Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Acute Transverse Myelitis as a Rare Neurological Complication of Dengue Fever: A Case Report

Muhammad Usman Iqbal
King Edward Medical University
Tabassum Ali
King Edward Medical University
Maria Qadri
Jinnah Sindh Medical University
Muhammad Zaid Iqbal
King Edward Medical University
Abdul Salam
King Edward Medical University
Naseeb Danaf
naseeb.danaf@st.ul.edu.lb
Lebanese University

Case Report

Keywords: Dengue fever, Acute transverse myelitis, Case report

Posted Date: November 1st, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10541099/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background/ Introduction

Dengue is a blood borne disease having a consistent presentation of fever, myalgia and thrombocytopenia while neurological involvement in dengue is rarely reported. This case report presents a rare case of dengue induced acute transverse myelitis (ATM) emphasizing the importance of early diagnosis and prompt treatment to prevent permanent neurological deficits.

Case presentation

The patient presented with fever, retro orbital pain and vomiting. A blood sample for dengue serology including IgM and IgG were positive. Later on, he developed sudden onset of paraplegia of lower limbs and loss of sensations below the level of umbilicus. Neurological examination revealed a power of zero in both lower limbs, positive Babinski sign, brisk lower limb reflexes and loss of control of bowel and bladder. Magnetic resonance imaging of the spinal cord revealed diffuse hyper intense signals. A possible diagnosis of ATM was made due to concurrent dengue infection and lack of other findings suggesting a diagnosis other than dengue. He was treated with IV methylprednisolone and tocilizumab and his condition started improving gradually.

Conclusion

This case underscores ATM as a rare yet reversible neurological complication of dengue fever. Early recognition and diagnosis through neuroimaging and exclusion of other causes, followed by timely initiation of high-dose corticosteroids and immunomodulatory therapy, can lead to favorable outcomes. Awareness of this association is very crucial in endemic regions of dengue to ensure prompt treatment and optimal recovery.

1. Introduction

Dengue fever is a painful, debilitating mosquito-borne disease caused by one of the four dengue viruses. Dengue fever is transmitted by the bite of an Aedes mosquito affected by a dengue virus. Dengue fever is a blood borne disease and has a broad spectrum of clinical manifestations and clinical syndromes ranging from subclinical infection to severe infection such as dengue hemorrhagic fever and dengue shock syndrome affecting 100 to 400 million people every year globally especially those residing in urban and semi-urban areas are affected[1, 2]. According to data from publicly accessible sources in the Americas, Africa, and the Western Pacific, more than 100,000 cases of dengue and more than ten dengue-related deaths were reported from 39 nations and territories worldwide in January 2026, reported by European Centre for Disease Prevention and Control[3].

Beyond its clinical manifestations, severe infection of dengue has also been associated with cardiomyopathy, hepatic injury, depression, pneumonia, iritis, orchitis, and neurological syndromes, including seizures, encephalopathy and transverse myelitis[4–6]. Neurological manifestations have been observed in 3 to 4% cases which can range from mild, leading to headache and dizziness, to more critically, causing encephalitis, encephalomyelitis, and seizures[7, 8]. Para-infectious transverse myelitis (TM) in dengue occurs during the acute viremic phase in first few days and is mainly due to direct viral neuroinvasion and replication in the spinal cord, whereas post-infectious myelitis occurs days to weeks after recovery and is caused by immune-mediated demyelination without active viral presence; in contrast, direct viral neuroinvasion specifies detection of dengue virus/antigen in CSF or neural tissue causing inflammation via in situ viral replication, irrespective of timing[9, 10].

However, acute transverse myelitis (ATM) due to dengue fever is particularly rare and underreported complication of dengue fever. Scarcity of data on dengue-induced TM underscores the need for further research and documentation. Herein, we describe a case of ATM caused by dengue fever. The case has been written in accordance with CARE guidelines[11].

