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SOMATIC AND PSYCHOLOGICAL COMPLICATIONS OF OBESITY IN THE PEDIATRIC POPULATION: CONTEMPORARY CHALLENGES FOR CLINICAL PRACTICE – A SYSTEMATIC REVIEW

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ABSTRACT

Obesity in children is a pandemic of the 21st century. It is a clinically significant problem and one of the most alarming health issues observed in children worldwide. Overweight and obesity result from the interaction of environmental, genetic, iatrogenic and endocrine factors, and their consequences significantly affect overall quality of life. Over the past few decades, it has been shown that obesity in young people influences the future development of diseases such as metabolic, respiratory, cardiovascular, endocrine and psychosocial disorders. This article focuses on the consequences of obesity in children and adolescents, including hypertension, obstructive sleep apnoea, insulin resistance, type 2 diabetes, dyslipidaemia, NAFLD, endocrine disturbances such as premature puberty or PCOS, and a range of mental health issues. We summarise the most important short- and long-term effects of obesity in children and adolescents and highlight the importance of early diagnosis and a multidisciplinary therapeutic approach to prevent short- and long-term complications.

KEYWORDS

Obesity, Childhood Obesity, Obesity Consequences, Hypertension, Dyslipidaemia, Polycystic Ovary Syndrome

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1. Introduction

1.1 Definition and significance of the problem

Obesity is defined as a disease characterised by the excessive accumulation of adipose tissue caused by a long-term positive energy balance. The scale of the problem continues to grow. In 2016, 340 million children worldwide were overweight, including 124 million who were obese [12]. Diagnosis is usually based on measuring height and weight, and then calculating the weight-to-height ratio in children under five years of age, and the BMI in older children. According to the WHO, in children under 5 years of age, overweight is diagnosed when this ratio is 2 SD above the median, while obesity is diagnosed when it is above 3 SD. In children aged 3-18, overweight is defined as a BMI deviation above the 85th percentile, and obesity above the 97th percentile [3, 13]. It is also worth mentioning that BMI is an imperfect indicator – it does not take into account the proportion of adipose tissue in total body weight, which means that in people with developed muscle tissue, the result may be falsely elevated. The distribution of body fat is also important in terms of metabolic complications – visceral fat is more significant for the risk of developing metabolic complications. BMI is a useful screening tool; however, changes over time and comparison with percentile charts are more informative than a single measurement, and the diagnosis of obesity should be supplemented with anthropometric and biochemical tests or body composition assessment.

1.2 Main causes of obesity in the pediatric population

The most common form of childhood obesity is simple obesity, which is linked to poor eating habits established early in life and reduced physical activity. In recent years, a number of genetic factors have been discovered that cause obesity. An important role is also played by disturbed circadian rhythms associated with excessive use of electronic devices and sleep disorders, mental and emotional disorders, and chronic stress [11]. In recent years, increasing attention has been focused on the role of the gut microbiota in regulating the body's energy balance. In children under 2 years of age with hyperphagia, genetic causes of obesity can be found, such as congenital leptin deficiency, proopiomelanocortin deficiency, prohormone convertase-1 deficiency, melanocortin 4 receptor, brain-derived neurotrophic factor (BDNF), angiopoietin-like protein 8 (ANGPTL8), and tyrosine kinase receptor [8, 10, 13, 14]. Another aspect is the occurrence of obesity in Prader-Willi syndrome, as well as in Bardet-Biedl, Alstrom, and Cohen syndromes. The differential diagnosis should include various endocrine diseases, as well as iatrogenic causes related to the chronic use of glucocorticosteroids and atypical neuroleptics.

2. Methodology

The aim of this article was to analyze current data on obesity complications in children and discuss their clinical significance, as well as to systematize prevention options and indicate new directions for therapeutic possibilities. This article was written based on publications available in the PubMed and Google Scholar databases. To ensure the accuracy of information, we searched and analyzed medical literature published between 2010 and 2025. The search was conducted using keywords such as childhood obesity, health consequences, type 2 diabetes, metabolic complications, and hypertension. The selection process included both review and original research articles. Duplicates, papers not directly related to the subject, and publications unavailable in full text were excluded. Publications in languages other than English were excluded. Particular emphasis was placed on the most recent and methodologically sound publications. The analysis was conducted taking into account the most common complications, focusing on the mechanisms of their development, their clinical significance, and referring to the possibilities of their prevention and treatment.

