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CASE REPORT

Acute Paraplegia with Dengue Fever: A Case Series

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ABSTRACT

Background: Common presentations of dengue fever include fever, thrombocytopenia, hemorrhage and shock. Sudden onset bilateral lower limb weakness i.e. paraplegia, in a patient with dengue fever is not an uncommon presentation in the emergency medicine department. Possible causes for dengue associated neuro-muscular disorders include, dengue related transverse myelitis, Guillain-Barre syndrome, myositis, rhabdomyolysis, hypokalaemic paralysis. We here present four such cases, patients with sudden onset bilateral lower limb weakness and dengue fever presenting to our emergency medicine department at a tertiary care centre in rajasthan.

Our reasons for highlighting this case: dengue related neuro-muscular disorders may be the presenting symptom in emergency medicine department, if identified early and promptly treated may result in favourable outcomes.

KEYWORDS

• Paraplegia • Paraparesis • Dengue fever related neuromuscular disorders • Transverse myelitis • Hypokalemic paralysis • Guillain-Barre syndrome

INTRODUCTION

Sudden-onset bilateral lower limb weakness is a frequent presentation in the emergency department and can result from a variety of etiologies. These include musculoskeletal conditions (e.g., myopathies, rhabdomyolysis), metabolic or toxic imbalances (e.g., electrolyte

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disturbances, vitamin B12 or D deficiencies, and alcohol or organophosphate toxicity), infectious processes, vascular events, autoimmune disorders, trauma, tumors, or neurological conditions such as spinal cord disorders, peripheral neuropathies, or motor neuron diseases[1,2,3]. Among these, dengue-related neurological complications, though rare, should be considered, particularly in endemic regions[4,5].

Dengue fever, caused by the dengue virus, is a mosquito-borne viral infection with a significant global burden. While the classic presentation includes febrile illness, arthralgia, and rash, the disease is increasingly recognized for its systemic and neurological complications[6]. Neurological manifestations associated with dengue include conditions such as transverse myelitis (TM), Guillain-Barré Syndrome (GBS), hypokalemic periodic paralysis (HPP), and other immune-mediated or metabolic syndromes[7,8,9]. Transverse myelitis, particularly the longitudinally extensive subtype (LETM), involves inflammation spanning three or more vertebral segments, leading to motor, sensory, and autonomic dysfunctions[10,11]. Dengue-associated LETM is thought to arise from immune-mediated mechanisms, potentially involving molecular mimicry and cytokine-induced neural inflammation[4,5]. The postpartum period poses additional risks due to heightened inflammatory and immunological changes, further complicating neurological involvement in dengue[12,13]. Early intervention with corticosteroids or immunoglobulin therapy has shown favorable outcomes, emphasizing the importance of prompt diagnosis through spinal imaging.[14]

Dengue can also present with hypokalemic periodic paralysis (HPP), characterized by acute flaccid paralysis resulting from significant hypokalemia due to increased renal potassium excretion, cytokine-mediated electrolyte channel dysfunction, and transient tubular injury[15,16]. Timely potassium supplementation leads to rapid recovery, underscoring the importance of electrolyte monitoring in severe dengue cases[15].

Additionally, dengue has been associated with Guillain-Barré Syndrome, an immune-mediated neuropathy triggered by molecular mimicry, where dengue antigens resemble peripheral nerve myelin[16]. This process results in an autoimmune response and nerve damage. Diagnosis relies on clinical evaluation and nerve conduction studies, with immunotherapy such as intravenous immunoglobulin (IVIG) or plasmapheresis proving effective[16]. Differentiating GBS from other causes of acute flaccid paralysis is critical for appropriate management and improved patient outcomes[16].

These neurological complications, while rare, reflect the expanding spectrum of dengue-related manifestations, necessitating vigilance, early diagnosis, and timely therapeutic interventions to mitigate potential long-term neurological sequelae.[6,7,8,10].

