Jack-o'-lantern sign in dengue encephalitis

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An 18-year-old previously asymptomatic man presented with high-grade fever with holocranial headache and vomiting. On the second day of illness, he became drowsy and subsequently developed altered sensorium. At presentation, he was not opening eyes even on painful stimuli and had decorticate posturing. Pupils were small and sluggishly reacting. Neck rigidity was absent and plantar reflexes were bilaterally extensor. The patient had total leukocyte count 9,700/ μ L with 76% polymorphs and platelet count of 34,000/ μ L. Other biochemical measures were normal. Nonstructural 1 (NS1) antigen by ELISA for dengue virus was positive. T2 and fluid-attenuated inversion recovery images in MRI brain showed symmetrical confluent hyperintensities in thalami, brainstem, and perimesencephalic and perichiasmatic regions, with confluent white matter hyperintensities in bilateral supra and deep periventricular region (figure 1). The lesion in pons in diffusion-weighted imaging and corresponding apparent diffusion coefficient maps gave an appearance of a Jack-o'-lantern due to selective involvement of specific tracts in pons (figure 2 and figure e-1, links.lww.com/CPJ/A21). The patient died on the 7th day of illness.

Dengue is a tropical vector-borne viral infection spread in humans by *Aedes* mosquito bite. CNS complications associated with dengue infection are reported in 4%–21.2% of cases. Dengue encephalitis, a dreaded CNS complication of dengue viral infection, is reported in 33%–43% of cases with neurologic manifestation. Possible mechanism of neurologic involvement in dengue infection may be secondary to systemic complications such as electrolyte imbalance, microcapillary hemorrhage, release of toxic products, or post-infectious immune-mediated mechanism or direct neurotropic effect of virus on CNS. Management of dengue infection is generally careful monitoring and replacement of intravascular fluid and electrolyte losses. For dengue encephalitis, other specific measures depending upon severity of symptoms and signs like maintenance of the airway for adequate oxygenation, proper hydration, antiepileptic drugs if seizures occur, and measures to decrease raised intracranial pressure are required. The overall mortality rates in dengue infection range from 3% to 5% but cases of encephalitis with widespread involvement of brainstem are usually fatal. Cause of death in dengue encephalitis is mostly because of increased vascular permeability, thrombocytopenia, and hemorrhagic manifestations.

Various imaging findings specific to dengue encephalitis have been described.¹ Earlier studies reported involvement of bilateral thalami, cerebellum, and basal ganglia, but few recent studies illustrated involvement of brainstem, specifically pons.² Selective pontine tract involvement gives the appearance of a Jack-o'-lantern sign in MRI brain in this fatal case of dengue encephalitis. The possible cause of involvement of specific areas and tracts in different viral encephalitis cases is debatable. The propensity to involve particular brain structures giving characteristic imaging patterns in different viral encephalitis cases helps in narrowing the differential diagnosis.

Author contributions

A. S. Singh: Drafting/revising the manuscript, study concept or design, analysis or interpretation of data.

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Figure 1 Fluid-attenuated inversion recovery (FLAIR) images

FLAIR images in MRI brain show symmetrical confluent hyperintensities in thalami, brainstem, and perimesencephalic and perichiasmatic regions, with confluent white matter hyperintensities in bilateral supra and deep periventricular region.

Figure 2 Diffusion-weighted imaging (DWI) and corresponding apparent diffusion coefficient (ADC) maps

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