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The effects of atomoxetine on weight, height, and body mass index in Turkish children and adolescents with attention deficit hyperactivity disorder

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ABSTRACT

Background: We aimed to examine the long-term effects of atomoxetine on height, weight, and body mass index in Turkish children and adolescents with attention deficit hyperactivity disorder (ADHD).

Methods: Participants (6–18 years, 146 boys, 52 girls) with ADHD who used atomoxetine for at least 1 year were included in a retrospective study. Weight, height, and BMI *z* scores were converted to age- and gender- corrected *z* scores at baseline and last follow-up.

Results: Atomoxetine treatment was associated with a notional reduction in height and weight standard deviation scores (SDS). There were no differences in BMI-SDS before and after atomoxetine treatment. Results of multiple linear regression analysis assess the possible contribution of the different treatment-related factors, age starting treatment, and duration of treatment predicted final height. And also, only the duration of treatment predicted final weight, not final height and BMI.

Conclusions: We conclude that atomoxetine shows a negative effect on height and weight in children. This study demonstrated that these findings obtained at the end of the study might be helpful in assessing the growth parameters that may facilitate the course of the ADHD.

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Atomoxetine; attention-deficit hyperactivity disorder; height; weight; BMI; growth

Introduction

Attention deficit hyperactivity disorder (ADHD) is defined as a neurodevelopmental disorder that reflects the persistence of ADHD symptoms such as inattention, overactivity, and impulsivity across lifespan [1]. Atomoxetine (Atx) is the first non-stimulant drug approved by the FDA in the treatment of ADHD. In recent years, it has been suggested that atomoxetine may have a role in the treatment of ADHD with an increasing understanding of the importance of noradernal mechanism in the aetiology of ADHD [2]. The most common side effects associated with atomoxetine include dry mouth (16–55%), decreased appetite (12–50%), insomnia (17–35%), irritability (35%), constipation (7–20%), nausea (12–40%), dizziness (6–15%), and fatigue (16–25%) [3].

The pathophysiological mechanisms underlying the loss of appetite observed due to atomoxetine use and the associated weight loss or the discontinuation of an expected weight gain are not yet known. The effects of atomoxetine on appetite are not known exactly, but different opinions have been proposed. The most emphasized of these opinions are abdominal pain and nausea which are seen in the early stages of treatment of food rejection and decreased appetite [4,5]. According to another opinion, the effect of atomoxetine on the central noradrenergic system causes a temporary decrease in appetite [6]. Wilens et al. showed that the weight and height averages of the patients using atomoxetine reached the expected level after 2 years. In the first 3 months of the treatment, the mean weight decreased by 0.3 kg, but the weight returned to normal after a 24-month treatment [7]. In a study by Kratochvil et al., it was determined that growth curves returned to their initial values after 2 years [8]. Although the literature is controversial, delay or retardation in growth is a common problem in the treatment of children with ADHD, whose growth percentages may be already low, but the effects of drugs used on height and weight gain remain unclear.

Thus, the goal of this study was to investigate the long-term effects of atomoxetine on height, weight, and body mass index in Turkish children and adolescents with ADHD. In this context, we hypothesized that (I) the atomoxetine would show a negative effect on weight, height, and body mass index standard *z* scores (II) and that the different treatment-related factors were associated with the final growth parameters.

Methods

For this study, we surveyed the medical records of 198 children and adolescents (6–18 years) with ADHD who received treatment with atomoxetine from March

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2012 to January 2019 at the Department of Child and Adolescent Psychiatry at Dokuz Eylül University Medical School. All children included in this study received treatment with atomoxetine for at least 1 year and met DSM-IV diagnosis criteria for ADHD. Exclusion criteria included via medical records (I) positive history of diseases that can repress growth, (II) past and/or current history of autism spectrum disorder and mental retardation, (III) past and/or current history of epilepsy, brain injury and cerebral palsy, and (IV) use of chronic medications which could affect growth (e.g. cortisol, stimulants, mood stabilizers). This led to the exclusion of 33 patients (3 children who were younger than 6 years in the baseline assessment, 4 adolescents who were older than 18 years in the follow-up assessment, and 26 children and adolescents who have exclusion criteria) out of the initial 231 participants and the final sample was 198 patients. Weight, height, and BMI *z* scores were converted to age- and gender-corrected *z* scores using norms from the Turkish Population [9] at baseline and last follow-up.

