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Адрес редакции: 140100, Узбекистан, г. Самарканд, ул. А. Темура 18.
Тел.: +998662333034, +998915497971
E-mail: **hepato_gastroenterology@mail.ru.**

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ЖУРНАЛ ГЕПАТО-ГАСТРОЭНТЕРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

Goyibova Nargiza Salimovna
Assistant at the Department of Pediatrics, Faculty of Medicine,
Samarkand State Medical University. Samarkand. Uzbekistan

FEATURES OF KIDNEY DAMAGE IN CHILDREN WITH OBESITY
(Literature review)

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ANNOTATION

The review provides information on kidney morphology and function in children with obesity. The influence and interrelationship of numerous pathogenetic factors affecting kidney function are discussed. Using early biomarkers of kidney pathology in obesity with an assessment of lipid, carbohydrate metabolism, insulin resistance, serum leptin, and adiponektin levels is promising for diagnosing kidney damage in children with obesity.

Key words: obesity, kidneys, children

Гойибова Наргиза Салимовна
Ассистент кафедры педиатрии лечебного факультета
Самаркандского государственного медицинского университета.
Самарканд. Узбекистан

ОСОБЕННОСТИ ПОВРЕЖДЕНИЯ ПОЧЕК У ДЕТЕЙ ПРИ ОЖИРЕНИИ
(Обзор литературы)

АННОТАЦИЯ

В обзоре приводятся сведения о морфологии и функции почек у детей при ожирении. Обсуждается влияние и взаимосвязь многочисленных патогенетических факторов, действующих на работу почки. Использование ранних биомаркеров патологии почек при ожирении с оценкой уровня показателей липидного, углеводного обмена, инсулинорезистентности, сывороточного лептина и адипонектина является перспективным для диагностики ренального поражения при ожирении у детей.

Ключевые слова: ожирение, почки, дети

G’oyibova Nargiza Salimovna
Samarqand davlat tibbiyot universiteti, Davolash fakulteti
pediatriya kafedrasi assistenti. Samarqand. O’zbekiston.

SEMIZLIKDA BOLALAR BUYRAGINING ZARARLANISH XUSUSIYATLARI
(Adabiyotlar sharhi)

ANNOTATSIYA

Sharhda bolalarda semizlikda buyraklarning morfologiyasi va funksiyasi haqida ma’lumotlar keltirilgan. Buyrak faoliyatiga ta’sir qiluvchi ko‘plab patogenetik omillarning ta’siri va o‘zaro bog‘liqligi muhokama qilinadi. Bolalarda semizlikda buyrak zararlanishini tashxislashda lipid, uglevod almashinuvi, insulinrezistentlik, zardob leptini va adiponektin ko‘rsatkichlari darajasini baholash bilan semizlikda buyrak patologiyasining erta biomarkerlaridan foydalanish istiqbolli hisoblanadi.

Kalit so‘zlar: semizlik, buyraklar, bolalar

In the last decade, the prevalence of obesity has acquired the character of a pandemic, which is a medical and social problem for healthcare worldwide [5,14]. According to the World Health Organization's global assessment in 2016, more than 340 million children and adolescents aged 5-19 and 41 million children under 5 had excess weight or obesity (WHO, 2016) [60,61].

The relevance of this problem also lies in the fact that the high prevalence of obesity contributes to the development of chronic diseases, including chronic kidney diseases [6,10,16,41].

Obesity is one of the main symptoms of metabolic syndrome, however, characteristic changes in kidney tissue during obesity are also detected in the absence of concomitant arterial hypertension and type 2 diabetes mellitus or when these conditions are compensated. Glomerulopathy associated with obesity (GPO) [obesity-related nephropathy] is a nosological unit recognized not only in therapeutic but also in pediatric nephrology [16,33].

Currently, the concept of end-stage renal disease (ESRD) or "terminal stage of renal failure" (TPN) is widely used in the literature, under which the development of renal failure is understood as the result of the influence of a whole range of various factors. The morphological manifestation of TPN is glomerulosclerosis and tubulointerstitial fibrosis.

The most common causes of TPN are considered to be systemic arterial hypertension with the development of a primarily wrinkled kidney and type 2 diabetes mellitus (T2DM), as shown in a number of experimental and clinical studies [4,20,21]. On the other hand, it has been determined that an increase in the risk of developing cardiovascular diseases, in particular arterial hypertension, correlates

with the level of pathological fat accumulation, and the development of DM2, as a rule, precedes obesity [31,44]. Moreover, it has been shown that the presence of obesity significantly reduces life expectancy, and in two out of three cases, death occurs from a disease associated with lipid metabolism disorder and excess weight[25].

