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Lumbar plexitis and rhabdomyolysis following abuse of heroin

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Summary

A young man was admitted following abuse of impure ('Chinese') heroin. He showed a number of the less common complications of heroin abuse. These are now more likely to be seen in England in view of the increasing incidence of heroin addiction.

Case history

A white man of 22 was found unconscious 24 hr after an intravenous injection of impure heroin. He had abused a variety of drugs since 1965 including, from 1968 till 1970, heroin, but had held down a job for 5 years and avoided opiates for the last 2 years. On admission he was unconscious, responding only to painful stimuli, and pyrexial, hypotensive, and tachypnoeic. There were widespread rales in both lungs, and no focal neurological signs save an absent right ankle jerk. His pupils were normal.

Investigations showed a hyperkalaemic metabolic acidosis—plasma urea 65 mg/100 ml; potassium 7.5 mEq/l; arterial pH 7.24; Pco₂ 31 mmHg; Po₂ 80 mmHg. There were atypical lymphocytes and a neutrophil leucocytosis in the stained film. Serum enzymes were grossly raised—SGOT 1240 units; HBD 3000 units. Unfortunately, serum muscle

enzymes and myoglobin levels were not measured. He was oliguric and the first urine sample was dark brown, benzidine-positive, but contained no red blood cells. Chest X-ray showed widespread pulmonary oedema, and the electrocardiogram (ECG) showed right bundle branch block with ST elevation. His hyperkalaemia was treated with intravenous calcium, insulin and glucose, and a calcium resonium enema, and his acute renal failure with intravenous frusemide and mannitol.

He was alert after 12 hr and over the next 3 days his temperature, ECG and chest X-ray reverted to normal. He remained oliguric, his plasma urea rose to 250 mg/100 ml after 3 days, and peritoneal dialysis was instituted for the next 7 days. High dose excretion urography showed changes compatible with acute tubular necrosis (Fry and Cattell, 1972). Four weeks after admission his creatinine clearance was normal.

The day after admission he had complained of pain in the right leg. The anterior compartment was swollen and indurated, and there was weakness of tibialis anterior, extensor hallucis longus, and extensor digitorum longus on the right. Three weeks after admission he began complaining of pain in both feet. Examination then showed weakness of the dorsiflexors of the left foot, and of flexor digitorum and the peroneal muscles on the right, in

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addition to the weakness noted following admission. There was hypalgesia in the distribution of L₅ and hyperpathia in that of S₁ in both legs. The plantar responses were flexor and reflexes normal. The cerebrospinal fluid was normal except for a protein of 56 mg/100 ml. Myelography was normal. Electromyography at this time showed a definite myopathic pattern in the right tibialis anterior with low amplitude, short duration, polyphasic motor unit potentials. The left tibialis anterior was normal. There was no evidence of partial denervation. Mixed ascending nerve action potentials in the lateral popliteal nerve were not recorded.

He was discharged 8 weeks after admission. Two months later power of the left foot was normal and that of the right improving. The sensory signs were unchanged. He had reverted to the occasional subcutaneous injection of heroin despite support from an addiction unit.

Comment

A large variety of medical complications may occur in the course of heroin dependency (Sapira, 1968). Most reports are from America where the prevalence of addiction is steadily increasing (*Medical World News*, 1972). In addition to pulmonary oedema (Frاند, Chang and Williams, 1972), this patient showed two of the less well recognized complications.

The swelling of the anterior compartment of the right leg was probably due to necrosis of the anterior tibial muscles. This was confirmed by the myopathic pattern seen on electromyography. Tissue necrosis must, however, have been more widespread to account for the gross elevation of serum enzymes and the hyperkalaemic metabolic acidosis found before the development of severe uraemia. Rhabdomyolysis may also have contributed to the ECG changes. The first urine sample was dark brown, benzidine-positive, and contained no red cells. This is compatible with acute myoglobinuria following acute rhabdomyolysis and this may well have contributed to the development of acute renal failure. Richter and his colleagues (1971) have recently described four cases of rhabdomyolysis with myoglobinuria in heroin addicts to which this case is very similar.

Clinical examination initially showed weakness of dorsiflexors of the right foot. This could have been due to muscle necrosis as subsequent electromyography suggested. However, 3 weeks later, with the onset of sensory symptoms in both feet, weakness

developed in further muscles but electromyography of dorsiflexors on the left was normal. Distribution of the sensory loss over L₅, S₁ with sparing of areas supplied by the sciatic nerve, indicated that the disturbance must have been in the lumbar plexus rather than the sciatic nerves or their branches. Richter's group in a short communication have also described lumbar and brachial plexitis occurring after the injection of impure heroin (Challenor, Richter and Pearson, 1971).

A case of brachial plexitis in an addict has recently been seen at our hospital. Other neurological complications include transverse myelitis (Pearson *et al.*, 1972) and polyneuritis.

These neuromyopathies have often occurred after resuming heroin use following a drug-free interim, as happened in this patient. The aetiology is obscure, and hypersensitivity or toxic reactions to contaminating chemicals, organisms or to heroin itself have been suggested. Penn, Rowland and Fraser (1972) consider the consequences of acute heroin intoxication—hypoxia, hypotension, hypothermia and pressure on dependent limbs—are more important. However, rhabdomyolysis and the prodrome of transverse myelitis have occurred in conscious patients following heroin injection. It would seem likely that a number of different factors operate in each patient to varying degrees.

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