CASE REPORT

DENGUE ENCEPHALITIS

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Abstract. Encephalitis is an uncommon manifestation of dengue fever. Here we present a 4-year-old female child from northeastern India with dengue encephalitis. To our knowledge, this is the first reported case of dengue encephalitis from this region.

INTRODUCTION

Dengue fever has varying clinical presentations ranging from asymptomatic infection to life threatening hemorragic fever and dengue shock syndrome (Koley *et al*, 2003). Dengue is an increasingly prevalent arboviral infection common in tropical countries including South and Southeast Asia. Fever, arthralgia, headache, petechial spots, rash and hemorrhagic manifestations are common features. However, neurologic manifestations are unusual (Ramos *et al*, 1998). We present a case of dengue shock syndrome with encephalitis.

CASE REPORT

A previously healthy 4-year-old female child from Arunachal Pradesh, India presented to Down Town Hospital, Guwahati, Assam, India in November 2005 with a history of high grade fever for 8 days associated with vomiting, headache, one episode of generalized tonic clonic seizures and altered sensorium for one day. The fever was continuous and not associated with chills, rigor or jaundice. The clinical examination on admission revealed a pulse of 140/minute, a respiratory rate of 43/minute, a blood pressure of 90/60 mmHg, a temperature of 38.5ºC, with a few petechial spots on the trunk and thighs. There was mild pallor but skin rash, icterus, lymphadenopathy. Clubbing and edema were absent. A neurological examination revealed coma grade 1 with absence of neck rigidity or kernig's sign. The tone was normal in all four limbs with brisk tendon reflexes in both upper and lower limbs. The plantar reflexes were extensor bilaterally. There were decreased breath sounds over the right chest.

Investigations revealed a hemoglobin of 8 g/dl on admission which later increased due to hemoconcentration. The initial platelet count was 26,000/mm^3, WBC was 4,200/ mm^3 with a normal differential count and normal WBC morphology. The serum sodium level was 128 mEq/l, SGPT was 280 IU/l, SGOT 150 IU/l, serum bilirubin was 0.8 mg/dl, serum albumin was 2.5 g/dl, DIC profile was mildly deranged and the blood urea and serum creatinine were normal. The cerebrospinal fluid (CSF) revealed a normal opening pressure, protein was 70 mg/dl and all 30 cells/mm^3 were lymphocytes. Gram staining, Zichl-Neelsen staining and fungal staining were negative. CSF adenosine deaminase (ADA) was normal and ELISA for Japanese and herpes encephalitis were both negative. Paired sera for dengue were positive for IgM antibodies. The CSF was positive for dengue IgM antibody. Blood cultures, urine culture and CSF culture were negative. ELISA for leptospirosis was negative. A chest x-ray showed a right sided pleural effusion. Ultrasonogra-

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phy showed minimal fluid in the peritoneal cavity. A CT brain was normal. A Widal test (Span Diagnostic, India), rapid slide test for malaria (OptiMAL) (Diamed and Cressier, Switzerland) and exam for typhoid (typhidot) (Malaysian Biodiagnostics Research, Malaysia) were negative. Virus isolation and typing was not done due to lack of facilities.

During hospitalization the patient had bleeding from the mouth, rectum and vagina, then developed shock. A diagnosis of dengue shock syndrome with encephalitis was made and the patient was treated in the intensive care unit with intravenous fluid, fresh frozen plasma, packed cells, anticonvulsants and other supportive measures. Intravenous antibiotics were given initially but after confirmation of the diagnosis, the antibiotics were stopped. The patient was discharged after 2 weeks and on subsequent follow-up she was found to be normal.

DISCUSSION

Most dengue cases are reported in epidemics in India and other parts of the world, but sporadic cases have been reported. The neurovirulent properties of dengue virus are not well known but there are some reports of nervous system involvement in children and adults in various parts of the world (Mehendale *et al*, 1989; Koley *et al*, 2003; Chotmonqkol and Sawanyawisuth, 2004; Witayathawornwong, 2005). Encephalitis in dengue fever is a rare entity (Koley *et al*, 2003; Chotmongkol and Sawanyawisuth, 2004; Witayathawornwong, 2005). The various nervous system manifestations reported are altered level of consciousness, seizures, pyramidal tract signs, meningeal signs, headache, encephalitis, myelitis, and Gullain Barre syndrome (Thisyakorn *et al*, 1999; Soares *et al*, 2006).

The exact pathophysiology in the nervous system is not clear. Since in dengue fever the virus mainly replicates in cells of the macrophage line, infiltration of virus infected macrophages into the brain is one pathway of entry into the brain in dengue encephalitis (Koley *et al*, 2003). Dengue virus type 2 has been demonstrated in the CSF of a dengue encephalitis patient (Ramos *et al*, 1998).

This is the first reported case of dengue encephalitis from this region of India. The case is presented not only because of the rare presentation of a common disease but also to emphasize the similarities of the clinical features of dengue encephalitis with that of cerebral malaria, meningitis, and Japanese encephalitis, which should be ruled out before a diagnosis of dengue encephalitis is made. A high index of suspicion is important to arrive at the correct diagnosis.

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