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CURRENT VIEWS ON URATE NEPHROPATHY IN CHILDREN

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Abstract:

The problem of urate nephropathy in children in modern literature sources is covered rather rarely and contradictorily. There are many disagreements in the issues of terminology and pathogenesis. Based on the results of long-term scientific research, analysis of modern literature concerning purine metabolism disorders in children, the authors made an attempt to express their point of view on the above-mentioned issues.

Keywords: hyperuricemia, urate nephropathy, urolithiasis, interstitial nephritis, uric acid, urates, uric acid diathesis, crystalluria, children.

INTRODUCTION. In the literature, nephropathies that develop on the basis of purine metabolism disorders (in particular, it is a violation of sodium monourate dynamics) are referred to by the terms: uric acid nephropathy, uricosuric nephropathy, uric acid diathesis, uraturia-type dysmetabolic nephropathy, urate nephrolithiasis; in adults, when combined with gout, - gouty kidney, etc.

Increased excretion of uric acid through the kidneys occurs both due to increased filtration of this metabolite, (which is observed in hyperuricemia), and as a result of a decrease in its reabsorption in the proximal tubule. Most researchers consider the renal pathomorphosis that occurs in connection with hyperuric-cemia [1, 2]. For example, B.T. Emmerson and P.G. Row [2] believe that with excessive production of uric acid, the kidneys take on increased (adequately to the degree of its hyperproduction) urate excretion, thus maintaining the optimal level of serum MC. In doing so, there is a risk of tubule damage, which ultimately leads to a decrease in renal excretion function of uric acid and an increase in its concentration in the blood.

At the same time, there is a primary impairment of renal excretion of uric acid. This is consistent with the data indicating that urate reabsorption in the tubule is associated with an active enzymatic process, which can be influenced by such substances as saline and glucose, which have common transport mechanisms in the proximal tubule, as well as loop diuretics, beta-adrenoblockers, etc.

The morphophysiological essence of the indicated nosology is most likely the same — it is a violation of the dynamics of sodium monaurate in the kidneys due to various reasons (primary or secondary). And also, the deposition of poorly soluble urate salts in the interstitial of the kidneys (and their crystallization), with the development of reactive inflammation (subsequently, immune and bacterial) and impaired renal function. Since we are talking about a factor with causal (direct and indirect) significance — the crystallization of urates, it is advisable to combine all the above—mentioned synonyms of the name of the same disease with the definition: urate nephropathy (UN). The latter may be primary when, due to the failure of renal tubular functions, the reabsorption of urates is impaired; and secondary, developing due to nephron overload by hyperconcentration of uric acid in the blood serum (for example, in hereditary hyperuricemia — purinosis, chronic alcoholism, treatment with cytostatics, in athletes, etc.). The diagnosis of urate nephropathy can be indicated by a cipher from ICD-10 - under the code N 16.3.

Urate nephropathy, depending on the stage progression, it can transform into urate interstitial nephritis, urolithiasis, glomerulonephritis, CRF.

In secondary urate nephropathy on the basis of purinosis, information about the presence of gout, urolithiasis, essential arterial hypertension, "metabolic syndrome", obesity, type 2 diabetes mellitus, idiopathic CRF, attacks of "acetonemic vomiting", etc. in the pedigree of the patient is of differential importance.

Many transformations of urate nephropathy, namely: urate interstitial nephritis, urolithiasis (urate), glomerulonephritis (more often on the background of gout), CRF (more often on the background of gout, interstitial nephritis), can be determined by other conditions, for example, immune factor, renal microanomalia, renal blood flow, etc. In practice, it is difficult to diagnose situations when 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 the

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diagnosis as urate nephropathy will not contradict the essence of clinical and morphological changes.

Clinically, urate nephropathy (UN) often manifests itself quite modestly. Urinary syndrome is detected randomly and manifests itself in isolated crystalluria (urate) or in combination with microproteinuria (sometimes moderate), microleukocyturia and microhematuria [7]. A persistent sharp acid reaction of urine is important — pH 4.5-5.5, with a norm of 7.4-7.5. There is hyperuricemia of varying severity in the blood. There may be manifestations of desuria, pollakiuria, pain during urination (urate cystitis phenomena). In some cases, especially in young children, orange uric acid crystals or an orange rim on the walls of a chamber pot can be visually detected in the settled urine.

