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Research Article

CHANGE OF FUNCTIONAL KIDNEY RESERVE IN CHILDREN IN DYSMETABOLIC NEPHROPATHIES

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ABSTRACT

The urgency of the problem. The development of renal failure depends on secondary hemodynamic metabolic factors than on the activity of the primary pathological process. Both non-modifiable and potentially modifiable risk factors for the development of renal failure have been identified (2, 7). Among the potentially reversible risk factors for the development of renal failure, glomerular hyperfiltration and interglomerular hypertension under the influence of angiotensin II (ANG II) are of high importance.

KEYWORDS

Renal failure, glomerular hyperfiltration, non-modifiable and potentially modifiable.

INTRODUCTION

The results of research conducted in recent years allowed to significantly expand the ideas about the mechanisms of development of kidney damage in urate dysmetabolism. In the induction of inflammation and fibrosis of tubulointerstitial structures, the increase in renin expression by yxtaglomerular cells under the influence of uric acid is of great importance,

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which leads to the activation of the local renal renin angiotensin-aldosterone system (RAAS).

RAAS activation leads to an increase in ANG II, causing systemic spasm of arteries, glomerular hyperfiltration, and proteinuria (8, 9). Given the above, as well as the fact that uric acid (UC) is a strong inducer of general endothelial dysfunction, metabolic correction of UC should be initiated early if possible. (4). However, angiotensin-converting enzyme (AAFI) inhibitors are currently used to reduce the expression of tubulo-interstitial fibrosis markers.

Thus, suppression of ANGII at the renal level in patients with urate nephropathy is a very important aspect of the problem.

The functional criterion of the state of glomerular hyperfiltration is to determine the functional reserve of the kidneys (BFZ), the level of which can also evaluate the effectiveness of measures aimed at its elimination (1, 6). The maximal rate of glomerular filtration (MF) in the hyperfiltration state and the difference between the maximum and basal HF is defined as BFZ.

Objective: To study the clinical significance: Identify the functional stock of kidneys in drugs in children.

Materials and research methods. 76 children, an optical nephropathy, were monitored. Including 27 children and pyelonephritis, including Corridor Nephropathy, were examined by 49 children. In the groups above, the condition of the functional reserve in the field of protein - the degree of water metabolism, the degree of the degree of endogenine Clients of the endogenine clearance was further studied.

77.8% of children with urate nephropathy and 86% of patients with impaired renal function have reduced functional renal reserve. The functional reserve index of the kidneys can serve as a measure of the effectiveness of therapy.

76 children with urate dysmetabolism aged 6 to 14 years were under our control. 38 of them are girls and 38 are boys. The control group consisted of 16 clinically healthy children with no family history of kidney pathology. Sick children were divided into 2 groups. Group I consisted of 27 children (35.5%) with dysmetabolic nephropathy (DZMN), 17 girls (63%), 10 boys (37%). The diagnosis of DZMN is based on the nature of the pathology in the family, the level of uric acid (MC) in the blood and urine, the presence of an isolated urinary syndrome of microhematuria and/or proteinuria, tubular dysfunctions, hyperstenuria, oliguria.

Group II included 49 children (64.5%) with hyperuricemia and urateria: 32 children had pyelonephritis (PN), 17 children had interstitial nephritis (IN), of which 27 (55.1%) were girls, and 27 (55.1%) were boys. children made up 22 (44.9%).

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A complete clinical and genealogical analysis was performed. Glomerular function was assessed by endogenous creatinine clearance, uric acid was determined by the Müller-Seifert method. The functional state of the tubes was assessed by the level of excretion of calcium, phosphorus, ammonia, and titrated acids.

BFZ was defined as the rate of increase in basal CF (% CF) after stimulation with increased protein and water intake: basal and stimulated CF were calculated from endogenous creatinine (Ccr) clearance. For this, 1 g/kg body weight of meat protein should be given orally. The study was carried out on an empty stomach after a night's sleep, and in the morning, medication was canceled. Between 8:30 AM and 8:30 AM, the patient drank 10 mL/kg of water, and then collected urine by voiding for one hour from 8:30 AM to 9:30 AM.

Thus, KF was determined. To determine the child's stimulated CF, the child was offered boiled meat at the rate of 1.0 g protein/kg body weight and drank another 10 ml/kg water for 30 minutes, from 9 hours to 30 minutes to 10 hours. then I collected urine for an hour, from 10 to 11 hours. Creatinine in blood and accumulated parts of urine were determined by the generally accepted Yaffe method, and clearance was calculated by Van Slick (E. A. Yuryeva, 2002)

Research and discussion results:

In the comparative analysis of the functional state of the kidneys and the composition of urine, a number of characteristics were revealed in the studied groups (Table 1).

Table 1.