Differences in all study variables were analysed using the Statistical Package for the Social Sciences (IBM, NY), version 22 for Windows. Before the statistical analysis was performed, it was checked whether the data met the assumptions of the parametric tests and the normal distribution and homogeneity of variance by using the Kolmogorov Smirnov test. In the interpretation of the variables, descriptive statistical techniques and quantitative data analysis were used. We analysed *z* scores before and after treatment with the paired Student *t*-test. Pearson correlation test was used to determine the direction and level of correlation between the variables, and the results were indicated by "*r*" (correlation coefficient) and "*p*" value (significance level). Linear regression analyses were used to analyse three factors, including age starting treatment, dosage of atomoxetine, and duration of treatment with height, weight, and BMI-SDS *z* scores as the dependent variable. $P < .05$ was considered statistically significant.

The Dokuz Eylul University Ethics Committee approved the study (date: 04.01.2018, number: 2018/01-13).

Results

Descriptive data

Table 1 shows the characteristics of the sample. Patients were 9.57 (2.58) years old when they started treatment. One hundred forty-seven (74.24%) were children and 51 (25.76%) adolescents. One hundred fifty (75.76%) were males and 48 (24.24%) were females. All of them were treated with atomoxetine. Mean dose was 29.81 (±13.29) mg/d or 1.25 (±0.54) mg/(kg/d). Median values and interquartile ranges (IQR, 25th–75th percentile) for the duration of atomoxetine treatment (months) are summarized in Table 1.

Table 1. Result of sociodemographic data and clinical assessment.

Variable	ADHD ($n = 198$)
Age, *M* (SD)	9.57 (2.58)
Age group	
Children: 6–12 years, *n* (%)	147 (74.24%)
Adolescents: 13–18 years, *n* (%)	51 (25.76%)
Sex	
Males, *n* (%)	150 (75.76%)
Females, *n* (%)	48 (24.24%)
Treatment: Atomoxetine	
Dose (mg/d) (SD)	29.81 (13.29)
Dose [mg/(kg/d)] (SD)	1.25 (0.54)
Duration (months) Md (IQR, 25th and 75th percentile)	25 (15.75–32)

Note: ADHD, Attention Deficit Hyperactivity Disorder; *M*, Mean; (SD), standard deviation; *n*, number of patients; Md, median; IQR, interquartile range.

Baseline data

Before starting treatment, weight-SDS (baseline weight-SDS [SDS]: 0.33 [1.25], $t = 2.89$; $p = .005$) and height-SDS (baseline height-SDS [SDS]: 0.61 [1.41], $t = 4.69$; $p < .001$) were above the average, and these differences were statistically significant. There were no differences in BMI-SDS (baseline BMI-SDS [SDS]: −0.02 [1.18], $t = -0.21$; $p = .836$) at baseline.

Atomoxetine and weight, height, and BMI by age

Considering the whole group, weight-SDS significantly decreased at follow-up (baseline weight-SDS [SDS] 0.33 [1.25], follow-up: 0.32 [0.68]; $p^{**} < .001$). Height-SDS [SDS] is also affected: 0.61 [1.41] at baseline and 0.43 (0.82) at follow-up, $p^{**} < .001$. There were no differences in BMI-SDS [SDS] before and after atomoxetine treatment: baseline −0.02 [1.18], follow-up: 0.09 (0.75), $p = .353$. The effect of ATX on growth is described in detail in Table 2, including pre and postdata of weight, height, and BMI *z* scores are shown.

