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CLINICAL AND LABORATORY DIAGNOSIS OF URICOSURIC NEPHROPATHY IN CHILDREN

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Objective
To study the clinical, genetic and laboratory characteristics of nephropathy in children against the background of hyperuricemia, hyperuricosuria .

Materials and Methods of Examination
The genealogy of 70 patients with nephropathy on the background of hyperuricemia, hyperuricosuria at the age of 1-15 years was studied, including 28 boys and 42 girls.

Results
The development of kidney disease was analyzed in 70 children with advanced nephropathy on the background of hyperuricemia and hyperuricosuria . The analysis showed that all patients were treated in the early stages of health care with a diagnosis of glomerulonephritis, pyeloneritis, or urinary tract infection, indicating that treatment was incorrect.

Conclusion
Hyperuricemic nephropathy is very common in pediatric practice, but despite the presence of biochemical markers, it is rarely diagnosed . Children with purine diathesis have been shown to be at increased risk of developing hyperuricosuric nephropathy during the summer due to hot climates and hyperinsulation. Early diagnosis of hyperuricemia is important in the treatment, prevention, and treatment of ureteral nephropathy.

Keywords: hyperuricemia, hyperuricosuria, pediatric nephropathy.

Introduction

Protecting children's health is one of the issues of strategic importance as children determine the future of the nation and the state. At present, the impact of adverse environmental factors, unfavorable socio-economic conditions has already exceeded the adaptability of the human body.

High morbidity rates in the pediatric population have long been not only a medical problem but also a serious socio-economic and demographic problem (GG

Onishchenko et al., 2001). Diseases previously detected or developed during adolescence are now being detected in young children and even newborns.

According to the WHO, urinary tract disease (UTI) is the second most common childhood pathology. In the last decade, the incidence of STK in children has increased 2.5-3 times. The increase in the frequency of chronic transition of these diseases is one of the current problems of modern pediatrics.

Dysmetabolic nephropathy (DMN) is a common disease in childhood (JI.A. Krivtsova,). DMN is one of the causes of the development of chronic pyelonephritis, interstitial nephritis (IN) and urolithiasis.

The prevalence of DMN and STK in environmentally unfavorable areas is considered to be one of the leading diseases (AD Tsaregorodtsev; MS Ignatova) Other authors emphasize the increase in endogenous factors in the development of this pathology (VA Tabolin et al., NA Dogadina) . Some researchers consider DMN to be a polygenetic hereditary membranopathy that occurs under the influence of adverse environmental factors (Yu.E. Veltishchev et al.).

The presence of early stages of DMN without clear clinical manifestations necessitates the need to actively identify risk groups and children with early signs of disease in order to take preventive measures.

At the same time, there is no doubt that there are real opportunities and prospects for successful primary prevention of chronic somatic diseases from childhood, the basis of which is the timely detection of hereditary predisposition - diathesis. One of these areas may be the timely detection and correction of uric acid (purine) diathesis based on impaired purine metabolism, and biochemical signs include increased levels of uric acid in the blood (hyperuricemia, GU), increased urinary excretion (hyperuricosuria, GUU) . The level of uric acid in the blood is shown to be related to the level of triglycerides (3). Among other conditions (dyslipidemia, insulin resistance), hyperuricemia is an important component of metabolic syndrome (MS) (3,6,7). The association of GU with cardiovascular and metabolic diseases has been proven in many randomized trials. In recent years, due to the increase in STCs, the study of hyperuricemia is gaining attention: arterial hypertension, type 2 diabetes, cardiovascular disease, gout, obesity, etc., 2/3 of children with hyperuricemia regardless of weight is the basis of MS (8,12,26). Other mechanisms that are more important than the deposition of urate crystals in the development of renal injury in hyperuricemia include the suppression of renal mechanisms of fibrinolysis, the development of general endothelial dysfunction, and the stimulation of the renin-angiotensin-aldosterone system (15). GU is associated with impaired metabolism of fats (hypertriglyceridemia) and carbohydrates. GU and hyperinsulinism (or insulin

resistance) are considered interrelated processes (7, 8,15). GU in children is mainly a hereditary trait that reflects the state of purine base metabolism. Therefore, the primary prevention of conditions that occur as a result of impaired purine metabolism occurs precisely in childhood (18).