CASE PRESENTATION

Case 1: Dengue-Associated Longitudinally Extensive Transverse Myelitis (LETM)

An 18-year-old male presented to the emergency medicine department with sudden onset paraplegia and urinary retention after a 4-day febrile illness characterized by high-grade fever, headache, and body aches diagnosed dengue fever. On neurological examination, **lower limb power was 2/5 bilaterally**, with **increased muscle tone** and **reflexes**bilateral lower limbs, consistent with upper motor neuron lesion. There was also sensory loss below the thoracic level. Ecg (*Fig. 1*) was normal, MRI (*Fig. 5*) revealed hyperintense T2-weighted signals in the spinal cord extending over multiple vertebral segments, confirming longitudinally extensive transverse myelitis. The patient was treated with high-dose intravenous methylprednisolone for five days, followed by an oral steroid taper. Over the next two weeks, his motor function and urinary retention improved significantly, although mild residual weakness persisted at discharge.

Case 2: Dengue-Associated Hypokalemic Periodic Paralysis (HPP)

A 19-year-old male presented with progressive weakness in his lower limbs after experiencing a 3-day febrile illness with myalgia and malaise. He reported difficulty walking and an inability to rise from a seated position. On physical examination, **lower limb muscle power was 2/5 bilaterally**, with **reduced muscle tone** and **diminished reflexes**. No sensory abnormalities or cranial nerve deficits were noted. EKG (*Fig. 2*) and Blood investigations were consistent with severe hypokalemia

with a serum potassium level of 2.1 mmol/L. Hypokalemic periodic paralysis secondary to dengue fever was diagnosed. The patient was treated with intravenous potassium chloride (KCl) and oral potassium supplementation. Remarkably, his motor function returned to normal within 48 hours of treatment. He was discharged in stable condition with advice to maintain regular follow-up for monitoring of potassium levels.

Case 3: Dengue-Associated LETM in a Postpartum Female

A 24-year-old postpartum day 7, female presented with a 5-day history of fever, chills, and generalized malaise, diagnosed dengue fever. The symptoms were followed by the sudden onset of paraplegia and bladder dysfunction. Neurological examination revealed **lower limb power of 1/5 bilaterally**, **raised muscle tone**, and **sensory loss below the thoracic level**. **Reflexes were brisk**, and there was no involvement of the upper limbs. Ecg (*Fig. 3*) was normal. MRI (*Fig. 6*) of the spine showed extensive longitudinal hyperintensities consistent with LETM. Laboratory investigations confirmed dengue fever, and other autoimmune markers, including aquaporin-4 antibodies, were negative. The patient was managed with high-dose intravenous methylprednisolone for five days. Gradual improvement in motor function was observed during the hospital stay, and she was discharged with physiotherapy and a tapering steroid regimen.

