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Study of partial kidney function in children of early age with nephropathy of metabolic genesis

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Abstract: 121 young children with pneumonia with dysmetabolic nephropathy, as well as 20 children with pneumonia with urinary syndrome, were studied. For these children, glomerular function was assessed by the clearance of endogenous formation, ammonia, titratable acidity, osmolarity of urine, and metabolites (oxalates, urates, uric acid). In children with pneumonia with dysmetabolic nephropathy changes in kidney partial functions, structural and functional states of erythrocyte cytomembrane as well as strengthening of lipid peroxidation were observed. In this regard, metabolic corrective therapy for these children was recommended.
Keywords: early age, urinary syndrome, dysmetabolic nephropathy.

Introduction Kidney diseases in children are a common pathology from 16.6 to 54: 1000 of the child population and, due to the frequency of the latent course, the tendency to chronicity, is an urgent problem in modern pediatrics. The widespread introduction of clinical genetic and biochemical research methods into clinical nephrology made it possible to establish a change in their nosological structure, an increase in multifactorically determined clinical forms [4]. Thus, according to epidemiological studies prevailing in the structure of nephropathy (34-40%) [2] are nephropathy exchange genesis, including 10-19% oxalate, 14-29% uranium nephropathy. At the same time, renal pathology manifests itself and is established in connection with pathologies of the respiratory organs - among newborns in the neonatal pathology department 34.2% [1], among children of early age being on treatment concerning various bronchopulmonary diseases 17-35 %, and at staphylococcal infections 78,6 %. Moreover, after elimination of the underlying disease, from 24.2 to 31.5% of children are discharged with residual urinary syndrome [3]. As a result of the above it becomes clear that the existing ideas about the urinary syndrome in children of early age, with the most widespread at this age bronchopulmonary pathology as a transient condition ("infectious kidney", "toxic kidney", "toxic-infectious kidney") contains a significant threat to the health of the growing organism. There are several reasons: first, it is known that the development of toxic conditions in general in children of early age (including those causing kidney damage) is not accidental and in their development play a significant role in the toxicosis of pregnant women, the presence of hereditary metabolic disorders, heredity burdened by nephropathy. Secondly, this initial manifestation is typical for the majority of hereditary, dysmetabolic and congenital kidney diseases with the most serious prediction [2,6]. In practice, even repeated episodes of urinary syndrome against a background of various intercurrent diseases continue to be evaluated as an infectious or toxic kidney. Meanwhile, modern methods of examining children and relatives, and the widespread introduction of genetic and biochemical research methods into nephrological practice make it possible to clarify the metabolic disorders that cause kidney damage with difficult to differentiate urinary syndrome [5].

The aim of this work was to develop the principles of an early differential diagnosis of metabolic renal lesions based on a comprehensive study of the state of partial renal function in young children with pneumonia with kidney damage.

Materials and Methods: Glomerular function was assessed by endogenous clearance on Van Slayke.

The state of tubular renal function was judged by the osmolarity of urine using the cryoscopic method on an OMK SH-01 apparatus, titratable acidity according to I. Todorov (1963).

The uric acid content in daily urine was determined according to the Mueller-Seifert method, based on the colorimetric determination of uric acid with Folin phosphoric tungsten reagent.

Quantitative determination of oxalates in urine was carried out according to N.V. Dmitrieva (1966), and the daily excretion of urates with urine was used by the Hopkins method in the description of O.V. Travina (1955).

Results and discussion: We studied partial renal function in 121 patients with pneumonia with dysmetabolic nephropathy (DMN) and 20 patients with pneumonia without dysmetabolic nephropathy (DMN). In all patients with pneumonia, there was a tendency to a decrease in daily diuresis, and in patients with pneumonia with DMN disorders it significantly decreased to 0.37 ± 0.013 compared with healthy children 0.450 + 0.038 ($p < 0.001$) and in patients with pneumonia without DMN 0.430 + 0.021 ($p < 0.05$). A decrease in glomerular filtration was found in patients with pneumonia with DMN to 0.97 + 0.17 ml/s ($p < 0.05$), which, apparently, is associated with hemodynamic disturbances on the background of infectious toxic lung damage, hyperthermia, microcirculatory disorders.

