Intoxication by large amounts of barium nitrate overcome by early massive K supplementation and oral administration of magnesium sulphate

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Abstract
Suicide by ingestion of barium is exceptionally rare. Adverse health effects depend on the solubility of the barium compound. Severe hypokalemia, which generally occurs within 2 hours after ingestion, is the predominating feature of acute barium toxicity, subsequently leading to adverse effects on muscular activity and cardiac automaticity. We report one case of acute poisoning with barium nitrate, a soluble barium compound. A 75-year-old woman was hospitalized after suicidal ingestion of a burrow mole fumigant containing 12.375 g of barium nitrate. About 1 hour post-ingestion, she was only complaining of abdominal pain. The ECG recording demonstrated polymorphic ventricular premature complexes (VPCs). Laboratory data revealed profound hypokalemia (2.1 mmol/L). She made a complete and uneventful recovery after early and massive potassium supplementation combined with oral magnesium sulphate to prevent barium nitrate absorption.

Keywords
barium nitrate, acute poisoning, hypokalemia, arrhythmia, potassium supplementation, oral magnesium sulphate

Introduction
Suicide by ingestion of barium is extremely rare. The adverse health effects of exposure to a barium compound depend on how well this compound dissolves in either water or acid. Barium nitrate, chloride, hydroxide, acetate and sulphide dissolve far more easily in water than barium sulphate and carbonate. However, barium carbonate dissolves within the acidic content of the stomach in contrast to barium sulphate. Therefore, barium sulphate is considered to be non-toxic and has been widely used for medical x-ray investigations of the gastro-intestinal tract.¹ We report one case of acute poisoning with barium nitrate, a soluble barium compound.

Case report
A 75-year-old woman with a medical history of major mood disorders treated by psychotherapy with no current adjunction of pharmacotherapeutic agents was hospitalized on 16 June 2008 after suicidal ingestion of a burrow mole fumigant containing 12.375 g of barium nitrate and 6.255 g of sublimed sulphur powder.

At the time of hospitalization (1 hour post-ingestion), she was only complaining of abdominal pain. The ECG recording demonstrated polymorphic ventricular premature complexes (VPCs). Laboratory data revealed profound hypokalemia (2.1 mmol/L). She made a complete and uneventful recovery after early and massive potassium supplementation combined with oral magnesium sulphate to prevent barium nitrate absorption.

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marked hypokalemia (2.1 mmol/L). Abdominal x-ray evidenced no radio-opaque foreign bodies. Oral magnesium sulphate (30 g) was given to prevent ongoing absorption by precipitating barium nitrate to insoluble non-toxic barium sulphate. Slow intravenous injection of 1 g of magnesium sulphate was given to stabilize the heart rhythm. The patient was then transferred to the Intensive Care Unit for aggressive correction of her hypokalemia and ECG monitoring. Within less than 24 hours, her kalemia normalized (4.8 mmol/L) after receiving an intravenous infusion of 322 mEq of potassium associated with potassium canrenoate, a potassium-sparing diuretic. Concomitantly, ECG returned to normal. The patient experienced neither muscular abnormalities nor hemodynamic disturbances. Her renal function remained normal (blood creatinin level: 61.1 µmol/L). The patient was discharged after 3 more days of uneventful observation.

The toxicological analysis (inductively coupled plasma-atomic emission spectrometry) of blood samples drawn shortly after admission confirmed the magnitude of her barium intoxication with blood barium level at 17.2 mg/L (125.5 µmol/L) and urine barium level at 28 mg/L.