Studies were included in the final analysis if they met criteria for methodological robustness, clinical significance, and alignment with the objectives of the review.

3. Overview of obesity complications

Obesity is a systemic disease whose consequences can affect most organs and systems in the human body. Excess adipose tissue is a hormonally active tissue that secretes substances affecting the body's homeostasis, and oxidative stress and chronic inflammation are the main causes of complications [16]. Currently, children of all ages are considered to be at risk of the long-term consequences of obesity. Modern diagnosis of these consequences is not solely based on identifying diseases typical of obese adults.

3.1 Metabolic complications

Metabolic complications constitute a large group of somatic consequences and include hyperglycaemic conditions such as type 2 diabetes, abnormal fasting blood glucose and abnormal glucose tolerance. Insulin resistance is the central link in the vicious circle of mechanisms leading to type 2 diabetes, non-alcoholic fatty liver disease (NAFLD), and dyslipidaemia. Adipose tissue secretes leptin and resistin, and the concentration of free fatty acids and interleukin 6 also increases. These substances, together with ectopic lipid deposition, reduce peripheral tissue sensitivity to insulin by increasing hepatic gluconeogenesis and reducing glucose uptake in skeletal muscles [7]. Initially, there is a compensatory increase in blood insulin concentration; however, prolonged stimulation can lead to dysfunction in insulin secretion and the development of prediabetes, which can progress to diabetes. Obese children may also experience reactive hypoglycaemia, which is associated with excessive insulin secretion in response to carbohydrate intake. This leads to hypoglycaemia, which is asymptomatic in most patients [17].

Children affected by obesity often develop dyslipidaemia, most commonly of a mixed nature. This is also the most common complication in overweight children. Adipose tissue insulin resistance causes increased lipolysis and elevated free fatty acid levels. These are substrates for the production of triglycerides and their conversion to triglyceride-rich lipoproteins and very-low-density lipoproteins (VLDL), which are then deposited in the walls of blood vessels. This is the first stage in the formation of atherosclerotic plaques. Additionally, elevated VLDL levels inhibit lipolysis in the liver, resulting in hypertriglyceridemia [15]. This process can develop from a very young age and lead to myocardial infarction, stroke and peripheral artery disease. High-density lipoprotein (HDL), on the other hand, prevents the formation of atherosclerotic plaques by inhibiting the oxidation of low-density lipoprotein (LDL) cholesterol [13]. The risk of cardiovascular complications increases with the development and persistence of obesity, especially when accompanied by hypertension, type 1 diabetes or familial hypercholesterolemia. Hyperuricaemia may also play a significant role in the development of insulin resistance [1].

Obesity and being overweight are the main causes of hypertension in children and adolescents. Hypertension should be diagnosed when the average result of three measurements taken during three separate visits exceeds the 95th percentile for the child's age, gender, and height. Hypertension has a multifactorial basis and is primarily associated with hyperinsulinemia, an imbalance between pro-inflammatory and anti-inflammatory factors, vascular endothelial dysfunction and increased activity of the renin-angiotensin-aldosterone (RAA) and sympathetic systems [18]. Hypertension may also occur as a clinical manifestation of renal dysfunction caused by hyperfiltration in the glomeruli [18]. Persistent hypertension leads to the progression of cardiovascular diseases.

The term 'metabolic syndrome' refers to the entirety of changes that occur during the development of subsequent obesity-related complications in children. However, there is no systematic definition of metabolic syndrome in children, due to the overlap between obesity and the growth and development period of the child. Most researchers include the following components: acanthosis nigricans, prediabetes, hypertension, dyslipidaemia and non-alcoholic fatty liver disease. It is crucial to identify obese patients and perform additional tests to diagnose possible components of metabolic syndrome and prevent the development of other complications associated with excess weight [10].