However, whether patients were children (6–12 years) or adolescents (13–18 years) when they started medication was considered. In the group of children, height was slightly affected by treatment (baseline height-SDS [SDS]: 0.72 [1.40]; follow-up height-SDS [SDS]: 0.54 [0.84]; $p^{**} < .001$). This effect was not observed if atomoxetine was started during adolescence (baseline height-SDS [SDS]: −0.14 [1.28], follow-up height-SDS [SDS]:−0.14 [0.44]; $p = .191$).

In the group of children, weight was affected by treatment (baseline weight-SDS [SDS]: 0.43 [1.23]; follow-up weight-SDS [SDS]: 0.39 [0.67]; $p^{**} < .001$). This effect was not observed if Atx was started during adolescence (baseline weight-SDS [SDS]: −0.20 [1.26], follow-up weight-SDS [SDS]:−0.08 [0.55]; $p = .551$).

In the group of children, BMI was not slightly affected by treatment (baseline BMI-SDS [SDS]: 0.01 [1.18]; follow-up BMI-SDS [SDS]: 0.09 [0.79]; *p*

Table 2. Comparison of height, weight, and BMI at baseline and follow up data of atomoxetine treatment by age.

	Total sample ($n = 198$)			Children (6–12 years) ($n = 147$)			Adolescents (13–18 years) ($n = 51$)		
	Baseline (T1)	Follow-up (T2)	p	Baseline (T1)	Follow-up (T2)	p	Baseline (T1)	Follow-up (T2)	p
Weight-SDS	0.33 (1.25)	0.32 (0.68)	<.001**	0.43 (1.23)	0.39 (0.67)	<.001**	-0.20 (1.26)	−0.08 (0.55)	.551
Height-SDS	0.61 (1.41)	0.43 (0.82)	<.001**	0.72 (1.40)	0.54 (0.84)	<.001**	-0.14 (1.28)	−0.14 (0.44)	.191
BMI-SDS	−0.02 (1.18)	0.09 (0.75)	.212	0.01 (1.18)	0.09 (0.79)	.252	-0.22 (1.25)	0.07 (0.55)	.611

Note: n, number of patients; SDS, standard deviation score; BMI, body mass index; *$p < .05$, **$p < .01$. p values from paired t-tests.

Table 3. Comparison of height, weight, and BMI at baseline and follow up data of atomoxetine treatment by gender.

	Girls ($n = 48$)			Boys ($n = 150$)		
	Baseline (T1)	Follow-up (T2)	p	Baseline (T1)	Follow-up (T2)	p
Height-SDS	0.62 (0.81)	0.43 (0.88)	.032	0.51 (1.40)	0.36 (0.77)	<.001**
Weight-SDS	−0.03 (0.92)	0.29 (0.59)	.034	0.35 (1.20)	0.27 (0.65)	<.001**
BMI-SDS	−0.46 (1.03)	0.11 (0.60)	.409	0.07 (1.10)	0.07 (0.72)	.307

Note: n, number of patients; SDS, standard deviation score; BMI, body mass index; *$p < .05$, **$p < .01$. p values from paired t-tests.

= .252). Also, this effect was not observed if atomoxetine was started during adolescence (baseline BMI-SDS [SDS]: −0.22 [1.25], follow-up BMI-SDS [SDS]: 0.07 [0.55]; $p = .611$) (Table 2).

Atomoxetine and weight, height, and BMI by sex

When the groups were divided into girls ($n = 48$) and boys ($n = 150$), girls showed height before starting treatment ($p = .002$), BMI ($p = .049$) and these differences were statistically significant.

Before starting treatment, the group of boys showed height ($p^{**} < .001$), weight ($p^{*} < .05$) and also these differences were statistically significant.

