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КАРДИОВАСКУЛЯРНАЯ СИСТЕМА У ДЕТЕЙ И ПОДРОСТКОВ ПРИ ОЖИРЕНИИ

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АННОТАЦИЯ

Обследовано 60 подростков с ожирением. Выявлено, что на развитие гипертрофии миокарда влияют масса тела, уровень АД, процессы вазоконстрикции, а также инсулинорезистентность, гиперурикемия и атерогенная дислипидемия. У части детей выявлен метаболический синдром, что требует терапии данного состояния для предотвращения ранних осложнений и инвалидизации подростков во взрослом периоде.

Ключевые слова: ожирение, артериальная гипертензия, гипертрофия миокарда левого желудочка, подростки, дети.
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SEMIZLIGI BOR BALALAR VA O`SMIRLARDA YURAK QON TOMIR TIZIMI

ANNOTASIYA

Semizligi bor 60 nafar bola va o'smir tekshirildi. Miyokardiyal gipertrofiyaning rivojlanishiga tana og'irligi, qon bosimi, vazokonstriksiya jarayonlari, shuningdek insulinga chidamlilik, giperurikemiya va aterogen dislipidemiya ta'sir ko'rsatishi aniqlandi. Ba'zi bolalarda metabolik sindrom ko`zatilib, bu kelajakda yuzaga kelishi mumkin bo`lgan erta asoratlar va nogironlikning oldini olish uchun ushbu holatni vaqtida davolashni talab qiladi. **Kalit so'zlar:** semirish, arterial gipertenziya, chap qorincha miyokardiyal gipertrofiyasi, o'spirinlar, bolalar.

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CARDIOVASCULAR SYSTEM IN CHILDREN AND ADOLESCENTS WITH OBESITY

ANNOTATION

60 adolescents with obesity were examined. It was revealed that the development of myocardial hypertrophy is influenced by body weight, blood pressure, vasoconstriction processes, as well as insulin resistance, hyperuricemia and atherogenic dyslipidemia. Some children have metabolic syndrome, which requires therapy for this condition to prevent early complications and disability in adolescents in the adult period.

Key words: obesity, arterial hypertension, left ventricular myocardial hypertrophy, adolescents, children.

Relevance. In recent years, cardiovascular diseases (CVD) associated with obesity have become one of the most significant medical and social problems in most countries worldwide (Dedov I.I., 2006; Roitberg G.E., 2007; Butrova S.A., 2008; Shilov A.M., 2008). Currently, the main risk factors contributing to the development of cardiovascular pathology in adults are arterial hypertension (AH) and abdominal obesity. It is well known that the risk of cardiovascular pathology originates in childhood; therefore, the increasing prevalence of overweight and obesity among children and adolescents contributes to a higher incidence of cardiovascular diseases and their complications in adulthood. This highlights the need for early detection and correction of the earliest signs of cardiac and vascular pathology associated with obesity and arterial hypertension [1, 3]. These considerations have led to studies aimed at determining the role of obesity and arterial hypertension in myocardial remodeling. According to several authors, eccentric left ventricular hypertrophy (LVH) develops earlier in children with borderline arterial hypertension in the presence of obesity [1, 2].

Objective. To determine the impact of certain risk factors on the development of myocardial remodeling and left ventricular hypertrophy in children with obesity.

Materials and Methods. The main study group included 60 children and adolescents aged 8 to 16 years with exogenous-constitutional obesity. Inclusion criteria were based on body mass index (BMI) and waist circumference measurements exceeding the 97th percentile for age and sex (WHO, 2006). The study included 27 girls

(45%) and 33 boys (55%), with a mean age of 16.87 ± 0.19 years. Participants were divided into groups according to BMI: Group 1 included 22 adolescents with overweight and grade 1 obesity (30.3 ± 1.2 kg/m²); Group 2 included 20 adolescents with a BMI of 33.4 ± 1.1 kg/m²; Group 3 included 18 adolescents with a BMI of 36.1 ± 1.4 kg/m². The control group consisted of 20 healthy adolescents of similar age with a BMI of 22.5 ± 0.9 kg/m².

