

ЖУРНАЛ

гепато-гастроэнтерологических
исследований



№3 (Том 6)

2025

ЖУРНАЛ ГЕПАТО-ГАСТРОЭНТЕРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

ТОМ 6, НОМЕР 3

JOURNAL OF HEPATO-GASTROENTEROLOGY RESEARCH

VOLUME 6, ISSUE 3





ISSN 2181-1008 (Online)

Научно-практический журнал
Издается с 2020 года
Выходит 1 раз в квартал

Учредитель

Самаркандский государственный
медицинский университет,
tadqiqot.uz

Главный редактор:

Н.М. Шавази д.м.н., профессор.

Заместитель главного редактора:

М.Р. Рустамов д.м.н., профессор.

Ответственный секретарь

Л.М. Гарифулина к.м.н., доцент

Редакционная коллегия:

Д.И. Ахмедова, д.м.н., проф;
А.С. Бабажанов, к.м.н., доц;
Ш.Х. Зиядуллаев, д.м.н., доц;
Ф.И. Иноятова, д.м.н., проф;
М.Т. Рустамова, д.м.н., проф;
Н.А. Ярмухамедова, к.м.н., доц.

Редакционный совет:

Р.Б. Абдуллаев (Ургенч)
М.Дж. Ахмедова (Ташкент)
А.Н. Арипов (Ташкент)
М.Ш. Ахророва (Самарканд)
Н.В. Болотова (Саратов)
Н.Н. Володин (Москва)
С.С. Давлатов (Бухара)
А.С. Калмыкова (Ставрополь)
А.Т. Комилова (Ташкент)
М.В. Лим (Самарканд)
М.М. Матлюбов (Самарканд)
Э.И. Мусабаев (Ташкент)
А.Г. Румянцев (Москва)
Н.А. Тураева (Самарканд)
Ф.Г. Ульмасов (Самарканд)
А. Фейзиоглу (Стамбул)
Ш.М. Уралов (Самарканд)
А.М. Шамсиев (Самарканд)
У.А. Шербеков (Самарканд)

