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CLINICAL CASE OF A PATIENT WITH DISTAL RENAL TUBULAR ACIDOSIS WITH HEARING LOSS

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Abstract. Distal renal tubular acidosis with deafness (OMIM 267300) is a rare disease characterized by severe metabolic acidosis due to impaired excretion of hydrogen ions in the distal nephron. Violation of physical and psychomotor development, deformation of limbs, electrolyte disturbances without proper and timely treatment can lead to disability at a very early age. The article describes a clinical case that reflects the complexity and insidiousness of diagnosing distal renal tubular acidosis in a child of the first year of life.

Key words: *distal renal tubular acidosis; children; tubulopathy; sensorineural hearing loss.*

КЛИНИЧЕСКОЕ НАБЛЮДЕНИЕ ПАЦИЕНТА С ДИСТАЛЬНЫМ РЕНАЛЬНЫМ ТУБУЛЯРНЫМ АЦИДОЗОМ С ТУГОУХОСТЬЮ

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Резюме. Дистальный ренальный тубулярный ацидоз с глухотой (OMIM 267300) — это редкое заболевание, характеризующееся тяжелым метаболическим ацидозом вследствие нарушения экскреции водородных ионов в дистальном отделе нефрона. Нарушение физического и психомоторного развития, деформация конечностей, электролитные нарушения без должного и своевременного лечения могут приводить к инвалидизации уже в самом раннем возрасте. В статье описан клинический случай, отражающий сложность и коварность диагностики дистального ренального тубулярного ацидоза у ребенка первого года жизни.

Ключевые слова: *дистальный ренальный тубулярный ацидоз; дети; тубулопатия; нейросенсорная тугоухость.*

Distal renal tubular acidosis (DRTA) is a heterogeneous group of genetic diseases characterized by impaired urinary acidification, leading to severe metabolic hyperchloremic acidosis with elevated urinary pH, hypokalemia, hypercalciuria and nephrocalcinosis [1–3]. Clinical features include delayed height and weight, rickets, nephrocalcinosis, polyuria, polydipsia and the possible development of chronic renal failure in patients not receiving therapy [4–7].

There are primary and secondary causes of the development of distal renal tubular acidosis. Primary diseases include genetically determined pathology of transport systems with an autosomal dominant or autosomal recessive type of inheritance.

- Autosomal dominant, mutation of the *SLC4A1* gene (chromosome 17q21-22), disruption of the structure of chloride-bicarbonate antiporter-1 (AE-1 — anion exchager 1) of the basolateral membrane of the cortical collecting ducts.
- Autosomal recessive with hearing loss, mutation of the *ATP6V1B1* gene (chromosome 2p13), disruption of the structure of the B1 subunit of the hydrogen ATPase of intercalary cells of population A of the apical membrane of the cortical collecting ducts.
- Autosomal recessive without hearing loss, mutation of the ATP6V0A4 gene (chromosome 7q33-34), encoding the alpha-4 subunit of the hydrogen ATPase of intercalary cells of population A of the apical membrane of the cortical collecting ducts [1, 2, 4].

Autosomal recessive forms of distal renal tubular acidosis are rare diseases with unknown prevalence in the population [7]. The presence or absence of hearing impairment is the main phenotypic criterion to narrow the genetic search to confirm the diagnosis. Causal mutations in the ATP6V1B1 gene are classically associated with early-onset sensorineural hearing loss, but cases of tubular acidosis with early-onset deafness have also been described in patients with ATP6V0A4 gene mutations. Along with familial forms of the disease, sporadic cases are also found.

Secondary (acquired) forms of distal RTA are described in many pathological conditions caused by disorders of calcium metabolism with nephrocalcinosis and hypercalciuria, primary hyperparathyroidism, drug and toxic damage, other renal diseases, including medullary cystic disease and sponge kidney, autoimmune diseases (hypergammaglobulinemia, Sjogren's syndrome, autoimmune hepatitis, thyroiditis, fibrosing alveolitis, systemic lupus erythematosus, periarteritis nodosa).

