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Case Report

Case Report of an Epileptic Nystagmus: Clinical Characteristics

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Abstract

Epileptic nystagmus is defined as a rapid repetitive jerky movement of one or both eyes associated with rhythmic eye oscillations that occur only during epileptic seizures. It is a rare epilepsy form, more frequently seen in the pediatric age group, and mostly associated with other oculomotor symptoms. We present an unusual case of epileptic nystagmus in a young male patient who showed a prominent response to low-dose antiepileptic drugs.

Keywords: Epilepsy, nystagmus, eye movements

INTRODUCTION

Nystagmus is rhythmic involuntary oscillations of the eye that could be binocular or monocular. Physiologic nystagmus is considered as a variant of normal eye movements that could be triggered by certain positions and movements such as holding far eccentric gaze or self-rotation. On the other hand, pathological nystagmus suggests an impairment or disruption among the nervous system or vestibular areas responsible for the oculomotor activity.[1] Pathological nystagmus may have been observed in seizures, and epileptiform discharges originating from the cortical areas involved in the generation of eye movements could lead to the formation of epileptic nystagmus (EN).

Epileptic nystagmus is defined as a rapid repetitive jerky movement of one or both eyes associated with rhythmic eye oscillations that occur only during epileptic seizures. Epileptic nystagmus is a rare phenomenon, often associated with other symptoms such as visual hallucinations, gaze deviation, cortical blindness, and more frequently encountered in the pediatric age group.[2] We present a young male patient with horizontal binocular IEN and a review of literature regarding the generation of EN.

CASE PRESENTATION

A 36-year-old right-handed male patient presented to our outpatient clinic with a complaint of dizziness that had begun 1.5 years ago.

He was referred by an otorhinolaryngology specialist who performed the first examination. Examination revealed horizontal nystagmus, and benign paroxysmal peripheral vertigo was suspected. The Dix-Hallpike maneuver elicited no response. Ophthalmological examination and optical coherence tomography were unremarkable. At the same time period, the patient had a neurological examination at a different outpatient clinic. Magnetic resonance imaging (MRI) scans of the brain were also found normal. Although the patient did not suffer from any headaches recently, he was diagnosed with basilar migraine at a different neurology outpatient clinic. Magnesium and vitamin B12 were started but showed no improvement.

At our clinic, detailed medical history was obtained. He experienced approximately 10 dizziness attacks in the last 10 months and stated that there were no triggers. The dizziness sensation had variable durations, lasting between 3 and 10 minutes. There was no loss of consciousness, accompanying motor symptoms. He did not experience any headaches before, during, or after these dizziness attacks. He would understand when the attacks would come and stated that an overwhelming sensation and nervousness always preceded these attacks suggesting a possible aura. He did not have any other symptoms during and after these attacks. He did not have any symptoms during sleep. The patient stated that he had experienced similar dizziness attacks 4 years ago that lasted for several days, but no further examination or treatment was initiated at that time period.

His medical history is remarkable for vascular headaches. He denied any sensory or motor disturbances before or during the headache; however, he occasionally experienced Deja vu feeling and visual disturbances in the form of flashing lights that occurred independently from the headache attacks. The last time he saw these flashing lights was 3 years ago. He had no other known illnesses, any central nervous system infections, head trauma, or a history of seizures. There was no history of epilepsy in his family. The patient showed a video clip of his attacks which revealed horizontal nystagmus that did not cross the midline with a fast phase toward the left (Video 1).

At our clinic, he had a thorough neurological examination. The examination was completely normal, and a new brain MRI, electroencephalography (EEG), and positron emission tomography (PET) were requested.

