

METABOLIC BASIS OF PREVENTION OXALATE NEPHROPATHY AND URINARY SYSTEM INFECTION IN CHILDREN

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Annotation

All over the world diseases of the urinary system are becoming one of the urgent problems. According to the World Health Organization, complications of kidney disease are of great importance for the public health system in all countries, depending on the place of residence, lifestyle, gender, age, nutritional status, under the influence of environmental factors, there is a risk to the lives of children and adults. In this regard, the problem of early diagnosis, the appointment of adequate dietary and drug therapy is relevant.

The article deals with the problem of etiology, pathogenesis, as well as the criteria for diagnosing dysmetabolic nephropathies in children. Attention is paid to the clinical manifestations of this pathology and the basic principles of treatment and prevention.

Keywords: children, dysmetabolic nephropathy, crystalluria, oxalaturia.

One of the urgent problems of pediatrics at the present stage is the diseases of the urinary system (OMS). Epidemiological studies conducted at the turn of the 20th-21st centuries showed that the prevalence of CMI pathology varies from 60:1000 to 187:1000 children in the child population, depending on the environmental situation in the area where the child lives [1,7,8]. At the same time, diseases of congenital and hereditary origin, having a latent onset and a torpid course, prevail in the structure of the pathology of OMS, among which a large proportion are metabolic, dysmetabolic nephropathies (DN).

Dysmetabolic nephropathies are understood as a large group of nephropathies with different etiology and pathogenesis, but united by the fact that their development is associated with metabolic disorders. Metabolic pathology leads to changes in the functional state of the kidneys or to structural changes at the level of various elements of the nephron. Dysmetabolic nephropathies in a broad sense combine diseases associated with severe disorders of water-salt metabolism that develop in gastrointestinal diseases with a toxic syndrome and hemodynamic disorders. These may include kidney damage occurring against the background of disorders of phosphorus-calcium metabolism in hyperparathyroidism, hypervitaminosis D and other diseases. The term "dysmetabolic nephropathy", used in a narrow sense, denotes

a polygenically inherited (multifactorially developing) nephropathy, which is associated with impaired metabolism of oxalic acid and manifests itself in conditions of family cytomembrane instability.

Healthy children excrete in the urine single small salt crystals (most often these are oxalates and tripelphosphates) of 0.03-0.055 microns in size, which do not cause damage to the renal tissue. It is believed that a damaging effect on the organs of the urinary system is possible if there are more than 10 crystals in the urine sediment in the field of view and if their size is more than 12 microns. Three fundamental factors play a role in the process of crystal formation: supersaturation of the tubular fluid beyond the limits of its stability, a decrease in the activity of supersaturation inhibitors, and the presence of precipitation activators.

Crystalluria is a variant of the urinary syndrome, in which, according to the results of laboratory tests, an increased content of salt crystals is found in the urine. In the daily practice of a doctor, this symptom is observed in almost every third child. The proportion of crystalluria in the structure of pediatric nephrological pathology exceeds 60% [1]. The most common is oxalate and oxalate-calcium crystalluria, which accounts for 75.0–80.0% [1,2,13].

For the formation of a crystal, the presence of an ion pair - an anion and a cation (for example, a calcium ion and an oxalate ion) is necessary. Oversaturation of urine with different types of ions eventually leads to their precipitation in the form of crystals and their subsequent growth. A huge role is played by the dehydration of urine, which entails an increase in the concentration of ions in the urine, even with their normal production. In addition to the degree of saturation, the solubility of ions is affected by ionic strength, ability to complex formation, urine flow rate, urine pH [14,17].

The problem of sporadic dysmetabolic nephropathies is very relevant in pediatrics and pediatric nephrology. This is due to the high frequency of the disease in the population, as well as the possibility of its progression up to the development of urolithiasis and/or interstitial nephritis.

It has been established that intermittent calcium oxalate crystalluria, detected in childhood and adolescence, leads to the progression of tubulointerstitial disorders in adults and an increase in the frequency of a mixed variant of the urinary syndrome, characterized by severe proteinuria, hematuria, signs of membranous changes of the tubular epithelium of the kidneys, functional and structural changes in the kidneys and bladder [5].