The violation of ammonium excretion in all cases is secondary. A bicarbonate reabsorption is quantitatively normal, but, consistent with the increase in urine pH, a certain degree of bicarbonaturia is necessarily present. In severe chronic metabolic acidosis, the neutralization of hydrogen ions occurs due to bone carbonate, which causes the release of calcium from the bone into the extracellular fluid, which leads to disruption of its normal structure and a variety of bone deformities. A citrate excretion in the proximal tubule is reduced, which is the basis for the formation of nephrocalcinosis.

CLINICAL PICTURE OF DISTAL RENAL TUBULAR ACIDOSIS

The disease more often manifests between the age of six months and two years. Usually the DRTA is characterized by nonspecific symptoms: delayed weight and height, rachitic-like skeletal changes, muscle hypotonia, polyuria, polydipsia.

LABORATORY AND INSTRUMENTAL METHODS FOR DIAGNOSIS OF DISTAL RENAL TUBULAR ACIDOSIS

Blood acid-base state. One of the main criteria for laboratory diagnosis is the identification of metabolic acidosis, due to a decrease in standard bicarbonate (HCO3–) in plasma < 15 mmol/l.

Urine analysis. The most characteristic is an alkaline reaction of urine (pH > 6.0); a decrease in the excretion of citrate and ammonium is also possible [3].

Biochemical blood test. Changes in blood chemistry for DRTA are nonspecific, but increases in alkaline phosphatase, parathyroid hormone, renin, and aldosterone levels may occur.

Genetic research. For the distal renal tubular acidosis, an autosomal dominant type of inheritance with a mutation in the *SLC4A1* gene (chromosome 17q21-22), as well as autosomal recessive types with a mutation in the *ATP6V1B1* (chromosome 2p13) and *ATP6V0A4* genes (chromosome 7q33-34) are possible.

Ultrasound. The distal renal tubular acidosis is characterized by echo signs of nephrocalcinosis ("hyperechoic pyramid syndrome") according to the results of ultrasound of the kidneys, as well as urolithiasis (the composition of urinary stones is calcium phosphate) [1, 2].

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In order to assess the severity of rachitic changes in the skeleton, it is recommended to conduct X-ray of the hands, determine bone age, densitometry, and X-ray of the tubular bones of the legs including the knee joints.

DIFFERENTIAL DIAGNOSTICS

Genetically determined forms of distal RTA (type I) must be differentiated from proximal RTA (type II), including those in the de Tony-Debre-Fanconi composition syndrome, pseudohypoaldosteronism, primary hyperoxaluria and some other variants of nephrocalcinosis.

TREATMENT OF DISTAL RENAL TUBULAR ACIDOSIS

According to clinical recommendations [1, 2], treatment of RTA II in children consists of using a 4% sodium bicarbonate solution or a citrate solution at a dose of 10–15 mmol/kg per day in several doses to compensate for metabolic acidosis.

Since untimely diagnosis of the disease significantly worsens the quality of life of patients, and in some cases can cause death, the team of authors considers it necessary to pay the attention of colleagues to the presence of such a pathology as distal renal tubular acidosis, and presents a description of a clinical case.

CLINICAL CASE DESCRIPTION

Patient V., 4 months old, was admitted for the first time as planned to the nephrology department of St. Petersburg State Budgetary Healthcare Institution Children's City Hospital № 2 of St. Mary Magdalene.

Anamnesis morbi. A screening ultrasound of the urinary system at 2 months revealed echo signs of nephrocalcinosis ("hyperechoic pyramid syndrome"). At 4 months bilateral sensorineural hearing loss was detected: degree III on the right, degree IV on the left.

Anamnesis vitae. A child from the third pregnancy, third birth. Labor at 39 weeks, Apgar score 8/9. Body weight at birth was 3560 g, body length was 53 cm. The mother suffered a new coronavirus infection (COVID-19) and received a course of nasal glucocorticosteroids at the 8th week of pregnancy. Heredity, according to the mother, is not burdened. The elder children (boy — 8 years old, girl — 4 years old) are healthy.

