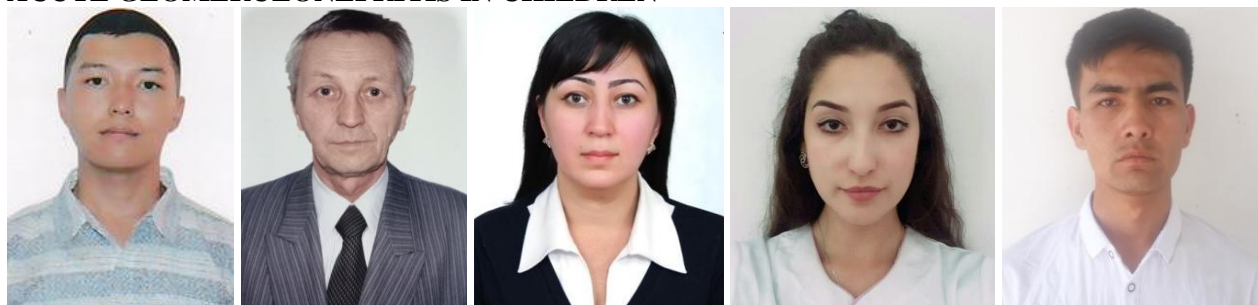


DYNAMICS OF CLINICAL AND LABORATORY PARAMETERS IN VARIOUS FORMS OF ACUTE GLOMERULONEFRITIS IN CHILDREN



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БОЛАЛАРДА ЎТКИР ГЛОМЕРУЛОНЕФРИТНИ ТУРЛИ ХИЛ ШАКЛЛАРИДА КЛИНИК - ЛАБОРАТОР КУРСАТКИЧЛАРНИНГ ДИНАМИКАСИ

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ДИНАМИКА КЛИНИКО-ЛАБОРАТОРНЫХ ПОКАЗАТЕЛЕЙ ПРИ РАЗЛИЧНЫХ ФОРМАХ ОСТРОГО ГЛОМЕРУЛОНЕФРИТА У ДЕТЕЙ

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Резюме. Болалик давридаги буйрак патологияси таркибида гломерулонефрит алоҳида ўрин тутди. Мақсад: Бу ишда бирламчи ва иккиламчи ГН билан касалланган болаларда буйракларнинг функционал ҳолатини баҳолаш ва клиник – лаборатор хусусиятларини ўрганиш. Тадқиқот материаллари ва усуллари. 70 нафар бемор бола кузатув остига олинган еди. Улардан 25 нафарда капилляр токсик нефрит (КН), 20 нафарда етакчи гематурик синдром билан бирламчи гломерулонефрит (БГН) ва 25 нафарда буйрак шикастланишсиз геморрагик васкулит (ГВ б/ш-сиз) бор еди. Натижалар. Капилляр токсик нефрит ва буйрак шикастланишсиз геморрагик васкулит билан оғриган беморларни солиштирганда (1 ва 3-гурuhlар) 3-гурuhlда (64%) қизлар ҳам устунлик қилиши аниқланди. Улар орасида мактаб ёшидаги болалар 1-гурuhlга қараганда камроқ еди. Гарчи улар аксарият қисмини ташкил этган бўлса-да (68%), улар буйрак шикастланиши (84%) билан кечувчи ГВ асоратлари ҳолатларида бўлгани каби унчалик устун бўлмади. Хулоса. КН нинг ерта салбий прогностик белгилари - касалликнинг экстра-ренал белгилари билан бошланиши, макрогематуриянинг протеинурия билан комбинацияси ва буйраклар фаолиятининг бузилиши.

Калит сўзлар: ўткир гломерулонефрит, капилляртоксик нефрит, гематурия, геморрагик васкулит.

Abstract. In the structure of renal pathology in childhood, a special place is occupied by glomerulonephritis. The aim of this work was to study the clinical features and evaluate laboratory parameters in children with primary and secondary acute GN. Material and research methods. Only 70 sick children were under observation. Of these, 25 had capillary toxic nephritis (CTN), 20 had primary glomerulonephritis (PGN) with a leading hematuric syndrome, and 25 had hemorrhagic vasculitis without renal damage (HV w/rd) without subsequent kidney damage. Results. When comparing patients with capillary toxic nephritis and hemorrhagic vasculitis without kidney damage (groups 1 and 3), it was found that girls also predominate in group 3 (64%). Among them, there were fewer children of school age than in the 1st group. Although they were in the majority (68%), they did not greatly prevail as in cases of HV complications with kidney damage (84%). Conclusions. Early unfavorable prognostic symptoms of CTN are the onset of the disease with extrarenal symptoms, a combination of gross hematuria with proteinuria, and functional impairment of the kidneys.

