

Case Report

Persistent hypokalemia masking distal tubular acidosis and hypothyroidism: a case report

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ABSTRACT

Hypokalemic paralysis is an important reversible cause of acute flaccid weakness and may result from either intracellular potassium shift or true potassium depletion. Persistent hypokalemia associated with metabolic acidosis should be promptly evaluated for renal tubular disorders. We report a case of a 49-year-old man who presented with acute progressive quadriplegia and severe hypokalemia. Initial evaluation revealed serum potassium level of 1.5 mmol/l, metabolic acidosis, elevated creatine phosphokinase, renal potassium wasting, hypocalcemia with secondary hyperparathyroidism, and hypothyroidism. Despite potassium replacement, the patient demonstrated persistent hypokalemia ranging from 2.0-3.0 mmol/l and low serum bicarbonate levels across four months of outpatient follow-up. Urine studies and persistent acid-base abnormalities suggested an underlying distal renal tubular dysfunction. The patient improved clinically with potassium supplementation, supportive management with ongoing clinical monitoring during follow-up. This case highlights the importance of distinguishing hypokalemic paralysis secondary to chronic renal potassium wasting from isolated hypokalemic periodic paralysis.

Keywords: Hypokalemia, Quadriplegia, Distal renal tubular acidosis, Renal potassium wasting, Hypokalemic paralysis, Thyroid dysfunction

INTRODUCTION

Hypokalemic paralysis is characterized by episodes of acute muscle weakness associated with low serum potassium levels. It may result from intracellular potassium shifts, as seen in hypokalemic periodic paralysis, or from true potassium depletion due to gastrointestinal or renal potassium loss.^{1,2} Distal renal tubular acidosis (dRTA) is a relatively infrequent cause of hypokalemia and may present with acute neuromuscular manifestations.^{3,4}

Distal renal tubular acidosis results from impaired distal urinary acidification and is characterized by metabolic acidosis, hypokalemia, and inappropriately alkaline urine.^{3,5} Severe hypokalemia may present with acute flaccid paralysis, and rhabdomyolysis has also been

reported in association with dRTA.⁴ Early recognition is important because the condition is potentially reversible with appropriate electrolyte correction and treatment of the underlying disorder.

CASE REPORT

A 49-year-old man presented to the emergency department with a two-day history of progressive weakness involving both upper and lower limbs. The weakness was insidious in onset and progressively worsened, resulting in difficulty performing activities of daily living. There was no history of fever, seizures, sensory disturbances, involuntary movements, bowel or bladder dysfunction, palpitations, breathlessness, vomiting, diarrhoea, recent infections, or diuretic use. There was no family history of similar episodes.

His medical history was significant for type 2 diabetes mellitus and hypertension, for which he was receiving oral hypoglycemic agents and antihypertensive medications.

On examination, he was conscious, alert, and hemodynamically stable. Pulse rate was 66 beats/minute, blood pressure was 140/80 mmHg, and oxygen saturation was 98% on room air. General physical examination was unremarkable without pallor, icterus, cyanosis, clubbing, lymphadenopathy, or pedal edema.

Neurological examination revealed symmetrical flaccid quadriparesis power 1/5 in all 4 limbs, hypotonia involving both proximal and distal muscle groups with absent deep tendon reflexes. Sensory examination was normal. Cranial nerve examination was unremarkable, and there was no evidence of respiratory muscle involvement.

Investigations

Biochemical investigations demonstrated severe hypokalemia (1.5 mmol/l) with low bicarbonate (17.8 mmol/l) levels. Serum magnesium was 1.8 mg/dl. Calcium levels were low at 6.7 mg/dl. Serum CPK levels were raised at 2253 ng/ml indicative of severe rhabdomyolysis. Urine studies demonstrated inappropriate renal potassium loss. Arterial blood gas analysis revealed a pH of 7.30, pCO₂ 35.4 mmHg, and bicarbonate 21.8 mmol/l, suggestive of metabolic acidosis. Spot urine sodium was 48 mmol/l. Spot urine potassium measured 29.8 mmol/l. Urine osmolality was 241 mOsm/kg, and urine pH ranged from 6.0-7.0, remaining inappropriately alkaline despite low bicarbonate levels. The patient also had mildly elevated thyroid-stimulating hormone (TSH) level with normal free thyroxine (FT4) and free triiodothyronine (FT3) levels suggestive of subclinical hypothyroidism. Ultrasonography of the thyroid revealed a colloid nodule in the right lobe.

Serial follow-up investigations demonstrated persistent hypokalemia despite potassium replacement therapy. Serum potassium remained below the normal range during outpatient follow-up over the next four months. Serum bicarbonate also remained persistently low or low-normal, suggestive of chronic acid-base disorder.

