DENGUE ENCEPHALITIS IN A CHILD - A CASE REPORT

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ABSTRACT

BACKGROUND

Though dengue is raging in epidemic proportions in India, not many reports of dengue encephalitis have been reported from this region. We report a case of encephalitis in a child, which turned out to be dengue encephalitis. Unlike many cases of viral encephalitis, the child recovered fast without any neurological outcomes with good supportive care. With the increasing dengue outbreaks in India recently, it should be considered as an aetiological agent in encephalitis also.

KEYWORDS

Viral Encephalitis, Dengue Fever, Dengue Encephalitis.

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BACKGROUND

Dengue fever has variable clinical spectrum ranging from asymptomatic infection to life-threatening dengue haemorrhagic fever and dengue shock syndrome. Fever, arthralgia, headache, petechial spots, rash and haemorrhagic manifestations are common features. Neurological manifestations during dengue infection are not uncommon.¹ The re-emergence of dengue as an important pathogen justifies its inclusion in the differential diagnosis of patients with acute onset of encephalitis in endemic countries or with a travel history suggestive of dengue exposure.² In the recent years, it has grown into an epidemic in various regions of India with varying degrees of morbidity and mortality.

Case Report

Ten-year-old boy presented with one day history of fever and headache along with two episodes of generalised tonic-clonic seizures on the day of admission. First seizure lasted 4 - 5 minutes and second episode happened on the way to the hospital and child was convulsing at the time of admission to PICU. No preceding history of irritability, abnormal behaviour or altered sensorium. No history suggestive of cranial nerve palsies, limb weakness or unsteadiness of gait. No history of any drug intake or travel. No past history of seizures, liver or renal disease. There was no history of hospital admissions. He was the second born in the family with normal development milestones and fully immunised according to the National Immunisation Schedule.

His convulsions got controlled with IV Lorazepam and Phenytoin and he remained drowsy, Glasgow Coma Scale (GCS) 9. Vital signs were normal and had no pallor or icterus. Neurological evaluation showed hypertonia in all four limbs

with well elicited deep tendon reflexes. He had blurring of both disc margins, but no fundal haemorrhages were noted. He had neck stiffness, although other meningeal signs were absent. After four hours of admission he had an episode of left focal seizures, which lasted about 5 minutes. Child was started on Acyclovir, Ceftriaxone, Mannitol, intravenous fluids and continued on anticonvulsants with a provisional diagnosis of acute viral encephalitis. CSF study was deferred on the day in view of the low GCS and focal seizures.

CECT brain showed abnormal enhancement of bilateral frontal, temporal, parietal cortical sulci, Sylvian fissure and basal cisterns. No hyper enhancing areas were noted suggestive of meningitis. Blood examination showed TC - 8500/mm³ (Polymorphs - 85%, Lymphocytes - 15%), Haemoglobin 12.4 gm%, PCV - 34.1%, Platelet count 1.9 lakhs/mm³, blood sugar 112 mg%, SGPT 24 IU/L, S. Sodium 118 mEq/L, S. Potassium 4.2 mEq/L, S. Calcium 9.9 mg% and S. Magnesium 1.2 mg%. MRI brain revealed subtle gyral hyperintensity on diffusion images in the high frontal lobe, possibly seizure related gyral oedema or meningoencephalitis.

As the imaging excluded a focal lesion, CSF study was done on the next day which showed 2 cells, lymphocytes, protein-78 mg/dL, sugar-78 mg/dL, Gram stain - no bacteria seen. CSF virology panel showed anti-JEV IgM ELISA negative and real time PCR was negative for HSV-1 and HSV-2, mumps, varicella and pan Enterovirus. Serology was positive for Dengue IgM (ELISA) and Dengue NS1 antigen (ELISA). CSF Dengue IgM was not done.

