Case Series

Occupational Metallic Mercury Poisoning in Gilders

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Abstract

Occupational exposure to elemental mercury vapor usually occurs through inhalation during its utilizations. This leads to a variety of adverse health effects. In some Islamic cities, this type of poisoning may occur during gilding of shrines using elemental mercury with gold. Herein, we report on three male patients aged 20–53 years, who were diagnosed with occupational metallic mercury poisoning due to gilding of a shrine. All patients presented with neuro-psychiatric disorders such as anxiety, loss of memory and concentration, and sleep disorders with high urinary mercury concentrations of 326–760 µg/L upon referring, 3–10 days after cessation of elemental mercury exposure. Following chelating therapy, the patients recovered clinically and their mercury concentrations declined to non-toxic level (<25 µg/L). Health, environmental and labor authorities, as well as the gilders should be aware of the toxicity risk of exposure to metalic mercury during gilding in closed environments and act accordingly.

Keywords: Mercury; Mercury compounds; Mercury poisoning; Occupational exposure; Mercury poisoning, nervous system

Introduction

Mercury is naturally produced in small amounts in the form of elemental mercury. Metallic mercury is a silver-colored liquid known as quicksilver. It is an extremely toxic element that possesses two different compounds including inorganic (*eg*, mercurous chloride, mercuric chloride, and mercuric oxide) and organic compounds (*eg*, methyl mercury and dimethyl-mercury).[1] Elemental or metallic mercury is the only metal existing in liquid form. It evaporates at room temperature, and if inhaled for a long time, may well induce toxicity.[2]

Absorption of elemental mercury occurs through inhalation, aspiration, subcutaneous exposure, and rarely, direct intravenous embolization. Volatilization of elemental mercury will drastically be enhanced when it is heated.[1] Occupational exposure to mercury usually occurs by inhalation during manufacturing (*eg*, using this element for fluorescent bulb factory).[3] Amalgam, which is used in restorative dentistry, contains 50% elemental mercury that may be absorbed and increase urinary mercury concentration.[4] There are also studies that illustrate positive relationship between occupational exposure to mercury vapors from amalgam and increased risk of infertility and miscarriage in women.[5,6]

In the processing and preparation of the liquid for gilding, huge amounts of metallic mercury are used to form a blend with gold. This mixture is then heated to vaporize the mercury after applying. This results in a substantial release of mercury leaving the gold to cover the desired surface.[7]

There are reports of acute mercury poisoning following exposure to mercury vapors in smelting of placer gold and extrac-

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tion process in gold mining,[8,9] however, to the best of our knowledge, no cases of elemental mercury toxicity in gilders has so far been reported. Herein, we report on three cases of occupational metallic mercury poisoning due to gilding.

We obtained approval of the University Research Ethics Committee as well as the informed consents from all the patients for their treatment and publishing their information.

Case Reports

General Findings

The patients were all Iranian professional gilders who had been employed to gild a shrine dome in another country for a couple of months. In the process of gilding, all guilders mixed liquid mercury with gold powder to create a blend of gold; they then daubed it on the surface of walls of interior surfaces of the shrine building. Then, the superficial layer was heated to extremely high temperatures to vaporize the mercury leaving the gold on the surface. Unfortunately, during this process, the workers had no suitable gloves, goggles and masks. They only used latex gloves and textile masks. The ventilation in the area was inadequate; the ventilation system was installed at top of the fourth floor of the four-story shrine—two floors above the place where the workers were gilding. Moreover, there was a canister of activated charcoal on the third floor, which was exchanged every 4–7 days. The three men worked 6–8 hours a day for approximately 20–50 days. The patients suffered from neuropsychiatric disorders after they returned home in Mashhad, where they were referred to a medical toxicologist for consultation. Following clinical examination, occupational metallic mercury poisoning was suspected. Urinary mercury concentration was measured by an atomic absorption spectrometer (Perkin Elmer, Model 3030) using the mercuric hydride system (MHS) in the Toxicology Laboratory of the Medical Toxicology Research Center.

For all patients, additional laboratory tests including urinalysis, serum sodium, potassium, calcium, creatinine, CBC, ESR, and CRP, were requested; all results were within the normal range. Further investigations such as spirometry, chest x-ray, NCV, and EMG were also normal in all patients. The patients were treated as outpatients.

Case 1

The patient, a 30-year-old male, worked as a guilder eight hours a day for 50 consecutive days. He then developed weakness, malaise, excessive diaphoresis, coughing, fever, shortness of breath, diarrhea, dysphasia, and polyuria. He therefore stopped working and returned to his home town, where he was visited a private physician and then a medical toxicologist. Clinical examination revealed anxiety, restlessness, memory weakness, insomnia, and depression. He had a blood pressure of 105/70 mm Hg, pulse rate of 78/min, and respiratory rate of 18/min. His deep tendon reflexes were exaggerated; no other sign was found. The initial urinary mercury concentration three days after cessation of elemental mercury exposure was 760 μg/L. Dimercapto succinic acid or succimer was administered orally 200 mg, tid, for two weeks, followed by 200 mg, bid, for another two weeks. After the first chelating therapy, his urinary mercury concentration reduced to 348 μg/L. After two weeks, another course of succimer was administered and the urinary mercury concentration declined to 130 μg/L. Garlic tablet, 400 mg allicin, tid, was then prescribed for three months. The final urinary mercury concentration almost six months of the treatment was 25 μg/L. The patient was then symptom free. He was advised to

avoid any chemical exposure particularly to mercury.

