An unusual cause of high density radiological opacities

Abstract

Introduction: Metallic mercury poisoning through intravenous injection is rare, especially as part of a suicide attempt. Diagnosis and treatment of the disease are challenging as clinical features are not specific.

Material and methods: A 41-year-old male presented with dyspnea, fatigue, loss of weight, and loss of appetite over two months. Routine radiological examination by chest X-ray and CT showed randomly distributed high density opacities with Hounsfield units (HU) around 500 HU all over the body. The diagnosis was then confirmed with a urinary mercury concentration of > 1000 mcg/24 h.

Results: The patient’s clinical condition was getting worse in spite of chelation therapy and hemodialysis. The patient eventually died because of respiratory failure.

Conclusion: Early diagnosis and appropriate treatment are critical for intravenous mercury poisoning especially because there are no specific signs or symptoms. There should be a high level of suspicion in drug abusers. Treatment should involve the combined use of chelating agents and other treatments such as hemodialysis and plasma exchange in advanced clinical settings.

Key words: intravenous injection, mercury poisoning, high density opacities

Introduction

High density opacities on chest X-ray are an infrequent finding and are highly nonspecific. When such a radiological picture is encountered the differential diagnosis includes pulmonary alveolar microlithiasis, pulmonary hemosiderosis, metal poisoning, barium aspiration, pellet injury, and possible remnants of contrast used for lymphangiography or myelography. However, a thorough history taking, physical examination and other supportive investigations will help in achieving a proper diagnosis. Though uncommon, mercury poisoning can be seen in patients who are frequent drug abusers as well as in individuals working in the manufacturing of industrial chemicals, paints, explosives, batteries, thermometers, electronic instruments, etc.

Mercury exists in elemental, inorganic, and organic forms. Elemental mercury causes pulmonary, neurologic and nephrotoxic injury and its poisoning results most commonly from vapor inhalation as it is absorbed (80%) throughout the lungs [1]. Poisoning due to intravenous injection of mercury usually occurs in connection with attempted suicide, by accident, or in drug addicts exploring new ways to become intoxicated [2].

Case details

A 41-year-old male presented with complaints of breathlessness and cough with watery expectoration for the past 2 months. He also complained of loss of appetite and loss of weight (around 6–7 kg in 2 months). He was a painter by occupation with no known comorbidities. He was a chronic smoker and alcoholic with events of binge drinking for the past 15 years. He also had a history of recurrent IV drug abuse in the last 6 months and it had increased in frequency over the past two weeks before presenting to emergency.

On examination, the patient was conscious, oriented, and afebrile. The patient had a blood pressure of 106/86 mm Hg, a pulse rate of 106/min, a respiratory rate of 26 cycles/min, and saturation was 93% in room air that improved to 96–98% with 2 L oxygen supplementation with nasal prongs. Puncture marks along with thrombosed veins were seen in the right antecubital fossa extending on to
the forearm (Figure 1). Chest auscultation revealed bilateral normal vesicular breath sounds. There were no signs and symptoms of other organ involvement. Blood urea, serum creatinine, and urine analysis were normal. Arterial blood gas analysis (ABG) showed a pH of 7.48, a pCO$_2$ of 24 mm Hg, HCO$_3$ of 23 mmol/L, and a PO$_2$ of 73 mm Hg which were all suggestive of respiratory alkalosis.

Chest X-ray showed multiple punctate and amorphous opacities of metallic densities noted in bilateral lung fields (predominantly in the lower lobes) and overlying the cardiac silhouette (Figure 2). An X-ray of the abdomen showed similar opacities over the liver, kidneys, and dorso-lumbar spine. A lateral radiograph of the skull was found to be normal. CT of the chest showed multiple diffusely distributed metallic densities in bilateral lungs, the pericardium, liver, and kidneys as well as in multiple vertebral bodies and foramina (Figure 3). These features were compared with similar scenarios in literature and thus, IV mercury poisoning was suspected [3–7]. CT Hounsfield unit (HU) values of the high density opacities were 179 to 1065 (mean 527 HU, median 566 HU). Hence, 24 hour urine mercury (ICP-MS) was measured and was found to be > 1000 mcg/24 h. The patient was prescribed the chelating agent penicillamine given at doses of 500 mg orally every 6 hours in combination with pyridoxine (vitamin B6) in doses of 20 mg/day. The patient’s condition was worsening in spite of chelation therapy hence hemodialysis and plasma exchange were used. Unfortunately, the patient’s condition deteriorated and he ultimately died due to respiratory failure.

Discussion

Elemental mercury, when injected intravenously, can cause widely varying presentations. It can be asymptomatic in some patients while causing respiratory failure, kidney damage, liver damage, neurologic symptoms, and even death in others. Once mercury enters the bloodstream, it’s quickly distributed throughout the body, particularly in the lungs as its absorption through the lungs is 80% [8]. Due to its high water–metal interfacial tension and lack of bonding to other materials, mercury either takes the form of tiny spherules or coalesces when it enters the plasma. Because of gas exchange and the extensive capillary network of the lungs through which blood flows back to the heart, the lungs can easily become a depository for the exogenous mercury. Because of this reason, the lungs are often more seriously affected than the other organs when examined on imaging and can give rise to a distinctive radiological appearance. In cases of intravenous self-administration in the forearm veins like in our case, aggregations of mercury at the site of injection may also be radiologically seen [9]. Radiologically small metallic spherules of different sizes scattered throughout the lungs or beaded chains along the pulmonary vasculature and other organs of the body can also be observed [10]. Mercury is mainly excreted from the body by the kidneys, but the rate of excretion is usually very slow and traces of mercury can be seen on radiographs and in urine even two years after the index event [8]. The total body burden of mercury can be determined by assessing twenty-four hour urinary levels of mercury. This total will serve as a useful marker to determine the
need for chelation therapy and gauge the prognosis in some cases. Dimercaprol (BAL, British anti-Lewisite) or succimer, an orally administered analogue of BAL, are effective chelators utilised for treatment. Chelation therapy is the mainstay of treatment because these agents increase the urinary excretion of mercury [11]. Combined use of chelating agents and other treatments like hemodialysis and plasma exchange will enhance the elimination of mercury from the body in severe poisoning.

**Conclusion**

A clinician must take mercury poisoning into consideration when the imaging results shows radiopaque deposits that are unexplainable as early diagnosis and treatment is critical. Findings in a patient’s history such as occupational exposure of mercury and drug abuse are very critical in the diagnosis of mercury poisoning and will affect the patient outcome significantly.

**Conflict of interest**

None declared

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