

Acute Mercury Vapour Poisoning in a Shipyard Worker-A Case Report

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Abstract

Acute mercury vapour poisoning is a serious, potentially fatal but fortunately rarely encountered problem. It is most commonly due to industrial accidents. The vapour is a direct respiratory tract irritant as well as a cell poison, exerting its greatest effects in the lungs, nervous system, kidneys and liver. We present a case of mercury vapour poisoning in a shipyard worker presenting as an acute chemical pneumonitis, which resolved with aggressive supportive therapy. Further investigations later revealed transient mild neuropsychiatric symptoms, and residual peripheral neuropathy. No chelation therapy was instituted. The detailed investigative work that led to the discovery of the source of mercury is also presented.

This case alerts us to the potential hazard to shipyard workers who may work in ships previously carrying oil contaminated with mercury. There have been no previous reports of mercury poisoning in shipyard workers. A high index of suspicion leading to early diagnosis and institution of appropriate supportive measures in suspected cases can be life-saving.

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Introduction

Despite improvements in industrial working conditions, mercury remains second only to lead as a cause of heavy metal poisoning.¹ Cases of poisoning by inhaled mercury vapour, though rare, are well documented in the literature. The manifestations differ with regard to the duration and concentration of exposure. Acute exposure to high concentrations predominantly affects the lung, causing respiratory symptoms and pneumonitis in severe cases, whereas chronic low-dose exposure affects the nervous system, causing tremors, neuropathy and psychological changes.^{1,2} In both settings, there have been many cases of morbidity and mortality.

The following is a case of acute mercury vapour poisoning in a shipyard worker presenting as a severe interstitial pneumonitis, as well as mild neuropsychiatric symptoms. Lung function improved without chelation therapy to near normal after nine months. There was, however, mild residual peripheral neuropathy.

Case Report

Clinical History

A 48-year-old Chinese man was admitted with symptoms of worsening dyspnoea after fume inhalation at work the previous day. He was a steelworker in a shipyard, and was cutting steel plates with an oxyacetylene torch in the hull of a ship, with only a towel over his face for protection. It was his first day working on this particular ship. That evening after work, he developed dyspnoea, which worsened gradually through the night, associated with mild left sided chest discomfort. He finally sought medical attention the next day, approximately 24 hours after exposure.

On admission, physical examination revealed a febrile and tachypnoeic middle-aged man, with tachycardia and bilateral basal crepitations on auscultation of the chest. Arterial blood gas analysis yielded the following results: pH 7.38, pCO₂ 38.7 mmHg, pO₂ 62.6 mmHg and oxygen saturation of 91.2% on 4 L/min supplemental

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oxygen. His chest radiograph showed diffuse infiltrates in both lung fields (Fig. 1). He also had marked leukocytosis of $29.5 \times 10^9/\text{L}$.

Based on the history and clinical features, a diagnosis of chemical pneumonitis secondary to fume inhalation was made and the patient was promptly transferred to the Medical Intensive Care Unit for closer observation, with preparations made for ventilatory support in the event of further deterioration. He was started immediately on intravenous hydrocortisone, and because the differential diagnosis of severe community-acquired pneumonia could not be ruled out, was also given intravenous ceftriaxone and erythromycin.

Over the next few days, there was gradual improvement in the patient's condition. He became less tachypnoeic, and arterial blood gas analysis on the fourth day after admission showed the following results: pH 7.40, pCO_2 33.7 mmHg, pO_2 84.0 mmHg and oxygen saturation of 96.4% on 60% supplemental oxygen. The patient had a high-resolution computed tomography (HRCT) scan of the thorax 1 week after admission which showed diffuse changes in the lung parenchyma. The prominent interlobular septal thickening, peribronchial thickening and subpleural lines were all consistent with interstitial lung disease (Fig. 2). A lung function test revealed the following results: FVC 1.73 L (51% of predicted value), FEV₁ 1.56 L (57%), FEV₁/FVC 90%, and DLCO 15.2 mL/min/mmHg (69%), corresponding to a restrictive defect.

As the patient's symptoms were suspected to be work-related, the Department of Industrial Health was notified and the work-site investigated. The possibility of heavy metals contaminating the paint coating the steel plates led to toxicological analyses for lead, mercury and cadmium, as both mercury and cadmium poisoning were known causes of acute pneumonitis. The patient's urine mercury levels were found to be grossly elevated at 284.8 ug/L (normal <50 ug/L), thus confirming the diagnosis of mercury poisoning. His blood cadmium and lead levels were normal.

