Spontaneous incarcerated herniation of the spinal cord into a vertebral body: a unique cause of paraplegia

Case report

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A case of progressive paraparesis is reported in which the normal spinal cord herniated through a defect in the ventral meninges and dura and became incarcerated in a cavity in the dorsal aspect of the body of T-7. Progress of the paraparesis was arrested by reduction of the hernia.

Key Words □ spinal cord □ herniation □ vertebral body □ arachnoid □ dura □ pia

This case is reported because it illustrates a unique cause of spinal cord compression.

Case Report

The patient was a diabetic 63-year-old farmer who had fractured his pelvis in 1933 and suffered several minor injuries to his back during 1968. Following the latter he first noticed aching pain in the lumbar spine and pelvis and easy fatigability while carrying his elderly mother in his arms. During April, 1968, he noticed loss of temperature discrimination in his right leg, followed by numbness in the calf and foot; there was also gradually increasing clumsiness of the feet and he began falling frequently.

First Examination. In January, 1969, he was admitted to the Toronto General Hospital where he was found to have a left ptosis and myosis, weakness of the lower abdominal muscles, a hyperactive left knee jerk, and an upgoing left toe. There was hypalgesia and thermalalgesia on the right below T-10, and vibration sense was absent at the right ankle. Plain films of the spine and a myelogram were considered normal; the cerebrospinal fluid (CSF) contained 79 mg% protein.

Between January, 1969, and November, 1970, the left leg became progressively weaker until he was forced to give up work and to walk with a cane. He developed urgency and occasional incontinence of feces, as
well as urgency and finally retention of urine. A transurethral resection of the prostate in August, 1969, improved function.

Second Examination. The patient was readmitted to the hospital on November 22, 1970. There was only a flicker of power in the dorsiflexors of the left ankle, nearly normal power in the flexors of the left hip and hamstrings, and slight weakness of other muscles in the left leg, which was slightly spastic. The right knee and ankle jerks were absent; those on the left were hyperactive. There was a positive left Babinski response. The patient had now developed bilateral hypalgesia and thermanalgesia below T-9. Plain spine films were again normal but upright views of a repeated myelogram (Fig. 1) now showed almost complete obstruction to the column of contrast material opposite the body of T-7, without lateral displacement of the cord. This suggested extradural compression of the subarachnoid space in the anteroposterior plane. Tomograms showed a corticated bony defect suggesting a benign lesion in the dorsal aspect of the T-7 vertebra opposite the myelographic block as shown in Fig. 2; the lateral projection of the myelogram (Fig. 3) shows the spinal cord displaced toward the intravertebral lesion.

Operation. On December 9, 1970, the right dorsal quarter of the body of T-7 was resected using a transthoracic approach through the bed of the seventh right rib. A smooth-walled, corticated, roughly spherical, nearly midline cavity 1 cm in diameter in the dorsal aspect of the cephalic portion of the body of T-7 was entered; this space communicated with the spinal canal through a narrow opening. It contained a smooth, rubbery, yellowish-white tumor-like sphere with a flimsy capsule whose contents grossly resembled nervous tissue. A minute biopsy showed reacting astrocytes,

Fig. 1. Anteroposterior view of cisternal myelogram in the upright position, November, 1970, showing nearly complete occlusion of the subarachnoid space at T-7 without lateral displacement of the cord, suggesting an extradural lesion.

Fig. 2. Lateral tomographic cut about the center of the bodies of T6–8 showing a corticated bony defect in the dorsal aspect of the body of T-7 at the level of the block.
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residual myelinated nerve fibers, and degenerating myelin, indicating unquestionably that this was central nervous tissue (Fig. 4). The intervertebral foramen was enlarged to visualize the spinal dura opposite the lesion. The spinal cord was then exposed through a longitudinal dural incision. The cord had been displaced toward the intravertebral nodule with which it connected on its midventral surface by means of a narrow neck passing through a smooth-edged, rounded dural defect into the bony cavity in T-7 (Fig. 5). With the use of × 10 magnification, the dura was then incised at right angles to the first dural incision along the side of the communicating neck toward the defect in T-7, whose small opening was enlarged. Figure 5 illustrates what was now clearly apparent; there was a partial hernia of the ventral surface of the cord so that it passed through a dural defect to become incarcerated within the cavity of the body of the T-7 vertebra. The hernia was not covered by dura, subarachnoid space, or leptomeninges, but only by flimsy pseudocapsular tissue. Upon wider opening of the dural and bony orifices, the hernia retracted spontaneously into the spinal canal whereupon the cord resumed a normal position and began to pulsate for the first time. The hernia could still be recognized as a yellowish, amorphous nodule on the midventral surface of the cord to which it was deeply attached. The dural defect was sutured and sealed with muscle.

