Case Report

RADIOPAQUE SHADOWS IN THE ABDOMEN

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Key word: Meracury poisoning.

A thirty-four-year old lady presented with the history of accidental ingestion of half to one spoon of mercury. The liquid mercury was being used in her household for many years as a grain preserver. Patient induced vomiting immediately after which she was taken to Sai Hospital, Moradabad where a gastric lavage was performed within 45 minutes. Patient was brought to IAH for further evaluation and management.There was no significant past medical history.

On presentation, she was conscious & oriented, afebrile, pulse rate116/min, blood pressure 120/80mm Hg, respiratory rate 20/min, the general and systemic examination was unremarkable. Patient was hospitalized and was started on supportive treatment with IV fluids and activated Charcoal and Peglac.

Her hematological and biochemical parameters including complete hemogram, liver function tests and renal profile were essentially normal, Chest X-ray was normal. Her abdominal X ray showed scattered opacities in the intestinal lumen (*Fig.*1) which on subsequent X-ray showed significant clearing (*Fig.*2).

Patient remained haemodynamically stable during the hospital stay and serial hematological and biochemical parameters were normal. She was sent home in a stable condition.

REVIEW OF LITERATURE

Mercury poisoning (also known as hydrargyria or mercurialism) is a disease caused by exposure to mercury or its compounds. Its zero oxidation state Hg^0 exists as vapor or as liquid metal, its mercurous state Hg^+ exists as inorganic salts, and its mercuric state Hg^{2+} may form either inorganic salts or organomercury compounds.

Mercury in any form is toxic. The difference lies in how it is absorbed, the clinical signs and symptoms, and the response to treatment modalities [1]. Mercury poisoning can result from vapor inhalation, ingestion, injection, or absorption through the skin.

Neurologic, gastrointestinal, and renal systems are the most commonly affected organ systems in mercury exposure.

- Organic mercury - Most devastating to the CNS
  - * Short-chained (methylmercury) - Affects the CNS
  - * Long-chained - Subacute/chronic effects similar to that of inorganic mercury exposure
- Elemental mercury - Primary neurologic toxicity
- Inorganic mercury salts
  - * Acute - Severe corrosive gastroenteritis, acute tubular necrosis
  - * Subacute or chronic - GI, neurologic, and renal dysfunction

Elemental mercury (Hg) is found in liquid form, which easily vaporizes at room temperature and is well absorbed (80%) through inhalation. Once inhaled, elemental mercury is mostly converted to an inorganic divalent or mercuric form by catalase in the erythrocytes [2]. This inorganic form has similar properties to inorganic mercury (e.g., poor lipid solubility, limited permeability to the blood-brain barrier, and excretion in feces). Small amounts of nonoxidized elemental mercury continue to persist and account for central nervous system toxicity.

Elemental mercury as a vapor has the ability to penetrate the CNS, where it is ionized and trapped, attributing to its significant toxic effects. Elemental mercury is not well absorbed by the GI tract and, therefore, when ingested (e.g., thermometers), is only

