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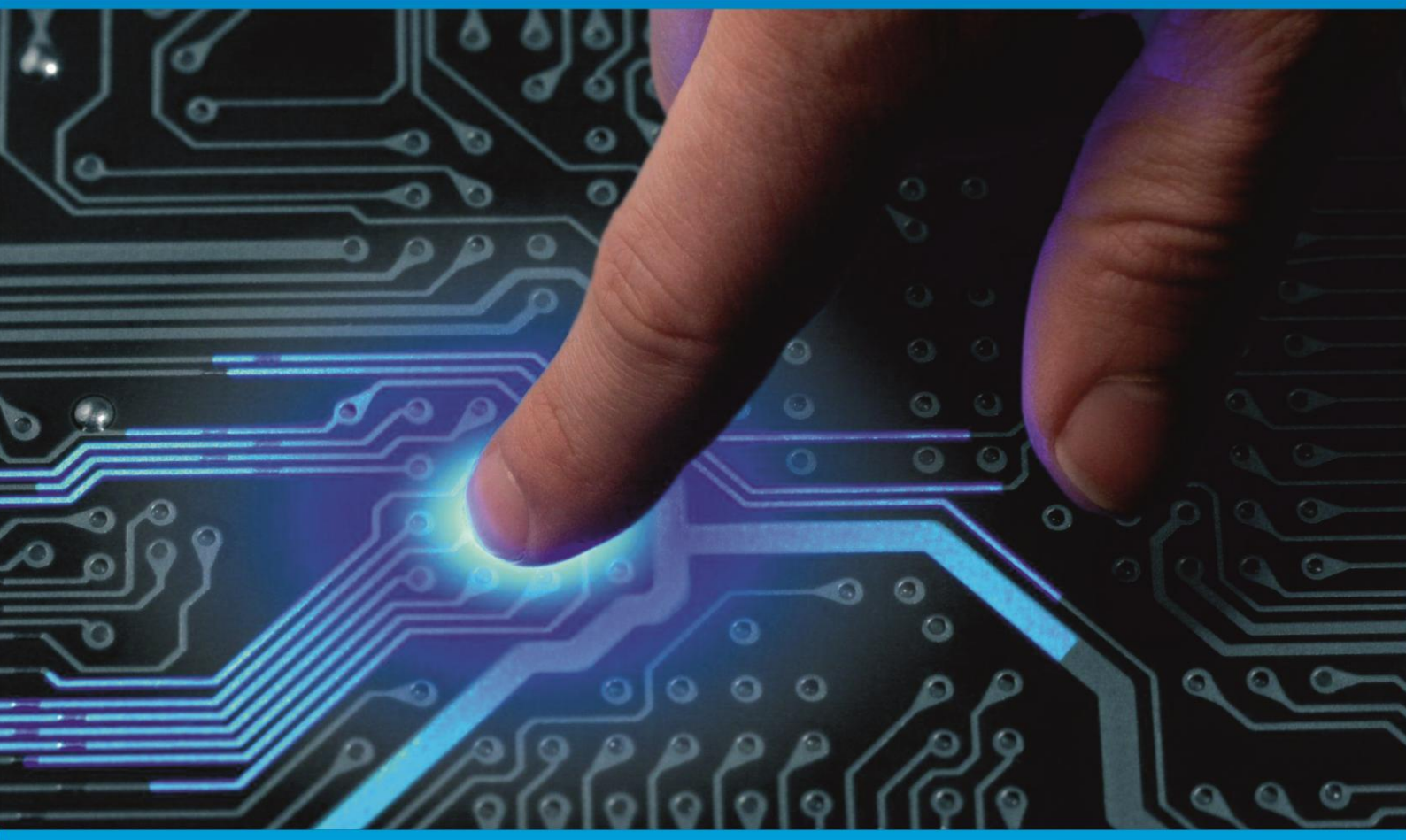

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Dysmetabolic Nephropathy and Clinical Definition of Functional Kidney Reserve in Children

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ANNOTATION: A major achievement of nephrology in the last 10-15 years was the clinical and experimental substantiation of the position that the progression of renal failure is more due to secondary hemodynamic and metabolic factors than to the activity of the primary pathological process. Unmodified and potentially modifiable risk factors for the progression of renal failure have been identified (2,7). So, dynamic observation for 6 years for 76 children who underwent nephropathy during the neonatal period. It showed that the majority of them, against the background of ongoing rehabilitation measures, later develop interstitial nephritis (IN), neurogenic bladder dysfunction or metabolic nephropathy and pyelonephritis. So, dynamic observation for 6 years for 76 children who underwent nephropathy during the neonatal period. It showed that most of them, against the background of ongoing rehabilitation measures, subsequently develop interstitial nephritis (IN), neurogenic dysfunction of the bladder or metabolic nephropathies and pyelonephritis. Among the potentially reversible risk factors for the progression of renal failure, a high value is attached to glomerular hyperfiltration and intraglomerular hypertension under the influence of angiotensin II (ANG II).

