Abstract

Objective: to review the medical literature in the last 5 years regarding obesity in children as well as its treatment.

Methods: literature review.

Results: obesity is a chronic disease that presents high percentages of therapeutic failure and recurrence, leading to serious physical and psychological consequences, especially in its most severe forms. Health care professionals and families usually neglect the treatment of obesity in the hope of a spontaneous reversal of the condition during adolescence. There is a great probability that obese children and adolescents will continue to be obese in adulthood, thus increasing morbidity and mortality of several diseases.

Conclusion: Pediatricians are supposed to early identify children at greater risk for obesity, taking effective control measures in order to achieve a more favorable prognosis.


Introduction

Obesity is a chronic multifactorial disease triggered by the combination of genetic and environmental factors. Obesity is present in 95 to 98% of cases, and only a small percentage (2 to 5%) originates from genetic syndromes that evolve into obesity (Prader-Willi, Bardet-Biedl), craniopharyngioma or endocrine disorders (hypothyroidism, Cushing’s syndrome).

Regarded as one of the major health problems in industrialized countries, obesity has become a growing threat to developing countries as well, coexisting with malnutrition. Obesity is not a recent phenomenon in human history; however, it had never been so epidemically spread as it is today. This significant increase in the number of obese individuals is related to changes in lifestyle and eating habits. We observed that the frequent use of high-calorie industrialized foods containing saturated fat and cholesterol, and technological advances encourage children and adolescents to adopt sedentary habits such as television viewing, playing videogames, and sitting in front of computers for hours.
Early weaning with inadequate food supplementation may lead to obesity during the first year of life in predisposed individuals.

Family disorders, especially mother-child relationship changes, are important determinants of obesity in children.\textsuperscript{8}

Obesity may initiate or be aggravated in adolescence due to a physiological increase in adipose tissue mass (especially in females), higher intake of high-calorie fast food, in addition to emotional disorders.

Family history of obesity is the most important factor that puts children at a higher risk of becoming obese due to genetic and environmental factors such as eating habits, which determine energy intake, family lifestyle, in relation to energy expenditure, and the whole family context.\textsuperscript{9} The probability of children becoming obese is greater if their parents are obese. This probability is lower if neither their mother nor their father is obese, high when only one parent is obese and very high if both parents are obese.

It is known that obesity in childhood and adolescence tends to continue in adulthood if not properly controlled, causing an increase in morbidity and mortality, and a reduction in life expectancy.\textsuperscript{10,11} Therefore, the earlier identification of obesity-prone children by pediatricians allows for effective control measures, thus helping prognosis in the long run.

Epidemiology

The prevalence of childhood obesity has been increasing in industrialized countries. In the United States, the comparison of data obtained in 1965 and 1980 showed a 67% increase in obesity among male children and a 41% increase among female children aged between 6 and 11 years.\textsuperscript{12}

Environmental theories are used to explain these changes from an epidemiological view, since no substantial changes in genetic characteristics of these populations have occurred in the last few decades, in opposition to significant changes in eating habits. The factors that should be considered when analyzing the increased prevalence of obesity among children include habits such as TV viewing and electronic gameplaying for long hours, and practices such as the suspension of breastfeeding, use of milk formulas and replacement of homemade foods with industrialized foods, which usually have higher energy density, are tastier and are always spearheaded by vigorous advertising campaigns.

In Brazil, some remarkable changes in leisure and eating habits have taken place in the last few years, but these changes do not involve a great share of the population, which is still squeezed out of the target market. As a result, the number of obese adults has been increasing. The comparisons of national surveys conducted in 1974 and 1989 showed an increase of respectively 75% and 60% in the number of obese men and women.\textsuperscript{2}

In 1989, there were approximately one and a half million obese children in Brazil with a slightly higher prevalence of obesity among female children (5% and 4.8%). The prevalence rates of obesity for the southern (7.2%) and southeastern (6.2%) regions were more than twice the rates observed for the northeastern region (2.5%), while the northern and mid-western regions presented intermediate prevalence rates. Among children born into high-income families, the percentage of obese individuals was around 8% compared to 2.5% in children who were born into low-income families. As far as age was concerned, obesity was more frequently present in the first two years of life, drawing our attention to the importance of breastfeeding and an adequate weaning diet.

