Acute myelopathy in a drug abuser following an attempted neck vein injection

Sir: Numerous neurologic complications have been described in association with intravenous drug abuse.1 Myelopathy was initially described in four patients by Richer and Rosenberg in 1968.2 Since then, several more cases have been reported.3,4,5 Our patient is unique in that the complica-
tion ensued within minutes after the injection, from a site quite close to the affected segments. Also, most cases of myelopathy reported in literature have occurred in association with heroin abuse and we are not aware of similar complications reported in association with methylenephendylate (Ritalin) or pentazocine (Talwin) abuse.

A 37-year-old black male who was known to have been a drug abuser of Ritalin and Talwin for nearly ten years was attempting to shoot up a crude tap water suspension of crushed tablets into one of his left sided neck veins when the needle inadvertently struck a spot and caused an acute severe tingling and numbness along his left side. In the next twenty minutes he developed a severe left sided weakness ascending from the toes to involve the whole arm. He could neither walk nor stand, and also experienced a strange feeling on the right side. The needle was withdrawn only after the contents were injected. There was no history to indicate that the patient had stopped abusing drugs prior to this episode. The next morning, 12 hours after the onset of symptoms was brought to the hospital. He was lucid, afebrile, normotensive and without distress though confined to bed. Generalised scars of the skin along veins, confirmed a long term intravenous drug abuse. The group of lymph nodes in the posterior triangle of the neck were slightly enlarged and moderately tender on both sides, suggesting chronic inflammation, but there did not seem to be an acute suppurative process. The rest of the general physical examination was unremarkable.

On neurologcal evaluation, mental status was normal though conforming to a personality disorder. All cranial nerve functions were intact. Hypotonia was noted on the left side, where extremities could be barely lifted against gravity. On the right side, the arm and leg could only be moved against minimal resistance. A definite upper sensory level was obtained bilaterally for pain and temperature at C7-C8 segments but vibration, position and touch sensations were preserved. Tendon reflexes were bilaterally sluggish without asymmetry and plantar responses were absent. The sphincter tone was flaccid. Signs of meningeal irritation and deformation or tenderness of the spine were absent.

A myelogram obtained soon after admission showed no spinal blockage or cord compression although there was a suggestion of swelling of the cord and nerve roots bilaterally at C6-C7 segmental levels. Spinal radiographs did not reveal osteomyelitis or other abnormality. Spinal fluid was colourless with a cell count of 100 WBC per ml (74% polymorphs, 26% lymphs) and 400 RBC per ml, protein was 37 mg/dl, glucose 57 mg/dl and Gram stain showed no organism. Blood, urine and spinal fluid cultures showed no growth. Serum and spinal fluid VDRL was non-reactive. Other investigations that included chest film, ECG, blood counts, electrolytes, liver function tests and urinalysis were normal. The patient, however, refused to have further investigations for possible vasculitis.

Therapy included a three day course of broad spectrum antibiotic, and a short five day course of corticosteroid and physical therapy. A significant improvement was noted from second day onwards. At the end of 2 weeks, he was able to walk unaided, dragging his left leg. Muscles innervated by C5-C6 segments, however, continued to be weak, bilaterally. Shoulder abduction and external rotation could not be performed against moderate resistance and elbow flexion was weak against full resistance. In the lower extremities, only a minimal weakness of left foot dorsiflexors was noted. There was mild increase in tone of all extremities and tendon jerks were bilaterally hyperactive associated with pathological features such as finger flexion in the upper extremities and crossed adductor response in the lower extremities. Plantar responses were equivocal on both sides while the sensory findings remained unchanged.

In most reported cases, the causal relationship of myelopathy to intravenous drug abuse has only been speculative. The proposed mechanisms of pathogenesis are multiple and may differ in individual cases.1 Infection including those of rare Gram negative organisms2 is a frequent complication of intravenous drug abuse. The incidence of spinal osteomyelitis and epidural abscess is much higher in this population and if untreated may result in myelopathy.3 Another known complication in this group is necrotising vasculitis4 which can affect arteries of multiple systems. At least in one patient myelopathy resulted from spinal vasculitis and was proved by biopsy.5 Since in some patients myelopathy occurred after resuming the drug abuse following a period of abstinence, a possible immunologic or hypersensitivity reaction affecting the spinal cord has been suggested, although not proved.6 Ischaemic myelopathy was suggested in some patients who had hypotension1 and developed myelopathy at segmental levels most vulnerable to ischaemia. Other proposed mechanisms include particulate...
embolism to the anterior spinal artery, hyperextension injury to the cord by prolonged stupor or coma, and direct toxic effects of drugs.

