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Case Report

Acute self-poisoning by ingestion of cadmium and barium

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Introduction

Acute poisoning as a result of exposure to cadmium has increased in industrialized countries. The salts of cadmium are used as pigments and stabilizers in plastics. Of the many reported cases of acute cadmium poisoning [1–6], most resulted from inhalation exposures, and the diagnoses were based on patients' histories and symptoms. Deliberate self-poisoning with oral cadmium is rare, and information regarding urine and blood cadmium concentrations is limited. The value of urine and blood cadmium concentrations as indicators of body burden or recent exposure remains uncertain. Previous case reports have shown even normal urinary cadmium concentrations at the time of diagnosis [2,4]. Some reports showed contradictory results—that there may be temporary elevations in urinary cadmium concentrations [3,6], but how long these elevations persist remains unclear.

Profuse diarrhoea, hypokalaemia and a history of use are clinical features, which may point to barium intoxication. To the best of our knowledge, acute self-poisoning with a combination of cadmium and barium has not been reported before. We report a case of acute cadmium and barium poisoning in a patient who presented with profuse diarrhoea, hypokalaemia, fever and thrombocytopenia after deliberately ingesting an industrial product containing cadmium and barium salts. Blood and urine cadmium concentrations were measured consecutively after exposure.

Case

A 42-year-old man presented to our emergency room with marked lethargy and fever. His vital signs were: blood pressure 129/72 mmHg, heart rate 87/min, respiratory rate 16/min and temperature 38°C. Physical examination was unremarkable, except for diminished deep tendon reflexes of the lower limbs. There were no focal neurological signs. Initial laboratory findings included negative serum cholinesterase and urine paraquat levels. Serum chemistry results were: sodium 142 mEq/l, potassium 2.2 mEq/l, chloride 112 mEq/l, BUN 11 mg/dl, creatinine 0.8 mg/dl, calcium 9.5 mg/dl and glucose 144 mg/dl. Serum CPK was 222 U/l, with the MB type 11 U/l; room air arterial blood had a pH of 7.35, HCO_3 19.4 mEq/l, pCO_2 35.5 mmHg, and pO_2 105 mmHg. His haemogram revealed leukocytosis (WBC 15 370/mm^3). A 12-lead electrocardiogram showed normal sinus rhythm at 87 beats/min and pathological u waves. Chest and abdominal X-rays were negative; a brain CT scan was unremarkable. Further diagnostic work-up yielded a negative serum acetaminophen and multiple negative blood cultures.

On further inquiry his wife stated that the patient had been depressed recently; and in the plastic factory where he worked he received some sedatives to relieve tension. In addition, he had taken ~50 cc of an industrial chemical solution at 1 am, 1 h prior to his admission. About 20 min after ingesting it, he began to have nausea, vomiting, abdominal pain and profuse diarrhoea, and his family noted the gradual onset of drowsiness ~20 min prior to his admission.

Owing to hypokalaemia and muscle weakness, barium intoxication was strongly suspected initially; in addition, the emergency room physicians were told that the industrial chemical solution he had taken contained a chromium salt. Intravenous half-normal saline, at a rate of 120–150 ml/h, and potassium chloride, 40 mg every 8 h, were administered, and samples of his blood and urine were subsequently sent to another laboratory for analysis of heavy metal levels. The patient was transferred to the intensive care unit for further management. Through further contact with the staff of his workplace, the industrial chemicals were found to contain 2–15% of cadmium and barium stearate. We then arranged for further tests on blood and urine samples taken previously. The blood cadmium concentration on day 1 was 24.9 ng/ml (normal range 0.2–6.0 ng/ml) and serum barium

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Table 1. The patient's consecutive blood and urine cadmium concentrations

Day	Blood cadmium (ng/ml)	Urine cadmium (ng/ml)
1	24.9	16.0
2	15.1	8.4
3	7.4	5.6
4	3.3	0.4
7	0.2	0.2

Normal blood and urine cadmium concentration: 0.2–6.0 ng/ml.

concentration on day 1 was 34.1 μg/dl (normal range 3.0–20.0 μg/dl).

After being stabilized on the second day, he was transferred to the medical ward. Diarrhoea, vomiting and abdominal pain subsided, but weakness and myalgias persisted. On hospital day 3, laboratory studies showed serum chloride 111 mEq/l, sodium 144 mEq/l, potassium 4.2 mEq/l and thrombocytopenia (platelet count of 77 000/mm^3). On day 7, his platelet count returned to the normal range. Consecutive blood and urine cadmium concentrations were measured. Blood cadmium concentrations on days 2, 3, 4 and 7 post-exposure were 15.1, 7.4, 3.3 and 0.2 ng/ml, respectively, and urine cadmium concentrations 8.4, 5.6, 0.4 and 0.2 ng/ml.

