

## Dengue encephalitis in children

Sir,

I read the editorial by Koshy and Pandian<sup>[1]</sup> as well as the article by Hira *et al.*<sup>[2]</sup> with great interest. Classically, dengue virus has not been described as neurotropic in nature, but its various neurological manifestations have been reported in adults, and such reports in pediatric population remain sparse. We hereby report two pediatric cases of dengue encephalitis.

Case 1: An 8-years-old boy presented with fever and body-ache for past 10 days; headache, vomiting and dizziness for 1 day. On examination, he was in shock; pallor and mild hepatosplenomegaly were present. Fluid resuscitation normalized BP within few hours. He received platelet transfusion on day 1 of admission due to hematemesis and thrombocytopenia ( $27 \times 10^9/L$ ). He had two episodes of generalized seizure at 8 hours and on day 3 of admission; and remained unconscious in between.

Case 2: A 3-years-old boy presented with fever for 5 days; vomiting and malena for 2 days; headache, decreased oral acceptance and cold extremities for 1 day. On examination, the child was in shock; pallor, pedal edema and mild hepatosplenomegaly were present. Fluid resuscitation started, and platelet concentrate given in view of thrombocytopenia ( $25 \times 10^9/L$ ). BP normalized within few hours. The child had one episode of generalized seizure 10 hours following admission.

In both of these cases, there were no signs of neck rigidity or any focal neurological deficit. Fundus examination, electrolytes, kidney and liver function tests were normal; and dengue serology was positive. Lumbar punctures were withheld due to thrombocytopenia. Neuroimaging was unremarkable.

The serotypes, most frequently implicated in causing neurological manifestations, are DEN2 and DEN3.<sup>[3]</sup> The spectrum of clinical manifestations has been classified by Murthy<sup>[4]</sup> into 3 categories, based on the pathogenesis. Firstly the ones, related to neurotropic effect of the virus *viz.* encephalitis, meningitis, myositis, rhabdomyolysis and myelitis. Secondly the ones, related to the systemic complications of infection like encephalopathy, stroke, hypokalemic paralysis and papilledema. Lastly, the post-infectious complications like ADEM, encephalomyelitis, myelitis, neuromyelitis optica, optic neuritis, Guillain Barré syndrome and various neuropathies. The most widely reported of these is encephalopathy (incidence, 0.5% to 6.2%).<sup>[5]</sup> The possible underlying pathophysiological mechanisms described include encephalitis, i.e., inflammation of brain as a result of direct viral invasion or due to non-encephalitic mechanisms like edema, cerebral hypoperfusion, hemorrhage, hyponatremia, hepatic failure, renal failure and cerebral hypoxia.<sup>[4,5]</sup> With isolation of dengue virus as well as anti-dengue IgM from CSF, the possibility of dengue virus being neurotropic cannot be ruled out.<sup>[5]</sup> This is further supported by the fact that dengue virus belongs to genus flavivirus, which includes neurotropic viruses such as, West Nile virus, Tick-borne encephalitis virus, Yellow fever virus, etc.

These cases were diagnosed as dengue encephalitis according to the case definition, suggested by Varathraj.<sup>[5]</sup> They had features suggestive of encephalitis *viz.* fever, headache, seizure and reduced consciousness, which were not explained by presence of any liver, kidney or electrolyte derangement; shock or any intracranial hemorrhage. This was further supported by positive anti-dengue IgM in the serum. These patients had complete neurological recovery as reported in most of the past studies.<sup>[5]</sup>

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