https://doi.org/10.29309/TPMJ/2026.33.01.10064 www.theprofesional.com

CASE REPORT

A diagnostic dilemma: Severe hypokalemia presenting with GBS-like clinical feature.

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ABSTRACT… Acute flaccid paralysis is a potentially life- threatening presentation necessitating prompt and precise diagnosis. Guillain-Barre' Syndrome (GBS) is often the first consideration due to its prevalence and severity. However, non-neurological conditions, particularly metabolic disturbances such as hypokalemia, can clinically and electrophysiologically mimic GBS. Hypokalemic paralysis, albeit rare, represents a reversible etiology of acute limb weakness. It may present with symptoms indistinguishable from GBS, including areflexia and ascending limb paralysis, resulting in a diagnostic dilemma. A misdiagnosis can delay appropriate treatment and subject the patient to unnecessary interventions. This case report elucidates a patient who exhibited classic features of GBS but was ultimately diagnosed with severe hypokalaemia. It underscores the imperative of good clinical assessments including correctly identifying serum electrolytes abnormalities in all patients presenting with acute neuromuscular weakness.

Key words: Acute Flaccid Paralysis, Diagnostic Challenge, Electrolyte Imbalance, Guillain-Barre Syndrome Mimic, Hypokalemic Paralysis, Quadriplegia, Reversible Paralysis.

Article Citation: Afridi IA, Virk B. A diagnostic dilemma: Severe hypokalemia presenting with GBS-like clinical feature. Professional Med J 2026; 33(01):187-189. https://doi.org/10.29309/TPMJ/2026.33.01.10064

CASE PRESENTATION

A 23-year-old previously healthy male presented to the emergency department of Farooq Hospital (Bahria Enclave Branch) Islamabad, Pakistan with a one-day history of progressive quadriplegia. The weakness commenced abruptly first in the lower limbs and quickly progressed to involve the upper limbs. He reported difficulty in walking, standing from a seated position, and lifting objects but there was no swallowing or breathing difficulty. There were no other symptoms such as sensory symptoms, fever or bladder or bowel dysfunction and there was no history of trauma, vaccination or toxin exposure prior to these symptoms. The only relevant history we could find was that the patient had 4 days of watery diarrhea episode approximately 6-7 days prior to hospital admission.

He had been referred from a tertiary care hospital in Islamabad, where he had already been diagnosed with Guillain-Barre Syndrome (GBS), based on clinical presentation. Baseline investigations conducted revealed a serum potassium level (K+) of 2.7mmol/L. He came to us specifically for the management and treatment of GBS, including lumbar puncture, electromyography (EMG), nerve conduction studies and comprehensive GBS treatment.

On Examination Revealed:	GCS 15. Neurological Examination
1.Motor	Flaccid Paralysis (Grade 1/5 Strength) in all LIMBS.
2.Reflexes	Absent Deep Tendon Reflexes
3.Sensory	Intact
4.Cranial Nerves	Normal
5.Gait	Not Testable Due to Weakness

It is noteworthy that Farooq hospital's Bahria Enclave branch had been operational for only one month at that time, and certain diagnostic facilities were not yet available on-site. Given the acute, symmetrical, ascending flaccid paralysis with areflexia, a clinical diagnosis of Guillain-Barre Syndrome was initially entertained. The patient was admitted for further evaluation. We repeated his baseline investigations, which revealed a critically low serum potassium level 1.1mmol/L.

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Article received on:
05/09/2025
Accepted for publication:
14/11/2025

Routine Laboratory Investigations Revealed	
Laboratory Investigations	**Results**
White Cell Count	7.2 (cell/μL)
Hemoglobin	15g/dL
Platelets	278 x 10^9/L
Serum Potassium	1.1 mmol/L
Serum Sodium	136mmol/L
Serum Magnesium	1.7mg/Dl
Renal Function	Normal
Arterial Blood GAS	Mild Metabolic Alkalosis

ECG revealed U-waves and flattened T-waves consistent with hypokalemia. Meanwhile, potassium chloride replacement therapy was immediately initiated. The patient was diagnosed with hypokalemic paralysis. Intravenous potassium chloride was administered with close monitoring in Intensive Care Unit. Remarkably, within 12 hours of starting treatment, the patient began to exhibit the signs of recovery with the return of reflexes. In the next few hours, we noted progressive improvement the patient's muscle power and reflexes and finally within 48 hours, the patient had regained full muscle strength of power 5/5 with normal tendon reflexes. By day third, he was discharged with oral potassium supplementation and dietary counselling. No further investigations such as Nerve Conduction Studies, MRI or Cerebrospinal Fluid (CSF) analysis were done.

DISCUSSION

Hypokalemia paralysis is a rare yet reversible cause of acute flaccid paralysis. It may be primary (often familial) or secondary to conditions such as renal losses, gastrointestinal losses, thyrotoxicosis, or certain medications. Hall mark features include acute-onset muscle weakness, hyporeflexia or areflexia, and significantly low serum potassium levels.

Clinically, hypokalemic paralysis can close resemble Gillian Barre Syndrome (GBS), particularly in cases presenting with symmetrical ascending paralysis, absent reflexes and a history of diarrhoea preceding the onset muscle weakness. However, distinguishing feature include a rapid and dramatic improvement with potassium correction, normal nerve conduction studies, and absence of sensory or cranial nerves involvement.

In contrast, GBS is an immune-mediated demyelinating polyneuropathy that usually follows an infection and requires treatment with intravenous immunoglobulin (IVIG) or plasmapheresis.

Misdiagnosis of hypokalaemia paralysis as GBS can lead to unnecessary interventions, prolonged hospital stays, and increased healthcare costs.

CONCLUSION

This case study underscores the need to consider severe hypokalemia as a differential diagnosis in patients presenting with symptoms suggestive of Guillain-Barré Syndrome (GBS). More importantly, it emphasizes the value of a thorough clinical and biochemical assessment in distinguishing between these conditions, particularly if resources are limited, as was in our case. A simple and relatively inexpensive step of repeating the serum electrolyte test as we did to eliminate the chances of lab errors, may save the day. This helped us in reaching a diagnosis quickly which led us to the early initiation of correct treatment without unnecessary investigations and potentially harmful treatments like immunoglobulin therapy, resulting in improved patient outcomes.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

SOURCE OF FUNDING

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

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AUTHORSHIP AND CONTRIBUTION DECLARATION	
1	**Imtiaz Alam Afridi:** Study concept, data analysis.
2	**Bella Virk:** Design, protocol writing, data collection, data analysis, manuscript writing.