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CHILDHOOD OBESITY

Sažetak: Pretilost se, po Međunarodnoj klasifikaciji bolesti (International classification of diseases, eleventh revision, ICD-11), svrstava u endokrine bolesti, bolesti prehrane i metabolizma (ICD-XI; E66). Predstavlja hroničnu multifaktorijalnu bolest praćenu nenormalnim ili prekomjernim nakupljanjem masnog tkiva koje predstavlja zdravstveni rizik.

Svjetska zdravstvena organizacija opisuje epidemiju dječije pretilosti kao: „jedan od najozbiljnijih javnozdravstvenih izazova 21. vijeka". Prema istoj, 41 milion djece mlađe od 5 godina i 340 miliona djece i adolescenata uzrasta od 5 do 19 godina ima prekomjernu tjelesnu težinu ili pretilost.

Pretilost može imati značajan uticaj na zdravstveno stanje, blagostanje (socijalno i psihološko) i samopoštovanje djece. Povezana je sa lošim akademskim uspjehom i nižim kvalitetom života koje dijete doživljava.

Pretilo dijete iziskuje individualno prilagođen multidisciplinaran pristup. Dijagnoza zahtjeva temeljan klinički pregled koji identifikuje etiologiju pretilosti, prisutne komorbitide, prehrambene navike, fizičku aktivnost, socijalne, porodične i psihološke faktore koji predisponiraju pretilost, spremnost i motivaciju za liječenje. Terapija pretilosti prioritetno ima za cilj trajnu promjenu prehrambenih navika i načina života djeteta.

Ključne reči: pretilost, djeca, zdravlje

Abstract: According to the International Classification of Diseases, Eleventh Revision, ICD-11, obesity is classified as an endocrine disease, dietary and metabolic disease (ICD-XI; E66). It is a chronic multifactorial disease accompanied by abnormal or excessive accumulation of adipose tissue that poses a health risk.

The World Health Organization describes the epidemic of childhood obesity as: "One of the most serious public health challenges of the 21st century." According to the same, 41 million children under the age of 5 and 340 million children and adolescents aged 5 to 19 are overweight or obese.

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Obesity can have a significant impact on health, well-being (social and psychological) and self-esteem of the child. It is associated with poor academic performance and the lower quality of life a child experiences.

An obese child requires an individually tailored multidisciplinary approach. Diagnosis requires a thorough clinical examination that identifies the etiology of obesity, comorbidities present, eating habits, physical activity, social, family, and psychological factors that predispose obesity, readiness, and motivation for treatment. Obesity therapy primarily aims to permanently change the child's eating habits and lifestyle.

Keywords: obesity, children, health

INTRODUCTION

Definition of childhood obesity

According to the International Classification of Diseases, Eleventh Revision, ICD-11, obesity is classified as endocrine disease, dietary and metabolic disease (ICD-XI; E66).[1] It is a chronic multifactorial disease accompanied by abnormal or excessive accumulation of adipose tissue health risk.[1]

There are no consensus criteria that define childhood obesity based on excessive adipose tissue [1,2]. In Bosnia and Herzegovina (other countries including the United States), obesity in children over two years of age represents physical nutrition above the 95th percentile for age and sex (as well as above the 85th percentile for overweight).[2] In children under two years of age, the limit is 97.7 percentile.[3]

In contrast, the World Health Organization (WHO) and the United Kingdom use limit values based on statistics corresponding to the number of standard deviations above the median.[2] According to them, obesity is described as body nutrition above 2 standard deviations (standard deviation, SD), which is equivalent to the more extreme 98th percentile.[2]

The third, strictest, approach was proposed by the International Obesity Task Force (IOTF).[5] According to the same, childhood obesity is defined as like bodily uh wounding above the 99th percentile (the threshold of the body mass index in childhood is analogous to the threshold of adults).[2]

Epidemiology of childhood obesity

The World Health Organization describes the childhood obesity epidemic as: "One of the most serious public health challenges of the 21st century".[4] According to the same, 41 million children under the age of 5 and 340 million children and

adolescents aged 5 to 19 are overweight or obese.[4] About 31 million of them live in developing countries.[4,5] The prevalence of obesity has increased since 1975 by three times.[4] The share of children in the highest percentiles of the body mass index records the fastest growth.[4]

Extremely high prevalence of malnutrition and childhood obesity was found in Mexico (64.0% of girls and 54.3% of boys), slightly lower in Middle East Bahrain, 38.5% and Kuwait, 31.8%).[6,7] According to the National Health and Nutrition Examination Survey, NHANES in the United States, obesity was found in 13.9% of children aged 2 to 5 years, 18.8% of children aged 6 to 11 years, and 17.4% of adolescents aged 12 to 19 years.[6,7] A slightly more modest prevalence has been established in the countries of Southeast Asia and the Western Pacific, including India, Malaysia, Vietnam, China, Australia, South Korea, and Japan.[6,7] The lowest prevalence rate was found in African countries.[6,7]

