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Case Report

Acute Hypoxemic Respiratory Failure due to Mercury Inhalation in a Two-Year Old Female

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ABSTRACT

Introduction: Mercury is a toxic trace metal that causes acute respiratory failure when vaporized and inhaled. We report the presentation and management of a two-year-old female with hypoxemic respiratory failure secondary to elemental mercury inhalation.

Case report: A 2-year-old female presented to our facility with acute respiratory distress. She developed hypoxemic respiratory failure requiring venoarterial extracorporeal membrane oxygenation. She was found to have high serum mercury levels and was managed with dimercapto succinic acid chelation therapy and high-dose corticosteroids. The patient survived and subsequently recovered.

Discussion: This is the first pediatric case of fulminant pneumonitis caused by elemental mercury inhalation managed with venoarterial extracorporeal membrane oxygenation reported in the medical literature.

Keywords: Mercury; Pneumonitis; Hypoxia; VA-ECMO

INTRODUCTION

Mercury is a non-essential trace elemental metal found ubiquitously in the environment. Three forms exist-organic, inorganic, and elemental – all of which are toxic [1]. Despite this toxicity, it remains present in medical equipment, including dental amalgams, blood pressure cuffs, and thermometers, as well as batteries and fluorescent light bulbs. Clinical toxicologic manifestations are dependent upon the exposure form and dose [1,2]. Pediatric populations are especially vulnerable to mercury toxicity. Exposure and toxicity are often higher due to body weight, body surface area, rapid growth, and development. Pulmonary dysfunction is the main cause of pediatric mortality [3-8]. While multiple pediatric post-mortem case reports exist, there are scant guidelines regarding treatment for acute pulmonary failure secondary to elemental mercury toxicity. We present the unusual case of a 19-month-old female who presented with fulminant pneumonitis and respiratory failure secondary to elemental mercury inhalation.

CASE REPORT

A 19-month-old female with an unknown past medical history and who had recently immigrated from Guatemala presented to an outside emergency department with a 6-hour history of tactile fever and vomiting. The child's 3-year-old brother was also ill with similar symptoms. Communication was limited as the mother spoke the Mayan dialect Popti. The mother endorsed that the patient had been transported illegally into the United States *via* the Arizona-Mexico border two weeks prior to presentation. In triage, the child was noted to be cyanotic and lethargic with an oxygen saturation of 66%. Pertinent laboratory results included normal hemoglobin, white blood cell count 22.6 k/μL, normal platelet count, normal creatinine, normal sodium, potassium 4.8 mEq/L, bicarbonate 15 mEq/L, normal chloride, calculated anion gap 19, glucose 116 mg/dL, erythrocyte sedimentation rate 43 mm/h, and carboxyhemoglobin 0.7%. Urinalysis showed 3+protein and 2+ketones. Urine drug screen was negative. Initial room air capillary gas showed a pH 7.11, pCO_2 40 mmHg, and lactate 3.3 mmol/L. The child and her brother were transferred to our tertiary-care facility. Her chest x-ray revealed bilateral pneumothoraces and an extensive pneumomediastinum. Her blood gas normalized on high-flow nasal canula with 100% FiO_2. Other notable labs obtained on admission were procalcitonin 6.94 ng/mL, C-reactive protein 192 mg/L, and methemoglobin 0.5%. The patient's respiratory status soon

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decompensated, and she required emergent intubation and chest tube placement. Respiratory support was escalated to high frequency oscillatory ventilation (HFOV). Despite HFOV and maximal inhaled nitric oxide, she continued to have persistent hypoxemic respiratory failure. Due to her tenuous state, she was placed on venoarterial extracorporeal membrane oxygenation (VA-ECMO). Our infectious and toxicology differential was broad and included hantavirus, histoplasmosis, coccidioidosis, blastomycosis, and paraquat ingestion and other farm chemicals. Piperacillin-tazobactam, vancomycin, and fluconazole were empirically initiated. Inhalational mercury exposure was considered after a negative infectious disease evaluation and after screening all agricultural chemicals used at the family residence. Eleven days after being placed on VA-ECMO, her serum mercury resulted at 110 mcg/L (reference range<10 mcg/L). The decision was made to pursue chelation therapy with dimercaptosuccinic acid (DMSA). The patient received a total of 19 days of DMSA (5 days 85 mg TID, then 14 days 85 mg BID). The patient also received 3 days of methylprednisolone (1 mg/kg) 3 days after being placed on VA-ECMO and 3 days of pulse-dose methylprednisolone (255 mg) after decannulation. Due to worsening renal function, the patient was also started on continuous renal replacement therapy for five days. She was decannulated from the VA-ECMO circuit after 20 days and then extubated 20 days later to high flow nasal canula. CT chest was performed with no evidence of pulmonary fibrosis. After a prolonged recovery period, she was transferred to the general pediatric floor and ultimately to pediatric rehabilitation. She was discharged home with family approximately four months after admission. Unfortunately, she has since been lost to follow-up. State health and Environmental Protection Agency sampling of the family's residence found elevated mercury levels in air, bedding, and other items, but no source was identified. The sink drain had the highest measured air level at 50 mcg/m^3, suggesting elementary mercury was poured down the drain. There was a high index of suspicion for methamphetamine production in the home but this was never confirmed. The patient's 3-year-old brother was admitted for 2 days with respiratory distress and had a mercury level of 231 mcg/L. Her asymptomatic parents refused chelation, though her mother was 6 weeks pregnant and had a mercury level of 313 mcg/L. Her father's level was 90 mcg/L. An asymptomatic 2.5-year-old girl had a level of 131 mcg/L and two other adults in home had levels of 72 and 61 mcg/L, respectively. All three received DMSA.

