



Arterial Hypertension as a Sign of Disorder of the Cardiovascular System in Children and Adolescents with Overweight and Obesity

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ABSTRACT

This paper makes analyses of the arterial hypertension as a sign of disorder of the cardiovascular system in children and adolescents with overweight and obesity. On this case, research has pinpointed on the various features of the research from both theoretical and methodological points. In conclusion, research has declared outcomes and shortcomings over the obesity topic.

Keywords:

overweight, obesity, children, left ventricular hypertrophy, left ventricular geometry, anthropometric markers

Introduction

According to the World Health Organization, obesity is one of the most obvious but neglected problems in modern medicine. Over the past 30 years, the number of obese and overweight people has doubled. In 2010, 43 million children were overweight and obese [1,3]. The last decade has been characterized by an increased interest of scientists in the course of various diseases against the background of obesity. Its frequency is so high that it has become a non-infectious pandemic. The influence of excess body weight on the formation of cardiovascular pathology is not in doubt, but the pathogenetic mechanisms of an increase in the level of blood pressure in obesity have not yet been studied. The prevalence of hypertension among children and adolescents when using various diagnostic criteria ranges from 0.4 to 8%. Certain risk factors for obesity and hypertension may be present from the earliest period of life; therefore, children and adolescents represent the optimal contingent for studying the onset of

the formation of diseases that are a complication of obesity and hypertension [2,6]. It is known that patients suffering from arterial hypertension for a long time are prone to damage to target organs, including left ventricular hypertrophy. According to the literature, the likelihood of developing left ventricular hypertrophy in persons with normal body weight is 5.5%, and in persons with obesity - 29.9% [1]. In this regard, there is an urgent need to identify early signs of structural and functional changes in the heart and blood vessels at the stages of formation and progression of obesity. Early markers of myocardial hypertrophy in patients with arterial hypertension can be hemodynamic (blood pressure, hydrostatic pressure, vasoconstriction processes) and non-hemodynamic factors (catecholamines, hereditary predisposition, insulin resistance, renin-angiotensin system, obesity) [2-7]. To obtain in-depth data on the pathogenesis of arterial hypertension in overweight individuals,

it is important to assess the state of the system of neurohumoral regulation of hemodynamic homeostasis, to study the relationship of the circadian rhythm of blood pressure, the state of the myocardium and the autonomic nervous system. The aim of the study is to establish the relationship of metabolic disorders and assess the relationship of structural and functional rearrangement of the heart and blood vessels with the identification of prognostic significant predictors of the formation of cardiovascular pathology in children and adolescents with excess body weight and obesity.

Materials And Research Methods

We examined 85 children and adolescents (39 girls and 44 boys) aged 8-17 years (average age 12.5 ± 1.3 years), of which 64 people were overweight (BMI, kg / m² within 75 - 97th percentile) and 21 - with obesity (BMI, kg / m² > 97th percentile). The proportion of prepubescent children (8-9 years old) was 16.8% (n = 19), adolescents (10-17 years old) - 83.2% (n = 66). The inclusion criteria for patients in the study were the absence of acute infectious or exacerbation of chronic diseases, previous metabolic and antihypertensive therapy for at least 6 months. Patients with secondary obesity and symptomatic hypertension were excluded from the study. To achieve this goal, the main research group was divided into two subgroups depending on the type of fat deposition. The 1st group comprised 41 (48.2%) people (19 girls and 22 boys) with visceros-abdominal fat deposition, diagnosed in children and adolescents aged 8-16 years when the OT of the 90th percentile was exceeded (IDF, 2007), and in patients 15-16 years old - with a ratio of OT / OB > 0.85 - in girls and > 0.9 - in boys. The 2nd subgroup (subsequently designated as the subgroup with uniform obesity) consisted of 44 (51.8%) people with gluteo-femoral and uniform fat deposition (23 girls and 21 boys) diagnosed in patients aged 8-15 years with OT 0.05). This made it possible to consider the designated subgroups statistically comparable in one of the main features affecting the studied clinical and metabolic disorders. The control group consisted of 33 children and adolescents 8-17 years old (mean age 12.7 ± 3.4

