Case Report

Acute barium intoxication following ingestion of soap water solution

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Abstract

We present a rare case in which a young girl ingested a solution of a hair-removing soap. The ingestion resulted in profound hypokalemia and severe acidosis leading to flaccid paralysis, respiratory arrest and ventricular arrhythmias. Ultimately the patient made complete recovery.The soapwas found to contain barium sulfide.The degree of paralysis and acidosis appeared to be directly related to serum potassium levels.

Key words: Barium, hypokalemia, poisoning

Access this article online

Website: www.ijccm.org

DOI: 10.4103/0972-5229.106513

Quick Response Code:

Case Report

A 24-year-old female was brought to the casualty department of our hospital with an alleged history of ingestion of a soapy solution 4 hours prior to admission. Her chief complaints were a sudden onset of severe cramping abdominal pain, vomiting and diarrhea. There was no previous history of influenza like illness, diarrhea or alcoholism.Her family history was noncontributory.

On examination, she was conscious and oriented with a blood pressure of 110/70 mmHg, a pulse rate of 92 beats/minute and respiratory rate of 16/minute. There were no external injuries.

As the chemical composition of the soap was not mentioned on the label, a prompt gastric lavage was done and the contents preserved for chemical analysis. The patient was treated symptomatically and vitals were monitoredcontinuously. On admission, her blood sugar was 100 mg/dl and her ABG was normal.

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One hour after admission she started complaining of increased difficulty in breathing and there was progressive deterioration of her level of consciousness. On examination by the ICU team in the casuality her GCS was 7 (E2 V1 M4), her pupils were dilated and sluggishly reacting. Her pulse rate was 100/minute, irregularly irregular and systolic blood pressure was 90 mmHg. Her respiratory rate was 24 breaths/minute with a paradoxical pattern of respiration. On auscultation of chest air entry was equalbilateraly. An ECG done in casualty revealed prolongation of PR interval, ST segment depression and T wave inversion in leads II, III, aVF and lateral chest leads with a normal rhythm.

The patient's trachea was intubated with a 7 mmI.D. endotracheal tube and ventilation started with 100% oxygen. The patient was then transferred to the ICU. In ICU she was put on ventilatory support with SIMV mode of ventilation and FIO_2 100%. Her ABG revealed severe metabolic acidosis with a pH of 6.97, PCO_2, 40.2 mmHg, HCO_3 8.4 and base excess of -25.2.

200 mEq of sodium bicarbonate infusion was started for correction of metabolic acidosis. Meanwhile, her ECG began showing frequent ventricular ectopy. Intravenous lidocaine 60 mg was given intravenously.

Laboratory examination revealed a serum potassium level of 1.6 mEq/l, serum bilurubin,AST and ALT, blood urea and serum creatinine were within normal range. Potassium supplementation was started immediately under strict cardiovascular monitoring and 80 mEq potassium was infused over the next 2 hours via central venous line. However, her motor paralysis progressed inspite of potassium correction at the rate of 40 mEq per hour.

When contacted,the manufacturer revealed that the soap contained barium sulfide.Saline diuresis was initiated with 0.9% saline and spironolactone 6 milligrams BD through ryel's tube. Magnesium levels were (1.7 mg/dl) intravenous magnesium supplementation 1 gm single dose was administered. Simultaneously, potassium correction was continued with 40 mEqof potassium per hour. An additional 280 mEqwas given in the first 24 hours and further 140 mEqgiven over the next 24 hours after which the potassium levels were 4 mEq/l.

Over the next 24 hours there was a dramatic improvement of her muscle power coincident with the rise in serum potassium levels and acidosis. She subsequently made a complete recovery with no residual neurological deficit.

Discussion

Barium sulfide is utilized as a constituent of various facial depilatory creams and hair-removing soaps. It is well known to physicians as a benign radio-opaque contrast agent and its insoluble sulfate salt has been utilizedfor X-ray diagnosis of colorectal and upper gastrointestinal conditions.[1]

Most of the cases reported in literature of acute toxicity due to barium are those due to ingestion of soluble salts of barium like barium carbonate (rodenticide) which allowsfor absorption of free barium ions.[2-6]

These free barium ions are severe irritants of the gastrointestinal tract and ingestion leads to excessive salivation, intractable vomiting, severe abdominal pain and diarrhea within 2-3 hours. Progressive toxicity causes muscle twitching, periorbital and extremity paraesthesias, weakness progressing to convulsions and flaccid paralysis, as well as life-threatening arrhythmias.[6]

The initial management of acute barium toxicity is induction of emesis and gastric lavage. Gastric lavage was done even 4 hours after ingestion as it is a protocol in our hospital to lavage all poisoning patients and to collect the samples for toxicological examination. The administration of activated charcoal has no role in barium toxicity as it does not bind barium and should not be used unless other agents are ingested or suspected. However, oral administration of magnesium sulfate or sodium sulfate leads to precipitation of ionic barium in gut to form insoluble barium sulfate and catharsis.[6] Saline diuresis has been recommended for management of barium poisoning.[3] We initiated a saline diuresis with intravenous normal saline 0.9% and utilized sprinolactone as a diuretic agent as it is a potassium-sparing diuretic to maintain a urine output of 200 ml/hr. Intravenous magnesium sulfate has also been used[6] as an antidote of Barium and also for correction of profound refractory hypokalemia.[7] This was our rationale for administration of magnesium though theoretically magnesium sulfate can precipitate barium in the kidneys leading to acute renal failure.

Aggressive respiratory assistance and bicarbonate administration are essentialto manage the life threatening respiratory paralysis and acidosis. Intravenous potassium administration reverses the hypokalemia as well as displaces barium from potassium channels, allowing it to be excreted in the urine.[4,9] Immediate administration of up to 400 m Eqof potassium evenly and over 24 hours, which might prove dangerous in other situations, is essential for rapid management of barium intoxication and for protection against fatal barium-induced arrhythmias.[4] Thomas *et al*.[5] and Phelan,[8] have emphasized on the value of early hemodialysis for the reduction in serum barium levels as they found a strong correlation between serum barium levels and the degree of weakness. However, we found a good correlation between the degree of paralysis and serum potassium levels. We could not do serum barium levels as facility to do this investigation was not available in our hospital.

Conclusions

Overdose of barium sulfide results in severe hypokalemia, acidosis and cardiac arrest. But respiratory assistance, aggressive intravenous potassium supplementation and saline diuresis may result in successful outcome.

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How to cite this article: Joshi N, Sharma CS, Sai, Sharma JP. Acute barium intoxication following ingestion of soap water solution. Indian J Crit Care Med 2012;16:238-40.

Source of Support: Nil, **Conflict of Interest:** None declared.