However, considering whether patients were girls or boys, in the group of girls, height (baseline height-SDS [SDS]: 0.62 [0.81]; follow-up height-SDS [SDS]: 0.43 [0,88]; $p^{*} < .05$) and weight (baseline weight-SDS [SDS]: −0.03 [0.92]; follow-up weight-SDS [SDS]: 0.29 [0,59]; $p^{*} < .05$) were slightly affected by treatment. These effects were observed in the group of boys also. In such cases, height (baseline height-SDS [SDS]: 0.51 [1.40]; follow-up height-SDS [SDS]: 0.36 [0,77]; $p^{**} < .001$) and weight (baseline weight-SDS [SDS]: 0.35 [1.20]; follow-up weight-SDS [SDS]: 0.27 [0,65]; $p^{**} < .001$) were slightly affected by treatment (Table 3).

Treatment-related factors and growth

Table 4 shows the correlations between age starting treatment, the dosage of Atx, and duration of treatment and growth parameters. There was a significant correlation between the dosage of Atx and differences in height-SDS between baseline and follow-up ($R = −0.192$, $p^{*} < .05$). The BMI-SDS did not correlate significantly with age starting treatment, but the younger the age of starting treatment, the higher the difference between baseline and follow-up height ($R = −0.393$, $p^{**} < .001$) and weight ($R = −0.227$, $p^{*} < .05$). On the other hand, BMI-SDS did not correlate significantly with duration of treatment, but the longer the duration of treatment, the higher the difference between baseline and follow-up height ($R = 0.286$, $p^{*} < .05$) and weight ($R = 0.215$, $p^{*} < .05$). Correlation analysis and the variance inflation factors (VIF) between age at starting treatment and duration of treatment show that may not be problematic (VIF score = 1.12).

The multiple linear regression models were used to evaluate the possible confounding factors as the independent variables (gender, age starting treatment, dosage of Atx, and duration of treatment) that might predict final height (adjust $R^2 = 0.191$), weight (adjust $R^2 = 0.057$), and BMI (adjust $R^2 = 0.013$). According to this regression analysis, age starting treatment ($B = −0.102$, −0.161 to −0.042, $p = .001$) and duration of treatment ($B = 0.017$, 0.006–0.028, $p = .003$) but not higher doses and sex predicted final height. And also, only the duration of treatment, but not higher doses, sex, and age starting treatment predicted final weight, not final height and BMI ($B = 0.011$, 0.001–0.021, $p = .033$) (Table 5).

Discussion

The findings of the current study represent the effects of long-term treatment with atomoxetine on height, weight, and body mass index of Turkish children and adolescents with ADHD. The effects of ADHD

Table 4. Results of the correlation model between growth parameters and treatment-related factors.

Correlations	Dif-Height	Dif-Weight	Dif-BMI
Age starting Atx-SDS	−0.393 (p < .001**)	−0.227 (p* < .05)	0.083 (0.371)
Dosage of Atx [mg]-SDS	−0.192 (p < .05*)	−0.088 (p = .342)	0.077 (0.406)
Duration of treatment-SDS	0.286 (p < .05*)	0.215 (p* < .05)	0.060 (0.517)

Note: BMI, body mass index; Atx, atomoxetine; SDS, standard deviation score. Dif-BMI, difference between baseline and follow-up BMI, in SDS; Dif-Height, difference between baseline and follow-up height, in SDS; Dif-Weight, difference between baseline and follow-up weight, in SDS. *$p < .05$, **$p < .01$. p values from Pearson correlation test.

Table 5. Results of the multiple linear regression analysis model between the growth parameters and treatment-related factors.

	Height			Weight			BMI		
Multiple linear regression	*B*	95% CI	*p*	*B*	95% CI	*p*	*B*	95% CI	*p*
Age starting Atx	−0.102	−0.161 to −0.042	.001	−0.048	−0.100–0.005	.074	0.022	−0.039–0.083	.470
Dose of Atx [mg]	−0.006	−0.018–0.005	.288	−0.002	−0.012–0.008	.667	0.002	−0.010–0.014	.728
Duration of treatment	0.017	0.006–0.028	.003	0.011	0.001–0.021	.033	0.004	−0.008–0.015	.511
Sex	0.056	−0.295–0.406	.753	0.067	−0.242–0.376	.670	−0.018	−0.377–0.341	.921