**Discussion**

Poisonings with soluble barium compounds typically produce profound hypokalemia. The majority of previously reported intoxications were related to epidemic food poisonings due to the mistaken use of barium carbonate instead of flour or salt.3-5 In such a setting, death occurred at least once with hypokalemia down to 0.8 mmol/L.5 Suicidal poisonings were much less often reported, and the involved compounds were barium nitrate,6-8 chloride,9-12 sulphide13-16 and carbonate.17-20 Whatever the soluble compound involved in suicidal poisonings, hypokalemia was consistently observed and was frequently below 2 mmol/L.7-11,14-19 Although severe gastroenteritis within the first 2 hours after ingestion is a common finding, it cannot be considered the cause of such a profound hypokalemia. Severe hypokalemia is indeed the predominant feature of acute barium poisonings even though gastro-intestinal symptoms are mild to moderate. Indeed, hypokalemia was also reported following extensive burns after explosion of the propellant barium styphenate and the spattering of molten barium chloride in an occupational setting.21,22 Barium can induce hypokalemia through two synergistic mechanisms. It is a reversible potassium channel antagonist that blocks the passive efflux of intracellular potassium and it can increase the activity of the cell membrane Na+-K+-ATPase pump.23-25 Therefore, hypokalemia is related to an intracellular shift of potassium. Indeed, the total body potassium load seems to remain constant.

The clinical features of barium poisoning are considered to reflect the rapid onset and progression of hypokalemia.26 They mainly include muscular deficiencies, which may lead to global paralysis and respiratory muscle failure.2-11,13,15-19 Rhabdomyolysis may occur.2,15 ECG abnormalities are common with flattening and inversion of the T wave, increased prominence of the U wave and sagging of the ST interval; they often result in ventricular arrhythmias.2,4,5,7-10,14,18,19 Some authors have suggested a direct effect of barium ions on muscle cells to account for their reported findings of muscular deficiency which subsided only after serum barium levels had decreased, whereas kalemia had normalized.16-18 However, they did not mention any serum phosphate levels. Hypophosphatemia, which can be as low as 0.48 mmol/L, is indeed a common finding in acute barium poisonings.2,6,13,15 The underlying mechanism is unknown. The clinical manifestations of severe hypophosphoremia include profound muscular weakness and rhabdomyolysis.27 It can also reduce the threshold to ventricular arrhythmias as hypokalemia. To substantiate a direct effect of barium ions on muscle cells, it would be helpful to monitor serum phosphate levels, especially when normalization of kalemia fails to achieve full recovery. The effectiveness of hemodialysis through improved barium elimination has been reported, no measurement of barium clearance across the dialyser has so far been described.7,8,10,16,18,19

The majority of acute barium poisonings evolved toward recovery after 24 hours of massive potassium supplementation and supportive treatment. It is noteworthy that intravenous magnesium sulphate should be avoided to prevent renal precipitation of insoluble barium sulphate, which may lead to renal failure.11,17 Large amounts of intravenous potassium, up to 440 mEq over the first 24 hours,9 have been reportedly required to normalize serum potassium levels. Hemodialysis with potassium-enriched dialyzing fluid allows to rapidly correct serum potassium levels.
and should be considered when severe hypokalemia does not respond to intravenous potassium supplementation. Rebound hyperkalemia is common and usually well tolerated.

Rebound hyperkalemia is common and usually well tolerated.\(^{2,11,15,19}\) Toxic effects can occur following ingestion of as little as 200 mg of soluble barium compound.\(^{24}\) The lethal dose is usually estimated to be in the range of 1 to 15 g.\(^{28}\) The rate of absorption following oral intake depends on the solubility of the barium compound. The time to peak plasma levels is usually 2 hours. Plasma barium levels rapidly fall within 24 hours. Renal elimination of the absorbed dose accounts for 10% to 28% of total barium excretion, the predominant route of elimination being the feces. Whereas activated charcoal is ineffective to prevent barium absorption, sodium or magnesium sulphate (25–30 g in adults) can be used orally as this is expected to precipitate non-absorbed barium ions to insoluble barium sulphate. In published case reports of acute barium poisonings, serum barium levels ranged between 3.7 mg/L and 41.1 mg/L.\(^{5,7,8,16,17,19,21,22}\) One death was recorded with a serum barium level of 9.9 mg/L.\(^{12}\)

Our case report presents typical features of acute barium poisoning. The benign outcome can be attributed to prompt and massive potassium supplementation. Acute barium poisonings are medical emergencies. Severe hypokalemia that occurs within 2 hours post-ingestion is the predominating feature. It is unclear whether mechanisms other than hypokalemia contribute to the neuromuscular and cardiovascular toxic effects of soluble barium compounds. In most cases, recovery is achieved with normalization of hypokalemia. It is recommended to measure serum phosphate levels when muscular weakness does persist despite normalized hypokalemia.

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References