A significant decrease in ammonia excretion was revealed in patients with pneumonia with DMN to 9.0 + 1.18 mmol/s compared with healthy children ($p < 0.001$) and in children with pneumonia without DMN 42.0 + 1.2 ($p < 0, 05$).

The decrease in excretion of titratable acids to 23.37 ± 4.25 mmol/s compared with healthy children 51.0 + 2.8 mmol/s ($p < 0.001$) and in children with pneumonia without DMN 48.8 + 2.2 . ($p < 0.05$).

A decrease in acidoammoniogenesis in patients with pneumonia with DMN indicates tubular renal dysfunctions that cause a violation of the adaptive-compensatory functions of the body under hypoxia, which increases metabolic acidosis in pneumonia.

A significant increase in urinary oxalate excretion was detected in children with pneumonia with DMN up to 244.0 + 1.8 μmol/s compared with healthy children 110.0 + 10.5 ($p < 0.001$), and with a group of children pneumonia without DMN 130.0 + 11.5 ($p < 0.05$), which may be secondary oxaluria, which is the result of instability of the cytomembranes of the renal epithelium due to intoxication of the body, hypoxia, electrolyte disturbances that accompany the development of the inflammatory process in the lungs .

Considering that 2/3 of patients with pneumonia with urinary syndrome had a hereditary burden on impaired metabolism of oxalic acid and uric acid, which was revealed by studying pedigrees and cross-interviewing relatives, increased excretion of oxalates and urates along with urate and oxalate -calcium crystalluria, we regarded as secondary oxaluria - uraturia, which is the result of hereditary instability of cytomembranes.

In patients with pneumonia with DMN, there was an almost twofold increase in the excretion of urates and uric acid, respectively 6.9 + 0.22 mmol/s and 6.64 + 0.36 mmol/s compared with those in healthy children ($p < 0.001$) and children with pneumonia without DMN 3.18 + 0.20 ($p < 0.05$) and 5.2 ± 0.3 ($p < 0.05$). Increased excretion of urate and uric acid can be associated with increased catabolic processes against the background of hyperthermia, hypoxia and the breakdown of purine bases, which causes an increase in the level of uric acid in the blood. Thus, the functional impairment of the kidneys is characteristic of pneumonia in patients with pneumonia without DMN, the concentration of nephrotoxic metabolites (urates, oxalates, MK) does not reach values that have a nephrotoxic effect and the concentration of urates, oxalates decreased as a result of targeted therapy for pneumonia,

diet and water regimen. uric acid, which corresponded to the disappearance of such clinical manifestations as pastiness of the eyelids, face, normalization of diuresis. A number of studies have shown the high effectiveness of the complex phytoniring drug Canephron in the treatment and prevention of microbial inflammatory and dysmetabolic processes of the urinary system (3).

It should be noted that in patients with metabolic genesis nephropathy with hereditary aggravation with layering of pneumonia, violations of the partial functions of the kidneys are revealed - oliguria, a decrease in the indicators of acid acid ammonia and an increase in nephrotoxic metabolites, oxalates, urates, uric acid, thereby exerting a nephrotoxic effect . This is possibly due to a violation of the adaptation process at the cellular level as a consequence of an individual inadequate response to the effects of a stress factor in this case of infection. Damage to the membrane structures of the renal tubules can lead to secondary changes in the processes of secretion and reabsorption in the tubules, causing the development of secondary tubulopathies.

The study of partial renal function depending on the nosological forms of dysmetabolic nephropathy was carried out in children with pneumonia with metabolic disorders.

