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THE IMPACT OF OBESITY ON THE DEVELOPMENT OF HYPERTENSION IN CHILDREN – A LITERATURE REVIEW

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ABSTRACT

Introduction: Childhood obesity is a growing global health concern that significantly increases the risk of developing hypertension during childhood, leading to serious health consequences. This review aims to present the current state of knowledge regarding the relationship between obesity and hypertension in children, with particular emphasis on pathophysiological mechanisms and prevention strategies.
Current Knowledge: Among all risk factors, overweight and obesity have the greatest impact, correlating proportionally with the degree of blood pressure elevation, especially systolic pressure. Studies have shown that the association between BMI and blood pressure is strongest during puberty. This relationship is influenced by complex mechanisms, including excessive activation of the sympathetic nervous system, the renin–angiotensin–aldosterone system (RAAS), hormonal imbalances, and chronic inflammation induced by adipokines. Additionally, excess adipose tissue mechanically compresses the kidneys, causing sodium retention and blood pressure elevation, while hyperinsulinemia enhances sodium reabsorption and vascular resistance, further contributing to hypertension development. These factors act synergistically, resulting in persistent dysregulation of blood pressure from an early age. Treatment primarily involves lifestyle modification, with pharmacotherapy (ACE inhibitors, ARBs, GLP-1 receptor agonists) reserved for cases resistant to non-pharmacological interventions.
Summary: The available literature unequivocally indicates that obesity constitutes a significant health threat from an early age and increases the risk of serious adult diseases. Given the involvement of numerous modifiable pathophysiological mechanisms and the rising prevalence of these conditions, early prevention, health education, and an interdisciplinary treatment approach are of paramount importance.

KEYWORDS
Childhood Obesity, Hypertension in Children, Hypertension Prevention

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Introduction and aim

Childhood obesity constitutes one of the greatest health challenges of the 21st century, reaching the scale of a global epidemic. Increasing availability of high-calorie, low-nutritional-value food, reduced physical activity, and lifestyle changes contribute to the rising number of children with excess body weight worldwide. It is estimated that the obesity rate among children and adolescents has increased fourfold compared to the previous century, and projections for the coming years suggest that one in six children will suffer from obesity [1,2]. According to the latest data from the World Health Organization (WHO), over 159 million children and adolescents aged 5–19 years worldwide are affected by obesity, with an additional 37 million children under the age of five struggling with excess body weight [3]. Beyond psychosocial aspects, this disorder leads to numerous serious health consequences. One of the most significant and frequently occurring complications of obesity is hypertension, which is increasingly diagnosed in pediatric patients, although until recently it was considered an adult condition [4]. Moreover, it is associated with early signs of target organ damage, such as in the heart and kidneys, and an increased risk of developing chronic diseases in adulthood [5].

Early diagnosis and understanding of the mechanisms underlying the relationship between obesity and hypertension in children are crucial not only from a treatment perspective but primarily for prevention. Studies have shown that reducing excess body weight and normalizing blood pressure values early in life significantly decreases the risk of hypertension in adulthood [6,7]. Attention must also be paid to environmental, genetic, behavioral, and social factors influencing the development of obesity in the pediatric population, which indirectly lead to serious health consequences.

The aim of this review is to discuss the impact of obesity on the development of hypertension in children and to present the current state of knowledge regarding pathophysiological mechanisms, risk factors, and associated consequences.

Materials and Methods

This review article was prepared based on an analysis of the available scientific literature regarding the relationship between obesity and the development of hypertension in children. A comprehensive search was conducted in the electronic databases PubMed, Google Scholar, and Scopus. Publications from 2010 onwards were considered, with particular emphasis on studies published in the last five years (2019–2024) to ensure data relevance. Articles were selected through screening of titles, abstracts, and full texts using the following keywords: “childhood obesity,” “hypertension in children,” “overweight,” and “cardiovascular risk.” The analysis included peer-reviewed scientific publications, original research articles, systematic reviews, and meta-analyses focusing exclusively on children and adolescents (0–19 years old). Only full-text articles published in English were included. Non-scientific articles and publications unavailable in full text were excluded from the review.

Current State of Knowledge

Definitions and Diagnostic Criteria

Childhood and adolescent obesity is diagnosed based on the Body Mass Index (BMI), calculated as the ratio of body weight in kilograms to the square of height in meters (kg/m²). To assess nutritional status, BMI values are referenced against age- and sex-specific percentile charts. According to criteria used, among others, by the Centers for Disease Control and Prevention (CDC), overweight is defined as a BMI between the 85th and 95th percentiles, while obesity is diagnosed when BMI exceeds the 95th percentile [8].

Unlike in adults, the diagnosis of hypertension in children and adolescents is not based on a fixed blood pressure threshold but on values exceeding the 95th percentile for age, sex, and height. Blood pressure measurements should be evaluated based on three independent readings taken under resting conditions, using a cuff of appropriate width and length.