Key words: acute glomerulonephritis, capillary toxic nephritis, hematuria, hemorrhagic vasculitis.

One of the important places in the pathology of childhood is occupied by inflammatory diseases of the urinary system, among which various forms of glomerulonephritis are of special attention [1, 2, 6].

This interest is caused by the diversity of hematuric nephritis according to the mechanisms of development and prognosis (primary, secondary, etc.) [3, 4].

Special diagnostic and therapeutic difficulties of hematuric nephritis are known. Hematuric nephritis often begins in childhood, can occur over several decades, and in adults transform into more severe forms of the disease [5, 7].

In literature of our and foreign countries over the past decades, many works have appeared on primary hematuric glomerulonephritis, various clinical and morphological variants have been identified among them, and therapy issues are being studied [10, 11].

At the same time, along with primary GN, among acquired hematuric nephritis (HN), renal damage in hemorrhagic vasculitis (HV), i.e. capillary toxic nephritis (CTN) occupies a special place [12].

The available works on CTN concern more the study of its chronic forms, the course in adults. Due to the fact that children often get sick with HV, the study of CTN, especially its early stages, is relevant in childhood. Meanwhile, the frequency of HV and accompanying kidney damage increases [8, 9].

The aim of this work was to study the clinical features and evaluate laboratory parameters in children with primary and secondary acute GN.

Material and research methods. Only 70 sick children were under observation. Of these, 25 were diagnosed with capillary toxic nephritis (CTN), 20 with primary glomerulonephritis (PGN) with a leading hematuric syndrome, and 25 with hemorrhagic vasculitis (HV w/rd) without subsequent kidney damage.

For the convenience of a comparative analysis of clinical and laboratory symptoms in the description of CI, the principles of the Vinnitsa classification of primary glomerulonephritis (1976) were conditionally used.

Of 25 patients with CTN, 10 had a disease duration of no more than 1 year by the time of observation ("acute CTN"). It should be noted that in the prevailing majority of acute CTN, the duration of the disease was less than 3 months (in 85.6%); 57.1% of patients were observed from the first days-weeks of the disease. In 6 patients, CI was more than 1 year old (up to 6 years).

Among 20 patients with primary GN, there were 14 children with acute GN and 6 children with hematuric form of chronic GN. Patients with nephrotic and mixed forms of GN were not included.

All patients underwent clinical and laboratory examination, including general blood analysis, general urinalysis, determination of total protein in blood serum, protein fractions, residual nitrogen, urea, creatinine, cholesterol, serum transaminases.

The study of the coagulogram included: determination of the type and amount of total fibrinogen, prothrombin complex, recalcification time, thrombin time, ethanol test, blood clotting time.

In addition, quantitative urine tests were studied: daily excretion of protein and formed elements according to Addis-Kakovsky, according to Nechiporenko. Glomerular filtration was determined by the clearance of endogenous creatinine concentration function - according to the Zimnitsky test. According to the indications, excretory urography, ultrasound and renography were performed, rarely cystography.

The state of the glomerular apparatus was determined according to the value of glomerular filtration (endogenous creatinine clearance), which was calculated using the Van-Slake formula. The degree of proteinuria was assessed by the amount of daily protein excretion, related to body weight in kilograms. The relationship between proteinuria and glomerular filtration was expressed by permeability index (the ratio of protein excretion per unit time to glomerular filtration).

The results of the research. 25 patients with hemorrhagic vasculitis, who did not have symptoms of kidney damage, had extrarenal symptoms (skin, articular, abdominal). For all groups of patients, age, gender, season at the onset of the disease, previous illnesses and some anamnestic risk factors (allergies in the family, in a child, heredity for kidney disease, frequency of colds) were analyzed. The analysis of clinical and main laboratory symptoms of capillary toxic nephritis was carried out, starting from the onset of the disease, depending on the subsequent outcome.

A comparative study of the main clinical and laboratory symptoms of capillary toxic nephritis in children (group 1) was carried out in comparison with the nephritic syndrome of primary glomerulonephritis (group 2) and hemorrhagic vasculitis without kidney damage (group 3, table I).

