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Quadriplegia in Accidently Diagnosed Distal Renal Tubular Acidosis with Pneumonia: Case Report

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ABSTRACT

Distal renal tubular acidosis (RTA1) is a rare disease. It confirms the lab's data and rules out other differential diagnoses. Late evaluation can interfere with healthy development. We explain a 38-year-old patient with a late and accidental RTA1 diagnosis. Correct treatment with alkali consumption and potassium citrate improves her general condition and eliminates further weakness attacks. Early diagnosis is crucial for patient development and morbidity avoidance with accurate treatments.

Introduction

Renal tubular acidosis (RTA) is an uncommon disease that refers to impaired filtered HCO3 reabsorption or acid excretion in the kidney, which disturbs normal acid-base homeostasis. RTA is divided into four different groups: proximal, distal, mixed, and hyporeninemic hypoaldosteronism [1].

Distal RTA (type 1) pathophysiological process is based on alpha-intercalated cell disability to eliminate additional hydrogen ions and potassium maintenance, resulting in hypokalemia and metabolic acidosis. Proximal RTA (type 2) occurs when proximal tubules are unable to reabsorb bicarbonate ions. It is accompanied by glycosuria and phosphaturia, similar to Fanconi syndrome. The clinical manifestations are similar to distal RTA; they are distinguished from each other by urine pH. The third subtype contains properties of both types one and two; it is rarer than the other main groups. Hyperkalemic RTA (type 4) results from aldosterone deficiency or receptor insensitivity [2].

RTA's complex conditions and various manifestations, as mentioned, have made diagnosis more difficult. Late diagnosis causes more complications and comorbidities.

The primary reason for distal RTA is associated with genetic mutations and systemic diseases, resulting

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in the secondary type, which can occur at any age, but the primary type is mostly diagnosed at younger ages. We introduce a rare new case of a 38-year-old woman with distal RTA and diabetes insipidus with viral pneumonia and other related comorbidities and complications [3].

Case Presentation

In May 2022, a 38-year-old woman came into the emergency department (ED) with a high fever, cough, dyspnea, and myalgia. She was admitted to the emergency ward for further evaluation. Her vital signs on arrival at the ED were as follows: HR of 110/min, temperature of 38.5 degrees Celsius, RR of 19/min, and SpO2 of 91% in room air. Physical examinations revealed coarse crackles with a sound reduction on auscultation at the base of the lungs.

Her past medical history revealed no underlying disease. She had been hospitalized several times due to weakness and was discharged after 24–48 hours without any definite diagnosis. The patient mentioned muscle weakness and fatigue over one hour after admission. Primary lab tests showed a low serum potassium level (Table 1). Her treatment began with a potassium chloride solution for hypokalemic periodic paralysis. However, despite several doses of intravenous potassium chloride solution with normal saline 0.9%, serum potassium wasn't corrected, and she suffered from flaccid quadriplegic paralysis. A nephrology consult was requested to correct electrolyte disturbances. Her condition worsened, and she was intubated for ventilation in the intensive care unit (ICU). A CSF sample was collected to measure absolute cell counts; the total protein and glucose cultures of the CSF specimen were negative (Tables 1, 2). Pneumonia treatment was started with antibiotics and oseltamivir according to a positive influenza PCR and evidence from a spiral chest CT scan without contrast. The nephrology consult suggested a diagnosis of distal renal tubular acidosis based on a high anion gap, metabolic acidosis, high urine pH, Cl, and previous medical records. She was treated with intravenous sodium bicarbonate, and her metabolic acidosis was corrected. Serum potassium increased due to metabolic acidosis modifications (Table 3). A few hours after treatment, her daily urine volume increased, presenting polyuria and hypernatremia. Serum and urine osmolality suggested nephrogenic diabetes insipidus. The water deficit was compensated with free water, dextrose 5% solution, and desmopressin. As her general condition improved, the patient was extubated. An ultrasound of the kidneys was done after transferring to the ward. The pathology report is as follows: kidneys are normal in size and echo pattern. The right kidney was 10.3 cm, and the left one was 10.2 cm; no hydronephrosis was seen. The urinary bladder is normal in size and shape. Left kidney stones 4mm, 9mm, and 10mm in size were seen in the

