intensive therapeutic intervention" for people at risk for repetitive self-harm. DBT is recommended for consideration, but only for people with self-harm and a diagnosis of borderline personality disorder. NSSI is likely to require specific psychotherapeutic interventions, beyond the treatment of depression and/or suicidality. Further, while treatments for borderline personality disorder are likely to be helpful in reducing NSSI in adolescents with

KEYWORDS

Self-harm; adolescence; psychotherapy

these personality characteristics, it is unknown if intensive treatments for borderline personality disorder, such as DBT, are equally effective or even necessary for adolescents with NSSI who don't have a personality disorder. In addition to developing treatments for adolescents with NSSI, we must develop dissemination pipelines to move evidence-based treatments out to practicing clinicians. Training clinicians in how to treat adolescents with NSSI is likely to be as great of an obstacle as creating the treatments in the first place. A recent study evaluating the effectiveness of DBT for adults with borderline personality disorder using routine community treatment settings found that therapists who received more intensive training had better outcomes than therapists who only received basic training. Most of the psychotherapeutic approaches to NSSI discussed in the literature focus on outpatient psychotherapy, with little focus on acute forms of treatment, such as inpatient, partial hospitalization, or residential treatment. Given the strong associations between NSSI, suicidal self-injury and suicide, developing effective psychotherapeutic interventions at acute levels of care is critical. Recent studies found that NSSI was a stronger predictor of future suicide attempts than prior suicide attempts among adolescents with depression. It is therefore likely that a substantial proportion of adolescents presenting to an acute level of care for suicidal behavior will also have either historical or current risk for NSSI. Effective approaches for the management and treatment of NSSI in acute levels of care are sorely needed. Although some preliminary evidence and guidance exists for the treatment and management of NSSI in residential settings and inpatient units, evidence-based strategies remain limited. Given that psychiatric discharges in the United States for adolescents increased from 683.60 to 969.03 per 100,000 adolescents between 1996 and 2007, it is important to develop evidence-based therapeutic practices for these higher levels of care.

[Abstract: 0609] [Others]

Problematic smartphone use and relations with psychopathology constructs

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ABSTRACT

Smartphones are used worldwide. Smartphones are used for positive reasons such as social use, and also non-social (process) use such as productivity management. In fact, smartphones are used productively in the workplace to enhance worker performance, and in schools to enhance learning. However, smartphones pose serious adverse disadvantages. Smartphone overuse is associated with academic impairment, traffic accidents and physical pain, as well as mental health problems. After defining problematic smartphone use (PSU), I discuss the bidirectional effects between it and mental health. I discuss theories on how PSU develops. I discuss Uses and Gratifications Theory that can explain how some people may be driven to PSU. I also discuss Compensatory Internet Use Theory involving PSU as a means of regulating negative emotion. Also discussed is how typical models of PSU that are tested in the literature map onto these theoretical frameworks. I briefly discuss three empirical studies I conducted in my lab. The first study examined process and social smartphone use as mediators in the relation between depression and anxiety with PSU. However, this study suffered from the limitation of failing to assess psychopathology beyond depression and anxiety. In the next study discussed, I assessed rumination and excessive reassurance seeking as mediators in the relation between depression and anxiety with PSU. I found support for rumination and excessive reassurance behavior as mediators in these relationships. However, this study was cross-sectional. In the third study, I discuss a repeated measures study assessing distress tolerance and mindfulness as mediators between depression and anxiety with PSU. I found support for these mediators in these relationships. However, I discuss the limitation that smartphone use was measured via self-report. After discussing briefly these studies, I discuss a fourth study in more detail. In this study, I measured smartphone use via objective measurement of phone logs. I assessed emotion dysregulation and depression in relation to objectively measured smartphone use over one week. Phone use was measured using the Moment app for iPhone. Results demonstrated that female gender was associated with more minutes of smartphone use at the beginning of the week, and more depression was associated with decreased smartphone use over the week. When excluding weekend days, expressive suppression of emotion was associated with more smartphone use at the beginning of the week, but less use over the week. Conclusions and limitations of this study are discussed. I discuss the main conclusions of these studies and the importance in studying psychopathology in relation to PSU.

KEYWORDS

Internet addiction; smartphone addiction; psychopathology

[Abstract: 0706] [ADHD]

ADHD as a later in life neuropsychiatric disorder

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ABSTRACT

Although follow-up studies in diagnosis, treatment and management of ADHD are popular in recent years, these studies are not included in late adulthood. For example, a 33-year follow-up study reported that the oldest person was 41 years old [1]. In particular, cognitive symptoms of individuals over the age of 65 are considered to be non-ADHD cognitive-related disorders, which in fact lead to the deprivation of pharmacotherapy in these patients with ADHD. The effects of ADHD on academic, occupational and social functioning have been well documented in childhood and adulthood, but the impairment in this functionality is beyond academic and occupational functioning in late adulthood [2]. To or best knowledge, there are 4 studies in the literature that meet all the diagnostic criteria of ADHD over 50 years of age which are only one 68-year-old woman, four women aged 62 to 91 years. Others are chart review and population studies in Israel and Sweden respectively. In terms of differential diagnosis of ADHD in older adults, mild cognitive impairment, dementia, Parkinson's disease, toxic / metabolic infections and secondary encephalopathies should be considered. Neurological examination of these diseases except for ADHD may include symptoms such as apraxia, aphasia, asymmetric motor deficits, delirium, and rigidity. Also In neuroimaging, different atrophy patterns, amyloid deposits, different infarct areas and white matter changes can be seen in these non-ADHD cognitive diseases [3]. Because of the systematic comprehensive studies of ADHD treatment are not yet sufficient in older adults, individual-specific interventions are recommended in this group. Medication and / or behavioral interventions should be evaluated depending on the age, medical condition, symptom severity and social support of the individual. Although studies showing the superiority of pharmacotherapy over behavioral interventions have been reported in children interventions have not been systematically compared in older adults. There are effective studies on how behavioral interventions improve coping strategies, reduce anxiety and increase daily functioning and satisfaction in older adults with ADHD [3,4].

KEYWORDS

ADHD; cognitive impairment; neuropsychiatric disorders; adulthood

References

[1] Klein RG, Mannuzza S, Olazagasti MA, Roizen E, Hutchison JA, Lashua EC, et al. Clinical and functional outcome of childhood attention-deficit/hyperactivity disorder 33 years later. Arch Gen Psychiatry. 2012;69 (12):1295–1303.

[2] Goodman DW. The consequences of attention-deficit/ hyperactivity disorder in adults. J Psychiatr Pract. 2007;13(5):318–327.

[3] Goodman DW. Clinical Presentation, Diagnosis and Treatment of Attention-deficit/ hyperactivity disorder (ADHD) in Older Adults. A Review of the Evidence and its Implications for Clinical Care. 2016.

[4] MTA Cooperative Group. A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. The MTA Cooperative Group. Multimodal Treatment Study of Children with ADHD. Arch Gen Psychiatry. 1999;56(12):1073–1086.

[Abstract: 0808] [Others]

Psychiatric disease modeling by organoids through stem cells

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ABSTRACT

Psychiatric illnesses are of prodigious communal and medical prominence. However, the intricacy of these diseases and the challenges of modeling the expansion and utility of the human brain have made these conditions problematic to study experimentally. It has proven that animal models are suitable for studying single nucleotide polymorphisms. Hence, the mainstream of psychiatric phenotypes contains mixed combinations of different alleles that are tremendously challenging to restructure in animal models. Furthermore, modified animal models are restricted by intrinsic differences in the improvement, construction and occupation of their brains. Therefore, the usage of endogenous human brain tissue is

KEYWORDS

Cerebral organoids; disease modelling; psychiatric illnesses; 3D culture; stem cells

complex in practice and in ethical apprehensions of tissue obtainability, growth and manipulation. The speech will focus on human pluripotent stem cell (hPSC)-derived *in vitro* models of psychiatric diseases and deliver a debate of the benefits, restrictions and promise that 3D human brain organoids could have as novel platforms for examining disease foundations and pathology. The present culture methods of 3D brain organoids made from human pluripotent stem cells deliver an encouraging technique for investigating the phenotypic foundations of these greatly complex illnesses and for realizing the involvement of discrete risk factors and multifaceted genetic background. They hold the promise of better relevance for understanding human brain development and disease than current rodent models. The failure of many neuro-therapeutic approaches to translate from animal models to clinical practice underscores the need for better predictive models, and brain organoids may help bridge this divide. In close, the ability of organoids to differentiate, self-organize, and custom separate, complex, biologically applicable structures makes them perfect models of development, disease pathogenesis, and areas for drug screening.

[Abstract: 0790] [Others]

COURSE 8

Principles and advances in stem cell therapy in neuropsychiatric disorders

Moderator: Erdem Tüzün

Clinical implications of stem cell as therapeutic targeting in schizophrenia and other psychotic disorders

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ABSTRACT

Schizophrenia is a devastating mental disorder afflicting 1% of general population. Recent genome-wide association studies (GWAS) of schizophrenia have identified more than 100 risk loci. However, the causal variants/ genes and the causal mechanisms remain largely unknown, which hinders the translation of GWAS findings into disease biology and drug targets. Most risk variants are non-coding, thus likely regulate gene expression. Excitatory dopaminergic neurons, inhibitory GABAergic neurons, microglia, and oligodendrocytes have all been implicated in schizophrenia network pathology. Still, schizophrenia has been a difficult disorder to study, not only because of the limitations of animal models in capturing the complexity of the human mind, but also because it is greatly polygenic, with high rates of variability across the population. The advent of patient-derived pluripotent stem cells and induced neural and glial cultures has brought hope for modeling the molecular dysfunction underlying schizophrenia pathology in a patient- specific manner. Induced pluripotent stem cells (iPSCs) provide an exciting opportunity to study schizophrenia in live patient-derived neuron iPSCs can be derived from somatic cells like dermal fibroblasts or hair follicle keratinocytes of patients and differentiated in vitro into a neural lineage to yield Neural Progenitor Cells (NPCs), glutamatergic neurons ,dopaminergic neurons, and other neural subtypes. Comparing the phenotypes of these patient-derived neurons to control neurons can demonstrate alterations in neuronal function which may be relevant to the neurobiology of schizophrenia. In this course, major knowledge regarding clinical implications of iPSC-based models of schizophrenia and other psychotic disorders will be presented. Last studies explore the phenotypes observed in schizophrenia and patient-derived NPCs and argue that, despite various technical and theoretical barriers, this work has already novel insights into the pathophysiology of schizophrenia. These initial findings provide evidence on the validity and utility of iPSC-based modelling in schizophrenia. To conclude, I discuss the limitations and opportunities of this work.

KEYWORDS

Genetics; induced pluripotent stem cells; schizophrenia; treatment

References

[1] Jones CA, Watson DJG, Fone KCF. Animal models of schizophrenia. Br. J. Pharmacol. 2011;164(4):1162–1194.

[2] Brennand KJ, Gage FH. Concise review: the promise of human induced plu- ripotent stem cell-based studies of schizophrenia. Stem Cells. 2011;29(12):371, 1915–1922.

[3] Buxbaum JD, Sklar P. Human induced pluripotent stem cells: a new model for schizophrenia? Cell Stem Cell. 2011;8(5):461–462.

[4] Brennand KJ, Simone A, Jou J, et al. Modelling schizophrenia using human induced pluripo- tent stem cells. Nature. 2011;473(7346):221–225.

[5] Malik N, Rao MS. A review of the methods for human iPSC derivation. Pluripotent Stem Cells. Springer; 2013. p. 23–33.

[Abstract: 0660] [Intellectual Disabilities]

Difficulties encountered by children with intellectual disability in schools: the other side of the mirror

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ABSTRACT

There are different environments where people communicate and interact with each other. School is one of the most important of these for children and young people. Children and adolescents' social skills, physical, cognitive and emotional capacity may increase with the help of the experiences at school. Successful adaptation to school is likely influenced by a number of factors including academic, social, emotional, behavioral and cognitive competencies. Children with an intellectual disability may encounter some difficulties because of both their cognitive deficits may influence adaptation and also their reduced acceptance by their peers. The reason for this less acceptance may be children and adolescents' knowledge with intellectual disability is not enough and youths usually reject differences. Children with an intellectual disability may encounter with peer bullying is one of the biggest challenges in school. Peer bullying is defined as the type of aggression that one or more students intentionally disturb the more vulnerable one student or group of students and results with psychological, social or physical consequences in that student or student group. Students who are exposed to bullying may have difficulty in adapting to school in a stressful environment. This problematic situation will negatively affect academic achievement. They may give undesirable reactions to this violence and bullying. These reactions may be absenteeism, self-closure against education and training, fear and anxiety and bullying behaviors to defend themselves against bullying. We evaluated peer bullying in our study, which included 46 children with intellectual disability and sex and age-matched children in primary and secondary school. We found that the peer bullying rate in children with intellectual disability had two times higher compared with the control group. Children with intellectual disability compared to the control group: 2-fold verbal bullying and 3-fold emotional bullying. The place where they most suffer from bullying is a classroom that there was no teacher. According to the results of our study, the children with intellectual disability stated that 15% of them do not like school, 23% of them are afraid of going to school, 17.5% of them had no good friends in the classroom. Children who exposed to bullying have more emotional problems than children not. This condition sometimes causes a life-threatening and unforgettable experience in children. The negative consequences of bullying can be seen not only in the school period but also in later years. It is important to determine the risk factors for bullying and how to intervene. It is also important to take precautions before bullying occurs. Firstly, all students and staff working in school must have knowledge about bullying in order to prevent bullying.

KEYWORDS

Children and adolescent; intellectual disability; ID; peer bullying

[Abstract: 0703] [Intellectual Disabilities]

Domestic difficulties encountered by children with intellectual disability and family approaches

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ABSTRACT

The family environment including the home situations and relationships with family members of a child remains at the center of his/her social world which is corresponding to the contexts for the emerging social interactions during the development and learning of a child about self and environment. The bi-directional interactions between child and caregiver and family may encourage or limit behaviors cause exploring and learning through the new experiences. A child with Intellectual Disability confronts with various environmental difficulties besides individual limitations. It is well-known that most of the families with intellectually disabled children have economic and social disadvantage. Children with Intellectual Disability may show serious maladaptive behaviors predicting their academic and social dysfunctioning, and challenging the capacity of family to manage them properly. The families also have to cope

KEYWORDS

Intellectual disability; maltreatment; mental disorders

with intensive caretaking and education needs, financial burden, impairing community accommodation, social isolation and rejection, and limited employment prospects. The parents of children with Intellectual Disability reported to experience increased difficulty related to the management of their children and regarding this, psychiatric disorders are more common among both parents and their children. On the other hand, due to the increased risk at family profile, the children with Intellectual Disability are found to be more likely to experience maltreatment. Poor socioeconomic statute, young parents, low education level, presence of psychopathology and disability of parents are risk factors associated with maltreatment. Some comorbidities (e.g. conduct disorder) or the age of onset may be risk factors related with child in terms of maltreatment. Additionally, the risk of maltreatment is also reported higher among the children having comorbid Intellectual Disability with congenital defects, cerebral palsy, autism and behavioral disorders. Considering all these issues, early differentiation and early intervention of children with Intellectual Disability not only enhance the efficiency of treatment but also reduce the emotional and financial strengths families confront with. The socioeconomical statute and social opportunities of child and family should be assessed and support services should be activated for essential situations. The government and society have also an important role in conserving intellectually disabled children and their families with support services ensuring their rights and well-being. Addition to social supports a multi-dimensional follow-up process is required. The treatment and follow-up process targeting development of social skills and management of negative emotions and behavioral problems of children with Intellectual Disability should be applied. Parent programs providing psychoeducation on both development, childrearing and individual difficulties of disabled child with realistic expectations should be also used to improve the impact of treatment. The parents who were assisted and supported to promote effective coping strategies may manage their stress and the behavior problems of their children successfully. Thus, the child risk for mental disorders may reduce in these families.

References

[1] Baker BL, Blacher J, Crnic KA, Edelbrock C. Behavior problems and parenting stress in families of three-year-old children with and without developmental delays. American Journal on Mental Retardation, 2002;107(6):433–444.

[2] Bronfenbrenner U, Morris PA. The ecology of developmental processes; 1998.

[3] Maclean MJ, Sims S, Bower C, Leonard H, Stanley FJ, O'Donnell M. Maltreatment risk among children with disabilities. Pediatrics. 2017;139(4):e20161817.

[Abstract: 0632] [Intellectual Disabilities]

Management of emotional and behavioral problems of children with intellectual disability (ID)

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ABSTRACT

Intellectual Disability (ID) that is characterized by significant limitations both in intellectual functioning and in adaptive behavior as expressed in conceptual, social, and practical adaptive skills originates before age 18. The prevalence of ID across the world is known to range from 1% to 3%. Although prevalence is higher in low and middle income countries (e.g., Turkey), no community-based studies have reported the rate of ID in our country. However, the rates of comorbid psychiatric disorders that cause additional challenges in terms of life quality are known to be higher in intellectually disabled children. Attention-deficit/ hyperactivity disorder, depression, anxiety disorders, oppositional defiant disorder, conduct disorder and autistic disorder are prevalently seen in about half of individuals with ID. Whereas psychiatric disorders present with roughly the same features in children with mild ID, recognition of more disorganized and unpredictable symptoms in those with moderate and severe ID is difficult because of their inability to describe own experiences and feelings. Timely identification and treatment of these comorbid disorders reduce disability, family burden and improve quality of life. There are scales available that help clinicians in the assessment of comorbid behavioral and emotional problems such as the developmental behavior checklist, and the aberrant behavior checklist. The behavioral and emotional problems especially in children with moderate and severe ID can present with restlessness (e.g., continuously moving around, unable to sit in one place), poor concentration, impulsiveness, temper tantrums, crying, withdrawn behavior (e.g., timidness, shyness), aggression, irritability, self-injury (e.g., head banging, ingestion or inhalation of foreign bodies), destroying objects, non-compliance, idiosyncratic habits (e.g., restricted range of foods), meaningless or stereotyped movements (e.g., rocking, teeth-biting, shouting, tearing

KEYWORDS

Behavioral intervention; challenging behaviors; children; emotional problems; intellectual disability

clothes, pulling hair) and socially inappropriate behavior (e.g., playing with the genitals). However, attention should also be paid to other underlying medical (e.g., unrecognized pain or discomfort, side effects of medications, substance abuse, physical illnesses such as epilepsy, behavioral phenotypes specific for a syndrome) and environmental (e.g., lack of stimulation, family conflict, bullying, change of school, death or separation, puberty, inability to communicate, carers not attuned to the young person's needs, inappropriate management that reinforces challenging behavior) conditions that can cause these symptoms while providing diagnosis and treatment. After being ascertained whether there are the other underlying causes, the management of behavioral and emotional problems requires to be conducted a comprehensive analysis. A detailed description of behavioral and emotional problems that includes time, place, activity, context, possible triggers, sequence and possible consequences of these problems, how others respond, whether these responses reinforce/extinguish these problems, how these problems affect potential needs, whether these problems serve a function or purpose for children with ID is made. As well as medication and education, behavioral intervention plan that targets to extinguish challenging behaviors should be designed to address specific problems, and involve active participation of families by taking into consideration the family's needs, strengths and weaknesses, and supporting and empowering them. Cognitive-behavioral therapy can also be used for comorbid psychiatric disorders in children with ID as in non-ID children.

[Abstract: 0677] [Intellectual Disabilities]

Use of medications in management of emotional and behavioral problems of children with ID

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ABSTRACT

Psychiatric comorbidities in patients with intellectual disability (ID) are more frequent than in general population. Psychiatric symptoms in ID are clinically relevant to cognition, function and social interaction, leading to a reduced quality of life for patients and their families. Behavioral problems such as aggressive behavior disorders, stereotypies, self-harm behaviors, emotional problems might be seen more frequently in ID population. The ID population requires a multidisciplinary and multimodal approach (from the psychopharmacological to behavioral interventions, psychoeducational, environmental and psychotherapeutical). In child and adolescent population there is limited information on the effectiveness of psychotropic medication. However, many psychotropic drugs such as antidepressants, antipsychotics, mood stabilizers, anxiolytics and stimulants could be used when it is necessary. Antipsychotics are the first choice of treatment for psychotic symptoms, and are also used very often for aggressive behavior disorders, stereotypies, vocal and motor tics. Atypical antipsychotics such as risperidone, olanzapine, clozapine, quetiapine and aripiprazole are often preferred and are also useful in treating aggressive and destructive behaviors, irritability, hyperactivity and social withdrawal. Antidepressants are used in patients with ID for the treatment of depressive, anxiety, obsessive–compulsive spectrum disorders, and behavioral disorders such as stereotypies, aggression, and self-harm behaviors. Tricyclic antidepressants (TCAs), have shown efficacy in self-harm behaviors and stereotypies in studies, but side effects are also common in patients with ID. Modern drugs with fewer side effects have overtaken these drugs. Selective serotonin reuptake inhibitors (SSRIs) have become first choice of treatment because of their better tolerability and efficacy profile compared with other antidepressants. Comparing with TCAs, SSRIs have lower anticholinergic effects, which imply lower risk of confusional states, less decreased seizure threshold, and lower risk of cardiological disturbances. In addition, its use has been demonstrated in children and adolescents with ID and it is known that is useful for treatment of behavioral problems in children and adolescents with ID. Mood Stabilizers also are used, especially lithium is used as a mood stabilizer in patients with bipolar disorder and psychosis in the context of ID. According to previous studies, lithium improves behavioral disorders such as aggressive behaviors. Valproic acid has shown efficacy in rapid cycling patients, self-harm behaviors and emotional lability which are more common in ID population in children and adolescents. Benzodiazepines could be an alternative for the treatment of anxiety disorders in this population. However, it is advisable to use them only in special situations but not as a long-term treatment. Methylphenidate is the most commonly used in attention-deficit/ hyperactivity disorder (ADHD), hyperkinesia, and impulsivity and even in resistant depression in patients with ID, and also atomoxetine appears to be useful in improving ADHD symptoms in patients, with ADHD and ID. In this panel, available psychopharmacological strategies for children and adolescents with ID, and behavioral problems were reviewed.

KEYWORDS

Intellectual; disability; behavioral; medication

[Abstract: 0770] [Others]

Cerebellar functions and their role in childhood psychopathology

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ABSTRACT
In recent decades, progress in molecular biology and advances in the development of functional neuroimaging analysis have shown that the evolution of the human cerebellum was accompanied by the acquisition of more functions than were previously deduced from human postmortem studies and animal experimentation. These new cerebellar functions included the control of attention and other cognitive functions, emotions and mood, and social behavior, which were all thought to represent cortical functions. This presentation will include brief anatomy and physiology of cerebellum and discuss the role review cerebellum in childhood psychopathology.

KEYWORDS
Cerebellum; child and adolescent; psychopathology

[Abstract: 0794] [Others]

Implantation of volumetric evaluation of brain structures on MRI in pediatric age group by stereologic method

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ABSTRACT
Human brain is the most complex organ that various anatomical and physiological factors interact with each other. Many pathological disorders lead to irreversible anatomical changes in macroscopic and microscopic anatomical structures. Although, pathological processes occur in three dimensional structures, researchers may only have information about two dimensional sections (radiological or histological). While two dimensional sections provide restricted and unrealistic data, three dimensional knowledge can be obtained by using stereological methods. Development of stereological methods in clinical and preclinical sciences improved the reliability of investigations in neuroscience. In recent years, stereological methods have been frequently used to examine the volumetric morphometric changes of brain structures in clinical and preclinical researches. Stereological methods provide to obtain quantitative data about irregular shaped three dimensional anatomical structures using by two dimensional radiological or histological sections. Cavalieri Point counting method is a design based stereological method, reliable, efficient and unbiased (Gundersen and Jensen, 1987). Additionally, this method is easy applicable, simple and inexpensive (Kipanyula and Sife 2018; Bas et al. 2008). The entire set of two dimensional sections through anatomical structure is essential for the application of this method. The slices should be separated at a known distance. Afterwards, the point counting method is applied randomly within the structure. The well described Cavalieri's principle is a stereological method that provides to measure the volume and volume fraction of various isolated central nervous system structures, cysts, or hematomas within the intracranial structures in living cases, in postmortem cases and in experimental studies (Perry et al. 2019, Bas et al. 2008).

KEYWORDS
Brain structures; stereologic method; pediatric age

References

[1] Gundersen HJG, Jensen EB. The efficiency of systematic sampling in stereology and its prediction. Journal of Microscopy. 1987;147:229–263.
[2] Kipanyula MJ, Sife AS. Global Trends in Application of Stereology as a Quantitative Tool in Biomedical Research. Biomed Res Int. 2018;13:1825697.
[3] Bas O, Acer N, Mas N, Karabekir HS, Kusbeci OY, Sahin B. Stereological evaluation of the volume and volume fraction of intracranial structures in magnetic resonance images of patients with Alzheimer's disease. Ann Anat. 2009;191(2):186–195.
[4] Perry JC, Pakkenberg B, Vann SD. Striking reduction in neurons and glial cells in anterior thalamic nuclei of older patients with Down syndrome. Neurobiol Aging. 2019;75:54–61.

[Abstract: 0763] [Schizophrenia and other psychotic disorders]

The cerebellum and the early onset schizophrenia

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ABSTRACT

Early-onset schizophrenia (EOS), defined as the onset of psychosis prior to age 18, is a severe and rare form of the disorder causing significant disturbances in perception, emotion, executive cognitive function and social relatedness [1]. A neurodevelopmental model suggesting dysfunctional connectivity involving the neural circuits within and between the prefrontal cortex, thalamus, and cerebellum, finds strong support for the explanation of the etiology of schizophrenia characterized by extreme etiologic heterogeneity [1]. The cortico-thalamic-cerebellar-thalamic-cortical loops have been proposed as a basis for a "cognitive dysmetria" underlying the cognitive deficits and symptoms seen during the course of EOS [2].

Cerebellar abnormalities have been reported, though inconsistently, in adult patients with schizophrenia. These include cerebellar atrophy, smaller anterior vermal lobe with Purkinje cell dropout, smaller total vermis area and significantly reduced total cortical and right cerebellar volume [3,4]. On the other hand, other neuroimaging studies have shown no significant structural and volumetric differences between patients with schizophrenia and comparison subjects [5,6].

In addition to regional cerebellar volumes in schizophrenia, some studies focused on cerebellar volumetric asymmetry as reflecting neurodevelopmental mechanisms underlying the etiology of the disorder. In their study exploring the presence of cerebellar asymmetry in adult schizophrenic cases, Szeszko et al. reported that male patients demonstrated significantly reversed anterior and posterior asymmetry compared with healthy male subjects although there were no differences in regional cerebellar volumes [7]. Although cerebellum has received interest in adult onset schizophrenia, there is limited data regarding its involvement in earlier onset forms of the illness.

In this presentation we present the findings of our study that aimed to explore whether EOS cases differed from healthy controls in terms of total cerebellar volume, volumes of the right and left cerebellar hemispheres and cerebellar volumetric asymmetry.

KEYWORDS

Cerebellum; early onset Schizophrenia; magnetic resonance imaging

References

[1] Kodish I, McClellan JM. Early onset schizophrenia. In: Dulcan, MK, editors, Dulcan's Textbook of Child and Adolescent Psychiatry, 2nd ed. Arlington: American Psychiatric Association Publishing; 2015. p. 389–402.

[2] Andreasen NC, Paradiso S, O'Leary DS. "Cognitive dysmetria" as an integrative theory of schizophrenia: a dysfunction in cortical-subcortical-cerebellar circuitry?. Schizophr Bull. 1998;24:203–218.

[3] Moberget T, Doan NT, Kaufmann T et al. Cerebellar volume and cerebellocerebral structural covariancein schizophrenia: a multisite mega-analysis of 983 patients and 1349 healthy controls. Molecular Psychiatry. 2017:1–9.

[4] Laidia C, d'Albisa M, Wessaf M, Linkef J, Phillips M, Delavest M, Bellivier F, Versace A, Almeida J, Sarrazin S, Poupon C, Le Dudal K, Daban C, Hamdani N, Leboyer M, Houenou J. Cerebellar Volume in Schizophrenia and Bipolar I Disorder with and without Psychotic Features. Acta Psychiatr Scand. 2015;131 (3):223–233.

[5] Andreasen NC, Flashman L, Flaum M et al.Regional brain abnormalities in schizophrenia measured with magnetic resonance imaging. JAMA. 1994;272:1763–1769.

[6] Staal WG, Hulshoff Pol HE, Schnack HG, Hoogendoorn MLC, Jellema K, Kahn RS. Structural brain abnormalities in patients with schizophrenia and their healthy siblings. Am. J Psychiatry. 2000;157:416–421.

[7] Szeszko PR, Gunning-Dixon F, Ashtari M, Snyder PJ, Lieberman JA, Bilder RM. Reversed Cerebellar Asymmetry in Men with First-Episode Schizophrenia. Biol Psychiatry. 2003;53:450–459.

[Abstract: 0765] [Schizophrenia and other psychotic disorders]

Cerebellar volumes in bipolar disorder: data obtained from a different measurement technique

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ABSTRACT

Bipolar disorder is a debilitating psychiatric illness characterized by severe changes in mood. Early onset bipolar disorder is represented by slow response to treatment, persistent mood fluctuations, elevated risk for suicidal attempts, and severe psychosocial impairment.

KEYWORDS

Early onset bipolar disorder; cerebellum; stereology; magnetic resonance imaging;

volumetric measurements; cerebellar asymmetry

Although the pathophysiology underlying the mood-state transitions in bipolar disorder remains largely unknown, many studies of the disorder using imaging techniques have implicated various brain structures such as the amygdala, anterior paralimbic cortices which are central to emotional processing.

Accumulating evidence have recognized the importance of cerebellum in emotional regulation and cognitive activities involving executive functioning, episodic memory and sensorimotor processing in addition to its well-known motor control functions. This feature provides a strong neuroanatomical basis for the involvement of the cerebellum in emotion regulation.

Prior brain imaging studies did not find significantly smaller cerebellar hemispheres compared with healthy subjects in adult bipolar patients. There is very limited data on the early on-set bipolar disorder. Adler et al. reported larger bilateral cerebellar gray matter volume in a mixed sample of adolescents and adults (age range 13–41 y) with bipolar I disorder in first-episode compared to healthy subjects. Controversially, in a longitudinal study, patients with bipolar disorder showed a decrease in cerebellar gray matter density over 4 years, and this decline was more rapid than that observed in healthy control subjects.

Throughout the literature, there are a variety of conflicting results regarding the cerebellar volumetric changes in patients with bipolar disorder. In this presentation findings of our study that aimed to explore whether early onset bipolar cases differed from healthy controls in terms of total cerebellar volume, volumes of the right and left cerebellar hemispheres and cerebellar volumetric asymmetry is going to be shown.

References

[1] Johnson CP, Christensen GE, Fiedorowicz JG, et al. Alterations of the cerebellum and basal ganglia in bipolar disorder mood states detected by quantitative T1ρ mapping. Bipolar Disorders. 2018;20:381–390.

[2] Biederman J, Mick E, Faraone SV, Spencer T, Wilens TE, Wozniak J. Pediatric mania: a developmental subtype of bipolar disorder? Biol Psychiatry. 2000;48:458–466.

[3] Baldaçara L, Nery-Fernandes F, Rocha M, et al. Is cerebellar volume related to bipolar disorder? Journal of Affective Disorders. 2011;135:305–309.

[4] Lin K, Shao R, Lu R, et al. Resting-state fMRI signals in offspring of parents with bipolar disorder at the high-risk and ultra-high-risk stages and their relations with cognitive function. Journal of Psychiatric Research. 2018;98:99–106.

[5] Rapoport M, van Reekum R, Mayberg H. The role of the cerebellum in cognition and behavior. J Neuropsychiatry Clin Neurosci. 2000;12:193–198.

[Abstract: 0575] [ADHD]

Treatment-resistant depression, depression-resisting psychiatry

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ABSTRACT

Treatment-resistant depression is a term that has been used since the 1970s to identify non-response to treatment in people with major depression. However, a common language about identification has not yet been established worldwide. Defined models; it is based on more experience and anecdotal impressions due to insufficient evidence-based data. There are some defined factors that may be associated with treatment-resistant depression: Differences in neuroendocrine and immune system, dysfunctional neuroanatomical circuits, neurotransmitter dysfunction, some clinical predictors (melancholic features, frequent and recurrent episodes, long duration of illness, comorbidity, bipolar-related features), environmental factors, genetic predictors, personality traits are some of them. The most comprehensive study on resistance to treatment in major depressive disorder is the STAR * D study. In the study, it was seen that unresponsiveness to treatment became evident from the third step (after 2 different drugs failed in sufficient dose and time). At this point, although there is a widespread opinion that resistance to treatment may be mentioned, the lack of a structured algorithm that is valid worldwide is evident. Recently, many new treatment methods have been identified and used. Some of those are; rTMU, intravenous/ intranasal ketamine, inhaled nitrous oxide; vagal nerve stimulation, deep brain stimulation and buprenorphine. The most important limitation to the implementation of some of these treatments is the lack of a standardized, ready-to-use treatment-resistant depression staging system. The steps to be taken in this regard will enable the use of new evidence-based, FDA-approved new and more aggressive treatments for treatment-resistant depression patients.

KEYWORDS

Treatment-resistant depression

References

[1] Murphy JA, Sarris J, Byrne GJ. A review of the Conceptualization and Risk Factors Associated with Treatment-Resistant Depression. Depression Research and Treatment. 2017;2017.

[2] Conway CR, George MS, Sackeim HA. Toward an Evidence-Based, Operational Definition of Treatment-Resistant Depression: When Enough Is Enough. JAMA Psychiatry. 2017;74(1):9–10.

[Abstract: 0695] [Sleep disorders]

Microbiota and sleep disorders

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ABSTRACT

Intestinal microbial flora plays critical role in the development and maintenance of brain/mental health. The neuroactive molecules produced by the gut microbiota has been found to modulate neural signals which affect neurological and psychiatric parameters like sleep, appetite, mood and cognition [1]. Approximately 1,000 types of microbiota are present in the adult intestinal tract. The most abundant species belong to the phyla Firmicutes and Bacteroidetes. Other abundant species belong to the Proteobacteria, Actinobacteria, Fusobacteria, Verrucomicrobia, and Cyanobacteria. The delicate balance of the microbiota is very important for health because dysbacteriosis increases the host's susceptibility to disease. There are more than 20 types of enteroendocrine cells in the intestine, which constitutes the largest endocrine organ in the human body [2]. The gut microbiome may affect the hypothalamic-pituitary-adrenal axis via regulating the secretion of neurotransmitters such as cortisol, tryptophan, and serotonin (5-HT). Microbial metabolism produces a variety of neurotransmitters, cytokines, and metabolites such as 5-HT, dopamine, GABA, short chain fatty acids, melatonin, and 90% of the 5-HT in the human body is derived from chromaffin cells in the gastroenteric tract. The intestinal nervous system also forms synaptic connections with the vagus nerve thereby affecting brain function, stress responses, and sleep structure. It is well known that these neurotransmitters are related both to the occurrence of REM sleep and to the development of sleep disorders [1–3]. Evidence suggests some of the microbiota that Clostridiales, Lactobacillales, Firmicutes, and Bacteroidales, show significant diurnal fluctuations that result in time-of-day-specific taxonomic configurations. Researches have shown that corticosterone and melatonin are involved in the interaction of intestinal microbiota with stress and anxiety conditions. Melatonin supplementation significantly increased the richness of the microbial community [2–4]. Stress is also related to sleep disorders. Sleep deprivation, and shift experience /workers change circadian clock gene expression and microbial community structure [5]. There is no doubt that the circadian clock genes are closely related to the development of sleep disorders such as insomnia, hypersomnia. As a result specific microbial taxa are associated with sleep / sleep disorder, and sleep / sleep disorders are also related intestinal microbiota. There have been need more research to identicate relation between each condition.

KEYWORDS

Microbiota; sleep; insomnia

References

[1] Kali A. Psychobiotics: An emerging probiotic in psychiatric practice. Biomedical Journal 2016;39:223–224.

[2] Li Y, Hao Y, Fan F, Zhang B. The Role of Microbiome in Insomnia, Circadian Disturbance and Depression. Front. Psychiatry 2018;9:669. doi:10.3389/fpsyt.2018.00669.

[3] Raybould HE. Gut chemosensing: interactions between gut endocrine cells and visceral afferents. Auton Neurosci Basic Clin. 2010;153:41–46. doi:10.1016/j.autneu.2009.07.007

[4] Chen CQ, Fichna J, Bashashati M, Li YY, Storr M. Distribution, function and physiological role of melatonin in the lower gut. World J Gastroenterol. 2011;17:3888–3898. doi:10.3748/wjg.v17.i34.3888

[5] Reynolds AC, Broussard J, Paterson JL, Wright KP, Ferguson SA. Sleepy, circadian disrupted and sick: Could intestinal microbiota play an important role in shift worker health? Molecular Metabolism 2017;6:12–13.

[Abstract: 0705] [Schizophrenia and other psychotic disorders]

Microbiota and psychotic disorders

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ABSTRACT

Intestinal ecosystem is a complex structure including trillions of microorganisms. This structure consists mostly of bacteria, fungi, viruses, protozoa and many different microbial populations. About 100 trillion microorganisms live in the human body. This number is 10 times more than the total number of human cells and in terms of the number of genes it contains is 150 times more than the human genome. The surface area they cover is 400 m^2 and the bacterial mass is approximately 1.5–2 kg. All these ecological assemblages formed by commensal, symbiotic and pathogenic microorganisms sharing our body and their genetic material are called Microbiome. In humans, the digestive system microbiota begins to form in the period of intrauterine; It has matured over time with the effect of factors such as type of birth, genetic structure, nutrition, lifestyle, antibiotic use, diseases, geographical region and bacteria species. The intestinal microbiota is like a fingerprint and has a unique composition, diversity and functional capability. In healthy people, species with Firmicutes and Bacteroide types are the main factors of intestinal microbiota composition (90%), whereas Proteobacteria, Actinobacteria, Fusobacteria and Verrucomicrobia species are small constituents. Microbiota-intestinal-brain axis is a two-way system between the brain and the gastrointestinal tract. With the discovery of this system, microorganisms, unlike known, are not only diseases; it also revealed that there are many beneficial effects of food digestion, the formation of vitamins, the production of short-chain fatty acids, and the strengthening of the immune system. However, this structure is deteriorated due to intervening antimicrobial treatments, vaccination, disinfecting, dietary changes. In addition, many neuropsychiatric disorders, including autoimmune diseases and metabolic diseases, are being prepared. This interaction between various bacterial species and brain is mediated the immune system response, by the metabolites of vagus nerve, short-chain fatty acid metabolites (acetic acid, propionic acid, butyric acid), enteroendocrine system and tryptophan metabolism.

With the emergence of data on the interaction of the microbiota with the brain, the relationship between anxiety, depression and mental disorders such as schizophrenia has been investigated. Epidemiological studies show that seasonality and urbanization have increased the risk of schizophrenia in many studies in the past. Microbial factors are the focus of efforts to explain these risk factors. With the discovery of Treponema pallidum in the developmental stages of biological psychiatry, it has been clearly shown that a microorganism can lead to a psychotic picture (paralyzed genital) by affecting the brain even after 10–20 years. Based on these observations, it has been suggested that microorganisms may affect the brain through their toxins rather than directly to other parts of the body, such as intestines and sexual organs. Increased psychotic symptoms following viral pandemics suggested that schizophrenia might be a post-viral encephalitis. To date, many microorganisms have been studied with schizophrenia because of their etiology. Protozoans such as Treponema pallidum, Borrelia burgdorferi, Hypersimplex virus, Epstein-Barr virus, Cytomegalovirus, Human immunodeficiency virus and Toxoplasmosis are the main microbial agents studied for this purpose.

As a result, how the microbiota affects human behavior and psychopathology has not yet been elucidated. It is believed that a number of neurotransmitters and neuromodulatory mechanisms, immune responses and genetic interactions are effective. Microbiota research is promising in terms of offering a different perspective on this issue and new treatment goals.

KEYWORDS

Microbiota; microbiome; intestinal-brain axis; schizophrenia; psychotic disorder

References

[1] Turnbaugh, PJ, Ley RE, Hamady M, Fraser-Liggett CM, Knight R, Gordon JI. The Human Microbiome Project. Nature. 2007;449:804–810.

[2] Evrensel A, Ceylan ME. Bağırsak beyin ekseni: Psikiyatrik bozukluklarda bağırsak mikrobiyotasının rolü. Psikiyatride Güncel Yaklaşımlar-Current Approaches in Psychiatry. 2015;7(4):461–472.

[3] Kartalcı Ş. Şizofreni EtyolojisindeMikrobiyotanın Rolü. Psikiyatride Güncel Yaklaşımlar-CurrentApproaches in Psychiatry. 2018;10(2):255–268.

[Abstract: 0713] [Mood disorders]

Microbiota and mood disorders

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ABSTRACT

The human body contains a community of microbial cells and genetic material, known as the microbiome. The gut microbiota is a known modulator of intestinal permeability and can be modulated to improve intestinal barrier integrity in humans. In adults, the gut microbiota consists of approximately 100 trillion organisms. Many recent studies over the last decade

KEYWORDS

Microbiome; microbiota; mood disorders; prebiotic; probiotic; gut-brain axis

have played an important role in recognizing the importance of gut microbiota in brain function. They showed that disruption to gut microbiota can impair physical and mental health. Because of the bidirectional interaction between microorganisms and the brain which affects various CNS activities such as stress response, behavior, and mood, the early life disruptions of the microbiome–gut–brain axis (MGBA) have been associated with an increased risk of developing many of the neuropsychiatric disorders in particular depression and the other mood disorders, autism and schizophrenia later in life, suggesting a link between gut microbiome, neurodevelopment, and disorders. So, it is now clear that the gut microbiota directly or indirectly affects neuropsychiatric illnesses which have collectively cause great burden of disease worldwide. In particular, mood disorders are among the top list, with major depressive disorder ranked the first, and bipolar disorder ranked the fifth. The studies using animal models showed that the dysbiosis factors in the MGBA interfere with emotional regulation, and may result in anxiety and depressive symptoms. However, there are few studies investigating the prenatal effect of drugs on the microbiota, more than a quarter of pharmaceutical drugs have been shown to affect the composition of the microbiota. For example, antibiotics are the most commonly prescribed drugs during pregnancy and reduce the diversity and bacterial load of the microbiome through bactericidal or bacteriostatic actions. Different classes of antidepressant drugs negatively impact bacterial growth. Prenatal exposure to selective serotonin reuptake inhibitors induces anxiety-like and depressive-like behavior during adulthood in rodents. The impact of substance-abuse on the microbiome has not yet been studied. Other pharmaceutical drugs may also alter the gut microbiota through secondary mechanisms of action. Understanding how bacterial commensals are involved in regulating brain function may lead to novel strategies for development of microbiota-based therapies for these neuropsychiatric disorders. Indeed, recent reports of trials using probiotics in healthy subjects demonstrated improvements in depression or anxiety outcome measures. Probiotic treatment has shown efficacy in suppression of animal depression models. Species of Lactobacillus and Bifidobacterium are particularly characterized as antidepressants. Mechanisms involved include attenuation of pro-inflammatory cytokines, regulation of tryptophan metabolism and CNS neurotransmitters. Additional studies are required to substantiate the clinical use of probiotics, prebiotics and Fecal Microbiota Translocation (FMT).

References

[1] Kuo P-H, Chung Y-CE. Moody microbiome: Challenges and chances. Journal of the Formosan Medical Association; 2018, doi:10.1016/j.jfma.2018.09.004

[2] Bruce-Keller AJ, Salbaum JM, Berthoud HR. Harnessing Gut Microbes for Mental Health: Getting from Here to There. Biological Psychiatry February 1. 2018;83:214–223.

[3] Liu L, Zhu G. Gut–Brain Axis and Mood Disorder. Front. Psychiatry. 2018;9:223. doi:10.3389/fpsyt.2018.00223

[Abstract: 0312] [Personality disorders]

Antisocial personality disorder and comorbidities

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ABSTRACT

Antisocial personality disorder (ASPD) is a pathology which has gained clinical significance as a personality pattern characterised by an absence of empathy and associated abuse of the right of others, repeated crimes, no feeling of regret and not learning from events, all kinds of manipulative behaviours which are superficial and removed from reality, compulsions accompanied by irritability and an irresponsible lifestyle at a noticeable level. In these cases, which can be easily confused with various forensic crimes and can be extremely life-threatening to social order, the most common comorbid condition is the abuse of psychoactive substances. The incidence of ASPD has been reported as 18%-40% in patients using psychoactive substances. Previous studies have shown that use of psychoactive substances is 13-fold higher in patients with ASPD, the most common diagnosis combination is of abuse of psychoactive substances, and in patients with severe mental disorders there is a relationship between a significant increase in behavioral problems and the rate of substance use of those with a co-diagnosis of ASPD. Other than abuse of psychoactive substances, one of the diagnoses often related to ASPD is Attention-deficit/ hyperactivity disorder (ADHD). In recent years there has been increased awareness of the diagnosis of ADHD both in children and adults. Just as in personality disorders, this disorder manifests with a range of symptoms from childhood onwards, and it is an extremely commonly encountered comorbidity, seen in children and adolescents at 5% and in adults at 4%.

KEYWORDS

Antisocial personality disorder; behavioral problems; comorbidities

Although the basic symptoms of this disorder are impulsivity, activity and attention problems, a series of behavioural problems, which are also seen in ASPD, such as aggression, novelty seeking behaviour, substance use, and behaviours that are harmful to the self or the environment, are frequently seen findings in this disease. The presence of these common clinical findings can sometimes create diagnostic confusion. It is also known that cases diagnosed with ADHD in childhood are more often diagnosed in young adulthood with psychoactive substance abuse and ASPD. As psychoactive substance abuse is frequently seen both in ASPD and adult ADHD, this creates problems in the diagnosis and treatment of this disorder. In particular, as impulsivity, which is one of the typical findings of ASPD, is also one of the basic symptoms of ADHD, it is of importance in respect of the combination with substance abuse. Furthermore, it has been reported that substance use starts at an earlier age in patients with ASPD and ADHD combination. In addition, comorbidities with ASPD such as primarily impulse control disorders, intermittent explosive disorder, pyromania, and pathological gambling can be seen together with several axis 1 psychiatric diseases. For example, the incidence of lifelong depression in ASPD has been found to be 34%. Follow-up studies of young patients diagnosed with major depression have reported that these patients have antisocial personality characteristics in early adulthood at a rate 10-fold higher than those without depression. As a result of studies conducted on large samples of patients with ASPD, it has been reported that rates of depression are 2-fold higher when there is substance use and addiction, a low economic level and marital or family problems.

References

[1] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.
[2] Glenn AL, Johnson AK, Raine A. Antisocial personality disorder: a current review. Curr Psychiatry Rep. 2013;15:427.
[3] Regier DA, Farmer ME, Rae DS, et al. Comorbidity of mental disorders with alcohol and other drug abuse. Results from the Epidemiologic Catchment Area (ECA) Study. Jama. 1990;264:2511–2518.

[Abstract: 364] [Personality disorders]

Antisocial personality disorders and forensic problems

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ABSTRACT

Antisocial personality disorder (ASPD) is characterized by impulsivity, difficulty in anger control, self-harm, self-esteem, and other psychiatric disorders such as alcohol and substance abuse. Criminal behavior, normative social order, and many criminal acts as well as breaking the laws are caused by the influence of more than one factor. Individuals who exhibit antisocial and violent behaviors are splendid, arrogant, emotional, dominant, superficial and manipulative. They reflect their sensuality in a short time, do not form strong emotional bonds with others and do not build empathy. Feelings of being right, impulsivity, general behavioral barriers, power and control needs, being asocial and antisocial are effective in committing crimes. Although many studies have shown that the rate of violent behavior in the community is not higher than that in patients with severe mental disorder (schizophrenia), this rate is significantly higher in patients with psychiatric comorbidity and addiction. These patients present a high risk of repetitive violent behavior. Substance abuse is common in individuals with ASPD. These individuals often tend to misuse substances during the crime-handling behavior. The most important factors that lead to criminal activity are having an antisocial group of friends, acquiring negative behaviors that lead to crime and using psychoactive substances. Benzodiazepine use also increases violent behavior in individuals with ASPD. Early onset alcohol abuse increases the effect of childhood adult behavior disorder on adult antisocial behavior in young adults. Alcohol and substance misuse in the family environment and in the environment where the individuals diagnosed with ASD are living, domestic violence, past criminal behavior, aggressive behavior towards oneself and others is associated with criminal conduct. These individuals have high levels of anger and self-esteem. The comorbidity of ASPD and Borderline Personality Disorder is mediated between childhood behavioral disorders and adult violent behavior. Individuals with ASPD differ from other psychiatric disorders when examined in terms of their crime characteristics. These individuals frequently commit crimes against theft, fraud, kidnapping and extortion, and their criminal capacity is complete. Homocidal behaviors can be observed in criminal antisocial individuals. These behaviors occur under the influence of substance abuse. Neuroimaging studies have shown a lower response time in the brain areas involved

KEYWORDS

Antisocial; antisocial personality disorder; crime; criminal behavior

in cognitive control, attention, language, and emotion processing, such as anterior cingulate cortex, dorsolateral prefrontal cortex, superior temporal cortex and postentral cortex, putamen, thalamus and amygdala. These regions are associated with aggression, impulsivity and lack of empathy. Antisocial personality characteristics decrease over time. However, comorbidities such as concomitant substance abuse, psychotic disorders, attention deficit and hyperactivity disorder may prolong this period. As a result, there are deficiencies in the inter-family relations of individuals with ASPD. The environment in which they grow, cause illegal behaviors such as criminal behavior and substance abuse. Even if they are aware of the consequences of their violent behavior, they cannot prevent themselves from committing a crime. Therefore, they maintain this attitude and behavior for a long time. These behaviors tend to decrease gradually over time. These stuations should be considered for clinicians working with these individuals.