KEYWORDS: Dysmetabolism, pyelonephritis, nephropathy, kidney, children, tubulointerstitium structures,

I. PURPOSE OF THE STUDY

To study the clinical significance of determining the functional reserve of the kidneys in children with dysmetabolic nephropathies.

II. MATERIALS AND METHODS

We observed 76 children with urate dysmetabolism at the age from 6 to 14 years. Of these, 38 are girls and 38 are boys. The control group consisted of 16 clinically healthy children without family history of renal pathology. Sick children were divided into 2 groups depending on the presence of renal pathology activity. Group I included 27 children with dysmetabolic nephropathy (DZMN), girls - 17 (63%), boys - 10 (37%). The diagnosis of DZMN was substantiated by the nature of the pathology in the pedigree, the level of uric acid (UC) in the blood and urine, isolated urinary syndrome - microhematuria and proteinuria, and the presence of tubular dysfunctions.

Group II included 49 children in whom hyperuricemia and uraturia were complicated by kidney diseases: pyelonephritis (PN) - in 32 children, interstitial nephritis (IN) - in 17 children. There were 27 girls (55.1%), and 22 boys (44.9%).

Clinical and genealogical analysis, general clinical laboratory studies were carried out. In the morning from 8 o'clock for 30 min. The patient drank at the rate of 10 ml / kg, then for an hour he collected urine by free urination - from 8:30 am. Until 9 o'clock. 30 min., From 9 h 30 min. up to 10 hours. Then within an hour, from 10 to 11 o'clock. Collected urine. Creatinine in blood and collected urine portions was determined by the standard Jaffe method, and clearance was calculated according to Wan Slayku.

III. THE RESULTS AND THEIR DISCUSSION

The results of recent studies have made it possible to significantly expand the understanding of the mechanisms of development of kidney damage in urate dysmetabolism. It turned out that the increase in the expression of renin by juxtaglomerular cells under the influence of uric acid, which leads to the activation of the local renal renin-angiotensin-aldosterone system (RAAS), is of greater importance in the induction of inflammation and fibrosis of the tubulointerstitium structures.

Activation of the RAAS leads to increased production of ANH II, causes systemic arterial spasm, glomerular hyperfiltration, and proteinuria (8,9). Taking into account this fact, also taking into account that uric acid (MK) is a

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powerful inducer of generalized endothelial dysfunction, there is a need for the earliest possible correction of metabolic disorders of MK (4). Along with this, angiotensin-converting enzyme (ACE inhibitors) inhibitors are currently used to reduce the expression of markers of tubulo-interstitial fibrosis.

Thus, the suppression of ANH II at the renal level in patients with urate nephropathy is a very significant aspect of the problem.

The functional criterion of the state of glomerular hyperfiltration is the determination of the functional reserve of the kidneys (FRP), by the level of which one can also judge the effectiveness of measures aimed at its elimination (1,6). The maximum level of glomerular filtration (CF) achieved under stimulation conditions is defined as the state of hyperfiltration, and the difference between the maximum and basal CF is defined as RFP.

A comparative analysis of the functional state of the kidneys and the composition of urine in the studied groups revealed a number of features (Table No. 1). In urate nephropathy, the layering of an active pathological renal process aggravates violations of urate homeostasis, phosphaturia, and tubular dysfunction according to clearance data.

In patients with urate nephropathy, a significant decrease in minute urine output compared with the control group (0.56 ± 0.03 ml / min. Vs. 0.72 ± 0.04 ml / min, P<0.05). The daily urate excretion in both groups was more than 2.4 times higher (5.74 ± 0.26 and 5.94 ± 0.15 mmol / day, respectively) than in the control group (2.41 ± 0.20 mmol / day).In 1/2 of patients with urate nephropathy, there was an increase in urinary excretion of oxalates compared with the control group (0.62 ± 0.03 and 0.51 ± 0.03 mmol / day versus 0.33 ± 0.05 mmol / day).

The daily excretion of calcium and phosphorus in both groups exceeded the value in the control group, an increase in their clearance compared to the norm was found (P<0.001). Endogenous creatinine clearance in all groups was reduced (P<0.001). In both groups, especially in the group with layering of PN and IN, an increase in ammoniumuria and a decrease in acidogenesis were observed.