The most common obesity-related gastrointestinal manifestation is non-alcoholic fatty liver disease (NAFLD), which is caused by the accumulation of triglycerides in hepatocytes associated with hyperinsulinemia. The gold standard for diagnosis is a liver biopsy; however, due to its invasive nature, ALT measurement and ultrasound examination are performed in children. NAFLD is diagnosed based on the detection of fatty liver in imaging tests and the presence of overweight/obesity, type 2 diabetes, or metabolic disorders (two of the following: waist circumference or blood pressure above the 95^(th) percentile, elevated triglyceride levels, reduced HDL, prediabetes, elevated HOMA-IR index, or elevated CRP) [1, 19]. Storage and infectious diseases that may mimic fatty liver disease should also be ruled out [13]. The risk of developing end-stage liver failure and consequently cirrhosis is lower in children with fatty liver disease than in adults.

In recent years, there has been increasing attention paid to the role of obesity in the development of gallstones, which can be influenced by weight gain or sudden weight loss. Gallstones are usually asymptomatic and are typically discovered during an ultrasound scan. Increased body weight also contributes to the development of gastro-oesophageal reflux, the symptoms of which worsen with increasing BMI [15].

The main factors impairing kidney function in children are overweight, obesity, type 2 diabetes, hypertension, dyslipidaemia and autonomic dysfunction, but their impact is not as clear as in adults [13, 20]. Obesity, on the other hand, is an independent risk factor for chronic kidney disease. Metabolic and cardiac complications in particular play a role in impairing nephron function. Obesity is a cause of focal segmental glomerulosclerosis. Excessive body weight probably contributes to micturition disorders in children, primarily urinary incontinence [13]. Beyond metabolic complications, excess weight also affects the musculoskeletal system.

3.2 Damage to the musculoskeletal system

Obesity in children and adolescents can have a significant impact on the musculoskeletal system due to the body's dynamic growth and development during this period. The most common problem is skeletal overload, which is associated with excessive mechanical stress and can result in pain in the lower limbs and spine, as well as a reduced range of motion in the joints. Limited physical activity and a sedentary lifestyle exacerbate these issues, increasing the likelihood of developing osteoarthritis in the future, particularly in the knee joint.

In overweight children, the risk of injury increases when they start to move around. Postural and limb defects are often observed, particularly knee valgus, flat feet and hyperlordosis. Impaired calcium-phosphate metabolism and vitamin D deficiency also increase the risk of bone fractures [1]. An imbalanced diet containing too much highly processed food can lead to hyperphosphatemia and hypocalcemia. Due to the excessive stress placed on the joints, there has also been a higher incidence of Blount's disease and femoral head exfoliation reported [4].

3.3 Respiratory disorders

Obstructive sleep apnoea is much more prevalent in obese children. OSA encompasses a range of sleep-related issues, including partial or complete blockage of the upper airway and reduced airflow through the upper airway. It is diagnosed in children when respiratory movements are present but airflow through the nose is not maintained. This manifests as mouth breathing and snoring. Symptoms associated with hypoxia and hypercapnia, such as excessive daytime sleepiness, fatigue, confusion, concentration difficulties, headaches and psychomotor hyperactivity, may be observed during the day. Polysomnography remains the gold standard for diagnosis. The cause-and-effect relationship associated with obesity can be seen in changes to breathing mechanics, such as larger fat pads that can act as a barrier to airflow, excessive chest weight and reduced chest mobility. Positive pressure can also close the throat and cause the airways to collapse during sleep. In recent years, the influence of gut microbiota and Th17 lymphocytes on the aetiology of OSA has been demonstrated [14]. Obstructive sleep apnoea causes hypoxia, which increases the risk of hypertension, left ventricular hypertrophy and pulmonary hypertension. Impaired alertness and reduced attention increase the risk of falls

and impair concentration, affecting academic performance. The coexistence of obstructive sleep apnoea and obesity significantly increases the risk of future cardiovascular events [10].

Children who are overweight are also more likely to suffer from hypoventilation syndrome, which primarily occurs in severe obesity. Hypoventilation leads to hypoxia and hypercapnia, resulting in headaches, impaired concentration, confusion, excessive irritability and behavioural disorders [13].

