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An Industrial Worker Hospitalized With Paralysis After an Aerosolized Chemical Exposure

CLINICAL PRESENTATION

A 54-year-old healthy male factory worker was brought to the emergency department reporting abdominal pain, nausea, vomiting, diarrhea, lethargy, and generalized muscle weakness 2 hours after accidental exposure to an aerosolized chemical. On arrival, he was breathing spontaneously and blood pressure was 121/78 mm Hg, pulse rate was 72 beats/min, and respiratory rate was 18 breaths/min. Physical examination was notable for alert consciousness, hyperactive bowel sounds, and symmetrical decreased muscle tone in all 4 limbs. Laboratory test results are listed in Table 1. Chest x-ray (Fig 1) and electrocardiogram (Fig 2) were obtained. The patient received 200 mEq of intravenous potassium chloride, but became drowsy 6 hours after arrival and respiratory failure ensued, requiring endotracheal intubation and mechanical ventilation.

Table 1. Laboratory Studies

Parameter	Value
Serum studies	
Sodium (mEq/L)	142
Potassium (mEq/L)	1.6
Chloride (mEq/L)	111
Bicarbonate (mEq/L)	17.4
Anion gap (mEq/L)	13
Serum urea nitrogen (mg/dL)	12
Creatinine (mg/dL)	1.52
eGFR (mL/min/1.73 m^2)	48
Calcium (mg/dL)	10.1
Phosphate (mg/dL)	2.4
WBC count[a] ($\times 10^3/\mu L$)	18
Hemoglobin (g/dL)	18.4
Arterial blood gas analysis[b]	
pH	7.308
$Paco_2$ (mm Hg)	35.5
Po_2 (mm Hg)	77.4

Note: Conversion factors for units: serum urea nitrogen in mg/dL to mmol/L, $\times 0.357$; creatinine in mg/dL to μmol/L, $\times 88.4$; eGFR in mL/min/1.73 m^2 to mL/s/1.73 m^2, $\times 0.01667$; calcium in mg/dL to mmol/L, $\times 0.2495$; phosphorus in mg/dL to mmol/L, $\times 0.3229$; hemoglobin in g/dL to g/L, $\times 10$. No conversion necessary for sodium, potassium, chloride, bicarbonate, and anion gap in mEq/L and mmol/L or WBC count in $10^3/\mu L$ and $10^9/L$.

Abbreviations: eGFR, estimated glomerular filtration rate; $Paco_2$, partial pressure of carbon dioxide, arterial; Po_2, partial pressure of oxygen; WBC, white blood cell.

[a]With 92.5% neutrophils.

[b]Room air.

- **What is the cause of the patient's profound hypokalemia and how can the diagnosis be confirmed?**
- **How can this condition be managed?**
- **What other complications can be seen with this exposure?**

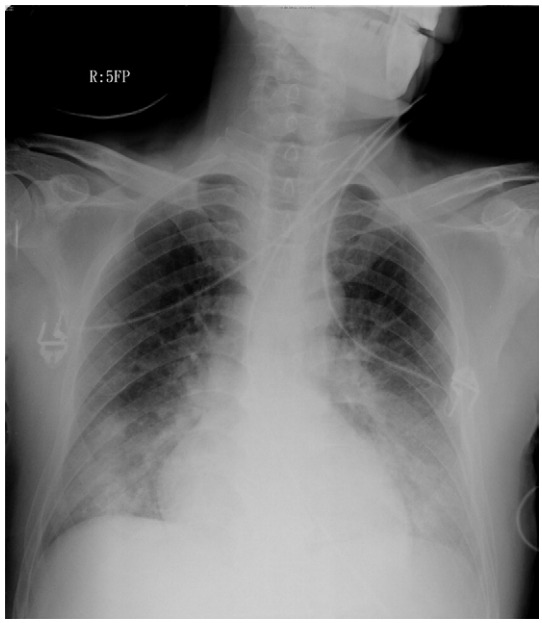

Figure 1. Chest x-ray showed marked patchy bilateral lung infiltrates.

