Case Reports

Alcohol Related Non-traumatic Rhabdomyolysis and Compartment Syndrome

JPL Ong & LA Thomas

Abstract

Rhabdomyolysis is a serious and life-threatening condition in which skeletal muscle is damaged, commonly resulting in acute renal failure. The causes of this clinical entity can be traumatic and non-traumatic. In the latter group, alcohol is the commonest cause. This report describes the case of a 25 year old man who presented with rhabdomyolysis leading to acute renal failure after an alcohol binge. He presented with painful legs and lower extremity compartment syndrome. The patient recovered with surgical fasciotomy and renal support. This case illustrates the importance of early recognition and treatment of alcohol related non-traumatic rhabdomyolysis and compartment syndrome.

Abbreviations

creatine kinase; CK

Keywords

Rhabdomyolysis, Compartment syndrome, Acute renal failure.

Teaching Points

- Rhabdomyolysis is an important cause of renal failure.
- Alcohol related non-traumatic rhabdomyolysis can present as compartment syndrome.
- Early recognition and prompt treatment of these conditions is important to prevent serious complications.

Case History

A 25 year old man presented with painful, swollen legs after a heavy alcohol binge the night before. He had consumed 9 cans of beer and 4 litres of cider. His girlfriend reported that he had spent the night drunk with his knees flexed under his thighs. He was in a state of unconsciousness for 7 hours. When he woke up his legs were extremely painful and he had difficulty in walking. He gave a history of epilepsy, well controlled on Carbamazepine, and a history of high alcohol consumption for many years. He had no history of recent seizure.

On admission, he had a normal temperature and had no clinical signs of liver disease. His blood pressure was 188/114 mmHg, and he was in extreme pain from both his legs. Both calves were painful, swollen, tense and tender on palpation and movement. His lower legs were cold and mottled, and peripheral pulses could not be felt in either foot.

Results of laboratory investigations are shown in the table. The marked elevation of Creatinine Kinase (CK) was strongly suggestive of rhabdomyolysis. Urine was positive for myoglobin. Other biochemical abnormalities (reference ranges shown in brackets) include a raised C-reactive protein at 249 mg/L (1–10mg/L), alanine amino transaminase at 997 U/L (0–40 U/L), phosphate 2.11 mmol/L (0.80–1.40 mmol/L) and uric acid of 0.52 (0.15–0.42) mmol/L.

Initially aggressive fluid replacement and alkalization of urine were undertaken. However, he became oliguric with deteriorating renal function (see table 1). Haemodialysis was started. He had bilateral four-compartment fasciotomies with split skin grafting four days later. His hospital stay was prolonged as a consequence of infection of the fasciotomy wounds. He was discharged from hospital 2 weeks after admission with a complete recovery of renal function. At follow-up clinic 2 months later, he remains well.

Discussion

This case clearly illustrates the association of alcohol related non-traumatic rhabdomyolysis and compartment syndrome.

Alcohol intoxication contributes to rhabdomyolysis due to a combination of immobilization and direct myotoxicity of ethanol.[3,4,6] During alcohol intoxication, the sedative effect of ethanol on the central nervous system leads to prolonged unconsciousness, which may result in muscle compression and ischaemia.

Due to rapid release of osmotically active agents into the interstitial space and rapid increase of compartment pressure, patients with non-traumatic rhabdomyolysis with alcohol intoxication are likely to present with severe muscle pain and compartment syndrome. Compartment syndrome is defined as a condition in which increased pressure within a limited space impairs capillary perfusion of the tissue within that space, resulting in ischaemia. In this case, the increase in compartment pressure may have

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resulted from his prolonged posture with knees flexed behind his thighs after an alcohol binge. The muscle injury led to oedema formation and further impairment of normal circulation.[5] Early decompressive surgical fasciotomy, as was undertaken in our case, is necessary to prevent irreversible tissue ischaemia.

The diagnosis of rhabdomyolysis is achieved through a combination of clinical features and laboratory findings. The urine often has a reddish-brown colour, indicating the presence of myoglobin, which may be confirmed on biochemical analysis. However, the main laboratory finding is a markedly increased serum CK level.

Renal impairment is a common complication of rhabdomyolysis.[1] Myoglobin, released from damaged muscle tissue, is freely filtered by the glomeruli and reaches the tubules, where it may cause obstruction, renal tubule necrosis and acute renal failure. Hypovolemia and the presence of acidic urine contribute to the development of renal failure following rhabdomyolysis.[2] In a 1988 review, Ward suggested that predictors for the development of renal failure include peak CK of > 6,000 IU/L, dehydration (haematocrit > 0.50), sepsis, hyperkalemia or hyperphosphatemia on admission.[8]

The main mechanisms leading to renal injury are renal vasoconstriction, intraluminal cast formation and direct heme protein-induced cytotoxicity.[2,9] Renal vasoconstriction is favoured by muscle necrosis, which leads to hypovolemia and the activation of cytokines. This increases capillary permeability and the binding of heme protein to nitric oxide.[9] Casts are produced after the filtration of myoglobin through the glomerular basement membrane, which causes water reabsorption and a rise in myoglobin concentration. Following this, in the presence of acidic urine, myoglobin precipitation takes place and causes obstructive cast formation.[3] It has been demonstrated that when urinary pH was kept at 8.0, 78% of the myoglobin was excreted by the kidneys.[9] Myoglobin may also induce the release of free iron, which catalyses free radical production and further enhances ischemic tubule damage.[2,9] Alkaline conditions prevent this effect by stabilizing the reactive ferryl myoglobin complex.[3]

The initial management of patients with rhabdomyolysis requires administration of intravenous fluid (e.g. normal saline) to maintain a high urine output (> 150ml /hour)[10] Alkalization of urine, by the intravenous administration of sodium bicarbonate will reduce the formation of myoglobin casts in the renal tubules.[10] Where urine output remains low the use of intravenous mannitol (1g/Kg as 20% solution) may help to achieve the necessary diuresis. Homsi et al., in a retrospective study, showed that progression to established renal failure following rhabdomyolysis could be totally avoided with prophylactic treatment in which volume repletion was achieved using saline solution.[11]

Day	1	2	3	6	8	10	12	16	22
Urea (mmol/L)	12.2	18.3	26.3	36.5	31.4	17.8	10.7	6.2	2.7
Creatinine (umol/L)	360	588	998	1266	1070	696	493	156	111
Potassium (mmol/L)	5.3	5.1	5.0	7.9	6.9	5.8	3.7	3.2	4.4
Sodium (mmol/L)	125	125	124	122	121	127	133	143	141
CK (IU/L)	130,000 0	69,934	34,880	17,920	1043	–	462		
Haemoglobin (g/dl)	19.5	–	13.9	8.8	6.9	7.3	–	9.9	13.0
Platelet (x10^9/l)	241	-	178	215	315	447	-	508	300

Table 1. Laboratory findings during hospitalisation

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