Reduced physical fitness can cause shortness of breath, an inability to catch one's breath after even minor physical exertion, wheezing and chest pain. Diagnoses of bronchial asthma and a deterioration in the management of the condition were also reported more frequently [2]. Excess weight has an impact on the development of bronchial asthma, which is a chronic inflammatory condition that induces the secretion of cytokines and blood cells involved in bronchial tree hypersensitivity. The combination of these factors with reduced lung compliance and bronchial hyperresponsiveness may result in poorer asthma control and an increased risk of exacerbations [13].

3.4 Endocrine disorders

Adipose tissue is a hormonally active organ that secretes various substances, including adipokines and cytokines. These substances affect the body's hormonal balance and individual organs. One of the main substances secreted by adipocytes is leptin, which reduces appetite, increases satiety and stimulates the secretion of gonadotropins. Paradoxically, leptin levels are elevated in obesity, while tissues show leptin resistance. Decreased adiponectin levels are associated with increased insulin resistance, inflammation and oxidative stress. Conversely, elevated resistin levels negatively affect the insulin signalling pathway. Obesity is also a form of chronic inflammation, with interleukin 6, tumour necrosis factor, visfatin and chemerin, among others, being secreted to stimulate this process. For this reason, obesity is now considered an inflammatory disease as well as a metabolic disorder, in which immunological factors and active substances play an important role.

A significant increase in the reported rates of type 2 diabetes and obesity has been observed, which is largely associated with insulin resistance and hyperinsulinemia and subsequent degeneration of pancreatic beta cells. Research has demonstrated that the younger the child diagnosed with obesity, the faster and more severely the beta cells are degraded. Oxidised low-density lipoproteins, hyperglycaemia, and elevated levels of free fatty acids play a major role in the pathogenesis of these changes [2]. Glucagon production and secretion are also disrupted. Elevated glucagon levels are caused by alpha cell resistance to the inhibitory effects of insulin, which exacerbates insulin resistance [9].

Obesity is recognised as a contributing factor to premature puberty, defined as the onset of puberty before the age of 8 in girls and 9 in boys. The relationship between obesity and premature puberty has been well documented in girls, but its impact on boys is not as well understood [10]. Leptin, secreted by adipose tissue, and kisspeptin, whose levels are elevated, play a role in the onset of premature puberty. It has been observed that there is a higher incidence of premature isolated adrenarche and pubarche.

The impact of obesity on the development of PCOS is significant. Insulin resistance and hyperinsulinemia are key factors in this process, as they stimulate the ovaries and adrenal glands to produce androgens. This, in turn, enhances the action of aromatase, which converts androgens into estrogens, and reduces SHBG synthesis. Research has also demonstrated a link between premature adrenarche in obese girls and the development of PCOS [10]. Hyperandrogenism in girls can lead to severe acne, hirsutism, premature pubarche and axillarche. Furthermore, obese girls are prone to irregular menstrual cycles and cycle disorders, which may include heavy, painful periods or very light bleeding, and even secondary amenorrhea. It is not uncommon to observe oligomenorrhea, otherwise known as no bleeding for 90 days. It is imperative to note that menstrual cycles that last less than 21 days or more than 45 days in older girls, and 21 and 35 days, respectively, require attention (depending on maturity level) [13]. In contrast, elevated estrogen levels are the primary cause of gynecomastia and fertility disorders associated with impaired spermatogenesis. Furthermore, it has been established that reduced levels of IGF-1 and testosterone are the cause of growth retardation in obese boys [2, 9].

One of the most common complications of obesity is a disorder of the hypothalamic-pituitary-thyroid axis. There are several concepts explaining thyroid dysfunction, in which leptin plays a major role by increasing TRH and TSH concentrations. Furthermore, increased TSH levels can be attributed to chronic inflammation, as evidenced by the inhibitory effect of cytokines on iodine transport to thyrocytes, consequently impairing thyroid hormone synthesis [9].

Adrenal axis dysfunction can present a number of diagnostic challenges due to its similarity in clinical features to Cushing's syndrome. Excessive cortisol secretion by the adrenal glands is often the result of excessive activation of the hypothalamic-pituitary-adrenal axis. However, in contrast to Cushing's syndrome, both cortisol concentration and its circadian rhythm are preserved, and the dexamethasone suppression test result is also normal. Excessive activation of the renin-angiotensin-aldosterone system at the systemic level and in local fat cells causes hyperaldosteronism, which may lead to developing secondary hypertension. Excessive activation of the adrenal glands can lead to their hypertrophy, resulting in increased secretion of cortisol and aldosterone, which in turn intensifies the metabolic-hormonal vicious circle [9].

