ISSN: 2754-4516

Journal of Medicine and Healthcare

SCIENTIFIC Research and Community

Case Report

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Dengue-Associated Encephalitis Mimicking LGI1 Autoimmune Encephalitis: Faciobrachial Motor Seizures Deep Gray Matter Injury Status Epilepticus and Neurological Recovery-A Case Report

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ABSTRACT

Neurological involvement in dengue virus infection encompasses a broad spectrum of central and peripheral nervous system manifestations, including encephalopathy, encephalitis, seizures, movement disorders, myelitis, and peripheral neuropathies. Dengue-associated encephalitis may produce clinical and neuroimaging abnormalities that overlap with autoimmune encephalitis. In particular, recurrent brief motor events involving the face and upper limb may resemble faciobrachial dystonic seizures, a characteristic seizure phenotype of leucine-rich glioma-inactivated 1 (LGI1) antibody encephalitis. However, faciobrachial motor seizures are a clinical phenotype rather than an etiologically specific finding.

Case Presentation: A 45-year-old previously healthy woman presented with 6 days of high-grade fever, 3 days of headache, and 1 day of generalized seizures followed by marked deterioration in consciousness. On admission, she had severe encephalopathy requiring airway protection and mechanical ventilation, with a Glasgow Coma Scale score of E4VtM1, quadriparesis, and recurrent brief focal motor events involving the face and upper limbs with a faciobrachial dystonic-like appearance. Dengue IgM was positive, while dengue NS1 antigen and IgG were negative. Japanese encephalitis IgM and Brucella serology were negative. Brain magnetic resonance imaging demonstrated bilateral symmetrical abnormalities involving the basal ganglia and thalami with susceptibility blooming, without acute territorial infarction or abnormal contrast enhancement. Electroencephalography demonstrated severe diffuse cerebral dysfunction. The clinical phenotype and deep gray matter abnormalities initially raised concern for LGI1-associated autoimmune encephalitis. The subsequent course was complicated by status epilepticus, quadriparesis, bicytopenia, dyselectrolytemia, Escherichia coli catheter-associated urinary tract infection, hospital-acquired pneumonia, grade 2 pressure injury, prolonged ventilatory and airway requirements, and later tracheal narrowing. She received prolonged antiseizure therapy, antimicrobial treatment for nosocomial infections, nutritional support, airway management, physiotherapy, wound care, and multidisciplinary rehabilitation.

Outcome: Despite severe neurological illness and prolonged critical care, the patient demonstrated substantial neurological recovery. Consciousness improved from E4VtM1 to E4V4M6, seizures were controlled, the pressure injury resolved, tracheostomy was successfully discontinued following airway evaluation, and she achieved functional independence at discharge.

Conclusion: Dengue-associated encephalitis may closely mimic LGI1 autoimmune encephalitis when it presents with faciobrachial motor seizures and bilateral deep gray matter abnormalities. In dengue-endemic regions, an acute febrile prodrome should prompt consideration of infectious encephalitis even when seizure semiology strongly suggests an autoimmune syndrome. Careful integration of clinical presentation, dengue testing, neuroimaging, EEG, exclusion of alternative infections, and longitudinal clinical evolution is essential. Severe neurological dengue may nevertheless be followed by substantial neurological and functional recovery with aggressive seizure control and coordinated multidisciplinary care.

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Received: October 07, 2026; **Accepted:** October 13, 2026; **Published:** October 21, 2026

Keywords: Dengue Encephalitis, Faciobrachial Motor Seizures, LGI1, Autoimmune Encephalitis, Status Epilepticus, Basal Ganglia, Thalamus, Case Report

Introduction

Dengue is one of the most important mosquito-borne viral infections in tropical and subtropical regions. Although the majority of infections are uncomplicated, neurological manifestations are increasingly recognized and include encephalopathy, encephalitis, seizures, cerebrovascular complications, myelitis, Guillain–Barré syndrome, myositis, and movement disorders [1,2]. Neurological dengue may occur through several proposed mechanisms, including

direct viral neuroinvasion, blood–brain barrier disruption, systemic complications, and immune-mediated mechanisms [1,2].

Dengue-associated encephalitis is an important but relatively uncommon neurological manifestation. In patients with encephalitis in dengue-endemic settings, dengue should remain part of the diagnostic evaluation even when classical systemic features of dengue are absent [1-3]. Neuroimaging findings are variable; reported magnetic resonance imaging (MRI) abnormalities include involvement of the basal ganglia, thalami, brainstem, cerebellum, cerebral cortex, and white matter, with susceptibility-weighted imaging sometimes demonstrating microhemorrhagic changes [4,5].