References

[1] de Tribolet-Hardy F, Vohs K, Domes G, Regli D, Habermeyer E. [Violent offenders with or without antisocial personality disorder. A comparison]. Nervenarzt. 2011;82(1):43–49.
[2] Howard R, McCarthy L, Huband N, Duggan C. Re-offending in forensic patients released from secure care: the role of antisocial/borderline personality disorder co-morbidity, substance dependence and severe childhood conduct disorder. Crim Behav Ment Health. 2013;23(3):191–202.
[3] Palijan TZ, Radeljak S, Kovac M, Kovacević D. Relationship between comorbidity and violence risk assessment in forensic psychiatry - the implication of neuroimaging studies. Psychiatr Danub. 2010;22 (2):253–256. Review.
[4] Palmstierna T. [Personality disorders, violence and criminal behaviour]. Lakartidningen. 2016;113. pii: DY6R.
[5] Pondé MP, Caron J, Mendonça MS, Freire AC, Moreau N. The relationship between mental disorders and types of crime in inmates in a Brazilian prison. J Forensic Sci. 2014;59(5):1307–1314.

[Abstract: 0371] [Personality disorders]

New developments and approaches in antisocial personality disorder

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ABSTRACT
Beginning from the early adolescent period, Antisocial Personality Disorder (ASPD) has devastating effects on the social and mental functionality of an individual. It is characterized by a behavior pattern of irresponsibility, impulsivity, guiltless and deception of others. Having the lifetime prevalence of ranging from 2% to 4%, the disorder is more common in male individuals. Different from the other personality disorders, criminal behavior is so close to the definition and thus the disorder arouses interest of public health. Childhood onset conduct disorder is associated with ASPD later on and epigenetic factors have a great impact on the disorder. Psychopathy is a distinct clinical definition. Individuals who score high points in Hare Psychopathy Checklist meet criteria for APSD but on the other hand, approximately % 10 of the ASPD individuals meet criteria for psychopathy [1]. Current literature available is reviewed and optimal results are obtained through clinical expertise. Despite it is a public health concern, limited data about the disorders is available in the literature. The disorder is associated with higher rates of comorbid mental disorders, mortality and morbidity. ASPD is common in prison settings and substance misuse/dependence is probably the most common co-occurring disorder with ASPD. Individuals with ASPD were three to five times more likely to misuse alcohol or illicit drugs when compared to other population. Genetic, imagining and studies focused on autonomic reactivity have been made, and a specific interest has been focused on the frontal lobes. Individuals with ASPD tend to externalize their difficulties and so, they rarely appeal to the healthcare settings. They are commonly sent for evaluation by some formal authority settings or have been forced into treatment by a relative. Knowing these "resistances for help" attitude in ASPD, therapeutic interventions become more difficult when compared with the other mental disorders. Also, comorbidity is associated with worse prognosis and treatment outcomes [2,3]. Recent advances in etiology and description of the Antisocial Personality Disorder will be carefully evaluated and current treatment approaches will be highlighted in this session.

KEYWORDS
Antisocial personality disorder; definition; treatment

References

[1] Black DW. The Natural History of Antisocial Personality Disorder. Can J Psychiatry. 2015;60 (7):309–314.
[2] Antisocial Personality Disorder: Treatment, Management and Prevention. Chapter II. The British Psychological Society & The Royal College of Psychiatrists; 2010.
[3] Goodwin D, Guze SB. Sociopathy (antisocial personality). In: Goodwin D, Guze SB, editors. Psychiatric diagnosis. 4th ed. New York (NY): Oxford University Press; 1989. p. 209–225.

[Abstract: 0779] [Others]

Evidence based therapies and non-medical treatments in ASD

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ABSTRACT

The definition of autism has evolved during the years. In the latest version of Diagnostic and Statistical Manual of Mental Disorders (DSM V); there is only one diagnostic classification, termed Autism Spectrum Disorder (ASD). In the DSM V, ASD is identified by two primary diagnostic markers: difficulties in social communication and restricted or repetitive behaviors and interests. Examples of difficulties in social communication include challenges in social reciprocity, non-verbal social behaviors, and establishment of social relationships. Restrictive and repetitive behaviors include stereotypic behavior or speech, excessive adherence to routines, and highly fixated interests. The increased prevalence of ASD, with an actual estimate of 1 in 59 children. *(Centers for Disease Control and Prevention*, 2018) has intensified the demand for effective educational and therapeutic services, and intervention science is now providing evidence about which practices are effective. In the latest National Professional Development Center on ASD (NPDC), a total of 456 studies were reviewed to outline practices that have sufficient empirical support to be termed "evidence-based" (EBP). Twenty-seven practices met the criteria for being evidence-based. The evidence-based practices consist of interventions that are fundamental applied behavior analysis techniques (e.g., reinforcement, extinction, prompting), assessment and analytic techniques that are the basis for intervention (e.g., functional behavior assessment, task analysis), and combinations of primarily behavioral practices used in a routine and systematic way that fit together as a replicable procedure (e.g., functional communication training, pivotal response training). Also, the process through which an intervention is delivered defines some practices (e.g., parent-implemented interventions, technology-aided interventions). PACT (Paediatric Autism Communication Therapy) therapy comprises some of these evidence based practices, it is a parent implemented , naturalistic and video-aided interventions, in which techniques as modelling and prompting are used. PACT therapy is the first autism intervention rigorously tested to show sustained impact on reduction in autism symptoms (Pickles et al. 2016). PACT has proven sustained effect on significantly reducing the difficulties in social, communication and repetitive behaviour/ restricted interests and improving communication initiation skills in the long-term (6 years after treatment end). PACT is efficient, relatively low cost on professional resources. PACT therapists train and supervise parents/carers embedding therapy in the child's naturalistic interactions within family life, leading to 'real world validity', generalisation and impact on child daily life and functioning. The PACT therapy is included in evidence for the NICE Clinical Guideline 170 (NICE 2013) and one of the leading evidence-based interventions for autism in the National Autism Project review, (2017). PACT is now a recommended intervention for core autism symptoms within the national Department of Health 'Improving Access to Psychological Treatment' (IAPT) curriculum for Autism and is being disseminated internationally.

KEYWORDS

Autism; evidence-based; therapy; PACT

References

[1] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.
[2] Wong, C, et al. Evidence-Based Practices for Children, Youth, and Young Adults with Autism Spectrum Disorder. Autism Evidence-Based Practice Review Group Frank Porter Graham Child Development Institute University of North Carolina at Chapel Hill; 2014.
[3] Pickles, et al. Parent-mediated social communication therapy for young children with autism (PACT): long-term follow-up of a randomised controlled trial. The Lancet. 2016;388:2501–2509.
[4] National Autistic Society. 2017. National Autism Project review. www.nationalautismproject.org.uk

[Abstract: 0659] [Autism]

Non-evidence based FAD treatments in ASD

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ABSTRACT

The prevalence of autism spectrum disorder (ASD) increases 123% in the last decade according to the last surveys. The increase in the prevalence of autism is astounding, as is the responsibility placed on parents and professionals to identify effective treatments for individuals with autism. A range of treatment approaches including relationship-based treatments, skill-based treatments, physiologically oriented interventions, psychopharmacologic treatments and combined programs were reviewed in the literature which revealed some treatments had supporting evidence, while others were understudied or had evidence indicating that the treatment was ineffective or even potentially harmful. Many terms have been used to describe treatments which have not been exposed to rigorous scientific study, including 'fad', 'controversial', 'alternative' and 'pseudoscientific'; however, for the most part these refer to treatments that are unsubstantiated or nonevidence based. Although the increasing rates of autism are staggering, the desperation of families to find the right treatment for their children can be overwhelming. This sense of urgency can increase parents' susceptibility to embracing fad treatments. Fad treatments, by definition, have no substantial body of research showing that they are effective in treating any aspect of autism. Fad treatments are interventions that can be based on anti-science or pseudoscience, which use scientific jargon, sound logical, are supported by celebrities, and are discussed in the media and on the internet where many parents can be easily exposed to them; however they become popular quickly, spread fast and often disappearing later frequently because of their reported harmful side effects or inefficiency. The use of such treatments not only waste time and money, but also prey upon the emotional vulnerability of parents and caregivers. From all that is known about the importance of early intervention to ameliorate the defects caused by autism, time spent on unproven treatments delay the implementation of therapy that can actually make a difference on the life-long consequences. So it gets important to educate parents and professionals in terms of treatment options, differentiation between a reliable intervention and a fad, or validation of an intervention scientifically, or criteria for valid evidence of effectiveness. In the literature of fad treatments in ASD, 'the biomedical treatment options' such as the diet and nutrition therapies of gluten-free and casein-free diets, the candida diet, the nutritional deficiency diet, the ketogenic diet, chelation therapy, intravenous immunoglobulin therapy, secretin therapy and use of folic acid, omega-3 or Vitamin B6-magnesium; 'speech and language therapies' such as the Fast Forward Program, the Hanen method, Lindamood-Bell learning processes, and SCERTs model; and some other complementary treatment options such as auditory integration training, craniosacral therapy, dolphin-assisted therapy, art therapy, music therapy, pet facilitated therapy, facilitated communication training, holding therapy, sensory integration therapy, vision therapy and etc. were widely discussed. In this presentation, number of issues including the conflicted and controversial results of established researches about nonevidence-based fad treatments, reported harmful side effects of fad treatments, the factors that influences parents and professionals' choices of treatment and comparison of them with evidence-based treatments in terms of cost and life-long results will be discussed.

KEYWORDS

ASD; non-evidence based treatments; fad; controversial; pseudoscientific; unsubstantiated

References

[1] Tuzikow J, Holburn S. Identifying Fad Therapies for Autism Spectrum Disorders and Promoting Effective Treatment; 2011. p. 307–319.

[2] Metz B, Mulick JA, Butter EM. Autism: A late-20th-century fad magnet. Controversial therapies for developmental disabilities: Fad, fashion and science in professional practice; 2005.

[3] Zane T, Davis C, Rosswurm M. The cost of fad treatments in autism. Journal of Early and Intensive Behavior Intervention. 2008;5(2):44.

[Abstract: 0747] [Others]

Recent genetic developments in ASD

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ABSTRACT
Autism spectrum disorder (ASD) is a neurodevelopmental disorder that manifests problems in social communication and interactions along with restricted interests or repetitive behaviors. It is estimated that the worldwide prevalence of ASD is about 1% in the general population. The origin of ASD is elusive, however growing evidence have indicated that genetic factors play a prominent role in the etiology. Family and twin studies reveal a strong genetic contribution. The concordance rate of autistic disorders in monozygotic twins is 70–90% while in dizygotic twins is 10–30%. There are approximately a hundred genetic disorders manifest features of ASD, like Fragile X and Rett syndromes. Cytogenetic studies indicated candidate regions that increase the risk for ASD like 15q11-q13. Common copy number variants in patients with ASD comprise 1q21.1,16p11.2, , 16p13.1, 17p11.2, 22q11.21 Furthermore, genetic studies detected hundreds of gene contribute to ASD risk like SYNGAP1, ADNP, NLGN4X, NRXN1, KATNAL2, GABRB3, GABRA5, CHD8, BAF155, SHANK2, SHANK3, MECP2,FMR1, DYRK1A are some of the associated genes with ASD. Several genetic variants may also increase ASD risk. However some results are conflicting and each risk gene or copy number variation implicated in ASD accounts for only <1% of cases. These findings indicate significant genetic heterogeneity. Siblings can carry different penetrant mutations. Results show that multiple genetic risk factors contribute to the etiology of ASD. Research reveal that most of the risk genes appear to be involved, in synaptic morphology, activity and plasticity. ASD-risk genes are enriched in broader functional groups consisting of RNA processing, and transcriptional regulation. Recent findings indicate critical roles of immune dysfunction and epigenetics. The elucidation of risk genes for ASD has provided important parts for the autism puzzle. However, causal role of the results is still elusive. .In this presentation, recent genetic findings regarding the etiology of ASD and possible future gene therapy options will be discussed.

KEYWORDS
Autism spectrum disorder; genetics; SNP; CNV

References

[1] Wiśniowiecka-Kowalnik B, Nowakowska BA. Genetics and epigenetics of autism spectrum disorder-current evidence in the field. J Appl Genet. 2019;60(1):37–47.
[2] Vorstman JAS, Parr JR, Moreno-De-Luca D, Anney RJL, Nurnberger Jr JI, Hallmayer JF. Autism genetics: opportunities and challenges for clinical translation. Nat Rev Genet. 2017;18(6):362–376.
[3] Woodbury-Smith M, Scherer SW. Progress in the genetics of autism spectrum disorder. Dev Med Child Neurol. 2018;60(5):445–451.

[Abstract: 0668] [Mood disorders]

Recent genetic developments in mood disorders

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ABSTRACT
Depression and bipolar disorder are complex disorders with a heritability of 37% and between 40–70% estimated from twin studies. Despite robust evidence for a genetic component, identifying the specific genetic variants involved in mood disorders have been a major challenge. According to the studies, several sets of variants or genes possibly associated with risk factors, brain structure and functions, response and tolerability to drugs in these disorders. At the beginning of this new era, many results have not been confirmed yet and clinical applications are still missing. In this presentation, recent genetic findings and developments focusing primarily on last five years will be discussed.

KEYWORDS
Depression; bipolar; GWAS; SNP; genetic

[Abstract: 745] [Mood disorders]

Current data on genetic and neurobiology of pediatric bipolar disorder

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ABSTRACT
Although bipolar disorder has been widely studied in adults, early-onset forms of the disease have started to attract attention. Recent neurobiological studies have raised hopes that it

KEYWORDS
Bipolar disorder; genetic; neurobiology

may be possible to identify biomarkers of BD. Such biomarkers would improve both the diagnosis and assessment of BD. Genetic studies of bipolar disorder is carried out by family and twin studies. Twins provide a valuable opportunity to look at the factors involved in human genetics. Family studies have consistently found a higher rate of bipolar disorder among the relatives of early-onset bipolar disorder patients than in relatives of later-onset cases, which supports the notion of a larger genetic contribution to the early-onset cases. [1] In addition to inherited genomic variants, recent evidence supports a significant role of de novo protein-damaging mutations in psychiatric disorders. Genome-wide association studies reveal the single nucleotide polymorphisms (SNPs) found in CACNA1C, ODZ4 and NCAN genes as promising candidate genes for bipolar disorder. The SNP rs1006737 in the gene CACNA1C is the most replicated and most studied common genomic variant associated with bipolar disorder to date. The A allele seems to increase the risk of bipolar disorder in some population subgroups, ODZ4 is a large transmembrane protein and its structure resembles signal transduction molecules. During brain development, ODZ4 appears to play a central role in the regulation of neuronal and synaptic connectivity. The common variant rs12576775 in the intron of gene ODZ4 has been associated with bipolar disorder. Researchers have studied the effect of rs12576775 in individuals with bipolar disorder and also in healthy individuals using functional and structural brain imaging. [2] Neuroimaging studies are performed in the form of anatomical and functional brain imaging. Neuroimaging studies indicate frontotemporal and frontostriatal pathology. In recent years, the development of two new neuroimaging techniques (diffusion tensor imaging MRI and fMRI connectivity analyses) has made it possible to investigate neural connectivity in BD. Some neuroimaging studies of BD have led to the formulation of neurobiological models based on dysfunctional connectivity between prefrontal and subcortical regions. With the increasing power of genetics and the new techniques used in neuroimaging, a new field, called imaging genetics, has emerged in recent years. Imaging genetics assesses the impact of allelic variation on brain structure and function. For example HTT promoter polymorphism has been associated with susceptibility to mood disorders in stressful conditions. Brain imaging studies exploring the impact of the s-allele have repeatedly demonstrated that this risk allele increases the reactivity of the amygdala to negative emotional stimuli. [3] Because of bipolar disorder is a complex and multifactorial disorder new studies are needed to understand the underlying mechanism.

References

[1] The Genetics of Pediatric-Onset Bipolar Disorder, 2003 Society of Biological Psychiatry. doi:10.1016/S0006-3223(02)01893-0

[2] Genetics of bipolar disorder, February 2014, The Application of Clinical Genetics: 33–42. doi:10.2147/TACG.S39297

[3] Neuroimaging biomarkers in bipolar disorder January 2012 Frontiers in bioscience (Elite edition) 4(2):593–606. doi:10.2741/40

[Abstract: 0649] [Mood disorders]

Current evidence for prodromal bipolar disorder and interventions

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ABSTRACT

Bipolar disorder (BD) is a common, chronic and highly morbid illness characterized by manic and depressive episodes, which often runs a relapsing and remitting course. With its onset in adolescents and young adults, BPD can severely derail young people from their normal developmental trajectory resulting in dropping out of education, work, relationships and returning them to a dependency on their parents. This can trigger additional mental health problems which are aggravated if there are premorbid adjustment difficulties, post manic depressive symptoms and cognitive impairment. BD is more prevalent than schizophrenia and affects 2% of adolescents. Although the schizophrenia prodrome has been well described, prodrome onset has been explored less in BD, despite substantial evidence that many people experience prodromal symptoms before the onset of BD. Currently, there is no consensus on the definition of the bipolar prodrome. The initial prodrome has been described as the time interval from the onset of the first noticeable symptoms and signs that deviate from a relatively stable or normal state of being to the onset of a fully developed and diagnosable disorder. Although initial attempts to define and describe early or prodromal BD have focused on clinical findings, more recent studies have considered a combined phenomenological and biological approach. There is evidence that early-stage BD

KEYWORDS

Bipolar; prodrome; risk syndrome

has significant biological and genetic correlates. When the clinical features of patients with prodromal BD were examined, studies of patients with established bipolar disorder indicate that the majority of bipolar patients experience subthreshold manic or depressive symptoms including elevated or irritable mood, racing thoughts, rapid speech, depressed mood, anhedonia, and thoughts of suicide or self-harm. Length of delay to first treatment is correlated with poorer longitudinal outcomes, including severity of depression, number of days depressed, number of euthymic days, number of episodes, and days of ultradian cycling. A judicious early intervention is the ultimate goal of identifying a BD prodrome, but there have been few studies examining primary or secondary prevention. A clinical staging model for algorithmic treatment endorses safer interventions, e.g., omega-3 fatty acids and family therapy in the prodromal phase, and then stepping up to pharmacologic agents for secondary prevention. Psychosocial interventions have been shown to lower a conversion rate after a 1-year follow-up. Family- Focused Therapy for High-Risk Children and Interpersonal and Social Rhythm Therapy, which targets social and sleep patterns, are two such programs, and a protocol for early cognitive-behavioral therapy specific for subjects at risk for BD is under evaluation. A pharmacological intervention is limited by the untested efficacy of medications in the prodromal or early phases. In this presentation, data regarding the current evidence for prodromal bipolar disorder and interventions will be discussed in the light of the recent literature.

[Abstract: 0673] [Mood disorders]

Current evidence for treatment and prognosis of pediatric bipolar disorder

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ABSTRACT

Bipolar Disorder (BD) in childhood is associated with significant morbidity and mortality, including psychiatric hospitalization, suicide ideation, suicide attempts, substance abuse, as well as academic and social behavior problems. Accurate assessment of BD is important for allocating appropriate treatment, but it is complicated by significant heterogeneity in symptom presentation and high rates of comorbidity. A recent meta-analysis of epidemiological of children and adolescents with BD published that the overall rate of BD was 1.8 %. Up to 65% of individuals with BD have illness onset before age 18, and 28% before age 13. The average onset of symptoms to diagnosis and therapy lasts 10 years which arises the need for early diagnosis and prompt treatment of symptoms. Longitudinal prospective studies are in early stage of seeing how BD children and adolescents develop into adulthood. Recent data suggests that episodic and chronic irritability in children and adolescents predict different outcomes in adulthood. Given high rates of genetic predisposition for BD, assessment of youth should focus on obtaining accurate family history of this illness. Poor outcomes were associated with low socioeconomic status, early onset and family history of BD and depressive disorder, among other factors. The current recommendations for the treatment of pediatric BD is primarily for psychopharmacological agents combined with psychosocial interventions Substantial increase in clinical trials pharmacological agents for BD in child and adolescents. Of 19 studies examining treatment of bipolar disorder, 15 reported on intermediate and 11 on effectiveness outcomes. Longitudinal Assessment of Manic Symptoms (LAMS) study results show that that, among a sample of youth selected for elevated symptoms of mania, manic symptoms decreased over a 24-month period for most youth. The Course and Outcome of Bipolar Youth Study (COBY) found that, BD diagnosed youth , 82% recovered from their index mood episode after 2.5 years; however, 1.5 years after recovery, 63% experienced recurrence. Monotherapy is generally recommended for the management of BD in childhood, combination therapy is often used due to partial response to treatment with a single agent Lithium and atypical antipsychotics (risperidone, olanzapine, quetiapine, aripiprazole, ziprasidone, asenapine) are generally effective and safe for the short-term management of acute manic/ mixed state. The depressive phase of BD in youth is more difficult to treat pharmacologically due to limited data available on the management of bipolar depression and to increased risk of mania with antidepressant treatment. Recent clinical study, lurasidone decreased depressive symptoms in youth with BD but quetiapine did not separate from placebo in depressed children with BD although it is different in adults. In this presentation, we will summarize BD prognosis and recent treatment modalities in childhood with all details.

KEYWORDS

Treatment; prognosis; pediatric; bipolar disorder

[Abstract: 0637] [Others]

New promises and new expectations from rTMS

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ABSTRACT

Transcranial magnetic stimulation (TMS) is a generally well tolerated and reliable procedure that uses magnetic fields to stimulate nerve cells in the brain. TMS uses electromagnetic stimulation to produce transient electric fields in the local cerebral cortex and this process causes depolarization and firing of local neurons. Although the use of TMS in medicine started 30 years ago [1], there has been a significant increase in the studies of this non-invasive brain stimulation technique (neuromodulation) over the last 10 years. TMS has the potential to investigate both the physiology and physiopathological processes of neural structures the, to be used in the treatment of many diseases and with the introduction of navigated transcranial magnetic stimulation to enable the activation / inactivation of functional cortical areas in certain anatomical regions are some of the reasons that explain the researchers' interest in TMS. The FDA approved the use of TMS in 2008 for depression, migraine headache in 2013 and recently for the treatment of obsessive-compulsive disorder (2018). In recent years TMS has been investigated as a treatment option for various neurological and psychiatric disorders. For example, many neuropsychiatric diseases such as stroke, treatment-resistant epilepsy, multiple sclerosis, tinnitus, conversion disorder, attention deficit and hyperactivity disorder, autism spectrum disorder, dementia, post-traumatic stress disorder, substance use disorders, dystonia and aphasia can be demonstrated [2]. Critical points about their use in these diseases; firstly determining which region of the brain should be stimulated, deciding how to change the function to make it compatible with the normal function of the brain (activation, inactivation), determining the frequency and pattern of stimulus to be applied, detection of the duration and recurrent doses that the application should continue to maintenance the remission. Different combination of these mentioned parameters during treatment applications, allowing rTMS to be used in the treatment of different diseases as alternative therapeutic choice. For example, it is reported that rTMS at low frequencies may cause cortical excitability suppression on target cortical tissue, whereas at frequencies above 5Hz may cause prolonged prolongation of cortical excitability. In addition, alternative modalities such as theta burst stimulation increase the frequency of the stimulus and make the application time shorter. Although Transcranial Magnetic Stimulation has been shown to be effective in treating the symptoms of various neurological and psychiatric disorders, there is a need for further research by comparing with large sample sizes and standard treatments.

KEYWORDS

RTMS; theta-burst; treatment of psychiatric disorders; efficacies; efficiency

References

[1] Barker AT, Jalinous R, Freeston IL. Non-invasive magnetic stimulation of human motor cortex. Lancet. 1985;1:1106–1107.

[2] Habib S, Hamid U, Jamil A, et al. Transcranial Magnetic Stimulation as a Therapeutic Option for Neurologic and Psychiatric Illnesses. Cureus. 2018;10(10):e3456.

[Abstract: 0744] [Psychotherapy]

Introduction to acceptance and commitment therapy

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ABSTRACT

ACT (Acceptance and Commitment Therapy) is a psychotherapy approach based on behavioral therapy traditions, supported by functional analysis of human language and Relational Frame Theory (RFT). There are more than 250 randomized controlled trials show effectiveness of ACT on several clinical conditions since 1986. ACT model is defined psychopathology as "psychological inflexibility", and goals of therapy is to improve "psychological flexibility" which means to increase functionality and valued-living. Psychological flexibility model includes six core process which interacts with each other. From ACT perspective, the purpose

of therapy is to focus on acceptance of inner experiences rather than reduce or control them and to help the clients act more functional and flexible way toward their values. Aim to this workshop is to introduce ACT model and some of the key concepts in ACT. After general theoretical information on behavioral therapy, the principles of contextual behavioral methodology and ACT will present. We plan to describe the concepts of psychological flexibility and experimentally retrieve the six processes of the hexaflex.
Look through ACT: K. Fatih Yavuz, MD
See with ACT: Zülal Çelik, MD
Educational Objectives:
1. Describe the concept of contextual behavioral methodology and ACT,
2. Describe behavioral analyses and ACT principles, experimentally retrieve six core processes,
3. Learn to formulate ACT-based behavior analyses,
4. Learn interventions to apply in their clinical practice through experimental exercises.
Components: Conceptual analysis, Didactic presentation, Experiential exercises, Role play

References

[1] Hayes SC, Villatte M, Levin M, Hildebrandt, M. Open, aware, and active: Contextual approaches as an emerging trend in the behavioral and cognitive therapies. Annual review of clinical psychology, 2011;7.
[2] Harris R. ACT made simple: An easy-to-read primer on acceptance and commitment therapy. New Harbinger Publications; 2009.
[3] Yavuz KF. Kabul ve Kararlılık Terapisi (ACT): Genel Bir Bakış. Turkiye Klinikleri Journal of Psychiatry Special Topics. 2015;8(2):21–27.

[Abstract: 0807] [Others]

The effects of antipsychotics on hormone and hematological system

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ABSTRACT
Antipsychotics cause many side effects such as extrapyramidal system, metabolic, endocrine, hematological system related side effects. Hematological side effects such as leukopenia and neutropenia could occur during treatment with atypical antipsychotics usually. These side effects could lead to life threatening situations and the mortality rate due to drug related agranulocytosis is about 5–10%. There are several hypotheses describing the mechanisms. Clozapine is the antipsychotic agent which has been most commonly associated with agranulocytosis. Patients who had hematological side effects during their previous antipsychotic drug treatments and who had lower baseline blood leukocyte counts, have higher risk to develop leukopenia or neutropenia during their current antipsychotic treatment. Once leukopenia and neutropenia develop, drugs thought to be responsible for this side effect should be discontinued or dosages should be lowered. Clinicians should avoid any combination of drugs known to cause hematological side effects. The endocrine side effects of antipsychotic drugs can limit their use. Especially atypical antipsychotics can cause many side effects such as those related with thyroid, blood sugar, level of sex hormones, growth rate and bone metabolism. Clinicians should present multidisciplinary approach to endocrine and hematologic side effects due to antipsychotic use and avoid multiple drug use in such patients.

KEYWORDS
Antipsychotics; side effects; hematological; endocrine

References

[1] Correll CU, Carlson HE. Endocrine and metabolic adverse effects of psychotropic medications in children and adolescents. J Am Acad Child Adolesc Psychiatry. 2006;45:771–791.
[2] Jerrell JM, McInytre RS. Adverse events in children and adolescents treated with antipsychotic medications. Hum Psychopharmacol Clin Exp. 2008;23:283–290.
[3] Flanagan RJ, Dunk L. Haematological toxicity of drugs used in psychiatry. Hum Psychopharmacol. 2008;23 (Suppl. 1):27–41.

[Abstract: 0717] [Psychopharmacology]

Effects of antipsychotics on metabolic system

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ABSTRACT

After the discovery of the chlorpromazine in 1950's, typical antipsychotics (D_2 antagonists) were started use for the treatment of schizophrenia and other psychotic conditions and with clozapine atypical antipsychotics ($5HT_{2A}/D_2$ antagonists) level upped. Although atypical antipsychotics (AAP) have low incidence of EPS and hyperprolactinemia and more effective in the treatment of negative, cognitive and affective symptoms of schizophrenia, AAP's like clozapine and olanzapine have more metabolic side effects like weight gain, dyslipidemia, Diabetes Mellitus (DM) and Metabolic Syndrome (MetS) than typical ones. İt's reported that the prevalence of MetS differs %32-68 in schizophrenia patients taking antipsychotics. MetS can be defined as three or more of; elevated blood pressure (>130/85 mmHg), elevated fasting glucose (>100 mg/dl), dyslipidemia (TG > 150 mg/dl; HDL cholesterol women <50 mg/dl, men <40 mg/dl) and central obesity (change due to ethnicity, women >80, men >94 in europid) and insulin resistance is one of the major underlying contributors to the MetS [1]. MetS is a suitable predictor for metabolic and cardiovascular diseases (CVD), because the definition of MetS contains most of the metabolic parameters. Studies demonstrated that patients with the MetS are three times more likely to have a cardiovascular disease and the risk for diabetes is up to fivefold higher in patients with the syndrome. Furthermore, the presence of the syndrome is associated with a significant increase in cardiovascular mortality. Serotonin receptors like $5HT_{2C}$, $5HT_{2A}$, H_1 histamine and M_1 muscarinic receptors in CNS mediate the metabolic effects of antipsychotics rather than dopamine receptors. Although CNS receptors mediate the metabolic effects of antipsychotics, neurotransmitters like dopamine and serotonin have receptors on peripheral tissues and these receptors enhance the metabolic effects of antipsychotics by reduction of leptin and adiponectin levels and raising adipogenesis, lipid accumulation, inflammatory cytokines and free fatty acids. İn a study comparing patients taking clozapine and less obesogenic antipsychotics (aripiprazole, haloperidol, amisulpiride and ziprasidone) and non-psychiatric group due to metabolic parameters and orexin-A levels; the prevalence of MetS was 36%, 20%, and 10% of the participants in the clozapine, less obesogenic antipsychotic, and non-psychiatric control groups, respectively. Study results revealed that the orexin-A level was upregulated in patients with schizophrenia treated with antipsychotics, especially for the group taking less obesogenic antipsychotics and higher orexin-A levels were independently associated with better metabolic profiles. These observations suggest that an upregulation of orexin-A has a protective effect against the development of metabolic abnormalities in patients with schizophrenia receiving antipsychotic treatment [2]. İn a 451 clozapine taking patients included study by Lappin et al. [3], the MetS prevalence was 58%, about 80% of patients were obese or overweight and impaired fasting glucose rate was 47%. İt is postulated that near the half of participants were smoking cigarette, having sedentary lifestyle and bad nutritional habits and not taking adequate health care for their physical problems. Because these habits are common among most of the schizophrenia patients, the cause of impaired metabolic parameters of schizophrenic patients taking antipsychotics remains unclear. Another AAP Aripiprazole is a partial D_2 and $5HT_{1A}$ agonist and has a different metabolic profile from other AAP's. Reviews suggested a protective effect of antipsychotic combinations which included aripiprazole for dyslipidemia and glucose metabolism, compared to other combinations and/or monotherapy. AAP's are widely used for the treatment of acute and prophylactic phase of bipolar disorder, treatment resistant and psychotic depression and insomnia. İt means that a huge proportion of psychiatric patient population is under risk for metabolic side effects and these patients fasting glucose, lipid profile, weight, waist circumference, BMI and blood pressure must be monitored closely. When starting AAP, the benefit/cost ratio for the patient should take account and the drug which is suitable for patients' metabolic profile should be chosen.

KEYWORDS

Atypical antipsychotics; schizophrenia; bipolar disorder; metabolic syndrome; dyslipidemia; DM

References

[1] Alberti KGMM, Zimmet P, Shaw J. Metabolic Syndrome. Diabetic Medicine, 2006;23:469–480.

[2] Chen P-Y, Chen C-H, Chang CK, et al. Orexin-A levels in relation to the risk of metabolic syndrome in patient with schizophrenia taking antipsychotics. 2018; doi:10.1093/ijnp/pyy075

[3] Lappin JM, Wijaya M, Watkins A, et al. Cardio-metabolic risk and its management in a cohort of clozapine-treated outpatients. Schizophr. Res. 2018. https://doi.org/10.1016/j.schres.2018.02.035

[Abstract: 0616] [ADHD]

The effects of antipsychotics on the movement system

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ABSTRACT

Antipsychotic drugs are the mainstay of treatment of schizophrenia and other psychotic disorders. The therapeutic efficacy of these drugs is well established. However, these drugs are associated with a wide range of side effects, including a variety of movement disorders. The newer antipsychotics have a lower propensity to cause acute extrapyramidal side effects and tardive dyskinesia.

Psychotic symptoms are successfully treated with antipsychotic drugs. However, the side effects of these drugs, especially those related to the motor system, seriously affect the patients and impair the compliance with the treatment.

Side effects of antipsychotic drugs related to the motor system:

1. Extrapyramidal syndromes

Akathisia

Acute dystonia

Parkinsonism

2. Chronic occurrences

Chronic akathisia

Tardive dyskinesia

Akathisia consists of motor restlessness accompanied by subjective feelings of inner tension and discomfort, mainly in the limbs.

Acute dystonia: Dystonias are involuntary movements characterized by intermittent or sustained muscle action.

Parkinsonism: Parkinsonian symptoms develop insidiously within days of starting antipsychotic treatment. The development of symptoms is dose dependent and emerges in about 20 to 40 percent of patients.

Tardive dyskinesia (TD) is the main late onset condition among the EPSEs. These are involuntary movements, mainly of the tongue and mouth with twisting of the tongue, chewing, and grimacing movements of the face.

[Abstract: 0804] [Others]

The effects of antipsychotics on enzyme system

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ABSTRACT

Changes in liver enzyme levels are one of the problems encountered during antipsychotic treatment. Recent studies have shown that liver enzyme abnormalities can occur not only with typical antipsychotics but also with atypical antipsychotics, which we have been using more and more frequently in recent years. Typical and atypical antipsychotics can cause liver enzyme abnormalities and rarely lead to liver toxicity. The mechanism of hepatotoxicity with antipsychotics is not known yet. High dose drug use, high drug plasma level, age, alcohol use and obesity are frequently reported risk factors for liver enzyme abnormalities associated with antipsychotics. It has also been suggested that the concomitant use of agents that cause an increase in liver enzymes significantly increases the risk. Therefore, the evaluation of liver enzymes at regular intervals is important in patients taking antipsychotics. Oxidative stress has been suggested to contribute to the pathophysiology of psychiatric disorders. Many inter-related mechanisms increase the production of reactive oxygen species and/or decrease antioxidant protection in psychiatry patients, thus provoking the free radical imbalance. Initial studies on psychiatric disorders suggest this imbalance may also be a consequence of the treatment with the first generation antipsychotic drugs, because treatment with these antipsychotics was found to cause oxidative injury in animals. Recent studies have reported that long-term treatment with both typical and atypical antipsychotics has an effect on the activity of antioxidant enzymes and lipid peroxidation levels. In general, typical antipsychotics may increase oxidative stress by attenuating antioxidant defense and increasing the conversion to free radicals.

[Abstract: 0676] [Neuroscience: Neuroimaging-Genetic Biomarkers]

Neuroimaging findings in childhood PTSD

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ABSTRACT

Almost two-thirds of adolescents in the USA have reported being exposed to one or more traumatic event during childhood and adolescence. Lifetime prevalence of post-traumatic stress disorder (PTSD) of traumatized adolescents is roughly 5%, with significantly higher rates of PTSD observed among females (7.3%) than males (2.2%) [1]. PTSD is associated with lower academic achievement, increased suicide, depression and substance use rates in adolescence. Structural and functional dysfunction were observed in individuals with PTSD in the frontolimbic circuity including dorsal anterior cingulate cortex(dACC), ventromedial prefrontal cortex (vmPFC), hippocampus, temporal pole, amygdala, insula responsible for emotion regulation and threat processing. In addition to the abovementioned circuity, dysfunction has also been shown in large brain networks responsible for self-referential thought and executive functions. Very little neuroimaging studies regarding the neurobiology of PTSD were conducted in pediatric individuals. Typical development of neural circuits' capacity supporting negative emotion processing and regulation changes with age in youth. Recent studies revealed the disrupting impact of childhood trauma exposure on development of threat processing and emotion regulatory systems resulting in compensatory mechanisms responsible for common PTSD symptoms such as negative emotional states and cognitions related to trauma. Childhood trauma exposure was also associated with increased rates of mood disorders. Moreover, DSM-5 offered the preschool (<6 years) subtype of PTSD considering the immaturity of the abovementioned systems, which requires fewer symptoms of avoidance and negative cognitions to meet the diagnostic threshold. There are a number of inconsistencies among results of structural neuroimaging studies in pediatric PTSD. While a decreased volume was reported in vmPFC, a study found higher volume in the same brain region as well as disrupted hippocampal development in youth with PTSD symptoms compared to typically developing youth [2,3]. Abovementioned differences may contribute to impaired regulation of threat via poor contextual gating and inhibition of threat responses, respectively. Studies about functional brain abnormalities during emotion processing have identified age-related abnormalities in amygdala-PFC coupling in pediatric PTSD. However, prior studies have shown that the variability in neuroimaging measures of brain function such as the amygdala reactivity to negative emotional information can be used as a marker for the treatment of PTSD [4]. For example, it has been shown that the response to trauma-focused cognitive-behavioral therapy (TF-CBT), known as the gold standard in adolescents, may vary depending on the activity in the brain regions before treatment [5]. Neurocircuitry models of PTSD have implications for understanding interindividual variability in reaction to treatment. As a result, changes in brain regions including the anterior cingulate cortex, amygdala and insula play a role in the neurobiology of PTSD and that changes in these regions anticipate psychotherapy treatment response using exposure therapies. An understanding of PTSD pathophysiology is an important issue for future research. It would be important to examine whether functional and structural integrity of brain regions predict susceptibility, chronicity and treatment response in PTSD and its developmental aspects.

KEYWORDS

Adolescents; children; neurodevelopment; neuroimaging; PTSD; trauma

References

[1] McLaughlin, K. A., Koenen, K. C., Hill, E. D., Petukhova, M., Sampson, N. A., Zaslavsky, A. M., & Kessler, R. C. (2013). Trauma exposure and posttraumatic stress disorder in a national sample of adolescents. Journal of the American Academy of Child & Adolescent Psychiatry, 52(8), 815–830.

[2] Carrion, V. G., Weems, C. F., Watson, C., Eliez, S., Menon, V., & Reiss, A. L. (2009). Converging evidence for abnormalities of the prefrontal cortex and evaluation of midsagittal structures in pediatric posttraumatic stress disorder: an MRI study. Psychiatry Research: Neuroimaging, 172(3), 226–234.

[3] Keding, T. J., & Herringa, R. J. (2015). Abnormal structure of fear circuitry in pediatric post-traumatic stress disorder. Neuropsychopharmacology, 40(3), 537.

[4] McLaughlin, K. A., Busso, D. S., Duys, A., Green, J. G., Alves, S., Way, M., & Sheridan, M. A. (2014). Amygdala response to negative stimuli predicts PTSD symptom onset following a terrorist attack. Depression and anxiety, 31(10), 834–842.

[5] Cohen, J. A., Bukstein, O., Walter, H., Benson, S. R., Chrisman, A., Farchione, T. R., Hamilton, J., Keable, H., Kinlan, J., Schoettle, U., Siegel, M., Stock, S., Medicus, J. Practice parameter for the assessment and treatment of children and adolescents with post-traumatic stress disorder., 2010/04/23. 2010. p. 414–430.

[Abstract: 0699] [Psychotherapy]

Trauma-focused cognitive behavioral therapy in children and adolescents with PTSD

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ABSTRACT

One of four children and adolescent reported exposure to a childhood trauma before age of 18. Approximately one third of this children and adolescents develop post-traumatic stress disorder (PTSD) [1]. PTSD is characterized by avoidance, re-experiment and hyperarousal symptom clusters. The treatment of such a common condition is also very important. In the choice of treatment, trauma-focused cognitive behavioral therapy (TF-CBT) is the first step in treatment guidelines, although many factors are important, such as the trauma type and severity, duration of trauma exposure, compliance with treatment and treatment options that the clinician can implement. Although there are few studies comparing efficacy among traumatic therapies, TF-CBT is the highest effectiveness in options of psychological therapies [2]. TF-CBT is a therapy ranging from 8 to 16 sessions, which is adapted to clients, considering the characteristics of the client, component-based, family and therapeutic engagement placed at the center of therapy. In therapy manuals, the components of TF-CBT are summarized by the acronym PRACTICE in order to guide practitioners. The first step, psychoeducation, provides training on situations in which the patient will be misunderstood, such as the frequency of traumatic events, reactions to the traumatic event, and risky groups for traumatic events. In relaxation sessions, the patient is taught relaxation techniques that will assist the patient in gradual exposure sessions. In the affect modulation sessions, the client is expected to recognize own feelings, to express feelings and to learn the connection in the triangle of emotion-thought-behavior. At the same time, they help to recognize their feelings about the traumatic event. In cognitive coping sessions, it is aimed that the client recognizes his / her unhelpful thoughts and becomes aware of the distress and thoughts and feelings and behaviors. While the trauma narrative sessions are the most important steps, the client is assisted about facing trauma reminders in an approached and safer environment and to help them combat unhelpful emotions and thoughts. In-vivo exposure sessions, the client is planned to face situations associated with the traumatic event, working together with the client, considering the safety and mental health of the patient. Other important sessions are the work with parents. Simultaneously, similar interviews with parent continue, while both are ready for joint sessions. Subsequent step is ensuring the client's safety and to help identify future risk situations and to prepare the client for these situations. While the main TF-CBT steps are as mentioned above, in the children and adolescents, the developmental stages of the client should be considered and revised according to the client. In younger children, psychoeducation should be changed so that the client can understand. Because of the different coping skills and treatment motivations, these number of gradual exposure sessions can be reduced or exposure with role play. If the child's age gets younger and behavior problems are higher, numbers of conjoint family sessions can be increased [3].

KEYWORDS

Trauma; focused; CBT; child; adolescent; therapy

References

[1] Fletcher KE. Childhood posttraumatic stress disorder. In: Barkley RA, Mash EJ, editors. Child Psychopathology. 2nd ed. New York: Guildford Press, 2003, 330–371.

[2] Gillies D, Taylor F, Gray C, O'Brien L, D'Abrew N. Psychological therapies for the treatment of post-traumatic stress disorder in children and adolescents. Evidence-based child health: a Cochrane review journal, 8(3), 1004–1116.

[3] Cohen, Judith A., Mannarino, Anthony P., ve Deblinger, Esther. Trauma-Focused CBT for Children And Adolescents Treatment Applications. New York: The Guildford Press, 2012, 1–29.

[Abstract: 0636] [Psychotherapy]

EMDR in children and adolescents with PTSD

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ABSTRACT

Eye movement desensitization and reprocessing (EMDR) was developed by Shapiro (1989). EMDR is an integrative evidence-based therapy, that for over twenty years, has been used to treat disorders relating to traumatic stress and distressing life experiences (Shapiro, 1995, 2001). EMDR relies on an AIP model that proposes that present problems are based on earlier experiences that have been dysfunctionally registered in the brain and need to be reprocessed (Shapiro, 2012). The primary usage of EMDR therapy was within the military personnel diagnosed with post-traumatic stress disorder. The EMDR therapy contains eight phases: History, Preparation, Assessment, Desensitization, Installation, Body Scan, Closure and Re-evaluation (Shapiro, 1995 and second edition 2001). Individuals who suffered traumatization can develop a series of problems like intrusive memories, nightmares, irritability, flashbacks, new fears, and avoidance strategies. When the individual is traumatized they experience such strong emotions that is thought to overwhelm the brain. Consequently the brain is unable to cope with it, or to process the information. The information processing theory suppose that this blocked information processing is facilitated by alternating bilateral stimulation (ABLS) using left/right eye movements, tactile stimulations or sounds (J. M.-Smith, M. Silvestre, 2014).

NICE (National Institute for Health and Clinical Excellence) guidelines recommend to "Using the EMDR therapy for children and adolescents (7–17 years old) with a diagnosis of PTSD or clinically important symptoms of PTSD who have presented more than 3 months after a traumatic event only if they do not respond to or engage with trauma-focused cognitive behavioral therapy" (NICE, 2018). Also AACAP recommends EMDR therapy for adults and children with PTSD, and explaining that it's effective for adults but the most randomized controlled trials for child EMDR have had serious methodologic shortcomings (Journal of The American Academy of Child & Adolescent Psychiatry Volume 49, number 4, April 2010).

KEYWORDS

Post-traumatic stress disorder; eye movement; desensitization and reprocessing; child psychiatry

References

[1] Journal of The American Academy of Child & Adolescent Psychiatry (April 2010) Volume 49, Number 4.
[2] J. M. Smith, M. Silvestre, EMDR for the Next Generation-Healing Children and Families. 2nd ed., 2: 2014.
[3] National Institute for Clinical Excellence. Post-traumatic stress disorder (PTSD): The management of adults and children in primary and secondary care. London: NICE Guidelines; 2018.
[4] Shapiro, F. (1989). Eye movement desensitization: a new treatment for post-traumatic stress disorder. Journal of Behavior Therapy and Experimental Psychiatry, 20, 211–217.
[5] Shapiro, F. (1995). Eye movement desensitization and reprocessing: basic principles protocols and procedures (1st ed.). New York: Guilford.
[6] Shapiro, F. (2001). Eye Movement Desensitization and Reprocessing: Basic Principles, Protocols, and Procedures (2nd ed.). New York: Guilford Press.
[7] Shapiro, E. Revue Européenne de Psychologie Appliquée 62 (2012): 241–251.

[Abstract: 0710] [PTSD]

Pharmacological treatments for PTSD in children and adolescents

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ABSTRACT

Approximately one third of children and adolescents exposed to various traumatic events have been reported to develop post-traumatic stress disorder (PTSD) [1]. Treatment strategies for children with PTSD consist of evidence-based trauma-informed interventions (behavioral cognitive therapies in individual, family and group formats) and psychopharmacology. The American Academy of Child and Adolescent Psychiatry suggests that psychoeducation and Trauma-Focused Psychotherapies are considered the first-line of treatments for PTSD in children and adolescents, and there were still no Federal Drug Administration (FDA) approved pharmacologic treatments for youth with PTSD. Research on the efficacy of psychopharmacological agents for pediatric PTSD is rather limited. A recent systematic review found three RCTs of selective serotonin reuptake inhibitors (SSRI) and one RCT of imipramine in children and adolescents with PTSD (two trials in total) [2]. Evidence-based guidelines for treating PTSD suggest that psychopharmacological interventions should be used cautiously and only after determining that the child or adolescent with PTSD may not benefit from psychotherapeutic interventions such as Trauma-Focused Cognitive Behavior Therapy (TF-CBT) [3,4]. Drugs prescribed by child and adolescent psychiatrists include SSRIs, tricyclic antidepressants, alpha and beta adrenergic agents, anxiolytics, antipsychotics and anticonvulsants. These interventions are often directed at attenuating symptoms in the core

KEYWORDS

Adolescents; children; PTSD; stress disorders post-traumatic; psychopharmacology

symptom domains of PTSD (intrusive, avoidance, hyperarousal). Some psychopharmacologic agents that have been shown to be effective in adult populations have failed to yield such results in children and adolescents. The differential efficacy of these agents in pediatric populations relates to pharmacokinetic or pharmacodynamic differences between children and adults or possibly to differences in the age-related neurobiological mechanisms of PTSD. A novel pharmacological augmentation strategy is based on data suggesting that NMDA partial agonist, D-cycloserine, enhances extinction of learned fear and the efficacy of cognitive behavioral and exposure-based therapies for PTSD in youth [5]. While TF-CBT is considered the first line of treatment for childhood PTSD, it is estimated that approximately 20% of youth who complete a course of TF-CBT will still meet diagnostic criteria for PTSD post treatment. Further blinded, systematic, placebo-controlled studies are needed to identify pharmacologic agents effective in the treatment of PTSD, and the augmentation of evidence-based psychotherapies. Preclinical and early clinical studies in adults suggest the utility of several potential novel agents, including nabilone, norbinaltorphimine, 7,8-dihydroxyflavone, oxytocin (OT) to target cannabinoids, opioids, brain-derived neurotrophic factor, and the OT receptor systems, respectively.

References

[1] Berman, S.L., et al., The impact of exposure to crime and violence on urban youth. American Journal of Orthopsychiatry. 1996;66(3):329–336.

[2] Keeshin, B.R., J.R. Strawn, Treatment of children and adolescents with posttraumatic stress disorder (PTSD): A review of current evidence. Child and Adolescent Psychopharmacology News. 2012;17(2):5–10.

[3] Cohen, J., The Work Group On Quality Issues ve AACAP Work Group on Quality Issues. Practice parameter for the assessment and treatment of children and adolescents with posttraumatic stress disorder. Journal of the American Academy of Child & Adolescent Psychiatry. 2010;49:414–430.

[4] National Collaborating Centre for Mental Health; 2005.

[5] Scheeringa, M.S. and C.F. Weems, Randomized placebo-controlled D-cycloserine with cognitive behavior therapy for pediatric posttraumatic stress. Journal of child and adolescent psychopharmacology. 2014;24(2):69–77.