Журнал зарегистрирован в Узбекском агентстве по печати и информации

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

ОРИГИНАЛЬНЫЕ СТАТЬИ

1	Ахмедова М.М. НАРУШЕНИЕ ЦЕЛОСТНОСТИ КЛЕТОЧНЫХ МЕМБРАН И УСИЛЕННОЕ ОКИСЛЕНИЕ ЖИРОВ (ПЕРЕКИСНОЕ ОКИСЛЕНИЕ ЛИПИДОВ) У ДЕТЕЙ С ЗАБОЛЕВАНИЯМИ ПОЧЕК, СВЯЗАННЫМИ С НАРУШЕНИЕМ ОБМЕНА ВЕЩЕСТВ.....	6
2	Ашурова М.Ж. СРАВНИТЕЛЬНАЯ ОЦЕНКА ЭФФЕКТИВНОСТИ СХЕМ ТЕРАПИИ ХОЛЕКАЛЬЦИФЕРОЛОМ У ДЕТЕЙ С ОЖИРЕНИЕМ ПРИ ДЕФИЦИТЕ И НЕДОСТАТОЧНОСТИ ВИТАМИНА D	9
3	Гарифуллина Л.М. ФАКТОРЫ РИСКА МЕТАБОЛИЧЕСКИХ НАРУШЕНИЙ У ДЕТЕЙ С ОЖИРЕНИЕМ: РОЛЬ ПЕРИНАТАЛЬНОГО ПРОГРАММИРОВАНИЯ.....	12
4	Goyibova N.S. MECHANISM OF INFLUENCE OF OVERWEIGHT AND OBESITY ON KIDNEYS IN CHILDREN.....	15
5	Ибрагимова М.Ф., Холмурадова Н.Дж., Шокирова Ш.Б. СОВЕРШЕНСТВОВАНИЕ МЕТОДОВ ЛЕЧЕНИЯ НЕОНАТАЛЬНОЙ ПНЕВМОНИИ НА ФОНЕ ВНУТРИУТРОБНОЙ ИНФЕКЦИИ.....	19
6	Ibragimova Yu.B. TURLI YOSHDAGI BOLALARDA O`TKIR REVMATIK ISITMANING RIVOJLANISH XUSUSIYATLARI.....	23
7	Исламова Д.С. ОПЫТ ПРИМЕНЕНИЯ ПРЕПАРАТА ВИСМУТА В ПЕРВОЙ ЛИНИИ ЭРАДИКАЦИИ HELICOBACTER PYLORI ПРИ ХРОНИЧЕСКОЙ ЯЗВЕННОЙ БОЛЕЗНИ ДВЕНАДЦАТИПЕРСТНОЙ КИШКИ У ДЕТЕЙ ПОДРОСТКОВОГО ВОЗРАСТА.....	26
8	Исламова Д.С. КЛИНИКО-ЭНДОСКОПИЧЕСКАЯ ХАРАКТЕРИСТИКА ГАСТРОДУОДЕНИТОВ У ПОДРОСТКОВ ПРИ ИНФИЦИРОВАНИИ HELICOBACTER PYLORI.....	31
9	Кудратова Г.Н. ЦЕРЕБРАЛЬНАЯ ИШЕМИЯ У НОВОРОЖДЁННЫХ: КЛИНИКО-НЕВРОЛОГИЧЕСКИЕ ОСОБЕННОСТИ И ДИАГНОСТИЧЕСКИЕ КРИТЕРИИ.....	34
10	Кудратова Г.Н. АЛЬФА ЛИПОВАЯ КИСЛОТА В ЛЕЧЕНИИ МЕТАБОЛИЧЕСКОГО СИНДРОМА У ДЕТЕЙ.....	37
11	Маматкулова Ф.Х. БОЛАЛАРДА ЎТКИР МИЕЛОИД ЛЕЙКЕМИЯНИ ДАВОЛАШДА МАҚСАДЛИ ПРЕПАРАТЛАРНИ КЎЛЛАШ.....	40
12	Набиева Ш.М. ХРОНИЧЕСКАЯ ГИПОКСИЯ ПЛОДА КАК ФАКТОР РИСКА РАЗВИТИЯ ПЕРИНАТАЛЬНОЙ ЭНЦЕФАЛОПАТИИ НОВОРОЖДЕННЫХ ОТ МАТЕРЕЙ С ПАТОЛОГИЕЙ АКУШЕРСКО-ГИНЕКОЛОГИЧЕСКОГО АНАМНЕЗА.....	43
13	Rasulova N.A., Rasulov A.S. A REASONABLE APPROACH TO THE TREATMENT OF RICKETS DEPENDING ON THE INTESTINAL MICROFLORA DISORDER.....	46
14	Rasulova N.A., Axmedova M.M. THE EFFECT OF MICROBIocenosis ON BIOCHEMICAL PARAMETERS IN CHILDREN WITH SIGNS OF RICKETS.....	49
15	Рустамов М.Р. ОЦЕНКА КАЧЕСТВА ЖИЗНИ И ОСОБЕННОСТЕЙ ПИТАНИЯ У ДЕТЕЙ И ПОДРОСТКОВ С ОЖИРЕНИЕМ.....	52

16	Рустамов У.М., Гарифулина Л.М. ФАКТОРЫ РИСКА РАЗВИТИЯ АРТЕРИАЛЬНОЙ ГИПЕРТЕНЗИИ И ОЖИРЕНИЯ У ДЕТЕЙ И ПОДРОСТКОВ.....	54
17	Файзуллаева Х.Б. ЯНГИ ТУҒИЛГАН ЧАҚАЛОҚЛАРДА ЛАКТАЗА ЕТИШМОВЧИЛИГИНИ ЭРТА АНИҚЛАШ ВА КОРРЕКЦИЯЛАШ.....	57
18	Kholmuradova Z.E. ARTERIAL HYPERTENSION IN OBESE CHILDREN AND ADOLESCENTS: PATHOPHYSIOLOGICAL MECHANISMS AND CLINICAL CORRELATIONS.....	61
19	Xolmurodova Z.E. SEMIZLIKNING BOLA PSIXOLOGIYASIGA TA`SIRI.....	65
20	Xusainova Sh.K. OPTIMIZING THE DIAGNOSIS AND TREATMENT OF NEONATAL JAUNDICE.....	68
ОБЗОР ЛИТЕРАТУРЫ		
21	Goyibova N.S. FEATURES OF KIDNEY DAMAGE IN CHILDREN WITH OBESITY.....	72
22	Turayeva D.X. SEMIZ BOLALARDA GEPATOBILIAR TIZIMDAGI PATOLOGIK O`ZGARISHLAR.....	77
ПАМЯТИ УЧИТЕЛЯ		
23	Ризаев Ж.А., Шавози Н.М., Рустамов М.Р. ЗАСЛУЖЕННЫЙ ДЕЯТЕЛЬ НАУКИ РЕСПУБЛИКИ УЗБЕКИСТАН, ДОКТОР МЕДИЦИНСКИХ НАУК, ПРОФЕССОР АБДУЛЛА ХАМРАЕВИЧ ХАМРАЕВ.....	80


JOURNAL OF HEPATO-GASTROENTEROLOGY RESEARCH

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)

 <http://dx.doi.org/10.5281/zenodo.17885247>

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 kafedrasini 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. Melanocortin 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 Cockcroft-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.