The patient was admitted to the intensive care unit because of severe hypokalemia and the associated risk of cardiac arrhythmias. Intravenous potassium chloride replacement was initiated using 60 mmol of potassium chloride diluted in 500 ml of normal saline, infused at 85 ml/hour. Intravenous magnesium sulfate (2 g) was also administered, and continuous electrocardiographic monitoring was maintained throughout treatment.

Following electrolyte correction, the patient showed gradual neurological improvement. Muscle strength progressively improved over the subsequent days, allowing independent ambulation before discharge. Despite satisfactory neurological recovery, serial

outpatient investigations continued to demonstrate persistent hypokalemia, necessitating ongoing potassium supplementation and further evaluation for an underlying.

Table 1: Serial electrolyte trend demonstrating persistent hypokalemia with low bicarbonate levels during follow-up.

Date	Sodium (mmol/l)	Potassium (mmol/l)	Chloride (mmol/l)	Bicarbonate (mmol/l)
April (admission)	135	1.5	102	17.8
May	140	2.1	104	21
June	139	2.0	103	21.8
July	136	3.0	102	19
August	133	2.8	102	19

DISCUSSION

Hypokalemia presenting with acute flaccid paralysis requires differentiation between disorders caused by intracellular potassium redistribution and those resulting from true potassium depletion. The principal diagnostic considerations in this patient included hypokalemic periodic paralysis and renal potassium-wasting disorders such as distal renal tubular acidosis.¹

Hypokalemic periodic paralysis usually presents with episodic weakness associated with a transient intracellular potassium shift.² In contrast, this patient demonstrated persistent hypokalemia over several months with repeatedly reduced bicarbonate levels, findings that are less consistent with isolated hypokalemic periodic paralysis.

Renal potassium wasting was suggested by inappropriate urinary potassium excretion despite severe hypokalemia. The absence of vomiting, diarrhea, or diuretic use further supported a renal etiology.¹

The simultaneous presence of persistent hypokalemia, metabolic acidosis, and inappropriately alkaline urine raised suspicion for distal renal tubular acidosis. Distal renal tubular acidosis results from impaired distal hydrogen ion secretion, leading to metabolic acidosis and renal potassium wasting.^{3,5}

The marked rise in creatine phosphokinase was consistent with hypokalemic myopathy associated with severe potassium depletion. Rapid improvement following potassium replacement, together with preserved sensory function and absence of cranial nerve involvement, favored hypokalemic paralysis over primary neurological disorders such as Guillain-Barré syndrome. Thyroid function testing also revealed subclinical hypothyroidism, although its contribution to the hypokalemia remains uncertain.

Acute hypokalemic quadriparesis secondary to dRTA has been described in association with underlying autoimmune conditions, including Sjögren syndrome and autoimmune thyroid disease.^{1,3,8} In contrast, our patient had no clinical evidence of connective tissue disease or other autoimmune disorder, and testing remained negative. The diagnosis was supported by persistent renal potassium wasting, normal anion gap metabolic acidosis, and an inappropriately alkaline urine.

The presence of thyroid dysfunction in our patient is also notable, as renal tubular acidosis has been reported in association with hypothyroidism.^{2,6} Previous reports have described hypokalemic paralysis in patients with overt hypothyroidism, including cases associated with Hashimoto thyroiditis and primary hypothyroidism (8-10). Tiwari and Kansal described an adult patient with primary hypothyroidism presenting with hypokalemic paralysis, in whom hyperchloremic, normal anion-gap metabolic acidosis and an inappropriately alkaline urine led to the diagnosis of dRTA.⁶ Diniz et al also reported dRTA as the initial manifestation of Hashimoto thyroiditis.⁷ Basak et al described a patient with dRTA, primary hypothyroidism, nephrocalcinosis, osteomalacia, and recurrent hypokalemic paralysis, while Mareedu et al recently reported Hashimoto thyroiditis presenting with dRTA and hypokalemic paralysis.^{8,9} Unlike these reports, our patient had only subclinical hypothyroidism with normal FT3 and FT4 concentrations, no nephrocalcinosis, no skeletal manifestations, and no evidence of autoimmune thyroid disease.

Another notable feature was the presence of marked rhabdomyolysis in the setting of profound hypokalemia. Rhabdomyolysis has previously been reported in patients with dRTA-associated hypokalemic paralysis, although it appears to be an uncommon manifestation.⁴ More importantly, serial follow-up in our patient demonstrated persistent hypokalemia and low serum bicarbonate over four months despite neurological recovery, supporting an underlying chronic renal tubular disorder.

CONCLUSION

The persistence of hypokalemia during long-term follow-up distinguishes this case from isolated hypokalemic periodic paralysis and emphasizes the importance of suspecting distal renal tubular acidosis in patients with recurrent electrolyte abnormalities.

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