The child improved over next 48 hours with GCS 15 without any focal neurological deficit. There was no seizure recurrence or any abnormal movements. Child was treated with Acyclovir, Ceftriaxone, Mannitol, anticonvulsants and intravenous fluids. His S. Sodium became 126 mEq/L after six hours of admission on Normal Saline infusion and remained normal thereafter. No hypoglycaemia was documented throughout the ICU stay. EEG done on fifth day was normal. Anticonvulsants were tapered and stopped in one week. Acyclovir and Ceftriaxone stopped once the virology reports were available and the child was discharged on the tenth day.

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DISCUSSION

In addition to the most recognised forms of the disease, which are Dengue Fever (DF), Dengue Haemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS), complications of dengue affecting specific organs and systems such as the brain, peripheral nerves, muscles, liver and pancreas, have recently been described.^{3,4}

Neurologic manifestations other than headache occur in approximately 4 - 6% of dengue cases. They have been attributed to different pathophysiological mechanisms, such as direct Central Nervous System (CNS) viral entry, metabolic disturbances affecting CNS function, haemorrhage and virus-generated autoimmune reactions leading to CNS inflammation and demyelination.^{5,6}

Patients with suspected dengue and neurological symptoms may present with headache (including dengue headache and dengue meningitis); altered sensorium with or without focal neurological signs (including encephalopathy, encephalitis, ADEM and stroke); and para- or tetra-paresis (including hypokalaemic paralysis, myopathy and GBS).⁷ The spectrum of neurological manifestations seen in dengue has been classified by Murthy,⁸

1. Those related to neurotropic effect: encephalitis, meningitis, myositis and myelitis.
2. Those due to the systemic complications: encephalopathy, stroke and hypokalaemic paralysis.
3. Post-infectious complications: encephalomyelitis, optic neuritis and Guillain Barré syndrome.

Dengue has classically been thought not to be neurotropic virus. The discovery of dengue virus and anti-dengue IgM in the Cerebrospinal Fluid (CSF) of patients with encephalopathy suggests that dengue is capable of Central Nervous System (CNS) infection.⁹ Various studies have described the onset of neurological symptoms ranging from 2 - 14 days from the onset of fever with median of 3 - 5 days. The common clinical features that characterise dengue encephalitis include fever, headache, reduced consciousness and seizures. Other features are meningism, extensor plantars, frontal release signs, abnormal posturing, facial nerve palsy and quadriparesis. Isolated cases with altered sensorium, bilateral hippocampal encephalitis presenting as amnesia has been reported. The pathogenesis of neurological complications and the contribution of viral and host factors are not well understood and can be related to neurotropic effect of the virus, systemic effects of the infection and can be immune mediated.⁸

Magnetic Resonance Imaging (MRI) is the modality of choice of brain imaging in dengue encephalitis. General findings consistent with viral encephalitis include cerebral oedema, white matter changes and (later) necrosis and brain atrophy. Infarction or haemorrhage may also be visible. Breakdown of the blood-brain barrier may be visualised as signal enhancement on MRI with gadolinium contrast. However, it is not uncommon for scans to appear normal early in the disease.¹⁰

In our case the diagnosis of dengue encephalitis was rather retrospective; as by the time the virology reports were available child had an uneventful recovery with fully retained neurological functions. This child had hyponatraemia, which is described in many reports of Dengue encephalitis.² Unlike in HSV encephalitis the neurological improvement was rapid, complete and was without any residual deficits. We could not do a CSF Dengue IgM, which is considered confirmatory of the disease.

In conclusion, neurological manifestations of Dengue infection are not uncommon. Dengue encephalitis should be suspected in children presenting with picture of encephalitis, especially in areas where the prevalence of dengue infection is high. Early intervention, monitoring and prompt supportive care can reduce the morbidity and mortality among these cases.

Contributions

SSV prepared the manuscript and was involved in case management and will be the guarantor of the paper, DSM critically reviewed the manuscript and managed the case. JMA, BKP and APR were involved in patient care and reviewed the manuscript.

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