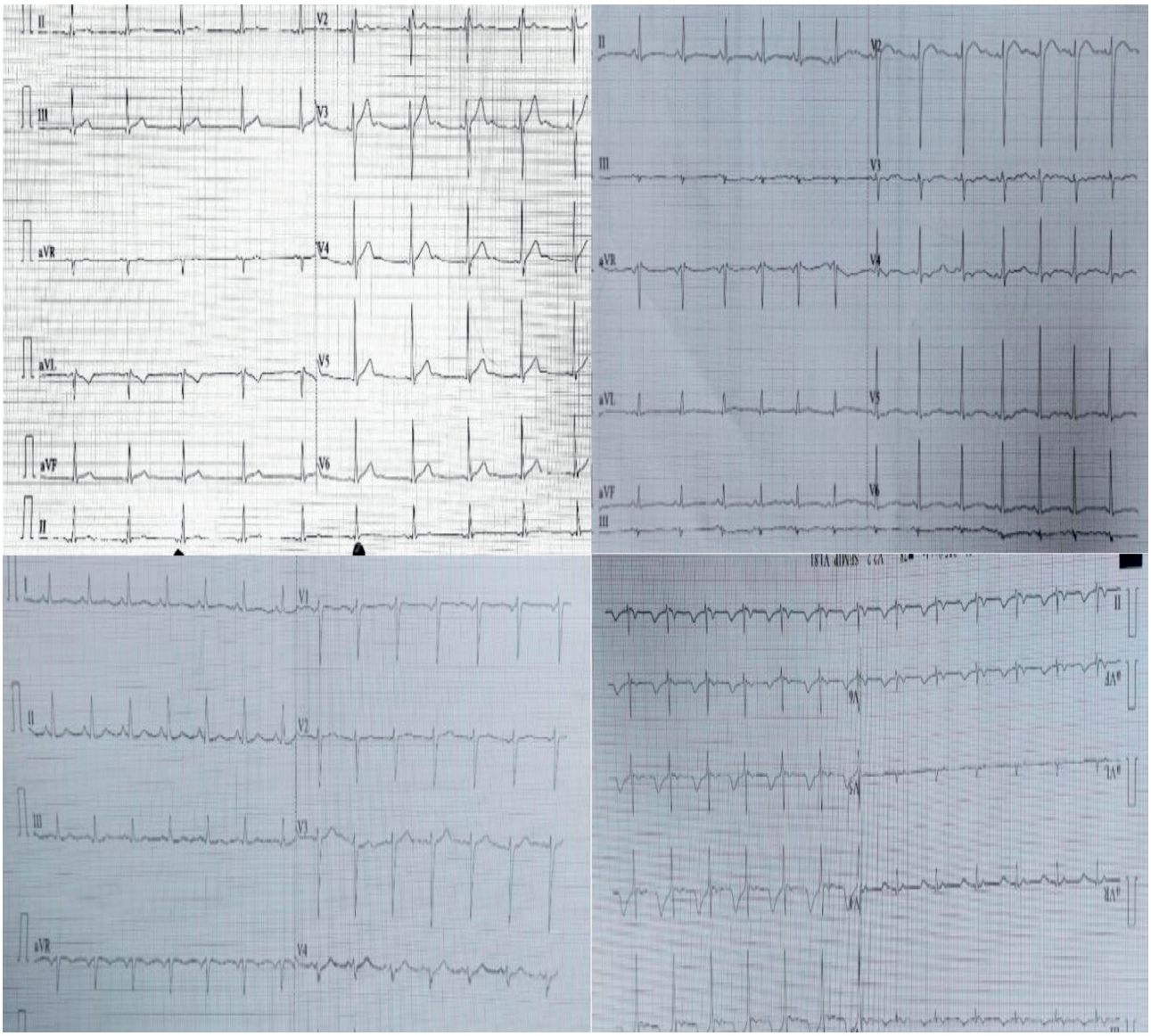


Figure 1-4: Showing ECG of the respective case, Showing flattening of the T wave(arrows).

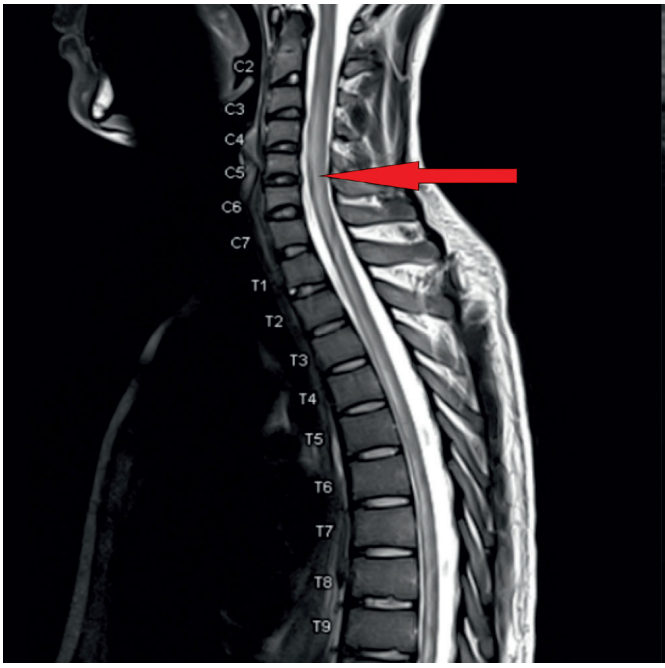


Figure 5: case 1-mri (non-contrast) cervicodorsal spine showing sagittal and axial planes of T2 weighted images indicating hyperintense lesion extending from c3 level to d10 level (indicated by red arrow)

