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CASE REPORT

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Hypoxia, hypotension, and bradycardia induced by povidone-iodine ingestion: A pediatric case report and literature review

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Abstract

Povidone-iodine (PI) is a disinfectant and antiseptic agent commonly used to sterilize skin, mucous membranes, and wounds. PI intoxication is very rare, but the complications may be severe. We report a pediatric patient who presented in the emergency department with hypoxia, hypotension, and bradycardia after unintentional PI ingestion.

1 | CASE PRESENTATION

An otherwise healthy 17-year-old boy drank a dark-brown colored liquid in a plastic coffee bottle from the refrigerator when he woke up at 4:00 am, believing the liquid to be coffee (Figure 1). He developed a headache and vomited extensively once about 4:30 am; he was immediately sent to the emergency department (ED) of a local hospital. His grandmother stated that povidone-iodine (PI) was stored in the beverage bottle.

Upon ED arrival at 5:00 am, the patient had a temperature of 36.6°C (normal 35.9–37.6°C), a pulse of 40 beats/min (normal 60–100 beats/min), and a respiratory rate of 20 breaths/min (normal 12–18 breaths/min). His blood pressure was too low to be measured (normal: 100/70–120/80 mmHg) and his oxygen saturation (OSAT) was 91.6% (normal 95%–100%).

Dyspnea commenced at about 7:00 am. Arterial blood gas (ABG) analyses revealed respiratory acidosis with a pH of 7.303, a pCO_2 of 51.2 mmHg, a pO_2 of 70.1 mmHg, a base excess of −2.2, and an HCO_3 level of 24.8 mmol/L. A chest X-ray revealed bilateral pulmonary edema (Figure 2A). Emergency endotracheal tube (ETT) intubation was performed, and furosemide (20 mg) was administrated intravenously 4 times daily for 3 days to treat the edema. An electrocardiogram (EKG) revealed a second-degree Mobitz type I atrioventricular block. Two doses of atropine (each 1 mg) were given intravenously to treat the

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FIGURE 1 The plastic coffee bottle used for storing povidone-iodine

bradycardia and a normal saline bolus (20 mL/kg) to treat the hypotension. The white blood cell (WBC) count was 12,010/μL, the platelet count was 438,000/μL, the glucose level was 228 mg/dL, and the serum level of sodium was 134 mEq/L. Brain computed tomography revealed no intracranial lesions. He was transferred to our pediatric intensive care unit (PICU) for further management at 11:00 am.

On the first hospital day in the PICU, aspiration pneumonia with fever developed. Amoxicillin/clavulanate (1.2 g/dose) was given intravenously 4 times on that day, but the fever persisted. The antibiotics were switched to piperacillin-tazobactam (4.5 g intravenously 4 times a day for 4 days); the fever gradually subsided. A thyroid function test was performed on the first hospital day to identify probable complications of a thyroid storm. The levels of free T4, T3, thyroid-stimulating hormone (TSH) were normal; there were no symptoms of hyperthyroidism (tachycardia or hypertension). Tests for other toxins including 3,4-methylenedioxy-methamphetamine (MDMA), ketamine, amphetamine, morphine, and benzodiazepines were negative. A chemical burn of the gastrointestinal tract was diagnosed on the third hospital day after panendoscopy revealed esophageal edema and gastric ulcers with oozing; esomeprazole (40 mg) was given intravenously every 12 h for 7 days.

No new symptoms had developed on the fourth hospital day and extubation was performed. The Glasgow Coma Scale indicated recovery; his temperature was 36.9°C, his pulse was 76 beats/min, his respiratory rate was 18 breaths/min, blood pressure was 110/45 mmHg, and the OSAT was 98%. The patient was transferred out of the PICU on the fifth hospital day, given his improved pulmonary condition (Figure 2B) and good oral intake, and was discharged on the ninth hospital day.

