Short report

Rhabdomyolysis and concomitant neurological lesions after intravenous heroin abuse

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SUMMARY Seven cases of rhabdomyolysis in heroin addicts are presented. All patients showed concomitant neurological symptoms suggesting mononeuropathy, incomplete plexus lesions or myelopathy. In most cases rhabdomyolysis occurred without preceding trauma to the muscles (for example tissue compression or coma). Five patients had a history of recently resumed heroin abuse after prolonged abstinence. An allergic or toxic reaction to heroin or adulterants seems to be more likely than trauma in the pathogenesis of these complications. Severe rhabdomyolysis can occur without visible muscular swelling. Routine screening of creatine kinase is recommended in heroin addicts with neurological complications, as rhabdomyolysis may lead to fatal renal failure and may easily fail to be diagnosed.

Many medical¹ and neurological²⁻⁷ complications have been recognised in heroin addicts. Heroin abuse (or adulterants) may cause plexus neuropathy, myelopathy, polyradiculoneuropathy, polyneuritis and leuco-encephalopathy. We present seven cases with rhabdomyolysis following intravenous heroin abuse with initially severe concomitant neurological complications. A conspicuous feature was the development of rhabdomyolysis together with nerve and plexus lesions without evidence of preceding muscle or nerve compression.

Case reports

Case 1

A 29-year-old man presented with severe pain and weakness in both legs following heroin abuse. On examination there was paraplegia with hyperalgesia of the trunk and upper legs and hypalgesia of the lower legs. The knee jerks were brisk and the ankle jerks were absent. Abnormal laboratory values were: creatine kinase (CK) 17000 U/l, sodium 123 mmol/l, potassium 8.2 mmol/l, BUN 78.4 mmol/l, creatinine 1000 µmol/l. Subsequently anuria developed necessitating haemodialysis.

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Case 2

A 22-year-old man who had taken heroin for six months presented with pain, weakness and distal paraesthesia in the right leg. On examination there was swelling of the right gluteal area. There was weakness of the finger flexors of the left hand with sensory loss in the palm. In the right leg there was weakness of the quadriceps and iliopsoas muscles and paralysis of the foot flexors and extensors, with sensory loss in the right foot and of the medial part of the calf. Creatine kinase level was 31000 U/l, serum myoglobin 92 µg/l; urinary myoglobin was not detectable.

Case 3

A 20-year-old man was found comatose after he resumed heroin abuse following an abstinence of three months. After administration of naloxone he regained consciousness and complained of weakness and sensory loss in the right hand and foot. On examination he had pin-point pupils and a swollen warm right upper leg. There was a paralysis of the right hand and finger extensors and weakness of the right foot and toe extensors. Sensation was diminished on the anterior part of the right lower leg. The right ankle jerk was absent. CK level was 7000 U/l; serum and urinary myoglobin were not detectable.

Case 4

A 24-year-old woman resumed heroin abuse after being abstinent for four months. She noticed weakness in the left leg and diminished sensation in the left foot after a few hours of sleep. On admission there was swelling of the left thigh. The muscles of the entire leg, including the ilio-

psoas were paralysed with exception of the knee and foot extensors. Sensation was diminished in the dermatomes L5 and S1 distally and Lasègue's sign was positive. The left ankle jerk was diminished. CK level was 22000 U/l, serum myoglobin 1880 µg/l, urinary myoglobin 15200 µg/l.

Case 5

A 29-year-old man with a long history of heroin addiction took a heroin dose after a long busdrive from Spain. He had been abstinent for eight months. After a few hours he noticed pain and weakness in the right leg. Examination showed gross swelling and tenderness of the right upper leg. There was severe weakness of the flexors and extensors of the right foot and sensation was diminished in the dermatomes L5 and S1. Lasègue's sign was positive and the right ankle jerk was absent. Laboratory findings were CK >100000 U/l, sodium 127 mmol/l, potassium 7.9 mmol/l, BUN 22 mmol/l, creatinine 420 µmol/l, serum myoglobin 59500 µg/l; urinary myoglobin was not detectable. Shortly after admission he became anuric and was treated with haemodialysis.

Case 6

A 36-year-old man resumed abusing heroin after being abstinent for twelve months. The next day he complained of weakness of the left arm and leg. He had flaccid paralysis of the entire left arm and weakness of the left quadriceps muscle. Sensation was diminished distally in the arm and on the medial aspect of the left leg. The left arm reflexes and the left knee jerk were absent. CK level was 12760 U/l, serum myoglobin 3000 µg/l, urinary myoglobin 14 µg/l.

Case 7

A 23-year-old man was unable to stand on his right leg after a two hour nap. He complained of a painful right leg. Although he denied heroin abuse during the last four months opiates were found in his urine. On examination the entire right leg was paretic with exception of the quadriceps muscle. Sensation was diminished below the knee and the ankle jerk was absent. CK level was 12500 U/l,

serum myoglobin 275 µg/l; urinary myoglobin was not detectable.

All patients recovered completely within two months except No 2, who had a residual foot drop and No 3, whose wrist extensors remained paralysed.

