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Acute Mercury Poisoning Revisited: Any Role for the Physician?

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Abstract

Mercury is a metal whose toxicity triggers neurological impairments at all ages, although the feti carry special risks. Mercury poisoning mostly occurs as a result of occupational accidents or attempted suicide. The bulk of evidence that addresses the health effects associated with long term postnatal mercury exposure is limited for all age groups. Public education on poisoning and the potential threats arising from mercury are of utmost importance for general well-being.

Keywords: Mercury poisoning; Intoxication; Management; Treatment; Elimination; Elemental mercury

What do we know about mercury and its poisoning?

Mercury is used in many industrial sectors to produce industrial chemicals, paints, explosives, batteries, thermometers, sphygmomanometers, electronic instruments, etc. It is also included in some drugs on the market such as Thiomersal, which is used to prevent contamination of vaccines.

Mercury is a metal with toxicity which triggers neurologic hazards at all ages, although the feti carry special risks [1]. Mercury poisoning mostly occurs as a result of occupational hazard or suicide attempt. In the last decades, researchers focused on environmental pollution as a source of mercury poisoning for the community as a whole. Interestingly Pirkle indicated that many northern American populations are deeply connected to the sea and rivers for food and medicine, while interestingly; they are often exposed to mercury via their diets, which incorporate wild foods from these ecosystems [2]. For example, some whales can accumulate considerably high concentrations of mercury (>1.0 g/g) [3].

Virtually all chemical forms of mercury are hazardous to human being. The entity varies in the absorption route, clinical presentation, and responses to therapy. Exposure to mercury is a threat to public health in either form, acute and/or chronic. Mercury poisoning is encountered following an occupational accident or attempted suicide. Mercury is silver-colored and liquid at room temperature. The element can be found in inorganic and organic forms. Neurotoxicity mostly stems from the soluble form of methyl mercury. Elemental (organic) mercury is especially hazardous for children since it is in liquid form and can easily be found [4].

Blood and hair represent the best media to use for measurements for the load of mercury in the body for evaluation of mercury ingestion in adults [2]. On the other hand, maternal blood and hair sampled during early pregnancy, and cord blood at delivery, are gold standards to evaluate prenatal exposure. In the last decade, researchers also demonstrated mercury thresholds of concern and appropriate clinical actions depending on the individual features [5].

The clinical effect of mercury poisoning changes in accord with the form and the route of the entry inside the body. The most severe impairments are noted in neurologic, gastrointestinal and renal systems, in accord with the route of exposure.

Elemental mercury attracts children with its bright gray appearance [6]. The compound rapidly distributes into body compartments and thus has a short half-life only two months.

Acute inhalations of mercury vapors can trigger pneumonia, respiratory distress, progressive pulmonary fibrosis and eventual death. Elemental (metallic) mercury can pass to systemic circulation via alveoli or directly through the skin. Nursing mothers can pass it directly to infants via breastfeeding [7].

All kinds of neurological findings can be seen in chronic mercury exposure. Some effects of high dose mercury inhalation are shown on (Table 1) [4,6]. Guidelines point out that *"if the elemental mercury was recently heated (e.g., from stove top, oven, furnace) in an enclosed area, all people within the exposure area should be evaluated at a healthcare facility due to the high risk of toxicity (Grade C)"* [4].

Central nervous system	Weakness, unconsciousness, headache, irritability, fatigue, confusion, insomnia, tremor, polyneuropathy, loss of hearing and/or vision
Cardiovascular system	Tachycardia, arrhythmia, problems with blood pressure, sometimes signs and symptoms resembling pheochromacytoma
Respiratory system	Cough, dyspnea, chest pain, pulmonary edema
Urogenital system	Tubular dysfunction, dysuria
Dermatological	Itching, erythema, rash
Digestive system	Stomatitis, colitis, abdominal pain, nausea, vomiting, diarrhea
Liver	Elevation of liver function tests, hepatomegaly, centrilobular vacuolization
Musculoskeletal system	Tremor, fasciculation, myoclonus, myalgia

Table 1: Signs and symptoms in patients with mercury exposure.

Small exposures to mercury can be fatal in young children and feti [8]. Inhalation of the heavy metal vapors by the infant or baby leading to necrotizing pneumonia and acute respiratory distress syndrome is thought to be the main reason of death in 24 h [9].

Poisoning due to self-injected metallic mercury has also been published in the literature. Local tissue or systemic consequences (i.e., mercurialism) can be seen in patients with deep tissue injection, while death due to pulmonary embolism and cardiac, brain, hepatic or renal toxicity may occur in cases of high dosage intravenous administration [10].

How should we treat mercury poisoning?

Treatment starts with keeping the patient away from the exposure and toxic agents. NAC can be beneficial for chelation of mercury. It binds mercury by its cystein groups (4). Most commonly used chelating drugs include dimercaprol (BAL), disodium ethylenediaminetetraacetate ($CaNa_2EDTA$), succimer (meso-DMSA), unithiol (DMPS), D-penicillamine (DPA), N-acetyl-D-penicillamine (NAPA), calcium calcium trisodium or zinc trisodium diethylenetriaminepentaacetate ($CaNa_3DTPA$, $ZnNa_3DTPA$), deferoxamine (DFO), deferiprone (L1), triethylenetetraamine (trientine), and Prussian Blue (PB). British Anti Lewisite (BAL) (2.5 mg/kg) is also commonly used in the treatment [4,11]. DMPS and DMSA are especially promising antidotes in mercury poisoning, whereas DMPS seems to be a more efficient agent against as poisoning. However, recent reports indicate that a combination of low-dosed BAL plus DMPS could be a preferred antidotal therapy to obtain mobilization of the intracerebral deposits into the circulation for rapid urinary excretion [12].

Intramuscular administration of dimercaptopropanol (BAL) has mostly been used in acute arsenic, lead, and mercury poisonings, but repeated BAL administration increased the brain uptake of heavy metals including mercury in experimental animals [13].

Conclusion

This review emphasizes that scientific research has culminated a significant amount of information on the mechanisms mediating Mercury-induced toxicity in the last decades. Further research in this area is well warranted. We cannot overemphasize the importance of public education on poisoning and specifically, potential hazards of mercury to protect public health.

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