Acute Respiratory Failure From Hypermagnesemia Requiring Prolonged Mechanical Ventilation

Khalid Sawalha, MD1 and Krishna Kakkera, MD1

Abstract

Electrolyte abnormalities are an underrecognized cause of respiratory failure in the intensive care unit. One such abnormality is a relatively rare phenomenon of hypermagnesemia resulting in paralysis. A 73-year-old Caucasian male patient presented to the emergency department with diffuse abdominal pain of 2-day duration. He received magnesium citrate and gastrointestinal cocktail for his constipation after initial imaging showed constipation. In view of acute worsening, follow-up computed tomography of the abdomen was done, which showed free air in upper abdomen along with free fluid. Hence, he was taken for emergent laparotomy with repair of pyloric ulcer perforation with omental patch. Post procedure course was complicated by sepsis, acute kidney injury, and respiratory failure with hypoxemia and hypercapnia. On physical examination the patient had flaccid paralysis in all his extremities along with absent brain stem reflexes. Extensive workup including imaging of brain failed to reveal diagnosis. On postoperative day 1, the patient was noted to have magnesium level of 9.2 mg/dL (1.6-2.3 mg/dL), which was thought to be cause of flaccid paralysis and respiratory failure. In view of his acute oliguric kidney injury, he was initiated on intermittent hemodialysis, until his magnesium levels were back to its physiologic limits. His paralysis gradually improved over next 48 to 72 hours and he was liberated from ventilator successfully.

Keywords

hypermagnesemia, flaccid paralysis, prolonged mechanical ventilation

Introduction

Magnesium is a major intracellular divalent cation. It is essential for multiple metabolic and physiological processes. It plays a crucial role in enzymatic actions, electron transporters, and the synthesis of nucleic acids. It influences other electrolytes such as sodium, calcium, and potassium.

Extracellular magnesium and calcium are vital for proper functioning of neuromuscular junction and electrophysiological activity of the myocardium. Normal serum values were found to be variable between 0.7 and 1.0 mmol/L (or 1.5 and 2.0 mEq/L, or 1.7 and 2.4 mg/dL).1

Hypermagnesemia is a relatively common problem noted in ~12% of hospitalized patients. However, clinically significant abnormality is relatively rare. We wish to present an extreme case of hypermagnesemia causing total flaccid paralysis causing prolonged respiratory failure in setting of oral magnesium supplementation with acute kidney injury.

Case

A 73-year-old Caucasian male patient with a past medical history of hypertension, diabetes mellitus type 2, chronic constipation, and chronic lower back pain presented to emergency department with 2-day history of diffuse abdominal pain. Acute abdominal series failed to show significant abnormality other than constipation and hence he was given 1 dose of oral magnesium citrate 300 mL, 1 dose of gastrointestinal cocktail (contains hyoscyamine, Maalox, and lidocaine) 30 mL and a fleets enema on day 1 of hospital stay. His home medication regimen included metformin, meloxicam, hydrocodone, and gabapentin. Overnight, the patient had a near syncopal episode and worsening of his abdominal pain while having a bowel movement. He was noted to be hypotensive with blood pressure of 84/47 mm Hg, heart rate of 132 beats per minute, and with a lactic acidosis level of 8.3 mmol/L; hence, an abdominopelvic computed tomography was done and showed free air in abdomen (Figures 1 and 2). The patient was taken for emergent laparotomy with repair of pyloric ulcer perforation with omental patch. Post procedure course was complicated by sepsis, acute kidney injury, and respiratory failure with hypoxemia and hypercapnia. On physical examination the patient had flaccid paralysis in all his extremities along with absent brain stem reflexes. Extensive workup including imaging of brain failed to reveal diagnosis. On postoperative day 1, the patient was noted to have magnesium level of 9.2 mg/dL (1.6-2.3 mg/dL), which was thought to be cause of flaccid paralysis and respiratory failure. In view of his acute oliguric kidney injury, he was initiated on intermittent hemodialysis, until his magnesium levels were back to its physiologic limits. His paralysis gradually improved over next 48 to 72 hours and he was liberated from ventilator successfully.

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to emergent laparotomy and underwent pyloric ulcer perforation repair with omental patch.

