

Critical care management of patients with barium poisoning: a case series

Jun-Feng Su¹, Dao-Ping Le², Chun-He Liu³, Jian-Dong Lin¹, Xiong-Jian Xiao¹

¹Department of Critical Care Medicine, The First Affiliated Hospital of Fujian Medical University, Fuzhou, Fujian 350000, China;

²Department of Critical Care Medicine, Ningde Hospital Affiliated to Fujian Medical University, Ningde, Fujian 352000, China;

³Department of Critical Care Medicine, Gutian County Hospital of Fujian Province, Ningde, Fujian 352000, China.

Acute barium poisoning is uncommon in clinical practice. It occurs primarily due to the ingestion of soluble barium compounds such as BaCl₂ that causes gastroenteritis (vomiting, diarrhea, and abdominal pain), hypokalemia, hypertension, cardiac arrhythmia, and skeletal muscle paralysis. Recently, several related cases of barium poisoning due to food contamination occurred in the vicinity of our hospital. We present the data for these cases and describe a representative case that we managed.

On March 13, 2018, several patients presented with symptoms including abdominal cramps, vomiting, diarrhea, partial paralysis (grade 2 to 4 according to the Manual Muscle Testing Grading System) after eating KomPyang, a traditional, charcoal-baked scallion cake made with flour, onions, and meat or vegetable stuffing, at a private roadside booth in Ningde city, Fujian province, China. In the following 24 h, 48 patients with suspected food poisoning were admitted to the hospitals at county level and above, of which 41 were hospitalized. Seven patients were observed in outpatient clinics since they had mild gastrointestinal symptoms and recovered quickly. The Fujian Provincial Center for Disease Control and Prevention (CDC) collected data for these cases. Owing to limited equipment, we could not acquire data for barium concentrations in the urine or blood. The serum potassium level, vital signs, main clinical problems, and ingested dose were considered as the determinants of the severity of barium poisoning. Based on our clinical experience, we classified cases with a blood potassium level <2.5, 2.5 to 3.0, and 3.0 to 3.5 mmol/L as severe, moderate, and mild, respectively. Supplementary Table 1, <http://links.lww.com/CM9/A176> summarizes the data for the 21 cases treated at Ningde Hospital (57% [12/21] severe, 19% [4/21] moderate, and 24% [5/21] mild). According to the Registry of Toxic Effects of

Chemical Substances (1985), the lowest lethal acute oral dose of BaCl₂ is 11.4 mg/kg body weight, and a dose as low as 5.8 mg/kg body weight causes flaccid paralysis, paresthesia, and muscle weakness.^[1] For an ordinary 70 kg adult, consumption of food containing ≥0.798 g BaCl₂ would be devastating. In this case, the CDC found that all barium-poisoned patients had eaten KomPyang with a meat filling containing 40 g/kg BaCl₂. Since the approximate weight of meat filling in one KomPyang is 10 g, each KomPyang contained 0.4 g BaCl₂. The average BaCl₂ intake in 21 patients was 1.257 g, with a latent period of 3 to 6 h after exposure, and median time in the hospital was 5 days. In Case 20, the patient consumed four KomPyangs at once, and the BaCl₂ dose was 1.6 g, which far exceeded the lowest lethal dose. The severity of the poisoning was determined by the quantity of KomPyang eaten. There was a significant positive correlation between the duration of hospitalization and dosage ($P < 0.05$). Although barium crosses the placenta,^[2] it may not be toxic to the human fetus. The pregnant patient in our study [Supplementary Table 1, <http://links.lww.com/CM9/A176>] delivered a healthy baby months after acute barium poisoning.

A 38-year-old man had a sudden attack of abdominal cramps and diarrhea 2 h after eating four KomPyangs. His symptoms included nausea, vomiting, stomach burning, dizziness, and diarrhea followed by heaviness of the limbs and weakness. Owing to life-threatening hypokalemia (1.4 mmol/L potassium), he received symptomatic treatment (continuous oxygen inhalation, intravenous [IV] potassium supplementation, and rehydration). Subsequently, he was transferred to the intensive care unit (ICU) for further treatment. On examination, he was conscious but completely paralyzed. He had a soft neck, no muscle strength (grade 0 according to the Manual Muscle

Access this article online

Quick Response Code:



Website:
www.cmj.org

DOI:
10.1097/CM9.0000000000000672

Correspondence to: Dr. Xiong-Jian Xiao, Department of Critical Care Medicine, The First Affiliated Hospital of Fujian Medical University, Fuzhou, Fujian 350000, China
E-Mail: xxj1495@163.com

Copyright © 2020 The Chinese Medical Association, produced by Wolters Kluwer, Inc. under the CC-BY-NC-ND license. This is an open access article distributed under the terms of the Creative Commons Attribution-Non Commercial-No Derivatives License 4.0 (CCBY-NC-ND), where it is permissible to download and share the work provided it is properly cited. The work cannot be changed in any way or used commercially without permission from the journal.

Chinese Medical Journal 2020;133(6)

Received: 4-11-2019 Edited by: Li-Min Chen.