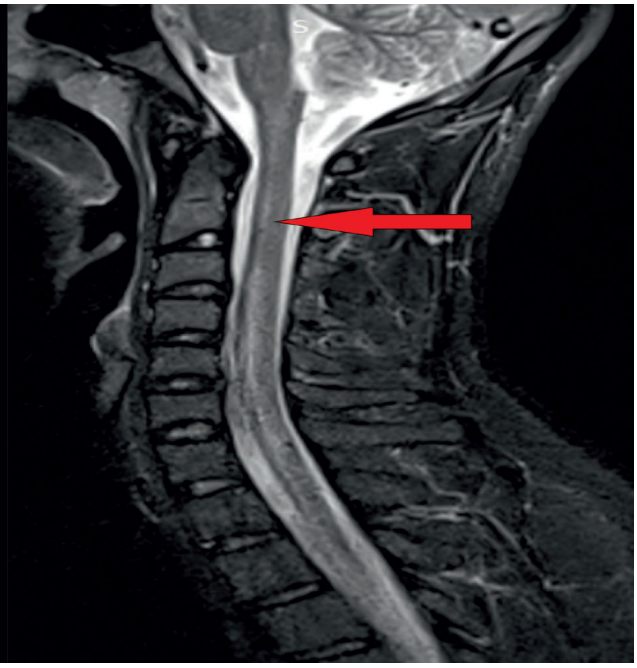


Figure 6: case 3-Long segment intra-medullary hyperi ntensity occupying more than 2/3rd of the cord diameter is noted from C6-7 level to D4-5 level – suggestive of longitudinally extensive transverse myelitis (indicated by red arrow).

Case 4: Dengue-Associated Guillain-Barré Syndrome (GBS)

A 22-year-old male presented with progressive weakness affecting all four limbs five days after recovering from dengue fever. He reported difficulty climbing stairs and gripping objects, accompanied by numbness in his hands and feet. Neurological examination revealed **flaccid quadriparesis**, with **power of 1/5 in all four limbs** and **absent reflexes**. Sensory examination revealed reduced vibration and proprioception in the lower limbs. E cg (*Fig. 4*) was normal. Nerve conduction studies revealed motor axonal and demyelinating affection in bilateral peroneal, tibial and median ulnar motor nerve suggesting a diagnosis of Guillain-Barré Syndrome with predominant axonal involvement. The patient was treated with intravenous immunoglobulin (IVIG) for five days and supportive care, including respiratory monitoring. He showed partial recovery during the hospital stay and was discharged for outpatient rehabilitation. At follow-up, gradual improvement in muscle strength was noted over three months.

Investigations	Case 1	Case 2	Case 3	Case 4	
ESR	50	48	35	40	1st Hr mm
Hemoglobulin	13.8	13.3	8.3	12	g/dl
Total Leukocyte Count	6.3	5.1	2.4	6.8	Thousand cu/mm
Platelets	78	70	60	30	Thousand/ cumm
S. Sodium	138.2	141.5	139.5	140	Mmol/L
S. Potassium	4.34	2.1	3.78	4.1	Mmol/L
Vit B12	766	623	596	711	Pg/ml
Urine Potassium	76.4	18.97	67.5	88	mEq/L per day
Anti Dengue IgM	Reactive	Reactive	Reactive	Reactive	

DISCUSSION

The cases presented in this series highlight a spectrum of dengue related neurological disorders, including longitudinally extensive transverse myelitis (LETM), hypokalemic periodic paralysis (HPP) and Guillain-Barré Syndrome (GBS)[1], each presenting unique diagnostic and therapeutic challenges.

Dengue-Associated Longitudinally Extensive Transverse Myelitis (LETM)

LETM, a rare inflammatory spinal cord condition spanning three or more vertebral segments, disrupts motor, sensory, and autonomic functions[2]. Though traditionally linked to autoimmune diseases like neuromyelitis optica (NMO), infectious agents such as dengue virus have been implicated in its pathogenesis[3]. Inflammation, immune dysregulation, and a dengue-induced cytokine storm are believed to underlie LETM[4].

Studies have described cases of dengue-associated LETM where MRI confirmed spinal cord inflammation[5]. Research has discussed the role of pro-inflammatory cytokines like tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) in causing endothelial damage and blood-spinal cord barrier breakdown, enabling immune cells to infiltrate the spinal cord 4,6. These findings align with our observations, as patients improved significantly following corticosteroid therapy, underscoring the critical role of early anti-inflammatory treatment.

Moreover, LETM in postpartum patients poses an additional challenge due to pregnancy-induced immune modulation[3]. Research has emphasized the altered immune landscape in postpartum dengue patients, which may exacerbate inflammatory damage, necessitating heightened surveillance and prompt intervention[6].