The diagnostic challenge becomes greater when dengue-associated neurological disease produces an autoimmune encephalitis-like phenotype. LGI1 antibody encephalitis is classically associated with brief, frequent faciobrachial motor events, traditionally termed faciobrachial dystonic seizures (FBDS), which often precede or accompany limbic encephalitis [6,7]. Recognition of this phenotype is clinically important because LGI1 encephalitis is highly treatable and early immunotherapy may reduce seizure burden and subsequent cognitive morbidity [3-8].

However, the term *faciobrachial motor seizure* describes a seizure semiology rather than a disease-specific diagnosis. The characteristic facial and upper-limb motor phenotype may include tonic, clonic, dystonic, or myoclonic activity [7]. Consequently, clinical anchoring on LGI1 encephalitis can potentially obscure an infectious or structural cause when the broader clinical context points elsewhere.

We report a patient with dengue-associated encephalitis presenting with recurrent faciobrachial motor seizures, severe status epilepticus, quadriparesis, bilateral basal ganglia and thalamic abnormalities with susceptibility blooming, and prolonged critical illness followed by substantial neurological and functional recovery.

Case Presentation

Patient Information

A 45-year-old previously healthy woman presented with a 6-day history of high-grade fever, followed by 3 days of headache and 1 day of generalized seizures associated with rapidly progressive alteration of consciousness.

There was no previously documented history of epilepsy or other chronic neurological disease.

On admission, she required immediate airway protection and mechanical ventilation. Her initial Glasgow Coma Scale score was **E4VtM1**.

Neurological examination demonstrated:

- Severe impairment of consciousness
- Quadriparesis
- Recurrent brief focal motor events involving the face and upper limbs
- Faciobrachial dystonic-like motor phenomenology during these events

Systemic examination revealed pallor and pedal edema.

Diagnostic Assessment

Infectious Evaluation

Dengue serology demonstrated:

- **Dengue IgM:** Positive
- **Dengue NS1 Antigen:** Negative
- **Dengue IgG:** Negative

Japanese encephalitis IgM and *Brucella* serology were negative. The combination of an acute febrile illness, subsequent encephalitic syndrome, recurrent seizures, positive dengue IgM, and compatible MRI abnormalities supported **dengue-associated encephalitis** as the leading diagnosis.

CSF dengue PCR/IgM and neuronal autoantibody testing, including anti-LGI1 antibodies, were unavailable because of resource limitations.

The absence of confirmatory neuronal antibody testing meant that LGI1 encephalitis could not be definitively excluded serologically. Nevertheless, the acute febrile presentation, positive dengue IgM, deep gray matter MRI abnormalities, and subsequent clinical course favored an infectious dengue-associated neurological syndrome over a primary autoimmune encephalitis.

Neuroimaging

Brain MRI

MRI demonstrated **bilateral, symmetrical signal abnormalities involving the basal ganglia and thalami**, accompanied by susceptibility blooming.

The susceptibility changes were compatible with microhemorrhagic/petechial changes reported in neurological dengue [4,5].

There was:

- No acute territorial infarction
- No abnormal contrast enhancement

The distribution of the lesions was particularly important because basal ganglia and thalamic involvement has been repeatedly described among the neuroimaging patterns of dengue encephalitis [4,5].

Non-contrast CT Brain

NCCT brain demonstrated:

- Bilateral symmetrical hypodensities involving the deep gray nuclei
- Diffuse cerebral atrophic changes

Spine MRI

MRI of the spine demonstrated:

- L4–L5 disc herniation
- L5–S1 disc herniation
- Mild thecal sac compression

These findings did not provide an anatomical explanation for the acute encephalopathy or quadriparesis.

CT Chest, Abdomen and Neck

Chest and abdominal CT demonstrated chronic pneumonitic changes and hepatosplenomegaly.

CT of the neck subsequently demonstrated tracheal narrowing at approximately the **C7–D1 level**, prompting specialist ENT/airway evaluation following prolonged airway support.

Electroencephalography

EEG demonstrated **severe diffuse cerebral dysfunction with background slowing**, consistent with severe encephalopathy.

The patient's recurrent clinical seizures and subsequent status epilepticus required prolonged antiseizure treatment and intensive neurological monitoring.

