CASE REPORT

Plexopathy in a Heroin Addict

Ahmed S., Hashem A., Bitar Z.I.

Medical Department and Intensive Care Unit (ICU)
Adan Hospital, Ministry of Health, Kuwait

Abstract:

Muscle and peripheral nerve disorders are important complications in intravenous heroin abusers. Rhabdomyolysis and acute lumbosacral plexopathy are important in the differential diagnosis of patients developing muscle weakness in an intensive care unit after intravenous overdoses of heroin. A 25-year-old man developed rhabdomyolysis with neuromuscular involvement, and consistent clinical and electrodiagnostic features of lumbar plexus neuropathy after an intravenous injection of heroin. The improvement occured slowly, over months, in spite of intensive physiotherapy.

Key words: Diacetylmorphine, Plexopathy, substance abuse, complications

Introduction:

Heroin is perhaps the best-known and most widely abused of the opiate narcotics. Neurological complications arising from heroin abuse are common and peripheral nerve disorders have also been noted. Acute lumbosacral polyradiculopathy is a devastating but potentially treatable neurological syndrome. Rhabdomyolysis is a recognized complication in patients admitted to an intensive care unit. The association of lumbosacral plexopathy and rhabdomyolysis is rare and the mechanism is unknown.

Case presentation:

A 25-year-old soldier was admitted to the ICU, Adan General Hospital, in a deep coma following an overdose of intravenous heroin. He was ventilated invasively because of severe hypoxia and hypotension secondary to an acute respiratory distress syndrome and dehydration. His urine opiates were markedly elevated and the urine contained hemosiderin. Other laboratory results are shown in ***Table 1***. Cerebrospinal fluid was normal and an HIV test was negative. Echocardiography was normal.

Table 1: Laboratory values

Variable	On admission	2 days later	7 days
Corrected Ca(mmol/l)	1.9	2.2	2.3
Total protein (gm/L)	75	65	77
Albumin (gm/liter)	25	22	28
Na (mml/l)	145	133	137
K (mmol/l)	3.2	5.2	4.5
BUN (mmol/l)	7.2	13	5.8
Creatinine (ummol/l)	174	223	88
Creatinine kinase (U/litre)	10,000	16,000	8,000
WBC	2.5	3.0	4.0
Aspartate aminotransferase (U/L)	320	340	240
Alanine aminotransferase (U/Litre	139	142	80

He was managed with intravenous fluid plus ceftriaxone 2 gm intravenously once daily and was weaned from the ventilator four days after admission. At that time there was generalized muscle wasting and tenderness, mainly in the lower limb. His upper limb power was 3/5 and lower limb 1/5 with generalized hyporeflexia. Urine porphobilinogen was normal.

Fourteen days later he was reassessed in the ward. He was unable to raise himself from a sitting to a standing position but he could walk with assistance. He complained of back pain radiating to the thigh. The power in the upper limb was 5/5 with normal reflexes. The lower limb showed a power of 1/5 in flexion, extension of the hip and flexion of the knee. The muscle power of the ankle joint was 5/5 with normal reflexes. Plantar reflexes were downward. There was no disturbance of the urinary sphincter. There was sensory loss to pain and superficial touch in the region of Lumbar 2, 3, 4 on both sides. Repeated CSF studies, CPK, renal profiles were all normal. Nerve-con-

Address for correspondence:
***Zouheir I. Bitar**, MRCP (UK)*
Medical Department, Adan Hospital, Ministry of Health
P.O. Box 46468, Fahahil 64015, Kuwait
*Tel.: (+965) 6043300; E-mail: **zbitar2@hotmail.com***

duction studies and an electro-myographic examination were performed on the 14th day ***(Tables 2 and 3)***. Ultrasound examination of the abdomen and MRI of the dorso-lumbo-sacral region were both normal.

Table 2: Results of nerve-conduction studies and electro-myographic examination in the 21st hospital day.