2. Case Presentation

2.1 History, examination and investigations

A 62-year-old male presented to the emergency department with complaints of fever, myalgia, retrorbital pain, vomiting and decreased appetite for the last week. On initial evaluation, he had tachycardia, a temperature of 101°F, and the capillary refill time was more than three seconds. A preliminary diagnosis of dengue fever was made, and he was admitted for observation. Initial blood investigations were within normal ranges except for decreased platelet counts, increased hematocrit levels and ALT levels given in Table 1. His dengue IgM and IgG were positive, and he was commenced on oral paracetamol.

Table 1
Initial Complete Blood Report showing Decreased Platelet count and raised Hematocrit and ALT levels

Test	Result	Reference Ranges
TLC (10*9/L)	10.7	4–11
HG (g/dl)	12.5	12–15
PLT (10*9/L)	138	150–450
HCT (%)	48	36–46
Urea (mg/dL)	31	10–50
Creatinine (mg/dL)	1.1	0.2–1.2
ALT (IU/L)	75	10–40
ESR (mm/h)	27	Less than 20
BSR (mg/dL)	116	100–125
eGFR (mL/min/1.73m^2)	72	More than 60

After 30 hours of admission, he developed sudden onset of paralysis of both lower limbs accompanied by loss of sensations below umbilicus and loss of bowel and bladder control. Neurological examination revealed a power of 5/5 of both upper limbs, a power of 0/5 of both lower limbs, positive Babinski sign and brisk lower limb reflexes. He had no signs of meningeal irritation, neck stiffness, or loss of muscle strength. Urgent magnetic resonance imaging (MRI) of the spinal cord was performed, revealing poorly delineated hyper intense signal (FLAIR/T2) sequence **(**Fig. 1**)**.

Detailed blood investigations revealed elevated lactate dehydrogenase, creatine phosphokinase, ferritin, and D-dimer. Viral serology was negative for herpes simplex virus (HSV) 1–2, human immunodeficiency virus (HIV), enterovirus, and varicella-zoster (VZV) virus. Blood culture and gram stain did not grow any organism. His urine screening was negative for any illicit drug use. His cerebrospinal fluid (CSF) analysis was performed, and the results are shown in Table 2.

Table 2
Cerebrospinal fluid (CSF) analysis

Tests	Results	Reference range
White blood cells (mm3)	2	0–5
Red blood cells (mm3)	130	0
Color	Colorless	Colorless
Protein (mg/dL)	66	15–45
Glucose (mg/dL)	81	40–70
Opening pressure (cmH2O)	12	05–20
Gram stain	Negative	Negative
VZV PCR	Negative	Negative
SARS-CoV-2 PCR	Negative	Negative
HSV 1–2 PCR	Negative	Negative
CMV PCR	Negative	Negative
VDRL	Nonreactive	Nonreactive
Bacterial antigen	Negative	Negative

VZV: varicella-zoster virus, PCR: polymerase chain reaction, SARS-CoV-2: severe acute respiratory syndrome coronavirus 2, HSV: herpes simplex virus, CMV: cytomegalovirus, VDRL: venereal disease research laboratory.

2.2 Differential Diagnosis

Differential diagnosis like Guillian Bare Syndrome and hypokalemic periodic paralysis were ruled out because of the presence of signs of upper motor neuron lesions sparing the upper limbs and definite sensory level at umbilicus which signifies the level of lesion in the spinal cord which was later on confirmed through hyperintense lesions in spinal cord on MRI while spinal cord compression was ruled out on MRI. A diagnosis of ATM was made due to concurrent infection of Dengue and fulfillment of Transverse Myelitis Consortium Working Group criteria of ATM. Due to the extension of hyperintense lesion in three spinal segments, it meets the definition of longitudinally extensive transverse myelitis.

