Movement Disorders
CLINICAL PRACTICE

CASE REPORT

Paroxysmal Tremor and Vertical Nystagmus Associated with Hypomagnesemia

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The clinical picture of hypomagnesemia can vary between asymptomatic presentations to life-threatening arrhythmia. Hypomagnesemia may have different causes, including low intake, pathological redistribution (ie, refeeding syndrome or treatment of diabetic ketoacidosis), and increased loss, for example, via the kidney (familial causes or acquired because of medication associated with hypercalcemia or as a result of alcohol abuse) or gastrointestinal tract (eg, because of diarrhea, vomiting, malabsorption, or proton pump inhibitors).[1] Classical features include cardiovascular complications, associated metabolic abnormalities (ie, hypokalemia and hypocalcemia), neuromuscular, and neurological manifestations.[2] Acute-onset movement disorders along with vertical downbeat nystagmus (DBN) is a further, under-recognized, presentation.

Case Report

A 68-year-old man was admitted to the emergency room because of repeated episodes of acute tremor, gait instability, and vertigo. He presented with a past medical history of bladder cancer diagnosed 15 years before, now in remission, and metabolic syndrome (type 2 diabetes, hypertriglyceridemia, and hypertension). His medications included metformin, fenofibrate, sitagliptin, valsartan, nicardipine, and rabeprazole. He was an active smoker and drank 2 to 3 units of alcohol per day. Between 2012 and 2018, he had experienced 4 similar episodes, each lasting 2 to 6 weeks. During those episodes, he also presented with transient cognitive disturbances and, on 2 occasions, with generalized epileptic seizures.

On neurological examination, spontaneous vertical downbeat nystagmus and action tremor of upper and lower limbs were observed as well as mandibular tremor (Video S1). There were no signs of cerebellar ataxia. He did not complain of diplopia or oscillopsia. The brain magnetic resonance image (MRI) revealed subcortical atrophy and nonspecific white matter lesions. An electroencephalogram (EEG) performed during the current episode showed normal background activity with slightly reduced reactivity to external stimuli and few diffuse theta waves. Laboratory investigations revealed mild hypokalemia (3.02 mmol/L) and hypomagnesemia (serum magnesium 0.09 mmol/L, normal range 0.66 – 1.07 mmol/L) with excessive renal magnesium loss (urinary magnesium 7 mmol/L). Calcium and parathyroid hormone were normal. There was no thiamine deficiency. A paraneoplastic panel and anti-GAD antibodies were negative. The diagnosis of renal hypomagnesemia associated with type 2 diabetes was established. Intravenous magnesium (initially 6 g per day), followed by oral supplementation resulted in a rapid recovery of the movement disorder (Video S2) beginning a few hours after treatment onset.

Discussion

Here, we report a case of hypomagnesemia presenting with relapsing episodes of acute tremor and vertical nystagmus. DBN is an unusual clinical sign that can result from lesions affecting the central vestibular system, in particular, located in the cranio-cervical junction or the cerebellar vermis (eg, Arnold-Chiari, syringobulbia), as well as demyelinating or cerebrovascular disorders, tumors, and brainstem encephalitis. Other etiologies of DBN include paraneoplastic and autoimmune diseases (eg, GAD-related autoimmunity),[3] genetic syndromes (eg, spinocerebellar ataxias),[4] toxic (eg, alcohol, lithium, and antiepileptic drugs), or metabolic causes and, more rarely, electrolyte imbalances such as hypomagnesemia.[5]

Clinical manifestations of magnesium abnormalities are heterogeneous. On the one hand, hypermagnesemia can be life-threatening, but it is almost exclusively observed in patients with substantially impaired kidney function and magnesium intake through supplements or other magnesium-based treatments (eg, antacids). On the other hand, hypomagnesemia may present with non-specific symptoms such as anorexia and nausea, cardiological abnormalities (including arrhythmia, ECG changes, and even sudden cardiac death), as well as neurological symptoms and signs,[6] including those resulting from neuromuscular hyperexcitability (Chvostek and Trousseau signs, tremors, fasciculation, and tetany), paresthesia, apathy, confusion, encephalopathy, epilepsy, opsoclonus, and ataxia.[7] Although, the underlying pathophysiology is unknown, vertical nystagmus, usually downbeat,

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has been described to be a typical manifestation of magnesium depletion.[5,8–10]

Our patient had experienced several previous episodes of tremor, vertical nystagmus, and different degrees of encephalopathy, but the diagnosis of hypomagnesemia was only established 6 years after his initial presentation, because serum magnesium is not part of the routine metabolic screening. Movement disorder specialists should be aware of this treatable condition, and assessment of the magnesium level in patients with acute tremor and vertical nystagmus should be considered.

Author Roles

(1) Research project: A. Conception, B. Organization, C. Execution; (2) Statistical Analysis: A. Design, B. Execution, C. Review and Critique; (3) Manuscript Preparation: A. Writing of the first draft, B. Review and Critique.

C.M.: 3A
V.D.: 3B
C.G.: 3B

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Ethical Compliance Statement: The authors confirm that informed consent from the patient has been obtained. We confirm that we have read the Journal's position on issues involved in ethical publication and affirm that this work is consistent with those guidelines.

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Supporting Information

Supporting information may be found in the online version of this article.

Video S1. Downbeat nystagmus and tremor. The video shows the tremor of the upper limbs and vertical downbeat nystagmus. Mandibular tremor and skew deviation are also shown.

Video S2. Recovery after treatment. There is still very mild intermittent intentional tremor in the left hand, but the vertical nystagmus has remitted.