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LABORATORY FEATURES OF URATE NEPHROPATHY IN CHILDREN

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Abstract: The interest in the study of urate nephropathies in children is determined by the following points: the significant prevalence of CHI diseases in both the pediatric and adult populations, the global trend towards an increase in the frequency of chronic kidney disease (CKD) and chronic renal failure (CRF) despite all the achievements of modern clinical nephrology. **The aim of the work** is to study the clinical, genealogical and laboratory features of urate nephropathies in children. **Material and methods.** We observed 86 children aged 1 to 14 years with a newly diagnosed urate nephropathy. Of these, 37 boys (43%) and 49 girls (57%). Under the age of 3 years, 24 (28%), 4-7 years, 32 (37.2%), 8-14 years, 29 (44.8%). Results: We analyzed the pedigrees of 60 probands with urate nephropathy. Information was obtained on 1,376 relatives, of whom 34.7% suffer from uricopathies, which is 3 times higher than those in the population. The proportion of nephropathies (including urolithiasis) of these was 20.5% (excluding probands). It should be noted that in the pedigree of ballroom patients with urate nephropathy, there is an exceptional incidence of cardiovascular diseases, including hypertension (43.3%), gastroduodenal (19.7%) and hepatobiliary systems (39.6%), diseases associated with salt deposition (26.7%). Allergic diseases accounted for (9.2%), obesity, diabetes 7.1% and 2.7% neuropsychiatric diseases. Conclusion. Thus, urate nephropathies have a significant share in the structure of nephropathies. In clinical diagnosis, it is important to study the pedigree (identification of the uricopathic spectrum of pathology), clinical features (early age, absence of extrarenal signs) and biochemical parameters (uric acid in blood and urine). Patients with urate nephropathy need constant medical supervision.
Key words: nephropathy, children, uraturia, oxalaturia

Relevance: The interest in the study of urate nephropathies in children is determined by the following factors: the significant prevalence of CHI diseases in both the pediatric and adult populations, the global trend towards an increase in the frequency of chronic kidney disease (CKD) and chronic renal failure (CRF) despite all the achievements of modern clinical nephrology. Almost every kidney disease in a child is CKD, which is characterized by a progressive course with the development, often in childhood, adolescence and young age, of chronic kidney disease, requiring very expensive renal replacement therapy (RRT), the costs of which have begun to exceed the financial capabilities of even highly developed countries.

The increase in CRF in childhood against the background of an increase in the frequency of hereditary and congenital pathology of MHI, the lack of opportunities for a radical cure of most CKD, the frequent unsatisfactory results of the so-called "active" therapy of nephritis, even such obviously associated with bacterial infection as chronic pyelonephritis in the presence of a huge number of antibacterial drugs (M.S. Ignatova; 2011).

The real difficulties in providing high-tech RRT to all those in need, and the unsatisfactoriness of the long-term results of even such therapy, an increase in mortality at a

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relatively young age not only from terminal CRF (ESRD), but also from cardiovascular pathology against this background (Ignatova, 2013), determines the relevance of studying this problem in childhood.

The aim of the work is to study the clinical, genealogical and laboratory features of urate nephropathies in children.

Material and methods. The establishment of the dysmetabolic genesis of nephropathy provides a real opportunity for etiotropic therapy. During the examination of patients, diseases such as chronic nephritis, hereditary nephritis, abnormalities of the development of MHI, tubulopathy, and other types of metabolic nephropathies (hyperoxaluria, hypercalciuria, cystinuria) were excluded. Speaking about the etiological diagnosis of nephropathy, we mean not only the isolation of pathogens of the disease, but mainly the specifics of the background, which made possible the realization of this infection, or a microbial-free pathological process. To distinguish this group, the characteristic spectrum of renal and extrarenal pathology in the pedigree (uricopathy) served as the basis, the biochemical criteria were the index of uricemia (over 0.320 mmol/l) and urinary excretion of uric acid (>1 mg/ml / urine / day).

We observed 86 children aged 1 to 14 years with a newly diagnosed urate nephropathy. Of these, 37 boys (43%) and 49 girls (57%). Under the age of 3, 24 (28%), 4-7 years old 32 (37.2%), 8-14 years old 29 (44.8%). All patients had previously been under the supervision of local doctors from 6 months to 4 years under the general diagnosis of glomerulonephritis (26.3%), pyelonephritis (PN) (64%), urinary tract infections (9.7%). The treatment was carried out according to the established diagnoses. Due to the ineffectiveness of treatment at different times from the time of manifestation (from 6 months to 4 years from the moment of manifestation), they were sent to a hospital to clarify the diagnosis. The quantitative determination of oxalates was carried out according to N.V. Dmitrieva, uric acid in blood and urine using the Muller-Seifert method [12]. Calcium, phosphorus, and creatinine were determined by conventional methods [28]. The determination of the excretion of ammonia, titrated acids was carried out in the description of I. Todorov.

