
Selected Topics: Toxicology

HYPERMAGNESEMIA-INDUCED FATALITY FOLLOWING EPSOM SALT GARGLES

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□ **Abstract**—Hypermagnesemia is a rare cause of coma in a patient with normal renal function. When present, it is often because of iatrogenic medication overdose. We report a fatal case of chronic Epsom salt gargles for halitosis that produced a serum magnesium of 23.6 mg/dL (9.8 mmol/L) and resulted in coma. We review the wide presentation of hypermagnesemia from subtle neurologic and cardiovascular signs to the major life-threatening clinical manifestations of shock, dysrhythmias, coma, and cardiopulmonary arrest despite emergency dialysis. © 2002 Elsevier Science Inc.

□ **Keywords**—hypermagnesemia; coma; Epsom salts; renal failure

INTRODUCTION

As the “forgotten cation,” magnesium chemistry abnormalities stand in the laboratory shadow of the other four major cations of the human body (1). Hypomagnesemia is much more common than hypermagnesemia. Clinically significant hypermagnesemia is rare in individuals with normal renal function, and its cause is often iatrogenic (2–4). Consumers and medical professionals alike often underestimate the potential toxic effects of hypermagnesemia. Clinical manifestations may be subtle and resemble many other diseases. Often, a seemingly harmless over-the-counter (OTC) medication such as a laxative, analgesic, or antacid is the culprit (5–9). A wide range of cardiac and neurologic presentations is possible,

with the hallmark being refractory hypotension (10). Fatalities have been reported.

CASE REPORT

A 31-year-old woman was brought to the Emergency Department (ED) by ambulance from home with a chief complaint of loss of consciousness. A neighbor reported that she had been in good health and was not taking any medications. Emergency Medical Services (EMS) did not find any medications, empty bottles, or other paraphernalia at the scene. As per coma protocol, she had been given 100 mg thiamine IV, 50 grams glucose IV, and 0.8 mg naloxone IV by EMS.

The patient was unconscious but withdrew to painful stimuli (Glasgow Coma Scale = 6). Vital signs were: blood pressure 86/52 mm Hg; pulse, 56 beats/min; respiratory rate, 14 breaths/min.; and temperature 33.8°C. The skin was atraumatic, dusky and cool. Pupils were equal but unreactive to light and accommodation. The cardiopulmonary examination was normal. The abdominal examination revealed mild distention, hypoactive bowel sounds and hard, guaiac-negative stool on rectal examination. The neurologic examination was notable for symmetrically depressed reflexes (1/4). Corneal reflexes were symmetrically present and the gag reflex was absent. No pathologic reflexes were noted. Laboratory studies included: white blood cell (WBC) of 8700/mm³ with normal differential, hemoglobin, hematocrit, plate-

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let count, activated partial thromboplastin, and prothrombin times; sodium, 135 mmol/L; potassium, 3.6 mmol/L; bicarbonate, 21 mmol/L; blood urea nitrogen, 21 mg/dL (7.5 mmol/L); creatinine, 1.1 mg/dL (93.4 μ mol/L); glucose, 76 mg/dL (3.8 mmol/L); calcium, 10.4 mEq/L (2.61 mmol/L); phosphorus, 2.8 mEq/L (0.655 mmol/L); chloride, 96 mEq/L (96 mmol/L); an anion gap of 23.5 mEq/L (23.5 mmol/L); an osmolal gap of 23 mosm/L; serum amylase, CPK, LDH, SGOT, SGPT, and alkaline phosphatase were normal; negative toxicology screen; pH 7.28; pCO₂, 44 mm Hg; pO₂, 144 mm Hg; and an oxygen saturation of 99% on 3L of oxygen by nasal cannula. The electrocardiogram (EKG) showed a sinus rate of 56 and a QRS axis of 60°. The PR interval was 0.23 s, the QRS interval was 0.12 s, and the QT interval prolonged for the rate (0.453 s). T waves were peaked and ST segments and voltage were normal. Computed tomography (CT) scan of the head was normal. Because a drug overdose was suspected, activated charcoal was administered through a nasogastric tube.

