A curious case of adult with accidental mercury ingestion presenting as foreign body in gastrointestinal tractwith isolated central nervous system toxicity

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ABSTRACT

Mercury is a heavy metal known for its toxicity and noted for inducing public health disasters in Minamata Bay, Japan, and Iraq. The toxic effects of mercury can either be acute and extremely severe or can manifest very subtly over a long period. Here, we report the case of a 56-year-old gentleman presented with complaints of sudden loss consciousness. On further evaluation and radiological examination, the patient had features suggestive of encephalopathy with a suspected foreign body in the gastrointestinal tract. Foreign body was found to be elemental mercury with elevated serum mercury levels. He was managed conservatively with chelation therapy with dimercaprol and prokinetic drugs such as polyethylene glycol which lead to the advancement of foreign body in the gastrointestinal tract on repeated abdominal x-rays with subsequent complete clearance.

Keywords: *Foreign body ingestion, Mercury poisoning, Toxicity.*

Mercury is a heavy metal known for its toxicity and noted for inducing public health disasters in Minamata Bay, Japan [1] and in Iraq [2–4].The clinical impact of smaller mercury exposures remains controversial. It exists in several forms: inorganic mercury, which includes metallic mercury and mercury vapor and mercurous or mercuric salts; and organic mercury, which includes compounds in which mercury is bonded to a structure containing carbon atoms (methyl, ethyl, phenyl, or similar groups).The biological behavior, pharmacokinetics, and clinical significance of the various forms of mercury vary with chemical structure. Inhaled elemental mercury vapor, for example, is easily absorbed through mucous membranes and the lung and rapidly oxidized to other forms (but not so quickly as to prevent considerable deposition of elemental mercury in the brain). Methyl mercury is easily absorbed through the gut and deposits in many tissues, but does not cross the blood-brain barrier as efficiently as elemental mercury; however, on entering the brain it is progressively demethylated to elemental mercury [5]. Mercury salts, in contrast, tend to be insoluble, relatively stable, and poorly absorbed.

Human toxicity varies with the form of mercury, the dose and the rate of exposure. The target organ for inhaled mercury vapor is primarily the brain.Mercurous and mercuric salts chiefly damage the gut lining and kidney, while methyl mercury is widely distributed throughout the body. Toxicityalso varies with dosage as large acute exposures to elemental mercury vapor induces severe pneumonitis, which in extreme cases can be fatal [6,7]. Here, we are presenting a curious case of 56-year-old adult who accidentally ingested approximately 200 grams of elemental mercury with isolated central nervous system toxicity.

CASE REPORT

A 56-year-old man, electrician by profession was brought by attendants with an alleged history of giddiness followed by sudden onset loss of consciousness, which was associated with the history of fall after which he was taken to a nearby hospital and evaluated for same. He had sustained head injury with a contused lacerated wound of 2centimeters on the left parietal region. The patient was intubated and put on ventilator support in view of cerebrovascular collapse/stroke. The patient is a known case of type 2 diabetes mellitus and is Australia antigen positive.

On further evaluation, the patient was restless and disoriented with a blood pressure of 140/70 millimeters of mercury, pulse 90/min, respiratory rate 16/min, and random blood sugar levels206milligram/deciliter. He was intubated in accident and emergency was put on put on pressure control ventilator mode. Glasgow coma scale was E_3 V_T M_4.

The blood investigations were as follows: hemoglobin 11.1gram/deciliter, leukocyte count10500, platelets130000 and prothrombin time/international normalized ratio (INR) 13.6/1.16. Alanine/ aspartate transaminases were 42/19 IU/ml, alkaline phosphatase was 140 IU/ml, sodium/ potassium/ chloride levels were 137/4.2/96 mEq/dl, and

