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**Research Article** 

# ON THERAPEUTIC TACTICS FOR CHRONIC SECONDARY **PYELONEPHRITIS IN CHILDREN**

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### ABSTRACT

Throughout the world, in recent years there has been a clear increase in the number of kidney diseases in both the adult (12.2) and pediatric populations (6.10). The frequency of urinary system infections (UTI) and their share in the nosological structure of kidney diseases is also increasing to 75.6% (9). The prevalence of pyelonephritis (PN) itself was 32.1:1000, i.e. 43.4% of the total number of nephrology patients. Latent forms of PN pyelonephritis, detected during active examination, are 3 times more common than manifest forms identified by referral. According to V.I. Naumova (1991), 15.3% of chronic renal failure (CRF) developed in childhood is the outcome of secondary chronic pyelonephritis.

## **K**EYWORDS

Chronic renal failure, pyelonephritis, children.

### INTRODUCTION

VOLUME 04 ISSUE 01 Pages: 78-85

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It was found that in 50-65% of children, inflammation causes irreparable damage to the kidney parenchyma, which leads to the development of chronic kidney failure along with the replacement of damaged areas with connective tissue. In addition, 41% of children without urodynamic disorders develop sclerosis. The direct cause of kidney damage is bacterial infection (2.6.2). Since the basis of pyelonephritis is undoubtedly an acute or chronic microbial inflammatory process, etiotropic treatment of pyelonephritis is the use of antibacterial therapy, including antibiotics, antibacterial chemotherapy drugs, and antiseptics (4,9,11).

Long-term (months and years) antibacterial therapy carried out in the last 3-4 decades is not considered harmless, and it leads to a decrease in the body's general resistance, causes allergies and has a toxic effect on parenchymal organs, including the kidneys. also has a toxic effect. In addition, there is a risk of developing dysbacteriosis and resistant forms of bacteria. Modern antibacterial therapy for PN is successful only in 50-70% of patients, in 30-50% of patients it does not lead to complete elimination of the pathogen and thus leads to the risk of subclinical development of nephrosclerosis and chronic kidney failure. . In addition, certain observations show that long courses of intermittent antibiotic therapy of 6 months and 2 months after the end of a course of 4-6 weeks of continuous antibacterial therapy do not prevent the chronic development of PN, do not increase the duration of remission, and do not increase the recurrence of PN. does not reduce the frequency (13, 2, 4).

The main reason for the failure of the treatment of PN is the insufficient identification of the causes that contribute to the development of PN and provoke its recurrence: dysplasia, metabolic, immunological, endocrine dysfunctions (3,5,23). addition, viruses, cytomegalovirus, and chlamydia play an important role in the etiology of PN, which are often neglected and therefore pose a serious threat to public health (8). Taking into account the above, it is optimal to correct the background conditions of the body for 2-3 weeks before antibacterial therapy. (11,9). Among the studied nephropathies, much attention was paid to pyelonephritis as a very common pathology in children, which turns the problem from a purely nephrological problem into a general pediatric problem, because almost every pediatrician has to deal with the diagnosis and treatment of pyelonephritis (9).

Nevertheless. children with pyelonephritis syndrome are often unsuccessfully treated for a long time by doctors of various specialties. This is explained by the fact that the disease can be accurately diagnosed only in a specialized department, where doctors use all the main methods of urological examination and evaluate the metabolic and immunological status of the patient. Despite the history of studying pyelonephritis for more than a century, there is still no unified point of view on the essence of this disease, and the question is often raised in the specialized literature today: does such an independent disease really exist or is this disease a myth? (23, 19,3). Today, the diagnosis of pyelonephritis is based only on clinical data and a

VOLUME 04 ISSUE 01 Pages: 78-85

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general urine test (even including bacteriological data), but it should be comprehensive and include genealogical analysis, obstetric history, X-ray urological examination, metabolic immunological status assessment. should include.

Thus, the urgency of the problem of urinary tract infection does not decrease, discussions about the nature of the disease, determining the number of layers of the lesion, etc. continue(2). The serious prognosis of chronic inflammatory conditions of the urinary system still requires the search for new effective methods of diagnosis optimization of therapy.

