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A Para-Infectious Neurological Syndrome Complicating Dengue Fever: A Case Report

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Case Report

Keywords: dengue fever, Expanded Dengue Syndrome, acute disseminated encephalomyelitis, Miller-Fisher syndrome, Sri Lanka, case report

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A Para-Infectious Neurological Syndrome Complicating Dengue Fever: A Case Report

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Abstract

Background: To our knowledge, this is the first reported case combining prominent central features with a peripheral, Miller-Fisher-like component in a single patient, illustrating that dengue-associated neurological syndromes may extend beyond the discrete categories of encephalitis, ADEM, or Miller-Fisher syndrome described individually in prior reports.

Case presentation: A previously well 39-year-old man presented with fever, arthralgia, and headache and was found to have NS1-positive dengue fever, with typical leucopenia, thrombocytopenia, and transaminitis. Fever recurred on the ninth day, with depressed consciousness, urinary retention, meningism, nonreactive pupils, nystagmus, multiple cranial nerve palsies, and areflexic quadriparesis. Cerebrospinal fluid showed lymphocytic pleocytosis with elevated protein, and imaging revealed nonspecific white matter lesions, consistent with a combined central and peripheral nervous system para-infectious syndrome. As the response to corticosteroids was inadequate, the patient received intravenous immunoglobulin and plasma exchange, with partial neurological recovery of upper-limb and sphincter function but persistent lower-limb weakness at two months.

Conclusions: This case broadens the recognized phenotype of para-infectious neurological syndromes complicating dengue fever, showing that central and peripheral features can coexist in a single patient. Clinicians evaluating atypical or overlapping neurological presentations during or after dengue fever should consider this broader spectrum when forming a differential diagnosis.

Key words: dengue fever; Expanded Dengue Syndrome; acute disseminated encephalomyelitis; Miller-Fisher syndrome; Sri Lanka; case report.

Background

Dengue fever is the most common arboviral infection in Sri Lanka; it is caused by four viruses DENV-1 to DENV-4 and is usually self-limiting. Apart from the typical presentations of dengue fever or dengue hemorrhagic fever, uncommon clinical presentations are grouped under Expanded Dengue Syndrome (EDS). EDS is characterized by cardiac, neurological, gastrointestinal, renal, and hematopoietic system involvement.(1,2)

Neurological complications of dengue are increasingly recognized; reported in approximately 0.5–21% of dengue cases, predominantly in association with DENV-2 and DENV-3.(3–5) These complications are divided into three categories according to the mechanism of injury: direct viral neuro-invasion (encephalitis, myositis), systemic or metabolic derangement (encephalopathy, stroke), and postinfectious, immune-mediated syndromes. The latter group includes acute demyelinating encephalomyelitis (ADEM), Guillain–Barré syndrome (GBS), Miller-Fisher syndrome and Bickerstaff brainstem encephalitis, and typically presents with a monophasic illness affecting the central and/or peripheral nervous system days to weeks after the infection.(4,6–8)

We report a case of an adult who developed a broad-spectrum neurological syndrome, combining central and peripheral features, during a single hospital admission for dengue fever, emerging shortly after the initial fever had settled. Such a combined presentation has not, to our knowledge, been previously described in the dengue literature, in which central (ADEM) and peripheral (Guillain-Barré/Miller-Fisher) syndromes are typically reported as distinct entities.

Case Presentation

A 39-year-old man with a history of recent travel to a dengue-endemic area presented with fever, arthralgia, giddiness, headache, retro-orbital pain, backache, diarrhea and poor appetite,

without any history of bleeding. He had no significant past medical history, was not on any regular medication, and there was no family history of neurological or autoimmune disease.

The patient was admitted on day 4 of illness. On admission, the patient was hemodynamically stable (pulse rate 85/min, blood pressure 135/75 mmHg, capillary refill time < 2 seconds) with a Glasgow Coma Scale (GCS) score of 15/15 and diagnosed with dengue fever confirmed with a positive dengue nonstructural protein 1 (NS1) antigen test.

Fluids were titrated at 100 mL/hour on days 4 to 8 of illness. Gallbladder wall edema was noted on ultrasound without evidence of plasma leakage by day 5, after which the patient developed constipation without vomiting or abdominal pain. Fever settled on the evening of day 6. By day 7, the patient had reduced urine output with persistent dizziness and a poor appetite, although a repeat scan was negative for free fluid in the abdomen; by day 8, the patient had been fever-free for 48 hours.

