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A case of acute renal failure and compartment syndrome after an alcoholic binge

N Sofat, S Bell, J Turner, A N Warrens

Abstract

A 25 year old man presented with anuria and bilateral leg pain two days after an alcoholic binge. He subsequently developed rhabdomyolysis causing acute renal failure, with compartment syndrome of both lower legs. This required urgent dialysis and fasciotomy respectively within six hours of admission. He remained dialysis dependent for three weeks and only after four months was he able to weight bear on both legs. Alcohol is a leading cause of rhabdomyolysis. Early recognition and prompt treatment is essential to prevent serious complications.

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Department of Medicine, Hammersmith Hospital
N Sofat
S Bell
J Turner
A N Warrens

Correspondence to:
Dr A N Warrens, Senior Lecturer and Honorary Consultant in Renal Medicine and Immunology, Hammersmith Hospital, Du Cane Road, London W12 OHS.

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Case report

A 25 year old man who had been previously fit and well presented to the accident and emergency (A&E) department with two days of pain and swelling in both his legs with anuria. Three days before presentation he had drunk 42 units of alcohol in a single evening. After 12 hours asleep on the floor, he was fully conscious but unable to walk owing to acute pain and swelling in his legs. There was no previous history of alcohol abuse and he was on no regular medication. He denied taking any recreational drugs. There was no other relevant past medical history.

On examination he was alert and orientated. He was pale and clammy with a temperature of 36.5°C. Clinically, he was dehydrated: he had a blood pressure of 130/60 mm Hg lying, 100/50 mm Hg sitting, with a tachycardia of 124 beats/min, and his jugular venous pressure was not detectable. Respiratory and abdominal examination was unremarkable. He had marked bilateral tense swelling of his legs below his knees. No pedal pulses were palpable and his pedal skin was mottled and cold. He had poor capillary return in both feet. Neurological examination revealed bilateral foot drop with absent ankle reflexes. He had bilateral downgoing plantars with paraesthesia below his knees.

Urethral catheterisation revealed only 20 ml of residual dark brown urine, which was positive for blood and protein on dipstick testing but on microscopy there were no red cells or casts. His urine pH was 6.0. Subsequently, the patient remained anuric.

Biochemical investigations were performed urgently (see table 1).

Electrocardiography showed tented T waves, consistent with hyperkalaemia as illustrated in fig 1.

The diagnoses of acute renal failure due to rhabdomyolysis and bilateral critical compartment syndrome of his lower legs were made. A central venous line was inserted to optimise fluid replacement with colloid. Emergency management of hyperkalaemia using 10 ml of calcium chloride 10% intravenously and 15 units of insulin in 50 ml 50% dextrose was initiated. Sodium bicarbonate intravenously at a concentration of 50 mmol (50 ml of 8.4% solution) was also given to achieve urinary alkalinisation. He was haemodialysed acutely for two hours owing to persistent hyperkalaemia and the need for urgent surgery. Doppler ultrasound of his legs confirmed critical ischaemia—both feet had weak dorsalis pedis pulses but absent posterior tibial impulses. The patient underwent a bilateral triple lower leg fasciotomy within six hours of presentation.

Postoperatively he underwent haemofiltration on the intensive care unit for 24 hours followed by haemodialysis for three weeks. His renal function resolved back to normal values during this time and his leg wounds healed. He was able to weight bear after four months of intensive physiotherapy and rehabilitation. Five months after admission he is now able to walk unaided and plans to resume work as a mechanic.

Table 1 Investigations performed

Sodium	121 mmol/l (135–145)	Creatinine kinase	>100 000 IU/l (25–70)
Potassium	7.4 mmol/l (3.5–5.0)	Haemoglobin	157 g/l (115–160)
Urea	20.2 mmol/l (2.5–6.7)	White cells	15×10^9/l (4–11)
Creatinine	446 μmol/l (70–150)	Platelets	305×10^9/l (150–500)
Calcium	1.70 mmol/l (2.12–2.65)	APTT	34 seconds (24–32)
Phosphate	3.21 mmol/l (0.8–1.5)	Thrombin time	11 seconds (15–19)
Bicarbonate	16 mmol/l (24–30)	Bilirubin	35 mmol/l (2–17)
Glucose	8.9 mmol/l (3.5–5.5)	Alkaline phosphatase	99 IU/l (30–130)
		Aspartate transaminase	2621 IU/l (5–35)
Arterial blood gases (on air)		Uric acid	1.02 mmol/l (0.1–0.4)
pH	7.38 (7.35–7.45)	Albumin	24 g/l (33–55)
P_{CO_2}	3.8 kPa (4.7–6.0)	C reactive protein	35 mg/l (<10)
P_{O_2}	14.2 kPa (>10.6)	Erythrocyte sedimentation rate	10 mm/hour
Base excess	−7.1 mmol/l (+/−2)	γ-Glutamyltransferase	205 IU/l (11–51)

Normal ranges are given in parentheses.
APTT=activated partial thromboplastin time.

Discussion

The case presented here illustrates how an alcoholic binge may cause acute rhabdomyolysis and compartment syndrome. The patient was managed as a medical and surgical emergency with multiple critical care needs.