Environmental theories were confirmed by the high prevalence of obesity in developed regions of the country where industrialization occurs much faster, resulting in remarkable changes in eating habits. Still in agreement with these theories, we found that obesity is more frequent during the first years of life, having an association with early weaning practices and inadequate dietary recommendations, which encourage overfeeding and even award obese infants.

The data obtained from the national survey carried out in 1996, which only included children younger than five years, revealed that our country is already going through the process called nutrition transition.\textsuperscript{3} In this process, mothers who have a better level of education tend to acquire adequate knowledge about nutrition (conveyed either by their doctors or the media), and end up providing their children with better nutrition, thus reducing the prevalence of obesity. Therefore, the prevalence of obesity among children younger than five years, born into mothers who have a better level of education, fell from 9.9% in 1989 to 4.5% in 1999.

On the other hand, the population in the northeastern region is still living the previous phase of the so-called nutrition transition. Nutritional inadequacy such as the intake of foods rich in salt and fat occurs due to the increased availability of industrialized foods that result from urbanization and lack of adequate information. Consequently, a 2.5% obesity prevalence rate in 1989 among children younger than five years reached 4.5% in 1996.

We conclude that, although nutritional disorders associated with insufficient food intake are still a priority for health policies and programs targeted on child nutrition, aggravation caused by excessive intake of food due to sedentary habits should not be overlooked. In times of demographic and epidemiological transition in Brazil, in which urbanization, life expectancy, deaths caused by chronic degenerative diseases, and violence are higher and higher, while fertility and deaths caused by infectious diseases are on the wane, nutrition transition should also be
considered, especially in regard to obesity, its determination, and measures aimed at its individual and collective control.

Pathophysiology

The pathophysiology of obesity is not totally clear. Breakthroughs have recently occurred in the field of molecular biology, helping to elucidate the disease.

Research usually follows two complementary approaches: a physiochemical approach, in which variations in energy balance are studied; and the molecular biology approach, which isolates specific genes responsible for controlling the different factors that determine this energy balance.13

Obesity is an energy metabolism disorder, in which triglycerides are excessively stored in the adipose tissue.

Body energy stores are regulated by the intake and expenditure of energy. When intake and expenditure of energy are balanced, body weight is maintained. A low positive balance causes low weight gain, but the chronic imbalance between intake and expenditure of energy leads to obesity with the passing of time.

In short, we can say that the causes of obesity are associated with excessive energy intake, reduced energy expenditure, and changes in the regulation of energy balance.14,15

Breakthroughs in molecular biology began after the observation of gene mutations in obese mice, which resulted in new forms of body weight regulation.16 There are several components that have been identified and participate in the regulation of body weight; however, their precise roles have not been defined yet. Therefore, we are going to discuss the mechanisms of two well-defined components: leptin and neuropeptide Y.

Leptin is a protein codified as the ob gene (in mice), produced and secreted by mature adipocytes, acting as an afferent feedback signal.17

Obese mutant mice (ob/ob) produce biologically inactive leptin. Therefore, they do not receive a feedback signal, continue to ingest excessive amounts of food, and gain excessive weight. The exogenous administration of leptin to these mice drastically reduces food intake and body weight.

Other obese mutant mice (db/db) produce normal leptin. However, there is a problem with the leptin receptor, and these mice are resistant to the effects of leptin. Consequently, they do not receive a feedback signal, continue to ingest food excessively, and end up gaining excessive weight.18

There was a great expectation after the discovery of leptin, and several research studies were then conducted. Nevertheless, studies with humans revealed that when obese and nonobese adults and children are compared, obese individuals present higher serum leptin concentrations, and these high concentrations are positively associated with adipose tissue mass.19 A hypothesis is that obese individuals could be less sensitized to leptin.

Neuropeptide Y, a neurotransmitter released by hypothalamic neurons, plays an important role in the control of body weight. Differently from leptin, the effects of neuropeptide Y, also studied in mice, are: increase in food intake, increase in serum insulin concentration, reduced sympathetic activity, thus reducing the release of energy, building up triglyceride stores in adipocytes, consequently leading to body weight gain. The secretion of neuropeptide Y is inhibited by leptin through a feedback mechanism.20

Several research studies on obesity are targeted on the assessment of the mechanisms involved in its physiopathology. Other components concerned with the control of body weight such as peptides (cholecystokinin-CCK, urocortin, CART); receptors (beta 3-adrenergic, glucocorticoid, and serotonin); and uncoupling protein (UCP) are also under study.21

Diagnosis

The diagnosis of obesity may be obtained through anthropometric methods, which are easy to use, harmless, relatively inexpensive, and ideal for daily practice.21 Normally, weight and height are used. Skinfolds and some circumference measurements may also be used.

