NOVATEUR PUBLICATIONS
JournalNX- A Multidisciplinary Peer Reviewed Journal
ISSN No: 2581 - 4230
VOLUME 10, ISSUE 01, January -2024

URATE NEPHROPATHY'S CAUSES AND CLINIC LABORATORY MANIFESTATIONS IN CHILDHOOD PERIOD

Karimova B. N.
Uzbekistan, Tashkent Medical Academy
99897-722-25-02 barnoshakbn@gmail.com

Abstract

Urate nephropathy in children is based on increased production of uric acid and, as a result, an increase in its concentration in the released blood and urine. According to various authors, this violation is recorded in 3-17% of the examined people. Environmental factors, urbanization, nutritional patterns can lead to the spread of such disturbances in the purine exchange. Urate nephropathy often proceeds covertly and it is detected by chance. This pathology is diagnosed in the stage of nephrosclerosis with slowly progressing nitrogemia and pronounced arterial hypertension and in climatically unfavourable seasons of the year when adapted. Such variants of dysmetabolic nephropathies are known econephropathy [1].

Violation of purine metabolism is quite widespread, and, according to various researchers, is recorded in 7-17% of the examined people. Currently, there is a certain tendency to increase this type of metabolic disorders, primarily due to the increased influence of environmental factors, such as the accumulation of excess lead in the body, as well as an increase in alcohol consumption, which can lead to the spread of urate dysmetabolism, which is population-based. In this regard, the problem of early diagnosis of this type of metabolic disorders, especially often manifested by kidney damage, becomes urgent. Early detection of urate nephropathy allows stopping its progression, development of nephrosclerosis and terminal renal failure [1, 2, 4].

Urate nephropathy, depending on the stage of progression, can be transformed into urate interstitial nephritis, urolithiasis, glomerulonephritis, CPN. In secondary urate nephropathy on the basis of purinosis, differential significance is given to the presence of gout in the pedigree of the patient, urolithiasis, essential arterial hypertension, "metabolic syndrome," fatty people, type 2 diabetes mellitus, idiopathic CKD, attacks of "acetonemic vomiting," etc. Many transformations of urate nephropathy, namely: urate interstitial nephritis, urolithiasis (urate), glomerulonephritis (more often against the background of gout), CPN (more often against the background of gout, interstitial nephritis), can be determined by other conditions, for example, immune factor, renal microanomalia, renal blood flow condition and In practice, there are difficulties in diagnosing situations where metabolic and morphological changes in the kidneys, in some cases, are a clinical manifestation of purinosis, in others - a consequence of renal tubulopathy. In these patients, the correction of metabolic disorders should follow the pathogenetic principle and the definition of diagnosis as urate nephropathy will not contradict the essence of clinicomorphological changes.Clinically, urate nephropathy (UN) is often quite modest [3, 5].Urinary syndrome is detected accidentally and manifested by isolated crystalluria (urate) or in combination with microproteinuria (sometimes moderate), microleucocyturia and microhematuria [5]. The urine persistent abrupt reaction is pH 4.5-5.5, at a rate of 7.4-7.5. In the blood - hyperuricemia of varying degrees of severity. There may be manifestations of disuria, pollakiuria, urinary rhesi (phenomena of urate cystitis). In some cases, especially in young children, orange crystals

of uric acid, or an orange rim on the walls of a night pot, can be visually detected in settled urine. In cases where, along with the above-described urinary syndrome, partial disorders of nephron function appear in the form of a violation of osmodiuresis, titrated acidity of urine, hypoisostenuria, the development of metabolic (urate) interstitial nephritis [1, 2, 4, 6] is not excluded.

In this regard, an urgent problem is the early diagnosis of this type of disorder, which will stop its progression, the development of nephrosclerosis and terminal renal failure.

The aim of the study is to identify urate nephropathy in children in the early stages.

