

Rare Magnetic Resonance Imaging findings in dengue encephalitis

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Abstract

Dengue encephalitis is caused by a nonneurotropic virus of *Flaviviridae* group. It is a very rare manifestation of dengue fever caused by direct invasion of neural tissue into brain parenchyma. It causes a spectrum of neurologic manifestations such as meningitis, encephalitis, myelitis, and stroke. Here, we discuss a 20-year-old antenatal woman who presented with acute-onset fever and altered sensorium for 1 day. Blood investigations showed mild thrombocytopenia. Magnetic resonance imaging showed characteristic hemorrhagic encephalitis involving bilateral thalami and pons with diffusion restriction. Cerebrospinal fluid for the meningoencephalitic panel was negative. Serology for dengue NS1 antigen and immunoglobulin M antibody were positive. Although considered as a nonneurotropic virus, acute clinical presentation of fever, and altered sensorium apart from herpes and Japanese encephalitis, dengue encephalitis should also be regarded as one of the differentials.

Keywords: Dengue encephalitis, *Flaviviridae*, hemorrhagic encephalitis

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INTRODUCTION

Dengue fever is caused by dengue virus, a single-stranded RNA virus belonging to *Flaviviridae* family. It includes clinical spectrum ranging from mild asymptomatic dengue fever to dengue shock syndrome. Dengue virus usually is a nonneurotropic virus.^[1] Neurological manifestations occur either secondary to systemic complications (e.g., encephalopathy) or direct neurotropic effect by the virus causing meningitis, encephalitis, myelitis, and stroke.^[2] Encephalopathy without encephalitis is the most common manifestation. Encephalitis is a very rare manifestation of dengue fever caused by direct invasion of virus into brain parenchyma. We report a case of dengue encephalitis in a 20-year-old female with characteristic magnetic resonance imaging (MRI) findings.

CASE REPORT

A 20-year-old pregnant woman, with no known comorbidities, presented with short febrile illness for 1 day followed by altered sensorium and inability to speak. On arrival, her Glasgow Coma Scale was 10/15 (E4-M6-V [aphasic]) with normal body temperature of 37°C, blood pressure of 120/80 mmHg, and her pulse rate of 84/min. She was pale with no skin rash and icterus. Neurological examination showed motor aphasia with a power of 3/5 in all four limbs and bilateral plantar extensor. Meningeal signs were negative. Rest of systemic examination was normal. She was diagnosed with encephalitis and evaluated for the same. Blood investigations – complete blood counts showed anemia (hemoglobin: 9.6 g/dl) with mild thrombocytopenia (128,000/mm³) and normal

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erythrocyte sedimentation rate. Serum electrolytes (sodium: 138 mmol/L, potassium: 3.8 mmol/L, magnesium: 1.8 mmol/L, and calcium: 2.45 mmol/L). Liver and renal functions were normal. Workup for fever – smear for malarial parasite was negative. Urine routine was normal. Blood and urine cultures were sterile. MRI brain axial images T2-weighted (T2W) [Figure 1] and T2 fluid-attenuated inversion recovery [Figure 2] showed bilateral symmetrical hyperintensities with a diffusion restriction [Figure 3] in bilateral thalami, external capsule, and pons with foci of blooming noted in susceptibility-weighted imaging and mild enhancement with contrast – suggestive of hemorrhagic encephalitis. Dengue NS1 antigen and dengue immunoglobulin M (IgM) antibody were positive. Cerebrospinal fluid (CSF) analysis showed no cells, and polymerase chain reaction for herpes and Japanese encephalitis virus was negative. Blood for HIV, hepatitis B, and hepatitis C viruses was negative. Initially, empirical antibiotics (ceftriaxone) and antivirals (acyclovir) were started and then stopped after CSF results, and supportive care was continued. At the time of discharge, she was ambulant with minimal support and able to speak few words.

DISCUSSION

Dengue fever is an arboviral infection affecting predominantly Asian population. The primary mosquito vector, *Aedes aegypti*, plays a significant role in global epidemics. Infection ranges from asymptomatic stage to severe hemorrhagic fever with multisystem involvement. Typical dengue fever symptoms are fever, myalgia, rash, bleeding manifestations, and headache. Neurologic signs do occur but with an unknown mechanism.^[3] Encephalopathy is a most common neurological manifestation and occurs secondary to systemic complications such as increased ammonia levels. Dengue encephalitis is caused by the direct effect of the virus on the central nervous system (CNS) and clinically presents with fever, decreased sensorium, seizures, headache, and focal neurological deficits. Varatharaj proposed a clinical criterion for diagnosis [Table 1].^[4]

Laboratory diagnosis is mainly done by two methods: first, the viral antigen (dengue NS1 antigen) detection is done by immunochemistry with 89% sensitivity and 100% specificity and second, the host immune response detection is done by serological testing of IgG and IgM antibodies with 80% specificity and 90% sensitivity. Serological testing is advised after 5–7 days of infection. They are considered as definitive diagnostic modalities.^[4,5] Brain imaging – MRI – is a superior modality to computed tomography. Among various studies, the prevalence of

