

## Heroin brachial plexopathy

Kongkiat Kulkantrakorn MD

Neurology Division, Department of Internal Medicine, Faculty of Medicine, Thammasat University Rangsit Campus, Pathumthani, and Bangkok Hospital Medical Center, Bangkok, Thailand

### Abstract

Neuromuscular complications from heroin abuse are rare. Most cases are caused by focal nerve compression and recover spontaneously. We report a 22-year-old Caucasian man who presented with shoulder pain and left arm weakness after recovering from coma due to heroin and phenobarbitone overdose. Electrophysiological findings were consistent with complete brachial plexopathy. Investigations did not reveal any other causes. Follow up examination at 4 weeks showed minimal improvement of his weakness. This case highlights an unusual complication of heroin abuse. Its pathophysiology remains unknown. Although heroin abuse is now less common, neurologists should be aware of this condition.

### INTRODUCTION

Heroin is a common drug of abuse, although its use is now less common. Neuromuscular complications, although rarely reported have included transient polyneuropathy, sensory axonal polyneuropathy, rhabdomyolysis, mononeuropathy, and plexopathy.<sup>1-4</sup> Some of these complications were seen concurrently with HIV infection. This report is a rare and interesting case of brachial plexopathy associated with heroin abuse.

### CASE REPORT

A 22 year-old right handed Caucasian man was admitted in a coma due to barbiturate and heroin overdose. He was last seen 2 hours prior to being found lying unconscious in bed. There were no signs of any external injuries. He regained consciousness after administration of flumazenil and naloxone and with supportive treatment. However, he was found to have severe weakness of his left arm. He had admitted to giving himself intravenous injections of phenobarbitone and heroin prior to becoming unconscious. As a regular drug abuser, he usually injected himself over both arms and had not previously had any local reaction after injections. He reported using intravenous heroin 2 to 3 times a week for 7 months and occasionally smoked marijuana. In the past few years, he had used barbiturates, “magic mushrooms”, inhaled ketamine, lysergic acid diethylamide (LSD), amphetamines and 3,4-methylenedioxymetamphetamine (MDMA or

“Ecstasy”). They were last used 6 to 12 months prior to this illness. He was otherwise well and was able to work as a teacher. He did not complain of fever, headache or any other neurological symptoms, nor was there a history of trauma or concurrent illness prior to this admission. He did not have any history of allergy.

General examination and vital signs were normal, except for multiple old puncture wounds over both arms. His mental status and cranial nerve examination were normal. His left arm was flaccid with almost no movement below left shoulder, and with only a flicker of movement over the fingers. There was mild loss to pain and light touch. Reflexes were absent over the left upper limb.

Investigations including complete blood count, blood sugar, liver function test, blood urea nitrogen, creatinine, electrolytes, creatinine kinase, thyroid function test, anti-HIV antibody, serum lead level and chest X-ray were all normal. Screening of his urine for cannabinoids, amphetamines and cocaine were negative while opiates were found in low concentrations. Magnetic resonance imaging (MRI) of the brain showed mild cortical and cerebellar atrophy while MRI of cervical spine and brachial plexus with gadolinium enhancement were unremarkable. He refused lumbar puncture for CSF examination.

Electrodiagnostic study was performed at 10 days after onset (Table 1). Motor nerve conduction studies showed normal distal latencies of both axillary and median nerves and the left ulnar nerve. However, the compound muscle action potential

Address correspondence to: Associate Professor Kongkiat Kulkantrakorn, Neurology Division, Department of Internal Medicine, Faculty of Medicine, Thammasat University Rangsit Campus, Pathumthani 12120, Thailand. Tel. +66-8-811-1476, +66-2-929-9794, Fax. +66-2-926-9793, +66-2-688-0237, Email: kongkiat1@gmail.com

**Table 1: Nerve conduction studies of patient**

Nerve	Stimulation site	Latency (ms)		Amplitude*		CV (m/s)		F-waves (ms)	
		R	L	R	L	R	L	R	L
Median motor (APB)Elbow	Wrist	3.8	4.0	5.3	0.3	50.0	49.1	27.3	NR
	8.8	9.5	4.8	0.1					
Ulnar motor (ADM)	Wrist	ND	2.7	ND	1.4	ND	49.0	ND	NR
	Below elbow	ND	7.8	ND	1.0	ND	55.6		
	Above elbow	ND	9.6	ND	1.0	ND			
Axillary motor (deltoid)	Erb's point	3.7	6.5	4.8	0.5				
Median sensory (second digit)	wrist	ND	3.4	ND	17	ND	63.6		
Ulnar sensory (fifth digit)	wrist	3.3	3.4	31	4.2	52.2	56.0		
Lateral antebrachial	forearm	1.58	NR	14	NR	70.8	NR		
Medial antebrachial	forearm	2.4	NR	12	NR	62.5	NR		