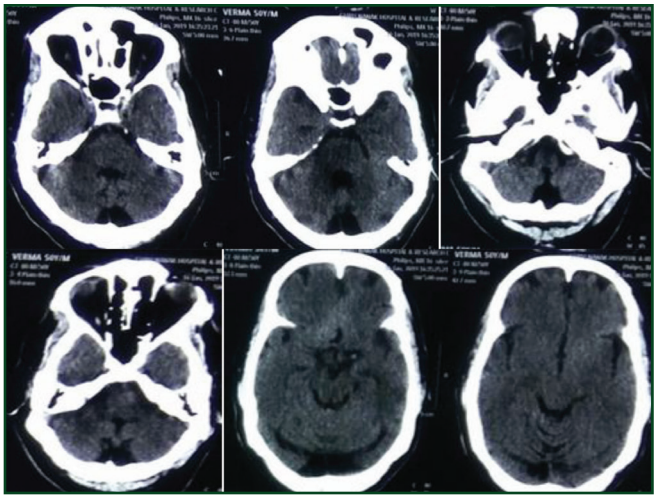

Figure 1: Bilateral cerebellar lacunar infarcts and no evidence of infarct/bleed or SOL were seen

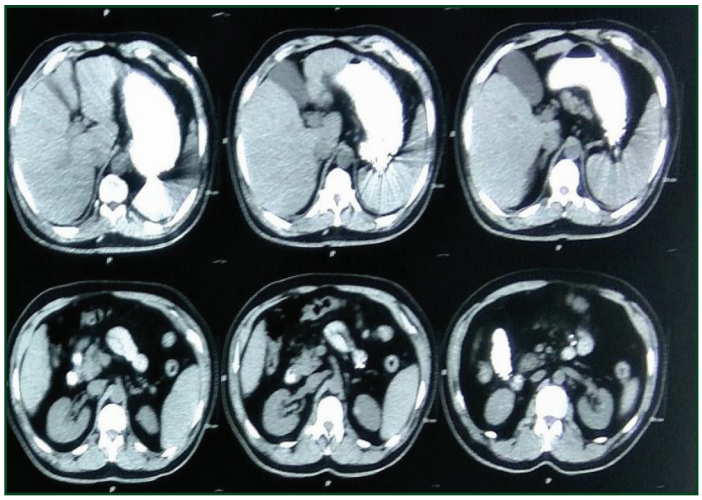

Figure 2: Cast beam hardening artifacts seen in the stomach and right-sided colon suggestive of foreign body

blood urea nitrogen was 10 mg/dl and serum creatinine was 0.94 mg/dl.

Computed tomography (CT) scan abdomen, pelvis, and brain revealed lacunar infarcts in bilateral cerebral hemispheres and no acute territorial infarct/bleed or space occupying lesion seen. Abdominal findings were consistent with a foreign body in the right-sided colon and stomach(Fig. 1 and 2). Multiple hyperdense foci were seen in the stomach and right-sided colon was seen. X-ray abdomen revealed a foreign body inthe stomach and right-sided colon (Fig. 3 and 4). Electroencephalogram showed an excess of slow activity over both sides, suggestive of bi-cerebral disturbance.

Patient in view of the above findings was evaluated for suspected foreign body ingestion with toxic encephalopathy due to unknown substance poisoning. A urine toxicology screen and blood heavy metal screening were ordered and was found to have isolated elevated benzodiazepine levels of around 550 ng/ml. Surgical reference was given in view of the foreign body in the stomach and right-sided colon. An upper gastrointestinal endoscopy was planned for assessment of the type of foreign body and if possible removal. Also, neurologist and nephrologist were involved in view of central nervous toxicity and to rule out renal toxicity. Vital signs, neurological checks, and electrolytes were measured on a regular basis.On day 2 of admission,the patient passed stool with silver colored droplets over it. It was suspected to be mercury and was confirmed on biochemical examination. The serum mercury levels sent subsequently were around 55 microgram/liter. X-ray done showed the advancement of the foreign body into small bowel from its previous location. Hence a decision to hold upper gastrointestinal endoscopic/colonic decompression with removal of foreign body was taken along with gastroenterologists.