2 | DISCUSSION

PI is a common surface disinfectant and antiseptic agent;[1-3] intoxication after ingestion can result in cardiovascular collapse, nausea, vomiting, acute renal failure, initial hyperthyroidism, metabolic acidosis, fever, liver function impairment, and iodine-induced hemolysis.[2,4-9] Our case initially presented with hypoxia, hypotension, and bradycardia after PI ingestion. Other sequelae including aspiration pneumonitis and corrosive gastroenteritis developed in subsequent days.[7]

Iodine toxicity develops after absorption through the skin, ingestion, or inhalation.[10] The lethal dose varies from 200 mg to > 20 g

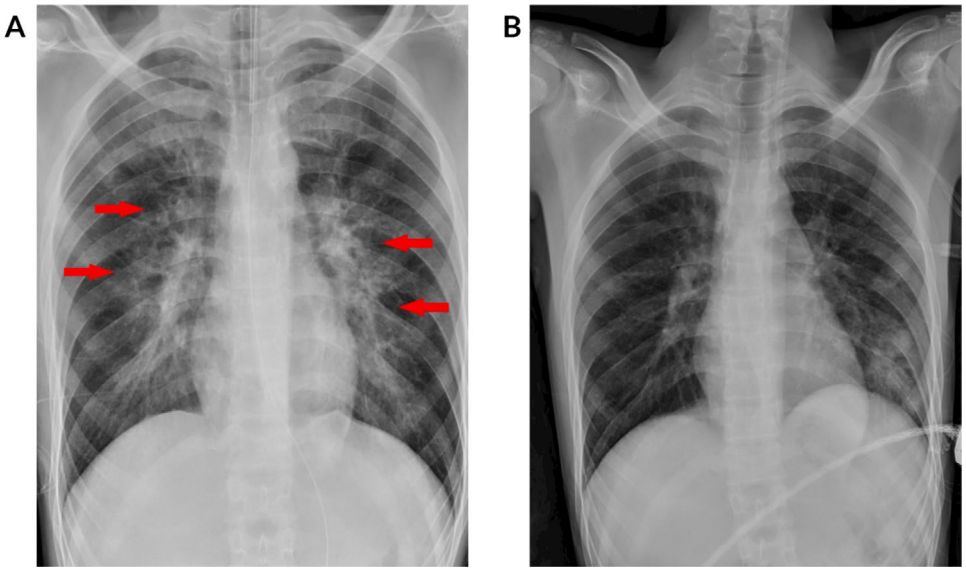

FIGURE 2 (A): Initial chest radiograph showing bilateral pulmonary edema. (arrows) (B): Chest radiologic finding at the fifth day after admission

(mean 2–4 g).[7,10] The extent of iodine absorption depends on the site and area of PI application; mucosal absorption is high, as is absorption through wounds.[11] The distribution volume is ≈ 23 L and the biological half-life is about 2 days.[11] After absorption, iodine is rapidly converted into the far less toxic iodide,[10,11] 97% of which is excreted by the kidneys with a half-life of 2 days.[11]

There are 4 reports of cardiovascular collapse with hypotension after iodine intoxication, of which 1 was pediatric.[5,6,8,9] In all cases, iodine was absorbed through mucosa or a wound during PI irrigation or topical treatment.[5,6,8,9] After PI ingestion, iodine can cause severe corrosive injury of the gastrointestinal tract; iodine is an oxidant.[10] Iodine is corrosive, causing chemical burns associated with esophageal edema and gastric ulcers with oozing.[10] Circulatory collapse after PI ingestion may be caused by the significant volume loss associated with corrosive gastroenteritis, vomiting, hematemesis, and/or diarrhea.[10] Vomiting may cause aspiration pneumonitis;[3] a previous animal study showed that PI aspiration can trigger lung injury or pulmonary fibrosis.[12] PI ingestion induces acute kidney injury.[2] Fortunately, our patient lacked such injury, perhaps because he spat out most of the PI.

A thyroid storm is a life-threatening clinical condition after iodine intoxication.[13] Our patient was closely monitored for signs of hyperthyroidism and altered thyroid function. Fortunately, no thyroid storm developed.[2–9]

When managing PI poisoning, supportive care is key.[10] The airway must be maintained; we performed endotracheal intubation to treat the progressive airway edema.[10] Fluid loss from the gastrointestinal tract must be aggressively treated to avoid possible sequelae.[10] Gastric decontamination is not recommended because of the corrosive effects of iodine.[10]

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