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Letter to the Editor

# Acute multifocal neuropathy following cocaine inhalation

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ABSTRACT

*Introduction:* We report a young man, not a habitual cocaine user, who developed an acute multifocal neuropathy following a second exposure to inhaled cocaine.
*Methods:* Case report.
*Results:* Clinical and electrophysiological findings suggested an acute multiple mononeuropathy following cocaine exposure. Imaging of the shoulder and pelvic girdles revealed multifocal denervation in selected proximal muscles. The patient was empirically treated with intravenous steroids to good effect.
*Discussion:* Cocaine use, although usually affecting the central nervous system, does produce peripheral nerve disease in rare instances. This unusual pattern of neurological involvement needs to be differentiated from the more common symptoms resulting from affection of the brain.


## 1. Introduction

Cocaine use has been associated with a variety of neurological complications, including central nervous system (CNS) vasculitis or vasoconstriction, intracerebral haemorrhage, ischaemic strokes, symptomatic seizures, transient movement disorders, acute severe headaches and myonecrosis or rhabdomyolysis [1–3]. Acute peripheral neuropathies, due to vasculitis or compression, are a rare complication of cocaine use and need to be differentiated from the more common CNS disorders [2–6]. We present a patient with acute multifocal neuropathy following non-habitual cocaine use, who showed significant improvement with intravenous steroid therapy.

## 2. Case report

A 17-year old male presented with acute onset of weakness of predominantly the right upper and lower limbs, of one week's duration. After inhaling (“snorting”) cocaine at a party, he fell asleep in his bed and awoke with weakness and severe pain in the affected limbs. He was not a habitual user, although he admitted to having inhaled cocaine once four months prior. He denied using any other substance. No progression of his deficit was noted. On examination, he had weakness of the right infraspinatus and right hand with more severe involvement of ulnar-innervated muscles. A right ulnar claw was noted, as was a right flail foot with severe weakness of all muscle groups in the leg and foot. Mild weakness (grade 4 to 4+/5 Medical Research Council grading) was present in the right iliopsoas, quadriceps and adductores whilst the right gluteus maximus and hamstrings showed more severe weakness (grade 2/5). Selective sensory loss was noted in the right fourth and fifth digits and over the medial palm and dorsum of the right hand; left hand over the anatomical snuffbox; the posterior right thigh; the lateral aspect of the right leg; and the dorsum and sole of the right foot. The right ankle reflex was absent. No muscle atrophy was noted. A clinical diagnosis of multifocal neuropathy following inhalation of cocaine was made, with a differential diagnosis of pressure palsies also being entertained.

Nerve conduction studies performed 12 days after onset showed absent motor potentials and F-waves in the right common peroneal and posterior tibial nerves, with low motor amplitudes and absent F-waves in the right ulnar nerve. The right ulnar, right superficial peroneal, right sural and left radial sensory potentials were absent (Table 1). These studies were interpreted as suggesting an axonal sensorimotor asymmetric neuropathy consistent with mononeuritis multiplex. Cerebrospinal fluid studies were normal, as was an MRI of the brain and spine. The MRI however revealed abnormal signal changes in selective proximal muscles (Fig. 1) suggestive of acute denervation. A vasculitic workup, including antinuclear antibodies, antineutrophil cytoplasmic

*Abbreviations:* ANCA, anti-neutrophil cytoplasmic antibody; CNS, central nervous system; MRI, magnetic resonance imaging.

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**Table 1**
Results of sensory and motor nerve conduction studies.

| Nerve | | Median | Ulnar | Radial | Common Peroneal | Posterior tibial | Sural |
|---|---|---|---|---|---|---|---|
| Distal motor latency (ms) | R | 2.92 | 2.29 | 2.8 | – | – | |
| | L | 2.81 | 3.02 | 2.4 | 3.23 | 5.94 | |
| | Normal† | <4.58 | <4.46 | <3.95 | <6.04 | <6.39 | |
| Distal motor amplitudes (mV) | R | 12.7 | 3.5 | 5.6 | 0 | 0 | |
| | L | 16.4 | 11.7 | 5.1 | 7.8 | 35.5 | |
| | Normal | >3.83 | >4.77 | >3.14 | >0.52 | >3.98 | |
| Proximal motor amplitudes (mV) | R | 10.9 | 9.4 | 4.1 | 0 | 0 | |
| | L | 13.1 | 11.5 | 3.9 | 4.0 | 25.7 | |
| | Normal | >2.17 | >3.9 | >2.95 | >0.81 | >2.99 | |
| Motor NCV (m/s) | R | 60.6 | 52.0 | 59.4 | – | – | |
| | L | 58.1 | 61.6 | 61.8 | 51.1 | 51.62 | |
| | Normal | >47.34 | >45.83 | >48.1 | >38.52 | >36.44 | |
| Sensory latency (ms) | R | 2.1 | – | 1.75 | | | – |
| | L | 1.83 | 2.0 | – | | | 2.79 |
| | Normal | <2.58 | <2.49 | <2.2 | | | <2.94 |
| Sensory amplitudes (μV) | R | 126.9 | 0 | 61.9 | | | 0 |
| | L | 118.4 | 100.9 | 0 | | | 27.2 |
| | Normal | >14.7 | >13.07 | >19.2 | | | >11.9 |
| Sensory NCV (m/s) | R | 60.1 | – | 51.4 | | | – |
| | L | 60.1 | 50.0 | – | | | 43.1 |
| | Normal | >52.47 | >50.46 | >49.4 | | | >41.05 |

† Normative data for our laboratory.

