Case Report

Recurrent Generalized Seizures with Postictal Todd's Paralysis Caused by Medication-Associated Severe Hypomagnesemia: A Case Report

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Keywords

Unprovoked first seizure · Hypomagnesemia-induced seizure · Todd's paralysis · Drugs associated with severe hypomagnesemia

Abstract

Hypomagnesemia is one of the electrolyte disturbances that can cause seizures. It is common in the hospitalized patients and can be induced by long-term usage of many medications. A 68-year-old male known to have hypertension and gastroesophageal reflux presented to the Emergency Department with an unprovoked first seizure at home followed by a temporary right-sided hemiparesis, dysphasia, and facial asymmetry. The hemiparesis, dysphasia, and facial asymmetry resolved within less than an hour after the seizure. His serum potassium was low with prolonged QT interval in the electrocardiogram (serum magnesium was not checked in the Emergency Department). He received intravenous IV potassium chloride infusion, and his serum potassium level was corrected, but he had a recurrent seizure after 10 h. At that time, his serum magnesium was found to be very low, he received IV magnesium sulfate infusion, and his indapamide, omeprazole, and metformin medications were stopped. He had no further seizures, the rest of his blood tests were normal, and his CT brain was unremarkable. He was treated for aspiration pneumonia, and his outpatient MRI brain and EEG came to be normal too.

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Published by S. Karger AG, Basel

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Introduction

Electrolyte disturbances are commonly encountered in our daily clinical practice. Seizures are more frequently observed with hyponatremia, hypocalcemia, and hypomagnesemia [1]. Postictal temporary limb weakness (Todd's paresis/paralysis) that lasts a few minutes and up to 36 h is known to happen occasionally after either a focal (partial) or a generalized tonic-clonic seizure [2]. There is a group of medications that are reported to cause hypomagnesemia. We are presenting a 68-year-old male who presented with recurrent seizures, postictal right hemiparesis, and dysphasia and was found to have severe hypomagnesemia caused by prescribed medications.

Case Presentation

A 68-year-old Caucasian male patient was escorted by an ambulance from home with a history of a sudden-onset slurred speech and right-sided upper limb weakness. His wife noted that the patient developed slurred speech and facial drooping, and then he became mute. She noted that the patient had jerky movements of the right arm, and then his whole body developed jerky movements before he developed the slurred speech. Paramedics could not assess the patient neurologically at home. The patient was confused and not obeying commands. His finger-prick blood glucose was 7.5 mmol/L, and his observations were normal.

More detailed history could be taken in the ED after the patient regained his power, speech, and orientation. The patient did not have any history of vomiting, diarrhea, or fever. He had no previous history of seizures or stroke. He had a past medical history of diabetes mellitus type 2, essential hypertension, hypercholesterolemia, and ischemic heart disease and had 3 coronary stents in 2019.

His drug list includes aspirin, nicorandil, atorvastatin, indapamide, omeprazole, bisoprolol, and metformin. He denies any illicit drug intake and claimed that his alcohol intake is only recreational.

In the Emergency Department, his speech was normal, he was oriented, and his Glasgow Coma Scale was 15/15. His power was 5/5 bilaterally in both upper and lower limbs. His cranial nerves were normal, the plantar reflex was flexor, and no sensory deficit or cerebellar signs could be detected. The rest of the clinical examination was unremarkable.

His full blood count revealed a white blood count of $15.0 \times 10^9/\text{L}$ and neutrophils $9.0 \times 10^9/\text{L}$. His renal profile was normal apart from potassium 2.7 mmol/L (3.5–5.0 mmol/L). His liver function tests, serum calcium, phosphate, thyroid function tests, parathyroid hormone, and coagulation profile were normal.

His C-reactive protein was normal, venous blood gases were within normal levels, no metabolic alkalosis, lactic acid was 2.6 mmol/L (up to 2.0 mmol/L), and glucose was 6.6 mmol/L. CT brain was unremarkable. His ECG showed normal sinus rhythm with a heart rate of 75/min, prolonged QT interval 525 ms (<440 ms in men), and corrected QTc was 551 ms (Fig. 1).

While the patient was in the ED, he developed a second attack of generalized tonic-clonic seizures after momentary jerky movements of the right side of the body and received lorazepam IM. He regained consciousness and was able to move 4 limbs and speak freely after 30 min of right upper limb weakness, and no facial asymmetry was noted. He was started on IV potassium chloride 40 mmol/L infusion over 4 h and was admitted to the acute medical unit. In the ED, the Stroke team assessed the patient, and the impression was focal seizures with secondary generalization and postictal temporary aphasia and right hemiparesis. His evening repeated serum potassium was 3.5 mmol/L.

Fig. 1. Twelve-lead ECG showed normal sinus rhythm with a heart rate of 75/min, prolonged QT interval 525 ms (<440 ms in men), and corrected QTc 551 ms.

