

A Rare Case of Dengue Fever with Bell's Palsy: A Case Report

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Cite this paper as: Dr. Vishalakshi Jorepalli, Dr. Ramachandran, Dr. Mullangi Chenchu Vinatha, Dr. Kishore, Dr. Vignesh, (2025) A Rare Case of Dengue Fever with Bell's Palsy: A Case Report, *Journal of Neonatal Surgery*, 14 (8), 147-149

ABSTRACT

Dengue fever is a mosquito-borne viral illness that commonly presents with fever, myalgia, and thrombocytopenia. Neurological complications, though rare, can occur and may include encephalitis, Guillain-Barré syndrome, and myelitis. However, isolated lower motor neuron (LMN) facial nerve palsy (Bell's palsy) is an exceptionally rare manifestation of dengue fever. Here, we present the case of an 18-year-old male who developed Bell's palsy as a neurological complication of dengue fever. Early recognition, supportive management, and a short course of corticosteroids led to significant clinical improvement.

Keywords: Dengue fever, Bell's palsy, Facial nerve palsy, Lower motor neuron (LMN) facial palsy, Neurological complications of dengue, Thrombocytopenia

1. INTRODUCTION

Dengue fever, caused by the dengue virus (DENV), is a major public health concern in tropical and subtropical regions. It is primarily characterized by fever, headache, rash, and thrombocytopenia. Although dengue is considered a systemic illness, neurological manifestations are uncommon but can significantly impact patient outcomes. The virus has been associated with encephalitis, myositis, acute disseminated encephalomyelitis (ADEM), Guillain-Barré syndrome, and mononeuropathies. Bell's palsy, an idiopathic acute facial nerve paralysis, is commonly linked to viral infections such as herpes simplex virus (HSV) or varicella-zoster virus (VZV). However, its association with dengue fever is extremely rare. The mechanism by which dengue virus leads to facial nerve dysfunction remains unclear, but possible explanations include immune-mediated nerve damage, direct viral invasion, or microvascular ischemia. Given the increasing incidence of dengue worldwide, recognizing such atypical presentations is crucial for timely diagnosis and management.

2. CASE PRESENTATION

Case Report An 18-year-old male presented with complaints of high-grade fever for four days, which was intermittent and relieved with medication. He also reported generalized body pain but denied any associated symptoms such as cough, cold, breathlessness, nausea, vomiting, abdominal pain, or urinary symptoms. There was no history of rash, petechiae, purpura, or ecchymosis. His past medical history was unremarkable, with no known congenital heart disease, diabetes mellitus, or tuberculosis. He was well-nourished, a non-smoker, and had no history of alcohol consumption. Family history was non-contributory. On examination, the patient was conscious, alert, and hemodynamically stable with a blood pressure of 120/80 mmHg, pulse rate of 82 bpm, oxygen saturation of 98% on room air, and a temperature of 100°F. Systemic examination was unremarkable, with normal cardiovascular, respiratory, abdominal, and neurological findings. Initial investigations revealed

a total white blood cell count of 4300/ μ L, hemoglobin of 13 g/dL, and thrombocytopenia with a platelet count of 55,000/ μ L. Dengue NS1 antigen was positive, confirming the diagnosis of dengue fever. Liver and renal function tests were normal, and urinalysis showed no significant abnormalities. Ultrasound of the abdomen did not reveal any significant findings. The patient was admitted for supportive management, including intravenous fluids and antipyretics, and close monitoring of platelet counts. On the second day of hospitalization, he experienced an episode of fever and developed mild itching over the back of the right earlobe. His platelet count dropped further to 45,000/ μ L. By the third day, the patient became afebrile but complained of sudden-onset right-sided facial weakness. Examination revealed deviation of the mouth to the left, inability to close the right eye, and flattening of the right nasolabial fold, suggestive of right-sided LMN facial nerve palsy (Bell's palsy). There were no complaints of limb weakness, slurring of speech, sensory loss, abnormal eye watering or dryness, drooling of saliva, or difficulty swallowing. Neurological examination confirmed Bell's phenomenon on the right side, with preserved deep tendon reflexes and normal muscle tone and power in all four limbs. Pupils were equal and reactive to light, and there was no other cranial nerve involvement. Dengue IgM serology returned positive, while standardized serological assays for malaria, scrub typhus, leptospirosis, typhoid, brucellosis, hepatitis B and C, and HIV were negative. Autoimmune markers, including ANA, C-ANCA, and P-ANCA, were negative. A comprehensive viral panel, including HSV-1, HSV-2, CMV, adenovirus, and VZV, was also negative. MRI of the brain was performed to rule out structural lesions or stroke and was reported as normal. A diagnosis of dengue fever with LMN-type facial nerve palsy was established. The patient was started on a short course of oral prednisolone at 1 mg/kg, tapered over 14 days, along with supportive care for fever and hydration. Physiotherapy, including facial muscle exercises, was initiated. He was also prescribed lubricant eye drops and an eye patch to prevent corneal exposure-related complications. Over the next few days, his platelet count improved to 100,000/ μ L, and by the time of discharge, his facial weakness had shown significant improvement. At a one-month follow-up, his facial nerve function had almost completely recovered.

