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ЖУРНАЛ ГЕПАТО-ГАСТРОЭНТЕРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

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MECHANISM OF INFLUENCE OF OVERWEIGHT AND OBESITY ON KIDNEYS IN CHILDREN

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ANNOTATION

Obesity and metabolic syndrome influence the functions of child's body's systems, provoke a progressive kidney damage and are development factors of chronic nephron number in reference to body mass. A long-term impact of these factors leads to glomerulosclerosis and chronic renal failure development. The detection of kidney disease. The kidney damage in case of obesity is related to the adipokine production failure, activation of the renin-angiotensin system, chronic inflammation, dyslipidemia, violation of kidney hemodynamics, reduction of early signs and biomarkers of the kidney damage at children is a necessary condition for prevention of the renal failure development.We studied impact of overweight and obesity in children and adolescents on renal tubular function and glomerular filtration rate.

Key words: obesity, children, kidney function.

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BOLALARDA ORTIQCHA VAZN VA SEMIZLIKNING BUYRAKLARGA TA'SIR MEXANIZMI

ANNOTATSIYA

Semizlik va metabolik sindrom bola organizmi tizimlarining funktsional holatiga ta'sir qilib, buyraklarning progressiv shikastlanishiga olib keladi va surunkali buyrak kasalligi rivojlanishida xavf omili hisoblanadi. Semizlikda buyrak shikastlanishining shakllanishi adipokin ishlab chiqarishning buzilishi, renin-angiotenzin tizimining faollashishi, surunkali yallig'lanish, dislipidemiya, buyrak gemodinamikasi buzilishi va tana vazniga nisbatan nefronlar sonining kamayishi bilan bog'liq. Ushbu omillarning uzoq muddatli ta'sir qilishi glomeruloskleroz va surunkali buyrak etishmovchiligining rivojlanishiga olib keladi. Bolalarda buyrak shikastlanishining dastlabki belgilari va biomarkerlarini aniqlash, buyrak shikastlanishining rivojlanishi va rivojlanishining oldini olish uchun zarur omil hioblanadi.Ushbu tadqiqotimizda biz bolalarda semizlik va ortiqcha tana vaznning buyrakning naychali apparati va glomerulyar filtratsiya tezligiga ta'sirini o'rganib chiqdik.

Kalit so'zlar: semizlik, bolalar, buyrak holati.

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МЕХАНИЗМЫ ВЛИЯНИЯ ИЗБЫТОЧНОЙ МАССЫ ТЕЛА И ОЖИРЕНИЯ НА ПОЧЕК У ДЕТЕЙ

АННОТАЦИЯ

Ожирение и метаболический синдром влияют на функциональное состояние систем детского организма, приводят к прогрессивному повреждению почек и являются факторами развития хронической болезни почек. Формирование поражения почек при ожирении связано с нарушением продукции адипокинов, активацией ренин-ангиотензиновой системы, хроническим воспалением, дислипидемией, нарушением почечной гемодинамики, уменьшением количества нефронов относительно массы тела. Длительное воздействие данных факторов ведет к развитию гломерулосклероза и хронической почечной недостаточности. Выявление ранних признаков и биомаркеров поражения почек у детей является необходимым условием для профилактики развития и прогрессирования ренального поражения.В нашей работе мы изучали влияние избыточной массы тела и ожирения у детей на функцию канальцевого аппарата почек и скорость клубочковой фильтрации.

Ключевые слова: ожирение, дети, состояние почек.

Actuality. In modern society, excess body weight and obesity are among the most pressing issues. The number of patients with excess weight is steadily increasing and, according to some data, doubles every three decades. Obesity is pathogenetically linked to several cardiovascular risk factors, such as diabetes mellitus and arterial hypertension, which often lead to kidney damage; obesity itself is

associated with unfavorable renal hemodynamics, which, regardless of these factors, can contribute to kidney pathology. In our work, we studied the influence of excess body weight and obesity on the function of the renal tubular apparatus and glomerular filtration rate in children and adolescents.

Relevance of the work Obesity is a major symptom of metabolic syndrome, but even in the absence of arterial hypertension, type 2 diabetes mellitus or a period of compensation for these conditions, specific changes in the renal tissue are observed. Obesity-related glomerulopathy (GPS), [obesity-related nephropathy], is a nosological entity recognized not only by therapeutics, but also by pediatric nephrology [16,3].

It has been established that the primary increase in glomerular filtration rate associated with obesity is an early compensatory response that helps restore salt balance despite the active continuation of reabsorption. Long-term glomerular hyperfiltration, especially in patients with concomitant arterial hypertension, causes damage to renal tissue. There are also studies showing that kidney tissue damage and glomerular hyperfiltration decrease with a decrease in body weight [7].

The identification of endothelial dysfunction markers is currently relevant for many diseases, including kidney diseases [12]. Endothelial dysfunction in patients with chronic kidney disease is considered as an imbalance between vasoconstrictors and relaxants, anti- and procoagulant mediators, growth factors and their inhibitors [16].

