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Two-Phase Development of Arterial Hypertension in Adolescents with Obesity: Results of a 24-Month Prospective Hemodynamic Study

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Abstract

The rising prevalence of obesity among adolescents has become a global health concern, directly contributing to the early development of cardiovascular risk. This prospective multicenter cohort study aimed to investigate the dynamics of hemodynamic mechanisms underlying the formation of arterial hypertension (AH) in this patient category during the critical period of pubertal development. The study included 498 adolescents aged 12-17 years from major cities in the North Caucasus, stratified by body mass index. Over 24 months, a comprehensive examination was conducted, including 24-hour ambulatory blood pressure monitoring (ABPM), echocardiography with calculation of cardiac output (CO) and total peripheral resistance (TPR), assessment of pubertal stage (Tanner), bioimpedance analysis, and metabolic marker profiling. The prevalence of AH at the end of the observation period in the obesity group was 34.5%, which was 7.2 times higher than in the normal weight group (4.8%). A two-phase pattern of hemodynamic disturbances was revealed: in the early pubertal stages, a hyperkinetic type with high CO (6.4 [5.9; 7.0] L/min) predominated, while in the late stages, the leading role shifted to increased TPR (1690 [1560; 1820] dyn·s·cm^{-5}), associated with insulin resistance (HOMA-IR 4.8 [4.0; 5.7]) and adipokine imbalance. Multivariate analysis confirmed that elevated TPR is an independent predictor of AH. The results demonstrate an evolution of the hemodynamic profile from a volume-dependent to a resistance-dependent form of AH with pubertal progression. This justifies the need for a differentiated approach to diagnosis and early intervention to prevent irreversible target organ damage.

Keywords: Adolescent obesity, Arterial hypertension, Hemodynamics, Puberty, Cardiac output, Total peripheral resistance

Introduction

Modern pediatrics faces a new non-communicable epidemic that is radically altering the health profile of the younger generation. This refers to the rapid increase in the prevalence of overweight and obesity among children and adolescents, a problem that has transcended the borders of developed nations and assumed global proportions (Nogueira-de-Almeida *et al*., 2024; Petridi *et al*., 2024; Zhang *et al*., 2024). This phenomenon poses not a delayed, but an immediate threat, predisposing adolescents to "adult" cardiometabolic diseases long before they reach maturity. One of the most significant and early manifestations of this threat is arterial hypertension (AH), once considered a rarity in adolescent clinical practice (de Simone *et al*., 2022; Welser *et al*., 2023).

The relevance of the problem is underscored by alarming statistics demonstrating a consistent negative trend in the Russian Federation as well. Data from official statistics and epidemiological studies indicate a marked rise in indicators over recent decades, with distinct regional nuances, such as in the North Caucasus, where this trend has unique features linked to demographic and socio-cultural factors (Pelevin *et al*., 2018; Baklanov *et al*., 2020; Kazakova, 2022). The dynamics of the prevalence of overweight/obesity and arterial hypertension among adolescents in Russia are summarized by decade in **Table 1**.

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Table 1. Dynamics of the Prevalence of Overweight/Obesity and Arterial Hypertension among Adolescents in the Russian Federation (Summary Data by Decade)

Period (Years)	Age Group	Prevalence of Overweight/ Obesity (%)	Prevalence of Arterial Hypertension/ Prehypertension (%)	Key Changes / Notes
2000-2010	12-17 years	~10-12% (combined)	~3-5%	Stable, low but rising rates. AH is often secondary.
2011-2020	12-17 years	~15-18% (combined)	~7-10%	Acceleration of growth, especially in urban centers. Increase in primary (essential) AH.
2021-Present	12-17 years	~20-25%	~12-18%	A sharp increase is associated with dietary changes and physical inactivity. AH in 30-40% of adolescents with obesity.

The relationship between obesity and impaired blood pressure (BP) regulation during puberty is not merely a statistical association but a complex pathophysiological nexus intertwining hormonal surges of growth, metabolic shifts, and key hemodynamic changes (Fan *et al*., 2024; Evripidou *et al*., 2025). Normal hemodynamics ensures adequate organ and tissue perfusion, forming the basis of vital activity. Its disruption in adolescents with obesity is systemic and serves as the starting point for a vicious cycle. The key element here is the alteration of fundamental parameters governed by the Frank-Starling law and defined as the product of cardiac output (CO) and total peripheral vascular resistance (TPR) (Hanft *et al*., 2021; Han *et al*., 2022; Kwarteng *et al*., 2023).

