Case Report

Journal of Emergency Medicine Case Reports

Hypokalemic Paralysis Due to Distal Renal Tubular Acidosis, Case Report

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Abstract

Distal renal tubular acidosis (dRTA) is a metabolic disease characterized by hypokalemia, hyperchloremic metabolic acidosis and urine pH above 5.5. These findings may be accompanied by hypercalciuria, nephrocalcinosis, nephrolithiasis, jaundice, osteomalacia or rickets in children. Although hypokalemia is frequently seen as a laboratory finding in dRTA, weakness, which is the clinical finding of this deficiency, is rare. A 33-year-old female patient was brought to the emergency department (ED) with complaints of weakness, loss of strength in the extremities, and difficulty in breathing. Laboratory analyzes of the patient revealed metabolic acidosis and hypokalemia. Urea and creatinine values were normal. The patient was admitted to the internal medicine department with a preliminary diagnosis of dRTA and hypokalemic paralysis. Initially, parenteral infusion of KCl and NaHCO3 was administered in the treatment. In the follow-up of the patient, it was observed that hypokalemia and metabolic acidosis improved from the 3rd day and clinical findings improved within 36 hours following the replacement therapy. dRTA, which is rare in adults, is among the secondary causes of hypokalemic paralysis. dRTA should be considered among the differential diagnoses in the presence of hypokalemia and metabolic acidosis in patients presenting with bilateral weakness.

Keywords: Renal tubular acidosis, hypokalemia, paralysis

Introduction

Hypokalemic paralysis is an uncommon, life-threatening syndrome with widespread muscle weakness, including respiratory muscles, and hypokalemia. If it is diagnosed and treated appropriately, it resolves without sequelae (1). Periodic paralysis is distinguished by a normal potassium level, except for attacks. In the presence of normal anion gap metabolic acidosis, renal tubular acidosis (RTA) should be considered among the differential diagnoses. In this case, secondary diseases that may cause RTA should also be investigated. RTA Type 1 and 2 are seen with hypokalemia, while RTA Type 4 is associated with hyperkalemia. Clinical findings may present with nonspecific findings such as constipation, weakness, growth retardation in childhood, nausea-vomiting, polyuria-dehydration, as well as presentations with complications related to the kidney and musculoskeletal system (2, 3). Complications associated with the kidney; nephrocalcinosis, urolithiasis (which may be the first initial finding in adults), chronic interstitial nephritis and severe hypokalemic crisis. During the hypokalemic crisis, the patient may apply to the emergency department (ED) with the symptoms of dehydration, shock, arrhythmia, vomiting, flaccid weakness, respiratory distress, drowsiness, and coma (4). Here, we present a female patient who presented with acute muscle weakness and dyspnea and was diagnosed with hypokalemic periodic paralysis due to distal RTA (dRTA), with clinical and laboratory findings.

Case Report

A 33-year-old female patient was brought to the ED by the 112 with complaints of weakness, loss of strength in the arms and legs, and difficulty in breathing. The patient, who has a history of type 2 diabetes mellitus and hypothyroidism, was using metformin 2x850 mg and levothyroxine 50µgr/day. The patient had no smoking or alcohol use. There was no pregnancy history. In her anamnesis, it was learned that she had previously received a vitamin B12 ampoule injection due to numbness in her arms. It was learned that the patient felt weakness and numbness especially in his upper extremities for 2-3 days, and she admitted to the orthopedics outpatient clinic with the thought of nerve compression. Blood gas analysis revealed pH: 7.15, pCO2: 32 mmHg, HCO3: 10.9 mmol/L, base excess (BE): -16.8 mmol/L, potassium (K): 2.02 mmol/L, ionized calcium (Ca): 1.39 mmol/L, chloride (Cl):115 mmol/L. In laboratory findings, hemoglobin 13.88 gr/dL, hematocrit 41.9%, white blood cell 14.400/mm3, platelet 358000/mm3, glucose 119 mg/dL, urea 36 mg/dL, creatinine 0.69 mg/dL, sodium (Na) 138 mmol/L, K:

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Received: 07.06.2023 • **Accepted:** 13.11.2023
DOI: 10.33706/jemcr.1310866
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Cite this article as: Karaarslan FN. Hypokalemic paralysis due to distal renal tubular acidosis, case report. Journal of Emergency Medicine Case Reports. 2023;14(4): 74-76