Discussion

Rhabdomyolysis is a release of muscle cell contents into the plasma resulting from damage to the muscle cell membrane.⁸ The diagnosis of rhabdomyolysis is mainly based upon the finding of greatly elevated CK levels. In addition to CK elevation, in five of the present cases myoglobinaemia or myoglobinuria was found (Table). The absence of myoglobin in serum or urine however, as in case 3, does not exclude the possibility of rhabdomyolysis, as myoglobin is rapidly cleared from the plasma.⁸ In two cases (1 and 5) acute renal failure developed, necessitating treatment with haemodialysis. Only in these two cases was the serum potassium elevated. High potassium levels in cases of rhabdomyolysis occur especially in renal failure.⁸ In three cases (1, 6 and 7) no diagnostic signs of muscle disease, such as swelling or tenderness of muscles, were found, indicating that severe rhabdomyolysis may be present in patients without visible muscle abnormality.

Rhabdomyolysis in intoxicated patients is generally regarded as caused by muscle compression during coma.^{6,9,10} In six of our seven patients however, rhabdomyolysis was not preceded by coma or other evidence of muscle compression. Consequently we believe that severe muscle compression is not a decisive factor in the development of rhabdomyolysis. This confirms the observations of Richter *et al*¹¹ who also described acute rhabdomyolysis in heroin addicts without preceding coma. Their patients had

Table Summary of seven cases of rhabdomyolysis and concomitant neurological lesions after intravenous heroin abuse

Case	Age (yr) sex	Period of abstinence (months)	Presentation	Possible muscle trauma	Muscular swelling	Neurological diagnosis	Creatine kinase (U/l)	Myoglobin (µg/l)
1	29/M	—	weakness both legs	—	—	myelopathy	17000	not tested
2	22/M	—	weakness right leg	—	right gluteal area	neuropathy median nerve, plexus neuropathy lumbosacral pl.	31000	serum 92 urine —
3	20/M	3	coma	coma	right upper leg	neuropathy radial nerve, sciatic nerve	7000	serum — urine —
4	24/F	4	weakness left leg	sleep	left upper leg	plexus neuropathy lumbosacral pl.	22000	serum 1880 urine 15200
5	29/M	8	weakness right leg	sleep	right upper leg	neuropathy, sciatic nerve	>100000	serum 59500 urine —
6	36/M	12	weakness left arm left leg	—	—	plexus neuropathy brachial pl. neuropathy femoral nerve	12760	serum 3000 urine 14
7	23/M	4	weakness right leg	sleep	—	plexus neuropathy lumbosacral pl.	12500	serum 275 urine —

been abstinent for some time before resuming heroin abuse which precipitated rhabdomyolysis. Similarly five of the present cases had been abstinent from three to twelve months.

The neurological diagnoses in the present cases were myelopathy, plexus neuropathy and single or multiple mononeuropathy. In two cases plexus neuropathy was present together with mononeuropathy in another extremity (table). The localisation of the lesions was based on clinical evidence. We assumed a plexus lesion to be present if there were extensive sensory and motor symptoms in one extremity and if these symptoms did not have the characteristics of a medullary or radicular lesion or of a peripheral neuropathy. In fact partial plexus derangement is hardly distinguishable from multiple incomplete neuropathies in one extremity. Cases 2, 4 and 7 for instance were diagnosed as lumbosacral plexus neuropathy, but similar sensory and motor symptoms may be caused by simultaneous incomplete proximal lesions of the femoral and sciatic nerves. The term "plexitis" is often used in this context.^{2,4} It suggests an inflammatory process which has never been proved to our knowledge. Electromyographic studies were performed in five cases. The only clear neurogenic disturbances were found in cases 2 (leg muscles) and 3 (radial nerve neuropathy). These were the only cases with permanent neurological sequelae. Probably the recovery in most patients was too fast to allow the development of neurogenic changes in the EMG. The cerebrospinal fluid was examined in four cases. No abnormalities were found except in case 6 who had a slightly elevated γ -globulin with oligoclonal bands on electrophoresis.

Peripheral nerve lesions are not uncommon in drug addicts, owing to injection of substances in the vicinity of a nerve, to local infection, or to nerve compression during coma.⁶ Cases of plexus lesions (without rhabdomyolysis) after heroin abuse have been described.² Combined plexus lesions and rhabdomyolysis in drug addicts have been reported in relation to possible trauma.^{4,12} The present case histories show that combined rhabdomyolysis and nerve or plexus lesions may occur in drug addicts without apparent trauma to muscles or nerves. The only possible trauma in three cases was a period of normal sleep and in three others no possible cause of limb compression was present. Consequently, toxic or allergic reactions to heroin (or adulterants) are probably more important causes of rhabdomyolysis and peripheral nerve lesions than limb compression.

Interestingly, resumed heroin abuse after abstinence has been reported not only in cases of rhabdomyolysis but also in patients with nervous system lesions.^{2,4-6,11,13}

The possibility of rhabdomyolysis and consequent renal failure in drug addicts presenting with some medical or neurological complication should be considered, even in the absence of muscle swelling or a history of limb compression. Routine screening of serum CK is advocated in such cases.

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