SHORT REPORT

Post-traumatic syringomyelia following uncomplicated spinal fracture

Andrew Bleasel, Paul Clouston, Nicholas Dorsch

Abstract

Two cases of post-traumatic syringomyelia presenting 10 and 41 years after spinal injuries that had caused lumbar vertebral fractures but no lasting neurological deficits are reported. In both patients the caudal end of the syrinx cavities, as shown by MRI, corresponded to the level of the previous vertebral fractures. Patients presenting with post-traumatic syringomyelia after uncomplicated spinal fracture are very rare, and the significance of the past history of spinal trauma may be overlooked.

Post-traumatic syringomyelia is an uncommon late complication of spinal cord injury occurring in 1·2–3·2% of patients with traumatic paraplegia or tetraplegia.[1–3] It has also been described in patients with a history of spinal trauma, but with no deficit or only transient neurological symptoms at the time of injury.[4] We report two cases of syringomyelia presenting years after spinal injuries that had caused vertebral fractures but no lasting neurological symptoms or signs.

Case 1

A 62 year old woman presented in November 1986 with an 11 month history of low back pain, left sciatica and progressive left leg weakness. In 1976 she had fallen from a stool, sustaining a crush fracture of the L1 vertebra. She had experienced some left leg weakness following the fall but eventually made a complete recovery. On examination in 1986, she walked with the aid of a stick, there was no wasting in the lower limbs but there was weakness of left knee extension and ankle dorsiflexion. The left knee jerk was reduced and the left ankle jerk absent. There was diminished sensation to pinprick and light touch in the left L5 and S1 dermatomes; joint position sense was normal. Electromyographic studies gave evidence of chronic partial denervation in the left vastus lateralis and tibialis anterior muscles. A lumbar myelogram showed a complete block at the lower border of the 12th thoracic vertebra; following a lateral cisternal injection, the upper end of the block was found at the lower border of the 10th thoracic vertebra (fig 1). An MRI scan showed a cystic cavity of the spinal cord extending from the upper T11 to the lower T12 level. No abnormality was seen at the craniocervical junction.

At operation, after complete laminectomy of T12 and L1, a cystic expansion of the conus medullaris was found to be a syrinx cavity; inspection showed no evidence of tumour within. Drainage was effected via a myelotomy along the length of the syrinx. Postoperatively, the patient lost her pain and had some improvement in the left lower limb weakness. When reviewed in 1988 she walked well without assistance; some weakness of the left lower limb persisted but the sensory examination was normal.

Case 2

A 61 year old man was first seen in July 1987; he had sustained a fracture of L1 during a motor vehicle accident in 1946. He could recall no neurological symptoms after the injury, but had been immobilised in a plaster spica for three months. In 1963 he had developed an acute demyelinating peripheral neuropathy three weeks after a smallpox vaccination; within 24 hours of the onset of symptoms he had become tetraplegic and required mechanical ventilatory support for four months. He had made an incomplete recovery from this illness, being left with distal wasting and weakness of all four limbs; he could walk with the aid of elbow crutches and had no residual sensory loss.

In January 1987 he developed left sided low thoracic pain, which by July had ascended to the upper border of the left trapezius muscle, and was exacerbated by coughing or straining. He had noted decreased temperature sensation in the left upper limb and later developed weakness about the left elbow. On examination, there was distal wasting and weakness in the four limbs and areflexia from his peripheral neuropathy. Pinprick and temperature sensation were lost on the left side from C3 to T11, while joint position, vibration and light touch sensation were normal.

A lumbar myelogram demonstrated a partial block to the flow of contrast just above the old vertebral fracture of L1, with marked expansion of the cord above. An MRI scan (fig 2) showed that a syrinx cavity extended from L1 to the high cervical area, but did not communicate with the IVth ventricle; there was no Arnold-Chiari malformation. A total laminectomy of T11 to L1 was performed and a syringopleural shunt inserted with the distal

Royal Prince Alfred Hospital, Sydney
Department of Neurology
A Bleasel
Department of Neurosurgery
N Dorsch
Concord Hospital, Sydney, Australia
Department of Neurology
P Clouston

Correspondence to: Dr Bleasel, Department of Neurology, Royal Prince Alfred Hospital, Missenden Road, Camperdown, NSW 2050, Australia

Received 5 April 1990.
Accepted 22 August 1990.

