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CASE REPORT

Repercussions of weight loss on the growth rate of children and adolescents: a case study

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Abstract

Report of the longitudinal evolution of the anthropometric indices of an obese child from preschool to the end of the puberty. It is about a 4 year-old boy with history of weight gain since the first year due to carbohydrate-rich and industrialized food intake, associated with physical inactivity and familiar history of obesity. At the first appointment he had body mass index (BMI) in Z-score + 7,36 and stature above the parental target. After orientations about lifestyle changes, he reached, at 7 years old, the lowest BMI and the growth rate became reduced. At this point, other causes of growth retardation have been ruled out, therefore the hypothesis was of growth stop secondary to weight loss and recanalization of the growth curve in response to the treatment of the exogenous obesity. In the evolution, the patient regained weight maintaining the drop on the stature, staying within the parental target. When puberty started, he presented progressive fall in BMI score and increased growth velocity. At 17, he reached BMI and stature within normality and parent target. Throughout the follow-up, the importance of healthy eating and realization of physical activity was reinforced. No medications were used, and there were no alterations at physical and complementary exams. The weight loss in obese children with tall stature for the parental target, can lead to slow growth rate when secondary to change to healthy habits and increase in physical activity, however without affecting the final height.

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INTRODUCTION

Monitoring a child's growth provides a longitudinal assessment of the patient and reflects their health status[1]. Altered growth may be the first manifestation of an underlying disease[2]. The causes of decreases in growth rate must be thoroughly investigated.

The target height is calculated based on the height of the parents. When growth patterns deviate from the expected genetic height, the potential occurrence of an underlying condition must be considered[2,3]. Deviations include excessive and insufficient growth.

Exogenous obesity in children and adolescents has been associated with the future development of chronic diseases and often correlated with rapid linear growth and accelerated bone maturation[2]. Therefore, reactive tall stature is expected in a child diagnosed with obesity at an early age.

This report describes the changes in the anthropometric indicators of an obese child initially diagnosed with tall stature compared with the parental target. Changes in lifestyle and treatment for obesity allowed the patient to return to within the range expected for his target height, despite a decrease in growth rate.

CASE REPORT

A 4-year-old boy was referred to the clinic for obese children and adolescents due to excessive weight gain from the first year of life.

He is the second child of a nonconsanguineous couple. He has a family history of exogenous obesity on the paternal side, a maternal grandmother and an uncle with type 2 diabetes, and a mother with systemic arterial hypertension. He was born full term, with height and weight suitable for his gestational age. He was exclusively breastfed until the age of four months and had breastmilk and other foods during the first year of his life. He had been followed by a pediatric pulmonologist since he was four years old for recurrent wheezing, which was controlled after the introduction of medium-dose inhaled corticosteroids and regular follow-up.

He did not present changes on physical examination on the first visit, except for a body mass index (BMI) z-score of +7.36 and a height (h) z-score of +1.78. The family channel was calculated at 167cm +- 8.5cm (Z-score between -2 and 0), which meant that the patient was obese and tall for his age[3].

His diet was rich in carbohydrates and industrialized foods and he did not eat fruits or vegetables. He did not perform regular physical activities and preferred to play while sitting down. He spent about four hours a day in front of a screen.

His family was advised about the need to decrease the screen time, the importance of an adequate diet, and the development of regular physical activities. Even though he was still selective about the food he ate, he started a routine of moderate intensity physical exercises two to three times a week. He started eating less carbohydrate-rich and industrialized foods and lost weight gradually, with maximum BMI reduction occurring at seven years of age (Z-score = +0.66) (Figure 1A). However, when he was seven, a phase in which he was expected to grow about 5 cm/year[3], he experienced a deceleration in height growth (and in the Z-score = +0.29) (Figure 1B) and grew only 0.48 cm/year. Despite the decrease in growth rate, the patient stayed within the parental target.

A: Body mass index x age in years. Point corresponding to the age of 7 years and 4 months, in which the greatest drop in the BMI was observed. BMI Z-score = +0.66[11]. B: Height in centimeters x age in years[11]. The red marking on the right refers to the parental target.

An investigation into the etiology of decreased growth rate was started. The tests performed to uncover a possible disease causing a slower growth rate came back normal (Table 1). The only altered parameter was an elevated IgE. Finding an IgE increase was consistent with the patient's atopic condition, but it did not justify the slower growth rate, because the patient's pulmonary symptoms had been under control since the introduction of inhaled corticosteroids. Therefore, growth arrest secondary to weight loss and rechanneling of the growth curve as a response to exogenous obesity therapy was considered.

