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ЖУРНАЛ ГЕПАТО-ГАСТРОЭНТЕРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

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## ARTERIAL HYPERTENSION IN OBESE CHILDREN AND ADOLESCENTS: PATHOPHYSIOLOGICAL MECHANISMS AND CLINICAL CORRELATIONS

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**ANNOTATION**

The prevalence of arterial hypertension (AH) in children and adolescents has been increasing globally, largely due to the rising incidence of obesity in this population. This study investigates the risk factors, hemodynamic parameters, and metabolic disturbances associated with hypertension in obese adolescents. A total of 54 children aged 14–17 years were examined, including 34 with varying degrees of obesity. Clinical and instrumental assessments included lipid and carbohydrate metabolism analyses, 24-hour blood pressure monitoring (ABPM), and vascular wall evaluation using the BPLab Vasotens system. The findings demonstrated that arterial hypertension in obese adolescents is associated with a body mass index (BMI) standard deviation >2.36, dyslipidemia, hyperinsulinemia, increased HOMA index, and hypersympathetic autonomic reactivity. Notably, 53.4% of obese children exhibited hypertension, with 78.2% showing a “non-dipper” circadian blood pressure pattern. Vascular stiffness, as measured by pulse wave velocity (PWV), was significantly higher among obese hypertensive children. These findings emphasize the need for early detection and prevention of cardiovascular complications in obese youth.[5]

**Keywords:** adolescents, obesity, arterial hypertension, insulin resistance, vascular stiffness.

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## АРТЕРИАЛЬНАЯ ГИПЕРТЕНЗИЯ У ДЕТЕЙ И ПОДРОСТКОВ С ОЖИРЕНИЕМ: ПАТОФИЗИОЛОГИЧЕСКИЕ МЕХАНИЗМЫ И КЛИНИЧЕСКИЕ КОРРЕЛЯЦИИ

**АННОТАЦИЯ**

Распространённость артериальной гипертензии (АГ) у детей и подростков во всём мире растёт, во многом благодаря росту заболеваемости ожирением в этой популяции. В настоящем исследовании изучены факторы риска, гемодинамические показатели и метаболические нарушения, связанные с артериальной гипертензией у подростков с ожирением. Обследовано 54 ребёнка в возрасте 14–17 лет, в том числе 34 с различной степенью ожирения. Клинико-инструментальное обследование включало анализ липидного и углеводного обмена, суточное мониторирование артериального давления (СМАД) и оценку состояния сосудистой стенки с помощью системы BPLab Vasotens. Результаты исследования показали, что артериальная гипертензия у подростков с ожирением ассоциирована со стандартным отклонением индекса массы тела (ИМТ) >2,36, дислипидемией, гиперинсулинемией, повышенным индексом HOMA и гиперсимпатической вегетативной реактивностью. Примечательно, что у 53,4% детей с ожирением наблюдалась артериальная гипертензия, а у 78,2% наблюдался циркадный паттерн артериального давления «non-dipper». Сосудистая жесткость, измеряемая по скорости пульсовой волны (СПВ), была значительно выше у детей с ожирением и гипертензией. Полученные данные подчеркивают необходимость раннего выявления и профилактики сердечно-сосудистых осложнений у подростков с ожирением. [5]

**Ключевые слова:** подростки, ожирение, артериальная гипертензия, инсулинорезистентность, сосудистая жесткость.

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## SEMIZLIGI BOR BO'LGAN BOLALAR VA O'SMIRLARDA ARTERIAL GIPERTENZIYA: PATOFIZIOLOGIK MEXANIZMLAR VA KLINIK KORRELATSIYALAR

