Drug abuse can result in several medical complications. Some of these complications may lead to multi-organ failure and are life-threatening. We report such a case after the use of heroin.

**Case report**

A 29-yr-old male with a known history of heroin abuse was found unconscious at home. There had been a period of enforced abstinence from drugs for nearly a year. He was unrousable, leaning forward on his thighs with legs crossed in the so-called ‘lotus’ position. Multiple puncture marks were seen in the left antecubital fossa. On arrival at the accident and emergency department, naloxone was administered i.v. and he regained consciousness. The patient complained of low back pain and he was unable to move his legs. There was no power or sensation in the lower limbs and lower limb reflexes were absent. The electrocardiograph showed intermittent ventricular tachycardia and aberrant broad complex rhythm. Initial blood investigations included a serum potassium concentration of 7.6 mmol litre$^{-1}$ and a serum creatinine concentration of 252 µmol litre$^{-1}$.

Shortly after admission, ventricular fibrillation developed which was reversed with electrical defibrillation. However, the cardiac rhythm remained unstable, with ventricular tachycardia and aberrant broad complex rhythms leading to further episodes of ventricular fibrillation, requiring electrical defibrillation. The patient was sedated with midazolam and mechanical ventilation of the lungs via a tracheal tube was started. Serum potassium concentration had increased to 10.9 mmol litre$^{-1}$ and was treated with i.v. calcium chloride, sodium bicarbonate and dextrose–insulin infusion. Fixed rate external cardiac pacing was used to control the grossly unstable rhythm. Continuous veno-venous haemofiltration (CVVH) was commenced because the cardiovascular instability precluded haemodialysis despite its superior control of electrolyte disturbances. Inotropic support with epinephrine was needed and the patient was transferred to the intensive care unit (ICU).

By day 2, the patient’s serum potassium concentration had decreased to 6.9 mmol litre$^{-1}$ but there was a marked coagulation abnormality, with an international normalized ratio (INR) of 3.3, activated partial thromboplastin time (APTT) of 142 and fibrin degradation products (FDP) greater than 10 mg litre$^{-1}$. The patient was treated with fresh frozen plasma (FFP). The coagulopathy persisted for 5 days. A diagnosis of rhabdomyolysis was confirmed by a serum myoglobin concentration of >400 000 µg litre$^{-1}$ (Latex agglutination Rapitex; Boehringer Ingelheim; minimum detectable 100 µg litre$^{-1}$; normal range 3–85 µg litre$^{-1}$) and serum creatinine kinase (CK) concentration of 122 000 iu (Fig. 1).
Unusual consequences of heroin overdose

Figure 1 details the time course of serum myoglobin and CK concentrations during the hospital stay. Haemodynamic instability and myoglobinemia resulted in the development of acute renal failure. Haemofiltration and subsequent haemodialysis were required for 26 days (Fig. 2). The diuretic phase of acute renal failure was complicated by hypercalcaemia (Fig. 2) which was treated with disodium pamidrate to slow calcium turnover from bone.

Mechanical ventilation of the lungs was maintained for six days. During this time, a right leg compartment syndrome became apparent. A through-knee amputation was required despite fasciotomies being performed on diagnosis of the compartment syndrome.

A neurological examination undertaken on day 12 revealed a diminished sensation of touch, temperature and pain below the groin creases, with totally absent sensation below the remaining (left) ankle. However, he was able to move the toes but no other limb movements were possible. The plantar, tendo-achilles, patellar and cremasteric reflexes were absent but abdominal reflexes in all four quadrants were present.

On day 34, the patient was discharged from the ICU to the surgical ward. He was kept under observation in the ward where his urea, creatinine, serum myoglobin and CK were within normal limits, although serum calcium was 2.49 mmol litre⁻¹ on day 40. He received physiotherapy and was finally discharged to a drug rehabilitation unit 8 weeks after admission. Overall muscle power in the remaining limb was still substantially below normal (grade 2).

Discussion

We have presented a case of severe rhabdomyolysis, resulting in myoglobinemia, hyperkalaemic cardiac arrest, renal failure, disseminated intravascular coagulation, paraplegia and eventual limb amputation after a prolonged period of immobility as a result of drug overdose. Grossly abnormal calcium metabolism was also seen during the diuretic phase of renal failure, leading to abnormal deposits of calcium all over the body. We believe this case is unique because serum myoglobin concentrations are the highest reported associated with survival and our patient suffered several life-threatening complications associated with heroin and cocaine overdose.