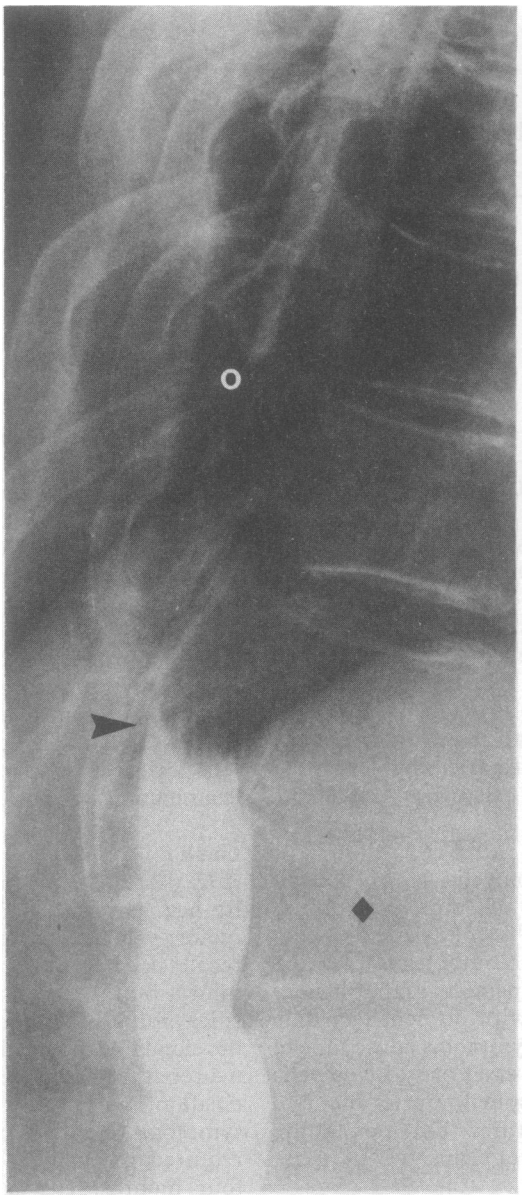

Figure 1 Lateral view of an Iohexol myelogram (case 1) following both lumbar and cervical punctures. There are blocks at T12 (arrowhead), just above the old L1 vertebral fracture (diamond), and at the lower border of T10 (open dot).

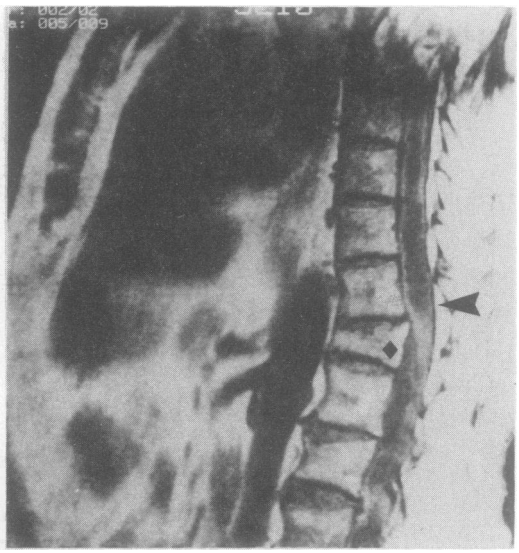

Figure 2 Sagittal MRI scan of the spine (case 2). A syrinx cavity begins at the conus (arrowhead), at the level of the previous crush fracture of L1 (diamond), and continues to where the spinal cord leaves the scan.

end of the shunt tube in the right pleural cavity. Postoperatively the patient had some improvement in his pain, and subjective improvement in the temperature sensation in his left upper limb. A repeat MRI scan demonstrated that the syrinx cavity had collapsed.

Discussion

We have described two cases of syringomyelia presenting years after spinal injuries resulting in crush fractures of lumbar vertebrae but no persistent neurological deficit. The lower level of the syrinx cavity corresponded to the level of the fracture in both cases. Neither patient had an Arnold-Chiari malformation and the close relationship between the syrinx cavity and the vertebral fracture argues strongly that the previous spinal injury was relevant to the pathogenesis of syringomyelia in these patients.

Barnett[5] defined three categories of post-traumatic syringomyelia; following traumatic paraplegia and tetraplegia, following minor or moderate spinal cord injury and as a late complication of spinal trauma producing adhesive arachnoiditis. Barnett and Jousse[4] reviewed six cases, one previously reported,[6] that they felt were sufficiently well documented to be accepted as examples of post-traumatic syringomyelia following mild to moderate trauma. Two of their patients had no neurological symptoms following the original spinal injury, one had a transient paresis of the right upper limb while the other three had persistent but mild sensory or motor symptoms, including one patient who was tetraplegic immediately after the accident yet recovered within the first month.[4] Although some of the more recently published series of post-traumatic syringomyelia have reported patients with incomplete cord lesions following the initial injury[2 3 7 8] no other cases with complete recovery after the spinal injury have appeared in the literature since Barnett and Jousse's review.[4]

The subsequent presentations of Barnett and Jousse's cases were similar to the syringomyelic syndrome that follows paraplegia and tetraplegia: pain, initially unilateral, segmental and possibly ascending, sensory disturbance (often dissociated), and new weakness with areflexia.[1 3 7 8] The interval between the original injury and the later presentation with syringomyelia varies greatly between patients. A range of three months to 34 years exists in the literature for paraplegia and tetraplegia patients[1 3 7 8] and four to 22 years for the mild to moderate spinal injury patients.[4] Our second case did not develop symptoms referable to the syringomyelia until 41 years after the spinal injury.