[Abstract: 0610] [Others]

Increasing productivity and efficiency in primary data analysis and manuscript writing

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ABSTRACT

In this workshop, I discuss strategies for increasing productivity and time management in accomplishing scientific manuscript writing. First, I discuss the importance of outlining analyses in advance before conducting them. I discuss using a checklist to ensure all steps are taken in the cleaning and analysis of research data. Next, I address the issue of whether to conduct data analysis before or after manuscript preparation, and how often conducting analysis prior to manuscript writing can be an efficient use of time. I discuss the use of cheat sheets and note-taking for data analysis education. Using such cheat sheets when learning a new data analysis technique makes it easier to remember how to use the technique years later when using that analysis again. I discuss model mapping and drawing. Visualizing models can assist in minimizing errors when analyzing data. Using a vector graphics app can assist with such diagrams. I discuss preserving computational resources for more efficient computer use when writings manuscript. For example, saving data analysis output files as PDFs makes it easier and more efficient to retrieve analysis output later when writing the manuscript. I discuss behavioral and environmental strategies to increase focus when manuscript writing. It is important to have a routine when writing, even though each individual's preferred routine may be slightly different. I discuss my preferred routine, involving writing at a coffee shop, while listening to music through headphones (instrumental music). I discuss the use of a text editor application when writing, to decrease the distraction that results from using a fully featured word processing application such as MS Word. I discuss using a pomodoro timer to assist in maintaining focus while writing, such as the Forest application. I discuss minimizing distractions while writing by setting one's phone on do not disturb or airplane mode. Efficient time management is also assisted by monitoring the time it takes one to complete their work, so engaging in time tracking is important and therefore discussed. I discuss using an outline when writing a paper. I discuss the importance of organizing a corpus of literature into a digestible spreadsheet, and how such practice makes it easier to write the introduction to a scientific manuscript. It is also

KEYWORDS

Productivity; manuscript writing; research

important to continue writing even when seeing the need to insert references; references can be added later, thus reducing task switching. Finally, I discuss the importance of using bibliographic management software such as Endnote.

[Abstract: 0683] [Dependencies]

Is internet addiction a clinical diagnosis?

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ABSTRACT
The pattern of use of internet can change from healthy use to problematic and addictive use. Internet addiction remains to be a topic of debate in scientific world in recent years. Following these debates and studies about the addictive use of internet, American Psychiatric Association (APA) included Internet Gaming Disorder (IGD) in the DSM-5 as a condition for further study. IGD has been reported to be associated with Attention-deficit/ hyperactivity disorder, depression, social phobia. The parent-child interaction is affected by IGD and parental attitudes have an important role both in development and treatment of IGD. World Health Organization has defined gaming disorder as a behavioral addiction in the 11th Revision of the International Classification of Diseaes (ICD-11) as "a pattern of gaming behaviour characterized by impaired control over gaming, increased priority given to gaming over the activities to the extent that gaming takes precedence over other interests and daily activities and continuation or escalation of gaming despite the occurrence of negative consequences. Addictive use of internet has been reported to be associated with school drop-out, social problems and psychopathology.

KEYWORDS
Internet addiction; internet gaming disorder; comorbidity

[Abstract: 0733] [Dependencies]

Epidemiology and neurobiology of internet addiction

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ABSTRACT
Internet addiction is considered as a public health problem by many countries, particularly among adolescents and young adults. The prevalence rates of internet addiction are reported to be increased by the recent epidemiological studies. The male to female ratio is reported as 2:1, and the prevalence rates are reported to be higher among Asian countries. The etiology of internet addiction is multifactorial; with a combination of biological and psychological factors. Neurobiological model is among the models trying to explain the etiology of internet addiction. There is a dual processing model of digital technology addictions according to the neurobiological model. The dual system includes the reflective and reactive systems. Hypodopaminergic activity in the reactive system is reported to be a risk factor for internet addiction. Neurobiological studies have reported that decreased DAT expression, decreased D2 receptor levels in caudate and putamen, dysregulation in orbital prefrontal cortex, low levels of N-acetyl aspartate, systolic, choline-containing compound (Cho) in internet addiction. Also, reduced capacity of prefrontal cortex, orbitofrontal cortex and anterior cingulate cortex , less grey matter in the anterior cingulate cortex ,posterior cingulate cortex dorsolateral prefrontal cortex, orbitofrontal cortex, and insula have been reported in internet addiction.

KEYWORDS
Addiction; epidemiology; internet; neurobiology

[Abstract: 0724] [Dependencies]

Psychosocial factors in internet addiction

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ABSTRACT

Like other addictions, internet addiction also comes with risk factors which can cause, increase and affect prognosis of addiction. There are also some protective factors against development of addiction. Both of these factors are subject to many studies. Different models try to explain psychosocial relations of addiction. Neurobiological, cognitive behavioral, deficiency in social skills, learning theory, Reward deficit hypothesis, rich get richer, I-PACE model are among some of models that try to explain internet addiction. According to Cognitive behavioral model positive experiences in internet usage leads to increased usage by the means of reinforcement. People with internet addiction has some negative cognitions about themselves, and they choose internet who seems to be less risky than real life. We looked at the psychologic model, stressful and/or traumatic life events and low resilience leads to dependencies. Some of the sociodemographic risk factors are starting using internet at young age, being in high school, male gender, high family income, immigration and living in rural areas. Divorced parents, hostile environment, absence of familial rules and rituals, poor attachment with parents are among familial risks. Some personnel variables related to internet addiction are impulsivity, high loss avoidance, new experience seeking, reward dependence, low social skills, low self-esteem, loneliness, boredom. Self-identify, self-control and good emotional regulation are protective against internet addiction. In this presentation we will try to explain psychosocial factors and models of internet addiction.

KEYWORDS

Internet addiction; psychosocial factors; risks; family

[Abstract: 0682] [Dependencies]

Treatment of internet addiction

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ABSTRACT

The Internet has become one of the most common, fast and important communication tools of our global community. With the rapid development of the Internet, Internet addiction (IA) has become a common and problematic phenomenon. IA is defined as excessive and compulsive use of the Internet, which causes problems and serious consequences in social and occupational problems. It is still one of the most common behavioral problems for adolescents who are more exposed to internet use and ultimately more vulnerable. Estimated Internet addiction rates are 0.8 to 11.8% in Western samples and 10% in some Asian samples.

There is no standardized Internet addiction treatment yet. Because of methodological limitations, including lack of control group and small sample size, there is little evidence for the most effective treatment approaches, in the literature. However, it has been seen that the best approach in the treatment of Internet addiction is the individual approach and the combination of psychotherapy and psychopharmacotherapy.

Various treatment modalities have been known, including medications, CBT, family-based therapies, reality therapy (R/T), mindfulness oriented therapy, virtual reality therapy, motivational interviewing, nature camps, multilevel counseling, electro acupuncture and a number of other eclectic approaches. Cognitive-behavioral strategies and perhaps motivational interventions have high levels of evidence for both adolescents and adults in the treatment of IA. Parental involvement has been found very important for a successful outcome. Especially, sport intervention can improve symptoms of withdrawal. What about medications? Escitalopram, methylphenidate, and atomoxetine have also been applied to treat Internet Gaming Disorder (IGD), however, little is known about medications for Internet addiction. There is a single open label study of escitalopram for treatment internet addiction. Clinical studies have demonstrated that both bupropion and escitalopram are effective in treating and managing IGD, even bupropion is more effective than escitalopram. Internet addiction has been associated with multiple comorbidities such as depression, anxiety, obsessive-compulsive symptoms, attention and hyperactivity disorder and aggression. The use of psychotropic drugs (including antidepressant drugs and psychostimulants) for comorbid psychiatric or developmental disorders has been effective in reducing the degree

KEYWORDS

Internet addiction; therapy; treatment

of Internet Addiction and symptoms. In this speech, there will be discussed, evidence-based treatment for Internet addiction based on recent experimental research including psychological, psychopharmacological and combined treatment approaches.

[Abstract: 0800] [Others]

Artificial intelligence and psychiatry

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ABSTRACT

Artificial Intelligence (AI) is one of the most popular tools for medicine, and there are discussions of fear and prejudice in these days. However, it is certain that AI will change our lives radically and generally positive way. It is clear that AI will permanently and drastically change medical interview, monitoring and treatment methods. In our presentation, we will talk about difficulties, prejudice, and new areas where AI is used in the field of medicine. We will also discuss potential uses in psychiatry and how it is applied to the masses. Therefore, we will explain the internet of things and deep learning, as well as the fields of study of patient diagnostics, treatment, and follow-up. Moreover, AI can provide guidance and counseling in many areas, making the problems that experts cannot see become more apparent. Today, it has much more advantages for experts (psychiatrist, psychologist, etc.). For showing it, we will introduce new intelligent psychiatric assistant (WeCureX) for assessing major symptoms of social and personal maladjustment, identifying suitable candidates for high-risk public safety positions, giving a strong empirical foundation for a clinician's expert testimony, evaluating participants in substance abuse programs and select appropriate treatment approaches, and supporting college and career counseling recommendations with high accuracy rates. Finally, it will be emphasized that the use of artificial intelligence and its current effects by showing the new solution examples.

[Abstract: 0702] [Autism]

Approach to ADHD and learning difficulties in ASD

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ABSTRACT

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by social-communication deficits and restrictive, repetitive, stereotyped behaviors, and interests that are typically detectable in early childhood and continue into adulthood. Its prevalence has multiplied by 4 in the past decade, reaching the current percentages of 1 in 68 children in USA. Attention-deficit/ hyperactivity disorder (ADHD) is another common neurodevelopmental disorder characterized by a persistent pattern of inattention and/or hyperactivity and impulsivity that leads to various degrees of functional impairment. Its prevalence is estimated between 5–13% in the general child and adolescent population. Children and adolescents with ASD have shown high rates of ADHD comorbidity (28–83%) which may be due to common etiological mechanisms. Recently, the Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) allowed the concurrent diagnosis of ASD and ADHD (ASD+ADHD), based on the high comorbidity presented by the two disorders and the co-occurrence of symptoms. Previous studies have described more severe autism symptoms, higher rates of cognitive impairment, more deficits in adaptive skills, higher rates of externalizing behavior problems, such as aggression, irritability, conduct problems, oppositional behaviors, and lower quality of life in individuals with

ASD and ADHD in comparison to ASD alone [1]. Therefore, clinicians should check the ADHD symptoms in all ASD cases and plan treatment strategies in diagnosed ones. In the treatment of ADHD in ASD population, optimizing behavioral/educational supports and initiating psychopharmacologic interventions are effective and important treatment modalities. Clinicians should use judgement in selecting medications according to the patient's needs. Stimulant medications, atomoxetine, alpha agonists (guanfacine, clonidine) and atypical antipsychotic medications (risperidone and aripiprazole) are useful options. Stimulant

KEYWORDS

ASD; ADHD; pharmacotherapy; treatment

medications (methylphenidate, amphetamine) enhance dopaminergic transmission by inhibiting or reversing dopamine reuptake. Children who have dual diagnosis of ASD and ADHD seem to have lower effect sizes with these medications and are more sensitive to side effects, including emotional lability, agitation, stomach pain and sleep problems. It is often preferable to start with a short-acting formulation methylphenidate to observe possible side effects before switching to the long-acting formulation [2]. In a recent study, long acting liquid methylphenidate at low to moderate doses has shown to be effective in reducing ADHD symptoms and well tolerated in young children with ASD and ADHD [3]. Atomoxetine is a selective norepinephrine reuptake inhibitor. Atomoxetine has some beneficial effects on ADHD symptoms, irritability, restricted, stereotyped behaviors and communication in ASD patients. A recent double blind placebo controlled trial has shown that atomoxetine is generally well tolerated except reduced appetite and fatigue side effects in these patients [4]. Alpha-agonists (guanfacine and clonidine) primarily target hyperactivity and impulsivity. They are frequently used in the treatment of ADHD symptoms in ASD. A recent placebo controlled study has shown that long acting guanfacine is effective at reducing hyperactivity, impulsivity and distractibility symptoms in ASD patients. Risperidone and aripiprazole are atypical antipsychotic medications. Studies have demonstrated that they could reduce some of the ADHD symptoms in children with ASD who have cooccurring irritability and agitation. Consequently, ADHD treatment is very important in ASD patients. If it is ignored, response to educational therapies and learning will be adversely affected, impulsivity and behavioral problems will increase, and quality of life will decrease.

References

[1] Avni E, Ben-Itzchak E, Zachor DA. The presence of comorbid ADHD and anxiety symptoms in autism spectrum disorder: clinical presentation and predictors. Front Psychiatry. 2018;9:717.

[2] Mahajan R, Bernal MP, Panzer R, et al. Clinical practice pathways for evaluation and medication choice for attention-deficit/hyperactivity disorder symptoms in autism spectrum disorders. Pediatrics. 2012;130(Suppl. 2):125–138.

[3] Kim SJ, Shonka S, French WP, et al. Dose-response effects of long-acting liquid methylphenidate in children with attention deficit/hyperactivity disorder (ADHD) and autism spectrum disorder (ASD): A pilot study. J Autism Dev Disord. 2017;47(8):2307–2313.

[4] Tumuluru RV, Corbett-Dick P, Aman MG, et al. Adverse events of atomoxetine in a double-blind placebo-controlled study in Children with Autism. J Child Adolesc Psychopharmacol. 2017;27(8):708–714.

[Abstract: 0711] [Autism]

Approach to autism spectrum disorders (ASD): new developments/new treatments Signs of anxiety and approach to quality of life in ASD

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ABSTRACT

Autism spectrum disorder (ASD) is a neurodevelopmental disorder characterized by impairments in social interactions and communications as well as restricted and repetitive patterns of behaviors, interests, or activities. Many studies have reported increased numbers of children and adolescents diagnosed with ASD. Recent estimates from the Centre for Disease Control place the prevalence at 1 in every 59 individuals. Many children with ASD also display symptoms of other psychiatric disorders, such as Attention-deficit/ hyperactivity disorder, depression, or anxiety disorders. A meta-analysis estimates that anxiety disorders may occur in approximately 40% of adolescents with ASD. This rate is more than two times higher than in adolescents without developmental disorders. Especially, specific phobias were found to be more common among children and adolescents with ASD. Previous studies proposed that many core ASD symptoms might lead to stressful experiences that promote anxiety. In line with this reasoning, children with ASD may be more likely to endorse specific phobias because of their ASD-related deficits. Despite their high prevalence and associated impairments, diagnosis and treatment of these co-occurring conditions in ASD are often underrecognized and undertreated, as symptoms of ASD can mask common anxiety symptoms. As anxiety disorders are highly prevalent among children with ASD, it is important to examine these symptoms during clinical evaluations.

Quality of life (QoL) refers to an individual's subjective perception of their personal well-being and encompasses multiple domains, generally representing physical, psychological, and social functioning. A study has declared that 65% of people with ASD report having a profound or severe disability with at least one of the functioning domains (communication, self-care, and mobility) which supports other studies reporting lower QoL in autistic people

KEYWORDS

Anxiety; approach; asd; autism; quality of life

compared to the general population. Some research has also shown that autistic people report relatively poorer adaptive functioning skills in comparison to their cognitive level. Children with anxiety disorders have been found to have a poorer QoL in the domain of emotional functioning compared to children with other disorders. There are also indications that QoL in children with ASD is poorer compared to children with chronic health conditions or children with other psychiatric disorders. Additionally, higher anxiety severity was reported to contribute to a lower QoL. In this context, clinicians should be aware of anxiety symptoms in ASD children during clinical examinations to improve QoL.

[Abstract: 0421] [Psychopharmacology]

Is psychiatry ready for clinical application of pharmacogenetic tests?

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ABSTRACT

Genes affect people throughout their lifetimes and are the most important elements characterizing our lives. Linked to how genes work, people display variations in resistance to diseases or responses to medication. At the same time, our gene functions affect development of diseases and medication metabolism linked to the polymorphic characteristics of our genes [1]. Pharmacogenetics is based on the interaction between genes and biopsychosocial environment. It aids in identifying diseases and adverse medication reactions that may form due to these interactions. Pharmacogenetic tests may create differences in medication effects due to the differences between individuals. Clinical application of pharmacogenetic test results ensures appropriate treatment choices, allows the possibility to perform dose optimization and reduces the incidence of adverse effects. Even today, clinicians determine the medication dose with the trial and error method and this situation causes unwanted medication reactions in many patients. Side effects of medications are a serious public health problem. Insufficient and mistaken medication treatment causes serious damage to the individual and the country's economy. Additionally, many patients are admitted to hospital due to adverse medication reactions linked to this situation and may even die [2]. Medication choices for psychiatric diseases are increasing every day. Psychiatrists still attempt to find the most appropriate medication for the patient's situation. However, in spite of all this effort, desired target outcomes are not achieved. The decisions to use a specific treatment for psychiatric diseases may be difficult due to variations in medication metabolism linked to the genetic complications and cytochrome p450 enzymes specific to the individual. The responses to antidepressant and psychotropic medications may be different between individuals. The medication choice and dose for psychiatric diseases must be specific to the patient. It is thought that different responses to medications in individuals are due to the cytochrome p450 enzyme family. These enzymes are responsible for adverse medication reactions. The use of pharmacogenetic tests to individualize the treatment and medications used for psychiatric diseases will reduce the failure rate of treatment and adverse medication reactions and ensure patients gain maximum benefit from medications. Pharmacogenetic tests are promising not just for psychiatric treatments, but for treatment specific to the person in all fields of medicine [3]. When research is investigated, the greatest deficiency noted is that the evidence for the clinical use of pharmacogenetic tests has not been proven at sufficient levels. As a result, there is a need for studies to strengthen consideration of the use of pharmacogenetic tests in clinical applications. Future studies should focus on finding specific genetic variations. Depending on how specific the genetic variations are, the treatment will be that individual. Individualization of treatment and use of pharmacogenetic tests with this aim will open new horizons for treatment not only in the science of psychiatry but in all areas of medicine. The future where pharmacogenetics is used to create treatment specific to the person for all types of disease is not far away. Hopeful advances have been and continue to be recorded in this area.

KEYWORDS

Pharmacogenetic; psychiatry; treatment; drug; genes

References

[1] Hızel C. Kanıta Dayalı, Öngörüsel ve Bireye Özgü Tıp Yeni Genetik ve Uygulamaları. İKU. 2010;24.

[2] Zanger UM, Turpeinen M, Klein K, Schwab M. Functional pharmacogenetics/genomics of human cytochromes P450 involved in drug biotransformation. Anal Bioanal Chem. 2008;392(6):1093–1108.

[3] Bousman C, Al Maruf A, Müller DJ. Towards the integration of pharmacogenetics in psychiatry: a minimum, evidence based genetic testing panel. Current Opinion in Psychiatry. 2019;32(1):7–15.

[Abstract: 0769] [Psychopharmacology]

The use of pharmacogenetic testing in patients with bipolar disorder

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ABSTRACT

Although the treatment options are increased with a great acceleration and the most suitable drug / drugs are selected, the desired treatment goals cannot be achieved in the patients. Pharmacogenetics is the study of the variability of drug response on the basis of heritability and is influenced by ethnicity, age and gender. Pharmacogenetic testing may help physicians to determine differences in the effects of drugs based on inter-individual variations. By using pharmacogenetic testing, genetic variations have been identified and linked to the risk of developing medical conditions as well as treatment responses. Applying pharmacogenetic results clinically may improve the medication choose, optimize dosage regimens, and reduce the side effects and this may reduce the health costs [1]. Psychiatry has a special position because the effect of many psychotropic drugs, especially antipsychotics, mood stabilizers, antidepressants, can be observed only after minimum three or four weeks. Even though the symptoms of the patients who applied to the psychiatric clinics seem to be similar, their medical and psychiatric histories, social situations, education, beliefs, ethnic groups and socioeconomic status, cognitive view and eating behaviors are different from each other. Although the proper medication for the patients is used at the appropriate dosage with the correct timing, sometimes the response to psychotropic treatment cannot be obtained or the response to the treatment may be insufficient or undesirable effects may occur. The most important question at this point is whether the selected drug or drugs are really the most suitable drug (s) for the patient. In another words, the success of the treatment may be possible by choosing the proper drugs to be used specific for the patient and not for the disease [2]. Another difficult area in psychiatry is that most of the etiologies of psychiatric disorders (schizophrenia, major depression and bipolar disorders) are polygenic. Especially in many psychiatric disorders, it is difficult to choose a good candidate gene because of the lack of well-known pathophysiology. The dopamine receptor DRD2 was the most widely studied gene among the patients with bipolar disorder. Also polymorphisms in the PDLIM5 gene that is expressed in the brain have been associated with bipolar disorder. In addition it has been revealed that PDLIM5 may be a good marker for bipolar disorder. Induced pluripotent stem cells in bipolar disorder was found to be directly associated with mania symptoms [3]. Choosing the proper treatment options for bipolar disorder is important. Because patients with bipolar disorder have to face many difficulties about drugs that may influence the treatment adherence. But the decision to use a particular treatment in bipolar disorder can be difficult because of the complexity of genetics and variations in drug metabolism with cytochrome P450 enzymes variations they may influence treatment response. Genetic differences in the induction or inhibition of enzymes they determine the individuals as slow or fast metabolizer are extremely important in both monotherapy and polypharmacy. In patients whose CYP2D6 enzyme, which metabolizes a large part of the psychotropic medications\s, is considered to be practically ineffective, toxic effects are frequently seen, but in patients with the same enzyme working too fast, the expected response to the drugs cannot be obtained. Therefore, the polymorphisms of the CYP system allow us to not only choose the proper dose of the drug for that patient and also to know the possible interactions, the adverse reactions and side effects [4]. One of the most important issue in treatment effectiveness is increasing the effects of drugs by inhibition of P-glycoprotein (P-gp) and developing the new treatment strategies. This protein, which regulates the passage of the blood-brain barrier of drugs, is formed by overexpression of the gene located on chromosome 7 (MDR-1) and shows polymorphism. P-gp inhibitor drugs compete with P-gp to bind to the drug and bind the substrate to P-gp instead of the drug. Many drugs are dual substrates for P-gp and CYP3A4, and this has a significant effect on the concentration of some drugs in the brain. P-gp is important for both pharmacogenetics and the interaction of psychotropic drugs. P-gp may play a role in treatment-resistant psychiatric disorders. Also, in patients with more than one disease which is called as syntrophic diseases (such as autism, bipolar disorder and schizophrenia), it is mentioned that there are syntrrophic genes and in these patients, individualization of treatment becomes more important [5]. In terms of psychotropic medications, the polymorphisms of enzymes involved in the synthesis of neurotransmitters, their receptors, post-receptor events and neurotransmitter carriers are also important. Although these genetic differences related to the drug target molecule are as important as the genetic polymorphism of the metabolism in order to individualize the treatment, they are not routinely used in clinic practice. Although the results of genetic studies vary considerably, they are very important in terms of understanding the brain function, pathology and bringing new perspective to treatment

KEYWORDS

Pharmacogenetic testing; bipolar disorder; personalized drug therapy; CYP2D6; antipsychotics; genetic polymorphism

[1–3]. Carriers are regulated at both cellular and molecular level. Maximum variability is observed at in single nucleotide polymorphism (single nucleotide polymorphism = SNP). Also Dopamine (D), noradrenaline (NA), adrenaline (A) and serotonin (5-hydroxytryptamine = 5-HT) and the enzymes such as tyrosine hydroxylase (TH), tryptophan hydroxylase (TPH), aromatic amino acid decarboxylase (AAD), dopamine beta hydroxylase (DBH), monoamine oxidase (MAO) and catechol-o-methyl transferase (KOMT) that are involved in the metabolism of these neurotransmitters are also important and thought to be good biomarkers [2]. In the treatment with antipsychotics, any proper effect is not observed among 30-40% of patients and serious side effects are seen in about 70%. This is due to either the ineffectiveness of the drug treatment due to the genetic mutation of metabolic enzymes, or the presence of toxic effects or the change in the effectiveness of the treatment due to the alter in the binding of receptors to the target receptors and the change in their functional capacity. Also while the differences in Synaptic Vesicle Protein 2C (SVC2) were found to associated with the response to olanzapine and quetiapine treatment, DRD2 SNP, rs2514218, was found to be associated with better antipsychotic response specifically with risperidone. The 16Gly allele of ARDB2 was significantly found to correlated with a higher risk of sexual adverse events in patients taking risperidone. A study comparing the Malaysian patients with Chinese and Indian patients in terms of treatment response to valproic acid therapy, it was revealed that Malaysian patients showed better treatment response to valproic acid therapy when they express the functional SCN1A IVS5Nþ5 polymorphism [2–4]. The use of SSRIs and MAOIs in long-term antidepressant therapy causes the sensitization of inhibitor somatodentritic 5-HT1A receptors and presynaptic inhibitor 5-HT1D autoreceptors. Tricyclic antidepressants, electroconvulsive therapy and non-SSRI antidepressants may increase the sensitivity of the inhibitor post-synaptic 5-HT1A receptors and reduce the expression of the stimulator 5-HT2A receptors [1]. It has been suggested that there is a significant decrease in Gs protein in depressive patients and it can be used as a biochemical parameter in these patients. It has been shown that long-term treatment with fluoxetine causes desensitization of postsynaptic 5-HT1A receptors in the paraventricular nucleus of the hypothalamus, and this effect is due to a decrease in Gαi, Gα, Gα2 protein levels. Subchronic therapy with tricyclic antidepressants did not alter G protein levels [2]. It was revealed that Gαs proteins were increased in bipolar disorders, especially in frontal cortex and Gαs proteins were reduced in the occipital cortex with lithium treatment. Again, lithium treatment showed decreased Gαi expression in the cerebral cortex and similarly Gαi1 / 2 decreased in cortex and hippocampus. Also, the decrease in Gαi expression in the cerebral cortex and the decrease in Gαi1 / 2 decreased in cortex and hippocampus. In addition, subchronic treatment of buspirone, a partial agonist of 5-HT1A receptors reduced Gαi1 and Gαi2 protein levels in cerebellum, and benzodiazepines such as alprazolam did not alter G protein levels in various brain regions [1]. Choosing the proper treatment options for bipolar disorder is important. But the use of pharmacogenetics testing is still lacking. By providing the most appropriate pharmacotherapy with pharmacogenetics, early diagnosis, severity of side effects and interactions can be determined, and possible adverse drug reactions can be predicted.

References

[1] Routhieaux M, Jessica J, Tillery EE. The use of pharmacogenetic testing in patients with schizophrenia or bipolar disorder: A systematic review. Ment Health Clin. 2018;8:294–302.

[2] Arıcıoğlu F, Çetin M. Psikiyatride Bireye Özgü Tedavi: Belki Biraz Eski Veya Biraz Yeni, Ama Geleceğin Tedavisi. Bulletin of Clinical Psychopharmacology. 2010;20:269–273.

[3] Goh LL, Lim CW, Sim WC, Toh LX, Leong KP. Analysis of genetic variation in CYP450 genes for clinical implementation. PLoS One 2017;12:e0169233.

[4] Azuma J, Hasunuma T, Kubo M, Miyatake M, Koue T, Higashi K et al. The relationship between clinical pharmacokinetics of aripiprazole and CYP2D6 genetic polymorphism: effects of CYP enzyme inhibition by coadministration of paroxetine or fluvoxamine. Eur J Clin Pharmacol. 2012;68:29–37.

[5] Zain MA, Jahan SN, Reynolds GP, Zainal NZ, Kanagasundram S, Mohamed Z. Peripheral PDLIM5 expression in bipolar disorder and the effect of olanzapine administration. BMC Med Genet. 2012;13:91.

[Abstract: 0430] [ADHD]

When ADHD is not treated

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ABSTRACT

Attention-deficit/ hyperactivity disorder (ADHD) is characterized by age-inappropriate levels of inattentiveness, hyperactivity, and impulsiveness, or a combination thereof. And this disorder is among the most commonly occurring psychiatric disorders of childhood. ADHD often persists

KEYWORDS

ADHD; low self-esteem; antisocial behavior; side effects

into adulthood and is a risk factor for other mental health. The timely recognition and treatment of this children opportunity to improve their long-term outcomes. Primary treatment of ADHD is psychopharmacology. The approach of families, teachers and other occupational groups, and even medical specialists outside of us, to ADHD's drug treatment is distant. However when ADHD is not treated, the possible negative consequences can affect the family and the community. Although many people are so resistant to the treatment, everyone thinks that she has a say in the diagnosis of ADHD. ADHD is a medical disorder and psychiatrists diagnose this disorder. If ADHD diagnosis is correct, medical treatment is used. In this context, ADHD is a public health problem. When ADHD is not treated, low self-esteem and unsuccessful social relationships, academic and professional failure, antisocial behavior, smoking and drug use, obesity, problems can develop [1,2].

Case 1: A 16-year-old male was brought with complaints of friend dissonance, disagreement with school rules, objection the authority, and smoking. In his story, until the 8th grade, overall school success was good, and he was accepted to a good school although he is not prepared for the examination. Since infancy, he has been described as a hyperactive and difficult child. In his 9th grade, his behavior problems increased, and he started to use drugs. He was expelled from school. For similar reasons, he was thrown out of different two other schools too, and one year was unable to attend school. Afterwards his drug use increased. There was a suicide attempt, and he wounded himself with a knife. For all these reasons and with the diagnosis of psychotic depression he was hospitalized. Ten sessions of ECT were performed. In the psychiatric follow-up and treatment history; risperidone was started when he was 9 years old because of his hyperactivity and impulsivity but he did not use regularly and did not continue the psychiatrist regularly. In the first period of substance use, he was hospitalized due to a "manic" episode. All evaluations and examinations were made but this was attributed to drug use and no other psychiatric diagnosis was made. EEG and MRI were normal. Risperidone was recommended but the family did not continue treatment.

Case 2: 17-year-old female adolescent. He was brought to our clinic with complaints of running away from home and school, constant discussion with his mother, problems in friendships. In the story, her parents separated when she was 10 years old. Since they she started school, she had not been listening to the lessons, but she had always been successful in their classes until middle school. After the 6th grade, he began to have problems with his friends at school and came out against his teachers. After class 7, she started to run away school and home. In the 8th grade, the course success decreased, smoking started, she opposed her mother, and her violent behavior were added. Her absenteeism was increased in the high school years and she never went to school at the final year. When she was 16, she had inappropriate relations with men and started using drugs. Her mother tried to take her to psychiatry from an early age, but the girl had most of the time refused. She was diagnosed with ADHD when she went to the psychiatrist within a short period of time and was offered treatment to her, but she did not use the drugs even if she accepted to treatment. Her family did not have any sanctions on treatment.

References

[1] Shaw M, Hodgkin's P, Caci H, Young S, Kahler J, Woods AG, Arnold LE A systematic review and analysis of long-term outcomes in Attention-deficit/ hyperactivity disorder: effects of treatment and non-treatment. BMC Medicine. 2012;10:99.

[2] Spencer T, Biederman J. Attention-Deficit/ Hyperactivity Disorder: Diagnosis, Lifespan, Comorbidities, and Neurobiology. Journal of Pediatric Psychology. 2007;32(6):631–642.

[Abstract: 0429] [ADHD]

ADHD difficult patients: when ADHD medications are not working

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ABSTRACT

Since ADHD is a serious public health problem that can cause significant psychiatric, academic and social problems, its diagnosis and treatment is becoming increasingly important. In this context, 'ADHD Implementation Guidelines' have been created to standardize the diagnosis and treatment of ADHD in North America, Europe and Turkey [1,2]. Since ADHD is a neurobiological disease, all guidelines provide a clear consensus that drug therapy, particularly stimulants, has a significant role in the treatment of ADHD. Stimulants are effective in 70–80% of patients with ADHD, however, 20–30% of cases do not have adequate efficacy or treatment cannot be continued due to side effects. Non-stimulant drug use may

KEYWORDS

ADHD; Diagnosis Stimulants; Anxiety; Medication

be required in some of the cases with alcohol-substance abuse disorder, anxiety disorder or tic disorder comorbid to ADHD. The findings further indicated that the combination of both treatments- compared with pharmacological treatment only- had benefits regarding non-ADHD symptoms, levels of functioning, and the need for lower drug doses [3]. Although the efficacy rates are high in ADHD treatment there are also some patients who do not respond to these medications or only partially respond. Stimulant and atomoxetine combination pharmacotherapy was used to maximize treatment effectiveness in patients classified as partial responders to stimulant or atomoxetine monotherapy, or to minimize intolerable side effects in patients requiring a reduction in stimulant dose because of intolerable side effects. Children with multiple comorbidities have significant academic, social, and occupational impairment, which may be targeted with psychosocial interventions. Using psychosocial interventions in combination with pharmacotherapy seems a reasonable approach for children with ADHD with multiple comorbidities.
Case Presentation: Patient is a 13-year-old, 8th grade female student with complaints of school failure, unable to focus on lessons and she has feeling of chest tightness, fear of not breathing, trembling of hands and feeling like fainting for 2 years. She went to a cardiologist and cardiologist said that she did not have any disorder. She said that her friends could not understand her, they didn't believe her when she had that anxiety symptoms. Her parents separated four years ago, and she cannot share the unhappiness resulting from the separation of her family with her friends. Her mood was depressive. Her school failure has existed since elementary school and she cannot prepare for the high school entrance examination. There was no delay in her developmental history, she started to walk and talk on time. Since elementary school, she has been a hyperactive and fidgety child. After the first interview. methylphenidate OROS 18 mg/day and fluoxetine 10 mg/day treatment started diagnosed with adhd, panic disorder and depressive disorder. Conners Parent rating Scale was 67 and Conners Teacher Rating Scale was 43 Second week, dosage of fluoxetine was increased to 20 mg/day. After two weeks, she had a fight with her teacher and had a panic attack. Cognitive behavioral therapy (CBT) sessions was started. In a month, methylphenidate dose increased to 27 mg/day because there was no decrease in attention deficit and she could not perform well at school. In the third month of treatment, adhd symptoms persisted, the frequency of panic attacks increased, only her depressive mood changed to euthymic. Methylphenidate treatment was discontinued, and atomoxetine 1.2 mg/kg started. During this time she did not to continue CBT sessions. Atomoxetine treatment was continued for 3 months. Her anxiety was diminished, she had not had a panic attack in the last month and Conners Parent rating Scale was 45 and Conners Teacher Rating Scale was 32.

References

[1] AACAP (American Academy of Child and Adolescent Psychiatry) Practice Parameter for the Assessment and Treatment of Children and Adolescent with Attention-Deficit / Hyperactivity Disorder. J Am Acad Child Adolesc Psychiatry. 2007;46(7):894–921.
[2] Ercan ES, Avcı A, Mukaddes NM ve ark. Dikkat eksikliği ve hiperaktivite bozukluğu klinik uygulama kılavuzu, 2008, Türkiye.
[3] Vitiello B, Severe J. B, Greenhill LL, Arnold LE, Abikoff, HB, Bukstein OG, Cantwell DP. Methylphenidate dosage for children with ADHD over time under controlled conditions: Lessons from the MTA. Journal of the American Academy of Child & Adolescent Psychiatry. 2001;40:188–196.

[Abstract: 0399] [ADHD]

Working with families of children with ADHD

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ABSTRACT
Attention-deficit/hyperactivity disorder (ADHD) is a complex condition that affects not only the individual who experiences the symptoms but also the individual's family system. Parents of children with ADHD report more family dysfunction along with family environmental adversity than families of children without a diagnosis. Family dysfunction in relation to communication, problem solving, and relationships has been noted in families with children who have a diagnosis of ADHD [1,2]. When assessing children with ADHD in clinic, it is important for clinicians to be aware of the high prevalence of parent mental health problems. Parents of children with Attention-deficit/ hyperactivity disorder (ADHD) have high rates of psychopathology, especially ADHD and depression. In families in which there are multiple members with psychiatric disorders, the complexity of systemic dynamics and the

KEYWORDS
ADHD; family; parent

need for accommodations increase exponentially. The presence of ADHD or another psychopathology in adults creates complicated layers of family dynamics and interactions, as well as associated comorbid symptoms, which can be difficult and challenging for the clinician. Parental psychopathology may also attenuate treatment effects of pharmacological interventions for child's ADHD. For instance, parental ADHD symptoms may be associated with poor medication adherence, and consequently, reduced medication response, through inconsistent implementation and monitoring of children's medication regime [3,4]. It is very important that both the evaluation and the treatment plan are made considering the family milieu of the individual. To accomplish this, the clinician must understand the struggles of all the family members, respecting their observations as well as their own unique resources for change.

References

[1] Everett CA, Everett SV. Family Therapy for ADHD: Treating Children, Adolescents, and Adults. Guilford Press; 2001. p. 1–10.

[2] Foley M. A comparison of family adversity and family dysfunction in families of children with Attention-deficit/ hyperactivity disorder (ADHD) and families of children without ADHD. Journal for Specialists in Pediatric Nursing. 2011;16(1):39–49.

[3] Margari F, Craig F, Petruzzelli MG, Lamanna A, Matera E, Margari L. Parents psychopathology of children with Attention-deficit/ hyperactivity disorder. Res Dev Disabil. 2013;34(3):1036–1043. doi:10.1016/j.ridd.2012.12.001

[4] Wang CH, Mazursky-Horowitz H, Chronis-Tuscano A. Delivering Evidence-Based Treatments for Child Attention-Deficit/Hyperactivity Disorder (ADHD) in the Context of Parental ADHD. Psychiatry Rep. 2014;6(10):474. doi:10.1007/s11920-014-0474-8.

[Abstract: 0390] [ADHD]

ADHD with multiple comorbidities: treatment principles

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ABSTRACT

Children with ADHD often exhibit behavioral, developmental, and psychiatric comorbidities. The comorbidity of ADHD with other psychiatric disorders is between 60% and 80%. The type of comorbidity varies and includes other neurodevelopmental problems, oppositional defiant and conduct disorders, anxiety disorders, depression, bipolar disorders, sleeep disorders, eating disorders and substance use disorders. Children with severe ADHD symptoms have a higher likelihood of developing other psychiatric disorders. The probability of comorbidity is especially high for children who are treatment resistant. ADHD is often the first psychiatric disorder that a child manifests, and the presence of ADHD increase risk for the development of a variety of future psychiatric problems. ADHD may also be caused by a similar genetic and neurobiological basis with some neurodevelopmental disorders. The risk for having two or more comorbidities is higher in children with ADHD. Multiple comorbidities significantly increase the morbidity and disease burden of ADHD. The presence of multiple comorbid psychiatric disorders greatly complicates and alters ADHD treatment, often necessitating multiple psychopharmacological and psychosocial interventions. There is not sufficient evidence of how to treat children with ADHD with more than one comorbidity. Therefore, individualized treatment profiles should be created for children with multiple comorbidities. Treatment should be arranged according to the number, type, cause, and severity of comorbid disorders, child's functionality and available treatment options. Because the treatment of a psychiatric disorder often results in the improvement of symptoms associated with the other psychiatric disorders, it is recommended that pharmacotherapy be initiated for only the most severe disorder. Due to the presence of faster and more effective treatment options, it is recommended to start treatment with ADHD in the presence of comorbid psychiatric disorders of similar severity. Moreover, effective treatment interventions for ADHD may prevent the development of future comorbid disorders. Comorbid disorders may alter response to ADHD pharmacotherapy. Rarely, pharmacotherapy given for ADHD may increase the severity of anxiety disorders, tic disorders, mood problems, sleep and eating disorders. Treatment of comorbid substance use disorders, bipolar disorders, and severe other comorbidities are usually the first priority, given the greater risks associated with such psychiatric disorders. Once pharmacotherapy is initiated and optimized for the most severe disorder, then symptomatology of the comorbid disorders should be assessed for need for pharmacotherapy. It is recommended that, whenever possible, only one change in pharmacotherapy be made at a time. These recommendations are based on expert consensus because minimal research data exist to support pharmacotherapy for ADHD with

KEYWORDS

ADHD; multiple comorbidities; treatment; pharmacotherapy; psychosocial interventions

multiple comorbidities. Children with multiple comorbidities have significant academic, social, and occupational impairment, which may be targeted with psychosocial interventions. Using psychosocial interventions in combination with pharmacotherapy seems a reasonable approach for children with ADHD with multiple comorbidties.

[Abstract: 0361] [Mood disorders]

The promise and limitations of genetics in the diagnosis and treatment of bipolar disorder

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ABSTRACT

Bipolar disorder (BD) is a severe psychiatric disorder characterized by mood swings that can strongly influence patients' and families' quality of life. Although environmental factors also are critical, many epidemiological studies have suggested that genetic factors have a key role in developing bipolar disorder, and the heritability of bipolar disorder is estimated to be approximately 0.8 [1].

Genetics has been one of the main driving forces in psychiatric research, as it has been clear for decades that there is heritability in many psychiatric conditions. Candidate gene studies and linkage analysis studies that can be successful in single gene diseases, but these methods have not shown this success in polygenic diseases. After the human genome project, studies on the etiology of polygenic diseases have accelerated. Over the last decade, Genome-wide association studies (GWAS) turned into an increasingly important approach to investigate the genetic architecture of diseases and traits.

There are currently 68 single nucleotide polymorphisms (SNP) tag for bipolar disorder in the GWAS catalog [2]. Some single nucleotide polymorphisms contain genes encoding ion channels and neurotransmitter transporters (CACNA1C, GRIN2A, SCN2A, SLC4A1), synaptic components (RIMS1, ANK3, MIR-137), energy metabolism and immune components [3]. The most striking fact from the results of the BD GWAS is that the effect size of the susceptibility SNPs is extremely small (e.g., odds ratio ~1.2), and the magnitude was similar to that of other psychiatric disorders. Additionally, numerous numbers of SNPs and probably their combination or gene environment interaction will contribute to development of BD. Therefore, current definitive "susceptibility SNPs" are not sufficient as diagnostic tools. In this session, clinic genetic evaluation in BD will be discussed in the light of the recent literature.

KEYWORDS

Bipolar disorder; diagnostic tool; genetic; GWAS; polygenic risk score

References

[1] Craddock N, Sklar P. Genetics of bipolar disorder. Lancet. 2013; 381: 1654–1662.

[2] https://www.ebi.ac.uk/gwas/diagram

[3] Stahl EA, Forstner A, McQuillin A, et al. Genome wide association study identifies 30 loci associated with bipolar disorder. bioRxiv; 2017.

[Abstract: 0546] [Mood disorders]

The promise and limitations of immunologic process in the diagnosis and treatment of bipolar disorder

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ABSTRACT

OBJECTIVES: Bipolar disorder is a chronic disease characterized by episodes of mania and depression with a prevalence of 1% in the community. Bipolar disorder is considered not only as a mood disorder nowadays but also as a multi-system disease with many dysfunctions such as cognitive, endocrine and metabolism. It is now known that immunological processes such as cytokine responses, inflammation, and oxidative stress are also effective in the occurrence or progression of bipolar disorder. The effect of immunological parameters on the neurobiological nature of bipolar disorder is based on

KEYWORDS

Bipolar disorder; immunology; inflammation; oxidative stress

observations from autoimmune diseases. Several groups have reported convincing clinical evidence suggesting that immune-related diseases are more frequently observed in patients with BD than in the overall population. Interestingly, patients with systemic autoimmune diseases have been shown to present a higher risk for BD, suggesting a significant cross-talk between autoimmune processes and an increased expression of psychiatric disorders At the same time, the prevalence rate of autoimmune diseases such as Hashimoto's disease in the children with bipolar disorder strengthens this relationship. Changes in the number and proportion of inflammatory cells are observed in patients with bipolar disorder. The decrease in leukocytes and changes in the neutrophil/lymphocyte ratio are the most prominent characteristics of these cells. In addition, T regulator and NK cells, as well as number and loss of function, are reported. Further studies with cytokines and free-circulating immune factors contributed to immunological processes in bipolar disorder. The studies with these molecules have shown that the pro-inflammatory mediators are increased relative to the anti-inflammatory mediators and are associated with the course of the disease. The increase in pro-inflammatory mediators is more frequently observed during exacerbation of the disease. Some of the important cytokines obtained in these studies include tumor necrosis factor (TNF) -alpha, soluble interleukin-2 receptor (sIL-2R), IL-1 beta, IL-6 and soluble TNF-type 1 receptor (STNFR1). A similar relationship is found in the oxidative stress balance. While oxidative damage increases in bipolar disorder patients, antioxidant protective factors decrease. In addition to the molecular findings, anti-inflammatory agents have been shown to contribute to treatment in patients with resistant bipolar disorder.

This information suggests that one of the missing parts of the environment(stressor)- biology (genetics-neurobiology) relationship in the etiology of bipolar disorder may be immunological processes. In this session, the contribution of immunological processes in the etiology of bipolar disorder will be discussed in the light of new findings and it is aimed to present a new framework for bipolar disorder in the axis of environment-biology.

References

[1] Fries GR, Walss-Bass C, Bauer ME, et al. Revisiting inflammation in bipolar disorder. Pharmaco Biochem Behav. 2018;177:12–19. doi:10.1016/j.pbb.2018.12.006.

[2] Barbosa IG, Machado-Vieira R, Soares JC, et al. The immunology of bipolar disorder. Neuroimmunomodulation. 2014;21(2–3):117–122. doi:10.1159/000356539. Epub 2014 Feb 14

[3] Brown NC, Andreazza AC, Young LT. An updated meta-analysis of oxidative stress markers in bipolar disorder. Psychiat Res. 2014;218(1–2):61–68. doi:10.1016/j.psychres.2014.04.005

[Abstract: 0561] [Mood disorders]

Neurocognitive markers in bipolar disorder

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ABSTRACT

Bipolar Disorder is a chronic psychiatric disorder with manic /depressive or mixed episodes. The etiology is unknown, and it is thought to be multifactorial. Numerous studies have provided important information about the underlying pathophysiological processes of bipolar disorder. It is known that bipolar disorder affected many cognitive functions. Those who have more than one episode are more likely to be affected by a single manic episode [1] It was understood that disturbances in attention, processing speed, memory and executive functions continued in euthymic episodes as well as in disease episode. It is known that neurocognitive deficits are determinant on the course of bipolar disorder and the severity of the disease [2]. There is evidence that patients with bipolar disorder have persistent cognitive impairment in areas related to attention, verbal memory, and executive functions. It has been emphasized that neurocognitive deficits are determinant in the course and severity of the disease in patients with bipolar disorder [3]. Neurocognitive impairment has been identified as a contributing factor in the negative psychosocial functioning of this population. Neurocognitive disorder in bipolar disorder has been associated with decreased occupational functioning [4]. Although cognitive impairment is considered to be an important clinical feature of bipolar disorder, there is no standard cognitive battery developed for use in bipolar disorder research. Standard tests may be important in understanding the pathophysiology of bipolar disorder in understanding both clinical and cognitive development. Recurrent attacks were associated with progressive cognitive destruction; severe cognitive destruction is considered a poor prognosis for bipolar patients. Early onset, low premorbid IQ, history of psychotic episode, type I bipolar disease, long clinical course and / or excessive manic episodes affect the severity of neurocognitive deficits likely to have occurred since the onset of the disease [5].

KEYWORDS

Bipolar Disorder; Neurocognitive; Cognition

References

[1] Tsitsipa E., Fountoulakis KN. The neurocognitive functioning in bipolar disorder: a systematic review of data. Tsitsipa and Fountoulakis Ann Gen Psychiatry 2015;14:42.
[2] Demirel A, Demirel ÖF. Ötimik Bipolar Hastalarda Nörobilişsel Defisitler. Psikiyatride Güncel Yaklaşımlar-Current Approaches in Psychiatry 2012;4 (3):381–395.
[3] Lima IMM, Peckham AD, Johnson SL. Cognitive deficits in bipolar disorders: implications for emotion. Clin Psychol Rev. 2018;59:126–136. Epub 2017 Nov 21
[4] Best MW, Bowie CR. Neurocognition and psychosocial functioning in adolescents with bipolar disorder. J Affect Disord. 2017;207:406–412.
[5] Bora E. Neurocognitive features in clinical subgroups of bipolar disorder: a meta-analysis. J Affect Disord. 2018;229:125–134.

[Abstract: 0697] [PTSD]

Introduction to eye movement desensitization and reprocessing (EMDR)

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ABSTRACT

Traumatic events experienced in different periods of life may soon alter negative psychological changes. These negative shifts might be indications of pathology depending on their duration and consequences. Eye Movement Desensitization and Reprocessing (EMDR) has been invented by Shapiro in 1980s for treating PTSD. EMDR treatment is an effective and safe intervention on PTSD that accompanies psychotic disorder; moreover, it shows effect in a short time. Auditory and verbal hallucinations, delusions, anxiety symptoms, and depressive symptoms could fade away and self-confidence can be enhanced through PTSD treatment on psychotic patients. Prolonged exposure and EMDR therapy help reduce side effects of PTSD among individuals diagnosed with schizophrenia and schizoaffective disorders.