Nerve & Site of Stimulation	Latency m/sec	Amplitude mV	Duration m/sec	Velocity m/sec
Motor nerve Right peroneal	3.6	1.7/ 1.4	12.7	42.0
Left tibial	3.9	21.7/18.0	11.6	42.0
Right median	3.0	10.4/10.2	10.4	57.0
Right ulnar	2.5	12.0/11.3	11.4	51.0

Sensory Nerves (Segment)	Amp. Nerve action pot. uV	Distance cm	Velocity m/sec
Right sural (midcalf)	10.0	14.0	36.0
Left sural (midcalf)	14.0	14.0	36.0
Right median (Dig II)	60.0	14.5	59.0
Right ulnar (Dig V)	30.0	11.7	50.0
Left ulnar (Dig V)	49.0	11.4	54.0

Table 3: Coaxial Needle Electromyography

Muscle	Spontaneous Potentials			Motor Unit Potentials			Volume recruited
	Fibrillation	Positive sharp wave	Faciculation	Polyphasia	Duration	Amplitude (mV)	
Rt. Rectus femoris	++	++	0	+	Normal	1.5	Markedly decreased
Lt. Rectus femoris	++	++	0	No MUPs can be recruited			
Rt. Tibialis anterior	+	+	0	++	Mildly increased	3.0-4.0	Moderately decreased
Rt. Gastrocnemius	+	+	0	+	Normal decreased	3.0-3.5	Mildly
Lt. Tibialis anterior	0	+	0	++	Mildly increased	5.0-5.5	Moderately decreased
Lt. Gastrocnemius	0	+	0	+	Mildly increased	4.0	Moderately decreased
Lt. 1st D1	0	0	0	++	Mildly increased	4.0-5.0	Mildly decreased
Lt. Biceps	0	0	0	0	Normal	3.0	Nearly full
Lt. Adductor magnus	++	++	0	++	Mildly increased	2.0	Moderately decreased
Lt. short head of biceps femoris	0	0	0	++	Increased	5.0-6.0	Moderately decreased

Ten months later there had been a slow improvement of power in the hip and knee regions but the patient still could not rise unaided from a sitting position. He was still on physiotherapy and a program for the management of heroin addiction.

Discussion:

Heroin, the diacetyl derivative of morphine, is three to five times more potent than morphine, has a shorter duration of action, and is the most popular street opiate[1]. It can be smoked, sniffed up the nose, or injected subcutaneously but the usual route is intravenous. After absorption it is rapidly converted into morphine or monoacetylmorphine which, being highly lipid soluble easily penetrates the blood brain barrier to cause morphine euphoria. Heroin also relieves pain, suppresses cough, depresses respiration, and clouds the sensorium.

Neurological complications arising from heroin abuse can be subdivided into those that stem from infection and those that are of non-infectious origin[2]. Those of non-infectious origin arise mostly from hypoxia and hypotension produced by the heroin overdose and they include post-anoxic encephalopathy, cerebral infarction, unilateral Parkinsonism, and acute transverse myelitis. Neurological complications related to infection are meningitis, cerebral abscess and embolic infarcts secondary to infective endocarditis.

Peripheral nerve disorders have been noted in heroin abusers. They were attributed to prolonged compression of the nerves during episodes of stupor or to direct trauma from injection[3]. Non-traumatic brachial and lumbosacral plexopathies have been reported in heroin addicts[4] but the cause of these plexopathies is not clear. Intense pain is a common clinical presentation, while weakness and sensory impairment are less prominent. Spontaneous recovery occurs slowly over weeks or months[5].

Cases of rhabdomyolysis and lumbosacral plexopathy in HIV patients with intravenous heroin addiction have been reported[6] and attributed either to a direct toxic effect of the substance or to the abuser's diseases such as HIV infection. In this reported case there was no HIV infection and the plexopathy was most likely due to the effect of the heroin alone. The absence of sensory level and upper-motor neuron signs and an MRI of the spinal cord helped to exclude a myelopathy. Sparing of the upper extremities and no involvement of the sphincters distinguished this disorder from other neuromuscular disorders seen in critically ill patients such myopathy, AID-wasting syndrome, Guillain Barre' syndrome, and other inflammatory neuropathies.

Toxic insult, potentially affecting both nerves and muscles, is an important consideration in this case. The patient was ex-

posed to many potentially myotoxic compounds, including drugs and substances of abuse, but the screening test for toxic compounds ruled out most of them. 3, 4- methylenedioxymethamphetamine (MDMA), like other illicit drugs, is a potential cause of rhabdomyolysis and myoglobinuria. A screening test for toxic drugs ruled out toxic myopathy due to MDMA as the cause of this patient's weakness. Although drug abusers mostly tend to lie about the nature and number of drugs that they abuse, we have a good reason to believe that this patient was giving reliable information and had not been taking other medications which might have enhanced the sensitivity to heroin.