The daily excretion of ammonia decreased significantly with a dismetabolic IN 23.6 ± 3.9 mmol/s Cp <0?05) and USD 29.6 + 3.2 mmol/s (p <0.05) and with PN 33, 8 + 2.6 (p <0.05), compared with healthy children. The most pronounced decrease in excretion of titratable acids was observed at IN 19.2 + 7.9 mmol/s 1.73 m, (p <0.05) and at PN and USD amounted to 24.7 + 3.85 mmol/s, respectively and 26.2 + 4.7 mmol/s (p <0.01).

The highest urinary osmolarity was observed in patients with USD 976 + 62.6 mmol/s (p <P} P5), ID 898 + 62.5 mmol/s (p <0.05) and PN 886 + 58.74 mmol/s (p <0.05) compared with healthy children 627 ± 61.7 mmol/s.

Thus, a decrease in the function of acidoid ammoniogenesis was detected in the group of patients with IN compared with patients with PN and USD, which characterizes the tension of the compensatory-adaptive capabilities of the kidneys to maintain the acid-base state (ABS) of the body in conditions of tissue hypoxia, respiratory and metabolic acidosis in pneumonia.

An increase in excretion of nephrotoxic metabolites (oxalates was noted at PN 226.0 ± 23.0 mmol/s (p <0.05), ICD 262.7 ± 49.8 mmol/s (p <0.01), and IN 243, 2 ± 28.4 mmol/s (p <0.05), compared with healthy children.The excretion of urates with PN was 6.22 ± 0, 52 mmol/s, ICD 8.3 ± 1.32 mmol/s (p <0.001), IN 6.36 ± 0.26 mmol/s (p <0.001), compared with healthy children. Urinary acid excretion is highest in patients with pyelonephritis 7.1 ± 0.3 mmol/s (p < 0.01), with an ICD of 6.26 ± 0.53 mmol/s (p <0.05) and IN 6.55 ± 0.37 mmol/s (p <0.05), compared with healthy children 4 13 ± 0.21 mmol/s.

The existing changes in the function of acidoid ammoniogenesis are associated with increased excretion of nephrotoxic metabolites, against the background of the hereditary burden of a family history of the exchange of UA, oxalates. Intoxications, dehydration, respiratory failure and hypoxia in the presence of pneumonia, exacerbating metabolic disorders, lead to a disorder of homeostatic renal function.

Accordingly, the risk of nephrotoxic effects of urates and oxalates on the kidneys also increases. A sharp increase in the concentration of urates and oxalates due to pneumonia in children with metabolic disorders poses a threat of tubular obstruction with subsequent deterioration of urine outflow, up to the development of renal failure. Clinically, in such children, pastes of the eyelids, faces, decreased diuresis were noted - to oliguria.

Conclusions: Thus, pronounced changes in the tubular renal function in young children with dysmetabolic nephropathy, the insufficiency of their adaptive reactions in conditions of

hypoxia and especially when exposed to infection, being a factor affecting the course of pneumonia, contribute to the chronicity of the pathological process in the kidneys and increase the risk of developing interstitial nephritis, microcrystallization, stone formation. Treatment in these cases without a differentiated correction of dysmetabolism, with a reference mainly only to urinary syndrome, can lead to stabilization and chronicity of the renal process, which acquire independent important medical and social significance.

One of the herbal preparations registered in the Republic of Uzbekistan is the Canephron N drug of the German company Bionorica, which allows to establish a normal passage of urine, a rhythm of urination, to avoid a high concentration of salts in the urine, the formation of large crystals. The drug improves blood supply to the kidneys, helps to reduce proteinuria and pathological crystallization of urine. The fluvonoid components that make up the Canephron N preparation have an angioprotective, anti-inflammatory and antispasmodic effect. Due to its antioxidant properties, Canephron N has a pronounced protective effect against damage by free radicals. Canephron N is prescribed for all types of metabolic nephropathies.

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