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LETTER TO THE EDITOR

Severe hypomagnesaemia as new potential stroke mimic: a case report

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Dear Editor,

Stroke is one of the most challenging conditions that neurologists must rapidly manage. In few minutes, physicians must analyse the clinical manifestations and distinguish between a stroke and a similar pathology, often called a stroke mimic. We present an interesting case of dysphasia and hemiparesis due to severe hypomagnesaemia, which may be proposed as a very rare cause of stroke mimic.

A 74-year-old female was admitted to the emergency department as a suspected stroke, presenting with a speech impairment. She was a heavy smoker (60 cigarettes/day for 30 years) and used to take aspirin, pantoprazole, and lorazepam.

She also manifested with generalized convulsions, treated by benzodiazepines with benefit.

Neurological examination revealed that the patient was stuporous with mixed dysphasia, left gaze deviation, right hemianopia, mild right facial paresis, and moderate right-sided limb weakness. The National Institute of Health Stroke Score (NIHSS) was 19. Brain CT and MRI excluded acute cerebral lesions. Few hours later, she displayed only expressive dysphasia. In the meantime, her relatives informed physicians that, two months earlier, the patient complained of an acute episode characterized by fine hand tremor and a fall to the ground, without loss of consciousness. The patient showed a normal neurological examination, but a slight impairment of cognitive functions.

A heart evaluation did not reveal any damage, as well as routine electrocardiogram (ECG) and transthoracic echocardiogram were unremarkable. An electroencephalography (EEG) showed normal electrical cerebral activity. Cerebrospinal fluid analysis was normal too.

Two weeks later, the patient was admitted again to the hospital because of mental confusion, speech impairment, and tonic–clonic seizures. An antiepileptic drug was started (Carbamazepine 400 mg/day). Few days later, she developed severe agitation, aggressiveness, and persecutory delusions, treated with antipsychotic therapy (Olanzapine 20 mg/day). Routine laboratory tests revealed slight hypocalcaemia (8.1 mg/dl [normal range: 8.2–10.4]) and moderate hypokalaemia (2.8 mmol/L [3.5–5.2]), which persisted despite substitutive therapy. After 10 days, all electrolytes were measured again and severe hypomagnesaemia was found (0.2 mg/dl [1.5–3.8]). She has been infused by a high intravenous magnesium charge and the proton pump inhibitor was stopped (PPI). After a week, neurological symptoms greatly improved, and she was discharged.

Six months later, her neurological and cognitive functions were totally normal, and therefore, magnesium, antiepileptic and antipsychotic drugs were suspended, allowing the patient to return to her normal daily activities.

Discussion

This patient was initially evaluated because of a possible acute stroke. In patients presenting with stroke-like symptoms, physicians must consider several other non-vascular pathologies, called stroke-mimics, that represent a real

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challenge in an emergency setting. Seizures, hypo-/hyperglycaemia, migraine, and tumours are the most common diseases within this group. Metabolic disorders, such as hypernatriaemia, hyponatriaemia, or hepatic encephalopathy, may also cause focal neurologic symptoms. To our knowledge, so far, only two clinical cases have been reported of hypomagnesaemia stroke-mimicking [1, 2].

In fact, low levels of magnesium are more often related to movement disorders. Patients with a chronic magnesium deficiency could manifest with weakness, tetany, and tremor, often potentiated by concomitant hypocalcaemia. Patients could present with positive Chvostek and Trousseau signs, muscle spasms, and cramps; all these symptoms are likely due to a lower threshold of nerve depolarisation. Choreiform and athetoid movements have also been described. Vertical nystagmus is a rare but helpful sign suggesting severe hypomagnesaemia, especially when there are no concomitant cerebellar lesions or vestibular alteration, and thiamine deficiency has been ruled out [3].

Convulsive seizures are included in the neurologic manifestations of hypomagnesaemia, usually with generalized and tonic–clonic features. It is not clear what is the exact pathophysiologic mechanism underlying the hypomagnesaemic seizures. It has been suggested that low levels of magnesium may reduce the inhibitory influence from the NMDA receptor, promoting neuronal depolarisation [3].

Moreover, hypomagnesaemia may manifest with subclinical cognitive impairment, with apathy, agitation, delirium, or dementia. Onset and severity of symptoms are heterogeneous and may depend on the causes of hypomagnesaemia. For example, in alcoholism-related hypomagnesaemia, cognitive symptoms prevail compared to motor deficits, although in non-alcoholic patients, mental alterations may be absent but also may be severe [3].

Low levels of magnesium could frequently cause severe cardiologic alterations. ECG modifications include widening of the QRS complex, prolonged QT, elongated PR interval, ST depression, and peaked T waves. Atrial fibrillation, supraventricular tachycardia and ventricular fibrillation represent the principal manifestations [3]. The herein described patient did not show heart problems.

Potassium deficiency is another consequence of hypomagnesaemia, which leads to urinary potassium wasting through renal ROMK (renal outer medullary potassium) channels. Decreased levels of potassium may delay the onset of tetany but promote the cardiac manifestations of hypomagnesaemia. A normal potassium concentration cannot be restored until the magnesium depletion is corrected [3].

Regarding the underlying cause for the hypomagnesaemia, this patient had no previous history of diarrhoea or malabsorption problems, no diuretics assumed, or any other drug that could explain a urinary loss of magnesium. On the other hand, the patient had been taking pantoprazole for at least 10 years. The chronic use of a proton pump inhibitor (PPI) was hypothesised as a possible cause of hypomagnesaemia. The association PPI-hypomagnesaemia was first described in 2006 and afterwards several papers have highlighted this relationship. The postulated mechanism seems to be due to impaired absorption of magnesium caused by PPI-induced inhibition of TRPM6 and TRPM7 channels on the intestinal epithelial membrane [4].

In addition, as abovementioned, smoking could have a role too. Our patient was a very heavy smoker. Actually, many studies found the plasma level of magnesium decreased significantly in chronic smokers (more than 10 cigarettes/day), compared with non-smoking healthy subjects [5].

Conclusion

Hypomagnesaemia may be more frequent than expected. Even if it is quite rare, low levels of magnesium could mimic an acute stroke. Stroke-mimics represent about 30% of all acute stroke presentations [1], so it is important to rule them out as fast as possible. In conclusion, we suggest considering hypomagnesaemia a possible cause of a stroke mimic and including magnesium dosage in emergency blood tests, mainly in the presence of heavy smokers or patients with hypokalaemia or with a longstanding PPI(s) use.

Author contribution AG, FG, and RFM conceptualized the study. AG and FG wrote the original draft. RFM and AT reviewed and critiqued the manuscript.

Declarations

Ethics approval The paper does not report on primary research. All data analysed were collected as part of routine diagnosis and treatment.

Informed consent Unfortunately, to account for the restrictive measures in place due to COVID-19, a handwritten signature is not possible. However, we obtained an audio recording of oral consent from the patient. We thank the patient and her family for their kind cooperation.

Conflict of interest The authors declare no competing interests.

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