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Case Report

Shock with Severe Rhabdomyolysis and Multiorgan Failure Following Detergent Ingestion

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Abstract

Detergent ingestion is typically associated with mild gastrointestinal symptoms but can occasionally result in severe systemic toxicity. We report the first Australian case of multiorgan failure following intentional detergent ingestion.

A 47-year-old male with schizophrenia ingested approximately 500 mL of dishwashing detergent. He presented with cardiorespiratory instability, progressing to inflammatory shock and multiorgan failure requiring prolonged intensive care. Early investigations revealed metabolic acidosis, elevated lactate, and hyperthermia refractory to cooling. Despite supportive measures, he developed severe rhabdomyolysis (peak creatine kinase: 488,650 U/L), acute kidney injury requiring dialysis, and respiratory failure necessitating prolonged ventilation. Imaging and endoscopic evaluation showed no significant airway or gastrointestinal damage.

The detergent contained sodium alkyl benzene sulfonate and cocamidopropyl betaine, surfactants known to disrupt cellular membranes and trigger systemic inflammation.

This case underscores the potential for severe systemic effects from large-volume detergent ingestion, even in the absence of overt corrosive injury. Prompt identification of ingested substances and aggressive organ support are critical for survival and recovery. The patient made a full recovery after 15 weeks of hospitalization.

Keywords: Detergent ingestion; Rhabdomyolysis; Acute kidney injury; Multi-organ failure

Introduction

Household cleaning products (detergents) generally have mild toxicity in the form of local irritant effects on the eyes and skin. Ingestion of detergent usually results in mild gastrointestinal manifestations but in some cases may cause severe corrosive injury.

Reports of early multiorgan failure with inflammatory vasogenic shock following detergent ingestion are limited and there are none from Australia. Here we present a case of early multiorgan failure requiring prolonged invasive cardiovascular, respiratory, and renal support following intentional detergent ingestion in a patient who made a full recovery.

Presentation and Clinical Course

A 47-year-old male with chronic and treatment resistant schizophrenia, type two diabetes mellitus, hypertension, and obstructive sleep apnoea presented to the Emergency Department (ED) approximately three to four hours after an intentional ingestion of approximately 500 ml of Aldi TANDIL ULTRA Concentrate Dishwashing Liquid (Lemon Anti-bacterial 900 ml). Prior to his presentation to ED, he had four episodes of vomiting and one episode of diarrhea at home. He had a further episode of vomiting (clear-white fluid) in the ED.

His medications included clozapine, fluoxetine, rosuvastatin, metoprolol, metformin SR, dapagliflozin, gliclazide MR, and

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melatonin MR. Recent dose adjustments of his clozapine had been made by his psychiatrist because of increasing anxiety and behavioural problems that his supportive family were having difficulty managing. He did not have any known drug allergies.

On examination in the ED, Glasgow Coma Scale (GCS) was 14 and he appeared diaphoretic. He was hypotensive on presentation with a blood pressure (BP) of 91/48 mmHg that responded to fluid resuscitation (BP increased to 131/74 mmHg). He was tachycardic with the heart rate of 124/min and in sinus rhythm. His oxygen saturations were 95% on room air and he was tachypneic with a respiratory rate of 32/min. His temperature was 37.3°C. There was no mucosal damage noted in oral cavity and his abdomen was soft and non-tender.

Initial laboratory investigations showed white cell count 22 (x10^9/L), platelet 326 (x10^9/L), serum creatinine 155 µmol/L, and potassium 5.0 mmol/L. His venous blood gas revealed pH 7.322, pCO2 53.3 mmHg, HCO3 26.8 mmol/L, and lactate 6.6 mmol/L. The serum clozapine level was 378 µg/L (reference 350-600 µg/L).

After two hours in the ED, he had a further deterioration with a requirement for oxygen of 15L/minute via a non-rebreather mask to maintain oxygen saturation of 92%. This was associated with hypotension and a further reduction in his GCS to 13. His venous lactate rose to 11.3mmol/L. He had no symptoms of stridor and was transferred to the operating theatre for an awake oral fibre-optic intubation due to concern regarding potential detergent induced airway swelling. Flexible trans-nasal endoscopy revealed mild oedema affecting the arytenoid, vocal cord, aryepiglottic fold, epiglottic and supraglottic areas. The carina was visualized with bubbly secretions coating mucosa, however there was no significant airway injury or obstruction detected.

He was admitted to the Intensive Care Unit (ICU) following intubation and developed a rapidly increasing temperature and circulatory failure. Bedside Transthoracic Echocardiography (TTE) on ICU admission revealed grossly normal contracting ventricles; no large pericardial effusion; and small appearing inferior vena cava (IVC). The working diagnosis for his haemodynamic instability was that it was most likely due to hypovolaemia and not of cardiogenic aetiology. Further intravenous fluids were administered, and he was initially responsive to fluids and vasopressors.