The Purpose of the Work

In children study of clinical ogenetic and laboratory features of nephropathy developed against the background of hyperuricemia, hyperuricosuria .

Methods of Control

In the study The genealogy of 70 patients aged 1 to 15 years with hyperuricemia, nephropathy developed against the background of hyperuricosuria was studied, including 28 boys and 42 girls. Renal glomerular function was assessed by endogenous creatinine clearance by the method of calculation using the Schwartz formula: Ccr (vk / vby / 1? 73m2) = K x Height (cm) / Scr where K is the coefficient of age, Scr is the concentration of creatinine in the blood (mmol / l). Phosphorus reabsorption was determined by Nordin and Fraser. Quantitative determination of oxalates in urine was carried out by NV Dmitrieva, Mueller-Seifert method of uric acid in blood and urine. The role of heredity in the occurrence of SKD was investigated using the Falconser static method. The significance of the difference was assessed using Student's t-test.

Results and Discussions

A study of the development of kidney disease in 7 0 children with nephropathy on the background of hyperuricemia (G U) and hyperuricosuria (G UU) showed that prior to this examination, all patients were diagnosed with the following diagnoses in primary care : glomerulonephritis 2 7 , 2 %, pyelonephritis diagnosis was 59 % , urinary tract infection was 13.8 % , and these patients were not adequately treated. The age distribution of patients was as follows: under 4 years - 57.8 % , under 5-7 years - 29.7%, under 8-15 years - 12.5%, of which boys - 32 (45.7%), girls - 38 (54.3). %). The sum of the leading clinical symptoms was urinary syndrome: protenuria (protein traces 0.165%), hematuria and leukyturia, erythrocyturia, macrohematuria in 1 in 7 patients under observation (2 4.28 %), dysuria ya 9 (pain during urination, 12.8 %) . Swelling, hypertension was not observed, 1 9 children (2 7.1 %) had a tendency to hypotension. Retrospective observations showed that 21 pregnancies (30%) had maternal toxicosis and 33 out of 70 (4 7 %) had severe toxicosis. When the genealogy was analyzed , information was obtained about 1,278 relatives . Of them, 317 relatives

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of I degree (parents and birth relatives) , 742 people of II degree, 219 people of III degree . Overall, hereditary family tree analysis revealed that 38.2 % of patients had various pathologies , including 33.8 % urinary tract diseases, urolithiasis ; 18.8% - diseases of the liver and biliary tract ; 4 8 % - diseases of the cardiovascular system (including hypertension-6 2 %, obesity, diabetes-1 8 , 4 %, allergic diseases- 5 .7 %, neurological diseases-1 3.9 %) .

1- Genetic predisposition to kidney disease in family tree analysis The Falconcer coefficient was calculated to be 70.0 ± 0.39%, including 7 6 % for parents and 5 4 % for siblings . In families with children with urate nephropathy, hereditary predisposition is urolithiasis (6 3.2 %), allergic diseases (5 6.4 %), urinary-salt diseases (7 3 %), liver and biliary tract (5 4 %). Families with uric acid diathesis in a child with metabolic (urate) nephropathy have a high coefficient of hereditary predisposition to hyperuricemia-related diseases: 5 6 to 7 9 %. Because these diseases are multifactorial in nature, their incidence is 2 4 -4 9 %, and it is important to identify environmental risk factors that provide a real opportunity in their primary prevention

Table -1. Laboratory findings for uricosuric nephropathy in children (M ± m)

Indicators	Healthy (n = 16)	SKD	R	Urate nephropathy	R
In the urine: Diuresis (l)	0.9 2 ± 0.3 4	0.6 3 0 ± 0.4 3	<0.001	0.6 35 ± 0.4 3	<0.05
also urinate Uric acid (mmol / l)	2.94 ± 0.27	5, 5 ± 0.02	<0.001	6, 2 ± 0.4	<0.05
Oxalates (mmol / l)	0.413 ± 0.059	0.48 ± 0.06	> 0.05	0.70 3 ± 0.051	<0.001
Calcium (mmol / l)	1.46 ± 0.12	1.9 ± 0.2	> 0.05	2.5 1 ± 0.18	<0.05
In the blood : Total protein in the blood (g / l)	72.0 ± 2.0	70.0 ± 1.4	> 0.05	6 5 , 0 ± 300	> 0.05
Cholesterol mmol / l	4.84 ± 0.4	6, 3 ± 0.8 3	<0.05	6.4 1 ± 0.8 4	<0.05
ASL -O (e d)	0.200 ± 0.05	0.1 78 ± 0.05	> 0.05	0.220 ± 0.03	> 0.05
EChT	7.1 ± 0.6	8.0 ± 0.6	> 0.05	14.2 ± 1.1	<0.05
Uric acid (mmol / l blood)	0.231 ± 0.04	0.3 12 ± 0.08	<0.001	0.335 ± 0.08	<0.05