KEYWORDS

EMDR; PTSD; Psychotherapy

[Abstract: 784] [Others]

Neuroprogression in psychiatric disorders: model, mechanisms interventions

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ABSTRACT

I will introduce the concept of neuroprogression and provide an overview of potential therapeutic interventions to arrest neuroprogression. Most major psychiatric disorders are associated with a clinical trajectory with progressive increase in the frequency and severity of illness episodes and treatment resistance. Treatment resistance remains one of the greatest challenges in the field of psychiatry and psychopharmacology. Treatments are often suboptimal or unsatisfactory together with measurable structural brain and cognitive changes. Neuroprogression is a theoretical framework explaining illness progression over time. There is molecular, neurocognitive and neuroimaging research supporting the neuroprogression model and it is assumed to be mediated by inflammation, oxidative stress, altered neurogenesis, apoptosis and mitochondrial dysfunction. These presentations will overview the model and provide recent data on the capacity of pharmacologic therapies to address neuroprogression. Likely pathophysiological mechanisms include a proinflammatory state, increased oxidative stress and deficits in neuroprotection and neuroplasticity. Brain-immune interplay involves monoaminergic, cholinergic, glutamatergic and GABAergic systems with associated disruptions in these systems. Neuroanatomical brain changes have been related to these dysregulatory mechanisms. Microglial activation contributes to persistent and progressive inflammation and drives neuroprogression. Acute mood episodes are associated with systemic toxicity, cognitive and functional impairment and biological changes; these effects are cumulative. Epigenetic consequences of early childhood abuse may prelude major psychiatric disorders. Arresting or reversing neuroprogression through pharmacologic and non-pharmacologic interventions holds great promise. A persistent pro-inflammatory status in unipolar or bipolar depression may delay, diminish, or inhibit antidepressant response and contribute to co-morbidity. I will discuss how modulation of immune system activation may arrest neuroprogression and provide a brief overview of studies reporting beneficial treatment outcomes after

KEYWORDS

Inflammation; celecoxib; cytokines; CRP; bipolar disorder

administration of cyclooxygenase-2 inhibitors thereby modulating the inflammatory response. Since immune system dysregulation has been described in bipolar disorder, we hypothesized that modulation of the inflammatory response in treatment resistant bipolar depression (TRD-BDD) by co-administration of the COX-2 inhibitor, celecoxib, with the SSRSI, escitalopram, would reverse treatment resistance, augment overall response and show a faster onset of antidepressant drug action. This was a randomized, double-blind, two-arm, placebo-controlled study. Our results confirmed our hypothesis. COX-2 inhibition via celecoxib co-administration led to augmented response and more remissions than SSRI monotherapy. Additionally, we observed a much faster onset (one week) of an antianxiey along with an antidepressant response. We concluded that modulation of the inflammatory response reversed TRD, and produced a statistically significantly better antidepressant response and a statistically significant week-1 response in subjects receiving the combination. Anxiety scores also showed a significantly greater improvement with combined treatment. Specific inflammation biomarkers (*hs*CRP, IL1β, TNFα, MCP-1) correlated with treatment response in the ESC + CBX group only. These results indicate that addition of a COX-2 inhibitor reverses treatment resistance in BDD while achieving an augmented and accelerated antidepressant response. Modulation of inflammation in depression holds great therapeutic promise for psychopharmacological interventions in psychiatric disorders aimed at arresting and possibly reversing neuroprogression.

References

[1] Halaris, Leonard B, editors. Neuroprogression in psychiatric disorders. Karger Verlag, Basel, 2017.
[2] Edberg D, Hoppensteadt D, Walborn A, et al. Plasma C-reactive protein levels in bipolar depression during cyclooxygenase-2 inhibitor combination treatment. J Psychiatr Res. 2018;102:1–7.
[3] Ruiz NAL, Del Ángel DS, Olguín HJ, et al. Neuroprogression: the hidden mechanism of depression. Neuropsychiatr Dis Treat. 2018;14:2837–2845.

[Abstract: 0791] [Others]

Inflammation, insulin resistance and neuroprogression in depression

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ABSTRACT

In recent years the emphasis on the possible pathophysiological basis of affective disorder has incorporated changes in the endocrine and immune systems in addition to role of the dysfunctional monoamine system. Such an approach has resulted in the consideration of changes in non-psychiatric physical disorders such as diabetes, heart disease and cancer which occur with increased frequency in those with chronic major depression (Sotelo and Nemeroff,2017). Chronic low-grade inflammation has frequently been observed in major depression and in other major psychiatric disorders and has been implicated in the metabolic changes which are commonly associated with these disorders. Thus, major depression may be classified as a disorder of stress, an endocrine and/or an immune disorder in addition to a disorder of neurotransmitter dysfunction. In addition, the long-term consequence of depression is often associated with dementia. While it is not the purpose of this review to consider these disparate aspects that comprise depression, it is apparent that there are critical links between them whereby endocrine and immune changes impact on central and peripheral neurotransmitter function to precipitate changes associated with metabolic dysfunction. Many genes that contribute to the depressive genotype target the immune and endocrine systems. In addition, the neurodegenerative changes in the brain which occur in major depression are qualitatively similar to those seen in the early stages of Alzheimer's disease (Sapolsky,1996; Leonard,2018). These observations form the basis of a working hypothesis whereby the metabolic changes associated with the chronicity of depression reflect the complex pathology of the disorder and a link with neuroprogression. In 1897, Henry Maudesley observed that diabetes and "insanity" were often co-expressed in families. Since that time evidence has accumulated to show that depressed patients have approximately 60% higher risk of type 2 diabetes and, conversely, diabetic patients are more likely to suffer from depression (Soleto and Nemeroff,2017). Thus, diabetes could provide an important link between depression, the metabolic syndrome and brain energy metabolism. At the cellular level, this is associated with a decrease in insulin receptor function which results from a combination of the stress induced rise in glucocorticoids and pro-inflammatory cytokines (McIntyre et al,2007). While the mechanism whereby insulin resistance remains to be fully elucidated, it is evident that dysfunctional insulin receptors and receptor pathways affect the transport of glucose across the blood brain barrier and the subsequent uptake into

KEYWORDS

Glucocorticoids; pro-inflammatory cytokines; brain glucose; insulin; neuroprogression; dementia

neurons and neuroglia (Reaven,1988, Werner et al,1989). Such changes could underlie neuronal apoptosis and thereby provide a structural basis for dementia (Raigon and Jarvik,2004). This review will consider the hypothesis that major depression, particularly in the elderly, is frequently a prelude to dementia. This emphasises the importance of defects in brain energy metabolism in both depression and dementia. Thus, an integrated approach involving targets in the immune, endocrine and metabolic pathways may offer new possibilities for future therapeutic advances.

[Abstract: 0785] [Others]

C-reactive protein: are we ready to introduce it into clinical practice?

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ABSTRACT

In 2012, 16 million United States adults experienced an episode of depression according to the National Institute of Mental Health. Major Depressive Disorder (MDD) is one of the most prevalent psychiatric conditions and is associated with a high level of morbidity and mortality. From causation to treatment, research has sought to identify the mechanisms mediating this disorder. Our understanding of MDD has recently been expanded to include the role of the immune system, notably the relationship between inflammation and depression. Inflammatory biomarkers, such as cytokines and C-Reactive Protein (hsCRP), have been shown to be elevated in MDD patients [3]. This link between inflammation and MDD raises questions about causation as well as long-term consequences, especially atherosclerosis and cardiovascular disease [1]. If compounds, such as hsCRP, can be proven to be specific and valid biomarkers of MDD, then new avenues for diagnosis and treatment that uniquely target inflammation can be further explored. The pentraxin protein family is a group of acute phase reactants involved in immune response. hsCRP is a short-chain pentraxin produced in the liver and upregulated by cytokines during early inflammation [2]. In contrast to hsCRP, Pentraxin 3 (PTX-3) is the only identified long-chain pentraxin and it is produced locally throughout the body by neutrophils and macrophages [2]. While there is extensive literature on the role of hsCRP in MDD, there is little information on the potential role of PTX-3 in MDD. This presentation reviews current literature on CRP and PTX-3 in MDD and presents new data from our study, which showed baseline PTX-3 was significantly higher in MDD patients compared to healthy controls.

KEYWORDS

C-reactive protein; pentraxin 3; major depressive disorder; inflammation

References

[1] Clearfield MB. C-reactive protein: a new risk assessment tool for cardiovascular disease. J Am Osteopath Assoc. 2005;105(9):409–416.

[2] Deban L, Jaillon S, Garlanda C, et al. Pentraxins in innate immunity: lessons from PTX3. Cell Tissue Res. 2011;343:237–249.

[3] Howren MB, Lamkin DM, Suls J. Associations of depression with C-reactive protein, IL-1, and IL-6: a meta-analysis. Psychosom. Med. 2009;71:171–186.

[Abstract: 0749] [Psychopharmacology]

Hirsutism and psychotropics

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ABSTRACT

The term 'hirsutism' is of Latin origin, meaning excess body or facial terminal hairs growth, such as coarse sexual or secondary hair, which grows in a typical male-like pattern in androgen-dependent areas of the female body under the appropriate endocrine stimulation. Hirsute patients have increased dermal activity of the enzyme 5a-reductase, which is responsible for conversion of testosterone to the more potent androgen dihydrotestosterone. The process of vellus hair transform into terminal hair takes time, and occurs progressively over many hair growth cycles so hirsutism develops slowly but progressively after drug initiation.

KEYWORDS

Carbamazepine; hirsutism; hyperprolactinemia; PCOS; psychotropic; sodium valproate

Interruption of the process sufficiently early (e.g. eliminating drugs) can reverse the effects observed.

Hirsutism evaluate and score by modified Ferryman Gallwey system. Nine body areas are scored from 1 to 4. Clinically terminal hair hairs can be distinguished from vellus hairs primarily by their length (greater than 0.5 cm) and the fact that they are usually pigmented . In evaluation of hirsute patients detailed history, clinical examination and follicular phase blood samples investigation are important. The reporting of drug induced changes in hair should be done on a more cautious basis, and include details of the relationship between the initiation of drug administration, that of hair changes, serum T, LH, FSH, PRL, A, DHEAS, SHBG level measurement and microscopic examination of the hair roots. An elevation of the plasma free testosterone level is the single most consistent endocrinologic finding in hirsutism. Iatrogenic hirsutism caused by androgen therapy or the administration of medications such as danazol, anabolic steroids, sodium valproate or carbamazepine. Carbamazepine (CBZ) induces hepatic cytochrome P450 enzymes and increases the level of SHBG, disrupting feed-back regulation in the pituitary by reducing the effect of biologically active sex hormones such as estradiol and testosterone. Valproate (VPA) inhibits steroid hormone metabolism, elevates androgens, and predisposes to phenotypic signs of hyperandrogenism-hirsutism, obesity, acne, and frequent anovulatory cycles. Valproate-induced hirsutism is in a small proportion of patients (1%). Patients with drug-induced hirsutism may also present with other dermatological signs of virilization such as acne, seborrhea oleosa (oily seborrhea), and androgenic alopecia. Hirsutism is a common clinical problem in women, the prevalence of the condition is 5–25% of women in reproductive age. Hirsutism may a symptom of the serious androgen excess pathologies such as the polycystic ovary syndrome (PCOS), non-classic adrenal hyperplasia and androgen-secreting neoplasms. Up to 80% of women with hirsutism are diagnosed with PCOS. Hirsutisms may also be a symptom of hyperprolactinemia which may develop due to psychotropic use. Hirsutism can be one of the distressing conditions as an intolerable cosmetic-dermatologic side effects and important predictors of a lower quality of life. Its presence does not require treatment, particularly in mild-to-moderate forms, and when an affected woman does not worry about it. Management is aimed at eliminating drugs that cause hirsutism when possible. However, before discontinuing a psychotropic drug or dosage reduction, the advice of a dermatologist should be sought. Cosmetic procedures and pharmacological intervention are commonly used in the treatment of hirsutism.

[Abstract: 0719] [Psychopharmacology]

Restless legs syndrome and psychotropics

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ABSTRACT

Restless legs syndrome/Willis-Ekbom disease (RLS/WED) is characterized by discomfort in the limbs associated with an urge to move the limbs, temporary improvement with movement, and worsening symptoms at rest and in the evening. About 70% of patients with RLS/WED have periodic limb movements (PLMs) of sleep. RLS/WED affects about 2.1 %–5% of the general population. The recognition of restless legs syndrome as a disease with considerable effects on quality of life is still low among clinicians. As a consequence when patients present with either sensory symptoms in their legs (with or without pain), an urge to move at rest mostly in the evening, and sleep disturbances, they are often undiagnosed and left untreated for years. RLS/WED pathophysiology occurs in a wide range of locations and systems. It seems to have largely metabolic abnormalities mostly involving iron and the consequences of iron deficiency including increased dopamine. RLS/WED like other common diseases may have multiple pathways to disease, some less common than others (e.g., hypoxia without iron deficiency producing tyrosine hydroxylase and dopamine increases). Selective serotonin reuptake inhibitors (SSRIs) and serotonin norepinephrine reuptake inhibitors (SNRIs) are frequently used as first-line agents in the treatment of depression and anxiety disorders. Unfortunately, these medications have also been associated with an increased incidence of RLS. Among the various antidepressants, mirtazapine may be associated with higher rates of restless legs syndrome and periodic limb movements. One small study of normal volunteers suggested that venlafaxine may be associated with an increase in restless legs syndrome symptoms and periodic limb movements. Sertraline, fluoxetine, and amitriptyline appear to increase periodic limb movements that do not disrupt sleep and are thus unlikely to be clinically significant. On the other hand, bupropion may reduce restless legs syndrome symptoms, at least in the short term. Sedating antidepressants such as trazodone, nefazodone, and doxepin do not seem to aggravate periodic limb

KEYWORDS

Psychiatric disorders; Psychotropics; Restless legs syndrome

movements. The atypical antipsychotics quetiapine, risperidone, and olanzapine have all been suggested as possible triggers for RLS symptoms, although most of these data are derived from uncontrolled case reports or series. On the other hand, aripiprazole, an atypical antipsychotic that has partial dopamine agonist activity, improved RLS symptoms in a few small case series. In this presentation relationship between psychotropics and RLS will be discussed in the view of current literature.

[Abstract: 0060] [Eating disorders]

Binge eating behavior, eating disorders among obese adults and their relationship with cognitive processes

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ABSTRACT

Obesity (BMI > 30 kg/m^2) is considered to be one of the 10 most risky disorders by the World Health Organization (WHO). There has been an increasing interest in the relationship between obesity and mental disorders such as depression, anxiety and eating disorders [1] Management of physical comorbidities and particularly surgical (bariatric) interventions have an important role in the treatment of obesity. However, clinical studies suggest that unless psychiatric comorbidities are detected and treated, physical interventions, in most cases, lead to limited benefit with recurrence of weight gain. Past studies indicate that the frequency of binge eating ranges from 6%to 64% in bariatric surgery patients. Binge eating is characterized by the occurrence of episodes of eating, in a discrete period of time, a large amount of food, with a sense of lack of control over eating behavior and what or how much one is eating. These episodes often related to emotional distress and some cognitive processes and may be seen as a maladaptive attempt to avoid or escape disturbing thoughts and emotions. The Binge Eating Scale (BES) is a commonly used self-report measure of binge eating severity. The aim of this panel to discuss the binge eating behavior, eating disorders among obese people and their relationship with cognitive processes in the light of current literature. We also would like to present the preliminary findings of our own study which is currently going on. We aimed to recruit 500 adults aged 18–65 with obesity from patients who presented to obesity outpatient clinic at the Department of Family Medicine. Patients were asked to fill out certain self-report scales: Binge Eating Scale, Rumination Thinking style Scale, Freiburg Mindfulness Inventory, Acceptance and Action Questionnaire. They were then underwent psychiatric assessment for psychiatric comorbidity including eating disorders according to DSM5. We will present our preliminary findings along with the current available literature on the topics described above.

Better understanding of the relation between obesity and psychological mechanisms including psychiatric comorbidity will undoubtedly contribute to the development of more effective preventative strategies and inform the treatment protocols for obesity.

KEYWORDS

Obesity; binging; experiential avoidance

References

[1] Rajan TM, Menon V. Psychiatric disorders and obesity: a review of association studies. J Postgrad Med. 2017;63 (3):182–190.

[2] Niego SH, Kofman MD, Weiss JJ, et al. Binge eating in the bariatric surgery population: a review of the literature. The Int J Eat Dis. 2007;40:349–359.

[3] Gormally J, Black S, Daston S, et al. The assessment of binge eating severity among obese persons. Addictive Behaviors. 1982;7:47–55.

[Abstract: 0608] [Eating disorders]

Eating disorders and related factors in patients presanting to obesity outpatient clinic

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ABSTRACT
Studies showing that obesity (BMI> 30 kg/m^2), which is considered to be one of the 10 most risky diseaSses by the World Health Organization (WHO), is associated with physical disorders such as cardiovascular and endocrine diseases, have been going on for many years. In addition, there is an increasing interest since current studies showing the relationship between obesity and mental disorders such as depression, anxiety and eating disorders [1] For many years, interventions on physical diseases and especially recent surgical interventions have an important role in the treatment of obesity. However, clinical studies suggest that other treatment results are partial and weight gain reappears in most cases, unless there are interventions for the detection and treatment of psychiatric disorders associated with or caused by obesity. In 2013, in the current version of the mental disorders diagnosis classification DSM-5, the diagnosis of eating disorders and criteria are detailed; Pica (in children, adults), Rumination disorder (retrieval), Avoidant / Restrictive food intake disorder, Anorexia nervosa (restricting type, binge eating / purging type), Bulimia nervosa, Binge eating disorder, Other specified feeding or eating disorder (atypical anorexia nervosa, bulimia nervosa (of low frequency and/or limited duration), binge eating disorder (of low frequency and/or limited duration), purging disorder, night eating disorder) and other unspecified feeding or eating disorder [2]. In addition, some researchers have suggested that obesity should be included in DSM-5 as a mental disorder. Even though obesity is not yet available to provide DSM criteria, this recommendation reinforces the approach that obesity is a mental disorder [3]. Diagnostic interviews for eating disorders in obese patients have an important role in the detection and treatment of comorbid disease and the long-term treatment of obesity. In order to evaluate this relationship, we determined the comorbid eating disorders and psychiatric diseases by interviewing 250 patients with SCID, who were admitted to the obesity outpatient clinic of family medicine in our hospital, with BMI> 30 and aged between 18–65 years.The aim of this session is to provide information about the relationship between obesity and eating disorders in the literature and to share the preliminary data of our study. Determining the relationship between obesity, which has become an ever-increasing threat in the world, with mental disorders and especially eating disorders, will contribute to the development of treatment protocols and the development of effective prevention methods.

KEYWORDS
Obesity; eating disorders; DSM 5; mental disorders

References

[1] Rajan TM, Menon V. Psychiatric disorders and obesity: a review of association studies. J Postgrad Med. 2017;63 (3):182–190.
[2] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 5th ed. Arlington (VA): American Psychiatric Publishing; 2013.
[3] Larkin D, Kirtchuk G, Yamaguchi M, et al. A proposal for the inclusion of 'obesity dysmorphia' in the diagnostic and statistical manual of mental disorders. Australian and New Zealand Journal of Psychiatry. 2017;51:1085–1086.

[Abstract: 0731] [Others]

Looking at the cause as much as the result: factors underlying childhood sexual abuse in Turkey

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ABSTRACT
Child sexual abuse is evidenced by this activity between a child and an adult or another child who by age or development is in a relationship of responsibility, trust or power, the activity being intended to gratify or satisfy the needs of the other person. CSA includes an array of sexual activities like fondling, inviting a child to touch or be touched sexually, intercourse, exhibitionism, involving a child in prostitution or pornography, or online child luring by cyber-predators. The exact prevalence of child sexual abuse (CSA) remains unknown. It is estimated that the worldwide prevalence of CSA ranges from 3 to 31%. In Turkey, although studies with nationwide samples are insufficient, the frequency of sexual abuse in children has been established as 9–13% [1]. CSA has profound consequences for the child. It is known to interfere with growth and development. CSA has also been linked to numerous maladaptive health behaviors, and poor social, mental and physical health outcomes throughout the lifespan. CSA's physical and psychosocial effects are felt by abused children, their families, and their communities. Approximately 40 million children worldwide are abused each year. Abuse occurs at every socioeconomic level, across all ethnic and cultural lines, within all religions, and at every level of education [2]. Why do people sexually abuse children? The answer proposed here will necessarily be incomplete because a great deal still

KEYWORDS
Childhood; sexual abuse; risk factors

needs to be learned about the causes of sexual abuse. In most cases the dynamics are complex and multiple factors lead to sexual abuse. However, perpetrators appear to share important characteristics that are prerequisites for sexual abuse. These are sexual attraction to children and the willingness to act upon that attraction. The probability of sexual abuse is increased by cultural, environmental, individual, and family factors. This talk aims to consider the prerequisites for sexual abuse, and will elaborate upon the contributing factors. Emphasis will be placed upon critically examining the role of contributing factors based upon our own case experience.

References

[1] Butchart A, Harvey AP. Preventing child maltreatment: a guide to taking action and generating evidence. Geneva: Switz: World Press; 2011.

[2] Fis NP, Arman A, Kalaca S, et al. Psychiatric evaluation of sexual abuse cases: a clinical representative sample from Turkey. Children and Youth Services Review. 2010;32(10):1285–1290.

[Abstract: 0651] [PTSD]

Oxidative stress mechanisms and its association with psychopathology in sexually abused children

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ABSTRACT

Sexual abuse is one most challenging adverse life events that a child or a teenager can encounter. There is an increased evidence of oxidative stress (OS) process in psychiatric disorders that may result due to intense chronic stress exposure. In addition to lipid peroxidation, chronic inflammation also increases OS according to the model of inflammation observed in psychiatric diseases. OS can also damage central nervous system by glutamate-mediated excitotoxicity and hyperstimulation of N-methyl-D-aspartate (NMDA) receptors. Direct studies examining the relationship between sexual abuse and OS are limited in the literature. In this study, we aimed to investigate the OS parameters, psychopathologies and related sociodemographic factors in adolescents exposed to sexual abuse. Adolescents between 10 and 17 years of age who has reported sexual abuse, were referred to our clinic as forensic cases were included in the study in addition to age and sex matched control cases. In addition to socio-demographic information, the assessment of psychopathology was carried out with the Turkish version of the Affective Disorders and Schizophrenia Interview for Children- Version of Life and Now (K-SADS). Adolescents were evaluated with "Beck Depression Inventory", "Beck Anxiety Scale", "Post Traumatic Response Scale", "Ways of Coping Inventory", "Strengths and Difficulties Scale", and "List of Negative Life Events". Our sample consisted of 50 cases of sexual abuse (42 girls and 8 boys) and 40 controls (32 girls and 8 boys). The mean age of the cases was found 14,88 ± 2,16 in the abuse group and 14,90 ± 2,18 years of age in the control group. School continuity in the sexual abuse group was significantly lower. Sexual abuse cases were coming from distressed families with relatively much lower monthly income. In cases of sexual abuse, penetration was 60% and physical violence was 46% reported during abuse. Suicide attempt was described 34% in abuse group. Prior to abuse, psychiatric referrals were present in 46% of the cases. The most frequent diagnoses were 78.2% Post Traumatic Stress Disorder (PTSD), depression and 60.8% specific phobia. Functionality scores were lower than controls whereas Beck Depression Inventory, Beck Anxiety Scale, Post Traumatic Response Scale, and scores of Negative Life Events scales were higher in the study group. The level of OS assessed by total oxidant level (TOS), total antioxidant level (TAS) and oxidative stress index (OSI) showed a significant higher OS and diminished antioxidant process profile in sexual abuse group. TOS and OSI values were significantly higher while TAS values were significantly lower than controls. The fact that the OS mechanism which accelerates the cell cycle, leads to premature cell death that may result in many neuropsychiatric illnesses has been described in this study of abused adolescents. A more detailed study of OS mechanisms in adolescents in terms of an increase in the risk of physical disease, as well as possible adverse effects on life span and deterioration in quality of life. The increased risk of acquiring a chronic illness would be a meaningful topic in seeking further answers to the possible epigenetic mechanisms.

KEYWORDS

Sexual abuse; adolescent; oxidative stress; total oxidative level; total antioxidative level; psychopathology

References

[1] Murray LK, Nguyen A, Cohen JA. Child sexual abuse. Child Adolesc Psychiatr Clin N Am. 2014;23 (2):321–327.
[2] Trickey D, Siddaway AP, Meiser-Stedman R, et al. A meta-analysis of risk factors for post-traumatic stress disorder in children and adolescents. Clin Psychol Rev. 2012;32(2):122–138.
[3] Şimşek S. Level of cortisol and oxidative stress and DNA damage in child and adolescent victims of sexual abuse with or without PTSD. Bulletin of Clinical Psychopharmacology. 2015;25(Suppl 1):S22–S23.

[Abstract: 0774] [Others]

Endocannabinoid system and psychiatry Endocannabinoid system elements and functions

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ABSTRACT

Endocannabinoid system (ECS) is a common neuromodulator system. This system is composed of endocannabinoid receptors, endogenous cannabinoids and enzymes of synthesis and degradation. It has two 2 main receptors that are Cannabinoid receptor type 1 (CB1) and Cannabinoid receptor type 2 (CB2). CB1 is found on the brain, adipose tissue, gastrointestinal tract, liver, heart, and skeletal muscles, also CB2 is found primarily in the immune system, and has not been found to play a role in appetite regulation. Anandamide (AEA) and 2-arachidonoylglycerol (2-AG) are bound these receptors. AEA is synthesized by the breakdown of N-arachidonic phosphatidylethanolamine by phospholipase D. 2-AG is synthesized by diacylglycerol lipase from diacylglycerol. Monoacylglycerol lipase (MAGL) has an important role in the destruction of 2-AG. AEA is degradation by enzymatic by the membrane-bound enzyme fatty acid amidohydrolase (FAAH). ECS has a widespread distribution in our body. Therefore, it suggests that there are regulatory effects in many systems. It has a role in the regulation of memory, neurogenesis, pain perception, appetite, stress response, social behavior, and sleep in the central nervous system (CNS). In addition to its effects on CNS; It affects energy metabolism, reproduction and immune system.

KEYWORDS

Endocannabinoid receptors; endogenous cannabinoids; neuromodulator

References

[1] Fowler CJ, Doherty P, Alexander SPH. Endocannabinoid turnover. Adv Pharmacol. 2017;80:31–66. doi:10.1016/bs.apha.2017.03.006. Epub 2017 May 25. Review.
[2] Muccioli GG. Endocannabinoid biosynthesis and inactivation, from simple to complex. Drug Discov Today. 2010;15(11–12):474–483. doi:10.1016/j.drudis.2010.03.007. Epub 2010 Mar 19. Review.
[3] Wang J, Ueda N. Biology of endocannabinoid synthesis system. Prostaglandins Other Lipid Mediat. 2009;89(3–4):112–119. doi:10.1016/j.prostaglandins.2008.12.002. Epub 2008 Dec 14. Review.
[4] Basavarajappa BS. Critical enzymes involved in endocannabinoid metabolism. Protein Pept Lett. 2007;14 (3):237–246. Review.
[5] Alptekin M, Keser A. Relationship between endocannabinoid system, insulin resistance and fatty acids. Bes Diy Derg 2016:44(2):144–153.

[Abstract: 0754] [Neuroscience: Neuroimaging-Genetic Biomarkers]

Synthesis and metabolism of endocannabinoids

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ABSTRACT

Endocannabinoids are endogenous fatty acid derivatives which can bind to cannabinoid receptors that play a regulatory role in many physiological processes. The cannabinoid system consists of G protein-coupled type 1 (CB1) and type 2 (CB2) cannabinoid receptors and endogenous lipid ligands (endocannabinoids) and enzymes involved in their synthesis and degradation. Anandamide (AEA, N-arachidonoylethanolamine) and 2-arachidonoyl-sn-glycerol (2-AG) are two of the most important endocannabinoids. The CB1 receptor is the G protein-coupled receptor found in the central nervous system. The cannabinoids show their

KEYWORDS

Endocannabinoids; anandamide; 2-arachidonoyl-sn-glycerol; synthesis; metabolism

behavioral and neuronal effects via the CB1 receptor. Oscillation mechanisms are not fully described. AEA and 2-AG are not stored in the cell. They are synthesized in case of need and released out of the cell. AEA and 2-AG are synthesized from arachidonic acid. The AEA is synthesized by the conversion of membrane phospholipids into arachidonic acid at a sn-1 position by a number of reactions including the rate limiting step N-acyltransferase and specific phospholipase D. 2-AG is synthesized by remodeling arachidonic acid at the sn-2 position with phospholipid or phosphatidic acid. Inactivation of AEA and 2-AG occurs by rapid cleansing from the extracellular space and subsequent destruction. Although AEA and 2-AG have diffusion capabilities along the plasma membrane, they are taken into the cell via protein carriers. The destruction of AEA in the cell is catalyzed by the fatty acid amide hydrolase (FAAH) enzyme and 2-AG is degraded by monoacylglycerol lipase enzyme and possibly by other lipases. In both cases, enzymatic hydrolysis of endocannabinoids releases arachidonic acid. The relationship between membrane transport and destruction of intracellular endocannabinoids is not fully understood. FAAH inhibits the production of AEA.

References

[1] Fowler CJ, Doherty P, Alexander SPH. Endocannabinoid turnover. Adv Pharmacol. 2017;80:31–66.
[2] Muccioli GG. Endocannabinoid biosynthesis and inactivation, from simple to complex. Drug Discov Today. 2010;15(11-12):474–483.
[3] Wang J, Ueda N. Biology of endocannabinoid synthesis system. Prostaglandins Other Lipid Mediat. 2009;89((3-4):112–119.
[4] Basavarajappa BS. Critical enzymes involved in endocannabinoid metabolism. Protein Pept Lett. 2007;14 (3):237–246.
[5] Alptekin M, Keser A. Relationship between endocannabinoid system, insulin resistance and fatty acids. Bes Diy Derg. 2016;44(2):144–153.

[Abstract: 0778] [Others]

The role of endocannabinoid system in psychiatric disorders

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ABSTRACT

Endocannabinoid system (ECS) is widespread throughout the body, and considerable evidence supports its modulatory role in many fundamental physiological processes and pathophysiology/ neurobiology of mental disorders. ECS elements perform an important role in central nervous system(CNS) development, synaptic plasticity, and the emotional homeostasis, cognitive function, behavioral, developmental, and physiological processes. These qualifications may contribute an important role for ECS in the neurobiology and pathophysiology of several psychiatric disorders [1].

ECS and the emotional homeostasis

The ECS regulates fear and anxiety-related behaviors in both humans and rodents [2]. The ECS is present in crucial structures within the brain such as prefrontal cortex (PFC), amygdala and hippocampus. Some components of the ECS have been found in depression and anxiety-related disorders. Chronic stress has been associated with a dysfunctional endocannabinoid (EC) signaling in the CNS [3].

ECS and depression

The effects of the ECS in depression result from observational findings regarding the mood-related effects of cannabis in humans. Human studies have supported the presence of an altered ECS activity associated to major depression [34,69]. The CB1 receptor density and mRNA is increased in the DLPFC with depression patients, in parallel to CB1 receptor functionality [4]. The serum ingredient of ECs is also altered in major depression. Some authors report lower levels of the circulating ECs AEA and 2-AG in the depression patients . The levels of the ECs AEA and 2-AG were not altered in reaction to SSRIs such as fluoxetine, while the chronic application of MAOIs induced a reduction in areas (PFC, hippocampus and hypothalamus) [4].

ECS and anxiety-related disorders

Several studies suggest a potential link between dysregulation of the ECS signaling and anxiety-related behavior in both healthy and patients with mental disorders in which anxiety is a core symptom (PTSD, social phobia, agoraphobia, etc.). The interaction between the serotonergic system and the ECS on anxiety disorders described in many preclinical and clinical studies [5]. Preclinical findings suggest that the pharmacological manipulation of endogenous either AEA or 2-AG levels under stressful conditions could represent a good strategy for treatment of anxiety-related disorders [108]. Thus, it is plausible to hypothesize

KEYWORDS

Endocannabinoid; anandamide; anxiety disorder; depressive disorder; schizophrenia

the existence of altered EC levels in the brain of patients diagnosed of psychiatric diseases in which anxiety is either core or a comorbid symptom.

ECS and schizophrenia

The ECS has become a hot-topic in schizophrenia research in the last years. Several studies starting from the 40 s up to nowadays agree that the ECS represents a major neuromodulatory system participating in tones of physiological processes. The endogenous hypothesis refers to the fact that deregulation of the ECS may contribute to the pathophysiology of schizophrenia, whereas the exogenous theory refers to the risk associated with cannabis abuse that could facilitate the onset of the disease in vulnerable individuals or aggravate the symptoms in schizophrenic patients [5]. Several studies have investigated the status of CB1 receptors in the brain of patients with schizophrenia. Both, imaging and postmortem brain studies, have reported different outcomes. PET studies have evaluated the CB1 receptor availability in schizophrenic patients compared to controls [121–123]. Postmortem brain radioligand binding studies consistently reported increased density of CB1 receptors in schizophrenia [5].

References

[1] Mechoulam R, Parker LA. The endocannabinoid system and the brain. Annu. Rev. Psychol. 2013;64:21–47.

[2] Ruehle S, Rey AA, Remmers F, et al. The endocannabinoid system in anxiety, fear memory and habituation. J. Psychopharmacol. 2012;26(1):23–39.

[3] Morena M, Patel S, Bains JS, et al. Neurobiological interactions between stress and the endocannabinoid system. Neuropsychopharmacology. 2016;41(1):80–102.

[4] Katzman MA, Furtado M, Anand L. Targeting the endocannabinoid system in psychiatric illness. J. Clin. Psychopharmacol. 2016;36(6):691–703.

[5] Ibarra-Lecue I, Pilar-Cuellar F, Muguruza C, et al. The endocannabinoid system in mental disorders: evidence from human brain studies. Biochem Pharmacol. 2018.

[Abstract: 0811] [Others]

Psychodynamic psychotherapy is effective in the treatment of major depressive disorder and anxiety disorders

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ABSTRACT

Depression and anxiety disorders are the most prevalent mental disorders worldwide. The lifetime prevalence ranges from 15% to 20%. It is well established that psychological interventions are effective in the treatment of these disorders. Cognitive behavioral therapy (CBT) aims at alleviating depressive symptoms by changing maladaptive thought schemata and errors in thinking in combination with engaging more in activities that affect mood positively, while psychodynamic therapy (PDT) assumes that gaining insight in unconscious emotions and relational functioning related to vulnerability for depression is curative.The most examined psychological treatment for depression is probably CBT, with interpersonal therapy coming next. PDT is probably the least examined treatment method of these three therapies. Evaluating the research results of the some trials, it can safely be said that PDT has proven to be effective in the treatment of depression. The results of the trials confirm no difference in efficacy between CBT and PDT [1,3]. Empirically substantiated clinical judgement underpins professional accountability and transparency in health care and increasingly so in mental health. Empirical knowledge in psychological therapies is multifaceted and complex, and requires sophistication in the scrutiny of research data. Evidence supports the use of PDT in the treatment of depression. There is evidence that the effects are maintained in both the short and long term. PDT may be a preferred alternative to pharmacotherapy and certainly adds to the effectiveness of medication. If CBT is more effective than PDT, this difference is neither large nor reliable. However, there are too few large-scale trials to fully establish equivalence [2]. In a study comparing the efficacy of CBT and PDT in the outpatient treatment of major depression, no statistically significant differences were found between these treatments in a large sample of patients treated for a major depressive episode, and less than one-fourth of the patients reached remission within 22 weeks of treatment. These findings extend the evidence base of psychodynamic therapy for depression but also indicate that time-limited psychotherapy is not sufficient for a substantial number of patients encountered in psychiatric outpatient clinics [3]. The effectiveness of PDT for anxiety is crucial in the debate between those who argue for specific treatment approaches, as in CBT, versus those who support a generic approach seeking to identify similar unconscious content across diagnostic groups. In relation to social anxiety and perhaps generalized anxiety disorder and panic disorder, promising emerging evidence

KEYWORDS

Psychodynamic therapy; cognitive behavioral therapy; depression; anxiety

supports the argument for a generic approach. Overall, there is considerable potential for further sound research aiming to identify the anxiety conditions for which PDT may be particularly helpful [2].

References

[1] Dekker JJM, Hendriksen M, Kool S, et al. Growing evidence for psychodynamic therapy for depression. Contemporary Psychoanalysis. 2014;50(1-2):131–155.
[2] Fonagy P. The effectiveness of psychodynamic psychotherapies: an update. World Psychiatry. 2015;14:137–150.
[3] Driessen E, Van HL, Don FJ, et al. The efficacy of cognitive-behavioral therapy and psychodynamic therapy in the outpatient treatment of major depression: a randomized clinical trial. Am J Psychiatry. 2013;170:1041–1050.

[Abstract: 0809] [Others]

Is psychiatry at a crossroads in terms of treatment interventions? What should be done besides pharmacotherapy and psychotherapies for effective treatment of psychiatric disorders?

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ABSTRACT
Lifetime prevalence of major psychiatric disorders such as major depressive disorder (MDD) is 16.2% and schizophrenia is 0.5–1.5%. Also, MDD direct medical costs is $26.1 billion dollars, suicide-related mortality costs are 5.4 billion dollars and workplace costs are $51.5 billion dollars and total cost is $83.1 billion per year in USA. Schizophrenia total cost is $83.1 billion per year 62.7 billion for 2006 in USA. In terms of Turkey antidepressant and mood stabilizing drug costs Turkish Lira (TL) 317.66 in 2015, while it costs 16% increase TL 367.92, in 2016; it cost 21% increase of TL 446.52 in 2017 and, an increase of 21% TL 527.67 in 2018. In Turkey, the use of antipsychotic costs TL 351.20, while for 2015, it cost TL 378.44 8% increase in 2016; In 2017, TL 435.60 increased by 15% and reached TL 526.49 with an increase of 21% in 2018. But remission rates are very low about 30–50 % for many psychiatric disorders. In addition, treatment adherence is very poor in psychiatric disorders. When we look at these facts, it is now time to take preventive medicine/ psychiatry measures as a community rather than a therapeutic medicine approach measure, etc. Unless these comprehensive primary preventive medicine / psychiatry studies are carried out, therapeutic health services will be costly and insufficient.

KEYWORDS
Direct medical costs; preventive medicine; antidepressant; workplace costs

[Abstract: 0813] [Others]

Child language disorders: language delay, primary developmental language disorders, and secondary developmental language disorders

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ABSTRACT
Language acquisition problems in children are variously labelled such as Language Disorder/Language Impairment/Language Impaired/Language Disability, Delayed Language/Language Delay (LD), Specific Language Impairment (SLI), or Developmental Language Disorders (DLD). It is aimed to define language acquisition problems in children within three different classifications; LD, primary DLD, and secondary DLD and discuss their underlying features for differential diagnosis and subgroups within the framework of Bloom and Lahey's taxonomy of language. The term of LD is regarded as a difficulty, rather than as a disorder. The level of language proficiency at 24 months is considered as a substantially important and the first indicator of language development. In this stage, language development is in a normal range if a child has more than 50 words, produces two-word utterances, and uses communicative gestures properly. Therefore, children whose language skills at approximately 24 months do not meet the criteria could be

KEYWORDS
Language delay; late bloomers; specific language disorder; developmental language disorder; Bloom and Lahey's taxonomy of language

identified as having LD. Children with LD mostly have no known pre/peri/postnatal underlying etiologies and show normal development in other domains such as motor, cognitive, and sensory. Or, they might be at a very early stage for a diagnosis of any neurodevelopmental disorder. Some of the children with normal development patterns and no known etiologies can generally catch up with a significant vocabulary growth (vocabulary spurt) by 30–35 months and are defined as late bloomers. However, some of the children with LD at emerging language abilities continue to have language problems at further ages. By 48 months, children are expected to acquire language almost fully and gain enough proficiencies in all linguistic aspects (form, content, and use) and modalities of language (receptive and expressive). If a child's language skills at 48 months fall short of the standards, the child is diagnosed with DLD/language impaired/SLI. Depending on the underlying causes of the disorder, the notion is classified as primary and secondary DLD. The term of primary DLD is used in the diagnosis of children who have problems only specific to language but no other developmental domains, whose disorders underlying causes have not yet known, or for whom any other diagnostic label is not appropriate. On the other hand, children whose language impairments are associated with or secondary to other developmental disorders such as autism spectrum disorders, intellectual impairment, sensory impairments etc., or to situations such as social isolation and abuse are diagnosed with secondary DLD. Bloom and Lahey's taxonomy of language suggests a comprehensive framework for the assessment of these disorders with different etiologies. Accordingly, language includes three aspects: (i) Form (structure of language: Phonology, morphology, syntax), (ii) Content (vocabulary knowledge: Semantics), and (iii)Use (social use of language: Pragmatics). Traditionally, the most impaired aspect of language in children with DLD is considered as the form. However, delays in the content is also observed. Impairments in the use and communicative gestures are not generally observed in children with LD and primary DLD but secondary DLD. DLD is further sub-grouped as DLD-Receptive (greater impairment in receptive language), DLD-Expressive (greater impairment in expressive language), and DLD-Both (impartment in both modalities).

References

[1] Gillam S, Kamhi A. Specific language impairment. In: M Ball, J Damico, N Mueller, editors. Blackwell Handbook in linguistics: the handbook of language and speech disorders. Hoboken, NJ: Wiley-Blackwell; 2010.

[2] Paul R, Norbury CF. Language disorders from infancy through adolescence. St. Louis (MO): Elsevier Health Sciences; 2012.

[3] Bloom L, Lahey M. Language development and language disorders. New York: John Willey & Sons; 1978.

[Abstract: 783] [Others]

Language and mind: in the context of atypical autism- symposia 40

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ABSTRACT

The significant characteristic of human being is our ability to consciously use our imagination to simulate reality and form fictional worlds. The mind in a subjective perspective includes the phenomenological concepts like thought, language, memory, decision making, emotions, self-awareness, etc. The implicit and explicit languages can be assumed as the abstract materials of mind. The inputs driven from environmental context (social or external facts, necessities, etc.) interact with the demands of internal milieu (personal urges, desires, values, targets, memory, etc.) in the interface of mind. Identical stimulus can cause vastly different consequences depending on the personal varieties of mind and language and situational context. In some instances, formation of the imaginative representations of "self" concept or emotions (e.g., autism) may never develop enough (e.g. ASD) or it becomes significantly disturbed (e.g. schizophrenia). Specifically the inferior parietal lobule is responsible for representing one's own mental states; superior temporal sulcus is specialized in the representation of the mental states of others. (Abu-Akel, 2003). The structures including amygdala, the anterior cingulate gyrus, ventral and dorsal medial prefrontal cortex are involved in both (Abu-Akel, 2003). Both autistic spectrum disorder and schizophrenia patients have problems in mentalizing, theory of mind and differentiating internal information from external. Pervasive Developmental Disorders (PDD), also called Autism Spectrum Disorders (ASD), are defined in terms of abnormalities in social and communication development in the presence of marked repetitive behavior and narrow interests (APA 1994). In a previous study (Karabekiroglu & Akbas, 2011) we aimed to investigate differential features of pervasive developmental disorder- not otherwise specified (PDD-NOS) (atypical autism) in terms of presenting symptoms, developmental history, and comorbidity with respect to autism and

KEYWORDS

Language development; autism; mentalizing; theory of mind

Attention-deficit/ hyperactivity disorder (ADHD). The prevalence rates of the most common presenting symptoms in the PDD-NOS and autism groups have shown a similar pattern of distribution from most common to the least, even when the results were corrected for age. However, almost all of these symptoms are reported significantly less in prevalence in the PDD-NOS group. The results suggested that PDD-NOS may be assumed as a quantitative partial subtype of autism, and it represents a less severe form that lies on a continuum of social-communication skills. The most common symptoms included poor social interaction, hyperactivity, language retardation, echolalia, lack of eye contact, etc. From autism to PDD-NOS, the cumulative symptoms decrease, and the mind and language appears. Thus, PDD-NOS can be considered as an optimal clinical model to focus on mind and language development and physiology. The goal of this presentation is to review areas in brain that have role in mind and language in the context of autistic spectrum disorders.

References

[1] Karabekiroğlu K, H. Yöney "Integrative Processing of Internal and External Information & Implications for Autism" Türkiye'de Psikiyatri Dergisi, 2007;9(1): 58–65.
[2] Abu-Akel A "A neurobiological mapping of theory of mind" Brain Research Reviews 2003;43: 29–40.
[3] Karabekiroğlu K, S. Akbaş. "Identifying and Differentiating PDD-NOS: A Comparison with Autism and ADHD" Yeni Symposium, 49(3), 141–152 (2011).
[4] American Psychiatric Assossiation (APA) (1994). Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition. Washington DC: American Psychiatric Association.

[Abstract: 0689] [ADHD]

ADHD and speech and language disorders

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ABSTRACT

Language is an assemblage of rules showing how a society has coalesced around symbols and the rules indicating how these symbols should be combined. Speech is the conversion of language by means of symbols into appropriate verbal expressions and their use in interpersonal communication. Data from epidemiological studies of speech-language disorders and studies involving different clinical samples have shown a close association between speech-language disorders and psychiatric disorders. The most common psychiatric diagnosis in children with speech-language disorders in these studies is Attention-deficit/ hyperactivity disorder (ADHD). ADHD is a disorder characterized by inattention, hyperactivity and impulsivity incompatible with age and level of development that leads to significant social, emotional academic losses in the individual. Although speech and language disorders are not regarded as one of the basic symptoms of ADHD, there is nevertheless significant relationality between ADHD and speech-language disorders. These disorders have been shown to accompany ADHD in 45% of ADHD cases in community samples and in 67% of clinical samples.

There are three basic components of language use as a communication tool - content (semantics), form (phonetics/phonology, morphology, and syntax) and social use (pragmatic). A review of studies in the field of language disorders and ADHD reveals various common areas. Studies of language disorders are divided into those involving *structural language disorders* and those concerned with *communication disorders,* such as story-telling and the pragmatic use of language. Problems may be seen in the structural linguistic and/or communication spheres in children with ADHD. However, some studies emphasize the need to differentiate the effect of language disorders seen in children diagnosed with ADHD. These studies emphasize that structural linguistic disorders are only seen in children with accompanying reading disorder, and that the general language-related deficiency in children with ADHD appears only in the area of story-telling and pragmatic language use under the effect of inhibitory linguistic control. Pragmatic language use refers to effective and appropriate language use in an interpersonal context. In traditional approaches, pragmatic language tends to be considered independently of structural language. Social problems frequently seen in individuals with ADHD are associated with difficulties in pragmatic language use. Problems in pragmatic language use may emerge in the form of logorrhea, weak reciprocity in speech, repetitive expressions, and difficulties in understanding implication and metaphor. These all have a significant impact on functionality in children's domestic, school and peer relations. Pragmatic language is multi-dimensional. Some difficulties in pragmatic language use can be identified through evaluation of higher order language (metalinguistic) skills. Higher order language skills require the use of reasoning and

KEYWORDS

ADHD; auditory sensory discrimination; executive functions; pragmatic language; reading skills; speech-language disorder

critical thinking executive functions, such as deduction, prediction, and establishing causal relations. In addition, other executive functions have to work effectively for appropriate use of pragmatic language, such as adaptation to the dynamic characteristics of social communication, response adaptation and flexibility. Similarly, the ability to maintain attention and working memory functions such as remembering what has been said during conversation must also be sound. All these neuropsychological functions require a greater cognitive performance than other components of language, such as syntax, semantics and grammar. Studies show that executive function defects seen in children with ADHD contribute to pragmatic language use problems. In addition, some children diagnosed with ADHD and with pragmatic linguistic problems may exhibit normal semantic and syntactic language development and a normal performance in traditional language tests. However, language functions must be considered as an integral whole at clinical evaluation. In contrast to the traditional approach, the idea of interaction between structural language, story-telling and pragmatic language is predominant in the literature. Deficiencies in expressive language skills and auditory and verbal memory functions can impair children's pragmatic adaptation by affecting response time, word selection and combination, and subject continuity. Insufficient higher order language skills in ADHD may have an adverse effect on responses to treatment by complicating the clinical manifestation. Whether higher order language skills should therefore be considered inside a broad ADHD phenotype, similarly to emotion regulation difficulties, is therefore controversial.

In addition to those examining the effect of executive function defects in ADHD on speech and language, a smaller number of studies have also concentrated on auditory processes and subcortical dysfunctions as an alternative cause of speech-language problems in ADHD. Some studies comparing healthy controls and children diagnosed with ADHD have revealed that auditory information processing does not function effectively in ADHD. It has been suggested that children with ADHD may have hyper- or hyposensitivity to sounds and that this compromises auditory information processing. However, the presence of auditory deficit in children with ADHD is also the subject of debate. Some studies have determined deviation in auditory cerebral cortex responses in some functions involving binaural integration in children with ADHD compared to controls. In conclusion, a holistic examination of the literature reveals that problems associated with receptive language development, expressive language development and pragmatic use of language frequently accompany ADHD. Some children diagnosed with ADHD have difficulties in areas such as understanding what they listen to, making deductions from texts, and sentence formation, despite exhibiting age-appropriate language development in standard tests. The acquisition of metalinguistic skills and learning the phonological aspect of language are among the speech-language problems observed in children with ADHD. It is therefore recommended that clinicians evaluate sub-domains of speech and language as an integral whole while remembering that different types of speech-language disorders may be seen in children with ADHD. Addressing speech-language difficulties in children diagnosed with ADHD with a complex manifestation and responding insufficiently to standard therapeutic options and including these difficulties in treatment strategies will therefore contribute to a reduction in social and academic problems in particular.

References

[1] Randell R, Somerville-Brown L, Chen W. How relevant is higher-order language deficit (HOLD) to children with complex presentations of attention-deficit hyperactivity disorder? Atten Defic Hyperact Disord. 2018. doi:10.1007/s12402-018-0279-4.

[2] Tien YM, Chen VC, Lo TS, Hsu CF, Gossop M, Huang KY. Deficits in auditory sensory discrimination among children with attention-deficit/hyperactivity disorder. Eur Child Adolesc Psychiatry. 2018 Sep 18. doi:10.1007/s00787-018-1228-7.

[3] Green BC, Johnson KA, Bretherton L. Pragmatic language difficulties in children with hyperactivity and attention problems: an integrated review. Int J Lang Commun Disord. 2014;49(1):15-29.

[4] Cohen NJ, Vallance DD, Barwick M, Im N, Menna R, Horodezky NB, Isaacson L. The interface between ADHD and language impairment:an examination of language, achievement, and cognitive processing. J Child Psychol Psychiatry. 2000;41(3):353–362.

[5] Geurts HM, Embrechts M. Language profiles in ASD, SLI, and ADHD. Autism Dev Disord. 2008;38 (10):1931–1943.