The causes of hemodynamic impairment in adolescent obesity are multifactorial and form a complex network of interactions. Their foundation lies in the development of dysfunctional, inflammatory, and endocrinologically active adipose tissue (Di Raimondo *et al*., 2021; LaCombe *et al*., 2023). The main pathogenetic components can be represented as follows **(Figure 1)**.

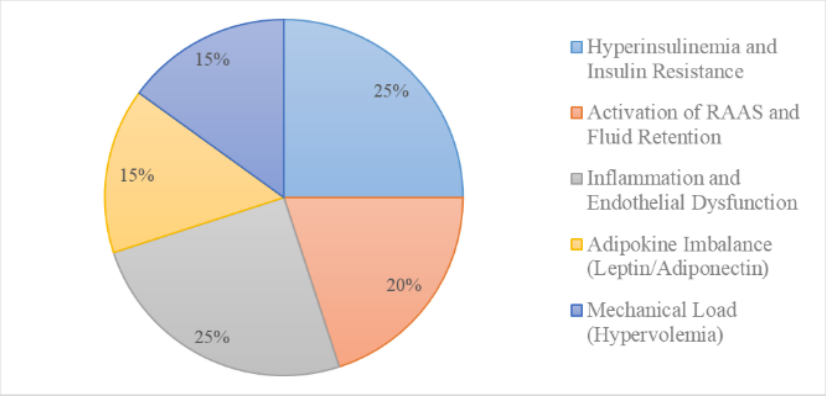

Figure 1. Main Causes of Hemodynamic Impairment in Adolescent Obesity

However, the precise temporal dynamics and contribution of these two hemodynamic determinants during the distinct stages of pubertal development in adolescents with obesity remain poorly characterized. It is unclear whether the initial phase is dominated by a hyperkinetic, high cardiac output state, which subsequently evolves into a vasoconstrictive, high-resistance phenotype with the onset of insulin resistance, or whether these mechanisms operate concurrently from the outset (Sadovoy *et al*., 2017; Cominato *et al*., 2021; Jebeile *et al*., 2022; Lister *et al*., 2023). Elucidating this sequence is critical for understanding the natural history of obesity-related hypertension in youth.

The answers to these questions are of paramount practical importance, as they determine the choice of preventive and therapeutic strategy: from a diet with restricted salt and volume to reduce preload to the prescription of drugs modifying vascular tone and insulin sensitivity. Existing data are fragmented, and there is a lack of prospective studies specifically dedicated to the dynamics of hemodynamic parameters during puberty.

Thus, this prospective cohort study aimed to conduct a comprehensive assessment of the dynamics of central and peripheral hemodynamic parameters in adolescents with overweight and obesity, depending on the stage of sexual maturation, to identify the pathogenetic features and staging of arterial hypertension formation in this patient category. The obtained data will allow a transition from a unified approach to personalized correction of early cardiovascular disorders, which is a cornerstone in preventing the future epidemic of cardiometabolic diseases.

Materials and Methods

Study Design

This work is based on a prospective comparative cohort design with a 24-month follow-up period. The study was organized as a multicenter project, which allowed for the formation of a representative sample of the adolescent population from major urban agglomerations in the North Caucasus region of the Russian Federation. Pediatric outpatient clinics and cardiology departments of city hospitals in Stavropol and Vladikavkaz were involved. The study protocol was approved by the local ethics committees of all participating institutions. Written informed consent was obtained from all legal representatives of the participants, as well as from the adolescents themselves.

