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Dengue encephalopathy

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Dengue fever is a viral infection transmitted in urban areas by *Aedes aegypti*. After an incubation period of 2 to 7 days, dengue fever begins with abrupt onset of fever, chills and headache. A transient macular rash may also be observed which usually resolves spontaneously. Complications of dengue fever are common and usually related to renal and hepatic dysfunction[1,2]. Encephalopathy and encephalitis are rare. We present a child with dengue encephalitis.

Case report: A 7 months old boy presented with fever for 14 days, irritability and excessive crying with poor feeding for seven days and altered sensorium for one day. There was no contact with a patient having tuberculosis or any other illness in the past. On examination, he was well-nourished, drowsy, had pallor and neck stiffness. Vital parameters were normal. Anterior fontanelle was closed. On central nervous system examination, he had hyperreflexia. On systemic examination, he had hepatosplenomegaly. Other systems were normal. Investigations showed hemoglobin of 8.1 g% hematocrit of 26, total leukocyte count of 22,600/mm^3 (58% polymorphs, 33% lymphocytes), and thrombocytopenia (platelet count of 77,000/mm^3). Erythrocyte sedimentation rate (ESR) was 10 mm at the end of one hour, serum glutamate oxaloacetate transaminase (SGOT) was 173 IU/L, serum glutamate pyruvate transaminase (SGPT) was 68 IU/L, serum albumin was 2.6 g/dl and both prothrombin time and partial thromboplastin time were deranged which corrected on Vitamin K administration. He had hyponatremia (serum sodium = 128 meq/L). Cerebro spinal fluid (CSF) examination showed proteins of 158 mg/dl, sugar of 65 mg/dl, 70 cells/hpf (20% polymorphs and 80% lymphocytes). CSF culture was negative. CSF Japanese B viral culture was negative. Other CSF viral polymerase chain reaction could not be done due to non-availability. CSF herpes ELISA could not be done due to non-affordability. MRI brain revealed mild communicating hydrocephalus. Mantoux test was negative. Peripheral smear for malarial parasite and malaria antigen tests were negative. Leptospirosis antibody was negative. Serum dengue IgM antibody was positive. Dengue serotype could not be done due to non-availability. After five days, hemoglobin dropped to 6.1 g%, repeat CSF examination showed 15 cells/hpf (100% lymphocytes), proteins of 134 mg/dl and sugar of 62 mg%. Patient was treated with Vitamin K, intravenous fluids and he had gradual improvement in his sensorium with normalization of liver enzymes. The child was discharged after two weeks of hospitalization.

Common clinical manifestations that we have found in children with dengue are thrombocytopenia, elevated liver transaminases and fever[3]. In last 20 years, there have been increasing neurological findings reported in association with dengue including mononeuropathies, polyneuropathies, and Guillain-Barré syndrome (GBS)[4–6]. Any virus serotype may be involved, but DEN-2 and DEN-3 are most frequently reported as the cause of severe neurological disease[7]. We could not do viral serotypes in our patient due to non-availability. The involvement of the central nervous system has always been thought to be secondary to vasculitis and leaky capillary syndrome with resultant fluid extravasations, cerebral oedema, hypoperfusion, hyponatremia, liver failure and/or renal failure. As such, it is usually called dengue encephalopathy. However, reports of virus isolation from brain tissue and CSF of patients with neurological symptoms suggest direct virus invasion of the CNS[7]. Our patient had hyponatremia and hepatic dysfunction. However, he had no signs of raised intracranial pressure, jaundice, serositis or hypoperfusion. Besides, the child took two weeks to recover and there were no changes of hyponatremia on MRI brain. There was also no intracranial bleed. Thus, the encephalopathy may have been related to direct viral invasion.

Three types of neurological manifestations have been associated with confirmed dengue infection, namely (a) classic signs with acute infection (headache, dizziness, delirium, sleeplessness, restlessness, mental irritability and depression); (b) encephalitis with acute infection (depressed sensorium, lethargy, confusion, somnolence, coma, seizure, stiff neck and paresis); and (c) post-infection disorder (epilepsy, tremors, amnesia, dementia, manic psychosis, Bell's palsy, Reye's syndrome, meningoencephalitis and GBS)[8]. Our patient presented with acute encephalitis.

The frequency of neurological changes as the presenting sign in dengue is unknown, but neurological compli-

cations associated with dengue infection have been recognized since the beginning of the 20th century and reported in almost every country in Asia and in many countries in the Americas[9]. In one study in Vietnam, 4% of patients admitted to a neurology ward with suspected CNS infections were infected with dengue virus[8] and in Thailand, 18% of children admitted to a hospital with encephalitis-like illness were confirmed as having dengue infection[10]. Mortality due to neurological involvement is low and patients may die due to other multi-system involvement[7,8]. Median coma recovery time for those admitted with a reduced level of consciousness as reported by Solomon *et al*[8] was 3.5 days. Our patient also responded without any active intervention and was completely alright in two weeks.

Neurological manifestations during dengue infection are not uncommon. The re-emergence of dengue as an important pathogen justifies its inclusion in the differential diagnosis of patients with acute onset of encephalitis in endemic countries or with a travel history suggestive of dengue exposure.

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