In 2019, obesity was found in 398,000 children aged 6 to 9 in Europe.[6,7] In southern European countries such as Greece, Italy, Malta, San Marino and Spain, almost one in five children is obese.[6,7]

At the age of 5 up to 9 years, a higher prevalence rate of obesity in boys was reported by 123 of 188 (65%) countries.[6,7] Between the ages of 10 and 19, the same pattern was observed in 112 (60%) countries.[6,7] Twice the prevalence of obesity in boys was found in 44 out of 88 (50%) countries with high and higher gross national income.[6,7] This pattern is significantly rarer in countries with medium and low gross national income.[6,7]

In Bosnia and Herzegovina, obesity is present in 17.4% of newborns, 9.6 % of boys aged 5 to 9 years, 6.3% of girls aged 5 to 9 years, 5.4% of boys aged 10 to 19 years, and 3.7% of girls aged 10 to 19 years.[6,7] Without significant interventions in prevention and treatment, in Bosnia and Herzegovina by 2030, an increase in obese children aged 5 to 19 years is projected to be 67.779% (29.031% of children aged 5 to 9 years, and 38.749% of children aged 5 to 9 years). 10 to 19 years).[6]

Etiology of childhood obesity

Obesity is a chronic multifactorial disorder of energy balance in which calorie intake exceeds their consumption.[8] The etiology includes genetic, endocrine, behavioral and environmental factors.[8]

Genetic factors

Monogenetic obesity occurs as a result of harmful mutations in genes encoding leptin, leptin receptor, pro-opiomelanocortin (POMC), prohormone convertase 1

(PC1), melanocortin receptor. melanocortin 4 receptor (MC4R) and brain-derived neurotrophic factor (BDNF).[8] Large chromosomal deletions result in hyperphagia, learning disorders, behavioral disorders and severe obesity.[8]

Monozygotic mutations in the leptin gene (serum leptin concentration below 5 ng/ml) result in excessive food intake and severe obesity.[9] Leptin therapy leads to withdrawal of symptoms.[9]

Inactivating mutations in allelic leptin receptor genes can cause central hypothyroidism, increased serum corticosteroids, and increased serum stagnation in sexual development.[8]

Mutations in pro-opiomelanocortin prevent its cleavage into alpha-melanocyte-stimulating hormone or adrenocorticotropic hormone (ACTH).[8,9] The same results in hyperphagia (secondary, absence of signaling at melanocortin receptors 3 and 4), red hair (inability of the peripheral alpha-melanocyte-stimulating hormone to bind to melanocortin receptor 1), and adrenal insufficiency (insufficient adrenocortical binding).[8,9] Prohormone convertase 1 deficiency causes obesity, adrenocorticotropic hormone deficiency, postprandial hypoglycemia (insufficient pro-insulin cleavage), hypogonadotropic hypogonadism, and small bowel malabsorption.[9]

Haploinsufficiency of neurotrophic factor derived from the brain was found in obese children with WAGR syndrome (Wilms tumor, Aniridia, Genitourinary abnormalities, Range of developmental delays, WAGR).[9]

Polymorphism in the gene locus associated with fat mass and obesity associated, FTO) leads to an increase in body mass index and serum lipid concentration.[9]

Extremely low percentage of obesity occurs in genetic syndromes.[8] Prader Willi, Bardet Biedl and Alstrom syndromes are particularly significant in hyperphagia.[8] Prader Willi syndrome is accompanied by high ghrelin concentrations, Bardet Biedl and Alstrom syndromes with cilia dysfunction.[8]

Endocrine factors

Endocrine disorders are thought to cause 2 to 3% of childhood obesity.[8]

Hypothyroidism causes increased permeability of capillary walls (extravascular outflow and water retention), slowed basal metabolism (storage of calories ingested), decreased linear growth (high body mass index although weight does not exceed the 95th percentile).[18] Associated with moderate obesity (increase body mass index by 1 to 2 units).[14]

Growth hormone deficiency (GH) is accompanied by a decrease in linear growth with a continuous increase in body weight (central obesity).[8]

Cushing's syndrome (Cushing's syndrome) is characterized by extremely reduced linear growth, increased gluconeogenesis, insulin resistance, inhibition of lipolysis and stimulation of lipogenesis [8]. It is extremely rare in children (1: 1,000,000).[8]

Insulinomas are accompanied by increased insulin production, consequent hypoglycemia and excessive food intake.[8] Extremely rare : 5,000,000, less than 10% occur before the age of 20).[8]

Pseudohypoparathyroidism causes resistance to parathyroid hormone (PTH), hypocalcemia and hyperphosphatemia.[8] Children are obese, short, retarded, have a round face, short metacarpal bones and calcified basal ganglia.[8]

Behavioral factors

Behavioral factors may predispose obesity through overeating and/or decreased physical activity.[11]

Outsourcing behaviors (increased impulsivity, sensitivity to reward, poor inhibitory control, and lack of self-regulation) are associated with obesity at age.[11] These can result in compulsive overeating, emotional overeating, eating in the absence of hunger, and demanding foods rich in sugars and fat, which is supposedly tastier than healthy food.[11]

