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Rhabdomyolysis and Acute Renal Failure Complicating Detergent Ingestion

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ABSTRACT

Rhabdomyolysis should be considered in the aetiology of all patients with unexplained acute renal failure (ARF). Early recognition provides the opportunity to initiate therapy aimed at preventing or limiting nephrotoxicity from the released heme pigment, myoglobin. We report an adult patient who developed ARF following the ingestion of a large amount of household detergent which, as far as we are aware, has not been previously described. The report illustrates the importance of measuring muscle enzyme levels and urinary myoglobin to confirm the possibility of rhabdomyolysis in any unusual presentation of ARF.

Keywords: rhabdomyolysis, acute renal failure, detergent.

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INTRODUCTION

Rhabdomyolysis is an important cause of ARF. Early diagnosis may provide the opportunity to reverse the decline in renal function and obviate the development of established ARF and need for dialysis support. Trauma, pressure necrosis in comatose patients, as well as epilepsy are well recognized causes and a careful history will readily raise the suspicion of rhabdomyolysis in such patients presenting in ARF. However, there are numerous other non-traumatic causes of rhabdomyolysis and this should therefore be considered at presentation of all patients with otherwise unexplained ARF. We report for the first time a patient who developed ARF in association with rhabdomyolysis after the ingestion of household detergent.

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Case Report

A 23-year-old Chinese male was admitted with a history of ingestion of about 250 ml of household washing detergent. He had a history of stable juvenile myoclonic epilepsy over the previous seven years for which he was on carbamazepine and sodium valproate. The recent bereavement at the loss of his father and an argument with his mother prompted him to attempt suicide by detergent ingestion. There was no history of ingestion of alcohol, any drugs or excess medication and he did not have any seizures prior to his presentation at the emergency department.

On admission he was fully conscious but febrile and hypotensive with extensive chemical burns of the oral cavity. He was commenced on iv crystalloids and empirical antibiotics. The diarrhoea which was present at admission subsided within 48 hours. Laboratory investigations at this time revealed a leucocytosis at 17.5 X $10^{(9)}$/L and evidence of renal impairment with a serum urea of 5.4 mmol/L and a creatinine of 212 umol/l. Initial urine output in the first 24 hours was satisfactory but he became oligoanuric over the subsequent 24 hours. This was associated with clinical deterioration and a rapidly worsening biochemistry, with the serum urea and creatinine rising to 37.1 mmol/L and 852 umol/L respectively, which prompted referral for a specialist renal opinion.

At this stage, he was found to be drowsy, dehydrated and acidotic, although hemodynamically stable. Further investigations revealed a metabolic acidosis with pH at 7.016, a bicarbonate of 6.5 mmol/L and high anion gap of 27 mmol/L. Severe hypocalcaemia (corrected calcium of 1.17 mmol/L), hyperphosphataemia (4.32 mmol/L and hyperkalaemia (6.4 mmo1/L) were also noted. The toxicology screening was negative and the blood levels of valproic acid and carbamazepine were within therapeutic range. Serum creatine phosphokinase (CPK) level was markedly elevated at 12,160 iu/L as were the Lactate dehydrogenase(LDH) at 2383 iu/L and Aldolase at 90 iu/L. No urine was available at this stage to document the presence of myoglobinuria.

A diagnosis of ARF complicating rhabdomyolysis was made and the patient commenced on intermittent haemodialysis. He became alert 72 hours after initiating dialysis and started diuresing about a week later. He received a total of nine dialysis sessions. The serum calcium rose to 3.3 mmol/L, which was managed conservatively. He was able to be discharged 22 days after his initial admission with a creatinine of 104 umol/L.

DISCUSSION

Rhabdomyolysis results in the release of myoglobin, a heme pigment, which through multiple effects can result in nephrotoxicity and ARF[1,2,3]. The main mechanisms are likely to be intraluminal obstruction by heme pigment casts, ischaemia[4] and a direct or iron-mediated proximal tubular injury. Hypoxia, volume depletion, hypotension, acidosis and disturbances of thermal regulation contribute to the occurrence of rhabdomyolysis[5].Treatment should be directed at reducing the nephrotoxic potential of the released pigments which can be achieved by rehydration, correction of acidosis, maintaining a good urine output (possible additional benefit from mannitol as a free radical scavenger) and establishing a urinary pH of 7.0 by forced alkaline diuresis[1,2,6,7].

Trauma is a well recognized cause of rhabdomyolysis as is ischaemic muscle pressure necrosis following drug overdoses and in comatose patients[1,5]. Other nontraumatic causes of rhabdomyolysis include alcoholism (particularly if complicated by hypophosphataemia), seizures and heat stroke as well as drugs,most notably, HMG CoA reductases, cocaine, phencyclidine, methadone, barbiturates or other sedatives, either singly or in combination[5,8,9]. Though clinical evidence of muscle injury such as swelling and tenderness of the involved muscle or localized pain and weakness are frequent, these findings may be absent, as evident in our patient.

This case report highlights the importance of considering rhabdomyolysis in all patients with unexplained ARF. At presentation, although his renal function was impaired with a serum creatinine of 212 umol/L, the patient was nonetheless maintaining a good urine output and the opportunity of limiting renal injury was missed. The diagnosis of rhabdomyolysis can be readily established by measuring serum CPK and detection of urinary myoglobin. CPK is directly proportional to the muscle injury, though other muscle enzymes like LDH and Aldolase are also elevated. Other biochemical features include severe hypocalcaemia related to calcium trapping in injured muscle though reduced production of 1,25-dihydroxycholecalciferol by hyperphosphataemia may be contributory[10]. Subsequent recovery phase hypercalcaemia is often seen and is supportive of the diagnosis. Typically there is also hyperphosphataemia and hyperkalaemia. A rapid rise in serum creatinine is a recognized feature of rhabdomyolysis and maybe related to its release from muscle and/or the release of creatine which is subsequently converted to creatinine[9].

Overdose with household detergent in adults is uncommon and more likely to be seen following accidental ingestion amongst children in whom only small amounts are usually ingested. Detergents contain non-soap surfactants (nonionic or anionic), in combination with inorganic ingredients such as phosphates, silicates and carbonates[11]. Recognised manifestations of toxicity include nausea, vomiting, diarrhoea, mild irritation of eyes, upper airway edema with respiratory distress, oral and oesophageal burns. Although the anticonvulsants sodium valproate and carbamazepine in themselves are not known to cause rhabdomyolysis, we cannot be certain whether this patient was predisposed in some way because of interaction of these drugs (although in therapeutic range) with the detergent preparation.We believe this is the first reported case of a household detergent-induced rhabdomyolysis complicated by dialysis-dependent ARF.

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