Acute renal failure following detergent ingestion

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ABSTRACT

A 42-year-old woman with a history of depression and epilepsy ingested two types of household detergent and developed gastrointestinal symptoms, and subsequently acute renal failure. Coingestants included nontoxic quantities of paracetamol and therapeutic doses of sodium valproate and fluoxetine. The patient developed acute renal failure, and also had fever and unilateral ear inflammation. The acute renal failure resolved four days later. Patients presenting with detergent poisoning are typically screened and treated for gastrointestinal and respiratory toxicity. We discuss the mechanism of development of acute renal failure in our patient, review the literature linking detergent poisoning and nephrotoxicity, and propose a direct relationship between detergent poisoning and acute renal failure.

Keywords: acute tubular necrosis, detergent ingestion, poisoning, renal failure

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INTRODUCTION

A patient with acute renal failure secondary to acute tubular necrosis following ingestion of household detergent is discussed.

CASE REPORT

A 42-year-old Chinese woman presented to the emergency department 90 minutes after ingestion of about 30 ml of dishwashing detergent ("Mama Lemon") and 80 ml of laundry detergent ("Dynamo"), and eight hours after ingestion of 3 g of paracetamol. She had taken them impulsively due to financial problems. She had a past medical history of idiopathic epilepsy and depression, and her regular medications were Epilim chrono 300 mg bd, folic acid 5 mg om, fluoxetine 20 mg om and lorazepam 1 mg on prn. She did not have any known drug allergy and did not consume alcohol, traditional medicines or any other over-the-counter medications.

On examination, her vital signs were normal and systemic examination was unremarkable. Laboratory investigations showed normal full blood count, serum electrolytes, serum creatinine of 63 μmol/L, serum urea of 5.2 mmol/L, normal liver function tests, and a paracetamol level of 27 mg/L at eight hours post-ingestion, which was in the nontoxic range. She was given intravenous hydration, and admitted to the medical ward later in the day. She complained of one episode of vomiting, passage of more than ten loose stools and epigastric pain. She had been given a tablet of loperamide/diphenoxylate in the emergency department for diarrhoea. On examination, she was tachycardic at 100 bpm, normotensive at 120/80 mmHg and clinically dehydrated. She was given magnesium trisilicate, omeprazole and intravenous hydration. Her regular medications were continued.

She had another two episodes of vomiting and loose stools subsequently, and also developed a fever of 38°C. When she was reviewed the next day, she complained of left ear pain, and otoscopy showed inflammation of the external auditory canal. Her charts showed that she had been given a total of 1.7 L of fluids since arrival to the hospital. She had been normotensive throughout. She was started empirically on intravenous amoxicillin-clavulanic acid and ciprofloxacin ear drops.

Laboratory work-up revealed an acute rise in serum creatinine to 447 μmol/L and serum urea to 14.8 mmol/L. Total white cell count had risen to 10.1 × 10^9/L from 5.7 × 10^9/L the day before. The rate of intravenous hydration was increased. Eight hours later, her serum creatinine had risen to 504 mmol/L and urea to 16.7 mmol/L, with evidence of metabolic acidosis (serum HCO_3 13 mmol/L), hyperphosphataemia (serum phosphate 2.8 mmol/L) and hypoalbuminaemia (serum albumin 31 g/L). Creatine kinase was normal at 168 U/L and aldolase was only marginally raised at 12 U/L. Urinalysis showed microscopic haematuria, 2+ proteinuria and no casts. Urine microscopy showed 25 epithelial cells/high power field (hpf), 10 white blood cells/hpf and 50 red blood cells/hpf. Ultrasonography of the kidneys showed normal-sized kidneys with no evidence of hydronephrosis.