On day 9 of illness, fever recurred (temperature 38.9°C), and the patient became poorly responsive, with no urine passed since the morning and no bowel motion. He did not report seizures. On reassessment the pulse rate was 106/min, the blood pressure was 135/88 mmHg, the GCS was 9/16 (Eye response 3 of 4, Verbal response 1 of 5, Motor response 5 of 6), oxygen saturation was 96% on room air, the respiratory rate was 20/min and clear lung fields were present. The bladder was palpable, in keeping with acute urinary retention, and the patient was catheterized, with an initial urine output of 1500mL. Neck stiffness was noted.

The patient responded nonverbally to commands and appeared to understand simple questions but was unable to articulate sounds or phonate. Cranial nerve examination revealed poor bilateral lateral eye movements with nystagmus greater on the left than on the right, and both pupils 3–4 mm and nonreactive to light and bilateral partial ptosis (cranial nerves III, IV, VI); poor mouth opening with sensory testing that was difficult to assess (V); inability to wrinkle the

forehead with no facial expression in the bilateral lower face (VII); normal hearing (VIII); absence of phonation with no palatal movement (IX, X); and poor tongue movements (XII).

Limb examination revealed reduction in tone and power, affecting proximal and distal muscle groups in all four limbs, with hyporeflexia and diminished plantar responses (Table 1).

(Table 1 should be located here)

Table 1. Limb examination.

	Right UL	Left UL		Right LL	Left LL
Tone	Reduced	Reduced	–	Reduced	Reduced
Power – proximal muscles	3/5	3/5	–	3/5	3/5
Power – distal muscles	2/5	2/5	–	2/5	2/5
Biceps reflex	1	1	**Knee reflex**	0	0
Triceps reflex	1	1	**Ankle reflex**	0	0
Supinator reflex	Equivocal	Equivocal	**Plantar response**	Diminished	Diminished

UL = upper limb, LL = lower limb. Muscle power was graded from 0–5 according to the Medical Research Council (MRC) scale (0 = no power; 1 = flicker or trace of contraction; 2 = active movement with gravity eliminated; 3 = active movement against gravity; 4 = active movement against gravity and resistance, 5 = normal power).(9) Deep tendon reflexes were graded 0–4 according to the NINDS Myotatic Reflex Scale (0 = absent; 1 = small, less than normal; 2 = lower half of the normal range; 3 = upper half of the normal range; 4 = enhanced, more than normal, including clonus).(10) The supinator response was equivocal and could not be reliably graded on this scale. Plantar responses are superficial (cutaneous) reflexes, not deep tendon reflexes, and are therefore reported qualitatively rather than on the NINDS scale.

Serial blood investigations (Table 2) revealed a thrombocytopenic nadir of 44 x 10^9/L on day 7 with recovery by day 11, alongside a secondary increase in white cell count and C-reactive protein (CRP) in the second week of illness. Blood and urine cultures were negative. HIV-1/2 antigen and *Treponema pallidum* antibody tests were negative.

(Table 2 should be located here)

Table 2. Hematological and biochemistry trends.

Parameter	Normal range	D3	D4	D5	D6	D7	D8	D9	D10	D20
RBC (x10¹²/L)	3.5–5.5	5.21	5.37	4.90	5.13	4.94	5.10	5.04	5.94	5.92
Hb (g/dL)	11.0–16.0	15.7	15.3	14.0	15.0	14.9	14.8	14.4	16.3	17.2
HCT (%)	37.0–54.0	44.8	45.7	41.5	42.8	41.2	42.8	42.0	48.6	49.6
WBC (x10⁹/L)	4.0–10.0	4.84	3.28	2.73	2.28	4.78	6.31	8.99	24.47	13.6
Neutrophils (x10⁹/L)	2.0–7.0	3.99	2.21	2.03	1.04	2.10	1.75	6.86	21.82	10.6
Lymphocytes (x10⁹/L)	0.8–4.0	0.34	0.67	0.50	0.89	1.86	2.65	1.61	1.23	1.92
Platelets (x10⁹/L)	150–450	169	134	110	68	44	55	166	194	329
ALT (U/L)	4.0–45.0	–	51.2	46.4	–	–	–	91.2	91.5	68.3
AST (U/L)	4.0–35.0	–	46.2	46.3	–	–	–	86.8	84.3	59.2
CRP (mg/dL)	<6	–	12.3	–	–	–	–	6	8.41	3.9
ESR (mm/first hour)	<15	–	–	–	–	–	–	35	–	25
Dengue NS1 Ag	–	–	Positive	–	–	–	–	–	–	–
Na (mmol/L)	135–145	–	139	–	–	–	–	141	140.5	134.8
K (mmol/L)	3.5–5.0	–	3.9	–	–	–	–	4.2	4.4	4.79
Creatinine (μmol/L)	57–97	–	95.4	–	–	–	–	–	277.5	78.5
Mg (mmol/L)	0.7–1.2	–	–	–	–	–	–	1.08	1.30	–
Total Ca (mmol/L)	2.15–2.5	–	–	–	–	–	–	1.95	2.37	–
Corrected Ca (mmol/L)	–	–	–	–	–	–	–	2.0	2.4	–
Total protein (g/dL)	6.0–8.3	–	–	–	–	–	–	7.3	–	–
Albumin (g/dL)	3.5–5.2	–	–	–	–	–	–	3.7	3.9	–
ALP (U/L)	40.0–147.0	–	–	–	–	–	–	58.8	–	–
GGT (U/L)	5.0–61.0	–	–	–	–	–	–	42.8	–	–
Total bilirubin (mg/dL)	0.3–1.2	–	–	–	–	–	–	0.6	–	–
Direct bilirubin (mg/dL)	0.1–0.3	–	–	–	–	–	–	0.2	–	–
Creatine kinase (U/L)	18–174	–	–	–	–	–	–	1120	–	482