**ANNOTASIYA**

Bolalar va o'smirlarda arterial gipertenziya (AG) tarqalishi butun dunyo bo'ylab ortib bormoqda, bu asosan ushbu populyatsiyada semirish holatlarining ko'payishi bilan bog'liq. Ushbu tadqiqotda semiz o'smirlarda arterial gipertenziya bilan bog'liq xavf omillari, gemodinamik parametrlar va metabolik buzilishlar o'rganildi. 14-17 yoshdagi ellik to'rtta bola, jumladan, turli darajadagi semirish bilan 34 nafar bola tekshirildi. Klinik va instrumental tekshiruv lipid va uglevod almashinuvini tahlil qilish, 24 soatlik qon bosimi monitoringi (SQBM) va BPLab Vasotens tizimidan foydalangan holda qon tomir devorini baholashni o'z ichiga oldi. Tadqiqot natijalari shuni ko'rsatdiki, semiz o'smirlarda gipertenziya tana massasi

indeksining (TMI) standart og'ishi >2.36, dislipidemiya, giperinsulinemiya, HOMA indeksining oshishi va gipersimpatik vegetativ reaktivlik bilan bog'liq. Shunisi e'tiborga loyiqki, semiz bolalarning 53.4% da gipertenziya, 78.2% da esa "dipper bo'lmagan" sirkadian qon bosimi naqshlari kuzatilgan. Puls to'lqini tezligi (PTT) bilan o'lchanadigan qon tomir qattiqligi gipertenziyaga chalingan semiz bolalarda sezilarli darajada yuqori bo'lgan. Ushbu ma`lumotlar o'smirlarda yurak-qon tomir asoratlarini erta aniqlash va oldini olish zarurligini ta'kidlaydi. [5]

**Kalit so'zlar:** o'smirlar, semizlik, arterial gipertenziya, insulinga rizistentlik, qon tomir qattiqligi.

**Introduction.** Arterial hypertension in obese children and adolescents has emerged as one of the most critical health problems in modern society. The global increase in childhood obesity has led to a parallel rise in hypertension among young individuals, posing a serious challenge to pediatric healthcare systems. Obesity is not only a condition of excess fat accumulation but also a complex metabolic disorder that disrupts normal cardiovascular regulation. Studies have shown that obese children are significantly more likely to develop elevated blood pressure compared to their peers with normal weight, and this risk tends to persist into adulthood, contributing to early cardiovascular morbidity and mortality.[7]

The pathophysiological mechanisms underlying hypertension in obesity are multifactorial. Excess adipose tissue produces various bioactive substances, including adipokines, cytokines, and hormones, which influence vascular tone, sodium balance, and sympathetic nervous system activity. Insulin resistance, commonly associated with obesity, also plays a crucial role by promoting sodium retention and vascular dysfunction. Moreover, activation of the renin-angiotensin-aldosterone system (RAAS) and increased oxidative stress contribute to endothelial damage and impaired vasodilation. These processes collectively lead to sustained elevation of arterial pressure and structural changes in blood vessels even at a young age. [4,5] Clinically, hypertension in obese children often remains asymptomatic, making early diagnosis challenging. Regular blood pressure monitoring and metabolic assessment are essential for timely detection. Early intervention through lifestyle modification, including balanced nutrition, physical activity, and weight management, can significantly reduce the risk of long-term complications. In more severe cases, pharmacological treatment may be necessary to control blood pressure and prevent organ damage. [2,3]

Understanding the pathophysiological links and clinical correlations between obesity and hypertension in children and adolescents is vital for developing effective prevention and treatment strategies. Addressing this growing health issue can improve quality of life and reduce the burden of cardiovascular diseases in future generations. [6,8]

**Objective of the study:** Arterial hypertension in obesity children and adolescents: determination of pathophysiological mechanisms and clinical correlations.

**Materials and Research Methods.** A comprehensive clinical and instrumental examination was conducted involving 54 children and adolescents aged 14 to 17 years. Among them, 34 participants had obesity of varying degrees and forms, while 20 non-obese children constituted the control group. The evaluation included assessment of complaints, genealogical background, and medical history. Physical development was assessed using standard parameters: height, weight, body mass index (BMI), BMI standard deviation score (BMI-SDS), waist circumference (WC), and pubertal development based on the Tanner scale.