Objective examination data upon admission to the hospital. The body length was 62 cm, body weight was 5500 g. The condition is satisfactory. A psychomotor development corresponds to age. The skin is naturally colored and clean. There was insufficiently of subcutaneous fat layer. A hypotonia of the lower extremities is noted. Heart sounds are clear and rhythmic. In the lungs, breathing is puerile, carried out in all sections. There was no wheezing. The abdomen is moderately swollen, accessible to deep palpation. The liver is + 1,5 cm from under the edge of the costal arch. The spleen is not palpable. The stool is mushy, 3–4 times a day, without pathological impurities. The urination is freely into the diaper.

Data from laboratory and instrumental examination methods.

The clinical blood test is in the age norm. No evidence of mineral metabolism disorders was identified (blood electrolytes are normal; urine calcium/creatinine, phosphorus/creatinine ratios are within normal limits). A general urine test revealed an alkaline reaction, without changes in urinary sediment. No disturbances in the acid-base status (ABS) of the blood were detected. The hormonal profile showed a decrease in the level of parathyroid hormone to 9,63 pg/ml, an increase in the level of renin (> 500.00 μIU/ml, reference values for renin for children under 1 year of age have not been established) and an increase in the level of aldosterone to 935,3 pg/ml , without clinical manifestations. The thyroid status, 17-OH-progesterone is normal. A nitrogen excretion function of the kidneys is satisfactory. A blood pressure is within target values.

An ultrasound of the urinary system showed echo signs of "hyperechoic pyramid syndrome", without negative dynamics.

The child was consulted by an endocrinologist; no evidence of endocrinological pathology was found at the time of examination. Then he consulted by a geneticist: taking into account bilateral sensorineural hearing loss, blood was drawn to study the mutation in the GJB2 gene and blood amino acids — in work at the time of discharge.

The child was discharged with the main diagnosis; nephrocalcinosis, and concomitant: bilateral sensorineural hearing loss — degree III on the right, degree IV on the left. The dynamic observation and control examination after 3 months were recommended.

A repeated hospitalization in August 2022. At the time of hospitalization, the child was 10 months old. The llow weight and height gains were observed over time. The hearing replacement was performed at 6 months.

Since July 2022, a polydipsia and polyuria have been noted. During control studies, the level of adrenocorticotropic hormone (ACTH) and aldosterone remained elevated. According to repeated monitoring, no electrolyte disturbances were detected. In urine tests over time, a persistent shift in urine pH towards the alkaline side is noted. When monitoring blood pressure, episodes of blood pressure increases up to 110/60 mmHg were recorded. According to the results of a genetic examination (from January 2022), disorders of amino acid metabolism were excluded, mutations in the GJB2 gene were excluded. Hearing correction was performed in 6 months age.