The weight/height ratio, which is the relation between measured weight and ideal weight for height/age in the 50th percentile (P50), multiplied by 100, is expressed in percentage. NCHS is used as reference standard.

Weight/height measurement is commonly used in children. When the value exceeds 110% and is less than 120%, children are considered to be overweight; when the values are equal to or higher than 120%, they are considered to be obese.

For adolescents, the most adequate method is the BMI (Body Mass Index), which is the relation between weight (kg) and squared height (m). The result obtained through this measurement must be checked against percentile tables according to gender, age (above 6 years), and race. A BMI above the 85th percentile (P85) and below the 95th percentile (P95) is interpreted as overweight, and above P95, as obesity.22

Laboratory measurements of body mass are more precise but more expensive, with sophisticated equipment commonly used in specialized centers. These lab measurements include electrical bioimpedance, infrared, and DEXA (dual-energy x-ray absorptiometry), which is a very reliable, not so invasive method that assesses lean and fat body masses. Female adolescents with regular menstrual cycles, and male adolescents should have respectively around 26% and 15% of fat mass.23
Consequences

Obesity is associated with important metabolic changes, which depend upon duration and severity, and whose consequences are more common in adults. However, obese children are at greater risk for some diseases, and psychosocial disorders, caused by the social stigmas surrounding obesity, are totally relevant at this stage of personality formation.

Hyperinsulinemia is one of the metabolic changes found in obesity, presenting a significant correlation with body fat mass. Fasting serum insulin levels are reduced due to weight loss and calorie restriction. The finding of basal hyperinsulinemia and postglucose-load, with normal or increased glycemia, suggests resistance to insulin. Insulin resistance seems to be related to changes in receptor levels in peripheral tissues.

Another important metabolic consequence of obesity is related to adverse lipid concentrations. Several studies have already shown an association between obesity and increase in total cholesterol levels and LDL cholesterol, with greater risk for the development of atherosclerotic disease and low HDL cholesterol levels; the risk is even greater because of antiatherotogenic fraction. Atherosclerosis may begin in childhood, and high cholesterol levels at this stage play an important role in the development of adult atherosclerosis.

The body distribution of adipose tissue implies different risks for the development of metabolic changes. Fat deposition in the trunk is clearly associated with a higher risk for diabetes, cardiovascular diseases, and high blood pressure.

There is also an association between obesity and high blood pressure; the mechanisms that predispose the occurrence of high blood pressure are not well known. Systolic and diastolic pressures increase as body mass index increases. Hemodynamic studies have shown increased heart deficiency in obese patients and reveal the expansion of blood volume as a cause for high blood pressure. There is a narrow relationship between blood pressure and body weight, in which blood pressure is reduced as weight is lost. Another mechanism that may be responsible for high blood pressure in obese individuals is reduced sodium excretion caused by hyperinsulinism. We observed that the renin - angiotensin - aldosterone system is stimulated in obese individuals, especially those with abdominal obesity, thus explaining the increase in blood pressure.

Obesity is one of the major causes of high blood pressure in children and adolescents, originating future cerebrovascular and cardiovascular diseases.

Orthopedic disorders are also frequent in obese individuals due to the trauma inflicted by excessive weight on their joints. Knee joints are the most commonly involved, and slipped capital femoral epiphysis is very common in obese individuals.

Obese individuals may present changes in their pulmonary function with reduction in the residual volume and maximum expiratory volume, and a tendency towards general reduction in pulmonary volume. Pickwickian syndrome, which is characterized by hypoventilation, diurnal sleepiness, and sleep apnea, may occur in severe cases of obesity.

Type II diabetes mellitus is an important chronic disease associated with obesity. This type of diabetes is rare in children and adolescents, but very common in obese adults.

Usual dermatological disorders found in obese individuals include striae, fragile skin in skinfold regions, with a tendency to fungal infection, and acanthosis nigricans, with skin darkening under the arms and around the neck.

Treatment

The best way to treat obesity is the use of a multidisciplinary approach due to the multifactorial nature of the disease. The treatment must be performed by a pediatrician, nutritionist, psychologist, and physiotherapist.

