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Delayed Spontaneuous Spinal Subdural Hemorrhage Following Dengue Fever: A Rare Case Report

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Abstract

Introduction: *Dengue fever is a mosquito-borne viral illness endemic to tropical and subtropical regions, including Southeast Asia. While most cases are self-limiting, severe complications such as bleeding diathesis and neurological involvement can occur. Neurological manifestations are rare, affecting less than 1% of patients, and spinal subdural hemorrhage (SSDH) is among the least reported.*

Case Presentation: *A 30-year-old man presented with acute spastic tetraparesis that began suddenly accompanied by sensory loss below the neck and urinary retention, one week after recovering from dengue fever. Neurological examination revealed global motor weakness, hypesthesia below C5, brisk reflexes, and positive bilateral pathologic reflexes, with no signs of meningeal irritation. Laboratory results showed mildly elevated liver enzymes and a history of thrombocytopenia during dengue infection. Cervical spine MRI with contrast demonstrated multifocal late subdural hemorrhage at C4–C7 with associated spinal cord myelopathy and mild canal stenosis.*

Discussion: *This case illustrates a rare neurological complication of dengue fever, where delayed spontaneous spinal subdural hemorrhage (SSDH) occurred despite only moderate thrombocytopenia. The proposed pathophysiology involves rupture of delicate intradural or bridging veins due to increased intraspinal venous pressure, compounded by dengue-associated endothelial dysfunction and coagulopathy. Hemorrhage into the subdural space may result from arachnoid membrane disruption or shear forces between the subarachnoid and subdural compartments. This supports the theory that even transient dengue-induced hemostatic imbalance can precipitate significant central nervous system bleeding.*

Keywords: *Dengue Fever, Spinal Subdural Hemorrhage, Tetraparesis, Thrombocytopenia.*

1. INTRODUCTION

Dengue fever, caused by a single-stranded RNA virus from the Flavivirus genus, is a mosquito-borne illness that remains a major public health concern in tropical and subtropical regions, including Southeast Asia. Although the majority of infections result in self-limiting febrile illness, dengue can occasionally progress to severe forms involving vascular leakage and coagulation abnormalities. One such complication is bleeding diathesis, which may lead to multisystem involvement, including renal impairment, cardiac dysfunction, shock, and electrolyte disturbances.[1] Neurological complications are uncommon, occurring in less than 1% of dengue cases, but when present, may involve encephalitis, myelitis, or hemorrhagic events within the central nervous system. Among spinal hemorrhages, spinal subdural hemorrhage (SSDH) is particularly rare compared to epidural or intramedullary types. The proposed mechanisms for neurological involvement include direct viral invasion of the CNS, systemic metabolic derangements, hemorrhage secondary to coagulopathy, immune-mediated demyelination, and inflammation. In this report, we describe an unusual case of paraparesis due to delayed spontaneous SSDH following dengue fever, representing a rare but serious neurological manifestation of the disease.[2]

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2. CASE PRESENTATION

A 30-year-old man was brought to Emergency Department with acute onset spastic tetraparesis that began seven days prior to admission. The weakness started suddenly on April 20th, 2025, while the patient was at rest. Initially able to move all four limbs, he abruptly lost motor function and developed a tingling sensation below the neck. He denied any back pain, fever, cough, or shortness of breath, but reported difficulty with urination and defecation following the onset of symptoms. One week earlier, he had been hospitalized for four days at a primary care facility with a diagnosis of dengue fever, from which he was discharged after clinical improvement. His medical history was notable for an ischemic stroke in May 2024, resulting in transient right-sided weakness that had resolved completely. He denied a history of hypertension, diabetes mellitus, cardiac or liver disease, autoimmune conditions, trauma, or malignancy. Home medications included acetosal 80 mg, simvastatin 20 mg, and piracetam 800 mg once daily.

On arrival, his vital signs were within normal limits, with a blood pressure of 129/84 mmHg, heart rate of 95 bpm, respiratory rate of 20 breaths/min, temperature 36.0°C, and oxygen saturation of 98% on room air. Neurological examination showed a fully alert and oriented patient (GCS 15), without signs of meningeal irritation. Cranial nerve assessment revealed no deficits except for a right-sided UMN-type lingual palsy, consistent with sequelae of his prior stroke. Motor strength was globally reduced to 2/5 in all limbs, and there was hypesthesia below the C5 dermatome. Reflexes were brisk (3+/4+) in all extremities, and pathologic reflexes including Hoffman, Tromner, Babinski, and Chaddock signs were all bilaterally positive. Urinary retention was evident, and a Foley catheter was in place.