DISCUSSION

Mercury is a non-essential elemental heavy metal found ubiquitously in the environment. All three forms, organic, inorganic, and elemental, are toxic, and can cause generalized and/or specific toxidromes [1]. Most mercury found in the environment is either elemental mercury or inorganic mercury [2].

In children, mercury toxicity generally results from exposure to food contaminated with organic mercury; however, other forms of exposure can occur. In aquatic environments, deposits of inorganic mercury are converted to organic mercury by various bacteria, fungi, and plankton. Biomagnification *via* the food chain results in amplification of organic mercury concentrations, with the most toxic levels occurring in larger fish and aquatic mammals that are eventually consumed by humans. Once consumed, approximately 90-100% of organic mercury is absorbed through the human gastrointestinal tract [1]. Elemental mercury is used commonly in a wide variety of industrial and household products, as well as in religious ceremonies (commonly Santeria, Voodoo, and Espiritismo) and in methamphetamine production [2,3]. It is a liquid at room temperature and easily vaporizes when heated, with toxicity occurring primarily from inhalation of these vapors [1,2]. More than 80% of inhaled elemental mercury is absorbed by the lungs, as the vapor readily diffuses across the alveoli to bind with red blood cells. Clinical manifestations of toxicity are dependent upon the form of exposure and the dose. A broad spectrum of symptoms, including fever, tremors, malaise, nausea, gingivitis, polyneuropathy, mental status changes, and acrodynia, often confound the diagnosis. Pediatric patients may develop severe muscle cramps, irritability, and dermal erythema. Pruritis, swelling, fever, tachycardia, hypertension, weakness, fever, insomnia, and excessive salivation or sweating have also been reported [2]. Acute exposures may cause central nervous system damage, respiratory distress, severe pneumonitis, and/or acute respiratory failure. Respiratory symptoms include cough, dyspnea, and chest tightness/burning. Chest x-rays often show diffuse infiltrates [1,2]. Airway obstruction, restriction, hyperinflation, and decreased vital capacity have been reported. In the most severe cases, pulmonary edema, lobar pneumonias, bronchiolar epithelium fibrosis, pneumothorax, respiratory failure, and death occur [3-9].

Mercury toxicity treatment is largely based upon chelation. There are no guidelines for chelation therapy, and the literature is limited to case reports [3-5,10]. Chelation therapy has been used for over 70 years, with British anti-Lewisite (BAL) developed in in wartime Britain. Unithiol (DMPS) and dimercaptosuccinic acid (DMSA), water-soluble analogs of BAL, were subsequently developed in the late 1950s. DMPS and DMSA have a higher therapeutic index and increase urinary metal excretion. The success of chelation is time-dependent, with an inverse relationship for the time interval between exposure and initiation of therapy and the therapy efficacy. DMSA is administered orally with rapid but variable absorption in the gastrointestinal tract. It should be noted that while chelation effectively removes renal mercury content, DMSA is inefficient at reducing mercury levels in the brain after elemental mercury inhalational exposure and does not mitigate neurological manifestations [2,10]. Corticosteroids have also been used in the treatment of acute mercury toxicity; however, their effect is variable [3-9]. While our patient did show improvement, the role of corticosteroids in her recovery is unclear as she also received chelation therapy and extensive respiratory support. To our knowledge, this adjunctive therapy has not been proven to show benefit. The exact source of our patient's mercury exposure is still unknown. Her exposure duration is also unknown, although in several adult case reports, exposure to mercury levels greater than 1-2 mg/m^3 for a few hours caused acute pneumonitis [4]. Pediatric case reports regarding elemental mercury toxicity have noted air samples

measuring as low as 0.193 mg/m^3 [6-8], and the CDC estimates the minimal risk level to be 0.0002 mg/m^3 [2]. Multiple reports exist regarding respiratory failure secondary to elemental mercury exposure [3-9]. Of these, only one pediatric case resulted in survival. We could not, however, find any cases of fulminant pneumonitis secondary to acute elemental mercury toxicity that required VA-ECMO.

CONCLUSION

In conclusion, it is important to consider elemental mercury toxicity when evaluating a patient with respiratory failure of unknown origin. This is the first known pediatric case of respiratory failure secondary to elemental mercury toxicity managed with VA-ECMO, chelation therapy, and corticosteroids. Given the rarity of inhaled mercury toxicity, it is difficult to propose this as a standard of care. We do not know what long-term sequelae our patient will have. However, given her survival, this approach should be considered in related cases.

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