Internalizing behaviors (fears, tension, shyness, feelings of dislike, inferiority, sadness, withdrawal, and depression) lead to decreased physical activity, excessive television viewing, and Internet use.[11]

Environmental factors

The family environment plays a crucial role in the development of childhood obesity.[5,12] The probability of developing childhood obesity is 80% if both parents are obese, 40% if only one parent is obese.[5,12] Only 14% of obese children have parents of normal nutritional status.[5,12]

Dietary habits, food availability and physical activity determine the nutritional status of family members.[13] Obesity in the family is affected by frequent consumption of fast food (with a significantly higher content of fat, saturated fat and sodium, less fiber, iron and calcium than meals at home), occasions to eat outside the home, large portions, significant consumption of beverages with high sugar content, skipping breakfast, lack of sleep, excessive television viewing and use of electronic devices (computer, tablet, mobile phone).[5]

Maternal obesity is an independent predictor of the development of childhood obesity.[14] Children of single mothers consume more total fat, saturated fat and sweetened beverages, watch TV longer and use electronic devices than children living with both parents.[12] Poor eating habits in pregnancy can negatively affect intrauterine environment.[14] Obesity in the first trimester of pregnancy doubles the risk of obesity in children aged 2 to 4 years.[13]

Maternal smoking in pregnancy (1 to 12 cigarettes per day in the first trimester of pregnancy) predisposes to obesity.[14] Breast milk has a protective effect on the development of obesity in later childhood.[14] Maternal education plays a significant role in the development of childhood obesity (lower education is associated with obesity).[14]

The impact of socioeconomic status on child obesity has not been fully elucidated.[5] High socioeconomic status creates an overweight environment.[5] On the other hand, lower socioeconomic status carries significantly more psychosocial stressors and food insecurity that contributes to obesity.[5]

Parents' efforts to create a healthy food environment are jeopardized by the high-budget advertising campaign of the food industry.[15] Social networking software vouchers and messages sent via mobile phones or embedded in the content of the Internet allow companies access to children without parental supervision.[15] The environment in which children grow affects the development of obesity.[16] Proximity to large supermarkets, the concentration of fast food and restaurants, availability of recreational areas for physical activity, distance from school can have a favorable or unfavorable effect on the eating behavior and patterns of physical activity of children.[16] Increased availability of fast food (longer opening hours, delivery options and convenient locations such as shopping malls and cinemas) negatively affects the nutritional status of children.[16]

Living in unsafe areas that do not have access to safe, well-lit walking routes reduces children's physical activity [15]. Hiking or cycling to school are becoming rarer.[15] Due to the long distance from school, unsafe walking routes and fear of the disappearance of a child over 50% of parents drove their children to school.[15] Socio-cultural factors can influence the development of obesity.[15] Tendency to use food as a reward, a means of controlling others, or part of socializing increases the risk of obesity.[15]

Iatrogenic obesity in children

Iatrogenic obesity may result from the use of insulin, corticosteroids, psychotropic drugs, including antipsychotics (olanzapine and clozapine), mood stabilizers (lithium), antidepressants (amygdalin, paroxetine), anticonvulsants (gabapentin, valproate, antiproperine, carbamazepine), antihistamines and chemotherapeutics.[17]

Pathophysiology of childhood obesity

With the industrial revolution and the growth of material consumption, food becomes richer in refined fats and carbohydrates.[18] The body does not have a mechanism capable of regulatory adaptation, which results in the accumulation of adipose

tissue and an increase in body mass index.[18] The modern science seeks to try to solve the problem of obesity by defining the physiological and biochemical mechanisms of hunger and satiety.[18]

The mechanism of satiety and hunger

The lateral hypothalamus, arcuate nuclei, and ventromedial hypothalamus within the middle (tuberal) region participate in the regulation of neurohormonal appetite and satiety.[5,18,19] The arcuate nucleus (ARC) unites two neuronal populations that communicate directly with peripheral peptide hormones.[5,18,19] One neurotic population is responsible for food intake, neuropeptide Y (NPY) coexpression, and agouti-related peptide (AgRP), while the other suppresses food intake and coexpresses a cocaine-related amphetamine transcript. cocaine and amphetamine-related transcript (CART) and pro-opiomelanocortin.[5,18,19]

The paraventricular nucleus (PVN) contains thyrotropin-releasing hormone (TRH) responsible for controlling energy balance and contributing to food intake and energy expenditure.[5,18,19]

The lateral hypothalamic area (LHA) produces orexigenic neuropeptides, melanin-concentrating hormone (MCH) and orexin (OX).[5,18,19] Orexin induces food demand (motivation to consume) energy-rich foods), while the concentrating hormone melanin functions during food intake, stimulating a high-calorie diet.[5,18,19]

The dorsomedial nucleus (DMN) participates in the control of food intake.[5,18,19] Its destruction results in hypophagia.[5,18,19]