The purpose of the study is to evaluate the importance of metabolic diseases in the development and progression of secondary chronic pyelonephritis in children and to optimize their treatment.

### RESEARCH MATERIALS AND METHODS

In this article, 163 children diagnosed with secondary chronic pyelonephritis aged 1 to 14 years were studied. 35 of them (21.4%) are under 3 years old, 59 (36.2%) are 4-7 years old, 69 (42.3%) are 8-14 years old. 68 boys (71.6%) and 95 girls (58.4%). 70 of 163 patients (49%) were diagnosed with pyelonephritis 2-5 years ago, and mostly antibacterial treatment had only a temporary effect, and then repeated relapses were observed. Among them, the initial stage of kidney failure (4.9%), 8 had a tendency to polyuria, hyposthenuria and hyperazotemia. The latter is undoubtedly due to inadequate consideration of contributing and precipitating

background conditions. From the total number of patients, 90 children were X-rayed, 32 of them were diagnosed with various anomalies of kidney development and vesicoureteral reflux (35.5%). Accordingly, obstructive secondary pyelonephritis was diagnosed. Including 24 children, at the same time, hyperoxaluria, uraturia and their combination were detected, i.e. In 755 cases, secondary chronic obstructive pyelonephritis had an obstructive-dysmetabolic character. The most frequently identified dysmetabolic variant of secondary chronic pyelonephritis is SIPN (80.4%). In order to identify the listed groups, the studies were conducted in the following order: the metabolic status of patients was evaluated based on the results of many studies carried out according to a multi-stage program, in which a genealogical analysis was carried out taking into account the kidney and kidney spectrum: pedigree, clinical screening and quantitative biochemical studies include extrarenal pathologies. Daily excretion of oxalates, uricosuria and uricemia are the main biochemical signs of metabolic disorders. Hyperoxaluria is defined as secretion of more than 0.5 mg/kg per day. Hyperuricemia is defined as a serum uric acid level of more than 5 mg% or 0.3 mmol/l, and uraturia is defined as excretion of more than 1 mg/ml of urine per day. Quantitative determination of oxalates in urine was carried out according to N.V. Dmitrieva (1966). Uric acid was determined by the Müller-Seifert method, and urate salts by the Hopkins method (11).

To estimate glomerular filtration, endogenous creatinine clearance was calculated using the Van

VOLUME 04 ISSUE 01 Pages: 78-85

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OCLC - 1368736135











Slyke formula and recalculated to adult standard surface area. Tubular functional status was assessed by Zimnitsky test, ammonia excretion rate, titratable acids and water reabsorption. Xray urological studies were performed and evaluated by pediatric surgeons according to generally accepted principles in nephrourology, followed by X-ray planometry.

### RESULTS AND DISCUSSION

The results of the study of the genealogical history of patients with chronic renal failure against the background of hyperoxaluria and uraturia showed that they are closer to the proband. Thus, in the presence of hyperoxaluria among first-degree relatives, the frequency of nephropathy in genealogy with the control group (3.57%). In patients with chronic kidney failure due to uraturia, this rate was 15.6%. These data indicate an unmistakable role of heredity in the development of DZMN. A retrospective study of the obstetric history of observed children showed that every fourth woman (26.7%) had diseases of

the urinary system (mainly cystitis, pyelonephritis) before pregnancy or during pregnancy. 40% of mothers had toxicosis in the first half of pregnancy, and 31.5% had gestosis during the entire pregnancy. The overall frequency of gestosis in pregnant women exceeded 70%, which is almost ten times higher than the level observed in the general population. All this confirms the truth. This indicates that children of this group are at potential risk for kidney pathology. The disease is often detected against the background of bronchopulmonary diseases (ORVI, pneumonia) and this is not Known. accidental. Relationship between pulmonary ventilation and renal hemodynamics. Maladaptive vascular reactions bronchopulmonary diseases lead to a decrease in glomerular filtration, that is, conditions are created for the appearance and recurrence of pyelonephritis.