2.3 Treatment

Initially, he was treated with intravenous acyclovir and lopinavir in addition to azithromycin empirically for any viral/ infectious myelitis suspicion. However, after the results of CSF analysis, antiviral medications were stopped, he was continued on azithromycin. In addition, intravenous methylprednisolone and tocilizumab were added. Tocilizumab was used off-label in this case in the setting of severe and rapidly progressive neurological deterioration, after weighing potential risks and benefits and was constantly

monitored for any possible adverse effects. His neurological symptoms started improving gradually after 12 to 24 hours. He was responsive, calm, and cooperative after 72 hours of admission. However, a slight postural tremor persisted. He was afebrile on day 5 with no respiratory signs and symptoms. He was discharged on oral azithromycin and dexamethasone with home self-isolation measures on day 12.

2.4 Conclusion and Results (Outcome and Follow-up)

ATM a rare but potentially curable neurological consequence of dengue infection, is highlighted by this case. Together with distinctive MRI findings and the exclusion of other possible causes, the patient's quick development of paraplegia after a confirmed dengue infection supports dengue as the most likely cause. Tocilizumab and high-dose corticosteroids both produced positive results, demonstrating the therapeutic value of early immunomodulatory intervention in reducing neuroinflammation and accelerating neurological recovery. On his recent visit two weeks later, he was doing well with no neurological signs and symptoms. Functional recovery was further supported by the patient’s ability to ambulate without assistance and perform activities of daily living independently at follow-up. He felt reassured and satisfied with the care provided.

3. Discussion

Acute inflammation of one or more spinal cord segments is the hallmark of acute transverse myelitis (ATM), an uncommon but potentially fatal neurological consequence of dengue infection. The characteristic picture of dengue-associated transverse myelitis (TM) in this 62-year-old patient includes sudden-onset paraplegia, sensory loss below the T10 level, and bladder-bowel dysfunction following initial dengue symptoms. The chronological correlation between the feverish illness and neurological deficits, serological confirmation of dengue infection, and characteristic MRI abnormalities all provide strong evidence for dengue as the etiology. Although 4–13% of dengue episodes result in neurological sequelae, ATM is still extremely rare; according to one systematic review, there were only 61 ATM occurrences among 2,672 dengue patients who had neurological symptoms[12–14]. This case contributes to the limited literature on this serious complication and highlights the importance of early recognition and intervention in dengue-endemic regions. Although cases of dengue-associated ATM have been documented over a wide range of ages, the epidemiological pattern shows a little male predominance, with a mean age of presentation of approximately 33.1 years. Although our patient was paralyzed much earlier in the course of the illness, the neurological symptoms usually appear 11.7 days after the onset of dengue symptoms[12].

The diagnosis of dengue-associated TM requires a comprehensive examination to identify the dengue etiology and rule out other causes of myelopathy[15]. The diagnostic procedure in this instance was carried out in accordance with accepted clinical guidelines for TM, starting with neuroimaging to rule out compressive lesions and validate inflammation of the spinal cord. Poorly defined T2 hyperintense signals on MRI that span three to four spinal segments are indicative of inflammatory myelitis and are in keeping with earlier results of dengue-associated TM[16]. Although lymphocytic pleocytosis is frequently seen in TM, the cerebrospinal fluid (CSF) study in this instance revealed a slightly higher protein level (66

mg/dL) without pleocytosis[12, 15]. Although NS1 antigen and IgM antibody testing are considered as specific tests for diagnosing acute dengue infection but NS1 antigen sensitivity starts declining after 5 days of symptoms onset [17], so only antibody testing was done in this patient because of more than a week's history of onset of symptoms.

Additionally given that many viral diseases can have identical presentations, the comprehensive viral serological testing and CSF PCR studies carried out in this instance are an essential part of the diagnostic workup for TM. The link with dengue infection was reinforced by the presence of positive dengue IgM and IgG in serum and the exclusion of other frequent viral etiologies, such as HSV, VZV, HIV, enterovirus, and CMV[18]. Although more conclusive proof of neurotropic invasion would be provided by the identification of dengue-specific IgM in CSF, this test is not always accessible. Mild protein elevation without pleocytosis may occur in early inflammatory myelitis and elevated RBCs likely due to traumatic tap.

