Iatrogenic hypermagnesemia following Epsom salt enema

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Abstract

Constipation is a common problem in the older population. Among other treatment modalities, Epsom salt enema has been used in emergency rooms. Since Epsom salts are 100% magnesium sulfate and excreted wholly through the kidneys, hypermagnesemia may result from their use, particularly in patients with impaired renal function. A case of fatal iatrogenic hypermagnesemia resulting from the administration of an Epsom salt enema is presented here. We suggest that magnesium-containing enemas should be avoided in patients with impaired renal function.

Keywords: enema; Epsom salts; hypermagnesemia; iatrogenic; renal failure

1. Introduction

Hypermagnesemia is a rare but potentially fatal condition if unrecognized. The majority of reported cases of hypermagnesemia are iatrogenic, arising from the administration of magnesium-containing laxatives or antacids,1–4 magnesium-containing enemas,5,6 or intravenous magnesium sulfate.7 Magnesium toxicity presents with nonspecific signs and symptoms8 and is therefore difficult to diagnose based on clinical findings. We present a case of iatrogenic hypermagnesemia resulting from the administration of an Epsom salt enema.

2. Case Report

An 85-year-old woman presented to the emergency department with a 3-day history of constipation and diffuse abdominal pain. Her past medical history was significant for amyloid light chain amyloidosis, hemodialysis-dependent end-stage renal disease, gastric angiodysplasia, and anemia. On presentation, her blood pressure was 147/69 mmHg, and all other vital signs were normal. Her cardiac, respiratory, and abdominal examinations were unremarkable. Complete blood count revealed a hemoglobin level of 99 g/L (120–160 g/L), with a normal white blood cell and platelets count; prothrombin and partial thromboplastin times were normal; and urea was 18.6 mmol/L (3.0–7.1 mmol/L), creatinine 972 μmol/L (60–130 μmol/L), sodium 133 mmol/L (135–145 mmol/L), potassium 4.6 mmol/L (3.5–5.0 mmol/L), chloride 99 mmol/L (98–110 mmol/L), calcium 2.36 mmol/L (2.14–2.66 mmol/L), phosphate 1.79 mmol/L (0.89–1.55 mmol/L), and magnesium 0.81 mmol/L (0.70–1.10 mmol/L). An abdominal X-ray revealed dilated small bowel loops and a minimally distended colon. No air–fluid levels or free air was seen.

Shortly after presentation, an enema containing water, glycerin, and 28 g of Epsom salts (approximately 3 g of elemental magnesium) was administered, with no evacuation of stool. Over the subsequent 7 hours, the patient developed worsening abdominal distension and bowel sounds became absent. A large ecchymosis appeared on the right side of the patient’s face, accompanied by the onset of rectal and vaginal bleeding. The patient’s neurologic status deteriorated, with...
progressive skeletal muscle paralysis, loss of deep tendon reflexes, and inability to speak. Breathing became shallow, and oxygen requirements increased to 10 L on a nonrebreather mask. Rectal temperature fell to a nadir of 33.8°C (Figure 1). Electrocardiography was performed, which showed first-degree atrioventricular block (PR interval 218 milliseconds) and a prolonged QRS interval (118 milliseconds). Piperacillin–tazobactam was administered due to concern of sepsis, followed by cryoprecipitate and platelets in an attempt to correct the bleeding. Blood work drawn 2 hours after the enema revealed the following: a calcium level of 2.94 mmol/L, a phosphate level of 1.72 mmol/L, and a magnesium level that exceeded the detectable range of the assay (>7.8 mmol/L). Unfortunately, the treating team of physicians was unaware of these results.

