RESEARCH PAPER

Acute Hypokalemic Quadriparesis with Respiratory Failure in a Middle-Aged Female: A Case Report with Autoimmune Serological Correlation

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ABSTRACT
Acute flaccid quadriparesis with respiratory compromise is a neurological emergency requiring rapid evaluation and intervention. Severe hypokalemia is a reversible yet potentially fatal cause of acute neuromuscular paralysis. We report a 41-year-old female who presented with sudden-onset quadriparesis and progressive breathlessness, requiring mechanical ventilation. Initial investigations revealed profound hypokalemia (serum potassium 1.58 mmol/L) with normal anion gap metabolic acidosis. Arterial blood gas analysis showed pH 7.25, HCO_3^- 12 mEq/L, and $PaCO_2$ 26 mmHg. Further evaluation revealed features suggestive of distal renal tubular acidosis with autoimmune association. Following aggressive potassium correction and ventilatory support, the patient demonstrated rapid neurological recovery within 48 hours. Autoimmune workup including ANA and lip biopsy supported underlying Sjögren's syndrome. This case highlights the importance of early recognition of hypokalemic paralysis, identification of renal tubular acidosis, and evaluation for autoimmune etiology.

Keywords: Hypokalemic paralysis, Acute quadriparesis, Respiratory failure, Mechanical ventilation, Autoimmune disease, ANA positivity
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INTRODUCTION

Acute flaccid paralysis presenting with respiratory compromise is a neurological emergency that necessitates immediate evaluation and intervention. The differential diagnosis is broad and includes immune-mediated neuropathies such as Guillain-Barré syndrome, neuromuscular junction disorders including myasthenic crisis, acute spinal cord pathology, toxin exposure, metabolic derangements, and severe electrolyte abnormalities. Among these causes, hypokalemia is an uncommon but entirely reversible etiology that must be recognized early to prevent morbidity and mortality.[1]

Potassium plays a critical role in maintaining resting membrane potential and neuromuscular excitability. Severe hypokalemia results in hyperpolarization of skeletal muscle membranes, impairing action potential generation and leading to symmetrical muscle weakness. When serum potassium levels fall below 2.5 mmol/L, patients may develop flaccid quadriparesis, hyporeflexia or areflexia, and in extreme cases, respiratory muscle paralysis. Cardiac arrhythmias and metabolic disturbances further compound the clinical risk.[2]

Hypokalemic paralysis may be primary (familial periodic paralysis) or secondary to gastrointestinal losses, renal tubular dysfunction, endocrine disorders, medications, or toxin exposure. Secondary causes are particularly important in middle-aged adults without prior history of periodic paralysis. Hypokalemia associated with metabolic acidosis raises suspicion for renal tubular acidosis, especially distal renal tubular acidosis, which may occur in association with autoimmune disorders such as connective tissue diseases.[3]

The acute presentation of quadriparesis often leads to initial consideration of neurological emergencies, and failure to identify electrolyte abnormalities early may delay appropriate treatment. Rapid correction of potassium typically results in dramatic neurological improvement, distinguishing metabolic paralysis from structural or inflammatory neurological conditions.

We report a case of a previously healthy 41-year-old female who presented with sudden-onset quadriparesis and impending respiratory failure due to profound hypokalemia with metabolic acidosis, highlighting the importance of early recognition, prompt ventilatory support, and evaluation for secondary etiologies.

Case Presentation

A 41-year-old female was brought to the emergency department in the early hours of Saturday at approximately 2:00 a.m. with complaints of sudden onset weakness affecting both upper and lower limbs. The weakness developed abruptly after she went to the washroom and progressed rapidly over the next two to three hours. Initially, she experienced difficulty standing and walking, which soon progressed to inability to move her limbs. Within a short period, she developed breathlessness that gradually worsened, prompting emergency medical attention.

She reported bilateral lower limb pain for three days prior to presentation. The pain was described as dull and

aching without radiation. She also complained of constipation for three days preceding the acute event. There was no history of fever, upper respiratory infection, diarrhea, vomiting, trauma, recent strenuous exercise, seizures, headache, or giddiness. There was no history suggestive of recent toxin exposure, ingestion of unknown substances, or envenomation.