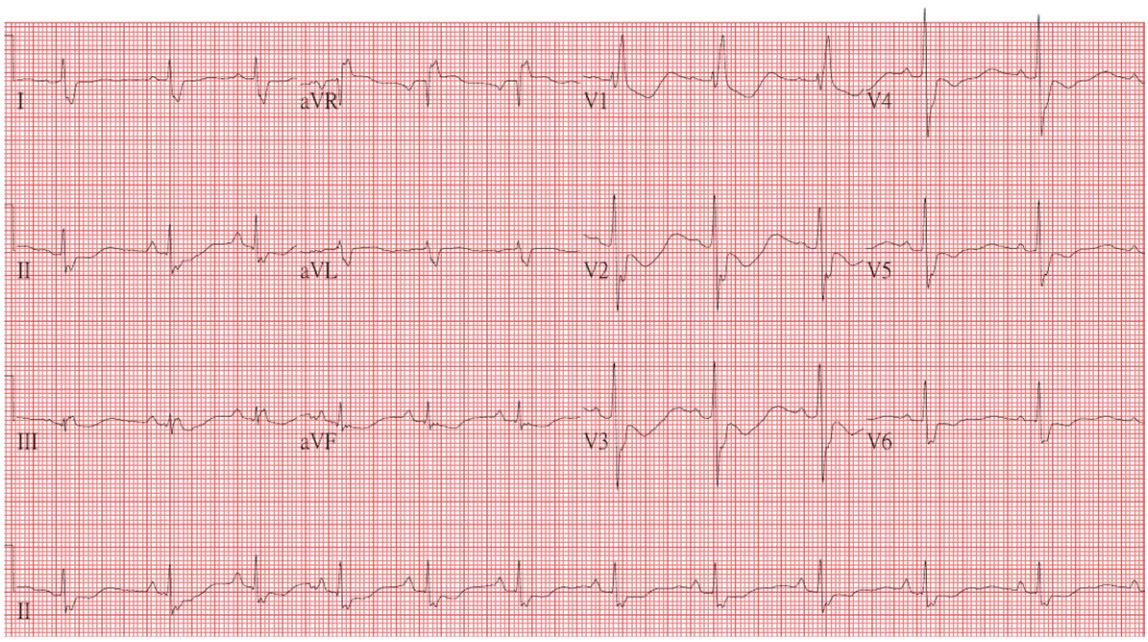

Figure 2. The electrocardiogram showed U waves and right bundle branch block.

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QUIZ PAGE SEPTEMBER 2010 ANSWERS

DISCUSSION

What is the cause of the patient's profound hypokalemia and how can the diagnosis be confirmed?

Hypokalemia frequently is encountered in clinical practice and can result from inadequate potassium intake, cellular redistribution into the cells, or, most commonly, excessive losses in the gastrointestinal tract or urine. Our patient experienced life-threatening hypokalemic paralysis and acute respiratory failure after accidental inhalation of an aerosolized chemical.

Accidental inhalation of soluble barium chloride used in manufacturing can result in profound hypokalemia, caused by inactivation of the passive potassium channels in the muscle cell membrane, thereby blocking efflux of intracellular potassium. Because activity of the adenosine triphosphatase sodium-potassium pump (Na^+-K^+-ATPase) is unchanged and continues to exchange sodium and potassium, potassium thus is trapped intracellularly.[1] In addition to barium intoxication, other chemical exposures that affect serum potassium levels include cesium, lead, cadmium, mercury, and copper. In our patient, serum and urine barium levels were 10,555.9 and 5,979 μg/L, respectively.

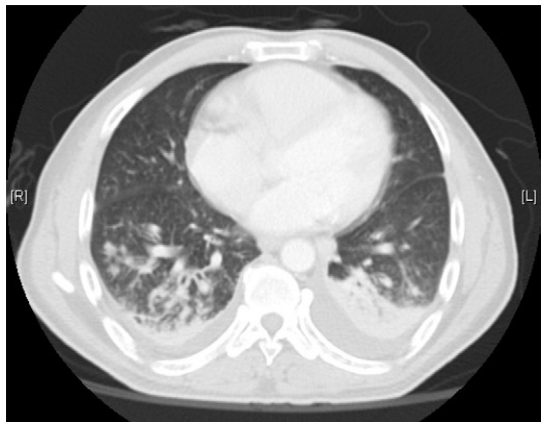

Figure 4. High-resolution computed tomography showed reticulonodular infiltration in bilateral lower lungs with consolidation, located mostly at the posterior segments of both lung lobes with prominent left-sided pleural effusion.