A serum insulin test was ordered at the start of follow-up to monitor the status of exogenous obesity. As the patient lost weight, serum insulin levels, which were within normal range, gradually decreased, reaching values below 2.0 uIU/ml in the period in which growth rate and BMI were at the lowest (Figure 2). A study including eutrophic children and adolescents showed that fasting insulin levels varied with age and grew until puberty, to then drop. However, there are no internationally accepted cutoff values for insulin levels in pediatrics[4]. Comparing data from different moments of our patient's life, during puberty he had an increase in serum insulin levels, which later decreased without jeopardizing his growth.

Relationship between serum insulin, BMI Z-scores, and growth rate. All decreased to a minimum at seven years of age.

The patient gained weight again and after two years reaching a BMI Z-score = +2.08 and sustaining a drop in height Z-score (Z-score = -0.21). At the age of nine years and nine months, he had a bone age of 9.1 years. From nine to twelve years of age, he maintained a high BMI (Z-score = +2.0) and saw further drops in height (Z-score = -1.07). He underwent a GH stimulation test and had a normal result. At this time, the patient was no longer performing regular physical activities and spent most of the time in front of screens. Although he was eating more fruit and vegetables, he was also eating an excessive amount of sweet foods.

At the age of 13 years and 2 months, in the period immediately before the pubertal growth spurt, a moment in which there is maximum growth deceleration before a peak in growth rate[3,5], he had a bone age of 11.4 years (-2.1 standard deviations). When the signs of pubertal development appeared