The day after admission, the patient developed a third attack of tonic-clonic seizure which lasted 60 s. He was chesty, and oxygen saturation came down to 88%.

He received a loading dose of IV levetiracetam, and blood was urgently taken for renal profile, bone profile, serum magnesium (which was not checked at ED the night before and not included within the bone profile), and venous blood gases. His chest examination revealed right basal crackles on chest auscultation.

The serum magnesium came to be 0.11 mmol/L (0.73–1.15 mmol/L). He received an IV magnesium sulfate infusion and was transferred to an ECG monitored bed. His portable chest X-ray AP view showed right basal patchy shadowing suggestive of an infection (Fig. 2).

The WBC increased to $30.0 \times 10^9/\text{L}$, and his CRP became 150 mg/L (0–5 mg/L). He was started on IV piperacillin/tazobactam for treating aspiration pneumonia in addition to levetiracetam 250 mg twice daily orally.

The CT angiography of the brain was normal, and patient's serum magnesium normalized with IV replacement. He did not have any further seizures, and his oxygenation improved. Gradually he felt better, did not require any oxygen, and his white blood cells and CRP trended down to normal.

On the sixth day after admission, he was switched to oral antibiotics and was discharged home. He had an MRI brain as an outpatient which was normal, and his EEG revealed a normal background rhythm at 8 Hz and no definite epileptiform discharges.

Discussion

Hypomagnesemia is one of the electrolyte abnormalities that can cause seizures, and it is common in hospitalized patients (7–11%) and is more frequent in critically ill patients and associated with higher mortality [3, 4]. Hypomagnesemia may be associated

Fig. 2. Portable (mobile) chest X-ray revealed right basal patchy shadowing suggestive of infection.

with other electrolyte disturbances mainly hypokalemia or hypocalcemia [5]. ECG changes of hypomagnesemia include prolonged QT interval, prolonged PR interval, u wave, and atrial and ventricular ectopic, and it may cause ventricular tachycardia [6]. The prolonged QT interval and prominent u wave are not specific for hypomagnesemia and maybe also noted with hypokalemia and carry a risk of torsade de pointes ventricular arrhythmia [7].

The American Academy of Neurology and the American Society of Epilepsy recommend checking blood glucose and serum electrolytes (sodium, potassium, calcium, and magnesium) for an adult patient presenting with unprovoked first seizures [8]. The incidence of Todd's paralysis following a seizure is 0.65–13% [9].

Our patient presented to the ED with a first seizure and without a history of trauma, drug withdrawal, or signs of meningeal irritation, and his CT head scan in ED was normal. His serum potassium was low, and his ECG showed prolonged QT interval. Initially, his serum magnesium was not checked in the ED. He was started on IV potassium chloride replacement which brought his potassium to 3.5 mmol/L.

He developed a third seizure the day after admission, and his serum magnesium was found to be very low. He did not have any further seizures after IV magnesium replacement and stoppage of the incriminated medications.

Hypomagnesemia induced by long-term proton pump inhibitors has been reported and can recur if it was replaced with another proton pump inhibitor, but not with a histamine type-2 receptor antagonist such as famotidine [10, 11]. The chronic usage of thiazides is also associated with hypomagnesemia and hypokalemia [12, 13].

Our patient was prescribed indapamide for essential hypertension and omeprazole for a suspected gastroesophageal reflux (both were prescribed for more than a year). Both drugs were canceled after the recurrence of seizures and the blood result showing severe hypomagnesemia. Metformin was also stopped because of the high lactic acid after recurrent seizures. The rest of the patient's blood tests, CT head, CT brain angiography, MRI brain, and EEG were unremarkable.

Conclusion

Checking serum magnesium and other electrolytes is one of the required tests in the general workup of any adult patient presenting with an unprovoked first seizure. Hypomagnesemia may be associated with other electrolyte disturbances such as hypokalemia or hypocalcemia and may show similar ECG changes.

A thorough review of medications is essential for patients coming with first seizure and hypomagnesemia or other electrolyte disturbances. Immediate IV replacement of magnesium and transferring to a monitored bed will help to prevent recurrent unprovoked first seizures in adult patients with severe hypomagnesemia.

Statement of Ethics

This case report is exempt from ethics committee approval as per the UK National Service research governance arrangements. The authors have obtained written informed consent from the patient to publish his case report including images. All attempts have been made to make the case anonymous.

Conflict of Interest Statement

Authors declare no conflicts of interest with respect to publication or authorship of this case report.

Funding Sources

Authors declare not receiving any kind of funding from the public, commercial, or nonprofit sector.

Author Contributions

Dr. A.K. wrote the preliminary draft of the case report. Dr. C.J., Dr. K.H., and A.T. contributed and edited the final revision.

Data Availability Statement

All data related to this case report are available to be sent for review if requested, but not publically available.

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