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CLINICAL AND LABORATORY FEATURES OFNEPHROPATHY IN CHILDREN WITH DIABETES MELLITUS

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Annotation
Diabetes currently occupies a leading position in pediatric endocrinology with diseases, disorders and complications that can lead to a decrease in life expectancy. According to the World Health Organization, the incidence of diabetes in the world doubles every 10-15 years, and the life expectancy of patients is lower than among the general population. As a result of type 1 diabetes, the patient's ability to work and their life are impaired, which indicates that the disease is an important medical, social and economic problem in modern society. Materials and research methods. 50 patients with type 1 diabetes mellitus aged 6 to 16 years, 28 boys and 22 girls. The control group consisted of 20 patients with type 1 diabetes mellitus who did not have hyperuricemia, hyperuricosis, protienuria, that is, normal urinalysis. ... Research results. The data obtained confirm that hyperuricemia, hyperuricosuria and urate nephropathy are common in children with diabetes. The onset of the disease is less pronounced, the course is relatively positive. However, if the diagnosis is not made at an early stage, that is, without dietary correction of purine metabolism, the process can become chronic (interstitial nephritis, DN, microbial inflammatory process of the kidneys, chronic renal failure can be observed). Conclusion: Given the type of disease, dysmetabolic changes are difficult to reverse, but we can conclude that dietary medico-time adjustments can be avoided in a timely manner.

Keywords: diabetes mellitus, nephropathy, children.

Relevance of the Topic

Diabetic nephropathy, one of the dreaded complications of type 1 diabetes, is now one of the main causes of death in children [4, 7]. DN mainly develops within 5-10 years after the onset of the disease, leading to a significant deterioration of the patient's quality of life, disability and, quite quickly, chronic renal failure (SBE). Every fourth to fifth of patients with type 1 QD die from SBE [4, 10]. Treatment of end-stage renal failure by extracorporeal methods (dialysis, kidney transplantation) requires a huge amount of money, amounting to several billion dollars every year, which raises kidney pathology in diabetes to the level of an important socio-economic problem [3, 5].

If children with diabetes develop symptoms of kidney disease, the first thing to consider is the possibility of developing diabetic nephropathy. In addition, such patients are not protected from dysmetabolic nephropathy with other genesis. According to observations, diabetic nephropathy can develop in children diagnosed with diabetes within 5 years (E. V. Melekhin, 2003). It is known that proteinuria observed in diabetes is not related to diabetic nephropathy in 10-30% of cases [4]. Metabolic nephropathy takes the main place among the non-infectious risk factors for kidney damage:

urate, oxalate, etc. [12]. According to scientists, urate nephropathy is a common option among dysmetabolic nephropathies, because carbohydrates with diabetic characteristics (obesity, MS, gout, atherosclerosis, arterial hypertension, insulin sensitivity) make up the whole range of pathological conditions associated with hyperuricemia (GU). There is a parallel between carbohydrate disorders and the level of manifestation of GU [11, 14, 1]. It was found that uric acid is structurally related to alloxan, a diabetogenic substance [19]. The etiological role of uric acid in the development of QD has been proven on the basis of experimental investigations: a single administration of SC to rats led to the development of uric acid diabetes. In this regard, timely initiation of treatment measures for dysmetabolism combined with urate dysmetabolism is of great medical, economic and social importance [6, 19].

The Purpose of the Work:

To study the clinical and laboratory characteristics of uricosuric nephropathy in patients with diabetes.

Case Study and Examination Materials:

50 patients with type 1 QD, 28 boys, 22 girls aged 6 to 16 years were taken. As a control group, 20 patients with type 1 QD and no hyperuricemia, hyperuricosuria, proteinuria, i.e., normal urinalysis, were taken. As a norm, 10 healthy children of the same age without aggravated family anamnesis (QD, metabolic syndrome) were taken.

The average duration of the disease is 3.24±0.8 years, that is, the duration of the disease is definitely not enough for the development of diabetic nephropathy. The disease was variable in 6 children, with decompensation 2 times a year, and in the rest 1-2 times a year, with relatively stable decompensation. When arriving at the hospital, all patients were in the stage of decompensation of carbohydrate metabolism, the amount of glycosylated hemoglobin (Nb A 1s) was determined in 12 patients (24%), from 3.8 to 14.4%, the average was 8.0 ± 3.57%. No ketosis was observed during special examination. According to the daily data of the glycemic profile, the average daily level of glycemia was from 3.2 to 14.1 mmol/l, the average value was 8.4 ± 2.4 mmol/l. According to the daily data of the glycemic profile, the average daily level of glycemia ranged from 2.5 to 28.1 mmol/l, with an average of 13.29 ± 4.8 mmol/l.

