EMERGENCY CASEBOOK

# Lower limb amputation with CPR in progress: recovery following prolonged cardiac arrest

**R Wise, I Higginson, J Benger, N Rawlinson**

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Intravenous drug users (IVDUs) often present to the emergency services with the medical complications of drug use. We report a case in which an acutely ischaemic lower limb of one such patient was thought to be the cause of cardiac arrest occurring during treatment in the emergency department (ED). Amputation of the limb was performed with cardiopulmonary resuscitation (CPR) in progress, spontaneous cardiac output was restored, and the patient made an excellent neurological recovery despite a total arrest time of 85 minutes. Possible causes of cardiac arrest, in relation to the release of potassium and metabolic toxins are discussed, as well as the decision making processes of the involved clinicians and other possible management strategies.

A 23 year old man was brought to the emergency department (ED) having being found by a housemate on the kitchen floor with an empty syringe and needle next to him. He was a known IVDU. There was no information available as to how long he had been lying in the kitchen, or in what position he was found. On arrival in the ED, he was maintaining his own airway with a respiratory rate of 21 breaths/min. His oxygen saturation was 85% on air, pulse 120 beats/min, blood pressure 112/74 mm Hg, Glasgow Coma Score 14/15, core temperature 29.4℃, and glucose 5.4 mmol/l. His pupils were bilaterally pinpoint, and naloxone was given. Full resuscitation and re-warming were commenced, but over the next 2 hours his right foot became cold and blue.

Doppler examination confirmed femoral and popliteal pulses, but none distal to these. The patient was administered heparin and an urgent surgical referral was made to arrange arteriography and transfer to a nearby vascular unit. His initial blood results were: sodium 146 mmol/l, urea 9.2 mmol/l, creatinine 305 μmol/l, and creatinine kinase 41 000 IU/l. The sample was haemolysed and no potassium result was reported.

The patient had a witnessed asystolic cardiac arrest while the surgeons were making their assessment. Cardiopulmonary resuscitation (CPR) was initiated, with tracheal intubation epinephrine and atrophine were administered. Blood gas measurement showed raised potassium (7.9 mmol/l). The patient regained sinus rhythm with good cardiac output and became restless. A propofol infusion was started. However, shortly afterwards, he developed pulsed ventricular tachycardia, and then a second cardiac arrest with varying rhythms including bradycardic pulseless electrical activity and asystole. It was felt that his ischaemic foot and rhabdomyolysis were causing hyperkalaemia and cardiac arrest. An arterial line showed that constant CPR was generating systolic pressures of 60–70 mmHg. Calcium gluconate, sodium bicarbonate, and dextrose and insulin were administered, but he failed to respond.

A decision was made by the ED consultant (NR), the consultant vascular surgeon, and the consultant intensive care physician to undertake an emergency amputation of the right leg in an attempt to stabilise the patient. This proceeded with CPR in progress. Immediately following the amputation the patient developed a pulseless bradycardia, which was successfully externally paced, resulting in return of a palpable output. An epinephrine infusion was commenced. He subsequently regained sinus rhythm with a good output, and pacing was discontinued. Potassium at this stage was 6.6 mmol/l. Total time in cardiac arrest was 85 minutes.

The patient was transferred to intensive care. He required further proximal amputation, but subsequently made a full neurological recovery.

## DISCUSSION

This was a very unusual clinical situation, and prompted us to examine the literature for more information about options in this circumstance. The cause of the patient's cardiac arrests is uncertain. Hyperkalaemia from rhabdomyolysis is well recognised in IVDUs. It is possible that this patient's potassium was rapidly rising as his limb reperfused, and that this was the cause of his cardiac arrests. This is described in crush syndrome, and would also be similar to the myonephropathic metabolic syndrome described after acute arterial occlusion.[1] It is also possible that other toxins were being released into the circulation, and that these were the cause. Options in this patient might have included haemodialysis[2] and cardiopulmonary bypass,[3] which have been described in the context of hyperkalaemic cardiac arrest. However, these would not have addressed the underlying cause of the hyperkalaemia. The reasoning in this patient was that the ischaemic leg was the cause of his adverse physiological status, and that the cause should be removed. We could find no reports of amputation with CPR in progress, although a case of recurrent hyperkalaemia secondary to fluid loss from leg ulcers, cured by amputation, has recently been reported.[4] Other options would have been clamping of the femoral vessels,[1] or application of a tourniquet (not described). It is not possible to state with certainty that amputation of the leg successfully treated this patient's cardiac arrest. However, there was certainly an improvement in his condition following amputation. In this patient's case there was clearly no harm caused, as the tissue removed was non-viable, and the procedure may have been of benefit.

We conclude that under some circumstances, acute limb ischaemia can also be immediately life threatening. In such cases, the ischaemic limb must be isolated from the circulation. Emergency amputation of non-viable tissue may be indicated, even if the patient is in cardiac arrest.

**Abbreviations:** CPR, cardiopulmonary resuscitation; ED, emergency department; IVDU, intravenous drug user

**Authors' affiliations**
**R Wise, I Higginson, N Rawlinson,** Emergency Department, United Bristol Healthcare Trust, Bristol, UK
**J Benger,** Academic Department of Emergency Care, Emergency Department, United Bristol Healthcare Trust, Bristol, UK

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Correspondence to: Dr J Benger, Academic Department of Emergency Care, Emergency Department, United Bristol Healthcare Trust, Bristol BS2 8HW, UK; jonathan.benger@ubht.nhs.uk

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