CV: conduction velocity; APB: abductor pollicis brevis; ADM: abductor digiti minimi; NR: no response; ND: not done.

\*Amplitude is measured in mV for motor studies and  $\mu$ V for sensory studies

(CMAP) amplitudes were much smaller on left side. Left ulnar and median F-waves were absent. Sensory nerve conduction studies showed reduced sensory nerve action potential (SNAP) amplitudes of left median and ulnar nerves. Left medial and lateral antebrachial cutaneous nerves were inexcitable. Needle electromyography showed prominent fibrillation and positive sharp waves at deltoid, biceps, triceps, extensor digitorum communis and first dorsal interosseous muscles. Only a few fast-firing motor units were recruited in each muscle. Interference pattern was markedly reduced. The results were consistent with an axonal lesion of the whole brachial plexus.

Despite supportive treatment and rehabilitation for 2 weeks, there was little improvement in his arm strength and hand function. However, his sensory function had improved. He was not given corticosteroid treatment because it was felt that there was little evidence of inflammation. Patient was subsequently referred to his home country for further follow up.

## DISCUSSION

In a previous report of 69 patients who experienced non-fatal heroin overdose, 49% of patients had peripheral neuropathy from lying on limbs for

prolonged periods while comatose. Twenty-six percent had temporary limb paralysis which lasted 55 minutes in average. Severe complications such as rhabdomyolysis and permanent nerve palsy were rare.<sup>5</sup>

Heroin-related plexopathy can either involve the brachial or lumbosacral plexus. In most cases, there is concurrent rhabdomyolysis. In these cases, it was postulated that there was a combination of compression and ischemia together with toxic damage to the muscles, resulting in focal myopathy and secondary compression of peripheral nerves. However, in cases where there is no compression, the pathophysiology is uncertain. Autoimmune, inflammatory or toxic mechanisms have been postulated.<sup>2</sup> Despite several routes for heroin abuse, almost all reported cases of neuromuscular complications have occurred with intravenous injections. This argues for a systemic mechanism as a cause of neuropathy, rather than a local cause. However, there is no temporal relationship between heroin injection and the onset of neuropathy. Most cases occur in the context of chronic injections and in one case, it occurred with heroin sniffing.<sup>2</sup> There was also no relationship between side of injection and side of plexopathy.<sup>2,3</sup>

An important differential diagnosis is lead poisoning which can cause brachial plexopathy.<sup>6</sup> Heavy metals and toxic chemicals are often impurities in heroin. In addition, risky administration techniques, such as injection of foreign material with diluents under non-sterile condition, and the use of a mixture of various drugs may also contribute to this nerve pathology.<sup>4</sup> Another possibility is the effect of phenobarbitone but this was unlikely. Despite several decades of use, there has been only one report of brachial plexus injury following barbiturate overdose, and this was thought to be due to local compression.<sup>7</sup> In our case, brachial plexopathy, was unlikely to be due to local compression during coma because of the relatively short duration of coma till medical attention. There was also no local evidence of compression viz. injury or inflammation. Compressive lesions at brachial plexus or spinal cord were also ruled out by normal MRI of brachial plexus and cervical spine. Due to the limited information about this condition, current treatment is limited to supportive treatment and rehabilitation. There is no evidence for the effectiveness of steroid treatment in this condition. Prognosis of these patients is generally poor, most likely due to severe axonal loss.

In summary, in heroin exposed patients with an acute neuromuscular weakness, the physician should consider the following:- rule out rhabdomyolysis and myoglobinuria, screen for serum lead levels and other relevant tests that may point to a specific cause. HIV antibody testing is also important. Electrodiagnostic tests will assist in localizing the lesion and provide more definitive diagnosis. Heroin toxicity should be added in the list of differential diagnoses of brachial plexopathy.

## DISCLOSURE

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