Unusual Manifestation of Mercuric Chloride Poisoning as Intratubular Nephrocalcinosis - A Rare Case Report

Abstract

Mercuric chloride salts are highly toxic compounds that have been linked to suicidal or accidental poisoning. Because of their high solubility, mercuric chloride salts can cause acute tubular injury, corrosive effects in the gastrointestinal system, hematemesis and hematochezia, circulatory collapse, and death. Here, we report an unusual case of mercuric chloride poisoning in a 17-year-old girl who manifested with acute tubular necrosis in association with intratubular nephrocalcinosis and emphasize the role of hemodialysis in the patient's successful recovery.

Keywords: *Acute renal failure, intratubular nephrocalcinosis, mercuric chloride poisoning*

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Introduction

Mercuric chloride compounds are classified as elemental mercury, organomercury (alkyl and aryl forms), and inorganic. Their toxicity, including effects on major target organs, varies due to their kinetics. Acute exposure to inorganic compounds can cause a life-threatening event.[1] Because of its corrosivity and high solubility, mercuric chloride is the most toxic of the inorganic mercury salts.[2] If not treated properly, mercuric chloride can cause toxic effects, primarily on its target organs, including the gastrointestinal tract (GIT) and kidney, in the form of corrosive injury, gastrointestinal disturbances, acute renal failure, later circulatory collapse, and death. The treatment plan includes immediate resuscitation with gastric lavage and close monitoring and aggressive supportive care such as hemodialysis or chelation with 2,3-dimercapto-1 propane sulfonate. We present the case of a young female who recovered successfully after receiving immediate heparin-free hemodialysis and fluid management for acute renal failure.

Case Report

A 17-year-old girl presented to the emergency department of a general hospital with Acute Kidney Injury (AKI) confirmed by elevated serum creatinine and decreased urinary output due to an alleged history of consumption of an unknown poison, quantity unknown (later revealed to be mercuric chloride). Her urine output was 100 mL/24 h at admission, and her blood urea and serum creatinine levels were 458 and 17 mg/dL, respectively. Table 1 includes other lab investigation reports. She was immediately resuscitated with gastric lavage. She was started on heparin-free hemodialysis to manage AKI. Her condition improved after 4 days, evidenced by a decrease in serum creatinine and an increase in urinary output. She was then transferred to a tertiary care hospital for further management. The patient had no history of fever. Laboratory investigations showed 6 mg/dL of serum creatinine and 70 mg/dL of blood urea. She was continued on heparin-free hemodialysis and other supportive therapy, and a renal biopsy was planned and submitted to the histopathology division. The biopsy revealed features of acute tubular injury, characterized by tubulitis with tubule loss and associated intratubular nephrocalcinosis with bluish-purplish nonpolarizable calcium phosphate crystals,[3] and a focus of epithelioid histiocyte collections, as well as mild to moderate interstitial inflammation [Figure 1a–c]. The glomeruli were unremarkable. On further inquiry with family members, the cause for sudden

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Table 1: Lab Investigations

Parameter	Day 1	Day 3	Day 7
Hb g/dL	8.7	10	na
TLC cells/mm^3	6000	9000	na
Platelets lac/mm^3	4.5	6	na
ESR mm/h	18	n.a	na
LDH U/L	824	774	na
Total Calcium mg/dL	10.6	9.7	na
Sr Uric acid	8.2	7.6	na
Urea mg/dL	458	111	70
Creatinine mg/dL	17	10	6
Na+/K+ mmol/L	132/6.3	133/6.5	na
Total CPK U/L	192	58	na
Serum Amylase U/L	124	105	na
Total Bilirubin mg/dL	0.4	0.3	na
SGOT U/L	116	39	33
SGPT U/L	77	59	29
ALP U/L	208	225	126

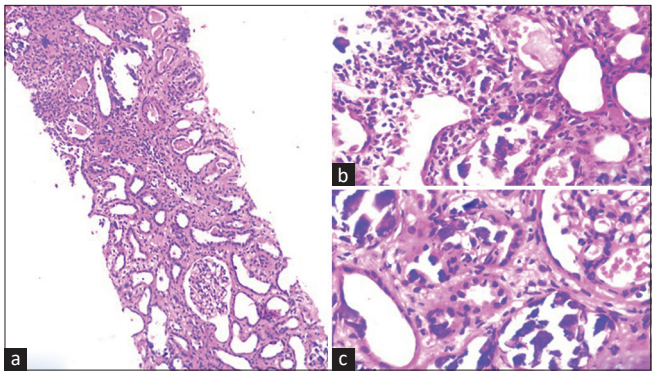

Figure 1: (a) Low-power view of hematoxylin–eosin (H&E)-stained renal biopsy section (100×) revealing evidence of acute tubular necrosis, intratubular nephrocalcinosis, and interstitial inflammation. (b) High-power view of H&E-stained section (200×) – acute tubular injury and intratubular nephrocalcinosis. (c) High-power view of H&E-stained section (400×) – intratubular nephrocalcinosis

AKI was found to be accidental consumption of mercuric chloride. With improved health conditions, she was discharged from the hospital. At the time of discharge, her serum creatinine level was 4.5 mg/dL.