Summary of Investigations

Investigation	Result	Clinical significance
Dengue IgM	Positive	Supports recent dengue infection
Dengue NS1	Negative	Does not exclude dengue, particularly outside the early antigen-positive phase
Dengue IgG	Negative	No serological evidence of established IgG response
Japanese encephalitis IgM	Negative	Reduced likelihood of Japanese encephalitis
Brucella serology	Negative	Reduced likelihood of neurobrucellosis
Brain MRI	Bilateral symmetrical basal ganglia and thalamic abnormalities with susceptibility blooming	Compatible with reported deep gray matter involvement in neurological dengue
NCCT brain	Bilateral deep gray matter hypodensities	Supports structural deep gray matter involvement
EEG	Severe diffuse cerebral dysfunction	Demonstrates severe global cerebral dysfunction
Spine MRI	L4–L5 and L5–S1 disc herniations with mild compression	Insufficient to explain encephalopathy or quadriparesis
Urine culture	*Escherichia coli*	Supported diagnosis of catheter-associated UTI
CT neck	C7–D1 tracheal narrowing	Relevant to prolonged airway management

Differential Diagnosis

Dengue-Associated Encephalitis

This was the leading diagnosis because of the acute febrile prodrome, positive dengue IgM, severe encephalitic syndrome, recurrent seizures, and bilateral deep gray matter abnormalities. Neurological dengue is known to produce seizures and abnormalities involving the basal ganglia and thalami [1,4,5].

LGI1 Antibody Encephalitis

LGI1 encephalitis was an important initial differential diagnosis because of the recurrent faciobrachial motor seizures and basal ganglia involvement. FBDS are highly characteristic of LGI1 encephalitis [6,7]. However, the patient's abrupt febrile onset, positive dengue IgM, severe acute encephalitic illness, deep gray matter injury involving both basal ganglia and thalami, and absence of available confirmatory LGI1 antibody testing made a definitive autoimmune diagnosis inappropriate.

Post-Infectious Inflammatory Encephalitis

A post-infectious immune-mediated mechanism was considered, particularly because dengue-associated neurological disease may involve both direct and immune-mediated mechanisms. [1,2]

Toxic-Metabolic Encephalopathy

Dyselectrolytemia and critical illness could have contributed to the severity of encephalopathy and neurological dysfunction. However, they did not adequately explain the combination of focal motor seizure phenomenology and structural bilateral deep gray matter abnormalities.

Therapeutic Interventions

The patient required prolonged multidisciplinary intensive care.

Antiseizure Therapy

Recurrent focal seizures progressed to status epilepticus and were managed with multiple antiseizure medications, including:

- Levetiracetam
- Sodium valproate
- Phenytoin
- Carbamazepine

The antiseizure regimen was subsequently transitioned according to clinical response and neurological recovery.

Treatment of Infectious Complications

During the prolonged hospitalization, the patient developed nosocomial infections, including E. coli catheter-associated urinary tract infection and hospital-acquired pneumonia.

Antimicrobial therapy included:

- Meropenem
- Linezolid
- Amikacin

Therapy was directed according to the clinical syndrome and microbiological findings.

Airway Management

Because of prolonged ventilatory and airway requirements, tracheostomy was performed.

Subsequent identification of tracheal narrowing at the C7–D1 level prompted ENT/airway evaluation. Following appropriate specialist

management and clinical stabilization, the patient was successfully decannulated.

Supportive and Rehabilitative Management

Management also included:

- Correction of electrolyte abnormalities
- Enteral nutritional support
- Pressure injury care
- Prevention and treatment of ICU-related complications
- Physiotherapy
- Progressive mobilization
- Neurological rehabilitation
- Multidisciplinary nursing and airway care

Timeline

Time	Clinical Event
Day −6	High-grade fever began
Day −3	Progressive headache developed
Day −1	Generalized seizures and deterioration of consciousness
Admission	GCS E4VtM1; quadriparesis; recurrent faciobrachial motor events; mechanical ventilation initiated
Early hospitalization	Dengue IgM positive; NS1 and IgG negative; Japanese encephalitis and *Brucella* serology negative
Early hospitalization	MRI demonstrated bilateral basal ganglia and thalamic abnormalities with susceptibility blooming
ICU course	Recurrent seizures progressed to status epilepticus; multiple antiseizure medications required
Subsequent course	Bicytopenia and dyselectrolytemia developed
Complications	*E. coli* CAUTI, HAP and grade 2 pressure injury
Prolonged recovery	Tracheostomy and prolonged airway support
Later course	C7–D1 tracheal narrowing identified; ENT/airway evaluation performed
Recovery phase	Progressive neurological improvement, seizure control and rehabilitation
Discharge	GCS E4V4M6; seizure-free; pressure injury resolved; tracheostomy discontinued; functional independence achieved

Clinical Course and Outcome

The patient's hospital course was prolonged and complicated by severe neurological dysfunction and multiple critical-care complications.

