He's been 'out' all night

GORDIAN FULDE
MB BS, FRACS, FRCS(Ed), FACEM, FRCS/RCP(A&E)Ed

Emergencies can spring up at any time and in many incarnations. Are you adequately equipped to deal with them? Each month we present a case study in emergency medicine based on real cases and events. Would you have been able to help this patient?

You are a GP doing a regular shift in the emergency department of an inner city hospital. It is the middle of the 'round' – an eight-hourly ritual of taking stock of patients and problems in the emergency department.

The 'bat phone' rings. All emergency departments have one. It is a phone without a dial; it has a peculiar ring or siren and is a direct line from ambulance. The emergency department is being notified about a critically ill patient 'three minutes out'.

The patient

The ambulance arrives and the patient is brought into a prepared resuscitation cubicle. The patient is a young man presumed to have taken a heroin overdose. He is unconscious and appears to have been 'down' for quite some hours.

The patient is transferred onto a resuscitation trolley, and the ambulance team give their report.

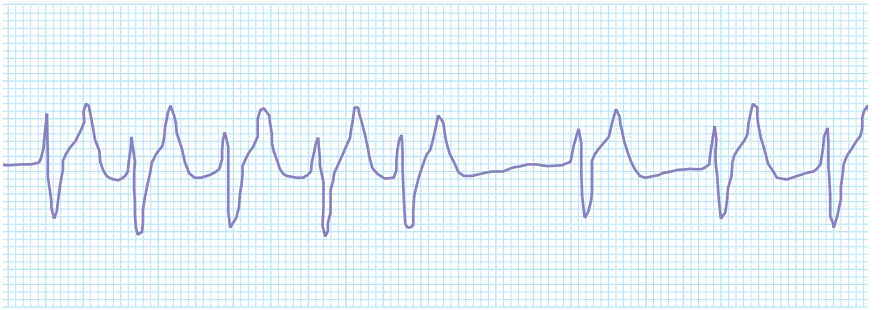

Figure 1. ECG showing signs of hyperkalaemia – T wave elevation.

Table 1. Emergency management of hyperkalaemia

- Calcium, 10 mL of 10% solution, to counteract the effect of hyperkalaemia on the myocardium
- Glucose, 100 mL of 50% glucose, with 20 units of short-acting insulin, to temporarily drive potassium into the cells
- Sodium bicarbonate, 100 mmol, again to temporarily drive potassium into the cells
- Ion exchange resin – for example, calcium resonium 30 to 60 mg every two to six hours, either orally or rectally
- Dialysis may be required for severe or resistant hyperkalaemia

Source: Reproduced with permission from Hillman K. Metabolic disorders. In: Fulde GWO, ed. Emergency medicine. 3rd ed. Sydney: MacLennan & Petty, 1998: 312.

The ambulance report

The patient was found collapsed on the floor in cheap accommodation. When seen by ambulance, he was unconscious, nonresponsive and cyanosed, and had:

- constricted pupils
- a Glasgow Coma Score (GCS) of 3 (lowest possible)
- a respiratory rate of 4 breaths per minute
- a pulse of 100 beats per minute
- no recordable blood pressure.

The ambulance team had some difficulty finding a good vein, but managed to do so and to administer naloxone 4 mg intravenously through the antecubital vein. This increased the respirations to a fast 44 breaths per minute and the GCS to 6 (normal 15), but there was still no recordable blood pressure.

The ambulance team also reported that the patient's left arm was grossly

MEDICI GRAPHICS

Figure 2. The classic colour of urine in crush syndrome.

Professor Fulde is Director, Emergency Department, St Vincent's Hospital and Associate Professor in Emergency Medicine, University of New South Wales, Sydney, NSW.

swollen, cold, hard to palpation, with no pulse and no movement from it. Elsewhere, the patient was flexing to pain.

An ECG was performed on site showed atrial fibrillation and very marked, peaked T waves (Figure 1). As per ambulance protocol, a venous tourniquet was applied to the arm to minimise the effects of muscle breakdown products (potassium and myoglobin) before transporting the patient to hospital.

In the emergency department

Under the direction of a very competent, young emergency medicine specialist, with plenty of support, everything seemed to be happening at once. The fact that it was 8.15 a.m. added to the flurry, because both night and day staff were present, as well as medical students.

Very early in the action, a femoral artery puncture was taken for arterial blood gases and an urgent potassium level. The patient's arm and T wave elevation made it clinically obvious that the patient had raised potassium. This suspicion was confirmed by the laboratory results – the patient's potassium was 9.3 mmol/L (normal range, 3.5 to 5.0 mmol/L).

Treatment for hyperkalaemia (outlined in Table 1) was continued.The patient was 'attacked' from all sides.

In order to get good oxygenation and control of the airway the patient needed to have an endotracheal tube. However, the usual drugs for rapid sequence induction could not be used because of the hyperkalaemia. Suxamethonium, a depolarising agent, can cause potassium levels

Table 2. General features of a crush syndrome

- Myoglobinuria
- Acute renal failure/acute tubular necrosis
- Hypovolaemia
- ECG changes of hyperkalaemia (T wave elevation, widened QRS complex, prolonged PR interval, deepened S wave)
- Disseminated intravascular coagulation

Source: Reproduced with permission from Woodruff P. The crush syndrome. In: Fulde GWO, ed. Emergency medicine. 3rd ed. Sydney: MacLennan & Petty, 1998: 177.

to rise, resulting from muscle fasciculations. Midazolam 5 mg IVI was used instead.

A large intravenous line was inserted into the patient's femoral vein because rehydration and fluid management were pivotal. A urinary catheter was inserted. The patient's urine was the classic colour seen in crush syndrome (see Figure 2). One by one all the features of crush syndrome became evident (see Table 2).

By now the surgeons had appeared, having been summoned urgently. The only hope for saving the patient's arm was to perform an immediate fasciotomy.

The background

During all this intense activity, the social worker was gathering background information by making phone calls to numbers in the patient's wallet.

It seems the patient, who had recently been discharged from prison, had broken up with his girlfriend. He had been attempting to commit suicide by heroin overdose.

The outcome

The fasciotomy was done very quickly and the patient transferred to intensive care postop. Dialysis was avoided by a forced alkaline diuresis and IV acetazolamide.

Over the next few days, the patient recovered physically and mentally; however, the left arm remained dead and serious discussions about amputation were under way.

What is crush syndrome?

Crush injuries arise from direct trauma to the muscle mass or from ischaemia, resulting in rhabdomyolysis. These injuries may be followed by a crush syndrome, in which there may be muscle swelling, myoglobinuria (hence, the red urine), acute renal failure, shock, hyperkalaemia (as potassium is released from damaged muscle) and disseminated intravascular coagulation.

Crush injuries may be caused by entrapment (e.g. earthquake), high energy exchange (e.g. car accidents) or pressure effects (e.g. coma). In this case, the patient had been lying unconscious on his arm for a long time before he was found. MT

Bibliography

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