Urinary excretion of nephrotoxic metabolites in forms various of chronic secondary pyelonephritis in children (М±м)

ChSP patients	Excretion with urine (mmol/day)		ay) Coef	ficients
	oxalates ura	tes calcium	oxalates/crea	tinine urates/creatinine
Obstructive	0,462±0,043	2,93±0,39 1	,81±0,2 0,48	±0,014 0,87±0,09
(n=32)	P>0,05	P>0,5 P<0,0	P>0,5	P>0,05
Obstructive				
Dysmetabolism	1,082±0,091	3,94±0,46	2,24±0,18 0,	249±0,04 1,38±0,14
(n=24)	P<0,001	P=0,05 P<	0,01 P<0,0	01 P<0,01

VOLUME 04 ISSUE 01 Pages: 78-85

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Dysmetabolism	0,72±0,051	5,63±0,24	2,53±0,18	0,206±0,04	1,96±0,16
(n=131)	P<0,001	P<0,001	P<0,001	P<0,001	P<0,001
Healthy (n=47)	0,413± 0,059	9 2,94±0,31	1,46±0,12	0,053±0,005	5 0,83±0,08

P - significance of the difference compared to the control group

As can be seen from the table, in contrast to the pure obstructive form of chronic renal failure, the obstructive-dysmetabolic form significantly increased the daily excretion of oxalates, urates and their ratio to creatinine (P<0.01).

In patients with oxalate nephropathy, the concentration of oxalates is 0.106±0.011 mmol/l, the daily excretion rate is 97.4 mg/day (more than 1.6 mg/kg), which is 3 times higher than in healthy people (co not 'p) more than 0.5 mg/kg/day). The ratio of oxalates to creatinine per day is  $1.38\pm0.14$ , the norm is  $0.053\pm0.83$ (P<0.001). In the group of patients with urate nephropathy complicated by chronic kidney disease, oxaluria was 63.2±4.6 mg/24 hours. creatinine ratio is 1.96±0, 16 is 0.83±0.08

(R<0.001) in norm. In cases of chronic renal failure associated with uraturia, a clear decrease in renal function was observed (Table 3). In the observed patients, the parameters of tubular reabsorption of water and glomerular filtration are not significantly different from the norm. We regularly observed a tendency to oliguria and a decrease in GFT (glomerular filtration rate) with the development of chronic renal failure against the background of uraturia. All groups of observed patients were characterized by a decrease in the relative density of urine and ammonia acidogenesis (P<0.05).

Treatment of patients with an obstructive form of chronic renal failure was carried out together with pediatric surgeons, and in some cases, with surgical correction of urinary bladder reflux (SQR).

Indicators	Healthy	OChSP	ChSP
Creatinine clearance	98,6±4,8	97,8±6,4	75,5±4,2
	$C_{cr}(ml/min,1)$	,73м²)	
Ammonia (g/d)	0,91±0,02	0,56±0,07	0,63±0,04
Titrated acids	45,4±2,8	29,0±3,0	27,2±1,4

VOLUME 04 ISSUE 01 Pages: 78-85

SJIF IMPACT FACTOR (2021: 5.478) (2022: 5.636) (2023: 6.741)

OCLC - 1368736135











Tubular reabsorption of water 98,6±0,06

98.2±0.3

98,3±0,3

(%)

Antibacterial therapy was carried out for 2-3 weeks under the control of the sensitivity of isolated microbes to them. Repeated courses of antibacterial therapy were carried out only in the presence of significant bacteriuria. In cases where dysmetabolism of oxalates and urates was detected, a diet was prescribed, which included the restriction or complete elimination of food products that are a source of oxalates and urates. Diet therapy is prescribed for a long period (6-12 more). Drug correction months or dysmetabolism has also been used. Taking into account that the lack of pyridoxal-5-phosphate is one of the causes of hyperoxaluria, B6 was prescribed 20-60 mg per day on an empty stomach, sodium benzoate 0.05-0.3 g/day to limit the synthesis of oxalate in the body; magnesium oxide 100-200 mg per day to improve solubility. Long-term use of this therapy complex (2-6 months) made it possible to normalize the excretion of oxalate with urine.