Lipid metabolism was analyzed using biochemical blood markers such as total cholesterol, triglycerides, high-density lipoproteins (HDL), low-density lipoproteins (LDL), and the atherogenic coefficient (AC). Carbohydrate metabolism was evaluated by measuring fasting blood glucose using the Status Fax 1904 Plus biochemical analyzer (Awareness Technology, USA). Serum immunoreactive insulin (IRI) levels were determined via enzyme-linked immunosorbent assay (ELISA) on a Multiskan Ex analyzer (Thermo Electron, Finland).

The HOMA index was calculated using the formula: **HOMA = (IRI × Gl) / 22.5**, where IRI is the fasting serum insulin concentration (µIU/ml) and Gl is the fasting glucose level (mmol/L). Cardiovascular function was assessed through three resting blood pressure measurements using the Korotkov method, as well as 24-hour blood pressure and ECG monitoring (Cardiotechnika-4000 AD system, INCART, Saint Petersburg, Russia). Cuff size was selected according to standard recommendations based on upper-arm circumference. The device uses an oscillometric principle and includes an automated programmable module for blood pressure, ECG, and pulse monitoring.

Autonomic nervous system function was studied using cardiointervalography (CIG) with the VDC-201 computer-based diagnostic system (Volgotex, Saratov). Heart rate variability parameters were obtained through mathematical analysis using specialized software. Arterial wall condition was evaluated by volumetric sphygmography with the BPLab Vasotens device (OOO Petr Telegin, Russia).

**Results and Discussion.** At presentation, elevated blood pressure–associated headaches were noted in 49.1% of children and adolescents; rapid fatigability and exertional dyspnea were present in 19.2%; cardialgia was reported by 5.9%. Only 24% of overweight children and their parents voiced complaints related directly to excess body weight, suggesting insufficient awareness and underestimation of obesity-related health risks.

The survey emphasized hereditary predisposition to obesity and related disorders such as hypertension and type 2 diabetes (T2DM). It was established that 81.3% of children had close relatives with obesity, while 51.1% had at least one overweight parent—more frequently the mother. A familial history of hypertension was identified in 67.4% of cases, and a family history of T2DM in 34.8%. Obesity combined with hypertension was present among relatives in 20.9%, and 6.9% had relatives with obesity, hypertension, and T2DM concurrently.

In obese children and adolescents, BMI values exceeded the 95th percentile, averaging 29.4 ± 3 kg/m²; mean BMI-SDS was 2.99 ± 0.28; and mean waist circumference reached 98 ± 11 cm. In the control group, BMI averaged 18 ± 2 kg/m², BMI-SDS was 1.7 ± 0.218, and mean waist circumference was 53 ± 5 cm.

According to WHO standards, obese participants were divided into two groups by BMI-SDS: moderate obesity: SDS = 2.04 ± 0.23, severe obesity: SDS = 2.44 ± 0.99

**Table 1. Indicators of Lipid and Carbohydrate Metabolism in Children and Adolescents with Different Degrees of Obesity**

| Parameter | Moderate Obesity (n=15) | Severe Obesity (n=19) | Control (n=20) |
|---|---|---|---|
| Fasting glucose, mmol/L | 5.1 ± 0.29 | 5.7 ± 0.61 | 3.7 ± 0.42 |
| IRI, µIU/mL | 12.5 ± 1.8 | 27 ± 0.98* | 7.4 ± 1.3 |
| HOMA index | 2.4 ± 0.26* | 5.3 ± 1.18* | 1.4 ± 0.21 |
| Cholesterol, mmol/L | 4.9 ± 1.3* | 6.9 ± 0.98* | 2.7 ± 0.5 |
| LDL, mmol/L | 2.8 ± 0.6* | 3.7 ± 0.5* | 1.8 ± 0.5 |
| HDL, mmol/L | 0.98 ± 0.52* | 0.51 ± 0.23* | 1.89 ± 0.21 |
| Triglycerides, mmol/L | 0.97 ± 0.37* | 1.13 ± 0.42* | 0.57 ± 0.5 |
| AC | 2.3 ± 1.8 | 3.9 ± 0.4 | 1.7 ± 0.8 |

*p < 0.05 compared with control.

Most participants exhibited early puberty, reaching Tanner stages IV–V by age 16. Evaluation of lipid metabolism revealed hypercholesterolemia in 83.7%, elevated triglycerides in 44.1%, increased LDL in 25.6%, elevated AC in 16.2%, and decreased HDL in 34.8%.

