Case Report

A case of quadriparesis with type 2 respiratory failure: A diagnostic conundrum

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Abstract

Background: Acute flaccid paralysis with respiratory failure is commonly attributed to Guillain-Barré Syndrome; however, other causes should be kept in mind.

Case Report: A 41-year-old woman presented with acute ascending weakness, areflexia, and respiratory failure requiring intubation. Initial clinical suspicion was Guillain–Barré Syndrome. However, Laboratory evaluation revealed severe hypokalemia (serum potassium 1.1 mEq/L) and normal anion gap metabolic acidosis (pH 6.9, HCO_3^- 14.6 mmol/L). Urine anion gap was positive, consistent with distal renal tubular acidosis. MRI showed pontine demyelination. In view of suspected combined central and peripheral demyelination syndrome, autoimmune workup was done, which revealed ANA, anti-SSA, anti-SSB, and anti-RNP positivity. The patient improved with intravenous potassium replacement, IVIG, Steroids and immunosuppressants. She was extubated on hospital day 4.

Why should an Emergency Physician be aware of this? Autoimmune polyradiculoneuropathy secondary to connective tissue disorder is a reversible cause of respiratory failure that can mimic Guillain–Barré Syndrome and hypokalemic periodic paralysis. Early recognition with prompt initiation of immunosuppression can be lifesaving.

Key words: Acute flaccid paralysis; Guillain–Barré Syndrome; Hypokalemic

Citation: Arpana Singh, Aravind Gautham, Meenakshi Paramasivan, Suresh Babu, APS Kannan, Adhitya, Prasanna Babu, Vidhya. A case of quadriparesis with type 2 respiratory failure - A diagnostic conundrum. Kauverian Med J. 2026;3(7):69—73.

Academic Editor: Dr. Venkita S. Suresh

ISSN: 2584-1572 (Online)

1. Introduction

Acute flaccid paralysis is a time-sensitive emergency presentation. While Guillain–Barré Syndrome is a common consideration, and metabolic causes are frequently ruled out, Distal Renal Tubular Acidosis is an under-recognized cause of hypokalemia and is frequently associated with concomitant polyradiculoneuropathy due to autoimmune conditions such as Sjogren's syndrome and other connective tissue disorder.

2. Case Presentation

A 41-year-old woman presented with two days history of progressive ascending weakness and worsening dyspnoea for one day. She also had slurred speech and altered mental status. There was history of fall due to limb weakness and injury to left 4th toe. There was a history of diarrhoea four days prior. On arrival, vital signs were blood pressure 140/90 mmHg, heart rate 140 beats/min, respiratory rate 40 breaths/min, oxygen saturation 78% on room air.

Neurologic examination revealed generalized areflexia, quadriparetic with power 0/5 in all 4 limbs, neck flexor weakness, and ptosis. Due to worsening respiratory distress, the patient was intubated.

3. Investigations

Laboratory evaluation showed:

Serum Potassium	1.1 mEq/L
Arterial PH	6.9
Bicarbonate	14.6 mmol/L

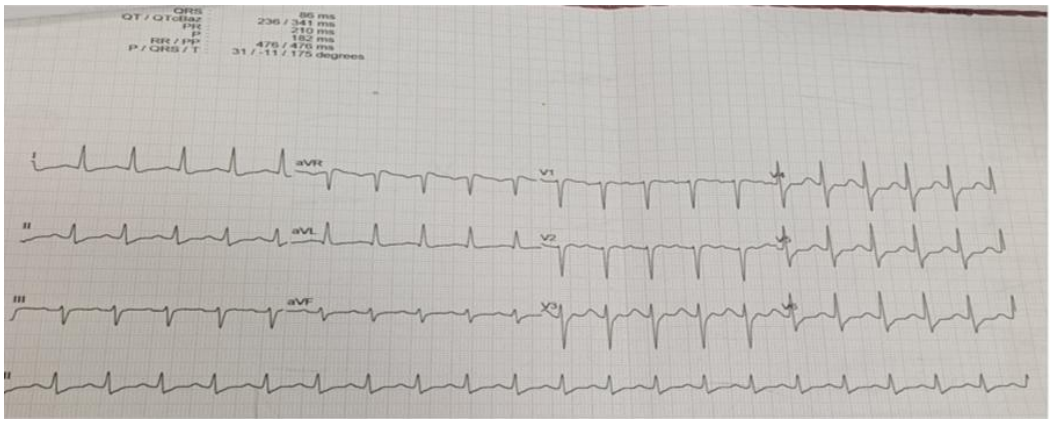


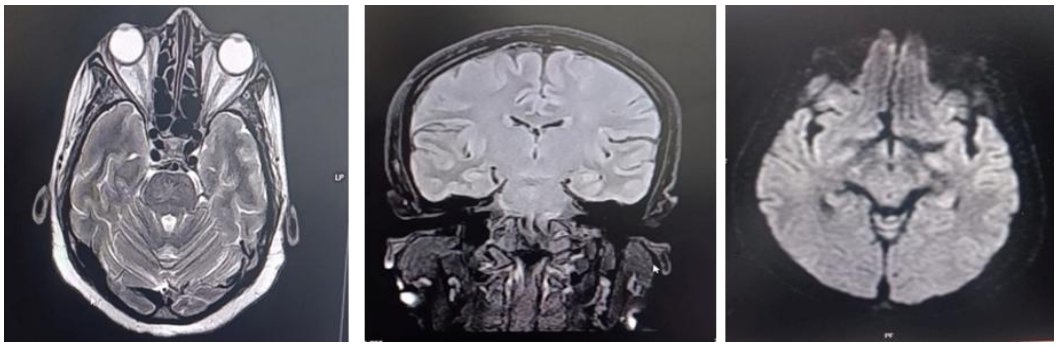
Fig (1): ECG-Sinus Tachycardia

Findings were consistent with Normal Anion Gap (Hyperchloremic) Metabolic Acidosis.