Patients need to establish a good relationship with the professionals involved since the treatment is long-lasting. Children and adolescents are not clearly aware of time; therefore, we should not show them just the potential risks of obesity but also assess its current implications, that is, the problems overweight is causing.

Treatment should be initiated as early as possible since the older the children and the more overweight they are, the more difficult it will be to reverse the case due to the assimilation of eating habits and metabolic changes.

The pediatrician establishes the first contact with the patient through anamnesis, which should emphasize the age of onset, development of the condition, and its possible triggering mechanisms. Previous family history of obesity, cardiovascular diseases, high blood pressure, dyslipidemia, and diabetes should also be investigated. The amount of physical activity performed by children should be assessed, in addition to disorders related to behavior, school, and family. On physical examination, blood pressure has to be checked, and possible complications such as orthopedic, postural, dermatological and respiratory problems have to be identified. Subsidiary tests include serum dosage of triglycerides, total cholesterol, and fractions. If possible, laboratory measurements of body mass should be conducted.

When lipid concentration is normal, the procedure has to be repeated the following year. If results are not normal, nutritional recommendations are made, and a new sample is collected within three months (Table 1.0). The lipid concentration in children and adolescents may be evaluated according to Kwiterovich method (1989).

In cases of severe obesity, hyperinsulinism may be detected through serum basal insulin dosage and fasting glycemia (insulin/glycemia 0.5).
After nutritional anamnesis, the patient is instructed to gradually reduce food intake, not to eat more than the recommended portion, to chew food properly, not to eat in front of TV, and to stick to meal time. In cases of dyslipidemia, patients are encouraged to limit their amount of calories obtained from fats that account for 30% of total calories required, reducing the intake of saturated fat and cholesterol, having a preference for polyunsaturated fats, complex carbohydrates and fibers.

In hypertensive obese children, the reduction in salt intake is important in order to normalize blood pressure. Children who have not entered the enhanced growth stage yet need to normalize their weight/height ratio, which can only be attained through development and maintenance of body weight.

Strict diets are contraindicated, and could lead to muscle tissue loss and reduce growth speed.

No medications should be used to treat childhood obesity due to their side effects and the risk of chemical and/or psychological dependence. Physical activity must always be recommended, respecting every patient’s limitations. Regular aerobic exercises are indicated, with the aim of energy expenditure and change in lifestyle.

Psychologists play a vital role in encouraging and supporting patients during treatment, which is slow and long-lasting.

Prognosis

Obesity is not easily controlled, presenting a high rate of therapeutic failure and recurrence, with possible organic and psychosocial consequences during its development, especially in more severe cases.

The treatment of obese children is usually neglected by the family and health care providers since a spontaneous resolution is expected. However, there is a great probability that obesity will persist in adulthood. Several studies were carried out to assess the evolution of obesity in children and its continuity in adulthood. The results of these studies are variable and hard to compare due to differences in sample selection, criteria used for the definition of obesity, analysis and presentation of data. These studies, however, tend to show that obese children and adolescents have a higher risk of becoming obese adults when compared to nonobese individuals.

The risk of childhood obesity extending into adulthood is related to its duration and severity. Resolution rates decrease with age and the increase in severity increases the risk for persistence. Approximately one third of obese adults were obese children, and when obesity is severe, the ratio increases to ½ or ¾.

A longitudinal study carried out in England to assess the weight and height of individuals up to the age of 26 years revealed that 40% of obese children aged 11 and 50% of them at the age of 15 were still obese by the age of 26. At all ages, the lower the relative weight, the lower the risk of becoming obese in adulthood.

In the study conducted by Bogalusa between 1973 and 1983, with patients aged on average 7.3 years at the beginning and 15.7 years at the end of the survey, 66% of the children who suffered from severe obesity and only 32% of those with moderate obesity at the beginning of the study continued to be obese. Severe obesity and the consecutive increase in its levels enhanced the probability of persistence.

In a follow-up study of obese children over a 40-year period, carried out in Stockholm (Sweden), the maximum weight for height ratio was achieved at the pubertal stage (higher than 3.5 SD) while 47% still presented obesity in adulthood. Family history of obesity (parents and grandparents) and the degree of obesity at the pubertal stage were the most important factors that influenced the persistence of obesity in adulthood. This study also showed that severe obesity in adolescents was associated with high morbidity and mortality in adulthood.

The probability of obese children becoming obese adults gradually increases with age. Therefore, the older obese children are, the greater their risk of becoming obese adults is.

References


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