[Abstract: 0671] [Mood disorders]

Unipolar/bipolar evolution of pediatric depression: neurobiological differences

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ABSTRACT
The differentiation of unipolar depression (UD) and bipolar depression (BD) is one of the major difficulties in psychiatric diagnoses because most bipolar patients first appear with depressive symptoms. Therefore, misdiagnosis is common in BD and leads to poor results and higher health costs that can be prevented by proper pharmacological treatment. A recent meta-analysis was compared to healthy controls (HC) in patients with UD, BD patients and HC, both voxel-based analysis (VBA) studies and those using channel-based spatial statistics (TBSS) approaches. The results suggest that corpus callosum and cingulate cortex show significant differences, especially in both diseases. Despite the need to find neurobiological markers that can help in distinguishing UD and BD, DTI measurements have not yet been used on a large measurement with strong results [3]. Recent research investigating the different neural substrates between BD and UD using fMRI used reward, emotion, or cognitive processing, and most studies focused on emotional tasks. Previous fMRI studies that directly compared individuals with BD and UD revealed differences in neural activity in limbic, PFC, subcortical and visuospatial regions during face-to-face jobs that were valued positively or negatively. In terms of shared neurobiological etiology in both UD and BD, both amygdala and ACC play a critical role in the dysfunction of emotion processing neural circuits in mood disorders. fMRI studies investigating brain-regional activation patterns have focused on emotional facial paradigms and found that differences in the neural activity pattern in the amygdala play a very important role in these tasks. Most studies have shown that the amygdala in UD has more activation against negative emotional stimuli than BD, and is in opposite order during positive emotional stimuli. Several proofs are distinctive amygdala activation pathologies during emotional facial tasks between UD and BD; In particular, there is a higher amygdala activation in UD compared to BD during negative emotional stimuli and an opposite pattern occurs during positive emotional stimuli. In this panel, we wanted to discuss the neurobiological aspect of unipolar/bipolar evolution of pediatric depression by the help of current debates.

KEYWORDS
Pediatric depression; neurobiological differences; unipolar depression; bipolar depression

References

[1] Phillips ML, Kupfer DJ. Bipolar disorder diagnosis: challenges and future directions. The Lancet. 2013;381 (9878):1663-71.
[2] Wise T, Radua J, Nortje G, Cleare AJ, Young AH, Arnone D. Voxel-based meta-analytical evidence of structural disconnectivity in major depression and bipolar disorder. Biological psychiatry. 2016;79(4):293–302.
[3] Repple J, Meinert S, Grotegerd D, Kugel H, Redlich R, Dohm K, et al. A voxel-based diffusion tensor imaging study in unipolar and bipolar depression. Bipolar disorders. 2017;19(1):23–31.
[4] Fournier JC, Chase HW, Almeida J, Phillips ML. Within-and between-session changes in neural activity during emotion processing in unipolar and bipolar depression. Biological psychiatry: cognitive neuroscience and neuroimaging. 2016;1(6):518–527.
[5] Wu M-J, Wu HE, Mwangi B, Sanches M, Selvaraj S, Zunta-Soares GB, et al. Prediction of pediatric unipolar depression using multiple neuromorphometric measurements: a pattern classification approach. Journal of psychiatric research. 2015;62:84–91.
[6] Han K-M, De Berardis D, Fornaro M, Kim Y-K. Differentiating between bipolar and unipolar depression in functional and structural MRI studies. Progress in Neuro-Psychopharmacology and Biological Psychiatry. 2018.

[Abstract: 0635] [Mood disorders]

Unipolar/bipolar evolution of pediatric depression: geno-typical and endopheno-typical differences

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ABSTRACT
Major depressive disorder (MDD) is one of the most common mental disorders and an important cause of impairment in functioning [1]. Threshold depressive symptoms are also associated with functional impairment and future psychological problems [2]. The family history of depression and the initial early age are associated with a worse prognosis in adults with MDD, but the factors and prognostic factors that affect depression in the youth period are not fully known [3]. Depression has a multifactorial complex etiology, including a moderate hereditary component [4]. The longitudinal studies and family studies show that the symptoms persist between adolescent-onset depression and adult depression, but there are also developmental differences between childhood, adolescence and adulthood depression [5]. Clinical follow-up studies of very early-onset depression have reported that

KEYWORDS
Paediatric depression; genotype; endophenotype

depression is frequently followed with a different type of clinical disorder, with high rates of heterotypic continuity [6]. In the literature, since at least half of adolescents with MDD have been shown to have manic transitions with or without antidepressant treatment, efforts to recognize young people with MDD who are at high risk for manic transitions have gained importance [7]. The family history of mood disorders is the most important risk factor for manic shift. These findings suggest that genetic factors may be effective for manic shift. Further evidence of the genetic effects of manic shift risk in pediatric MDD comes from studies documenting the history of mood disorders in the extended family as risk factors for such shifts (8). In this panel, we wanted to discuss the genetic aspect of unipolar/bipolar evolution of pediatric bipolar affective disorder by the help of current debates.

References

[1] Vos T, Flaxman AD, Naghavi M, Lozano R, Michaud C, Ezzati M, et al. Years lived with disability (YLDs) for 1160 sequelae of 289 diseases and injuries 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. The lancet. 2012;380(9859):2163–396.

[2] Angold A, Costello EJ, Farmer EM, Burns BJ, Erkanli A. Impaired but undiagnosed. Journal of the American Academy of Child & Adolescent Psychiatry. 1999;38 (2):129–137.

[3] Musliner KL, Trabjerg BB, Waltoft BL, Laursen TM, Mortensen PB, Zandi PP, et al. Parental history of psychiatric diagnoses and unipolar depression: a Danish National Register-based cohort study. Psychological medicine. 2015;45(13):2781–2791.

[4] Flint J, Kendler KS. The genetics of major depression. Neuron. 2014;81(3):484–503.

[5] Thapar A, Collishaw S, Pine DS, Thapar AK. Depression in adolescence. The Lancet. 2012;379(9820):1056–367.

[6] Rutter M, Kim-Cohen J, Maughan B. Continuities and discontinuities in psychopathology between childhood and adult life. Journal of Child Psychology and Psychiatry. 2006;47(3-4):276–95.

[7] Etain B, Lajnef M, Bellivier F, Mathieu F, Raust A, Cochet B, et al. Clinical expression of bipolar disorder type I as a function of age and polarity at onset: convergent findings in samples from France and the United States. Journal of Clinical Psychiatry. 2012;73(4):e561–e566.

[8] Biederman J, Petty CR, Byrne D, Wong P, Wozniak J, Faraone SV. Risk for switch from unipolar to bipolar disorder in youth with ADHD: a long term prospective controlled study. Journal of affective disorders. 2009;119 (1-3):16–21.

[Abstract: 0737] [Mood disorders]

Unipolar / bipolar evolution of pediatric depression: neurocognitive differences

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ABSTRACT

The presence of cognitive symptoms in bipolar disorder(BD) and unipolar depression(UD) is widely accepted. Although the UP-BP distinction is primarily phenotypic, genetic, biological and cognitive data support this approach. However, considering that both groups have important common points, it is appropriate to consider UD - BD disorder as subtypes of mood disorders rather than two different disorders. In cognitive characteristics of unipolar depression; impairments in executive functions such as attention, planning, working memory have been reported [1]. Patients with bipolar disorder have been shown to have sustained impairment in executive functions such as prolonged attention, verbal memory, processing speed, inhibition response [2]. Few studies have examined the neuropsychological functions in pediatric UD-BD. In studies; It was found that children and adolescents with bipolar disorder had neurocognitive impairments similar to adult bipolar patients and the most obvious deficits were in verbal memory [3]. A study using the Wisconsin Card Sorting Test showed impaired executive functions in children and adolescents with bipolar disorder [4] and decreased response inhibition scores were observed in the Stroop Word Color Test and Continuous Performance Test [5]. In a study comparing the neurocognitive performance of euthymic patients with bipolar and unipolar depression; prolonged attention was found to be more specific for bipolar disorder [6]. Mixed / manic bipolar patients; showed significant impairments in spatial attention, episodic and working memory and problem solving. On the other hand, bipolar and unipolar depressive patients showed disruption only in episodic memory. These findings emphasize the different cognitive profiles of mania and depression, and demonstrate similar models of neuropsychological impairments in bipolar and unipolar depression. Neuropsychological impairments affect the more pronounced and larger cortical function sequence in the mixed / manic period than in the depressive period. In a few studies comparing unipolar and bipolar patients, it was reported that bipolar patients performed worse in all areas [7]. Neuropsychological impairments have been shown to be more prominent in male patients in bipolar disorder. While gender-related factors change

KEYWORDS

Adolescent; bipolar; child; depression; neurocognitive; unipolar

the severity of neuropsychological impairments in bipolar disorder, this is not the case in unipolar patients.

In family studies, healthy 1st degree relatives of bipolar patients were found to have impaired verbal memory and some areas of executive functions; This suggests that cognitive impairment may be an endophenotype in bipolar development. Are these cognitive symptoms related to the nature of the disease or the side effects of drugs used for treatment? Are there neurocognitive markers in the development of unipolar depression into bipolar disorder? Finding answers to these questions may shed light on bipolar pathophysiology. In order to answer these questions, we planned a study on Endophenotypes, Prevalence of Psychopathology and Related Factors: Five-Year Follow-up Study in Children of Parents with Bipolar Disorder Diagnosed with Selcuk University Medical School Child Psychiatry and Adult Psychiatry. Our aim is to determine the endophenotypic markers for bipolar disorder in children and adolescents and to determine the prevalence of psychopathology in the high-risk group.

References

[1] Austin MP, Mitchell P, Goodwin GM. Cognitive deficits in depression: Possible implication for functional neuropathology. Br J Psychiatry 2001;178:200–206.

[2] Thompson JM, Gallagher P, Hughes JH, Watson S, Gray JM, Ferrier IN. Neurocognitive impairment in euthymic patients with bipolar affective disorder. Br J Psychiatry 2005; 186: 32–40.

[3] Horn K, Roessner V, Holtmann M. Neurocognitive performance in children and adolescents with bipolar disorder: a review. Eur Child Adolesc Psychiatry. 2011;20 (9):433- 450.

[4] Doyle AE, Wilens TE, ve ark KA. Neuropsychological functioning in youth with bipolar disorder. Biol Psychiatry 2005;58:540–548.

[5] Rucklidge RR (2006) Impact of ADHD on the neurocognitive functioning of adolescents with bipolar disorder. Biol Psychiatry 60:921–928.

[6] Sole B, Bonnin CM, Torrent C, Martinez-Aran A, et. al. Neurocognitive Impairment Across the Bipolar Spectrum. CNS Neuroscience & Therapeutics, 2012; 18: 194–200.

[7] John A. Sweeney, Julie A. Kmiec, and David J. Kupfer. Neuropsychologic Impairments in Bipolar and Unipolar Mood Disorders on the CANTAB Neurocognitive Battery. Biological Psychiatry, 2000; 48: 674–685.

[Abstract: 0732] [Mood disorders]

Unipolar/bipolar evolution of pediatric depression: phenotypical differences

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ABSTRACT

Dramatic increases in the frequency of bipolar disorder have been reported in the light of recent studies in the last 20 years [1]. In most patients, mood symptoms appear before the age of 20 (10-20% were reported before 10 years of age, and 60% were reported to have been onset before 20 years of age). It should be kept in mind that children who are diagnosed with depressive disorder during their adolescence may be experiencing first-episode bipolar depression. In prospective studies, the rate of conversion of major depressive disorder to bipolar disorder ranges from 7.7% to 49% [2–4], this rate increases to 50% if there is a family history. The delicate assessment of depressive symptoms in young people is important in the early diagnosis of bipolar disorder, in order for the child to complete normal development steps and to prevent serious losses in psychosocial development and education. Bipolar disorder indicates that the subthreshold findings are dense in the baseline, and show mixed and depressive symptoms. In the study Course and Outcome of Bipolar Illness in Youth (COBY) showed that children and adolescents with bipolar disorder had threshold and subthreshold mood changes in 60% of the 4 years, and only 40% had subthreshold and mostly depressive mood symptoms [5]. In the studies, the symptoms such as lability, hopelessness, social withdrawal, sweet food craving, lethality of suicidal behavior, non-suicidal physical self-injurious acts and insensitivity to pain while performing these behaviors were higher in young people with bipolar depression compared to young people with unipolar depression. In addition, those with a diagnosis of psychotic depression, those having a drug-induced manic / hypomanic episode and a family history of bipolar disorder have a high risk of developing bipolar disorder. The severity and the number of depressive episodes were higher in these adolescents than in unipolar youth. Again, young people with bipolar depression experience more and severe subthreshold manic symptoms compared to unipolar. These symptoms may include more psychomotor activity, grandiosity, distractibility, reduced sleep need, inappropriate laughs, jokes, increased productivity, and flight of ideas,

KEYWORDS

Unipolar; Bipolar; Depression; Phenotype; Child; Adolescent

pressurized speech, and hypersexuality. Child and adolescent onset Bipolar Disorder is a heavier clinical picture and has a worse prognosis. Therefore, carefully identifying mood symptoms during an acute depressive episode is important in terms of capturing important clinical clues about bipolar disorder in young people, even if there is no history of hypomania or mania. In this symposium, the symptomatology based distinction between bipolar and unipolar depression in young people with acute depressive episode will be discussed in the light of literature.

References

[1] Moreno C, Laje G, ve ark BC. National trends in the outpatient diagnosis and treatment of bipolar disorder in youth. Archives of general psychiatry 2007;64:1032–1039.
[2] Strober M, Carlson G. Bipolar illness in adolescents with major depression: clinical, genetic, and psychopharmacologic predictors in a three to four-year prospective follow-up investigation. Arch Gen Psychiatry 1982;39(5):549–555.
[3] Akiskal HS, Walker P, Puzantian VR, King D, Rosenthal TL, Dranon M. Bipolar outcome in the course of depressive illness: phenomenologic, familial, and pharmacologic predictors. J Affect Disord 1983;5(2):115–128.
[4] Rao UMA, Ryan ND, Birmaher B, et al. Unipolar depression in adolescents: clinical outcome in adulthood. J Am Acad Child Adolesc Psychiatry 1995;34(5):566–578.
[5] Birmaher B, Axelson D, Goldstein B, Strober M, Gill MK, Hunt J, Houck P, Ha W, Iyengar S, Kim E, Yen S, Hower H, EspositoSmythers C, Goldstein T, Ryan N, Keller M. Four-year longitudinal course of children and adolescents with bipolar spectrum disorders: The Course and Outcome of Bipolar Youth (COBY) study. Am J Psychiatry 2009;166:795–804.

[Abstract: 0738] [Psychopharmacology]

Psychopharmacological treatments in male sexual dysfunctions

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ABSTRACT

Male sexual dysfunctions are a common health problem that can affect men of all ages. According to DSM-5, male sexual dysfunctions include premature (early) ejaculation (PE), delayed ejaculation (DE), erectile disorder (ED), and male hypoactive sexual desire disorder (HSDD). Psychopharmacological treatments are an important treatment option for men who suffering from sexual dysfunction. PE is the most common sexual dysfunction in men, and there are various oral or topical pharmacological approaches for the treatment of PE. Pharmacotherapies for PE include selective serotonin reuptake inhibitors (SSRIs), tricyclic antidepressants, topical anesthetics, tramadol, and phosphodiesterase type 5 (PDE-5) inhibitors. SSRIs stand out as the first-line treatment for pharmacological treatment of PE. Although current clinical studies suggest that paroxetine may be the most effective drug among SSRIs, only dapoxetine has been formally registered for the treatment of PE. Dapoxetine is currently the only SSRI approved for on demand treatment for PE because of its rapid absorption and short initial half-life. If SSRIs are ineffective or cannot be tolerated, clomipramine can be used. Topical anesthetic creams and sprays, such as benzocaine, lidocaine or prilocaine, can be used to treat PE. Topical anesthetic agents are effective and well-tolerated, but they have potential side effects such as temporary loss of sensitivity and decreased sexual pleasure. Tramadol, an analgesic agent that is effective on opioid receptors, also inhibits serotonin and neuropinephrine reuptake. It can be prescribed when SSRIs and clomipramine are not effective. PDE-5 inhibitors also help in treating PE. Although there are a number of drugs to treat patients with DE (testosterone, cabergoline, bupropion, amantadine, cyproheptadine, midodrine, imipramine, ephedrine, pseudoephedrine, yohimbine, buspirone, oxytocin, and bethanechol), there is currently no pharmacological agent that has been shown to be effective for treatment. ED is one of the most common disorder affecting middle-aged and older men, and treatment of it is carried out with pharmacological treatments for many patients. PDE-5 inhibitors are first-line therapy if there are no specific contraindications (such as taking nitrates) to their use for most men with ED. The PDE-5 inhibitors used for the treatment of ED are sildenafil, tadalafil, vardenafil and avanafil. There is no evidence of significant differences in efficacy, safety, and tolerability between the PDE-5 inhibitors. While tadalafil has a longer duration of action, avanafil have a more rapid onset. Intracavernosal injection therapy with alprostadil or intraurethral and topical alprostadil are effective and well tolerated treatments for men with ED. Treatments with alprostadil should be recommended to patients as second-line therapy for ED. Several studies have suggested that yohimbine

KEYWORDS

Delayed ejaculation; erectile disorder; hypoactive sexual desire disorder; premature ejaculation; psychopharmacology

may be more effective than placebo in the treatment of psychogenic ED. In addition, in case male HSDD, which is another sexual dysfunction in men is due to low testosterone levels, testosterone replacement therapy should be recommended.

[Abstract: 0716] [Psychopharmacology]

Psychopharmacological treatments in female sexual dysfunctions

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ABSTRACT

Female sexual dysfunctions (FSD), which have a major impact on a women's and couples' quality of life, can occur throughout the lifespan. Despite the high prevalence rates, FSD is frequently underdiagnosed and untreated. FSD classified into four domains in DSM-5: Combination of former hypoactive sexual desire disorder (HSDD) and female sexual arousal disorder (FSAD) into female sexual interest/arousal disorder (FSIAD), female orgasmic disorder (FOD), genito-pelvic pain/penetration disorder (GPPPD) and substance/ medication-induced sexual dysfunction. As somatic, psychosocial and neurobiological factors contribute to FSD, treatment should follow a biopsychosocial model with consideration of physical, psychological, relational, and situational determinants. Sex therapy has been the standard treatment and there are only few pharmacologic treatments available for FSD. The most common sexual dysfunction in women is HSDD and flibanserin is the only approved medication to treat premenopausal women with generalized acquired HSDD by Food and Drug Administration (FDA). However, there is no treatment for FSD approved outside the USA. Flibanserin is a multifunctional serotonin agonist and antagonist (MSAA), increases levels of norepinephrine and dopamine while reducing levels of serotonin in prefrontal cortex, nucleus accumbens and medial preoptic area, all three of which are brain regions that regulate sexual desire in women. It has demonstrated statistically significant efficacy in terms of increasing sexual desire and satisfying sexual events while decreasing sexually related distress (Goldstein et al., 2017). Bupropion, a norepinephrine-dopamine reuptake inhibitor (NDRI), and buspirone, a serotonin 5-HT1A partial agonist, may be considered off-label, non-hormonal, centrally acting medications for HSDD, despite limited safety and efficacy data. Testosterone, estrogen and tibolon treatment are hormonal therapies for FSIAD. Another drug candidate for HSDD is the injectable bremelanotide. It acts as an agonist at the melanocortin 3 and 4 receptors and has recently successfully completed two phase 3 multicenter trials (Miller, Smith, Norman, & Clayton, 2018). Vasodilator drugs/ phosphodiesterase type 5 inhibitors, testosterone/buspirone combination (Lybridos) testosterone/sildenafil combination (Lybrido), topical lubricants are other options to enhance treatment of FSIAD. There is not any approved medication specifically for FOD. Sex therapy is the standard treatment for GPPPD. Pharmacotherapy can only be used in conjunction with psychotherapy for patients with high levels of anxiety. Pharmacological treatment for females has been more difficult due to complexity of defining and objectively measuring FSD in laboratory. Safety concerns, difficulty with FDA approval, cultural views on treating FSD and socio-cultural bias against women's sexual and reproductive health are other barriers to development of new pharmacological treatments. Future targets with possible effect on FSD are DRD3 dopamine agonist, apomorphine, bremelanotide, oxytocin, testosterone products, alprostadil, dehydroepiandrosterone, L-arginine. Due to significant prevalence and limited available treatments of FSD, a greater effort is needed for new studies. FSD can be complex with multifactorial etiology and their treatment should be individualized with multidisciplinary and multimodal approach.

KEYWORDS

Female sexual dysfunction; hypoactive sexual desire disorder; flibanserin

References

[1] Goldstein I, Kim NN, Clayton AH, et al. Hypoactive Sexual Desire Disorder: International Society for the Study of Women's Sexual Health (ISSWSH) Expert Consensus Panel Review. Mayo Clin Proc. 2017;92 (1):114–128.

[2] Miller MK, Smith JR, Norman JJ, et al. Expert opinion on existing and developing drugs to treat female sexual dysfunction. Expert Opin Emerg Drugs. 2018;23 (3):223–230.

[Abstract: 0727] [Psychopharmacology]

Sexual side effects and management due to psychopharmacological treatments

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ABSTRACT

The prevalence of sexual dysfunctions (SD) is highly prevalent in patients with psychiatric disorders compared to the general population, particularly in patients treated with psychotropic drugs. In these patients, SD may be related to their premorbid sexual activity, to the psychiatric disorder itself, to comorbid mental disorders, to comorbid physical diseases or concomitant medications and in particular to their psychotropic medication. Many sexual symptoms resolve as the improvement of the psychiatric disorder, but SD due to psychotropic drugs tend to persist throughout the treatment and are underrecognized by clinicians. It has been reported that 30–60% of patients with schizophrenia who use antipsychotic medication and 80% of patients with depression and anxiety disorders suffer from sexual side effects (SSE). Most psychotropic drugs may give rise to SD including decreased sexual desire, erectile dysfunction (ED), delayed ejaculation, anorgasmia, and, rarely, retrograde ejaculation (RE), painful ejaculation, priapism, restless genital syndrome and vaginal or penile anesthesia. The incidence and severity of SD caused by psychotropic drugs may be different in relation to differences in their mechanisms of action. Antidepressants with predominantly serotonergic activity, antipsychotics that may cause hyperprolactinemia, and mood stabilizers with hormonal effects are usually linked to moderate or severe SD. In the literature, it has been reported that SSE are not common with the antidepressants agomelatine, amineptine, bupropion, moclobemide, mirtazapine, nefazodone, vilazodone and vortioxetine. It has been found that nearly all other antidepressants are significantly associated with SD. Antipsychotics causing hyperprolactinemia such as haloperidol, risperidone, paliperidone and amisulpride are more likely to be associated with sexual problems (decreased libido and/or arousal difficulties). In contrast, aripiprazole, quetiapine, olanzapine and ziprasidone have been associated with relatively lower rates of sexual problems. It is known that lithium, one of the first treatment options in bipolar disorder, may cause SD (reduced sexual desire, erectile dysfunction and decreased sexual satisfaction). Valproate increases serum testosterone, androstenedione and dehydroepiandrosterone sulfate levels, resulting in reduced sexual desire in women. Carbamazepine leads to SD (decreased sexual desire, reduced libido, erectile problems) by reducing the levels of estradiol, progesterone and testosterone and increasing sex hormone binding globulin levels. Although there are some papers of anorgasmia and RE, oxcarbazepine and lamotrigine are not generally associated with hormonal alterations and SSE. Maintaining normal sexual life and treating the SSE (or at least talking about the burden of SSE) in medicated patients can affect positively quality of life, mood, self-esteem, attitude toward taking medication, and adherence. Importantly, Patients may tend not to talk about their sexual life with their clinicians. Therefore, clinicians should take initiative to talk about the patient's sexual life to be informed about drug-induced sexual problems. In this session, SSE due to psychopharmacological treatments and its optimal management will be discussed.

KEYWORDS

Sexual side effects; psychotropic drugs; sexual dysfunction; psychopharmacological treatments

References

[1] Waldinger MD. Psychiatric disorders and sexual dysfunction. Handb Clin Neurol. 2015;130:469–489.

[2] Just MJ. The influence of atypical antipsychotic drugs on sexual function. Neuropsychiatr Dis Treat. 2015;11:1655–1661.

[3] Montejo AL, Montejo L, Navarro-Cremades F. Sexual side-effects of antidepressant and antipsychotic drugs. Curr Opin Psychiatry. 2015;28(6):418–423.

[Abstract: 0627] [Neuroscience: Neuroimaging-Genetic Biomarkers]

Neurocognitive changes in emotional dysregulation: challenging evaluation for children and adolescent with psychopathology

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ABSTRACT
Emotion and cognition are two entities that have intriguingly connected each other. As cognition refers many domains of high-ordered actions like attention, executive functions (planning, problem solving and behavioral inhibition, for instance), memory and verbal fluency occurring in mainly hippocampal and prefrontal cortical region of the brain, emotion defines feelings experienced by at the end of some mental processes developing as a reciprocal interaction of limbic system (including amygdala, ventral striatum, thalamus, hypothalamus and periaqueductal gray) and higher cortical regions such as medial, orbital, and inferior lateral frontal cortices. Emotional regulation is a collection of behavioral spectra which is developed, initiated and modified with the use of sensing the sensory stimuli from the outer environment through the subcortical amygdala, categorizing them by processing in the frontal cortex via cognitive elements (such as memory) in the hippocampus, reward processes in the ventral striatum and collecting them in the anterior cingulate cortex and inferior frontal gyrus triangularis. Within emotional regulation, two cognitive elements, including cognitive reappraisal and expressive suppression occurring within the prefrontal cortex, play a considerable role [1]. Since the maturation of emotional regulation continues throughout developmental periods of childhood and adolescence, any disturbances of its regulation could result in a risk for developing or triggering of a psychopathology, or a psychiatric disorder could cause emotional dysregulation, vice versa. Studies related to this issue revealed that inefficient emotion regulation or emotional dysregulation (ED) is a condition that develops as a consequence of the problems in the strategies used to regulate the emotion and can occur either under-regulation (implicit that there is not efficient control of emotions displaying itself as impulsive, disruptive or aggressive behavior) or over-regulation (means that there is much more control than that of optimum emerging itself internalizing symptom as depression, anxiety or social withdrawal) of emotion [2]. The effects of emotional dysregulation on cognitive processes of attention or memory could be detected via related cognitive tasks. Attention and inhibitory control could be evaluated with go/no-go test, span and Stroop tests. Short or long-term memory deficits, executive dysfunctions, psychomotor speed and spatial abilities could also be examined by cognitive assessment batteries. Cambridge Neuropsychological Test Automated Battery (CANTAB) is one of objective measurement tool of cognitive functions [3]. In one thing that accounts for consideration is that ED might a contributor factor for a varied psychiatric impairments from anxiety disorders, Attention-deficit/ hyperactivity disorder to eating disorder in children or adolescence yet it was not primarily a consequence of psychopathology. Here, there is another issue that whether this cognitive symptom is because of emotional dysregulation itself or this symptom results from underlying psychiatric disorder. Consequently, there is an apparent need for discrimination of cognitive symptoms whether it is based on emotional regulation or psychiatric disorder itself. In this presentation, there will be summarized cognitive symptoms in emotional dysregulation and differential diagnoses will be discussed.

KEYWORDS
Emotional dysregulation; cognitive functions; CANTAB; psychiatric disorders

References

[1] Gross JJ. Emotion Regulation: Affective, Cognitive and Social Consequences. Psychophysiology 2002; 39: 281–291.
[2] Wenar C, Kerig P. Developmental Psychopathology From Infancy Through Adolescence. 5th revised ed., McGraw Hill Higher Education; 2005, p. 696.
[3] Robbins TW, Sahakian BJ. Computer Methods of Assessment of Cognitive Function. In Copeland JRM, Abou-Saleh MT, Blazers DG, editors. Principles and Practice of Geriatric Psychiatry. Chichester: Wiley; 1994. p. 205–209.

[Abstract: 0750] [Neuroscience: Neuroimaging-Genetic Biomarkers]

Neuroimaging studies in emotional dysregulation

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ABSTRACT
Emotional regulation definition is described as a group of intrinsic and extrinsic processes which observe, assess, and alter an emotional reaction to a stimulus. Emotional dysregulation (ED) can be considered as the deficiency of these processes. These processes are concluded from underlying neurophysiology and are also determined by environmental, biological, and genetic factors and the interactions between these factors [1]. This presentation will aim to explain the neurological pathways in children and adolescents. Firstly, we will discuss the brain regions involved in emotional regulation and then ED in some specific psychiatric disorders.

KEYWORDS
Emotion dysregulation; neuroimaging; amygdala; prefrontal cortex; children

Functional emotional regulation requires the coordination of multiple high-level processes. Development of these cognitive processes need to the protracted development of some areas of prefrontal cortex (PFC) and the connections between prefrontal and limbic regions. PFC is the main center of generation and maintenance of emotion regulation strategies. Dorsolateral prefrontal cortex (DLPFC) is associated with cognitive control processes such as coordinating thoughts and behaviors in compliance with internally represented goals. This is implicated in the down-regulation of negative emotion. Inferior frontal gyrus traingularis (IFGT)/ ventrolateral prefrontal cortex (VLPFC) take part in when reducing subjective negative affect during effortful regulation. The VLPFC is also thought to play significant role in inhibition of emotional and non-emotional stimulant. Medial prefrontal cortex (MPFC) integrates inputs from subcortical regions related to memory, cognition, attention, and emotion. This region also plays role in Theory of Mind and understanding social emotions. Anterior cingulate cortex (ACC) has bidirectional neuronal connections with dorsolateral, orbitofrontal, and insular regions of the cerebral cortex. Orbitofrontal cortex (OFC) is linked to the expected rewards and punishments of an action and is important for adaptive learning. Finally, amygdala is a collection of nuclei which has extensive connections with several regulatory regions. It is associated with learning and expressing fear response as well as determining prominence of emotional stimuli [2]. Studies evaluating children with antisocial and aggressive behavior indicated that functional connectivity between specific brain areas may take part in ED. These researches showed that there has been an inverse correlation between right ACC thickness and bilateral striatal volume in aggressive children and adolescents, suggesting that these brain areas are crucial for aggression and emotional impulsivity. Moreover, it is thought that functional connectivity between amygdala and PFC might be associated with poorer processing of emotional expressions which is found in children with the callous unemotional traits. Several neuroimaging studies suggest that while youths with anxiety disorders show less functional connectivity in prefrontal-amygdala connections, adolescents with externalizing disorders exhibit less functional connectivity between prefrontal cortex and striatum [3].

References

[1] Geise C, Barzman D, Strakowski S. Pediatric Emotion Dysregulation : Biological and Developmental Evidence for a Dimensional Approach. The Psychiatric Quarterly 2014; 85(3): 383–389.

[2] Ahmed SP, Bittencourt-Hewitt A, Sebastian CL. Neurocognitive Bases of Emotion Regulation Development in Adolescence.Developmental Cognitive Neuroscience 2015; 15: 11–25.

[3] Beauchaine TP. Future Directions in Emotion Dysregulation and Youth Psychopathology. Journal of Clinical Child and Adolescent Psychology 2015; 44(5): 875–896.

[Abstract: 0644] [Others]

Transdiagnostic approach to emotional dysregulation

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ABSTRACT

Emotions and their regulation are related to various interconnected processes, including biological, psychological, cultural, and interpersonal domains. Better emotional regulation skills in children are associated with better peer relationships, enhanced prosocial behaviors, and social competence [1]. Emotional dysregulation (ED) is defined as problems in monitoring, evaluating, or controlling an emotional reaction [2]. There have been lots of studies demonstrating relationship between ED and internalizing and externalizing psychopathology in childhood. Attention deficit and hyperactivity disorder (ADHD) has been related to increased emotional reactivity/negativity and problems in emotional regulation. 25% to 50% of children/youths with ADHD seems to have difficulties in emotional regulation. Intriguingly, ED is one of the main reasons of functional impairment in children with ADHD. Children/youths with depression or anxiety disorders have been also reported to have poorer emotional understanding and difficulties in managing negative emotions. Furthermore, a recent diagnosis, in which difficulties in controlling negative emotions is a core symptom, is proposed by DSM-5: Disruptive mood dysregulation disorder. This new diagnosis is characterized by chronic and severe persistent irritability along with tempur tantrums. Children with disruptive mood dysregulation disorders are more likely to develop depressive disorder or anxiety disorder in adulthood.

In addition, women with eating disorder have difficulties regarding describing their emotions. Anorexia nervosa has been tried to be explained by Linehan's bio-psychosocial model of emotion dysregulation [3]. This model suggests that emotional dysregulation

KEYWORDS

ADHD; anxiety; childhood; depression; emotion dysregulation; transdiagnostic

emerges by a transactional process which is between emotional vulnerabilities of an individual and invalidating responses to this individual from his/her environment. Individual vulnerability describes temperamental features (i.e. emotional sensitivity, reactivity) and transient factors (i.e. sleep habits, diet, physical health etc.). Invalidating emotions describe the situation when an individual's emotional needs are ignored, misunderstood, or criticized by others (e.g. family members, teachers, peers etc.). Linehan's model is also used to enlighten emotional dysregulations in borderline personality disorder as well. Some researchers suggest that eating behaviors in eating disorders may be a maladaptive emotional regulation technique similar to self-harm behaviors in borderline personality disorder [3]. Importantly, ED plays a central role in the etiology of non-suicidal self-harm behaviors. In line with this, alleviating unwanted emotions is the most frequently reported reason of non-suicidal self-harm behaviors. In addition to this, individuals with substance use disorder have heightened levels of ED. When non-suicidal self-harm behaviors accompany to substance use disorder, problems in emotional dysregulation increases.

A longitudinal study that examined reciprocal relationship between ED and symptoms of psychopathologies showed that ED during adolescence predicted increases in anxiety symptoms, aggressive behavior, and eating pathology. In conclusion, ED is a significant transdiagnostic situation that influence course of psychopathologies. In this session, it is aimed to be discussed the mechanisms of emotion generation and emotion regulation; emotional regulation strategies across psychiatric disorders; prevalence and effects of emotional dysregulation among different psychopathologies.

References

[1] McLaughlin KA, Hatzenbuehler ML, Mennin DS, Nolen-Hoeksema S. Emotion dysregulation and adolescent psychopathology: A prospective study. Behav Res Ther 2011;49(9):544–554.

[2] Thompson RA, Calkins SD. The double-edged sword: Emotional regulation for children at risk. Dev Psychopathol 1996;8(01):163.

[3] Crowell SE, Beauchaine TP, Linehan MM. A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. Psychol Bull. 2009;135(3):495–510.

[Abstract: 0799] [Others]

The genesis of artistic creativity

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ABSTRACT

Creativity is the innate capacity of man and genius is the instinctive and extraordinary capacity for creation. It requires massive persistent, high levels of energy, motivation and great capacity for observation with attention to detail. Einstein, who had autism had a massive capacity for visual imagination. These persons with autism reject received wisdom and are emotionally immature. This is different from savantism, where a person with autism and low intelligence possesses an unusually high skill in for example, arithmetic. Indeed, autistic intelligence is unconventional, unorthodox akin to the intelligence of true creativity. In the last one thousand years, Isaac Newton was the greatest figure with autism and the past hundred years, Einstein. Others in history would include Lewis Carroll, Ludwig Wittgenstein, Archimedes, Telsa, Cavendish, Mendeleyev, Darwin, Dirac, Fisher, Jefferson, Joyce, Kinsey, Kubrick, Michelangelo, Mozart, Beethoven, Yeats, Hans Christian Anderson, Shakespeare, Dick, Capote, Schiller, Ibsen, Pirandello, Chekhov, Disney, Hitchcock, Marx, Sellers, Niven, Garbo, Valentino, Sinatra, Burton, Guinness, Houdini.

KEYWORDS

Autism; creativity; mathematics; science; literature; philosophy

[Abstract: 0728] [Psychotherapy]

Psychotherapy, dreams, and theoretical basis of PDSM

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ABSTRACT
Whether it is in traditional or modern context, dreams have a special place in both psychology and psychotherapy studies. 'The Traditional Interpretive Dream Approach' model which we had come across its first remains in Mesopotamia and is also the inspiration for psychoanalysis, is based on the interpretation of the symbols in the dream and the principle of reaching the hidden meaning. However, what is done with this interpretation is to move away from the phenomenal subjective reality of the client. In modern psychotherapy applications, a reliable and effective method for working with dreams has not been developed yet. Although most therapists think that dreams are therapeutically valuable, the number of therapists who work with dreams is few. Especially young therapists find the theories of dreams as speculative, mysterious, confrontational, and difficult to understand. All these problems are related to two basic assumptions on which dream theories in modern psychology are based. The first is the belief that dreams originate from the unconscious. The second is the idea that mental processes in wakefulness and dreams are qualitatively different. Rapidly growing phenomenological and neuroscientific studies of dreams in recent decades show that these two assumptions are not true. Both the difficulties in implementation and the latest scientific data on dream and consciousness force clinicians to develop a new dream model. In this respect, we have developed a new dream model that we call the PDSM based on the dream work that we have been continuing with a phenomenological attitude for many years. The feedback of the therapists who use the model is that the model's dreams are effective and reliable in therapy. In the forthcoming period, studies are planned to test the effectiveness of the model in different groups of clients. As the name implies, the PDSM is a self-focused model. The model takes dreams as a life and does not distinguish their dream life from qualitative vigilance experiences. In dreams, as in wakefulness, there is an accessible self through consciousness. The dream self is not different from the awakening self in terms of basic phenomenological qualities This dream self is like the waking self, it is at a "moment", in a "place", in "perception", and in "emotion". This dream self is always in an intentionality for an object just like the waking self. If dreams are the experiences of a self, the most reliable way to understand life is to describe life as what it is. The importance of phenomenology in our sense is that it gives a method of depicting life. With a four-stage process, the PDSM examines the client's experience of the dream self first and then the experience of waking self, with phenomenological sensitivity. The most important point that differentiates PDSM from the previous dream theory and models is that it doesn't separate the individual from the phenomenal experience in all four phases and it is avoided to impose something that the clients haven't said about their dreams by depending on some theories and assumptions.

KEYWORDS
Phenomenological dream self-model; dream works; psychotherapy; self

[Abstract: 0761] [Neuroscience: Neuroimaging-Genetic Biomarkers]

Current neuroscience and PDSM studies about dreams

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ABSTRACT
Dream can be described as a special state of consciousness which is comprise of complex internal activities of specific brain regions leading to sensory, emotional and cognitive experiences during sleep. Dreams can be detected both in REM and NON-REM stages of sleep but mostly reported during REM stage. Studies revealed particular decrease in low-frequency activity in posterior cortical regions in both NREM and REM stages during reported dreams. Brain imaging studies determined particular regional brain activities. specific to REM stage. Limbic and paralimbic structures, basal forebrain, thalamus, pontine tegmentum, amygdala, anterior cingulate cortex and hippocampal formation are the regions those have increased activity during REM sleep. Studies have also shown that temporal visual cortex , primary motor, premotor cortex and cerebellum and basal ganglia reveal high activation during REM sleep thought to be consistent with the sensory visual and motional perception in dreams. The regions such as the medial prefrontal cortex, the medial temporal lobe, the lingual gyrus and the caudate nucleus which are active in REM sleep, are also found to be more active when compared to resting state during waking. Since the regions of default mode network (DMN; brain activity network of regions revealing increased activity and connectivity during resting state) has been asserted to form an imagery network, it has been proposed that the DMN associated with all types of mental imagery and it may be acceptable for the visual imagery that occurs while dreaming.

KEYWORDS
Dreams; Neuroscience; REM sleep

References

[1] Desseilles M. et al. Cognitive and emotional processes during dreaming: A neuroimaging view. Consciousness and Cognition 20;2011:998–1008.
[2] Domhoff GW, Fox KCR. Dreaming and the default network: A review, synthesis, and counterintuitive research proposal. Consciousness and Cognition 33;2015:342–353.
[3] Siclari F. et al. The neural correlates of dreaming. Nat. Neurosci. 2017;20(6):872–878.

[Abstract: 0730] [Psychotherapy]

General framework of PDSM: process and technique

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ABSTRACT

The Phenomenological Dream Self Model (PDSM) is a four-stage model. According to this model, in the first stage of the dream work, the life of the dream self is described in terms of feelings, thoughts, intentions and behaviors without any interpretation. In the second stage, it is described that how would the waking self-experience or behave in the situations similar to dream experiences. In the third stage, the dream and waking selves are compared over the descriptions in the first two stages. In the fourth stage, the possibilities of new and different meaning layers of the dream life are searched based on the description in the first stage and the life story of the client. Since both dream life and waking life experiences belong to the client's self, by using PDSM the client can discover or reconstruct the basic intentions of him/herself. The purpose of the therapist working with the PDSM is not to tell the client something new but to try to make them to hear his/her own voice. For many dreams, the first three phases of PDSM are sufficient for therapeutic work. Studying the fourth phase becomes important especially in the dreams where metaphoric and symbolic elements are dominant. For PDSM, metaphors and symbols are not the means of hiding something; on the contrary they are the means of being able to reveal and explain. The model requires the therapist to stay on the phenomenological descriptive attitude in the first three stages. Before these four stages, there is the preparation phase. The therapist should listen to the client's assumptions and beliefs about dreams and explain the basic assumptions of the model to the client in this preparation phase. The purpose of this preparation phase is to provide a partly reconciliation related to dreams between the therapist and the client. With a four-stage process, the PDSM examines the client's experience of the dream self first and then the experience of waking self, with phenomenological sensitivity. In the third phase, the parallels and angle differences are interpreted by comparing two dream experiences and finally the associations about daily/past experiences are examined without departing from the dream self-experience. The most important point that differentiates PDSM from the previous dream theories and models is that it does not separate the individual from the phenomenal experience in all four phases. PDSM work avoids imposing something that the client has not said about his/her dream by depending on some theories and assumptions. Thus, the client's subjectivity is respected and kept safe from the risk of therapist's intervention. In conclusion, we think that PDSM is a practical and effective dream model for clinicians who want to perform self-consciousness-based dream work in psychotherapy practices. In future studies, we aim for qualitative and quantitative research on the clinical benefits of PDSM.

KEYWORDS

Phenomenological dream self-model; dream; self; psychotherapy

[Abstract: 0698] [Psychotherapy]

Trauma-oriented approach in treatment-resistant depression

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ABSTRACT

Traumatic events experienced in different periods of life may soon alter negative psychological changes. These negative shifts might be indications of pathology depending on their duration and consequences. In cases of Post-Traumatic Stress Disorder with delayed expression (PTSD with delayed expression), which is a trauma-related disorder, stress related symptoms are

KEYWORDS

EMDR; depression; psychotherapy

observed after 6 months or later following traumatic event. Although the clinical picture be complicated, scrutinizing the patient's existing symptoms and psychiatric history, detecting underlying problems and traumas, and then conducting a proper treatment accordingly may help provide recovery for its patients. In this case study, treatment of a PTSD with delayed expression comorbid Major Depressive Disorder (MDD) resistant to pharmacotherapy, which emerged after a childhood sexual trauma had become triggered in adulthood, will be discussed. EMDR is a feasible treatment for recurrent and/or long-term depression. Research on treatment efficacy and effectiveness is now required.

[Abstract: 0751] [Psychotherapy]

Trauma-oriented approach in social anxiety disorder

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ABSTRACT

Etiological models of social anxiety disorder (SAB) show that early childhood trauma contributes to the development of this disease. However, in SAB, surprisingly little is known about the connection between different childhood trauma forms and adult clinical symptoms. Healthy operation with this study generalized SAB compared the levels in adult and childhood trauma SAB 'have also examined the relationship between specific types of childhood trauma to adult clinical symptoms. Participants were included in a generalized study that completed the criteria of childhood trauma, social anxiety, trait anxiety, depression and self-confidence. Compared to healthy controls, individuals with SAD reported more emotional abuse and emotional neglect in childhood. In the SAD group, emotional abuse and neglect in childhood were associated with sexual abuse, physical abuse or physical neglect, social anxiety, susceptibility to depression, and lack of self-esteem. In this presentation, we will look at social anxiety disorder in terms of trauma and how to approach trauma-oriented treatment.

KEYWORDS

Trauma; social phobia; psychotherapy

References

[1] Wilson B, Butler LD. Running a gauntlet: A review of victimization and violence in the pre-entry, post-entry, and peri-/post-exit periods of commercial sexual exploitation. Psychological trauma: theory, research, practice, and policy 2014;6(5):494.

[2] Nachar N, Lavoie ME, Marchand A, Guay S. The effect of talking about psychological trauma with a significant other on heart rate reactivity in individuals with post-traumatic stress disorder. Psychiatry research 2014;219 (1):171–176.

[Abstract: 0669] [OCD]

OCD from trauma window

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ABSTRACT

Obsessive-Compulsive Disorder (OCD) is a common, chronic and long-lasting disorder in which a person has uncontrollable, reoccurring thoughts (obsessions) and behaviors (compulsions) that he or she feels the urge to repeat over and over. Obsessions are recurrent and persistent thoughts, impulses or images that are experienced in an intrusive and inappropriate way, cause marked anxiety and distress, and persist despite all attempts to try to ignore or suppress them. Compulsions are repetitive behaviors or mental acts that a subject feel driven to perform in response to obsessions and are aimed at preventing or reducing anxiety. The frequency of OCD is reported to be 1–3% (1). The etiology is not known exactly. Research is ongoing in this area. Regarding psychopharmacological therapy, serotonergic agents, especially selective serotonin re-uptake inhibitors (SSRI), are used as a first-line treatment in children and adolescents. But, between 40% and 60% of OCD patients fail to respond to SSRI treatment and require augmentation therapy. Cognitive behavioral therapy (CBT) based on exposure and response prevention (ERP) is recommended by clinical guidelines as a first-line psychological treatment for this condition. Also, recent studies have considered whether eye movement desensitization and reprocessing (EMDR) could be a

KEYWORDS

Childhood trauma; EMDR; Obsessive-Compulsive Disorder

helpful alternative treatment for OCD (2). Eye Movement Desensitization and Reprocessing (EMDR) therapy has been widely recognized as an efficacious treatment for post-traumatic stress disorder (PTSD). In the last years more insight has been gained regarding the efficacy of EMDR therapy in a broad field of mental disorders beyond PTSD. The cornerstone of EMDR therapy is its unique model of pathogenesis and change: the adaptive information processing (AIP) model. The AIP model developed by F. Shapiro has found support and differentiation in recent studies on the importance of memories in the pathogenesis of a range of mental disorders beside PTSD. People who have experienced abuse (physical or sexual) in childhood or other trauma are at an increased risk for developing OCD. There is evidence that in some cases, OCD may originate in the wake of stressful life events and that stressful life events increase the risk of OCD relapse (Steketee, 1993). It has been reported that there may be a relationship between childhood trauma and OCD among people's past experiences with others and difficulties in emotion processing. Based on the notion that EMDR works to resolve disturbing memories of traumatic events, it could be that other types of anxiety disorders that develop following a distressing event may also be responsive to EMDR. Some study is consistent with some evidence that childhood trauma may play a role in the development of these disorders. In this verbal session speech, it is planned to explain the development of OCD on the basis of trauma in the light of literature.

References

[1] American Psychiatric Association. Diagnostic and statistical manual of mental disorders. DSM-5 diagnostic criteria for OCD disease. 1st ed. Washington (DC): American Psychiatric Association; 2013.
[2] Nazari H, Momeni N, Jariani M, Mohammad JT. Comparison of eye movement desensitization and reprocessing with citalopram in treatment of obsessive–compulsivedisorder. International Journal of Psychiatry in Clinical Practice 2011;15:270–274.
[3] Steketee, G. Social support and treatment outcome of obsessive compulsive disorder at 9-month follow up. Behavioural and Cognitive Psychotherapy 1993;21:81–95.

[Abstract: 0752] [Anxiety disorders]

Considering diagnosis from a different perspective can make a big change: panic disorder case study from a trauma focused approach and treatment with EMDR therapy

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ABSTRACT
This oral presentation begins with the diagnostic definition of 'panic disorder' according to DSM-V criteria. Then, it proceeds with the detailed information about the presented case; the demographical and familial information, patient's presenting problem, the onset, history and the development of panic disorder. The treatment formulation especially focuses on patient's trauma history, how some of the traumatic experiences contributes to patient's ongoing panic disorder and anxiety symptoms and the current triggers of the presenting problem. The presenter then mentions how EMDR (Eye Movement and Desensitization and Reprocessing) Therapy formulates and works with panic disorder; the 'panic disorder protocol'. Finally, the presentation propounds session by session showing how the treatment progressed by working with patient's related traumatic memories and triggers, how symptoms improved and therapy process had been terminated.

KEYWORDS
Panic Disorder; panic attacks; EMDR; case study

[Abstract: 0812] [Others]

Auditory processing in ADHD

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ABSTRACT
Attention-deficit/ hyperactivity disorder (ADHD) is a neurobiological condition characterized by symptoms of inattention, hyperactive, and impulsive behaviors. It is the most common psychiatry disorder with 5.3% prevalence in school children population and high association with comorbidities, such as Oppositional Defiant Disorder (ODD). Auditory Processing (AP) refers to the perceptual processing of auditory information in the Central Nervous System (CNS), including sound localization, auditory discrimination, auditory pattern recognition, temporal aspects of hearing during exposure to competitive, and degraded acoustic signs. Auditory Processing Disorder (APD) refers to auditory perceptual difficulties that are not related to peripheral hearing deficits or language and cognitive dysfunctions. APD children may have difficulties hearing in noisy environments, understanding instructions, reading, and spelling, as well as poor concentration and impaired memory. Studies have suggested that ADHD children show sensory processing deficits, in which the underlying pathophysiology is poorly understood. Although APD and ADHD have overlapping clinical characteristics, they are distinct entities, requiring accurate diagnoses and appropriate interventions. According to some studies methylphenidate MPH may improve the deficits of auditory information processing in ADHD children with no influence over information inputs. However, MPH effect data on the auditory processing in ADHD children are limited.

