CASE REPORT

A rare case of dengue encephalitis

Sachin Rao, Manish Kumar, Soumik Ghosh, Adesh Kumar Gadpayle

Department of Medicine,
PGIMER Dr Ram Manohar
Lohia Hospital, New Delhi,
India

Correspondence to
Dr Soumik Ghosh,
soumik13joy@gmail.com

SUMMARY

Dengue fever has a variable clinical spectrum ranging from asymptomatic infection to life-threatening dengue haemorrhagic fever and dengue shock syndrome. However, neurological complications, in general, are unusual. Dengue encephalopathy is not an unknown entity; however, dengue encephalitis, a direct neuronal infiltration by the dengue virus, is an extremely rare disease. Although dengue is classically considered a non-neurotropic virus, there is increasing evidence for dengue viral neurotropism, suggesting that, in a proportion of cases, there may be an element of direct viral encephalitis. An MRI brain rarely shows focal abnormalities in dengue encephalitis. We report an interesting case of dengue encephalitis during an outbreak in Delhi, India. The diagnosis was confirmed by blood and cerebrospinal fluid dengue serology and (NS1) antigen assay. The case showed extensive lesions involving the midbrain, cerebellum, thalamus and medial temporal region on both sides of the MRI brain, which is an uncommon manifestation of dengue fever.

BACKGROUND

- Dengue encephalopathy is usually secondary to multisystem derangement like shock, hepatitis, coagulopathy and concurrent bacterial infection.
- Encephalitis as a cause of dengue encephalopathy is extremely rare. We report a case of dengue encephalitis.
- This case is presented to highlight the possibly extensive involvement of the brain by the dengue virus. Involvement of the thalamus, midbrain and cerebellum are usually not a feature of dengue encephalitis. This is a rare case with extensive MRI brain findings. Only two other case reports with this type of extensive brain lesions of dengue encephalitis are reported by Kamble *et al* and Borawake K *et al*.
- This documentation is presented because of a rare manifestation of a common disease. It also highlights an important, potentially fatal complication of this disease.

CASE PRESENTATION

A 23-year-old woman presented with fever for 3 days with altered sensorium for 1 day. There was no history of seizure and rash. On examination, her temperature was 100.4 °F, pulse 124/min; blood pressure 86/48 mm Hg, O₂ saturation 74%. Pallor was present, but oedema and icterus were absent. There was evidence of some vaginal bleeding. Glasgow Coma Scale was E1M1V1; the pupils were bilateral, equal and reacting to light. Bilateral plantars were mute. The cardiovascular and

respiratory systems were normal. She was intubated and put on mechanical ventilation.

INVESTIGATIONS

Investigations revealed haemoglobin 8.5 g/dl, thrombocytopenia (platelets 40 000/mm³), raised lactate dehydrogenase LDH (734 U/l), deranged kidney function tests (creatinine 1.9 mg/dl, urea 88 mg/dl), raised serum glutamic oxaloacetic transaminase (SGOT)/serum glutamic pyruvic transaminase (SGPT) (499/341 U/L). Malaria antigen-rapid test was negative. x-Ray chest was normal. Ultrasound abdomen showed bilateral minimal pleural effusion with mild ascites. (NS1) antigen was positive.

Cerebrospinal fluid (CSF) analysis revealed protein 158 mg/dl, sugar 70 mg/dl, 20 cells, mainly lymphocytes. Blood culture was sterile. Paired sera for dengue serology (MAC ELISA) were positive for IgM antibody. IgM antibody for dengue was also detected in CSF by immunoabsorbent assay. ELISA for tuberculosis was negative. PCR for both herpes simplex virus (HSV)-1 and HSV-2 DNA was negative. Blood serology for Japanese encephalitis virus was negative. EEG showed generalised low amplitude discharges and non-specific slowing suggestive of diffuse encephalopathy.

The MRI brain showed altered signal intensity in the midbrain, deep cerebellar white matter, bilateral thalamus and medial temporal region, with the periventricular and corona radiata on both sides showing scattered areas of restriction on diffusion-weighted imaging and patchy areas of enhancement in the bilateral thalamus, corona radiata and deep

Figure 1 T2-weighted image of the coronal section of the brain showing hyperintense signals in the bilateral thalamus, periventricular and medial temporal and deep cerebellar white matter, corona radiata.

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Figure 2 T2-weighted image of the sagittal section of the brain showing hyperintensities of white matter of the periventricular region, thalami and the midbrain.

Figure 4 T1-weighted image of the sagittal section showing white matter involvement of the subcallosal structures, mid brain and pons.

cerebellar white matter (figures 1–3). The area of altered signal intensity in the pons shows focal areas of blooming on gradient echo (GRE) (blood degradation products) with a restriction on diffusion and complete marginal enhancement on postcontrast images suggestive of acute necrotising meningoencephalitis (figures 4–6).

TREATMENT

The patient was managed conservatively with intravenous fluids, antipyretics and other supportive treatment.

OUTCOME AND FOLLOW-UP

Over 14 days, she was put on tracheostomy and weaned off, as spontaneous breathing resumed. The patient at present is alive, but with only slight improvement in her neurological status after 42 days of illness onset.

Figure 3 T2/fluid attenuated inversion recovery (FLAIR) image of the transverse section shows maintained grey-white differentiation with signal hyperintensities in the bilateral corona radiata and centrum semiovale.

DISCUSSION

Dengue encephalopathy is a rare entity, with the incidence ranging from 0.5% to 6.2%. Encephalopathy has been thought to result from the multisystem derangement that occurs in severe dengue infection, with liver failure, shock and coagulopathy causing a cerebral insult. Dengue has classically been thought to be not neurotropic.¹ However, there is increasing evidence regarding direct dengue viral neurotropism.

The MRI brain is usually normal in dengue. MRI in a cohort of patients with dengue-associated neurological involvement revealed cerebral oedema in most of the patients; encephalitis-like changes were less common and one patient had intracranial haemorrhage.² Globus pallidus involvement has also been reported.³

This patient's case of febrile encephalopathy had positive dengue serology and NS1 antigen assay, features suggestive of dengue encephalopathy. What characterises our case is the extensive MRI brain finding. Involvement of the thalamus,

Figure 5 T1-weighted image of the coronal section of the brain showing a white matter hyperintense lesion of the brainstem as focal areas of blooming.

Figure 6 T1 postcontrast image showing a necrotising lesion in the bilateral pons with a rim of peripheral enhancement with scattered areas of restriction on the diffusion-weighted imaging suggestive of encephalitis.

midbrain and cerebellum are usually not a feature of dengue. Only two other case reports with this type of extensive brain lesions of dengue encephalitis are reported by Kamble *et al*⁴ and Borawake K *et al*.⁵

Competing interests None.

Patient consent Obtained.

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Learning points

- ▶ Neurological manifestations in dengue infection are increasingly recognised.
- ▶ Dengue encephalopathy, though a rare diagnosis, should be considered a differential in cases of febrile encephalopathies, especially at the times of a dengue epidemic.
- ▶ Although acute dengue encephalopathy was previously considered to be non-encephalitic, the increasing evidence of viral neurotropism suggests that a proportion is partially or wholly encephalitic.
- ▶ Dengue encephalitis must be thought of in differentials of encephalopathy, in patients with dengue. Dengue encephalitis is thought to be benign, but it can be fatal at times.
- ▶ Extensive brain lesions as seen in MRI, although not a feature of dengue encephalitis, are not completely unknown.

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