The ventromedial nucleus (VMN) is involved in reducing food intake.[5,18,19] Ventromedial nucleus lesions cause hyperphagia and obesity.[5,18,19]

Nutrient (energy) intake is biochemically explained by peptide hormones, which can be appetite stimulants and appetite suppressants.[5,18,19]
Adipokine (leptin, adiponectin and resistin) via the hypothalamus affect food intake and energy expenditure.[5,18,19]

Leptin (satiety hormone) circulates in concentrations proportional to fat mass.[5,18,19] Its deficiency causes severe obesity which can be ameliorated by peripheral application.[5,18,19]

Insulin is secreted in pancreatic cells in response to a glucose load of.[5,18,19] Increases the storage of glycogen, fat and protein.[5,18,19] Like leptin, the level of insulin in the circulation reflects a fat mass of.[5,18,19]

Resistin reduces the sensitivity of target tissues to insulin (increased concentration in obese individuals).[5,18,19]

Gastrointestinal peptide hormones are sensitive to nutrient content in the gut (coordinated changes in circulating concentrations partially mediate short-term feelings of satiety and hunger).[5,18,19]

Cholecystokinin (CCK) is released in duodenal and ileum cells after a meal.[5,18,19] Inhibits food intake and increases leptin concentration.[5,18,19] Glucagon-like peptide-1 (GLP-1) is secreted by the L cells of the interstitial mucosa of the ileum and colon after ingestion of foods rich in carbohydrates and fats.[5,18,19] Significantly reduces appetite by peripheral and central mechanisms.[5,18,19] Oxyntomodulin, OXM) are secreted by intestinal L cells during meal.[5,18,19] Mediates at the end of a meal after ingesting a sufficient amount of food.[5,18,19] Ghrelin, a hunger hormone (leptin antagonist) secreted by the endocrine glands of the stomach.[5,18,19] It has a short-term effect on the daily feeling of hunger before a meal.[46] Peptide YY3-36 (Peptide YY3-36, PYY3-36) in proportion to the amount of food ingested postprandially secrete intestinal L cells.[5,18,19] Helps suppress appetite (action slower than cholecystokinin, faster than leptin).[5,18,19] Pancreatic polypeptide (PP) regulates endocrine and exocrine pancreatic secretion, liver glycogen levels, and gastrointestinal secretion.[5,18,19]

Indirectly participants in the regulation of appetite are secretin (establishes the desired pH for the action of digestive enzymes and prevents heartburn) and gastrin (the main hormonal regulator of gastric acid secretion).[5,18,19]

Fat accumulation

Adipose tissue (AT) is an endocrine organ with a high degree of plasticity (response to body deviations and changes in the environment).[20,21] Distributed in metabolically diverse regional depots, it represents 5 to 60% of total body weight.[20] It is made up of adipocytes (the main type of parenchymal cells) and a vascular stroma fraction (SVF) that includes preadipocytes, fibroblasts, endothelial cells, multipotent cells and immune cells such as macrophages, neutrophils, lymphocytes and T cells.[20] In the form of white (visceral adipose tissue, WAT) or brown/beige adipose tissue (BAT) stores excess triglycerides, releases free fatty acids, participates in the immune response, thermogenesis, fertility and lactation.[20]

The main stores of white adipose tissue include visceral adipose tissue (VAT), subcutaneous adipose tissue (SCAT) and ectopic adipose tissue (EAT).[20,21] Subcutaneous fat is located under the skin (more than 80% of total body fat) and includes gluteal, femoral and abdominal (superficial and deep) adipose tissue.[20] Visceral fat (10% to 20% of total body fat) is located intra-abdominal around the internal, especially digestive organs.[20] Includes omental fats associated with the stomach, mesenteric fats associated with the small intestine and epiploic fats around the colon.[20]

Ectopic fat stores (intramyocellular fats, intrahepatocellular fats, myocardial fat and pancreatic tissue) excess lipids.[20,21]

Accumulation of visceral and deep subcutaneous adipose tissue is an independent risk factor for metabolic and cardiovascular disorders associated with obesity (insulin resistance, non-insulin-dependent diabetes mellitus, high triglycerides, low

HDL cholesterol, hypertension, metabolic syndrome and carcinoma-carcinoma-surface).[20] Superficial and gluteo-femoral adipose tissue has a protective role.[20,21] Ectopic adipose tissue plays a significant role in the development of endothelial dysfunction.[21] Lipolysis, increased sensitivity to catecholamines, transport of free fatty acids via the portal vein in the liver, accumulation of inflammatory cells, changes in adiponectin production, abnormalities in γ-peroxisome signaling are cited as responsible pathophysiological mechanisms. activated receptor proliferator, lower angiogenetic capacity and hypoxia.[20]