[Abstract: 0810] [Others]

The role of cerebellum in ADHD

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ABSTRACT
Attention deficit/hyperactivity disorder is a persistent pattern of inattention and/or hyperactivity/impulsivity behavior. ADHD-related difficulties affect other functional skills for daily living, for example, problem solving, emotional/motivational self-regulation, deficits in motor coordination, or time management. Deficits in fine motor coordination affect up to 30–50% of ADHD cases. Furthermore handwriting performance in children with ADHD were adversely affected by stimulant therapy. But the association between ADHD and motor coordination problems are still unclear. Difficulties in motor co-ordination can result in clumsiness, perhaps explaining the high rates of injury in children with ADHD and poor handwriting and performance in sports. Fenollar-Cortés et all (2017) showed the children with ADHD had poorer fine motor performance than controls across all fine motor coordination tasks and this result was related inattention dimension. Anatomical, physiological and functional neuroimaging studies suggest that the cerebellum participates in the organization of higher order function, but there are very few descriptions of clinically relevant cases that address this possibility. neuropsychological and anatomical neuroimaging studies show that cerebellum plays an important role in executive functions such as planning, set-shifting, verbal fluency, abstract reasoning and working memory; visual-spatial organization and memory, language and attention. Although several studies suggest heritability of ADHD, Rapin et al. (2017) show that parents of children with ADHD exhibited significantly increased neural activations in the posterior lobes of the cerebellum. These findings suggest the fronto-cerebellar circuit's abnormalities in parents of children with ADHD. Poissant et al. also showed that ADHD children have more activation in the cerebellar vermis during incongruent stimuli from cartoon stories representing sequences of action. to compensate activation of prefrontal cortex. A high seroprevalence of Yo antibodies targeting cerebellar Purkinje cells was recently reported in children with attention-deficit/ hyperactivity disorder (ADHD). An Italian study has suggested a high prevalence of cerebellar Purkinje cell specific antibodies in children with ADHD. Yo antibodies were detected in the serum of 26 out of 30 ADHD children, and in none of the 27 sex- and age-matched healthy controls. But no associations between ADHD and serum Yo antibodies or other antibodies associated with PNS were found in another study with 169 adult ADHD patients. However, as such studies are still lacking for ADHD, it is not possible to conclude that such serum antibodies have a pathogenic role in this disease. Cerebellar dysfunction is evident in several developmental disorders, including autism, attention deficit-hyperactivity disorder (ADHD), and developmental dyslexia, and damage to the cerebellum early in development can have long-term effects on movement, cognition, and affective regulation. Further studies are needed to clarify the role of cerebellum in ADHD.

KEYWORDS
ADHD; cerebellum; anti-Yo; motor coordination; inattention

References

[1] Schmahmann JD, Sherman JC. The cerebellar cognitive affective syndrome. Brain: a journal of neurology 1998;121(4):561–579.
[2] Passarelli F, Donfrancesco R, Nativio P, Pascale E, Di Trani M, Patti AM, Villa MP. Anti-Purkinje cell antibody as a biological marker in attention deficit/hyperactivity disorder: a pilot study. Journal of neuroimmunology 2013;258(1):67–70.
[3] Rapin L, Poissant H, Mendrek A. Atypical activations of fronto-cerebellar regions during forethought in parents of children with ADHD. Journal of attention disorders 2017;21(12):1050–1058.

[Abstract: 0685] [Disruptive behavior disorders]

Listening to kids with ADHD in virtual environments: technology and ADHD

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ABSTRACT

On the one hand, we can say that the technology has its benefits and the losses on the other hand. While technology does not stop improving, is moving on the path of continuous development, we can define technology as the blending of the knowledge and skills necessary to make the tools suitable for the needs of human beings in general. Society and technology are always evolving. It's not that technology itself is bad, but rather what we do with it. As a relatively new phenomenon, most popular outcomes of technological advances adolescents use, social media and games have no concrete rules to governing these platforms or a set age for when children should be allowed to join. Attention-deficit/hyperactivity disorder (ADHD) is a brain disorder characterized by a pattern of inattention and/or hyperactivity-impulsivity that interferes with daily functioning. People with ADHD often struggle with time management, organization, completing tasks and not paying attention to details. This can make it difficult to stay functionally in school. A study of 29 ADHD children (and 21 children in a control group) between 6 and 16 years old found that ADHD children were more vulnerable developing a dependence on video games. In another study conducted by 535 primary school students, it was found that there was a significant relationship between the level of ADHD symptoms and the severity of internet addiction in children. This study suggests that the presence of ADHD symptoms may be important risk factors for Internet addiction. To prevent the technology from dominating life, a number of arrangements must be made. It is possible to limit the use of these tools at sometimes of the day and in some places. While living with children and adolescents with ADHD, it is important for parents to emphasize these limits. There are time reminder softwares that restricts usage in technology tools. Proper use of these will prevent many possible negative consequences. The effects of technology should not be addressed in one way. For example, applications and computer programs can help people with ADHD stay organized, reach goals, and even deal with distraction. For children, electronic timers help students stay on the job and accelerate themselves as they work. In addition, the use of technology in the classroom helps prevent distraction in some cases. A study of third and fourth grade students with ADHD found that computer-based instruction in mathematics led to an improvement in mathematics performance and an increase in behavior in the task.

When used correctly, digital tools can help people with ADHD to remember, to improve focus, to increase productivity, and do their work on time.

KEYWORDS

ADHD; technology as a help; technology as a hindrance

[Abstract: 0621] [Autism]

What potential role could genetics have in management of ASD?

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ABSTRACT

Autism spectrum disorder (ASD) is characterized by socio-communicational deficits and restricted/ repetitive behaviors, with early onset. There is a growing body of research in autism genetics and progress has been made in recent years with the developing molecular genetic analysis technology. Genetic variations now can be identified in almost one third of individuals with ASD and single gene disorders account up to 5% of cases. Genetic

KEYWORDS

Autism; gene therapies; genetics; pharmacogenomics

mechanisms underlying ASD seems to be as complex and heterogenous as its clinical heterogeneity, including single gene disorders to polygenic mechanisms to epigenetic alterations. Thousands of related genetic variations and more than 100 genes have been identified so far [1]. While it will be undoubtfully challenging, translation of this ever-increasing genetic information into clinical care would offer enormous opportunities in different aspects of management of individuals with ASD. To name a few, knowledge of genetic background of ASD may indicate certain biological pathways and may allow targeted pharmacological interventions and gene therapies. Precision of pharmacogenomic changes may influence pharmacological treatment choices [2]. Cognitive and behavioral profile may also vary between different subtypes of genetic variations and guide our choice of non-pharmacological intervention [3]. Moreover, understanding certain causative genetic variants would also ensure more precise information for genetic counselling [3].

References

[1] Huguet G, Benabou M, Bourgeron T. The Genetics of Autism Spectrum Disorders, in A Time for Metabolism and Hormones. In Sassone-Corsi P, Christen Y, editors. Cham: Springer International Publishing; 2016, p. 101–129.

[2] Bowers K, Lin PI, Erickson C. Pharmacogenomic medicine in autism: challenges and opportunities. Paediatr Drugs 2015;17(2):115–124.

[3] Vorstman JAS, et al., Autism genetics: opportunities and challenges for clinical translation. Nat Rev Genet 2017;18(6):362–376.

[Abstract: 0622] [Autism]

Importance of genetic consultation in clinical practice of autism spectrum disorder

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ABSTRACT

Many factors may contribute to the occurrence of autism. One of the most important factors is genetic. Genetic factors can cause autism by altering neurodevelopment, especially neural connectivity. Genetic consultation for autism is important in clinical practice. The American Academy of Child & Adolescent Psychiatry (AACAP)'s Practice Parameter for ASD states that clinicians should coordinate an appropriate multi-disciplinary assessment of children with ASD, including genetic consultation. Because, in any periods of the development of children with ASD, clinical care needs may arise due to genetic causes. For instance, a tumor follow-up program should be applied in the comorbidity of PTEN and autism. However, the diagnostic yield of genetic consultation varies according to the type of test applied. In a previous study, McGrew et al. found that diagnostic yield was 2.5% for karyotype testing, 0.57% for Fragile X testing and 24% for CMA. Clinicians should endeavor to keep in mind that patients with ASD may also have comorbidity of genetic syndromes in order to have chance for early diagnosis and necessary interventions.

KEYWORDS

Autism; genetic consultation; clinical practice

References

[1] Volkmar F, Siegel M, Woodbury-Smith M, King B, McCracken J, State M, American Academy of Child. Practice parameter for the assessment and treatment of children and adolescents with autism spectrum disorder. *Journal of the American Academy of Child & Adolescent Psychiatry* 2014;53(2): 237–257.

[2] McGrew SG, Peters BR, Crittendon JA, Veenstra-VanderWeele J. Diagnostic yield of chromosomal microarray analysis in an autism primary care practice: which guidelines to implement? *Journal of autism and developmental disorders* 2012;42(8):1582–1591.

[Abstract: 0688] [Others]

Regional and international collaboration for bipolar disorders: EABF, ANBD, and ISBD

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ABSTRACT
The East Asian Bipolar Forum (EABF) consists of experts of bipolar disorders from China, Japan, South Korea, and Taiwan, and has worked together to promote education and research of bipolar disorders in East Asia since 2007. It also had its biennial symposia from 2010, and was successful to conduct a multi-national collaborative research which resulted in publishing a paper. The Asian Network of Bipolar Disorders (ANBD) also started to be developed in 2007, and was successful to have its first conference in 2008, and had its 10th conference in 2017. The EABF and the ANBD contributed greatly to promote bipolar disorders in the respective area, especially to increase awareness, to enhance accurate and early diagnosis, to disseminate knowledge related with proper management of bipolar disorders, including the issue related with unopposed antidepressants prescription in bipolar depression. The EABF and the ANBD collaborated with the International Society for Bipolar Disorders, as a regional representative. What's noteworthy is the World Bipolar Day, which is cerebrated each year on March 30th, the birthday of Vincent Van Gogh, who was posthumously diagnosed as probably having bipolar disorder. The World Bipolar Day was initially proposed by the ANBD modifying the Bipolar Screening Day in South Korea, under the leadership of the past Chairman Pichet Udomratn. The proposal was adopted and developed into the World Bipolar Day from 2014, by the supportive collaboration of the International Society for Bipolar Disorders, and the International Bipolar Foundation. The initiative, development progress, difficulties and limitations of regional collaboration will be presented. Suggestions for future development will be also discussed from the experience of East and Southeast Asian collaboration in the field of bipolar disorders.

KEYWORDS
Bipolar disorder; international collaboration; early diagnosis

[Abstract: 0691] [Psychopharmacology]

Psychopharmacology & polypharmacy

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ABSTRACT
Despite that clinical guidelines recommend monotherapy as the preferred therapy for the treatment of pediatric psychiatric disorders, the use of psychotropic polypharmacy (≥2 medications) is quite common in clinical practice [1]. Overall the prevalence of psychotropic polypharmacy ranges from 14% to 73% among pediatric population [2]. It is important to clearly define the current terms used for simultaneous psychotropic prescription. *Copharmacy*, describes the pharmacologic treatment of different disorders with two or more medications. *Concomitant psychotropic medication,* is the use of two or more medications for either the same or different psychiatric symptoms or disorders [3]. In this context, the risk of drug-drug interaction is increased in the presence of both kinds of polypharmacy. Therefore, in this study, psychotropic drug-related problems (polypharmacy, drug-drug interactions, off-label psychotropic drug usage) in pediatric inpatients were evaluated in a university hospital by the child and adolescent psychiatrists and clinical pharmacists. This retrospective study included 200 inpatients who were consulted to the Child and Adolescent Psychiatry clinic and that received psychotropic medication between January 2016 and September 2017. The mean age (standard deviation) was 11.88 (4.13) years and 5.41 (3.52) months. Of the patients, 118 (59%) were female, 4 (2%) were smoker, 4 (2%) were alcohol consumer and 2 (1%) were substance abuser. Sixteen patients (8%) had a psychiatric family history, 13 (6.5%) were exitus. While the average total number of psychiatric drugs they used during hospitalization was 1.29 (0.55), the total number of drugs was 7.39 (4.45). The majority of patients were in general pediatric service (61; 30.5%), intensive care unit (30; 15%) and hematology unit (20; 10%). The most commonly used psychotropic drug was escitalopram (69; 26.74%). 49 (24.5%) patients prescribed concomitantly 2 or more psychotropic drugs during hospitalization. A total of 336 drug interactions were observed (1.68 interactions / patients). Of these, 53 (15.77%) were between two psychotropic drugs. At the same time, there was a significant correlation between the number of psychiatric drugs and total number of drug-drug interactions (r: 0.680, $p < 0.01$). These results suggest that as the psychiatric polypharmacy increases, the potential drug-drug interactions that can cause harm to the patients are increasing. Considering the increased risk of drug-drug interactions, side effects, and drug noncompliance with the increased polypharmacy, it is thought that simplification of drug therapy can be beneficial in pediatric inpatients.

KEYWORDS
Pediatrics; inpatient; psychotropic polypharmacy; drug-drug interaction; clinical pharmacy

References

[1] AACAP. Prescribing psychoactive medication for children and adolescents. Am. Acad. Child Adolesc. Psychiatry. 2001.

[2] Chen H, Patel A, Sherer J, Aparasu R. The definition and prevalence of pediatric psychotropic polypharmacy. Psychiatr Serv. 2011;62:1450–1455.

[3] Zonfrillo MR, Penn JV, Leonard HL. Pediatric psychotropic polypharmacy. Psychiatry (Edgmont). 2005;2:14–19.

[Abstract: 0662] [Psychopharmacology]

Psychotropic-psychotrop and psychotropic-non-psychotropic drug interactions: which one is more important?

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ABSTRACT

Psychiatric consultation requests and psychotropic medication use are rapidly increasing in pediatric practice for children and adolescents with non-psychiatric medical conditions. On the other hand, knowledge about psychopharmacology practice in hospitalized children and adolescents for other medical reasons is quite limited. Psychopharmacological research mostly focuses on specific psychiatric disorders and little is conducted in the field of child and adolescent psychopharmacology in inpatient settings for other medical reasons. However, psychotropic agents are frequently used in inpatient settings and require rational prescribing practices based on research results addressing available evidence, alternatives and risks beyond anecdotal clinical experience. Polypharmacy is defined as the simultaneous use of multiple drugs and due to possible drug interactions, children and adolescents have been shown to have significant risks for drug-related adverse events. Psychotropic drug interactions in children and adolescents may occur in two different ways. Firstly, different clinicians can often prescribe other medical drugs to a child who is currently using a psychotropic agent. Secondly, a psychotropic drug can often be augmented with other psychotropic drugs, because psychiatric disorders are usually chronic, and are difficult to diagnose categorically in children and adolescents, and psychiatric treatment is often symptomatic. There are potentially numerous interactions with psychotropic drugs, but many of these do not have clinically significant results. The lack of established methods to investigate the safety of psychotropic drug interactions in children and adolescents is a cause and result of limited research into this area. Drug interactions are particularly important because they can be predicted and prevented based on previously reported case reports, clinical trials and understanding pharmacological principles. The epidemiology of significant pediatric drug interactions is not fully known because they vary greatly across studies. The aim of our study was to evaluate the frequency and characteristics of psychotropic drug interactions in children and adolescents in an inpatient medical setting. A total of 336 psychotropic drug interactions were observed in 200 patients who received any psychotropic medication (1.68 interaction/patient). Of these interactions, 53 (15.77%) were between two psychotropic drugs. Computerized physician order entry systems are said to control and manage drug interactions and have become common for medication ordering. However, it is difficult to classify drug interactions according to severity levels. Even drug combinations labeled as contraindicated are not necessarily an absolute contraindication. So, clinicians become over saturated with warnings of computer-based drug interactions, which in general may lead to disregard of these warnings. Even when requested by computer-based alerts, clinicians ignore certain interactions and do not complete the recommended follow-up for subsequent negative events. In addition, if clinicians avoid the use of specific drugs due to carefree concerns about drug interactions, the effectiveness of treatment may be compromised. For these reasons, future research should continue to prioritize drug interactions depending on the likelihood of occurrence and the extent of possible harm to patients, and then develop plans for specific high priority drug interactions.

KEYWORDS

Adverse drug events; children; drug interactions; inpatient; psychotropic medication

[Abstract: 0789] [Others]

Mental health and exercise

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ABSTRACT
World Health Organization predicts that depression and anxiety-related mental illnesses will be one of the most important factors threatening the quality of life in 2020. Researches show that physical activity is effective in the treatment or prevention of mental disorders. It is possible to compare the neuropsychobiology pool that affects our behavior to a kind of seesaw, with physical activity at one end causing tremors that affect the entire system. Inactive life style has increased all over the world. It is accepted by the authorities that the physical and mental illnesses and problems caused by the inactive life style are at a worrying level. The social cost is increasing because of people suffering, loss of productivity and health concerns. Physical activity has both preventive and curative effects for many diseases. Physical activity is beneficial to psychological, mental, social and emotional health as well as physical health. Moreover, the direct impact of physical activity on quality of life is indisputable. In addition to enhancing cardiovascular stability, muscle strength and flexibility and body composition; physical activity is also related to positive psychological effects such as decreasing anxiety, increasing self-esteem and respect, distancing from negative thoughts, coping with stress, adapting to stressful events and improving sleep hygiene. Economic benefits of physical activity such as reducing health expenses should not be underestimated as well. Studies show that physical activity is effective in improving symptoms, especially in dementia, depression, anxiety and psychotic disorders. Neurotransmitter release, neurotrophic factors, neurogenesis and alterations in brain blood flow are some of the possible mechanisms that may explain the effects physical activity on mental health. Including physical activity in treatment guidelines is a new approach in the treatment of mental disorders.

KEYWORDS
Mental health; exercise; physical activity

References

[1] Fariz B. Psikiyatrik hastalıklar ve egzersiz Spor Hekimliği Dergisi 2015;50:131-38.
[2] Salzman C, Jeste DV, Meyer RE, Cohen-Mansfield J, Cum-mings J. Elderly patients with dementia-related symptoms of severe agitation and aggression: consen sus statement on treatment options, clinical trials met- hodol-ogy, and policy. J Clin Psychiatry. 2008;69(6):889–898.
[3] Guallar-Castillon P, Bayan-Bravo A, Leon-Munoz LM, Balboa-Castillo T, Lopez- Garcia E, Gutierrez-Fisac JL, et al. The association of major patterns of physical activity, sedentary behavior and sleep with health-related quality of life: a cohort study. Preventive medi-cine 2014;67:248–254.

[Abstract: 0656] [Forensic Psychiatry]

SYMPOSIUM 50

Impulse, crime and punishment: an update for psychiatric patients

Discussant Moderators: Musa Tosun, Mustafa Solmaz

The relationship between crime and impulsivity in psychiatric patients

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ABSTRACT
Impulsivity is a component of the initiation of behavior and predisposition to have rapid and unplanned reactions to internal and external stimuli without regard to the negative consequences of these reactions to individuals and others [1]. Since it is a basic part of disruptive behavior disorders, it may play a role in pathogenesis of neuropsychiatric disorders. It has been found as a significant contributing factor for many psychiatric disorders such as mood disorders, personality disorders (borderline personality disorder, antisocial personality disorder), attention-deficit/hyperactivity disorder (ADHD), conduct disorder, subtance-alcohol use disorder [2]. It is also associated with aggression, self-injury, suicide attempts, domestic violence and risk-taking behaviors [3]. The link between impulsivity and offending behavior is well studied in the criminological, psychological literature. However, the relationship between impulsivity and criminal attempts has been overlooked and it is still uncertain that how impulsivity often contribute to offending behavior can also create further problems for offenders after they come into contact with the criminal justice system. According to Heilbrun violent crimes such as murder, assault, and rape were more likely to be classified as impulsive. Heilbrun also found that those who committed impulsive homicides had a higher recidivism rate (62%) compared with pre-meditated murderers (45%) [4]. Here in this talk, we aim to shed some light on this neglected area, reviewing the main symptoms of impulsivity, and how it affects as individual's experiences and his way through criminal justice system.

KEYWORDS
Impulsivity; psychiatric disorder; criminal; forensic psychiatry

References

[1] Patton JH, Stanford MS, Barratt ES. Factor structure of the Barratt impulsiveness scale. J Clin Psychol. 1995;51 (6):768–774.
[2] Bayard S, Raffard S, Gely-Nargeot MC. Do facets of self-reported impulsivity predict decision-making under ambiguity and risk? Evidence from a community sample. Psychiatry Res. 2011;190(2–3):322–326.
[3] Black RA, Serowik KL, Rosen MI. Associations between impulsivity and high risk sexual behaviors in dually diagnosed outpatients. Am J Drug Alcohol Abuse. 2009;35(5):325–328.
[4] Heilbrun AB. Psychopathy and violent crime. J Consult Clin Psychol 1979; 47: 509–516.

[Abstract: 0756] [Forensic Psychiatry]

SYMPOSIUM 50

Impulse, crime and punishment: an update for psychiatric patients

Discussant Moderators: Musa Tosun, Mustafa Solmaz

Impulse and punishment: a view on the axis of forensic psychiatry

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ABSTRACT

Each legal system has an exception to criminal responsibility when defendants fail to understand wrongfulness of their actions. This exception relies on the assumption that punishment is morally correct only if the defendants could choose between right or wrong. There lies a bigger question of deciding what's right or wrong? Which behaviors should be classified as wrong and be punished and how much punishment is required conscience of society? There is also the "irresistible impulse" cases where the defendants understand wrongfulness of their action but fails to suppress the action created by this impulse. This defense is recognized in some jurisdictions but not others. This defense raises the question of "Is it possible a person can understand wrongfulness of their action but be powerless to stop committing it?" When the literature examined there are cases which this phenomenon is recognized. Especially when patient has experienced head trauma or frontal lobe abnormalities, there is an increased chance of impulsivity and violent behavior and other antisocial behaviors. There is also a relation between impulsivity and lowered prefrontal, heightened subcortical activity. Historically attempts creating a brain correlate of responsibility have failed. Jurisdictions simply used a right or wrong test and in 1962 American Law Institute included control test in its Model Penal Code. Resistance to this test and defense grow and have reached peak when John Hinckley who have shot Reagan in 1981, was found not responsible under MPC standards. After this case control tests were rejected by most of United States jurisdictions, Canada, New Zealand and Hong Kong. The court permitted indeterminate confinement of dangerous individuals as long they have a personality disorder that renders them unable to adequately control their antisocial conduct in Kansas *vs.* Hendricks case. a fifth time child molester who claims if released the only sure way he will stop molesting children is to die.

KEYWORDS

Impulse; punishment; crime; responsibility

[Abstract: 0781] [Others]

SYMPOSIUM 50

Impulse, crime and punishment: an update for psychiatric patients

Discussant Moderators: Musa Tosun, Mustafa Solmaz

Neurobiological substrates of impulsive aggression: a holistic approach to criminal behavior

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ABSTRACT

Aggression and violence are common symptomatic manifestations that the mental health experts are frequently requested to identify, evaluate, and treat in both clinical and forensic settings. Violence is rather a sociological phenomenon representing the interpersonal

KEYWORDS

Aggression; criminal behavior; violence

expression of aggression while the term aggression scientifically indicates a more internal state of living being with its numerous components. Human aggression is any behavior directed toward another individual that is carried out with the proximate (immediate) intent to cause harm. In addition, the perpetrator must believe that the behavior will harm the target and that the target is motivated to avoid the behavior. Aggression has been studied from a variety of perspectives, including the political, legal, sociological, psychological, and neurobiological. Aggression has multiple social and psychological determinants and identifying neurobiological correlates of them may ultimately lead to the development of clinically informatic biomarkers and rational treatment design. The environmental and psychological roots of aggression have been studied for several centuries, yet it is only in the past few decades that we have explored in a systematic fashion possible neurobiological vulnerability to aggression. Much of the knowledge has been learned from the animal studies about the role of neurostructural, neurohumoral and neurotransmitter systems as inhibitors and facilitators of aggression. However, extrapolating the results of these studies to human aggression considered to be difficult. Other species have detailed taxonomies for aggressive behavior, but the lack of a consensus human analogue hinders research progress. A longstanding simple system for codifying aggression has been the impulsive/ instrumental dichotomy. The impulsive/ reactive subtype best resembles as an emotionally charged, uncontrolled type of aggressive display in response to frustration or perceived provocation with a motivation of the more rudimentary purpose of quelling unpleasant affect states. Instrumental/ proactive/ premeditated aggression, on the other hand, characteristically represents planned, controlled, and unemotional acts typically initiated by the offender, rather than provoked, and is explicitly motivated by an expectation of obtaining something of value. Reactive and proactive forms of aggression frequently coexist and are highly intercorrelated. Moreover, these two forms of aggression have been observed in adult populations with personality disorders. A substantial literature has developed linking various brain regions. Neurobiological studies have identified a subset of hypothalamic and limbic brain areas that tend to facilitate aggressive behavior in rodents and primates. In contrast, neural activity in the frontal cortex generally acts to inhibit aggressive behavior. Based on more recent studies, it is acknowledged that aggression is generally associated with impairments in several cognitive systems, typically, the abilities to resist impulses, to modulate behavior, and to understand the consequences of behavior, among other functions. The neural basis for these executive skills resides in the prefrontal cortex. Imaging studies have revealed reduced volume and metabolism of the prefrontal cortex in people with higher aggression. The amygdala, a phylogenetically ancient structure, is the seat of emotion. This limbic site in which emotional responses register and modulated is also involved in the acute threat response and responsible for the emergence of aggression. Contrary to impulsive aggression, the data on neural substrates and clinical characteristics of human proactive aggression is limited. Probably, the primary characteristic that distinguishes an individual who displays impulsive aggressive behavior from one whose aggression is premeditated in nature is the level of behavioral control. Aggressive behaviors in animal models and humans are known to be regulated by serotonin neurotransmission. Behaviour can be modified at several levels, including regulation of serotonin release, reuptake and sensitivity (via serotonin receptors). In humans, the neurotransmitter research on aggression has centered around the involvement of serotonin hypofunction in impulsive aggressive behavior. Serotonin modulates activity in areas of the prefrontal cortex, including the orbitofrontal cortex and anterior cingulate, which are implicated in "top-down" control of limbic responding to stimuli. The neurobiological vulnerability to aggression is also a subject of a debate between the naturalistic and moralistic approach. The main discussion relies upon the argument that despite biological findings point to aggressive behavior as a natural part of the human behavioral repertoire, it is also a part of our evolutionary baggage that we must learn to control with free-will Adaptation to the society requires acquiring resources peacefully by working, and people can rely on the trusted criminal justice system to protect what they have. Novel pharmacological treatments would be able to target specific subtypes of aggression to have improved effectiveness. An appreciation of the contribution of environmental stressors to aggressive phenotypes is necessary for further advancements in the successful management of maladaptive aggression. The lack of well-designed studies of the interaction between neurobiology and an individual's environment in the genesis of human aggression is a major concern. However, neuroimaging methods have a great potential to enhance our knowledge of both neurotransmitter and neurocircuitry function in human aggression. In these symposia, the presenter aimed to stress that professionals who work with criminal individuals are advised to have sufficient understanding of the universality of aggression violence in social mammals, the functions it performs, and the ideas which have been generated to explain these phenomena.

References

[1] Houston RJ, Stanford MS, Villemarette Pittman NR, Conklin SM, Helfritz LE. Neurobiological Correlates and Clinical Implications of Aggressive Subtypes. J Forensic Neuropsychol 2003;3:67–87.

[2] Nelson RJ, Trainor BC. Neural mechanisms of aggression. Nat Rev Neurosci 2007;8:536–546.

[3] Kavoussi R, Armstead P, Coccaro E. The Neurobiology of Impulsive Aggression. Psychiatr Clin North Am 1997;20:395–403.

[4] Coccaro EF, Fanning JR, Phan KL, Lee R. Serotonin and impulsive aggression. CNS Spectr 2015;20:295–302.
[5] Blake P, Grafman J. The neurobiology of aggression. Lancet 2004;364:12–13.
[6] Blair RJR. The Neurobiology of Impulsive Aggression. J Child Adolesc Psychopharmacol 2016;26:4–9.
[7] Anderson CA, Bushman BJ. Human Aggression. Annu Rev Psychol 2002;53:27–51.
[8] Pustilnik AC. Violence on the Brain: A Critique of Neuroscience in Criminal Law. Wake Forest Law Rev 2013;44:183–237.
[9] Gunn J, Taylor PJ. Forensic psychiatry: clinical, legal and ethical issues. 2nd ed. New York: CRC Press; 2014.

[Abstract: 0715] [Sleep disorders]

Normal sleep in children and differences from adults

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ABSTRACT

Sleep is a self-repeating and easily reversible dynamic process that presents with major alterations with respect to its qualitative and quantitative qualities in healthy individuals. This recurrent periodic condition is defined as "circadian rhythm". From infancy through adulthood, sleep and circadian rhythm evolve as a reflection of central nervous system (CNS) maturation. Alterations regarding sleep structure of an individual have long been accepted to emerge during the last trimester of pregnancy. Even though major portion of sleep is made up of undifferentiated sleep at 32nd gestational week, it is possible to encounter REM and non-REM (n-REM) sleep phases. Between 32nd and 40th gestational weeks, amount of undifferentiated sleep decreases while n-REM sleep increases. Until the last trimester, no change is observed in REM sleep. In addition to these changes in sleep phases observed during the final trimester, circadian rhythms also become more evident. Even though sleep-related alterations start during prenatal period, most dramatic changes do not emerge until the first year of life. During this time, major changes occur in total sleep time, sleep structure and 24- hour sleep- wake cycle. Newborns typically sleep for 16–17 h a day. In the first 3-6 months of life, no significant changes is observed, regarding total duration of sleep per day. Nevertheless, significant alterations with respect to the distribution of sleep within a day of 24 h emerge and major portion of 24-hour sleep shifts to night hours. Total amount of unfragmented sleep the infant has increases as well from 3–4 hours at 1 month to 6 h around 6th month of life. When newborns are evaluated from the perspective of sleep structure, one major difference one might observe is that contrary to adults, babies enter their sleep cycle through REM phase. In addition to this, newborns exhibit a sleep pattern that contains significantly higher rates of REM compared to that observed in adults, nearly where 50% of total sleep duration would involve REM phase. Total amount of REM sleep gradually decreases as life progresses, reduced to 20% of total sleep time and stays pretty much in those limits, for the rest of life. In small children, REM and non-REM (n-REM) sleep repeat all night, within approximately 50- minute cycles (ultradian cycle). As a consequence of normal ultradian cycle, children tend to wake up 4–6 times at night during infancy. As they get older, total length of an ultradian cycle increases, reaching almost 90 min, in adults. Following sleep time and structure, another significant alteration regarding sleep is within the sleep-wake cycle. In the first few months of life, light-dark/daytime-nighttime cycle is not present. Sleep is evenly distributed between nighttime and daytime. In general, phases that would involve 3–4 h of sleep and 1–2 h of being awake are observed. Most infants tend to start sleeping more and longer during nighttime, around 3rd month of their lives. With this presentation, we have aimed to describe normal sleep processes in children, as well as the differences when compared to sleep characteristics of adults.

KEYWORDS

Adult; children; circadian rhythm; differences; normal sleep

References

[1] Iglowstein I, Jenni OG, Molinari L, et al. Sleep duration from infancy to adolescence: reference values and generational trends. Pediatrics. 2003;111:302–307.
[2] Mirmiran M, Maas YG, Ariagno RL. Development of fetal and neonatal sleep and circadian rhythms. Sleep Med Rev. 2003;7:321–334.
[3] Mirmiran M, Lunshof S. Perinatal development of human circadian rhythms. Prog Brain Res. 1996;111:217–226.

[Abstract: 0768] [Sleep disorders]

Current approaches to treatment of sleep disorders in children

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ABSTRACT

Sleep disorders occur frequently in children. It is estimated that various sleep difficulties (bedtime resistance, delayed sleep onset, frequent waking after sleep onset, lack of sleep consolidation, early waking, daytime sleepiness) may affect 25–62% of children depending on development phase. Those can present as a primary problem or coexist with other disorders. The most common types of sleep problems in children are difficulties falling asleep and difficulties maintaining sleep. Inadequate sleep in children may have a negative impact on their cognitive development, mood regulation, attention, behavior, and quality of life. Not only are children affected, but also parents and caregivers are affected in their wellbeing and daily working activities because of sleep deprivation. Therefore, there is an urgent need to identify and treat sleep disorders in children. Current management strategies for sleep disorders start with educating parents about sleep hygiene and adequate sleep routines. Other behavioral therapies, such as cognitive behavioral therapy, have also shown to improve sleep quality in young children. When sleep hygiene and behavioral interventions fail to have an effect, pharmacologic treatment with, for example, antihistaminic agents, alpha-agonists, or benzodiazepines may be considered. It should be emphasized that these drugs are often used off-label, as there exist no approved drugs for treating sleep disorders in children. Furthermore, these drugs should be prescribed with caution for children, as they are associated with a risk of side effects such as daytime sedation, dizziness, change in behavior, memory deficits, and paradoxical hyperactivity. Since pharmacologic treatment strategies for insomnia are limited, parents may seek other, natural products to meet the medical needs of their children.

KEYWORDS

Sleep disorders; children; attention

[Abstract: 0741] [Sleep disorders]

Approach to sleep disorders in adolescent period

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ABSTRACT

Adolescence is a transitional period and a period of significant changes in sleep and wake rhythm. During this period, the night sleep time increases and start time to sleep shifts to late hours. Despite the increased need for sleep, biological, psychiatric and social factors specific to this age period lead to shortening of sleep time and many problems related to it. There is an increase in sleep-related symptoms such as sleepiness, frequent awakening, and daytime sleepiness in adolescents. The increase in social activity during the adolescence, the intensification of academic functions, the beginning of the lessons in the early morning hours and the shift of the beginning to sleep to the late hours cause less sleep. Adolescents who sleep 9 h or more in the night and who enter school later in the morning have been shown to have significantly higher school achievement than adolescents with less sleep, independently of age, gender, stress and eating habits. It was shown that depressive symptoms increased compared to those who sleep less than six hours a day, and anxiety scores increased in late sleepers. Changes sleeping start hours in adolescents, the elimination of the compulsion of parents to sleep time, using internet, television and so on. , the use of alcohol, smoking and caffeine, starting competition in school and social life, conflicts with friends and parents are the main reasons leading to difficulty in initiating and sustaining sleep. The main sleeping problems in adolescence are classified under two main headings. Dyssomnia on; Insomnia disorder, hypersomnolence disorder, narcolepsy, respiratory-related sleep disorder, circadian rhythm sleep-wake disorders. Parasomnias; Non-Rem Sleep Awakening Disorders (sleep terror, sleepwalking), nightmare disorder, Rem Sleep Behavior Disorder, Restless Leg Syndrome, substance / drug-induced sleep disorder. In adolescents, the frequency of problems related to sleep also increases in psychiatric disorders. Seventy percent of children diagnosed with ADHD have different levels of sleep problems. In children diagnosed with ADHD, there was a significant increase in sleep-related problems compared to healthy peers such as bedtime resistance, difficulty in initiating sleep, night waking, and

KEYWORDS

Sleep disorders; adolescent period; DSM-V sleep and wake disorders; Dyssomnia; Parasomnias

difficulty in waking up in the morning, breathing problems, and drowsiness during the day. Sleep problems are quite common in children diagnosed with ASD and rates between 40–80% are reported. In studies, based on parental reports of children diagnosed with ASD, the most common reported sleep problems are falling asleep, maintaining sleep, and consequently reducing the amount of sleep. In a field study conducted in the adolescent age group, the presence of insomnia in the initial evaluation revealed that the risk of major depressive disorder (MDD) increased 2-3 fold. In another study, it was reported that 83% of the children with anxiety disorder had intermittent problems and 46% had sustained at least one sleep problem. In this talk, sleep disorders and treatment approaches in adolescents will be discussed based on DSM-V sleep and wake disorders classification.

[Abstract: 757] [Sleep disorders]

Current approach to treatment of sleep disorders in infants and toddlers

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ABSTRACT

In this speech; we aim to describe the developmental changes in sleep across three years of life, most common sleep disturbances seen during these ages, sleep disturbances in the context of other biological and psychological disorders and common interventions used to treat infant and toddler sleep problems. Typical sleep for babies is characterized by different patterns. Regulation and timing of sleep change across the first three years of life. Active sleep has a potential role in brain development. Most infants sleep between 12 and 14 h per day through the age of 2 years. Sleeping through the night is a misnomer for most infants and toddlers across the first 3 years of life. Sleep disturbances in infants and toddlers generally occur in the context of parent- child relationships. However; in this age group definition of sleep disorders is clinically complicated. How a parent defines " normal" sleep is quite variable. The most common sleep complaint in the infant- toddler parent population is night waking. The most common etiology for problematic night waking is termed " behavioral insomnia of childhood, sleep- onset association of Sleep Disorders-2 (ICSD-2). The second most common sleep disorder found in the infant/toddler populations is termed behavioral insomnia of childhood, limit setting subtype in the International Classification of Sleep Disorders-2 (ICSD-2). This condition is defined as delaying or resisting bed time. It is generally seen children 2 aged or older. The studies about this are found that significant correlation between bed time resistance and day time resistance to parental behaviors.

Sleep problems in infants and toddlers can have a significant impact on some families, but not for others. Also familial factors such as maternal depression, parental fatigue, general disruptions to family life, poor maternal and physical health and less parental well-being correlate to sleep problems in infants and toddlers. The high prevalence rates for sleep problems found in children with neurodevelopmental disorders, ranging from 13 to 85%. It has been estimated that significant sleep problems occur in 30–80% of children with severe mental retardation and 50–70 % of children with autism spectrum disorders. The mainstay of treatment for bedtime struggles and night wakings in infant and toddlers should be consisting behavioral interventions. Studies about pharmacological interventions for sleep problems in this age group are limited. However, a few studies have examined the effect of pharmacological treatment, usually combination of with behavioral treatments. Sedating antihistamines are commonly used for sleep problems in clinical practice. Melatonin can be used for sleep latency in children with Attention-deficit/ hyperactivity disorder (ADHD) and with other neurodevelopmental disorders. Alternative treatments such as infant massage may be safe and simple when used adjunctively in the treatment of infant sleep problems. Recent studies have shown that massage in the newborn period may have a long- term effect on melatonin synthesis and the development of normal circadian rhythms.

[Abstract: 0704] [Psychosomatic medicine - Liaison psychiatry]

What is burning mouth syndrome? diagnosis and treatment approaches

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ABSTRACT
Burning mouth syndrome is a chronic, idiopathic condition characterized by pain and burning sensation in the oral mucosa, which is described as unbearable in the oral cavity, especially in the tongue, without a demonstrable pathology. This may be a symptom accompanied by burning and painful lesions, as well as in individuals with healthy oral tissues. The typical age is postmenopausal women between the ages of 55–60. It is thought to affect% 1–5 of the general population. It is reported that it is rarely seen in males and in the pre-30 age period and never seen in children. Etiopathogenesis has not been fully elucidated. Local (candidiasis, dentures, tooth extraction, local irritation, chronic traumatic irritations) and systemic (hormonal disorders, immunological disorders, hematological diseases, nutritional deficiencies, vitamin deficiencies, diabetes, drugs, infections) are believed to be effect of many factors. Although the clinical findings variability, the findings are frequently observed; pain in the mouth, burning sensation, dry mouth and the presence of foreign bodies in the mouth feeling. The pain is usually bilateral and localized to the two thirds of the tongue. In addition, changes in taste sensation, dry mouth, dizziness and nausea frequently accompany the table. Pain can increase with drinking, and sometimes it can affect other parts of the body, such as the hand-foot of the body. In more than half of the cases, the pain starts spontaneously, increases during the day and reaches the maximal level in the evening hours. Still , one third of the cases stated that the symptoms developed due to a dental procedure, disease, drug use, familial problem or a stressful condition. They describe the foreign body feeling as sand, hair, paste, yarn or rough matter. Diagnosis is based on the patient's subjective complaints, systemic (Sjogren's syndrome, diabetes, fungal infections, iron, zinc, vitamin B deficiency) or local (lichen planus, fissured tongue, erythema, erosion skin lesions) secondary to exclusion of secondary causes It is placed as. Detailed anamnesis should be taken during clinical evaluation. The history of the burning sensation, duration, severity, when it started, how it travels during the day, exacerbating or relaxing factors should be questioned. Oral mucosal examination should be performed carefully and required laboratory tests should be requested. Treatment of burning mouth syndrome is a challenging process for the physician and the patient. The treatment should be person-specific. The patient should be told that the disease may have idiopathic reasons, that the treatment can last a long time, but the disease has nothing to do with cancer. It is very important for the success of the treatment that the patient trusts the physician and thinks that the physician understands it. The biggest obstacle to treatment is the lack of a sufficient number of studies and a consensus-based treatment plan has not yet been established. Possible treatment options include systemic pharmacological agents such as lidocaine gel, capsaicin mouthwash, benzidamine mouthwash, antidepressants, antipsychotics, antiepileptics, anxiolytics and alpha lipoic acid. The treatment options of the pharmacological treatment as well as the side effects of psychodynamic behavioral psychotherapy and psychodynamic behavioral therapy. As a result, burning mouth syndrome seems to be an area open to further study in terms of multifactorial nature, diagnostic criteria, and treatment options.

KEYWORDS
Burning mouth myndrome; diagnosis; treatment; oral health; mental disorder

References

[1] Toptaş O, Akkaş İ, Özan F. Yanan Ağız Sendromu. Acta Odontol Turc 2014;31(2):102–105.
[2] Erdoğan B, Zorlu MY. Oral Hastalıklarda Güncel Tedavi: Yanan Ağız Sendromu, Türk Derm 2012; 46 Özel Sayı 2:119–122.
[3] Ritchie A, Kramer JM. Recent Advances in the Etiology and Treatment of Burning Mouth Syndrome J Dent Res 2018;97(11):1193–1199.

[Abstract: 0624] [Others]

What is psychogenic halitosis? diagnosis and treatment approaches

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ABSTRACT
Halitosis is used to describe oral malodor and brings the need for professional psychiatric and dental care. Of note, "oral malodor" is accepted to describe halitosis, not bad breath. Halitosis is classified under 5 types. According to halitosis classification, halitosis is classified / described in Type I to V, and Type 0 is physiological. Type V halitosis includes psychogenic and neurogenic causes. Neurogenic type is the result of neurological conditions. The data of the American Association of Dentists reveals that this problem is seen in 50% of the adult population and 25% is a chronic problem. Today's commercial advertising pressures have changed society's

KEYWORDS
Anxiety; depression; oral malodor; olfactory reference syndrome; psychogenic halitosis

orientation towards problems such as halitosis, resulting in a greater impact on negative psychosocial changes. Women are more worried about their bad breath than men, and it is emphasized that the role of mouth is very important in interpersonal relations. In many studies based on personal reports, it was emphasized that halitosis is not often perceived by the individuals themselves. A few studies have emphasized that there may be a relationship between halitosis and mood (e.g. anxiety, depressive disorder). However, clinical observations have also been shown that the concentration of volatile sulfur compounds (VSC) can be increased in especially anxiety states. it has been shown that people have a very important and beneficial effect on appearance, comfort, sleep, mood and social life-relationship in their social environment. In general, self-awareness is defined as the person's perception of himself / herself, and the tendency to evaluate against the encounter with stressful life events (such as dental stimulation). Therefore, the perception of why intra-oral applications stimulate more anxiety is strengthened (extreme sadness, restlessness, perception of damage, experiencing acute symptoms of anxiety), leading to avoiding dental treatment. Halitosis means that perception can be caused or changed by personal differences. The most common psychiatric problem associated with halitosis is anxiety. Depressive states may play a role in this issue. In addition, it is another finding that thyroid problems, which have been reported to be associated with the problem of halitosis and their relationship with anxiety, especially in women. Type V, psychogenic type halitosis has not yet been fully established in psychiatric classifications. The patient group of the olfactory reference syndrome (ORS) consists of patients who do not refer to psychiatry, but rather visit other medical branches and dentists (doctor-shopping). The psychogenic type of halitosis increases with the awareness in modern society. Psychogenic type halitosis classification: it is more complex and difficult to treat because of the causes involved (since it has a very close relationship with the mental processes, including current or past or both). The olfactory reference syndrome, which is not yet fully understood, has continued to confuse our minds at the level of diagnosis or comorbidity.

References

[1] Özen ME, Aydin M. Subjective halitosis: definition and classification. J N J Dent Assoc 2015;86(4):20–24.

[2] Aydin M, Derici MC, Yeler DY, et al. Criteria to distinguish subjective halitosis. Compend Contin Educ Dent 2017;38(10):e5–e8.

[3] Aydın M. Ağız kokusunu anlamak ve sınıflamak. Turkiye Klinikleri J Oral Maxillofac Surg-Special Topics 2016;2(1):5–16.

[4] Zaitsu T, Ueno M, Shinada K, et al. Social anxiety disorder in genuine halitosis patients. Health Qual Life Outcomes 2011;9:94.

[5] Suzuki N, Yoneda M, Naito T, et al. Relationship between halitosis and psychologic status. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 2008;106(4):542–547.

[Abstract: 0424] [Motor disorders]

What is bruxism? diagnosis and treatment approaches

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ABSTRACT

Bruxism is a clinical condition characterized by tooth tightening and / or squeaking and is one of the most common reasons for referral to psychiatry and dentists. However, this clinical condition is often not detected and often leading to pain in the jaw and tooth damage. In this article, Searching will be done in the databases using the KEYWORD "bruxism". The current information for the diagnosis and treatment of bruxism will be compiled in light of the articles found. In the literature, it is reported that bruxism is between 8% and 14%. Although this is so common, the main cause of bruxism is unknown. In the other hand, recent evidence suggests that the imbalance between dopaminergic and serotoninergic system may be associated with the pathophysiology of bruxism. These serotonergic and dopaminergic drugs are frequently used in psychiatry practice. Therefore, these drugs can reduce the symptoms of bruxism in some patients, while others increase it. For the treatment of bruxism, it must be diagnosed first. Because most patients may not notice bruxism. Treatments such as Mouth guard, medications, botulinum toxins, stress reduction, counseling, lifestyle changes, and hypnotherapy can be used to treat bruxism. Although there is a frequent complaint in the psychiatric outpatient clinics of bruxism, there is a need for detailed research for diagnosis and treatment.

KEYWORDS

Bruxism; diagnosis; treatment

[Abstract: 173] [Anxiety disorders]

What is misophonia? diagnosis and treatment approaches

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ABSTRACT

Misophonia is defined as hate of sound. It is characterized with intense emotional reactions like anger, anxiety, or disgust to and avoidance behavior from special sounds such as mouth sounds while eating or chewing gum, nasal sounds like breathing, smelling or blowing or some other sounds done with fingers like playing with a pen, writing or drumming on the table, especially, made by other people. Although it has been known for years, misophonia which is described as hate of sound, was first described by Jastreboff in 2001 but, it has not been well recognized in the field of mental health up to now [1]. Misophonia has never been present in any of the psychiatric classification systems. Some authors suggest that misophonia should be regarded as a new mental disorder. There is limited information about misophonia. Only few cases of misophonia have been reported. The causes of misophonia, risk factors, the relationship between mental disorders and treatment of misophonia are not fully known. Relationships and co-morbidity between misophonia and other mental disorders have not been fully elucidated yet. A study investigating the relationship between misophonia and OCD, anxiety and depressive symptoms, reported strong associations and also emphasized the relationship between misophonia and anger outbursts. Misophonia symptoms may cause nervousness, marked anxiety, disgust, anger, hatred, physical violence, loss of control, avoidance behavior, or even suicidal thoughts. The knowledge on the treatment of misophonia is also limited. It has been pointed out that coping strategies with CBT, exposure and response prevention, cognitive restructuring, awareness and acceptance based therapy methods may have positive results in the treatment [2–4]. The knowledge about psychopharmacological treatment is not available. In our case study, selective serotonin reuptake inhibitors (SSRIs) were preferred in the treatment of the cases. Another remarkable issue is the doses of antidepressant were the same as doses used in OCD treatment [5]. Misophonia is a recently described, poorly understood and neglected condition. Future studies will focus on investigating the epidemiology, phenomenology, neurophysiology, and treatment of the misophonia. In this presentation, I describe what is known about the symptoms, epidemiology, and assessment of misophonia, including a discussion of its diagnostic boundaries and neuropsychological profile.

KEYWORDS

Diagnosis; mental disorders; misophonia

References

[1] Jastreboff MM, Jastreboff PJ. Components of decreased sound tolerance: hyperacusis, misophonia, phonophobia. ITHS News Lett. 2001; 2:5–7.

[2] Cavanna AE, Seri S. Misophonia: current perspectives. Neuropsychiatr Dis Treat. 2015; 11:2117–2123.

[3] Dozier TH. Etiology, composition, development and maintenance of misophonia: a conditioned aversive reflex disorder. Psychological Thought. 2015; 8:114–129.

[4] Edelstein M, Brang D, Rouw R, Ramachandran VS. Misophonia: physiological investigations and case descriptions. Front Hum Neurosci. 2013; 7:296.

[5] Cavanna AE. What is misophonia and how can we treat it? Expert Rev Neurother. 2014;14:357–359.

