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Case Report

Poisoning as a Result of Barium Styphnate Explosion

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Background *Acute barium poisoning is most often the result of accidental or suicidal ingestion of the rodenticide, barium carbonate. We describe a trauma patient whose condition was complicated by severe acute barium toxicity from an explosion of the propellant, barium styphnate. In addition to critical injuries, the patient manifested classic signs of barium toxicity including repeated profound hypokalemia, cardiac arrhythmias, respiratory failure, prolonged gastrointestinal dysfunction, paralysis, myoclonus, hypertension, and profound lactic acidosis.*

Methods *The patient required lidocaine for ventricular bigeminy, massive infusions of potassium, prolonged ventilatory support, and parenteral nutrition to manage the effects of barium toxicity.*

Results *He is the first reported case to demonstrate recurrent profound hypokalemia as an effect of blood transfusions.*

Conclusions *Considering the paucity of information concerning management of this life-threatening problem, the pathophysiology of barium toxicity, including the transfusion related hypokalemia, and its management is reviewed.* Am. J. Ind. Med. 41:285–288, 2002. © *2002 Wiley-Liss, Inc.*

A previously healthy 50-year-old male, who worked in a munitions factory with the propellant barium styphnate, was placing about 1 kg of the compound behind a 2″ thick transparent blast shield when it exploded, throwing him 40 feet and moving the building's sand reinforced blastproof wall.

He was found combative and unresponsive, with burns to the face and abdomen, and marked deformity of the right upper extremity. He was promptly intubated and transported to the hospital. Upon arrival to the trauma resucitation area, his initial arterial blood gas (ABG) on an FiO_2 of 1.0 his saturation was 92%, pH 6.84, pCO_2 35 mmHg, pO_2 213 mmHg, and lactate 21.8 mmol/L. Serum potassium was 3.5 mmol/L, bicarbonate 10 mmol/L, sodium 141 mmol/L, and chloride 107 mmol/L. His face had burns and tattooing, with singed eyebrows and nasal hairs. The corneas were burned and the right eardrum ruptured. Partial and full thickness burns covered the abdomen. The right upper extremity was mangled and pulseless, with an open fracture of the humerus and closed comminuted fractures of the radius, ulna, humeral condyles, and hand. Spectrometric analysis of the serum and urine for barium showed concentrations roughly 20 and 80 times higher than normal. The serum and urine concentrations in our patient were 370 mcg/dL and 1600 mcg/L, respectively. A serum concentration of greater than 20 mcg/dL is considered abnormal.

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The initial acidosis was corrected over 90 min with bicarbonate drip and ventilator manipulation. An ABG at that time recorded a serum potassium of 2.9 mMol/L. Fifty milliequivalents (mEqs) of potassium was started at 10 mEqs an hour, but a repeat ABG 1 hr later showed the serum potassium had dropped to 2.3 mMol/L. Despite the pH correction to 7.36; the pCO_2 fell to 18mmHg and the base excess was minus 14. The remaining 40 mEqs of potassium were infused over 30 minutes. Despite this rapid infusion, the potassium fell to 1.5 mMol/L. A bolus of 100 mEq of KCl was administered over thirty minutes, only correcting the serum potassium to 4.2 mMol/L.

During irrigation, debridement and fasciotomy of his right upper extremity, the patient's serum potassium was 1.8 mMol/L. Rapid infusion of potassium (120 mEq KCl over 30 minutes) raised the level to 6.3 mMol/L. Although his serum potassium rose to 8.5 mMol/L postoperatively; no ventricular ectopy was noted on continuous EKG or 12 lead EKG. Serum potassium declined to 6.0 mMol/L over the following 7 hr, and further dropped to 2.2 mMol within 2 hr of transfusion of packed red blood cells (RBC) to replace intraoperative blood losses. Two hundred and ten mEqs of KCl were administered over the next 6 hr and the potassium increased to above 4.0 mMol/L (Fig. 1).

During the first 4 days the barium toxicity manifested as profuse diarrhea, severe muscular weakness, including muscles of respiration, clonus, persistent but diminishing lactate levels and a recurring pattern of sudden hypokalemia after transfusions. He required frequent, large infusions of potassium in addition to maintenance in the hyperalimentation fluid to keep his serum potassium over 3.5 mMol/L. He was discharged 4 weeks after the explosion.