Table 1. Patient lab data to confirm and rule out other diagnosis

Test	Result	Reference value
BS	159	140-200 mg/dl
BUN	17	7-18 mg/dl
CR	1.3	0.6-1.3 mg/dl
NA	143	135-145 mEq/L
K	2.5	3.5-5.1 mEq/L
ESR	45	UP TO 20 mm/h
AST	40	<31 U/L
ALT	16	<31 U/L
ALKP	129	64-306 IU/L
TOTAL.B	1.3	0.1-1.2 mg/dl
DIRECT	0.2	<0.3 mg/dl
ALB	3	3.5-5.5 g/dl
CA	8.5	8.6-10.3 mg/dl
MG	2.1	1.7-2.2 mEq/L
Urine Citrate	55	150-450 mg/24h
P	4.5	2.6-4.5 mg/dl
CSF GLU	86	50-80 mg/dl
CSF LDH	107	<40 U/L
CSF Pr	170	15-60 mg/dl
TSH	1.3	0.3-4.7 µIU/ML
T4	4.5	4.5-12.6 µg/dL
Ferritin	41	4-104 ng/ml
Urine URICACID	232	250-700 mg/24h
C3	140	90-180
C4	20	10-40

Table 2. Patient results to confirm and rule out other diagnosis

CSF CULTURE	NEG
ANA	NEG
Anti-ds-DNA	NEG
Anti RO	NEG
Anti La	NEG
RF	NEG
Anti CCP	NEG
P/C/ANCA	NEG
wright	NEG
Coombs/wright	NEG
HIV Ab	NEG
HBS Ag	NEG
HCV Ab	NEG

Table 3. The course of the patient's VBG

VBG	The first day	3 days after treatment	The day of discharge
PH	7.03	7.11	7.39
PCO2	47	31.8	37.2
HCO3	11	9.7	22.5

pelvicalyceal system. The diagnosis of distal renal tubular acidosis was confirmed. Potassium citrate and sodium bicarbonate were prescribed. Patient evaluation after six months reported no recurrence of previous symptoms, and her general condition was noticeably good. Her follow-up continued.

Discussion

Distal renal tubular acidosis is the most common group of tubular acidosis, which is an infrequent disease that can happen at any age during a patient's life span. Distal renal tubular acidosis was an accurate diagnosis in this case. Optimal management occurs when considering alkaline urine pH, urinary acid, and alkaline secretion. Patient treatment was challenging and required potassium solution and Na bicarbonate together [4].

Decreased oxalate, fructose, animal protein, vegetable, and Na consumption benefits patients with distal RTA and nephrolithiasis. However, recent studies suggest that using foods high in potassium and restricting the intake of acid-generating food supplements can modify serum acidosis. Dehydration, sepsis, nausea, vomiting, and exacerbations of acid-base imbalance can occur; the first presentation may also be different based on the patient. Hypokalemic periodic paralysis was inaccurately diagnosed at first, playing an important role in containing treatment, and may result in worsening hypokalemia [5,6].

Other secondary reasons for distal RTA, such as autoimmune diseases, are ruled out. Clinical association with electrolyte changes should be interpreted for a well-informed response to choose treatment before life-threatening complications occur [7,8]. Treatment started with potassium citrate and Na bicarbonate to prevent wasting potassium. The patient had recurrent hospitalizations secondary to distal RTA, and the main problem was easily underdiagnosed with an inaccurate approach. A good response to alkali consumption replacement occurred; however, nephrogenic diabetes insipidus has been noted due to aquaporin 2 channel dysfunction in hypokalemia [9].

In the six-month follow-up after receiving the correct treatment, the general condition of the patient had clearly improved.

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Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this article.

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Conflict of Interests

The authors declare that they have no conflict of interests.

Consent statement

Written informed consent for publication was obtained from the patient in accordance with the journal's patient consent policy.

Ethical statement

For publishing this case report, we asked Rasoul Akram hospital ethical committee for approval.

Authors' contributions

All of the authors responsible for gathering data and writing the manuscript. Dr. Maryam Ghaffari Rahbar is the lead nephrologist who suggest the correct diagnosis.

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