Participants

The study included 540 adolescents of both sexes aged 12 to 17 years at the time of the initial examination. The criterion for forming the main group (the "risk" cohort) was the presence of overweight or obesity, diagnosed according to the universal criteria of the World Health Organization (WHO) using body mass index (BMI) (Safaei *et al*., 2021; Cole & Lobstein, 2022; Celletti *et al*., 2026). For screening and stratification, gender- and age-specific BMI percentile charts were used. The observation group consisted of 360 adolescents whose BMI exceeded the 85th percentile. The comparison group comprised 180 conditionally healthy adolescents with BMI ranging from the 15th to the 85th percentile, matched by sex and age. The key exclusion criterion for all participants was the presence of established congenital heart defects, severe endocrine pathology (type 1 diabetes mellitus, decompensated thyroid disease), chronic kidney disease, as well as the use of medications with systemic effects on blood pressure and metabolism.

Examination Procedure and Methods

Each participant underwent a comprehensive three-stage examination upon inclusion in the study (visit V0), after 12 months (V1), and after 24 months (V2). Anthropometric assessment included standard measurement of height, weight, waist circumference (WC), and hip circumference (HC) with calculation of BMI and WC/HC ratio (Casadei & Kiel, 2022; Mandalidis *et al*., 2022). Body composition was analyzed using bioelectrical impedance analysis on the "ABC-01 Medass" device to determine absolute and relative masses of fat, muscle, and lean body mass, as well as total body water content.

Assessment of Pubertal Development was performed by a pediatrician using the Tanner scale, with separate assessment of breast and genital development stages (in boys) or pubic hair (Emmanuel & Bokor, 2022). For statistical analysis, stages were grouped into two categories: early puberty (Tanner stages II–III) and late puberty (Tanner stages IV–V).

The Gold Standard for Arterial Hypertension Diagnosis was 24-hour ambulatory blood pressure monitoring (ABPM) using "Cardiotechnika-04" devices or equivalents validated for pediatric practice. Monitoring was conducted on a typical school day with a measurement interval of 15 minutes during wakefulness and 30 minutes during sleep. An appropriately sized cuff for age and arm circumference was used. ABPM data analysis included calculation of average systolic (SBP), diastolic (DBP), and mean arterial pressure (MAP) over 24 hours, daytime, and nighttime, hypertension time index, and assessment of the circadian rhythm (dipper status).

Central to the Study Was the Echocardiographic Assessment of Hemodynamics

All adolescents underwent transthoracic echocardiography (EchoCG) on expert-class ultrasound scanners (e.g., Vivid E95, Philips EPIQ 7) according to a standard protocol. In addition to assessing the structural state of the heart, special attention was paid to calculating hemodynamic parameters. Stroke volume (SV) was calculated as the product of the left ventricular outflow tract (LVOT) area and the velocity-time integral (VTI) in the LVOT obtained by pulsed-wave Doppler. Cardiac output (CO) was determined by the formula: $CO = SV \times HR$. Total peripheral vascular resistance (TPR) was calculated based on MAP data from ABPM and CO: $TPR\ (dyn \cdot s \cdot cm^{-5}) = (MAP / CO) \times 80$. Additionally, left ventricular myocardial mass (LVMM) indexed to $height^{2.7}$, as well as diastolic function assessed by tissue Doppler imaging, were evaluated.

Assessment of Vascular Wall Status was performed by measuring carotid-femoral pulse wave velocity (PWV) using the "Artlab" device. This parameter is a recognized marker of large artery stiffness.

Laboratory Tests included fasting venous blood sampling to determine basic biochemical parameters (glucose, lipid profile) and specific hormonal-metabolic markers. To assess insulin resistance, the HOMA-IR index was calculated using the formula: (fasting glucose, mmol/L × fasting insulin, μU/mL) / 22.5. Levels of leptin, adiponectin, testosterone, and estradiol were determined by enzyme-linked immunosorbent assay.

Collection of Anamnestic Data was carried out using structured questionnaires completed jointly by parents and the adolescent. The questionnaire included sections on family history of cardiovascular and metabolic diseases, dietary habits, level of physical activity (using the validated IPAQ-A questionnaire for adolescents), and lifestyle details.

