



Sepsis-induced Hypermagnesemia Resulting in Hypotensive Crisis and Bradycardia in an Octogenarian

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Hypermagnesemia with potential life-threatening occurs rarely in clinic. We present the case of an 85-year-old man with community acquired pneumonia with sepsis. After antibiotic treatment, fever subsided. Four days later, his blood pressure and heart beat dropped. Follow-up laboratory investigations revealed a serum magnesium level of 8.2 mg/dL (reference range, 1.7-2.55 mg/dL). Computed tomography (CT) of the abdomen demonstrated few small calcific lesions in the descending colon, which were presumed to be undigested magnesium oxide tablets on account of his history of constipation. Hypermagnesemia was successfully treated and hypotension improved after administration of intravenous calcium chloride and gastrointestinal decontamination with a magnesium-free enema. The possibility of hypermagnesemia should not be neglected when patients present with hypotension and bradycardia. Early diagnosis of hypermagnesemia is very important for preventing life-threatening effects of this condition.

Key words: hypermagnesemia, hypotension, bradycardia

INTRODUCTION

Constipation is a common complaint of older adults. Treatment for constipation includes lifestyle changes, increasing fiber intake, and using laxatives or enemas. Laxatives are taken as a daily medication by 10 percent of older adults.¹ Magnesium oxide is a common medication that can be used as laxative and antacid in patients with normal renal function. Our patient without pre-existing renal dysfunction, sustained hypermagnesemia after acute infection. Sepsis induced renal dysfunction and ileus could be predisposing factors of hypermagnesemia by reducing magnesium excretion and increasing gastrointestinal absorption. Magnesium-containing laxatives for treatment of constipation should be administered with caution in infected patients.

CASE REPORT

An 85-year-old man was admitted to our hospital with a 2-day history of fever, dyspnea and disturbed consciousness. He had a 1-year history of constipation and no pre-existing renal dysfunction. On admission, the patient's temperature was 38.2 °C; pulse, 92 beats per minute; respiratory rate, 22 breaths per minute, and blood pressure, 104/62 mmHg. On examination, there were diffuse crackles over bilateral lungs and abdominal distention. Laboratory test results were as follows, a white blood cell count of $13.52 \times 10^3 / \mu\text{L}$, with 89.3% neutrophils, 7.8% lymphocytes, and 2.8% monocytes, a hemoglobin of 11.8 g/dL, a blood urea nitrogen level of 24 mg/dL (reference range, 6-20 mg/dL) and a serum creatinine level of 2.0 mg/dL (reference range, 0.7-1.2 mg/dL). Electrocardiogram was normal (Figure 1A) and a chest radiography showed increased interstitial marking and ground-glass opacities of both lungs. Antibiotics with intravenous imipenem 250 mg every six hour was given for sepsis.

Two days later after antibiotic administration, his fever subsided and blood pressure was in normal range. Four days later, the patient's blood pressure and heart beat dropped. He had a temperature of 36.2 °C; pulse, 42 beats per minute; respiratory rate, 14 breaths per minutes, and blood pressure, 74/38 mmHg. Follow-up

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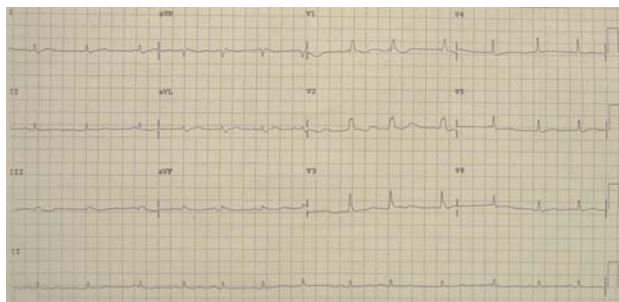


Fig. 1A Electrocardiogram showed sinus rhythm with complete right bundle branch block pattern (Heart rate: 80/min; PR interval: 0.12 sec; QTc interval: 0.378 sec).

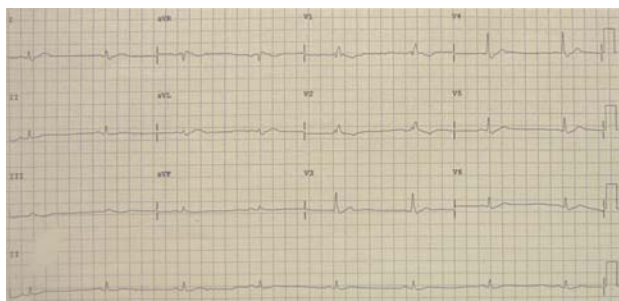


Fig. 1B Electrocardiogram showed sinus bradycardia with complete right bundle branch block pattern (Heart rate: 47/min; PR interval: 0.148 sec; QTc interval: 0.452 sec).

physical examination revealed diminished bowel sounds. Deep tendon reflexes of all extremities were decreased. Laboratory investigations were as follows, a white blood cell count of $8.52 \times 10^3 / \mu\text{L}$, with 70.3% neutrophils, 6.2% lymphocytes, and 3.4% monocytes, a hemoglobin of 12.3 g/dL, a blood urea nitrogen level of 18 mg/dL, a serum creatinine level of 1.2 mg/dL, a serum sodium level of 145 mmol/L (reference range, 136-145 mmol/L), a serum potassium level of 3.5 mmol/L (reference range, 3.5-5.1 mmol/L), a serum free calcium level of 3.9 mg/dL (reference range, 4.5-5.3 mg/dL), and a serum magnesium level of 8.2 mg/dL (reference range, 1.7-2.55 mg/dL). Electrocardiogram showed sinus bradycardia with a prolonged QT interval (Figure 1B). Plain film of the abdomen revealed gas retention and large amounts feces in colon. On account of the impression of ischemic bowel disease, computed tomography (CT) of the abdomen was arranged which demonstrated few small calcific lesions (Figure 2, arrows) in the descending colon, which were presumed to be undigested magnesium oxide tablets on account of his history of constipation. Intravenous