The satisfaction of recognized diagnostic criteria for TM, such as bilateral sensorimotor deficits, a clearly defined sensory level, an inflammatory CSF profile, the exclusion of compressive etiology, and progression to nadir within 21 days, further supports the diagnostic certainty in this instance[19]. Furthermore Dengue-associated transverse myelitis is caused by intricate immunological processes that are still being understood. Two main mechanisms are supported by current evidence: post-infectious immune-mediated damage and direct viral invasion of brain tissues. The discovery of dengue virus antigens in CSF and spinal cord tissue during the early stages of infection supports the direct neurotropic hypothesis by proving that the virus can penetrate the blood-brain barrier. By breaking down endothelial tight junctions and encouraging the breakdown of the blood-brain barrier through TLR4-mediated inflammation, the dengue non-structural proteins NS1 and NS3 seem to aid in this neuroinvasion[20].

However this case illustrates the need for immediate immunomodulatory medication in addition to supportive care for the treatment of dengue-associated transverse myelitis. The patient was appropriately started on high-dose intravenous corticosteroids, usually methylprednisolone 1g daily for 3–5 days, which is still the first-line treatment for TM. This strategy seeks to inhibit the inflammatory cascade that causes brain damage, and some case studies have shown positive results in dengue-associated TM. Given the growing evidence of tocilizumab's effectiveness in cytokine-mediated neurological injury, specifically through IL-6 receptor inhibition, its inclusion in this instance constitutes a fresh approach[21].

Notably as shown in this instance, prompt immunomodulatory treatment in addition to supportive care is necessary for the treatment of dengue-associated transverse myelitis. As was appropriately started in this patient, high-dose intravenous corticosteroids, usually methylprednisolone 1g daily for 3–5 days, continue to be the first-line treatment for TM. Several case studies have shown positive results in dengue-associated TM using this strategy, which attempts to inhibit the inflammatory cascade that causes neurological damage. In this instance, tocilizumab's addition is a unique strategy that is backed

by growing evidence of its effectiveness in preventing cytokine-mediated neurological injury, namely through IL-6 receptor blocking [7]. Given the concomitant administration of high-dose corticosteroids, it is not possible to attribute the observed clinical improvement solely to tocilizumab, and the therapeutic effect likely reflects a combined immunomodulatory response.

Moreover plasma exchange (PLEX) is a well-established second-line treatment for individuals who do not respond adequately to initial steroid therapy. It is thought to function by eliminating inflammatory mediators and harmful autoantibodies. When PLEX is not available or in situations that are refractory, intravenous immunoglobulin (IVIG) may also be taken into consideration. This case demonstrates a remarkable neurological improvement after 14 hours of starting therapy, with full symptom clearance at the 2-week mark. This positive result is consistent with earlier studies showing that substantial functional recovery can be facilitated by early, vigorous immunomodulatory therapy[22]. Although recovery patterns vary, the prognosis for dengue-associated TM is generally excellent with timely care. Within the initial few days after symptom onset, the majority of patients experience a maximum deficiency, which gradually improves over the course of weeks to months. Although ongoing improvement may be shown for up to two years, the majority of neurological healing usually takes place in the first three to six months. Rapid symptom progression, full paraplegia at presentation, and spinal shock are factors linked to a worse outcome. This case's good outcome—complete remission within two weeks—highlights the possibility of full functional recovery with rapid detection and treatment[23].