Discussion

Mercuric chloride compounds are seen in three forms: elemental mercury, organomercury (alkyl and aryl forms), and inorganic compounds. Because of their kinetics and toxicity, the effects on major target organs differ. Acute exposure to inorganic compounds can cause a life-threatening event.[1] Due to its corrosivity and high solubility, mercuric chloride is the most toxic form of inorganic mercury salts.[2] Mercuric chloride can cause toxic effects, primarily on its target organs, including the GIT and kidney, in the form of corrosive injury, gastrointestinal disturbances, AKI, later circulatory collapse, and death, if not treated appropriately.

Mercuric chloride ingestion results in extensive precipitation of intestinal mucosal proteins, mucosal necrosis, generalized abdominal pain, bloody diarrhea, and shock. If the patient survives, he or she may develop AKI.[4] In $HgCl_2$ nephrotoxicity, tissue and mitochondrial calcium overload does not occur during the relatively long pre-lethal stage of injury and only develops after morphological evidence of cell necrosis develops.[5] The use of mercuric tablets is a popular method of suicide. An adult's lethal dose of mercuric chloride is 0.2–1 g.[6,7] The term "nephrocalcinosis" refers to the generalized deposition of calcium phosphate or calcium oxalate in the kidney, which can occur within the tubular lumen (intratubular nephrocalcinosis) or in the interstitium (interstitial nephrocalcinosis). Furthermore, depending on the location, it is subdivided into cortical and medullary nephrocalcinosis. Chronic glomerulonephritis, acute cortical necrosis, pyelonephritis, and trauma are common causes of cortical nephrocalcinosis. However, in the present case, we saw evidence of acute tubular injury and associated intratubular nephrocalcinosis, which we cannot totally attribute to mercuric chloride poisoning in the absence of cortical necrosis.[8]

According to Yoshida *et al*.,[9] a similar case of mercuric chloride ingestion was observed, and the patient presented with hematemesis, melena, and severe AKI. Yoshida *et al*.[9] have documented that the role of hemodialysis in the removal of mercuric chloride is minimal. Hemodialysis in combination with plasma exchange therapy, supported by chelation therapy, yields good results in treating mercuric chloride poisoning cases. In the current study, the patient did not receive any chelation therapy. She was managed with hemodialysis for AKI and supportive therapy, and she recovered well. The increased solubility, its manifestation as tubular necrosis and consecutive nephrocalcinosis, and the variations in mercuric chloride concentration may have contributed to the difference in therapy success.

Dhanapriya *et al*.[10] reported a case of mercury salt poisoning with disseminated intravascular coagulation and AKI; however, in our case, we found acute tubular injury with intratubular nephrocalcinosis. The most common lesion is acute tubular necrosis, but tubulointerstitial nephritis and immune-mediated glomerular damage can also occur.[11] It can cause hypertensive encephalopathy, nephrotic syndrome, chronic tubulointerstitial nephritis, or isolated tubular dysfunction in rare cases.[12]

The release of lactate dehydrogenase (LDH) is a highly reliable test for determining the degree of *in vitro* cell death. In their experimental study, Zager *et al*.[13] discovered that with severe AKI, there is a >65% decrease in cortical LDH and a corresponding increase in plasma and urinary LDH, and they concluded that the renal cortical LDH assay determines the extent of the experimental acute ischemic

and toxic renal injury. Similarly, we observed a significant increase in LDH in the present case, indicating toxic renal injury.

Conclusions

Accidental ingestion of mercuric chloride can result in acute kidney injury by tubular injury with associated intratubular nephrocalcinosis in the absence of cortical necrosis and needs to be evaluated further scientifically. Though the role of hemodialysis in the management of mercuric chloride poisoning is minimal, the increased solubility, nature, and dose differences of mercuric chloride, as well as the clinical presentation in the form of acute renal failure, made hemodialysis a key player in successfully managing our case.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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