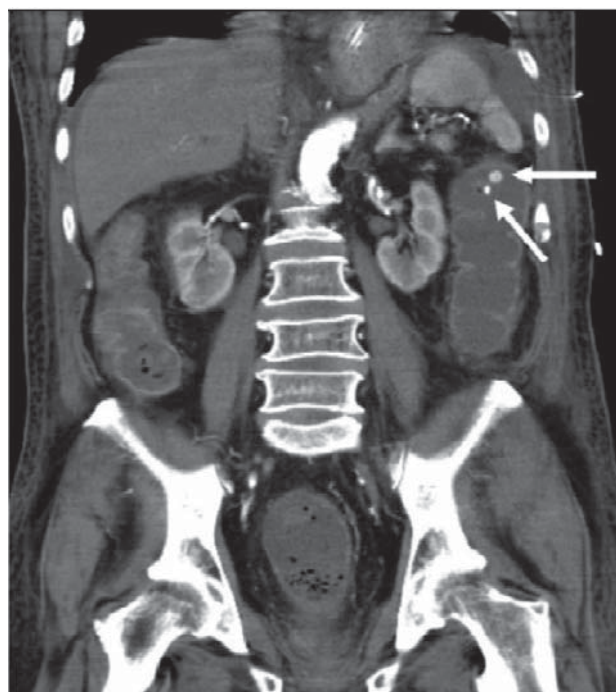


Fig. 2 Computed tomography of abdomen demonstrated large amounts feces and few small calcific lesions in descending colon (arrows).

calcium chloride 20 mg every eight hour for 3 days and gastrointestinal decontamination with a magnesium-free enema were administered for hypermagnesemia. Three days later, the follow-up serum magnesium level was 2.3 mg/dL. After 3-day of therapy with gastrointestinal decontamination and intravenous calcium chloride, and 14-day of antibiotic treatment, the patient recovered well and was discharged in stable condition. After discharge, magnesium-free laxatives, such as sennoside, was prescribed for his constipation.

DISCUSSION

Hypermagnesemia is a rare and life-threatening clinical condition that is caused by magnesium injection, oral ingestion of magnesium-containing laxatives or antacids, chronic kidney injury, primary hyperparathyroidism, Milk-alkali syndrome or adrenal insufficiency. Magnesium hemostasis is regulated mainly by gastrointestinal absorption and renal excretion. The excretion of magnesium is reduced in patients with kidney injury. Bowel movement disorders was reported to be the most common etiology of hypermagnesemia by Clark and Brown that may increase absorption of non-excessive amounts

of magnesium-containing cathartics or antacids.²⁻³ Sepsis can cause acute kidney injury due to reductions in renal blood flow and glomerular filtration.⁴ Upon questioning the patient eventually admitted to covert use of oral magnesium oxide 500 mg twice daily as laxative for one year. Our patient had experienced sepsis with acute kidney injury, leading to reduced magnesium excretion. Sepsis would also cause bowel movement decreasing. Colonic retention of magnesium oxide tablets could have caused prolonged magnesium release and gastrointestinal absorption.

Magnesium is a physiological calcium channel blocker, and hypermagnesemia can lead to blockage of several cardiac channels and reduced impulse transmission at the neuromuscular junction. Hypermagnesemia would suppress the secretion of parathyroid hormone and inhibit the reabsorption in the loop of Henle which lead to hypocalcemia.⁵ Electrocardiograms during hypermagnesemia and hypocalcemia have shown a prolonged QT interval and atrioventricular block.⁶⁻⁷ The clinical manifestations of hypermagnesemia include nausea, vomiting, and flushing. Severe hypermagnesemia can cause absent deep tendon reflexes, hypotension, bradycardia, and respiratory depression.⁸ When patients presented with hypotension, bradycardia, or respiratory depression, the possibility of hypermagnesemia should not be neglected.

The initial management of hypermagnesemia is the discontinuation of magnesium administration. Calcium acts as an antagonist of magnesium, and intravenous calcium is thereby effective for treating hypermagnesemia. In cases of severe hypermagnesemia with an unstable hemodynamic state, hemodialysis can rapidly lower the serum magnesium level. Hemodialysis is the definitive treatment for patients with renal dysfunction or life-threatening hypermagnesemia.⁹⁻¹⁰ Our patient was successfully revived by gastrointestinal decontamination and the administration of intravenous calcium chloride 20 mg every eight hour for 3 days without hemodialysis. The patient's blood pressure and bradycardia recovered after the first dose of intravenous calcium chloride 20 mg and fluid resuscitation without inotropic agents. Electrocardiogram could be a method to monitor hypermagnesemia. Intravenous calcium can thus be used as the first line of treatment for hypermagnesemia.

The case reminds physicians that the possibility of hypermagnesemia should be considered when hypotension and bradycardia develop in patients with renal insufficiency and taking magnesium-containing laxatives. Early diagnosis of hypermagnesemia is very important for preventing the life-threatening effects of this condition.

DISCLOSURE

All authors declare no competing financial interests.

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