Despite the fact that both of the most common causes of TPN are directly related to excess body weight, obesity has only recently been considered a possible cause of renal failure [54], and its connection with kidney pathology is only discussed in separate clinical studies. Thus, the significance of obesity as a predisposing factor in the development of focal-segmental glomerulosclerosis has been shown [38,56]. In nephropathy associated with immunoglobulin A deposition, excess body weight is considered an independent risk factor affecting overall and renal survival [24,42]. In patients after kidney transplantation, the role of obesity in the development of chronic transplant rejection and the deterioration of the overall prognosis is discussed [37,47,62]. In the literature, only a few works describe structural and functional changes in the kidneys during obesity. They are expressed in focal glomerulosclerosis and other changes resembling the morphological picture in DM2 [39].

The mechanisms of development of the pathological process in the kidneys under the influence of excess body weight have been little studied and are known only in isolated, mainly experimental, works on research in this area [29,35,43]. However, the data accumulated to date allow us to form an understanding of the contribution of obesity and its accompanying metabolic, hormonal, and hemodynamic disorders to the formation of pathological changes in the function and structure of the kidneys.

Framingham Heart Study and 18-year observation of patients showed a higher risk of developing the third stage of chronic kidney disease (CKD) in patients with obesity (body mass index (BMI) > 30 kg/m2) compared to overweight patients (BMI 25-30 kg/m2) [32]. It has been proven that in patients with nephropathies against a background of visceral obesity, the lipid spectrum of blood serum is characterized by an increase in lipid atherogenicity with a decrease in high-density lipoprotein cholesterol [18], an increase in serum triacylglycerols, total cholesterol, very low-density lipoprotein cholesterol in combination with hyperleptinemia and impaired glucose tolerance [18]. It has been established that an increase in serum leptin and a disruption in lipid metabolism indicators in children with nephropathy against a background of visceral obesity are associated with changes in echographic indicators of the kidney's structural state, intrarenal hemodynamics, and a partial decrease in kidney function [2,3].

It has been proven that kidney diseases progress in patients with hypertension with the formation of nephrosclerosis and the development of a primarily wrinkled kidney. In type 2 diabetes mellitus, tubulointerstitial fibrosis, glomerulosclerosis, and diabetic nephropathy develop [9].

Kidney disease associated with obesity and diabetes mellitus develops when several metabolic and hemodynamic factors interact, activating general intracellular signals, which, in turn, cause the production of cytokines and growth factors that form renal failure. The mechanisms underlying glomerular hyperfiltration against the background of obesity are widely discussed in the literature [7].

A recognized mechanism is the increase in sodium reabsorption near the tubules or Henle loop, leading to the development of tubuloglomerular feedback - an indirect decrease in afferent arterioles' resistance, an increase in intracapsular pressure, and glomerular filtration rate [48].

Among the main factors contributing to the progression of kidney damage in obesity, the following are distinguished: insulin resistance (IR), hyperinsulinemia, dyslipidemia, disruption of systemic and renal hemodynamics, renal tissue ischemia, and auto- and paracrine effects of fat tissue hormones [7, 13,17]. Currently, when assessing the pathophysiological mechanisms of kidney damage, special attention is paid to studying the role of metabolic syndrome (MS).

Many authors believe that the main pathogenetic link in kidney damage is the production of biologically active substances - adipocytokines by adipose tissue, which allows us to consider adipose tissue as an active endocrine organ. Among adipokines, leptin and adiponektin are given special attention [28].

It has been established that the initial increase in glomerular filtration rate associated with obesity is an early compensatory response that contributes to the restoration of salt balance, despite continued activation of reabsorption. Prolonged glomerular hyperfiltration is the cause of kidney tissue damage, especially in patients with hypertension. There are studies showing a decrease in glomerular hyperfiltration and kidney tissue damage during weight loss [27].

Determining markers of endothelial dysfunction is currently relevant for many diseases, including kidney diseases [12]. Endothelial dysfunction in patients with CKD is considered as a disbalance between vasoconstrictors and relaxing factors, anti- and procoagulant mediators, growth factors, and their inhibitors [46].

The connection between endothelial dysfunction (ED) and kidney damage appears to be regular, but not sufficiently studied. The pathological role of endothelial dysfunction has been described in chronic pyelonephritis, chronic glomerulonephritis [1].

Currently, endothelial dysfunction is understood as a disruption of the balance between the production of vasodilating, athrombogenic, antiproliferative factors on one side and the production of vasoconstrictor, prothrombotic, and proliferative substances produced by the endothelium - on the other [1]. Markers of endothelial dysfunction include decreased endothelial synthesis of nitrogen oxide (NO), increased levels of endothelin-1, circulating von Willebrand factor, plasminogen activator inhibitor, homocysteine, thrombomodulin, soluble vascular intercellular adhesion molecule B1, C-reactive protein, microalbuminuria, and others [8].