Her past medical history was notable only for a hysterectomy performed ten years earlier. She was not on any regular medications and denied the use of diuretics, laxatives, herbal supplements, or over-the-counter drugs. There was no prior history of similar weakness episodes. She had no known history of hypertension, thyroid disease, type 2 diabetes mellitus, cerebrovascular accident, chronic kidney disease, ischemic heart disease, or tuberculosis. There was no significant family history of neuromuscular disorders or periodic paralysis.

Examination at Presentation

The patient was conscious and oriented to time, place, and person. Higher mental functions: Normal. No cognitive impairment noted

Vital signs were as follows:

Blood pressure: 110/70 mmHg
Heart rate: 60 beats per minute
Respiratory rate: 34 breaths per minute
Oxygen saturation: 88% on room air
Temperature: 37°C
Random blood glucose: 126 mg/dL

The patient was conscious but drowsy, likely secondary to respiratory compromise.

Systemic Examination

Cardiovascular system: Normal S1 and S2 heart sounds were heard without murmurs, rubs, or gallops.

Respiratory system: Bilateral air entry was present without added sounds; however, tachypnea was evident.

Abdominal examination: Abdomen was soft and non-tender with no organomegaly.

Neurological Examination

Neurological assessment revealed severe generalized flaccid paralysis.

Higher mental functions: Conscious but drowsy
Cranial nerves: Grossly intact
Motor examination: Power 0/5 in all four limbs
Tone: Reduced
Deep tendon reflexes: Absent (areflexia)
Plantar responses: Bilaterally Flexor
Single breath count: 6

Worsening breathlessness indicated significant respiratory muscle involvement and impending respiratory failure.

Given the rapid progression of weakness and oxygen desaturation, the patient was intubated and placed on mechanical ventilation for airway protection and respiratory support.

Clinical Course in ICU

Arterial Blood Gas (ABG):

pH: 7.25 ↓
HCO_3^-: 12 mEq/L ↓
$PaCO_2$: 26 mmHg ↓
→ Suggestive of **metabolic acidosis with respiratory compensation (normal anion gap)**

Serum Electrolytes:

Potassium: 1.58 mmol/L ↓
Chloride: **Elevated (hyperchloremia)**
→ Supporting **hyperchloremic metabolic acidosis**

Urine Analysis:

Urine pH: **>5.5** (inappropriately alkaline)
Urine potassium: Elevated
Urine chloride: Elevated
Urine calcium: Elevated (hypercalciuria)

Urine Electrolytes:

Na^+, K^+, Cl^- levels consistent with renal potassium loss

Urine Anion Gap:

Positive
→ Suggestive of **distal renal tubular acidosis (Type 1 RTA)**

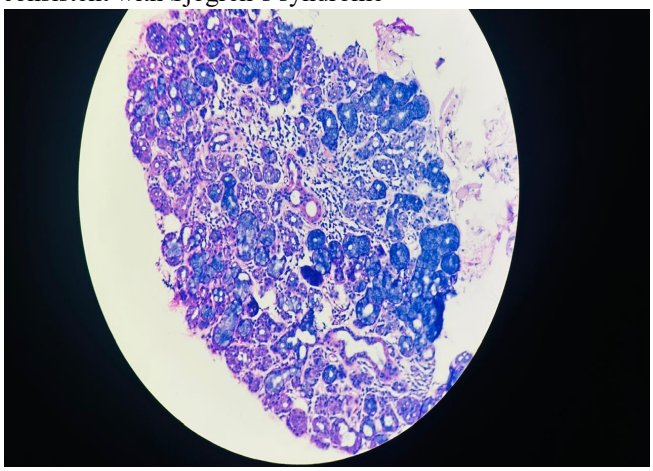
Over the next 24 hours, the patient demonstrated marked neurological improvement. By Sunday evening (approximately 8:00 p.m.), she was conscious and oriented. Motor power improved to 3/5 in all four limbs, and deep tendon reflexes returned as 1+.

