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Integrated clinical and metabolic assessment of children with obesity and arterial hypertension

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Abstract.

Introduction. Childhood and adolescent obesity is one of the most important medical and social challenges in modern pediatrics and pediatric cardiology. The increasing prevalence of abdominal obesity is accompanied by a growing incidence of arterial hypertension, insulin resistance, dyslipidemia, and early cardiovascular disorders. Despite numerous studies, the relationships between clinical, metabolic, and cardiovascular alterations in obese children remain insufficiently understood, emphasizing the need for a comprehensive assessment of cardiometabolic risk factors. **Objective**. To investigate the clinical and metabolic characteristics of children with obesity, evaluate the relationships between anthropometric parameters, disorders of carbohydrate and lipid metabolism, arterial hypertension, and cardiovascular changes, and determine the structure of metabolic syndrome components in pediatric patients. **Materials and Methods**. A prospective clinical and laboratory study included 55 children aged 10–18 years with exogenous constitutional obesity. The control group consisted of 20 age-matched healthy children. All participants underwent comprehensive clinical examination, anthropometric assessment, body mass index calculation, waist circumference and waist-to-hip ratio measurements, blood pressure evaluation, electrocardiography, and biochemical investigations including carbohydrate and lipid metabolism parameters, serum uric acid, immunoreactive insulin, and HOMA-IR index determination. Statistical analysis included correlation analysis between clinical and laboratory variables. **Results and Discussion.** Children with abdominal obesity and arterial hypertension demonstrated an earlier onset of the disease, accelerated physical growth and pubertal development, a higher prevalence of functional cardiovascular abnormalities, and persistent elevation of arterial blood pressure. Disorders of carbohydrate metabolism were detected in approximately one-third of the patients and were accompanied by elevated HOMA-IR values, indicating insulin resistance. Hypertriglyceridemia, increased total cholesterol and low-density lipoprotein cholesterol, together with reduced high-density lipoprotein cholesterol, were significantly more common in children with abdominal obesity. Serum uric acid concentrations were increased and showed significant positive correlations with body mass index and blood pressure. Correlation analysis demonstrated close associations between obesity severity, carbohydrate and lipid metabolism disorders, hyperuricemia, and cardiovascular abnormalities, confirming the common pathogenetic mechanisms underlying metabolic syndrome and cardiovascular disease in obese children. Conclusion. Children with abdominal obesity, particularly those with concomitant arterial hypertension, exhibit the most pronounced clinical, metabolic, and cardiovascular abnormalities. The development of insulin resistance, dyslipidemia, hyperuricemia, and functional cardiovascular changes is closely associated with increasing obesity severity and elevated blood pressure. These findings highlight the importance of early identification of metabolic syndrome components and comprehensive assessment of cardiometabolic risk factors to prevent long-term cardiovascular complications in obese children.

Key words: obesity, abdominal obesity, arterial hypertension, metabolic syndrome, insulin resistance, children.

Introduction. Childhood and adolescent obesity has become one of the most important public health challenges worldwide. The increasing prevalence of obesity, together with the growing incidence of arterial hypertension and metabolic abnormalities affecting carbohydrate and lipid metabolism, highlights the importance of investigating metabolic syndrome (MS) in the pediatric population [2. 3. 6].

Although numerous studies have examined metabolic syndrome in adolescents and a limited number have focused on younger children, many pathogenetic mechanisms underlying its development remain insufficiently understood. Furthermore, the application of the International Diabetes Federation (IDF) diagnostic criteria to pediatric patients

remains controversial. One of the major limitations is the absence of universally accepted definitions of abdominal obesity in children, despite its recognized role as the principal component of metabolic syndrome and an independent predictor of future metabolic and cardiovascular complications in adulthood [1.5].

Arterial hypertension represents one of the key components of metabolic syndrome. Children with concomitant obesity and hypertension are at particularly high risk of developing persistent metabolic disturbances and cardiovascular diseases later in life. Therefore, this group represents an important target population for investigating the mechanisms responsible for the development and progression of obesity-related cardiovascular and metabolic complications [4.7].