Examination Methods:

Clinical and laboratory characteristics of the examined patients are presented in Table 1. As can be seen in this table, there is no difference in age among the examined patients ($r>0.05$). Clinical examination methods include: general examination of patients, objective examination of organs and systems by palpation, percussion (comparative, topographical), auscultation and measurement of arterial blood pressure by the Korotkov method.

Table 1. Clinical and laboratory characteristics of patients

Indicators	Duration of illness	
	up to 2 years	2-4 years
Age, year	11,9±2,1	12,1±2,2
TMI, kg/m2	18,8±2,3	19,1±1,6
Glycemia, mmol/l	12,6±3,4	13,5±4,1
NvA 1s	9,8±2,5	11,8±3,7
Total cholesterol, mmol/l	4,7±1,1	4,49±0,9
Blood creatinine mmol/l	0,086±0,07	0,091±0,04
Average AQB mm.sm.us.	110,0±6,8	114,6±7,4
Diastolic AQB mm.sm.us.	62,2±6,8	68,4±8,4
Uric acid mmol/l	0,310±0,05	0,360±0,06
Insulin daily dose ED/milk	30,2 ±10,1	36,3±12,6
Urea mmol/l	5,7±4,1	36,3±12,6

Quantitative examination of proteinuria: application of Beuringer-Ingel Austrian company Nephur-type test strips. With the help of strip tests, in addition to determining the amount of proteins in urine, the following indicators were studied: reaction, specific gravity, leukocytes, bacteria, erythrocytes, glucose. Sulfosalicylic acid was used for qualitative and quantitative evaluation of proteinuria.

Partial kidney function in children with uricosuric nephropathy on the background of type 1 diabetes. According to the task, the patients were divided into 2 groups: 1 group (control) included patients with hyperuricemia, without hyperuricosuria, without pathological changes in urine, with 20 QD with a negative microalbuminuria test; Group II included 30 patients with hyperuricemia, hyperuricosuria and uricosuric nephropathy. The analysis of the functional state of the kidneys was carried out using the Roberg-Tareev test, the test of the functional reserve of the kidneys and the concentration function of the kidneys were carried out using the Zimnitsky test. Also, calciuria, tubular reabsorption indicators of phosphorus were used to evaluate the function of kidney tubules. (table 2). According to Table 2, glomerular filtration rate (FFT) in both groups is significantly increased ($r<0.001$) and refers to glomeruli in a state of hyperfiltration (FFT>140 ml/min/1.73m2). Water reabsorption tended to decrease, but was not statically reliable ($r<0.005$), TRP was reliably decreased in both groups. Uric acid was 8.4±0.08 ml/min/1.73m2 in patients of group I, 6.6% in group I, 10.3% in group II. Accordingly, the fractional clearance of uric acid in group I was 14.6±1.2 ml/min/ha (normally 8.4%±0.4, $r>0.05$) and in group II up to 16.2±1.2% ($r<0.001$) increase was observed. These data indicate a better functional capacity of the kidneys in the excretion of uric acid from the body in the observed patients, which explains the almost 2-fold increase in the daily secretion of uric acid in uricosuric nephropathy ($r<0.001$).

Table 2 Clearance and reabsorption of certain substances in diabetes mellitus complicated by uricosuric nephropathy (M±m)

Indicators	Healthy ones (n=10)	Group I (n=20)	Group II (n=30)
Creatinine clearance (ml/min/1.73m2)	108,0±4,8	160,0±6,3 p<0,001	156,4±11,2 p<0,001
Water reabsorption (%)	98,2±0,7	96,1±1,1 p>0,05	96,6±1,2 p>0,05
Sa-clearance (ml/min/1.73m2)	0,8±0,04	1,2±0,08 p<0,05	1,6±1,0 p<0,001
Uric acid clearance (ml/min/1.73m2)	8,4±0,8	14,6±1,2 p>0,05	16,1±0,9 p<0,05
Phosphorus clearance (ml/min/1.73m2)	10,4±0,8	16,0±0,4 p<0,05	18,1±2,0 p<0,001
Ammonia (mol/milk)	88,2±9,2	76,2±6,7 p<0,05	62,6±5,1 p<0,001
Titratable acid (mol/milk)	56,5±4,1	34,6±2,6 p<0,05	28,0±4,5 p<0,001
Uric acid (mol/l)	46,6±2,4	36,2±2,7 p<0,001	31,4±2,5 p<0,001
Fractional clearance of uric acid (%)	3,4±0,25	4,6±0,5 p<0,05	6,5±0,6 p<0,001
	8,7±0,4	12,0±0,8 p>0,05	20,5±1,2 p<0,001

The concentration function of the kidneys is evaluated by the Zimnitsky test. A more accurate assessment of this function can be carried out by eating dry products. However, due to the possibility of developing dehydration and increased blood glucose in patients with QD, it was not possible to transfer. The average weight of the studied groups is presented in (table 3.2).