Statistical Analysis

Data processing was performed using the IBM SPSS Statistics 26.0 software package. Description of quantitative indicators was performed by calculating the median and interquartile range (Me [Q25; Q75]), as the distribution of most parameters deviated from normal, which was checked using the Shapiro-Wilk test. For comparison of independent groups, the Mann-Whitney U test was used; for comparison of related measurements over time, the Wilcoxon test was applied. Assessment of correlations between features was performed using Spearman's rank correlation coefficient. To build multivariate models predicting BP level or hemodynamic type, logistic and linear regression analyses were used. The level of statistical significance (p) in all tests was set at less than 0.05. This comprehensive methodological approach ensured high measurement accuracy, reproducibility of results, and depth of analysis of multifactorial relationships underlying hemodynamic disorders in adolescents with excess body weight.

Results and Discussion

Following the 24-month prospective observation, data from the comprehensive examination of 540 adolescents were obtained and systematized. Results from 498 participants who completed all three visits according to the protocol were included in the analysis (attrition rate was 7.8%, mainly due to relocation).

Cohort Characteristics at Baseline

Baseline anthropometric and demographic indicators of participants grouped by body weight status are presented in **Table 2**. The groups were comparable in age and sex, ensuring the validity of further comparisons. As expected, adolescents with obesity had significantly higher absolute and relative fat mass values measured by bioimpedance analysis, as well as central obesity, evidenced by significantly greater waist circumference and WC/HC ratio. Notably, the distribution by pubertal stage (Tanner) across groups also showed no statistically significant differences, excluding the influence of this factor on intergroup comparisons of baseline parameters.

Table 2. Baseline Anthropometric and Demographic Characteristics of the Examined Adolescents (Visit V0)

Parameter	Normal Weight Group (n=167)	Overweight Group (n=183)	Obesity Group (n=148)	p-value*
Age, years	14.2 [13.1; 15.8]	14.5 [13.3; 15.9]	14.3 [12.9; 15.6]	0.451
Males, n (%)	85 (50.9%)	92 (50.3%)	78 (52.7%)	0.891
Pubertal Stage (Tanner), n (%): **Early (II-III)** **Late (IV-V)**	71 (42.5%) 96 (57.5%)	80 (43.7%) 103 (56.3%)	68 (45.9%) 80 (54.1%)	0.832
BMI, kg/m²	19.1 [17.8; 20.5]	24.8 [23.1; 26.3]	31.5 [29.2; 34.1]	<0.001
Waist Circumference (WC), cm	68.5 [65.0; 72.0]	82.1 [78.5; 86.0]	98.3 [92.7; 104.5]	<0.001
WC/HC Ratio	0.81 [0.78; 0.84]	0.88 [0.85; 0.91]	0.96 [0.92; 0.99]	<0.001
Fat Mass, %	18.2 [15.1; 21.5]	28.4 [25.7; 31.8]	38.9 [35.2; 42.5]	<0.001

Note: *Kruskal-Wallis test (quantitative data) and χ^2 test (categorical data) were used for group comparison.

Dynamics of Hemodynamic Parameters and AH Prevalence

The results of 24-hour ABPM and key calculated hemodynamic indicators, assessed dynamically over the two-year observation period, are summarized in **Table 3**. By the end of the study (V2), the proportion of adolescents with AH diagnosed by ABPM in the obesity group reached 34.5%, which was significantly higher than in the overweight group (18.0%) and the normal weight group (4.8%). Isolated systolic AH predominated in the obesity group. Analysis of hemodynamic mechanisms revealed clear age and group dynamics. Adolescents with obesity already at baseline had a significantly higher cardiac output associated with increased stroke volume. However, by visiting V2, this same group also developed a statistically significant increase in total peripheral vascular resistance compared to the normal weight group. Such a pronounced increase in TPR was not recorded in adolescents with overweight. The left ventricular mass index progressively increased from the normal weight to the obesity group, reaching maximum values by visit V2, indicating the development of concentric hypertrophy as a marker of long-term hemodynamic load.

