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Correspondence

Acute disseminated encephalomyelitis and dengue fever: Comment

Sir,

We read with interest the case of dengue encephalopathy by Kanade and Shah[1] and have the following comments to offer:

The authors have correctly listed the three types of neurological manifestations associated with dengue infection[1]. We would like to add 'acute disseminated encephalomyelitis' (ADEM) to the list of post-infection neurological manifestation of dengue fever. ADEM is a monophasic, post-infectious or post-vaccine acute inflammatory demyelinating disorder of central nervous system[2]. The pathophysiology involves transient autoimmune response directed at myelin or other self-antigens, possibly by molecular mimicry or by non-specific activation of autoreactive T-cell clones[2]. ADEM is often preceded by a viral or bacterial infection; usually in the form of a non-specific upper respiratory tract infection[3]. Neurologic symptoms of ADEM commonly appear 4 to 13 days after the infection. ADEM can affect any part of the nervous system and thus the clinical presentation is variable. Patients can present with altered mental status, seizures, pyramidal dysfunction, cerebellar ataxia, brainstem syndromes, optic neuritis, myelitis, and rarely myeloradiculopathy and extrapyramidal syndromes[2]. The diagnosis of ADEM is based on clinical presentation and neuroimaging[2,3]. ADEM following dengue infection has been described in literature[4,5]. Although, ADEM following dengue infections is rare, it deserves mention as the management and prognosis depend on early recognition and treatment. It has been seen that the recovery is incomplete in patients with ADEM not receiving any form of immune modulation treatment (high dose methylprednisolone, intravenous immunoglobulin or plasmapheresis)[2,3]. The clinical presentation of the infant and cerebrospinal fluid analysis described by Kanade and Shah[1] is similar to that of ADEM. However, the normal magnetic resonance imaging findings and the complete recovery without immune modulation treatment goes against ADEM.

In conclusion, we would like to highlight that ADEM should be suspected in a child who develops multifocal neurologic abnormalities with encephalopathy, especially if onset occurs one to two weeks after dengue fever.

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Author's Reply

The authors have correctly mentioned that acute disseminated encephalomyelitis' (ADEM) should be added to the list of post-infection neurological manifestation of dengue fever. However, ADEM is a rare neurological manifestation of dengue[1]. ADEM has an abrupt onset and a monophasic course. Symptoms usually begin 1–3 weeks after infection or vaccination. Major symptoms include fever, headache, drowsiness, seizures and coma. Additional symptoms include hemiparesis, paraparesis, and cranial nerve palsies[2]. Though our patient had fever and altered sensorium at presentation, he also had neck stiffness suggestive of a meningeal irritation[3]. The cerebrospinal fluid (CSF) findings in ADEM are highly variable[4]. Most patients have cell counts below 800 leukocytes/mm^3, and many of these are below 200 leukocytes/mm^3. However, cell counts of 400 to 1500 leukocytes/mm^3 have been reported. The CSF leukocytosis is usually lymphocytic or monocytic predominant. protein is usually elevated but is <100 mg/dl[5–7]. Our patient had pleocytosis on CSF and high proteins which can be seen in ADEM and also in encephalitis and meningitis. Magnetic resonance imaging

(MRI) has become the single most important test in the early identification of ADEM[8]. Three distinct categories of disease can be classified using MRI criteria. The first is multifocal lesions in the white matter with or without basal ganglia involvement. The second is single or multifocal lesions only in the gray matter. The last category includes localized lesions in the brain stem, basal ganglia, or cerebellum[8]. In our patient, MRI was not showing any lesions and thus a diagnosis of ADEM is not considered in the child.

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