Among 25 patients with capillary toxic glomerulonephritis, 15 had acute CTN (60%) and 10 (40%) had chronic CTN. Capillarotoxic nephritis developed more often in school-age children (acute CI - in 82.5%, chronic - in 96.1%) (Table 1).

Thus, in terms of age distribution, CI was close to primary glomerulonephritis, in which school-age children accounted for 84% and 65%, respectively (Fig. 1). It should be noted that there are some differences between chronic CTN and chronic GN; Chronic capillary toxic nephritis occurred exclusively in school age children while among the primary chronic hematuric nephritis, children of this age accounted for 65%.

The prevalence of boys among primary glomerulonephritis is a well-known fact: according to our data, they amounted to 75% (Fig. 1).

In contrast, it turned out that capillary toxic nephritis develops both in girls and boys with some prevalence in girls (57.5%). Moreover, the frequency of females is growing among chronic CTN (65.3%).

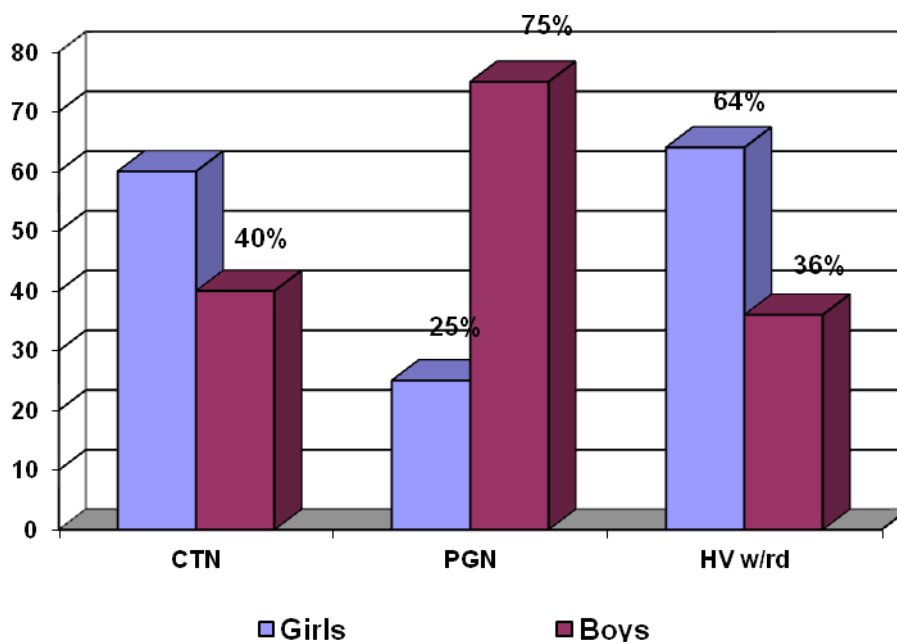


Fig. 1. Distribution of examined children depending on gender

Table 1. Distribution of patients depending on age and forms of GN

GN form	Age of patients		
	from 3 to 6 years	from 7 to 15 years	Total
CTN	4 (16%)	21 (84%)	25 (35,7%)
PGN	7 (35%)	13 (65%)	20 (28,6%)
HV w/rd	8 (32%)	17 (68%)	25 (35,7%)
Total	19 (27,1%)	51 (72,9%)	70 (100%)