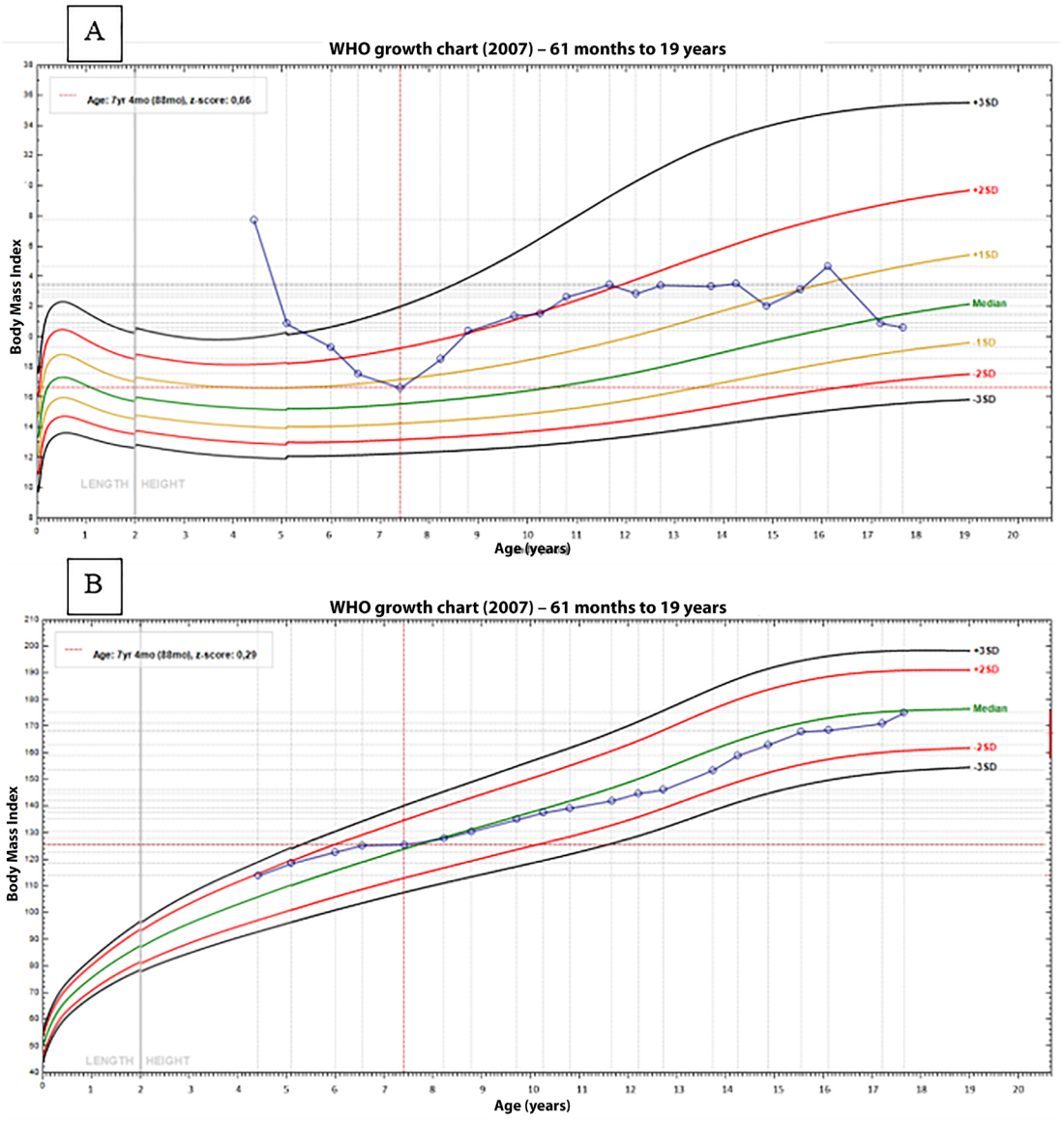


Figure 1. A: BMI; B: Height growth.

at 13 years of age, he showed a progressive decrease in BMI Z-scores and an increase in growth rate (7.4 cm/year), with a consequent increase in height (Z-score = -0.74).

At the age of 17, he achieved a BMI and a height with Z-scores of -0.34 and -0.12, respectively, within the normal range for his sex and age and within the parental target. During puberty, he performed regular physical activity and had a healthy diet. Table 1 summarizes the chronology of events.

After potential diseases were ruled out, the following diagnostic hypothesis was retrospectively considered: exogenous obesity with reactive tall stature, followed by growth arrest secondary to weight loss after the introduction of diet changes and physical activity. The patient returned to his parental target (rechanneling), remaining at the upper limit of the channel. From the onset of puberty, he showed adequate growth, reaching the expected final height. During the entire follow-up period, the importance of eating a healthy diet and performing physical activity was reinforced, and at no time was medication used.

DISCUSSION

Growth is a continuous process affected by intrinsic and extrinsic factors on the individual. An individual's genetic potential can only be reached if these factors are concordant with the growth process[5].

Obesity, in turn, is defined as excessive accumulation of body fat with harmful consequences to one's health. It has a multifactorial etiology and, like growth, is dependent upon on genetic, nutritional, metabolic-hormonal, psychosocial, and

Table 1. Clinical signs, anthropometric indicators, intervention, and test results.

Age	Follow-up and clinical complaints	Anthropometry Z-scores; Growth rate and bone age	Proposed Management	Supplementary tests
4 to 5 years	Start of follow-up: patient gained weight since the first year of life	BMI = +7.36; H = +1.78	Lifestyle changes: advice on diet and physical activity	Total cholesterol: 163 mg/dl; HDL-cholesterol: 57 mg/dl; LDL-cholesterol: 94 mg/dl; Triglycerides: 60 mg/dl; Blood glucose: 73mg/dl; Insulin: 8.6 uUI/ml TSH: 2.71 uIU/ml; Free T4:1.41 ng/dl IgE: 2120 IU/ml;
6 years	Treatment successful with lifestyle changes	BMI = +1.37 H = +1,18 GR = 4.28 cm/year	Encouragement to continue with the diet and physical activity	Total Cholesterol: 140 mg/dl; HDL-cholesterol: 51 mg/dl; LDL-cholesterol: 81 mg/dl; Triglycerides: 39 mg/dl; Blood Glucose: 72 mg/dl; Insulin: <2.0 uIU/ml
7 to 8 years	"He is not growing"	BMI = +0.66; H = +0,29; GR = 0.48 cm/year ; Bone age = 4 years and 6 months (Body age = 7 years and 7 months)	Initial investigation on growth retardation: No change. Advice on diet and physical activity	Hb: 14.2; MCV: 84; MCH: 29; TSH: 2.41 uIU/ml; Free T4:1.41 ng/dl; Anti-HIV: negative; Alpha-1-acid glycoprotein: 77.4mg/dl; CRP: 0.03; ESR: 2.0 mm; Creatinine: 0.4; Urea: 26; Albumin/creatinine: 6.27 mg/g; AST: 26; ALT:11; Uric acid: 1.8; Urine 1 no changes; negative urine culture; Na and Cl in sweat: normal. IGF1: 262 ng/ml; IGBP3: 4.5 ug/ml;
9 to 11 years	Resumed weight gain	BMI = +2.08; E = -0.21; GR=4.9 cm/year; Bone age = 9 years and 1 month, (Body age = 9 years and 9 months)	Investigation with GH stimulation test with clonidine and other tests. Advice on diet and physical activity	Dynamic GH test: Baseline - 0.06 ng/ml; 30 minutes - 0.08 ng/ml; 60 minutes - 7.37 ng/ml; 90 minutes: 4.78 ng/ml; 120 minutes - 1.06 ng/ml - Normal response. Anti-thyroglobulin: Negative; Anti-TPO: Negative Anti-transglutaminase: Negative;
12 years	Period just before pubertal growth spurt, phase of lowest height Z-score	BMI = +1.79 H = -1.07 GR = 2.6 cm/year	Advice on diet and physical activity	
13 to 14 years	Pubertal growth spurt	BMI = +1.42 H = -0.74 GR = 11 cm/year Bone age = 11 years and 4 months; body age = 13 years and 2 months	Advice on diet and physical activity	Total cholesterol: 131 mg/dL; HDL-cholesterol: 44 mg/dL; LDL-cholesterol: 80 mg/dL; Triglycerides: 36 mg/dL; Blood glucose: 74 mg/dL; Insulin: 2.06 uIU/mL TSH: 2.871 uIU/ml; Free T4:1.3 ng/dl
17 years	Eutrophic, stature at the upper limit of the parental target.	BMI = -0.34 H = - 0.12	Advice on diet and physical activity. Discharge from the obesity clinic	