The study involved standard clinical examinations. Body weight was assessed using percentile tables correlating height and weight or BMI (Quetelet index) for age and sex (WHO, 1998). Waist circumference (WC) and hip circumference (HC) were measured, and their ratio (WC/HC) was used as an indicator of abdominal obesity. Values of WC/HC >0.85 in girls and >0.9 in boys were considered indicative of abdominal obesity (IDF, 1997). Arterial hypertension was diagnosed according to the criteria established by the Expert Committee of the All-Russian Scientific Society of Cardiology and the Association of Pediatric Cardiologists (Moscow, 2009) [5].

Morphometric parameters of the myocardium (left ventricular mass — LVM, left ventricular mass index — LVMI, interventricular septal thickness — IVST, and posterior wall thickness of the left ventricle — PWT) were assessed using echocardiography (Aloka Alpha-7 ultrasound scanner with a cardiology package). Laboratory tests included measurement of total cholesterol, high-density lipoproteins (HDL), and triglycerides in serum using a biochemical analyzer. Serum insulin levels were determined by enzyme-linked immunosorbent assay (ELISA). Insulin resistance (IR) was evaluated using the HOMA-R index, calculated from fasting glucose and insulin levels. A threshold value >2.7 was considered indicative of insulin resistance.

Statistical analysis included descriptive and inferential methods. Mean values (M ± m), correlation coefficients (r), and significance levels (p) were calculated. Differences between groups were considered statistically significant at $p < 0.05$. Correlation analysis was used to assess the relationships between anthropometric, hemodynamic, and metabolic parameters.

Results and Discussion. First, in accordance with the study objective, we assessed the relationship between BMI and systolic and diastolic blood pressure in adolescents. The results showed that systolic and diastolic blood pressure levels were significantly higher in adolescents of Group 3 (135.2 ± 9.1 mmHg, $p < 0.05$) compared with Groups 1 and 2 (116.1 ± 7.2 mmHg and 123.2 ± 6.7 mmHg, respectively). A direct correlation was found between BMI and systolic, diastolic, and mean daily blood pressure ($r = 0.601$; $r = 0.589$; $r = 0.603$, respectively; $p < 0.01$ for all).

Among adolescents with overweight and obesity, “white coat hypertension” was detected in 22.9% of cases, labile hypertension in 16.3%, and stable hypertension in 13.1%. Stable hypertension was more frequently observed in adolescents with grade 3 obesity (6.5%) compared with grades 1 and 2 (4.5%).

Echocardiographic evaluation demonstrated that obesity combined with arterial hypertension leads to structural and geometric remodeling of the left ventricle, primarily manifested by increased wall thickness. Significant correlations were found between BMI and posterior wall thickness ($r = 0.588$; $p < 0.01$), as well as interventricular septal thickness ($r = 0.501$; $p < 0.05$).

Left ventricular hypertrophy initially develops as an adaptive response to pressure overload, maintaining adequate contractile function. The main indicators of LVH are left ventricular mass and its index.

The prevalence of LVH was 40.9% in Group 1, 50% in Group 2, and 61.1% in Group 3. No significant differences in LVMI were observed depending on the type of hypertension: 35.7 ± 3.4 g/m².7 in white coat hypertension, 35.9 ± 4.7 g/m².7 in labile hypertension, and 36.4 ± 4.6 g/m².7 in stable hypertension. These findings suggest that obesity plays a key role in increasing left ventricular mass.

Left ventricular geometric remodeling was observed in nearly one-third of obese adolescents: 30.4% in Group 1, 35.0% in Group 2, and 33.3% in Group 3. Eccentric LVH was diagnosed in 16.3% of patients, while concentric remodeling was found in 11.4%. Concentric LVH, which is associated with the highest cardiovascular risk, was observed in 4.9% of cases and only in adolescents with grade 3 obesity.

