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A 3 Year-Old Male Child Ingested Approximately 750 Grams of Elemental Mercury

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Background: The oral ingestion of elemental mercury is unlikely to cause systemic toxicity, as it is poorly absorbed through the gastrointestinal system. However, abnormal gastrointestinal function or anatomy may allow elemental mercury into the bloodstream and the peritoneal space. Systemic effects of massive oral intake of mercury have rarely been reported.

Case Report: In this paper, we are presenting the highest single oral intake of elemental mercury by a child aged 3 years. A Libyan boy aged 3 years ingested approximately 750 grams of elemental mercury and was still asymptomatic.

Conclusion: The patient had no existing disease or abnormal gastrointestinal function or anatomy. The physical examination was normal. His serum mercury level was 91 µg/L (normal: <5 µg/L), and he showed no clinical manifestations. Exposure to mercury in children through different circumstances remains a likely occurrence.

Keywords: Intoxication, mercury, supportive therapy

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 (1-3). Elemental mercury intoxication usually occurs via inhalation, with less than 0.1% being absorbed through the gastrointestinal system following oral ingestion (1-3). The accidental oral intake of excessive elemental mercury in children has not been encountered in the literature. Here, we are presenting a child aged 3 years who ingested approximately 750 grams of elemental mercury and remained asymptomatic.

CASE PRESENTATION

A Libyan boy aged 3 years who ingested approximately 55 cc (750 grams) of elemental mercury was admitted to the Pediatric Emergency Service for further diagnosis and treatment. We learned that his uncle, who worked in silver processing, kept 60 cc of liquid mercury at home. The patient's family reported that he drank from the bottle and only 5 cc of mercury remained. He was immediately taken to a nearby emergency unit after receiving gastric lavage and the administration of penicillin; the boy was transferred to our clinic for further diagnosis and treatment.

The patient had no symptoms when he was admitted to our clinic after 2 days following oral mercury intake. The patient had no acute or chronic gastrointestinal or other systemic diseases.

In the physical examination, the patient was conscious, cooperative, and orientated. The respiratory and cardiovascular system examinations showed no signs of abnormality, and the abdomen was soft and non-distended. There was no tenderness, rebound or hepatosplenomegaly. Neurological and other system examinations were normal. There were particles of mercury on the patient's diaper.

Full blood count, electrolytes, renal and hepatic function tests were normal (WBC: 10200/µL, AST: 46 U/L, ALT: 112 U/L, GGT: 87 IU/L, Glu: 89 mg/dL, Na: 138 Meq/L, K: 4.1 Meq/L, BUN: 15 mg/dL, creatinine: 0.4 mg/dL).

Occult blood in the stool test was negative. Urine analysis had no features. In the Chest X-Ray, there were no foreign bodies and the parenchyma showed no pathological findings. In the abdominal X-Ray, dense opacities were found in all intestinal segments due to mercury ingestion (Figure 1).

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The patient was followed-up in close clinical, biochemical, and radiological monitoring. The patient was fed fiber-rich foods and defecated mercury particles spontaneously every day. The blood mercury level was 91 µg/L (normal: <5 µg/L). Urine mercury levels could not be measured due to technical malfunctions.

Dimercaptosuccinic acid (Succimer (Chemet); Kremers Urban Pharmaceuticals Inc.; IN, USA) treatment was started orally with 10 mg per kg every 8 hours for 5 days because blood mercury levels were high, even though the patient was asymptomatic. The treatment was continued at 12-hour intervals for 2 weeks.

The clinical signs and biochemical parameters were within normal values.

The radio-opacities due to mercury in the abdomen X-ray decreased gradually and vanished by the seventh day (Figure 2).

The blood mercury level on the 7th day after the initial measurement was 7 µg/L. The patient was re-evaluated 15 days after discharge. The patient was asymptomatic and had no complaints involving his gastrointestinal system. In follow-up tests, the blood mercury level in the 3rd month after the initial measurement was 5.5 µg/L, the urine mercury levels were 11.3 µg/L (normal: <5 µg/L) and urine creatinine 9.5 mg/dL (normal: 25-180 mg/dL).