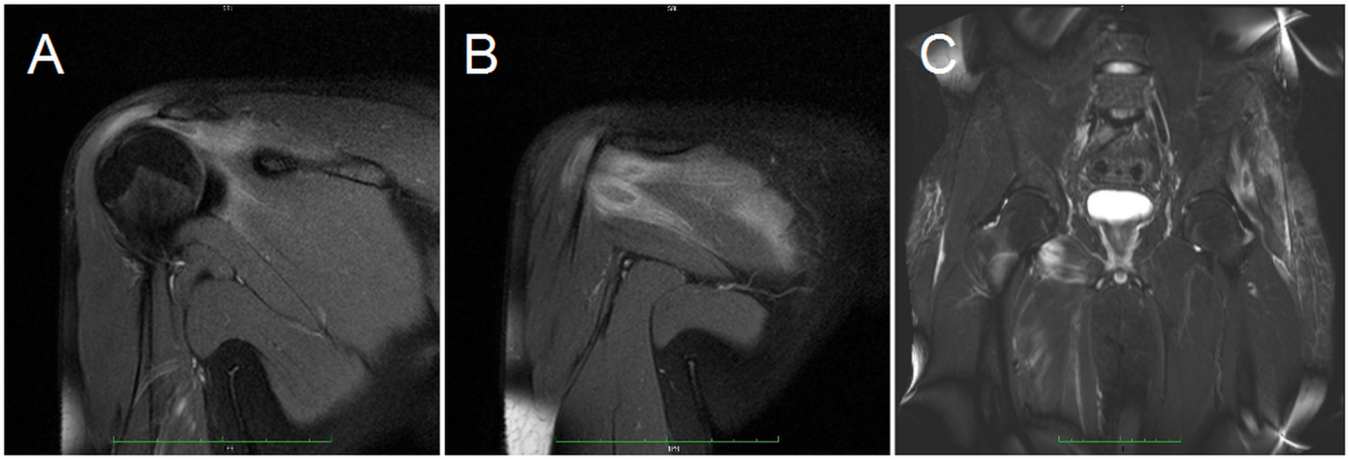


**Fig. 1.** (A and B) Proton density fat saturated image coronal image through the right shoulder joint reveals abnormal signal within the deltoid, supraspinatus and the infraspinatus muscles. (C) Coronal STIR image reveals abnormal high signal in the right adductor brevis and magnus muscles and the left gluteus medius and minimus muscles.

antibodies (ANCA), and rheumatoid factor was negative. Human immunodeficiency virus, hepatitis B surface antigen and hepatitis C serology were negative. Serum creatine kinase levels were 156 IU/L (normal, <195). A nerve biopsy was refused by the patient.

The patient was treated with intravenous methylprednisolone in a dose of one gram per day for five days, followed by oral prednisolone in tapering doses over one month along with intensive physiotherapy. Fifteen days after the start of treatment, substantial improvement was noted in the right upper limb and in proximal muscles of the right lower limb, as well as in neuropathic pain and the cutaneous anaesthesia in both upper limbs. The foot remained very weak, and an ankle–foot orthosis was prescribed.

## 3. Discussion

This 17-year old man presented with weakness of the right upper and lower limbs after a second exposure to inhaled cocaine. The pattern of involvement and electrophysiological studies both confirmed that sensory and motor neurological deficits were in discrete multiple nerve distributions consistent with a mononeuritis multiplex. Clinically, involvement of the sciatic, posterior femoral cutaneous and inferior gluteal nerves suggested a right sacral plexopathy. In addition, MRI of the shoulder and pelvic girdles showed signal abnormalities in discrete proximal muscles consistent with multifocal nerve involvement and consequent acute denervation. The lesion in this patient may therefore best be described as a "plexo-neuropathy". The proximal involvement, away from the usual sites of compression, rendered the possibility of pressure palsies unlikely.

Vasculitis affecting the peripheral nervous system has rarely been reported after cocaine use. Nerve trauma due to compression or during intravenous injection is considered more frequent [3]. Ischaemia due to vasoconstriction has also been proposed: cocaine blocks the presynaptic reuptake of norepinephrine and dopamine, producing vasoconstriction. However, this is transient, and there is reperfusion when the effect of the drug fades away [3]. Patients with mononeuritis multiplex associated with habitual cocaine inhalation or freebase smoking were found to have ANCA-positive medium-vessel vasculitis with antiphospholipid antibody