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FUNCTIONAL STATE OF KIDNEYS IN ADOLESCENTS WITH OBESITY

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ANNOTATION

Overweight and obesity are the most actual problems nowadays. Number of overweight patients steadily raises and duplicates every three decades. Obesity is associated with some factors of cardiovascular risk like diabetes mellitus and arterial hypertension, frequently leads to kidney disfunction. Obesity itself can result in poor renal hemodynamics, well-known risk factor of kidney dis-ease. We studied impact of overweight and obesity in children and adolescents on renal tubular function and glomerular filtration rate.

Keywords: obesity, children, kidney function.

In the last decade, obesity among children has been growing rapidly all over the world. Therefore, in the assessment of anthropometric indicators of children in developed countries, it was found that high body mass and obesity were observed in 28% of schoolchildren and 12% of preschool children [1]. In recent years, a lot of information has been revealed that proves that obesity is a direct factor leading to chronic kidney disease. Body mass index (BMI) has been suggested as a direct predictor of chronic kidney disease (CKD) progression [2]. Obesity is inextricably linked with hypertension and diabetes, which are common causes of end-stage renal failure. In addition, metabolic syndrome, which is the main consequence of obesity, is an important factor in end-stage renal failure [3]. Recent data show that insulin sensitivity and hyperinsulinemia are important factors in kidney damage [4].

The purpose of the examination is to determine the effect of obesity on kidney function in children.

Patients to be examined and methods of examination. We studied 35 patients aged 10-17 years (30 boys and 5 girls) with varying degrees of obesity who were treated for periodic hypertension in the nephrology department of Children's City Hospital No. 1 in Samarkand. child) we checked. Body mass index (VBI = M/R2, V- body weight in kg, height of patient B in cm) was determined from clinical examinations - anthropometry (body weight, height, waist circumference) data for all patients. Waist circumference (BA) was measured after full exhalation in a standing position along the axillary line at the point between the iliac crest and the rib cage. The evaluation complex of paraclinical tests included: determination of serum glucose, insulin, creatinine, uric acid, lipid spectrum indicators, and microalbuminuria. Children were divided into 3 groups according to body mass index: group 1 (high body mass) 8

patients (7/1 boys/girls) with VBI 25–30 kg/m2 (1SD) ; 2nd group (obesity Id) – 18 boys and 2 girls with BMI 30–35 kg/m2 (2SD); 3 groups (obesity II and III d (3SD)) – BMI 35–40 kg/m2 – 3 boys and 2 girls, body mass index i in 2 boys was above 40 kg/m2, which we included in group 3 for statistical analysis. All children were subjected to daily monitoring of blood pressure using an oscillometer, based on auscultatory detection of AQB. According to the form of arterial hypertension, the patients were divided into 4 groups: 1st group consisted of 9 children who did not have arterial hypertension according to the daily monitoring of arterial hypertension; A normal increase in arterial blood pressure was observed in 8 patients (group 2); AG I level – in 15 children (group 3); AG II level was observed in 3 children (group 4). A positive diagnostic value of albumin excretion was taken as 50 mg/l in morning urine. We conducted this inspection 2 times with an interval of 7 days. We considered a positive result only when microalbuminuria was detected in the morning urine twice. Later, we determined albumin in the morning urine by the immunoturbodimetric method on the Integra Analyzer (Roche, Basel, CH). Based on the obtained results (albumin concentration in 1 ml of urine), albumin excretion was calculated (mg/l). The test was considered positive when the amount of albumin in the morning urine was higher than 30 mg/l. Glucose and insulin levels were determined in all children using a standard glucose tolerance test (GTT) [5]. The amount of insulin in blood serum was determined by immunoenzymatic method. Insulin values were evaluated according to the recommendations of the American Society of Cardiology, according to which the insulin level was considered normal when it was less than 15 μMED/ml, borderline – 15–20 μMED/ml, and high – more than 20 μMED/ml [6]. We used the updated HOMA 2 homeostasis computer submodel to determine insulin secretion, insulin sensitivity (S%) and insulin resistance by pancreatic cells (B%). This method is considered to be the best informative method worldwide, unlike other previously conducted methods, it allows detection of latent IR based on fasting glucose and insulin levels in the morning [7, 8]. In addition, the presence of insulin resistance was also determined based on the HOMAR index: Index IR (IIR) HOMAR = G0 × INS0 / 22.5, G0 – morning plasma glucose concentration, mmol/l; INS0 – morning serum insulin concentration, μMED/ml. Determination of the lipid spectrum consisted of determination of total cholesterol (UMX), high (YUZLP) and low (PZLP) density lipids, triglycerides. Based on the obtained results, the formula atherogenic index (AI) was calculated: IA = UMX- YUZLP / YUZLP. AI was considered pathological when it was higher than 3. MS criteria presented in IDF 2007 (International Diabetes Federation) were used [9]. Along with these, abdominal obesity in children aged 10-16 (waist circumference above 90th percentile) occurs with two or more of the following signs: TG > 1.7 mmol/l; YUZLP < 1.03 mmol/l; arterial blood pressure exceeds 95 percentile; hyperglycemia > 6.1 mmol/L or postprandial levels higher than 7.8 mmol/L, or other carbohydrate metabolism disorders, VBI > 85th percentile also