RBC, red blood cell; Hb, hemoglobin; HCT, hematocrit; WBC, white blood cell; ALT, alanine amino transferase; AST, aspartate aminotransferase; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; NS1 Ag, nonstructural protein 1 antigen; Na, sodium; K, potassium; Mg, magnesium; Ca, calcium; ALP, alkaline phosphatase; GGT, gamma-glutamyl transferase.

Cerebrospinal fluid (CSF) was colorless and slightly turbid. Microscopy revealed a pleocytosis of 110 cells/μL, predominantly lymphocytic, with only 3 polymorphs/mm³, alongside 96 red blood cells/μL. Glucose was 3.70 mmol/L, against a paired random blood sugar of 9.76 mmol/L; CSF-to-blood glucose ratio was 0.38. Protein was 99.7 mg/dL (reference range 15.0–45.0 mg/dL). CSF bacterial culture revealed no growth. CSF for Japanese encephalitis virus and herpes simplex 1 and 2 virus serology were negative. CSF GeneXpert was negative.

Electroencephalography revealed generalized theta slowing as the background rhythm, with runs of diffuse epileptiform discharges, suggestive of an encephalopathic/encephalitic process. MRI brain revealed a few deep white matter lesions and periventricular white matter lesions without diffusion restriction or contrast enhancement and no mass effect (Figure 1).

(Figure 1 should be located here)