The patient was started on oral penicillamine 250mg thrice a day and activated charcoal was started via Ryles tube to which he developed an allergic reaction and was subsequently started on dimercaprol 200 milligrams for 10 days. Egg whites were given through Ryles tube in view of chelation along with polyethylene glycol for promoting passage foreign body through gastrointestinal tract along with continuous intravenous fluid and electrolyte replacement. Subsequent X-ray abdomen revealed the movement of foreign body distally. The patient improved

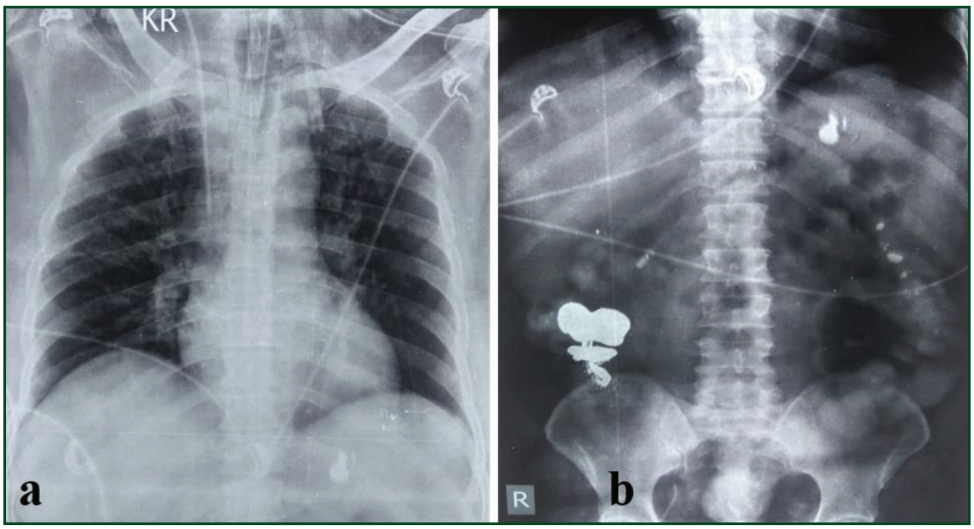

Figure 3: (a) Foreign body seen in stomach and (b) right colon and stomach

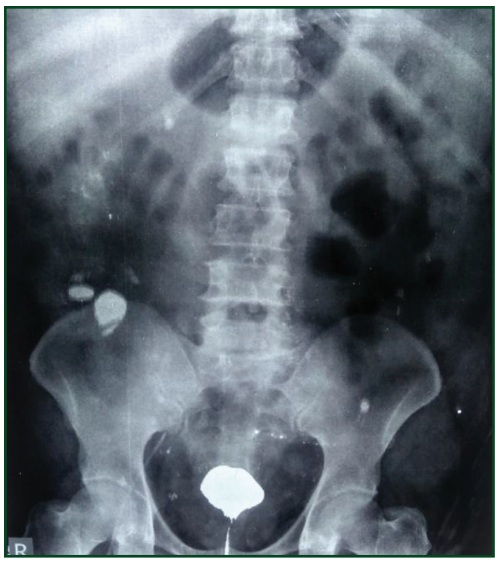

Figure 4: Advancement of foreign body in rectum with residual amount seen in the right-sided colon