indicate the presence of metabolic syndrome. In our investigation, we relied primarily on the association of abdominal obesity with hypertriglyceridemia and insulin resistance-type disturbances of carbohydrate metabolism. We determined the glomerular filtration rate by the SHvars formula: KFT = height × K × 88.4/Cr serum (μmol/l), K = 0.55 in boys and girls under 14 years of age, and 0. 7 in boys older than 14 years. We used Statistica 7.0 (StatSoft, USA) statistical software package to evaluate the test results. Results are presented as arithmetic mean ± standard error. Statistical significance of both means is Student's t-test; frequency - χ2 - was determined using Pearson's criterion. The strength of relationships between quantitative traits was assessed using the Pearson correlation coefficient. The null statistical hypothesis of no differences and relationships was rejected at p0.05. In addition, it was found that in children with obesity of the first degree, AG of the II degree, in obesity of the II and III degrees, only 2 patients of the AG of the I degree were detected (Table 1). 22 (62.9%) patients were found to have insulin resistance, the degree of its manifestation depends on the degree of obesity and the amount of YUZLP (table; fig. 1, 2). However, lipid metabolism was only slightly related to the level of obesity (r=0.2; p>0.05). As can be seen from the table, obvious changes in the form of an increase in the atherogenic index were observed in children with obesity of the first degree. To assess the functional state of the kidneys, we determined the glomerular filtration rate and microalbuminuria in a single urine sample. When calculating the glomerular filtration rate, 10 (28.6%) patients were found to have an abnormal value between 80 and 87 mL/min/1.73 m2, indicating "initial decrease in glomerular filtration rate." will give. These data were found equally in all three groups: 3 (37.3%) adolescents with high body mass; 5 (25%) in children with obesity of degree I and III and 2 (25%) in children with obesity of degree II. KFT was negatively correlated with the index of atherogenicity, that is, it was observed that KFT was significantly lower in adolescents with clearly disturbed lipid metabolism dominated by PZLP (r= -0.37;). It should be noted that microalbuminuria was not high in all groups. The maximum value of MAU was 187 mg/l. High indicators were observed in all three groups, but mainly in group 2 with obesity of the first degree. This principle was not important. However, we found a direct correlation between MAU indicators and atherogenicity index, i.e., higher MAU content was observed in adolescents with stronger lipid metabolism (r=0.42;) , while determining the amount of KFT and MAU, we found out that patients with low late diastolic arterial pressure in the 10% had a higher amount of MAU (r= –0.37; p<0.05).

Analysis of Results

Obesity among children is becoming a global epidemic. It has been proven that obesity and accompanying insulin resistance are factors that cause the development of cardiovascular diseases [10].

In our research, 62.9% of patients had a violation of carbohydrate metabolism leading to the development of insulin resistance. The reason for such a high percentage of carbohydrate metabolism disorders was the fact that the examined patients were teenagers, at this age physiological insulin resistance is typical [5]. As a result, we did not find a correlation between insulin resistance and microalbuminuria and CFT. The formation of nephropathy in obesity and its aggravation is primarily associated with the harmful effect of adipokines - mediators, which are actively produced and released by white adipocytes, on the structure of kidney tissue, mainly in abdominal obesity. Leptin is the cause of target organ damage in obesity. Leptin is a peptide hormone that plays an important role in energy homeostasis by signaling to the brain about adipose tissue reserves. Leptin is a satiety hormone. It stimulates the synthesis of several factors that reduce appetite [11,16]. In obese patients, resistance to leptin develops and leads to its excessive production. An increase in leptin, in turn, begins to have a damaging effect on the myocardium, vascular wall, and kidney tissue. Leptin induces renal fibrogenesis primarily by activating the expression of transforming growth factor-β (TFR-β) and its receptors on the membrane of mesangiocytes and endotheliocytes. The resulting TFR-β expression is one of the endothelial dysfunctions induced by leptin, which is diffuse in obese patients and is important in the pathogenesis of kidney damage [15]. An important factor in the development of endotheliocyte dysfunction in hyperleptinemia is the simultaneous release of endothelin-1, angiotensin I by these cells, and depression of endothelium-dependent vasodilation cascades. A marker of endothelial dysfunction is microalbuminuria, which is an early sign of the regressive stage of obesity-related nephropathy. The consequence of renal glomerular endotheliocyte dysfunction is a violation of intrarenal hemodynamics, which is manifested by a decrease in the functional reserve of the kidneys [12, 13,14]. Our investigation analyzed this pathophysiological process developed in obese children. Correlation of the atherogenic index with microalbuminuria and glomerular filtration rate confirms the correlation between lipid metabolism and kidney dysfunction. A decrease in the functional reserve of the kidneys is manifested by a violation of the circadian rhythm of arterial blood pressure in a person, in which a decrease in evening arterial blood pressure is observed. The degree of manifestation of evening arterial blood pressure decrease in microalbuminuria indirectly shows the activation of the renin-angiotensin-aldosterone system in the evening in our patients. It should be noted that lipid metabolism was not associated with the level of obesity, which must be taken into account in health care practice.

CONCLUSION

Thus, in adolescents with high body mass and obesity, the functional state of the kidneys is correlated with the degree of manifestation of lipid metabolism disorders, and low-density lipoproteins predominate in it. Dyslipidemia does not depend on the

degree of obesity and is a direct predictor of the formation of nephropathy in adolescents with high body mass.

Foydalanilgan adabiyotlar

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