In the first 48 hours following ICU admission, his temperature peaked at 40.8ºC and his peak noradrenaline dose was 0.35 mcg/kg/min; concomitantly he received adrenaline (peak dose 0.08 mcg/kg/min) and fixed dose vasopressin infusion (2.4 units/hr) to provide further pharmacological circulatory support. An urgent Computed Tomography (CT) scan of his chest, abdomen and pelvis was performed which demonstrated bilateral lower lobe consolidation and no evidence of upper gastrointestinal perforation or inflammation. His electrocardiogram showed sinus tachycardia with nonspecific ST and T wave changes. A further TTE showed hyperdynamic biventricular function without significant valvular abnormalities. Microbiology results including blood and other cultures were negative.

Between 48 and 72 hours after ICU admission, he developed dark coloured urine, a rise in his serum creatinine from 152 to 269 µmol/L and a creatinine kinase of 417,500 U/L. Lactate Dehydrogenase (LDH) was elevated to 14,897 U/L. His Aspartate Transaminase (AST) increased to 3,150 U/L and Alanine Transaminase (ALT) increased to 299 U/L; these were most likely from muscle injury and there were no other features suggestive of liver injury. He also developed thrombocytopenia which was thought to be likely consumptive due to acute rhabdomyolysis and acute inflammation. Peripheral blood smear examination revealed some hyposplenic features, but no schistocytes or fragmentation. There was no evidence of disseminated intravascular coagulation or thrombotic microangiopathy. Repeat microbiology test results were also negative. Trends of temperature, inotropic and vasopressors doses are shown in **Figure 1**. The relevant laboratory tests and their trends are shown in **Figure 2**.

Circulatory support was ceased on day 10 after ICU admission. He was liberated from the invasive respiratory support on day 20 and was discharged to the ward on day 28. His last haemodialysis session occurred on day 29. His recovery was complicated by profound weakness which manifested as severe symmetric flaccid quadriparesis likely secondary to rhabdomyolysis and delayed clearance of sedatives due to his organ dysfunction. His antipsychotic medications were gradually reintroduced by the psychiatric team. His renal function recovered completely in 6 weeks with normal serum creatinine and estimated glomerular filtration rate (eGFR). He underwent gastroscopy in week 12 which was normal. He was referred to the rehabilitation unit and subsequently discharged home after spending 15 weeks in the hospital.

Table 1: Detergent composition.

Ingredients	Proportions
Sodium Laureth-3 Sulphate	<20%
Sodium Alkyl Benzene Sulphonate	<10%
Cocamidopropyl betaine	<10%
Ethanol	<10%
Lactic Acid	<2%
Water	60-90%
Also contains other components, including fragrance and dyes, which are not classified as hazardous	To 100%

Table 2: Physical and chemical properties.

Physical State	Clear liquid
Colour	Yellow – Lemon Anti-bacterial
pH	3.0 - 4.0
Specific Gravity or Density	1.04 - 1.06
Percent Volatiles	67 – 69%
Solubility	Soluble in water

The product information and the safety data sheet for the detergent ingested is shown below in **Table 1** and **Table 2** [1].

Discussion

Our patient developed severe shock, hypoxic respiratory failure, and severe metabolic derangement within hours of detergent ingestion, requiring early invasive respiratory and cardiovascular organ support. This was in the absence of oesophageal injury, infection, or evidence of a drug-induced hyperthermic syndrome. There was an initial improvement in cardiovascular function in the first 48 hours, however, the acute pathophysiological process did not abate. There was a subsequent deterioration on day four with an increase in cardiovascular support. This was associated with rhabdomyolysis and progression to

