Rhabdomyolysis and heroin addiction

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In recent years, there has been an increased number of reports of rhabdomyolysis associated with heroin addiction and other drugs such as cocaine, amphetamines, salicylates, and alcohol. Early diagnosis and treatment can prevent serious complications such as acute renal failure. We describe a case of an intravenous heroin addict with rhabdomyolysis and acute renal failure which required acute haemodialysis. A high index of suspicion for rhabdomyolysis and myoglobinuria is essential in drug overdose patients when admitted to hospital. The prognosis for adequately treated cases of rhabdomyolysis is often excellent.

HKMJ 1995;1:266-268

Key words: Rhabdomyolysis; Heroin dependence; Creatine kinase; Diagnosis

Introduction

Rhabdomyolysis is a clinical syndrome which results from the release of damaged skeletal muscle fibre content (myoglobin) into the bloodstream. While the causes of the muscle damage vary, renal failure is a common and potentially fatal complication. A case of heroin-associated rhabdomyolysis complicated by acute renal failure is presented and a discussion of the diagnosis and treatment made. We stress the need for early recognition of the disease as the extent of serious complications will greatly depend on early diagnosis and adequate therapy.

Case report

A 44-year-old Chinese man known to be an intravenous heroin addict was admitted to the Medical Department, Tuen Mun Hospital, with right lower limb weakness and swelling. He gave a history of intravenous injection of heroin followed by an unknown period of unconsciousness one day before admission. On admission, he was afebrile and fully conscious. His blood pressure was 140/80 mmHg. Physical examination revealed marked tenderness, swelling, and weakness over the right lower limb. The power was grade 0/5 for hip flexion and extension, grade 2/5 for knee flexion and extension, grade 2/5 for ankle flexion and extension, and grade 3/5 for toe flexion and extension. There was decreased sensation over the L2 to S5 dermatomes of the right leg. Knee and ankle jerks were absent on the right side. Both dorsalis pedis pulses were present. The bladder was not palpable and the anal tone was intact.

An initial diagnosis of compartment syndrome was made. Laboratory investigations revealed the following values: serum potassium 5.2 mmol/L, urea 16 mmol/L, and creatinine 346 μmol/L. Blood gas analysis showed metabolic acidosis with pH 7.29, pCO_2 3.9 kPa and base excess of -10.5 mmol/L. The creatine kinase level was greater than 16 000 U/L. The urine showed a strongly positive reaction to red blood cell by the Hemastix method (Bayer Diagnostics/Bayer Australia Ltd, Victoria, Australia) with no red blood cells being demonstrable microscopically in the urine sediment.

The orthopaedic team was consulted; the compartment pressure for the anterior and posterior compartments were measured to be 38 and 42 mmHg respectively, using the Whitesides technique. Emergency exploration and decompression of the right gluteal compartment was performed on the patient. On exploration, the gluteal muscles were found to be oedematous and ischaemic, particularly the gluteus medius.

Muscle biopsy of the gluteus muscle showed marked oedema among the skeletal muscle fibres, with

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isolated but large areas of acute fibre degeneration (necrotic and atrophic fibres) with the presence of polymorphonuclear leukocytes. Histology confirmed acute rhabdomyolysis. Figure 1 shows normal muscle histology and Fig 2 shows muscle biopsy of our patient with acute rhabdomyolysis.

