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Original Research Paper

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OCULAR FLUTTER IN DENGUE FEVER

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ABSTRACT Ocular flutter is an eye movement disorder characterized by purely horizontal rapid saccadic oscillations without intersaccadic interval lasting for few minutes which stops spontaneously. Truncal ataxia and post infectious ocular flutter in dengue fever are a rare entity. Multiple cases of opsoclonus, ataxia and myoclonus are reported in dengue fever. However, there are only very few reported cases of truncal ataxia and para-infectious ocular flutter in dengue fever. Thus, we report a 50 years old dengue virus infected female who had ocular flutter and truncal ataxia.

KEYWORDS : Ocular flutter, saccade, omnipause neuron, dengue fever.

INTRODUCTION

Ocular flutter is an eye movement disorder characterized by purely horizontal rapid saccadic oscillations without intersaccadic interval lasting for few minutes which stops spontaneously.[1] Opsoclonus is multidirectional and is influenced by fixation, which differentiates it from ocular flutter. Truncal ataxia and Postinfectious ocular flutter has been reported after infections with mumps, enterovirus, human immunodeficiency virus and cytomegalovirus. Multiple cases of opsoclonus, ataxia and myoclonus are reported in dengue fever.[2] However, very few cases of para-infectious ocular flutter and associated truncal ataxia have been reported in dengue fever. Thus, we report a 50 years old dengue virus infected female who had ocular flutter and truncal ataxia.

Case Report

A 50-year-old female presented with complaints of fever, giddiness, vomiting and generalised tiredness for duration of 4-5 days. On examination her vitals were stable. She was drowsy and responding to oral commands. Patient was diagnosed as Acute Febrile Illness and patient was started on IV fluids, IV antibiotics and antipyretics. On day 2 of admission, patient sensorium improved and patient complained of difficulty in going to restroom without support. So patient was examined for cerebellar signs. Finger nose test and knee heel shin test were normal. Patient had oculomotor abnormality in the form of sudden conjugate horizontal saccadic oscillations without intersaccadic interval (ocular flutter) occurring spontaneously, mainly in horizontal direction of gaze. She had truncal and gait ataxia. Other cranial nerves were normal. Motor and sensory examination were normal. Other system examination was normal. In view of cerebellar signs and increased BP (150/90 mmHg), patient was suspected of posterior circulation stroke and MRI brain was taken. MRI brain came out to be normal. Complete hemogram showed elevated WBC count and platelet count was normal. On day 3 of admission, there was a drop in platelet count from initial value. But both the values were within normal range. Dengue NS1 antigen was positive. LFT was normal. Patient was started on oral steroids. Patient had symptomatic improvement - her ataxia improved and ocular flutter decreased. She was able to walk without support and she was discharged with oral antibiotics and tapering dose of oral steroids. When patient came for follow-up after 2 weeks, patient recovered completely.

DISCUSSION

Ocular flutter is the saccadic intrusions characterized by pathological fast saccade, followed by corrective saccade without an intersaccadic interval in horizontal direction.[1] Opsoclonus is the rapid conjugate oscillations of the eyes in horizontal, rotatory and vertical directions often made worse by voluntary movement or the need to fixate the eyes.[4] Excitatory burst neuron which initiates a saccade, for horizontal gaze is parapontine reticular formation (PPRF) and for vertical gaze is rostral interstitial medial longitudinal fasciculus. Nuclear integrator for horizontal gaze are medial vestibular nucleus (MVN), Nucleus Propositus Hypoglossi (NPH) and for vertical gaze is interstitial nucleus of Kajal. The function of omnipause neuron located in caudal pons is to inhibit the excitatory burst neuron. Opsoclonus and ocular flutter are due to the loss of inhibitory control of excitatory burst neurons by pontine omnipause cells.[3] They are usually associated with parainfectious or paraneoplastic disease. Most common causes include breast, lung and testicular cancer in adults and neuroblastoma in children. Other less frequent causes include poststreptococcal infection, HIV, rickettsial infections and West Nile virus encephalitis.

CONCLUSION

Although ocular flutter and truncal ataxia are alarming findings in dengue fever, they show good response to steroids. But it is always mandatory to exclude structural brain disease before concluding that the cause of CNS manifestation is purely due to parainfectious etiology. 3.

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