Structural remodeling also involved the left atrium. Significant differences in left atrial size were observed between groups (31.4 ± 1.2 mm, 31.8 ± 0.8 mm, and 34.5 ± 1.4 mm in Groups 1, 2, and 3, respectively). A significant correlation between left atrial size and BMI was found ($r = 0.608$; $p < 0.01$), suggesting that left atrial changes may represent an early stage of myocardial remodeling.

Compensatory cardiovascular responses to obesity also involved central hemodynamics. Cardiac output increased progressively with obesity severity (5.5 ± 1.1 L/min, 5.8 ± 0.9 L/min, and 6.2 ± 1.1 L/min in Groups 1, 2, and 3, respectively), indicating an increase in circulating blood volume. This was accompanied by a decrease in total peripheral vascular resistance (TPR) with increasing body weight (1318.8 ± 289.1; 1299.9 ± 274.3; and 1287.4 ± 284.1 dyn·s·cm^{-5}, respectively).

TPR also depended on the type of hypertension: 1287.8 ± 250.7 dyn·s·cm^{-5} in labile hypertension and 1325.6 ± 301.5 dyn·s·cm^{-5} in stable hypertension, reflecting exhaustion of adaptive mechanisms and increased vascular resistance.

Metabolic parameters were also assessed. Impaired glucose tolerance was detected in 22.9% of adolescents, predominantly in Groups 2 and 3 (30% and 44.4%). Serum insulin levels were significantly higher in obese children (14.2 ± 1.2; 16.7 ± 1.5; and 19.3 ± 2.1 μIU/mL in Groups 1, 2, and 3, respectively) compared with controls (9.3 ± 0.8 μIU/mL), despite normal fasting glucose levels.

The prevalence of insulin resistance was 24.5%, increasing with obesity severity (13.6% in Group 1, 25% in Group 2, and 38.8% in Group 3). Significant correlations were found between insulin levels and BMI ($r = 0.545$; $p < 0.01$), as well as between BMI and HOMA index ($r = 0.704$; $p < 0.01$).

Insulin resistance was detected in 3.2% of adolescents with white coat hypertension, 8.1% with labile hypertension, and 11.4% with stable hypertension, indicating its key role in the development of hemodynamic and metabolic disturbances.

Lipid profile analysis showed that with increasing obesity, triglycerides ($r = 0.621$; $p < 0.01$) and low-density lipoproteins (LDL) ($r = 0.501$; $p < 0.05$) increased, while high-density lipoproteins (HDL) decreased ($r = 0.703$; $p < 0.001$).

The combination of obesity, arterial hypertension, insulin resistance, and dyslipidemia indicates the formation of metabolic syndrome. Both complete and incomplete forms were identified, with incomplete forms being more prevalent, reflecting the gradual and progressive development of metabolic disturbances.

Overall, the results emphasize that cardiovascular and metabolic alterations in obese adolescents are interrelated and progress in parallel, forming a complex pathological continuum.

Conclusions. The study demonstrates that obesity in children and adolescents is a major determinant of early cardiovascular remodeling, particularly affecting myocardial structure and function. Increased body weight, elevated arterial pressure, vasoconstriction processes, insulin resistance, and atherogenic dyslipidemia act synergistically to promote the development of left ventricular hypertrophy.

These factors should be regarded as early predictive markers of cardiovascular pathology, as they reflect both structural cardiac changes and systemic metabolic disturbances. The high prevalence of metabolic syndrome, including its incomplete forms, highlights the progressive nature of obesity-related complications.

The findings underline the importance of early diagnosis, regular monitoring, and comprehensive management of obese children and adolescents. Preventive and therapeutic strategies should include lifestyle modification, dietary correction, physical activity enhancement, and, when necessary, pharmacological intervention.

Early and targeted intervention is essential to prevent the progression of cardiovascular and metabolic disorders, reduce the risk of long-term complications, and improve quality of life. Thus, pediatric obesity should be considered not only a metabolic condition but also a significant cardiovascular risk factor requiring multidisciplinary clinical attention.

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