

Original Article

Evaluation of statural growth and puberty of children and adolescents with overweight and obesity

Avaliação do crescimento estatural e da puberdade de crianças e adolescentes com sobrepeso e obesidade

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ABSTRACT: Objectives: In females, being overweight is associated with the anticipation of puberty. However, in males, few studies have been done and they are controversial. Therefore, this study aimed to evaluate growth and puberty in overweight boys and girls. **Methods:** In this retrospective study, we analyzed medical records of patients aged 5 to 19 years, overweight or obese. The data collected were height (H) and weight, body mass index (BMI), waist circumference (WC), Waist-to-Hip Ratio (WHR) and pubertal staging. **Results:** There was no impact of obesity severity on the onset of puberty or height. The mean final height of the female patients remained within the family target, and the final height of the male patients remained above the genetic standard. **Discussion:** Female patients show greater growth in the prepubertal period and less growth during puberty, which is consistent with previous studies. However, these patients do not present weight gain after menarche, contrary to the literature. In male patients, the severity of BMI did not impact the chronological age (CA) at puberty, and BMI decreased during puberty. There was no loss of final height in any of the groups. Therefore, that treatment should be individualized, since body composition is different according to gender.

Keywords: Obesity; Child obesity; Puberty.

RESUMO: Introdução: A obesidade infantil, além de fator de risco para outras doenças metabólicas, pode também alterar o crescimento e os padrões de puberdade. **Objetivos:** No sexo feminino, a obesidade é associada à antecipação da puberdade. No sexo masculino, existem poucos estudos e há divergências. Este estudo, assim, busca avaliar o crescimento e puberdade em crianças e adolescentes acima do peso. **Métodos:** Estudo retrospectivo, com análise de prontuários médicos de pacientes em idade pré-puberal e puberal com sobrepeso ou obesidade. Os dados obtidos foram: peso, estatura (E), índice de massa corporal (IMC), circunferência abdominal (CA), e estadiamento puberal. **Resultados:** Não houve impacto da gravidade da obesidade na idade cronológica (IC) de entrada na puberdade ou na estatura. A estatura final média tanto das meninas quanto dos meninos ficou dentro do seu padrão genético. **Discussão:** As pacientes do sexo feminino apresentam maior crescimento no período pré-púbere e menor crescimento durante a puberdade, fato consistente com estudos anteriores. Porém, essas pacientes não apresentam ganho de peso após a menarca, contrariando a literatura. Nos pacientes do sexo masculino, a gravidade do IMC não impactou a IC de entrada na puberdade, tendo este reduzido ao longo da puberdade. Em nenhum dos grupos houve perda de estatura final.

Palavras-chave: Obesidade; Obesidade infantil; Puberdade.

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INTRODUCTION

Child obesity is one of the main public health problems in Brazil and all over the world. A study projects that by 2025 there will be 206 million children and adolescents with obesity in the world, and this will reach 254 million by 2030. This same study estimates that by 2030, in Brazil, there will be 7.6 million children and adolescents aged 5 to 19 years old with obesity. This would put Brazil in 5th place in a world childhood obesity ranking. In response to this problem, the World Health Organization announced a goal to have no increase in the prevalence of global childhood obesity by 2025. However, the chances of Brazil achieving this are around 2%^{1,2}. It is already well defined that obesity is a risk factor for many other diseases, such as diabetes mellitus type 2 (DM2), cardiovascular diseases and metabolic syndrome, implying an increase in mortality and morbidity in all age groups³.

Besides these metabolic abnormalities, it is known that obesity can change growth and pubertal patterns. Growth in children and adolescents is controlled by genetic, endocrine and nutritional factors. Although the growth hormone (GH) axis is very important to normal growth, it's well known that the growth plate plays an important role. A lot of factors can act in the growth plate such as GH and IGF1, thyroxine, cortisol, estrogen and androgens hormones, cortisol, cytokines, insulin, leptin and nutritional factors⁴. Children with overweight show a reduction in the total production of growth hormone (GH), but maintain normal concentrations of IGF-1 and its binding proteins, even generating tall stature before the onset of puberty. So, they usually are taller than their peers in the prepubertal period and they can be taller than their genetic pattern in this period as well.

Puberty is the transition period between childhood and adulthood, characterized by the appearance of secondary sexual characters. Studies show that excess adipose tissue influences pubertal and height development. This relation results mainly from the production of pro-inflammatory substances in this tissue, with the ability to act on the adrenarche and the onset of puberty. In females, the higher prevalence of overweight and obesity is associated with the anticipation of puberty. However, in the male sex, few studies have been carried out and present divergences on the topic^{5,6}.