Aim of the Study: The aim of this study was to identify the clinical and metabolic characteristics of children with obesity and arterial hypertension and to determine the prevalence and structure of metabolic syndrome components in this patient population.

Materials and Methods

A total of 55 children with exogenous constitutional obesity were enrolled in this study. Patient selection was based on the presence of overweight or obesity, defined as a body mass index (BMI) and waist circumference above the 97th percentile for age and sex according to the World Health Organization (WHO) growth standards (2006). The study population included 30 boys (55%) and 25 girls (45%), aged 10–18 years, with a mean age of 14.35 ± 0.21 years.

All participants underwent a comprehensive clinical examination using standard pediatric assessment protocols. Anthropometric evaluation included measurements of body weight, height, BMI (Quetelet index), waist circumference (WC), and hip circumference (HC). BMI was interpreted using age- and sex-specific percentile reference values recommended by the WHO (1998). The waist-to-hip ratio (WHR) was calculated as an indicator of abdominal fat distribution. Abdominal obesity was defined as a WHR greater than 0.85 in girls and greater than 0.90 in boys, in accordance with the International Diabetes Federation (IDF) recommendations.

Based on fat distribution and blood pressure status, children with obesity were divided into two principal groups. Group I included 17 children with generalized obesity but without abdominal fat accumulation. Their mean waist circumference was 80.11 ± 1.36 cm, and the mean WHR was 0.87 ± 0.01. Group II consisted of 38 children with abdominal obesity, characterized by a mean waist circumference of 99.82 ± 1.30 cm and a mean WHR of 0.92 ± 0.009. Within this group, 20 children had normal arterial blood pressure (Group IIA), whereas 18 children had confirmed arterial hypertension (Group IIB). The differences in WHR between Groups I and II were statistically significant ($P < 0.05$).

The overall mean BMI of the study population exceeded the 97th percentile and was 31.27 ± 0.51 kg/m^2, ranging from 23.5 to 47.2 kg/m^2. Children in Group I had a mean BMI of 28.85 ± 0.52 kg/m^2, whereas those in Group II demonstrated significantly higher BMI values (35.37 ± 0.63 kg/m^2; $P < 0.01$).

The control group consisted of 20 healthy children without obesity (11 boys and 9 girls), with a mean age of 14.31 ± 0.63 years. Their mean waist circumference was 64.0 ± 1.51 cm, and the mean WHR was 0.81 ± 0.02. WHR values differed significantly from those observed in both Group I ($P < 0.01$) and Group II ($P < 0.001$). All children in the control group were classified as Health Group I according to routine pediatric health assessment. The mean BMI in the control group was 19.44 ± 0.47 kg/m^2 (range: 18.2–20.4 kg/m^2), which was significantly lower than that of the study groups ($P < 0.001$).

Biochemical analyses included determination of fasting serum glucose using the glucose oxidase method. Total cholesterol and high-density lipoprotein cholesterol (HDL-C) concentrations were measured by enzymatic colorimetric assays. Low-density lipoprotein cholesterol (LDL-C) and very-low-density lipoprotein cholesterol (VLDL-C) were calculated using the Friedewald equation. Serum and urinary uric acid concentrations were determined spectrophotometrically according to the method of Morimont and London (1982). Serum immunoreactive insulin (IRI) levels were measured using enzyme-linked immunosorbent assay (ELISA), and insulin resistance was assessed by calculating the Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) index using the standard formula [8].

Results and Discussion. The analysis of the clinical characteristics of children with obesity demonstrated significant differences in the age of disease onset among the study groups. In children with generalized obesity (Group I), the first signs of excessive weight gain followed by the rapid development of obesity were most frequently observed between 12 and 14 years of age, corresponding to the onset of puberty. The mean age at disease

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manifestation in this group was 12.2 ± 0.8 years. In contrast, children with abdominal obesity (Group IIA) and those with abdominal obesity combined with arterial hypertension (Group IIB) exhibited a significantly earlier onset of obesity, occurring at 9.5 ± 0.6 years and 8.9 ± 0.5 years, respectively ($P < 0.01$ compared with Group I).