In our patient spinal fluid pleocytosis was unequivocal evidence of an acute inflammatory reaction in the spinal column. Infection seemed unlikely since cultures showed no growth and other evidences of sepsis were lacking. In view of the immediate sequel and the proximity of the affected segments to the site of injection, a possible entry of the needle into the spinal canal resulting in trauma or inflammation of the cord, due to direct toxic effects of the injected contents were considered. More importantly, neurological findings were suggestive of an anterior spinal artery syndrome especially as the posterior column sensations were spared. Ischaemia of this arterial territory could have resulted from vasospasm or particulate embolism. Hypotension was not a feature in our patient. Penetration of the needle into the ascending thyrucervical or deep cervical arteries in the neck would have the same effect since these vessels supply radial branches to the anterior spinal artery. Either trauma or the direct irritant effect of the contents could also induce vasospasm. In addition, methylphenidate (Ritalin) could cause vasospasm by its sympathomimetic action and also, is known to cause necrotising vasculitis. Such adverse effects are not documented with pentazocine (Talwin). Prolonged stupor or coma was also lacking in our patient. The possibility of hypersensitivity and immunological reaction producing myelopathy could not be excluded completely.

The exact pathophysiology of the myelopathy in patients with intravenous drug abuse is not well understood. We feel that the spinal cord ischaemia produced by vasospasm secondary to Ritalin was responsible for the cervical myelopathy seen in our patient. It is important to ascertain the cause of myelopathy in this group of patients so that appropriate therapy is promptly instituted.

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References


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Letters

Solitary neurofibroma of the lumbosacral plexus

Sir: There are few reports on the subject of benign neurofibromas in the peripheral nervous system. These tumours have occasionally been encountered in patients with multiple neurofibromatosis. Solitary neurofibromas with no other signs at all of von Recklinghausen’s disease, however, are uncommon, rarely painful and most frequently found in the upper limbs. We report such a tumour located in the lumbosacral plexus.

The patient was a 50-year-old woman, whose first experience of pain occurred during a routine gynaecological examination two months prior to hospitalisation in February, 1983. The digital vaginal examination caused a pain that radiated into the back and the right lower extremity. Since the patient’s right breast had been amputated four years previously, for a carcinoma with limited risk of recurrence, an exploratory examination was recommended. On admission to the Department of Gynaecology and Obstetrics, she complained of pain in the inner thigh that was unaffected by posture and pressure. Ultrasound examination revealed a 3.5 × 4.5 cm smooth tumour mass firmly attached to the right wall of the pelvis. Intravenous pyelography demonstrated a soft tissue mass in the right medial pelvic cavity causing displacement of the ureter. During a laparotomy, an encapsulated tumour was found attached to the piriform muscle and enclosing the branches of the deep pelvic veins. Excision of the tumour was not thought to be feasible, and only a hysterectomy was performed with the removal of the adnexes.

In the course of the following weeks, the patient experienced increasing pain, again in her right inner thigh, and her right leg distinctly lagged when walking. Surgical removal of the tumour, which had meanwhile been diagnosed as a benign neurofibroma was therefore undertaken. The tumour’s upper pole extended to the aortic bifurcation and its lower pole to the obturator fossa, where it was firmly attached to the wall of the pelvis. Dissection began at the level of the obturator fossa. After careful mobilisation, the tumour was totally removed. Pathological examination revealed an encapsulated tumour mass measuring 4 × 4 × 3 cm. On sectioning, the cut surface was of a grey-yellowish colour. Microscopically, the tumour’s capsule was fibrous. The tumour itself was very cellular with scattered fibrillar, cell-poor areas. Elongated oval cells with spindle-shaped nuclei were arranged compactly parallel forming sheets, loose whorls and interlacing bundles (fig 1). There were no atypical nuclei, nor was there any appreciable mitotic activity. In parts, the matrix was myxoid and contained spindle-shaped cells with cytoplasmic cell processes. It surrounded both Schwann cells, many of which contained axons, and bundles of collagen. Axons were found traversing a twisted course within the tumour (fig 2).

Two weeks after operation, the patient was free of pain. Her right leg still lagged slightly but only after prolonged standing or walking. An extremely mild paesthesia of the adductors, abductors and internal rotators of the right lower extremity was observed. When compared with the left, the right adductor reflexes were somewhat weaker. Neither sensory impairment nor muscle atrophy was found. In conformity with the neurological clinical examination, there was electromyographic evidence of denervation in the adductor magnus, gluteus medius and tensor fasciae latae. This denervation indicated involvement of the obturator and the superior gluteal nerves, which innervate these muscle...
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