According to guidelines from the American Academy of Pediatrics (AAP, 2017) and the European Society of Hypertension (ESH, 2016) the criteria are presented in Tabele 1.

For adolescents aged ≥ 13 years (AAP) and ≥ 16 years (ESH), adult thresholds may be used, facilitating cardiovascular risk identification during the transitional period [9,10].

Table 1.

Blood Pressure Category	Definition
Normal Blood Pressure	Systolic and/or diastolic BP < 90th percentile for age, sex, and height
Elevated Blood Pressure	Systolic and/or diastolic BP ≥ 90th percentile and < 95th percentile
Stage 1 Hypertension	Systolic and/or diastolic BP ≥ 95th percentile to < (95th percentile + 12 mmHg)
Stage 2 Hypertension	Systolic and/or diastolic BP ≥ (95th percentile + 12 mmHg) or ≥ 140/90 mmHg

BMI and Hypertension

The development of hypertension in children and adolescents is influenced by numerous factors, including gestational age and birth weight, a positive family history of cardiovascular diseases, as well as lifestyle-related elements such as diet, physical activity, and stress exposure. Among all known risk factors, overweight and obesity are considered the most significant. Due to their modifiable nature, early identification and implementation of appropriate preventive interventions constitute a key element in strategies aimed at preventing long-term complications.

A study conducted by K. Babińska demonstrated a proportional relationship between Body Mass Index (BMI) and the severity of hypertension assessed by ambulatory blood pressure monitoring (ABPM), both in terms of average 24-hour and daytime values [11]. Similarly, research by Sachdeva et al. confirmed a proportional association between BMI and hypertension severity, particularly during daytime measurements [12].

An analysis of thirteen studies examining the relationship between BMI and blood pressure values in adolescents revealed a positive correlation between BMI and systolic blood pressure (SBP), with correlation coefficients (r) ranging from 0.18 to 0.48 (mean r = 0.34). For diastolic blood pressure (DBP), the correlation coefficients were lower, ranging from 0.05 to 0.36 (mean r = 0.21). These findings indicate a stronger association of BMI with systolic than with diastolic blood pressure, which aligns with previous observations that overweight adolescents more frequently present isolated elevated SBP or concurrent increases in both blood pressure components rather than isolated elevation of DBP [13,14].

Data from a study by Chiolero et al., involving 7,746 adolescents aged 12–16 years, suggest that the contribution of excess body weight as an explanatory factor for hypertension development is greatest during puberty. The attributable fraction (AF) for overweight and obesity reached its highest values among boys aged 12–13 years and girls aged 15–16 years—39% in both groups. In comparison, among children aged 5–6 years, increased BMI accounted for only 5% of hypertension cases in girls and 9% in boys. Notably, AF values in adult populations are lower—in two independent studies, they were 28% and 11%, respectively [15].

Koebnick and colleagues confirmed these findings in their study, showing that the association between body weight and elevated blood pressure was slightly but statistically significantly stronger ($P < 0.001$) in

individuals aged 12–17 years compared to children aged 6–11 years. They also demonstrated that sex-related differences, commonly observed in adult populations, were not evident in this younger cohort [16].

Pathophysiological Mechanisms of Obesity-Induced Hypertension Development in Children

Overweight and obesity significantly increase the risk of hypertension in the pediatric population through multiple pathomechanisms [17]. It is believed that the most critical pathways leading to hypertension in obesity are closely interrelated and primarily involve metabolic, renal, and neuroendocrine mechanisms [18].

The key factors implicated in the development of hypertension include heightened activity of the sympathetic nervous system (SNS), activation of the renin-angiotensin-aldosterone system (RAAS), and mechanical compression of the kidneys caused by fat tissue accumulation, which results in enhanced sodium reabsorption in the kidneys and impairment of pressure natriuresis [17,18].

Adipose tissue is hormonally active and secretes hormones and cytokines known as adipokines. In conditions of excess adiposity, there is an overproduction of adipokines due to an increased number and size of adipocytes. At this stage, pro-inflammatory adipokines (leptin, resistin, IL-6) predominate over anti-inflammatory adipokines (adiponectin), initiating a chronic inflammatory state in the body [19]. Numerous analyses have reported a statistically significant inverse correlation between adiponectin levels and blood pressure values in obese children, independent of the amount of adipose tissue or BMI, suggesting that adiponectin may play a crucial role in blood pressure regulation within this population [20].

Retinol-binding protein 4 (RBP4) concentrations were significantly elevated in obese adolescents and correlated with average 24-hour systolic blood pressure, regardless of the degree of adiposity. However, after adjusting for BMI, the association between RBP4 and random blood pressure values was attenuated, indicating that body mass itself influences this relationship.