However, once advanced bone age occurs and consequent early onset of puberty, there is an early closure of the epiphyses, which can influence the reach of target height in adulthood⁷.

Therefore, this study aimed to evaluate growth and puberty in overweight patients.

METHODS

A retrospective study was carried out, with a

survey of medical records of patients with overweight or obesity from the Pediatric Endocrinology ambulatory of the Irmandade de Misericórdia da Santa Casa de São Paulo. Patients, prepubertal and pubertal aged between 5 and 19 years were included, and patients who presented incomplete medical records or impossible access to those records, patients with genetic syndromes and patients using medications that could interfere with growth and puberty were excluded (glucocorticoids, growth hormone, GnRH analogues).

The data obtained from the medical records at each pubertal stage were: measures of weight and height, used to calculate the body mass index - BMI, waist circumference (WC) and waist-to-hip ratio (WHR). The measures of height and BMI were expressed in standard deviation score - SDS (2) being considered overweight when BMI SDS was between +1 and +2DP, obesity when between +2 and +3DP and severe obesity, above +3DP. The measurement of waist circumference (WC), obtained at the midpoint between the lower edge of the last rib and the iliac crest, was used for the ratio WC/height and considered normal when less than 0.5 (6). Pubertal staging was evaluated according to Tanner's criteria⁸. Final height was considered when the growth rate was less than 2 cm/year and/or bone age was over 15 years.

The complementary exams analyzed were: bone age (Greulich and Pyle), total cholesterol and fractions, triglycerides, glycemia and insulin, at the first consultation and, when possible, at each stage of puberty.

This study was approved by the ethical board of our institution (CAAE: 52953115.6.0000.5479)

For statistical analysis, the SigmaStat 3.2 software was used. *T student test* was used for analysis between distinct and independent groups, for variables with normal distribution and Mann Whitney test was used for nonparametric variables. To assess the same individual in different periods of time, we used paired t test or Wilcoxon test, depending on the normal distribution of the variable. To assess the correlation between two variables, the Pearson test was used. $P < 0.05$ was considered statistically significant and the confidence intervals were constructed with 95% statistical confidence.

RESULTS

Female participants

We were able to access 90 medical records of girls with overweight and obesity. The girls were divided according to the pubertal stage at the first consultation: prepubescent/Tanner I (n = 34), Tanner II (n = 11), Tanner III (n = 23), Tanner IV (n = 9) and Tanner V (n = 13). Table 1 contains the summary of clinical data from the first consultation.

Table 1: Clinical data of overweight girls, expressed as mean and standard deviation (SD) according to pubertal stage

Tanner	n	Chronological Age (SD)	Height SDS (SD)	BMI SDS (SD)	Bone Age - Chronological Age (SD)	Target Height SDS (SD)
I	34	7,5 (1,6)*	1,3 (1,0)	2,8 (0,9)	1,7 (0,9)	-0,6 (0,9)
II	11	9,8 (1,2)	1,3 (0,9)	2,7 (0,4)	1,1 (0,8)	0,3 (1,3)
III	23	11 (1,2)	1 (1,4)	2,7 (0,5)	1,4 (1,1)	-0,4 (1,0)
IV	9	11,5 (1,2)	0,6 (0,7)	3 (0,6)	-	-0,6 (0,6)
V	13	13,5 (1,6)*	0,8 (1,3)	2,4 (0,4)	-	-0,9 (0,8)

p < 0,5; one way analysis of variance. Analysis shows no difference between groups regarding height SDS, BMI SDS and target height SDS. There were no data records of bone age from patients with Tanner IV and V.

The mean (SD) of chronological age (CA) that girls started puberty (Tanner II) was 9.8 (1.2) years. There was no correlation between CA and BMI SDS ($r = 0.0575$, $p > 0.05$), suggesting that there was no impact of the severity of obesity in pubertal onset. There were only 3 patients that had Tanner II before 8 years old. When comparing

the different groups according to the pubertal stage, no difference was observed in relation to the height SDS or the BMI SDS ($p > 0.05$). Of the 90 patients, 43 reached their final height. The initial and final data are summarized in Table 2.