The data obtained in sick children were compared with those of 32 children of the same age with chronic nephritis, hematuric form, as well as 27 healthy children. As a result, 21 (24.4%) children with uraturia and isolated urinary syndrome (IUS) were identified, 18 (20.1%) children were diagnosed with dysmetabolic chronic interstitial nephritis (ChIN) and 43 (50%) children with secondary chronic pyelonephritis (HCPN) in the acute stage. On the basis of the interest in both cases of interstitial kidney tissue, patients (IN, HCPN) are united in one troupe. This also included 4 children with uric acid lithiasis and secondary pyelonephritis. The obtained results were processed by the statistical method of Student and Fisher. The arithmetic mean of the indicators (M), its average error (t) and the confidence index (P) were calculated.

Results: We analyzed the pedigrees of 60 probands with urate nephropathy. Information was obtained on 1,376 relatives, of whom 34.7% suffer from uricopathies, which is 3 times higher than those in the population. The proportion of nephropathies (including urolithiasis) of these was 20.5% (excluding probands). It should be noted that in the pedigree of ballroom patients with urate nephropathy, there is an exceptional incidence of cardiovascular diseases, including hypertension (43.3%), gastroduodenal (19.7%) and hepatobiliary systems (39.6%), diseases associated with salt deposition (26.7%). Allergic diseases accounted for (9.2%), obesity, diabetes 7.1% and 2.7% neuropsychiatric diseases. Such a spectrum of extrarenal pathology in the pedigree should certainly direct the diagnostic search to the study of metabolic status and, above all, to the exclusion of dyspurinosis. Despite the paucity of clinical symptoms

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of urate nephropathies, careful comparison of a number of signs is undoubtedly important for diagnosis (Table 1).

As can be seen from Table 1, urate nephropathy is characterized by the actual absence of extrarenal symptoms (edema, hypertension) in the presence of isolated urinary syndrome (IMS), detected against the background of intercurrent diseases and often accidentally. With the layering of microbial - inflammatory pain syndrome, dysuria and other signs.

Table 1

Some clinical and laboratory parallels in patients with glomerulonephritis and urate nephropathy (frequency of the sign, g)

	Urate nephropathies		
Signs	**ChGN(п = 32)**	**IUS (и = 21)**	**IN + PN (п = 65)**
The age at the time of detection is up to 3 years	-	14 (0,67)	31 (0,48)
4-7 years	7 (0,22)	5 (0,24)	25 (0,38)
8-14 years	25 (0,78)	2 (0,09)	9(0,14)
Conditions that contributed to the detection of the disease			
(ARVI, pneumonia, etc.)	29 (0,9)	14 (0,67)	42 (0,65)
Accidental detection	3	7(0,33)	23 (0,35)
Attitudes towards intercurrent diseases of diseases	The interval is 2-3 weeks	Against the background of intercurrent	
Prevalence of nephropathies in pedigrees	1,0%	7,1%	7,3%
Extrarenal manifestations of Edema	30(0,91)		
Hypertension	7 (0,22)	-	-
Hypotension	-	5(0,24)	14(0,22)
Pallor	32(1,0)	2 (0,09)	42 (0,65)
Enuresis	-	-	7(0,11)
Dysuria	-	7 (0,33)	20 (0,31)
Abdominal syndrome Urinary syndrome:	-	6 (0,29)	17(0,26)
Proteinuria (%)	0,33- 1,65	0,066	0,099
Leukocyturia (in n/a)	10-150	10-22	15-30
Hematuria (in n/a)	5-10-30	5-8-10	10-20

Diuresis in patients with urate nephropathy, as a rule, is significantly reduced (P<0.001), slightly higher in the presence of an interstitial process (570.4±12.4 and 610.6±15.4, respectively, P<0.01) (Table 2).

The daily excretion of urates is 2.5-3 times higher than normal (respectively 5.63±0.46-6.5±0.56 mmol / day at a rate of 2.94± 0.13 mmol / day). In 1/3 of patients with urate nephropathy, hyperoxaluria is also observed (0.702±0.051, P<0.001, with a norm of

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0.413±0.05 mmol/day). Urinary excretion of calcium and phosphorus is slightly increased (P<0.05). Creatinine clearance and water reabsorption in the renal tubules were not significantly changed (P>0.05), however, tubular phosphorus reabsorption was reduced compared to the norm (65.4±9.7% and 52.6± 11.2% with a norm of 88.2±17.4%P<0.05).

Oxalate coefficients: creatinine were significantly increased (0.152±0.03-0.157±0.013 at a norm of 0.053±0.05, P<0.001), the ratio of urine urates to creatinine was 2.6-2.76±0.41 at a norm of 0.83±0.08 (P<0.001). A decrease in the ammonio-acidogenetic function of the kidneys is characteristic: both in the early stage of urate nephropathy (36,0x4,6 and 34.6±6.5 mmol/day), and especially with the development of IN and stratification of PN (P<0.001). The noted excess of neurotoxic metabolites in biological fluids, in addition to their direct nephrotic effect, creates an additional load in all departments of the nephron, irritate the entire urinary tract the system depletes their compensatory capabilities.