The patient recovered well without chelation therapy. His respiratory symptoms improved gradually, a 24-hour urinary creatinine clearance test was normal and no proteinuria was detected. He was well enough to be discharged 18 days after admission. A repeat urine mercury analysis before discharge showed a level of 63.3 ug/L.

Neurobehavioural testing using the World Health Organisation Neurobehavioural Core test battery (WHO-NCTB)³ performed one month after the incident showed poor scores for motor speed (Santa Ana test, digit symbol, trail making) and steadiness (aiming) compared to the reference group. He also complained of irritability, restlessness, depression and numbness of fingers. Nerve

conduction studies showed diminished velocities and amplitudes for both median nerves suggesting some degree of peripheral neuropathy. A follow up visit three months after the incident showed that he had no further complaints of irritability or restlessness. However, there was no significant improvement in the nerve conduction studies at that time.

The patient's pulmonary function continued to improve. Serial lung function tests showed significant improvement at 3 months, and a return to normal 9 months after exposure (Table I), while a repeat high resolution CT scan at 9 months post-exposure showed only very mild residual interstitial shadowing in the upper zones (Fig. 3), indicating a significant recovery.

Occupational History and Exposure

The patient's job as a steelworker was to cut away old corroded steel plates in a storage tank at the bottom of the ship's hull using an oxyacetylene flame, and then replace them with new ones. The workers were provided with ventilation through a portable ducting and cartridge respirators, which they did not use. The ventilation was only adequate for non-toxic metal fumes as the steel plates were not expected to contain toxic metals.

However, chemical analysis of the surface scrapings from the steel plates revealed a very high mercury content ranging from 900 to 3500 ppm (0.09% to 0.35%). By comparison, the scrapings from another ship was 3 ppm (0.0003%). Industrial hygiene monitoring of mercury vapour-in-air during simulated flame cutting of the steel plates showed levels exceeding 30 times the permissible occupational exposure limit of 0.025 mg/m³.

Identification of the Source of Mercury

Initially, it was thought that the mercury originated from the old paint coating the steel plates. However, subsequent discussion with the ship owners revealed that it was most likely to have come from a previous

TABLE I: SERIAL PULMONARY FUNCTION TEST RESULTS

	During admission	2 months	9 months
FVC (L)	1.73 (51% predicted)	2.54 (75%)	3.00 (91%)
FEV ₁ (L)	1.56 (57%)	2.10 (78%)	2.60 (100%)
FEV ₁ /FVC (%)	90	83	85
VC (L)	1.79 (59%)	2.54 (69%)	2.4 (64%)
DLCO (mL/min/mmHg)	15.2 (69%)	6.36 (152%)	Not done due to technical fault

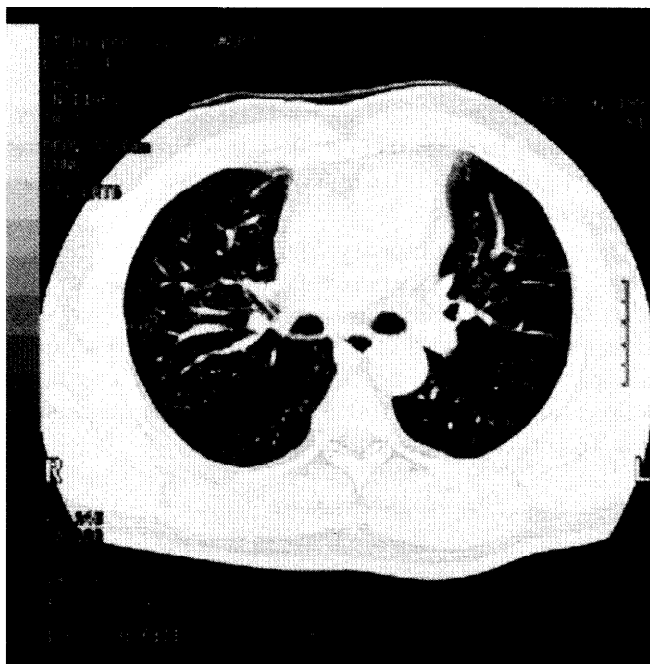


Fig. 1 Chest X-ray of the patient on admission showing diffuse bilateral infiltrates

cargo. This 30-year-old ship owned by an oil company had been used as a floating storage unit to store oil condensate from an oil/gas field in a foreign country. It was last used for storage a few months prior to coming to Singapore for repair work. Normal crude oil may have mercury content ranging from 0.02 to 30 ppm (0.000002% to 0.003%). This particular oilfield was known to be rich in elemental mercury, and some of it could have been deposited on the inner surface of the storage tanks. With high temperature work such as flame cutting, vaporisation of the mercury could occur and result in poisoning by inhalation, especially inside the confined space of a storage tank.