Список литературы/ Iqtiboslar / References

1. Боровкова Н.Ю., Боровков Н.Н., Теплова Н.О. Состояние вазодилатирующей функции эндотелия при артериальной гипертензии у больных хроническим гломерулонефритом с сохраненной функцией почек. *Клиническая медицина* 2009; (4):57-61

2. Вялкова АА, Савельева ЕВ, Кулагина ЕП, Белова МА. Особенности патологии почек при сахарном диабете и ожирении у детей. *Материалы конференции педиатров-нефрологов, урологов «Памяти А.В. Папаяна посвящается»*(Санкт-Петербург, 5 февраля 2016).
3. Вялкова АА, Николаева СН, Лебедева ЕН и др. Характеристика липидного обмена при ожирении у детей с нефропатиями. *Материалы III научно-практической конференции «Педиатрия и детская хирургия в Приволжском федеральном округе»* (Казань, 2006) ;107-108
4. Дедов И.И., Шестакова М.В. Диабетическая нефропатия М. «Универсум Паблишинг»: 2000.
5. Дедов И.И., Петеркова В.А. Федеральные клинические рекомендации (протоколы) по ведению детей с эндокринными заболеваниями. М.: Практика, 2014. – 442 с.
6. Заболевания почек и ожирение: молекулярные взаимосвязи и новые подходы к диагностике/ Вялкова А.А с соавт// Нефрология. 2017. Том 21. №3, С 25 – 38.
7. Клинико-патогенетические аспекты повреждения почек при ожирении (обзор литературы)./ Вялкова А.А.// *Нефрология* 2014; (3): 24- 33
8. Конюх ЕА, Парамонова НС. Клинические особенности течения острого и хронического гломерулонефритов у детей с дисфункцией эндотелия. *Журнал ГрГМУ* 2010; (2): 149-151
9. Кутырина ИМ, Краснова ЕА, Федорова ЕВ, Фомин ВВ. Поражение почек при ожирении: клинические, патогенетические и терапевтические аспекты. *Врач* 2005; (6):6-9 .
10. Мельник А.А. Метаболический синдром и риск хронической болезни почек. *Почки*. -Том 6, № 2, 2017. С. 80-90
11. Мухин НА, Балкаров ИМ, Моисеев СВ и др. Хронические прогрессирующие нефропатии и образ жизни современного человека. *Тер арх* 2004; (9): 5-11
12. Мартынов АИ, Аветяк АИ, Акатова ЕВ и др. Эндотелиальная дисфункция и методы ее диагностики. *Российский кардиологический журнал* 2005; (4): 94-98.
13. Наточин ЮВ. Нефрология и фундаментальная наука. *Нефрология* 2012; (16): 9-21.
14. Нетребенко О.К., Украинцев С.Е, Мельникова Ю.И. Ожирение у детей: новые концепции и направления профилактики. // Вопросы современной педиатрии том 16, №5. – С 399-405.
15. Николаева СН, Лебедева ЕН, Вялкова АА и др. Клиническая оценка уровня лептина и инсулина в крови у детей с ожирением. *Современные вопросы педиатрии* 2007; (5):485-486
16. Смирнова НН, Куприенко НБ. Нефропатия ожирения в педиатрии. *Нефрология* 2013 (6): 37-45
17. Смирнов АВ, Шилов ЕМ, Добронравов ВА. Национальные Рекомендации. Хроническая болезнь почек: основные принципы скрининга, диагностики, профилактики и подходы к лечению. *Нефрология* 2012; (16): 89-115
18. Смирнов А.В. Характеристика дислиппротеинемий у больных гломерулонефритом. *Нефрология* 1998; (2):76-83
19. Смирнов А.В, Румянцев АШ, Добронравов ВА, Каюков ИГ. XXI век – время интегративной нефрологии. *Нефрология* 2015; 19(2): 26-31
20. Тареева И.Е. Механизмы прогрессирования гломерулонефрита. *Терапевтический архив* 1996; 6: 5-10
21. Швецов М.Ю. Особенности синдрома артериальной гипертензии у больных волчаночным нефритом. Диссертация на соискание ученой степени кандидата медицинских наук. - Москва. - 1998
