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CASE REPORT

Mercury poisoning with acute kidney injury

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ABSTRACT

Exposure to inorganic mercury or mercuric salt can occur as an occupational hazard or suicidal attempt and can cause vomiting, severe abdominal pain, gastrointestinal bleeding, hypovolemic shock and renal tubular necrosis leading to oliguria or anuria. Hemodialysis is used in severe cases of toxicity when renal function has declined. This report aims at highlighting the clinical presentation and course of a case of mercuric poisoning who was treated with hemodialysis. This article reports a case of mercury poisoning whose renal failure improved with high flux hemodialysis. A 25 years old girl ingested a heavy metal compound containing Mercuric chloride obtained from her place of work in a deliberate suicidal attempt, following which she developed massive hematemesis, hypotension and developed renal failure with anuria. She was treated with broad spectrum antibiotics, IV pantoprazole and high flux hemodialysis. Renal biopsy was suggestive of acute tubular necrosis. After 7 hemodialysis her urine output began to improve and dialysis was stopped. Her renal function gradually improved and her blood mercury level also decreased. We have here by presented a case of mercury intoxication with acute tubular necrosis in a 25-year old woman, with an excellent improvement of the renal failure and normalization of laboratory results with high flux hemodialysis.

***Key words**: high flux dialysis, renal biopsy, acute tubular necrosis*

INTRODUCTION

Acute poisoning with mercuric salts (typically $HgCl_2$) generally targets the gastrointestinal tract and the kidneys. Extensive precipitation of enterocyte proteins occurs, with abdominal pain, vomiting, and bloody diarrhoea with potential necrosis of the gut mucosa. This may produce death either from peritonitis or from septic or hypovolemic shock. Surviving patients commonly develop renal tubular necrosis withanuria.[1] Historically, mercuric chloride ($HgCl_2$) was used as a preservative and for development of photographic film and was ingested accidentally or as a suicide measure. It is a component of some skin-lightening creams. Only about 2% of ingested mercuric chloride is absorbed initially,[2] although it is believed that its corrosive effect on the intestine may increase permeability and hence, absorption, with prolonged exposure.[3] Much of the body burden of mercuric mercury resides in the proximal convoluted renal tubule[4] bonded to metallothionein.[5] Significant deposition also occurs periportally in the liver[6] and lesser amounts in epithelial tissues, choroidal plexus, and testes. Excretion of mercuric mercury is largely through urine and stool, although significant amounts are shed through sweat, tears, breast milk, and saliva.[7] Half-lives appear to be multiphasic, as with metallic mercury, with human studies suggesting an effective half-life of 42 days for 80% of an oral tracer dose.

Previous reports on the use of extracorporeal procedures such as haemodialysis and haemoperfusion have shown no significant removal of mercury. One case report showed the successful use of the chelating agent 2,3-dimercaptopropane-1-sulphonate (DMPS), together with continuous veno-venous haemodiafiltration (CVVHDF), in a patient with severe inorganic mercury poisoning.[8] Another case report showed the effectiveness of plasma exchange with dimercaprol chelation in normalizing the renal function in acute mercuric chloride poisoning.[9] We present here a case of mercuric chloride poisoning where renal failure was successfully treated with high flux hemodialysis.

CASE REPORT

A 25 years old girl ingested a heavy metal compound containing

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Mercuric chloride obtained from her place of work in a deliberate suicidal attempt. 1hour following ingestion she was taken by her colleagues to a nearby Railway Hospital with complaints of burning pain in the abdomen and burning sensation in the throat. Immediately gastric lavage was done and patient was managed conservatively with intravenous fluids and proton pump inhibitors. Patient did not have any other significant medical history and the patient was not on any regular medication.

Two hours following ingestion she had vomiting followed by a large hematemesis for which she received 2 units of blood transfusion. When the patient continued to have hematemesis despite all conservative measures, she was referred to a higher centre for further management.

On presentation to the emergency department of the Tertiary Care Hospital $4^{1/2}$ hours after ingestion of the mercuric compound she was afebrile, fully conscious but irritable. Her heart rate was 150 beats per minute, respiratory rate 32 breaths per minute and blood pressure was 96/68 mm of Hg. There was no cyanosis or pallor. Initial investigations showed TC- 13512/mm^3, Hb- 9.6g/dl, Platelet count- 139000/mm^3, RBS – 104mg/dl, urea- 44mg/dl, Creatinine- 1.6mg/dl, AST- 212U/l, ALT- 243U/l, ALKP- 360U/l, total bilirubin-1.2mg/dl, total protein 7.2 gm%, albumin 3.7 gm/dl, globulin 3.5 gm/dl and PT- 20. 9 sec with an INR of 1.6. Her ECG was normal except sinus tachycardiaand radiology including X-ray chest and soft tissue neck was normal. The initial blood mercury level by ICPMS was 35 µgm/dl (N=<1µgm/dl) and urinary mercury levels were 24 µgm/dl (N=<10 µgm/dl).