Regardless of body mass index, each person has a critical visceral adipose tissue threshold (CVATT) associated with metabolic syndrome.[20,21] On the other hand, there is the metabolically obese normal weight (MNO).[21] Insulin sensitivity is thought to be related to fat cell size.[21] A marked increase in body nutrition at a younger age leads to early fat cell hypertrophy and a higher incidence of metabolic syndrome in children.[21]

Complications of childhood obesity

Obesity can have a significant impact on children's health, well-being (social and psychological) and self-esteem.[22] It is associated with poor academic performance and the lower quality of life a child experiences.[22]

Diseases and conditions associated with obesity

Metabolic and cardiovascular complications of obesity (primarily dyslipidemia, hypertension, and insulin resistance) are more prevalent in childrens.[23-26]

Obese children often have an abnormal lipid profile with high concentrations of triglycerides and low levels of high-density lipoprotein cholesterol (HDL cholesterol).[23] Low-density lipoprotein cholesterol (LDL cholesterol) levels are usually normal, but qualitative changes can occur (they become smaller, denser and more atherogenic).[23]. Obese children are three times more likely to develop hypertension than children with normal nutrition.[23] Adipose tissue dysfunction, associated with an imbalance in pro-inflammatory and anti-inflammatory activities of adipocytes is the most important pathophysiological mechanism.[24] Childhood obesity can cause subclinical atherosclerosis with thickening of the arterial wall and compromised arterial elasticity.[25] Changes in myocardial geometry and function (enlargement of the left and right ventricles, thickening of the left ventricular wall, increase in left ventricular mass) indicating the early onset of potentially adverse changes in the myocardium.[26] Intramiocellular fat deposition and a relatively high percentage of visceral fat relative to subcutaneous abdominal fat has been associated with significant insulin resistance in obese children.[27] Insulin resistance combined with relative insulin deficiency in obese

children may result in the development of insulin-independent diabetes mellitus type 2 (T2DM).[27] Patients may develop microvascular and macrovascular complications at a young age (compared to those diagnosed in adulthood).[28] The occurrence of type 1 diabetes mellitus (T1DM) in obese children explains the "accelerator hypothesis" "(being overweight accelerates insulin resistance, resulting in type 1 diabetes mellitus in a genetically predisposed child).[29]

Obese children often develop sleep apnea [30]. It occurs as a result of adenotonsillar hypertrophy, altered neuromuscular tone and excessive mechanical load on the chest wall.[30] Central obesity and consequent insulin resistance often favor the development of nonalcoholic fatty liver disease (NAFLD).[31] Nonalcoholic steatohepatitis (NASH), fibrosis and cirrhosis are significantly less common in obese children.[31] Although gallstones are less common among obese children and adolescents, almost 50% of cholecystitis cases in adolescence are associated with obesity.[32]

Obese children are about 30% more likely to develop bronchial asthma.[33] The disease is affected by increased concentration of inflammatory mediators in the circulation, oxidative stress, the mechanical load on the chest wall, narrowing of the airways and the presence of obesity comorbidities.[33]

Obese children tend to be taller, advanced in bone age, and mature earlier than normally fed children.[34] Childhood obesity alters bone metabolism resulting in an increased risk of fracture (avulsive injury at puberty, vertebral fracture in early adulthood), change in pattern and severity of fractures, conservative and operative treatment (increased risk of dislocations and postoperative complications).[34] Increases the risk of Blount's disease (varus deformity of the tibia) and slipped capital femoral epiphysis (SCFE) and medial rotation of the affected joint).[34]

Obese children, even in the absence of diabetes, may have glomerulomegaly, thickening of glomerular basement membranes, the proliferation of the mesangial matrix, focal segmental glomerulosclerosis (FSGS) and increased microalbuminuria marked as obesity-dependent glomerulopathy (ORG) in both normotensives and hypertensive.[35]

In obese children, an increased incidence of gastroesophageal reflux disease (GERD) and irritable bowel syndrome (IBS) was found to be.[36]

With its effects on the production and bioavailability of sex steroids, follicle development and peripheral steroid metabolism, obesity predisposes to the development of polycystic ovary syndrome (PCOS).[37] The coexistence of obesity and polycystic ovary syndrome in adolescence is significantly associated with higher concentrations. menstrual dysfunction.[37] Obesity can accelerate puberty in girls and delay puberty in boys.[37]

There is a significant association between childhood obesity and several malignancies in adulthood, including leukemia, Hodgkin's disease, colon cancer and breast cancer.[38]

Idiopathic endocrine rare but potentially serious condition that may cause permanent vision loss.[39] Prevalence and risk of recurrence increase with obesity.[39] Obese children have a higher prevalence of episodic or pon migraines, daily chronic headaches and tension headaches.[39]

Social and psychological consequences

Obese children are often teased and/or abused for their weight of.[40] They face a number of other difficulties, including negative stereotypes, discrimination and social marginalization.[40] They possess low social competence, are shy, prefer the company of adults and avoid participating in activities with schoolmates.[40] Obese children have fewer friends than children with normal body weight, resulting in less social interaction, lack of play, and significant time spent in sedentary activities.[40] They seek protection from negative comments and attitudes in safe places, such as their homes, where they spend time in inactivity and/or eating.[40]