Laboratory results on April 25th showed hemoglobin 12.0 g/dL, white blood cell count 8,670/mm³ with neutrophil predominance (84.3%), platelet count 334,000/mm³, PT 13.0 sec, APTT 25.0 sec, sodium 134 mmol/L, potassium 5.2 mmol/L, AST 82 U/L, and ALT 152 U/L. Inflammatory markers were within normal range. Serial platelet counts during his prior admission showed thrombocytopenia, with the lowest value recorded at 58,000/mm³ on April 15th and the result of the NS1 antigen test for dengue was positive.. MRI of the cervical spine with contrast (April 23rd) revealed multifocal late subdural hemorrhage at the C4 and C7 levels, with associated myelopathy and mild spinal canal stenosis on on T1W (Figures 1) and T2W (Figures 2). Based on clinical presentation, imaging, and history of recent dengue fever, the patient was diagnosed with tetraplegia due to delayed spinal subdural hemorrhage secondary to dengue-associated coagulopathy. Surgery was not preferred due to small and multifocal lesion in spinal subdural space, hence the patient was treated with conservative treatment and was discharged a few days later.

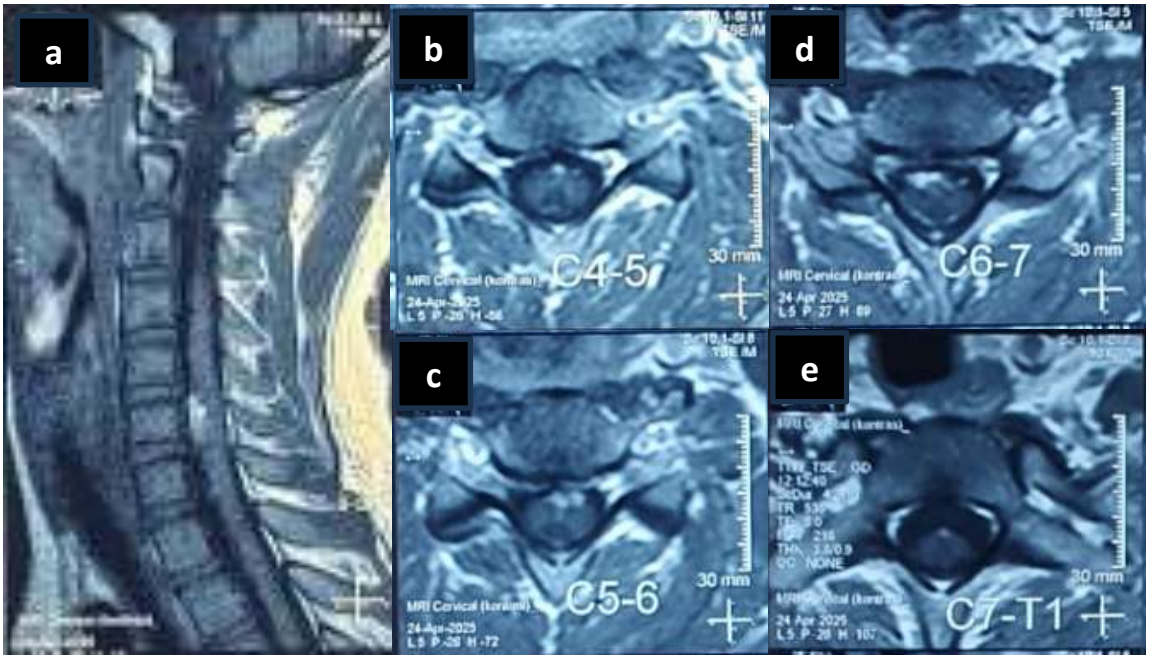

Figure 1. Magnetic Resonance Imaging (MRI) of the cervical spine.

Sagittal (a) and axial (b,c,d,e) views in T1-weighted post-contrast show multifocal late subdural hemorrhage at the C4 and C7 levels.