Sex-specific analysis revealed differences in both the prevalence and timing of obesity onset. Among boys, the highest proportion of obesity cases was recorded at 15–16 years of age (36.6%), whereas the average age at disease onset was 8.3 ± 0.5 years. In girls, the greatest prevalence was observed between 13 and 14 years of age (36.6%), with obesity developing significantly earlier, at a mean age of 7.4 ± 0.3 years.

Anthropometric assessment showed that the body mass index (BMI) of all examined children exceeded the 97th percentile for age and sex. The mean BMI for the entire study cohort was 31.27 ± 0.51 kg/m^2, with values ranging from 23.5 to 47.2 kg/m^2. Children with generalized obesity had a mean BMI of 28.85 ± 0.52 kg/m^2, whereas significantly higher values were recorded in children with abdominal obesity (35.37 ± 0.63 kg/m^2; $P < 0.01$).

Waist circumference increased progressively with increasing severity of obesity. In Group I, BMI demonstrated a weak positive correlation with waist circumference ($r = 0.456$), whereas no meaningful association was observed between BMI and the waist-to-hip ratio ($r = 0.341$). Similar findings were obtained in children with abdominal obesity. Waist circumference showed a significant positive correlation with BMI both in Group IIA ($r = 0.640$; $P < 0.01$) and Group IIB ($r = 0.708$; $P < 0.01$). In contrast, the waist-to-hip ratio was only weakly associated with BMI ($r = 0.124$ and $r = 0.199$, respectively). These findings suggest that waist circumference is a more reliable indicator of abdominal fat accumulation in children than the waist-to-hip ratio, which is consistent with previous reports.

Evaluation of arterial blood pressure revealed marked differences between the study groups. In children with generalized obesity, systolic and diastolic blood pressure values exceeded the age- and sex-specific 95th percentile at the initial examination in four patients (23.5%). However, elevated blood pressure was not confirmed on repeated measurements, and these cases were therefore classified as white-coat hypertension. Among children with abdominal obesity, white-coat hypertension was identified more frequently, affecting 11 patients (28.9%). In addition, 18 children (47.3%) fulfilled the diagnostic criteria for Grade 1 arterial hypertension and were assigned to the subgroup with abdominal obesity and hypertension. In these patients, both systolic and diastolic blood pressure values consistently exceeded the 97th percentile for age and sex.

Correlation analysis demonstrated a significant positive relationship between BMI and blood pressure. In children with generalized obesity, BMI correlated with systolic blood pressure ($r = 0.602$) and diastolic blood pressure ($r = 0.589$). Similar associations were observed in Group IIA (SBP: $r = 0.618$; DBP: $r = 0.602$), while the strongest correlations were identified in children with abdominal obesity and hypertension (SBP: $r = 0.701$; DBP: $r = 0.658$).

Assessment of physical development showed that accelerated linear growth was common among obese children. Tall stature was identified in 17 children (47.0%) with generalized obesity and in 19 children (50.0%) with abdominal obesity. Within the abdominal obesity group, tall stature was observed in 12 children (60.0%) without hypertension and in seven children (38.8%) with concomitant arterial hypertension.

Dermatological examination revealed characteristic obesity-associated skin manifestations. Striae, ranging in color from pale pink to dark purple and predominantly located on the abdomen, shoulders, and thighs, were identified in 29.4% of children with generalized obesity. Acanthosis nigricans was observed in 11.7% of these patients. Among children with abdominal obesity, the prevalence of these cutaneous findings was comparable between subgroups: striae were detected in 35.0% of patients without hypertension and 33.3% of those with hypertension, whereas acanthosis nigricans was present in 15.0% and 16.6% of patients, respectively.

Assessment of pubertal development according to the Tanner classification (1969) demonstrated that the majority of both male and female adolescents with obesity had reached stages IV and V of sexual maturation. Children at Tanner stage I were virtually absent from the study groups, indicating that obesity was predominantly diagnosed during the later stages of pubertal development.