Obesity is often accompanied by psychological comorbidities, including depression, attention-deficit hyperactivity disorder (ADHD), anxiety, and oppositional defiant disorder (ODD).[41] Obesity and depression are considered inflammatory conditions characterized by increased serotonin concentration, impaired stress regulation, and impaired glucocorticoid negative feedback.[42] There are significant overlaps in neurobehavioral circles between obesity and attention deficit hyperactivity disorder.[42]

Psychological comorbidities are predisposed to the presence of eating disorders including bulimia nervosa, compulsive binge-eating disorder (BED) and night-eating syndrome. NES).[42]

Academic implications

Childhood obesity has also been found to negatively affect school performance.[40] Obese children are four times more likely to develop incidental situations at school than their normal-weight peers.[40] More frequent absences from school (primary children with comorbid diabetes and asthma) can negatively influence academic results.[40]

Quantification of body fat in obese children

The choice of method for quantification of body fats and definition of their distribution takes into account the costs of use, the level of personal skills required for proper application, the time required for performance, the susceptibility of the population and possible health risks.[5,43]

Anthropometric measurements

Anthropometry is the most commonly used method, due to its ease of execution, low cost, and inexorability.[5,43] It is recommended to measure the weight with an anthropometric scale in kilograms (child without shoes, with light clothes, in orthostatic position in the middle of the scale), height with a stadium meter in centimeters, to the nearest 0.1 cm, (child without shoes, back to the wall, parallel, connected feet, in an upright position and looking forward in the horizon line).[5] Body mass index (BMI) is obtained by dividing body weight in kilograms and height in square meters.[43] Stratification of nutritional status is obtained from the percentile in relation to body mass index, age and sex.[43]

Percentiles are correlated with total body fat, especially with the amount of internal fat.[43] It does not distinguish fat from lean mass, which makes it difficult to distinguish obesity with excess fat from that with muscle hypertrophy.[43] In addition, does not determine the distribution of body fat and does not reflect growth retardation (common among children of low socioeconomic status sa).[43]

Waist Circumference, (WC) is measured in centimeters using a flexible and inelastic measuring tape (child in orthostatic position, underclothing, at the end of a normal exhalation, midway between the iliac crest and the lower edge of the arch posterior rib).[43] Represents a significant predictor of visceral obesity and benefits in the identification of high-risk children for the development of chronic non-communicable diseases in adulthood.[43] It is non-invasive, requires simple and inexpensive equipment, easy to use, and very affordable, can be used as an alternative or addition to determining the percentile of children.[43]

The hip ratio (HR) is measured at the widest part of the hip at the level of the large trochanter of the femur. Waist-to-hip ratio (WHR), although correlated with visceral fat, is rarely used in children due to constant growth and the inability to create criteria and curves.[5,43] Skinfold thicknesses it is measured with a caliper in millimeters on the dorsal side of the upper arm above the triceps, below the lower edge of the shoulder blade, at the height of the navel and 2 cm lateral to it.[5,43] It is a quick, simple, inexpensive method that provides useful information on body fat distribution.[5] In combination with the body mass index, it has a higher predictive value of.[43] Anthropometric measurements, although common and simple procedures, must be performed carefully, with standardization and calibrated measuring instruments.[43]

Bioelectric impedance

Bioelectric impedance assay (BIA) is based on the significantly higher electrical resistance of adipose tissue compared to other tissues in the body.[5] The measurement procedure is simple, two electrodes are placed on the hand and wrist, two on the foot

and ankle.[5] Although popular, it is not recognized in academic circles due to high variability (results are influenced by meals, physical activity and other factors that change the state of hydration of subjects, such as menstrual cycle, acute illness, kidney disease, water and electrolyte imbalance).[5]

Other methods

Hydrodensitometry, dual-energy x-ray absorptiometry (DEXA), densitometry, computed tomography (CT), nuclear resonance nuclear magnetic resonance, NMR) are used for research purposes.[4]

Access to an obese child

An obese child requires an individually tailored multidisciplinary approach.[1] Prospective, longitudinal monitoring of child growth and development is the first prerequisite for objective diagnosis of obesity.[1] Diagnosis requires a thorough clinical examination (history, inspection, physical examination) that identifies the etiology of obesity (primary or secondary obesity), comorbidities, eating habits, physical activity, family and psychological factors that predispose obesity, readiness and motivation for treatment.[1]

Secondary obesity

Secondary (endocrine, hypothalamic, genetic, iatrogenic) obesity is indicated by: the development of obesity before the age of five and/or its rapid progression (primarily in the presence of signs of genetic etiology of obesity), continuous and/or rapid weight gain associated with decreased height or short stature, retarded cognitive development, dysmorphic features, use of drugs that cause hyperphagia (corticosteroids, sodium valproate, risperidone, phenothiazines, cyproeptadine).[3,39] Obesity that occurs early in a child with delayed psychomotor development, cognitive impairment, short stature, cryptorchidism or hypogonadism, dysmorphisms and facial features, eye and/or auditory changes, indicates a syndromic form of obesity.[3,39]

