



ACUTE KIDNEY INJURY DUE TO ACCIDENTAL MERCURY POISONING –A CASE REPORT

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Abstract:

Mercury poisoning can occur as result of industrial poisoning, consumption of sea food, suicidal intent or accidentally as a part of folk remedies used for various purposes. We present a case of a 50 year old male who upon consumption of an unknown substance used as a folk remedy developed typical features of acute kidney injury and oropharyngeal ulceration. The diagnosis was reached at by correlating the history, physical examination, various lab investigations, imaging and renal biopsy results.

Keywords: Mercury poisoning, acute kidney injury.

Introduction

Mercury (Hg) atomic number 80 is a liquid metal at room temperature and pressure. Mercury freezes at -38.9°C and boils at 357°C. It is sometimes called quick silver and is easily alloyed with many other metals, such as gold, silver and tin [1]. It exists in the environment in three forms: elemental mercury (poisonous as vapor), organic mercury (methyl mercury and ethyl mercury) and inorganic mercury (mercuric mercury) and all these forms have toxic health effects [2]. Most human exposure results from fish consumption (organic mercury) or dental amalgam (metallic mercury). Kidneys are the prime target of mercury toxicity as it is primarily excreted through them [3]. Here, we describe a patient who consumed mercuric chloride unintentionally through ingestion of some unknown substance used as an aphrodisiac and presented with typical manifestation such as acute kidney injury (AKI) and oropharyngeal ulceration.

Case Report

A 50 year old male was admitted with 3 days history of oliguria progressing to anuria, lower limb edema and facial puffiness. He gives history of ingestion of some unknown substance (? DAAL CHIKNA) used in folk remedies as 5 days back. It was associated with odynophagia due to oropharyngeal ulceration.

On examination he was conscious, well oriented. His heart rate was 78 bpm, blood pressure of 140/90 mmHg. There was pitting pedal edema and facial puffiness. There was patchy oropharyngeal ulceration. There were no pallor, cyanosis, lymphadenopathy or skin lesions. Systemic examination was normal.

Laboratory investigations showed hemoglobin 13.5; total leukocyte count of 12,000; platelet count of 240,000; urea 217 mg/dl; creatinine 14.8 mg/dl; Na138mEq/L; K 4.7 meq/L; calcium 8.7 mg/dl; phosphate 6.2 mg/dl; total bilirubin 0.56 mg/dl; AST 28 mg/dl; ALT 62 mg/dl; ALP 53; INR 1.2, aPTT34.2; HIV ½ negative; HBsAg negative; Anti HCV negative; Urine showed protein 3+, RBC 3-4, leukocytes 6-8; urine protein creatinine ratio 3.27; ANA negative; p-ANCA c-ANCA negative; Anti GBM negative; C3/C4 normal;

USG showed normal sized kidneys with increased cortical echogenicity; ECG was normal; chest X-ray was normal; Serum mercury level was 250 ug/L.

Renal biopsy revealed severe acute tubular injury. Urine mercury levels were sent once patient started forming urine and the level was 3.7ug/ml.

He was treated with hemodialysis and supportive measures. He received 6 sessions of hemodialysis over 2 weeks after which his urine output started improving.

Discussion

Humans exposure to mercury usually take place via eating mercury contaminated food, dental care procedures (using amalgams in endodontics) using mercury based, thermometers, and sphygmomanometer), occupational exposure (e.g. mining) and others (using fluorescent light bulbs and batteries) [4]. Metallic mercury intoxication was known in ancient times by Aristotle, but that large scale occupational poisoning with mercury had occurred when the great statue of Buddha of Nara was constructed in 8th century in Japan [5]. Mercury is a metallic element, distributed naturally in the environment [6]. There are three classes of mercury: metallic elemental mercury, inorganic mercurial salt (mercurous and mercuric salts),