Table 3. Dynamics of BP, Hemodynamic, and Cardiac Structure Indicators (ABPM and EchoCG Results)

Visit	Normal Weight Group (n=167)	Overweight Group (n=183)	Obesity Group (n=148)	p-value* (Intergroup at V2)
		AH Prevalence, n (%)		
V0	5 (3.0%)	15 (8.2%)	32 (21.6%)	<0.001
V2	8 (4.8%)	33 (18.0%)	51 (34.5%)	
		Mean Daytime SBP, mm Hg		
V0	108 [103; 113]	112 [107; 117]	118 [112; 124]	<0.001
V2	110 [105; 115]	116 [110; 121]	125 [119; 131]	
		Stroke Volume (SV), ml		
V0	65 [59; 72]	72 [66; 78]	81 [74; 88]	<0.001
V2	72 [66; 79]	78 [71; 84]	86 [78; 93]	
		Cardiac Output (CO), L/min		
V0	4.8 [4.3; 5.3]	5.2 [4.7; 5.7]	5.9 [5.4; 6.5]	<0.001
V2	5.1 [4.6; 5.6]	5.5 [5.0; 6.0]	6.2 [5.6; 6.8]	
		TPR, dyn·s·cm^{-5}		
V0	1450 [1320; 1580]	1520 [1400; 1650]	1480 [1350; 1620]	0.009
V2	1490 [1360; 1620]	1550 [1420; 1690]	1620 [1480; 1770]	

		LVMI, g/m²·⁷		
V0	32.5 [29.0; 36.1]	35.8 [32.0; 39.5]	40.2 [36.0; 44.5]	<0.001

Note: *Kruskal-Wallis test was used to compare values between the three groups at visit V2.

Relationship between Hemodynamic Profile, Pubertal Stage, and Metabolic Markers

Stratification of data by pubertal stage within the obesity group revealed important pathogenetic features **(Table 4)**. In adolescents with obesity at early pubertal stages (Tanner II-III), the leading hemodynamic phenomenon was increased cardiac output with still normal TPR and PWV. In contrast, in adolescents with obesity at late pubertal stages (Tanner IV-V), although cardiac output remained elevated, the leading role in maintaining AH was played by a significant increase in peripheral resistance and arterial stiffness, as evidenced by the rise in PWV. This hemodynamic profile in older adolescents was closely associated with laboratory signs of insulin resistance (high HOMA-IR) and dyslipidemia, as well as with adipokine imbalance – a significant increase in leptin and a decrease in adiponectin.

Table 4. Hemodynamic and Metabolic Parameters in Adolescents with Obesity Depending on Pubertal Stage (Data from Visit V2)

Parameter	Obesity, Early Puberty (Tanner II-III, n=68)	Obesity, Late Puberty (Tanner IV-V, n=80)	p-value*
Cardiac Output (CO), L/min	6.4 [5.9; 7.0]	6.0 [5.5; 6.5]	0.012
TPR, dyn·s·cm⁻⁵	1550 [1420; 1680]	1690 [1560; 1820]	<0.001
Pulse Wave Velocity (PWV), m/s	5.2 [4.9; 5.5]	5.8 [5.5; 6.2]	<0.001
HOMA-IR	3.5 [2.8; 4.2]	4.8 [4.0; 5.7]	<0.001
Leptin, ng/mL	32.5 [26.1; 39.8]	38.9 [31.0; 47.5]	0.003
Adiponectin, µg/mL	8.1 [6.5; 9.8]	6.2 [5.0; 7.5]	<0.001
Triglycerides, mmol/L	1.2 [1.0; 1.5]	1.5 [1.2; 1.9]	<0.001

Note: *Mann-Whitney U test was used for group comparison.

Factors Associated with the Development of Arterial Hypertension

To identify independent predictors of AH formation by the end of the observation period, a multivariate logistic regression analysis was performed, including significant variables from univariate comparisons **(Table 5)**. The model demonstrated that the presence of obesity, transition to a late pubertal stage, and an increase in the HOMA-IR index are independent risk factors for the development of AH. At the same time, an increase in TPR, unlike cardiac output, was a significant hemodynamic predictor directly associated with the AH diagnosis after adjustment for other variables. This confirms the hypothesis of a change in the leading hemodynamic mechanism with the progression of puberty and metabolic disorders.