Post procedure the patient was in severe septic shock and acute oliguric kidney injury. After fluid resuscitation, his blood pressure and sepsis improved. However, on postoperative day 1 he was noted to have flaccid paralysis including lack of deep tendon and brain stem reflexes. Due to concerns of anoxic brain injury, brain imaging was done with no acute findings. Ionized calcium, phosphate, and potassium levels were within normal limits. He was noted to have magnesium level of 9.2 mg/dL (Figure 3). This was thought to be cause of his flaccid paralysis and hence was initiated on intravenous fluids along with lasix. In view of oliguric kidney injury, emergent hemodialysis was initiated. The patient received 2 hemodialysis sessions with magnesium levels returning to physiologic levels. Electrolytes are shown in Table 1. Over the next 48 to 72 hours the patient has significant improvement in his hypotension and bradycardia. His mentation and muscle strength and acute kidney injury also improved. He was successfully liberated from the ventilator and subsequently discharged to rehab.

**Discussion**

Respiratory failure is a common cause of intensive care unit admission and electrolyte imbalance is a very underrecognized cause. Our patient had acute respiratory failure with failure to extubate due to flaccid paralysis of his respiratory muscles. This was found to be a result of extremely high serum magnesium levels, which was unexpected given he received only 2 doses of magnesium-containing agents (magnesium citrate and Maalox) within the last 2 weeks. A detailed chart review and interview with spouse confirmed this fact.

Hypermagnesemia is most commonly seen in the setting of renal impairment along with concomitant excessive magnesium intake (via any route). Neuromuscular and cardiac toxicity are common complications of hypermagnesemia. Hypocalcemia can also be seen in the setting of hypermagnesemia due to inhibition of parathyroid hormone secretion, which could also aggravate cardiac toxicity and muscular activity. Our patient had all the features usually described in hypermagnesemia, but with only a small intake of magnesium in setting of acute kidney injury. We hypothesize that the pyloric perforation allowed for increased uptake of magnesium through the inflamed peritoneum (normal gastrointestinal tract absorption is only 30% to 40% of oral intake). Regardless of etiology, this case illustrates an important but rare cause of respiratory failure in the intensive care unit, which can be treated easily and successfully.

Most cases of hypermagnesemia can be prevented by avoiding magnesium-containing agents in patients with renal impairment. However, if a magnesium-containing agent must be administered, the patient should be monitored more frequently for the signs and symptoms noted above. In patients with mild acute kidney injury, initial medical treatment consists of intravenous isotonic fluids, a loop diuretic such as furosemide, and cessation of the agent. This will enhance renal excretion of magnesium. Dialysis is often reserved for patients who have severe oliguric/anuric kidney injury.

As was seen in this case, electrolyte disturbances can be associated with increased morbidity and mortality among critically ill patients. Early recognition and goal-directed therapy of hypermagnesemia can result in a fewer adverse
outcomes such as prolonged intensive care unit stay, higher cost of care, and increased mortality as seen in this case.1

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Ethics Approval
Our institution does not require ethical approval for reporting individual cases or case series.

Table 1. Table showing electrolytes on admission, post-operatively day 1 and after the first dialysis session.

| Electrolyte                  | Admission  | Postoperative day 1 | After first dialysis session |
|------------------------------|------------|---------------------|-----------------------------|
| Sodium (137-145 mmol/L)      | 131        | 128                 | 130                         |
| Potassium (3.5-5.1 mmol/L)   | 4.5        | 5                   | 4.9                         |
| Chloride (98-107 mmol/L)     | 93         | 95                  | 94                          |
| Carbon dioxide (22-30 mmol/L)| 29         | 23                  | 29                          |
| Creatine (0.8-1.5 mg/dL)     | 0.7        | 2.4                 | 2.9                         |
| Magnesium (1.6-2.3 mg/dL)    | 1.9        | 8.3                 | 4.6                         |
| Calcium (8.4-10.2 mg/dL)     | 9.9        | 6.3                 | 6.8                         |
| Lactic acid (0.7-2.0 mmol/L) | N/A        | 3.8                 | 1.7                         |

Figure 3. Graphic line showing daily magnesium levels with green line indicating level on admission, yellow line indicating when the laparotomy was done, and a black line indicating when the patient was extubated.

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Informed Consent
Informed consent for patient information to be published in this article was obtained directly from the patient.

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