In cases where, along with the above-described urinary syndrome, partial violations of nephron function appear in the form of impaired osmodiuresis, titrated urine acidity, hypoisostenuria, the development of metabolic (urate) interstitial nephritis (IN) is not excluded. For any etiology, in addition to the above signs, an increase in the content of polar lipids in urine, increased urinary excretion of ethanolamine, phosphonic acids, lysine, B-lysinuria, betta-2-microproteinuria, the appearance of acetyl-betta-D-glucosoamidase (NAG), malate dehydrogenase (MDG), high phospholipase activity in urine (blood).

The stages of development of this process can be represented as follows: crystalluria (uraturia) microerythro (lympho)cyturia microproteinuria tubular fermenturia partial disorders of tubular kidney function increased hyperuricemia ^ increased urinary syndrome ^ impaired glomerular kidney function and the appearance of arterial hypertension ^ CRF.

In some cases, the clinical picture of UN is manifested by isolated glomerular proteinuria of varying severity (more than 1 g in urine per day), up to the development of nephrotic syndrome or mesangioproliferative nephritis. Urate nephropathy can also be complicated by the development of arterial hypertension, secondary pyelonephritis. The latter usually proceeds undularly, with little symptoms [3].

In cases where arterial hypertension (AH) is associated with severe hyperuricemia and urate nephropathy, its course can become persistent and severe, especially with the appearance of renal failure. Filtration disorder usually appears at the stage of CRF. With the progression of the disease, lower back pain may appear, as well as a clinic for renal colic, due to the development of nephro- or urolithiasis.

Uric acid stones with severe hyperuricemia, especially in gout patients, occur in 538% of cases [4], and in combination with the calcium oxalate form of urolithiasis - in 80% [5]. In about 20% of cases, stones in these patients consist of calcium oxalate and phosphate. However, in most cases, a central urate "core" is detected in stones of this composition [6] and this explains the decrease in the frequency of formation of calcium stones during treatment with allopurinol.

The morphology depends on the stage of urate nephropathy, its complications and clinical transformation. In far-reaching cases, diffuse fibrosis with rare infiltrates consisting of lymphocytes and macrophages is seen. Characteristic areas of urate deposition are the interstitial tissue of the medulla, pyramids or papillae, where they can be surrounded by lymphocytes and histiocytes. These deposits of urates are rarely found, as they are located very deep, mainly in the papillary zone. On autopsy, these deposits are found in all patients.

In severe urate nephropathy, the canal lesions are characterized by atrophy, dilation and focal regeneration of the epithelium of the Henle loops. The width of the cortical layer can be reduced. The cerebral layer and pyramids can also be reduced and contain white lines of uric acid deposits located horizontally. These deposits occur as a result of the concentration of urine occurring in these departments, when an oversaturated solution of uric acid salts is formed in an acidic environment. The precipitation of crystals of these salts is also observed in collecting tubes. At the same time, the tubule wall is damaged, and the fallen crystals penetrate into the interstitial tissue, accumulate in it and cause the development of interstitial nephritis [3]. Sometimes uric acid stones can be found in the pelvis [7].

Vascular damage is often found in the form of thickening, hyalinization of the arterial wall and the development of arteriosclerosis. Changes are also found in the glomeruli, in the form of thickening of the basement membrane and an increase in the size of the cytoplasm in endothelial and epithelial cells [3]. There are glomeruli that are anatomically normal and partially or completely hyaline.

Basically, such changes in the kidneys are observed in adult patients with gout. As for children, there is very little information about pathomorphosis in patients with urate nephropathy. I.A. Mukhin et al. [8] indicate that one of the most important aspects of kidney damage in gout is glomerulonephritis, the presence of which is confirmed by a number of lifetime morphological studies of the kidneys.

CONCLUSIONS: Thus, this glomerulonephritis is characterized by a latent course, with a predominance of proteinuria and hematuria (episodes of

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macrohematuria are not uncommon), and a steady progression towards chronic renal failure. There may be episodes of reversible renal dysfunction caused by transient uric acid blockade of the renal tubules, developing under conditions of dehydration and decreased diuresis.

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