

*Short report*

# Polyradiculoneuropathy associated with heroin abuse

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**SUMMARY** Neurological complications of heroin addiction have occurred only sporadically in the United Kingdom. We report the case of a young female patient who developed an acute polyradiculoneuropathy when she recommenced intravenous heroin after four years abstinence. Another possible aetiological factor was a preceding flu-like illness but, after full investigations, we concluded that heroin abuse was the most likely cause of the neurological symptoms.

The medical complications of drug addiction have been reviewed by several authors in North America (Cherubin, 1967; Louria *et al.*, 1967; Louria, 1974). A variety of neurological complications including peripheral nerve lesions have been reported in heroin addicts (Richter and Pearson, 1975). There are only two reports in the British literature of neurological complications of narcotic abuse (Weller and Perry, 1973; Greenwood, 1974) which relate to peripheral neuropathy and to lumbar plexitis with rhabdomyolysis respectively. The Guillain-Barré syndrome has been observed in some of the heroin addicts reported (Richter *et al.*, 1973; Smith and Wilson, 1975). All these patients developed the syndrome during an uninterrupted period of heroin abuse ranging from months to years, and one of them had been on methadone maintenance (Richter and Pearson, 1975).

## Case report

This 22 year old woman (JB) came under our care in January 1977. At the age of 17 years, while living in London, she became addicted to heroin which she took intravenously for six months. She then abstained for four years but continued to smoke marijuana. In September 1976 she recommenced almost daily use of intravenous heroin. On one occasion she used morphine, on five occasions methadone, and on two occasions she sniffed methedrine. In mid-November 1976 she had a flu-like illness from which she recovered fully. On 16 and 20 December 1976, and on 1 January 1977, she took intravenous methadone prepared by crushing the tablet in tap water. Approximately 12 hours after the last injection, she awoke

with cramp-like pains in her legs. Over the next nine days she developed severe lumbosacral pain radiating to the thighs and calves, and severe burning and tingling sensations in her feet and calves and, to a lesser extent, in her mouth and hands. She also experienced difficulty in walking and passing urine. She felt numb from the waist down, had tenderness in her calves, and felt nauseated and dizzy on standing up.

On 10 January 1977 JB was admitted to another hospital where she was found to have a peripheral neuropathy in the legs and urinary retention requiring catheterisation for a short period. Examination of the CSF showed normal pressure, a raised protein content at 2 g/l and 140.10<sup>6</sup>/l lymphocytes with normal glucose and no organisms. Full haematological, biochemical, and viral studies were normal. The ESR was raised at 30 mm in the first hour (Westergren). She was transferred to the North Staffordshire Royal Infirmary on 28 January. The pain in her feet and legs was worse but she had normal bladder function and the paraesthesiae in the hands and mouth had subsided. General physical examination was normal but the skin of her legs was dry and scaly. Neurological examination showed normal cranial nerves and upper limbs. Abdominal reflexes were absent. There was a symmetrical peripheral sensory neuropathy involving all modalities of sensation in the legs. There were contact dysaesthesiae in the feet and calves, and delayed appreciation of pain and temperature sensation in the feet. There was a mild symmetrical distal motor weakness with depression of knee jerks and absence of the ankle jerks and of the plantar responses. Haematological, virological, serological, biochemical, immunological, and plain radiological investigations were normal. The serum creatine kinase was 85 iu/l (upper limit of normal). The repeat CSF examination

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showed a raised protein level at 1.2 g/l (IgG 0.14 g/l) with  $52.10^6$ /l nucleated cells, mostly lymphocytes. There was a normal glucose content and negative tests for syphilis. Nerve conduction studies showed prolonged distal motor latencies in both median nerves and the right lateral popliteal nerve (6.6 ms, 5.5 ms, and 12.1 ms respectively) with reduced conduction velocity of the right median and lateral popliteal nerves (41 m/s and 33 m/s respectively). The right extensor digitorum brevis muscle showed evidence of denervation. Sensory nerve action potentials were present but reduced in amplitude in both arms (3–10  $\mu$ V).

She was treated with sedatives, analgesics, an antibiotic for a urinary tract infection, and ACTH injections. Improvement was slow, and she was allowed home five weeks after her original admission. On follow-up six months after her initial presentation she was much better but still complained of painful paraesthesiae in the feet; she had persistent loss of cutaneous sensation and vibration sense in her feet, and the right ankle jerk was still absent.

## Discussion

The patient presented with an acute sensorimotor polyradiculoneuropathy with autonomic involvement. Two possible identifiable aetiological factors were the preceding flu-like illness (six weeks earlier) and the use of intravenous narcotics (heroin and methadone). Although a viral cause could not be totally discounted, no rise in viral antibody titres in blood was found, the viral hepatitis antigen was negative, and no viruses were isolated from the CSF. Other causes of peripheral neuropathy were excluded by appropriate investigations.

We concluded that heroin abuse was the most likely cause of this patient's neurological syndrome. Richter and co-workers have described a variety of peripheral nerve lesions in heroin addicts including plexitis, mononeuropathy, acute and subacute polyneuropathy (Challenor *et al.*, 1973; Richter *et al.*, 1973; Richter and Pearson, 1975). One of the patients described by Richter and Pearson (1975) developed a Guillain-Barré polyneuropathy while on methadone maintenance, and it should be noted that our patient had taken methadone on the three occasions before the onset of her symptoms. Also, her polyradiculoneuropathy developed on recommencing the use of heroin after a period of abstinence, in contrast to the patients described by Richter and co-workers, and the two patients described by Smith and Wilson (1975) who had been using heroin continuously. Sural nerve biopsy in a patient with acute

polyneuropathy referred to by Richter and Pearson (1975) showed segmental demyelination and axonal degeneration. A patient described briefly by Weller and Perry (1973) developed a myocardial infarct and a sensorimotor neuropathy in the legs after ingestion and inhalation of uncontaminated heroin and cocaine. The sural nerve biopsy showed total acute axonal degeneration. No nerve biopsy was done in our patient but the nerve conduction studies and muscle EMG sampling were compatible with a mixed demyelinating and axonal neuropathy.

There is no satisfactory explanation of the mode of action of heroin in causing these and other neurological syndromes. Possible mechanisms are a direct toxic action, a hypersensitivity reaction to the heroin adulterant mixture, or the introduction of infection. The quinine adulterant has been reported as being the cause of amblyopia in a heroin addict (Brust and Richter, 1971). In heroin addicts developing the nephrotic syndrome complement and immunoglobulins can be shown in glomeruli, implying an immunologically mediated response (Kilcoyne, 1975). In the case of transverse myelitis, plexitis, and rhabdomyolysis developing in addicts on restarting the use of heroin, a hypersensitivity reaction has been postulated (Richter and Rosenberg, 1968; Richter *et al.*, 1971; Challenor *et al.*, 1973; Richter and Pearson, 1975). A direct toxic effect on blood vessels or drug allergy have been considered in the case of strokes developing in heroin addicts (Brust and Richter, 1976).

Whatever the mechanism, heroin addiction may be associated with severe neurological complications. Such complications are likely to be seen more commonly as the problem of drug addiction in the United Kingdom increases.

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