Acute Respiratory Distress Syndrome Caused by Mercury Inhalation: A Case Report

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Abstract

Keywords

- ▶ acute respiratory distress syndrome
- ▶ child
- ▶ inhalation injury
- ▶ mechanical ventilation
- ▶ mercury

Inhalation of mercury vapor is an uncommon cause of acute respiratory distress syndrome. It may go unnoticed if appropriate environmental/occupational history is not taken. A 2-year-old child presented with severe respiratory distress and progressed to develop acute respiratory distress syndrome, necessitating high-pressure mechanical ventilation. On detailed enquiry, history of exposure to mercury fumes was obtained (at home), during gold extraction by the father. Diagnosis of mercury poisoning was confirmed by blood levels, and despite treatment with dimercaprol (mercury chelator) and high-frequency ventilation, the child subsequently succumbed within 26 hours. This case reiterates the need for awareness regarding unusual environmental toxic exposures and need for stricter regulations for sale or use of toxic products.

Introduction

Acute respiratory distress syndrome (ARDS) is a known complication of mercury vapor inhalation, but rare. It was commonly reported in literature till early 1990s, but there has been a decline in such cases since past two decades. Furthermore, ARDS caused by elemental mercury has been reported in adults but sparingly in children.^{1–4}

We describe ARDS caused by inhalation of mercury vapor in a child, where elemental mercury was used to extract gold from the ore.

Case Report

A 2-year-old boy was brought to the emergency room with fever, cough, and breathlessness for 3 days. There was no other significant history. He was admitted earlier to a peripheral hospital for 2 days, where he was treated with oxygen, nebulizations, and intravenous antibiotics. On admission, he had tachycardia (heart rate of 160/minute), dyspnea (respiratory rate of 88/minute), and weak peripheral pulses. There were bilateral coarse crepitations in lungs and other systems

were normal. A chest radiograph revealed bilateral pulmonary infiltrates (▶ Fig. 1). In view of shock and impending respiratory failure, he was intubated and mechanically ventilated by using conventional ventilation. His hemoglobin level was 11.6 g/dL with leukocyte counts of 13,300/mm³ (polymorphic predominance—72%) and platelet counts of 3,00,000/mm³. The renal and liver functions were normal. Initial clinical impression was bronchopneumonia of infective etiology and he was treated with broad-spectrum antibiotics. However, due to persistent hypoxemia (PaO₂/FiO₂ ratio of < 300) and worsening chest radiography (despite high FiO₂ and higher pressure requirements [peak inspiratory pressure and positive end expiratory pressure]), he was put on high-frequency ventilation (▶ Fig. 2). On further enquiry, history of exposure to some chemical fumes (3 days back) was obtained after which all the family members had vomiting and diarrhea. The sample of the chemical (used for gold extraction) looked to be elemental mercury when examined. The father, a goldsmith, had used mercury for gold extraction for the first time at home (enclosed space of 23.22 m², air-conditioned). After sending samples for urine and blood mercury levels, first dose of dimercaprol (mercury chelator) at 5 mg/kg was

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Fig. 1 Chest radiograph (on admission). Note the bilateral pulmonary infiltrates.

given deep intramuscular 18.5 hours after admission, on clinical suspicion of mercury inhalation-related pulmonary injury. Urine and blood mercury levels were 13.92 $\mu\text{g/L}$ (normal $< 10 \mu\text{g/L}$) and 4.30 $\mu\text{g/dL}$ (normal $< 1 \mu\text{g/dL}$), respectively. However, the child succumbed to the illness after 26 hours of admission before the next doses of dimercaprol could be given. Postmortem histopathology of the lungs

Fig. 2 Chest radiograph (after 18 hours). Increase in bilateral pulmonary fluffy shadows suggestive of acute respiratory distress syndrome.

confirmed ARDS. Blood and urine levels of mercury in the father and sibling were normal.

Discussion

Mercury exists in three forms in the environment: elemental, inorganic, and organic mercury. Elemental mercury is volatile even at room temperature.⁵ When heated, its airborne concentration increases further due to its high density.⁶ Exposure to mercury vapor can be residential (accidental breakage of mercury containing devices such as thermometers), occupational (improper handling of mercury-containing materials, poor ventilation, and accidental spills), and cultural (folk remedies).⁵ Children are more susceptible than adults to toxicity due to such exposure, as mercury vapor being heavier than air tends to settle closer to the ground; increasing inhalation load and the minute ventilation per unit weight is more in them.⁵

Inhalational exposure to large doses of elemental mercury can cause fulminant pulmonary manifestations, as approximately 80% of elemental mercury is absorbed by lungs causing rapid cell damage, whereas there is minimal absorption by skin contact or ingestion.⁵ It may manifest as interstitial pneumonitis, necrotizing bronchiolitis, pulmonary edema, and even severe respiratory failure.⁵ Patients may present with wide range of manifestations including sudden cough, breathlessness, chest pain, fever, nausea, vomiting, diarrhea, salivation, conjunctivitis, colitis, gingivitis, stomatitis, and metallic taste.⁵

Earlier literature reports many cases of inhalational injury in children caused by mercury exposure (as early as 1948) often due to heating mercury on a stove at home.^{4,7,8} Gold being an expensive commodity, there have been instances of mercury poisoning from gold ore processing at home. Lien et al⁹ reported six patients including three children who were admitted due to respiratory symptoms subsequent to such an exposure; all survived. In addition, the baby delivered to a pregnant woman 26 days after the exposure was healthy and asymptomatic despite evidence of transfer of mercury through placenta.⁹ Soni et al¹⁰ have also described a 3-year-old child admitted with respiratory distress after similar exposure at home; the child survived, but the sibling died within 6 hours of exposure at home. Thus, an amateur attempt to retrieve gold can have disastrous consequences if the danger of such exposures is unknown.

In the United States, many areas have banned sale of mercury-containing devices and mercury thermometer exchange programs have been undertaken to get rid of this potential source.⁶ The problem has also been recognized in India, and attempts have been made to decrease use of mercury-containing devices at hospital level, but there is no active legislation. Despite a decline in reported exposures to mercury inhalation over the last few decades and clean-up by skilled personnel in attempts to decrease the impact of contamination (in countries such as the United States), newer cases are still being observed.^{1,6,11}

In our case, the father who was a goldsmith was unaware of the toxicity of mercury vapor and attempted unprotected extraction from gold ore at his home, due to which one child succumbed.

Present case emphasizes the need for public awareness and legislative regulations regarding sale and use of mercury-containing chemicals and devices. It also underscores the importance of detailed history for unusual occupational or environmental exposures in pediatric illnesses without an obvious etiology.

Key Learning Points

Inhalation of mercury is an uncommon cause of ARDS. Exposures can be residential, occupational, or cultural. Despite a decline in reports of such accidental exposures, mainly due to legislation in many countries and increasing public awareness, the threat still remains.

There is a need to increase awareness of toxicity caused by metal exposure among physicians. Pediatric history taking should routinely include history of environmental exposures (as it is often overlooked). Active education and participation of health personnel as well as patients is essential to gradually phase out mercury-containing instruments, so as to minimize such accidental exposures.

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