CASE REPORT

Mercury: Is it elemental my dear Watson?

M Poulden

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A case of poisoning with a mercury "solution" is reported. The solution was known to contain mercury but its exact nature was unknown. Despite appropriate initial management, various assumptions were made that resulted in a "toxic" level. The presentation and management is discussed.

Mercury toxicity has been recognised for thousands of years; its effects depending on the type and mode of exposure.

Exposure can be acute or chronic. Acute exposure usually follows ingestion, inhalation of vapour or rarely parenteral exposure.

Mercury exists as either elemental mercury (quicksilver), inorganic salts or organic compounds.

Exposure may be as a result of attempted suicide, occupational exposure (fireworks industry), medical exposure (sphygmomanometers), food (contaminated fish, Minamota¹) and other sources (button batteries²).

Toxicity depends on type of mercury, route of exposure, load and rate of administration and patient age.^{3,4}

CASE REPORT

A 40 year old woman presented to accident and emergency (A&E) with a history of deliberate self poisoning four hours previously.

She admitted to taking an unknown quantity of paracetamol, diazepam and some "mercury solution from her husband's toolbox". The exact identity of the solution was unknown; it was unavailable for analysis. No information was available from her husband.

She had a past history of deliberate toxic drug ingestion. Her usual medications were fluoxetine and diazepam. She complained of some nausea on arrival. On examination she was alert. The airway was patent. Her pulse was 96, BP 106/71, RR14, oxygen saturations 98% on air. Chest, abdominal and neurological examination was normal.

Supplemental oxygen was supplied, intravenous access established and blood sent for U&E, FBC, liver function test and INR (all normal), paracetamol (<10 mg/l), salicylate (not detected). Blood and urine were also sent for mercury estimation. An abdominal radiograph showed radio-opaque globules throughout the stomach and small intestine (fig 1).

She was referred to the medical team for further treatment. The medical team discussed the case with the National Poisons Information Service (NPIS). They were informed that elemental mercury is negligibly absorbed from the gastrointestinal tract and therefore requires no treatment.

After review by the duty psychiatrist the next morning the patient was discharged home. Two weeks later the chemical pathology technician contacted A&E with the blood and urine mercury levels (they had been sent to a regional heavy metal laboratory). Blood mercury was 815 nmol/l (normal <30 nmol/l, toxic >250 nmol/l), urine mercury 640 nmol/mmol creatinine (normal <2 nmol/mmol creatinine).

She was recalled to A&E where neurological examination was unremarkable, abdominal radiograph showed no evidence of retained mercury, U&E was normal and blood mercury (done as an emergency) was 178 nmol/l. The NPIS advised to monitor U&E until blood mercury normal. At this stage the patient refused further follow up. Her general practitioner was contacted because of the risk of late sequelae.

DISCUSSION

Elemental mercury ingestion is considered benign because systemic absorption is unlikely. The exceptions are patients with intestinal problems such as diverticulosis where mercury may be retained in the gastrointestinal tract for prolonged periods. This permits bacterial conversion of elemental to organic mercury. This can then be systemically absorbed.

Also, because of its specific gravity and free flowing properties it can overcome the normal swallowing pathway to pass into the bronchial tree. Here its significant vapour pressure, together with potential for oxidation to inorganic mercury gives a high risk of toxicity,⁵ leading to endobronchial haemorrhage and death.⁶

Our patient did not have a chest radiograph to exclude this as a potential, but unlikely, site for absorption.

Elemental mercury intoxication usually results from inhalation of vapour. This results in respiratory distress with pneumonitis, nausea and vomiting, salivation, headache and occasionally thrombocytopenia.⁷

Subcutaneous administration can cause local reaction⁸ but the slow absorption may produce no acute toxic effect.⁹ Chronic toxicity, however, may occur leading to delayed neuropsychological sequelae.¹⁰

Figure 1 Plain abdominal radiograph showing radio-opaque material in stomach and small intestine.

Similarly intravenous injection can lead to pulmonary and systemic microemboli resulting in both acute and chronic toxicity.¹¹

Organic compounds tend to have a less dramatic acute effect but again delayed neuropsychological sequelae are important.¹²

Acute ingestion of mercuric salts causes discolouration of the mucous membranes and a severe irritant gastroenteritis resulting in vomiting, diarrhoea, haematemesis, and massive fluid loss.

After acute or chronic mercury exposure delayed sequelae (usually neurological and renal dysfunction) tend to manifest several weeks later.

Two overlapping neurological syndromes recognised are neurasthenia (fatigue, headache, depression, and psychomotor complaints), and erethism (anxiety, emotional lability, insomnia, irritability and delirium). This classically affected those working in the felt hat industry coining the term the Mad Hatters syndrome.¹³

Reports vary as to whether these manifestations resolve or are permanent. Investigation of mercury toxicity should include FBC, U&E and blood and urine mercury estimation. For acute ingestion of inorganic mercury blood type and cross match should be taken because of the potential for gastrointestinal tract haemorrhage or perforation.

Most laboratories that measure mercury levels do so on a weekly basis, although samples can be done immediately in an acute exposure.

The assumption of elemental ingestion after the finding of radio-opaque globules on a plain radiograph is incorrect, as other forms of mercury are also radio-opaque. Abdominal, chest or soft tissue films can be obtained depending on exposure.

As our patient had no acute symptoms, nor any absorption modifying gastrointestinal disease, it must be assumed that she ingested an organic compound to account for her high levels.

Treatment of mercury poisoning entails having an initial high index of suspicion. As with all acute poisonings general supportive measures are paramount. Chelating agents are

used to increase elimination. Plasma exchange¹⁴ or haemodialysis can aid these. Treatment should be considered in all patients because of the possibility of long term neuropsychiatric problems.

Authors' affiliations

M Poulden, Emergency Unit, University Hospital of Wales, UK
Correspondence to: Mr M Poulden, c/o Accident and Emergency Department, Morriston Hospital, Swansea SA6 6NL, UK;
Markpoulden@hotmail.com

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