Microalbuminuria is a proven highly sensitive marker of prognostically unfavorable kidney damage, and also reflects the presence of endothelial dysfunction. The detection of non-selective proteinuria indicates gross damage to kidney structures and, moreover, becomes a direct damaging factor contributing to the progression of nephrosclerosis. The damaging effect of systemic arterial hypertension on the kidneys is realized through a disruption of renal hemodynamics under the influence of a cascade of changes in the renin-angiotensin-aldosterone system (RAAS). The appearance of the indicated clinical symptoms indicates a pronounced, often irreversible, damage to the kidney tissue. In this regard, the active study of early biological indicators of kidney damage continues, among which markers of endothelial dysfunction are actively being studied [1].

Morphological changes in the nephron in obesity are similar to those in oligomeganephrony. According to the "three blows" concept [19], a small number of nephrons at birth can be the "first blow," while the "third blow" is the development of obesity and insulin resistance. In the context of kidney pathology in children, a decreased mass of nephrons and the risk of developing a terminal stage of renal failure are associated with the birth of a child with a low body weight compared to gestational age, or with premature birth with a body weight corresponding to gestational age [58].

Many studies have presented confirmations of the hypothesis that birth weight is related to diseases in subsequent life (Barker's hypothesis). In particular, a correlation has been established between a decreased birth weight and an increased risk of coronary heart disease, type 2 diabetes mellitus, hypertension, hyperlipidemia, stroke, and heart attack [36]. In advanced cases, secondary focal-segmental glomerulosclerosis (FSGS) may develop [26]. This form is distinguished by the fact that it is not characterized by massive proteinuria corresponding to the nephrotic syndrome, and swelling is practically absent. In individuals with pronounced obesity, with preserved kidney function, biopsies reveal morphological changes including glomerulomegaly, hypertrophy and fusion of podocytes, expansion of the mesangial matrix, and proliferation of mesangial cells [53].

Glomerulomegaly is a primary histopathological trait that distinguishes GPO from primary FSGS [52]. The thickening of the glomerular basement membrane (GBM), previously considered an early manifestation of hyperglycemia and diabetic nephropathy, can also be an additional pathological finding in obesity. Thickening of the GBM is found during biopsy in patients with nephrosclerosis associated with

essential arterial hypertension and in patients with GPO with normal glycemia. The thickness of GBM is directly correlated with the level of cholesterol and triglycerides [40]. According to American pathomorphologists who studied 6818 kidney biopsies over 15 years, the frequency of GNOs increased 10 times: from 0.2% of all studied biopsies in 1986 to 2% in 2000 [50].

Fat tissue produces a number of peptides of the blood pressure regulation system: angiotensinogen, angiotensin I and II, renin. These peptides directly affect renal blood flow and nephron function. The pathogenetic relationship between arterial hypertension (AH) and obesity is not fully understood. Obesity-related arterial hypertension is clearly the result of a combination of many factors. Obesity increases the risk of developing hypertension by 65-75% [49]. A hypothesis has been put forward that increased concentrations of insulin and leptin can activate obesity-related arterial hypertension by stimulating the centers of the sympathetic nervous system (hypothalamus or nucleus tractus solitarius in the midbrain). The connecting link between leptin and the sympathetic centers of the midbrain includes two transmitters known as the neuropeptide Y and melanocortin. Melanokortin receptor mutation has been found in families with early-onset obesity [30].

The key link connecting obesity and hypertension is the increase in tubular sodium reabsorption. An important determinant of tubular reabsorption is glomerular hyperfiltration.

Obviously, patients with obesity are not homogeneous in the nature of renal hemodynamic changes. Thus, during the examination of adult men with obesity (BMI > 36) by Israeli nephrologists (Rabin Medical Center), two groups of patients were identified who differed in the level of filtration fraction of sodium (FFNa). In both groups, CFT was significantly higher than in the group of individuals with normal body weight. In the group of patients with a high FFNa level, postglomerular oncotic pressure was 13% higher, and the fractional excretion of lithium (a marker of proximal sodium reabsorption) was 33% lower than in the control group.

In the second group with normal FFNa levels, postglomerular oncotic pressure and fractional lithium excretion remained normal. The authors believe that the mechanism of hyperfiltration in pronounced obesity is heterogeneous [27]. Previous observations by these authors of patients with pronounced obesity showed that renal plasma flow (RPF) changes to a lesser extent compared to CKD.