Opioid overdose has been associated with rhabdomyolysis. The pathophysiology of rhabdomyolysis in heroin addiction is obscure and it has been suggested that there is a multifactorial pathogenesis, including acidosis, systemic hypoxia, hypothermia, muscle compression and a direct toxic or immunological effect of the drug or its contaminants. In our case, there was a substantial contribution from direct pressure on the muscles of the lower limbs as a result of the unusual, prolonged ‘lotus’ posture.

Other complications from heroin overdose have been reported, including renal failure associated with rhabdomyolysis, disseminated intravascular coagulation, paraplegia, but all of these complications occurring in one patient is uncommon. Our patient also had repeated cardiac arrests in the early phase, compartment syndrome and calcium deposition as secondary consequences from rhabdomyolysis and renal failure.

Myoglobin was detected by immunoprecipitation at very high levels in the blood for 4 days and only gradually returned towards normal over the next 16 days. Unfortunately, blood and urine specimens obtained during the acute management of our patient were not saved for further, more accurate, analysis by radioimmunoassay for myoglobin. Nevertheless, the immunoprecipitation technique involves agglutination of serially diluted specimens, and thus gives a range within which the actual myoglobin concentration lies, confirmed by testing alongside controls. Our results showed that the maximum myoglobin concentration was more than 400,000 µg litre⁻¹ (i.e. well above the maximum dilution reference range recommended by the manufacturer of Rapitex). There appear to be no reports of survival with serum myoglobin concentrations greater than 100,000 µg litre⁻¹, although one case had post mortem serum myoglobin concentrations of 118,000 µg litre⁻¹ and another survived with concentrations of 58,000 µg litre⁻¹.

Although it was apparent that extensive muscle destruction had taken place, it was hoped initially that the more severely affected limb might still be viable. Early amputation may, in retrospect, have reduced the severity of some of the
complications, as ongoing muscle destruction contributed to the persistently increased myoglobin concentrations in the first 4 days. Lack of renal function was probably the cause of the slow return towards normal serum myoglobin concentrations, as the kidneys seem intimately involved with myoglobin metabolism.\textsuperscript{13} \textsuperscript{14}

Renal management followed established practice, relying initially on haemofiltration because of severe cardiovascular stability, but changing to full haemodialysis from day 7. Failure of haemofiltration to control serum creatinine played a major part in the decision to change to haemodialysis, when cardiovascular stability had been established. Serum creatinine concentrations mirrored serum urea concentrations. The diuretic phase started on day 20 but was complicated by severe derangement of calcium metabolism.

In renal failure caused by rhabdomyolysis and tissue injury, there is often hypocalcaemia in the anuric phase followed by hypercalcaemia in the diuretic phase.\textsuperscript{15} Hypercalcaemia has been reported to occur between the third and 55th day of the diuretic phase. In our patient, it occurred on day 7 of the diuretic phase. Calcitonin is ineffective in severe cases. Calcium-free dialysate solutions are helpful but unfortunately were not available. It has been suggested that if hypercalcaemia occurs towards the end of dialysis, disodium pamidrate is the drug of choice\textsuperscript{6} and we used a single dose with success. Calcium therapy for hypocalcaemia in post-traumatic renal failure is only of temporary benefit and should not be used in view of the problem of hyperkalaemic cardiotoxicity, as in our patient.\textsuperscript{15} The result of abnormal calcium metabolism was deposition of calcium in soft tissues and along blood vessel walls.

Neurological complications ranging from monoparesis to tetraparesis, cases mimicking transverse myelitis\textsuperscript{9} and rhabdomyolysis–lumbosacral plexopathy\textsuperscript{8} \textsuperscript{10} have been reported in cases of heroin toxicity. The aetiology is not clear but mechanical, toxic and immunological factors have been implicated. Our patient was paraplegic below T12 and this contributed significantly to his long-term morbidity.

### References

12. Laurence AS, Vanner GK. Serum and urinary myoglobin following an aborted malignant hyperthermia reaction. \textit{Anaesthesia} 1996; 51: 958–61