There is a logical connection between kidney damage and endothelial dysfunction, but it has not been fully studied. The pathological role of endothelial dysfunction has been shown in chronic pyelonephritis, chronic glomerulonephritis [1].

Currently, endothelial dysfunction is understood as a violation of the balance between the production of vasodilating, athrombophilic, antiproliferative factors, on the one hand, and the production of vasoconstrictor, prothrombotic and proliferative substances, on the other. Markers of endothelial dysfunction include decreased endothelial synthesis of nitric oxide (NO), increased levels of endothelin-1, increased levels of circulating von Willebrand factor, plasminogen, homocysteine, thrombomodulin, soluble vascular cell-cell adhesion molecule B1, S-reactive protein, microalbuminuria, and others [8].

Materials and methods. We examined 54 patients aged 8 to 18 years suffering from obesity and excess body weight, who were treated in a stationary setting at the Endocrinology Dispensary of Samarkand region. Among the examined children, 29 were boys and 25 were girls. The study used the obesity classification, according to which the Body Mass Index (BMI) was considered to be overweight if it exceeded 85-95 percentile, and obesity if it exceeded 95 percentile. A BMI of more than 35 indicates morbid obesity [1]. In the study, overweight was detected in 12 children, obesity in 32, and morbid obesity in 10 children.

As a control group, 18 healthy children aged 10 to 18 years with no kidney pathology and normal body weight were examined(tab.-1).

Table-1

Obesity rate	Number of children	
	boys	girls
Overweight	7	5
Obesity	18	14
Morbid obesity	7	3
Control group	10	8

A general clinical examination included a complete blood count and urinalysis. The function of the glomerular apparatus was assessed by glomerular filtration rate. The state of the proximal tubules was assessed by daily excretion and clearance of calcium and phosphorus. Albuminuria was determined by visual test strips in the morning urine for microalbuminuria (MAU) using the semi-quantitative method(tab.-2).

Table-2

Daily urinary albumin excretion and tubular filtration rate in obese and overweight children and adolescents according to the degree of obesity				
Indicator under investigation	Control group (n=18)	Overweight group (n=12)	Obesity group (n=32)	Morbid obesity group (n=10)
MAU, mg/l	0,0 (0,0; 10,0)	20,0 (0,0; 50,0)	20,0 (15,0; 20,0)	20,0 (0,0; 100,0)
KFT, ml/min	93,4 (81,8; 102,3)	104,4 (75,0; 149,0)	111,05 (93,8; 137,0)	122,3 (96,5; 136,0)

A general clinical examination included a complete blood count and urinalysis. Calcium excretion and clearance in obese and overweight children and adolescents according to the degree of obesity(tab.-3)

Table-3

Calcium excretion and clearance in obese and overweight children and adolescents according to the degree of obesity				
Investigated indicator	Control group (n=18)	Overweight group (n=12)	Obesity group (n=32)	Morbid obesity group (n=10)
Blood calcium level, mmol/l	2,48 (2,43; 2,5)	2,48 (2,4; 2,5)	2,44 (2,35; 2,5)	2,38 (2,25; 2,45)
Daily urinary calcium excretion mmol/l	0,43 (0,25; 0,77)	2,01 (1,4; 2,1)	1,02 (0,6; 1,58)	1,12 (0,55; 1,89)
Calcium clearance, ml/min	0,07 (0,05; 0,18)	0,45 (0,3; 0,9)	0,24 (0,13; 0,39)	0,3 (0,12; 0,52)

The obtained data were statistically analyzed using the Statistics 8.0 software. Considering that the results of the obtained medical-biological indicators, especially those from a small sample, are not suitable for statistical analysis, non-parametric methods of variation statistics (median and percentages) and the Mann-Whitney test were used to compare independent samples. The statistical significance of the differences was assessed when the probability of the null hypothesis was less than 0.05 ($p < 0.05$). The data in this text and tables are presented as Me (25; 75) (where Me is the median, 25th and -75th, the interval between the 25th and 75th percentiles).

Research results: Glomerular filtration rate (GFR) analysis revealed a gradual increase in GFR with increasing body weight. Statistically significant differences were found in the groups with morbid obesity and obesity compared with the control group (in the group with obesity 111.05 (93.8; 137.0) ml/min compared with the control group 93.4 (81.8; 102.3) ml/min,) and in the group with morbid obesity (122.3 (96.5; 136.0) ml/min; in the control group – 93.4 (81.8; 102.3).

When comparing albuminuria indicators with the control group, it was found that the indicator increased statistically significantly in the groups with obesity and morbid obesity. The frequency of detection of MAU in the control group was 0 cases, in the group of children with excess body weight - 6 (30%), in the group with obesity - 18 (22.2%), in the group with morbid obesity - 7 (20%).