In children with excess body weight, visfatin levels positively correlated with elevated blood pressure values (above the 90th percentile), suggesting its potential involvement in hypertension pathogenesis. Conversely, apelin concentrations did not differ significantly between groups and showed no association with cardiovascular risk [20].

Leptin, produced by adipocytes, acts on the hypothalamus to modulate sympathetic nervous system activity and vascular tone, suggesting a possible role in the development of hypertension in children. However, research findings indicate that leptin's effect on blood pressure largely depends on BMI, implying that it likely does not play an independent role in obesity-related hypertension pathogenesis [20].

Excessive Activation of the Sympathetic Nervous System (SNS)

Numerous adipokines, including leptin—which is notably elevated in obese hypertensive children—have been shown to increase sympathetic nervous system activity. This, in turn, significantly contributes to elevated blood pressure by accelerating heart rate, increasing cardiac output, and enhancing renal tubular sodium reabsorption [17–19].

It has also been demonstrated that SNS is activated in obesity. A urinary analysis study revealed significantly higher levels of norepinephrine in obese children compared to their normal-weight peers. [18–20] Additionally, the same study showed reduced vagal nerve activity in obese children. These two components play a critical role in the pathogenesis of elevated blood pressure in the pediatric population [20].

Abnormal adipokine secretion, excessive activation of the renin-angiotensin-aldosterone system (RAAS), insulin resistance, and baroreceptor dysfunction are the primary factors driving sympathetic nervous system overactivity in obesity [18]. Moreover, obstructive sleep apnea (OSA), commonly associated with obesity, causes recurrent hypoxia, which stimulates chemoreceptors and further increases sympathetic activity. Some of these mechanisms may contribute to hypertension development independently of SNS activation [18,21].

Individuals with obesity exhibit a chronic inflammatory state characterized by macrophage infiltration into adipose tissue and elevated free fatty acid levels. Dyslipidemia, defined by increased low-density lipoprotein (LDL) and triglycerides with reduced high-density lipoprotein (HDL), promotes atherosclerosis, chronic inflammation, sympathetic overactivity, and RAAS activation. Enhanced oxidative stress further amplifies sympathetic tone and damages endothelial cells. Consequently, these disturbances lead to metabolic dysfunction, impaired vascular function, and the development of hypertension [19].

Activation of the renin-angiotensin-aldosterone system (RAAS)

Despite increased blood volume and sodium retention, which physiologically should inhibit the RAAS, studies have shown that obese individuals have higher levels of renin, angiotensinogen, ACE, and aldosterone compared to lean individuals [22,23]. Activation of RAAS enhances the production of angiotensin II, which constricts blood vessels and stimulates aldosterone secretion. Both hormones increase sodium reabsorption and water retention, raising blood volume and contributing to the development of hypertension in children [17,18].

In obesity, activation of the renin-angiotensin-aldosterone system occurs mainly through two key mechanisms: mutual stimulation of the sympathetic nervous system and RAAS, and mechanical compression of the kidneys by excess adipose tissue, which leads to increased renin secretion. Further stimulation of renin is caused by tubuloglomerular feedback triggered by reduced blood flow and decreased sodium delivery to the macula densa. Adipose tissue cells also possess a local RAAS, being the main source of angiotensinogen and angiotensin II, as well as producing mineralocorticoid factors that independently stimulate aldosterone production, thereby promoting hypertension [18].

It has been demonstrated that even a 5% weight loss reduces circulating levels of RAAS components (angiotensinogen, renin, aldosterone, and angiotensin-converting enzyme), leading to a decrease in systolic blood pressure by up to 7 mmHg. However, this study was conducted in peri-menopausal women and does not directly apply to the pediatric population [19,24].

Structural changes in the kidneys caused by adipose tissue compression

An important role is played by visceral and retroperitoneal adipose tissue located around the kidneys, which exerts mechanical pressure on the renal parenchyma—specifically the renal medulla. This leads to impaired pressure-dependent natriuresis and causes increased sodium reabsorption in the renal tubules, resulting in sodium retention, elevated renin levels, activation of the RAAS in the subsequent mechanism, and increased blood pressure [17-19].

Hypothalamic-Pituitary-Adrenal (HPA) Axis

Cortisol, whose secretion is stimulated by adrenocorticotropic hormone (ACTH) as part of the hypothalamic-pituitary-adrenal (HPA) axis, plays a crucial role in the regulation of metabolism and blood pressure. Studies in children and adolescents with excess body weight have demonstrated that elevated morning levels of ACTH and cortisol are significantly correlated with increased systolic and diastolic blood pressure values, independent of BMI. Furthermore, hypertensive obese patients exhibited increased urinary excretion of cortisol metabolites, suggesting enhanced HPA axis activity and increased conversion of cortisone to its active form. These findings emphasize the importance of chronic HPA axis activation, augmented stress response, and hormonal dysregulation in the pathogenesis of hypertension among young individuals with obesity [20,25].