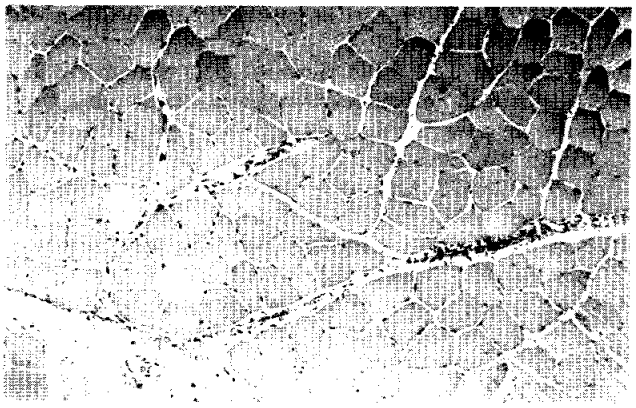

Fig 1. Histology showing normal skeletal muscle

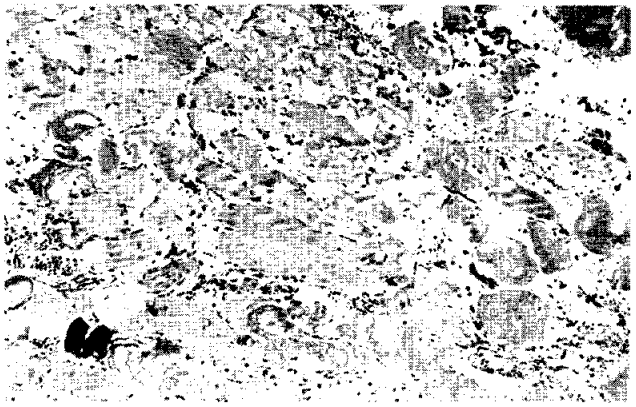

Fig 2. Histology of the gluteus muscle showing muscle fibre degeneration

The patient subsequently had two sessions of haemodialysis because of his deteriorating renal function and acute renal failure. The peak urea and creatinine levels were 41.4 mmol/L and 1141 μmol/L, respectively. The patient's recovery was uneventful. His leg swelling subsided and renal function gradually returned to normal. He was given regular physiotherapy and rehabilitation exercises. On subsequent follow up, both motor and sensory functions had returned to normal.

Discussion

Rhabdomyolysis is a well-known complication of heroin addiction and overdose. A review of the literature showed that it occurs in 7.7% of patients admitted for acute intoxication to a general hospital from an urban area, and in 11.7% of patients (22 of 188) admitted for acute opiate intoxication.[1,2] We may have underestimated the occurrence of this complication among drug overdose patients admitted to the hospital.

Many other drugs, such as cocaine, alcohol, amphetamines, strychnine, phencyclidine and arsenic—which are commonly mixed by the drug abuser deliberately or by the vendor—are also reported to be associated with rhabdomyolysis.[3,4]

Heroin intoxication, apart from causing respiratory arrest and coma, is associated with other complications, such as aspiration of gastric contents, myocarditis, rhabdomyolysis, pulmonary oedema, convulsion, spongioform leukoencephalopathy, transverse myelopathy and other neurological complications.

The exact aetiology for rhabdomyolysis is not known. Hypotheses as to the causes of the muscle damage include prolonged coma and immobilisation in one position, which results in either occlusion of the regional vascular supply or direct compression to the muscle; a direct or immunologically-mediated toxic effect from heroin or an adulterant to the muscle;[5,6] acidosis, hypoxia, or hypothermia.[7] The above factors could all have been involved in causing rhabdomyolysis in our patient. When injury to the muscle occurs, the normal function of the sarcolemma and ion transport systems are disrupted, resulting in a net influx of calcium ions into the cell. This high concentration of calcium ions in the muscle cell will activate intracellular protease leading to destruction of the myofibrils and further muscle damage.

The exact aetiology for renal failure in rhabdomyolysis is not known. Proposed mechanisms include ischaemia to renal tissue resulting from hypovolaemia and shock, a direct blocking effect of myoglobin-containing casts in the renal tubules, nephrotoxic effects of the myoglobin metabolites, and proteases or vasoactive kinins from injured muscle cells, resulting in renal damage.

Most patients with rhabdomyolysis have an excellent prognosis, provided that the diagnosis is made early and prompt treatment initiated. Clinicians should be alert to the possibility of rhabdomyolysis and myoglobinuria in patients who have drug overdose due to heroin or other associated drugs. Most of these patients will present with classical features of fever, muscle pain, stiffness, swelling, weakness, dark-

coloured urine, hyperkalaemia, hypocalcaemia, hyperphosphataemia, and renal failure, but some may have only vague symptoms of malaise. A fatal case of myoglobinuria complicating heroin addiction, with no overt symptoms of rhabdomyolysis and no period of unconsciousness, has been described in the literature.[8]

In an emergency situation, one should always consider the need for exploration, decompression, and fasciotomy. A compartment pressure of greater than 30 mmHg using the Whitesides technique suggests a likelihood of compartment syndrome. A raised serum creatine kinase level and the detection of urine haematic pigments without red blood cells in the urine sediment are useful for the early diagnosis and treatment of this complication.[8,9] The serum creatine kinase level rises in 2 to 12 hours from the onset of muscle injury, peaks in one to three days, and declines within three to five days. Muscle biopsy reveals muscle necrosis and degeneration. Electromyography may show myopathic changes in the acute phase.

Acute management involves correction of fluid and electrolyte disturbances, cardiac monitoring for dysrhythmias, fasciotomy for compartment syndrome, and dialysis for acute renal failure. Following acute management, physiotherapy and orthopaedic aids are important in achieving full functional recovery in these patients.

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