years) without excess body weight (BMI <75th percentile), without AH and metabolic disorders. There were 13 girls and 20 boys. This contingent was selected in the city family polyclinics of Samarkand. All children were assigned to health groups I and II. In the complex examination of patients of the main and control groups, laboratory and instrumental research methods were used, and all children underwent anthropometry (length and weight, abdominal circumference, hips) with the calculation of body mass index (BMI), percentage of deficiency / excess body weight, circumference ratio abdomen to thigh circumference and abdominal circumference to growth. Daily monitoring of blood pressure was carried out. Echocardiography was carried out according to the standard technique with the calculation of the mass of the left ventricular myocardium (LVMM), the LV myocardial mass index (LVMI). The percentile tables were used to identify the gradations of LVMI. The relative wall thickness of the LV myocardium was calculated and the phenotypes of the LV geometry were determined. Results. In the main group, the assessment of the correlation of the severity of obesity with clinical and metabolic disorders revealed the presence of linear correlations between BMI and WC (r = 0.67; p = 0.001), WC / OB (r = 0.24; p = 0.009), levels SBP (r = 0.47; p = 0.001) and DBP (r = 0.38; p = 0.001), serum MC concentration (r = 0.47; p = 0.001), lipid spectrum indicators - total cholesterol (r = 0.23; p = 0.015) and TG (r = 0.27; p = 0.007), as well as fasting glycemia (r = 0.25; p = 0.007) and IRI level (r = 0.41; p = 0.032). At the same time, these correlations were more significant in visceral obesity than in uniform obesity. So, in the 1st subgroup, MTI correlated with OT (r = 0.80; p = 0.001), OT / OB (r = 0.46; p = 0.001), with SBP (r = 0.57; p = 0.001), DBP (r = 0.55; p = 0.001), MC level (r = 0.63; p = 0.001), total cholesterol (r = 0.41; p = 0.04) and fasting glycemia (r = 0.34; p = 0.002). The results obtained indicate a direct correlation between the severity of obesity and the nature of visceralization of fat deposits, the level of blood pressure, HG, dyslipidemia and fasting GGL. Regardless of age, the first clinical complication of overweight is hypertension, the degree and

nature of the course of which significantly affect the further prognosis of obesity and determine the likelihood of early cardiovascular complications. In children and adolescents of the main group, AH was documented in 78.0% (n = 66) of the subjects without significant differences depending on the type of fat deposition: in 82.9% (n = 44) of cases - with visceral and in 70.3% (n = 22) - with uniform obesity (p = 0.121). However, the average SBP / DBP levels in hypertensive patients of the 1st subgroup were significantly higher than those of the 2nd: $146.0 \pm 13.7 / 88.8 \pm 7.1$ mm Hg. against $131.9 \pm 6.5 / 81.9 \pm 7.3$ mm Hg. respectively (p = 0.001). In turn, in the main group, the blood pressure level was linearly associated with the experience of obesity (r = 0.38; p = 0.02), and SBP / DBP - with its severity (BMI, kg / m²) (r = 0.47 ; p = 0.001 / r = 0.38; p = 0.001) and the degree of visceralization of fatty deposits (r = 0.39; p = 0.001 / r = 0.33; p = 0.001). In children and adolescents with excessive fat deposition, the following features of AH were revealed: AH grade II was diagnosed 6.8 times more often (87.2%) than I (12.8%), and in the upper type of fat deposition - more often than in uniform: in 92.6% (n = 63) and 73.1% (n = 19) cases, respectively (p = 0.013). Stable hypertension was recorded 1.5 times more often (60.6%) than labile (39.4%), and also in visceral obesity - more often than in uniform: 70.6% (n = 48) versus 34, 6% (n = 9) cases, respectively (p = 0.002). In terms of structure, the dominant positions were occupied by systolic-diastolic hypertension (60.6%; n = 57) (mean values of IV SBP were $56.9 \pm 9.2\%$, and IV DBP - $31.6 \pm 7.5\%$), which visceral obesity was diagnosed 2 times more often than with uniform obesity: in 70.6% (n = 48) versus 34.6% (n = 9) cases, respectively (p = 0.002). In turn, isolated systolic hypertension was detected in 39.4% (n = 37) of hypertensive patients in the main group (mean IV SBP - $59.7 \pm 5.6\%$), of which 65.4% (n = 17) had uniform obesity. Isolated diastolic hypertension was not recorded. In the main group, the ABPM parameters reflecting the "quality" of the course of AH were linearly associated with metabolic parameters. Thus, direct reliable correlations were obtained between the average SBP level