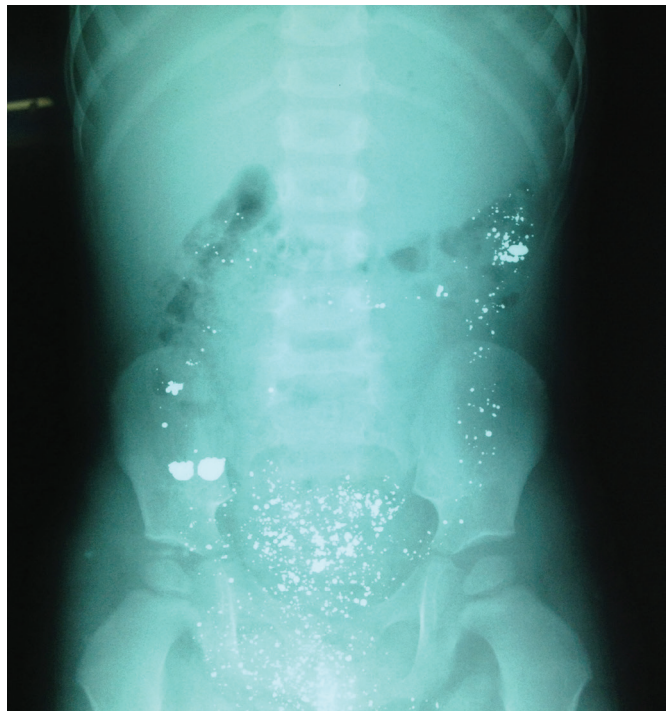

FIG. 1. Radiography of the abdomen, first day

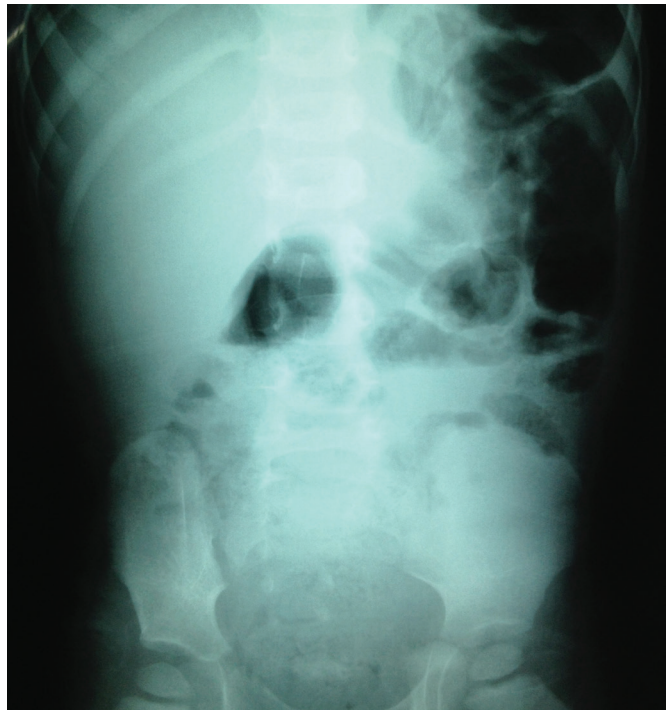

FIG. 2. Radiography of the abdomen, seventh day

DISCUSSION

The toxic effects of mercury can either be acute and extremely severe, or can manifest very subtly over a long period. The toxicity level is determined by the chemical form, degree of exposure and route of administration, distribution through the body, accumulation in target organs, and the elimination of mercury, age of the patient, and co-morbidities (4). Elemental mercury exposure occurs frequently and might cause neurological symptoms (5). Yilmaz et al. (6) have shown that serum neuron-specific enolase (NSE), S100B and glutamate receptor have been found to be significantly higher in these patients with neurological symptoms. In our case, although the patient ingested a large dose of elemental mercury, there were no clinical symptoms present.

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 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 (2,4). In our case, the patient had no existing GIS pathologies and consequently had no clinical manifestations.

The dose of mercury ingested and 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 (2,4,5). Toxic levels

of oral intake of elemental mercury and the treatment for mercury poisoning in children are not clear. It has been reported that the oral intake and ingestion of elemental mercury typically contained in a thermometer (approximately 0.1 mL or 1 gram) does not cause intoxication. Therefore, elemental mercury levels in thermometers are below toxic levels (7). To the best of our knowledge, this case reports one of the highest single doses of mercury taken orally without presenting any clinical symptoms. In our case, on the 7th day, the blood mercury levels had returned to normal and the excretion of mercury through the feces had stopped. The patient was treated with laxatives, and was recommended bed rest and monitoring. It was considered that such an amount of mercury ingested orally would not have been absorbed significantly (8).

The presence of mercury in the blood, urine, hair or tissues is accepted as evidence of poisoning. It has been reported that there is no correlation between clinical signs and mercury concentration in the blood and urine (9). In our case, the blood mercury level was 91 μg/L and mercury poisoning was a certain diagnosis.

Abdominal X-rays should be performed if the patient has a history of exposure over a long-term exposure, decrease in gastrointestinal motility, inflammatory bowel disease, or a fistula (2). The patient was put on a fiber-rich diet and continued to excrete feces with mercury; the excretion of mercury ceased on the 7th day.

Gastrointestinal lavage, a controversial and unclear method, is usually not recommended as orally ingested mercury is not absorbed effectively. Although active carbon and cathartic treatments are also controversial, they may be useful if other poisoning agents are considered to be present (4). In patients with gastrointestinal fistula, if mercury is observed in direct X-rays, oral polyethylene glycol along with fiber-rich food may be given for bowel cleaning in order to prevent long-term exposure to mercury. Total bowel washing with tamponed electrolyte solution may be performed.

Signs of mercury intoxication with a history of exposure are an indicator for chelation treatment. Chelation treatment should be started earlier if blood and urine mercury levels are high, or if there is respiratory distress or acrodynia. Early chelation can reduce or prevent the widespread effects of intoxication. Chelates used in mercury intoxication should have two reciprocal sulfhydryl groups to bind free ionic Hg^{2+} in the blood or mercury stored in tissues. Dimercaptosuccinic acid may be used as a treatment from 10 mg per kg for 5 days every 8 hours, followed by every 12 hours for 14 days (2,10).

Unfortunately, children's exposure to mercury by different routes is still likely to occur. Ours is a rare case in which no clinical toxic signs developed after the oral intake of mercury; however, other cases might present with serious toxic effects after mercury poisoning. Therefore, we aimed to review the current approach to elemental mercury poisoning in the hope of further contribution to its treatment.

Ethics Committee Approval: N/A.

Informed Consent: Written informed consent was obtained from the parents of the patient who participated in this study.

Peer-review: Externally peer-reviewed.

Author contributions: Concept - M.U., Y.Y. ; Design - M.U., Y.Y., G.P.; Supervision - A.C., N.U.; Resource - Y.Y., G.P.; Materials - M.U., Y.Y., G.P.; Data Collection and/or Processing - M.U., G.P., Y.Y.; Analysis and/or Interpretation - M.U., Y.Y., G.P.; Literature Search - M.U., Y.Y.; Writing - M.U., Y.Y.; Critical Reviews - A.C., M.U.

Conflict of Interest: No conflict of interest was declared by the authors.

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