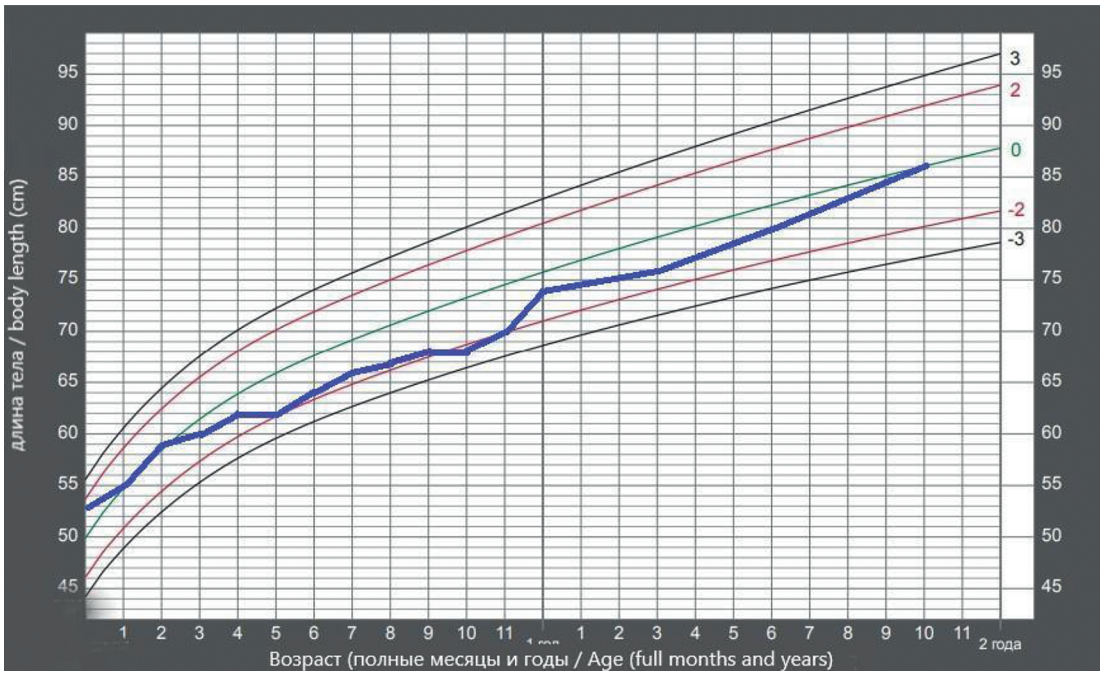

Fig. 1. Patient growth chart from birth to 22 months
Рис. 1. График динамики роста пациента от рождения до 22 месяцев

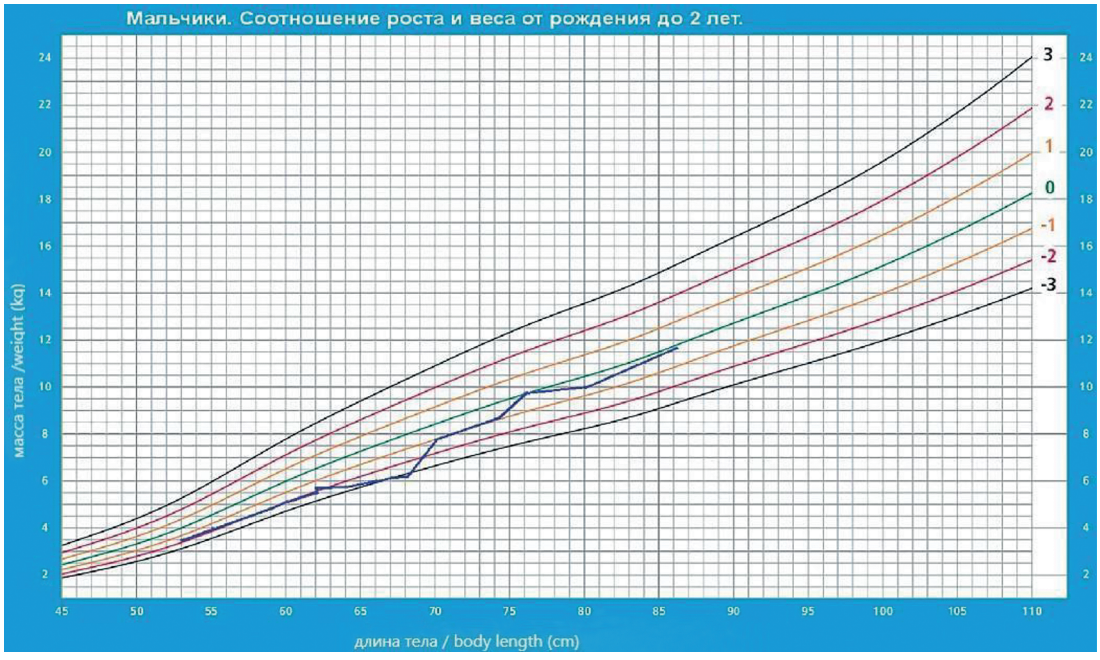

Fig. 2. The ratio of the patient's body length and body weight from birth to 22 months
Рис. 2. Соотношение длины тела и массы тела пациента от рождения до 22 месяцев

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Table 1. Laboratory indicators of the patient over time

