Bilateral thalamic involvement in dengue infection

Sir,

A patient of 11 years presented with fever, headache and vomiting for two days, an episode of tonic clonic seizures, ecchymosis and altered sensorium. Her pupils were 3mm, constricted and reacting and all ocular movements were restricted. She had spasticity with tonic posturing and extensor plantars. The CSF showed protein of 265mg%, glucose-109mg%, no cells and random blood sugar was 213mg%. Tests for malarial parasites, leptospira, Japanese encephalitis, Herpes simplex virus (CSF) and salmonella were negative. Dengue IgM antibody test was done twice one week apart and showed rising titers (92 units, 126 units) with normal bleeding and clotting time and low platelet counts (40,000 and 37,000). Ultrasound abdomen and chest X-ray were normal. However, EEG was not done. The CT scan of brain at admission [Figure 1] showed cerebral edema with symmetrical non-enhancing hypodensities in bilateral thalami, midbrain and posterior part of pons. The patient developed respiratory distress and was on mechanical ventilator for two days. Repeat CT four days later [Figure 2] showed persistent findings. Patient’s sensorium improved and third CT done two weeks after first scan [Figure 3] showed the thalamic lesions had resolved, whereas the lesions in the posterior pons and midbrain had become more hypodense. Marked generalized cerebral atrophy and ventricular dilatation were seen. At discharge she had mild cognitive impairment and tonic posturing.

Dengue viruses are single-stranded RNA viruses of the flaviviridae family causing dengue fever and dengue hemorrhagic fever. The pathophysiology of neurological symptoms in dengue fever is attributed to cerebral edema, hemorrhage, hemoconcentration due to increasing vascular permeability, coagulopathy and release of toxic substances. Neurotropic potential of dengue virus leading to encephalitis has been suggested.

In our case, the CT scan showed cerebral edema with non-enhancing hypodensities in the thalami, posterior pons and midbrain. Follow-up CT scan after two weeks showed that the lesions in the thalami had resolved, but lesions in the posterior pons and midbrain persisted. These imaging findings, classically described in Japanese encephalitis (JE)\[^3\] have not been reported in dengue infection. Japanese encephalitis was ruled out in our patient by immunological tests. Cerebral edema, encephalitis-like changes (edema and scattered focal lesions), intracranial hemorrhages on MRI\[^1,4\] have been reported. Yeo et al. have reported selective involvement of bilateral hippocampus in dengue infection in patient presenting with amnesia\[^5\].

While perivenous encephalitis described in dengue infection\[^4\] could be the cause for infarcts in the posterior pons and midbrain, vasogenic edema may have caused thalamic hypodensity that disappeared on subsequent scans. This finding has not been previously reported in dengue infection and may be missed during its temporal evolution.

In conclusion, dengue infection should be considered in the differential diagnosis of viral encephalopathies with bilateral transient thalamic involvement.

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