Although there is a substantial clinical and chronological correlation between dengue illness and TM, the strength of the causal conclusion is limited by some diagnostic limitations. The first is that direct confirmation of viral neuroinvasion was not done in the cerebrospinal fluid (CSF) in this patient however it did not alter the diagnostic certainty because dengue specific IgM antibodies were detected in serum. Second, due to the individual diversity in immune responses and clinical course, the results may not be applicable to all dengue patients, as is the case with most case reports. Third, the absence of measurement of IL-6 levels limits the ability to directly justify targeted IL-6 inhibition by tocilizumab. Fourth, the lack of long-term follow-up data on recurrence or remaining neurological damage restricts the evaluation of long-term therapy efficacy. Lastly, extensive viral and autoimmune testing that could improve diagnostic confidence is frequently hindered by resource limitations in many dengue-endemic areas. This case thus highlighted the significance of early detection, suitable diagnostic assessment, and vigorous treatment of dengue-associated TM in endemic areas and offered important clinical insight into the growing neurological spectrum of dengue.

Learning points:

- Dengue fever can present with Acute Transverse Myelitis (ATM), causing sudden paralysis, sensory loss, and bowel/bladder dysfunction.
- Spinal MRI confirms spinal cord inflammation, while serum Dengue IgM/IgG tests help confirm the infection when presentation occurs past the first week.

- Early high-dose IV corticosteroids (e.g., methylprednisolone)—alone or combined with targeted immunomodulators like Tocilizumab—are critical to stop spinal damage and allow full neurological recovery.
- In dengue-endemic areas, physicians should immediately evaluate any sudden weakness or neurological deficits following a feverish illness.

Declarations

Ethics approval and consent to participate

It is a case report exempted from Ethical Approval by the institutional Board of Review, King Edward Medical University Lahore. The patient consented to the use of his data for this publication.

Consent for Publication

Written informed consent was taken from the patient to publish this rare under-reported case presentation and a copy of the written consent will be available for review by the Editor-in-Chief of this journal on request.

Competing interests

The authors have no competing interests to report.

Funding

The authors received no funding for this case report from any organization.

Author Contribution

M.U.I: Conceptualization, Methodology, Data curation, Writing Original draft preparation, Reviewing and Editing; T.A: Methodology, Writing Original draft preparation, Reviewing, and Editing; M.Q: Writing Original draft preparation, Reviewing, and Editing; M.Z.I: Writing Original draft preparation, Reviewing, and Editing; A.S: Writing Original draft preparation, Reviewing, and Editing; N.D: Writing Original draft preparation, Reviewing, and Editing.

Acknowledgement

The authors thank the patient for granting permission to share his clinical information for educational purposes.

Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

References

1. Dengue n.d. https://www.who.int/news-room/fact-sheets/detail/dengue-and-severe-dengue (accessed October 17, 2025).
2. Guzman MG, Gubler DJ, Izquierdo A, Martinez E, Halstead SB. Dengue infection. Nat Rev Dis Primer 2016;2:16055. https://doi.org/10.1038/nrdp.2016.55.
3. Dengue worldwide overview 2026. https://www.ecdc.europa.eu/en/dengue-monthly (accessed March 29, 2026).
4. Kularatne SAM, Rajapakse MM, Ralapanawa U, Waduge R, Pathirage LPMMK, Rajapakse RPVJ. Heart and liver are infected in fatal cases of dengue: three PCR based case studies. BMC Infect Dis 2018;18:681. https://doi.org/10.1186/s12879-018-3603-x.
5. Giri S, Agarwal MP, Sharma V, Singh A. Acute hepatic failure due to dengue: A case report. Cases J 2008;1:204. https://doi.org/10.1186/1757-1626-1-204.
6. Li G-H, Ning Z-J, Liu Y-M, Li X-H. Neurological Manifestations of Dengue Infection. Front Cell Infect Microbiol 2017;7:449. https://doi.org/10.3389/fcimb.2017.00449.
7. Murthy JMK. Neurological complications of dengue infection. Neurol India 2010;58:581. https://doi.org/10.4103/0028-3886.68654.
8. Badat N, Abdulhussein D, Oligbu P, Ojubolamo O, Oligbu G. Risk of Transverse Myelitis Following Dengue Infection: A Systematic Review of the Literature. Pharmacy 2018;7:3. https://doi.org/10.3390/pharmacy7010003.
9. Naderian R, Paraandavaji E, Maddah AH, Keshavarzi S, Habibian A, Naderian R, et al. Pathophysiology and clinical implications of dengue-associated neurological disorders. Front Microbiol 2025;16:1536955. https://doi.org/10.3389/fmicb.2025.1536955.
10. Pires BF, de Souza CGN, de Campos ECM, Rajab T, de Albuquerque MF, do Carmo JVP, et al. Post-dengue transverse myelitis: a challenging case of neurological and therapeutic evidence. Oxf Med Case Rep 2026;2026:148–50. https://doi.org/10.1093/omcr/omag005.
11. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D, et al. The CARE guidelines: consensus-based clinical case reporting guideline development. BMJ Case Rep 2013;2013:bcr2013201554. https://doi.org/10.1136/bcr-2013-201554.
12. Karishma F, Harsha F, Rani S, Siddique S, Kumari K, Mainka F, et al. Acute Transverse Myelitis as an Unusual Complication of Dengue Fever: A Case Report and Literature Review. Cureus 2024. https://doi.org/10.7759/cureus.54074.