During the course of workup the patient became progressively hypotensive and apneic. The patient was intubated and, shortly thereafter, developed asystole, punctuated by episodes of ventricular fibrillation. Five doses of epinephrine 1 mg IV were administered. A 100 mg IV lidocaine bolus was given, followed by an infusion of 2 mg/min. Spontaneous cardiac activity returned with a sinus rhythm of 64 and a blood pressure of 108/66 mm Hg. Five minutes later asystole, alternating with ventricular fibrillation, again developed. Two additional doses of 1 mg epinephrine IV were given, plus 1.5 mg IV atropine sulfate, a fluid bolus of 2L NS, 40 mg IV furosemide, dopamine infusion up to 10 μ g/kg/min (titrated for a systolic pressure > 90 mm Hg), defibrillation once at 200 joules, and insertion of a transvenous pacer. Pupils were fixed and dilated, but there were no lateralizing neurologic signs. An extremely high serum magnesium level, 9.8 mmol/L (23.6 mg/dL), was reported and confirmed.

Two hours and 6 h after presentation, the patient received 600 mg of IV 10% calcium chloride. She was given vigorous non-alkaline IV fluids and furosemide to induce diuresis. A stormy resuscitation period followed, marked by declining urine outputs (250 mL over 6 h), acute renal failure, and multiple episodes of asystole and ventricular fibrillation requiring repeated defibrillation (total of 17). Seven hours after admission to the ED, the patient was emergently dialyzed. Repeat chemistries showed the serum magnesium was 7.32 mg/dL (3.05 mmol/L); serum chloride, 103 mEq/L (103 mmol/L); serum bicarbonate, 27 mEq/L (27 mmol/L); serum blood urea nitrogen, 22 mg/dL (7.7 mmol/L); serum creatinine, 0.9 mg/dL (79.4 μ mol/L). The patient's neurologic status

remained unchanged. A postdialysis arrest occurred from which she could not be resuscitated.

Upon careful questioning of her 9-year-old daughter, it was determined that the patient had been concerned with halitosis for about one month. Progressively more frequent and larger gargles with Epsom Salts had been used over the intervening weeks. An entire box had been used in the 2 days preceding her illness.

DISCUSSION

Clinical interest in magnesium, particularly by the specialty of Emergency Medicine, has grown significantly over the last several years (11). It has been used as an anticonvulsant in pre-eclampsia and eclampsia, as an antidysrhythmic, an antacid, a laxative, an antihistamine drug, as a cathartic to treat some poisonings, and has been recommended as routine replacement therapy in the potential hypomagnesemic states, such as chronic alcoholism. As the fourth most common cation in the body (1700–2300 mEq/70kg), only 1% of total body magnesium is present in the extracellular fluid, and of this, one-third is plasma protein bound (1,12). Therefore, spuriously low plasma levels are recorded in the presence of hypoalbuminemia. The major site for magnesium absorption is the proximal small bowel (13–15). The normal serum value of magnesium is 0.60 to 0.90 mmol/L (1.5 to 2.2 mg/dL). Magnesium is, however, the second most abundant intracellular cation after potassium (16). Because intracellular and serum values may vary independently, a normal serum magnesium level does not exclude a total body deficit nor excess. Magnesium is a nonspecific, natural calcium channel blocker. Renal homeostasis maintains the magnesium serum level. Seventy-five to 90% of the magnesium reabsorption occurs at the proximal tubule and ascending loop of Henle so that a myriad of magnesium-dependent enzyme systems critical to cellular metabolism (e.g., amino acid activation, oxidative phosphorylation, protein synthesis, glucose utilization, ATP reactions, DNA reactions) are preserved.

Hypomagnesemic states secondary to dietary deficiencies, diuretic therapy, diabetes, renal losses, and malabsorption are common clinically, particularly with alcoholism. Hypermagnesemia is much less common, especially in the setting of normal renal function. Its presentation is frequently subtle and nonspecific and often the symptoms are attributed to other illnesses. Hypermagnesemia is associated with a high morbidity and may be life threatening. Eighty-six percent of such cases may be missed when serum levels are determined only at the request of a physician based on clinical suspicion (e.g., history [see below], paresis, altered mental status, abnormal electrocardiogram (EKG)) (17).