22. Avramoglu RK, Qiu W, Adeli K. «Mechanisms of metabolic dyslipidemia in insulin resistant states: Dereglulation of hepatic and intestinal lipoprotein secretion. *Front Biosci* 2003; 8: d464-d476
23. Bagby SP. Obesity-initiated metabolic syndrome and the kidney: A recipe for Chronic kidney disease? *J Am Soc Nephrol* 2004; 15: 2775-2791
24. Bonnet F., Deprele C., Sassolas A. et al. Excessive body weight as a new independent risk factor for clinical and pathological progression in primary IgA-nephritis. *Am J Kidney Dis* 2001; 37 (4): 720-727
25. Calle E., Thun M., Petrelli J. et al. Body-mass index and mortality in a prospective cohort of U.S. adults. *B Engl J Med* 1999; 7 (15):1097-1105
26. Carolin LA, Rodriguez MM. Obesity-related Nephropathy in Children. *Pediatr Health* 2009; 3(2): 141-153
27. Chagnac A, Herman M, Zingerman B et al. Obesityinduced glomerular hyperfiltration: its involvement in the pathogenesis of tubular sodium reabsorption. *Nephrol Dial Transplant* 2008; 23: 3946-
28. Chdek J, Adamczak M, Nieszporek T. et al. Adipose Tissue as an Endocrine Organ-A Nephrologists Perspective. *Obesity and kidney. Gunter Wolf* 2006; 15 (1): 70-90.
29. Cortes P, Zhao X, Riser B, Narins RG: Regulation of glomerular volume in normal and partially nephrectomized rats. *Am J Physiol* 1996; 270: F356 -F370
30. Greenfield JR, Miller JW, Keogh JM et al. Modulation of blood pressure by central melanocortinergic pathways. *NEJM* 2009; 360: 44-52
31. De Simone G., Devereux R.B., Roman, et al. Relation of obesity and gender to left ventricular hypertrophy in normotensive and hypertensive adults. *Hypertension* 1994; 23: 600-606
32. Foster MC, Hwang SJ, Larson MG et al. Overweight, obesity, and the development of stage 3 CKD: the Framingham Heart Study. *Am J Kidney Dis* 2008; 52(1): 39-48. doi: 10.1053/j. ajkd.2008.03.003
33. Freemark M. *Pediatric Obesity: Etiology, Pathogenesis, and Treatment*. Humana Press, New York, 2010; 27-30
34. Hall JE. The kidney, hypertension, and obesity. *Hypertension* 2003; 41: 625-633
35. Henegar J. R., Bigler S. A., Henegar L. K., Tyagi S. C. and Hall J. E. Functional and Structural Changes in the Kidney in the Early Stages of Obesity *J Am Soc Nephrol* 2001; 12: 1211-1217
36. Hovi P, Andersson S, Raikonen K et al. Ambulatory blood pressure in young adults with very low birth weight. *J Pediatr* 2010; 156: 54–59
37. Johnson D.W., Iabel N.M., Brown A.M. et al. The effect of obesity on renal transplant outcomes. *Transplantation* 2002; 74: 675-681
38. Kambham N., Markowitz G.S., Valeri A.M., Lin J., D'Agati V.D. Obesity-related glomerulopathy: An emerging epidemic. *Kidney Int* 2001; 59(4): 1498-1509
39. Kasiske BL, Crosson JT: Renal disease in patients with massive obesity. *Arch Intern Med* 1986; 146: 1105 -1109
40. Kato S, Nazneen A, Nakashima Y et al. Pathological influence of obesity on renal structural changes in chronic kidney disease. *Clin Exp Nephrol* 2009; 13: 332-340
41. Kiortsis, D.N. Management of Obesity-Induced Kidney Disease: A Critical Review of the Literature / D.N.Kiortsis, M.A. Christou // *Obes Facts*. - 2012 Nov. - Vol. 29; 5(6). - P.821-832. (Epub ahead of print).

