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Effects on childhood obesity and kidney failure

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Conclusion. *obesity have reached epidemic proportions, affecting almost 60% of adults, with 7.9% of children under 5 years of age suffering from this pathology. At the present stage, the problem of obesity in children and adolescents has become one of the significant problems of medicine. One in three school-age children and one in four children aged 10 to 19 are overweight or obese [1]. Today it is known that obesity is also an independent risk factor for the development of chronic kidney disease (CKD) [2,3], contributing to kidney damage through direct (hemodynamic and hormonal effects of adipose tissue) and indirect (hypertension and type 2 diabetes) mechanisms [4]. Despite this, there is very little literature data on the structural and functional state of the*
Keywords: *obesity, children, kidney function.*

Introduction. **Materials and research methods:** We examined 93 patients aged 8 to 18 suffering from obesity and excess body

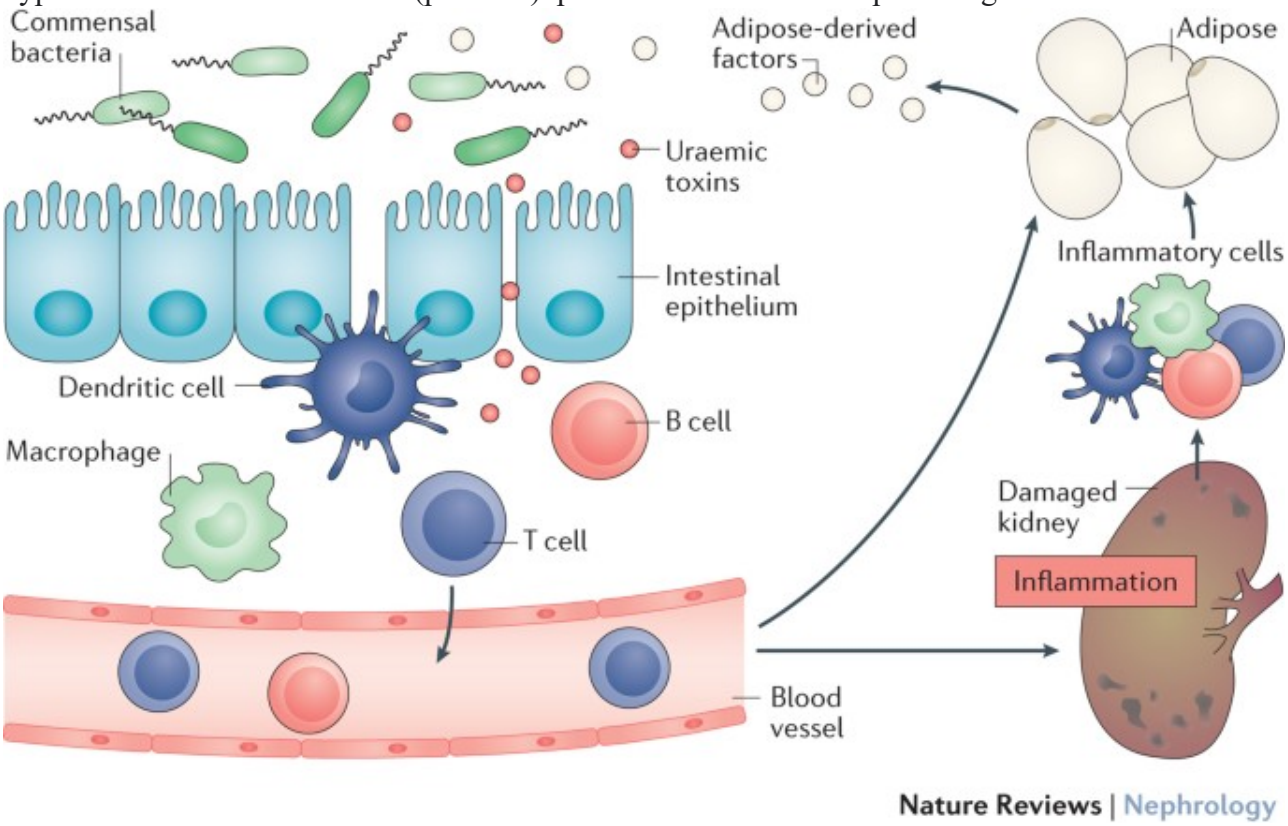
weight who were treated inpatient at the endocrinology dispensary of the Samarkand region. Among the examined children, 48

are boys and 45 are girls. The classification of obesity was used in the conducted study, according to which the Body Weight Index (BMI) exceeded 85-95 percent as excess body mass, and if it exceeded 95 percent, it was considered obesity. If the TVI exceeds 35, it indicates morbid obesity [1]. In the conducted research, 10 children were overweight, 63 were obese, and 20 were morbidly obese.
As a control group, 18 healthy children aged 10 to 18 years with no kidney pathology and normal body weight were examined.