The most significant neurological complication was status epilepticus, occurring in the setting of the acute encephalitic illness and bilateral deep gray matter abnormalities.

Her clinical condition subsequently improved with aggressive seizure management and prolonged supportive and rehabilitative care.

Consciousness improved from GCS E4VtM1 at admission to E4V4M6 at discharge. Recurrent seizures were brought under control. Quadriparesis improved progressively with rehabilitation. The grade 2 pressure injury resolved completely.

Following evaluation and management of the airway abnormality, the patient was successfully decannulated. At discharge, she had achieved functional independence and was continued on oral antiseizure therapy with follow-up and rehabilitation recommendations.

The longitudinal course demonstrated substantial neurological recovery despite the severity of the initial encephalitic illness, prolonged status epilepticus, deep gray matter abnormalities, and extended ICU stay.

Discussion

This case illustrates a diagnostically challenging presentation of dengue-associated encephalitis in which the seizure semiology and neuroimaging initially strongly resembled LGI1 antibody encephalitis.

Neurological complications of dengue are diverse and include encephalitis, encephalopathy, seizures, movement disorders, myelitis, myositis, Guillain–Barré syndrome, and other peripheral neurological syndromes [1,2]. The distinction between dengue-associated encephalitis and encephalopathy can be clinically difficult because systemic complications of severe dengue may themselves produce altered consciousness. Nevertheless, the presence of recurrent focal seizures, focal neurological abnormalities, and structural CNS abnormalities should increase suspicion for a primary neurological process [1-3].

The MRI findings in this patient were particularly informative. Bilateral involvement of the basal ganglia and thalami has been repeatedly reported in neurological dengue. In a series examining MRI findings in neurological dengue, the basal ganglia–thalamus complex was the most commonly affected region, with additional involvement of the cerebellum, cortex, white matter, and brainstem. Focal hemorrhagic abnormalities have also been described [4]. A subsequent imaging series of dengue encephalitis similarly reported deep gray matter involvement and susceptibility blooming corresponding to microhemorrhagic changes [5].
These findings provided important contextual support for dengue-associated neurological disease but were not, in isolation, disease-specific.

The major diagnostic challenge was the seizure phenotype.
LGI1 antibody encephalitis is strongly associated with brief, frequent faciobrachial dystonic seizures. In the original description by Irani and colleagues, these events predominantly involved the arm and ipsilateral face and frequently preceded the development of limbic encephalitis [6]. Recognition of this seizure phenotype has substantial clinical relevance because LGI1 encephalitis is treatable with immunotherapy [6-8].

In the present patient, recurrent brief facial and upper-limb motor events closely resembled this phenotype. However, the term faciobrachial motor seizure is more appropriately viewed as a description of seizure semiology rather than an etiological diagnosis. Faciobrachial events associated with LGI1 antibodies may have tonic, clonic, dystonic, or myoclonic characteristics [7].

The clinical context therefore became decisive.
Unlike the typical subacute presentation of LGI1 encephalitis, this patient had a prominent acute febrile prodrome followed

by severe encephalopathy, generalized seizures, quadriparesis, and structural bilateral deep gray matter abnormalities. Dengue IgM was positive, while other tested regional infectious causes were negative. The combination supported dengue-associated encephalitis as the principal diagnosis.

Importantly, the absence of anti-LGI1 antibody testing should not be interpreted as proof that autoimmune encephalitis was excluded. Rather, it represents a limitation of the diagnostic evaluation. Similarly, because CSF dengue PCR and CSF dengue antibody testing were unavailable, direct CNS infection could not be virologically demonstrated. The most scientifically defensible interpretation is therefore dengue-associated encephalitis based on the clinical syndrome, serological evidence, neuroimaging findings, exclusion of selected alternative infections, and longitudinal evolution.

The patient's susceptibility-weighted MRI abnormalities are also noteworthy. Microhemorrhages and susceptibility blooming have been described in dengue encephalitis and may reflect hemorrhagic involvement of the CNS in the setting of dengue-associated vascular and inflammatory injury [4,5].

Another important feature was the subsequent development of extrapyramidal manifestations during recovery. Given the prominent bilateral basal ganglia involvement, transient movement abnormalities may plausibly reflect disruption of basal ganglia motor circuits. However, because the precise phenomenology and electrophysiological correlation were not systematically characterized, these manifestations should not be assigned a specific movement-disorder diagnosis retrospectively.