[Abstract: 0793] [Others]

Use of Turkish scales to evaluate subjective recovery, social functioning, and self-stigmatization in patients with schizophrenia and their families

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ABSTRACT

The assessment of the social functioning of the patients with schizophrenia, the evaluation of the patients' perceptions of recovery and self-stigmatization are the areas to be considered in the treatment process. In the same way, families should also be concerned about for their self-stigmatization. The scales used in these areas are generally the ones that have been validated in western countries. At the translation and adaptation of these scales, there may be difficulties in understanding for the patients and their relatives and this might result in incorrect evaluation. In

KEYWORDS

Schizophrenia; social functioning; subjective recovery; self-stigmatization; scales

fact, the evaluation of the patients is most likely possible with the use of scales developed from their own culture. In this course, the characteristics and usage of the scales developed in accordance with Turkish culture will be explained.
Scale 1: *Social Functioning Assessment Scale (SFAS)*
The scale consists of 4 factors (1-Interpersonal relations and recreation, 2-Self-care, 3-Independent living, and 4-Occupation) with 19-item. The Cronbach's alpha coefficient is 0.84 for the SFAS total score. Each item is assessed with 3-point Likert-type ratings. A high score shows higher functionality.
Scale 2: *Subjective Recovery Assessment Scale (SubRAS)*
The scale consists of one factor with 17-item. The Cronbach's alpha coefficient is 0.98 for the SubRAS. Each item is assessed with 5-point Likert-type ratings. A high score shows higher recovery feeling.
Scale 3: *Self-Stigma Inventory for Families (SSI-F)*
The scale consists of 3 factors (1-Social withdrawal, 2-Perceived devaluation, and 3-Concealment of the illness) with 14-item. The Cronbach's alpha coefficient is 0.88 for the SSI-F total score. Each item is assessed with 5-point Likert-type ratings. A high score shows higher self-stigmatization for the family members.
Scale 4: *Self-Stigma Inventory for Patients with Schizophrenia (SSI-P)*
The scale consists of 3 factors (1-Internalized stereotypes and social withdrawal, 2-Perceived devaluation, and 3-Concealment of the illness) with 17-item. The Cronbach's alpha coefficient is 0.93 for the SSI-P total score. Each item is assessed with 5-point Likert-type ratings. High score shows higher self-stigmatization for the patients.

References

[1] Yıldız M, Kiras F, İncedere A, Esen D, Gürcan MB, Abut B, İpçi K, Tural Ü. Development of Social Functioning Assessment Scale (SFAS) for People with Schizophrenia: Validity and Reliability Study. Anadolu Psikiyatri Derg, 2018; 19(Suppl 2):29–38.
[2] Yildiz M, Erim R, Soygur H, Tural U, Kiras F, Gules E. Development and validation of the Subjective Recovery Assessment Scale for patients with schizophrenia. Psychiatry and Clinical Psychopharmacology 2018; 28 (2):163–169.
[3] Yildiz M, İncedere A, Kiras F, Abut FB, Kircali A, İpci K. Development of Self-Stigma Inventory for Families of the patients with schizophrenia (SSI-F): validity and reliability study. Psychiatry and Clinical Psychopharmacology 2018.
[4] Yildiz M, Kiras M, Incedere A, Abut FB. Development of self-stigma inventory for patients with schizophrenia (SSI-P): reliability and validity study. Psychiatry and Clinical Psychopharmacology, 2018.

[Abstract: 0725] [Dependencies]

Neurobiology of nicotineuse disorder

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ABSTRACT
Nicotine addiction is a disease where biological mechanisms, along with psychological, behavioral and social elements having active roles as well. In order to have a clear understanding of the treatment, one must know the mechanisms which lie underneath. When the reasons for its occurrences are examined, neurobiological and behavioral setups seem to play important roles. Nicotine is a very potent addictive agent, and nicotine addiction is the most common, most deadly and costly among addictions. There are many neurobiological mechanisms under the nicotine addiction. During the animal trials, the main focus has been on glutamate, GABA, acetylcholine and dopamine neurotransmitters; brain's ventral tegmental area(VTA), amygdala and prefrontal cortex area. This system, which is interrelated in several ways, can sometimes hinder our progress on understanding addiction mechanisms. The mutual pathway of the brain's reward mechanism (reinforcement mechanism) is the mesolimbic(ML) dopamine pathway. Nicotine shows activity directly through the nicotinic cholinergic receptors of the reward cycle. The nicotine in the ventral tegmental area, is the relation to addiction. Nicotine causes a dopamine release from the nucleus accumbens by connecting to the postsynaptic alpha4beta2 nicotinic receptors at VTA's dopamine neurons. In addition, by connecting to the glutamate neurons' presynaptic nicotinic alpha7 receptors, yet another wave of dopamine is released from the nucleus accumbens. Meanwhile, the postsynaptic beta2 receptors which are located on the VTA's GABA interneuron, become desensitized by the nicotine and therefore GABA's overall neuronal transmission decreases. This decrease, in turn, increases the dopamine release from the nucleus accumbens by reducing the inhibition of the ML dopamine neurons. The essence of reinforcing effect relates to conditioning processes. In psychology there are two

KEYWORDS
Nicotine addiction; neurobiology; reward function; classical conditioning; operant conditioning

types of conditioning; classical and operant. Classical conditioning is a model put forward by Pavlov. The sound of a bell, which initially means nothing to a dog, when combined as a stimulant with meat to get the dog to salivate as a response, is later observed to respond and salivate to the sound of the bell alone. Operant conditioning is the teaching of the behavior which leads to reward or avoids punishment. The stimulants which make a positive impact on the organism and therefore increase the occurrence of a specific behavior are called reinforcements. As such, substances with reinforcing properties are observed to cause an increase in their usages initially which, at a later time, and with the help of the classical conditioning process, becomes a learned behavior. Cigarettes and nicotine are identified as both positive and negative reinforcements. Because the act of smoking reinforces both distress and pleasure. Early nicotine withdrawal is associated with decreased expression of presynaptic inhibitor metabotropic glutamate 2/3 receptors and increased expression of postsynaptic glutamate receptors in the limbic and frontal area. Prolonged deprivation might be related to the increased glutamate response to nicotine related stimulants. Pharmacological interventions which reduce glutamate transmission while increasing GABA transmission, have resulted in diminishing the effects of nicotine through reward mechanism and alleviating the behavior of nicotine cravings.

References

[1] Markou, A. Neurobiology of nicotine dependence. Philosophical Transactions of the Royal Society B: Biological Sciences 2008;363(1507):3159–3168.
[2] Stahl SM. Stahl'in Temel Psikofarmakolojisi. Tunç Alkın (Çeviri Ed.) 1.Baskı, İstanbul: İstanbul Tip Kitabevi, 2015; 547-551.
[3] Koob GF, Le Moal M. Drug addiction, dysregulation of reward, and allostasis. Neuropsychopharmacology, 2001;24(2):97.
[4] Türk Toraks Derneği Tütün Kontrolü Çalışma Grubu. Sigara Bırakma Tanı ve Tedavi Uzlaşı Raporu. Miki Matbaacılık, Ankara 2014.

[Abstract: 0674] [Dependencies]

Pharmacotherapy in nicotine (cigarette) use disorder

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ABSTRACT

Tobacco use remains a significant threat to public health due to its strong association with morbidity and mortality. People with severe mental illness die 25 years younger than the general population, and most of this premature mortality is due to tobacco use. Smoking also complicates treatment of some mental disorders by reducing blood levels of some neuroleptics. While there has been a steady decline in the percentage of adults who smoke from 31.2 % in 2008–27.1% in 2012 in Turkey, the rate of successful smoking cessation or quit attempts remains low. Currently, pharmacological approaches to smoking cessation follow three lines: (a) first line nicotine replacement therapy (NRT), bupropion and varenicline, (b) second-line treatment with nortriptyline and clonidine and (c) immunization against nicotine. NRT is a classical pharmacotherapy approach for smoking cessation that aims to replace the inhaled or ingested nicotine from cigarettes pharmacologically, thereby reducing the withdrawal symptoms that occur with smoking cessation. Bupropion, as well as its active metabolite, (2S,3S)-hydroxy bupropion, is a noncompetitive antagonist on nAChRs, particularly those containing α4β2 and α3β2 subunits. It is also a dopaminergic and noradrenergic reuptake inhibitor. Varenicline is a selective α4β2-containing nAChR partial agonist and a full agonist at the homomeric α7-containing nAChR and also exerts some antagonistic properties on these receptors with nicotine co-administration. Sustained release bupropion was approved by the Food and Drug Administration (FDA) in 1997 and varenicline in 2006. Nortriptyline is a selective NE reuptake inhibitör, that has been tested as a therapy in smoking cessation and shown to have adverse events, including depression and increase of suicidal behavior. Clonidine is a centrally-acting α-adrenergic receptor agonist with higher affinity for α2 than α1 receptors. It has also been tested as therapy in smoking cessation and showed increased rate of adverse effects, such as dry mouth and suppression of the central nervous system, leading to depression. Other potential treatments including treatment with rimonabant (a cannabinoid-1 receptor antagonist) and nicotine vaccines are currently investigated, however, not proved for clinical use. Another treatment option of e-cigarette use was found to be associated with unsuccessful rather than successful smoking cessation.

Some studies have shown that varenicline is associated with higher rate of continuous abstinence from smoking, causes fewer withdrawal symptoms, and has the highest rate of success compared to NRTs or bupropion [1]. Further, it is suggested that varenicline is safe

KEYWORDS

Bupropion; nicotine; pharmacotherapy; smoking cessation; varenicline

and efficacious for increasing smoking abstinence rates in smokers with alcohol use disorders and also may decrease alcohol consumption in this population of smokers [2]. Also a recent meta-analysis suggested that bupropion is effective and tolerable for smoking cessation in adults with serious mental illnesses. Smokers treated with bupropion are less irritable, better able to concentrate, have less desire to smoke and experience less negative affect. In conclusion, bupropion and varenicline have been reported to be more effective than placebo and NRTs when used as either single agents or with NRTs in combination [1,3].

References

[1] Anthenelli RM, Benowitz NL, West R, St Aubin L, McRae T, Lawrence D, et al. Neuropsychiatric safety and efficacy of varenicline, bupropion, and nicotine patch in smokers with and without psychiatric disorders (EAGLES): A double-blind, randomised, placebo-controlled clinical trial. Lancet. 2016;387(10037):2507–2520.

[2] Hurt RT, Ebbert JO, Croghan IT, Schroeder DR, Hurt RD, Hays JT. Varenicline for tobacco-dependence treatment in alcohol-dependent smokers: A randomized controlled trial. Drug Alcohol Depend [Internet]. 2018;184(October 2017):12–17.

[3] Cahill K, Stevens S, Perera R, Lancaster T. Pharmacological interventions for smoking cessation: An overview and network meta-analysis. Vol. 2013, Cochrane Database of Systematic Reviews. 2013.

[Abstract: 0721] [Dependencies]

Cognitive behavioral therapy in nicotine (tobacco) use disorder

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ABSTRACT

Tobacco is the most commonly used and addictive substance in the world. The World Health Organization estimates that 1.2 billion people worldwide use tobacco products. Turkey is among the top ten tobacco consuming countries and is estimated to accommodate 17 million smokers. In our country, about 100,000 people die due to smoking-related diseases. These numbers suggest that nicotine addiction is a serious public health issue. All addictions should be treated with biopsychosocial approaches. Pharmacologic agents alone are insufficient in the treatment of nicotine addiction, among the foremost preventable causes of mortality, and psychological support and treatment is necessary. Psychological treatment includes cognitive behavioral psychotherapy. Cognitive behavioral therapy aims to treat nicotine dependence by establishing a new manner of thinking and behaving using cognitive techniques and behavioral interventions. Motivational interview techniques also hold an important place in psychological treatment. Studies have shown that 70% of smokers have a desire to quit. Despite these high rates of desire to quit, the rates of quitting smoking are low. Additionally, further studies have shown that 70-80% of people who managed to quit smoking began smoking again within 6–12 months. Cigarette-related cognitions, triggers and sustainers, and the lack of interventions on what cigarettes represent to smokers are thought to contribute to the high rates of recurrence. The individual's self-assessment of these fields should be questioned, including their thoughts on what smoking or quitting contributes to them, cognitions on what they expect to gain should be exposed, and cognitive intervention should be implement against erroneous assessments. For example, quitting will be more difficult for individuals who say that smoking gives them relief and reduces distress. As long as this individual's positive thoughts on smoking continue, smoking addiction will persist. At this stage, cognitive intervention aims to generate functional and rational thoughts instead of positive cognitions attributed to smoking, procuring evaluation of overlooked negative, and establishing long-term losses instead of short-term gains. Behavioral intervention aims to generate lifestyle changes, establish individual behavior and attitude associated with situations and environment, and change them. Therefore, the beliefs and behaviors responsible for the problem interrelated to each other. Interventions in these fields reduce both quitting and recurrence rates. Since personality traits and personal differences effect the experiences of individuals treatments should be planned individually. However in many occasions' treatment may be applied as both individual or group psychotherapy. The cognitive model views nicotine addiction as a learned process with increased use based on various positive and negative reinforcers, and plans interventions on these grounds. Therefore, it assumes that this behavior could be developed in terms of non-smoking. Once addiction has developed, the cycle will be difficult to break, therefore cognitive and behavioral interventions towards not starting should not be ignored. The prevention of smoking is the most important step in preventing long-term dependence, especially in youth and adolescence, and at this point cognitive behavioral therapy is necessary.

KEYWORDS

Tobacco; dependence; treatment; cigarette quitting therapies; cognitive behavioral therapy

References

[1] Fiore MC, Bailey WC, Cohen SJ, Dorfman SF, Goldstein MG, Gritz ER, Mecklenburg RE. Treating tobacco use and dependence: clinical practice guideline. Rockville (MD): US Department of Health and Human Services; 2000, 00-0032.

[2] WHO report on the global epidemic, 2008: The MPOWER package. Geneva, World Health Organization. 2008.

[3] Ryan F. Cognitive therapy for addiction: Motivation and change. John Wiley & Sons; 2012.

[Abstract: 0736] [Dependencies]

Hypnosis for nicotine cessation

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ABSTRACT

Hypnosis has been defined as 'a state of consciousness involving focused attention and reduced peripheral awareness characterized by an enhanced capacity for response to suggestion'. Although hypnosis has been known about for centuries, there is still widespread misunderstanding about what it actually is, and it has been viewed with distrust and skepticism by many health professionals. However, recent advances in neuroscience have enabled us to understand the hypnotic state, and evidence is building for the use of hypnosis as a useful tool to help patients and health professionals manage a variety of conditions, especially smoking. Hypnosis continues to be viewed with great interest among smokers considering treatment options for smoking cessation and hypnosis can enhance treatment gains of cognitive behavioral therapies. The success rates reported for hypnosis for smoking cessation typically ranging between 25–35%. To achieve higher rates, hypnosis interventions need to be more individualized (based on patients' needs and preferences) and intensive (minimum four sessions lasting more than 10 min each).

KEYWORDS

Hypnosis; nicotine dependence; smoking cessation; tobacco use; complementary therapies

[Abstract: 0397] [Psychosomatic medicine-Liaison psychiatry]

Bidirectional communication between the brain and gut

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ABSTRACT

It has been reported that the gut microbiota have approximately 40,000 bacterial species and 10^{14} microorganisms. The ~100 trillion microorganisms that reside in the digestive tract, and the wide assortment of metabolites they produce, are critical for maintaining health. The presence of microbiota along the gastrointestinal (GI) tract is also critical for the maintenance of the intestinal barrier. By altering expression levels of tight junction proteins along the epithelial wall, and thus the level of bacterial infiltration in the mucosal layer, the gut microbiota can adjust finely the level of intestinal permeability. The regulation of the intestinal barrier by gut microbiota determines their role as mediators of the intestinal and peripheral immune response. It is known that psychological status and physiological stimuli play an important role in the development of GI disorders such as inflammatory bowel disease (IBD) and irritable bowel syndrome (IBS). In response to stress, the inflammatory cytokines, various chemicals, short chain fatty acids (SCFAs), and microbial products can alter the autonomic nervous system leading to secretion of cortisol and adrenaline from adrenals via CRF and ACTH-dependent pathways. The gut microbiota and neuroactive chemicals including histamine released from mast cells and 5-hydroxytryptamine are known to impair the intestinal secretion and intestinal mucosa in response to stress. Various stressors make the intestinal mucosa more permeable, more absorbable to bacterial cytotoxins and neurotransmitters (norepinephrine), and contribute to the development of local inflammation. In conditions associated with stress, bacteria can undergo translocation from intestinal lumen into systemic circulation affecting the central and peripheral organs. There is a growing evidence of the role of the gut microbiota in all aspects of health and disease, including brain health. Roles for the bacterial commensals and BGA in various psychiatric and neurological conditions, such as depression, autism, stroke, Parkinson's disease, and Alzheimer's disease, are emerging. Microbiota dysregulation has been documented in all of these conditions or in their animal models. Besides, several studies shows

KEYWORDS

Brain; gastrointestinal diseases; gut microbiota; mental disorders; microbiota

consistent effects of microbial states on behavior of mice, supporting a role for microbiota in modulating behavior. Over the past decade, it has become clear that the bi-directional communication pathway between gut bacteria and the central nervous system exerts a deep influence on some important brain processes, such as neuroinflammation, activation of the stress axes, neurotransmission, and neurogenesis. Gut bacteria influence these processes through their ability to synthesize neurotransmitters (e.g., GABA, noradrenaline, and dopamine) and modulate activation of the immune system, along with their ability to produce metabolites, such as SCFAs, that have neuroactive properties. Moreover, the gut microbiota and the brain are linked through additional pathways, such as the vagus pathway, enteroendocrine signaling and through the modulation of key dietary amino acids, such as tryptophan. The recent researches about gut-brain communication attempted to understand how the microbiome may shape microglial identity and function and how the microbiome, via microglia, may modulate the pathogenesis of the diseases. In conclusion, understanding the BGA will provide the basis for potential novel therapeutic approaches in the years ahead.

References

[1] Abdel-Haq R, Schlachetzki JC, Glass CK, Mazmanian SK. Microbiome–microglia connections via the gut–brain axis. J Exp Med 2019;216(1):41–59.
[2] Sherwin E, Sandhu KV, Dinan TG, Cryan JF. May the Force Be With You: The Light and Dark Sides of the Microbiota-Gut-Brain Axis in Neuropsychiatry. CNS drugs 2016;30(11):1019–1041.
[3] Sherwin E, Dinan TG, Cryan JF. Recent developments in understanding the role of the gut microbiota in brain health and disease. Ann N Y Acad Sci 2017.

[Abstract: 413] [Psychosomatic medicine-Liaison psychiatry]

Gastrointestinal signs and symptoms in mental disorders

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ABSTRACT

Medically unexplained gastrointestinal symptoms had a high prevalence in the general population (6-25%) [1]. There are also increased reporting of gastrointestinal symptoms (GIS) in many mental disorders. In a community study, it is reported that subjects with two GIS had even higher lifetime rates of depression (13.4%), panic (5.2%), or agoraphobia (17.8%) [1]. It is also suggested that women more prone to have any gastroistinal complaint. Nearly half of women with mania, schizophrenia, or panic disorder reported two or more GIS, and more than one fourth of women positive in any diagnostic category reported two or more symptoms [2]. It is widely known that GISs are strongly correlated with anxiety and depression [3,4]. Remission in the anxiety symptoms with psychiatric treatment results in reduction GIS [5]. The association between psychiatric disorders and GIS can be observed in different spectrum. It is suggested that eating disorder patients report many somatic symptoms related to the gastrointestinal tract, which may sometimes be interpreted as somatization [6]. Patients with eating disorders present with various gastrointestinal disturbances such as postprandial fullness, abdominal distention, abdominal pain, gastric distension, and early satiety, with altered esophageal motility sometimes seen in patients with anorexia nervosa. Fibromyalgia is another disease which is undertook in psychiatry clinics. It is reported in patients with fibromyalgia, the severity scores of dyspepsia symptoms, constipation, and dyspepsia-related "quality of life" disturbance were higher than in patients with romatoid artritis and controls [7]. Autism have been also studied in terms of brain-gut axis. A previous data suggest that a neurobehavioral rather than a primary organic gastrointestinal etiology may account for the higher incidence of these GISs in children with autism [8]. GISs widely manifested in various psychiatric conditions allow us to better understand the brain-gut axis.

KEYWORDS

Gastrointestinal symptoms; mental disorders; brain-gut axis

References

[1] Walker EA, Katon WJ, Jemelka RP, Roy-Byrne PP. Comorbidity of gastrointestinal complaints, depression, and anxiety in the Epidemiologic Catchment Area (ECA) Study. The American journal of medicine 1992;92(1):S26–S30.
[2] North CS, Alpers DH, Thompson SJ, Spitznagel EL. Gastrointestinal symptoms and psychiatric disorders in the general population, 1996.
[3] Haug TT, Mykletun A, Dahl AA. Are anxiety and depression related to gastrointestinal symptoms in the general population?. Scandinavian Journal of Gastroenterology 2002;37(3):294–298.
[4] Mussell M, Kroenke K, Spitzer RL, Williams JB, Herzog W, Löwe B. Gastrointestinal symptoms in primary care: prevalence and association with depression and anxiety. Journal of psychosomatic research 2008;64 (6):605–612.
[5] Noyes R, Cook B, Garvey M, Summers R. Reduction of gastrointestinal symptoms following treatment for panic disorder. Psychosomatics, 1990; 31(1), 75–79.

[6] Chami TN, Andersen AE, Crowell MD, et al. Gastrointestinal symptoms in bulimia nervosa: effects of treatment. Am J Gastroenterol. 1995;90:88–92.
[7] Pamuk, Ö. N., Ümit, H., & Harmandar, O. (2009). Increased frequency of gastrointestinal symptoms in patients with fibromyalgia and associated factors: a comparative study. The Journal of rheumatology, jrheum-090024.
[8] Incidence of Gastrointestinal Symptoms in Children With Autism: A Population-Based Study Ibrahim SH, Voigt RG, Katusic SK, et al (Mayo Clinic, Rochester, MN) Pediatrics 124:680-686, 2009.

[Abstract: 0726] [Psychosomatic medicine-Liaison psychiatry]

Psychiatric factors and psychosomatic symptoms in functional gastrointestinal disorders

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ABSTRACT
Functional medical disorders are defined as the body symptoms without objective physical, metabolic, or neurological explanations for their presence. Irritable bowel syndrome (IBS), non-ulcer dyspepsia, premenstrual syndrome, chronic pain syndromes, tension headaches, fibromyalgia, chronic fatigue syndrome, interstitial cystitis, reflex sympathetic dystrophy, temporomandibular joint syndrome, and various chemical and food sensitivities are examples of functional disorders in many organ systems [1]. These unexplained conditions are commonly associated with anxiety, depression, stressful life events, and psychological trauma in childhood or adulthood. Furthermore, patients with functional somatic symptoms are more likely to have an increased rate of current and lifetime psychiatric disorders [2]. The functional gastrointestinal disorders (FGIDs) are common conditions with an overall FGID prevalence burden estimated at approximately one-third of the population. The role of psychological factors has also been studied extensively in FGIDs. Traumatic events during childhood and higher levels of neuroticism are associated with elevated rates of FGIDs. Also, FGIDs are strongly associated with both anxiety and depression in cross-sectional studies [3]. However, the exact relationship between psychological and biological processes remains elusive. Finally, there is a substantial body of research supporting that there is a significant overlap between different functional medical disorders which make us think that there may be a shared common pathophysiology under these situations.

In this presentation; cutting edge findings about psychiatric entities in functional gastrointestinal diseases will be reviewed. Also, it is aimed to present possible etiologic mechanisms by addressing other psychosomatic symptoms that occur in functional gastrointestinal diseases.

KEYWORDS
Psychosomatic; functional gastrointestinal diseases; psychiatric comorbidity

References

[1] North CS, Hong BA, Alpers DH. Relationship of functional gastrointestinal disorders and psychiatric disorders: implications for treatment. World J Gastroenterol 2007;13(14):2020–2027.
[2] Afari N, et al., Psychological trauma and functional somatic syndromes: a systematic review and meta-analysis. Psychosom Med 2014;76(1):2–11.
[3] Jones MP, et al., Mood and Anxiety Disorders Precede Development of Functional Gastrointestinal Disorders in Patients but Not in the Population. Clin Gastroenterol Hepatol 2017;15(7):1014–1020.e4.

[Abstract: 0417] [Psychosomatic medicine - Liaison psychiatry]

Psychosomatic signs and symptoms in organic gastrointestinal diseases

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ABSTRACT
Depression and anxiety are common symptoms in clinical practice and depression has been projected to be the second leading cause disability worldwide by 2020. Moreover these two conditions are very important for the consultation and liaison psychiatry practice. In this study we aim to demonstrate effects of depression and anxiety on the course of

KEYWORDS
Depression; anxiety; antidepressant; inflammatory bowel disease; bio-psycho-social approach

inflammatory bowel disease (IBD), which is prototype of organic gastrointestinal disease. IBD is a chronic clinical course with relapsing remitting character. It typically has early life onset. Two main groups of IBD are Chron's disease and ulcerative colitis. Depression, and anxiety are common in IBD population. These symptoms are more severe during the onset and flare of IBD. Increased level of depression and anxiety is related to poorer quality of life, decreased drug treatment adherence, more frequent recurrence of the disease, and more common sexual dysfunction. The relationship between psychiatric conditions and IBD is not well understood. Immune system dysfunction and inflammatory process have been linked with both IBD and psychiatric conditions. Maladaptive coping strategies, insecure attachment style, and impaired social interactions were defined as psychosocial component related to IBD and psychogenic distress. Antidepressant treatment may be given for symptoms of depression, anxiety, and decreased level of quality of life. Higher risk for gastrointestinal bleeding were determined when antidepressant agent taken together with non-steroidal anti-inflammatory drugs. Psychotherapy may be useful for the heightened pain sensitivity, depression, anxiety, and reduced self-motivation to overcome difficulties associated with IBD. Thus, bio-psycho-social approach may be important for organic gastrointestinal problems and collaboration with mental health care specialists may be related to better outcome.

References

[1] Yanartas O, Kani HT, Bicakci E, Kilic I, Banzragch M, Acikel C, Atug O, Kuscu K, Imeryuz N, Akin H. The effects of psychiatric treatment on depression, anxiety, quality of life, and sexual dysfunction in patients with inflammatory bowel disease. Neuropsychiatr Dis Treat. 2016;12:673–683.

[2] Graff LA, Walker JR, Bernstein CN. Depression and anxiety in inflammatory bowel disease: a review of comorbidity and management. Inflamm Bowel Dis. 2009;15(7):1105–1118.

[3] Filipovic BR, Filipovic BF. Psychiatric comorbidity in the treatment of patients with inflammatory bowel disease. World J Gastroenterol. 2014; 20(13):3552–3563.

[Abstract: 0748] [ADHD]

Contribution of neuro- and bio-feedback in treatment of ADHD

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ABSTRACT

Attention-deficit/ hyperactivity disorder (ADHD) is a disorder that manifests in childhood with symptoms of hyperactivity, impulsivity, and/or inattention. The symptoms affect cognitive, educational, behavioral, emotional, and social functioning. Medications, with or without behavioral/psychologic interventions, are the first-line therapy for school-aged children and adolescents who meet diagnostic criteria for ADHD. The treatment plans for children with ADHD vary according to age. For preschool children (age 4 through 5 years) who meet the diagnostic criteria for ADHD, we suggest behavior therapy rather than medication as the primary therapy. For most school-aged children and adolescents (≥6 years of age) who meet the diagnostic criteria for ADHD and specific criteria for medication, we suggest initial treatment with stimulant medication combined with behavioral therapy to improve core symptoms and target outcomes. We recommend behavioral interventions for children with problematic behavior who do not meet the diagnostic criteria for ADHD. Response to treatment is demonstrated by objective measurement of reduction in core symptoms and/or improvement in target goals (e.g., 40 to 50 percent reduction in core symptoms compared with baseline; the decreased proportion of missing assignments from 60 to 20 percent per week). There are some treatment modalities; Behavioral interventions; Behavioral techniques that are used for children with ADHD include positive reinforcement, time-out, response cost (withdrawing rewards or privileges when unwanted or problem behavior occurs), and token economy (a combination of positive reinforcement and response cost), Pharmacotherapy; Medications, with or without behavioral/psychologic interventions, are the first-line therapy for school-aged children (≥6 years) and adolescents who meet diagnostic criteria for ADHD, Combination therapy; Combination therapy uses both behavioral/psychologic interventions and medications, School-based interventions; School-based interventions may include the provision of tutoring or resource room support (either in an exclusive setting or within the classroom, classroom modifications, accommodations, or behavioral interventions. EEG biofeedback (neurofeedback) began in the late 1960s as a method for retraining brainwave patterns through operant conditioning. Since that time a sizable body of research has accumulated on the effectiveness of neurofeedback in the treatment of uncontrolled epilepsy, ADD/ ADHD, anxiety, alcoholism, posttraumatic stress disorder, and mild head injuries. Since the

KEYWORDS

ADHD; neurofeedback; biofeedback

first reports of neurofeedback treatment in Attention-deficit/ hyperactivity disorder (ADHD) in 1976, many studies have investigated the effects of neurofeedback on different symptoms of ADHD such as inattention, impulsivity and hyperactivity. This technique is also used by many practitioners (Hammond, 2007), but the question as to the evidence-based level of this treatment is still unclear. Complementary and alternative medicine therapies other than physical activity, elimination diets, fatty acid supplementation, and mindfulness that have been suggested in the management of ADHD include vision training, megavitamins, herbal and mineral supplements, neurofeedback/biofeedback, chelation, and applied kinesiology, among others. Most of these interventions have not been proven efficacious in high-quality randomized controlled trials.

References

[1] American Psychiatric Association. Attention-deficit/ hyperactivity disorder. In: Diagnostic and Statistical Manual of Mental Disorders, 5th ed.. Arlington (VA): American Psychiatric Association, 2013. p. 59.
[2] Subcommittee on Attention-Deficit/Hyperactivity Disorder, Steering Committee on Quality Improvement and Management, Wolraich M, et al. ADHD: clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. Pediatrics 2011; 128:1007.
[3] Hammond DC. What is neurofeedback? Journal of neurotherapy 2007;10(4):25–36.

[Abstract: 0617] [ADHD]

Occupational therapy on ADHD

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ABSTRACT

Attention-deficit/ hyperactivity disorder (ADHD) affects approximately % 5–10 children and manifests in childhood with symptoms of hyperactivity, impulsivity, and/or inattention. The symptoms affect cognitive, academic, behavioral, emotional, and social functioning [1]. The purpose of this project is to investigate the occupational therapy interventions are effective for school-aged children with sub threshold ADHD symptoms.

ADHD is a syndrome with two categories of core symptoms: hyperactivity/impulsivity and inattention. Each of the core symptoms of ADHD has its own pattern and course of development. The complaint regarding symptoms of ADHD may originate from the parents, teachers, or other caregivers. Hyperactive and impulsive symptoms typically are observed by the time the child reaches four years of age and increase during the next three to four years, peaking in severity when the child is seven to eight years of age [3,4]. After seven to eight years of age, hyperactive symptoms begin to decline; by the adolescent years, they may be barely discernible to observers, although the adolescent may feel restless or unable to settle down. In contrast, impulsive symptoms usually persist throughout life. Thirty sub threshold ADHD patients were included in this study. Eight sessions of occupational therapy were given once a week. Dunn sensory profile was applied before and after occupational therapy sessions. Conners forms were given to their teachers pre- and post-therapy sessions. There was a significant improvement in the Dunn sensory profiles after the occupational therapy. ADHD can have a negative impact on occupational performance such as on activities of daily living (ADLs), instrumental activities of daily living (IADLs), education, rest and sleep, leisure, play and social participation [5]. Occupational therapists are able to provide interventions to improve engagement in daily activities on social skills, play, executive functioning, impulsivity, inattention, and motor coordination [6]. Occupational therapy on ADHD has a significant role in treatment and some intervention areas. The parent or child-focused intervention programs can focus on cognition, motor, sensory or play areas. ADHD can have a limiting impact on a child's occupations in many areas including, ADLs, IADLs, academic, rest and sleep, leisure, play, and social participation. It was found that performance skills/ client factors were discussed more in depth than areas of occupations in terms of areas to address in intervention. Although the first step treatment is medication for intervention of ADHD, therapy is advised as a co-treatment of intervention for those individuals affected. The four main areas were explored, which are cognition, motor, sensory, and the area of occupation of play and with that, multiple interventions were found for therapists to improve those performance skills/client factors.

KEYWORDS

ADHD; Dunn's sensory profile; occupational therapy; sub-threshold ADHD

References

[1] American Psychiatric Association. Attention-deficit/ hyperactivity disorder. In: Diagnostic and Statistical Manual of Mental Disorders, 5th ed., Arlington (VA): American Psychiatric Association, 2013. p. 59.
[2] Subcommittee on Attention-Deficit/Hyperactivity Disorder, Steering Committee on Quality Improvement and Management, Wolraich M, et al. ADHD: clinical practice guideline for the diagnosis, evaluation, and treatment of attention-deficit/hyperactivity disorder in children and adolescents. Pediatrics 2011; 128:1007.
[3] Applegate B, Lahey BB, Hart EL, et al. Validity of the age-of-onset criterion for ADHD: a report from the DSM-IV field trials. J Am Acad Child Adolesc Psychiatry 1997; 36:1211.
[4] Lahey BB, Applegate B, McBurnett K, et al. DSM-IV field trials for Attention-deficit/ hyperactivity disorder in children and adolescents. Am J Psychiatry 1994; 151:1673.
[5] Kaneko, F., & Okamura, H. (2005). Study on the social maturity, self-perception, and associated factors, including motor coordination, of children with Attention-deficit/ hyperactivity disorder. Physical & Occupational Therapy in Pediatrics, 25(4) 2005.
[6] Wehmeier, P.M., Schacht, A., & Barkley, R.A. (2010). Social and emotional impairment in children and adolescents with ADHD and the impact on quality of life. Journal of Adolescent Health 46, 209–217.

[Abstract: 684] [ADHD]

Contribution of family-based approaches in treatment of ADHD

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ABSTRACT

Many behavioral interventions have a psychoeducational component that provides information about the nature of the disorder. One of the aims of the parent education programs is to increase the knowledge of parents about the nature, possible causes, general course of ADHD and treatment options of the disorder. This may be on a stand-alone target, but may also be on a necessary basis for subsequent therapeutic intervention.

Parental education programs for ADHD often focus on reducing the problems associated with ADHD and impaired functioning rather than ADHD symptoms. The reasons for applying for families are often these problems, and the main goal of many treatment modalities is in fact about these behavioral problems. Parent training-based behavioral interventions for individuals with ADHD are trying to focus on reducing these behavioral problems, which are very common in these children. Meta-analyzes confirm that behavioral interventions reduce behavioral problems in children with ADHD. In other studies, it has been shown that various behavioral interventions, including parent training, reduce the symptoms of core ADHD [1]. However, there are studies indicating that parental education does not significantly reduce ADHD symptoms when administered individually and single-blind, therefore it is not recommended to reduce the symptoms of ADHD. But also, these studies support the fact that parent trainings change parent perceptions of the child's behavior, and in fact, even though they do not actually reduce the core ADHD findings, these programs are highly effective. Patients with more symptomatic and comorbid ADHD can make parental programs less effective. A recent study compared two programs aimed at developing general parenting skills and treating misbehavior with a specific intervention to ADHD. The first program was found to be more effective in comorbid ADHD and oppositional defiant cases [2,3]. As a result, behavioral interventions involving parenting programs can be used for children with ADHD regardless of the severity of symptoms. Comorbidity may change the effects of these behavioral interventions, but these programs are still recommended for children with comorbidities.

KEYWORDS

Parenting programs; parents plus programs; ADHD; behavioral interventions; complementary treatment

References

[1] Montoya A, Colom F, Ferrin M. Is psychoeducation for parents and teachers of children and adolescents with ADHD efficacious? A systematic literature review. Eur Psychiatry. 2011;26(3):166–175.
[2] Van Den Hoofdakker BJ, Van der Veen-Mulders L, Sytema S, Owens EB, Hinshaw SP, Kraemer HC, Arnold LE, Abikoff HB, Cantwell DP, et al. Which treatment for whom for ADHD? Moderators of treatment response in the MTA. J Consult Clin Psychol. 2003;71 (3):540.
[3] Forehand R, Parent J, Sonuga-Barke E, Peisch VD, Long N, Abikoff HB. Which type of parent training works best for preschoolers with comorbid ADHD and ODD? A secondary analysis of a randomized controlled trial comparing generic and specialized programs. J Abnorm Child Psychol. 2016;44(8):1503–1513.

[Abstract: 0614] [ADHD]

Contribution of supplements in treatment of ADHD

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ABSTRACT

Attention-deficit/ hyperactivity disorder (ADHD) is one of the most common psychiatric disorders of childhood. The prevalence in school-age children is 5%. In children, stimulant and non-stimulant drugs are used safely in the treatment of ADHD. Short and medium term efficacy of these drugs in the course of treatment has been reported. Currently, multimodal approaches combining pharmacological and psychological approaches are recommended primarily for treatment. Drug therapies have been shown to be effective in a variety of conditions in patients with ADHD, including core symptoms, comorbid behavior problems, functioning, executive functions, executive and non-executive memory, response time, response time variability, response inhibition, delayed reward avoidance, and intrinsic motivation. However, the need for non-pharmacological interventions that affect both the main symptoms and neuropsychiatric disorders in the treatment of ADHD has been raised because of the different limitations of drug therapies such as partial response, adverse effects, negative attitudes of the patients and their relatives, indefinite long-term benefits and treatment costs. To date, various non-pharmacological treatment modalities such as dietary treatments, psychological interviews and neurofeedback have been discussed in the treatment of ADHD. Alternative therapies for neurodevelopmental disorders including both ADHD and Autism Spectrum Disorder (ASD) such as dietary interventions have increasingly been drawing interest in recent years. Recommended treatments include different approaches such as adjusting diet by removing certain foods or adding different group of food supplements. Studies on dietary regimens in children with ADHD, diets containing high levels of fat, refined sugar and sodium, and low amounts of fiber, folate and omega-3 fatty acids were reported to exacerbate ADHD symptoms. Foods containing artificial dye (e.g., petrochemical dyes), artificial flavors and fragrances, some preservatives, artificial sweeteners and salicylates have been recommended to be removed from the diet. In addition, an individualized oligoantigenic elimination diet excluding foods such as cow's milk, cheese, eggs, chocolate and hazelnut, which are known to be allergic, has been recommended as an effective treatment for ADHD. Omega-3 fatty acids were found lower in plasma and erythrocyte membranes of children and adults with ADHD and the essential fatty acids omega-3 and omega-6 have been suggested to be added to the treatment. Decreased metabolic activity due to the deficiency cofactors regulating neurotransmitter enzyme function such as vitamins and minerals (iron, zinc, magnesium), has been suggested to be an underlying factor and recommended to be supplemented in the treatment. Several commercial products are available as a combination of the recommended fatty acids, minerals and vitamins.

All these alternative therapies recommended in the treatment of ADHD provided limited efficacy in clinical studies. Larger-scale studies including evaluation of long-term outcomes are needed. The efficacy of this dietary adjustment in different groups of children is not clear and available data is not sufficient to draw a conclusion regarding these approaches.

KEYWORDS

Attention-deficit/ hyperactivity disorder; supplements; treatment; dietary treatments; minerals and vitamins

[Abstract: 0626] [Schizophrenia and other psychotic disorders]

Psychotic disorders of childhood

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ABSTRACT

Psychotic disorders, particularly schizophrenia, are among the most severe mental disorders that can lead to delays in maturation of neuropsychological functions, impairments in psychosocial functioning and in the long term cognitive ,emotional and behavioral deficits in adolescents (Schimmelmann ve ark. 2012). Thus, early detection of psychotic disorders and shortening the duration of untreated period is important for good prognosis. It is reported that adolescent onset psychosis is associated with worse outcomes compared to adult onset psychosis. Current literature revealed that, with early diagnosis and treatment, adolescent

KEYWORDS

Childhood; prepsychotic; prodromal

onset psychosis has similar short term prognosis (Schimmelmann ve ark. 2007),, and better long term prognosis (Amminger ve ark 2011). Prodromal symptoms of psychotic disorders and schizophrenia are characterized by nonspecific symptoms such as depression, high level of anxiety, irritability and impairment in social functioning. In addition, presence of history of psychotic disorder in the first degree relatives, presence of schizotypal personality disorder, having attenuated psychotic symptoms or brief limited intermittent psychotic symptoms, impairment in psychosocial functioning during adolescence are considered as ultra-high risk symptoms (Schimmelmann ve ark. 2012). When considering the fact that the highest risk of developing schizophrenia is around 20 years of age (Amminger ve ark. 2006), the importance of early detection of prepsychotic symptoms is well understood. On the other hand, false positive diagnosis can lead to stigmatization and increase the risk of side effects due to long term use of antipsychotic medication. This presentation aims to discuss prepsychotic symptoms, early detection and intervention of psychosis during the adolescence under the highlight of recent literature.

References

[1] Schimmelmann BG, Schultze-Lutter F. Early detection and intervention of psychosis in children and adolescents: urgent need for studies, 2012.
[2] Schimmelmann BG, Conus P, Cotton S, McGorry PD, Lambert M. Pre-treatment, baseline, and outcome differences between early-onset and adult-onset psychosis in an epidemiological cohort of 636 first-episode patients. Schizophrenia research 2007;95(1-3):1–8.
[3] Amminger GP, Henry LP, Harrigan SM., Harris MG., Alvarez-Jimenez M, Herrman H, McGorry PD. Outcome in early-onset schizophrenia revisited: findings from the Early Psychosis Prevention and Intervention Centre long-term follow-up study. Schizophrenia research 2011;131(1-3):112–119.
[4] Amminger GP, Harris MG, Conus P, Lambert M, Elkins KS, Yuen HP, McGorry PD. Treated incidence of first-episode psychosis in the catchment area of EPPIC between 1997 and 2000. Acta Psychiatrica Scandinavica 2006;114(5):337–345.

[Abstract: 0665] [Schizophrenia and other psychotic disorder]

Clinical presentation and prognosis of childhood psychosis

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ABSTRACT

Schizophrenia is a complex disorder in which genetic and environmental factors contribute both to the risk of developing the illness. Its clinical features are positive symptoms (hallucinations and delusions), negative symptoms, disordered thoughts and speech and disorganized behaviors. When the disorder begins under 18 years old it is termed early onset schizophrenia (EOS), with some cases beginning even earlier (under 14 years old) and called child onset schizophrenia (COS). The National Institute of Mental Health cohort showed that *children* with psychotic symptoms are *often misdiagnosed* and diseases such as bipolar disorder, depression, post-traumatic stress disorder, anxiety disorders may present with psychotic symptoms in children [1]. The prevalence of all psychotic symptoms increase in adolescence. *An* estimated 20% of patients with EOS experience only one episode and the full recovery occurs mostly in first three months. 25% of children who exhibit psychotic symptoms at age 11 meets the criteria of any *schizophreniform* disorder at age 26. Therefore periodic assessments of all children, who display any psychotic symptom, namely "at risk mental states (ARMS)" are essential. *There is some evidence to* suggest that EOS is a severe form of adult onset schizophrenia (AOS). Early onset schizophrenia is characterized with a greater disorganization and a worse outcome than AOS, which makes a large contribution to the burden of disease. *Early intervention* improves *long*-term outcomes for children with EOS. *Impaired premorbid social functioning*, motor delay, a family history of schizophrenia are more *common in EOS* (*than in AOS*). Severity of negative and cognitive symptoms are the best predictors of long term outcome. Indicators of a better outcome are rapid recovery and acute onset. In contrast, male gender, substance use, having a neurodevelopmental disorder, premorbid social and cognitive impairments, history of trauma or maltreatment, insidious onset, a prolonged first episode, extended duration of untreated psychosis are associated with a poorer prognosis [2]. Psychiatric *comorbidities* are common among children with EOS. Comorbidities such as anxiety disorder or depression may lead to persisting of positive symptoms. The risk for suicide, homicide, substance use should be evaluated at first and following examinations. Cannabis use is associated with cognitive deterioration. *Antipsychotic medications are* the *cornerstone* of schizophrenia *treatmen*t of which a *low* initial *starting dose* is *recommended*. Children are more vulnerable to side effects than adults. Preventing and managing side effects increases the patient's compliance to the treatment. Prevalence of

KEYWORDS

Adolescents; childhood-onset schizophrenia; children; prognosis; schizophrenia; treatment outcome

cardiovascular diseases and diabetes is increased in patients with schizophrenia and may be the reasons of premature death. Clozapine used in treatment resistance schizophrenia may cause life threatening side effects. Sedation and cognitive slowing may cause withdrawal of treatment. Weight, height, pulse, blood pressure should be monitored at first and following assessments. Besides medication, psychoeducation also reduces the rate of relapse [3]. Psychoeducation, family interventions help the patients to recognize the early signs and warning symptoms. Improved patient compliance have a major effect on treatment outcomes.

References

[1] Nicolson R, Rapoport JL. Childhood-onset schizophrenia: rare but worth studying. Biol Psychiatry. 1999;46(10):1418–1428.
[2] Stentebjerg-Olesen M, Pagsberg AK, Fink-Jensen A, Correll CU, Jeppesen P. Clinical Characteristics and Predictors of Outcome of Schizophrenia Spectrum Psychosis in Children and Adolescents: A Systematic Review. J Child Adolesc Psychopharmacol. 2016;26(5):410–427.
[3] Xia J, Merinder LB, Belgamwar MR. Psychoeducation for schizophrenia. Schizophr Bull. 2011;37(1):21–22.

[Abstract: 0767] [Schizophrenia and other psychotic disorders]

Psychotherapeutic approaches in early onset psychotic disorders

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ABSTRACT

Psychotic disorders and schizophrenia are long term debilitating disorders that have serious implications on the mental, behavioral and psychosocial development of children and adolescents (Jepsen et al. 2010). Early onset psychotic disorders that first become symptomatic in childhood and adolescence differ from adult onset psychotic disorders in that they are characterized with undifferentiated symptoms and diagnostic challenges (Werry 1992). Mood symptoms, social withdrawal, decrease in personal hygiene, academic deterioration and withdrawal from peer relations are frequently encountered psychosocial changes (Iyer et al, 2008). Cognitive developmental delay, learning disorders and social adjustment issues are often found to accompany early onset psychotic disorders. Bizarre behavioral patterns and negative symptoms are reported to be more frequently encountered in early onset psychotic presentations compared to adult onset psychotic disorders. Amongst negative symptoms, blunted affect is reported to be more prevalent than other symptoms (Ballageer et al. 2005). Early onset psychotic disorders are commonly comorbid with Attention-deficit/ hyperactivity disorder, Mood Disorders, Anxiety Disorders and Substance Misuse and Abuse. Early onset psychotic disorders are reported to have worse long term prognosis than adult onset types, given that they have an insidious prodromal period with a longer time until diagnosis (Joa et al. 2009). Joa et al. (2009) have reported that depressive symptoms and lifelong suicide contemplations are encountered more frequently in early onset psychotic disorders. Literature review reveals that insidious onset, absence of positive symptoms at the prodromal or early stages, equivocal symptoms that resemble other disorders are the reasons why diagnosis and treatment of psychotic disorders that present in childhood and adolescence are delayed. Early recognition of psychotic presentations are as important as early and effective medical and psychotherapeutic approaches. Antipsychotic pharmacotherapy with adjunct cognitive behavioral therapy for 9 months duration are demonstrated to be effective in decreasing delusional symptoms in psychotic patients, although CBT has not been shown to be effective for hallucinations (Durham et al, 2003). Psychodynamic therapy in psychotic disorders can be beneficial as a supporting approach that could help patients with understanding the relationship between psychotic episodes and interpersonal issues, as well as providing insight into drug resistance (Johannessen et al. 2006).

KEYWORDS

Child; adolescent; early onset; psychosis; psychotherapy

References

[1] Jepsen JRM, Fagerlund B, Pagsberg AK, Christensen AMR, Hilker RW, Nordentoft M, Mortensen EL. Course of intelligence deficits in early onset, first episode schizophrenia: a controlled, 5-year longitudinal study. European child & adolescent psychiatry 2010;19(4):341-351.
[2] Iyer SN, Boekestyn L, Cassidy CM, King S, Joober R, Malla AK. Signs and symptoms in the pre-psychotic phase: description and implications for diagnostic trajectories. Psychological Medicine 2008;38(8):1147–1156.
[3] Werry JS. Child and adolescent (early onset) schizophrenia: a review in light of DSM-III-R. Journal of Autism and Developmental Disorders 1992;22:601–624 114(5), 337-345.

[4] Ballageer, T., Malla, A., Manchanda, R., Takhar, J., & Haricharan, R. (2005). Is adolescent-onset first-episode psychosis different from adult onset?. Journal of the American Academy of Child & Adolescent Psychiatry, 44(8), 782–789.
[5] Joa, I., Johannessen, J. O., Langeveld, J., Friis, S., Melle, I., Opjordsmoen, S., … & Larsen, T. K. (2009). Baseline profiles of adolescent vs. adult-onset first-episode psychosis in an early detection program. Acta Psychiatrica Scandinavica, 119(6), 494–500.
[6] Durham, R. C., Guthrie, M., Morton, R. V., Reid, D. A., Treliving, L. R., Fowler, D., & MacDonald, R. R. (2003). Tayside–Fife clinical trial of cognitive–behavioural therapy for medication-resistant psychotic symptoms: results to 3-month follow-up. The British journal of psychiatry, 182(4), 303–311.
[7] Johannessen, J. O., Martindale, B. V., & Cullberg, J. (Eds.). (2007). Evolving psychosis: Different stages, different treatments. Routledge.

[Abstract: 0792] [Others]

Functional neuroimaging in childhood schizophrenia

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ABSTRACT
Childhood-onset schizophrenia (COS) is pretty rare but debilitating disorder. With the advance of imaging technologies, many researchers have provided data with regards to functional brain network abnormalities in children with schizophrenia. Two main fMRI techniques (functional and resting state) have been implemented to assess the dynamic response of neural networks to a given neurocognitive task and the integrity of functional connectivity between discrete brain regions during resting-state, respectively. Hypo activity in the cortices of dorsolateral prefrontal, ventromedial cortex, parietal, anterior cingulate and temporal lobe has been reported in functional fMRI studies in children and adolescents with schizophrenia. Additionally, functional connectivity abnormalities in the default mode network (DMN), sensory and motor cortex, frontoparietal network were also reported in the COS literature. In this presentation, the association of fMRI findings and schizophrenia symptoms will be discussed in the lights of corollary discharge theory.