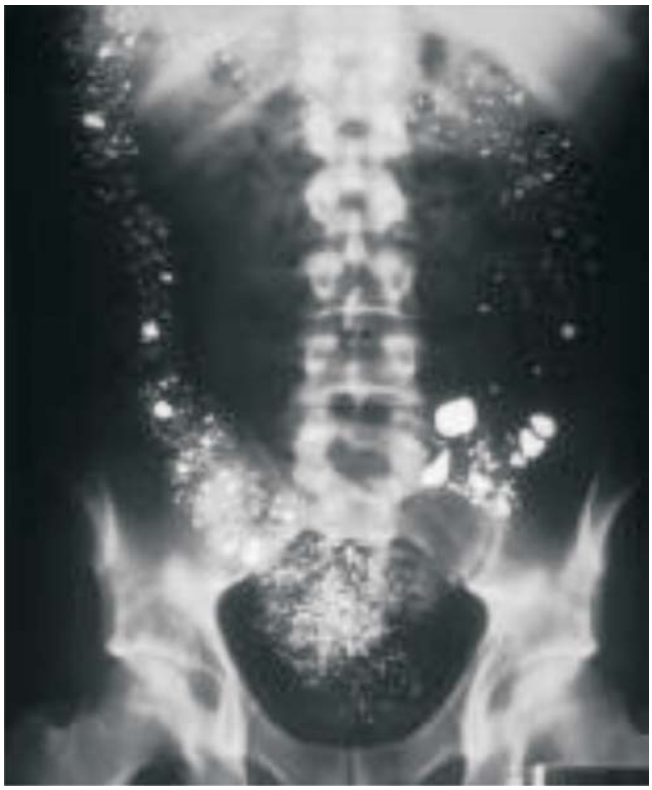

Fig.1. Plain X-ray abdomen AP view

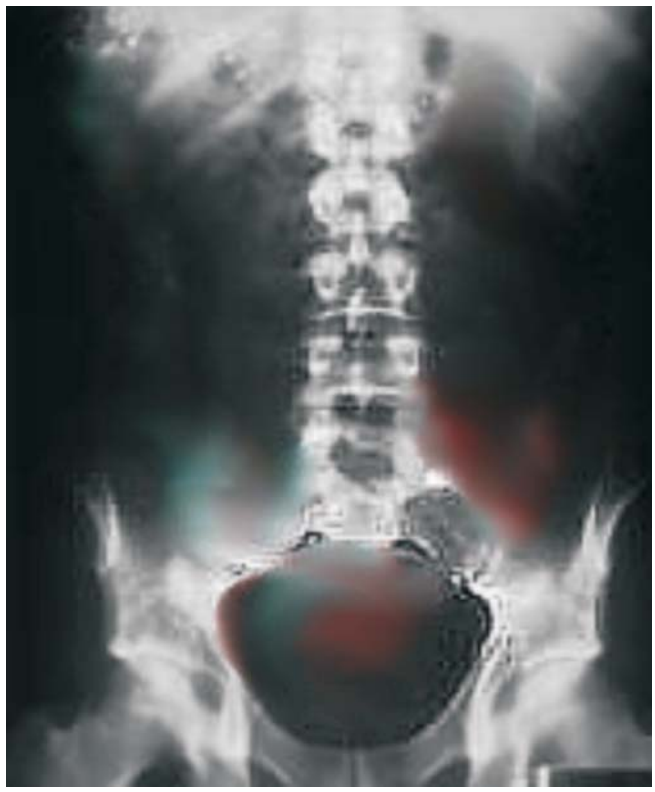

Fig. 2. X-ray abdomen after 2 days of hospitalisation

mildly toxic. Animal data indicate that less than 0.01% of ingested mercury is absorbed through the intact gastrointestinal tract; though it may not be true for individuals suffering from ileus. Cases of systemic toxicity from accidental swallowing are rare, and attempted suicide via intravenous injection does not appear to result in systemic toxicity. Some mercury vapour is absorbed dermally but uptake by this route is only approximately 1% of that by inhalation. Although people can be exposed to elemental mercury as a dust or a liquid, it is especially dangerous when it is inhaled as a vapor. In particular, crawling children are at a greater risk because mercury is heavier than air, and will settle near the floor. Natural sources of elemental mercury include release from volcanic eruptions, and erosion of mercury-containing ores.

Causes of elemental mercury toxicity include barometers, batteries, bronzing, calibration instruments, chlor-alkali production, dental amalgams, electroplating, fingerprinting products, fluorescent and mercury lamps, infrared detectors, the jewelry industry, manometers, neon lamps, paints, paper pulp production, photography, silver and gold production, semiconductor cells, and thermometers. Mercury can be found in a number of items, including switches used in machinery, equipment, and explosives (*Fig.*3).