during the day and the content of TG (r = 0.56; p = 0.02), LDL cholesterol (r = 0.43; p = 0.011) and MC (r = 0, 53; p = 0.02); and also between IV SBP during the day and the level of MC (r = 0.52; p = 0.047). An inverse correlation was determined between the mean daytime DBP and postprandial glycemia (r = -0.55; p = 0.003). In visceral fat deposition, the average daily SBP and DBP levels directly correlated with fasting glycemia (r = 0.22; p = 0.047 and r = 0.26; p = 0.019, respectively), the daytime SBP level - with TG (r = 0.66; p = 0.012), and SBP levels during the day and night hours were in inverse relationship with HDL cholesterol (r = - 0.63; p = 0.014 and r = - 0.66; p = 0.019, respectively). In addition, IV DBP was in direct relationship with the levels of IRI (r = 0.47; p = 0.001) and C-peptide (r = 0.38; p = 0.002), and IV SBP in the daytime and night hours - with the content MC in serum: r = 0.56; p = 0.04 and r = 0.54; p = 0.004, respectively. With a uniform fat deposition, no reliable correlations between metabolic parameters and ABPM parameters were determined. From the results obtained, it follows that in children and adolescents with overweight and obesity (mainly with viscerobdominal fat deposition), disorders of carbohydrate, fat and purine metabolism, underlying the development of IR syndrome, were associated with the formation, stabilization and progression of hypertension. In children with obesity, significantly higher than in the control group were revealed indicators reflecting the dimensions of the LV (posterior wall, interventricular septum, end-diastolic and end-systolic dimensions of the LV), as well as LVMM and LVMMI. LV hypertrophy (LVH) developed in 42.3% of overweight children and 58.3% of obese children. Among children with normal body weight, 73.1% had normal LV geometry, 19.2% had concentric remodeling, and 7.7% had eccentric LVH. In the group of overweight children, normal LV geometry was determined in 34.5% of cases, concentric remodeling was found in 7.7% of children, concentric LVH was observed in 19.2%, and eccentric LVH was observed in 38.6%. In obese children, the distribution of different types of LV remodeling was as follows: 23.3% / 3.3% / 15% / 58.4%, respectively.

LVMM correlated more closely with body area, thigh circumference and shoulder circumference, and LVMI with BMI, percentage of excess body weight, shoulder circumference and lower segment length. Abdominal circumference in children is less associated with LVH.