It is assumed that in diabetes mellitus [57] and obesity [27] in the kidneys, the proximal reabsorption of sodium increases under the influence of an unknown factor that activates tubuloglomerular feedback and thus causes glomerular hyperfiltration.

The authors believe that a vicious circle is created: increased sodium reabsorption increases CFT, which, in turn, leads to an increase in FF, an increase in postglomerular oncotic pressure, stimulation of sodium reabsorption, and again - an increase in CFT. This effect of hyperfiltration on sodium reabsorption reduces its excretion associated with high CFT levels and supports both phenomena - salt retention and hyperfiltration. There are other mechanisms that increase sodium reabsorption in individuals with obesity. Obesity is associated with the activation of RAAS caused by many factors, including the secretion of angiotensin II by adipocytes. Increasing the concentration of angiotensin II increases the proximal reabsorption of sodium without affecting the FF level of this hormone [57].

Another determinant of sodium excretion is the pressure in the interstitial tissue of the kidneys. It is assumed that the increase in pressure in the interstitium may be caused by subcapsular fat infiltration and abdominal fat deposits. In this case, the tubules are compressed, the urine flow slows down, and sodium reabsorption increases in the loop of Henle [34] and the proximal part of the nephron [57].

It must be acknowledged that high CRF values cannot be considered a universal sign of obesity. Indian pediatricians, comparing this indicator in school-age children (average age -9 years, i.e., before puberty) with excess body weight with the control group of children who did not differ by age and gender, but with normal body weight, found no significant differences in either CFT values, blood pressure values, or albumin excretion [59].

In some adult patients with clinically significant obesity, there is a decrease in CFT, an increase in renal vascular resistance, and a decrease in effective renal blood flow [23]. Increased intra-abdominal pressure is a common phenomenon in individuals with high values of the "waist-hip" index. It can cause renal vein compression and thus increase venous pressure and reduce renal perfusion. In addition, increased intra-abdominal pressure can increase pressure in the inferior vena cava, further worsening the outflow of renal veins. In confirmation of this hypothesis, when measuring ileofemoral venous pressure, its high values were found in pronounced degrees of obesity and a positive correlation of this indicator with intra-abdominal pressure was found [59].

Increased intra-abdominal pressure can have other hemodynamic consequences: increased intrathoracic pressure, increased load on the right heart chambers, pulmonary hypertension, and reduced cardiac output. All these conditions can also reduce renal perfusion [22].

Diagnosing kidney damage in patients with obesity is generally not difficult. Urinary changes in them, as a rule, are less informative: leukocyturia, erythrocyturia are not characteristic (it is necessary, nevertheless, to keep in mind that this category of patients is more susceptible to the risk of nephrolithiasis, primarily urate), proteinuria does not exceed the "trace" level. A significantly more accurate method for diagnosing the early stage of kidney damage is the quantitative determination of albumin in urine, which allows for the timely detection of microalbuminuria [45].

At the screening examination stage, test strips (micral-test) can be used. It is also necessary to determine the serum creatinine concentration and calculate the glomerular filtration rate using Cockroft-Gault or MDRD formulas, although it is believed that the diagnostic accuracy of these tests decreases in patients with obesity. It is necessary to assess the indicators characterizing lipoprotein metabolism (serum concentration of total cholesterol, low, very low, and high-density lipoproteins, triglycerides), gastric glycemia, and uricemia, as well as to conduct diagnostic tests used to diagnose insulin resistance [3,15].

Conclusion

Obesity is the earliest and most noticeable sign of a metabolic disorder in a child. It appears significantly earlier than arterial hypertension, insulin resistance, and diabetes mellitus. The prerequisites for all these conditions are laid during the intrauterine period. Even in the early stages of excessive fat accumulation in the body, significant changes occur in the target organs. The kidneys are one of the first organs to take on the compensatory function with increasing body weight and simultaneously undergoing pathological changes.

The main links in the pathogenesis of obesity nephropathy are hemodynamic disorders, endothelial dysfunction, exposure to biologically active substances secreted by adipocytes, lipotoxicity, and latent inflammation. Pathological factors affecting the kidney are closely interconnected, complementing and activating each other, forming a complex interweaving. In pediatrics, it is practically important to distinguish a risk group for the formation of obesity nephropathy, metabolic syndrome, and cardiorenal syndrome. The risk group should include children born with low body weight at gestational age, children with signs of early obesity, children from families with obesity, carbohydrate metabolism disorders, and arterial hypertension. Further research will allow individualizing the approach to each child with excess body weight, diagnosing and correcting the leading link of impaired metabolism.

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