In the general clinical examination, a general blood and urine analysis was used.

The function of the glomerular apparatus was evaluated by the glomerular filtration rate. The condition of the proximal part of the tubules was evaluated based on the daily excretion and clearance of calcium and phosphorus. Albuminuria was determined by visual test-lines of semi-quantitative microalbuminuria (MAU) in morning urine. Calcium and phosphorus clearance was calculated using the formula recommended by Shyuk O. (1981):
C = U*V/P *1.73/S(m2), where
C is the clearance of the tested product (ml/min),
U is the composition of the substance to be tested in urine (µg/l, mg/l),
V – minute diuresis (ml/min),
R is the composition of the tested substance in the serum
(µg/l, mg/l),
C - skin level.
The obtained data were statistically processed using Statistics 8.0 software. Taking into account that the results of the obtained medical-biological indicators, especially the indicators of a small sample, are disproportionate for statistical processing, non-parametric methods of variational statistics (median and percentages) and the Mann-Whitney test were used to compare independent samples.

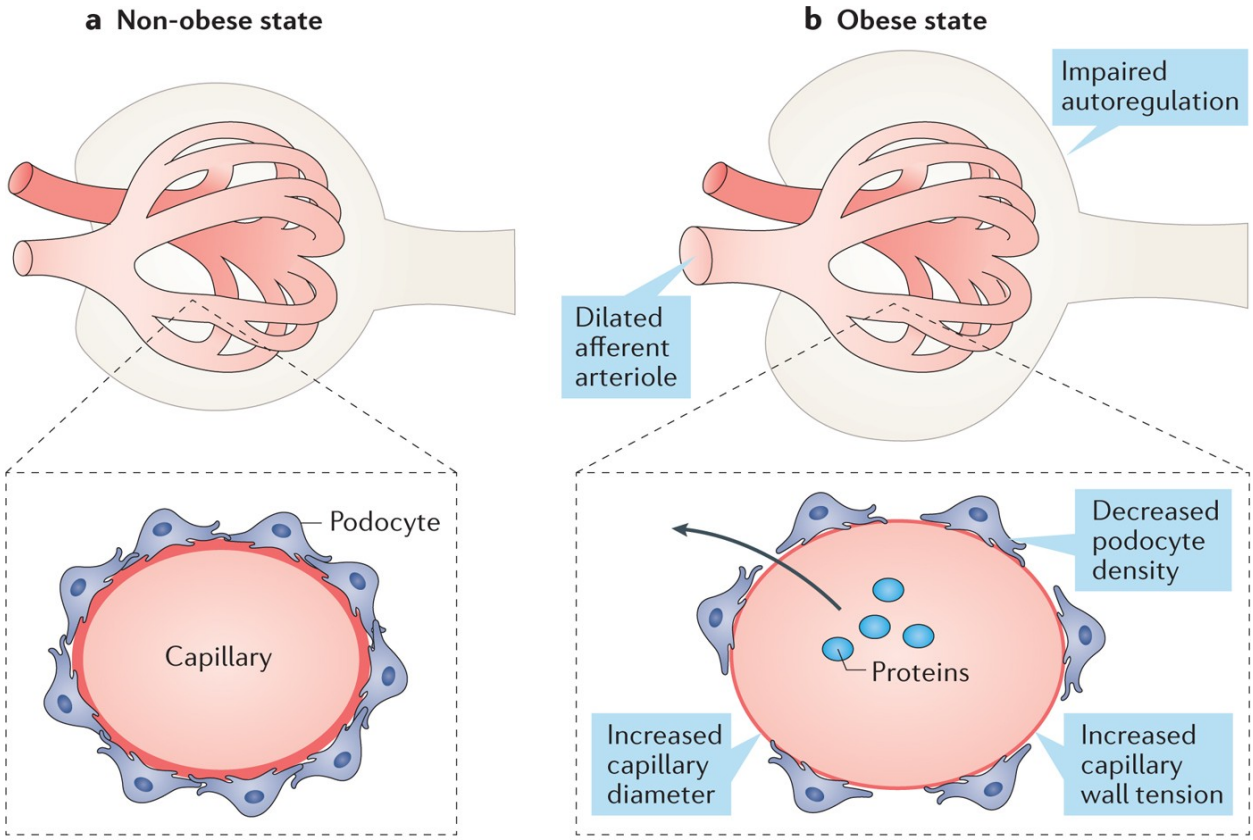
The statistical significance of the differences was assessed when the probability of validity of the null hypothesis was lower than 0.05 ($p < 0.05$). presented in the form of percentage interval



Research results

Glomerular filtration rate (GFR) analysis revealed a gradual increase in GFR with increasing obesity. Statistically significant differences were found when comparing the morbidly obese and obese groups with the control group (obese group 111.05 (93.8; 137.0) ml/min compared with the control group 93.4 (81.8; 102.3) ml /min, p2=0.01) 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), p3=0 ,04) was found.