Discussion

Cadmium is a severe pulmonary and gastrointestinal irritant, which can be fatal if inhaled or ingested. Acute cadmium poisoning causes gastrointestinal tract erosion, pulmonary, hepatic or renal injury and coma, depending on the route of poisoning [7,8]. After acute ingestion, symptoms usually appear in 15–30 min. These include: abdominal pain, burning sensation, nausea, vomiting, salivation, muscle cramps, vertigo, shock, loss of consciousness and convulsions. Our case had vomiting, diarrhoea, anorexia and drowsiness after ingesting cadmium-containing chemicals. Acute ingestion of as little as 10 mg of inorganic cadmium has caused severe symptoms. Ingestion of >100 mg of its soluble salts can be lethal [7]. Our patient was estimated to have taken ~1 g of inorganic cadmium, which is a lethal dose. Given the clinical course and the elevated blood and urine cadmium levels, it is all but certain that this patient's symptoms were related to acute cadmium poisoning.

Our case is unique due to the combination of cadmium and barium. We are unsure how much cadmium and barium our patient took, but a blood level of 24.9 mEq/l is a marked elevation, and is consistent with excessive cadmium salt ingestion. Blood cadmium levels are a reflection of acute cadmium exposure; urine levels appear to provide a better measure of chronic exposure [9]. Serum cadmium levels will help confirm the diagnosis, but do not correlate with the severity of intoxication. Many of our patient's symptoms could also be ascribed to barium poisoning (gastro-enteritis and paralysis). Physicians are familiar with barium in its inert and insoluble form as a contrast medium, barium sulfate. While barium sulfate is not absorbed systemically, other water-soluble barium salts (carbonate, chloride, sulfide and nitrate) can be absorbed and cause barium poisoning [10]. Barium stearate, the presumed culprit in our case, is a water-soluble barium salt and can cause acute poisoning. The serum barium concentration of 34.1 μg/day could have contributed to our patient's clinical manifestation; however, serum barium assays are not routinely available in hospital laboratories, and do not contribute to the management of this poisoning.

Because cadmium has a high affinity for the metal-carrying protein metallothionein, the chelation of cadmium is very difficult. Risk–benefit analysis suggests that chelation therapy should not be performed; therefore, all treatment is supportive. Hydration and the correction of hypokalaemia are the main therapies [9].

In conclusion, cadmium and barium intoxication in our patient was suspected initially, based on the symptoms and hypokalaemia, and then confirmed by blood and urine tests and a detailed medical history. We therefore suggest considering cadmium and barium intoxication in the differential diagnosis of any patient with profuse diarrhoea, vomiting and hypokalaemia. The clinician should be diligent in eliciting a drug intake history to diagnose early a potentially fatal poisoning.

Conflict of interest statement. None declared.

References

1. Barnhart S, Rosenstock L. Cadmium chemical pneumonitis. *Chest* 1984; 86: 789–791
2. Beton DC, Andrew GS, Davies HJ, Howells L, Smith GF. Acute cadmium fume poisoning: fives cases with one death from renal necrosis. *Br J Ind Med* 1966; 23: 292–301
3. Lucas PA, Jariwalla AG, Jones JH, Vale PT. Fatal cadmium fume inhalation. *Lancet* 1980; 2: 205
4. Yates DH, Goldman KP. Acute cadmium poisoning in a foreman plater welder. *Br J Ind Med* 1990; 47: 429–431
5. Seidal K, Jorgensen N, Elinder C-G, Sjpgren B, Vahter M. Fatal cadmium-induced penumonitis. *Scand J Work Envrion Health* 1993; 19: 429–431
6. Blejer HP, Caplan PE, Alcocer AE. Acute cadmium fume poisoning—a fatal and a nonfatal case in California. *Calif Med* 1966; 105: 290–296
7. Baselt RC, Cravey RH. *Disposition of Toxic Drugs and Chemicals in Man*, 4th Edn. Year Book Medical Publishers, Chicago, IL, 1995; 105–107
8. Baselt RC. *Disposition of Toxic Drugs and Chemicals in Man*, 5th Edn. Chemical Toxiology Institute, Foster City, CA, 2000
9. Shannon MW. *The Toxicology of Other Metals. Clinical Management of Poisoning and Drug Overdose*, 3rd Edn. W. B. Saunders Company, Philadelphia, PA, 1998; 856–863
10. Jacobs IA, Taddeo J, Kelly K, Valenziano C. Poisoning as a result of barium styphnate explosion. *Am J Ind Med* 2002; 41: 285–288

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