Table 1. Dose-Related Effects of Magnesium (13,15,18,27-31)

Serum Magnesium Level		Effect
mg/dL	mmol/L	
1.7-2.4	0.7-1.05	Normal serum level
5-8	2-3.5	Nausea, flushing, headache, hyporeflexia, lethargy
9-12	4-5	Somnolence, loss of deep tendon reflexes, prolongation of QRS, PR, QT intervals, bradycardia, and hypotension
>15	>6	Complete heart block, respiratory paralysis, coma, shock
>20	>8	Asystole, death

Mild hypermagnesemia has been observed in patients receiving lithium therapy, or those with underlying hyperparathyroidism, hypothyroidism, diabetic ketoacidosis, viral hepatitis, in certain neoplasms with skeletal involvement, in pituitary dwarfism and the milk-alkali syndrome, or with adrenocortical insufficiency (18). The most common cause is iatrogenically encouraged excessive intake of magnesium containing laxatives, cathartics, or supplements, particularly in the presence of a perforated viscus, constipation, small-bowel hypomotility disorder, ureteral irrigation, parenteral replacement magnesium, excessive magnesium in dialysate solutions, or concomitant use of anticholinergic and narcotic agents and multiple doses of magnesium-containing cathartic therapy given in conjunction with activated charcoal (6,8,14,19-23). Epsom Salt, a therapeutic agent originally derived from a well in Epsom, England in 1695, is almost 100% magnesium sulfate. Other common magnesium-containing OTC products may also contain carbonate, dioxide, gluconate, hydroxide, oxide, trisilicate, citrate, salicylate, and lactate derivatives of magnesium. In the setting of coexisting renal failure (GFR < 30 mL/min), excessive intake of magnesium containing antacids is the most frequent precipitating etiology (24). Additionally, hypermagnesemia has occurred in patients from intentional overdoses.

Nausea, headache, flushing, warmth, and lightheadedness characterize minor elevations of serum magnesium. The cardiovascular, respiratory, and neuromuscular systems are affected at higher doses (Table 1). In large doses, magnesium acts like curare at the neuromuscular junction and a parasympathetic blocking agent (25). Paralytic ileus has been reported (4). Although the correlation between symptoms, signs, and serum level can be extremely variable and dependent on comorbid states, in most cases respiratory failure precedes cardiac collapse (26). Muscle weakness, an abnormal EKG (prolonged PR, QRS, QT intervals, bradycardia, asystole) and altered mental status with a normal CT scan should suggest

hypermagnesemia. The highest published serum magnesium level in a patient who survived was 10.65 mmol/L (26.0 mg/dL) (14).

The toxic effects of acute hypermagnesemia are readily reversible. Emergent treatment for severe hypermagnesemia consists of magnesium elimination by forced diuresis and supportive treatment. All exogenous magnesium containing medications, nutritional supplements, and dietary sources should be withheld (12). Emergent renal dialysis utilizing a magnesium-free dialysate in the setting of renal impairment is the definitive therapy. All patients should receive general supportive care, especially aggressive airway management. Because magnesium is a natural physiological calcium-channel blocker, calcium can reverse this antagonistic action. Calcium is especially effective for hypotension, dysrhythmias, and respiratory distress and also enhances excretion of magnesium (20). The usual dose for treatment is 5-10 mEq of calcium (CaCl₂) rapidly IV over 30 s, repeated as necessary every 5-10 min.

CONCLUSION

We report a case of severe hypermagnesemia and normal renal function following chronic gargling with Epsom Salt that resulted in coma and cardiopulmonary arrest. The patient expired despite aggressive medical intervention including renal dialysis. While patients with higher serum magnesium levels have survived, the metabolic effects of chronic hypermagnesium are probably profound.

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