DISCUSSION

Acute barium poisoning is well described in the study. It is most often the result of accidental or suicidal ingestion of the rodenticide, barium carbonate [Johnson and VanTassel, 1991]. Barium intoxication has also been reported as a result of absorption through a chemical burn [Stewart and Hummel, 1984] and inhalation of metal fumes [Hicks et al., 1986]. This case was caused by an explosion of barium styphnate with transcutaneous and pulmonary absorption.

Physicians are familiar with barium as a contrast medium in its inert and insoluble form, barium sulfate. While barium sulfate is not absorbed systemically, water-soluble barium salts (carbonate, chloride, sulfide, and nitrate) can be absorbed and cause barium poisoning. These salts are widely used in industry [Budavari, 1996].

Barium styphnate is a 2,4,6-trinitroresorcinol salt made from styphnic acid, a compound often used in the production of explosives [Budavari, 1996]. It has an

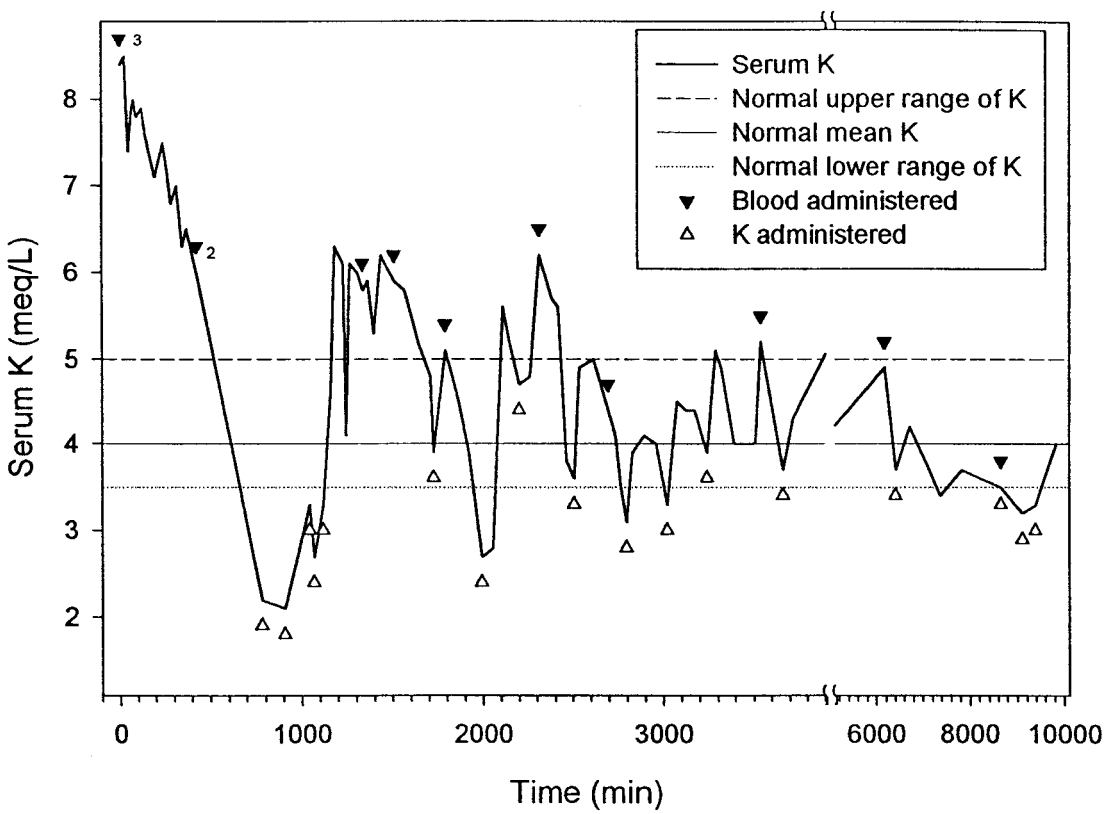

FIGURE 1. Serum potassium levels in a barium-poisoned patient.

explosive temperature of 341°C and a static sensitivity of 2 J. This is relatively stable when compared to other common explosives with a static sensitivity of two microjoules and therefore barium styphnate is classified as a propellant, rather than an explosive. However, in large quantities, propellants can explode.