Table 3 Maximum specific gravity of urine according to the Zimnitsky test in type I QD

	Group I	Group II	p
Average index of maximum specific gravity	1028±3,8	1020±4,2	>0,05
Daily diuresis (ml/milk)	1324±134,0	1408±92,0	>0,05

According to Table 3, patients with uricosuric nephropathy tend to increase diurnal diuresis and decrease specific gravity, but both cases are not statistically significant (r>0.05). When the groups were compared with the main clinical-laboratory parameters, in patients with uricosuric nephropathy compared to the first group, the level of glycemia in the morning was reliably higher (9.3±0.2% and 6.3±0.1%, r<0.001), glycosylated hemoglobin (7 ,9±0.2 and 6.0±0.1%). Clear changes were detected in the chemical composition of blood and urine. In patients with urate nephropathy, diuresis is significantly reduced (r<0.001), which is more evident when abacterial and bacterial interstitial nephritis are added, accordingly, it was found that although the glomerular filtration rate at this stage of QD is high, the specific gravity of urine is relatively low (table 3.3).

Table 4 Characteristics of urine composition and partial kidney function in children with QD with hyperuricemia (M±m)

Group indicators	Healthy ones (N=16)	Group I (N=20)	Group II (N=30)
Creatinine clearance (ml/min/1.73)	108,6±4,8	160,0±6,3	156,4±12,3p<0,001
Water Reabsorption(%)	98,2±0,06	96,6±0,11 p>0,05	96,6±0,2p<0,05
Urates (mmol/milk)	3,16±0,38	5,6±0,5 p<0,001	6,5±0,6 p<0,001
Oxalates (mmol/milk)	0,36±0,04	0,57±0,05 p<0,05	0,74±0,06 p<0,001
Phosphorus clearance (ml/min/1.73m2)	10,4±0,8	16,0 ±4,3	18±5,0 p<0,001
Calcium clearance (ml/min/1.73m2)	0,82±0,04	1,6±0,8 p>0,05	2,1±1,3 p<0,001
Calcium (mg/milk)	31,3±3,5 p<0,05	74,6±2,2 p<0,05	76,8±1,2 p<0,05
Phosphorus (mg/milk)	478,7±9,1	575,4±7,0 p<0,05	610,0±54 p<0,001
Ammonia (mmol/milk)	56,5±8,3	36,0±4,6 p<0,05	22,0±4,5 p<0,001

According to Table 4, the daily excretion of urates in children with hyperuricemia and hyperuricosuria of the first type QD was 2.5-3 times higher than that of healthy children (5.6±0.5-6.5±0.6mmol/milk, in healthy children 3.16- 0.38 mmol/day; s<0.001), more than half of these patients have hyperoxaluria (0.57±0.005-0.74±0.06 mmol/day, 0.36-0.04 mmol/day in healthy children, r<0.001). Creatinine clearance is high (r<0.001, water reabsorption in renal tubules is not significantly changed (0.05), but tubular reabsorption of phosphorus is reliably reduced when compared with healthy children (r<0.001). A decrease in ammonia acidogenetic function of the kidneys is characterized by a decrease in ammonia excretion and titratable acids (r<0.001). Overpayment of nephrotoxic metabolites (urates, oxalates, calcium) was found in biological fluids, which may cause additional stress to all parts except for their nephrotoxic effect, and may be a factor accelerating the formation of dysmetabolic and later diabetic nephropathy. The obtained data confirm that QD is hyperuricemia, hyperuricosuria, and urate nephropathy are common in children. The onset of the disease is mild, and the course is relatively positive. However, if it is not diagnosed early, i.e., if purine metabolism is not corrected by diet and medication, the process may become chronic (interstitial nephritis, kidney (including microbial inflammatory process, oxidative stress, DN and membrnolysis as a result of chronic renal failure) can be observed.

Conclusion

As a result of the above investigations, it can be said that in nephropathies caused by diabetes, some substances include; increased clearance of calcium, phosphorus, ammonia, creatinine, decreased water reabsorption, increased urate and oxalate salts in urine. This, in turn, leads to an increase in the symptoms of the disease and an increase in the rate of the disease. Taking into account the type of disease, it is difficult to stop dysmetabolic changes, but we can conclude that it can be prevented by timely dietary and drug correction.

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