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Case Report

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Renal cortical necrosis complicating laundry detergent ingestion

Leonardo Vidal Riella[1], Sunitha Golla[4], Grigore Dogaru[5], Helmut G. Rennke[2] and Kenneth Christopher[3]

[1]Renal Division, [2]Renal Division, [3]Pathology Division, Brigham and Women's Hospital, Boston, MA, USA, [4]Department of Medicine, MetroWest Medical Center and [5]Department of Nephrology, University of Medicine and Pharmacy, Targu Mures, Romania

Abstract

Accidental oral detergent ingestion usually causes mild gastrointestinal manifestations including nausea, vomiting and diarrhea as well as upper airway irritation. There are a limited number of reported oral detergent ingestions leading to acute kidney injury, mainly due to rhabdomyolysis. We present an ultimately fatal case of laundry detergent ingestion leading to biopsy proven severe cortical necrosis and irreversible renal damage. Detergent ingestion was associated with widespread endothelial injury leading to a picture of thrombotic microangiopathy. Among the detergent ingredients ingested by the patient, sodium borate raised the highest concern as a potential toxin exacerbated further by severe hypovolaemia and consequent decrease in renal toxin excretion. As sodium borate is dialyzable, haemodialysis should be a consideration early after laundry detergent ingestion.

Keywords: acute kidney injury; cortical necrosis; detergent

Introduction

Nephrotoxicity caused by contrast media and drugs is a frequent cause of renal failure in medical practice. However, there are only sporadic cases of renal failure caused by chemical ingestions [1–3]. Accidental oral detergent ingestion usually causes mild gastrointestinal manifestations including nausea, vomiting and diarrhoea as well as upper airway irritation with mucosal lesions and risk of upper airway oedema. There are a limited number of reported oral detergent ingestions leading to acute kidney injury, mainly due to rhabdomyolysis [3]. We now present a case of detergent ingestion leading to a biopsy-proven severe cortical necrosis and irreversible renal damage.

Correspondence and offprint requests to: Leonardo Vidal Riella, Medical Research Building 4th floor, Renal Division, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115, USA. Tel: +00-1-617-732-5252; Fax: +1-617-732-6392; E-mail: lriella@rics.bwh.harvard.edu

Case report

A 49-year-old man with a history of hypertension and mild mental retardation presented to the emergency department with nausea, vomiting and diarrhoea. He reported some crampy diffuse abdominal pain on midepigastrium intermittently associated with copious diarrhoea (>10 bowel movements/day), anorexia and fever for 24 h. Initially, there was no evidence of blood in the stools, but on admission, there was evidence of bright red blood. He denied any sick contacts but the father reported that the patient had eaten an undercooked hamburger 3 days prior as well as the inadvertent ingestion of laundry detergent 30 h prior to presentation. The estimated amount was ∼200–250 mL. There was no accompanying chest pain, dyspnoea, lightheadedness, haematemesis, odynophagia, haematuria, dysuria or headache.

His prescription medications were atenolol, levetiracetam and carbamazepine, and he denied any history of antibiotic use. Physical examination revealed normal vital signs, a clear oropharynx and no rashes. The abdomen had increased bowel sounds and was soft, but tender to deep palpation throughout without guarding. Lower extremities demonstrated trace oedema around the ankles.

At presentation, creatinine was 141 μmol/L from baseline of 70 μmol/L 3 months prior. White blood cell count (WBC) was 23 000/mm^3 with 55% band forms, haematocrit was 35.5 and platelets were 128 000/mm^3. On peripheral smear, there were 1+ schistocytes. Coagulation studies were normal. Creatine kinase was 249 IU/L (normal range 25–200 IU/L) and myoglobin was 1007 mg/dL (normal range 0–100). Urinalysis revealed a specific gravity of 1.023, pH of 7.0, 3+ protein, 1+ leukocyte esterase, 3+ blood and no nitrites. Urine sediment demonstrated many red blood cells per high-powered field but no evidence of cellular/granular casts or dysmorphic red blood cells. Over the next 48 h (Day 2), the creatinine increased despite fluid resuscitation and the patient became oliguric. He remained haemodynamically stable.