Materials and methods. An analytical study of 720 medical histories and outpatient maps (form 112-U) of children aged 5-12 years performed, with selection of study subjects from 46 patients with recurrent uraturia. Of these, 26 are girls, 20 are boys. We divided these children into two groups: 1-group 20 children from 5-7 years old, 2- group 22 children from 8-10 years old. The control group was 20 children who, according to medical records, had a first or second health group with no urinary system pathology. In parallel, 20 mothers with family history burdened by disease were examined and observed. The work done at the Tashkent Medical Academy. The examination of patients carried out based on the nephrology department and polyclinic. A genealogical history was analyzed with the identification of leading risk factors, a history of the course of pregnancy, childbirth, the incidence and nature of feeding children in the first year of life, the nature of nutrition and water-salt regime during life in the families surveyed, suffered and comorbid diseases, as well as the peculiarities of the clinical picture. All children underwent a comprehensive clinical-laboratory and instrumental examination, biochemical blood analysis, urine pH. The functional state of the kidneys assessed according to the results of a Zimnitsky sample, a dry-eating sample, clearance of endogenous creatinine, and serum urea levels. All patients underwent ultrasound examination of the gastrointestinal tract, kidneys and bladder.Study results and discussion. It established that in children from families with a history aggravated by diseases of impaired metabolism, urate nephropathy occurs in 91% of cases. The incidence of kidney disease in relatives is generally higher than in the control group, with maternal kidney and urinary tract diseases occurring 2 times more frequently than in the father. A survey of 20 mothers revealed that half of them had an increase in blood pressure (70%). 55% of women were diagnosed with chronic gastroduodenitis, 15% with chronic cholecystitis, and according to the results of ultrasound, 5% of them first showed bile stone disease, in 5% of mothers - urolithiasis.

We have determined that disruption of salt metabolism in the form of an increase in uric acid in the blood and its increased excretion with urine in a number of children is associated with their early transfer to artificial and mixed feeding. Risk of hyperuricosuria in children on artificial feeding increases by 2.5 times

With the age of children, the nature of nutrition in the families surveyed also changes. There is an abuse of food rich in preservatives, which increases the risk of urate nephropathy by 10 times.

The clinical picture of preschool children dominated by complaints of pastosity of the eyelids, dysuric disorders (72%), precipitation of salts in the urine (66%), increased sweating, increased nervous excitability (37%), sleep disturbance in the form of difficult falling asleep, night fears (22%). Arthralgia, mainly at night, were recorded in 27%, myalgia in 17% of children, acetonemic vomiting in 7% of children.

With age, the number of complaints among children 8-12 years old decreases. The most common complaints were of abdominal and lumbar pain (62%). Increased nervous excitability, emotional

lability (45%), sleep disturbance (33%), decreased appetite (57%) were detected. For the first time, an increase in blood pressure recorded in 7% of the patients examined.

In general blood tests, eosinophilia was significantly more common in the children examined than in the comparison group. The main and most significant symptom of urate nephropathy in children is hyperuricosuria. Most of the children tested (80%) showed an increase in urinary uric acid levels from 800 to 1000 mg/day. Combined urate-oxalate crystalluria was significantly more common in children aged 6-8 years. On average, 85% of children showed a sharply acidic urine reaction in general analyses. During crystallographic examination, urates (67.7%) and sodium urates (47.5%) were most often detected in an open drop of urine, mainly large crystals, uric acid crystals (25%) of various sizes prevailed.In 69.9% of children, protein was traced as a thin rim along the edge.

Conclusion: It was established that the main risk factors for urate nephropathy are: aggravation of hereditary history in the maternal and paternal pathology of metabolism, early artificial feeding, violation of the water and salt regime. New risk factors have been identified: abuse of food rich in preservatives. Lowering urine pH less than < 5.75 is an independent risk factor for developing urate nephropathy in children.

References

1. Ignatova M.S., Lengn V.V. Hereditary kidney diseases occurring with hematuria. Russian Journal of Perinatology and Pediatrics 2019; 3: 59: 82–90.
2. Length V.V., Ignatova M.S., Osmanov I.M. and others. Dysmetabolic nephropathies in children. Russian Journal of Perinatology and Pediatrics 2018; 58: 5: 36–45.
3. Novikova N., Kovalchuk M., Stepina N. et al. Distinct effect of xenobiotics on the metal-binding properties of protein molecules. J Synchrotron Rad 2015: 22: 1: 1001–1007.
4. Parish L.S., Length V.V., Ignatova M.S. Glomerulonephritis primary. Pediatric nephrology. Ed. M.S. Ignatov. M.: Honey. Heart attack. Agency, 2011; 263–297.
5. Yurieva E.A. SuhorukovV. Page, A.D. Tsaregorodtsev, etc. Alteration of protein molecules in endogenous body intoxication as a risk factor for chronic metabolic diseases. Molecular Medicine 2018; 3: 45–52
6. Yurieva E.A., Yablonskaya M.I., Raba G.P. and others. Pathogenetic aspects of urinary (purine) diathesis in children. Russian Journal of Perinatology and Pediatrics 2017; 58:4:40–46.