Urine anion gap was positive, suggesting impaired ammonium excretion consistent with distal renal tubular acidosis.

4. Neurologic workup

Cerebrospinal fluid	Normal
Nerve conduction studies	Diffuse demyelinating polyradiculoneuropathy
MRI brain	Pontine Demyelination



5. Autoimmune evaluation

Anti-SSA	Positive
Anti-SSB	Positive
Anti-RNP	Positive
Anti -Ro-52	Positive

6. Differential Diagnosis

- Guillain–Barré Syndrome
- Hypokalemic paralysis
- Myasthenia gravis
- Periodic paralysis

7. Management and Outcome

Given the initial suspicion of demyelinating polyradiculoneuropathy, the patient was started on intravenous Immunoglobulin. In view of coexisting comorbidities, non-sucrose based IVIG formulation was administered cautiously over 7 days, with close monitoring of cardiac, renal function and electrolytes.

The patient was treated with:

- Mechanical ventilation
- Aggressive intravenous potassium replacement, Steroids, IVIg, azathioprine and continuous cardiac monitoring

Neuromuscular strength improved significantly within three days. The patient was successfully extubated on hospital day 4 and discharged without neurologic deficits after 2 weeks of hospital stay.

8. Discussion

This case highlights a diagnostic and therapeutic challenge. The initial presentation strongly mimicked Guillain-Barré Syndrome due to ascending flaccid paralysis, areflexia, with respiratory and bulbar involvement. However, severe hypokalemia and metabolic acidosis indicated a concomitant Metabolic aetiology as well. As patients altered sensorium and plantar extensor on left side, MRI was done to rule of central etiology. MRI was suggestive of pontine demyelination but still could not explain quadriparesis with areflexia. Altered sensorium recovered following intubation. Hence encephalopathy was attributed to hypercarbemic encephalopathy secondary to type 2 respiratory failure with CO2 retention rather than ascending reticular activating system involvement in pons. Clinical pictures were more in favour of polyradiculoneuropathy. Hence a diagnosis of combined central and peripheral demyelination syndrome was suspected.

Severe hypokalaemia is a well-recognized cause of acute flaccid paralysis and can involve respiratory muscles, leading to life-threatening respiratory failure. Potassium levels below

2.5 mEq/L are strongly associated with neuromuscular weakness; in this case, a level of 1.1 mEq/L explains the severity of presentation. However, since pt failed to improve after potassium correction. Patient was continued on IvIg following which weakness recovered. Patients continued to have recurrent hypokalemic attacks during the hospital stay over 3 days and persistent distal RTA, despite adequate potassium correction. Within a day after pulse steroids were initiated, metabolic parameters recovered. This highlights the prompt initiation of immunosuppressants which address the underlying pathology rather than correction of hypokalemia alone.

Distal renal tubular acidosis is characterized by impaired hydrogen ion secretion, resulting in normal anion gap metabolic acidosis and inability to acidify urine. A positive urine anion gap reflects reduced ammonium excretion and supports the diagnosis.

Patients also had an ejection fraction of 28 %. At presentation, Myocarditis was suspected. As patient's cardiac function recovered with supportive care within 3 days, stress induced cardiomyopathy was thought to be more likely. Chest imaging was in favour of Interstitial lung disease likely non-specific interstitial pneumonia (NSIP). Multidisciplinary team evaluation and interventions favored patients' early recovery.

Autoimmune disorders, particularly Sjogren's syndrome, are common causes of distal renal tubular acidosis due to tubulointerstitial injury and interstitial lung disease.

This case mimicked Guillain-Barré Syndrome due to ascending weakness, areflexia, and respiratory involvement. This case also underscores the clinical dilemma regarding Intravenous Immunoglobulin use in renal and cardiac dysfunction. While IVIG is standard treatment for demyelinating polyradiculoneuropathy, it carries potential renal risks. In this patient, IVIG was administered cautiously due to renal and cardiac involvement.

9. Why should an Emergency Physician be aware of this?

- Hypokalemia is a reversible cause of acute paralysis. However, treatment of underlying causes of hypokalemia is more important.
- Autoimmune polyradiculoneuropathy secondary to connective tissue disorder Can mimic Guillain–Barré Syndrome clinically

- Early electrolyte evaluation is critical
- Prompt treatment leads to rapid recovery
- First presentation of autoimmune disease can be quadriparesis with respiratory distress

10. Conclusion

Hypokalemic paralysis due to autoimmune distal renal tubular acidosis and demyelinating polyradiculoneuropathy due to Connective tissue disorder, both are reversible causes of respiratory failure that can mimic Guillain–Barré Syndrome. Early diagnosis and treatment are essential to improve outcomes.

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