

Rhabdomyolysis following acute alcohol intoxication

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SUMMARY

The case of a fit young man who developed rhabdomyolysis after a short period of immobilization following acute alcohol intoxication is described. Rhabdomyolysis should be considered in an intoxicated patient presenting with muscle tenderness, particularly after immobilization.

Key words: alcohol, compartment syndrome, rhabdomyolysis

INTRODUCTION

Rhabdomyolysis has been previously described in chronic alcoholics and also in binge drinkers in association with other drugs.^{1,2} We report a case of rhabdomyolysis occurring in a previously healthy male in whom the precipitating cause appears to be acute alcohol intoxication and immobility.

CASE HISTORY

A 22-year-old man presented with a painful weak right leg and inability to pass urine. The night before admission he had consumed over 20 units of alcohol and fallen asleep at the top of the stairs for approximately 4 h.

On admission he was drowsy (Glasgow Coma Scale 14), with a tachycardia of 98, blood pressure of 110/75 mmHg and peripheral cyanosis. He had a palpable tender bladder which was catheterized with a residual of 900 ml of dark brown urine. His right leg was swollen and tense with petechial haemorrhages. He was unable to move the leg but passive movement resulted in severe pain. Diminished pulses were palpable in the right foot.

Abnormal laboratory findings included the following: hyperkalaemia (7.0 mmol L⁻¹), leucocytosis, and acidosis (bicarbonate 13.6 mmol L⁻¹) with raised urea (12.3 mmol L⁻¹) and creatinine

(148 mmol L⁻¹) levels. Serum creatinine kinase was grossly elevated at over 120 000 IU L⁻¹.

Following aggressive intravenous fluid management including mannitol and bicarbonate, low-dose dopamine (2.5 µg kg⁻¹ min⁻¹) and surgical decompression of the leg, the patient's renal function recovered. His recovery was complicated by a pulmonary embolism which occurred whilst he was awaiting skin grafting of the operated leg.

DISCUSSION

Rhabdomyolysis occurs following injury to skeletal muscle resulting in leakage of the intracellular contents into the circulation. It has been reported in association with a variety of seemingly unrelated insults, and may be multifactorial.¹ A number of drugs and toxins have been implicated.³ Alcohol-related rhabdomyolysis has been attributed to a direct toxic effect on muscle, or secondary metabolic changes associated with alcohol abuse.^{1,4} However, its action as a central nervous system depressant resulting in immobilization cannot be discounted.

Massive rhabdomyolysis can produce myoglobinuria and acute renal failure, hyperkalaemia, metabolic acidosis and disseminated intravascular coagulation. Damage to muscle results in the release of osmotically active agents into the interstitial space of fascial compartments, leading to elevated pressures. The development of compartment syndrome may be delayed for several days after the initial insult to muscle.¹

Renal failure has also been reported in association with binge drinking and the use of non-steroidal anti-inflammatory drugs. In this situation the mechanism may be alcohol-induced volume depletion, resulting in compromised renal haemodynamics, together with inhibition of renal prostaglandin synthesis. Overt rhabdomyolysis did not occur in the patient examined in this report.²

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The combination of limb compression and alcohol intoxication can be associated with extensive rhabdomyolysis, and the associated renal failure has been shown to be preventable by early volume replacement, together with mannitol and bicarbonate infusion.⁵

In addition to the prevention of renal failure, a high index of suspicion should be maintained for the development of compartment syndrome as the cause or effect of rhabdomyolysis. Adequate distal pulses do not rule out the presence of a compartment syndrome, and emergency fasciotomy may be required.

In this case, sufficient alcohol was consumed to produce depression of consciousness. The period of immobilization in our patient was relatively short, and alcohol may have contributed to the development of rhabdomyolysis and compartment syndrome. In an intoxicated patient presenting with muscle tenderness following a period of

immobilization it is important to consider rhabdomyolysis, as tenderness may be incorrectly attributed to local trauma.

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