Evaluation of health-related quality of life revealed that recurrent headache was the most frequently reported complaint among obese children. Headaches were typically associated with emotional stress and occurred predominantly during the afternoon and evening. This symptom was reported by 70.5%, 80.0%, and 88.8% of children in Groups

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I, IIA, and IIB, respectively, whereas only 20% of children in the control group experienced occasional headaches.

Episodes of discomfort or tingling sensations in the precordial region were also commonly reported. Such symptoms occurred both at rest and during physical activity and were observed in 29.4%, 35.0%, and 44.4% of patients in Groups I, IIA, and IIB, respectively. In contrast, only 10% of children in the control group reported transient cardiac discomfort associated with emotional or physical exertion.

Electrocardiographic examination identified functional cardiac abnormalities in a considerable proportion of obese children. Sinus tachycardia and/or sinus bradycardia were detected in 15 patients (21.8%). An ectopic atrial rhythm, which converted to accelerated sinus rhythm upon assuming the upright position, was observed in four children (7.2%). Incomplete bundle branch block was diagnosed in six patients (10.9%). In the control group, sinus rhythm disturbances were detected in only one child (5.0%), while incomplete bundle branch block was likewise identified in one participant.

The worldwide increase in childhood obesity has been accompanied by a growing prevalence of metabolic syndrome (MS). Depending on the diagnostic criteria applied, the reported prevalence of MS in children and adolescents ranges from 0.4% to 25%. Therefore, one of the principal objectives of the present study was to evaluate the frequency and characteristics of metabolic syndrome components among children with obesity[9,10].

Analysis of carbohydrate metabolism demonstrated that the mean fasting glucose, postprandial glucose, and immunoreactive insulin (IRI) concentrations remained within the normal reference ranges in all study groups. Nevertheless, the HOMA-IR index was significantly elevated ($P < 0.05$), indicating the presence of insulin resistance despite apparently normal glycemic parameters. Furthermore, fasting glucose, postprandial glucose, and IRI values increased progressively with increasing severity of obesity and were significantly higher than those observed in the control group ($P < 0.05$) (Table 1).

Table 1. Indicators of Carbohydrate Metabolism in the Study Groups

Parameters	Control Group	Group I	Group IIA	Group IIB
Fasting plasma glucose (mmol/L)	3.25 ± 0.41	4.02 ± 0.68	4.92 ± 0.52	5.08 ± 0.41
2-hour postprandial plasma glucose (mmol/L)	6.07 ± 0.52	6.62 ± 0.61	7.02 ± 0.55	7.94 ± 0.51
Serum insulin (μIU/mL)	7.89 ± 0.80	9.36 ± 0.80	10.91 ± 0.80	10.97 ± 0.98
HOMA-IR index	2.57 ± 0.83	2.89 ± 0.83	3.68 ± 0.78	4.39 ± 2.11*

Note: $P < 0.05$ compared with the control group.

Evaluation of the prevalence of carbohydrate metabolism disorders showed elevated fasting plasma glucose in 17.6%, 20.0%, and 27.7% of children in Groups I, IIA, and IIB, respectively. Elevated postprandial glucose concentrations were identified in 5.8%, 15.0%, and 22.2% of patients in the corresponding groups. Increased serum immunoreactive insulin levels were detected in 23.5%, 25.0%, and 27.7% of patients, respectively. No clinically significant carbohydrate metabolism abnormalities were observed in the control group; however, two children (10%) demonstrated mildly elevated HOMA-IR values due to fasting glucose and insulin concentrations approaching the upper limit of the normal range.

Abdominal obesity represents the principal clinical manifestation of metabolic syndrome and is strongly associated with disturbances in lipid metabolism, particularly hypertriglyceridemia. Similar findings were obtained in the present study. Overall, approximately one-third of obese children exhibited abnormalities of lipid metabolism.

Hypertriglyceridemia was identified in 29.4%, 30.0%, and 38.8% of patients in Groups I, IIA, and IIB, respectively. Mean triglyceride concentrations were 1.56 ± 0.25 mmol/L, 1.92 ± 0.16 mmol/L, and 2.30 ± 0.23 mmol/L in the respective groups.