Case 2

This 20-year-old man complained of a metallic taste and mild anorexia during his gilding work. He experienced 8 kg weight loss after 60 days of gilding (8 hours daily). No obvious neuropsychiatric, pulmonary or gastrointestinal signs were found on physical examination. His initial urinary mercury concentration measured one week after cessation of elemental mercury exposure, was 635 µg/L; the level reduced to 351 µg/L after the first course of the treatment with succimer as described for the first patient. We also prescribed garlic as was done for the first case, however unfortunately, the patient did not return on his appointments and just informed us in a telephone conversations that he was doing fine.

Case 3

This 53-year-old man had two periods of metallic mercury exposure, each of which lasted for almost 50 days. He had persistent cough, dyspnea and insomnia. His vital sign was normal except for a blood pressure of 165/100 mm Hg. On his physical examination, deep tendon hyperreflexia was found the only abnormal finding. His urinary mercury concentration 10 days after cessation of elemental mercury exposure was 326 µg/L; the level declined to 20 µg/L after the treatment given as described for case 1.

Discussion

For centuries, mercury has been used in industry. Being a frequent component in numerous medications, mercury has also been used widely in medicine. Widespread use of metallic mercury in manufacturing fluorescent light bulbs, thermometers, barometers, dental amalgam, and glass-blowing industries has made the elemental mercury vapor, an important cause of occupational, accidental and even intentional exposures.[10,11] Clinical manifestations and route of exposure in some patients presented with metallic mercury poisoning are presented in Table 1.

Cultural and ritual use of metallic mercury may also lead to exposures to high levels of mercury vapor.[19] Mercury has also been used in gold mining to achieve a stable compound of gold and mercury, which is then heated to melt the mixture and separate the gold. Workers in such jobs may expose to significant amounts of mercury if the workplace sanitation control is not properly considered. Exposure to mercury vapor can occur through using paints containing phenyl-mercuric compounds. Mercury vapors also release while the paints are drying.[20]

The only biologically significant route of absorption of elemental mercury is inhalation. Being quickly partitioned to other tissues, the initial half-life of metallic mercury following a single exposure is around three days. Because of this short half-life in the blood, there is trivial correlation between blood and urinary mercury concentrations. Normally, mercury is excreted by the kidneys. Nevertheless, in exceedingly high exposures, the major route of elimination of mercury is exhalation.[20] Neurotoxic effects of metallic mercury vapor are attributable to the divalent mercury ions formed through oxidation in the brain, which are more toxic than monovalent elemental mercury compounds. One possible mechanism is interference with enzyme functions by binding to their sulfhydryl groups. Transport through the cell membrane via the formation of carrier complexes can be considered as another possibility.[21]

Studies have revealed that humans exposed occupationally to metallic mercury or amalgams had a significantly more in-

cidence of lymphocytic aneuploidy but not structural chromosomal aberrations compared to a control group. Chronic absorption due to handling of mercury or exposure to its vapors has led to a characteristic discoloration of the anterior surface of the lens.[22] In another study on 75 workers exposed to mercury vapor in a glass manufacturing factory, six experienced insomnia and one had tremors. One-third of the workers had hyper-excitability and 20% had tremors. Based on such exposures and hazards, mercury is listed as a hazardous air pollutant generally known or assumed to cause serious health risks. According to the World Health Organization,[23] the 8-hour time-weighted average (TWA) for elemental and inorganic mercury is 25 μg/m^3. According to the National Institute for Occupational Safety and Health (NIOSH), the Recommended Exposure Limit (REL) for mercury is 50 μg/mL, as a TWA exposure for up to 10 hours a day, 40 hours a week.[24]

Table 1: Clinical manifestations and route of exposure in some patients with metallic mercury poisoning reported from different countries.

Age (yrs)/ sex	Route of exposure	Clinical manifestations	Elemental mercury concentration (μg/L)	Treatment	Country	Reference
67/Male	Ingestion, inhalation (suicidal attempt)	Severe pneumonitis, ARF*, anuria	Blood: 1577	Hemodialysis, No chelating therapy	Japan	12
46/Female	Manometer used in the arterial line	Severe pain, ischemia, erythematous lesions, cyanosis of the left hand	Blood: 192.9	Nitroglycerin patches, codeine, acetaminophen, and penicillamine	Brazil	13
40/Male	Self-injection of elemental mercury	Schizophrenia and inflammatory soft tissue lesions	24-h urine: 6	Broad-spectrum antibiotics, surgical debridement of necrotic tissue	USA	14
21/Male	Self-injection of elemental mercury by breaking thermometers	Granuloma in the antecubital fossa	Serum: 11	Surgical removal of mercury	Georgia	15
22/Male	Repeated self-administration of metallic mercury injection	Arthromyalgias, fever, weakness, chest pain (multiple punctuates metallic densities in radiographs)	Blood: 370	Mercury micro-emboli management	Spain	16
36/Male	Chronic mercury vapor exposure and possible iv injection	Rash, sore throat, fever, chills, cough and diarrhea	N/A	Chelating therapy with DMSA† and DMPS‡	USA	17
36/Female	Heating the liquid form of mercury	Abdominal pain, diarrhea and fever	Not tested on admission—blood: 300 at discharge	Symptomatic chelation treatment with N-acetyl cysteine (NAC)	Turkey	18