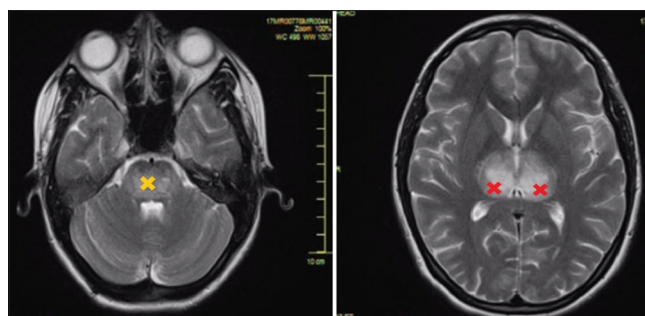


Figure 1: Axial T2-weighted magnetic resonance imaging showing bilateral thalamic (right image) and pontine hyperintensities (left image) with some blooming in susceptibility-weighted imaging suggestive of hemorrhagic encephalitis

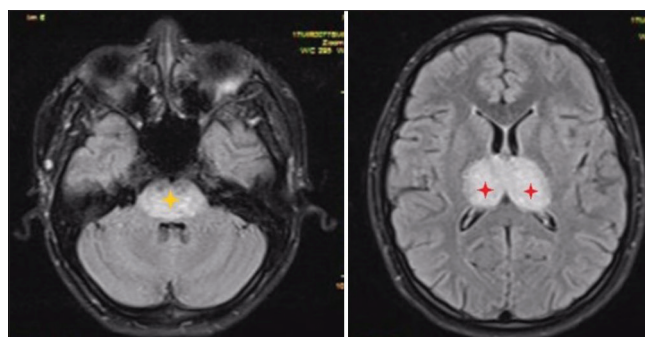


Figure 2: Axial T2 fluid-attenuated inversion recovery magnetic resonance imaging showing bilateral thalamic (right image) and pontine hyperintensities (left image) with some blooming in susceptibility-weighted imaging suggestive of hemorrhagic encephalitis

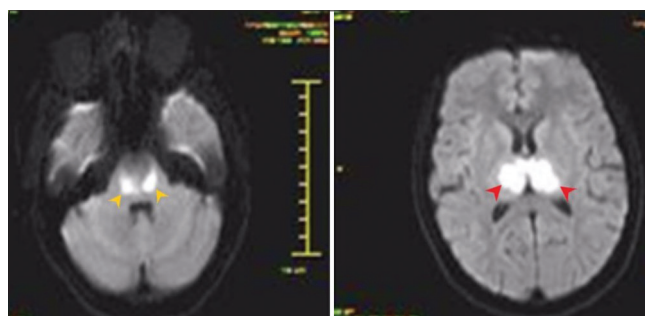


Figure 3: Axial diffusion-weighted imaging sequence showing hyperintensity (restriction) in the thalami (arrowheads on the right image) and the pontine region arrowheads on the left image)

encephalitis constituted about 4%–21% of all dengue cases.^[6,7] In our case, MRI showed classical hemorrhagic encephalitis involving bilateral thalamus and also bilateral pons.

MRI findings in dengue encephalitis commonly involve bilateral thalamic regions and rarely affect the cerebellum and brainstem as seen in our case.^[8,9] A case series have been reported by Soni *et al.* describing involvement of the thalami, pons, and upper half of the medulla in dengue hemorrhagic encephalitis.^[10] Few case reports showed involvement of the bilateral basal ganglia,

Table 1: Criteria for dengue encephalitis^[5]

Presence of fever
Acute signs of cerebral involvement such as altered consciousness or personality and/or seizures and/or focal neurological signs
Reactive IgM dengue antibody, NS1 antigen, or positive dengue PCR on serum and/or cerebrospinal fluid
(choice of test can be chosen depending on time of presentation)
Exclusion of other causes of viral encephalitis and encephalopathy
PCR - Polymerase chain; IgM - Immunoglobulin M

hippocampus, temporal lobe, midbrain, pons, and spinal cord in dengue infection.^[11] Involvement of the basal ganglia and thalamus was noted in children with dengue infection and has been reported by Liyanage *et al.*^[12] It is also used to exclude other differentials such as Japanese encephalitis (bilateral basal ganglia and thalamic regions), West Nile, and herpes encephalitis (bilateral medial temporal and basifrontal regions) which also mimic hemorrhagic encephalitis involving thalamus and brainstem.^[13] Another close differential to be considered in this case both clinically and radiologically is chikungunya fever with hemorrhagic encephalitis. However, MRI in chikungunya encephalitis shows T2W hyperintense white matter lesions with restricted diffusion. No basal ganglia or white matter involvement has been reported in this case.^[14] Other differentials to be considered are immune-mediated conditions such as adult demyelinating encephalomyelitis and Behcet's disease which are differentiated by their temporal course of illness and clinical manifestations. Other diagnostic modalities for dengue encephalitis include CSF positive for viral RNA than serum and positive viral RNA and antigens in CNS biopsies. Management is mainly supportive therapy. Symptomatic management includes antiepileptic medications for seizures, antipyretics, antiedema measures for raised intracranial pressure, and empirical antibiotics for the prevention of secondary bacterial infections.

Dengue virus, though being a nonneurotropic virus, is known to attack CNS directly. Dengue infection must be considered as one of the differentials among people who present with fever and encephalopathy with typical MRI features suggestive of hemorrhagic encephalitis, mostly in tropical endemic countries such as India. Mostly, it is a benign condition, and it may be considered fatal with the unfavorable outcome if there is extensive brain parenchymal involvement.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given her consent for her images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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