When comparing albuminuria indicators with the control group, it was found that the indicator increased statistically 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 - 3 (30%), with obesity. in the group - 14 (22.2%), in the group with morbid obesity it was 4 (20%) (Table 2).



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As the degree of obesity increases, the amount of calcium in the blood and daily urine decreases in obese children. In children with normal body weight and a little obesity, the difference in the amount of calcium in the blood is statistically significant compared to children with morbid obesity. At the same time, the amount of calcium excreted in the urine gradually increased as the level of obesity increased, and the difference was statistically significant even when children in the overweight group were compared with the control group. It is also important that calcium clearance increased statistically in all investigated groups.

Analyzing the excretion and clearance of inorganic phosphorus, it was possible to identify similar trends: a decrease in phosphorus in the blood (the difference is statistically significant when comparing the control group with the group with morbid obesity, $p = 0.047$), an increase in the excretion of phosphorus in the urine (the control group and a statistically significant difference was found between the obese group $p = 0.04$, compared with the morbidly obese group, $p = 0.002$). Also, phosphorus clearance gradually increases from the control group to the morbidly obese group.

Discussion of results

In recent years, epidemiological studies have clearly shown that obesity is an independent risk factor for chronic kidney disease [10]. In studies of adult patients with metabolic syndrome, the presence of MAU was noted in groups with obesity and morbid obesity. It should be noted that according to the results of the study conducted by Chen B. and co-authors, the frequency of MAU in patients with metabolic syndrome reached 20.3% [6]. The presence of obesity is expressed by the widespread and obvious manifestation of MAU, and also indicates the rapid development of kidney diseases in the analysis of an older population [12]. In 572 obese patients examined by Atshinnia F. and co-authors, the decrease in body mass was associated with proteinuria and MAU of 1.7 g (confidence interval 0.7–2.6 g) and 14 mg (11–17), respectively ($r< 0.05$) was found to decrease [4].

Complex metabolic changes are detected in chronic kidney diseases; these include vitamin D deficiency, metabolic acidosis, inflammatory processes, and accumulation of "uremic toxins"[15].

In a study of 171 patients with chronic kidney disease, it was found that the average amount of 25-(OH) D was 22.1 +/- 13 ng / ml, and only 18.7% of patients had 25- (OH) D of the normal amount,

58.5% of vitamin D content decreased and 22.9% of the studied showed a significant decrease in its amount, in which 47.3% of patients suffered from obesity [9].

According to research conducted by Hultin H. and co-authors, the average amount of 25- (OH) D3 in blood serum was 53 nmol/l in 108 patients with morbid obesity [11]. The increase in calcium clearance found in our study may be related to vitamin D deficiency in chronic kidney disease. 40% of calcium in blood serum is bound to protein, 10% to bicarbonate and phosphate, and 50% of calcium is in the form of free fraction. Reabsorption of calcium in the kidneys occurs mainly in the proximal tubules and the knee of Henle's loop by means of passive diffusion along the electrochemical gradient, partially with sodium and water [2]. Accordingly, an increase in calcium excretion and clearance indicates impaired reabsorption in the renal proximal tubules [2]. Calcium reabsorbed in the distal tubules of the kidney is transported in a vitamin D-dependent manner using Ca2+-binding protein [2].

Also, vitamin D deficiency 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 the failure of the kidney mechanism to regulate the level of phosphate [8]. In the literature, there are publications showing the 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 pmol/l), then in the group of patients with normal body mass (12.1 pmol/l), in the group of patients with excess body mass (14 .0 pmol/l) and in obese patients (17.5 pmol/l) [7]. Parathyroid hormone reduces reabsorption of phosphates in the proximal and distal tubules of the kidney, leading to hypophosphatemia and phosphaturia. Increases reabsorption of calcium in distal tubules. [2]. In our study, the increase in calcium clearance was probably related to impaired calcium reabsorption in the distal tubules with vitamin D deficiency[3]. Obesity is often manifested by changes in blood lipid spectrum, glucose metabolism disorders and hypertension in adults [3].

Dyslipidemia is a clear risk factor for atherosclerosis, and it also occurs in adults and children with chronic kidney disease. In one of the conducted studies (391 children aged 1 to 16 years), it was found that there is a relationship between dyslipidemia and proteinuria [14]. A decrease in lipid catabolism or an increase in excretion can cause the development of atherosclerosis, as well as glomerulosclerosis and tubulointerstitial kidney damage [16].

According to the information in the literature, the glomerular filtration rate increases and proteinuria develops in obesity [13], which is consistent with the data obtained from our study. The fact that calcium and phosphorus clearance does not decrease with an increase in GFR and the lack of correlation between these parameters may indirectly indicate the mechanism of deterioration of renal tubular functions independent of filtration in overweight and obesity.

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