Epidemiological Investigations

Screening of the other workers who had carried out similar work on this vessel during the same period revealed another 7 with high urine mercury content exceeding the biological threshold limit value of 50 µg/L. Four of them had symptoms suggestive of mild pneumonitis and of these, 3 had urine mercury levels exceeding 100 µg/L. A random sample of 4 steelworkers who had not worked on this particular ship showed urine mercury levels of 5 µg/L or less.

Implementation of Control Measures

Following confirmation of the diagnosis, all hot work on that particular ship was stopped and the surfaces of the steel plates were blasted to remove the mercury prior to further work. All workers with high urine mercury levels were not allowed to work on the ship, while mercury vapour respirators were provided for those

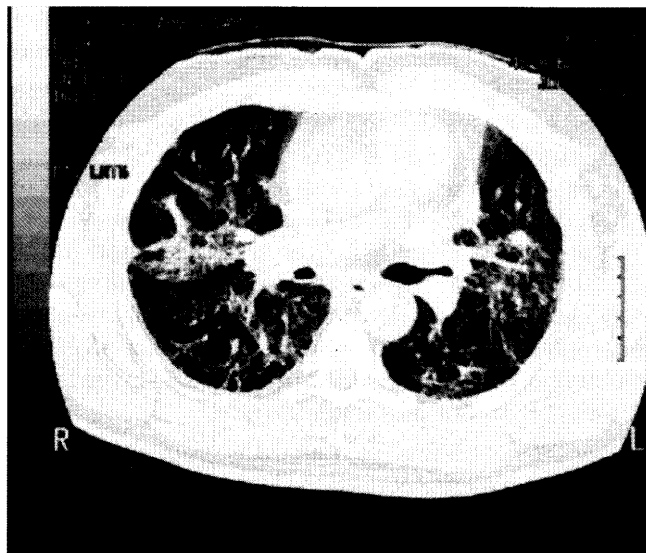


Fig. 2 High resolution CT scan of the thorax 1 week after admission showing typical changes of diffuse interstitial lung disease.

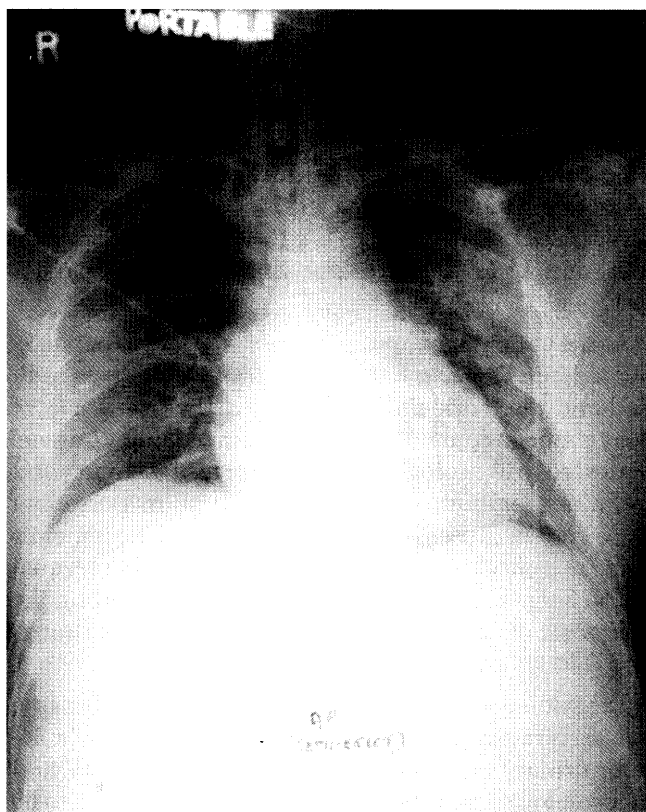


Fig. 3 High resolution CT scan of the thorax 9 months post-exposure. Near normal CT study indicating recovery.

involved in hot work. Regular monitoring of the mercury-in-air levels and urinary mercury levels of workers performing hot work showed that there was no further mercury hazard. A health hazard alert circular was sent to all shipyards informing them of this case and the need to check for any mercury hazard before carrying out hot work on floating storage units and oil tankers.