The dyslipidemia associated with metabolic syndrome is characterized by elevated total cholesterol and low-density lipoprotein cholesterol (LDL-C), accompanied by reduced high-density lipoprotein cholesterol (HDL-C). This lipid profile substantially increases the risk of future cardiovascular disease, including coronary artery disease and myocardial infarction. Moreover, dyslipidemia promotes the accumulation of atherogenic lipoproteins, increases blood viscosity and peripheral vascular resistance, and contributes to the persistence of elevated arterial blood pressure[12.13.14].

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Elevated or borderline total cholesterol concentrations were detected in 35.2%, 35.0%, and 44.4% of children in Groups I, IIA, and IIB, respectively. Mean total cholesterol levels progressively increased with abdominal obesity, reaching 4.56 ± 0.58 mmol/L in Group I, 5.01 ± 0.33 mmol/L in Group IIA, and 5.76 ± 0.52 mmol/L in Group IIB, all of which exceeded the values recorded in the control group.

A reduction in HDL-C concentration was observed in 17.6%, 25.0%, and 22.2% of children in Groups I, IIA, and IIB, respectively. Mean HDL-C levels were 1.22 ± 0.12 mmol/L, 1.13 ± 0.09 mmol/L, and 1.03 ± 0.07 mmol/L. Conversely, LDL-C concentrations increased progressively with disease severity, averaging 3.04 ± 0.23 mmol/L, 3.66 ± 0.18 mmol/L, and 4.14 ± 0.39 mmol/L in Groups I, IIA, and IIB, respectively. Elevated LDL-C values were detected in 29.4%, 35.0%, and 44.4% of patients.

Recent evidence suggests that hyperuricemia constitutes an additional component involved in the pathogenesis of metabolic syndrome, including in the pediatric population. In the present study, mean serum uric acid concentrations remained within the reference range but were significantly higher than those of the control group. Serum uric acid levels demonstrated significant positive correlations with both obesity severity ($r = 0.592$, $P < 0.001$) and arterial blood pressure ($r = 0.446$ and $r = 0.369$, $P < 0.001$).

Children with generalized obesity had a mean serum uric acid concentration of 0.324 ± 0.011 mmol/L compared with 0.180 ± 0.013 mmol/L in healthy controls ($P < 0.01$). Hyperuricemia was identified in three children (17.6%) within this group, with a mean uric acid concentration of 0.366 ± 0.010 mmol/L. Among children with abdominal obesity, hyperuricemia was detected in approximately one-quarter of cases, with a mean concentration of 0.415 ± 0.021 mmol/L and an overall group average of 0.369 ± 0.012 mmol/L. The highest uric acid levels were observed in children with abdominal obesity combined with arterial hypertension, in whom the mean concentration reached 0.398 ± 0.130 mmol/L, while hyperuricemia was present in 55.5% of patients, with a mean value of 0.413 ± 0.030 mmol/L.

Conclusions. The present study demonstrated that abnormalities of carbohydrate metabolism were identified in approximately one-quarter of children with generalized obesity and in nearly one-third of those with abdominal obesity combined with arterial hypertension. Disturbances of lipid metabolism were detected in approximately one-third of patients across all obesity groups and were accompanied by elevated serum uric acid concentrations. Increased uric acid levels showed significant positive correlations with both body mass index (BMI) and arterial blood pressure, suggesting their close association with the severity of metabolic disturbances [11,15,16].

Children with abdominal obesity and concomitant arterial hypertension exhibited more pronounced clinical manifestations than those with generalized obesity alone. These patients were characterized by accelerated linear growth, advanced pubertal development, impaired quality of life, and a higher prevalence of cardiovascular abnormalities, including functional cardiac disorders and persistent elevation of arterial blood pressure. These findings emphasize the importance of early identification of metabolic and cardiovascular risk factors in obese children to prevent long-term complications.

Study Transparency.

The study was not sponsored. The authors are solely responsible for submitting the final version of the manuscript for publication.

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All authors participated in the conception and design of the study and in writing the manuscript. The final version of the manuscript was approved by all authors. The authors did not receive any fees for the study.

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