Test	Patient Value	Reference Range	Interpretation
Hemoglobin (Hb)	11.0 g/dL	13.5-17.5 g/dL (men)	Low (Anemia)
White Blood Cells (WBC)	4,300 / μ L	4,000-11,000 / μ L	Normal
Platelets	55,000 / μ L	150,000-450,000 / μ L	Low (Thrombocytopenia)
Urea	25 mg/dL	8-23 mg/dL	Slightly Elevated
Creatinine	0.7 mg/dL	0.6-1.2 mg/dL	Normal
Uric Acid	5.0 mg/dL	4.0-8.0 mg/dL	Normal
Sodium (Na ⁺)	135 mEq/L	136-145 mEq/L	Low-Normal
Potassium (K ⁺)	4.5 mEq/L	3.5-5.0 mEq/L	Normal
Chloride (Cl ⁻)	105 mEq/L	98-106 mEq/L	Normal
Total Bilirubin	1.0 mg/dL	0.3-1.2 mg/dL	Normal
Direct Bilirubin	0.4 mg/dL	0.1-0.3 mg/dL	Slightly Elevated
Indirect Bilirubin	0.6 mg/dL	0.2-0.8 mg/dL	Normal
SGOT (AST)	27 U/L	10-40 U/L	Normal
SGPT (ALT)	26 U/L	7-56 U/L	Normal
Alkaline Phosphatase	80 U/L	44-147 U/L	Normal
Dengue NS1 Antigen	Positive	Negative	Positive (Acute Dengue)
Dengue IgM Antibody	Positive	Negative	Positive (Recent Dengue)
Leptospirosis	Negative	Negative	Negative

Scrub Typhus	Negative	Negative	Negative
HSV	Negative	Negative	Negative
CMV	Negative	Negative	Negative
EBV	Negative	Negative	Negative
ANA	Negative	Negative	Negative
ANCA	Negative	Negative	Negative

3. DISCUSSION

Neurological complications of dengue fever are increasingly recognized but remain underreported. The spectrum of dengue-associated neurological manifestations includes encephalitis, acute flaccid paralysis, Guillain-Barré syndrome, myelitis, and mononeuropathies. Bell's palsy is rarely reported in dengue patients, making this case unique...

The proposed mechanisms underlying dengue-associated Bell's palsy include:

1. Immune-Mediated Nerve Damage – Viral antigens may trigger an aberrant immune response, leading to inflammation and demyelination of the facial nerve.
2. Direct Viral Neurotropism – Although uncommon, dengue virus may invade Schwann cells or neurons, causing localized nerve dysfunction.
3. Microvascular Ischemia – Dengue-induced endothelial dysfunction and thrombocytopenia may contribute to ischemic damage to the facial nerve.

While the exact pathophysiology remains unclear, it is essential to differentiate dengue-associated Bell's palsy from other causes of facial nerve paralysis, such as HSV/VZV infections, Lyme disease, Guillain-Barré syndrome, and stroke. In this case, normal neuroimaging and the exclusion of alternative causes strongly suggested a dengue-related etiology. Treatment for dengue-associated Bell's palsy is primarily supportive, with a short course of corticosteroids often used based on their benefit in idiopathic Bell's palsy. Physiotherapy plays a crucial role in preventing long-term complications such as muscle atrophy and synkinesis.

4. CONCLUSION

This case highlights a rare but important neurological complication of dengue fever—Bell's palsy. Dengue fever should be considered a potential cause of acute facial paralysis in endemic regions, especially when other common etiologies have been ruled out. Early recognition and prompt management, including hydration, corticosteroids, and physiotherapy, can lead to excellent recovery outcomes

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