The patient's outcome is also clinically significant. Dengue-associated neurological disease may result in substantial morbidity, including persistent neurological deficits and death [1,3]. Nevertheless, recovery can be substantial, particularly when seizures and systemic complications are aggressively managed. Published cases of dengue encephalitis with extensive MRI abnormalities have also demonstrated neurological recovery, supporting the possibility of meaningful reversibility despite severe radiological disease [4,9].

This case therefore reinforces an important diagnostic principle: a striking seizure phenotype should not be allowed to override the temporal and systemic context of the illness. In a patient with fever followed rapidly by encephalopathy and seizures, infectious encephalitis remains a major consideration even when the seizure semiology resembles an autoimmune syndrome.

Strengths and Limitations

The principal strength of this case is the detailed longitudinal clinical course linking an acute febrile illness with a distinctive seizure phenotype, deep gray matter MRI abnormalities, severe status epilepticus, multiple ICU complications, and substantial neurological recovery.

The case also highlights an important diagnostic pitfall: the possibility that a faciobrachial motor seizure phenotype may mimic a highly characteristic autoimmune seizure syndrome in the setting of an infectious encephalitis.

Several limitations should be acknowledged. First, CSF dengue PCR and CSF dengue IgM testing were unavailable, preventing direct virological confirmation of CNS infection. Second, serum/CSF neuronal antibody testing, including anti-LGI1 antibodies, was unavailable, preventing definitive serological exclusion of autoimmune encephalitis. Third, the available EEG demonstrated severe diffuse cerebral dysfunction, but the clinical seizure semiology and status epilepticus were not necessarily captured as electrographic seizures throughout the entire course. Finally, because this is a single case, the findings cannot establish the frequency with which dengue produces faciobrachial motor seizure phenotypes [10].

Conclusion

Dengue-associated encephalitis can present with a striking autoimmune encephalitis-like phenotype, including recurrent faciobrachial motor seizures, severe status epilepticus, quadriparesis, and bilateral basal ganglia and thalamic abnormalities.

Faciobrachial motor seizures are highly characteristic of LGI1 antibody encephalitis but should be interpreted as a clinical seizure phenotype rather than an etiological diagnosis. In a patient with an acute febrile prodrome, infectious causes must remain prominent in the differential diagnosis.

In dengue-endemic regions, integration of the clinical timeline, dengue testing, neuroimaging, EEG, exclusion of alternative infections, and longitudinal response is essential. This case also demonstrates that severe neurological dengue with deep gray matter injury and prolonged status epilepticus may be followed by substantial neurological and functional recovery when aggressive seizure management, intensive supportive care, airway management, prevention of secondary complications, and multidisciplinary rehabilitation are provided.

Key Learning Points

- Dengue-associated encephalitis can present with severe focal and generalized seizures, including status epilepticus.
- Fever followed by acute encephalopathy should prompt consideration of infectious encephalitis even when the seizure semiology resembles an autoimmune syndrome.
- Faciobrachial motor seizures are highly characteristic of LGI1 antibody encephalitis but are not, by themselves, etiologically diagnostic.
- Bilateral basal ganglia and thalamic abnormalities, including susceptibility blooming, are recognized neuroimaging patterns in neurological dengue but are not disease-specific.
- When anti-LGI1 antibodies and CSF dengue testing are unavailable, diagnosis requires careful integration of clinical, serological, radiological, and longitudinal evidence rather than overinterpretation of any single test.
- Severe neurological dengue with status epilepticus and deep gray matter injury can nevertheless be followed by substantial neurological and functional recovery.

Patient Perspective

The patient expressed gratitude for the multidisciplinary care received during her prolonged hospitalization and for the recovery of consciousness, seizure control, airway independence, and functional autonomy.

Informed Consent

Written informed consent was obtained from the patient for publication of this case report and its associated clinical information and images, with appropriate de-identification.

Ethics Statement

This case report was prepared in accordance with the CARE (CAse REport) guidelines for case-reporting transparency and completeness. [10] Institutional requirements for publication of a de-identified case report were followed.

Conflict of Interest

The authors declare no conflicts of interest.

Funding

No external funding was received for this case report.

Author Contributions

Dr. Bimalesh Yadav: Conceptualization, clinical management, literature review, manuscript drafting and final review.
Dr. Saphiur Rahaman: Clinical management, multidisciplinary care, manuscript review and revision.
Dr. Rakesh Yadav: Radiological assessment and interpretation, imaging review and manuscript review.
Dr. Sushmita Yadav: Clinical care, data collection, manuscript review and revision.
All authors approved the final manuscript and agreed to be accountable for all aspects of the work.

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