Discussion

Thus, obesity and arterial hypertension have many points of contact. Most scientists share the opinion that obesity is one of the leading factors affecting the cardiovascular system [1, 7]. The relationship between obesity and arterial hypertension was documented in the Framingham Study, which showed that as body weight increases in relation to height (Quetelet index), the prevalence of hypertension in different age groups in both sexes increases significantly. According to our research, each extra kilogram leads to an increase in systolic blood pressure by 0.36 mm Hg, and diastolic - by 0.1 mm Hg. and as the weight increases, the frequency of diagnosing stable hypertension increases. The relationship between obesity and blood pressure cannot be adequately explained only by hemodynamic changes in the body, since in this case, obese patients would have increased circulating blood volume and cardiac output index, while they remain within normal limits after correction for body weight. The increase in circulating blood volume is adaptive in the early stages of weight gain. In response to the increase in the volume of circulating blood, there is a gradual decrease in the total peripheral vascular resistance, while the amount of decrease is directly proportional to the mass of the patient's adipose tissue. The mechanisms of the possible dependence of arterial hypertension on hyperinsulinemia and insulin resistance are widely studied by the world community. There is still no consensus on this issue, but during puberty there is a close relationship between hyperinsulinemia and arterial hypertension, regardless of the degree of obesity [1]. The analysis of the conducted studies indicates that insulin does not affect the level of blood pressure at physiological concentrations, but statistically significant correlations between the level of insulin and

blood pressure are observed in chronic hyperinsulinemia. It is possible that an increase in blood pressure against the background of hyperinsulinemia occurs as a result of stimulation of the sympathetic division of the autonomic nervous system, an increase in adrenaline release, an increase in cardiac output and general peripheral vascular resistance. An increase in the tone of the sympathetic part of the autonomic nervous system is indirectly evidenced by the revealed violations of the circadian profile of blood pressure in the form of an absence of an adequate nighttime decrease in systolic pressure during sleep and an increase in pressure and pulse variability. Structural adaptation of the heart to overweight is manifested by dilatation of the left atrial cavity, moderate thickening of the left ventricular myocardium, and myocardial remodeling, more often by the type of eccentric hypertrophy. The Framingham study demonstrated a highly significant relationship between body mass index, cavity dimensions, and left ventricular wall thickness. These relationships retained their reliability even after adjusting for age, gender, and blood pressure, which was confirmed in our study. According to the literature, the likelihood of developing left ventricular hypertrophy in individuals with arterial hypertension and normal body weight is 5.5%, and in obese individuals - 29.9% [1]. According to our study, the frequency of diagnosis of left ventricular hypertrophy in obese adolescents in combination with arterial hypertension was 39.3%. Thus, it has been proven that geometric rearrangement of the myocardium as an indirect marker of functional disorders of the left ventricle occurs not only in pathology of the cardiovascular system, but also with an increase in body weight. An increase in the mass of the left ventricular myocardium may be due to an increase in the metabolic requirements of the increased tissue mass. Eccentric left ventricular hypertrophy develops as a result of an increase in the volume of circulating blood against a background of obesity and an increase in cardiac output, which leads to dilatation of the left ventricle and, in accordance with Laplace's law, to an increase in the tension of its walls. Many authors consider

this mechanism as adaptive, designed to normalize the tension of the left ventricular wall. An increase in blood pressure leads to a change in biochemical markers of endothelial dysfunction - atherogenic changes in the lipid spectrum of blood serum, an increase in the level of immunoreactive insulin while maintaining normal serum glucose values. Changes are most pronounced at II and III degrees of obesity and are interrelated with insulin resistance. Violation of the lipid spectrum of blood serum in obese adolescents is characterized mainly by an increase in triglyceride levels and a decrease in the content of low density lipoproteins. These changes are more common in patients diagnosed with insulin resistance, and their frequency increases with the progression of obesity.

Conclusion

Thus, the development of myocardial remodeling and endothelial dysfunction in adolescents is influenced by both hemodynamic (blood pressure level, vasoconstriction processes) and non-hemodynamic factors (atherogenic dyslipidemia, hyperfibrinogenemia, insulin resistance, body weight). These parameters may serve as early markers of myocardial hypertrophy and endothelial dysfunction. Thus, it has been undeniably proven that overweight and metabolic disorders occurring against the background of obesity have an effect on an increase in blood pressure, and are also non-hemodynamic risk factors for the development of cardiac and vascular remodeling. For the purpose of early diagnosis of cardiovascular disorders in obese adolescents, it is necessary to carry out echocardiography, 24-hour blood pressure monitoring, and study of markers of metabolic syndrome and endothelial dysfunction. Overweight and obesity from childhood are risk factors for LVH with the development of various phenotypes of LV geometry. The use of anthropometric markers of myocardial remodeling is an affordable way of early stratification of cardiovascular risks in children with overweight and obesity.