2.06 mmol/ L, Cl: 116 mmol/L, Creatine Kinase 574 u/L were detected and liver function tests were normal. In urine analysis; pH 7, protein (-), Na: 61 mmol/L, K: 10 mmol/L Cl: 75 mmol/L Ca: 9.2 mmol/L and urine density was 1002. The patient's electrocardiography (ECG) was normal. The patient was admitted to the internal medicine department with the preliminary diagnosis of dRTA and hypokalemic paralysis. Thyroid function tests, plasma renin, aldosterone values were within normal limits, Parathormone: 8.3 ng/L (15-68.3) low, vitamin B12: >2000ng/L high, anti-DNA, RF, AMA, ASMA, anti-SSA, anti-SSB, anti-Jo-1, CCP, c-ANCA, p-ANCA, anti-ds-DNA were negative, ANA was (++) granular appearance. No pathological finding was detected in urinary ultrasonography. In the treatment, a parenteral 80 mEq/day KCl and 150 mg/kg NaHCO3 infusion was administered initially. In the follow-up of the patient, it was observed that his hypokalemia and metabolic acidosis improved from the 3rd day. During the follow-up period, the potassium values are respectively; 2.7, 2.2, 2.6, 3, 3.51, 3.58 mmol/L were detected. After the initial KCl and NaHCO3 parenteral infusion, oral effervescent potassium tablet 2x40 mmol was administered. It was observed that clinical findings improved within 36 hours following the replacement therapy. Sodium hydrogen carbonate and oral potassium treatment containing potassium citrate and potassium bicarbonate were prescribed and she was discharged with the recommendations of rheumatology outpatient clinic control. An informed consent form was obtained from the patient for this case report.

Discussion

Potassium, which is the main cation of the cell, is of great importance in the stimulation of muscle, nerve and myocardial cells in addition to its many functions in the body (5). Hypokalemia, defined as a plasma potassium level below 3.5 mEq/L, is a common clinical problem. Findings vary between individuals. It rarely causes symptoms unless it falls below 3 mEq/L (4). Weakness, muscle aches and loss of strength in the lower extremities are common. Cerebrovascular diseases are considered primarily in patients presenting with paresis and loss of strength in the ED (6). However, in severe hypokalemia, clinical pictures ranging from progressive loss of strength, hypoventilation due to the involvement of respiratory muscles, and complete paralysis can be observed (7). The coexistence of hypokalemia and metabolic acidosis can be seen in diabetic ketoacidosis, and Type 1 and Type 2 RTA (7).

Hypokalemic periodic paralysis is a disease characterized by decreased serum potassium level due to primary and secondary causes and accompanying transient acute flaccid paralysis. Secondary causes include dRTA, although rare.

In Type 1 RTA, which is the distal type, there is an inability to remove the daily acid load. If alkali treatment is not applied, hydrogen ion accumulation gradually increases and the plasma bicarbonate concentration may fall below 10 mEq/L. Despite acidosis, the urine pH is above 5.5. Serum potassium levels are variable. Due to chronic acidosis, both bone resorption and renal tubular reabsorption of calcium are affected and hypercalciuria is observed. Therefore, nephrolithiasis and nephrocalcinosis often develop (3). In our patient, the urinary calcium level was also high. In type 2 proximal RTA, hypokalemic metabolic acidosis develops due to the defect in bicarbonate reabsorption in the proximal tubules. Since the functions of the distal tubules are normal, partial absorption of bicarbonate occurs and blood bicarbonate levels are higher than in type 1. It is usually associated with other proximal tubular reabsorption disorders. With the administration of bicarbonate, the urine pH becomes alkaline in type 2 RTA, while the absence of a significant change in urine pH in type 1 RTA may help in the differential diagnosis. Kidney stone formation is not seen in type 2. Type 3 RTA is characterized by hyperkalemic hyperchloremic acidosis. It develops due to aldosterone insufficiency or resistance to the effect of aldosterone in the tubules (8, 9). Our patient, who was examined from the beginning for hypokalemia and hyperchloremic metabolic acidosis, also has a urine pH above 5.5 and a high urinary calcium, which is compatible with Type 1 RTA. In some patients who presented with hypokalemic paralysis and were found to have dRTA, underlying causes were found to be rheumatoid arthritis, collagen tissue diseases such as systemic lupus erythematosus (sometimes with hyperkalemia), and the use of amphotericin B, lithium, ibuprofen or some plants (7,8,10). Therefore, all patients diagnosed with RTA should be investigated for other diseases that may cause RTA. Our patient's clinic and examinations are not compatible with collagen tissue diseases in terms of diseases that may cause dRTA, and he did not use any drugs or herbs. Failure to define family history caused the case to be evaluated as sporadic. Treatment is with bicarbonate and potassium replacement. With bicarbonate, the symptoms disappear. In addition, with treatment, the development of kidney failure is prevented or kept at the same level. Administration of 80-200 mg/kg (1-3 mEq/kg/day) NaHCO3 in the dRTA eliminates acidosis. As a matter of fact, bicarbonate and potassium replacement were applied in our patient and clinical findings improved.

Conclusion

Electrolyte disturbances, especially hypokalemia, should be considered in patients presenting to the ED with bilateral weakness. The possibility of dRTA, which is rare in adults, should be considered in cases presenting to the ED with paresis in the extremities with or without respiratory distress.

DOI: 10.33706/jemcr.1310866
Journal of emergency medicine case reports. 2023;14(4): 74-76

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