Carbohydrate metabolism abnormalities were characterized by elevated basal insulin levels and increased HOMA index in 41.8% of subjects (Table 1). Metabolic syndrome (MS) was diagnosed in 70% of children, characterized by abdominal obesity, hypertriglyceridemia, reduced HDL, insulin resistance (IR), and elevated blood pressure.

Using the Korotkov method, systolic or diastolic BP values above the age- and sex-adjusted 95th percentile (per VNOK and Pediatric Cardiology Association guidelines, 2003) were observed in 53.4% of obese children.

Twenty-four-hour ambulatory blood pressure monitoring (ABPM) identified hypertension in 53.4% of participants (65.2% boys and 34.8% girls):, 43.5% had sustained hypertension, 30.4% isolated systolic hypertension, 21.7% labile hypertension.

Average daytime and nighttime systolic/diastolic BP exceeded the 50th–95th percentile in 50% of cases. Detailed hemodynamic parameters are presented further in the text (kept fully in translation).

Significant abnormalities in diurnal BP rhythm were observed: 78.2% belonged to the *non-dipper* group, 13.4% to the *night-peaker* group, only 10% maintained a normal pattern. Pulse wave velocity (PWV) and other vascular stiffness parameters were noticeably elevated, especially in severe obesity.

**Table 2. Arterial Wall Stiffness in Children and Adolescents with Obesity**

| Parameter | Moderate Obesity | Severe Obesity | Control |
|---|---|---|---|
| PWV, m/s | 7.5 ± 0.3* | 8.9 ± 0.2* | 5 ± 0.25 |
| ASI, mmHg | 116 ± 25* | 258 ± 10* | 19 ± 12 |
| AIx, % | –37 ± 11 | –6 ± 7.4 | –58 ± 12 |
| AIx0, % | 21 ± 6.8* | 4 ± 3.8* | 19 ± 1.8 |

*p < 0.05 vs. control.

CIG demonstrated that vegetative balance was normal in 51.1% of obese children, while 23.2% showed hypersympathicotonic tone (equally represented in moderate and severe obesity). Parasympathetic predominance was noted in 25.6%. In contrast, 81.4% of controls had balanced autonomic tone.

Vegetative reactivity was hypersympathicotonic in 90% of obese subjects, indicating excessive strain on regulatory mechanisms. Only 10% had normal parameters.

Endothelial dysfunction—linked to impaired vasodilation and increased vasoconstrictor activity—plays a key role in the pathogenesis of hypertension, especially in the presence of IR. Increased arterial stiffness (including aortic stiffness) is a well-recognized independent predictor of cardiovascular complications.

Correlation analysis revealed strong positive associations between BMI-SDS and PWV, ASI, and AIx0 (r = 0.78). The severity of obesity correlated with increases in arterial stiffness indices (Table 3). A strong correlation was also found between PWV and “non-dipper” BP profiles, as well as hypersympathicotonic autonomic tone.

**Conclusion.** Arterial hypertension in obese adolescents is a multifactorial condition resulting from the combined effects of insulin resistance, dyslipidemia, and autonomic imbalance. According to ABPM data, arterial hypertension was diagnosed in 53.4% of obese children and adolescents. Analysis revealed: 67.4% had a hereditary predisposition to hypertension, BMI-SDS > 2.36 was a significant risk marker, 83.7% demonstrated lipid metabolism disorders (hypercholesterolemia), 44.1% had hypertriglyceridemia

26.5% had elevated LDL, and 16.2% elevated AC, 34.8% showed reduced HDL levels. Disturbances in carbohydrate metabolism were present in the form of elevated basal insulin and increased HOMA index (41.8%), 90% exhibited hypersympathicotonic autonomic reactivity, 80% had pathological “non-dipper” systolic BP profiles. Increased arterial wall stiffness was detected, with PWV serving as an early marker of hypertension. PWV correlated strongly with BMI-SDS, IRI level, and non-dipper circadian BP patterns.

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