Table 2. Anthropometric and laboratory data at the beginning of the follow-up and at the time of final height of 43 overweight patients, expressed as mean (SD)

	Height SDS (SD)	Height SDS - TH SDS	BMI SDS	WC/H	TC	LDLc	HDLc	TG	G	Insulin
initial	0,71 (0,9)	1,4 (1,1)	2,7 (0,6)	0,6 (0,06)	164,8 (29,8)	99,6 (26,3)	40 (9,5)	120,3 (83,6)	86,3 (7,8)	15,8 (10)
final	-0,07 (1)*	0,7 (1,2)*	2,4 (0,8)	0,6 (0,06)	163,8 (37,9)	95,8 (31,5)	47,3 (9,4)	105,2 (67,7)	84,5 (9)	16,7 (8,8)

p < 0.05, paired t-test. Height SDS - TH SDS: difference between the z score of patient height and the z score of the target height; BMI SDS: z score of body mass index; WHR: Waist-to-Hip Ratio; TC: total cholesterol; LDLc: low density lipoprotein; HDLc: high density lipoprotein; TG: triglycerides; G: blood glucose.

The mean final height of these patients was equal to their family target, with 159.9 (6.2) and 158.3 (5.9) cm, respectively ($p = 0.229$), showing that they achieved their expected familial height.

No patient had DM2 during the follow-up. The laboratory data are shown in Table 3, showing that there was no difference between the beginning and the final height in any of the variables analyzed.

Male participants

Regarding to male patients, we obtained data from 82 medical records of boys with overweight and obesity.

We separated them according to their first consultation pubertal stage: Tanner I (n=53), Tanner II (n=7), Tanner III (n=15), Tanner IV (n=5) e Tanner V (n=1) (Table 4).

The puberty entry chronological age could be determined in 23 boys, with a mean (SD) of 11.7(1.2) years; with height SDS 1.4 (1) and BMI SDS 2.9(0.4). As observed for girls, there was a reduction in the height SDS during puberty, as shown in Table 4. Next, they were clustered in the following way for data analysis: Group 1 (pre-pubertal): Tanner I; Group 2: Tanner II and III and Group 3: Tanner IV and V (Table 4).

Table 3. Clinical data of overweight boys, expressed as mean and standard deviation (SD) according to pubertal stage.

Tanner	n	Chronological Age (SD)	Height SDS (SD)	BMI SDS (SD)
I	53	8,97 (2)	1,4 (0,8)	3,3 (0,8)
II	7	12,5 (0,6)	1,5 (1,2)	2,8 (0,5)
III	15	12,6 (1,6)	1,1 (1)	2,8 (0,7)
IV	6	13,8 (0,9)	0,9 (0,2)	3,3 (0,8)

Table 4. Clinical and laboratorial data of the groups, according to puberty stage, expressed as mean.

Group	n	Chronological Age (SD)	BMI SDS (DP)	Cholesterol Total (DP)	LDL (DP)	HDL (DP)	TG (range)
Group 1 (Tanner I)	53	8,97 (2)	3,3 (0,8)	174,1 (37)	102,9 (30,3)	45 (8,4)	98
Group 2 (Tanner II + III)	22	12,5 (1,4)	2,8 (0,6)	159,7 (32)	93,7 (25)	37,4 (8)	103
Group 3 (Tanner IV + V)	6	13,8 (0,9)	2,9 (0,4)	121,5 (29,7)	65 (26,7)	43,5 (1,9)	71

One way analysis of variance shows that BMI SDS reduced at the onset of puberty and remained stable until the end ($P = 0,010$), Cholesterol values were higher at the onset of puberty, with a progressive reduction ($P = 0,020$), as well as LDL values, which showed no significant difference between the prepubertal and onset of puberty groups, but were higher in the prepubertal group when compared to the end of puberty ($P = 0,049$). HDL values reduced at puberty onset, and then returned to higher levels at the end

of puberty ($P = 0,017$). Triglyceride values increased at the onset of puberty but decreased towards the end ($P = 0,044$). Data collected regarding insulin values were insufficient to perform the analysis.

14 male participants reached their final height. Similar to the girl's results, the average final height of these patients was equal to their family target, showing that there was no loss of final height in relation to the standard genetic (Table 5).

Table 5. Anthropometric and laboratory data at the beginning of the follow-up and at the time of final height of 43 overweight patients, expressed as mean (SD)

	Height SDS (SD)	Height SDS - TH SDS	BMI SDS (SD)
initial	1,2 (0,9)	1,36	2,7 (0,6)
final	0,3 (0,9)	0,46	2,5 (1,1)

DISCUSSION

Studies show that overweight girls tend to anticipate puberty. In our sample, we observed that the age of the thelarche was, on average, 9.8 years, very close to the chronological age of the general population, described as 10.3 years⁸. Of this group, only three girls had pubertal onset before 8 years of age and three others, between eight and nine years old.