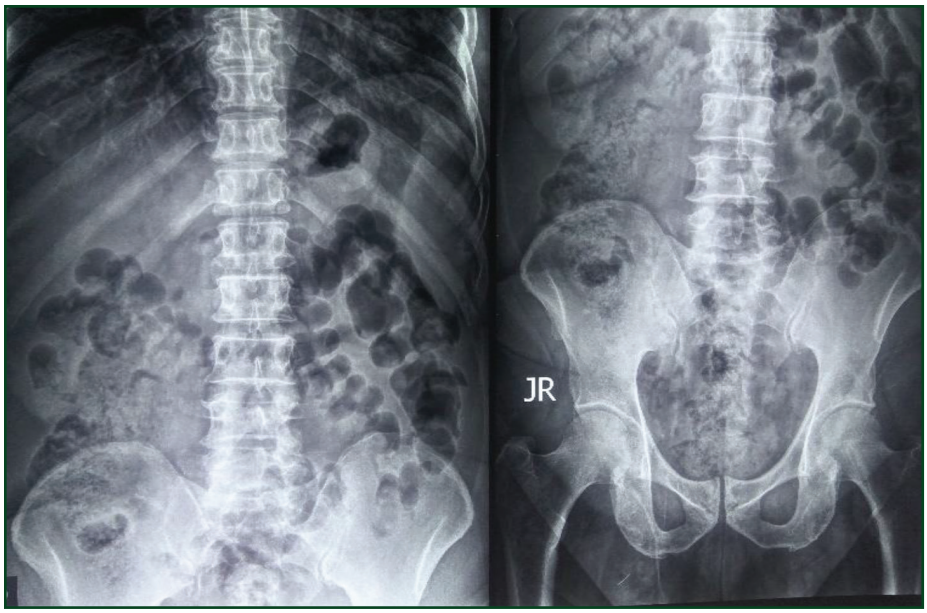

Figure 5: Complete removal of foreign body from gastrointestinal tract

hemodynamically and neurologically and hence, extubated on the 10th day. The patient was started on oral feeds which he tolerated well and all vital parameters were within normal limits. X-ray abdomen done on 12th day revealed clearance of entire foreign body from the colon (Fig. 5).

The patient was showing slow progress in term of feeling weak, fatigue, and imbalance during his stay but remained hemodynamically stable. He was shifted out of the intensive care unit to the ward. The patient was having recurrent bowel movements on a daily basis, and X-rays showed a continued decrease in the amount of mercury in the gastrointestinal tract. Retrospectively,the patient was asked for the accidental occupational exposure or ingestion of mercury before the cerebrovascular event, and he revealed not to remember any of such exposure or accidental ingestion. On subsequent follow-up visit after one month, he was doing well with all vital parameters within normal range and no neurological deficits.

DISCUSSION

The toxic effects of mercury can either be acute and extremely severeor can manifest very subtly over a long period. The toxicity level is determined by the chemical form, degree of exposure, route of administration, distribution through the body, accumulation in target organs, elimination of mercury, age of the patient, and co-morbidities [8,9,10]. Elemental mercury exposure occurs frequently and might cause neurological symptoms [11]. Elemental mercury is a shiny, silver-colored liquid, which evaporates at room temperature. Thermometers, sphygmomanometers, barometers, and batteries contain elemental mercury. Elemental mercury is also used in gold and silver processing. Elemental mercury intoxication usually occurs via inhalation, with less than 0.1% being absorbed through the gastrointestinal system following oral ingestion [12-15]. Absorption, distribution, excretion and toxicity depend upon the compound to which the patient is exposed.

Acute mercury poisoning is best managed by discontinuing the exposure, providing supportive therapy and enhancing the removal of the metal from the body. Elimination of mercury from the body is achieved by chelation therapy and self-excretion. Lowering the body level concentration is fundamental in selected cases by using chelating agents. Chelating agents increase the urinary excretion of mercury which includes thiol-based agents such as dimercaprol, penicillamine, unithiol (2,3-dimercaptopropane-1-sulfonate), and succimer (dimercaptosuccinic acid) [16].

The absorption rate of elemental mercury is lower than 0.01% through a healthy intestine; therefore, it is suggested that gastrointestinal absorption of mercury is clinically unimportant and oral intake is usually not toxic [2, 7]. In general, the risk of systemic toxicity from ingestion is considered to be low, but there are conditions under which ingesting elemental mercury can be dangerous (e.g., obstruction with a delayed passage or intestinal perforation allowing absorption from the peritoneum). Furthermore, elemental mercury may accumulate in the appendix and be converted into organic mercury compounds such as methyl-mercury by bacterial flora, which can cause toxic effects due to the increase in absorption [11,12]. The dose of mercury ingested and the level of exposure is the most important factor in mercury poisoning. Even if the patients are exposed to the same mercury dose, clinical manifestation in acute and chronic poisonings may differ [11, 12, 13].

Toxic levels of oral intake of elemental mercury and the treatment for mercury poisoning in adultsare not clear. It has been reported that the oral intake and ingestion of elemental mercury typically contained in a thermometer (approximately 0.1 milliliters or 1 gram) does not cause intoxication [15]. The presence of mercury in the blood, urine, hair or tissues is accepted as evidence ofpoisoning. It has been reported that there is no

correlation between clinical signs and mercury concentration in the blood and urine [17].