Note: BMI, body mass index; Atx, atomoxetine; SDS, standard deviation score; CI, confidence interval; *$p < .05$, **$p < .01$. *p* values from multiple linear regression test.

treatment on growth still remain controversial. Some long-term follow-up studies are associated with significant growth suppression during treatment [8,10–12] while others do not find significant alterations [7,13,14]. At baseline, height SDS and weight SDS *z* scores were higher than expected in our study which is not coherent with the studies suggesting that patients with ADHD show physical growth delay intrinsic to the disorder [15,16]. According to the "maturational delay" in ADHD, a delay in prefrontal cortical regions has been described [17,18] if the same condition was not associated with an intrinsic delay of physical development. Our results are coherent with other studies which compared the prevalence of higher body weight or body mass index in children and adolescents with ADHD [19,20]. Our hypothesis in these differences could be explained by comorbidities. Some studies suggested an association between overweight and obesity with comorbidities such as mental retardation, adjustment disorders, and oppositional deficit disorder [19,21]. Therefore, mental retardation is a common exclusion criterion from the study, but the other comorbidities have not examined because of the retrospective design. It is unclear if and in what dimension comorbidities have an influence on weight problems in examined patients with ADHD.

A significant decline between baseline and follow-up weight and the significant decrease in the *z*-score for height were observed in the total sample, but the decline was not observed in all subgroups supporting the current literature that has evaluated the clinical importance of reduced parameters [22]. Several mechanisms explain the reason of Atx on height and weight. Nausea and somnolence are frequently reported during the early stages of treatment with atomoxetine, and it was possible that there may be a situation which makes the patients to refuse to eat. Alternatively, atomoxetine could have directly affected neuroendocrine systems involved with the inhibition of growth hormone. Another possible explanation is parental height values which influence the growth speed. When making a clinical assessment of an individual child, exact measurements are of great importance, but clinicians have to rely on at least one reported parental height or weight. This area requires further studies which can be evaluated in terms of these explanations.

Our study focused on the effect of atomoxetine in the different age subgroups who are children (6–12 years) and adolescents (13–18 years). In the children subgroup, height and weight become reduced significantly by treatment. Our results coherent with the findings of another study by Kratochvil et al. [8] determined that atomoxetine treatment resulted in a decrease in the growth curves of the patients in the early period, but the growth curves returned to their initial values after 2 years [8]. Despite the central noradrenergic system immaturity, the putative loss of appetite could have been due to a temporary perturbation of these systems involved in hunger or satiety [6]. Patients who are children when they use atomoxetine should be monitored closely based on these results.

Few studies have examined the relationships of the age of onset, duration, and growth parameters of the ADHD sample. The present study suggests that younger age at first atomoxetine order was associated with a decline in anthropometric scores. Our results of significant delay in the rate of physical growth do not support the findings of the study by Gustafsson et al. [23] which suggest that children with ADHD are less mature at baseline as they show a maturation catch-up rapidly [23]. The possible reasons for findings may be nutritional status or hormonal mechanisms which can modify the relationship between physical growth and development. Due to the earlier age of atomoxetine use and the long treatment episode, the drug may have a more permanent effect on the nutritional status or hormonal mechanisms.

We recorded no effect of Atx dosage (mg/day) on differences in all *z*-scores between baseline and follow-up; but the literature is incoherent and it is unknown whether these effects are potential dose-dependent growth effects. These findings along with no correlations between all *z* scores, especially BMI-SDS and atomoxetine dosage, suggest evaluation of suppression of appetite in children taking atomoxetine. Accounting for the mechanisms suggested by Cortese et al. [24], BMI may be an indicator of ADHD symptoms [24]. An important bias here is that the reduction in BMI-SDS may be associated with a reduction of ADHD symptoms.

