Acute kidney injury and disseminated intravascular coagulation due to mercuric chloride poisoning

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ABSTRACT

Mercury is a toxic heavy metal and occurs in organic and inorganic forms. Inorganic mercury includes elemental mercury and mercury salts. Mercury salts are usually white powder or crystals, and widely used in indigenous medicines and folk remedies in Asia. Inorganic mercury poisoning causes acute kidney injury (AKI) and gastrointestinal manifestations and can be life-threatening. We describe a case with unknown substance poisoning who developed AKI and disseminated intravascular coagulation (DIC). Renal biopsy showed acute tubular necrosis. Later, the consumed substance was proven to be mercuric chloride. His renal failure improved over time, and his creatinine normalized after 2 months.

Key words: Acute tubular injury, disseminated intravascular coagulation, hemodialysis, mercuric chloride, renal failure

Introduction

Mercury is the only metal that is liquid at room temperature. All forms of mercury viz., organic, elemental, and mercury salts are toxic and manifestations depend on nature, intensity and the chemical form of mercury.[1] 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.[2] Here, we describe a patient who consumed mercuric chloride with suicidal intent, and presented with typical manifestation such as acute kidney injury (AKI) and gastrointestinal erosion. In addition, he had disseminated intravascular coagulation (DIC), a rare complication of mercury poisoning.

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Case Report

A 36-year-old male was admitted with 2 days history of oliguria progressing to anuria, facial puffiness, edema legs, bleeding gums, hematochezia, and fever. He gave a history of consumption of unknown substance (around 500 mg) a week back used for folk remedies and rituals. He developed dysphagia and oral ulcerations soon after consumption of substance. His heart rate was 92/min, respiratory rate was 15/min, and blood pressure was 130/90 mm of Hg. There was no cyanosis, pallor, lymphadenopathy, or skin lesions. Systemic examination was normal.

Laboratory investigations showed hemoglobin 11.2 g/dl; total count 6000/mm^3; platelet count 100,000/mm^3; blood urea 124 mg/dl; serum creatinine 6.7 mg/dl; sodium 135 mEq/L; and potassium 6.1 mEq/L; arterial blood gas analysis showed high anion gap metabolic acidosis. Liver function tests, electrocardiography, and

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X-ray chest were normal. Ultrasonography showed normal sized kidneys with increased echoes. Upper gastrointestinal endoscopy revealed erosive gastritis. His coagulation profile showed prothrombin time 18 s, INR 1.6; activated partial thromboplastin time 60 s; fibrin degradation products 10 μg/ml; D-dimer levels 1 μg/ml; and serum fibrinogen 250 mg/dl. Diagnosis of overt DIC was made as per criteria proposed by the International Society of Thrombosis and Hemostasis with total score of five at admission.

He was treated with hemodialysis, fresh frozen plasma transfusion, and supportive measures. His coagulopathy improved after 5 days. He received eight sessions of hemodialysis over 2 weeks after which his urine output started improving. Renal biopsy done 2 weeks after admission showed markedly dilated tubules with sloughed off epithelium and cell debris within lumen [Figure 1], interstitial edema, and mild inflammatory infiltrate in the interstitium consistent with acute tubular necrosis. Glomeruli and vasculature were normal. Consumed substance brought by the patient 3 weeks later was white colored powder, called "VEERAM" in local language. Toxicological analysis of the compound revealed it to be mercuric chloride. At the end of 2 months, his serum creatinine was 1 mg/dl.

Discussion

Mercury is a metallic element, distributed naturally in the environment.[3] There are three classes of mercury: metallic elemental mercury, inorganic mercurial salts (mercurous and mercuric salts), and organic mercurials. 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.[4] 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.[5]

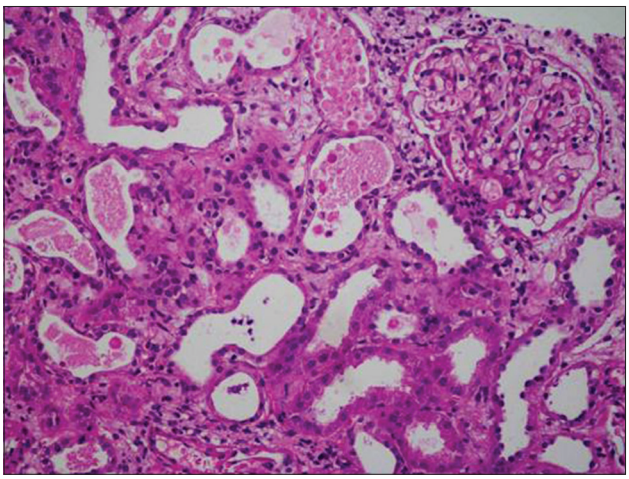

Figure 1: Renal biopsy showing acute tubular injury (H and E)

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.[6] 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[7] include (a) mercuric ions precipitate proteins that cause direct necrosis of tissues. About 85–90% of mercury in the body accumulate 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.[8] Rarely, it can present as hypertensive encephalopathy especially in children,[9] 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. Franco *et al*.[10] 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.

Our patient had features of DIC which is rarely reported in mercury toxicity. The International Society of Thrombosis and Hemostasis criteria, which has 91% sensitivity and 97% specificity, was used for the diagnosis of overt DIC.[11] A score of five or higher is compatible with DIC while a score below five is suggestive of DIC. Our patient had a score of 5. Murphy *et al*.[12] reported a patient with mercuric chloride poisoning and DIC. The possible explanation for DIC was the lowered fibrinolytic activity due to inhibition of plasma plasminogen activator or the inhibition of plasminogen activation reaction catalyzed by this enzyme as demonstrated in experimental rat models.[13] Patients coagulation parameters improved after 5 days with supportive treatment.

Measurement of mercury levels in blood (>3.6 μg/dl) and urine (>15 μg/dl) may be helpful in diagnosis.[6] 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.[14] Chelating agents include dimercaprol (BAL), 2,3-dimercaptopropane-1-sulfona te (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.[15]

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. We did not do urine mercury levels, and the patient had not received any chelating agents as toxicological analysis was done much later. He was treated with hemodialysis and supportive measures. His renal function improved in 2 months and still on regular follow-up. We conclude that mercury poisoning should be considered in case of AKI and DIC though it is a rare complication. Prompt treatment with chelating agents guided by measurement of mercury levels will have an impact on the favorable clinical outcome.

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Conflicts of interest
There are no conflicts of interest.

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