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Bilateral facial nerve palsy: a rare complication of dengue fever – a case report

Mandeep Singh¹, R K Cheema²

From, the Department of 1 Medicine and 2 Pediatrics, Liberty Hospital, SAS Nagar, Punjab, India

Correspondence to: Dr. Mandeep Singh, Liberty Hospital, Phase 3b2, SAS Nagar, Punjab, 1799-1800, India. E-mail: drmandeep1980@gmail.com.

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Case Report

ABSTRACT

Dengue is the most common mosquito-borne viral infection worldwide. There is increased evidence for dengue virus neurotropism, and neurological manifestations could make part of the clinical picture of dengue virus infection. Pathogenic mechanisms include systemic complications and metabolic disturbances resulting in encephalopathy, direct effect of the virus provoking encephalitis, and post infectious immune mechanisms causing immune-mediated syndromes. Dengue viruses should be considered as a cause of neurological disorders in endemic regions. Standardized case definitions for specific neurological complications are still needed. We report a patient with dengue fever who later developed bilateral facial nerve palsy.

Keywords: Dengue, Facial nerve palsy, Infection

Dengue is an acute viral infection with potential fatal complications. Dengue viruses (DV) belong to family Flaviviridae and there are four serotypes of the virus referred to as DV-1, DV-2, DV-3 and DV-4. It is transmitted mainly by Aedes aegypti mosquito and also by Ae. albopictus. All four serotypes can cause the full spectrum of disease from a subclinical infection to a mild self limiting disease, the dengue fever (DF) and a severe disease that may be fatal, the dengue haemorrhagic fever/dengue shock syndrome (DHF/DSS).

Recently, neurological manifestations have been increasingly described in dengue fever, making it challenging to correlate neurological symptoms to the infection [1]. The incidence of infection associated to neurological manifestations ranges from 1% to 5% [2-3]. The most common neurological presentations are encephalitis and encephalopathy, although every year cases of meningitis, Guillain-Barré syndrome (GBS), myelitis, acute disseminated encephalomyelitis, acute motor weakness, myositis, neuritis and neuropathy have been reported [4-6]. DENV-2 and DENV-3 serotypes are frequently associated with neurological most complications [7-9]. Immunoreactivity to DENV-4 has also been detected in neurons, microglia and endothelial cells [10]. The role of viral factors in dengue neuropathogenesis was demonstrated by mutating three amino acids in DENV-1, mapping to the structural protein E and non-structural protein NS3 helicase domains. These mutations produced a neurovirulent virus, and the result was an extensive encephalitis and leptomeningitis in mice [11].

Analysis of CSF contributes to the neurological diagnosis associated with dengue by the demonstration of: i) inflammatory reaction in the CNS (pleocytosis, hyperproteinorhachia, blood-CSF barrier dysfunction, and intrathecal synthesis of total IgG) in cases of encephalitis, myelitis, and meningitis; ii) protein-cytological dissociation in Guillain Barré syndrome; iii) hemorrhagic CSF in cases of cerebromeningeal hemorrhage; iv) exclusion of other infectious diseases; and v) detection of specific antibodies and viral antigens [12-14]. However, normal CSF does not exclude the possibility of neurological complications associated with dengue.

CASE REPORT

A 41 year old man presented with complaints of high grade fever, myalgia, arthralgia, retro orbital burning pain, abdominal pain, weakness and anorexia of 5 days duration. On evaluation, patient was haemodynamically stable with a blood pressure of 110/74 mm-Hg, pulse rate of 96/min. He had high grade fever with maximum temperature up to 104[°] F. His laboratory parameters showed leucopoenia (4300/mm³), thrombocytopenia (71000/mm³) and liver function tests showed elevated transaminases (AST/ALT 104/55 IU/L). Dengue serology showed positive NS1 antigen test while chikungunya serology was negative and rest of the investigations were within normal limits. Patient was diagnosed with dengue fever, managed conservatively and discharged in a stable condition after three days of in hospital care. Platelet count at discharge was 1.25lakh/mm³ and patient was afebrile.

One week after discharge patient started having difficulty in speaking, chewing and inability to close eyes along with recurrence of myalgia and arthralgia. On evaluation, patient was having normal higher mental functions; although, he had bilateral lower motor neurone facial palsy, his upper and lower limb muscles had a power of grade 4+, deep tendon reflexes were normal with no sensory deficits, babinski was bilateral flexor response with no cerebellar signs.

On investigations, serum potassium level was 4.1 mEq/L, CSF analysis revealed no cells with normal glucose level (69.8mg/dl, corresponding plasma glucose-84.9mg/dl), raised protein (113.94mg/dl) and normal gram stain, india ink and CSF culture. CSF was also evaluated using ELISA based kit for dengue and was found to be IgM positive. His MRI brain was normal and MRI cervical spine showed mild edema along superior end plate of C5 vertebra. Patient subsequently underwent nerve conduction which suggestive study was of symmetrical, motor, predominantly predominantly axonal polyneuropathy. Patient was subsequently started on intravenous methylprednisolone 1 gm OD over 5 days. Patient showed improvement in muscle power and was

subsequently discharged in stable condition. He was doing normal on follow up at 1 month of discharge.

DISCUSSION

Dengue viruses now affect almost every country between the tropics. Neurological manifestations received little attention initially, but in the last twenty years there has been increasing recognition of their possible importance. Neurological findings reported in association with dengue include mononeuropathies, polyneuropathies, and Guillain-Barré syndrome. The patient in our case had a motor axonal polyneuropathy. He had high grade fever, headache, arthralgia, retroorbital burning and myalgia during the acute presentation, which were consistent with an acute dengue, and this was supported by the positive serology (NS1 Ag). There have been many other case reports of dengue infection associated with CNS involvement. A case was reported from Jamaica of a 51 year old man with dengue fever who presented with encephalopathy, cranial nerve palsy, and severe hepatitis [15]. Diagnosis of neurological dengue needs to be revaluated; especially for clinicians, who are unfamiliar with its wide array of clinical presentations.

Diagnosis is currently based on the detection of specific IgM antibodies or antigens in the blood. In addition, analysis of CSF (for the detection of specific antibodies and viral antigens) has demonstrated great potential as a diagnostic tool for neurological dengue as well as for providing a better understanding of the dengue neuropathogenesis. In endemic areas or in cases of recent travel to endemic regions, dengue infection should be considered in the differential diagnosis of encephalitis, myelitis, Guillain-Barrè syndrome, and meningitis, even in the absence of a previous history of dengue fever. The neurological complications associated with dengue may be underestimated, especially in cases in which there was a prior asymptomatic dengue infection. Improving the accuracy of diagnosis is important for the early treatment of the neurological complications, thereby avoiding unnecessary therapy and longer hospitalization, while searching for the presence of other diseases.

CONCLUSION

Our case highlights the importance of high index of suspicion to diagnose neurological involvement of dengue infection. It should be considered in the differential diagnosis of encephalitis, myelitis, Guillain-Barrè syndrome, and meningitis, even in the absence of a previous history of dengue fever.

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