

Case Report

A rare case of Homicidal Mercury poisoning

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ABSTRACT: Mercury poisoning results from exposure to mercury in various forms. We report a case of homicidal ingestion of elemental mercury. Patient was managed with gastric lavage and chelation with British anti-Lewisite (BAL). Patient remained asymptomatic and developed no complications. He was discharged after 5 days in stable condition. Homicidal cases of mercury poisoning are rare. Elemental mercury ingestion usually does not produce complications as absorption from gastrointestinal tract is minimal. Patient with mercury poisoning is managed with abstinence of exposure, gastric lavage and chelation therapy. Although elemental mercury poisoning usually does not cause complications but should be managed with care to minimize the risk if any.

Key words : Mercury, elemental, poisoning, homicidal, oral.

INTRODUCTION

Mercury poisoning is a type of metal poisoning, due to exposure to mercury,^[1] which is found in the environment in three basic states: elemental mercury or mercury vapour, inorganic mercury, and organic mercury (ethyl-, methyl-, alkyl-, or phenylmercury).^[2] Mercury poisoning can occur as a result of occupational hazard or suicide attempt. Symptoms depend upon the type, dose, method, and duration of exposure.^[1]

We report a case of homicidal mercury poisoning as a result of ingestion of elemental mercury.

CASE REPORT

A 35 years married male patient presented to emergency department with alleged history of ingestion of elemental mercury dissolved in milk. Patient was given milk to drink by his paramour. He noticed silvery white material in the glass after he finished last sip of milk. He immediately rushed to hospital. There was no history of any abdominal pain, vomiting or bowel upset. On examination patient was very apprehensive. His vitals were normal (BP-130/80 mmHg, PR-94/min, RR-14/min). Systemic examination was unremarkable. On investigations; his hemoglobin was 12gm/dl, TLC- 9000/mm³, Platelet count- 2.5 lac/mm³, blood urea- 20 mg/dl, serum creatinine- 0.6 mg/dl, serum Na⁺-136 meq/l, serum K⁺ -4 meq/l, serum Cl⁻-106 meq/l, serum

bilirubin-0.6 mg/dl, SGOT-30 mg/dl, SGPT-23mg/dl, serum Calcium 9.2 ml/dl. Urine examination was within normal limit. X-ray abdomen revealed elemental mercury in gut loops (Pics 1 and 2). Patient was managed by nasogastric tube insertion and gastric lavage. Chelation was done by administering British anti-Lewisite (BAL). Patient remained asymptomatic throughout his stay in hospital. He was discharged after five days in stable condition. On follow-up after 1 week, patient was asymptomatic, stable and feeling well.

DISCUSSION

Elemental mercury (Hg) is a dense, silver-white, odourless, heavy metal that is liquid at room temperature.^[3] Mercury exists in three forms: the metallic element, inorganic salts, and organic compounds. The source, biological properties, and toxicity between these three forms differ.^[4] Table 1 and table 2 shows sources of mercury and clinical manifestations of mercury exposure.^[5] The absorption of elemental mercury from intact skin^[6] or the gastrointestinal tract is negligible.^[7] Elemental mercury vapor is 70–80% absorbed by the lungs^[8] and inhalation is the primary route for systemic toxicity. It distributes into red blood cells, other tissues, and crosses the blood-brain barrier to accumulate in the central nervous system. It also crosses the placenta.^[9] In our case, route of poisoning was oral. As the absorption of elemental mercury

from GIT is negligible, so the patient remained asymptomatic. Wright et al [7] reported that large amounts of elemental mercury (e.g. 15 mL, 204 g) have been ingested without adverse clinical effect. Diagnosis of elemental or inorganic mercury poisoning involves determining the history of exposure, physical findings, and an elevated body burden of mercury. Blood and urinary levels of mercury may be elevated, but these levels may be misleading because chelation therapy itself may lead to transiently elevated urinary mercury levels.^[10] Levels of mercury in blood and urine does not correlate well to the body burden of mercury.^[11] Elemental mercury can be seen in abdominal viscera on roentgenography examination.^[12] In our patient diagnosis was confirmed by history of exposure and X-ray abdomen. X-ray abdomen of the patient revealed elemental mercury in gut loops (fig1 and 2). Removal of source of exposure is the first step for treatment of mercury poisoning. There are no specific blood or urine levels above which treatment with a chelating agent is indicated.^[13] Chelation therapy for acute mercury poisoning can be done with dimercaptosuccinic acid (DMSA), 2,3-dimercapto-1-propanesulfonic acid (DMPS), D-penicillamine (DPCN), or dimercaprol (BAL).^[14] In our patient gastric lavage was done followed by chelation with dimercaprol. Patient remained asymptomatic and was discharged after five days.

Mercury poisoning through ingestion of elemental mercury may cause no complications, because of minimal absorption through gastrointestinal tract. But systemic effects may be produced if there is any inflammation of the gut, due to facilitation in mercury absorption through inflamed gut.^[7] There are studies in which quantifiable doses of elemental mercury resulted in toxicity from the gastrointestinal route.^[15,16]

Elemental mercury can be methylated by micro-organisms in the soil, water and in the human gut to the methyl mercury that has the toxic effect of denaturing biological proteins, inhibiting enzymes, and interrupting membrane transport and the uptake and release of neurotransmitters.^[17,18]

CONCLUSION

We present a rare case of homicidal elemental managed simply by gastric lavage and chelation therapy. Oral poisoning when identified early and managed properly can minimize the risk of any complications.

TABLE 1. Sources of mercury exposure

TYPE OF MERCURY	EXAMPLES
Elemental mercury	Thermometers, barometers, sphygmomanometers, dental amalgams, gold-mining industry
Inorganic mercury	Antiseptics, whitening creams, Chinese herbs, mercury batteries, paint industries
Organic mercury	Fish, biocides, vaccines

TABLE 2. Clinical manifestations of mercury toxicity

	Acute exposure	Chronic or subacute exposure
Elemental mercury	Dyspnoea, cough or chest pain signifying pneumonitis, bronchiolitis, pulmonary oedema or frank respiratory failure, pruritic rash, conjunctivitis, gingivitis or stomatitis	Classic triad of tremor, gingivitis, and erethism
Inorganic mercury	Caustic gastroenteric symptoms like abdominal pain and bleeding; and renal impairment, if severe enough, acute renal failure	Kidneys (proteinuria or nephritic syndrome), nervous system (tremor, neurasthenia, erethism, neuropathy, ataxia, tunnel vision, anosmia), digestive tract (nausea, gingivostomatitis), skin (acrodynia)
Organic mercury	Malaise, paraesthesia, ataxia and impaired visual, auditory, olfactory and gustatory senses	Peripheral neuropathy



Pic.1 and 2: X-ray Abdomen showing mercury in the Gastrointestinal Tract.

Consent: Written informed consent was obtained from the

patient for publication of this case report and accompanying images.

Conflict of interest: None

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