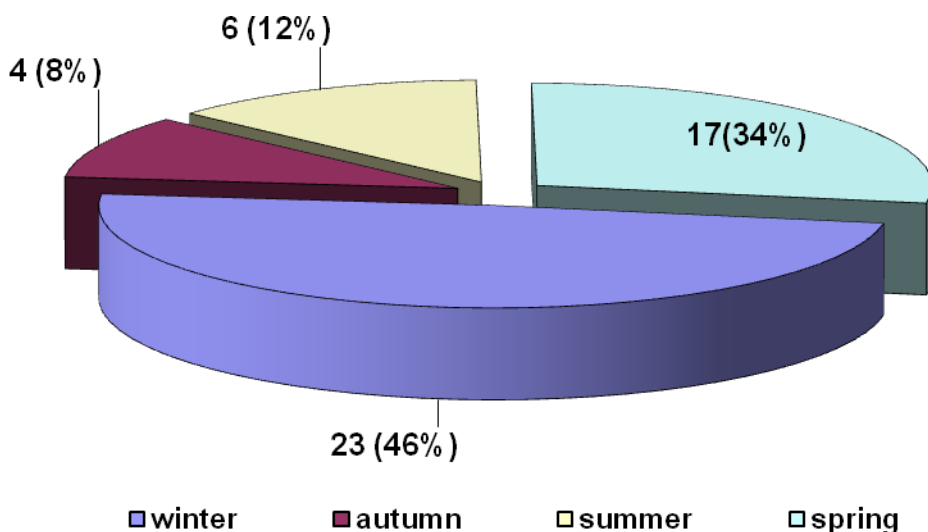


Fig. 2. Distribution of HB patients by seasons of the year

When comparing patients with capillary toxic nephritis and hemorrhagic vasculitis without kidney damage (groups 1 and 3, Table 1), it was found that girls also predominate in group 3 (64%). Among them, there were fewer children of school age than in the 1st group. Although they were the majority (68%), they did not greatly prevail as in cases of complications of hepatitis B with kidney damage (84%).

Hemorrhagic vasculitis without kidney damage develops in 32% of children aged 3-6 years (with 16% among CTN), occasionally - in children under 3 years of age (4%) in the absence of such damage among CTN.

Thus, among patients with hemorrhagic vasculitis, children of school age are most susceptible to nephritis, especially boys.

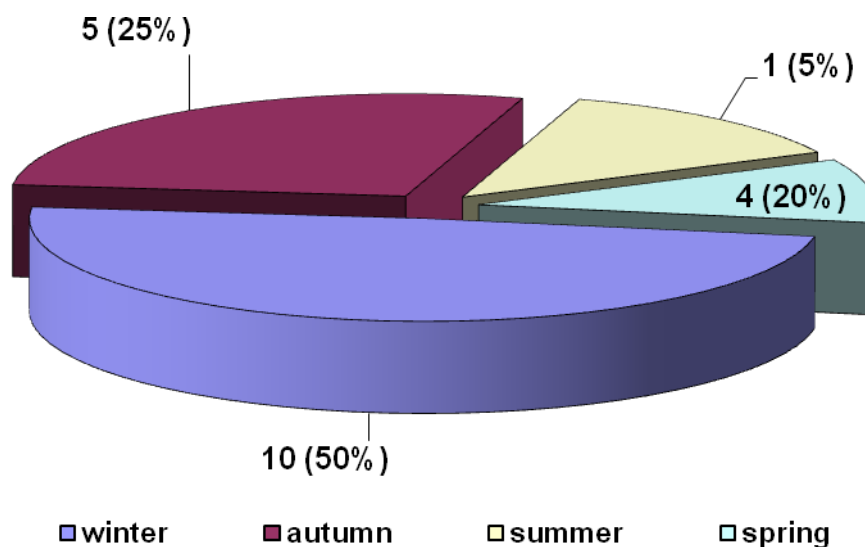


Fig. 3. Distribution of PHN patients by seasons

Table 2. Clinical syndromes of the initial period in patients with acute capillary toxic nephritis, primary glomerulonephritis and hemorrhagic vasculitis without kidney damage

№	Group of patients	Onset syndromes							
		Nephritic syndrome (%)	Of nephritis «urinary syndrome» (%)			Of vasculitis			
			Total	Hematuria + protein	Hematuria	skin (%)	Skin-articular (%)	Skin-abdominal (%)	Skin-articular-abdominal (%)
1	CTN n=25	8 (32)	17(68)	7 (28)	10(40)	3(12)	5(20)	7(28)	10(40)
2	PGN n=20	15(75)	5(25)	4(20)	1(5)	-	-	-	-
3	HV w/rd n= 25	-	-	-	-	1(4)	12(48)	4(16)	8(32)
	Total	23 (32,8)	22 (31,4)	11 (15,7)	11 (15,7)	4 (5,7)	17 (24,2)	11 (15,7)	18 (25,7)

In all three groups, the disease was most often preceded by ARVI and tonsillitis. Other factors (injury in all three groups - in 4-5%, etc.) were rare. In a fairly large percentage the disease began for no apparent reason (24-31.8%).

According to anamnestic data, children were prone to colds, especially often patients with hemorrhagic vasculitis (group 2 - 22.7%, group 3 - 44%, with 19.2% in group 1). Particularly noteworthy was the unfavorable allergic anamnesis in both groups of patients with hemorrhagic vasculitis (30.3% in the 2nd and 40% in the 3rd group).