On Day 3, the chemistries were notable for creatinine 1,122 μmol/L, lactate dehydrogenase (LDH) 1706 IU/L (normal range 100–300 IU/L) and creatine kinase (CK) 249 IU/L (Table 1). Haptoglobin was 12 (normal range 30–200 mg/dL) and stool analysis was negative for *E. coli*

Table 1. Relevant laboratory results during hospitalization

Day	Urea (mmol/L)	Creatinine (μmol/L)	LDH (IU/L)	CK (IU/L)	Haptoglobin (mg/dL) (normal range 30–200 mg/dL)	Haematocrit smear	Platelet (k/mm^3) (normal range 150–400)	AST (IU/L)	ALT (IU/L)
1		141				35; 1 + schistocytes	128	42	42
2		627		266		38	115		
3 HD		1122	1706	249	12	35	105	144	342
4	26	937	4046			35; 1 + schistocytes	106	211	211
5						30	94	235	235
6 HD	35	1016				30	114	582	582
7 HD	32	884				35	145	825	767
8	27	769	2216	180		36	162	664	772
9 HD	40	1060	1580	484		33	195	525	665
10 HD	32	804			54	31	189	292	428

LDH: lactate dehydrogenase; CK: creatine kinase; AST: aspartate aminotransferase; ALT: alanine aminotransferase; HD: haemodialysis.

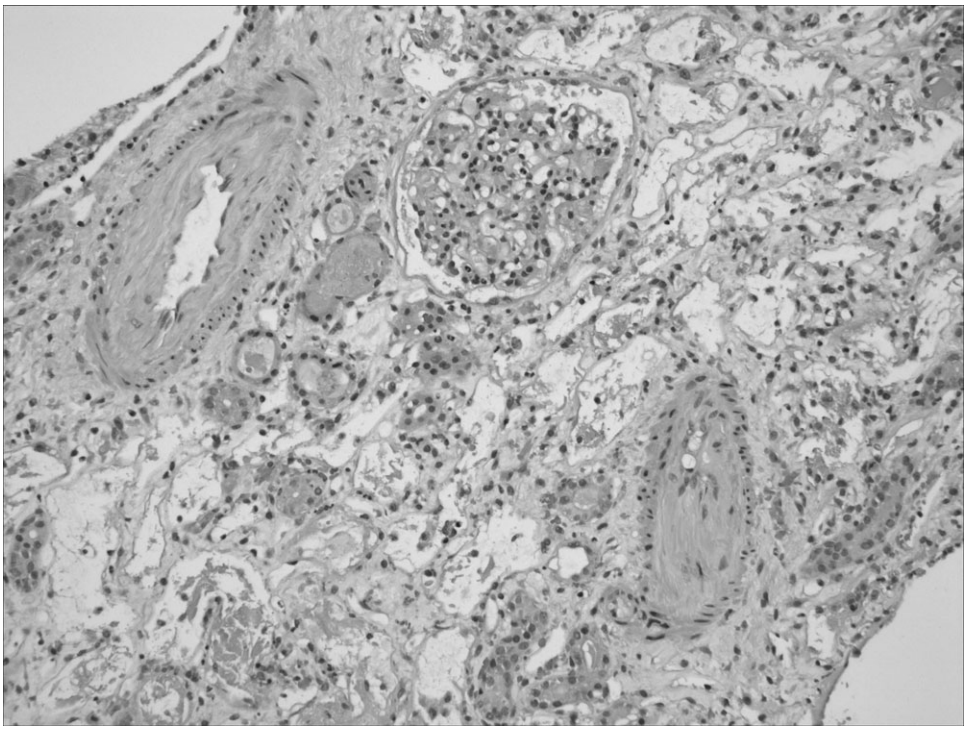

Fig. 1. Light microscopy of kidney biopsy showing extensive focal cortical necrosis with tubular necrosis and fibrin thrombi in the glomerulus. Small arteries reveal very pronounced narrowing and near occlusion of the lumen by loose connective tissue, lesions that are most suggestive of a chronic thrombotic microangiopathy.

O157:H7. Renal ultrasound was unremarkable. Haemodialysis was initiated on Day 3 following ingestion. The patient was subsequently transferred to our hospital on Day 4 and noted to be anuric with continuous diarrhoea.

A decision was made to perform a renal biopsy to clarify the aetiology of his severe renal dysfunction. Light microscopy was notable for extensive acute tubular necrosis, focal cortical necrosis with widespread distribution of fibrin in the glomeruli, a finding that is highly suggestive of thrombotic microangiopathy. (Figure 1). Arteries and arterioles showed marked sclerosis of the vascular wall with occlusion of the lumen in several intra-renal vessels by organizing loose connective tissue. These lesions may also have predisposed to the acute ischaemia and cortical necrosis observed. Ultrastructural analysis showed evidence of extensive parenchymal and endothelial injury, intracapillary congestion, fibrin and platelet thrombi and inflammatory cells (mainly mononuclear elements); there was no evidence of pigment deposits on tubular cells. The diffuse endothelial injury was likely to be secondary to a direct toxic effect. ADAMTS13 activity in the serum was 47% (reference >67%). The review of the material safety data sheet from the detergent ingested revealed potential toxins including ethanol, 2-aminoethanol and sodium borate (Table 2, available online at http://ndt.oxfordjournals.org).

