Case Report

Alcohol-induced Rhabdomyolysis: A Disease With Potential Pitfalls

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Abstract

Non-traumatic rhabdomyolysis induced by alcohol appears to cause serious clinical implications and could result in catastrophic consequences. We describe a patient who developed rhabdomyolysis following acute alcohol intoxication which led to severe kidney injury requiring dialysis therapy. This case also illuminates the fact that deranged liver function can result from a significant skeletal muscle injury in the absence of a concomitant liver pathology.

Keywords

Acute kidney injury, Alcohol, Dialysis, Liver dysfunction, Rhabdomyolysis

Key learning points

- Alcohol-induced rhabdomyolysis can lead to fatal consequences, such as cardiac arrhythmias, electrolytes imbalances, and kidney failure
- Alcohol intoxication can exacerbate rhabdomyolysis- related AKI, and associated with worse clinical outcomes
- Rhabdomyolysis should be included in differentials for abnormal liver function test in the absence of apparent causes of liver injury
- Further studies should examine the long-term prognosis of injured kidney and skeletal muscles after rhabdomyolysis

Case history

A 38-year-old homeless man was hospitalized after being found on the floor with depressed consciousness at a local night shelter. He was an ex-intravenous drug user and had past history of excessive alcohol consumption and chronic hepatitis C infection. He was heavily intoxicated with strong alcohol breath when he was attended by paramedics. On arrival, he had Glasgow Coma Scale 5/15 with low capillary glucose level (3.8 mmol/L), for which he was treated with a bolus intravenous dextrose and a trial of naloxone injections. His GCS had subsequently improved to 12/15. Physical examination was unremarkable. He was afebrile with a pulse rate of 90/min and blood pressure 110/80 mmHg. Initial laboratory results were as follows: white cell 18.6×10^9/L (4-10), haemoglobin 154 g/L (120-150) , platelets 172×10^9/L (150-410), C-reactive protein 135 mg/L (0-10), serum creatinine 197 µmol/L (45-84), urea 4.7 mmol/L (2.5-7), potassium 4.1 mmol/L (3.5-5.5), adjusted calcium 2.03 mmol/L (2.2-2.6), phosphate 1.31 mmol/L (0.8-1.4), creatine kinase (CK) was 7121 U/L (55-170), lactate dehydrogenase 1592 U/L (125-243). His liver function was deranged with alanine aminotransferase (ALT) of 1189 IU/L (5-65), alkaline phosphatase 113 IU/L (30-130), gamma-glytamyl transpeptidase 488 IU/L (11-50), total bilirubin 10 umol/L (<20). He had normal coagulation, with INR of 1.1 (0.9-1.3). He received aggressive intravenous fluids and a broad spectrum antibiotic to cover for an infection. Over the next 48 hours, he developed oliguria with a progressive deterioration in his renal function, with a creatinine rise to 593 µmol/L, in parallel with CK of 35364 U/L and ALT of 2336 IU/L. Abdominal ultrasonography showed a mild fatty liver, otherwise unremarkable. Virology screen including Hepatitis A and E, Epstein-Barr Virus, Cytomegalovirus revealed no evidence of active infection. Full autoimmune hepatitis panel was also normal. He was commenced on acute intermittent haemodialysis, and has remained dialysis dependent to date as his renal function has failed to recover. His CK, LDH, and ALT levels had gradually returned to their baseline.

Discussion

Rhabdomyolysis is a clinical syndrome which commonly occurs as a consequence of significant skeletal muscle injury. It can lead to severe tissue necrosis, and a subsequent release of intra-muscular contents into the circulation may potentially result in life-threatening complications, such as acute kidney injury (AKI), disseminated intravascular coagulation, electrolyte disturbances, cardiac dysrhythmias, and limbs ischaemia due to compartment syndrome.[1-2] The leakage of myoglobin, creatine phosphokinase (CK), lactate dehydrogenase (LDH), potassium, and phosphate from damaged muscle into circulation is observed, along with typical clinical presentations; muscle pain, weakness, and tea-coloured urine.[1-2]