Postoperative Course. The postoperative course was stormy, complicated by coagulase-positive staphylococcal meningitis and empyema, and treated with antibiotics and drainage. The diabetes was aggravated, and insulin was required. Methicillin nephropathy, proven by open biopsy, responded to steroid therapy, and by February 26, 1971, recovery began. At first the patient was only able to walk with great difficulty, both legs being weak, but rehabilitation proceeded steadily until, by

Fig. 3. Lateral projection of myelogram showing displacement of the shadow of the spinal cord (arrows) toward the body of T-7 at the site of the lesion shown in Fig. 2.

Fig. 4. Photomicrograph of the surgical biopsy revealing residual myelinated axons lying among reacting astrocytes and macrophages. Combined H & E, and Luxol fast blue, × 225.

J. Neurosurg. / Volume 41 / November, 1974
November, 1971, he had virtually regained his preoperative neurological level. He resumed working on the farm early in 1972, though he required a cane. By November, 1972, his neurological status had become stable. He had normal power in the right leg, 2-3+ power in the dorsiflexors of the left ankle, flexors of the left hip, and left hamstrings. The left knee and ankle jerks were absent, the right hyperactive, while the right plantar reflex was unresponsive, the left upgoing. Vibration sense was absent at the right ankle, and there was bilateral analgesia and thermanalgesia below T-9, incomplete on the right. Bowel and bladder function were normal.

Discussion

To understand the lesion found in this patient, two phenomena must be considered: first, the pathogenesis of the defect in the leptomeninges and dura, and, second, the mechanism of herniation of cord tissue through the defect.

Spinal dural defects that are not iatrogenic or posttraumatic are rarely encountered, are usually situated anteriorly, and are due to intrathecal rupture of disc material. Blikra added two cases in the lumbar spine to the nine similar ones found in the literature, Tovi and Strang described one in the thoracic spine, and we have seen a similar case. In one of Fisher's patients, sequestrated disc was found actually embedded in the anterior surface of the cord while in another case there was extensive dural erosion dorsally over three vertebral levels, secondary to posterior displacement of the cord caused by herniated disc material. In our patient there was nothing to suggest disc disease other than the history of trauma. Truly spontaneous defects in the spinal dura have not previously been reported. A review article on morphological abnormalities of the spinal dural space does not mention this type of lesion, nor do the standard text books. We have not encountered any references in the literature to defects in the leptomeninges.

The most familiar type of spinal cord herniation, the meningocele, involves the spinal cord and its meninges. Intrathoracic meningoceles, 64% of which are associated with neurofibromatosis, have been reported with increasing frequency; review articles now describe some 72 cases. However, meningoceles are situated laterally and associated with local dural dysplasia or preexisting skeletal defects and usually protrude through an intervertebral foramen. They are frequently an incidental finding in chest films where they appear as rounded paravertebral soft tissue masses. However, herniations of this sort are extensions of the subarachnoid space so that they are covered with meninges, contain cerebrospinal fluid, and fill with contrast medium during myelography. Our case lacks all these essential characteristics of a meningocele.

One might have expected that hernias of the spinal cord with its leptomeninges would occur commonly through dural defects since postoperative decompressions are so frequently left and since it has been shown that the cord possesses sufficient mobility to enter such defects. But Cobb and Ehni, in describing a unique case of myelopathy caused by an incarcerated herniation of the cervical cord into an iatrogenic dorsal dural defect, have pointed out how rare such occurrences actually are. We suggest that the rarity of this type of spinal cord hernia is due to the restraint exerted on the cord by the tensile strength of the arachnoid and pia mater, which are usually left intact at surgery.

If, however, the cord surface is incised, a different type of herniation involving cord tissue alone, devoid of its coverings, can apparently occur. In the autopsy room, hernias of the spinal cord occur readily through inadvertent nicks in its collagenous outer cov-
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neurological deterioration and radiological paradox of cord displacement toward an apparently compressive extradural lesion. The chronicity of the process is emphasized by the cortication of the cavity in the body of T-7.

References

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