The EMG study reveals pure neurogenic features. The changes are in the distribution of first lumbar vertebrae down to first sacral. The brunt of the lesion is bilateral in the distribution of L2, L3. A bilateral femoral nerve lesion is quite possible but the involvement of muscles outside its distribution makes it unlikely to be the sole reason for all the changes.

Neuropathies often cause weakness in critically ill persons. The Guillain-Barre'syndrome , the most common cause of acute non-traumatic paralysis, typically occurs one to three weeks after infectious illness[7]. In this case it is a plausible diagnosis because of the rapid evolution of weakness, the greater involvement of motor than of sensory nerves but the normal CSF study (which was done twice) and the electromyographic findings, which did not strongly implicate demyelination, makes this diagnosis highly improbable. Critical-illness polyneuropathy, a sensorimotor axonopathy, may be the most common cause of weakness in patients in the intensive care unit[8,9]. Because this syndrome probably alters perfusion of the brain and other organs, critical-illness neuropathy is usually associated multi-organ failure. In our patient, this disorder is ruled out as the primary diagnosis by the presence of normal sensory-nerve action potentials and the absence of spontaneous electromyographic activity.

Attacks of acute intermittent porphyria can be precipitated by heavy alcohol consumption and by numerous medications, many of which, such as diazepam and barbiturates, are commonly administered in the intensive care unit but the absence of spontaneous electromyographic activity and normal urinary porphobilinogen exclude intermittent porphyria. Other neuropathic causes of rapidly progressive generalized weakness, such as hypophosphatemic neuropathy, severe renal failure, motor neuron disease, lead intoxication, paraproteinemic neuropathy, human immunodeficiency virus infection, are incompatible with the clinical features of this case. Elevated serum levels of creatine kinase and the myoglobinuria suggest a necrotizing myopathy. Toxic myopathy is a well-known cause of generalized muscle weakness in a critically ill patient but cannot explain the whole picture.

Heroin, like other illicit drugs, is a potential cause of rhabdomyolysis, myoglobinuria and lumbar plexus neuropathy and toxic insult, potentially affecting both nerves and muscles, and was an important consideration in this case in which the neuropathy is slowly recovering. This association of rhabdomyolysis-lumbosacral plexopathy has been reported occasionally[6] and should be considered in any patient admitted to an ICU. In spite of this being only one case it seemed important to alert other physicians to this possibility.

References:

1. *Ashley R (ed): Heroin. St Martins Press, New York, 1972 (1)+54.*
2. *Pearson J, Richter RW: Addiction to opiates: Neurologic aspects P 365. In: Vinkin PJ, Brugn GW(eds): Handbook of Clinical Neurology, Vol 37: Intoxications of the nervous system, Part 111. North Holland, Amesterdam, 1979.*
3. *Finelli PF, Taylor GW: unusual injection neuropathy in heroin addict: Case report. Milit Med 142: 704, 1977.*
4. *Hall JH, Karp RH: Acute progressive ventral pontine disease in heroin abuse. Neurology 23: 6, 1973.*
5. *Walter GB, Robert BD, Gerald MF: Neurology in clinical practice, the Neurological disorders. In: David MC (editor). Disorder of peripheral nerves. 2nd ed. 1996. p 1940-1952.*
6. *Diaz GJ, Pastor VC, Gil GR, et al. Rhabdomyolysis and lumbosacral plexopathy in intravenous drug addict: Report of a case. An Med interna. 1996 Feb; 13(2): 84-6.*
7. *Ropper AH, Wijdicks EFM, TruaxBT. Guillain Barre' syndrome. Philadelhia: F.A. Davis, 1991.*
8. *Bolton CF, Gilbert JJ, Hahn AF, Sibbald WJ. Polyneuropathy in critically ill patients. J Neurol Neurosurg Psychiatry 1984; 47: 1223-31.*
9. *Zochodone DW, Bolton CF, Wells GA, et al. Critical illness polyneuropathy: A complication of sepsis and multiple organ failure. Brain 1987; 110: 819- 41.*