In our case, the blood mercury level was 55 microgram/liter and mercury poisoning was a certain diagnosis. Incidence of accidental ingestion of mercury in such large amount in an adult is not quoted anywhere in literature searched through PubMed, Medline, google scholar, which makes this case a rare one.

CONCLUSION

The accidental oral intake of elemental mercury in large amounts approximately of 200grams is extremely rare in adults. Ours is a rare case in which adult had an accidental ingestion of mercury in large quantity and developed isolated central nervous systemencephalopathywith no other system involved.

REFERENCES

1. Environmental Health Department, Ministry of the Environment. Minimata Disease: The History and Measures. Tokyo, Japan: Ministry of the Environment, Government of Japan; 2002.
2. Bakir F, Damluji SF, Amin Zaki I. Methylmercury poisoning in Iraq: an interuniversity report. Science 1973;181:230-41.
3. Skerfving SB, Copplestone JF. Poisoning caused by the consumption of organomercury dressed seed in Iraq. Bulletin of the World Health Organization 1976;54:101-12.
4. Clarkson TW, Magos L, Cox C. Tests of efficacy of antidotes for removal of methylmercury in human poisoning during the Iraq outbreak. Journal of Pharmacology and Experimental Therapeutics 1981;218:74-83.
5. Berlin M, Zalups RK, Fowler BA. Mercury. In: Nordberg GF, Fowler BA, Nordberg M, Friberg LT, editors. Handbook on the Toxicology of Metals. 3rd edition. Chapter 33. New York, NY, USA: Elsevier; 2007.
6. Worth DP, Davison AM, Lewins AM, Ledgerwood MJ. Haemodialysis and charcoal haemoperfusion in acute inorganic mercury poisoning. Postgrad Med J 1984;60:636-8
7. Sauder PH, Livardjani F, Jaeger J, Kopfershmitt J, Heimburger R, Waller CH, *et al*. Acute mercury chloride intoxication, effects of hemodialysis and plasma exchange on mercury kinetic. ClinToxicol 1988;26:189-97
8. Counter SA, Buchanan LH. Mercury exposure and child development outcomes. Pediatrics 2004;198:209-30.
9. Carl RB. Treatment of mercury intoxication. CurrOpinPediatr 1999;11:265-8.
10. Yoshida M, Satoh H, Igarashi M, Akashi K, Yamamura Y, Yoshida K. Acute mercury poisoning by intentional ingestion of mercuric chloride. Tohoku J Exp Med 1997;182:347-52.
11. McKay CA, Perez A, Goldstein R. Mercury. In: Erickson TB, Baum CR, Ling LJ, editors. Pediatric Toxicology: Diagnosis and Management of the Poisoned Child. NewYork, PA: The McGraw-Hill Companies; 2005. pp. 468–75.
12. Mahajan PV. Heavy Metal Intoxication Mercury. In: Kliegman RM, Geme JW, Schor NF, Behrman RE, editors. Nelson Textbook of Pediatrics. 19th ed. Philadelphia PA: Elsevier Saunders; 2011. p. 2448.http://dx.doi.org/10.1016/B978-1-4377-0755-7.00701-6
13. Carman KB, Tutkun E, Yilmaz H, Dilber C, Dalkiran T, Cakir B, *et al*. Acute mercury poisoning among children in two provinces of Turkey. Eur J Pediatr 2013;172:821-7.
14. Yilmaz FM, Yilmaz H, Tutkun E, Uysal S, Carman KB, Dilber C, *et al*. Serum biochemical markers of central nerve system damage in children with acute elemental mercury intoxication. ClinToxicol (Phila) 2014;52:32-8.
15. Agency for Toxic Substances and Disease Registry - ATSDR Public Health Service. Toxicological Profile for Mercury 1999:1–676.
16. Nayfeh A, Kassim T, Addasi N, Alghoula F, Holewinski C, and Depew Z. A Challenging Case of Acute Mercury Toxicity, Case Rep Med 2018;1010678.
17. Ruha AM, Tanen DA, Suchard JR, Curry SC. Combined ingestion and subcutaneous injection of elemental mercury. J Emerg Med 2001;20:39-42.

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