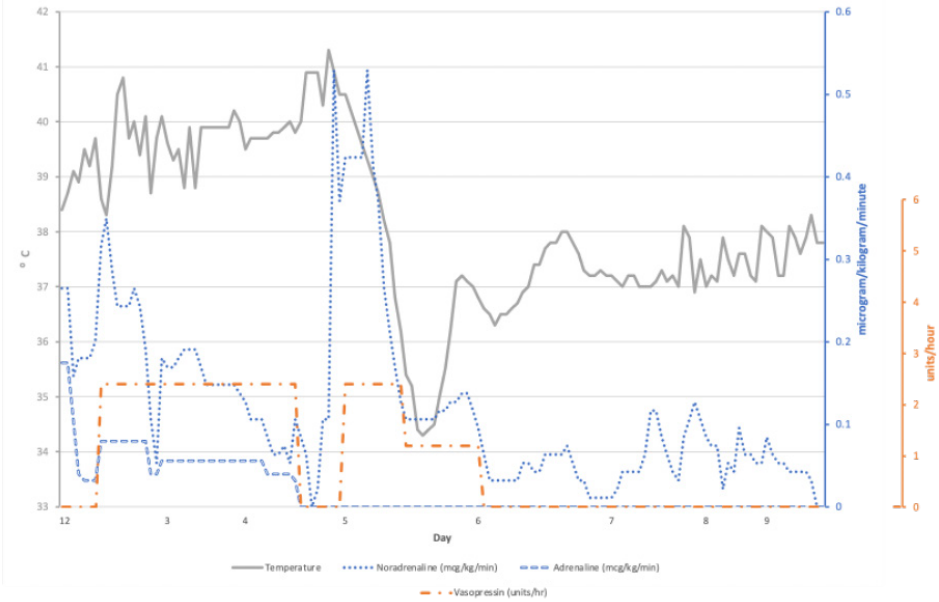

Figure 1: Trends of temperature, inotropic and vasopressors doses in first 9 days.

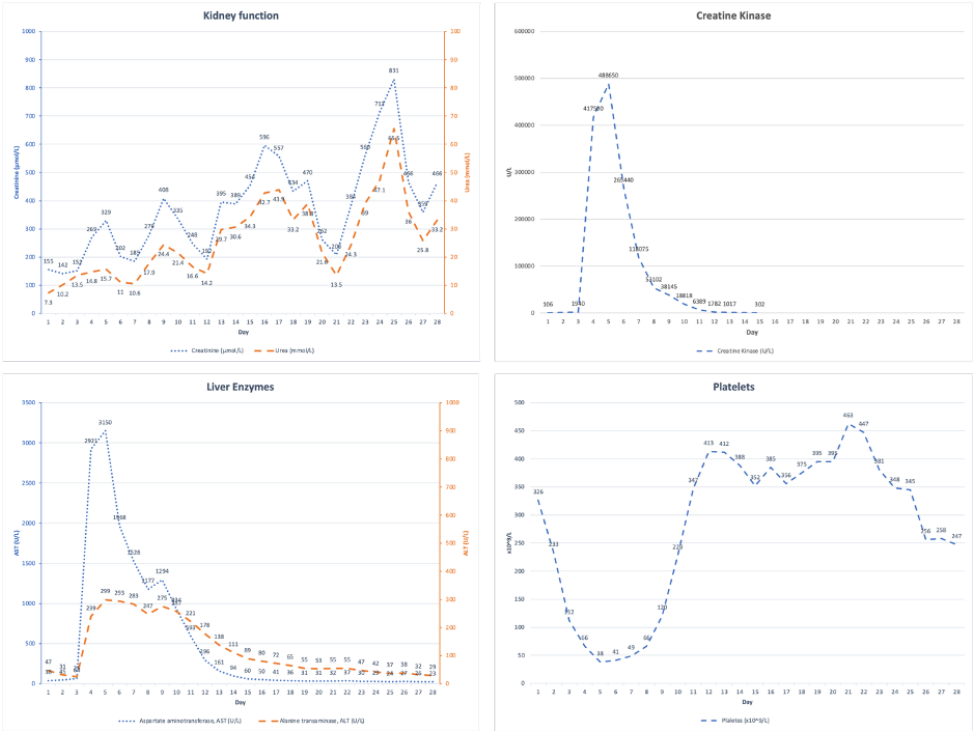

Figure 2: Biochemical trends during the ICU stay.

renal failure that required additional invasive renal support. Microbiology test results including blood and other cultures were negative for both initial and secondary deteriorations.

Of the 164,363 exposures reported to four Australian Poisons Information Centres (PIC) in 2015, 18,542 (10.2%) were for household cleaning substances, and bleach accounted for 2,685 (14.5%) of these exposures [2]. The incidence of unintentional poisoning due to corrosive substances, soaps, and detergents in preschool children in New South Wales, Australia was even lower [132 (4%) of 3436 cases of poisoning] [3].

Detergents contain surfactants (surface active agents) and builders. The surfactant molecules contain both polar and non-polar regions, which effectively decrease the surface tension of water. This helps water to wet surfaces more effectively and results in more efficient cleaning and washing. Surfactants can also help remove dirt, disperse soil, and emulsify oil or grease in the wash water. There are 4 types of surfactants in the household detergents as shown below in Table 3.4 Nonionic and amphoteric surfactants have the lowest toxicity, anionic surfactants have intermediate toxicity, and the cationic surfactants have the highest toxicity [4].

