

Visual Diagnosis in Emergency Medicine



ACUTE-ONSET VERTICAL NYSTAGMUS AND LIMB TREMORS IN CHRONIC RENAL FAILURE

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CASE REPORT

A 77-year-old woman presented to the emergency department for abrupt onset of intermittent involuntary eye movements and limb tremors. Her medical history was characterized by chronic respiratory and renal failure due to diabetic nephropathy, left ventricular hypertrophy with 62% ejection fraction, hypertension, hyperuricemia, and obesity. At age 40 years, she had an unspecified uterine tumor treated with surgery and chemotherapy, and episodes of paroxysmal atrial fibrillation. At our observation, she was on furosemide, ramipril, gliclazide, insulin, allopurinol, and digoxin.

The neurologic examination disclosed intermittent vertical downbeat nystagmus (Video 1) and dysrhythmic limb tremors. After each episode of nystagmus, she appeared mildly lethargic (Glasgow Coma Scale score of 13: eye response to verbal command, oriented verbal response, and pain localization). Computed tomography scan of the brain was normal. Electrocardiography revealed normal sinus rhythm. Electroencephalography (EEG) performed while the patient was experiencing symptoms showed no epileptiform abnormalities (Figure 1). Laboratory investigations revealed high levels of creatinine (3.8 mg/dL), hypocalcemia (1.45 mEq/L), and severe hypomagnesemia

(under the sensitivity limits, i.e., <0.10 mEq/L [normal values: 1.5–3.0 mEq/L]). The patient improved after magnesium levels normalized 2 days later.

DISCUSSION

Acute-onset involuntary eye movements and limb tremors in a lethargic patient should be promptly evaluated to exclude ongoing epileptic seizures. Epileptic nystagmus is a short-lasting (<1 min) focal seizure spreading from the occipital and temporoparietal regions. It may develop as an isolated symptom or accompany more-complex phenomena, such as dizziness, hallucinations, head turning, cortical blindness, motor automatism, also evolving to generalized tonic-clonic seizures. Epileptic nystagmus is typically short lasting (<1 min), occurs multiple times per day, and may be either horizontal or vertical. In children, it is most often observed in non-lesional epilepsies (e.g., idiopathic childhood occipital epilepsy), while in adults it is commonly associated with brain lesions (e.g., trauma, cerebrovascular disorders, and brain tumors) (1,2). To perform EEG during symptoms is of paramount importance, as the absence of epileptiform abnormalities allow ruling out epileptic seizures (3).

A vertical downbeat nystagmus may also result from brain lesions interfering with the central vestibular system, especially affecting the craniocervical junction or the cerebellar vermis, such as malformations (e.g., Arnold-Chiari