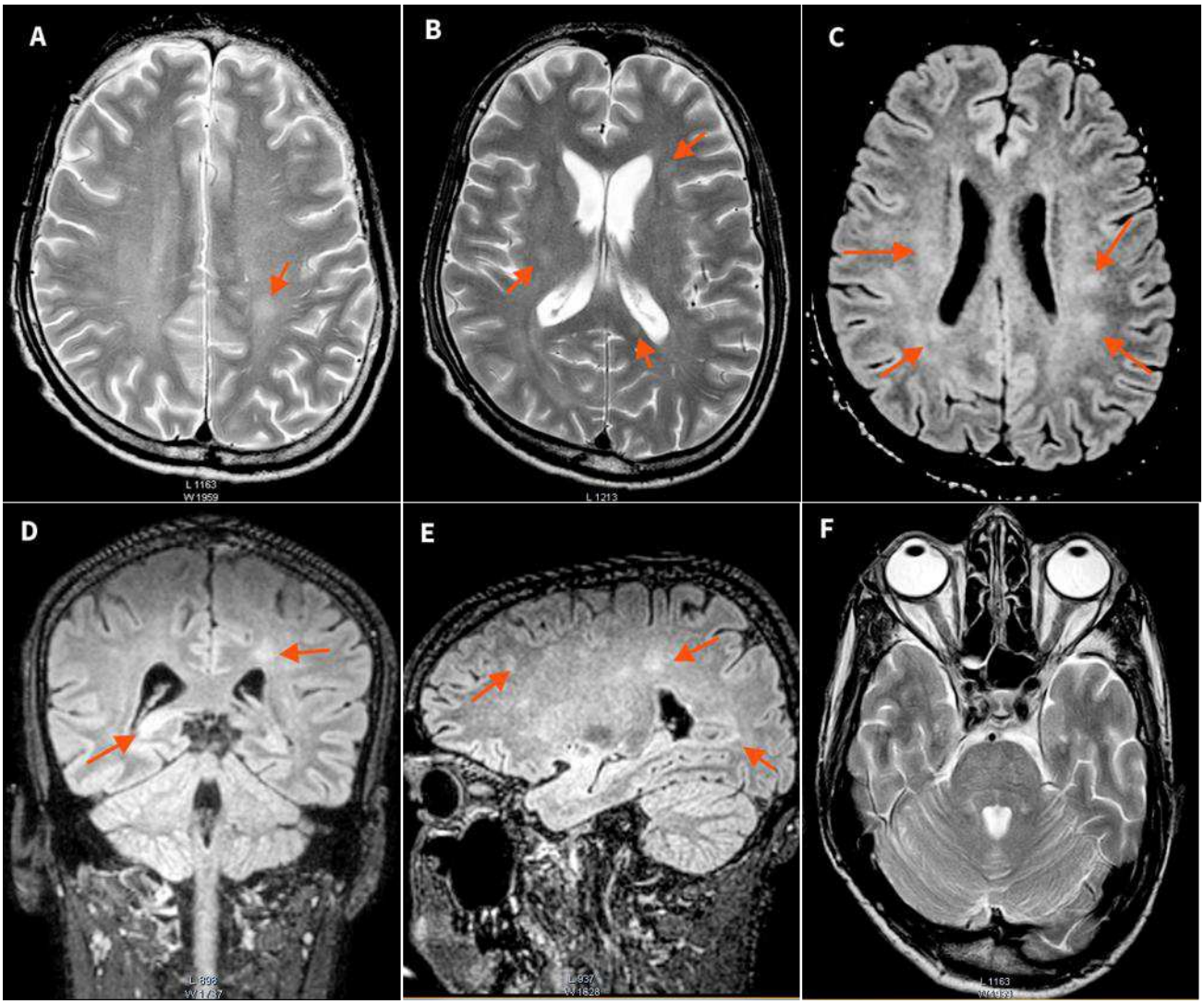


Figure 1. MRI brain shows periventricular and subcortical white matter, and hippocampal hyperintensities. [A and B, T2-weighted MRI images show periventricular and sub-cortical white matter hyperintensities. C, D and E, FLAIR images reveal white matter hyperintensities periventricular regions (C and E), and the right hippocampus and fornix (D). F, T2-weighted MRI images show normal orbits, pons and cerebellum.]

Serotype-specific PCR, anti-ganglioside antibody testing, and nerve conduction studies were not locally available at the time of admission, which limited further diagnostic characterization of this presentation.

The differential diagnosis at the time of neurological deterioration included dengue encephalitis, classical acute disseminated encephalomyelitis (ADEM), Miller-Fisher spectrum disorders, and

metabolic encephalopathy secondary to the recent dengue illness. Direct viral encephalitis was considered less likely because of the interval after fever defervescence, the absence of a demonstrable pathogen in CSF, and the MRI pattern of non-enhancing white-matter lesions. Pure ADEM did not fully account for the multiple cranial-nerve palsies, nystagmus and areflexic quadriparesis, while an isolated peripheral-nerve syndrome could not explain the encephalopathy and white-matter changes. An immune-mediated central–peripheral overlap syndrome was therefore favored.

With the onset of neurological deterioration on day 9 of illness, initial treatment comprised ceftriaxone and acyclovir to treat possible meningoencephalitis and a single dose of intravenous dexamethasone 8mg. Following definitive diagnosis of demyelination, intravenous methylprednisolone was initiated at 1000 mg/day for 5 days, followed by oral prednisolone 40 mg/day on a tapering regimen and intravenous immunoglobulin (IVIG) 2g/kg body weight divided over 5 days. Therapeutic plasma exchange was commenced for six cycles after the initial administration of methylprednisolone and IVIG, owing to an inadequate clinical response. Methylprednisolone and plasma exchange were well tolerated, with no infusion reactions or other adverse events observed during treatment. The patient recovered partially following completion of plasma exchange and required an additional intravenous immunoglobulin dose of 2g/kg and comprehensive physiotherapy; he developed a minor allergic reaction to this second dose of IVIG.

Following the acute phase of treatment, the patient was transferred to a rehabilitation hospital, where he underwent comprehensive physiotherapy and occupational therapy for one month. At follow-up, two months from the onset of his condition, he remained bedbound due to lower-limb weakness, although upper-limb activity and bladder and bowel function had returned to normal. Given the ongoing clinical improvement, full functional recovery is anticipated. A follow-up MRI of the brain is planned at six months to assess radiological resolution of the white-matter

lesions. The clinical course, investigations, and treatment escalation are summarized in Figure 2.