His course was further complicated by an ischaemic stroke and sepsis culminating with cardiac arrest and death on Day 12 following ingestion.

Discussion

Detergent is a mixture of multiple components including surfactants, builders, fillers, bleach and antifoaming agents. The common builder agents are compounds containing sodium phosphate or borates. Among the detergent ingredients ingested by the patient, sodium borate raised the highest concern as a potential toxin [4–7].

Sodium borate is a naturally occurring alkaline mineral and has many industrial uses including natural laundry booster, preservative, pesticide and disinfectant. The majority of single acute boric acid and borate ingestions are considered benign and asymptomatic [5,6]. However, acute fatal single boric acid ingestions are reported [4,7]. The mechanism of toxicity is unknown but most likely involves direct cellular toxicity secondary to oxidative properties and the degree of toxicity is dose dependent. Main organs affected include skin, gastrointestinal tract, brain, liver and kidneys. It is mainly removed by the kidneys and has a half-life of ∼12–27 h. The clinical presentation of toxic ingestion includes nausea, vomiting and diarrhoea with significant dehydration and renal failure. Renal involvement in acute boric acid intoxications is reported with boric acid ingestion of over 400 mg/kg with abnormal urinary sediment and/or the presence of oliguria, anuria and azotaemia [4]. Histological findings in severe acute boric acid intoxication consist of swelling or degeneration of tubular epithelial cells as well as dilation and congestion of glomerular tufts in the majority of cases [4]. There is no antidote available and treatment is mainly supportive, and haemodialysis might be helpful after massive ingestions since the compound has a low molecular weight and small volume of distribution [8].

The clinical features seen in our case are likely multifactorial. Surfactant toxicity led to diarrhoea through the local effect on bowel mucosa. The renal failure was most likely due to direct toxic action of detergent components (especially borate) on renal tubular epithelial cells and endothelium exacerbated further by hypovolaemia and consequent decrease in renal toxin excretion. The extensive detergent-related endothelial injury also resulted in thrombotic microangiopathy with low schistocytes on peripheral smear and low platelets, confusing the picture especially with the history of hamburger ingestion [9].

Conclusion

Toxins may damage the kidney at both tubular and endothelium locations, leading to tubular necrosis and thrombotic microangiopathy, respectively. Laundry detergent ingestion is generally considered to have minor consequences and there is little literature on the subject. However, it is always important to be vigilant about toxic ingestions, and attempts should be made to investigate immediately the constituents of ingested materials and dialysis should be initiated early if the contents are thought to be dialyzable.

Supplementary data

Supplementary data is available online at http://ndt. oxfordjournals.org.

Conflict of interest statement. All authors were involved clinically with this case. Dr Riella prepared the case presentation and the discussion. Dr Christopher helped with interpretation of data and revision of the report adding important clinical analysis. Dr Rennke processed the biopsy and analysed all the histological findings. Sunitha did background literature review and wrote an initial draft and Grigore Dogaru reviewed the inital draft.

References

1. Abuelo JG. Renal failure caused by chemicals, foods, plants, animal venoms, and misuse of drugs. An overview. *Arch Intern Med* 1990; 150: 505–510
2. Cecchin E, De Marchi S. Does ethanol have toxic effects on the kidney? *Nephrol Dial Transplant* 1994; 9: 1522–1523
3. Prabhakar KS, Pall AA, Woo KT. Rhabdomyolysis and acute renal failure complicating detergent ingestion. *Singapore Med J* 2000; 41: 182–183
4. Ishii Y, Fujizuka N, Takahashi T *et al*. A fatal case of acute boric acid poisoning. *J Toxicol Clin Toxicol* 1993; 31: 345–352
5. Linden CH, Hall AH, Kulig KW *et al*. Acute ingestions of boric acid. *J Toxicol Clin Toxicol* 1986; 24: 269–279
6. Litovitz TL, Klein-Schwartz W, Oderda GM *et al*. Clinical manifestations of toxicity in a series of 784 boric acid ingestions. *Am J Emerg Med* 1988; 6: 209–213
7. Restuccio A, Mortensen ME, Kelley MT. Fatal ingestion of boric acid in an adult. *Am J Emerg Med* 1992; 10: 545–547
8. Naderi AS, Palmer BF. Successful treatment of a rare case of boric acid overdose with hemodialysis. *Am J Kidney Dis* 2006; 48: e95–e97
9. Fakhouri F, Fremeaux-Bacchi V. Does hemolytic uremic syndrome differ from thrombotic thrombocytopenic purpura? *Nat Clin Pract Nephrol* 2007; 3: 679–687

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