Table 5. Results of Multivariate Logistic Regression Analysis of Factors Associated with AH Development by Visit V2 (n=498)

Factor	Regression Coefficient (β)	Standard Error	Odds Ratio (OR)	95% Confidence Interval for OR	p-value
Presence of Obesity (vs. normal)	1.85	0.31	6.36	3.46 – 11.68	<0.001
Late Pubertal Stage (Tanner IV-V)	0.92	0.29	2.51	1.42 – 4.43	0.002
HOMA-IR (per 2 units)	0.67	0.18	1.95	1.37 – 2.78	<0.001
TPR (per 100 dyn·s·cm⁻⁵)	0.21	0.08	1.23	1.05 – 1.44	0.009
Cardiac Output (per 1 L/min)	0.31	0.22	1.36	0.88 – 2.10	0.165
Age	0.15	0.09	1.16	0.97 – 1.39	0.098

Thus, the obtained results demonstrate a clear relationship between excess body weight, stage of sexual maturation, and specific changes in central and peripheral hemodynamics, which collectively lead to a high prevalence of arterial hypertension in adolescents with obesity.

The conducted prospective cohort study allowed for a detailed characterization of the features of hemodynamic impairment formation in adolescents with overweight and obesity within the context of pubertal development. The obtained data not only confirm the close epidemiological link between obesity and early arterial hypertension (AH) but, more importantly, uncover the dynamic pathophysiological mechanisms underlying this link (Czogała *et al*., 2021; El-Ayash & Puyau, 2023). The results indicate that the hemodynamic profile of an adolescent with obesity is not static but evolves predictably with the progression of puberty and the worsening of metabolic disorders (Hans *et al*., 2021; Smigoc Schweiger *et al*., 2021; Su *et al*., 2024). Our data on the high prevalence of AH, reaching 34.5% in the obesity group by the end of observation, are consistent with the alarming global trend noted in several major epidemiological studies in recent years (Guo *et al*., 2023; Bongers-Karmaoui *et al*., 2024; Zhou *et al*., 2025). Similar studies in various populations also report that the risk of developing AH in adolescents with obesity is 3-4 times higher than in peers with normal weight (Neves *et al*., 2021; Petro *et al*., 2022; Morales-Ghinaglia *et al*., 2023). However, the present study adds an important qualitative dimension to this statistic by showing that pubertal status acts not merely as a background but as an active modulator of this risk.

A key finding of our work is the identification of a two-phase hemodynamic adaptation. In the early stages of puberty (Tanner II-III), adolescents with obesity exhibit a dominance of a hyperkinetic type of circulation, characterized by a significant increase in stroke volume and cardiac output (up to 6.4 [5.9; 7.0] L/min) with still relatively preserved total peripheral vascular resistance (TPR) (Szakály *et al*., 2024; Zhang *et al*., 2025). This phenomenon is well explained by the classical model of volume overload associated with an increase in total blood and plasma volume, confirmed by bioimpedance data on elevated total body fluid content (Hans *et al*., 2021; Frías *et al*., 2023). A similar hyperkinetic state early in puberty has been described in several fundamental physiological studies on obesity in young individuals and is considered a compensatory mechanism aimed at ensuring perfusion of the increasing body mass (Falkner, 2018; Bujanowicz & Skrzypczyk, 2023; Tran *et al*., 2025).

However, with the transition to late pubertal stages (Tanner IV-V), a fundamentally different component begins to dominate the pathogenesis. We recorded a statistically significant increase in TPR (up to 1690 [1560; 1820] dyn·s·cm^{-5}) and pulse wave velocity (PWV), which unequivocally indicates increased vascular tone and arterial wall stiffness. This transition from a volume-dependent to a resistance-dependent form of AH is a critically important point. The results of our regression analysis confirm that it is TPR, not cardiac output, that acts as an independent hemodynamic predictor of established AH (Litwin, 2024). This evolution of the pattern is fully consistent with the conclusions of a number of pathophysiological studies showing that, over time, neurohormonal factors associated with obesity begin to prevail over purely mechanical ones (Hall *et al*., 2021; Aryee *et al*., 2023; Moran-Lev *et al*., 2023).