(Figure 1 should be located here)

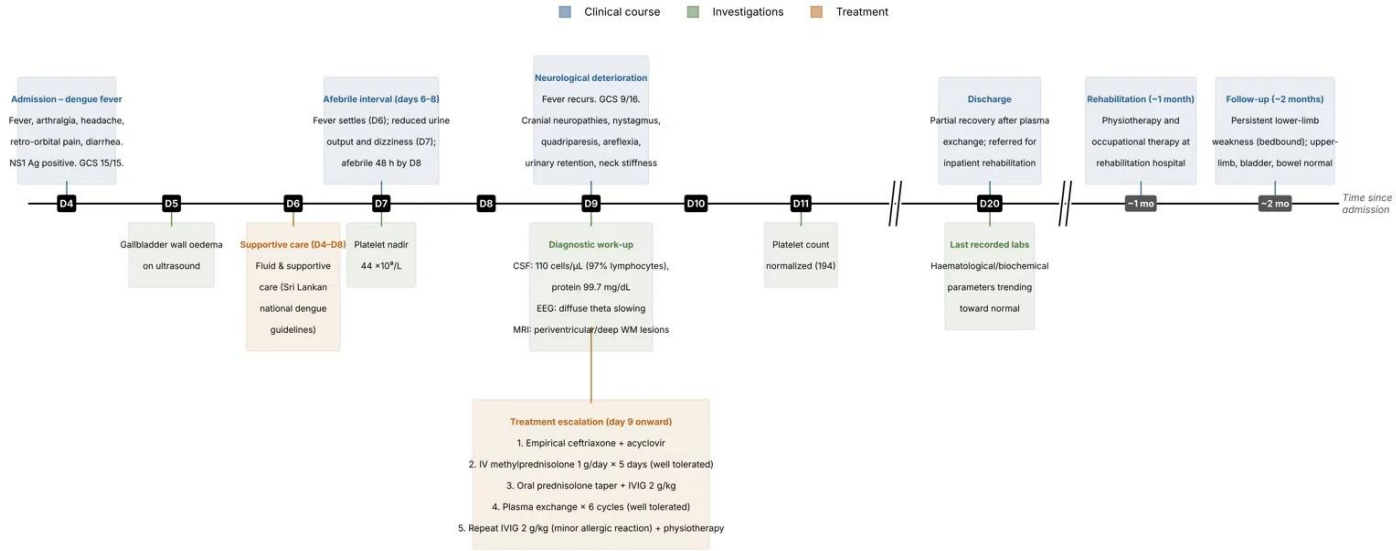


Figure 2. Timeline of the clinical course, investigations, and treatment escalation. [The illness-day axis (D4–D20) is spaced ordinally rather than to linear scale. Checkpoints beyond acute admission (~1 month, ~2 months) denote approximate time since admission. Boxes are color-coded by category: blue, clinical course; green, investigations; orange, treatment. CSF, cerebrospinal fluid; EEG, electroencephalography; GCS, Glasgow Coma Scale; IVIG, intravenous immunoglobulin; MRI, magnetic resonance imaging; NS1 Ag, dengue nonstructural protein 1 antigen; WM, white matter.]

(Table 3 near here – placed at the end of the text file, after the references, as it exceeds one page)

Discussion and conclusions

This case adds to the growing body of literature describing para-infectious neurological syndromes complicating dengue fever and is notable for its neurological involvement combined

with encephalopathy, multiple cranial neuropathies, oculomotor signs, and quadriparesis with areflexia, together with lymphocytic pleocytosis, elevated CSF proteins and nonspecific periventricular white matter changes, which is most consistent with immune-mediated neurological syndromes of EDS.

Among the ADEM cases summarized in Table 3, the onset of neurological symptoms ranged from day 4 to day 20 after the onset of dengue. This is broadly consistent with the day 3–19 window (median day 7) reported across pooled cases by Kamel et al.(11) ; our patient's day-9 onset falls within both ranges. Dengue-associated ADEM remains rare, estimated at 0.4% of dengue infections and 6.8% of dengue cases with neurological complications.

Reported cases of dengue-associated ADEM typically present with limb weakness and isolated cranial nerve palsy (usually facial); our patient's combination of multiple bilateral cranial neuropathies, nystagmus and quadriparesis with diminished reflexes is considerably broader than the pure ADEM phenotype described elsewhere. This pattern overlaps with descriptions of Miller Fisher–GBS spectrum, a continuum for which dengue itself has been implicated as a trigger(4), raising the possibility that this represents a combined central–peripheral autoimmune process rather than classical ADEM alone.

Soares et al. demonstrated that dengue-associated GBS and myelitis share a common CSF signature, a raised albumin quotient indicating blood–CSF barrier dysfunction, distinct from the CSF profile of dengue encephalitis, which is more often normal or mildly pleocytosis.(12) Our patient's CSF (lymphocytic pleocytosis, markedly elevated protein) sits closer to the myelitis/GBS pattern than to the typically unremarkable CSF of dengue encephalitis, lending some support to an overlap-syndrome interpretation.