As obesity increased, it was found that the amount of calcium in the blood and daily urine decreased in obese children. The difference in blood calcium levels in children with normal body weight and with a slight degree of obesity was statistically significant compared to children with morbid obesity. At the same time, as the degree of obesity increased, the amount of calcium excreted in the urine gradually increased, and the difference was also statistically significant in the overweight group compared to the control group. It is also important to note that calcium clearance increased statistically in all groups studied.

When analyzing the excretion and clearance of inorganic phosphorus, it was possible to identify similar trends: a decrease in phosphorus in the blood (the difference was statistically significant when comparing the control group with the morbid obesity group), an increase in phosphorus excretion in the urine (a statistically significant difference was found between the control group and the obese group, p, when comparing the morbid obesity group). Also, phosphorus clearance gradually increases from the control group to the morbid obesity group.

Analysis of results

In recent years, epidemiological studies have clearly shown that obesity is an independent risk factor for chronic kidney disease [10]. Studies conducted in adult patients with metabolic syndrome have shown that MAU is present in obese and morbidly obese groups. It is worth noting that according to the results of a study by Chen B. et al., the frequency of MAU in patients with metabolic syndrome reached 20.3% [6]. The presence of obesity is reflected in the prevalence and severity of MAU, and also indicates the rapid development of kidney disease in the analysis of the elderly population [12]. In a study of 572 obese patients, Atshinnia F. et al. found that weight loss was associated with a 1.7 g (confidence interval 0.7–2.6 g) and 14 g (11–17) reduction in proteinuria and MAU, respectively ($p<0.05$) [4].

Chronic kidney disease is characterized by complex metabolic changes; these include vitamin D deficiency, metabolic acidosis, inflammation, and accumulation of "uremic toxins"[15]. A study of 171 patients with chronic kidney disease found that the mean serum 25-(OH) D was 22.1 +/- 13 ng/ml, with only 18.7% of patients having normal levels of 25- (OH), 58.5% having decreased vitamin D levels, and 22.9% having significantly decreased levels, with 47.3% of patients suffering from obesity [9]. A study by Hultin H. et al. found that the mean serum 25- (OH) D3 level in 108 morbidly obese patients was 53 nmol/l[11]. The increased calcium clearance observed in our study may be related to vitamin D deficiency, which is common in chronic kidney disease.

In the blood serum, 40% of calcium is bound to protein, 10% to bicarbonate and phosphate, and 50% to the free fraction. Calcium reabsorption in the kidneys occurs mainly in the proximal tubules and the loop of Henle by passive diffusion along an electrochemical gradient, partly together with sodium and water [2]. Accordingly, increased calcium excretion and clearance indicate impaired reabsorption in the proximal tubules of the kidneys [2]. Calcium reabsorbed in the distal tubules of the kidneys is transported by vitamin D-dependent Ca2 + binding proteins [2]. Vitamin D deficiency also increases daily urinary calcium excretion and clearance.

Inorganic phosphorus is mainly reabsorbed in the proximal tubules (80%), 10% in the distal tubules, and 10% is excreted in the urine [2]. Accordingly, chronic hypo- and hyperphosphatemia may be a consequence of impaired renal phosphate regulation [8]. There are also publications in the literature showing a relationship between obesity and hyperparathyroidism [7]. When 1628 patients were examined, the median parathyroid hormone level was the lowest in the group of patients with the lowest body weight (10.2 mmol/l), then in the group of patients with normal body mass (12.1 mmol/l), in the group of patients with excess body mass (14.0 mmol/l), and in the group of patients with obesity (17.5 mmol/l) [7]. Parathyroid hormone reduces phosphate reabsorption in the proximal and distal tubules of the kidney, leading to hypophosphatemia and phosphaturia. Increases calcium reabsorption in the distal tubules. [2]. In our study, the increase in calcium clearance was probably due to impaired calcium reabsorption in the distal tubules in vitamin D deficiency[3].

Obesity is often associated with changes in the blood lipid spectrum, impaired glucose metabolism in adults, and hypertension [3].

Dyslipidemia is a well-established risk factor for atherosclerosis and is also found in adults and children with chronic kidney disease. One study (391 children aged 1 to 16 years) found an association between dyslipidemia and proteinuria [14]. Decreased lipid catabolism or increased lipid excretion may contribute to the development of atherosclerosis, as well as glomerulosclerosis and tubulointerstitial kidney damage [16]. According to literature data, obesity increases glomerular filtration rate and proteinuria [13], which is consistent with our findings. The lack of a decrease in calcium and phosphorus clearance with increasing GFR and the lack of correlation between these parameters may indirectly indicate a mechanism of impaired renal tubular function independent of filtration in overweight and obesity.

Conclusion:

1. As obesity increases, glomerular filtration rate and microalbuminuria can be observed in children and adolescents.
2. Impaired renal tubular function is observed in overweight children.

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