Muscle accounts for approximately 40% of total body mass and falls victim to a wide variety of toxic, ischaemic, infectious, inflammatory, and metabolic insults. Acute rhabdomyolysis was originally described as a distinct entity by Bywaters and Beall, who described the "crush syndrome" after the bombing raids

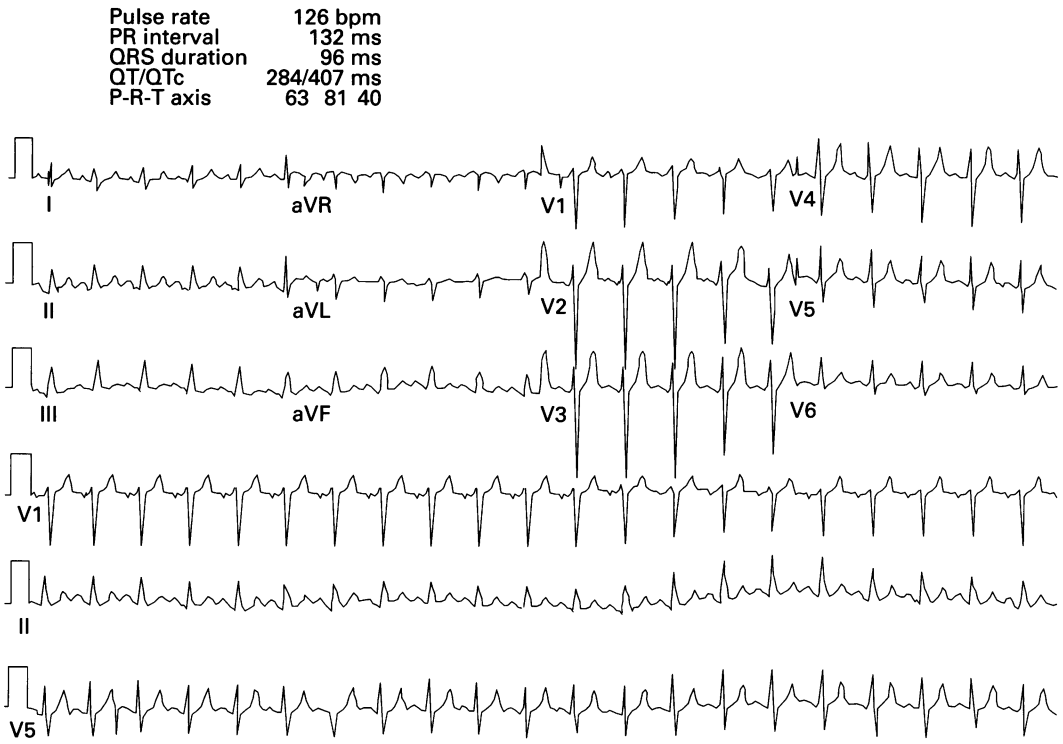

Figure 1 Electrocardiogram of patient showing tented T waves consistent with hyperkalaemia.

of London during second world war.[1] The role of alcohol is of particular importance as it accounts for at least 20% of all cases of acute rhabdomyolysis.[2]

Rhabdomyolysis is a form of muscle necrosis triggered by derangements in oxidative or glycolytic energy production and the resulting ATP depletion. In the case described, the earliest insult occurred in the form of a "pressure-stress myopathy", in which external pressure/tension on muscle increases the influx of cellular sodium and calcium into the intracellular compartment down their concentration gradients.[3] External pressure induced occlusion of the microcirculation also occurs, rapidly depleting the ambient and myoglobin oxygen content. Cells may remain viable for considerable periods of time, since the pressure induced vascular occlusion limits calcium delivery to ischaemic tissues, which itself delays the onset of necrosis.[4] Mitochondrial oxygen free radical production is markedly reduced during ischaemia and acidosis, both intracellular and extracellular, also occurs. The restoration of blood flow results in an influx of neutrophils, which can then reocclude the microcirculation and release proteases and free radicals into the microenvironment. Therefore, although the crush lesion sets the stage for rhabdomyolysis, many of the critical events occur during the reperfusion period. It is during this latter period that myoglobin gains ready access into the circulation. Extracellular fluid depletion also results because of fluid sequestration in muscle and other soft tissues.

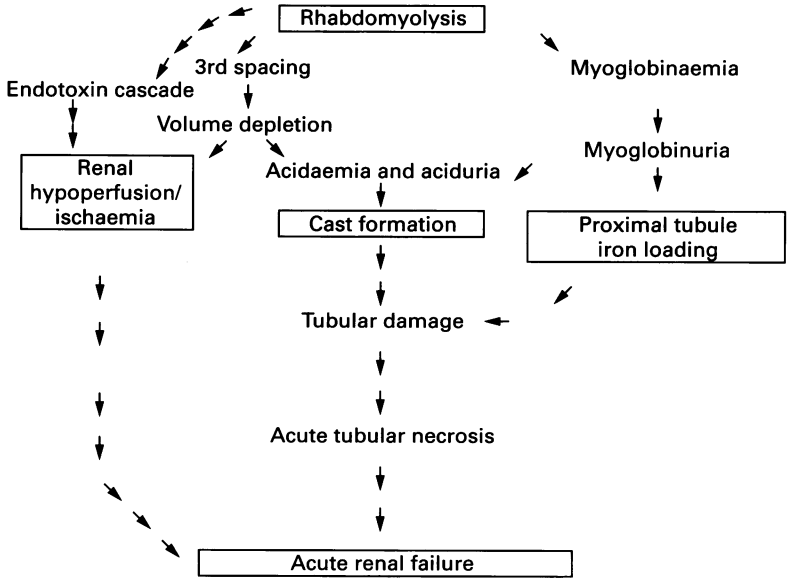

Figure 2 Pathways underlying rhabdomyolysis induced renal injury.