and organic mercurial. Elemental mercury enters systemic circulation via the skin or inhalation. Acute poisoning of this leads to corrosive bronchitis, pulmonary edema/fibrosis, diarrhea, renal dysfunction, visual and neuropsychiatric disturbances, and in severe cases, death due to respiratory failure [7]. Organic mercury compounds are absorbed completely from the intestine, converted to inorganic forms, and possess similar toxic properties. Most organic mercury exposures leave neurological sequelae [8]. Mercury compounds exert toxic health effects by different mechanisms such as; interruption of microtubule formation, changing intracellular calcium balance and membrane potential, altering cell membrane integrity, disturbing or inhibition of enzymes, inducing oxidative stress, inhibition of protein and DNA synthesis and disturbing immune functions [9]. Mercury binds to phosphoryl, carboxyl and amide groups in biological molecules [10]. Methyl mercury induces oxidative stress and the free radicals may cause neurotoxicity. On the other hand, it has been reported that accumulation of serotonin, aspartate, and glutamate has a role in mechanism of methyl mercury induced neurotoxicity [11]. Inorganic mercury is highly toxic and corrosive. Mercuric chloride is still used as wood preservative, photographic intensifier, disinfectants and also in indigenous drug formulation, and folk remedies in Asian countries [12]. Once ingested, 10% of mercuric chloride is absorbed through the gut. The lethal dose of mercuric chloride is 300–1000 mg. Mechanism of mercury toxicity [13] include (a) mercuric ions precipitate proteins that cause direct necrosis of tissues. About 85–90% of mercury in the body accumulates in the kidneys causing acute renal failure due to necrosis of the proximal tubular epithelium. (b) Inorganic mercury complexes sulfhydryl groups and causes metabolic acidosis, vasodilatation, and shock. Though acute tubular necrosis is the most common lesion, tubulointerstitial nephritis and immune-mediated glomerular damage can also occur [14]. Rarely, it can present as hypertensive encephalopathy especially in children [15], nephrotic syndrome, chronic tubulointerstitial nephritis, or with isolated tubular dysfunction. Our patient consumed mercuric chloride with suicidal intention and developed gastrointestinal erosion, anuric renal failure, and DIC. Renal biopsy showed acute tubular injury [figure1] [figure2] Franco et al[16] reported one patient with mercuric chloride poisoning who developed two consecutive episodes of acute renal failure by two different mechanisms, one toxic and the other immunological. Renal biopsy done in that patient, showed acute tubular necrosis initially and granulomatous interstitial nephritis in the second biopsy. Measurement of mercury levels in blood (> 3.6 µg/dl) and urine (> 15 µg/dl) may be helpful in diagnosis [12]. Treatment for acute ingestion includes activated charcoal for gastrointestinal decontamination.

Emesis is contraindicated. Chelation therapy should be considered for any symptomatic patient with a history of acute elemental mercury exposure [17].

Chelating agents include dimercaprol (BAL), 2, 3-dimercaptopropane-1-sulfonate (DMPS), dimercaptosuccinic acid, and penicillamine. These agents contain thiol groups, which compete with endogenous sulfhydryl groups. Hemodialysis is not effective in removing mercury, but can enhance the removal of the dimercaprol-mercury complexes [18]. The outcome depends on the form of the mercury compound and severity of the exposure. Complete recovery occurs after mild exposure but usually fatal in severe toxicity. Our patient was treated with hemodialysis and supportive measures. His renal function started improving in 2 weeks and is still on regular follow-up. He didn't receive chelating agents in view of anuria /oliguria.

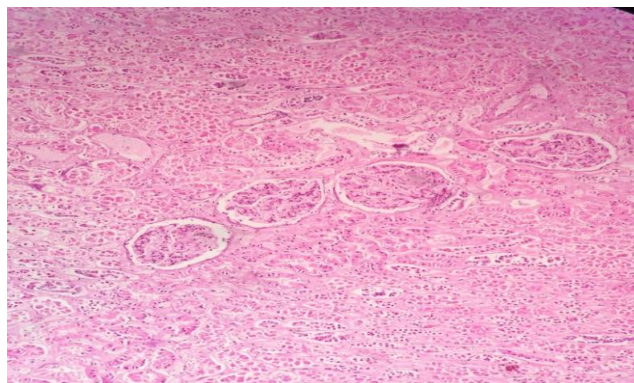


Figure 1: showing necrotic tubular epithelial cells in tubules (H & E 10X)

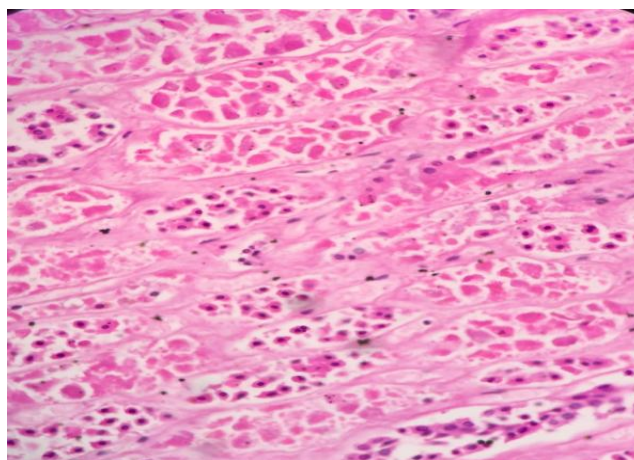


Figure 2: Acute tubular injury showing necrosis of tubular epithelial cells, with many of them detached from the basement membrane and sloughed into the tubular lumen.(H&E 40X)

Conclusion

Besides the obvious causes of mercury poisoning like the consumption of sea food, occupational poisoning, suicidal or homicidal intent, mercury poisoning can occur as a result of consumption of various folk remedies for various purposes such as used as an aphrodisiac in our patient. We conclude that mercury poisoning should be considered in case of AKI with history of exposure to any unknown substance. Prompt treatment with chelating agents guided by measurement of mercury levels will have an impact on the favorable clinical outcome and would prevent the end organ damage thus obviating the need of hemodialysis.

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