Parainfectious Ocular Flutter and Truncal Ataxia in Association with Dengue Fever

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INTRODUCTION

Ocular flutter is an eye movement disorder characterized by purely horizontal rapid saccadic oscillations lasting for few beats and stops spontaneously.[1] Opsoclonus and nystagmus are multidirectional and are influenced by fixation, thus can be differentiated from ocular flutter. Postinfectious ocular flutter and truncal ataxia are a rare entity which has been described after infections with enterovirus, mumps, cytomegalovirus, and human immunodeficiency virus. There are reported cases of opsoclonus myoclonus ataxia in association with dengue virus infection.[2] However, there are no reported cases of parainfectious ocular flutter and truncal ataxia in association with dengue virus infection. Hereby, we report a patient with dengue fever who had ocular flutter and truncal ataxia and responded to intravenous steroids.

CASE REPORT

A 14-year-old male child presented with fever associated with chills of 3 days duration. He also had dull aching continuous holocranial headache associated with 3–4 episodes of vomiting. On the 3rd day of fever, he noticed giddiness associated with difficulty in sitting and standing; needing support of family members; and swaying while walking. There was blurring of vision but no dysphagia, dysarthria, weakness or incoordination of limbs, sensory symptoms, or myoclonic jerks. On examination, his vitals were stable. He was conscious and oriented. The speech was normal. Pupils were equal and reactive. Oculomotor abnormality in the form of sudden conjugate horizontal saccadic oscillations without intersaccadic interval (ocular flutter) occurring spontaneously, with eyes open and closed, during pursuit and saccades, and mainly in horizontal than vertical direction of gaze was seen [Video 1]. Other cranial nerves were normal. Motor examination showed normal tone and muscle power with sluggish deep tendon reflexes. Sensory examination was normal. He had titubation with truncal, stance, and gait ataxia. Plantar response was mute. Other system examination showed mild hepatosplenomegaly. Complete hemogram showed low platelet count (0.76 lakh). Liver function test showed transaminitis (aspartate aminotransferase: 110 IU/L; alanine aminotransferase: 156 IU/L). Serum dengue nonstructural 1 antigen and immunoglobulin M antibodies were positive. Magnetic resonance imaging brain plain was normal. Cerebrospinal fluid (CSF) analysis was normal. He was treated with antipyretics and intravenous fluids. His fever subsided, but ocular flutter and ataxia persisted. He was given 5 days course of intravenous methylprednisolone after fever subsided. The patient had symptomatic
improvement in the form that his ataxia improved and ocular flutter decreased [Video 2]. He was able to walk without support and was discharged with tapering dose of oral steroids.

**DISCUSSION**

A syndrome of benign encephalitis with ocular oscillation and truncal ataxia was described in the 1960’s by Smith and Walsh and later Cogan.[3,4] Patients developed ocular flutter or opsoclonus and shivering movements of the head and body with preserved consciousness following a prodrome of malaise and mild fever. CSF protein and cell count were often elevated. Usually, within a few weeks or months, the symptoms resolved. Ocular flutter is the saccadic intrusions characterized by pathologic fast saccade, followed by corrective saccade without an intersaccadic interval. It is usually seen in lesions involving cerebellum and/or brainstem. The role of omnipause neurons in the brainstem is to inhibit the excitatory burst neurons which are required to initiate a saccade. Opsoclonus and ocular flutter are due to the loss of the inhibitory control of saccadic burst neurons by pontine omnipause cells.[1] Involvement of cerebellar vermis was responsible for tremulousness of head and body.

Wiest et al. reported three patients with similar syndrome as reported by Smith, Walsh, and Cogan secondary to enterovirus infection. Patients had ocular flutter and truncal ataxia with the symptoms following an upper respiratory infection and resolved without sequelae within a few weeks. One patient had a significant rise in antibody titers to enterovirus.[5] Touray et al. reported a patient in acute disseminated stage of Lyme disease who had ocular flutter.[6] Idris and Sokrab reported two patients with falciparum malaria infection who had severe ocular flutter and cerebellar ataxia. They had good response to prednisolone.[7]

The most common etiologies of ocular flutter are paraneoplastic syndrome, brainstem encephalitis, metabolic-toxic disturbance, and idiopathic. In children, 50% of cases prove to be a paraneoplastic manifestation of neuroblastoma. In adults, paraneoplastic causes include malignancies, such as small cell lung cancer, testicular germ cell tumor, and breast cancer.

**CONCLUSION**

The parainfectious occurrence of ocular flutter and cerebellar ataxia in association with dengue fever has not been reported previously. They show good response to steroids.

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**Conflicts of interest**

There are no conflicts of interest.

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