Distinguishing ADEM from dengue encephalitis is a recurring difficulty in literature; both can present with altered consciousness and nonspecific imaging in the febrile period or immediate post-febrile period (13,14). Normal CSF cellularity cannot reliably exclude dengue encephalitis:

Soares et al. (2006) (12) reported pleocytosis in only 28.5% and elevated protein (>45g/dL) in 43% of patients with dengue encephalitis, while GBS and myelitis patients together presented higher rates of both (pleocytosis 50%, elevated protein 66%). In a larger series, Soares et al. (2011) (15) similarly reported pleocytosis in 25% of dengue encephalitis patients, with elevated protein in 63%. The pleocytosis and elevated protein observed in our patient are not diagnostic but are somewhat more pronounced than typically described in dengue encephalitis patients and may indicate an immune-mediated pathology.

The peripheral component of this overlap syndrome is supported by the wider literature on dengue-associated Guillain-Barré syndrome (GBS). A systematic review of 56 reported cases found a median latency of 7 days from dengue onset to GBS, with albuminocytologic dissociation in 62.5% of cerebrospinal fluid samples and an axonal electrophysiological pattern in almost half of cases; most patients were treated with IVIG, with full or partial recovery in the majority and a reported mortality of 5.4%.(16) Reported adult cases of dengue-associated GBS and MFS have been attributed to differing mechanisms. In one Sri Lankan case, normal CSF and neuroimaging, together with detectable dengue viral RNA in serum, were interpreted as evidence of direct viral neurotropism rather than a para-infectious process.(17) In another, a more typical demyelinating GBS with albuminocytologic dissociation followed the expected post-infectious pattern and resolved fully with IVIG alone.(18) Our patient's lymphocytic pleocytosis, markedly elevated CSF protein, and demyelinating-pattern white matter lesions are more consistent with an immune-mediated, para-infectious mechanism than with direct neuroinvasion, supporting the overlap-syndrome interpretation proposed here for the peripheral component of this presentation.

ADEM lesions on MRI generally follow one of three patterns: multifocal white matter involvement (with or without basal ganglia extension); grey-matter–restricted lesions; or localized brainstem/basal ganglia/cerebellar disease.(5) Our patient's periventricular white matter lesions fall within the first pattern. Several features support a para-infectious rather than

a direct infective process: the interval between fever resolution and neurological deterioration, pleocytosis, the absence of a demonstrable pathogen in CSF, and the pattern of white matter change on the MRI scan.

Kamel et al.'s (2017) pooled analysis of dengue-associated ADEM revealed 16 complete recoveries, 7 partial, and 3 deaths, with no association between outcome and steroid regimen.(11) The case reported by Diallo et al. received oral rather than pulsed intravenous steroids and progressed to loss of brainstem reflexes and death.(5) Our patient's escalation to IVIG and plasma exchange after an inadequate steroid response is unusual among reported cases but fits the overlap-syndrome interpretation: trial evidence in GBS shows plasma exchange and IVIG, but not steroids alone, to alter outcome.(4)

The dengue serotype was not determined, precluding correlation with the serotypes (DENV-2 and DENV-3) most frequently implicated in neurological complications; CSF dengue antigen and antibody testing were not performed. Anti-ganglioside antibodies and nerve conduction studies were not performed, limiting formal characterization of the peripheral component of this presentation. The strengths of this report include detailed serial clinical, hematological, and biochemical data spanning the full course of illness, cerebrospinal fluid analysis obtained at the time of neurological deterioration, and a clearly documented treatment-escalation pathway, allowing close correlation between the clinical course and the immunomodulatory response.

This case illustrates that dengue-associated neurological syndromes can extend beyond the discrete categories of encephalitis, ADEM, or MFS described individually in prior reports and may instead present as an overlap syndrome spanning the central and peripheral nervous systems, sitting at the more severe end of the EDS spectrum. To our knowledge, this is the first reported case of dengue-associated ADEM combining prominent central features with a peripheral, MFS-like component in a single patient. Recognizing this broad phenotype is clinically important: an inadequate response to first-line corticosteroid therapy in a patient with

combined central and peripheral or brainstem involvement, as in this case, may indicate a more extensive underlying process than isolated ADEM or Miller-Fisher syndrome.

Patient's perspective

I am deeply shocked and saddened by the situation in which I was confined to bed for more than two months because of dengue fever and neurological impairment. Nevertheless, I am striving to strengthen my resolve day by day, with the goal of regaining the ability to stand on my own feet and return to my former life with my wife and children. In the meantime, I make every effort to perform all tasks that I am currently capable of handling on my own. My only goal has been to get back on my feet. I extend my heartfelt gratitude to everyone who has provided me with strength and encouragement throughout this ordeal. Having been in a life-and-death struggle with this disease, I understand the value of life very well. My only wish is that no living beings should ever have to endure illness or suffering.