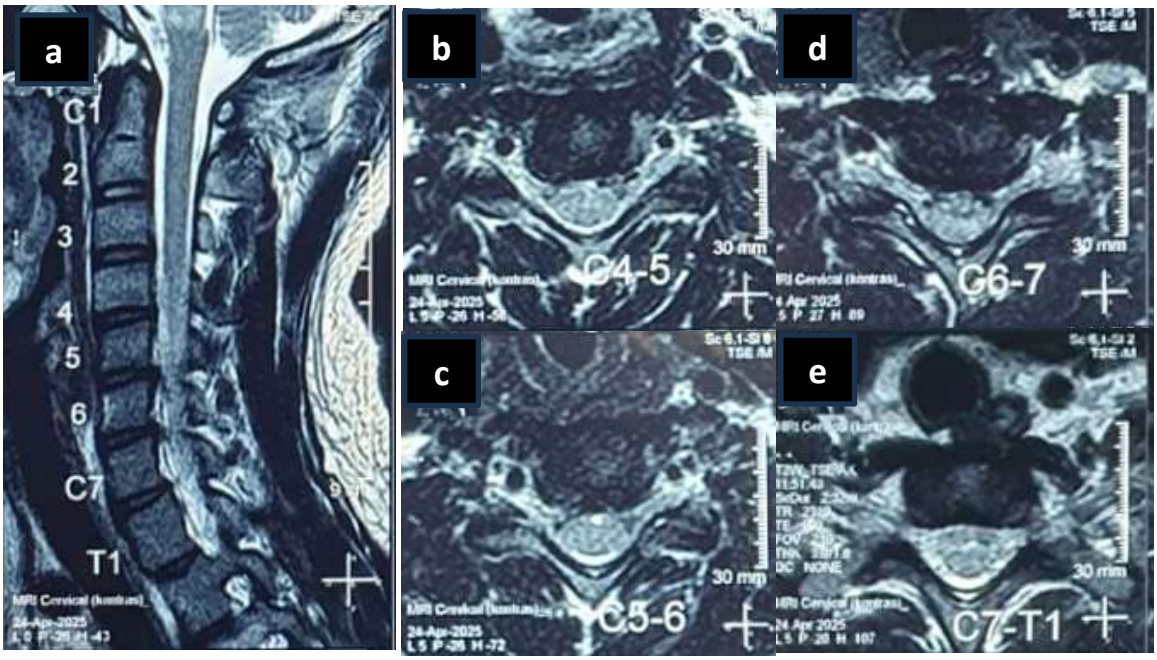

Figure 2. Magnetic Resonance Imaging (MRI) of the cervical spine.

Sagittal (a) and axial (b,c,d,e) views in T2-weighted post-contrast show multifocal late subdural hemorrhage at the C4 and C7 levels

3. DISCUSSION

This case presents a rare instance of nontraumatic spontaneous spinal subdural hemorrhage (SSDH) associated with recent dengue fever, manifesting as acute spastic tetraparesis with sensory deficits and autonomic dysfunction. Although dengue fever is a well-recognized cause of hemorrhagic complications due to thrombocytopenia, spinal subdural hemorrhage remains an exceptionally rare neurological manifestation as a presenting feature of dengue infection.3

The patient had experienced only moderate thrombocytopenia during his dengue illness, suggesting that even non-severe coagulopathy may predispose to spinal hemorrhage. This aligns with observations that the spinal subdural space is avascular, and that SDH may result from bleeding in the subarachnoid space that dissects into the subdural space following rupture of bridging veins or arachnoid membrane compromise. It is plausible that dengue-induced vascular fragility, platelet dysfunction, and transient thrombocytopenia contributed to this pathogenesis.4 Some authors propose that a sudden increase in intraspinal venous pressure, possibly triggered by minor movements or postural shifts, may cause rupture of fragile radiculomedullary or dural veins, particularly when cerebrospinal fluid (CSF) pressure is reduced.4,5

Interestingly, the cervical location (C4–C7) and absence of back pain in this case differ from the more common thoracic presentation of SSDH, which often features sudden severe pain radiating to limbs or trunk followed by progressive neurological decline.6 The lack of back pain may have contributed to a delayed recognition of the spinal etiology in this case. MRI remains the diagnostic modality of choice, with T1 and T2-weighted sequences revealing convex, crescentic lesions surrounding the cord, as seen

here. However, as previously described, atypical imaging appearances may mimic tumoral hematomas or intradural masses, potentially leading to diagnostic confusion.
Given the multifocal and small nature of the lesions, conservative management was deemed appropriate in this case. The decision to avoid surgical decompression is supported by the absence of progressive neurological decline and the patient's stabilization after supportive care. This case highlights the importance of considering spinal hemorrhagic complications in dengue patients, especially in those presenting with new-onset myelopathy even in the absence of trauma or severe thrombocytopenia.

4. CONCLUSION

Spinal subdural hemorrhage (SSDH) is a rare but important neurological complication of dengue fever, even in the absence of severe thrombocytopenia or trauma. This case underscores the potential for dengue-associated coagulopathy and vascular fragility to precipitate spinal bleeding, leading to serious neurological deficits such as spastic tetraparesis and autonomic dysfunction.

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