Hypokalemic Periodic Paralysis (HPP) in Dengue Fever

HPP is an acute motor paralysis condition caused by hypokalemia, commonly associated with hereditary channelopathies or thyrotoxicosis. In dengue fever, HPP occurs due to metabolic derangements such as renal tubular dysfunction, inappropriate potassium excretion, and cytokine-mediated electrolyte imbalance. This mechanism differentiates dengue-associated HPP from its familial counterpart[12].

Previous studies highlighted the high prevalence of hypokalemia among dengue patients presenting with flaccid paralysis[13]. These studies underscore the importance of distinguishing dengue-associated HPP from hereditary causes through history, laboratory findings, and the rapid reversibility of symptoms with potassium supplementation. In our case, there was no apparent cause of hypokalemia (renal loss, gastrointestinal loss, or any history of similar presentation), and the patient's symptoms resolved entirely following intravenous potassium chloride (KCl) therapy, leading to the diagnosis of dengue-associated hypokalemia[14].

A retrospective analysis revealed that 8–10% of dengue patients experience significant electrolyte disturbances, including hypokalemia, which could mimic neuromuscular disorders[15]. The reversible nature of dengue-induced HPP and its favorable prognosis with timely electrolyte correction highlight the importance of maintaining a high index of suspicion for this condition. Further studies are required in this regard[16].

Dengue Fever and Guillain-Barré Syndrome (GBS)

GBS, an acute immune-mediated polyneuropathy, is characterized by ascending paralysis and areflexia. Infectious triggers such as Campylobacter jejuni, Zika virus, and dengue virus have been implicated[8,9]. Dengue-associated GBS is thought to result from molecular mimicry, where dengue antigens elicit cross-reactive immune responses targeting peripheral nerve myelin[10].

Research has highlighted the rarity of GBS in dengue but stressed its severe implications, including respiratory compromise requiring intensive care[11]. Diagnostic nerve conduction studies (NCS) and cerebrospinal fluid (CSF) analysis revealing albuminocytological dissociation remain pivotal in confirming GBS[7]. Our fourth case demonstrated typical features of GBS, including quadriparesis and nerve conduction abnormalities. The patient responded well to intravenous immunoglobulin (IVIG) therapy, aligning with existing literature[9].

Further studies reported that early treatment with IVIG or plasmapheresis significantly reduces morbidity in dengue-associated GBS

[10,11]. They also highlighted the importance of differentiating GBS from other causes of acute flaccid paralysis, such as hypokalemia, to guide appropriate therapy[11]. Dengue-related GBS, though rare, underscores the systemic and immune-mediated nature of the disease [10,11].

Broader Implications of Neurological Complications in Dengue

The neurological spectrum of dengue reflects the systemic and immune-mediated impact of the virus. Studies emphasize that neurological complications such as encephalitis, myelitis, and neuropathies often correlate with severe disease and increased mortality. The role of pro-inflammatory cytokines, endothelial activation, and molecular mimicry in the pathogenesis of these complications necessitates further research to develop targeted therapies.

Timely recognition and management of these complications can significantly improve patient outcomes. The cases in this series underscore the importance of interdisciplinary collaboration, involving neurologists, infectious disease specialists, and intensivists, to manage such complex scenarios.

CONCLUSION

In conclusion, dengue fever, though primarily recognized for its febrile and systemic manifestations, presents a growing spectrum of neurological complications, including transverse myelitis, hypokalemic periodic paralysis, and Guillain-Barré Syndrome. These conditions, while uncommon, can result in significant morbidity if not promptly identified and treated. Early recognition and management, including interventions such as corticosteroids, immunoglobulin therapy, or potassium supplementation, are crucial in mitigating long-term neurological sequelae.

Given the expanding burden of dengue worldwide, especially in endemic regions, there is a critical need to enhance awareness among healthcare providers regarding its neurological manifestations. Furthermore, additional research is essential to better understand the underlying mechanisms, risk factors, and optimal treatment strategies for these complications. By advancing our knowledge through dedicated studies, we can improve diagnostic accuracy, treatment outcomes, and overall patient care in the context of dengue-associated neurological disorders.

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