1-as can be seen from the table, the presence of P D is confirmed by the presence of GU (0.312 ± 0.08 and 0.335 ± 0.08 mmol / l at 0.231 ± 0.04 mmol / l, p <0.001) and GUU () in children. 5.5 ± 0.02 mmol / s at a rate of 2.94 ± 0.27 mmol / s, p <0.001). Children with PD are characterized by oliguria (p <0.001). According to age -related physiology , there is a lot of information about the effect of high temperature and hyperinsulation on metabolic processes in the growing young organism.

However, studies have not shown that children with nephropathy in P D have adaptive properties to high temperatures . We conducted seasonal studies of renal function and urine composition in children aged 6–1 5 years to determine the effect of heat load on children with nephropathy in the background of PD .

The hottest months were June, July, and August, with average monthly maximum temperatures ranging from 39.2 to 44.2 ° C. The number of sunny hours is 2,976, of which 72% fall in the months from May to October. A sharp decrease in relative humidity began in April and fell to a minimum in June (average 40 %) , during which time relative humidity was observed to decrease from 1.89 % to 13% at noon . In the summer heat, the air temperature in the buildings rose from 28 to 36 ° C , relative humidity decreased from 12-3 to 3 % .

To achieve comparable results in all seasons, we recommended that children eat the same diet according to their age. Urine was taken within 24 hours for the study, and blood was tested on the same day. No additional water loading was performed. C. The amount of daily urine in young children varies precisely depending on the season: diuresis during the period of maximum heat load is 6 5.6 % in winter . In the same comparison, the endogenous creatinine clearance was 7 3.2 %. Urinary excretion of uric acid, oxalates, phosphates increased significantly, calcium excretion decreased moderately. In healthy children, these changes are seen as a manifestation of the functional state in summer , while in healthy children we see them as a manifestation of functional metabolic adaptation to heat stress in summer.

By adhering to optimal hygiene standards (microclimate conditions, drinking regimen), they are easily tolerated by healthy children and do not manifest themselves clinically. The effectiveness of homeostasis mechanisms during adaptation to high external temperatures depends on the initial characteristics of the child's metabolic state. Thus, in patients with metabolic nephropathy on the background of PD in all seasons, daily urine output and glomerular filtration were low . Urinary excretion of urate, oxalates, and phosphorus was increased in these children compared to healthy children.

Thus, differences in metabolic shifts in healthy children and observant children with metabolic nephropathy (P D, urate li nephropathy, interstitial nephritis, secondary dysmetabolic pyelonephritis) under conditions of high external temperature and hyperinsulation indicate the presence of endogenous causes of disadaptation in patients. The characteristic spectrum of hereditary pathology in patients with PD includes the properties and interactions of different levels of uric acid , including the nervous system, connective tissue, immune system, etc. is denoted by,. urate oxalates, phosphorus in the urine against the background of a decrease in diuresis leads to an

additional load on all parts of the nephron, reducing their compensatory capacity . Given that the incidence of PD in the pediatric population is 3–5% and hyperuricemia 1 2 –1 8%, early detection of children with G U , targeted monitoring, and establishment of a therapeutic diet and phytoprophylaxis are of practical importance. In this regard, a certain psychological attitude is necessary not only for the child himself, but also for his parents, as they often underestimate the importance of these measures.

Conclusion

1. Uric uric (urate) nephropathy is a common, but rarely diagnosed, disease in pediatric practice in the presence of clinical, genetic, and biochemical signs .
2. In summer, heat load and hyperinsolation uric acid diathesis are real risk factors for the development of uricosuric nephropathy in children.
3. Timely detection of hyperuricemia in the prenatal period provides a real opportunity to prescribe a complex diet and medication correction of its level and primary prevention of the development of urate nephropathy.

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