Case Report

Primary dengue fever with acute transverse myelitis - A rare case

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Abstract

An 8 month girl presented as a case of acute febrile encephalopathy with hepatomegaly. Investigations showed thrombocytopenia, positive IgM for dengue and normal liver echotexture on ultrasonography. During hospital stay she developed paraplegia with no signs of abnormal hemostasis or plasma leakage. MRI showed signs of acute tranverse myelitis at T8-T12 spinal level. This is first infantile case of primary dengue fever with acute tranverse myelitis to be reported. Autoimmune demyelination and direct viral invasion are suggested pathophysiological mechanism.
Keywords: dengue fever; acute transverse myelitis.

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INTRODUCTION

Dengue fever and dengue hemorrhagic fever are one of the most important human viral infection prevalent in South East Asia, Africa and America. The tropical population is mainly at risk along with the travelers at these areas. The incidence of DHF is steeply rising in past two decades with simultaneously rise in morbidity and mortality.[2] About 3.6 billion people worldwide (i.e. 55% of world population) in 124 endemic countries are at risk of dengue infection, according to Pediatric Dengue Vaccine Initiative.[1] Although dengue has been considered as a non neurotropic virus[3], there are reports describing neurological complication involving both central and autonomic nervous system. The various neurological manifestation includes acute disseminated encephalomyelitis, encephalitis, Guillian Barre syndrome and Miller Fisher syndrome and neuropathies.[4-7] The spinal cord involvement (especially transverse myelitis) with dengue viral infection is rarely mentioned and only five cases have previously reported.[8-12] These cases were observed in the age group of 14-65 years.[8-12] and we hereby report a first infantile case of primary dengue fever with acute transverse myelitis.

CASE REPORT

8 month female born by non- consangious marriage was referred for intermittent fever since 8 days and generalized tonic clonic convulsions 2 episodes in three days. There was no history of vomiting, loose motion, cough and cold, trauma to head or spinal cord, irritability. No significant family and birth history. Anthropometric measurements were acceptable.

On examination; baby was febrile and hemodynamically stable. Facial flush and conjunctival congestion were present. The sign of raised intracranial tension or meningeal irritation were absent. Abdominal examination revealed hepatomegaly of 4 m which was soft, smooth and non tender. Other systemic examination was normal.

The hematological parameter showed thrombocytopenia (platelet count 6300/mm^3). The biochemical parameter were normal. Cerebrospinal fluid analysis was within normal limit. CSF culture showed no bacterial growth and Indian ink stain for cryptococcus was also negative. Dengue fever serology was ordered which showed IgM positive but negative IgG. Chest X ray was normal; USG abdomen showed hepatomegaly . A diagnosis of primary dengue fever with acute encephalopathy was kept and conservative treatment was started as per WHO guidelines.[4] However on day 3 of admission, we noticed decreased movements of both lower limbs; the power of the bilateral hip, knee, ankle joint flexors and extensors was grade 0/5 on Medical Research Scale. The bilateral plantar reflexes were

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upgoing. Abdominal reflexes were absent; but scapular reflexes were present. Beside this patient was hemodynamically stable and no metabolic disturbance were documented. Her MRI spine which showed edematous spinal cord at T8 –T12 level with abnormal signal intensity suggestive of infective etiology s/o acute transverse myelitis. Baby was started on pulse therapy of methyl predisolone for 5 days. She improved over 10 days and recovered completely over period of 2 months.

DISCUSSION

Dengue is an arboviral disease caused by four antigenically distinct viral serotype (DEN1, DEN2, DEN3 and DEN 4). The dengue virus belongs to flavivirus group in the Flaviviridae family and transmitted by bite of infected female Aedes mosquitoes. The dengue virus infection results in spectrum of clinical presentation from subclinical to fatal manifestations called dengue hemorrahgic fever and dengue shock syndrome. The primary dengue infection is more common in infant who possess maternal IgG dengue antibody.[3,4] The differential diagnosis includes bacteremia, leptospirosis, rickettsial fever, malaria and acute HIV infection syndrome. Diagnosis is confirmed by isolation of virus from blood during the viremic (febrile) phase and the presence of IgG and IgM antibody by ELISA during the post febrile peroid. The treatment aims at maintaining adequate hydration and managing potentially fatal complications.[2,12] Increasing spread of dengue infection has lead to rise in atypical manifesatation of dengue which were previously under reported due to lack of awareness. Dengue virus is an non neurotropic virus however there are recent reports of neurotropism or neuroinvasion.[3] The virus has been detected in CSF and demonstrated by immune histochemistry in CNS biopsies of fatal cases.[5] Acute transverse myelitis is an acute intramedullary spinal cord dysfunction involving both halves. Dengue myelitis showed differential gray matter involvement corresponding to anterior horn cell involvement similar to poliomyelitis which differ in sensory dysfunction.[12,13] The duration between the onset of infection and development of acute tranverse myelitis ranged from 2 to 16 days in all the previously reported cases. The development of neurologic symptoms in close association with the initial dengue infection (peri infectious) and flaccid paraplegia are attributed to direct viral invasion of the nervous tissue.[13] Whereas the late appearance of neurologic disorders (post infectious) and spastic paraplegia are considered immunologically mediated neural injury.[4,13] Reports of isolation of dengue virus, detection by PCR, dengue immunoglobulin M assays in CSF indicates the virus is able to directly invades the brain and cause encephalitis. Occurrence of delayed neurological manifestation suggest autoimmune demyelination may be further mechanism of neurological damage.[4,5] History of fever and convulsioin with transient decrease in platelet count and positive IgM and negative IgG dengue suggests that the patient is suffering from acute febrile encephalopathy with primary dengue infection. In this particular infant, the clinical picture was that of complete loss of spinal cord function at T8-T12 level along with platelet count of 63000/mm3 and no clinical sign of abnormal hemeostasis or the evidence of plasma leakage. There was no metabolic disturbance, while the spinal MRI at corresponding level showed edema with no bone abnormalities. All this indicates that bleeding or no other systemic factor was responsible for the development of transverse myelitis in this patient. Unfortunately tests to detect the virus in CSF was not available.

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