The girls, when they enter puberty, have more advanced bone age and height above the family standard. However, the height remains within the expected range, at the end of puberty. This suggests that they grow more in the prepubertal period and the growth rate slows down during puberty. This fact is consistent with studies such as that of Qing He and Johan Karlberg⁹, who observed that an increase in BMI from 2 years to 8 years of age is associated with greater height in childhood and growth during adolescence, and such factors do not affect height in adulthood. Obesity during childhood causes the growth curve to shift to the left, but as individuals show less growth during puberty, this does not result in effects on the final height¹⁰.

There was no difference between the BMI SDS at the beginning and the end of puberty, indicating that

these patients do not show worsening in weight gain after menarche, contrary to the literature. The girls' weight spurt occurs after menarche and at the expense of gaining adipose tissue. There was also no worsening of the WC/H ratio, remaining stable during puberty, but at high values (above 0.5). However, despite prolonged follow-up in a specialized service, there was also no reduction in the BMI SDS, showing little adherence to conventional treatment, which guides lifestyle changes.

Regarding the laboratory variables, we did not have any patient who developed DM2, and we also did not observe any differences between the values obtained at the beginning and at the end of puberty. Some studies suggest that in healthy adolescents, insulin resistance and compensatory hyperinsulinemia are higher at the beginning of puberty, returning to normal after the end of puberty. We observed in this group of girls that there was no difference in insulin concentrations, which may suggest that they will be adults with a higher risk of developing metabolic syndrome and cardiovascular disease.

In boys with overweight and obesity, our results show an average of 11 years old for Tanner II and 12/13 years old for Tanner III, therefore it didn't show precocity or delay in relation to the average, going against studies that show that heavier boys have a greater risk of having

an earlier pubertal initiation^{11,12}.

In relation to BMI, our male patients started puberty more obese and reached the end of puberty less obese. It was different to what happened to girls. Boys usually have a higher weight gain at the middle/end of puberty and their body composition is also different than girls. They have a higher increment of muscle mass compared to girls and it leads to a higher energy expenditure. Cholesterol was higher at the beginning of puberty than at the end, as well as LDL, which had no difference between the prepubertal and early puberty groups but was higher in the prepubertal groups when compared to the end of puberty. There was a drop in HDL values and an increase in TG during puberty. These data, therefore, are in agreement with the literature, in which it is said that there is a decrease in total cholesterol, LDL and HDL levels and an increase in TG levels in boys of normal weight during adolescence¹². The drop in HDL and the increase in TG throughout puberty suggest a greater insulin resistance mechanism in those who entered puberty than in those who are prepubertal. At the end of puberty, TG decreases and HDL increases again. Insulin analysis, although limited by the very small n, gives the impression that it increases and gets taller. Both the increase in insulin resistance and insulin levels are analyzed already described in the literature¹³, which are corroborated by the study.

The analysis of heights showed that at the beginning of puberty the height SDS is higher (1.2 SD), and at the end of puberty, it falls by 1 SD (being 0.2 SD), analysing final height and target height, the SDS showed that the final height was within the expected range for the familiar target; although they start out tall, the height does not exceed the family value. The start height SDS is greater than the target height SDS (1.3 SD above).

individualized between genders.

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Masarwi et al.¹⁴ showed that leptin, encoded by the obese gene, is produced and secreted mostly by adipose tissue and can regulate growth by stimulating aromatase in the presence of testosterone. The increase in aromatase expression enhances the aromatization process of testosterone to estrogen, increasing locally production of estrogen, which culminates in growth decline due to the estrogen effect of declining the growth plate chondrocyte proliferation rate. Obesity is associated with high serum leptin levels due to the increase in adipose tissue concomitant with central resistance to circulating leptin. The peripheral effect of leptin may explain the accelerated growth of pre-pubertal obese children. Pre-pubertal children with obesity are taller than their peers, often start puberty earlier and show earlier maturation of the growth plate, which may be also associated with loss of the pubertal growth spurt and final short stature. In this study, however, there has been no loss of stature at the end of puberty.

CONCLUSION

We concluded that both female and male patients started puberty within the expected age, with height above the family target. However, the final height was compatible with the target height, due to the reduced growth rate throughout puberty. In contrast to what many studies point out, there was no difference between the BMI SDS at the beginning and at the end of puberty in female patients, which indicates that there was no significant increase in weight after the end of puberty in these patients. But boys can reduce their BMI SDS at the end of puberty. These data suggest that treatment should be different and

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