environmental factors[6]. The most common type is exogenous obesity, characterized by low physical activity and excessive intake of foods rich in fats and carbohydrates, which demonstrates the important role of the environment[7].

Growth rate provides information about a child's growth pattern. Deviations from normal growth may be the first manifestation of an underlying disease[2]. Decreases in growth rate must be thoroughly investigated. Genetic inheritance is also important in determining growth, and when a child has a height that deviates from familial patterns, an underlying disease could be the culprit. For example, a child who is short vis-à-vis the parental target or with a low growth rate may not be short in height[8].

Exogenous obesity is accompanied by high stature for age and for the parental target, and a trend toward showing advanced bone age[2,9]. A longitudinal follow-up of a series of

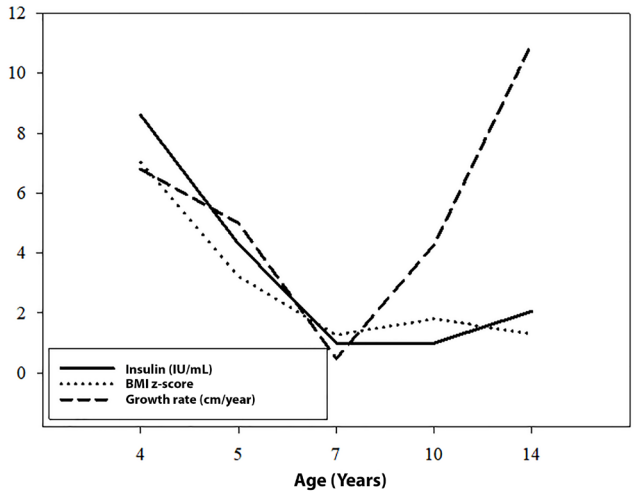


Figure 2. Relationship between insulin, BMI z-scores and growth rate.

randomized clinical trials on weight control evaluated 158 children aged 6 to 12 years up to 10 years after treatment for obesity in order to assess their height development. Although these children had a reduction in height percentile compared with their height at the beginning of treatment, there was no difference in growth at the end of 10 years compared with non-obese children[10].

A study following malnourished children on nutritional rehabilitation showed that daily weight gain was directly related to increases in growth rate, which consequently allowed them to catch up with their genetic growth potential[11]. Similarly, the child described in our report had severe obesity at an early age, experienced rechanneling after losing weight and becoming eutrophic, as if after undergoing nutritional rehabilitation, he was able to reach his genetic potential.

A child with obesity at an early age undergoes accelerated growth. When successfully treated, this child may experience a decrease in growth rate until they reach their genetic target height, and a rechanneling of growth occurs.

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