Testing Grading System), insufficient respiration, and diminished neural reflexes with excessive salivation and perspiration. Potassium chloride (IV; 10% KCl) at 15.0 mmol/h coupled with magnesium sulfate (25 g in 250 mL of 5% glucose saline) was administered to precipitate the Ba^{2+} . Approximately 2 h later, the patient became unconscious with shortness of breath, nodding, cyanosis, and profuse facial sweating. Because muscle paralysis leads to respiratory failure, emergency tracheal intubation was performed, and ventilator-assisted breathing was initiated. Subsequently, the potassium level was 1.8 mmol/L, bicarbonate (HCO_3^-) level was 16.7 mmol/L, and pH was 7.24. In addition to KCl and magnesium sulfate, sodium bicarbonate (125 mL) was administered to correct acidosis. The patient soon regained consciousness and sinus rhythm (95 beats/min), but he could barely nod. In the following hours, the amount and rate of potassium infusion were continuously adjusted according to the urine volume and serum potassium concentration results. About 8 h after ICU admission, the potassium level was normal (5.4 mmol/L), and potassium supplementation was stopped. Rehydration was continued. Two hours later, we disengaged the ventilator and removed the tube. The patient made a miraculous functional recovery without any complications and was discharged after another 3 days. Inadvertent consumption of barium-contaminated food is the leading cause of barium poisoning, and it may occur when $BaCl_2$ is mistakenly used instead of salt, flour, or baking powder. In this case series, the chef unknowingly used $BaCl_2$ instead of potato flour.

According to the US CDC, a suspicious dietary history and profound hypokalemia associated with generalized muscle weakness are key indicators of barium poisoning. The clinical manifestations of barium poisoning include gastrointestinal symptoms and hypokalemia which may progress to cardiac arrhythmia, skeletal muscle weakness, and respiratory muscle paralysis caused by the deleterious effects of barium on the potassium channels and sodium-potassium pump.^[3] Moreover, hypokalemia is caused by frequent vomiting, severe diarrhea, and overuse of diuretics. As shown in Supplementary Table 1, <http://links.lww.com/CM9/A176>, every patient with moderate or severe poisoning was hypokalemic to some extent. Additionally, the loss of potassium leads to T-wave morphology, ST-segment depression, and U waves (ST-T-U) changes on electrocardiography and arrhythmia.^[4] The laboratory diagnostic criteria consist of elevated urinary barium concentration or detection of barium compounds in environmental samples. Although we lacked the data of urinary or blood barium concentration, the detection of excess $BaCl_2$ in KomPyang clarified the diagnosis.

Attention to vital signs and blood biochemistry is the first step in the successful rescue of barium-poisoned patients. For barium ions in the intestines, precipitation by oral administration of sodium sulfate, sodium thiosulfate, or magnesium sulfate is effective. Magnesium ions maintain normal intracellular potassium levels and promote potassium retention in potassium-deficient conditions. Therefore, for patients with hypokalemia, oral magnesium supplementation is recommended (250 mg/kg for children and 300 mg/kg for adults). Although precipitation of barium in blood vessels may theoretically lead to renal failure, there were no abnormalities in creatinine and urea nitrogen or renal failure signs in our cases. Potassium infusion is used clinically to reverse the toxic effects of barium. The highest rate of KCl supplementation in the representative case was 20.0 mmol/h, with a total dose of about 160 mmol (11.2 g) in 8 h. It was remarkable that barium-poisoned people tolerate potassium supplementation and recover quickly upon potassium restoration. Blood chemistry testing should be performed hourly, and attention to changes in electrocardiograms and urine volume will help to prevent rebound hyperkalemia when direct channel block is relieved followed by a backward shift of cellular potassium.^[3] Moreover, comprehensive treatments that protect the myocardium, maintain homeostasis, mechanically assist ventilation, and combat arrhythmia are essential for rescuing barium-poisoned patients.

The collective cases described in this report highlight the timely and appropriate management of barium-poisoned patients at the Ningde Hospital. Multifaceted treatments that rapidly increase the level of serum potassium ensure the successful rescue of barium-poisoned patients and prevent death.

Conflicts of interest

None.

References

1. Harlal C, Richard C. Barium and Barium Compounds. Geneva: World Health Organization; 2001.
2. Schroeder HA, Tipton IH, Nason AP. Trace metals in man: strontium and barium. *J Chronic Dis* 1972;25:491–517. doi: 10.1016/0021-9681(72)90150-6.
3. Bhoelan BS, Stevering CH, van der Boog AT, van der Heyden MA. Barium toxicity and the role of the potassium inward rectifier current. *Clin Toxicol (Phila)* 2014;52:584–593. doi: 10.3109/15563650.2014.923903.
4. Wang X, Han D, Li G. Electrocardiographic manifestations in severe hypokalemia. *J Int Med Res* 2018;0:1–7. doi: 10.1177/0300060518811058.

How to cite this article: Su JF, Le DP, Liu CH, Lin JD, Xiao XJ. Critical care management of patients with barium poisoning: a case series. *Clin Med J* 2020;133:724–725. doi: 10.1097/CM9.0000000000000672