Among the DN associated with impaired water-salt, carbohydrate, phosphorus-calcium and other types of metabolism, special attention, due to the highest prevalence, reaching 20% of the total number of OMS pathologies, is drawn to oxalic acid metabolism disorders, the so-called dysmetabolic nephropathy with oxalate - calcium crystalluria (DN with OKC) [1,2,5]. The variability in the prevalence of DN with OCC, according to various authors, is due to differences in the environmental

situation in the area where children live and can reach 31.4% in preschool children [1,3,5].

The progression of oxalate nephropathy often leads to the development of abacterial interstitial nephritis (IN), and as a result of the addition of a secondary infection, pyelonephritis develops. The most pronounced dysmetabolic disorders can cause the occurrence of urolithiasis (UCD), even in the first years of a child's life [3,12,15].

Currently, the prevalence of crystalluria in the child population in non-endemic areas is 32%, and in environmentally unfavorable areas it reaches 47%; 68-71%, urate 9-15%, phosphaturia 9-10% and others from 3 to 5% [9].

One of the most important scientific and practical areas of social pediatrics and healthcare organization is a regional approach to studying the state of children's health. Its formation is influenced by climatic, geographical, environmental and economic living conditions, the degree of population migration, ethnic and sociocultural characteristics, as well as significant differences in the material and technical base of medical institutions by region.

According to some experts, dysmetabolic nephropathy with calcium oxalate crystalluria is considered as a model of an eco-dependent disease [11].

Pushkareva E.Yu. when studying the clinical and pathogenetic features of the formation and mechanisms of progression of dysmetabolic nephropathy in children, depending on age, she found that living in areas with a high anthropogenic load increases the chance of developing DN with OCC by 2 times. The author has proved that the use of filtered drinking water can be considered as a measure to prevent the development of calcium oxalate crystapluria in the child population of industrial areas [12].

The variability in the prevalence of dysmetabolic nephropathy with calcium oxalate crystalluria, according to various authors, is due to differences in the environmental situation in the area where children live and can reach 31.4% [7,8]. On average, in Russia, the prevalence of dysmetabolic nephropathy is 1.4:1000 of the child population and, due to the deterioration of the environmental situation, tends to increase [10].

Allocate endogenous and exogenous causes of oxalate dysmetabolic nephropathy in children. Endogenous causes include: increased oxalate biosynthesis, hyperuricemia, cystine metabolism disorders, phosphaturia, diabetes mellitus, vitamin metabolism disorders, ischemic nephropathy, electrolyte disturbances, hyperparathyroidism. Exogenous causes include dietary habits, drinking habits, ecopathogens (cadmium, lead, uranium, organic solvents, etc.), medicinal substances, climatic features of the region of residence. Persistent crystalluria should be considered a specific sign of impaired calcium metabolism at the cellular level; its presence is usually combined with salt diathesis [1,2].

The trigger mechanism for damage to tubulointerstitium is the crystallization of calcium oxalate in the tubules, due to its local toxicity and poor solubility [18]. Damaged cells of the renal epithelium actively bind to the crystals, inducing the

processes of regeneration and repair. Proliferating urothelial cells express “crystal-binding molecules” on their surface, which act as stimulators of crystal adhesion to the surface of epitheliocytes [20].

There are two etiopathogenetic variants of hyperoxaluria - primary and secondary. Primary hyperoxaluria is a hereditary disease that includes three rare types of genetically determined disorders of glyoxylic acid metabolism, which are characterized by increased oxalate excretion, recurrent calcium oxalate urolithiasis and/or nephrocalcinosis, and a progressive decrease in glomerular filtration rate with the development of chronic renal failure [2]. Oxalate-calcium crystals are deposited in all tissues of the body, which leads to oxalosis at the age of 10-30 years. The disease is inherited in an autosomal recessive manner, but cases of dominant inheritance are known. These forms are diagnosed using biochemical methods, their clinical manifestations are identical.