Dental amalgams also contain elemental mercury (*Fig.*4). Dental professionals who are in contact with amalgam must follow specific guidelines to avoid exposure to toxic amounts of aerosolized elemental mercury [2]. Patients with dental amalgam fillings have slightly elevated levels in their urine, but these findings have not correlated with any systemic disease [3].

Diagnosis of elemental or inorganic mercury poisoning involves determining the history of exposure, physical findings, and an elevated body burden of mercury. Although whole blood mercury concentrations are typically less than 6μg/L, diets rich in fish can result in blood mercury concentrations higher than 200μg/L; it is not that useful to measure these levels for suspected cases of elemental or inorganic poisoning because of mercury's short half-life in the blood [4]. If the exposure is chronic, urine levels can be obtained; 24-hour collections are more reliable than spot collections. Urine mercury levels are typically less than 10-20 mcg/L. Excretion of mercury in urine is a good indicator of inorganic and elemental mercury exposure but is unreliable for organic mercury (methylmercury) because elimination occurs mostly in the feces [5]. No absolute correlation exists between the urine mercury levels and the onset of symptoms; however, levels higher than 300 mcg/L are associated with overt symptoms [6].

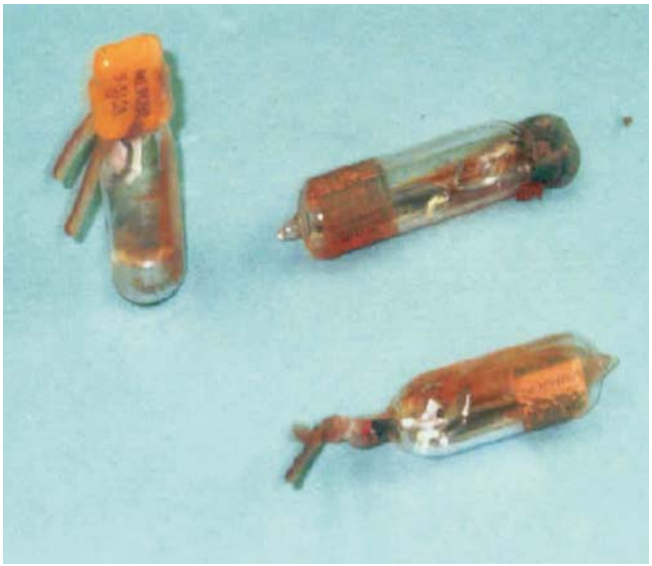

Fig.3. Switches can contain a teaspoon or more of liquid mercury.

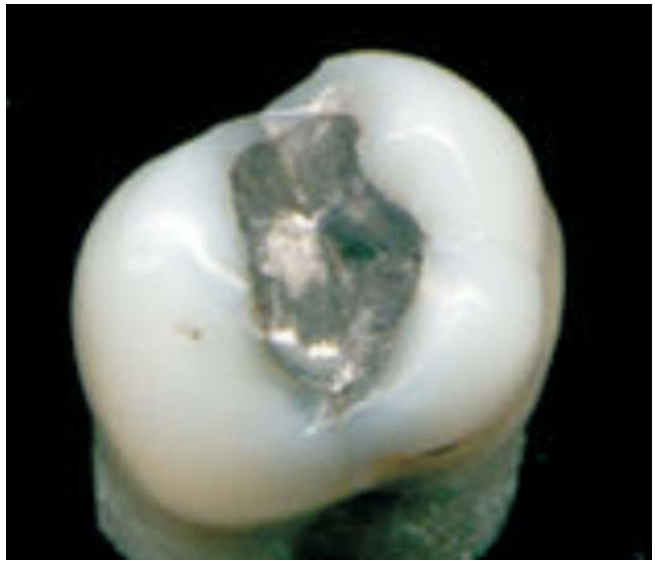

Fig.4. Amalgam filling

Supportive care begins with the ABCs, especially when managing the inhalation of elemental mercury and the ingestion of caustic inorganic mercury, both of which may cause the onset of airway obstruction and failure [7]. The next step in supportive care is the removal of contaminated clothing and copious irrigation of exposed skin. Aggressive hydration may be required for acute inorganic mercury poisoning because of its caustic properties [8].