On Day 2 motor strength improved to 5/5 in all limbs with restoration of normal reflexes (2+). Plantar responses remained down going. Respiratory parameters improved significantly, and she was successfully weaned from mechanical ventilation. By 2:00 p.m., she

was extubated and maintained oxygen saturation of 99% on nasal prong oxygen at 4 L/min, subsequently sustaining adequate saturation on room air.

ANA Profile:
ANA by IF: 3+ (**Positive)**
Pattern: Speckled pattern
Titer: **1:1000**

Specific Antibodies:
Anti-SSA (Ro-52): Positive
Anti-SSB (La): Positive
Schirmer's test: **Reduced tear production (<5 mm in 5 minutes).**
The rapid and complete neurological recovery within 48 hours following potassium correction confirmed the diagnosis of **acute hypokalemic paralysis**.
Histopathology-Histopathological image showing focal lymphocytic infiltration of minor salivary glands consistent with Sjögren's syndrome



Lip Biopsy Report:
Chronic lymphocytic sialadenitis
Focus score >1.
Additional biochemical investigations, including spot urine chloride estimation, did not reveal evidence of renal dysfunction or electrolyte imbalance. The absence of proteinuria, significant renal abnormalities, or metabolic derangements further supported the notion of an early or incomplete autoimmune process rather than an established systemic connective tissue disease.

Treatment
Immediate **mechanical ventilation** for respiratory failure
Intravenous potassium chloride infusion with cardiac monitoring
Correction of metabolic acidosis
Oral potassium supplementation after stabilization
Alkali therapy (oral sodium bicarbonate) for RTA
Supportive care including oxygen therapy post-extubation
Clinical Course
Within 24 hours: Improvement in sensorium and muscle power (0/5 → 3/5)
Within 36–40 hours: Further neurological recovery
Within 48 hours: Complete recovery (5/5 power), extubated successfully

Discussion

Acute hypokalemic paralysis represents a metabolic neuromuscular emergency resulting from severe depletion of extracellular potassium. Potassium is the principal intracellular cation and is critical in maintaining resting membrane potential across skeletal muscle fibers. When serum potassium levels decline significantly, membrane hyperpolarization occurs, increasing the threshold required for depolarization and thereby impairing action potential generation. This results in reduced muscle fiber excitability and manifests clinically as symmetrical flaccid weakness.
In severe hypokalemia (serum potassium <2.5 mmol/L), muscle weakness can progress rapidly to quadriparesis, areflexia, and involvement of respiratory muscles. In the present case, the patient's serum potassium was profoundly reduced to 1.58 mmol/L, explaining the abrupt onset of generalized paralysis and the absence of deep tendon reflexes. The single breath count of zero and progressive desaturation reflected diaphragmatic and intercostal muscle dysfunction. Hypokalemia impairs respiratory muscle contractility, and when combined with metabolic acidosis, may precipitate early ventilatory failure.
Unlike primary neurological disorders such as Guillain–Barré syndrome, hypokalemic paralysis typically demonstrates rapid reversibility following potassium correction. The dramatic improvement from motor power 0/5 to 5/5 within 48 hours strongly supports a metabolic etiology rather than structural or inflammatory neuropathy.
An important feature in this case was the presence of metabolic acidosis with markedly reduced bicarbonate levels. Hypokalemic periodic paralysis classically presents with metabolic alkalosis due to intracellular potassium shift. However, the coexistence of hypokalemia and metabolic acidosis suggests a secondary etiology, most notably renal tubular acidosis (RTA).[3,4]

Metabolic acidosis reduces intracellular pH, influencing potassium redistribution and renal potassium handling. In states of acidosis, potassium shifts from intracellular to extracellular compartments; however, when total body potassium depletion is severe, serum levels remain low despite this shift. Persistent urinary potassium loss in the presence of metabolic acidosis strongly indicates renal potassium wasting. The correction of acidosis observed by Day 2 in this patient paralleled neurological recovery, reinforcing the link between acid-base disturbance and neuromuscular dysfunction.
Renal tubular acidosis refers to a group of disorders

characterized by impaired renal acidification despite preserved glomerular filtration. Of particular relevance in this context is distal (Type 1) RTA, in which there is defective hydrogen ion secretion in the distal nephron. This defect results in:

Inability to acidify urine

Chronic metabolic acidosis

Enhanced renal potassium loss

Severe hypokalemia

In distal RTA, increased sodium delivery to distal nephron segments promotes potassium secretion via aldosterone-sensitive channels, leading to significant potassium wasting. Chronic hypokalemia may remain asymptomatic until acute depletion triggers paralysis.