It has been established that the function of gastrointestinal hormones, which play an important role in energy metabolism and appetite regulation, is also disrupted. Following a meal, the level of ghrelin, a hormone that stimulates hunger, does not decrease. Reduced GLP-1 secretion results in increased appetite and accelerated gastric emptying. The introduction of GLP-1 agonist drugs to the pharmaceutical market proved revolutionary, as they reduce excess body weight by suppressing appetite and slowing down gastric emptying. Concurrently, reduced PYY levels have been shown to increase appetite.

The impact of obesity on the pituitary gland affects virtually all hormones synthesised and secreted by it. Growth hormone levels are reduced due to an increase in somatostatin, which acts as an antagonist to growth hormone (GH). A decline in growth hormone levels in children can lead to premature closure of the epiphyses of long bones, resulting in reduced overall height. Decreased GH levels also reduce lipolysis, which in turn increases fat deposition in tissues [9].

3.5 Neurological disorders

It has been demonstrated that obesity exerts a significant influence on the development of intracranial hypertension of unknown aetiology. This condition, frequently termed pseudotumor cerebri, presents with symptoms similar to those observed in intracranial hypertension, including projectile vomiting, which is often not preceded by nausea, morning headaches that disturb sleep, and visual disturbances. It is essential to exclude the possibility of other pathologies that may present with similar symptoms; the diagnosis is made subsequent to the exclusion of other potential etiologies. It is hypothesised that elevated intra-abdominal pressure may contribute to the pathogenesis of pseudotumor cerebri [13].

Furthermore, migraine is more frequently diagnosed in obese children, and obesity itself exacerbates its course. This relationship is likely due to the pro-inflammatory effect of adipokines and neuromediators secreted by adipose tissue, which results in inflammation in the CNS. Obstructive sleep apnoea, a prevalent condition associated with headaches and heightened migraine severity, is a salient factor [13].

In obese children, low levels of physical activity and a sedentary lifestyle have been demonstrated to result in cognitive impairment [10].

3.6 Dermatological and immunological complications

Dermatological complications may include the development of dark keratosis, which has been associated with insulin resistance. Stretch marks and purulent inflammation of the sweat glands are often visible on the skin [10].

Furthermore, individuals suffering from obesity have been shown to have a compromised immune system. This phenomenon can be attributed to an imbalance between immune tolerance and the abnormal secretion of specific adipokines by adipose tissue, including leptin. Furthermore, the prevalence of Helicobacter pylori infection was found to be increased in these subjects.

3.7 Mental health disorders

The issue of obesity in children is a multifaceted one, with ramifications for both their physical and mental health. Excessive body weight has been demonstrated to have a detrimental effect on the mental health of children, manifesting in a lack of self-acceptance and reduced understanding and acceptance from their peers. This, in turn, has been shown to contribute to discrimination and stigmatisation, engendering feelings of loneliness, rejection and frequent isolation. Obese children are frequently marginalised in terms of participation in play activities, and are subject to ridicule and mockery from their peers. Such social exclusion may further exacerbate the emotional difficulties experienced by affected children. The development of eating disorders, characterised by emotional binge eating, is a consequence of stigmatisation, which perpetuates a vicious circle. This, in turn, contributes to further weight gain. It has been demonstrated that children who are obese are more prone to developing depression, anxiety disorders, and suicide. In instances of improper

treatment of obesity, the occurrence of anorexia is also a possibility, should the patient begin to engage in obsessive calorie counting and excessive calorie correction by means of following a highly restrictive diet [10]. It is also noteworthy that rapid weight loss is frequently accompanied by a subsequent relapse. Furthermore, obesity has been demonstrated to be a contributing factor to the development of sleep disorders, such as obstructive sleep apnea, and to the deterioration of sleep quality. This, in turn, has been shown to exert a substantial influence on bodily functions and cognitive abilities [4].