The influence of insulin on blood pressure values in obese children

So far, only one study has been conducted in the pediatric population aiming to examine the correlation between fasting insulin levels and systolic blood pressure values in children. The results indicated a significant correlation between fasting insulin concentration and increased systolic blood pressure in obese children compared to the pediatric population with normal weight.

However, the authors emphasized that this study had many doubts and inconsistencies, so it is not fully reliable regarding the influence of insulin on the development of hypertension in children [20,26].

From a pathophysiological perspective and considering the mechanism of insulin action, it can be assumed that insulin has a significant impact on the development of hypertension in children with obesity.

Obesity promotes the development of insulin resistance and chronically elevated insulin levels, which may lead to increased blood pressure through activation of the sympathetic nervous system and enhanced sodium retention in the kidneys. Insulin increases sodium reabsorption in the proximal renal tubule by stimulating the sodium-hydrogen exchanger (NHE3), resulting in a greater circulating blood volume and elevated blood pressure [18]. Additionally, in obese individuals, chronic hyperinsulinemia impairs the vasodilatory effect of insulin due to endothelial dysfunction, promoting increased vascular resistance and contributing further to the development of hypertension [18].

Treatment Methods

Two main approaches are distinguished in the management of obesity-related hypertension. The first focuses on treating obesity by reducing body mass index (BMI) to below the 85th percentile for age and sex, while the second addresses the direct management of elevated blood pressure. According to current guidelines, all pediatric patients—regardless of the stage of hypertension—should undergo lifestyle modifications, including adherence to the DASH (Dietary Approaches to Stop Hypertension) diet and engagement in regular moderate-to-vigorous physical activity [9,10].

A meta-analysis of non-pharmacological interventions for childhood obesity demonstrated that combining dietary changes with physical activity is effective in reducing the risk of obesity among children aged 0 to 5 years. In contrast, interventions based solely on increasing physical activity did not produce significant results in this youngest age group. However, among children aged 6 years and older, physical activity-focused interventions alone were effective in lowering obesity risk [27].

Pharmacological treatment in children, according to the latest recommendations, should not typically be initiated as a first-line therapy. It is reserved for cases where lifestyle interventions (including dietary changes and physical activity) have failed to yield satisfactory results [28].

In children and adolescents requiring pharmacologic treatment of hypertension, the recommended first-line agents include angiotensin-converting enzyme (ACE) inhibitors, angiotensin receptor blockers (ARBs), long-acting calcium channel blockers, or thiazide diuretics. Among these, ACE inhibitors and ARBs have demonstrated the highest efficacy in recent meta-analyses of pediatric hypertension management [28, 29].

In addition, glucagon-like peptide-1 receptor agonists (GLP-1 RAs) have shown promise in treating pediatric obesity and thereby reducing associated hypertension. The latest meta-analysis confirms the safety of GLP-1 RAs in children and their moderate efficacy in reducing body weight, BMI, glycated hemoglobin (HbA1c), and systolic blood pressure in children and adolescents with obesity in clinical settings—although an increased incidence of nausea has been reported [30].

Conclusions

Based on a review of the available literature, it can be stated that there is a clear and strong association between obesity and the development of hypertension in children. The rising prevalence of excess body weight in the pediatric population directly translates into an increased number of hypertension diagnoses, which until recently was considered a disease of adults. It has been proven that the higher the BMI, the greater the risk of elevated blood pressure, particularly systolic pressure, which may have serious health consequences both in childhood and later in life. Childhood obesity is no longer merely an aesthetic or social issue — it has become a real public health threat requiring coordinated actions at multiple levels.

The pathogenesis of hypertension in children with obesity involves many coexisting mechanisms, including neuroendocrine disturbances, chronic inflammation, abnormal secretion of adipokines, activation of the sympathetic nervous system, and the renin-angiotensin-aldosterone system. Additionally, the presence of insulin resistance, dyslipidemia, and sleep apnea further increases the risk of hypertension. Importantly, many of these factors are modifiable, which highlights the significance of early prevention and intervention beginning in childhood.

In light of the increasing prevalence of both obesity and hypertension among children and adolescents, the role of primary care physicians, pediatricians, dietitians, and health educators becomes especially important. It is crucial not only to treat already diagnosed cases but above all to implement effective prevention strategies conducted both at home and in school environments. Raising public awareness about the impact of lifestyle factors — including diet, physical activity, and sleep hygiene — on children's cardiovascular health is essential.

Equally important is the continuation of research to better understand the mechanisms linking obesity and hypertension, as well as the development of effective, long-term therapeutic strategies tailored to the age of patients. Only a holistic approach — taking into account biological, psychological, and environmental factors — offers a real chance to halt the epidemic of lifestyle-related diseases in the youngest generations.

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