She continued to have hematemesis and 1 day following ingestion she gradually became drowsy with hypotension and decreasing urine output. She was commenced on broad spectrum antibiotics, intravenous pantoprazole (40 mg twice daily) and nasogastric sucralfate (onegm three times daily) and had no further gastrointestinal bleeding. By the third day she was completely anuric and was put on inotropic support. Her serum creatinine increased from 1.6mg/dl on the first day to 5.0mg/dl by the third day. She received 6 units of FFP and 4 units of blood transfusion. On Day 4 when the patient was hemodynamically stable and there was no further episode of hematemesis, high flux bicarbonate hemodialysis was initiated and continued every alternate day for 14 days. Renal biopsy was done at this time, which was suggestive of acute tubular necrosis (**Figure 1**). After 7 sessions of hemodialysis her urine output started to increase and gradually her creatinine levels started to fall till it reached 1.2mg/dl 7 days there after without any further sessions of hemodialysis. Her blood mercury level on the 25^{th} day post ingestion was 5 µgm/dl.

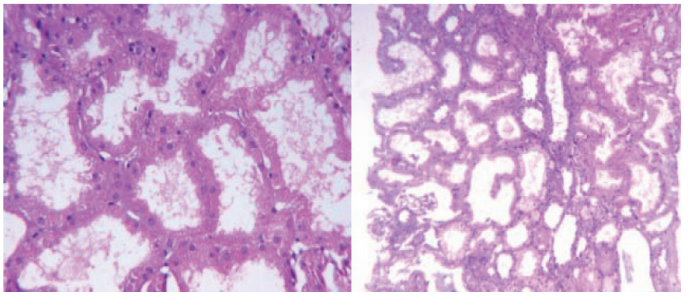

Figure 1 Renal biopsy showing acute tubular necrosis

She was transfused 15 U bloods over the first 20 days. Her blood parameters on Day 25 showed normal renal function, mildly deranged liver function with leucocytosis. However on the 26^{th} day she again had a large bout of hematemesis and despite all conservative measures she succumbed to her GI bleed.

DISCUSSION

The clinical picture of acute mercury poisoning in our patient was characteristic. It includes sudden, profound circulatory collapse with tachycardia, hypotension and peripheral vasoconstriction and vomiting. Renal failure usually develops within 24 hours and is associated with albuminuria, epithelial cell casts and red cells in the urine, glycosuria, and aminoaciduria. Oliguria may proceed to complete anuric failure. There is a neutrophil leucocytosis of up to 20 x 10^6/L due to tissue necrosis. Histologically the classic renal lesion of acute mercury poisoning lies in the terminal part of the proximal tubule. Mercury ions are probably specifically concentrated there and have a direct toxic effect on the cell membrane. In fatal cases the tubular epithelial cells show a spectrum of degeneration, fragmentation and necrosis with areas of bared basement membrane. The tubular lumen is blocked by large casts and granular debris. There is variable interstitial oedema but no consistent vascular or glomerular changes. Mercury is also widely taken up by body tissues, so that in fatal cases other organs may show non-specific changes, viz., colitis, liver necrosis and cerebral petechial haemorrhages. Death in our patient was attributed not to renal failure, for which appropriate and adequate treatment was instituted, but to massive hematemesis and consequent cardiovascular collapse. Rate of excretion of inorganic mercury is complex, which occurs in three phases: 35% of single dose is excreted within few days, a second phase with half-life of 30 days account for 50%, and rest is excreted in slow third phase with a half-life of 100 days.[10] Renal failure due to acute tubular necrosis usually becomes evident within few hours, but in our case it occurred after 2 days. In severe cases, death may occur due to cardiovascular collapse. Because of its solubility, mercuric chloride is the most toxic of the inorganic salts with a mean lethal dose in the adult 0.2–1.0 gm. Acute mercury poisoning is best managed by discontinuing the exposure, providing supportive therapy and enhancing the removal of the metal from the body. Elimination of mercury from the body is achieved by chelation therapy and self-excretion. Hemodialysis, haemoperfusion and peritoneal dialysis have been reported to have little efficacy or to be completely ineffective in removing mercury.[9] Historically, the appropriate treatment for acute inorganic mercury poisoning is chelation therapy using dimercaprol (BAL), if renal failure develops, dialysis may be needed.[9, 10] One case report showed a 26 years old woman treated by plasma exchange, hemodialysis and peritoneal dialysis in combination with continued dimercaprol chelation. Dimercaprol mobilizes tissue mercury by forming soluble complexes, which are excreted in urine. While hemodialysis was ineffective in removing the mercury, plasma exchange effectively eliminated mercury. After two plasma exchange therapies, mercury concentration in the blood decreased linearly on a log scale with half-lives of 23.1 days for whole blood and 19.1 days for plasma, using first-order kinetics. One

month after ingestion, renal function recovered to normal as judged by serum creatinine and blood urea nitrogen levels, at a follow-up examination four months later, renal function was found to be completely normal. This indicates that the renal damage caused by acute mercuric chloride poisoning may not be permanent. In our case on the contrary High flux hemodialysis was found to be highly effective in treating the renal failure of mercury chloride poisoning.

CONCLUSION

We have hereby presented a case of mercury intoxication with acute tubular necrosis in a 25-year old woman, with an excellent improvement of the renal failure and normalization of laboratory results with high flux hemodialysis.

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