Comorbidity screening

It is recommended to measure the blood pressure of all obese children at the age of three years.[39,44] At the age of less than three years, blood pressure measurement is recommended for: history of neonatal complications, heart malformations, genetic

diseases, acquired or congenital kidney diseases, neoplasms, use of drugs (corticosteroids, erythropoietin, theophylline, beta-stimulants, cyclosporine, tacrolide, antipsychotics, monoamine oxidase inhibitors, nasal decongestants, oral contraceptives and androgens), diseases that cause increased intracranial pressure.[39,44] The diagnosis of hypertension requires further laboratory processing (urea, creatinine, glucose, lipids and electrolytes in the blood, urine examination, microalbuminuria), measurement of glomerular filtration and echocardiography.[39,44]

Fasting blood glucose measurement (as an initial step in the diagnosis of diabetes mellitus) is recommended for obese children aged 6 years.[39] The measurement is repeated after three years, except in the case of accelerated weight gain and/or development metabolic and cardiovascular comorbidities.[39] Oral glucose tolerance test (OGTT) is advised at the age of ten or at the beginning of puberty.[39] There is no consensus on the use of glycosylated hemoglobin (HbA1c) age.[39] Diagnosis of diabetes mellitus requires assessment of autoimmune markers (extremely genetic screening) in the exclusion of type 1.[39]

Measurement of cholesterol and triglycerides is recommended for obese children aged 6 years.[39] Measurement is repeated after three years, except in the case of accelerated weight gain and/or or the development of metabolic and cardiovascular comorbidities.[39]

Transaminase measurement and liver ultrasound are recommended for obese children aged 6 years.[39] Persistence of hepatic hyperechogenicity, ie non-alcoholic fatty liver disease and/or elevated alanine aminotransferase after weight loss requires further diagnostic processing (exclusion of viral hepatitis, Wilson's disease, autoimmune hepatitis, alpha 1 anti-trypsin deficiency).[39] There is no evidence to recommend screening for cholelithiasis.[39]

The diagnosis of gastroesophageal reflux disease is made using the standardized questionnaire (Reflux Disease Questionnaire, RDQ).[39] Persistence or worsening of symptoms (pyrosis, heartburn, regurgitation) after weight loss requires further diagnostic processing (gastrointestinal contrast study, endoscopy and pH monitoring, esophageal impedance).[39] Components of polycystic ovary syndrome, hyperandrogenism (acne, hirsutism and alopecia) and ovarian dysfunction (oligomenorrhea, amenorrhea) should be considered in obese adolescents.[39] Treatment failure requires further diagnostic processing (exclusion of congenital adrenal hyperplasia and androgen-secreting tumors, Cushing's syndrome/diseases).[39]

Obese children require evaluation of symptoms and signs of respiratory diseases (rapid breathing, dyspnea after moderate exertion, whistling, chest pain, hypoxemia, hypercapnia, snoring / noisy breathing, pauses in breathing, mouth breathing, frequent waking, morning persistent and daytime headaches, daytime sleepiness, inability to concentrate, poor academic performance, hyperactivity, cognitive deficits, anamne-

stic data on atopy) including asthma, obstructive sleep apnea syndrome (OSAS) and obesity hypoventilation syndrome (OHS).[39]

In the presence of musculoskeletal pain and limited movement of the joints of the lower extremities, screening of orthopedic complications of obesity is recommended.[39] Despite the increased prevalence of fractures, osteodensitometry is not recommended.[39]

Children need to determine the presence of vomiting headache, photophobia, transient blurred vision and diplopia.[39]

Unsatisfactory body image, symptoms of depression and anxiety, loss of control over diet, weight concerns, dysfunctional social relationships, inactivity due to problematic body image, stigma associated with obesity, low self - esteem and poor academic failure of obese children require psychiatric evaluation.[39]

Prevention of childhood obesity

Prevention of childhood obesity requires a multidisciplinary approach.[5,45]

Primary prevention is aimed at the individual, but also at the entire population with optimal body weight to promote, educate and adopt healthy eating habits.[45] It begins preconception and prenatally, continues in preschool and school age.[5] In the prevention of childhood obesity, education of pregnant women about the protective effect of breastfeeding and maintaining optimal body weight is of great importance.[45] Primary prevention of obesity during preschool age is aimed at adopting healthy eating and living habits (eating in a certain place and at a certain time, regularity of meals, varied diet in accordance with energy and nutritional needs, encouraging daily physical activity, time limit for watching television, playing video and computer games).[45] At school age, it is important to maintain healthy eating habits, regular physical activity, limit the use of restrictive diets, prevent smoking, consume alcohol, narcotics, unhealthy and uncontrolled dietary supplements.[45] Primary obesity prevention is a lengthy, comprehensive process that requires the active participation of the entire socio-political community.[5,45] Secondary prevention is targeted at groups at risk for developing obesity.[5,45] It includes children with a sedentary lifestyle, a history of obesity in the family, endocrinopathies and syndromes that predispose to obesity.[45] Secondary prevention includes regular anthropometric monitoring, clinical and laboratory supervision.[5,45] Tertiary obesity prevention is aimed at children with a body mass index above the 97th and 99th percentiles, in whom there are consequences of obesity.[5] A multidisciplinary approach is aimed at preventing and monitoring the further deterioration of obesity comorbidities.[45]