13. Trivedi S, Chakravarty A. Neurological Complications of Dengue Fever. Curr Neurol Neurosci Rep 2022;22:515–29. https://doi.org/10.1007/s11910-022-01213-7.
14. Noronha AK, T. AM, Rupali P. Dengue encephalitis: what's new? Curr Opin Infect Dis 2025;38:364–71. https://doi.org/10.1097/QCO.0000000000001128.
15. Kumar N, Mishra R, Bargali A, Bhavana RI. Dengue-associated longitudinally extensive transverse myelitis. BMJ Case Rep 2024;17:e259388. https://doi.org/10.1136/bcr-2023-259388.
16. Mirbagheri S, Eckart Sorte D, Zamora CA, Mossa-Basha M, Newsome SD, Izbudak I. Evaluation and management of longitudinally extensive transverse myelitis: a guide for radiologists. Clin Radiol 2016;71:960–71. https://doi.org/10.1016/j.crad.2016.05.020.
17. Hu D, Di B, Ding X, Wang Y, Chen Y, Pan Y, et al. Kinetics of non-structural protein 1, IgM and IgG antibodies in dengue type 1 primary infection. Virol J 2011;8:47. https://doi.org/10.1186/1743-422X-8-47.
18. Kabir MA, Zilouchian H, Younas MA, Asghar W. Dengue Detection: Advances in Diagnostic Tools from Conventional Technology to Point of Care. Biosensors 2021;11:206. https://doi.org/10.3390/bios11070206.
19. Li G-H, Ning Z-J, Liu Y-M, Li X-H. Neurological Manifestations of Dengue Infection. Front Cell Infect Microbiol 2017;7:449. https://doi.org/10.3389/fcimb.2017.00449.
20. De Sousa AM, Alvarenga MP, Alvarenga RMP. A Cluster of Transverse Myelitis Following Dengue Virus Infection in the Brazilian Amazon Region. Trop Med Health 2014;42:115–20. https://doi.org/10.2149/tmh.2014-06.
21. Shrestha K, Poudel B, Shrestha S, Rai BP, Rajbhandari P, Mishra DK. An unusual case of transverse myelitis in dengue fever: A case report from Nepal. Clin Case Rep 2024;12:e8461. https://doi.org/10.1002/ccr3.8461.
22. Queiroz ALGD, Soares Neto HR, Kobayashi TT, Silva SMCDA. Plasma exchange in inflammatory demyelinating disorders of the central nervous system: reasonable use in the clinical practice. Arq Neuropsiquiatr 2023;81:296–307. https://doi.org/10.1055/s-0042-1758447.
23. Schaefer TJ, Panda PK, Wolford RW. Dengue Fever. StatPearls, Treasure Island (FL): StatPearls Publishing; 2025.

Figures



Figure 1

T-2-weighted-MRI-spinal cord-showing-hyperintense-signals-extending for 3-4 spinal segments