*ARF: Acute renal failure; †DMSA: Dimercaptosuccinic acid; ‡DMPS: Dimercapto-propane-sulfonic acid; N/A: Not available

Measurement of blood mercury level is important in acute mercury poisoning, if the samples are taken within a few days of exposures. However, a 24-hour urine sample is more indicative of the exposure.[25] Based on our experience, collection of 24-hour urine is not reliable in Iranian outpatients; we thus decided to use morning urine samples. The normal range of mercury concentration in whole blood and urine is generally considered <10 µg/L and <25µg/L, respectively.[20] Nonetheless, since organs other than blood can concentrate the mercury at higher levels in chronic mercury poisoning, there are reports that indicated no precise correlation between urinary mercury concentration and clinical manifestations.[26]

Although our patients were poisoned with metal mercury vapor via inhalation, they had no clinically significant signs; they presented with only mild clinical manifestations, mostly different symptoms (Table 1). In contrast, their urinary mercury concentrations were much higher than the upper normal limit. This might be due to the fact that when exposure to mercury vapor is stopped, the blood mercury reduces quickly. Therefore, blood testing is only useful for continuous or very recent exposures. For the metallic mercury in the body eliminates mainly via the urinary system, urine samples are the best indicators to assess the chronic exposure to metallic mercury. Although, urinary mercury concentrations above 25 µg/L indicates exposure to mercury, clinical symptoms and signs of toxicity will not necessarily appear for some times after chronic exposure.[25]

The initial symptoms of acute elemental mercury inhalation may occur within hours of exposure in some patients. The symptoms include cough, chills, fever, and shortness of breath. Gastrointestinal complaints such as nausea, vomiting, and diarrhea might also happen, accompanying by a metallic taste, dysphagia, salivation, weakness, headaches, and visual disturbances. However, the lethal dose of inhaled elemental mercury has not been described.[20]

In addition to the previously mentioned symptoms, numerous neurological, reproductive, pulmonary, renal, muscular, and dermal symptoms have also been reported following exposure to metallic mercury;[27-30] none of these symptoms was observed in our patients.

Diagnosis of chronic mercury poisoning is usually based on a history of occupational exposure, clinical manifestations, and high concentrations of urinary elemental mercury. However, the initial diagnosis can be complicated, as many of the clinical findings resemble those of neurological diseases, vitamin or mineral deficiencies, and psychological disorders.[1,29]

Generally, the initial step in the management of occupational poisonings is to withdraw the patient from the site of the exposure, which may be sufficient in mild symptomatic patients. However, in those with high urine or blood mercury concentrations and in patients with significant clinical manifestations, chelating therapy must be considered.[29] We used "meso 2,3-dimercaptosuccinic acid or DMSA," commonly known as succimer, which has been approved by the US Food and Drug Administration (FDA).[31] DMSA can eliminate mercury stronger than N-acetyl-D, and L-penicillamine, by decreasing mercury levels in tissues and improving its excretion.[32]

We also prescribed garlic tablets since it has been shown that garlic can reduce accumulation of some heavy metals and effectively reduce clinical manifestations in occupational poisoning.[33] Moreover, garlic can effectively protect rats' liver against accumulation of cadmium and mercury while administering before, with or after exposure to such heavy metals. Possible mechanisms of action are perceived as

For more information on occupational exposure to mercury among workers in a fluorescent lamp factory see http://www.theijoem.com/ijoem/index.php/ijoem/issue/view/21

involving in chelating and preventing the absorption, uptake, accumulation, and excretion of heavy metals.[34]

Protection guidelines for workers should be the first step in preventing mercury vapor poisoning. In areas with excessive exposures to mercury, respiratory protection must be applied by full-face canister type mask or supplied air respirator, depending on the concentration of mercury vapors. Concentrations above 50 mg elemental mercury/m^3 of elemental mercury in work environment requires supplied air and use of positive-pressure full-face respirators. Full bodywork clothes including shoes or shoe covers, goggles, suitable gloves and hats must also be applied.[35]

In conclusion, occupational poisoning due to exposure metallic mercury may occur during gilding process. Training the gilders in potential health hazards of mercury exposure and utilizing suitable protection is imperative. Moreover, the health, environmental and labor authorities should be aware of the risk of toxicity of metal mercury exposure during gilding, particularly in closed environments, and act accordingly.

Conflicts of Interest: None declared.

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