Builders are chemical compounds that are added to a detergent product to improve its cleaning properties. Builders are alkaline compounds that are irritants at low concentrations but can be corrosive at higher concentrations. The corrosive injury becomes a risk at a pH less than 2 or more than 12 [5].

Our patient ingested dishwashing detergent and we believe the two ingredients, sodium alkyl benzene sulfonate and be-

Table 3: Surfactants used in synthetic household detergents:

Anionic • Alkyl sodium sulfates • Alkyl sodium sulfonates • Dioctyl sodium sulphosuccinate • Linear alkyl benzene sulfonate (Na^+) • Sodium lauryl sulfate	**Cationic** • *Quaternary ammonium compounds* o Benzalkonium chloride o Benzethonium chloride • *Pyridinium compounds* o Cetylpyridinium chloride • *Quinolinium compounds* o Dequalinium chloride
Nonionic • Alkyl ethoxylate • Alkyl phenoxy polyethoxy ethanols • Polyethylene glycol stearate	**Amphoteric** • Imidazolines • Betaines

taines (anionic and amphoteric surfactants respectively), were responsible for the systemic toxicity and organ failures. Corrosive injury to the upper GI tract has previously been reported and although our patient had some gastrointestinal symptoms (vomiting and diarrhoea) at the time of admission, fibreoptic intubation only revealed mild oedema of the upper airway, and subsequent CT ruled out significant structural upper gastrointestinal pathology. The pH of the ingested compound was 3.0 – 4.0 and this explains the absence of obvious corrosive injury in our patient.

The lactic acid (<2%) and ethanol (<10%) in this detergent composition may have contributed to the initial hyperlactatemia and metabolic acidosis. We suspect that the initial hypoxia may have been due to aspiration or recumbency associated bilateral lower lung collapse as he had a mildly reduced level of consciousness and early episodes of vomiting. Aspiration pneumonitis with systemic inflammation is a possible differential diagnosis and has also been reported with laundry pod exposures [6].

A key point in this patient's presentation is the very large volume (up to 500 mls) of detergent ingested. Most calls to Poisons Information Centre (PIC) involve small, accidental ingestions. Even in the available case reports, the largest volume reported is 250 mls [7,8]. The volume may be relevant for two reasons: (1) The large volume of surfactant ingested may be sufficient to account for the severity of the acute presentation; and (2) there may be an unknown ingredient that we do not know about that is hazardous in large volumes as ingredients that are <1% of the composition are not required to be listed. For example, propylene glycol is commonly used in cleaning products and can be associated with cardiovascular depression and renal dysfunction.

Our patient developed persistent hyperthermia from admission (peak temperature of 41.4°C) which was refractory to surface cooling with a cooling blanket, suggesting that there was systemic inflammation occurring. Continuous Renal Replacement Therapy (CRRT) was commenced during the second deterioration on day four with the joint purpose of managing anuric renal failure and temperature control.
Acute renal injury following detergent ingestion is rare and we identified only four cases in the literature. Two papers reported ingestion of laundry detergent [8,9] and one reported ingestion of dishwashing and laundry detergent [10]. The type of detergent was not mentioned in one of these reports [7]. In addition, acute kidney injury (AKI) due to intravenous detergent poisoning has also been reported [11].

Laboratory studies have shown interactions between surfactants and the cell membrane. The surfactants enter the lipophilic part of the membrane and increase its fluidity resulting in cell disruption [12,13]. This mechanism may explain the initial generalised systemic inflammatory response and vasoplegia, in addition to the delayed muscle injury and rhabdomyolysis in our patient. Various mechanisms have been described for the acute kidney injury. Lim et al attributed the AKI to renal tubular damage [10] while Riella et al attributed it to direct toxic action of detergent components on renal tubular epithelial cells and endothelium [8]. In a case reported by Prabhakar et al, rhabdomyolysis was the cause of AKI [7]. The highest CK in their patient was 12,160 U/L. Our patient had severe rhabdomyolysis (peak CK of 488,650 U/L) and we believe this was the main reason for AKI although the initial systemic inflammatory response and hypotension may also have contributed. Other differentials such as neuroleptic malignant syndrome (NMS) and serotonin syndrome were also considered but ruled out as our patient did not display any muscle rigidity which is an essential criterion for these syndromes.

We believe this is the first reported case from Australia of an ingestion of household detergent resulting in acute multiorgan failure, manifest as early shock and respiratory failure with subsequent rhabdomyolysis and renal failure. It is important in these cases to urgently investigate the constituents of the ingested material and monitor renal function. Early invasive organ support can facilitate survival with a good outcome.

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Bash Jagarlamudi: review and editing
George Mangos: review and editing
Manoj Saxena: review and editing, supervision

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