The obesity prevention program requires a multidisciplinary, uniform approach of the family, all levels of the education and health system, with emphasis on the promotion and adoption of healthy eating and living habits.[45] The implementation of

the prevention program requires active participation of relevant ministries, the wider community, media, industry.[45]

Childhood obesity therapy

Obesity therapy primarily aims to permanently change the eating habits and lifestyle of the child.[39] Optimal therapy provides: maintaining an appropriate growth rate and balanced weight-to-height ratio, weight loss without necessarily achieving ideal weight (with emphasis on reducing fat while maintaining muscle mass), maintenance or promotion of good mental health (self-esteem, correct attitude towards food and body image), treatment of obesity complications as soon as possible, prevention of relapse.[39]

With growth, there is a normalization of the relationship between body weight and height, and often a slight reduction in caloric intake may be effective if the child and his family are motivated to change their diet.[39]

Diet

Obese children should avoid energy-rich but nutritionally poor foods and beverages (fruit juice, sports drinks, fast foods, snacks, confectionery).[5,46,47] It is recommended to control caloric intake, reduce the consumption of saturated fats, sugars and salts, increase the intake of whole grains, low-fat proteins, fruits and vegetables, optimal intake of micronutrients (iron, calcium, zinc, copper, magnesium, folic acid, vitamins) [5,46,47,] age requires a diet with a balanced ratio of carbohydrates, fats and proteins.[5] The overall reduction in caloric intake has a much stronger effect on weight loss than selective reduction of carbohydrates or fats.[5,48] The diet of obese children requires harmony between reducing caloric intake (weight reduction) and maintaining normal growth and development.[5,48] Immoderate restriction calories are contraindicated in childhood, because it can slow down the growth and development of a child.[5]

Physical activity

Permanent weight loss in addition to reducing caloric intake also requires increased physical activity.[5,39] Physical activity contributes to weight reduction, results in improvement of overall body composition, reduction of blood pressure, LDL-cholesterol, triglycerides, insulin sensitivity and enables psychological well-being.[5] Intensity interests and needs of the child.[47] The frequency of activities must be moderate (moderate onset and gradual increase).[47] Obese children are recommended recreational

activities and sports that involve a large group of muscles such as swimming, football, basketball, volleyball, handball, rugby or require anaerobic and neuromuscular strength, such as gymnastics or judo.[39] It is advisable to avoid exercises with constant load or repeated impact on the child's legs, feet and hips.[39]

Pharmacological therapy

Pharmacological therapy is used in severely obese children with cardiac, metabolic, hepatic or respiratory comorbidities in whom interventions based on dietary and lifestyle habits did not lead to weight loss.[39] Orlistat (tetrahydro-lipstinate) is the only drug approved for the treatment of obesity in childhood.[39] Orlistat inhibits intestinal lipases and reduces gastrointestinal fat absorption by 30%.[49] In randomized, controlled studies, its use has been associated with significant weight loss and cardiovascular risk reduction.[49] It is absorbed in small amounts and is considered quite safe.[49] Side effects include bloating, diarrhea, malabsorbable stools, decreased vitamin D levels and increased bone resorption.[49]

Surgical treatment

Bariatric and metabolic surgery (gastric obstruction with adjustable silicone tape, gastric bypass, sleeve resection, and Scopinar surgery) are the ultimate solution in therapeutically resistant severe obesity accompanied by serious complications.[5,39] In addition to a multiple body mass index, surgical treatment requires skeletal maturity or stage IV puberty according to Tanner, confirmation of the ability to change eating habits and lifestyle and a stable psychosocial environment.[50] Medically solvable cause of obesity, documented substance abuse problem or planned pregnancy, breastfeeding, and unwillingness to understand the consequences of the procedure delay treatment.[50] Surgical therapy allows for greater weight loss in the long run (50% to 60% in the first year and up to 75% by the end of the second year) compared to other treatments.[50] Complications include gastrojejunal anastomotic stricture, anastomotic leakage, dehydration and the nutritional deficiency (lack of protein, calcium, hydro and liposoluble vitamins).[50]

Behavioral therapy

Behavioral therapy provides an individual approach to the treatment of obesity (analysis of previous attempts to reduce body weight, degree of motivation, social and family predictors of obesity).[5] It solves gradually isolated problems (not all at

once), sets realistic and accessible goals, reduces stressful situations thus facilitating weight loss.[5] Social support is provided by group therapy in which people with similar problems participate.[5]

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