3.8 The prevalence of general complications within society and healthcare systems

The issue of obesity in children has been demonstrated to exert an influence on the functioning of society as a whole, with implications for both families and the healthcare system. The rising prevalence of obesity among children, who are increasingly being diagnosed with the disease at younger ages, is associated with a greater frequency of visits to various specialist clinics and more frequent hospitalisations [2]. Moreover, the social implications of obesity are manifold. Obese children are frequently the subject of social exclusion, ridicule, and the absence of healthy interpersonal relationships.

3.9 Methods of treating obesity complications

The treatment of obesity in children should be comprehensive and tailored to the patient's needs. It is essential that complications are frequently monitored, detected early, and treated promptly in order to ensure a successful outcome.

Lifestyle modifications represent a pivotal aspect in the mitigation of complications and the prevention of further ones, which primarily encompass weight reduction and the incorporation of regular physical activity, meticulously tailored to the age and fitness level of the child. When implemented in accordance with the established protocol, this approach has been demonstrated to effectively halt the progression of severe complications and reverse alterations at an early stage. The diet should be limited in simple carbohydrates and highly processed foods, while being rich in protein, monounsaturated and polyunsaturated fats, and fibre. It is imperative to consume meals in a manner that is conducive to good health, and to do so at regular intervals. The consumption of snacks between meals should be restricted, and meals should comprise a minimum of two servings of fruit, two servings of vegetables, and dairy products per day. In younger children, due to the continuous dynamic development of the body, it is recommended to maintain body weight. In older children, however, a gradual reduction in body weight is recommended. The process of weight reduction should take into account a number of factors that influence its course, such as the degree of obesity, the coexistence of other diseases, and the age of the child. It is also recommended that regular physical activity appropriate to the child's abilities be introduced, and that screen time be limited [13].

Pharmacological intervention and bariatric surgery have limited application in the pediatric population and are intended for older children. The only GLP-1 analog that can be used in pediatric patients is liraglutide, which may be considered if the desired weight loss goals are not achieved. The medication is only approved for use in adolescents over 12 years of age with a BMI above 30. Pharmacological treatment is also used to treat complications such as hypertension, diabetes, and hyperlipidaemia that do not respond to dietary interventions.

A multidisciplinary approach, incorporating a proper diet and adequate physical activity, is recommended. It has been demonstrated that even a modest amount of weight reduction can lead to a decline in triglyceride levels and an increase in HDL levels. This, in turn, has been shown to result in a reduction in the incidence of cardiovascular complications in the future. In the case of PCOS, the administration of antiandrogenic drugs necessitates weight loss in order to achieve a therapeutic effect [13]. Bariatric surgery is only performed in centres that meet stringent criteria for indications, including a BMI above 40 or BMI above 35 with comorbidities or complications arising from obesity. Prior to the consideration of surgical intervention, an attempt at lifestyle modification and pharmacotherapy should be made for a period of no less than one year.

It is also recommended that psychological support be provided, with such support improving the child's adaptive and coping skills during the treatment process, increasing motivation, and improving the patient's self-acceptance. Cognitive-behavioural psychotherapy is the most commonly used approach [13].

Early detection of obesity complications and laboratory monitoring of overweight patients also play a significant role. Given the potential for dyslipidaemia to manifest in patients from a very young age, it is recommended that total cholesterol, LDL, HDL, and triglyceride levels be checked from the age of two in overweight children. Research suggests that regular screening for glycaemia should be performed, including measurement of fasting glucose. In certain cases, the measurement of glycated haemoglobin may also be

required. It is recommended that blood pressure be measured during periodic check-ups. Monitoring of liver enzymes – AST, ALT, GGTP – is recommended. It is also recommended to check basic renal parameters – GFR, general urine test [13]. In the event of clinical indications, the assessment of hormone levels is recommended, as well as the performance of echocardiography and ECG. It is recommended that trends in BMI be monitored, and that periodic check-ups include waist circumference and body composition tests. The chronic nature of the disease and its tendency to recur should be borne in mind, necessitating regular and long-term monitoring of patients and cooperation between the physician and patient. It is recommended that individuals contemplate the potential benefits of consulting with a range of professionals, including dieticians, psychologists, and physiotherapists.