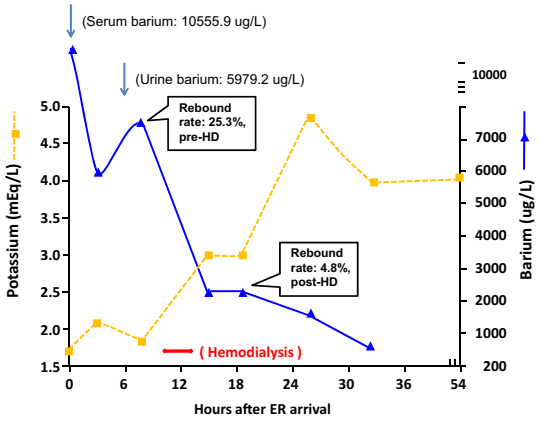

Figure 3. A series of changes in serum potassium and barium levels during hospitalization (barium reference range: serum <200 μg/L; urine <3.85 μg/L, measured by inductively coupled plasma mass spectrometry). No units of conversion are necessary for serum potassium in mEq/L and mmol/L. Abbreviations: ER, emergency department; HD, hemodialysis.

How can this condition be managed?

Early recognition of the symptoms and signs of barium intoxication and a detailed history of occupational exposure allowed us to offer prompt treatments. The typical clinical manifestation is gastrointestinal hypermotility, rapidly followed by areflexia, flaccid paralysis, and respiratory failure.[2] Laboratory findings include severe hypokalemia.

In an attempt to accelerate the removal of barium from the bloodstream, emergent hemodialysis was initiated.[3] Rapid correction of potassium levels and reestablishment of transmembrane potentials stabilized our patient, while the barium was readily dialyzed because of its molecular weight of 137.3 g/mol. Pharmacokinetic analysis of serum barium showed that hemodialysis short-

ened the serum half-life of barium and decreased the degree of barium rebound from 25.3% to 4.8%. This highlights the role of hemodialysis in barium removal (Fig 3).

What other complications can be seen with this exposure?

Acute pneumonitis induced by barium chloride inhalation is very rare and distinctive and differs from oral barium salt intoxication.[4] Our patient's case also was complicated by hypoxia and acute chemical pneumonitis diagnosed by examination, arterial blood gas analysis, and lung image studies (Fig 4). Pulmonary function tests showed mild restrictive ventilatory impairment. The abnormal chest x-ray and pulmonary function test result resolved completely within 2 weeks.

Acute kidney injury has been observed in an animal model of barium intoxication and possibly is related to kidney hemodynamics, regional hypoxia caused by vasoconstriction of glomeruli, and direct toxic injury to the tubulointerstitium.[5]

This case highlights the role of emergent hemodialysis in acute barium intoxication.

FINAL DIAGNOSIS

Hypokalemia secondary to acute inhaled barium chloride exposure.

ACKNOWLEDGEMENTS

The authors thank Wen-Huei Lee, MD, and Yu-Che Tsai, MD, for helpful advice.

REFERENCES

1. Roza O, Berman LB: The pathophysiology of barium: hypokalemic and cardiovascular effects. *J Pharmacol Exp Ther.* 1971;177(2):433-439.

2. Ellenhorn MJ, ed. *Ellenhorns Medical Toxicology: Diagnosis and Treatment of Human Poisoning.* 2nd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 1997.

3. Schorn TF, Olbricht C, Schüler A, et al. Barium carbonate intoxication. *Intensive Care Med.* 1991;17(1):60-62.

4. International Programme on Chemical Safety, Barium and Barium Compounds (CICADS 33, 2001). http://www.inchem.org/documents/cicads/cicads/cicad33.htm. Accessed August 31, 2009.

5. National Toxicology Program (NTP), Public Health Service, US Department of Health and Human Services. Toxicology and carcinogenesis studies of barium chloride dihydrate (CAS no. 10326-27-9) in F344/N rats and B6C3F1 mice (drinking water studies). NTP TR 432, 1994. http://ntp.niehs.nih.gov/ntp/htdocs/LT_rpts/tr432.pdf. Accessed August 31, 2009.

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doi:10.1053/j.ajkd.2010.02.004

SUPPORT: None.

FINANCIAL DISCLOSURE: The authors declare that they have no relevant financial interests.