Таблица 1. Лабораторные показатели больного в динамике

	Кислотно-основное состояние капиллярной крови / Acid-base state of capillary blood				
	01.2022	**08.2022**	**12.2022**	**03.2023**	**06.2023**
pH	7,45	7,30	7,43	7,46	7,43
Напряжение CO_2, мм рт.ст. / CO_2 tension, mmHg	46,6	21,5	32,4	33,3	34,6
Напряжение O_2, мм рт.ст. / O_2 tension, mmHg	65	84	78	77	69
Лактат / Lactate	–	2,43	–	–	–
Гематокрит, % / Hematocrit, %	39	33,0	33,0	–	36,0
Концентрация Na^+, ммоль/л / Na+ concentration, mmol/l	138	132	138	139	137
Концентрация K^+, ммоль/л / K^+ concentration, mmol/l	4,5	3,0	3,8	4,5	4,6
HCO_3^-, ммоль/л / HCO_3^-, mmol/l	32,4	10,5	21,6	23,7	23,1
cBase (B), ммоль/л / cBase (B), mmol/l	4	–14,0	–2,0	0	–1
$ctCO_2$ (B), ммоль/л / $ctCO_2$ (B), mmol/l	34	11,0	23,0	25,0	24,0
sO_2, %	93	95,0	96,0	96,0	94,0
	Общий анализ мочи / General urine analysis				
Кислотность / Acidity	Щелочная / Alkaline	Щелочная / Alkaline	Нейтральная / Neutral	Слабощелочная / Slightly alkaline	Нейтральная / Neutral
Удельный вес / Specific gravity	1025	1020	1018	1020	1015
Белок / Protein	0	0	0	0	0
Глюкоза / Glucose	0	0	0	0	0
Эпителий плоский / Flat Epithelium	2–3	0–1	1–2	2–3	1–2
Лейкоциты / Leukocytes	5–7	2–3	3–5	2–3	0–1
	Биохимический анализ крови / Biochemical blood test				
Ренин, мкМЕ/мл / Renin, μIU/ml	>500,00	>500,00		84	28
Альдостерон, пг/мл / Aldosterone, pg/ml	953,3	158,5		147,5	
Паратгормон, пг/мл / Parathyroid hormone, pg/ml	9,63	24,41		26,8	
Общий белок, г/л / Total protein, g/l	72	67	66	62	67
Альбумин, г/л / Albumin, g/l	53	46	43	42	45
Мочевина, ммоль/л / Urea, mmol/l	4,8	4,5	7,0	6,7	3,9
Глюкоза, ммоль/л / Glucose, mmol/l	6,2	4,9	4,6	4,8	3,8
Мочевая кислота, ммоль/л / Uric acid, mmol/l	200	157	165	183	248
Креатинин, мкмоль/л / Creatinine, μmol/l	29	27	25	27	30
Кальций общий, ммоль/л / Total calcium, mmol/l	2,.74	2,58	2,72	2,44	2,67
Фосфор, ммоль/л / Phosphorus, mmol/l	2.00	1.24	2,53	1.89	1.53
Железо, мкмоль/л / Iron, μmol/l	13.9	7.0	8,6	36	18.8
Ферритин, мкг/л / Ferritin, μg/l	56	54	12	6,3	
Калий, ммоль/л / Potassium, mmol/l	4.50	3.7	3.8	4,6	4.9
Натрий, ммоль/л / Sodium, mmol/l	135	132	138	137	139
Хлор, ммоль/л / Chlorine, mmol/l	107	107	109	106	108
Аспартатаминотрансфераза, Ед/л / Aspartate aminotransferase, U/l	36	40	24	28	
Аланинаминотрансфераза, Ед/л / Alanine aminotransferase, U/l	24	13	15	20	
Витамин D, 25-гидрокси (кальциферол), нг/мл / Vitamin D, 25-hydroxy (calciferol), ng/ml	31,32				
17-гидроксипрогестерон (17-ОПГ), нг/мл / 17-hydroxyprogesterone (17-OPG), ng/ml	0,85				

Objective examination data upon admission to the hospital. The height was 68 cm and weight was 6,1 kg (dynamics of physical development are presented in Fig. 1, 2). There is a delay in psychomotor development within two months (assessed using the Griffiths scale). The condition is satisfactory. The skin is naturally colored, dry, and there is a decrease in skin turgor. The subcutaneous fat layer is not sufficiently expressed in the abdomen and thighs. Muscular hypotonia (hypotonia of the lower extremities, "frog belly") is noted. Heart sounds are clear and rhythmic. SS at the top. In the lungs, breathing is puerile, carried out in all sections. No wheezing was detected. The abdomen is moderately swollen, accessible to deep palpation. The liver is at the edge of the costal arch. The spleen is not palpable. The stool is mushy, 1–2 times a day, without pathological impurities. The urination is freely into the diaper.

