CASE REPORT

Dengue fever with encephalitis: a rare phenomenon

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SUMMARY

The clinical profile and presentation of patients with dengue fever may differ from asymptomatic infection to the dreadful complications like dengue shock syndrome. However, neurological complications are very rare. Dengue encephalitis occurs by a direct involvement of central nervous system by the dengue virus which is an extremely rare complication. A 33-year-old man presented with fever, vomiting and severe headache. He had one episode of generalised tonic-clonic seizure followed by an altered sensorium on the day of admission to the hospital. The diagnosis of dengue fever was confirmed by dengue serology (IgM) and (NS1) antigen assay. MRI brain was suggestive of encephalitis. Thus, the patient was treated symptomatically and discharged in stable condition with minimal neurological deficit.

BACKGROUND

Dengue fever is one of the most common tropical diseases which is caused by single-stranded RNA viruses of four different serotypes: DEN-1, 2, 3 and 4; under the family of *Flaviviridae* which is often transmitted by *Aedes aegypti*.¹ Usually, it presents with acute febrile illness and has complete recovery without much complications.

Encephalopathy is often secondary to multisystem dysfunction which includes shock, acute respiratory distress syndrome, hepatitis and secondary bacterial infection.² Encephalitis as a result of dengue fever is quite rare and not many cases have been reported to date.

This case provides information on extensive involvement of central nervous system by the dengue virus which is an extremely rare phenomenon. Interestingly, literature suggests that simultaneous involvement of the thalamus, midbrain and cerebellum is extremely unusual in dengue encephalitis. Only two other such case reports with this type of extensive brain lesions of dengue encephalitis are reported (Kamble *et al*³ and Borawake K *et al*⁴).

This present case reports a rare manifestation of dengue, highlighting a significant, and potentially a very fatal, complication: encephalitis.

CASE PRESENTATION

A 33-year-old man presented with fever, vomiting and severe headache for past 5 days; generalised tonic-clonic seizure since past 2 days followed by altered sensorium for 6 hours. On examination, body temperature was 38.2°C, pulse 58/min, blood pressure 100/60 mm Hg and O₂ saturation

90%. Pallor was present, but oedema and icterus were absent. The relatives gave a history of haematuria from day 1 without any active bleeding sites. Glasgow Coma Scale score was E₂M₃V₂, and the pupils were bilaterally equal and reacting to light. Babinski reflex was present. The cardiovascular and respiratory systems were normal. The patient was intubated and put on mechanical ventilation and started on supportive treatment.

INVESTIGATIONS

Investigations revealed haemoglobin of 16.1 g/dL, thrombocytopenia (platelets 30 000/mm³), total leucocyte count of 4.59/mm³, serum creatinine of 1.2 mg/dL, raised serum glutamic oxaloacetic transaminase/serum glutamic pyruvic transaminase (821/432 U/L) ratio. Rapid malaria antigen test and *Leptospira* IgM were negative. Procalcitonin level was normal. Ultrasound abdomen showed bilateral minimal pleural effusion with mild ascites. Furthermore, dengue NS1 and IgM were positive. Serum electrolyte levels were normal. Electroencephalogram showed diffuse generalised slowing in delta range suggestive of diffuse encephalopathy. Cerebrospinal fluid (CSF) was positive for dengue IgM, mild lymphocytosis with normal sugars and CSF proteins (cells-16 (all lymphocytes), protein 40 mg/dL, sugar 60 mg/dL). CSF was negative for herpes, tuberculosis and *Cryptococcus*.

The MRI brain (figure 1) showed diffuse signal abnormality, and swelling and abnormal enhancement in the cerebral cortex, hippocampus, amygdala, thalami and cerebral vermis. Also, the foci of petechial haemorrhage were noted in the left lateral frontal cortical region, left amygdala, superior vermis and left superior cerebellar hemisphere. Extensive leptomeningeal enhancement was also observed. These features suggested the diagnosis of encephalitis.

TREATMENT

Thus, the patient was managed conservatively with intravenous fluids, antipyretics and other supportive treatment. Gradually, the patient's general condition improved, platelet counts started improving and liver enzymes concentration was reduced. The patient was extubated 4 days after the spontaneous mode trial, as consciousness and respiratory efforts improved significantly.

OUTCOME AND FOLLOW-UP

The patient had no major neurological deficit except for the mild gait ataxia 4 weeks after discharge. On

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Figure 1 MRI of the brain: (A) abnormal gyral hyperintensity in the left frontoparietal region; (B) similar pattern of abnormal hyperintensities in the hippocampal and amygdala regions on both sides; (C) thalami on both sides show hyperintensity on diffusion-weighted MRI (DWI); (D) DWI showing abnormal signal intensities in the vermis; (E) blooming is noted in the occipital region suggestive of petechial haemorrhage.

follow-up MRI (after 2 weeks), reduction in size of the lesions were noted (figure 2).

DISCUSSION

Dengue encephalitis is one of the rarest possible entities. Dengue fever has always been considered to be non-neurotropic.⁵ However, due to ever-increasing data, with regard to direct dengue viral neurotropism, especially if presented as encephalopathy with multiorgan involvement and hypoperfusion due to dengue shock syndrome, it needs to be seriously considered as a differential diagnosis in case neurological symptoms are persistent.

The most common clinical features of dengue encephalitis are headache associated with altered consciousness and seizures.⁶ The usual symptoms of dengue fever like rashes, muscular pain and bleeding are generally not seen in more than 50% of the total patients with encephalitis.⁷ So, it is wise to consider dengue as a diagnosis in all encephalitic cases, especially in areas that are endemic to dengue, irrespective of the presence or absence of the clinical features. Our patient however did have the classic symptoms of dengue.

This patient's case of febrile encephalopathy had positive serum NS1 antigen and IgM, and positive CSF dengue IgM suggestive of dengue encephalitis. What characterises our case is

Figure 2 Follow-up MRI (after 2 weeks): (A) Fluid-attenuated inversion recovery (FLAIR) MRI images showing a reduction in the size of the lesion in the right Sylvian region; (B) FLAIR MRI images showing a reduction in the size of the lesion in bilateral thalami; (C) follow-up diffusion-weighted MRI did not show the diffusion restriction as was seen before.

the extensive MRI brain finding (involvement of the thalamus, midbrain and cerebellum) which are usually not a feature of dengue fever or encephalitis. Only two other case reports with this type of extensive brain lesions of dengue encephalitis are reported (Kamble *et al*³ and Borawake *et al*⁴).

Learning points

Neurological manifestations in dengue infection are increasingly recognised and potentially fatal, if not treated promptly. If diagnosed and treated on time, it may have a good prognosis. Dengue encephalitis, though a rare diagnosis, should be considered a differential in cases of febrile encephalopathies, especially at the times of a dengue epidemic.

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