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Letter to the Editor

Sufficiently Rule out all Differentials before Attributing Myopathy, Ataxia, Tremor, Nystagmus, and Myoclonus to Hypomagnesemia

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We read with interest the article by Ray *et al*. on a 62-year-old male with myopathy, ataxia, tremor, myoclonus, and downbeat nystagmus, being attributed to hypomagnesemia [1]. The cause of hypomagnesemia has not been elucidated, but chronic use of proton pump inhibitors(PPI) or diuretics or GAD65 antibody-associated autoimmune disease were suspected causative [1]. The patient benefited from oral calcium and intravenous magnesium [1]. The study is excellent but has limitations.

The major limitation is that the cause of hypomagnesemia was not elucidated. Only speculation was presented. In general, hypomagnesemia may be due to reduced intake (starvation, alcohol misuse, anorexia nervosa, total parenteral nutrition, malignancy), drugs (loop and thiazide diuretics, PPIs, aminoglycosides, amphotericin-B, pentamidine, digitalis, cisplatin, cyclosporine, cetuximab, matuzumab, panitumumab, laxatives, insulin), redistribution from extra- to intracellular compartment (refeeding syndrome, correction of metabolic acidosis, pancreatitis, alcohol withdrawal), gastrointestinal or renal loss due to tubular dysfunction (diarrhoea, hunger bone syndrome, gastric bypass, post-kidney transplant, post-obstructive diuresis, recovery from tubular necrosis), or genetic causes (Gitelman syndrome, Bartter syndrome, diabetes due to *HNF1*-variant, *EGF* variants, mutations in *ATP1A3, Kv1.1, CNNM2*). Only some of these causes have been discussed.

A second limitation is that the cause of the muscle weakness and the increase in creatine-kinase is not clear. Needle-electromyography and muscle biopsy results are missing. We should know whether motor unit action potentials were myogenic, neurogenic, or mixed. Because muscle weakness could also be due to plexopathy, radiculopathy, or transmission disease, it is important to know the results of nerve conduction studies (NCSs) and repetitive nerve stimulation. Were the acetyl-choline receptor antibodies elevated? Has pyridostigmine improved muscle function?

Since hypomagnesemia can be complicated by ECG abnormalities [2], it is important to know the results of the ECG recordings. Hypomagnesemia can lead to QT-prolongation and is thus a risk factor for malignant ventricular arrhythmias and sudden cardiac death [2].

Several differential diagnoses were considered but not sufficiently ruled out [1]. Although encephalitis was considered, the patient did not undergo cerebrospinal fluid (CSF) testing [1]. Wernicke encephalopathy was suspected, but no vitamin-B1 levels were reported. Channelopathy due to *CACNA1A* variants has been considered, but genetic testing has not been reported [1]. Reversible, cerebral vasoconstriction syndrome was suspected but no magnetic resonance angiography or digital subtraction angiography was performed. There is no EEG to clarify whether or not myoclonic jerks were due to epileptiform discharges. Hereditary myopathy has not been adequately ruled out.

We disagree that hypocalcemic myopathy is associated with myalgia in each case. There are several cases of hypocalemic myopathy without myalgia [3]. In addition, it is highly questionable whether creatine-kinase normalised from >5000U/l to normal within five days. How is that explained?

There is no explanation for the exaggerated tendon reflexes in the upper limbs and the simultaneous muscle wasting in the forearm.

After what time did muscle weakness disappear completely?

Which PPIs, insulin, diuretics and other medications were taken and in what dosage?

Without the presence of reference limits in Table 1, it is difficult to interpret the reported values.

Overall, addressing these issues would strengthen the conclusions and could improve the status of the study.

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