The lethal dose of most barium salts is between 1–30 g taken orally. Toxicity has been reported with as little as 200 mg. The concentration in air that is considered immediately dangerous is 250 mg/m^3. The onset of action is minutes to hours after ingestion, inhalation, or absorption through a burn. Barium is excreted in stool and, to a lesser extent, in urine, the remainder is largely incoporated into bone. Victims develop gastrointestinal, cardiovascular, respiratory and neuromuscular symptoms [Newton et al., 1977; Stewart and Hummel, 1984; Shankle and Keane, 1988; Johnson and VanTassel, 1991; Olson, 1994; Downs et al., 1995].

The hallmark of barium poisoning is life-threatening hypokalemia; first described by Roza and Berman [1971] when he noted a net uptake of potassium by muscle cells. Once absorbed, barium blocks a cell's passive efflux channel for potassium, but has no effect on the sodium-potassium ATP pump which actively transports potassium into the cell [Roza and Berman, 1971]. The net effect is rapid, intracellular sequestration of potassium and subsequent depolarization of cellular membranes causing gastrointestinal dysfunction, cardiac dysrhythmias, skeletal muscle, and respiratory paralysis [Layzer, 1982; Johnson and VanTassel, 1991]. Olson, however, implicates rapidly developing severe hypokalemia as the principle etiology of death in these patients and claims that studies have excluded intracellular sequestration of potassium as the cause of hypokalemia. He does not offer another etiology of the hypokalemia [Olson, 1994].

Managing this severe hypokalemia requires large, rapid infusions of KCl. Schorn et al. [1991] reports giving 400 mEq of KCl in 1 day to a patient who ingested barium carbonate. Others have also noted the necessity to give rapid infusions of 50–75 mEq of KCl [Stewart and Hummel, 1984]. Our maximum bolus was 120 mEq over 30 min with a maximum 24 hr dose of 380 mEq. Furthermore, as in our case, both Schorn and Johnson overcorrected their patient's serum potassium to levels considered cardiotoxic, yet observed no evidence of cardiotoxicity by 12 lead EKG as well as continuous EKG monitoring. The cardioprotective role of barium during severe hyperkalemia was demonstrated by Roza and Berman [1971] who noted that electrocardiograms of hyperkalemic dogs (serum K greater than 9 mEq/L) normalized when 1:30 barium chloride/potassium chloride infusions were started.

The hyperkalemic potassium levels fell slowly until transfusions of red blood cells were given; then they plummeted. The introduction of RBC's provided a new reservoir within which the potassium was sequestered as the passive potassium efflux channels are blocked. This mechanism is suggested by Roza and Berman [1971] who noted an intracellular potassium shift in canine red blood cells exposed to barium. This seems to be the first major trauma patient with barium poisoning to require transfusions and demonstrate this effect. In addition to blocking passive potassium channels, barium directly affects vascular, cardiac, and gastrointestinal smooth muscle, and the diaphragm. Ventricular tachyarrhythmias, hypertension, vomiting, and diarrhea are reported in both laboratory and clinical series [Roza and Berman, 1971; Johnson and VanTassel, 1991]. Subsequent prolonged ileus and respiratory failure are also reported. Lactic acidosis is also reported by Schorn et al. [1991] who attributes it to inadequate tissue perfusion due to barium induced intense vasoconstriction.

It is suggested that serum barium may precipitate in the renal tubules when exposed to sulfates, thus causing renal failure [Wetherill et al., 1981]. Our patient received many boluses of both morphine and magnesium sulfate without evidence of renal compromise. A pregnant patient in Johnson's series received magnesium sulfate tocolysis with no adverse renal effect [Johnson and VanTassel, 1991].

If treated promptly with infusions of potassium, fluid resuscitation, and ventilatory support when needed, most patients recover fully within a few days. Although barium has a serum elimination half-life of 3.5 days, the intracellular effects are not yet fully described and some respiratory and gastrointestinal effects seem to last for much longer periods.

In summary, a knowledge of the treatment regimens for barium toxicity and its hallmark hypokalemia is valuable given the widespread use of barium salts in industry. This is the first reported case of a victim of a barium styphnate explosion in addition to demonstrating recurrent profound hypokalemia after blood transfusions. Aside from traditional management of these injuries and complications, physiologically lethal doses of potassium were required to treat his barium toxicity. Overcorrection of the serum potassium to cardiotoxic levels had no adverse affect, bearing out the fact that barium toxicity makes all cells resistant to the effects of high doses of potassium.

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