KEYWORDS
Child-onset schizophrenia; child; adolescent; psychosis; fMRI

[Abstract: 0529] [Others]

AutoImmune neuropsychiatric disorders in childhood: current treatments and clinical experiences

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ABSTRACT
Significant interest has recently focused on autoimmunity and neuropsychiatric disorders, especially Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections (PANDAS). However, in current literature suggest that the presence of immune system alterations in obsessive-compulsive disorder (OCD), tics and other abrupt presentation of neuropsychiatric disorders probably triggered by different agents, such as infections and stress rather than Goup A Beta Hemolytic Streptococ (GABHS). It is called as Pediatric Acute Onset Neuropsychiatric Syndrome (PANS).The pathophysiology of PANS/PANDAS are still unknown efficiently. Different studies support the hypothesis that this disorder may have an immunogenetic etiology, suggesting the production of autoantibodies in response to a streptococcal infection; these antibodies would be able to cross the blood-brain barrier and react against neurons, especially against those located in the basal ganglia. In addition, familial autoimmunity, environmental and genetic factors may trigger neuropsychiatric disorders in some individuals. The clinical symptomatology may depend on patients' age, comorbidity, gender and genetic susceptibilities. ADHD, Tourette, Separation Anxiety and abrupt problematic eating patterns are mostly seen clinical conditions besides OCD and tics in PANS cases. In this presentation, we aim to discuss phenomenology, current treatment modalities and clinical approaches to PANDAS/PANS cases who shown different symptomatology.

PANDAS/PANS cases who have different symptomatology:

KEYWORDS
Pediatric Autoimmune Neuropsychiatric Disorders Associated With Streptococcal Infections; Pediatric Acute Onset Neuropsychiatric Syndrome; autoimmunity; obsessive-compulsive disorder; tics

A. Pediatric bipolar disorder case; 13-year-old boy. He did not benefit from the standard treatment for bipolar disorder. With the injection of penicillin once every 21 days, there were mild attacks of less than 4 months.
B. Attention deficit and hyperactivity disorder case; A 10-year-old boy. Penicillin injection was added to the current stimulant treatment every 21 days. Symptoms decreased, increased functionality.
C. Sleepwalking Disorder case: 12-year-old boy who responded penicillin prophylaxis.
D. Psychotic Disorder with Mental Difficulties: 6-year-old boy who responded partial remission after IVIG treatment.

Current studies have shown that immunomodulator and neuroprotective molecules are candidates for protecting recurrent inflammation of central nervous system. Treatment with antibiotics, corticosteroids, non-steroidal anti-inflammatory drugs, intravenous immunoglobuline and plasmapheresis may be effective for in PANS/PANDAS although there are no standardazitated pharmacological agents in these cases.

References

[1] Calaprice D, Tona J, Murphy TK. Treatment of pediatric acute-onset neuropsychiatric disorder in a large survey population. J Child Adolesc Psychopharmacol, Mar;28 (2):92–103, 2018.
[2] Frankovich J, Swedo S, Murphy T, Dale RC, Agalliu D, Williams K, Daines M, Homig M, Chugani H, Sanger T, Muscal E, et al; Clinical management of pediatric acute-onset neuropsychiatric syndrome: part II-use of immunomodulatory therapies. J Child Adolesc Psychopharmacol, 27(7):1–16, 2017.
[3] Swedo SE, Leonard HL, Garvey M, Mittleman B, Allen AJ, Perlmutter S, Lougee L, Dow S, Zamkoff J, Dubbert BK. Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections: clinical description of first 50 cases. Am J Psychiatry, 155:264–271, 1998.
[4] Thienemann M, Murphy T, Leckman J, et al. Clinical Management of Pediatric Acute-Onset Neuropsychiatric Syndrome: Part I-Psychiatric and Behavioral Interventions. J Child Adolesc Psychopharmacol. Sep;27(7):566–573, 2017.
[5] Wilbur C, Bitnun A, Kronenberg S, et al. PANDAS/PANS in childhood: Controversies and evidence. Paediatrics & Child Health pxy145, 2018.
[6] Baytunca, MB, Donuk T, ve Erermis S. Evaluation of a neuropsychiatric disorder: from PANDAS to PANS and CANS. Turk Psikiyatri Derg, 2016;27(2):0
[7] Greenberg R. How pediatric bipolar disorder and pandas/pans symptoms overlap and can cause diagnostic confusion. European Psychiatry 2013;1(1):2666. Abstracts of the 21th European Congress of Psychiatry. https://www.nimh.nih.gov/news/science-news/2017/guidelines-published-for-treating-pans-pandas.shtml
[8] Mink J, Kurlan R. Acute postinfectious movement and psychiatric disorders in children and adolescents. Journal of child neurology. Feb 2011;26(2):214–217.
[9] Swedo SE, Leonard HL, Garvey M, Mittleman B, Allen AJ, Perlmutter S, et al. Pediatric autoimmune neuropsychiatric disorders associated with streptococcal infections: clinical description of the first 50 cases. American Journal of Psychiatry. 1998;155(2):264–271.

[Abstract: 0657] [Others]

The concept of social cognitions and adolescence

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ABSTRACT

Adolescence is a period of physical, psychological and social change from childhood to adulthood. Adolescents are more social than children; they constitute more complex peer relationships and are more susceptible to acceptance and rejection by their friends. İmportance of peer relations and understanding of others increase during the adolescence. Social cognitive functions include the ability to understand the beliefs and intentions of others, to recognize that other people have a different mind than their own and, to be able to interact with appropriate responses. Behaviors related to social cognition changes prominently during adolescence. Two major changes were showed in the brain before and after puberty. The first one is that axonal myelination in some cortical regions increase during adolescence. This process increases the speed of transmission of neuronal information. The second change is that grey matter volume decrease as a result of synaptic elimination (pruning) and thus often used connections are strengthened and rare used connections are eliminated [1]. The brain regions associated with social cognition are medial prefrontal cortex, posterior superior temporal sulcus, amygdala, the anterior cingulate cortex, anterior insula, the temporoparietal junction, inferior frontal gyrus, interparietal sulcus [2]. The synaptic rearrangement in these cortical regions is likely to have effect on social cognitive processes such as mentalizing, perspective taking and related processes [3] Remodeling process in the brain especially in prefrontal, parietal and superior temporal cortex facilitate the required social skills during adolescence.

KEYWORDS

Adolescence; brain development; social cognition

References

[1] Blakemore SJ, Choudhury S. Development of the adolescent brain: implications for executive function and social cognition. Journal of child psychology and psychiatry 2006;47(3-4):296–312.

[2] Blakemore SJ. The social brain in adolescence. Nature Reviews Neuroscience 2008;9(4):267.

[3] Choudhury S, Blakemore SJ Charman T. Social cognitive development during adolescence. Social cognitive and affective neuroscience 2006;1(3):165–174.

[Abstract: 0679] [Others]

Social cognition in adolescent depression: cause or result?

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ABSTRACT

Major depressive disorder (MDD) one of the most debilitating and prevalent disease worldwide is characterized by persistent and severe low mood and/or loss of enjoyment and interest and impaired functioning in multiple domains, including social and interpersonal functioning. Previous reports have shown that social cognitive deficits can contribute particularly to poor social functioning in several disorders as well as in depression. In perspective of developmental psychopathology, it is of importance to determine whether social cognitive impairment has a progressive developmental course or likewise prevails in early stages of MDD in childhood or adolescence. However, to date most studies examining social cognition in MDD have been conducted among adult patients. Thus, little is known about the trajectory of social cognition in adolescent depression, while most MDD begins emerging in this early stage of life. Moreover, it is also not clear which particular aspects of social cognition are impaired in MDD. In this presentation, it is aimed to explore social cognitive abilities in MDD in the point of view of developmental psychopathology.

KEYWORDS

Adolescent depression; developmental psychopathology; social cognition

[Abstract: 0643] [Mood disorders]

Bipolar disorder and theory of mind from developmental perspective

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ABSTRACT

In the current presentation, it is aimed to be discussed the deficits of social cognition and theory of mind in bipolar disorder from a developmental perspective. Bipolar disorder is a chronic and recurrent psychiatric disorder that is characterized by cyclic changes between mania and major depressive episodes. Since bipolar disorder causes the high rates of hospitalization, suicidal attempts, substance abuse, and psychiatric comorbidity, it is the fourth leading reason of disability adjusted life years (DALYs) in 15 to 19 years old adolescents worldwide. Social cognition is a complex psychological function that enables individual to interact with his/her social environment in an adaptive way. It involves the representation of internal somatic state, the perception of self and others, and interpersonal motivations. Theory of mind, which is the ability to attribute mental states to others and understand and predict others' behavior based on their mental states, is one of the components of social cognition. Deficits in social cognition can cause impairments in psychosocial functionality in psychiatric disorders. Social cognition impairment were found to be related to persistent poor social functioning in individuals with SCZ. Social cognition deficits mediated the relationship between neurocognition and functional impairment in these individuals. In addition, a significant link between functional impairment and social cognition deficits has been found in adolescents with attention deficit and hyperactivity disorder [1]. Social cognitive impairments including emotion recognition and theory of mind have been well defined in schizophrenia (SCZ) and neurodevelopmental disorders, especially in autism. On the other hand, recent studies have also shown social cognitive deficits in individuals with bipolar disorder. Social cognition deficits have been associated with bipolar disorder with medium effect size in a recent meta-analysis. Compared to healthy controls, significant impairments

KEYWORDS

Bipolar disorder; development; pediatric bipolar disorder; social cognition; theory of mind

were found in faux pas task, hinting task, the false beliefs task, reading the mind in the eyes task, and facial emotion recognition in bipolar disorder. Although social cognition deficits were more apparent during acute episodes, these deficits were persistent in remitted patients. Structural abnormalities in VLPFC (ventrolateral prefrontal cortex), MPFC (medial prefrontal cortex), and amygdala; and altered functional connectivity between these regions may be related to impaired social cognition in bipolar disorder [2]. Less is known concerning the social-cognitive profiles of pediatric bipolar disorder. Impairments in false belief task and facial emotion identification, deficits in social inference ability have been reported in pediatric bipolar disorder, in line with adult studies. Moreover, social cognitive deficits have been detected both in euthymic and symptomatic patients with pediatric bipolar disorder. Importantly, earlier onset of pediatric bipolar disorder was found to be associated with increased theory of mind deficits. This finding indicates that, pediatric bipolar disorder can interfere with the development of social cognition abilities, with potentially long-term impact on social skills. Therefore, interventions focusing on social cognition deficits is crucial both to improve interpersonal functioning and to impede long term effects of pediatric bipolar disorder [3].

References

[1] Bora E, Bartholomeusz C, Pantelis C. Meta-analysis of Theory of Mind (ToM) impairment in bipolar disorder. Psychological medicine 2016; 46(2):253–264.

[2] Samamé C, Martino DJ, Strejilevich SA. Social cognition in euthymic bipolar disorder: systematic review and meta-analytic approach. Acta Psychiatrica Scandinavica 2012; 125(4):266–280.

[3] Schenkel LS, Marlow-O'Connor M, Moss M, Sweeney JA, Pavuluri MN. Theory of mind and social inference in children and adolescents with bipolar disorder. Psychological Medicine 2008 ;38(6):791–800.

[Abstract: 0798] [Others]

Biological and clinical markers in bipolar disorder

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ABSTRACT

According to the World Health Organization (WHO), in terms of loss of functioning, when all medical diseases are evaluated, it is stated that the bipolar disorder is in the sixth position (Phlips et al. 2013. Lancet).

Diagnosis and management of bipolar disorder (BD) are limited by the absence of available laboratory tests. A major limitation in the diagnosis and management of bipolar disorder is the exclusive reliance on subjective clinical information in the absence of available laboratory tests (Akiskal et al. 2007). Diagnostic criteria are arbitrary, often leading to wrong and delayed diagnoses and it is difficult to assess whether affective symptoms are indicative of emerging affective episodes. Identification of peripheral blood biomarkers of disease and disease activity has the potential to both advance the understanding of pathophysiological processes and to improve clinical treatment of bipolar disorder .Although our understanding of its biological background is inadequate, growing evidence indicates that inflammatory disturbances, altered neuroplasticity , oxidative stress and disturbances related to mitochondrial function are associated with bipolar disorder. Apart from a study (Barbosa et al. 2012), it has been shown that there is a correlation between elevation of proinflammatory cytokine levels in Bipolar Disorder patients and impaired cognitive functions such as memory, executive functions, attention, language and visual spatial memory (Lotrich et al., 2014). 2015, Hoseth et al. 2016). Treatments and clinical observations indicate the importance of early diagnosis of the disease in reducing disability and hospitalization. The course of the disease in the form of attacks, their family and environment according to the definition of the subject creates difficulties in terms of treatment. Particularly, patients and family have difficulties in drug compliance. The use of disease-specific biomarkers in patients with bipolar disorder seems to be of great importance in terms of both the cost and the course of the disease. The presence of specific biomarkers prior to the disease, in acute periods and in the follow-up of the efficacy of the treatment, is important for clinicians and patients. Until this time, many scientific research have been done on this subject from the past to the present. This issue is very current and exciting in our country and the world. Our aim is to share with my collages the current scientific studies in our country and around the world. To raise awareness about our colleagues and to share the excitement of new ideas with them.

[Abstract: 0797] [Others]

Imaging findings in bipolar disorder

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ABSTRACT
Bipolar disorder is a chronical mental disorder which is characterized by recurrent attacks and which significantly disrupts the functionality of the patient. Although there has been a growing number of studies on the neurobiological basis of the disorder, its pathophysiology has not been clearly understood yet. Increasingly in the last decade, brain imaging has been applied to the study of psychiatric disorders and, most notably, to research on schizophrenia and depression, leading to new clues on its cause and treatment. In contrast, bipolar disorder has received relatively less attention. The neuroimaging methods used in psychiatric studies are mostly cross-sectional methods that help the anatomical examination of the tissues. Initially, Computerized Tomography (CT) was used in the studies but currently, mostly Magnetic Resonance Imaging (MRI) is preferred. Recently, the tendency for visualizing the properties of physiological tissues has shifted towards functional imaging methods such as functional Magnetic Resonance Imaging (fMRI), Diffusion Tensor Imaging (DTG), Single-Photon Emission Tomography (SPECT), and Positron Emission Tomography (PET). With the help of these methods, blood flow distribution and metabolism in the brain can be measured indirectly. Structural and functional imaging techniques have significantly contributed to a better understanding of the etiology of bipolar disorder and to the development of a diagnostic approach for it. The most consistently and frequently observed findings include increased white matter hyperintensities, cerebellar structural abnormalities, mild sulcal prominence and ventricular enlargement, and decreased VMPFK cell density with partial mood stabilizing drugs. However, there has been no specific pathophysiologic mechanism to unify these findings yet.

KEYWORDS
Bipolar disorder; neuroimaging; structural and functional imaging

[Abstract: 0802] [Others]

Cognitive functions in bipolar disorder

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ABSTRACT
Bipolar disorder is a chronic, episodic mental disorder with periods of mania, hypomania mixed episodes and depression. Bipolar disorder is associated with high mortality, morbidity and loss of functioning. Recently, there has been a large number of evidence indicating an impairment in cognitive functions like attention, verbal memory, psychomotor and executive functions in bipolar disorder. Recurrent attacks were also associated with progressive cognitive dysfunction and severe cognitive impairment is considered a poor prognosis for bipolar patients. The cognitive impairment may persist in remission periods, even after the end of treatment. Also there are evidences about these cognitive impairments were found even before the onset of the illness and neuro-cognitive deficits were directly related to early onset age and the number of long-term disease hospitalizations. In this presentation cognitive functions in bipolar disorder will be discussed.

KEYWORDS
Bipolar disorder; cognitive dysfunction; remission; functioning

[Abstract: 0803] [Others]

Metacognitions in bipolar disorder

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ABSTRACT
Metacognition is defined as "an orchestra conductor, who appraises, monitors or controls cognitions". It is assumed that people have positive or negative beliefs (metacognitions) of their appraisal of the events they encounter, and such metacognitions are believed to be the underlying process of the maintenance of psychopathology. According to metacognitive theory, psychological disorders are based on toxic cognition mode called "Cognitive Attentional Syndrome" (CAS). CAS consists of rumination and worry, focusing attention on threats and maladaptive coping behavior (thought control strategies, behavioral, cognitive and emotional avoidance). CAS is controlled by person's beliefs (metacognitive beliefs) about thought process. Positive metacognitive beliefs are related to the advantages and benefits (e.g. worrying about future will help me avoid threats) of making cognitive activities that lead to CAS. Negative metacognitive beliefs are related to uncontrollability, importance, meaning and dangerousness of thoughts and cognitive experiences (e.g. worrying will damage my mental health) [1]. In bipolar disorder, which has two opposite poles regarding its clinical phenomenology, it is highly important to investigate the specific metacognitions. Metacognitive beliefs and thought control strategies could thus be phenomena that underlie increased sensitivity to stress, and contribute to emotional symptoms and disorder, including bipolar disorder [2]. Although the cognitive model of unipolar depression is extensively studied, little is known about the distinctive features of the cognitive model of bipolar depression. From the view point of a cognitive behavioural psychotherapist, bipolar depression seems to be very similar to unipolar depression, as observed by previous researchers. Some of these similarities can be summarized as increased rumination, an implicit pessimistic attributional style, low self-esteem, and dysfunctional attitudes towards the self. Patients with unipolar depression and bipolar disorder in a depressed episode have been found to report higher levels of unhelpful metacognitive beliefs than controls, specifically believing thoughts are uncontrollable dangerous, that one needs to control thoughts, being more cognitive self-conscious, and having less confidence in cognitions [2]. In bipolar disorder, research suggests that the number of previous depressive episodes explains negative cognitive styles [3], indicating that number of mood episodes may also play a role in metacognitive processes. Understanding whether – and if so how – metacognitive beliefs or thought control strategies are linked to illness-factors in bipolar disorder could inform further theory development of the disorder.

KEYWORDS
Bipolar Disorder; Metacognitions

References

[1] Wells A. Metacognitive Therapy for Anxiety and Depression. New York: Guilford Press; 2009. p. 1–3.
[2] Batmaz S, Kaymak SU, Kocbiyik S, et al. Metacognitions and emotional schemas: a new cognitive perspective for the distinction between unipolar and bipolar depression. Comprehensive Psychiatry. 2014;55:7:1546–1555.
[3] Reilly-Harrington NA, Alloy LB, Fresco DM, et al. Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. Journal of abnormal psychology 1999;108:4:567.

[Abstract: 0743] [Disruptive behavior disorders]

Approach to management of disruptive behavior associated with ASD and intellectual disabilities that are unresponsive to standard treatments

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ABSTRACT
Youths with autism spectrum disorder (ASD) and other developmental disabilities often display disruptive behaviors, yet few of the available treatments for such behaviors have been effective with this clinical population. Aggression is associated with negative outcomes for children with these children and their caregivers, including decreased quality of life, increased stress levels, and reduced availability of educational and social support. Pharmacologic treatments, particularly the use of second-generation antipsychotics, may also be of some benefit in reducing aggression in those individuals. First-generation antipsychotics, antiepileptic medications, mood stabilizers, and several glutamatergic modulators are also frequently employed for the treatment of ASD and other developmental disabilities-associated aggressive behavior. Development of effective therapeutic and pharmacologic methods for preventing and treating aggression are essential to improving outcomes in this disorder. Rapid titration of clozapine has recently been shown to be safe and effective for the treatment of drug-refractory aggressive behavior. Several case reports demonstrate its safety

KEYWORDS
Aggressive behaviors; clozapine; children; adolescents; autism spectrum disorder; developmental disabilities

and effectiveness in reducing aggression in ASD and other developmental disabilities. Clozapine carries the potential for severe adverse effects, including agranulocytosis. In particular, agranulocytosis is life-threatening and requires frequent blood draws to monitor white blood cell counts. Because blood draws can be especially difficult in highly irritable or aggressive individuals with ASD and other developmental disabilities, clozapine is rarely used in this population. In the present study, the authors examined resistant to common treatment modalities for disruptive behaviors in youths with ASD and other developmental disabilities. For this study, database systems were searched such as YOK thesis data bank, Dergi Park academic, NIH public, ResearchGate, EBSCO, Jstor and ProQuest. Also, author give information on her Bakırköy Mental Health Hospital and Balıklı Rum Hospital experiences of treatment of severe aggressive behaviors resistant to common treatment modalities in individuals with ASD and other developmental disabilities. Findings indicated that clozapine was more effective than polypharmacy use in improving family functioning and ameliorating youth aggressive behaviors in individuals with ASD and other developmental disabilities. A retrospective analysis of 26 children and adolescents' adults with ASDs, aged 14 to 19 years (mean age, 15 years), showed that treatment with clozapine led to decreased aggression, a reduction in the number of psychotropic drugs needed to manage behavior, and a decrease in the dose of concomitantly administered antipsychotic drugs. Clozapine was well tolerated, with no significant reductions in white blood cell count or EPS, although common adverse effects included constipation and weight gain. Only one subject experienced decrease of WBC numbers to 3000 and clozapine treatment needed to discontinue in that patient. Clozapine treatment was associated with reduction in aggressive behaviors and resulted in the reduction of number and dose of concomitant psychotropic medications prescribed. Subjects reported no extrapyramidal side effects, and no cases of agranulocytosis occurred. But despite evidence of effectiveness as a treatment for aggression, particularly to rapidly control symptoms, there have been no controlled studies of clozapine in individuals with developmental disability.

[Abstract: 0646] [Psychopharmacology]

Approach to ADHD patients who are unresponsive to standard treatments

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ABSTRACT

Attention-deficit/ hyperactivity disorder (ADHD) is the most common childhood neurodevelopmental disorder leading to disease burden on indiviuals' lives, families and school system. Most of the patients respond to stimulants such as methylphenidate (MPH) and non-stimulants such as atomoxetine well. However about 30% of patients remain treatment resistant (TR) in which they dont respond well or they experience unwanted side effects. When the patients with ADHD do not respond to MPH, we should consider other medications, including other classes of psychostimulants, the nonstimulant atomoxetine, bupropion, tricyclic antidepressant, alpha 2 adrenergic agents (clonidine, guanfacine) and atypical antipsychotics. Also psychosocial interventions including psychoeducation, social skills training, cognitive behavioral techniques for ADHD, school interventions and mindfullnes for aggression are also added to conventional medical treatment.

If there is a concern of TR, the clinican has to identify the sources of it. Prevention of misdiagnosis, considering adverse effects of the medication and optimizing the treatment are important issues [1]. Accompanying comorbid psychiatric disorders are also major concerns leading to TR cases. Oppositional defiant disorder, conduct disorder and anxiety disorder are the most common psychiatric comorbidities. Children with special conditions such as epilepsy, chronic diseases, preschool aged Preschool aged children, cases with epilepsy, This presentation provides an overview of current knowledge about the clinical and neurobiological factors responsible for TR ADHD cases [2]. This presentation is aimed to address the challenges of treatment resistant ADHD in children with medical and psychiatric comorbidities and special conditions.

KEYWORDS

Attention-deficit/ hyperactivity disorder; clonidine; disruptive behaviors; psychostimulants

References

[1] Barnard-Brak L, Roberts B, Valenzula E. Examining breaks and resistance in medication adherence among adolescents with ADHD as associated with school outcomes. J Atten Dis 2018 Mar 1:1087054718763738.

[2] Shim SH, Yoon HJ, Hahn SW, Kim YK. Clinical and neurobiological factors in the management of treatment refractory Attention-deficit/ hyperactivity disorder. Prog Neuropsychopharmacol Biol Psychiatry 2016;70:237–244.

[Abstract: 0681] [Cultural psychiatry]

"Is it herbal ?" heightened doubt/mistrust as an indicator of treatment resistance in the age of information

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ABSTRACT

Nowadays, an increasing number of people are suspicious of medical interventions, treatments, recommendations, vaccines. In the information age, this can be paradoxical at first place, while information is so easily accessible. However, accessing to any information is facilitated, not just to scientific data, but also to "thrash" information. "Is it herbal" question mainly means less "human hand" which is likely to be evil time to time! It is the declaration saying that the one, the product of the "pure nature" is preferable. Good perception of the nature, liking things "coming from nature" does not mean that people who prefer herbal medicine totally lack the logic. It would be easy to reach such a conclusion. If we go deep down here, one of the things we will find is the concern for technological advances, the news making us even difficult to follow-up in nanotechnology, "the threat" of the artificial intelligence to the existing status of almost all jobs, including medicine. The fact that "the future was unknown, unpredictable" had never been so hard. The increase in the concern of "if it is herbal, OK" "in recent years can be a sign of a defensive attitude, underlining the needs to be protected from this "hurricane". How do these resistances of our patients appear when they are sitting across us in the outpatient room? So, what are the behavioral outcomes of this attitude when we implement our daily routine? Indecisiveness, astonishment, stubbornness, rejection. Is our work hard, yes. Are we angry? No doubt. It is quite hard to manage it and seems to be even more difficult in the future. Because we, as physicians, as well as people who are exposed to these developments, are not different from the patients. Physician-patient relationship may have to be rebuilt. Over the next decade, we will probably learn the results of our biochemistry test, the exercises we have to do, the food we need to eat or avoid, the date of the next appointment, probably from a "machine". Please see the verbal humiliation by saying adjective "machine" we often use while talking about AI's. This might be some kind of scornful, disdainful, debilitating language of a "war" that will break down soon. At this point, "herbal medicine" which is known as the old, which belongs to the tradition, is considered safe. Things are more complicated in terms of psychiatry. İf the prescribed drug is a "chemical" acting on the brain, interrogation and resistance is increasing. A partly unknown "organ" which makes us to be "us", a human being, an individual. We all know that brain gets a quite "unique" and more attention than the kidney or the liver do. Why are painkillers also acting on the brain used so common? Because there is no claim they offer to change "feelings"! This is a presentation not having answers but lots of questions.

KEYWORDS

Herbal treatments; information age; resistance to treatment/therapy; mistrust to medicine; technological advances

[Abstract: 0655] [Non-biological treatments]

Psychotherapeutic approaches with PTSD patients: recent development

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ABSTRACT

Post-traumatic stress disorder (PTSD) is a widespread psychological problem which can come along with traumatic experiences or witnessed to traumatic events. All kinds of uncontrollable traumatic life experiences like natural disasters; earthquakes, hurricane, flood, war or terrorist actions, accidents, rape or any sorts of abuse are the widely known events. As well as straightforward ways to expose a trauma, it is possible to be indirectly exposed. After traumatic experience has occurred, some individuals could not handle with it. For this reason, overwhelming thoughts and feelings such as fear, unhappiness, temper management issues related to event can manifest. In addition to this, some distressful symptoms which accompany to PTSD come alone with flashbacks, nightmares and anxiety, hyperarousal to noise or voice. Related to these symptoms of PTSD, according to DSM-5 defined 4 behavioural criteria for the diagnosis; experiencing or witnessing a stressful life events; re-experiencing of symptoms; avoidance to the reminders of the traumatic situations; and hyperarousal symptoms (APA, 2013; Sareen, 2014). PTSD restrain people's daily life by

KEYWORDS

Trauma; PTSD; Exposure; Psychotherapies

changing their life style in terms of avoiding the traumatic cues. It is quietly possible to change direction of a street which the traffic accident experience has occurred or to give a sudden reaction of noise or to be startled. Besides the availability of medical treatment to PTSD, there are many different psychotherapeutic theories and application with respect to the clients' need. Most of the PTSD psychotherapeutic interventions are based on cognitive behavioural therapy which is scientifically empiric treatment (Schnyder U., 2015). In this article, very common psychotherapeutic approaches such as Cognitive Behavioural Therapy, Exposure Therapies, Eye Movement of Desensitization and Reprocessing (EMDR), Stress Inoculation Training and Group Therapy are mentioned for trauma related disorder treatments (Schnyder, 2015).

References

[1] American Association of Psychiatry. Diagnostic and statistical manual of mental disorders 5th ed. Arlington (VA): American Psychiatric Publishing; 2013.
[2] Sareen J. Posttraumatic Stress Disorder in Adults: Impact, Comorbidity, Risk Factors, and Treatment. The Canadian Journal of Psychiatry 2014;59(9):460–467.
[3] Schnyder UEA. Psychotherapies for PTSD: What do they have in common? European Journal of Psychotraumatology 2015;6(28186).

[Abstract: 0734] [ADHD]

Slow in motion, slow in cognition, slow in organizing, slow in everything; What is sluggish cognitive tempo?

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ABSTRACT

SCT was mentioned for the first time in the 18th century however the first contemporary studies on SCT have started in early 1980s. In 1985 Lahey et al. compared 20 ADD+H children and 20 ADD-H children. ADD-H children showed significantly anxiety, shyness, and withdrawn . Besides this group attracted the attention with the symptoms of somnolency, non-registration to the stimulus and greater sluggish. This group had the symptoms of behavior disorders, disunity with peers and the level of social rejection less. In spite of that ADD+H children was seen as the children with more social problems [1]. In 2001 a review of the literature about these two attention disorders was published by Milich, Balentine and Lyam . The authors broadly reviewed research concerning the subtypes and concluded that ADD-H (ADHD-I Type) was a significant and unrelated disorder to ADD+H (ADHD Combined Type). The authors discussed that a subset of children which characterized as being in the I-Type might be an entirely separate disorder and be best distinguished by their SCT symptoms[2]. In 2001, a factor analysis of 692 children referred to a specialty pediatric clinic for ADHD was conducted by McBurnett et al. found, that SCT symptoms formed a distinct dimension from the two traditional ones including ADHD. The comparison was based on whether the children were high or low in SCT symptoms. Both of the groups had similar levels of inattention and learning new things. But children with SCT had higher levels of anxiety, depression, unhappiness, withdrawn behavior, social dysfunction and less externalizing symptoms. They discussed for usage of SCT symptoms to identify a more homogeneous group of inattentive children who were apart from those having ADHD [3]. Increased request for such empirically-based knowledge is supposedly to occur due to increasing clinical referrals of cases with this condition driven by increased awareness of the general public about SCT. Children whom researchers have labeled as having SCT has no official diagnostic term. There are no official criteria available for its clinical recognition, but researchers have identified the most apparent SCT symptoms [4]. These are: (1) daydreaming, (2) trouble staying awake/ alert, (3) mentally foggy/easily confused, (4) stares a lot, (5) spacey, mind is elsewhere, (6) lethargic, (7) under-active, (8) slow-moving/sluggish, (9) doesn't process questions or explanations accurately, (10) drowsy/sleepy appearance, (11) apathetic/withdrawn, (12) lost in thoughts . These twelve symptoms appear to be immensely useful for making such distinctions. SCT symptoms are considerably but moderately correlated with the ADHD symptom dimensions, especially so for the IN dimension of ADHD. Furthermore, these symptoms identify a distinctive group of children even inclusive of samples that have ADHD I-Type [5]. Recent studies which is made in the past years supports the fact that SCT can be seen with ADHD but a completely separate disorder [5]. SCT, still has not been included as a separate diagnosis in DSM-5, causes disagreements in the literature.

KEYWORDS

ADHD; diagnosis; sluggish cognitive tempo

References

[1] Lahey BB, Schaughency E, Frame CL, Strauss CC. Teacher ratings of attention problems in children experimentally classified as exhibiting attention deficit disorders with and without hyperactivity. Journal of the American Academy of Child Psychiatry. 1985;24: 613–616.
[2] Milich R, Ballentine AC. Lynam ADHD/combined type and ADHD/predominantly inattentive type are distinct and unrelated disorders. Clinical Psychology: Science and Practice. 2001;8: 463–488.
[3] McBurnett K, Pfiffner LJ, Frick PJ. Symptom properties as a function of ADHD type: an argument for continued study of sluggish cognitive tempo. Journal of Abnormal Child Psychology. 2001;29; 207–213.
[4] Barkley RA. Distinguishing sluggish cognitive tempo from ADHD in children and adolescents: executive functioning, impairment, and comorbidity. Journal of Clinical Child and Adolescent Psychology. 2013;42: 161–173.
[5] Marshall SA, Evans SW, Eiraldi RB, Becker SP, Power T J. Social and academic impairment in youth with ADHD, predominantly inattentive type and sluggish cognitive tempo. Journal of Abnormal Child Psychology. 2013;42; 77–90.

[Abstract: 0694] [ADHD]

What is happening in the brain of children with sluggish cognitive tempo? Neurocognitive aspects of sluggish cognitive tempo?

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ABSTRACT

Since its definition in 1980's, Sluggish Cognitive Tempo (SCT) has been subject to many studies. It has been increasingly suggested that SCT can be seen with ADHD but is a distinct disorder. Recent studies have focused on distinguishing SCT from ADHD by investigating neuropsychological and executive functioning, neuroimaging and genetic inheritance of these two clinical condition.

Deficits in executive functions are characteristic of ADHD. Evaluating neuropsychological and executive functions may be beneficial to determine different or common aspects. Because of SCT's phenotypic characteristics, SCT implies a slower rate of cognitive processing. For that reason processing speed and reaction time are expected to be a kind of hallmark for neurocognitive measures related to SCT. Several studies did not find any deficits in processing speed and reported that SCT cases are not slow in fact during their performance in neuropsychological tasks. Although one study reported slower reaction time for SCT, several studies found no significant differences. Children with higher SCT symptom scores present both deficient early selective attention processes and decreased sustained attention. SCT has also been associated with increased variability in spatial memory. Additionally higher SCT symptom scores appeared to be possessing greater deficits in cognitive flexibility. From the clinical perspective, it was reported that deficits in cognitive flexibility and attentional shifting are implied with academic impairment. Consistent with this finding SCT was reported to be associated with academic impairment in the literature.

Recent evidence from neuropsychological studies is contradictory. Some researchers reported that SCT is not strongly related to executive functions in the relevant neuropsychological tests. It has been indicated that ADHD symptoms were more associated with disruptions in executive functions but the relationship of SCT is quite small. From this point of view, some authors suggested that SCT is not a primary disorder of executive functions but may be a disorder of vigilance. Further studies may address this issue.

KEYWORDS

Adolescents; children; neurocognition; sluggish cognitive tempo

References

[1] Becker SP, Burns GL, Leopold DR, et al. Differential impact of trait sluggish cognitive tempo and ADHD inattention in early childhood on adolescent functioning. J Child Psychol Psychiatry. 2018 Oct;59(10):1094–1104.
[2] Baytunca MB, Inci SB, Ipci M, et al. The neurocognitive nature of children with ADHD comorbid sluggish cognitive tempo: Might SCT be a disorder of vigilance? Psychiatry Res. 2018 Dec;270:967–973.
[3] Barkley RA. Distinguishing sluggish cognitive tempo from ADHD in children and adolescents: executive functioning, impairment, and comorbidity. J Clin Child Adolesc Psychol. 2013;42(2):161–173.

[Abstract: 0638] [ADHD]

Biological evidence of a distinct clinical entity: Sluggish Cognitive Tempo?

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ABSTRACT

Despite a voluminous neurobiological literature, the etiology and pathophysiology of Attention Deficit and Hyperactivity Disorder (ADHD) are not completely understood. ADHD is a multifactorial and polygenic disease with high inheritance. Up to now, neuroimaging findings and genetic variants associated with the diagnosis of ADHD can explain a very small proportion of pathogenesis. The most important reason for this is the phenotypic heterogeneity in ADHD cases. Studies to reduce ADHD heterogeneity have increased interest in researching the concept of Sluggish Cognitive Tempo (SCT). The construct of SCT is characterized with daydreaming, mental confusion, staring blankly and hypoactivity. Previous studies showed that SCT is a distinct disorder from ADHD but highly comorbid with it. In the beginning, the majority of SCT studies focused on the distinctiveness of the SCT symptoms and impairment. The growing evidence showed that SCT is a distinct clinical entity in terms of demographics, comorbidities, functional impairments, and cognitive and neuropsychological functioning. After these important results, researchers started to investigate biological factors in relation to SCT. These studies are important to know the underlying mechanisms of SCT and also to decrease inconsistencies in ADHD studies. Genetic differences underlying SCT have evaluated in a twin study and SCT showed less heritability than ADHD and large non-shared environmental influences. ADHD-inattentive, ADHD-hyperactive/impulsive dimensions, and SCT were reported as distinct and partly correlated at the genetic level of analysis. To date, there are only a few studies investigating differences in brain function related to SCT symptoms using functional magnetic resonance imaging (fMRI) in children with ADHD. In the first study, 16 ADHD patients compared with 13 Typically developing controls (TD). They reported that higher SCT symptoms were associated with hypoactivity in the left superior parietal lobe and higher inattention symptoms were associated with altered activity in the supplementary motor area and thalamus. Another neuroimaging study investigating SCT symptoms was conducted in primary schools with 178 children. This study differentiated SCT and ADHD symptoms at the level of cortical and subcortical level. Although the diagnosis of ADHD was associated with altered white matter microstructure, this study was not able to find a significant difference in the white matter microstructure for SCT symptoms. The authors reported that altered cortical structure (especially in the frontal lobe) in the form of increased regional volume anomalies were located at the cortical level only, that point out that the neuroimaging findings of SCT symptoms differ from that in ADHD presenting distinct patterns of etiological factors. Overall, ADHD is a heterogeneous disorder with various clinic presentations, impairment domains, comorbidities, and biological traits. ADHD studies are aiming to reduce this heterogeneity by controlling comorbidities. Future studies may benefit for also including an assessment for SCT. We also need more studies to understand the differences between SCT and inattention. These results have importance for defining appropriate therapeutic and educational services specific to these different phenotypes.

KEYWORDS

Sluggish cognitive tempo; genetics; neuroimaging; biomarker

References

[1] Becker, S. P., Leopold, D. R., Burns, G. L., Jarrett, M. A., Langberg, J. M., Marshall, S. A., … Willcutt, E. G. (2016). The Internal, External, and Diagnostic Validity of Sluggish Cognitive Tempo: A Meta-Analysis and Critical Review. J Am Acad Child Adolesc Psychiatry, 55(3), 163–178.

[2] Camprodon-Rosanas E, Pujol J, Martínez-Vilavella G. Brain Structure and Function in School-Age Children With Sluggish Cognitive Tempo Symptoms. J Am Acad Child Adolesc Psychiatry. 2018;58

[3] Fassbender C, Krafft CE, Schweitzer JB. Differentiating SCT and inattentive symptoms in ADHD using fMRI measures of cognitive control. Neuroimage Clin. 2015;8:390–397

[Abstract: 0735] [ADHD]

Ongoing debates of sluggish cognitive tempo and possible treatment options

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ABSTRACT

In the last years, there is a growing interest in a constellation of symptoms that is known as Sluggish Cognitive Tempo (SCT) in child psychopathology. Though, the precise definition of which symptoms compose the syndrome is still not clear. SCT first presents in childhood and is an impairment of attention in daydreamy, hypoactive-appearing individuals. Even though the symptom dimensions forming SCT are distinct from Attention Deficit and Hyperactivity Disorder (ADHD) especially its inattention dimension partially correlated with ADHD [1]. SCT is described as significant impairment, most reliably in social impairment. Primarily social withdrawal is a well-known problem among patients diagnosed with SCT which affects individuals' daily life. It also makes some contribution to difficulties with academic performance in children. It is also shown that SCT significantly associated with risk for internalizing symptoms, especially depression and anxiety disorders.

The etiologies of SCT are not well understood, but some evidence suggests a strong heritability to the disorder. Evidence supports the view that SCT is distinct from ADHD, but the two conditions can overlap in nearly half of all cases [1]. As with the etiology of SCT only a few studies have investigated possible treatments. Early studies on stimulants for treating ADHD inattentive type did not find them to be particularly effective in improving the inattention linked to SCT. In a double blind placebo controlled trail, it was found modest positive response to MPH, mainly at low doses [2]. Unfortunately no stimulant medication studies have been done specifically with SCT. A recent research reported that higher SCT symptoms predicted a poorer response to MPH [3].

To date, only one study has examined a nonstimulant ADHD medication for treating SCT symptoms specifically [4]. Atomoxetine was effective at reducing SCT symptoms in patients having both ADHD and dyslexia, ADHD only, and dyslexia only. In this session, considering overlapping symptom dimensions with ADHD and comorbidities, it is aimed to discuss possible treatment options of SCT along with ongoing debates in diagnosing this distinct disorder.

KEYWORDS

ADHD; comorbidity; sluggish cognitive tempo; symptom dimensions; treatment

References

[1] Barkley Sluggish Cognitive Tempo Scale—Children and Adolescents (BSCTS-CA). Russell A. Barkley. Copyright © 2018. Guilford Publications.

[2] Barkley RA, DuPaul GJ, McMurray MB. Attention deficit disorder with and without hyperactivity: clinical response to three dose levels of methylphenidate. Pediatrics, 1991;87(4):519–531.

[3] Froehlich TE, Becker SP, Nick TG, Brinkman WB, Stein MA, Peugh J, Epstein JN. Sluggish Cognitive Tempo as a Possible Predictor of Methylphenidate Response in Children With ADHD: A Randomized Controlled Trial. J Clin Psychiatry, 2018;79(2). pii: 17m11553.

[4] Wietecha L, Williams D, Shaywitz S, Shaywitz B, Hooper SR, Wigal SB, Dunn D, McBurnett K. Atomoxetine improved attention in children and adolescents with attention-deficit/hyperactivity disorder and dyslexia in a 16 week, acute, randomized, double-blind trial. J Child Adolesc Psychopharmacol. 2013; 23 (9):605–613.

[Abstract: 0663] [Autism]

Feature selection and classification on application log data as machine learning procedure of emotion recognition Processes of ADHD, ASD, and healthy controls

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ABSTRACT

In this work we focused on classification of the participants with Attention-deficit/ hyperactivity disorder (ADHD), Autism Spectrum Disorder (ASD) and typically developing children, according to their performance during an emotion recognition experiment that we developed. We prepared an experiment environment in which, participants were shown images of faces of people exhibiting certain emotions up to a certain strength and then they answered the question "what is the emotion of this person?'". The response and response latency of the participants were recorded and used for the classification process. Relief feature selection algorithm was used in order to select the relevant images which are used as features in this study. Observation of the eye track fields were determined between the study groups to calculate the difference between emotion recognition process. Machine learning feature selection and classification algorithms were used on different definitions of the classification problem where the differentiation between two classes against each other or one class

KEYWORDS

Relief feature selection; human-computer interaction; classification; emotion recognition; Autism; ADHD

against the other two classes were aimed. This study was executed in Marmara University School of Medicine Hospital Child and Adolescent Psychiatry Outpatient Department. In our dataset there were 18 participants with ASD, 30 participants with ADHD and 13 participants in control group. Average age of each group is 10.5, 9.46, and 9.22 respectively. The patients are recruited from the outpatient clinic referrals and randomly assigned. ADHD diagnosis is based on Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime Version (K-SADS-PL). Consequently, by doing data analysis; we could separate ADHD, ASD and control groups from each other ($p < 0.5$). Selecting relevant and informative features increased the classification accuracy. Also there was a better classification performance resulting from group redefinition. The most accurate result was 80% for "others" group definition, on response correct data. It is achieved by selecting 15 features and implementing KNN algorithm. On the contrary, if feature selection processes were not applied; the accuracy decreased to 68.89%. Higher degrees of performance are obtained for the ASD- others group on response time data. Namely we could distinguish participants with ASD from the other groups which included participants with ADHD and controls. Also by 90% accuracy; we could differentiate participants with ADHD from participants with ASD by using Adaboost algorithm with and without feature selection process on response time data. In addition to this, on RC&RT data ASD and ADHD participants can be separated by 80% accuracy.

References

[1] First MB. "Diagnostic and statistical manual of mental disorders," DSM IV-4th edition. APA, p. 1994, 1994.
[2] Kanade T, Cohn JF, Tian Y. "Comprehensive database for facial expression analysis," in Automatic Face and Gesture Recognition, 2000. Proceedings. Fourth IEEE International Conference on. IEEE, 2000, pp. 46–53.
[3] Kononenko I, ˇSimec E, Robnik-ˇ Sikonja M. Overcoming the myopia of inductive learning algorithms with RELIEFF. Applied Intelligence 1997;7 (1):39–55.

[Abstract: 0616] [ADHD]

Attention problems of children with ADHD in classroom Environment

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ABSTRACT
The assessment of attention performance and executive functions in individuals with ADHD can be done with various neuropsychological tests and scales, but also by eye tracking technique which is an objective measurement tool [1]. It is important to assess the performances of these individuals in the areas of important functionality in daily life activities as well as in various tasks [2]. The aim of this study is to show the attention performance and executive function deficits of children with ADHD as close as possible to real time(in classroom) by eye tracking method. Our study consisted of 30 healthy control subjects and 30 children with ADHD. Development and Well-Being Assessment (DAWBA) and Conners parent and teacher rating scales were applied and DSM-V based clinical interviews were conducted. A video task consisting of lesson content was shown to all participants and their eye tracking parameters (dwell time, fixation duration, fixation counts, first fixation duration and average fixation duration) were measured with eye tracking device. These parameters represent bias in sustainable attention and sustainable attention. On the task, 5 areas of interest were identified: the area of the distractor, the teacher's area, the board area, the area where the other students were located, and the area consist of outside of the other areas. The parameters of eye tracking were analyzed separately on each region of interest and compared with statistical methods among groups. ADHD group had shorter first and average fixation periods in the areas that were expected to be focused in general during the course flow (teacher, board); and had longer dwell time, fixation duration and more fixation counts in the unrelated areas of course content(distractor, students, other area). Distractors have different effects on the attention performance of the groups according to their area (within / outside the learning area). The distractor within the learning area affected the attention performance positively(increase in dwell time, fixation duration and average fixation duration in the teacher area) in the ADHD group ($p = 0.02$, $p = 0.018$, $p = 0.003$). A negative effect (decrease in dwell time and fixation duration in the board area) was observed in the control group ($p = 0.037$, $p = 0.036$). The distractor outside the learning area had a negative effect (decrease in dwell time and fixation duration in the teacher area, increase in first fixation duration in the distractor area and increase in average fixation duration in the other area) on the attention performance of the ADHD group ($p = 0.00$, $p = 0.00$, $p = 0.039$, $p = 0.041$). In the

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ADHD; Eye tracking; Attention performances; Executive functions

control group, a significant increase in the average fixation duration was observed in the teacher area ($p = 0.22$). Our study showed that eye tracking technique is a very informative method in the real-time evaluation of attention performance and executive function of individuals with ADHD. Further research is needed that evaluating the eye tracking technique in terms of prediction of ADHD diagnosis, ability to assess the effectiveness of treatment interventions.

References

[1] Gioia GA, et al. Test review behavior rating inventory of executive function. Child Neuropsychology. 2000;6 (3):235–238.

[2] Barkley RA. ADHD and the nature of self-control. Guilford Press; 1997.

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Change blindness in children diagnosed with ADHD

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ABSTRACT

Attention-deficit/ hyperactivity disorder (ADHD) is one of the most common neuropsychiatric disorder in childhood. Although it has been described as a clinical condition for many years, its etiology is still unclear. However it is commonly considered to develop as a result of interactions between genetic and environmental multifactorial factors. It was demonstrated in the studies that ADHD is associated with several difficulties in cognitive functions. Researches that evaluated visual memory have been showed that observers do not often detect the large and repeated changes in a scene from one viewpoint to the next. In common real world situations, a change in a scene creates a signal and it provides an automatic and internal cue for the subject. However, when the change occurs during an event (e.g. an eye movement or an eye blink or a blank screen), people almost never notice the change. This inability to detect changes of an object or a scene is known as change blindness. It has been argued that change blindness phenomenon is commonly related to attentional mechanisms, visual perception and visual working memory. It is emphasized that focused attention is necessary in order to detect changes. When there is no focused attention, the visual memory cannot make comparison between scenes (original and changing scenes), and the change cannot be detected [1].

In studies evaluating change blindness, different methods are used to create change. For example, during eye movements or blinking, or a blank screen enters between two scene, or by interrupting a movie, or an event in real life. Several studies using eye tracking methodology to investigate inhibition control in ADHD argued that children diagnosed with ADHD tend to show deficits in voluntary eye movement control and poor inhibition of saccades. Neuroimaging studies claimed that there is a strong relationship between the oculomotor and attentional brain systems, and impairments in saccadic control were expected to result in visual attentional deficits. Therefore, when a task requires shifting the attention from the high-level interest area (central interest region) to the low-level interest (marginal interest region), children diagnosed with ADHD might have more difficulty due to their voluntary eye movement control and attention shifting mechanisms while detecting changes in the marginal interest regions which might result in higher accuracy differences between central and marginal changes. The results of the interactions between cognitive processes and oculomotor behavior suggested that it is important to investigate the visual scan paths of children diagnosed with ADHD with eye tracking equipment during visual discrimination tasks that require systematic search strategies like change detection [2,3]. In this symposium, the results of our study evaluating change blindness in children diagnosed with ADHD will be discussed in the light of the literature.

KEYWORDS

Attention-deficit/ hyperactivity disorder; change blindness; child; eye tracking

References

[1] Rensink RA, O'Regan JK, Clark JJ. To see or not to see: The need for attention to perceive changes in scenes. Psychological science. 1997;8(5):368–373.

[2] Cohen AL. Performance on the flicker task and Conners CPT in children with ADHD. Auburn: Auburn University; 2009.

[3] Sonuga-Barke EJ, Elgie S, Hall M. More to ADHD than meets the eye: observable abnormalities in search behaviour do not account for performance deficits on a discrimination task. Behavioral and brain functions. 2005;1(1):1.