Comparative characteristics of the functional status of kidneys and the composition of urine in children with urate nephropathy (m±m)

Indicators	Control group (n=16)	Children with DZMN	
		Children with urate nephropathy with isolated urine syndrome (n=27)	Kidney function activity (n=49)
Diuresis (ml/min.) Urates (mmol/day)	0.72±0,04 2.41±0,20	0,56±0,03 P<0,001 5,74±0,26 P<0,001	0,64±0,05 P<0,05 5,94±0,15 P<0,001

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Oxalates (mmol/milk)	0.332±0.05	0,62±0,03 P<0,001	0,51±0,03 P<0,001
Calcium (mmol/milk)	1,42±0,18	1,66±0,09 P<0.05	1,52±0,09 P<0,05
Inorganic phosphorus (mmol/milk)	10,4±1,24	16,2±1,3 P<0,001	18,6±0,86 P<0,001
Creatinine clearance (ml/min. 1.73m²)	115,8±7,1	81,9±13,9 P<0,001	61,4±3,9 P<0,001
Calcium	0.78±0,1	1,26±0,24 P<0,05	1,32±0,24 P<0.05
Phosphorus	9.4±2.0	12,6±0,94 P<0,05	16,4±10,94 P<0,05
Ammonia (mmol/milk)	91,9±12,6	101,6±21,4 P<0.05	127,7±14,8 P<0,001
Titrated acid (mmol/milk)	28,4±2,7	22,4±4,5 P<0,05	22,4±5,4 P<0,05

Note: p-relative to control group

The table shows that the minute diuresis in patients with uratic nephropathy (0.72±0.04 ml/min) is significantly reduced (0.56±0.03 ml/min) compared to the control group (p<0.05). min). The daily excretion of urates is 2.4 times higher than both groups (5.74±0.26 and 5.94±0.15 mmol/day, respectively) compared to the control group (2.41±0.20 per day). mmol.).

It should be noted that 1/2 of patients with urate nephropathy had increased excretion of oxalates in the urine, so the daily excretion of oxalates in the urine was 2 times higher (0.332±0.05 mmol/day) compared to the control group (0.62±0.03 and 0.51±0.03 mmol/day).). In both groups, the daily excretion of calcium and phosphorus exceeds the values in the control group, and their clearance also increases compared to the norm (p<0.001). Creatinine clearance decreased in all groups (p<0.001). An increase in ammonium urea and a decrease in acidogenesis were observed in both groups, especially in the group with PN (pyelonephritis) and IN (interstitial nephritis) layers.

Thus, layering the active pathological renal process in the Nephropean nephropathy strengthens the disorders of uratinene, phosphaturia, phosphaturia, dysfunction, which is likely to bring to tubulo-intensive disorders.

When the protein-water content, the RFR identified in healthy children. The Basal KF (DKF) growth rate was 13.7 ± 2.2% in healthy children. In patients with Uritic Nephropathy, RFR (renal fundal reserve) is based on DKF control values, the following scderable values, remain 9% of the score dkf \ u003e 9%; DKF-4.5 - 9% - reduced bfz; DKF <4.5% - no RFR.

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Information on the distribution of studied patients according to %KF is presented (Table 2).

Table 2

Distribution of patients according to RFR (Renal Functional Reserve) status

RFR	Patients with urate nephropathy	
	With isolated urine syndrome (n=27)	Kidney function activity (n=49)
RFR saved RFR decreased RFR no	6 (0,22) 12 (0,45) 9 (0.33)	7 (0,14) 22 (0,45) 20 (0.41)

Note: the frequency of occurrence of the symbol in parentheses.

Table 2 shows that RFR urinary syndrome with isolated uratic nephropathy is preserved in 22.2%, and 77.8% of them are reduced or absent. The latter indicates the presence of glomerular hyperfiltration and the risk of developing glomerulosclerosis at this stage of the disease. Therefore, at this stage of urate nephropathy, measures aimed at eliminating hyperuricemia (correction of diet and drugs) and hyperfiltration (angiotensin-converting enzyme inhibitors) are necessary.

The addition of PN and IN sharply worsens the situation, increases the risk of developing progressive renal failure. Thus, the preservation of RFR in this group was determined in 14.3%, its decrease in 44.9% was not possible in 40.8%. Patients with slightly elevated creatinine (above 125 µmol/L) also had no BFZ. The decrease in RFR is associated with the loss of working parenchyma with the development of compensatory hyperfiltration. This is confirmed by the dependence of RFR loss on the severity of the disease (Table 3).

Table 3

Distribution of patients by RFR status depending on the duration of nephropathy

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Duration of nephropathy	Number of patients			
	With isolated urine syndrome		Kidney function activity	
	RFR saved (n-6)	RFR decreased or not maintained (n=21)	RFR saved (n-7)	RFR decreased or not maintained (n-42)
4 to 1 year 1-3 years 3 years	3 2 1	2 5 14	6 1 0	3 8 31

Table 3 shows that as the duration of the disease increases, the number of patients with preserved BFZ decreases and the number of patients with reduced or absent RFR increases dramatically).

After diet-drug therapy of urate nephropathy with isolated urine syndrome and after one month of PN and targeted therapy including angiotensin-converting enzyme inhibitors, RFR in all DZMN patients with isolated urine syndrome From 4.5 to 9% and increased in 36 of 42 patients (85.7%) in the group of patients with renal function.

Therefore, the decrease or absence of RFR does not exclude the possibility of its rebellion with successful treatment and means a decrease in the rate of development of kidney disease.

CONCLUSIONS

1. In patients with urate nephropathy, the partial functions of the kidney are disturbed in the early stages of the disease and increase with the addition of kidney diseases (pyelonephritis, interstitial nephritis).
2. The functional reserve of the kidneys decreases in the early stages of the development of urate nephropathy and increases when the active renal process is added.
3. A decrease in the functional reserve of the kidneys and the appearance of previously absent urate nephropathy during successful therapy indicate the prognostic value of this indicator.

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