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In the majority of cases, the culprits of rhabdomyolysis can be easily identified through a careful history and physical examination, especially traumatic causes due to a crush injury or prolonged immobilization.[1] However, more comprehensive clinical works and laboratory evaluation may be required in non-traumatic aetiologies, such as drugs, toxins, infections, metabolic disorders, and hereditary myopathies.[1] In our case, the precipitant of rhabdomyolysis was likely to be an excessive alcohol intake, although a period of immobilization due to depressed consciousness following intoxication could be taken into consideration. Acute ethanol overdose has been recognised as one of the rare forms of non-traumatic rhabdomyolysis, which was demonstrated in previous case studies, and in one animal model, it was proven to exacerbate rhabdomyolysis related organs dysfunction, especially kidney injury.[3]

The clinical implication of rhabdomyolysis may vary from raised serum CK without any symptoms to multi-organ involvement. Acute renal impairment is considered the most common manifestation which occurs in about 10-50% of all cases, and in one retrospective study involving 475 hospitalized patients with rhabdomyolysis, the incidence of AKI was 46%.[4] It usually occurs as a result of renal vasoconstriction, direct tubular injury and tubular obstruction due to pigmented casts. There is a weak correlation between CK values and risk of AKI; patients with CK levels at admission less than 20,000 U/L are associated with a lower incidence of AKI, although other contributing factors including sepsis, volume depletion, and acidosis may still precipitate AKI even at the low levels of CK.[5] Early recognition and timely implementation of an appropriate intervention is a key in management of rhabdomyolysis to prevent fatal consequences, such as refractory hyperkalaemia and volume overload requiring acute renal replacement therapy and an admission to intensive care unit for vital organs support. The cornerstone of the management is an aggressive intravenous fluids therapy with crystalloid (preferably isotonic saline), then followed by urine alkalinisation and osmotic diuresis with bicarbonate and mannitol; the latter options remain controversial in practice and appear less effective in oliguric state.[1-2] Although there is a good chance of renal recovery in the majority of the cases, further clinical studies are still required to better inform the long-term prognosis of AKI in rhabdomyolysis in terms of mortality and risk of progression to chronic kidney disease, which has not been well described in the current literature.

It has been observed that about 25% of patients with rhabdomyolysis may experience some degree of deranged liver function, mainly presents with elevated aminotransferases, along with other intramuscular constituents such as CK and LDH.[1,6] Little is known about an underlying mechanism, but has been postulated that the leakage of proteases after muscle breakdown is thought to be responsible.[6] Clinicians would normally assume that a coexisting liver pathology is present when a significant elevation of aminotransferases is observed in the setting of rhabdomyolysis, even without supporting clinical evidence, or pre-existing liver conditions. This may render a patient into undergoing further unnecessary investigations, such as comprehensive blood works, imaging studies, and liver biopsy. In previous observational studies, the relationship between raised liver enzymes and rhabdomyolysis was addressed and demonstrated a strong association, which in turn implied that abnormal AST and ALT may not be reliable markers of liver injury in the context of muscle injury.[6-8]

In our patient, the striking feature is a massive rise in serum ALT level at above 60 times upper limit of normal, which would be suggestive of hypoxic-ischaemic hepatitis in normal circumstances. However, there was no evidence to support a circulatory or hypoxic failure, toxins, and viral hepatitis in our case. Except for the presence of a background history of chronic hepatitis C infection with stable liver function, the cause of deranged liver function was not evident from a full viral screen, autoimmune serology, and ultrasound examination. Some might argue that acute alcohol overdose as a possible cause of acute liver injury, but it may partially be accountable given the fact that in alcoholic hepatitis AST/ALT ratio is usually more than 2, and AST is less than 500 IU/L.[9] On this basis, we conclude that our patient's profoundly abnormal liver function is largely a representative of rhabdomyolysis.

In summary, we present a case of rhabdomyolysis sustained after acute alcohol intoxication and a period of immobilization, which was complicated with acute kidney injury requiring an immediate dialysis support and became dialysis dependent due to a failed renal recovery. Early recognition and timely intervention is a key in the management of rhabdomyolysis to prevent lethal consequences. This case also reminds acute care physicians to raise their awareness about the crucial fact that an elevation in serum aminotransferases of any degree can be observed in rhabdomyolysis without accompanying hepatic injury. To our knowledge, this is a rare clinical entity that has only been mentioned in a very few case reports.

Conflict of interest

None declared.

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