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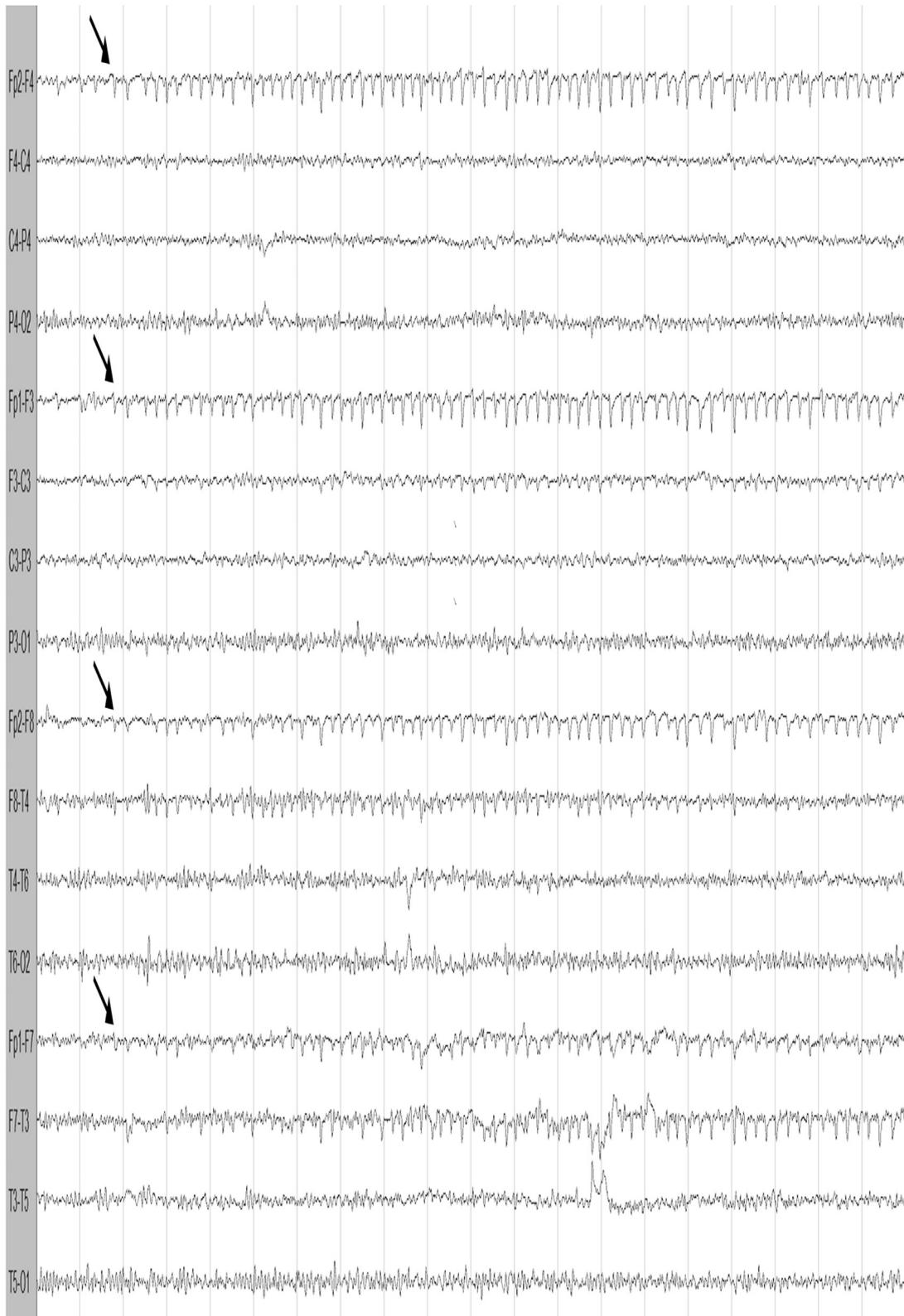


Figure 1. Electroencephalogram showing normal background activity with no epileptic discharges. Note the nystagmus artifact on the frontal electrodes (arrows).

type I, platybasia, syringomyelia), tumors (e.g., meningioma, cerebellar hemangioma), demyelinating diseases (e.g., multiple sclerosis), cerebrovascular disorders (e.g., stroke), and brainstem encephalitis. It can also result from genetic syndromes (e.g., spinocerebellar ataxias) and from paraneoplastic/autoimmune disorders. However, all of these etiologies yield a persistent nystagmus that does not fluctuate, as we observed in our patient. In most cases, a brain CT scan can disclose underlying structural abnormalities (4,5). Rarely, vertical downbeat nystagmus results from electrolyte imbalances, especially hypomagnesemia.

Hypomagnesemia can yield variable neurologic symptoms characterized by neuromuscular hyperexcitability, including tremors, fasciculation, tetany, and epileptic convulsions, as well as apathy, delirium, and even coma (6). Vertical downbeat nystagmus can be considered a useful diagnostic hint of hypomagnesemia in a patient with normal neuroimaging (7,8). Although the underlying pathophysiology is unknown, vertical downbeat nystagmus could originate from a transient cerebellar dysfunction induced by severe hypomagnesemia (9).

In summary, we suggest promptly investigating hypomagnesemia in patients with vertical nystagmus and limb tremors, especially after normal EEG and neuroimaging and in presence of a putative hypomagnesemia etiology, such as renal failure.

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SUPPLEMENTARY DATA

Supplementary data associated with this article can be found, in the online version, at <https://doi.org/10.1016/j.jemermed.2018.10.001>.

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