List of abbreviations

ADEM, acute demyelinating encephalomyelitis; ALP, alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CRP, C-reactive protein; CSF, cerebrospinal fluid; DENV, dengue virus; EDS, Expanded Dengue Syndrome; EEG, electroencephalography; ESR, erythrocyte sedimentation rate; GBS, Guillain–Barré syndrome; GCS, Glasgow Coma Scale; GGT, gamma-glutamyl transferase; Hb, hemoglobin; HCT, hematocrit; IVIG, intravenous immunoglobulin; IVMP, intravenous methylprednisolone; LL, lower limb; MFS, Miller-Fisher syndrome; MRC, Medical Research Council (scale); MRI, magnetic resonance imaging; NINDS, National Institute of Neurological Disorders and Stroke (Myotatic Reflex Scale); NS1, nonstructural protein 1 antigen; RBC, red blood cell; TPE, therapeutic plasma exchange; UL, upper limb; WBC, white blood cell; WM, white matter.

Declarations

Ethics approval and consent to participate: Institutional policy does not require formal ethics committee approval for the publication of a single, de-identified case report.

Consent for publication: Written informed consent for publication of this case report and accompanying clinical images was obtained from the patient. A copy of the written consent form is available for review by the Editor-in-Chief of this journal.

Availability of data and materials: All data generated or analyzed during this case are included in this published article.

Competing interests: The authors declare that they have no competing interests.

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Authors' contributions: RNCTF conceived the report, managed the patient's clinical care, performed the neurological assessment, drafted the manuscript, prepared the figures and tables, and critically revised it. HDY contributed to conception of the report, data acquisition, and the neurological examination, drafted sections of the manuscript, and critically revised it. DGHR contributed to the clinical management of the patient, data acquisition, and obtained MRI images. ABNNP, as consultant neurologist, supervised clinical management, examined the patient, interpreted the neuroimaging, provided expert opinion on the differential diagnosis, and critically revised the manuscript. RMADBA, as consultant physician, supervised clinical management, examined the patient, and provided expert opinion on the differential diagnosis, and critically revised the draft. NGGCSN, WMSW, and LKM contributed to the clinical management of the patient, and data acquisition. All authors read and approved of the final manuscript.

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Table 3. Comparison of clinical profiles, investigations and management of reported dengue-associated ADEM cases in adults.

Author, Year, Country	Age/Sex; Onset (day)	Key clinical features	Investigations (CSF / imaging)	Management and outcome
Gupta et al., 2013 — India(19)	26/F; D6	Coma (GCS 8/15), fever rash, long tract signs	CSF: 10 cells (all lymphocytes), protein 58 mg/dL. MRI: temporal lobe, cerebellum, callosal-septal interface, subcortical and periventricular WM of frontal and parietal lobes	IVMP 1 g/day x 5 days. Recovered after 2 weeks; no long-term data.

Karoli et al., 2013 — India(20)	32/F; D14	Seizures, altered level of consciousness	CSF: 320 cells/μL (85% lymphocytes), protein 200 mg/dL. MRI: patchy areas of subcortical WM lesions	IVMP 1 g/day x 5 days → oral taper. Recovered after 2 weeks; no long-term data.
Pal et al., 2016 (Case 7) — India(21)	19/M; D8	Headache, fever, altered sensorium, meningism, quadriparesis	CSF: 16 cells/μL (all lymphocytes), protein 62 mg/dL. MRI: periventricular, brainstem lesions, cerebellar peduncle, parietal lobe, cord lesions	IVMP 1 g/day x 5 days. Full recovery
Viswanathan et al., 2016 (Case 1) — Malaysia(22)	61/F; D4	Coma (GCS 4/15)	CSF: unknown cell count (33% lymphocytes), protein 100 mg/dL. MRI: periventricular WM, brainstem lesions	IVMP 1 g/day x 5 days → oral taper. Partial recovery over 2 months
Viswanathan et al., 2016 (Case 2) — Malaysia(22)	30/M; ~D18–20	Ophthalmoplegia, quadriparesis, seizures	CSF: unknown cell count (lymphocytic pleocytosis), dengue NS1+/IgM+, normal protein. MRI: bilateral thalami, brainstem, corpus callosum	IVMP 1 g/day x 5 days → oral taper. Near-complete recovery
Diallo et al., 2020 — France(5)	39/F; D10	Urinary retention, altered level of consciousness, dysautonomia, loss of brainstem reflexes	CSF: 132→7500 cells/μL (83% → 8% lymphocytes), protein 49.4 mg/dL. MRI: brainstem/cerebellar peduncle, corpus callosum, cord lesions with enhancement	Ceftriaxone/amoxicillin/acyclovir → oral prednisone 2 mg/kg body weight/day. Died (brain death, day 3 of steroids)