The severity of the original injury was sufficient to produce a vertebral fracture in each of our patients; four of the six patients reviewed by Barnett and Jousse had also sustained vertebral fractures, and they postulated that post-traumatic syringomyelia following mild to moderate trauma shared pathogenetic mechanisms with that following paraplegia or tetraplegia.[4] Factors involved in the produc-

tion of the early cord cavitation may include central cord softening due to trauma, ischaemia and intramedullary haemorrhage.[7 9 10] Cyst extension, leading eventually to the development of new neurological symptoms and signs, has been thought to be due to the transmission to the cyst cavity of cerebrospinal fluid pressure fluctuations occurring with flexion and extension of the spine and straining.[10] Any local arachnoid adhesions tethering the spinal cord enhance the transmission of CSF pressure fluctuations and cyst extension, and may contribute to cord ischaemia.[9 10] Indeed, two of the cases reviewed by Barnett and Jousse[4] had severe adhesive arachnoiditis at the site of the original trauma without vertebral fracture. Neither of our patients had significant arachnoiditis at the site of trauma as determined by inspection at the time of surgery.

A different type of operation was carried out in each of our patients. In one, with a syrinx occupying only a short length of the cord at the conus, a syringostomy of the whole length of the cavity ("terminal ventriculostomy") was, at least in a two year follow up, sufficient treatment. The other had a much more extensive syringomyelia, involving most of the spinal cord; in this situation a syringopleural or syringoperitoneal shunt probably gives the best results. Surgery is most likely to be useful where there is progressive deficit as in our cases, and should be avoided if the patient is not deteriorating.[11]

Patients presenting with post-traumatic syringomyelia years after a spinal injury that caused no lasting neurological deficit are very rare, and the significance of a past history of spinal trauma in such patients may be overlooked.

We thank Drs J E Hassall and D Sharp (case 1) and Dr I Johnston (case 2) for providing additional clinical information, and Professor J G McLeod for his help in preparing the paper.

1 Barnett HJM, Jousse AT. Syringomyelia as a late sequel to traumatic paraplegia and quadriplegia—clinical features. In: Barnett HJM, Foster JB, Hudgson P, eds. *Syringomyelia*. London: WB Saunders, 1973:129–53.
2 Watson N. Ascending cystic degeneration of the cord after spinal cord injury. *Paraplegia* 1981;**19**:89–95.
3 Rossier AB, Foo D, Shillito J, Dyro FM. Posttraumatic cervical syringomyelia. *Brain* 1985;**108**:439–61.
4 Barnett JHM, Jousse AT. Posttraumatic syringomyelia (cystic myelopathy). In:Vinken PJ, Bruyn GW, eds. *Handbook of clinical neurology*, Vol 26. Amsterdam: North-Holland, 1976:113–57.
5 Barnett HJM. Trauma and syringomyelia. In: Barnett HJM, Foster JB, Hudgson P, eds. *Syringomyelia*. London: WB Saunders, 1973:127–8.
6 Bischof W, Frowein RA. Zur Spaetmyelomalazie nach Kompressionsfraktur. *Zentralblatt fuer Neurochirurgie* 1967;**28**:61.
7 Shannon N, Symon L, Logue V, Cull D, Kang J, Kendall B. Clinical features, investigation and treatment of post-traumatic syringomyelia. *J Neurol Neurosurg Psychiatry* 1981;**44**:35–42.
8 Anton HA, Schweigel JF. Posttraumatic syringomyelia; the British Columbia experience. *Spine* 1986;**11**:865–8.
9 Barnett HJM Jousse AT, Ball MJ. Pathology and pathogenesis of progressive cystic myelopathy as a late sequel to spinal cord injury. In: Barnett HJM, Foster JB, Hudgson P, eds. *Syringomyelia*. London: WB Saunders, 1973: 179–219.
10 Williams B, Terry AF, Jones F, McSweeney T. Syringomyelia as a sequel to traumatic paraplegia. *Paraplegia* 1981;**19**:67–80.
11 Peerless SJ, Durward QJ. Management of syringomyelia: a pathophysiological approach. *Clin Neurosurg* 1983;**30**: 531–76.