Discussion

Acute mercury inhalation exposure tends to occur in 3 settings, namely industrial accidents (61%), novice attempts to extract precious metals from amalgams (24%) and home accidents (15%).² In Singapore, industrial accidents are almost solely responsible for the rare cases that occur.

Mercury is readily and rapidly absorbed through the alveolar membrane and may be deposited in the kidneys, liver, brain and oral mucosa. The main toxic effects in acute poisoning involve the lungs and kidneys,² but tissue levels of mercury do not correlate well with organ toxicity.⁷

Mercury vapour acts as a direct airway irritant and a cellular poison.^{2,5} Chemical pneumonitis following acute exposure is well documented, presenting as dyspnoea and chest discomfort with or without cyanosis, as in our patient.^{2,5} Cough is not a major feature. Pulmonary toxicity seems to be related to local irritant effects created by oxidised mercury ions.² There may be direct damage to bronchial and parenchymal cells with resultant acute lung injury. Mercuric ions are believed to bind predominantly to sulfhydryl groups and may thus disrupt sulfhydryl containing enzyme systems, contributing to the injury cascade. Post-mortem studies have shown damage to the bronchi and bronchioles, with marked alveolar oedema and areas of necrosis.^{4,5} In fatal cases, the clinical picture of adult respiratory distress syndrome has been well documented.^{2,4} Complications such as pneumothorax and pneumomediastinum can occur and are usually pre-terminal events.² Lung function tests usually reflect a restrictive picture, manifesting as loss of lung volumes and diminished diffusing capacity,⁵ as seen in our patient. Some patients also present with an obstructive picture, possibly due to bronchiolitis, but this usually appears later in the course of the disease.^{5,7} Good short-term clinical recovery of lung function usually occurs in survivors, as seen in our patient again, but mild permanent damage from bronchiolitis obliterans or residual interstitial fibrosis is a possibility.^{4,8}

Extra-pulmonary toxicity following inhalation of mercury vapour has also been reported, with the main pathology being acute tubular necrosis involving the proximal renal tubule.⁴ However, not all patients show laboratory evidence of renal dysfunction, despite subsequent post-mortem findings of acute tubular necrosis. In our patient, both 24-hour urinary total protein and creatinine clearance tests were normal, and thus we were unable to determine if renal damage had occurred clinically. Neurological abnormalities described include irritability, delirium, ataxia, seizures and neuropathy,^{4,8} some of which were found in our patient.

The generally accepted treatment of mercury poison-

ing is chelation therapy with penicillamine or dimercaprol (BAL), which was found to be effective in increasing its excretion by some authors,^{4,7} but not by others, who question its value.^{2,5} Our patient improved significantly without chelation therapy, thus illustrating, along with 2 other cases,^{8,9} that chelation therapy, though well established, is not absolutely necessary in acute mercury poisoning. Corticosteroids are also of unproven benefit. Even though they may retard progression to severe interstitial fibrosis, they apparently do not improve the outcome of patients with acute lung injury.^{6,8}

As in all industrial hazards, location of the source is of great importance once a definite diagnosis is made. If this worker had not been correctly diagnosed to have mercury poisoning and the source identified, more could have been exposed, some chronically, as work on this ship had been expected to last for several months. As it were, several other workers had already been exposed, though none developed severe symptoms. Early diagnosis led to the cessation of hazardous work and introduction of preventive measures, thus eliminating further cases of acute and possibly chronic poisoning.

To our knowledge, there have been no previous reports of mercury poisoning among shipyard workers. Occupational exposure to mercury has been reported in a natural gas plant,¹⁰ and is a known hazard in the petroleum industry but little known in the shipbuilding and repair industry. In this case, the early diagnosis and identification of the source of mercury enabled us to alert the shipbuilding and repair industry to this little known hazard and hopefully prevent further cases of mercury poisoning in this occupational setting.

Conclusion

In conclusion, acute mercury vapour poisoning, a severe and potentially fatal condition, should be borne in mind when faced with cases of pneumonitis following inhalation of fumes in a shipyard worker involved with hot work. A high index of suspicion leading to early diagnosis and prompt and aggressive institution of supportive therapy can be life-saving and prevent further cases of poisoning.

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