Seven hours after the administration of the magnesium-containing enema, the consulting internal medicine team noted the patient’s elevated magnesium level. Repeat blood work confirmed the diagnosis of hypermagnesemia (>7.8 mmol/L), and revealed normal partial thromboplastin time (PTT) and International normalised ratio (INR), hemoglobin level 89 g/L, white blood cell count $10.7 \times 10^9$/L, and platelet count $139 \times 10^9$/L. The patient was sent urgently for dialysis. Post dialysis, the patient’s oral temperature

![Figure 1. Vital signs in relation to magnesium levels. DBP = diastolic blood pressure; HR = heart rate; RR = respiratory rate; SBP = systolic blood pressure.](image-url)
normalized and oxygen requirements declined to 3 L by nasal prongs. She was alert and speaking, patellar reflexes were reduced bilaterally, and strength had improved throughout. Repeat electrocardiography revealed normal sinus rhythm, a PR interval of 178 milliseconds, and a QRS interval of 84 milliseconds. The postdialysis serum magnesium level was 4.35 mmol/L, and a second hemodialysis treatment on the same day reduced the magnesium concentration to 2.47 mmol/L. The patient received hemodialysis every other day for the management of her end-stage renal disease, and serum magnesium levels gradually normalized. However, the patient continued to experience fatigue, poor appetite, and muscle weakness. On Day 9 post admission, the patient declined further hemodialysis and care was transferred to a palliative care physician. She died peacefully.

3. Discussion

Renal excretion is the sole route of magnesium elimination, therefore, hypermagnesemia most frequently occurs in the context of renal impairment. However, magnesium toxicity has also been reported in patients with normal renal function, and impaired gastrointestinal motility has been cited as an additional risk factor. In the present case, end-stage renal disease and impaired intestinal motility were predisposing conditions for the development of magnesium toxicity. The patient received approximately 28 g of Epsom salts, which are 100% magnesium sulfate. This is equivalent to 2.8 g of elemental magnesium, a considerable amount given that the recommended daily allowance for an adult is 300–400 mg per day.

Hypermagnesemia presents with nonspecific signs and symptoms. Neurologic effects include lethargy, loss of deep tendon reflexes, skeletal muscle paralysis, and coma. Cardio-respiratory effects are bradycardia, hypotension, first-degree atrioventricular block, QT interval prolongation, respiratory depression, and ultimately cardiopulmonary arrest. Hypermagnesemia has also been associated with paralytic ileus. The toxic effects of supraphysiologic magnesium levels are due to antagonization of calcium-dependent acetylcholine release at neuromuscular junctions and calcium-channel blocking effects on cardiac myocytes. With the exception of hypotension, bradyarrhythmia, and cardiopulmonary collapse, the patient in the present case report displayed all the aforementioned features of hypermagnesemia.

Several treatment options are available to correct hypermagnesemia. Since magnesium competitively binds to calcium channels and hence acts as a calcium-channel blocker, administration of intravenous calcium can reverse cardiac arrhythmia, respiratory depression, and hypotension. Adequate hydration and use of diuretics may help eliminate excess magnesium through diuresis. Hemodialysis can be lifesaving in cases of severe toxicity and in patients not responding to other less invasive measures.

Several aspects of the patient’s presentation are noteworthy. First, the patient became hypothermic shortly after the administration of the magnesium-sulfate enema. Hypothermia associated with hypermagnesemia has been described previously; however, it is not widely recognized as a clinical manifestation of magnesium toxicity. The onset of hypermagnesemia in the present report was associated with a concomitant rise in serum calcium concentration, which has been reported only twice previously. This is likely because of the exchange of calcium at the bone surfaces with magnesium in the circulation. In contrast, hypermagnesemia is more frequently associated with hypocalcaemia, an effect that is mediated through depression of parathyroid hormone levels due to hypermagnesemia. Finally, the patient developed significant bruising and bleeding following the onset of magnesium toxicity. Although uremia and amyloidosis would have predisposed the patient to bleeding due to platelet factor X dysfunction, it is possible that hypermagnesemia may have been a precipitating factor. This is supported by the experimental observation that magnesium inhibits platelet aggregation both in vitro and in vivo.

3.1. Learning points

(1) Iatrogenic hypermagnesemia may be associated with hypothermia, elevated serum calcium levels, and bleeding diathesis. Hypothermia and hypercalcemia are rare sequelae of magnesium toxicity.

(2) This case provides additional evidence for avoiding magnesium-containing enemas in patients with impaired renal function or gastrointestinal motility disorders.

(3) Physicians should consider tap water or polyethylene glycol-containing enemas as alternatives in these clinical contexts.

Conflicts of interest

None of the authors have any financial conflicts of interest to declare.

References