42. Kuiper J.J. Effects of weight reduction and angiotensin-converting enzyme inhibition on IgA nephropathy-associated proteinuria. *Nephron* 1996; 74(2): 462-463
43. Landsberg L, Krieger DR: Obesity, metabolism, and the sympathetic nervous system. *Am J Hypertens* 1989; 2: 1255-1325
44. Levy D., Garrison R.J., Savage D.D. et al. Left ventricular mass and incidence of coronary heart disease in an elderly cohort. *Ann Intern Med* 1989; 110: 101-107
45. Looker HC. Adiponectin concentrations are influenced by renal function and diabetes duration in Pima Indians with type 2 diabetes. *J Clin Endocrinol Metab* 2004; 89:4010-4017
46. Malyszko J, Bachorzewska-Gajewska H, Malyszko JS et al. Prevalence of Chronic Kidney Disease in Elderly Patients with Normal Serum Creatinine Levels Undergoing Percutaneous Coronary Interventions. *Gerontol* 2010; (56): 51-54.
47. Meier-Kriesche H.U., Vaghela M., Thambuganipalle R. et al. The effect of body index on long-term renal allograft survival. *Transplantation* 1999; 68 (9): 1294-1297
48. Miyazaki Y, Cersosimo E, Triplitt C, DeFronzo RA. Rosiglitazone decreases albuminuria in type 2 diabetic patients. *Kidney Int* 2007; 72 (2):1367-1373. doi: 10.1038/sj.ki.5002516/
49. Naumnik B, Mysliwiec M. Renal consequences of obesity. *Med Sci Monit* 2010; 16(8): RA163-170
50. Neeraja KH, Markowitz GS, Anthony MV et al. Obesity-related glomerulopathy: an emerging epidemic. *Kidney Int* 2001; 59: 1498-1509
51. Praga M., Hernandez E., Morales E. Clinical features and long-term outcome of obesity-associated focal segmental glomerulosclerosis. *Nephrol Dial Transplant* 2001; 15: 1790-1798
52. Rea DJ, Heimbach JK, Grande JP et al. Glomerular volume and renal histology in obese and non-obese living kidney donors. *Kidney Int* 2006; 70: 1636-1641
53. Serra A, Romero R, Lopez D et al. Renal injury in the extremely obese patients with normal renal function. *Kidney Int* 2008; 73: 947-955
54. Saxena A.K., Chopra R. Renal risk of an emerging «Epidemic» of obesity: the role of adipocyte-derived Factors. *Dialysis and Transplantation* 2004; 33: 11-20
55. Sharma A.M. Ожирение и риск сердечно-сосудистых заболеваний: новые аспекты. 10th European Congress on Obesity, May 2000. Ожирение. Актуальные вопросы 2001; 5: 4-6/
56. Verani R.R. Obesity-associated FSGS: Pathologic features of lesion and relationship with cardiomegaly and hyperlipidemia. *Am J Kidney Dis* 1992; 20: 629-634
57. Vallon V, Blantz RC, Thomson S. Glomerular hyperfiltration and the salt paradox in early type 1 diabetes mellitus: a tubulocentric view. *J Am Soc Nephrol* 2003; 14:530-537
58. Vikse BE, Irgens LM, Leivestad T et al. Low birth weight increases risk for end stage renal disease. *J Am Soc Nephrol* 2008; 19:151-157
59. Weinberg J.M. Lipotoxicity. *Kidney Int* 2006; 70: 1560-1566
60. World Health Organization. Childhood overweight and obesity. <http://www.who.int/dietphysicalactivity/childhood/en/> 2013
61. who.int [Internet]. Global Health Observatory (GHO) data. Overweight and obesity [cited 2017 Aug 9]. Available from: http://www.who.int/entity/gho/ncd/risk_factors/overweight/en/.
62. Yamamoto S., Hanley E., Hahn A.B. et al. The impact of obesity in renal transplantation: An analysis of paired cadaver kidneys. *Clin Transplant* 2002; 16 (4):252-256

ЖУРНАЛ ГЕПАТО-ГАСТРОЭНТЕРОЛОГИЧЕСКИХ ИССЛЕДОВАНИЙ

ТОМ 6, НОМЕР 3

JOURNAL OF HEPATO-GASTROENTEROLOGY RESEARCH

VOLUME 6, ISSUE 3

Editorial staff of the journals of www.tadqiqot.uz

Tadqiqot LLC The city of Tashkent,
Amir Temur Street pr.1, House 2.

Web: <http://www.tadqiqot.uz/>; Email: info@tadqiqot.uz
Phone: (+998-94) 404-0000

Контакт редакций журналов. www.tadqiqot.uz

ООО Tadqiqot город Ташкент,
улица Амира Темура пр.1, дом-2.

Web: <http://www.tadqiqot.uz/>; Email: info@tadqiqot.uz
Тел: (+998-94) 404-0000