Data from laboratory and instrumental examination methods.

The clinical blood test is within the age norm. In the urine analysis, a shift in the pH of urine to the alkaline side. The urine Ca/Cr ratio is hypercalciuria. The urine P/Cr ratio is normal. Blood ABS is metabolic acidosis (data are presented in Table 1). Biochemical blood test shows hypokalemia. Kidney function is satisfactory. The level of cortisol, aldosterone, parathyroid hormone, ACTH is normal. A thyroid status — subclinical hypothyroidism. When monitoring hydrobalance, polydipsia is noted up to 1800 ml/day, polyuria up to 1900 ml/day. An ultrasound of the urinary system shows echo signs of "hyperechoic pyramid syndrome", without negative dynamics. Taking into account clinical and laboratory data (low height and weight gain, bilateral sensorineural hearing loss, nephrocalcinosis, persistent shift in urine pH to the alkaline side, metabolic acidosis, polydipsia, polyuria, hypercalciuria, hypokalemia), the diagnosis was interpreted as distal renal tubular acidosis with hearing loss. An exome sequencing has been carried out — in progress. In therapy, a citrate solution (blemaren at the rate of 5 mmol/kg per day for citrate), ACE inhibitor therapy (Enap 2,5 mg/day), against this background, in the 2nd week there was a normalization of ABS, blood electrolytes, a decrease in the volume of consumed and excreted liquids.

The third hospitalization was in October 2022, against the background of therapy with a citrate solution. The positive dynamics in weight and height indicators were noted, the data are presented in Fig. 1, 2.

The laboratory data at the time of hospitalization: capillary blood ABS, ionogram, biochemical and clinical blood tests were within the reference values (the results of laboratory data are presented in Table 1). A general urinalysis reveals a slightly alkaline urine reaction. Based on the results of exome sequencing, a heterozygous ATP6V1B1 mutation was identified, which confirms the previously made clinical diagnosis.

Readmissions in December 2022, March and July 2023. During each of the hospitalizations, compensation of metabolic acidosis was noted (laboratory data are presented in Table 1) and the absence of clinical manifestations, satisfactory weight and height indicators (Fig. 1, 2).

CONCLUSION

The diagnosis of tubulopathy, including renal tubular acidosis, is a rather difficult task for a pediatrician and nephrologist, since in practice it is quite difficult to establish the type of disease, the described clinical case proves this. Thus, taking into account clinical and instrumental data, including general signs of distal renal acidosis and the presence of nephrocalcinosis and sensorineural hearing loss in the boy, the clinical diagnosis of DRTA was confirmed genetically (heterozygous variant ATP6V1B1). A timely treatment started and leaded to compensation of metabolic acidosis with use of citrate solution, and timely hearing aids for the patient made it possible to prevent retardation of physical and psychomotor development.

ADDITIONAL INFORMATION

Author contribution. Thereby, all authors made a substantial contribution to the conception of the study, acquisition, analysis, interpretation of data for the work, drafting and revising the article, final approval of the version to be published and agree to be accountable for all aspects of the study.

Competing interests. The authors declare that they have no competing interests.

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Consent for publication. Written consent was obtained from the patient's legal representatives for publication of relevant medical information within the manuscript.

ДОПОЛНИТЕЛЬНАЯ ИНФОРМАЦИЯ

Вклад авторов. Все авторы внесли существенный вклад в разработку концепции, про-

ведение исследования и подготовку статьи, прочли и одобрили финальную версию перед публикацией.

Конфликт интересов. Авторы декларируют отсутствие явных и потенциальных конфликтов интересов, связанных с публикацией настоящей статьи.

Источник финансирования. Авторы заявляют об отсутствии внешнего финансирования при проведении исследования.

Информированное согласие на публикацию. Авторы получили письменное согласие законных представителей пациента на публикацию медицинских данных.

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