The spectrum of potential complications after acute rhabdomyolysis includes:

- Metabolic: hyperkalaemia, a rapidly rising serum creatinine, hyperuricaemia, hypocalcaemia and hypercalcaemia, hyperphosphataemia, metabolic acidosis.
- Haematological: disseminated intravascular coagulation.
- Cardiovascular and respiratory: cardiomyopathy and respiratory failure.
- Localised oedema: leads to the development of compression syndromes in the anterior tibial, soleal, peroneal, lateral thigh, gluteal, deltoid, and volar forearm compartments with consequent peripheral neuropathies.[5]

The cause of acute renal failure after muscle necrosis is complex and the mechanisms described fall into three categories:

(1) The impairment of renal vascular flow due to the activation of the renin-angiotensin system with increased sympathetic activity, altered prostaglandin production, high circu-

lating concentrations of antidiuretic hormone, and the deposition of microthrombi.[3]

(2) The tubular destruction by myoglobin casts or uric acid crystals, with passive back diffusion of glomerular filtrate, leading to acute tubular necrosis.[3 5]

(3) Direct tubular toxicity of ferrihaemate, which is a product of the dissociation of myoglobin at pH 5.6. Animal studies have shown that ferrihaemate infusion produces a dose dependent deterioration in renal function.[5] There is also depression of renal tubular transport mechanisms,[5] cell swelling, and death.[6 7]

The mechanisms described above are illustrated in fig 2.

Successful management of rhabdomyolysis depends on early diagnosis and, therefore, a low threshold of clinical suspicion. The initial treatment is aimed at correcting:

- Acute metabolic disturbances: calcium gluconate is given to protect the myocardium against arrhythmias and insulin and dextrose to correct hyperkalaemia.
- Hypovolaemia: is best managed by establishing central intravenous access and optimum fluid replacement.
- Elimination of toxic substances may need to be encouraged by diuresis, haemofiltration, or haemodialysis.
- Myoglobinuric acute renal failure: it is often suggested that this should initially be managed with intravenous sodium bicarbonate. The object is to maintain an alkaline urine and prevent dissociation of myoglobin to its toxic metabolite, ferrihaemate. Diuretics, notably mannitol,[8] also have a role by promoting a diuresis, thereby diluting nephrotoxic substances, and "flushing through" blocked renal tubules.[5 7] Data supporting the use of mannitol include studies that have shown that it is highly effective in promoting haem protein/haem iron excretion by working as a proximal acting diuretic.[9] Mannitol is also a potent renal vasodilator, and may therefore improve renal perfusion, possibly preventing renal ischaemic damage.[10]
- Disseminated intravascular coagulation usually only requires treatment with clotting factors if there is bleeding.
- Hypocalcaemia will usually correct itself spontaneously and attempts at treating it can result in rebound hypercalcaemia.
- Compartment syndrome: complications can be prevented by careful clinical and/or intracompartmental pressure monitoring. The diagnosis is based on clinical assessment of tissue perfusion.[11] The common symptom is pain, in particular, painful passive stretching of the compartment. The presence of distal pulses in relation to the lesion does not exclude the diagnosis since a compartment pressure of 40 to 60 mm Hg may be sufficient to block capillary and arteriolar flow, but not to compress large arteries. Doppler studies can be performed in the acute situation, but more accurate measurements of compartment pressures can be made with devices such as the slit catheter.[9] The slit catheter was developed by Bourne and Rorabeck, and consists of five 3 mm long slits approximately 60 degrees apart, cut into the tip of a length of polyethylene tubing. The catheter is inserted into the tissue via a 14 gauge needle. The saline filled slit catheter is connected to a sterile transducer. A drop of saline is raised at the tip of the catheter, and the catheter is threaded through the needle into the compartment. The needle is then withdrawn. Tissue measurements are checked within the compartment by palpation, which evokes brisk responses from the readout. A compartment pressure of 30 mm Hg is generally considered as the critical pressure for fasciotomy. This technique was not used in the case described in view of the obvious clinical signs and the need for urgent decompressive fasciotomy.

In conclusion, we have presented a case of acute renal failure resulting from alcohol induced rhabdomyolysis with associated compartment syndromes of the lower legs. The mechanisms of tissue injury and management have been discussed. The case described highlights the importance of early recognition of the warning symptoms and signs in managing such patients who present via an A&E department, and the need for swift acute care and specialist referral.

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