Discussion. The significant prevalence of CHI diseases turns this problem from a purely nephrological one into a general pediatric problem: almost every district doctor has to deal with their diagnosis and treatment [4,7]. The data obtained confirm that the onset of dysmetabolic nephropathies is manosymptomatic, the course is relatively favorable. However, in the absence of early diagnosis, correction of dysmetabolism and treatment of complications, the mechanisms of chronization and nephrosclerosis (IN, microbial-inflammatory process, oxidative stress, activation of cancer, membrane destruction) continue to operate with an outcome in chronic renal failure, which corresponds to the literature data [4,8]. If the importance of metabolic disorders in development and progression is undeniable today, then taking into account the prevalence of secondary DMN [up to 32 - 120 per 1000 children in the population], their social significance rises to its full height [11,19].

Consequently, urate nephropathies belong to the category of common but difficult to diagnose diseases, and therefore require increased alertness. The general principles of the treatment of urate nephropathies, along with diet, with the exclusion of foods rich in purines and stimulation of diuresis (high-liquid regime), are the appointment of antioxidant and membrane stabilizing agents (vitamin A, E, dimephosphone), enterosorbents (pterosgel, activated charcoal, alyserb), drugs to normalize calcium metabolism (ksidiphon).

Table 2

Features of urine composition and partial renal function in urate nephropathies (M±w)*

Indicators	Health (и = 27)	**IUS** (” = 21)	IN + PN (п = 65)
Diuresis (ml/day)	884,7±22,4	570±12,4 P <0,01	610,6±15,4 P<0,01
Osmolarity (mosm/kg)	880 ± 41,5	771,0±65,7 P<0,05	570,0±57,5 P<0,01
Calcium (mmol/day)	61,3±1,5	64.3±1,2 P>0,05	70,0±1,9 P<0,01
Inorganic phosphorus (mmol/day)	458,7 ±6,8	570±7,0 P<0,01	610,0±5,4 P<0,001
Ammonia (mmol/day)	46,5±8,3	36,0±4,6 P>0,05	22,0±4,5 P<0,001
Titrated acids (mmol/day)	45,4±2,8	34,6±6,5 P<0,05	18,2±7,9 P<0,001
Urates (mmol/day)	3,16±0,38	5,63±0,5 P<0,01	6,5±0,6 P<0,01

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Oxalates (mmol/day)	0,36±0,04	O,57±O,O5 P<0,05	0,74±0,06 P<0,01
Clearances (ml/min/1.73 m2):			
Calcium	0,82±0,04	1,8±1,3	2,9±1,3
Phosphorus	13,4±0,8 P>0,05 P<0,001	16,0±43	18,1±5,0
Water reabsorption (%)	98,6±0,06	97,9±0,11 P>0,05	97,6±0,10 P>0,05
Tubular phosphorus reabsorption (%)	88,2± 17,4	65,4±9,7 P<0,01	52,6±11,2 P<0,01
Coefficients:			
Urates/ Creatinine	0,83±0,08	2,76±0,41 P<0,01	2,6±0,45 P<0,01
Oxalates/ Creatinine	0,053±0,005	0,152±0,03 P<0,01	0,157±0,013 P<0,001
Calcium/ Creatinine	0,12±0,003	0,15±0,003 P>0,05	0,15±0,003 P>0,05

P - the reliability of the difference compared to healthy

Patients with uraturia and urate nephropathies need constant dispensary supervision and require special attention during the period of heat stress and with any intercurrent infection. Pathogenetic treatment for non-correctable hyperuricemia diet therapy is based on the suppression of uric acid synthesis (MC) by uricodepressants. Allopurinol, an inhibitor of the enzyme xanthine oxidase, synthesis of MK, has become the most widespread. It is prescribed at the rate of 5-10 mg / kg (children under 6 years of age 0.15, over 6 years of age 0.2) once a day during the period of the highest concentration of MC in the blood, i.e. in the morning, the duration of treatment can be 3-6 months [16].

The consistent use of drugs that improve the solubility and excretion of urates (phytolysin, normurate, uralite, magurlite, kansfron) is of great importance. The remaining measures depend on the stage of evolution of the disease. One of the most common mistakes in outpatient practice is the restriction of prescribing only antibacterial treatment with an established diagnosis of PN. Meanwhile, it is known that the presence of PANIN, GN already means the presence of oxidative stress with the accumulation of by-products (superoxide anion - O2, hydrogen peroxide, etc.), which contribute to the progression and chronization of renal pathology [13]. At the same time, changes in the components of lipid peroxidation (POL) and the body's antioxidant defense (AOP) occur at the earliest stages of the development of kidney pathology [18]. Therefore, the treatment complex usually included vitamin E at a dose of 2 mg / kg body weight and vitamin A at 1000 units per year of a child's life.

Conclusion. Thus, urate nephropathies have a significant share in the structure of nephropathies. In clinical diagnosis, it is important to study the pedigree (identification of the uricopathic spectrum of pathology), clinical features (early age, absence of extrarenal signs)

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and biochemical parameters (uric acid in blood and urine). Patients with urate nephropathy need constant medical supervision.

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