Gastric lavage is recommended for organic ingestion, especially if the compound is observed on the abdominal X-ray series [9]. Activated charcoal is indicated for GI decontamination because it binds inorganic and organic mercury compounds to some extent.

Whole bowel irrigation may be used until rectal effluent is clear and void of any radiopaque material. However, effectiveness in decreasing the GI transit time of elemental mercury is doubtful because of the high density of elemental mercury and the low density of the whole bowel irrigant solutions [10]. Likewise, whole bowel irrigation has no adsorptive effect on any type of mercury within the GI tract.

Use chelating agents if the patient is symptomatic, if systemic absorption is anticipated, or if increased blood or urine levels are present. Chelating agents contain thiol groups, which compete with endogenous sulfhydryl groups [11,12].

Hemodialysis is used in severe cases of toxicity. In very rare cases, mercury can become trapped in the appendix or intestine and require surgical removal.

Systemic effects of massive oral ingestion of mercury have been infrequently reported [13]. The observation suggests that massive and prolonged retention of metallic mercury may facilitate the conversion of metallic, elemental mercury to divalent mercury and its subsequent absorption with development of hepatic dysfunction.

REFERENCES

1. Bronstein AC, Spyker DA, Cantilena LR Jr, Green JL, Rumack BH, Heard SE. 2007 Annual Report of the American Association of Poison Control Centers' National Poison Data System (NPDS): 25th Annual Report. Clin Toxicol (Phila). 2008; 46(10): 927-1057.

2. Public Health Service. Dental amalgam: a scientific review and recommended Public Health Service strategy for research, education, and regulation. Public Health Service. January 1993ATSDR. Agency for Toxic Substances and Disease Registry. Toxicological Profile for Mercury. ATSDR. August 1997.

3. Food and Drug Administration. Consumer Update on dental amalgams. United States Food and Drug Administration (FDA).

4. Belson MG, Schier JG, Patel MM. CDC. Case definitions for chemical poisoning. MMWR Recomm. 2005; 54(RR-1): 1-24.

5. Young-Jin S. Mercury. *In* Flomenbaum NE, Goldfrank LR, Hoffman RS, Howland MA, *et al*, eds. Goldfrank's Toxicologic Emergencies. 8th ed. New York: The McGraw-Hill Companies; 2006: 96.

6. Charlton N, Wallace KL. American College of Medical Toxicology - Position Statement: Post-Chelator Challenge Urinary Metal Testing. July 27, 2009.

7. Bates B. Heavy metals and inorganic agents. *In* Clinical Management of Poisoning and Drug Overdose. Vol 55. WB Saunders; 1998; 55: 750-756.

8. Clifton JC 2nd. Mercury exposure and public health. Pediatr Clin North Am. 2007; 54(2): 237-269.

9. Ford M. Heavy metals. *In* Tintinalli JE, ed. Emergency Medicine: A Comprehensive Study Guide. 4th ed. McGraw-Hill; 1996; 158: 839-841.

10. Goyer RA. Toxic effects of metals. *In:* Casarett LJ, ed. Casarett and Doull's Toxicology: The Basic Science of Poisons. 5th ed. New York: McGraw-Hill; 1996: 709-713.

11. Taueg C, Sanfilippo DJ, Rowens B, *et al.* Acute and chronic poisoning from residential exposures to elemental mercury—Michigan, 1989-1990. J Toxicol Clin Toxicol. 1992;30(1): 63-67.

12. Watson WA, Litovitz TL, Klein-Schwartz W, *et al.* 2003 annual report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. Am J Emerg Med. 2004; 22(5): 335-404.

13. Young J. Mercury. *In* Goldfrank LR, ed. Goldfrank's Toxicology Emergencies. New York: McGraw-Hill; 1994; 74: 1051-1062.