In pediatric practice, secondary or spontaneous hyperoxaluria is most common, which can be transient (with a monotonous diet, against the background of acute respiratory viral infections, intercurrent diseases) or permanent. There are several mechanisms of its development. Alimentary hyperoxaluria is associated with excessive consumption of foods containing

oxalic and ascorbic acids [13].

Risk factors for secondary hyperoxaluria include hereditary predisposition, which is observed in 70% of cases in children with hyperoxaluria. This is manifested not only by the pathology of oxalate metabolism, but also by a tendency to instability of cytomembranes [13]. In the genesis of membrane destabilizing processes, an important role belongs to the processes of intensification of lipid peroxidation, activation of endogenous phospholipases and oxidative metabolism of granulocytes. With the destruction of acidic phospholipids of cell membranes, oxalate precursors are formed.

Recently, the local formation of oxalates in the kidneys has been discussed in connection with the destruction of cell membrane phospholipids, resulting in the formation of oxalate precursors (serine), as well as phosphates, with which calcium forms insoluble salts [16].

The first manifestations of hyperoxaluria in children can be already in the first year of life. Most often, hyperoxaluria is recorded during periods of intensive growth of a child 7-8 and 10-14 years old. In most cases, the oxalate crystal Riya is found by chance, sometimes against the background of acute respiratory viral infections, intercurrent diseases.

Often, parents notice in a child a decrease in the volume of urine during the day, the precipitation of a large amount of salts. When questioned in children, recurrent abdominal pain is detected. Sometimes inflammation of the genitals develops due to constant irritation of the skin and mucous membranes, while urinating, there may be a burning sensation or other dysuric disorders. Against the background of crystalluria,

an infection of the urinary system is often formed. A visual assessment of urine indicates its saturated character, spontaneous sedimentation is possible. Hyperstenuria (urine relative gravity greater than 1030) in the absence of glucosuria should alert for hyperoxaluria. In the future, against the background of crystalluria, slight microhematuria and / or proteinuria, abacterial leukocyturia appear, which indicates damage to the kidneys and is referred to as “dysmetabolic nephropathy” [4]. Biochemical study of daily urine (transport of salts) allows you to clarify the presence of hyperoxaluria and hypercalciuria. The normal level of oxalates is less than 0.57 mg/kg/day, calcium is less than 4 mg/kg/day. To diagnose hyperoxaluria and hypercalciuria, one can also use the definition of such an indicator in a single portion of urine as the ratio of calcium to creatinine and the ratio of oxalates to creatinine [4]. In children with hyperoxaluria in nephrological hospitals, an analysis is carried out for the anti-crystal-forming ability of urine to calcium oxalate, which is reduced. The test for peroxide in the urine allows you to evaluate the activity of the processes of lipid peroxidation of cytomembranes.

An ultrasound examination of the kidneys in some children reveals echopositive inclusions in the pelvis and calyces.

For the prevention of DNOCK and calcium nephrolithiasis, dispensary observation of children from families with a hereditary predisposition to urolithiasis is recommended, with regular preventive treatment, including diet therapy, drinking regimen, vitamin therapy (A, E, B6) and other types of treatment, primarily herbal medicine .

Authors Dlin V.V., Ignatova M.S., Osmanov I.M., E.A. Yuryeva, S.L. Morozov (2015) prove that the observation of 130 children for 5 years showed the effectiveness of this rehabilitation scheme both in the treatment and prevention of recurrence of pyelonephritis and in reducing the severity of metabolic disorders in children.

Despite recent advances in the treatment of dysmetabolic nephropathy, the problem of improving treatment methods, preventing the most severe outcomes of the disease, and implementing effective preventive measures remains one of the most important in modern pediatric nephrology.

The versatility of the pathogenetic mechanisms of damage to the urinary system, the severity of the consequences caused by metabolic disorders, such as urolithiasis, pyelonephritis, etc. aim scientists to look for new modern technologies for the treatment and prevention of these diseases.

Thus, the study of risk factors and the main etiopathogenetic mechanisms for the formation of dysmetabolic nephropathy in children is of particular importance due to their high prevalence and serious prognosis.

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