There have been limited researches into the association between boys and girls growth with ADHD. In the current study, our sample, which included 150

boys and 48 girls with ADHD, had a 3.13:1 ratio. There was a significant effect between only boys and differences in height-SDS *z*-scores and weight-SDS *z*-scores between baseline and follow-up, but in the regression analysis, gender did not have an effect on predicted difference and final anthropometric values. Our results were coherent with the findings of the study by Kweon et al. [22] which did not find interaction between boys and girls in the effect of Atx on growth [22]. Also Biederman et al. [25] showed the effect of emotional disorders on *z*-scores which are greater weight in girls and with smaller height in boys [25]. In our study, this effect was not evaluated by researchers.

Several mechanisms have been proposed to explain these findings, but we suggested that atomoxetine may affect the pattern of delay in the rate of physical growth. Therefore, no statements could be made on the influence of gender on weight and height change in the ADHD sample.

In our study, a statistically significant positive correlation was found between age starting Atx and differences in weight-SDS and height-SDS between baseline and follow-up, duration of treatment and differences in weight-SDS and height-SDS, also the dosage of Atx and differences in height-SDS between baseline and follow-up. A possible explanation of these results may be associated with the duration of ATX treatment. The use of atomoxetine in the earlier age may indicate the severity of impairment in functionality as well as increasing the chronological age and weight of the cases will increase drug use doses. These three factors that interact with each other may have a linear effect on growth. The results of multiple linear regression analysis which examine the possible contribution of the different treatment-related factors, only age starting treatment, but not higher doses and sex predicted final height, not final weight and BMI. And also, the duration of treatment predicted final height and final weight, not BMI. Nevertheless, in most previous studies, there has been no difference between the effect of atomoxetine and target height reduction in children and adolescent with ADHD [11,26]. On the other hand, these results are not coherent with previous studies that concluded that Atx may be associated with the reduction of growth velocity and target height [22,27]. In contrast to the studies mentioned above, the reason of controversial results could be due to differences in study designs including those of retrospective or prospective, self-reported evaluation, ADHD subtypes, phone interviews, other medication use, comorbidities, etc.

Limitations

When considering the results of the current study, some limitations need to be taken into account. First, our study was a retrospective chart review. Participants with no clinically diagnosed possible conditions associated with growth retardation were selected for the study sample, but there may have been differences between health conditions of the participants, which may affect the anthropometric values. Secondly, we did not consider past treatment regimes which are very important to assess if patients were taking stimulants before atomoxetine. An alternative possible limitation is that we do not evaluate the parental anthropometric values, socioeconomic status, ethnicity, and genetic factors of the ADHD group, which have an effect on our findings. Moreover, we did not consider if comorbid features, such as mood disorders, other neurodevelopmental disorders, or eating disorders, can be associated with *z* scores' variations. Another issue of concern is defining other psychopharmacological medications which also influence growth parameters; the impact of this bias is unknown. Another limitation is the availability of chart records which include changes in growth parameters in monthly or three-month intervals with longitudinal growth curves.

Additionally, the pattern of the current study prevents any continuous explications considering the intercourse between Atx and growth parameters. Prospective and longitudinal studies would allow for a deeper understanding of whether and how complexities on growth parameters subscribe to the improvement and preservation of the ADHD.

Conclusion

The current study evaluated the long-term effects of atomoxetine on height, weight, and body mass index in Turkish children and adolescents with ADHD. The results of this study may have clinical variables in the follow-up of growth parameters in ADHD patients. Although the literature is controversial, our findings concluded that Atx may be associated with the reduction of growth velocity, target height, and target weight. It was thought that the findings obtained at the end of the study might be helpful in assessing the growth parameters that may facilitate the course of the ADHD, and in the improvement of more efficient and permanent treatment approaches, and the adherence of patients to the treatment.

Disclosure statement

No potential conflict of interest was reported by the authors.

Key points

- The evaluation of the long-term effects of atomoxetine on height, weight, and body mass index has been found to be critical in terms of both the aetiology and clinical course of ADHD.

• Atomoxetine may be associated with the reduction of growth velocity, target height, and target weight.

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