4. Discussion

The prevalence of obesity in children is a growing concern, with increasing complications both in the short and long term. The prevailing paradigm regarding obesity has shifted from a narrowly defined metabolic issue to a more comprehensive understanding that encompasses a range of diseases and health consequences. A comprehensive review of the extant literature has unequivocally demonstrated a robust correlation between weight gain and an elevated risk of carbohydrate and lipid metabolism disorders. A relationship has been demonstrated between obesity and the development of insulin resistance, hypertension, endocrine, musculoskeletal, neurological, and pulmonary complications, as well as psychiatric disorders. The principal mechanisms underlying obesity-related complications comprise chronic inflammation, oxidative stress, and endothelial dysfunction. Studies have shown that understanding the pathophysiological interrelationships underlying obesity-related complications is essential for developing personalised therapeutic strategies. Research into the impact of obesity on the development of the young body is ongoing, and these findings may be reflected in treatment and prevention strategies in the near future. The early detection of obesity-related complications, in addition to the identification of high-risk groups, is of paramount importance in the prevention of both somatic and mental illnesses. In the future, the delivery of obesity therapy should be individualised, effective, and based on modern technologies. This will not only limit complications but also allow for more precise monitoring of the effects of the therapy used, which will in turn improve the quality of life of patients.

The obesity pandemic also has financial implications. Research has demonstrated that obese children utilise healthcare services more frequently and experience prolonged hospitalisation durations in comparison to children of normal weight. This has significantly increased healthcare system costs. It is important to implement effective strategies aimed at the prevention of obesity and its associated complications, with the ultimate objective of achieving a reduction in the prevalence of both obesity and its attendant complications. Taken together, these findings underscore the urgent need for comprehensive, early interventions to mitigate the long-term consequences of paediatric obesity.

5. Conclusions

Obesity in children is a complex, chronic, and multisystem disease that significantly affects physical, metabolic, psychological, and social functioning. Its complications—ranging from insulin resistance and hypertension to sleep disorders, orthopaedic problems, and mental health challenges—often emerge early and may persist or worsen in adulthood. The cumulative duration of excess weight during development remains one of the strongest predictors of irreversible long-term outcomes, underscoring the need for timely identification of at-risk children.

Effective management must therefore prioritise early diagnosis, regular monitoring, and comprehensive intervention strategies. Family-based lifestyle modification, integrating balanced nutrition, adequate physical activity, and reduced sedentary behaviours, remains the cornerstone of treatment. Multidisciplinary cooperation between pediatricians, endocrinologists, dietitians, psychologists, physiotherapists, and educators is essential to address the diverse consequences of obesity and to support long-term behavioural change. Providing psychological support and fostering a safe environment—both at home and in school—play an important role in improving motivation, treatment adherence, and overall well-being.

Given the growing prevalence of childhood obesity and the increasing burden it places on healthcare systems worldwide, preventive strategies should be prioritised at the population level. Further research on the molecular and environmental mechanisms underlying obesity and its complications is needed to refine diagnostic tools, identify high-risk groups earlier, and develop targeted, personalised therapeutic approaches. Advancing such knowledge will contribute to more effective prevention, improved clinical outcomes, and better quality of life for children affected by obesity.

6. List of abbreviations
BMI – Body Mass Index
NAFLD – Non-Alcoholic Fatty Liver Disease
HDL – High-Density Lipoprotein
LDL – Low-Density Lipoprotein
VLDL – Very Low Density Lipoprotein
RAA – Renin-Angiotensin-Aldosterone System
ALT – Alanine Aminotransferase
AST – Aspartate Aminotransferase
GGTP – Gamma-Glutamyl Transferase
USG - Ultrasonography
HOMA – IR – Homeostatic Model Assessment of Insulin Resistance
CRP – C-Reactive Protein
OSA – Obstructive Sleep Apnea
PCOS – Polycystic Ovary Syndrome
SHBG – Sex Hormone Binding Globulin
TRH – Thyrotropin-Releasing Hormone
TSH – Thyroid-Stimulating Hormone
GLP 1 – Glucagon-Like Peptide 1
PYY – Peptide YY
GH – Growth Hormone
GFR – Glomerular Filtration Rate
EKG – Electrocardiogram

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