The close association found between the rise in TPR, insulin resistance (HOMA-IR = 4.8 [4.0; 5.7]), and adipokine imbalance (hyperleptinemia and hypoadiponectinemia) in the late puberty subgroup allows us to propose a holistic cause-and-effect model **(Figure 2)**. The accumulation of visceral adipose tissue, which worsens during puberty, triggers a cascade of metabolic events (Morais *et al*., 2022). Insulin resistance and compensatory hyperinsulinemia, on one hand, directly stimulate the sympathetic nervous system and sodium reabsorption in the kidneys (Hall *et al*., 2021; Xu *et al*., 2024). On the other hand, dysfunctional adipose tissue produces an excess of proinflammatory cytokines and leptin while being deficient in anti-inflammatory adiponectin (Engin, 2024). This leads to endothelial dysfunction, impaired nitric oxide-mediated vasodilation, and structural remodeling of the vascular wall (Koenen *et al*., 2021). Sex hormones, whose concentrations peak in late puberty, may modulate this process, which requires further study. The interconnection of these processes is reflected in the following scheme:

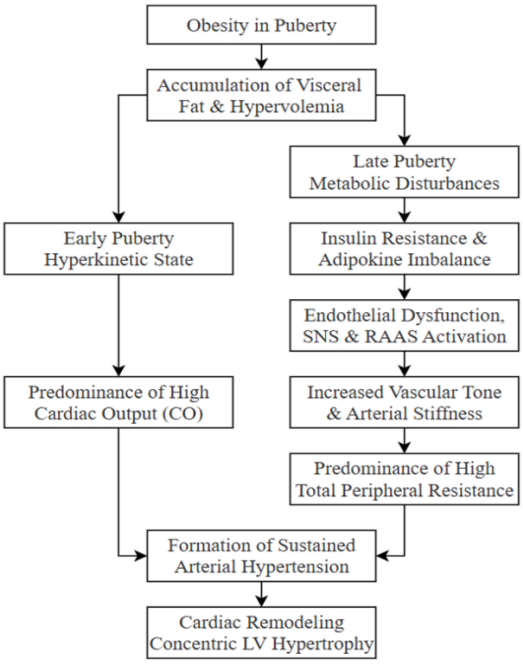

Figure 2. The proposed two-phase model of hemodynamic evolution in adolescents with obesity. The model illustrates the temporal relationship between metabolic and vascular changes during puberty. Abbreviations: SNS, sympathetic nervous system; RAAS, renin-angiotensin-aldosterone system; LV, left ventricular.

The revealed progressive increase in the left ventricular mass index (LVMI) to 45.1 [40.5; 49.8] g/m$^{2.7}$ in the obesity group is a direct consequence of prolonged combined hemodynamic load—both volume and resistive. Concentric left ventricular hypertrophy in adolescents with obesity is not merely a marker of current AH but a powerful independent predictor of cardiovascular events in adulthood, as confirmed by data from long-term cohort studies. Thus, our results indicate that puberty is a critical window during which subclinical target organ damage is established (Constantin *et al*., 2022; Mojsak *et al*., 2022; Sugimori *et al*., 2022; Essah *et al*., 2024; Frost *et al*., 2024; Kajanova & Badrov, 2024; Lee & Ferreira, 2024; Rosellini *et al*., 2024; Umarova *et al*., 2024).

Several study limitations should be acknowledged. First, the 24-month observation period, while significant for a prospective study, is still insufficient to assess long-term cardiac outcomes. Second, the assessment of physical activity and diet, despite using validated questionnaires, may carry an element of subjectivity. Third, genetic factors that may influence both predisposition to obesity and vascular wall sensitivity were not studied (Delcea *et al*., 2024; Essah *et al*., 2024; Ribeiro *et al*., 2024; Sanlier & Yasan, 2024; Uneno *et al*., 2024).

The practical significance of the obtained results lies in the potential for a personalized approach to the prevention and early therapy of AH in adolescents with obesity. Screening should be aimed not only at detecting elevated blood pressure but also at assessing the hemodynamic profile using accessible methods (EchoCG, PWV) and metabolic status. For adolescents in early

puberty with a hyperkinetic type of circulation, non-pharmacological strategies aimed at reducing circulating blood volume may come to the forefront: a low-sodium diet, increased aerobic physical activity. For patients in late puberty stages with signs of elevated TPR, insulin resistance, and endothelial dysfunction, in addition to lifestyle modification, a more active strategy may be considered, including pharmacological correction of vascular tone and metabolism under the close supervision of a specialist (Adeleke, 2022; Sri *et al*., 2022; Simonyan *et al*., 2023; Tsiganock *et al*., 2023; Sanlier & Yasan, 2024).

