Isolated Lower motor neuron type facial nerve palsy: An unusual manifestation of Dengue haemorrhagic fever

Sunantha S¹, Peranantharajah T¹, Nisahan B¹
¹ Teaching hospital, Jaffna.

Introduction

Dengue is a febrile illness caused by infection with one of four dengue viruses (DENV). It is highly endemic in many tropical countries. Nervous system involvement is seen with serotypes 2 and 3 [2]. Neurological manifestations in dengue fever are rare and include encephalopathy, meningitis, acute pure motor weakness, mononeuropathies, transverse myelitis, stroke (both hemorrhagic and ischemic), acute disseminated encephalomyelitis, Guillain Barre Syndrome, hypokalemic paralysis and neuromyelitis optica [3]. Only a few cases of isolated facial nerve palsy in dengue haemorrhagic fever has been reported.

Case presentation

A previously healthy 28 year old army officer presented with fever of 2days duration associated with myalgia, arthralgia, dropping platelets with evidence of leak and managed as dengue haemorrhagic fever. On 6th day of admission while in the recovery phase of dengue haemorrhagic fever, he complained of inability to close his left eyelid, drooling of saliva and left sided facial weakness with drooping of the angle of the mouth was noticed. There was no history of any limb weakness or paraesthesia. He did not have any ear problems or parotid enlargement. He denied any history of trauma. On examination he was conscious, alert and haemodynamically stable. A detailed neurologic examination was unremarkable except left sided lower motor neuron type of facial nerve palsy. All other systemic examinations were normal.

His full blood count on admission showed a total white blood cell count of 5350/mm³ with normal differentials. The platelet count was 116000/mm³. The white blood cell count gradually dropped to 4490/mm³ and platelet count dropped to 10,000/mm³. Inflammatory markers such as ESR and CRP were normal. His aspartate transaminase level was 173 U/L and alanine transaminase level was 126 U/L. His renal function tests, electrolytes and random blood sugar level, chest x-ray and electrocardiogram were normal. Ultrasonography of abdomen revealed mild ascites with right sided mild pleural effusion. He was also found to have positive dengue IgM antibody on the 6th day of fever.

He was treated for dengue fever according to the Ministry of Health dengue guidelines and his platelets gradually improved in the next 7 days. His facial nerve palsy was managed with oral prednisolone at a dose of 1 mg/kg and tapered off over the subsequent 4 weeks. Facial nerve stimulation therapy was given. Facial nerve palsy improved after one month without any residual effect. The liver enzymes and platelet count also returned to normal. IgM antibody became negative after the 4th week of fever.

Discussion

Atypical manifestations associated with dengue virus infection are increasingly reported due to the higher incidence. They are acute kidney injury, myocardial impairment, fulminant myocarditis, arrhythmia, retinal vasculitis & haemophagocytic lymphohistiocytosis.

Though dengue had been regarded as a non-neuropotopic virus, there are recent reports on neuropotropism or neuroinvasion of the dengue virus, causing various central nervous system manifestations [4]. Permanent neurological sequale also has been described in dengue virus infection [4]. The pathogenesis of neurological manifestations are multiple includes both neuropotropic effects of the dengue virus and due to immune mediated effects [5]. The neurological spectrum of dengue patients has been limited because of a small number of case reports, lack imaging and neurophysiological studies in developing countries. We recommend dengue fever can be considered as a cause for lower motor neuron type facial nerve palsy in an endemic area.
References