Literature

1. Plotnikova I.V. Regularities and risk factors for the formation of essential arterial hypertension in adolescence: Author's abstract. dis. ... Dr. med. sciences. Tomsk 2009; 45.
2. Shlyakhto E.V., Konradi A.O. Causes and consequences of activation of the sympathetic nervous system in arterial hypertension. *Arterial hypertension* 2003; 3- 81-88.
3. Strand A.H., Gudmundsdottir H., Os I. et al. Arterial plasma noradrenaline predicts left ventricular mass independently of blood pressure and body build in men who develop hypertension over 20 years. *J Hypertens* 2006; 24— 5— 905— 913.
4. Horio T., Miyazato J., Kamide K. et al. Influence of flow highdensity lipoprotein cholesterol on left ventricular hypertrophy and diastolic function in essential hypertension. *Am J Hypertens* 2003; 16— 11— 938—944.
5. du Cailar G., Pasquile J.L., Ribstein J., Mimran A. Left ventricular adaptation to hypertension and plasma rennin. *J Hum Hypertens* 2000; 14— 3— 181—188.
6. Sundukova, E.L. Physiological and endocrinological aspects of adipose tissue, quantitative and topographic methods of its diagnosis in clinical practice / E.L. Sundukova, N.N. Minyailova, Yu.I. Rovda // *Mother and Child in Kuzbass*. -2009. - No. 3 (38). - S. 3-8.
7. Risk factors for the development of primary arterial hypertension in children and adolescents / E.G. Bunin, N.N. Minyailova, Yu.I. Rovda, E.L. Sundukova // *Doctor*. - 2010. -№1. - S. 40-43.
8. Leontyeva I.V., Moreno I.G., Neudakhin E.V. Metabolic disorders in the syndrome of arterial hypertension in adolescents / *Abstracts of the 9th Russian National Congress "Man and Medicine"* .- M., 2012.- P.261.
9. Nazhmitdinovna K. G. et al. Costs Syndrome in Children, Causes, Comparative Diagnosis and Rational

- Therapy (Review of the article) //The Peerian Journal. – 2022. – Т. 6. – С. 8-13.
10. Mukhin N.A., Balkarov I.M., Shonichev D.G., Lebedeva M.V. Normalization of arterial hypertension with urate tubulointestinal kidney damage // Ter. archive. 2013; 6: 23-27.
 11. Maratovna G. L., Ergashevna K. Z. Integrated clinical and metabolic evaluation of the condition of children with obesity and arterial hypertension //Достижения науки и образования. – 2020. – №. 8 (62). – С. 79-84.
 12. Starostina E. Biguanides: a second birth // New medical journal.-2012.-№1.-С3-11.
 13. Stroyev Yu.I., Churilov LP, Chernova LA, Bel'gov A.Yu. Obesity in adolescents. - S-Pb: "ELBI-S-Pb". - 2013. - 216 p.
 14. Холмурадова З. Э., Гарифулина Л. М. Semizligi bor osmirlarda yurak-qon tomir tizimining holati //журнал гепато-гастроэнтерологических исследований. – 2022. – Т. 3. – №. 3.
 15. Almazov V.A., Blagosklonnaya Ya.B., Shlyakhto E.V., Krasilnikova E.M. // Metabolic cardiovascular syndrome - SPb.: Publishing house of SPbGMU, 2011. - 208 p
 16. Garifulina L. M. et al. Psychological status and eating behavior in obese children //Science and education issues. – 2020. – Т. 110. – №. 26. – С. 45-50.