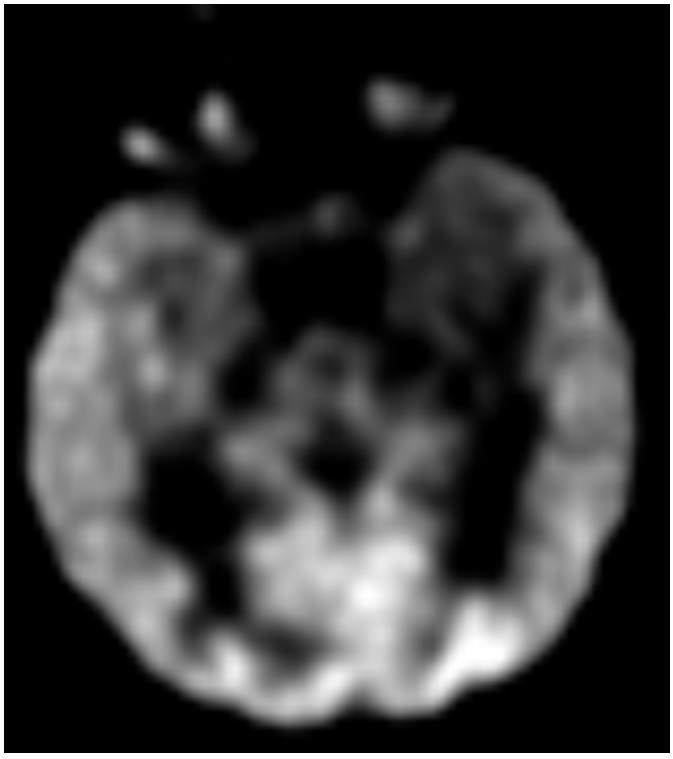

Figure 1. Bilateral temporo-occipital hypometabolism, prominently on the left side.

Brain MRI revealed suspicious gyral anatomy of the occipital cortex, thus occipital cortical dysplasia was suspected. However, this was not confirmed by the neuroradiologist. Electroencephalography and PET scan were performed. Inter-ictal EEG was found normal. Positron emission tomography confirmed bilateral temporo-occipital hypometabolism but more prominent on the left side. Hypometabolism was also evident on both median parietal lobes. Figure 1 shows a section of the above-mentioned PET scan.

Fifty milligrams of lamotrigine twice daily was started. The patient did not have any further attacks while on this treatment.

DISCUSSION

Generating eye movements and maintaining steady gaze involves various mechanisms operating together, and any disruption among these can result in nystagmus.[2] Frontal cortex has 3 regions involved in generating eye movements: the frontal eye field (FEF), the supplementary eye field (SEF), and the dorsolateral prefrontal cortex (DLPFC). Frontal eye field is responsible mainly for the intentional saccadic movements and aids pursuit. Supplementary eye field is connected with all regions involved in eye movement and also controls saccade sequences, whereas DLPFC is responsible for saccade inhibition.[3] Frontal eye field and SEF also participate in smooth pursuit eye movements (SPEM), characterized by slow, voluntary movements depending on visual stimulus. Frontal eye field works closely with temporo-parieto-occipital regions including Brodmann areas 19, 37, and 39 to generate SPEM.[4]

Epileptic nystagmus is mostly manifested as horizontal nystagmus in the posterior cortex. Weber et al[4] analyzed 40 EN cases, and all Fourty patients had horizontal nystagmus, with fast phase beating contralateral to the ictal site. Majority of these ictal discharges were associated with the posterior regions, especially the occipital cortex.[4] In addition, Lee et al.[5] studied 37 EN patients and 17 of them had ictal discharges originating from the occipital cortex and 5 patients from the temporo-occipital cortex.[5] Although EEG findings support the diagnosis of EN, it should be kept in mind that EEG might be completely normal in various epilepsy cases.

There are several proposed mechanisms regarding EN generation. Activation of the saccadic regions (FEF and SEF) results in nystagmus with a fast phase beating away from the ictal site, whereas slow phase caused by a defect in gaze holding mechanism results in centering eye back to its position without crossing the midline.[4] When epileptic discharges generate from the smooth pursuit areas, it results in slow ipsiversive eye movement crossing the midline, followed by the reflexive quick phase.[5] Midline crossing and the direction of the fast phase could be used for identifying potential locations of the nystagmus. In our case, the fast phase was toward the left and did not cross the midline, and hence, disruption in cortical saccades could be the underlying cause. However, the localizing value of the fast phase could not be assessed since bilateral temporo-occipital lobe hypometabolism was observed.

Although a rare phenomenon, EN should be kept in mind in the differential diagnosis of nystagmus. It could be the sole sign of epileptic seizures, thus requiring further investigation.

Informed Consent: Written informed consent was obtained from the patient.

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Video: You can access the video at https://doi.org/10.54614/ArchEpilepsy.2022.70883

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