Determination of FRP in healthy children using a protein-water load revealed a distinct stimulating effect. The degree of increase in basal CF (ΔKF) in healthy children was 13.7 ± 2.2%. Based on the control values of ΔKF for assessing RFP in patients with urate nephropathy, the following scale was used: ΔKF>9% - FRP is preserved; ΔKF = 4.5-9% - FRP is reduced; ΔKF <4.5% - there is no FRP. Table 2 shows data on the distribution of the studied patients depending on Δ% CF.

FRP was preserved in 22.2% of patients with urate nephropathy with isolated urinary syndrome and in 77.8% of them it was reduced or absent. Therefore, already at this stage of urate nephropathy, measures are needed to eliminate hyperuricemia (dietary drug correction) and hyperfiltration (angiotensin-converting enzyme inhibitors).

The addition of PN and IN sharply aggravate the situation, increase the risk of developing progressive renal failure. Thus, the preservation of FRP in this group was revealed only in 14.3%, its decrease - in 44.9%, absence - in 40.8%. Patients with even a slight increase in creatininemia (over 125 μmol / L) had no RFP.The decrease in FRP is apparently associated with the loss of a functioning parenchyma and the development of compensatory hyperfiltration. This was evidenced by the dependence of the loss of FRP on the duration of the disease (Table 3). With an increase in the duration of the disease, the number of patients with preserved FRP decreased, and the number of persons with a decrease in FRP (or its absence) increased sharply.

After dietary-drug therapy of urate nephropathy with isolated urinary syndrome and targeted therapy of PN and IN with the inclusion of angiotensin-converting enzyme inhibitors for one month, FRP increased from 4.5 to 9% in all patients with DMD with isolated urinary syndrome and in 36 of 42 (85.7%) - in the group of patients with the activity of the renal process.

Consequently, a decrease or absence of FRP does not exclude the possibility of its restoration with successful treatment and suggests a slowdown in the rate of progression of renal disease.

Table 1.
Comparative characteristics of the functional state of the kidneys and the composition of urine in children with urate nephropathy (M±m)

Indicators	Thecontrolgroup	DZMN patients	
		Urate nephropathy with isolated urinary syndrome (n=27)	With the activity of the renal process (n=49)
Diuresis (ml / min)	0,72±0,04	0,56±0,03 P<0,001	0,64±0,05 P<0,05
Urates (mol/day)	2,41±0,20	5,74±0,26 P<0,001	5,94±0,15 P<0,001

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Oxalates	0,332±0,05	0,62±0,03 P<0,001	0,51±0,03 P<0,001
Calcium	1,42±0,18	1,66±0,09 P<0,05	1,52±0,09 P<0,05
Inorganic phosphorus	10,4±1,24	16,2±1,3 P<0,001	18,6±0,86 P<0,001
Creatinine clearance (ml / min. 1,73m^2)	115,8±7,1	81,9±3,9 P<0,001	61,4±3,9 P<0,001
Calcium	0,78±0,01	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±0,94 P<0,05
Titrated acidity(mmol/day)	28,4±2,7	22,4±4,5 P<0,05	P<0,05

Note: P - compared to the control group.

Table 2.
Distribution of patients according to the state of FRP

FRP	Patients with urate nephropathy	
	With isolated urinary syndrome (n=27)	With the activity of the renal process (n=49)
FRPsaved	6(0,22)	7(0,14)
FRP reduced	12 (0,45)	22(0.45)
FRP ismissing	9(0,33)	20(0,41)

Note: In parentheses, the frequency of occurrence of the trait

Table 2.
Distribution of patients according to the state of FRP depending on the duration of nephropathy.

Durationofnephropathy, years	Numberofpatients			
	Isolatedurinarysyndrome		With the activity of the renal process	
	FRPsaved(n=6)	FRP is reduced or absent (n=21)	FRPsaved(n=7)	FRP is reduced or absent (n=42)
Upto 1 year	3	2	6	3

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1-3 yearsold	2	5	1	8
Morethan 3 years	1	14	0	31

IV. CONCLUSIONS

1. Partial renal functions in patients with urate nephropathy are impaired in the early stages of the disease and are aggravated by the addition of kidney diseases (pyelonephritis, interstitial nephritis).
2. The functional reserve of the kidneys is reduced in the early stages of development of urate nephropathy and is aggravated by the addition of an active renal process.
3. An increase in the previously reduced functional reserve of the kidneys and the appearance of urate nephropathy, which was previously absent with successful therapy, indicate the prognostic value of this indicator.

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