Thus, this study demonstrates that arterial hypertension in adolescents with obesity is the result of a dynamic interaction between puberty, hemodynamics, and metabolism. Understanding this evolution from a hyperkinetic to a vasoconstrictor type opens new avenues for timely, pathogenetically grounded intervention aimed at breaking the vicious cycle and preventing the formation of irreversible cardiovascular risk in the younger generation (Razhaeva *et al*., 2022; Rojas *et al*., 2022; Al Abadie *et al*., 2023; Guzek *et al*., 2023; Lee *et al*., 2023; Ncube *et al*., 2023; Oran & Azer, 2023).

Conclusion

The conducted prospective multicenter study, which included 498 adolescents from the North Caucasus region, yielded new data deepening the understanding of the pathogenesis of arterial hypertension in the context of the modern obesity epidemic among youth. The obtained results convincingly demonstrate that the relationship between excess body weight and elevated blood pressure in adolescence is not a static phenomenon but a dynamic process that naturally evolves in parallel with pubertal development. It was established that by the end of the two-year observation period, the prevalence of AH confirmed by 24-hour monitoring among adolescents with obesity reached 34.5%, which is more than seven times higher than the rate in their normal-weight peers (4.8%).

A key scientific achievement of this work is the identification of a two-phase hemodynamic restructuring. In the early stages of puberty (Tanner II-III), hypertension formation is primarily driven by a hyperkinetic type of circulation with a significant increase in cardiac output to 6.4 [5.9; 7.0] L/min against a background of relatively preserved peripheral resistance. However, with the transition to the late stages of sexual maturation (Tanner IV-V), a fundamentally different mechanism begins to dominate the pathogenesis—a significant increase in total peripheral vascular resistance to 1690 [1560; 1820] dyn·s·cm^{-5}. Multivariate analysis confirmed that an increase in TPR by every 100 units raised the odds of developing hypertension by 23%, while cardiac output was not an independent predictor.

The obtained data allow for the integration of hemodynamic and metabolic aspects into a unified pathogenetic model. The evolution towards a resistance-dependent form of hypertension is closely linked to the progression of insulin resistance, as evidenced by the rise in the HOMA-IR index to 4.8 [4.0; 5.7] in the late puberty subgroup, which is almost one and a half times higher than in the early stages. A clinical marker of prolonged hemodynamic load was the revealed progressive increase in the left ventricular mass index to 45.1 [40.5; 49.8] g/m$^{2.7}$ in adolescents with obesity, indicating the onset of structural cardiac remodeling already in adolescence.

The practical significance of the study lies in substantiating the need for a differentiated approach. The results show that one in three adolescents with obesity develops persistent arterial hypertension requiring active intervention. Meanwhile, the risk of developing hypertension in adolescents with overweight (18.0%) also remains significantly elevated compared to the norm. Screening should extend beyond a one-time blood pressure measurement and include an assessment of the hemodynamic profile, especially in patients with a body mass index exceeding the 85th percentile.

Thus, puberty in the context of obesity represents a critical window during which the phenotype of future sustained arterial hypertension is formed. The obtained numerical indicators—from a prevalence of 34.5% to specific values of TPR, HOMA-IR, and myocardial mass—objectively confirm the scale of the problem and the need for a systemic approach. Timely identification of the stage of hemodynamic restructuring opens the possibility for early, pathogenetically grounded intervention. The development of personalized programs based on the identified patterns is a necessary step to break the vicious chain linking adolescent obesity with the epidemic of cardiovascular diseases and to shape a healthier cardiometabolic future for the new generation.

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Ethics statement: The study was conducted in accordance with the ethical principles for medical research involving human subjects outlined in the Declaration of Helsinki. Written informed consent was obtained from the legal guardians (parents or custodians) of all adolescent participants prior to enrollment. Additionally, written assent was obtained from each participating adolescent aged 12 years and older, ensuring they understood the study's purpose, procedures, potential discomforts, and their right to withdraw at any time without affecting their future medical care. The consent process emphasized the voluntary nature of participation.

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