At the same time, indications for the pathology of the kidneys and urinary tract, including nephropathy in pregnancy, were most in the group of children with primary glomerulonephritis (34% at 13.6 and 8% in others).

Capillarotoxic nephritis and hemorrhagic vasculitis without kidney damage occurs more often in winter and spring, and primary glomerulonephritis occurs in autumn and winter. We attribute this to

ARVI, a factor of hypothermia, and the beginning of HV - with an increase in allergenic factors in the spring, a decrease in the body's immune responses.

Capillarotoxic nephritis differs from primary GN by initial syndromes: in a significant number of cases, 92.5% (Table 2) it manifested itself as an isolated urinary syndrome. Occasionally, pastosity of the face was only noted. Of 3 patients whose manifestation was attributed to the "nephritic syndrome", 2 also did not have edema, they had arterial hypertension as an extrarenal symptom, it also did not reach high numbers in our observations (from 120/90 to 140/100 mm Hg. Art. .) and was short-lived (1-2 days). Only 1 boy had both extrarenal symptoms, and subsequently developed persistent nephrotic syndrome.

Gross hematuria was only observed in 30% of acute CTN cases. Isolated urinary syndrome was represented by isolated hematuria or its combination with proteinuria less than 1%. The combination of hematuria (mainly macrohematuria) with proteinuria marked more than 1% (table 2).

Thus, the prevailing number of CTN patients, starting from the onset, had urinary syndrome, the duration of which lasted from several days to months or longer. Attention was drawn to the fluctuation of symptoms: with periods of decrease and recurrence of hematuria.

Our observations have shown that sometimes short-term normalization of urine tests is possible in this microhematuria, which does not allow us to think about a complete regression.

More or less complete remission can be said about by observing the urinary syndrome in patients no less than 2 years after the onset.

Primary acute glomerulonephritis, on the contrary, mainly began with more marked symptoms, more often in combination of urinary syndrome with extrarenal symptoms. So, in 75% acute nephritic and only in 25% - isolated urinary syndrome were observed. Differences in the frequency of syndromes between groups 1 and 2 were statistically significant ($P < 0.001$). The very nature of the nephritic syndrome was different: in all cases there was distinct edema. In 7 of 10 cases, they were combined with arterial hypertension (up to 180/90 - 140/100). Although high BP values were also short-lived, moderate hypertension sometimes lasted up to 7–14 days.

The leading urinary syndrome, as in the 1st group, was hematuria (gross hematuria in 6 out of 10 cases). Isolated urinary syndrome, was observed only in 5 patients, in 1 of them it was in the form of isolated hematuria, in 4 - its combination with proteinuria 7 more than 1%.

Symptoms of vasculitis in patients with capillary toxic nephritis (1 - gr.) are presented in table 2.2 in comparison with group 3. The onset of hemorrhagic vasculitis with the subsequent development of nephritis (gr. 1) and without nephritis (gr. 3) differed in the symptoms of vasculitis. In children of the 1st group, combinations of skin-articular, skin-abdominal and skin-articular-abdominal syndromes were almost the same. And in hemorrhagic vasculitis without nephritis, skin-abdominal syndrome was observed somewhat less frequently. In this group, joint damage was more common than in the 1st (48% and 20%, respectively). These groups differed more clearly in the frequency of liver damage. In patients with capillary toxic nephritis, we noted a more frequent increase in the size of the liver in the initial period of the disease (17.5%) than in HV without nephritis (4%). A significant difference between the groups was found in the level of ALT, the increase of which in the 1st group was observed in 34.3%, and in the 3rd only 4.7% ($P < 0.01$). The blood test for HbS Ag was negative and subsequently the enzyme was eliminated, which suggests that these changes were caused by the active period of hemorrhagic vasculitis.

The difference between the 1st and 3rd groups was also revealed in the higher frequency of recur-

rence of non-renal syndromes: in children of the 1st group (with kidney damage). Skin syndrome recurred most frequently (12% in group 1, 4% in group 3, $P < 0.01$).

Moreover, 60.8% of patients with CTN had more than 2 relapses (8% in the 3rd group). Rarely (4.5% and 0%, respectively), there was a recurrence of the articular syndrome. The frequency of recurrence of abdominal syndrome in two groups did not differ significantly (5% in the 1st group and 4% in the 3rd group).