Farooque et al., 2020 — Pakistan(23)	18/M; ~D4	Altered sensorium, headache, vomiting; normal neuro exam	CSF: 36 cells/μL (80% lymphocytes), protein 125 mg/dL. MRI: bilateral centrum semiovale, open-ring enhancement	IVMP 1 g/day x 5 days. Full recovery
Qayyum et al., 2022 (Case 1) — Pakistan(24)	22/M; D8	Altered level of consciousness, tachycardia, sluggish pupils	CSF: 133 cells, protein 64.5. MRI: bilateral centrum semiovale, brain stem, bilateral temporal lobes, bilateral periventricular white matter, T1 hypointensities	IVMP 1 g/day x 4 days. Died (raised intracranial pressure, day 4 of admission)
Qayyum et al., 2022 (Case 2) — Pakistan(24)	50/F; D10	Hemiplegia, altered level of consciousness, sluggish pupils, long-tract signs,	CSF: not done MRI: bilateral basal ganglia, insula, frontotemporal lobes, contrast enhancement	IVMP 1 g/day x 2 days. Died (raised intracranial pressure, day 2 of admission)
Qayyum et al., 2022 (Case 3) — Pakistan(24)	27/M; D14	Aphasia, altered sensorium, drowsiness, bladder incontinence, seizures; later coma	CSF: 146 cells (lymphocyte predominant), protein 59.9. MRI: frontal, parietal and occipital lobes; contrast enhancement	Pulse steroid + TPE x 5 cycles. Died (brain death, day 23 of admission)
Qayyum et al., 2022 (Case 4) — Pakistan(24)	52/M; D4	Altered sensorium, fever, seizures	CSF: <5 cells, protein 69.8. MRI: frontal, parietal and temporal lobes; subcortical and deep white matter.	Pulse steroid + TPE x 4 cycles. Died (brain death, day 7 of admission)

Qayyum et al., 2022 (Case 5) — Pakistan(24)	57/F; D20	Coma (GCS 4/15), mid-dilated non-reactive pupils	CSF: <5 cells, protein 69.8. MRI: bilateral frontoparietal lobes, cerebellum, pons, contrast enhancement	Pulse steroid x ~ 4 days. Died (cardiac arrest, day 4 of admission)
Chakraborty et al., 2024 — India(13)	38/M; D14	Paraparesis, bladder/bowel incontinence, sensory level, facial nerve palsy	CSF: 10 cells/μL (All lymphocytes), protein 56 mg/dL. MRI: cerebellar peduncles, corpus callosum, periventricular white atter, spinal myelitis	IVMP 1 g/day x 5 days → oral taper. Improving, ambulating with support
Lahari et al., 2025 — India(25)	18/(Sex not known); D5	Altered sensorium, fever, vomiting, diplopia	CSF [done on 14th day of illness]: 1 cell/μL (All lymphocytes), protein not available MRI: subcortical white matter, brain stem, cerebellum	Dexamethasone 40mg/day x 1 day → IVMP 1 g/day x 5 days → IVIG 2 g/kg body weight + oral taper of prednisolone. Full recovery
Torioni et al., 2025 — Brazil(26)	21/M; D6	Diplopia, acute urinary retention, hallucination, altered sensorium, nystagmus, hemiparesis, diminished reflexes	CSF: 135 cell/μL, protein 15.5 mg/dL. MRI: cerebral and spinal cord lesions	Pulse IVMP x 5 days → oral taper of prednisolone → Azathioprine. Full recovery after 3 months
Quelho Filho et al., 2026 — Brazil(27)	25/F; D15	Seizures, headache, vomiting, altered level of consciousness,	CSF data not available MRI: extensive lesions in all lobes, cerebellum, corpus callosum	Pulse IVMP + TPE + Decompressive craniectomy. Partial recovery
Our case, 2026 — Sri Lanka	39/M; D9	Cranial nerve palsies, nystagmus, mid-dilated non-reactive pupils,	CSF: 110 cells/μL (97% lymphocytes), protein 99.7 mg/dL.	IVMP 1 g/day x 5 days → IVIG 2 g/kg body weight + TPE x 6 cycles →

	diminished reflexes, quadriparesis, acute urinary retention	MRI: periventricular white matter lesions	repeat IVIG 2 g/kg body weight + oral taper of prednisolone. Partial recovery

CSF, cerebrospinal fluid; GCS, Glasgow Coma Scale; IVIG, intravenous immunoglobulin; IVMP, intravenous methylprednisolone; MRI, magnetic resonance imaging; TPE, therapeutic plasma exchange; WM, white matter.