Dengue Encephalitis

Sir,

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. However, neurologic complications in general are unusual. In this documentation manifestations are common features. However, neurologic complications in general are unusual. In this documentation we present a case of dengue encephalitis, which is an uncommon manifestation of dengue fever. A previously healthy 30-year-old man presented with fever for six days with headache, vomiting and altered sensorium for one day. Fever was not associated with chills and rigor. Fever was of continuous type and it was associated with mild dry cough. There was no history of seizure, rash and bleeding episodes. He was a non-smoker and drank very little alcohol. The clinical examination on admission revealed pulse 66/min, BP - 130/90 mm Hg, temperature 101°F. Respiration was normal. No skin rash or petechiae were noted. There was mild pallor and icterus was absent. Lymph nodes were normal. Rest of the general physical examination was unremarkable.

Neurological examination revealed: Coma grade II with absence of neck rigidity. Kernig’s sign was negative. Plantars were extensor bilaterally. There was bilateral 6th cranial nerve palsy. Tone was increased in all the four limbs with brisk tendon reflexes in upper and lower limbs. Examination of spine was normal. Other systems were also normal. Tourniquet test (capillary fragility test) was negative.

Routine haematology and biochemistry results were normal apart from raised SGOT (180 IU/L) and SGPT (340 IU/L). Blood culture was sterile. BT and CT were normal. Mantoux test was normal. CSF examination revealed - pressure raised, protein 130 mg%, cytology 70 cells/cumm and all the cells were lymphocytes. Elisa for tuberculosis and Japanese encephalitis was negative. TORCH test was negative. Paired sera for dengue serology (MAC Elisa) were positive for IgM antibody. IgM antibody for dengue was also detected in CSF by immunabsorbent assay. CT scan head was normal. Viral isolation and typing was not done due to lack of facility.

A diagnosis of dengue encephalitis was made and patient was treated with intravenous fluids and intravenous mannitol. Paracetamol was used as antipyretic. Intravenous ampicillin given to prevent bacterial infection during hospital stay. Steroids and aspirin were not given. Patient had uneventful hospital stay and recovered completely without any neurological deficit. He was discharged after a week and subsequent follow up did not reveal any abnormality.

Neuroviral properties of dengue virus are not well known but there are some reports of nervous system involvement in both children and adults from various parts of the world. The various nervous system manifestations reported are alteration of consciousness, seizures, pyramidal tract signs, meningeal signs, headache, etc. Dengue encephalitis was reported by Hommel et al from French Guiana. The exact pathogenesis of nervous system involvement is not yet clear. Dengue virus type 2 has been demonstrated in CSF of dengue encephalitis patient. Dengue virus serotype 4 has been detected by immunohistochemistry and by RT-PCR in inferior olivary nucleus of medulla and granular layers of cerebellum. Immuno-reactivity has been observed in endothelial cells, astrocytes, neurons and microglia. Extended immunohistochemical studies have shown the virus positive cells located mostly with Virchow Robin space of medium size and small veins, infiltrating the white and gray matter are often close to neurons displaying cytopathic features. Since in dengue fever the virus mainly replicates the cells of macrophage lineage, it seems that infiltration of virus infected macrophages into the brain is one of the pathways of entry of virus into the brain in dengue encephalitis. However, it is not clear whether virus infected macrophages or virus free particles cause the lesions in nervous system by immune, metabolic and/or direct cytopathic effect.

Dengue is present since ancient times in India but encephalitis is not common. Dengue encephalitis was not observed in the 1996 epidemic, which ravaged several parts of India. After extensive review of literature we could not find a documented report of dengue encephalitis in an adult Indian patient. The documentation is presented not only because of a rare presentation of a common disease but also to emphasize upon the similarity of clinical features of dengue encephalitis with that cerebral malaria, meningitis, Japanese encephalitis, etc which should be ruled out before the diagnosis of dengue encephalitis is made. A high index of clinical suspicion and prompt investigations are important to arrive at the correct diagnosis.

The exact pathogenesis of dengue encephalitis is not yet clear. A lot of further studies are required to understand the pathophysiology of the disease.

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