In the treatment of chronic kidney failure in patients with uricopathy, we use a diet with the maximum exclusion of food products rich in purine bases from the diet. Fruits, fruit juices, lemons, and a high fluid regimen are common to alkalinize the urine. To limit the synthesis of uric acid in the body, we used allopurinol 0.05x 1 time a day for children under 7 years old, 0.1x 1 time a day for children over 7 years old for 1 month. Potassium orotate helps to improve the excretion of uric acid in the urine. In all cases herbal

medicines in various forms are widelv recommended. In recent years, we have widely used Kanefron-N complex drug, which has antiinflammatory, antibacterial, spasmolytic and diuretic effects (1,15,16). The drug was prescribed to babies 10 drops 3 times a day. 15 drops for preschool children, 25 drops 3 times a day for school-aged children. If necessary, repeated courses of treatment were conducted. Immunomodulating drugs (Viferon 1.2, Reaferon, lysostim, Immunal) were prescribed to all children with a slow, recurrent course of the disease. Immunomodulators were used in all patients with the development of chronic renal failure against the background of uraturia, because dysnucleotidosis is characterized by a violation of molecular processes that ensure the proliferation and maturation immunocompetent cells (7,14). In addition, all patients received concomitant antioxidant therapy (vitamins A, E, and C) due to the stimulation of lipid peroxidation and deficiency of antioxidant protection, which are common factors in chronic renal failure, which are factors that aggravate depression of immunological homeostasis, absence, preparations containing selenium - triovit, vitrum, centrum) for 2-3 weeks. If there are symptoms of mitochondrial insufficiency, Kudesan or Elkar is recommended. Such complex therapy (and not limited to prescribing antibacterial therapy) helped to significantly increase the effectiveness treatment. Against the background of 2-3 weeks

VOLUME 04 ISSUE 01 Pages: 78-85

SJIF IMPACT FACTOR (2021: 5.478) (2022: 5.636) (2023: 6.741)

OCLC - 1368736135











of complex therapy, a significant decrease in oxaluria, uraturia and, in parallel, proteinuria, leukocyturia and bacteriuria was observed.

We have seen that in the dysmetabolic variant of chronic renal failure, the initial acceptance of clinical and even clinical laboratory remission is not a guarantee against further relapses of the disease. Therefore, patients with dysmetabolic chronic renal failure require continuous targeted monitoring and corrective dietary therapy. In this regard, a certain psychological "mood" is necessary not only for the child, but also for his parents, because they often stop treatment on their own, considering that the child is cured, and often ignore the importance of dietary measures. they ignore.

### Conclusions

The most common source of diagnostic and treatment-tactical errors is the management of outdated concepts of pyelonephritis by general practitioners as an independent disease with microbial-inflammatory properties, which helps to implement only antibacterial therapy. The diagnosis of pyelonephritis should be established after a comprehensive clinical, genealogical, X-ray and urological examination, assessment of the metabolic, immunological and endocrine status of patients. Without this, it is impossible in relation to SIPN, the main deontological principle of clinical medicine should be observed "to treat the patient, not the disease". Obstructive chronic secondary pyelonephritis is often combined with metabolic diseases (hyperoxaluria, uraturia,

hypercalciuria, etc.), which requires surgical correction of the malformation, as well as correction of metabolism with diet and drugs, and targeted antibacterial therapy.

Non-obstructive chronic secondary pyelonephritis is mainly caused by the accumulation of nephrotoxic metabolites against background of metabolic diseases. antibacterial therapy is also insufficiently effective without appropriate correction of the metabolic background.

Isolated antibacterial therapy for chronic secondary pyelonephritis is often not effective enough, it should be comprehensive and, in addition to surgical and metabolic correction, should include immunomodulatory therapy and antioxidant protection of the organism.

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VOLUME 04 ISSUE 01 Pages: 78-85

SJIF IMPACT FACTOR (2021: 5.478) (2022: 5.636) (2023: 6.741)

OCLC - 1368736135











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