She was diagnosed with acute renal failure secondary to acute tubular necrosis. Intravenous and

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oral hydration was continued, and her renal function began to improve by Day 4 of admission. Her serum creatinine normalised by Day 6. Blood valproate level, which was not checked earlier, on Day 6 of admission was sub-therapeutic at 29 mg/L, while the patient had been maintained at the same dose of sodium valproate throughout admission. Blood and urine cultures did not yield any bacterial growth, and she was switched to oral ciprofloxacin. She was discharged on Day 7 with a normal serum creatinine of 86 μmol/L.

DISCUSSION

Our patient ingested two types of household detergent, which contained anionic and non-ionic detergent compounds, oxidants, brighteners, softeners, colours and perfumes. She presented to the emergency department within two hours of ingestion, and soon after developed abdominal pain, vomiting and diarrhoea. Despite intravenous hydration, she became dehydrated, but was not hypotensive. On the second day of admission, she developed fever, unilateral ear inflammation and acute renal failure. The clinical picture and laboratory features were consistent with acute tubular necrosis. She was given intravenous hydration and antibiotics for ear inflammation, and recovered fully.

Could the detergent she ingested have caused the renal failure? The gastrointestinal, respiratory and ocular effects of detergent poisoning by ingestion, inhalation and ocular exposure, respectively, are widely known,[1] but renal complications are not often mentioned. To the best of the author's knowledge, no reports of acute tubular necrosis secondary to oral ingestion of detergent exist. Prabhakar et al reported a patient who developed rhabdomyolysis and acute renal failure after detergent ingestion.[2] Their patient was similar to ours in that he had epilepsy treated with carbamazepine and sodium valproate, diarrhoea on admission which subsequently resolved, normal serum creatinine on admission, and acute renal failure surfaced 24–48 hours after admission. However, unlike our patient, he had frank rhabdomyolysis, with a serum creatine kinase level of over 10,000 IU/L, and his renal failure was more severe—complicated by metabolic acidosis, hyperkalaemia, hypocalcaemia, hyperphosphataemia and reduced mentation—necessitating dialysis, and recovery occurred later, at Day 22.

Acute renal failure has been reported in patients with detergent poisoning by non-oral routes. Okumura et al reported a patient who developed acute renal failure, cardiac arrhythmia, rhabdomyolysis, haemolysis and coagulopathy after intravenous injection of detergent.[3] Renal biopsy of their patient showed acute tubular necrosis without deposits of haemosiderin or myoglobin, suggesting that acute renal failure was not a result of rhabdomyolysis. Burnhill in 1985 wrote that chemically-induced abortions via transcervical introduction of soap, phenol and cresol compounds was a fairly common practice in the time before abortion was legal, and described lower nephron changes in the kidneys of these patients, with resulting irreversible renal damage.[4] Other authors similiarly described acute renal failure in women who had induced abortion using intrauterine instillation of soap or phenolic antiseptics, and reviewed their management.[5,6]

What else could have caused acute renal failure in our patient? Dehydration probably contributed, but was unlikely to have been the sole cause. She was given intravenous hydration during her hospital stay, had unrestricted access to oral fluids, and was not hypotensive at any point in time. She appeared to have suffered an otitis externa which may have been of bacterial or viral aetiology, but this was not associated with severe sepsis, as there was no viral prodrome, and acute renal failure occurred on the same day she had the ear symptoms. Hence, it was unlikely that an infection had resulted in acute renal failure. The paracetamol level was not high and she did not appear to have ingested any other nephrotoxic agent.

Hence, in this case, it was possible that the ingested detergent had been systemically absorbed and filtered through the kidneys, and had caused direct toxicity resulting in acute tubular necrosis and acute renal failure. With the removal of the toxin from the system, as the ingested detergent passed out of the gastrointestinal tract and the possibly affected tubular cells were sloughed and excreted in urine, the remaining tubular cells and renal function recovered. Pathological evaluation might have been useful from an academic viewpoint, but renal biopsy was not performed as the patient improved rapidly. In conclusion, this case report highlights the need to consider renal injury in patients presenting with detergent poisoning, via mechanisms that are not completely clear at present.

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