The presence of **normal anion gap metabolic acidosis, hyperchloremia, urine pH >5.5, positive urine anion gap, and renal potassium wasting strongly supports distal renal tubular acidosis (Type 1 RTA).** The association of distal RTA with **positive ANA, anti-SSA/SSB antibodies, reduced Schirmer's test, and confirmatory lip biopsy** establishes **Sjögren's syndrome as the underlying etiology.** Hypercalciuria further supports distal RTA due to impaired hydrogen ion secretion and calcium phosphate precipitation risk.

Although classical distal RTA is associated with normal anion gap metabolic acidosis, overlapping metabolic disturbances may occur in critically ill patients, and high anion gap states can coexist transiently due to tissue hypoxia or lactic acidosis secondary to respiratory compromise. [5,6]

The clinical context of this case—middle-aged female, severe hypokalemia, metabolic acidosis, absence of gastrointestinal losses, no diuretic use—raises strong suspicion for renal potassium wasting, potentially due to distal RTA.

Distal renal tubular acidosis is frequently associated with autoimmune disorders, particularly:

Sjögren's syndrome

Systemic lupus erythematosus

Mixed connective tissue disease

Autoimmune thyroid disease

Autoimmune-mediated damage to intercalated cells of the distal nephron impairs hydrogen ion secretion, resulting in chronic metabolic acidosis and secondary potassium wasting. In some cases, hypokalemic paralysis may be the initial presenting manifestation of an underlying autoimmune condition.

The absence of classical systemic symptoms does not exclude autoimmune pathology. Renal tubular dysfunction may precede overt manifestations such as sicca symptoms, arthritis, or cutaneous findings. Several case series have reported hypokalemic paralysis as the first presentation of Sjögren's syndrome secondary to distal RTA.

Therefore, this case warrants further evaluation including:

Urine pH assessment

Urinary potassium excretion

Serum aldosterone and renin levels

Anti-SSA (Ro) and Anti-SSB (La) antibodies

Thyroid function tests

Identification of an autoimmune etiology has prognostic significance, as recurrence of hypokalemia may occur if the underlying inflammatory process remains untreated.

Respiratory muscle involvement is uncommon but life-threatening in hypokalemic paralysis. Diaphragmatic weakness leads to hypoventilation, hypercapnia, and hypoxemia. The patient's rapid tachypnea and declining oxygen saturation indicated early ventilatory fatigue. Mechanical ventilation was lifesaving and allowed safe correction of electrolyte imbalance.

The rapid weaning and extubation following potassium normalization further differentiate metabolic paralysis from neuromuscular junction disorders, which typically require prolonged respiratory support. [6]

Clinical Significance

This case highlights several critical teaching points:

Severe hypokalemia can mimic acute neurological emergencies.

Respiratory muscle paralysis may develop rapidly and requires early airway protection.

Hypokalemia with metabolic acidosis strongly suggests renal potassium wasting rather than primary periodic paralysis.

In middle-aged women, autoimmune-related distal RTA must be actively investigated.

Rapid neurological recovery after potassium correction confirms metabolic etiology.

Conclusion-

Acute hypokalemic paralysis is a rare but reversible cause of sudden flaccid quadriparesis and respiratory failure. This case highlights distal renal tubular acidosis secondary to Sjögren's syndrome presenting as life-threatening hypokalemic quadriparesis with respiratory failure. The combination of normal anion gap metabolic acidosis, hyperchloremia, alkaline urine, positive urine anion gap, and autoimmune markers is diagnostic. Early recognition and prompt correction of hypokalemia are lifesaving, while identification of autoimmune etiology is essential to prevent recurrence.

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