Kidney damage in our observations appeared after non-renal symptoms, quite early, more often (33.3%) within 1 month (of which in half of the cases in the first days), in 28.5% - 2 months after the onset of skin and other symptoms, rarely at a later period.

Although it is possible that other non-renal syndromes may have preceded kidney damage, there were no such cases in our observations with acute capillary-toxic nephritis.

The most frequent disorders both in the group of patients with capillary toxic and primary glomerulonephritis there was a decrease in the function of osmotic concentration (in 50% and 78.5%, respectively), as well as changes in the general analysis of peripheral blood in the form of normochromic anemia (50% in both groups.) and an increase in ESR (55.9% and 78.6%, respectively).

Most patients had moderate anemia (Hb 90-70 g/l), ESR within 15-25 mm/hour, rarely up to 40 mm/hour. Slightly more frequent decrease in concentration function and high ESR in patients with primary GN due to the frequency of their nephritic syndrome, while in patients with CI these changes were combined with an isolated urinary syndrome.

These laboratory symptoms, as is known, characterize the severity of the inflammatory process, a decrease in the function of concentration, and partly anemia, mainly due to morphological changes in the tubulointerstitial tissue of the kidneys.

Discussion of the research results. If in acute nephrotic syndrome one can assume the severity and evidence of the inflammatory process in the tubulointerstitial tissue, respectively, with an acute marked inflammatory change in the glomeruli of the kidneys, the changes revealed in capillary toxic nephritis, where isolated urinary syndrome prevailed, may be evidence of a predominant lesion of the tubulointerstitial apparatus and confirm the position of involvement of various structural elements of the nephron in the process of this disease, sometimes with a predominance of changes in the tubulointerstitial tissue and vessels over glomerular lesions. Biochemical studies revealed azotemia in 25% of patients with acute CTN and in 38.4% of acute GN, also caused by the acute period of glomerulonephritis. At the same time, it should be noted that a significant decrease in glomerular filtration in pa-

tients with CTN was not revealed, and among patients with GN, 2 of 7 examined showed a moderate decrease in glomerular filtration (54-62 ml/min) at the onset. For the onset of the acute nephritis examined by us, hypoproteinemia was a slight characteristic symptom. In total, in single patients, its moderate decrease was revealed (often not lower than 51-55 g/l). This is due to the selected contingent of patients (hematuric nephritis). Only at the onset, 1 patient with CI with severe edematous syndrome and proteinuria had short-term hypoproteinemia. Another symptom, hypoalbuminemia, was more frequently noted. Dysproteinemia was more often due to a relative increase in the fraction of globulins.

Conclusions. Thus, the study of clinical and laboratory symptoms in capillary toxic nephritis showed that CTN develops more often in children older than 7 years, equally often in boys and girls. Early unfavorable prognostic symptoms of CI are the onset of the disease with extrarenal symptoms, a combination of gross hematuria with proteinuria, and functional impairment of the kidneys.

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ДИНАМИКА КЛИНИКО-ЛАБОРАТОРНЫХ ПОКАЗАТЕЛЕЙ ПРИ РАЗЛИЧНЫХ ФОРМАХ ОСТРОГО ГЛОМЕРУЛОНЕФРИТА У ДЕТЕЙ

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Резюме. В структуре почечной патологии в детском возрасте особое место занимают гломерулонефриты. Целью настоящей работы явилось изучение клинических особенностей и оценка лабораторных показателей у детей с первичным и вторичным острым ГН. Материал и методы исследования. Под наблюдением находилось всего 70 больных детей. Из них у 25 капилляротоксический нефрит (КН), у 20 - первичный гломерулонефрит (ПГН) с ведущим гематурическим синдромом, у 25 - геморрагический васкулит (ГВ б/пн) без последующего поражения почек. Результаты. При сравнении больных капилляротоксическим нефритом и геморрагическим васкулитом без поражения почек (1 и 3- группы) установлено, что в 3 - группе также преобладают девочки (64%). Среди них меньше, чем в 1-группе было детей школьного возраста. Хотя их было большинство (68%), но не сильно преобладали как в случаях осложнений ГВ поражением почек (84%). Выводы. Ранними неблагоприятными прогностическими симптомами КН являются начало болезни с экстраренальных симптомов, сочетание макрогематурии с протемнурией и функциональными нарушениями почек.

Ключевые слова: острый гломерулонефрит, капилляротоксический нефрит, гематурия, геморрагический васкулит.