Posttraumatic pseudomeningomyelocele (enlarging fracture?) in a vertebral body

Case report

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An unusual case is reported in which a posttraumatic pseudomeningomyelocele developed over many years inside the body of a fractured lumbar vertebra, eroding the pedicle and causing progressive neurological deficit. The wall of the sac was mostly formed by the scalloped bare bone, and partly by a membrane resembling the dura. The terminal part of the conus medullaris and some nerve roots of the cauda equina formed the contents and parts of the wall of the outpouching of the subarachnoid space into the vertebral body. A comparison is drawn between this lesion and formation of an "enlarging fracture" of the skull. The surgical technique used for obliteration of this pseudomeningomyelocele is described.

KEY WORDS □9 meningocele □9 posttraumatic meningocele □9 posttraumatic meningomyelocele □9 spinal trauma □9 enlarging fracture

The term "traumatic meningocele" was used by Murphey, et al.,18 to describe myelographic outpouchings from the subarachnoid space during the investigation of a patient with cervical nerve root avulsion. Various other names have been given to more or less similar lesions found mostly on myelographic studies and occasionally at surgery. Some of the terms used have been "meningeal pseudocysts," "spurious meningocele," "extradural pseudocyst," and "post-traumatic arachnoid diverticula."

We present a unique case of a posttraumatic pseudomeningomyelocele developing insidiously inside the body of a fractured vertebra, diagnosed many years after initial injury, and causing progressive neurological deficit.

Case Report

This 44-year-old woman was involved in an automobile accident in November, 1963. At the time of impact, she was thrown out of her car, landing on the ground on her right gluteal region. She experienced severe pain in her back and was unable to move her right leg. The right lower extremity below the level of the groin felt numb. X-ray films at a local hospital reportedly revealed a compression fracture of the L-1 body. After 5 days, during which time she made no neurological improvement, she was transferred to another hospital where a repeat set of lumbar spine films was reported to show "fracture of the body of L-1 vertebra with compression deformity of the upper plate as well as a line of increased density in the midportion of the body caused by trabecular condensation." There was a "discontinuity of the left pedicle suggesting extension of the fracture through the pedicle."

The patient was placed in a body cast for the next 6 weeks. There was no return of power in the right lower extremity, and the limb continued to feel numb. There were no symptoms referred to the left leg, and bladder and bowel sphincter control was normal. After the body cast was removed, a myelogram was performed. This was reported as showing "a long narrowing of the opaque column at the level of the body of L-1 most likely the result of reactive changes secondary to trauma." In January, 1964, she underwent surgery at another hospital. "A complete laminectomy of L-1 was performed . . . adhesions between dura and bone
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FIG. 1. Plain films of the thoracolumbar spine showing scalloping and cavitation of the posterior part of the body of L-1 encroaching on the right pedicle. Left: Lateral view showing the lobulated lesion with sclerotic margins (arrowheads). Right: Anteroposterior view showing the scalloped lesion (single arrowheads) and encroachment on the right pedicle by the lesion. Double arrowheads point to the lobulated left border.

were freed . . . intradural exploration was normal . . . dura was closed." Two weeks after the laminectomy she began to notice return of sensation in the right lower extremity, and within the next few weeks there was progressive improvement in motor power. Within a year she was able to walk without support, although with a limp. She resumed gainful employment and continued to do well for 9 years.

FIG. 2. Tomograms of the thoracolumbar spine showing the sclerotic margins of the lesion (arrowheads). The old fracture of the superior and inferior end-plates of the vertebral body is visualized (arrows). Left: Frontal view through the posterior part of the body of L-1.

In 1973, she started to notice pain around the right knee, radiating to the inner side of the right leg, and frequent cramps involving the muscles of the right leg. Approximately 2½ years later, 18 months prior to admission to Mount Sinai Hospital, she noted progressive weakness of the right leg, mainly in dorsiflexion of the right foot and flexion and extension of the right knee. A myelogram was performed at another hospital, but was considered inadequate. She was admitted to Mount Sinai Hospital on October 1, 1977.

Examination. Neurological examination revealed almost complete absence of dorsiflexion and eversion of the right foot, and moderate weakness of plantar flexion and extension at the right knee and hip. There was marked atrophy of the anterior tibial and calf muscles, and flexors and extensors of the knee and hip. Her spine was straight, and the laminectomy scar was well healed. All sensory modalities were markedly diminished from the L-2 to L-5 dermatomes on the right side, most marked in the L-4 and L-5 dermatomes.

Investigations included plain x-ray films of the lumbar spine (Fig. 1), polytomography (Fig. 2), computerized tomography (CT) of the spine (Fig. 3), and a full-column Pantopaque myelogram of the lumbar spine (Fig. 4).

Operation. Lumbar reexploration (V.S.) was carried out on October 5, 1977. The laminectomy was completed from T-12 to L-3. The articular facets at L1-2 on the left side were markedly deformed, and the dura was strongly adherent to the bone. By means of microsurgical technique, the dura was opened in the midline. The right pedicle and body of L-1 were seen to have a large smooth cavity filled with cerebrospinal fluid (CSF). Some nerve roots of the cauda...
FIG. 4. Preoperative Pantopaque myelogram. **Left:** Lateral view. Cavity of the sac (S) in the posterior part of the body of L-1 is continuous with the spinal subarachnoid space. The conus medullaris (C) is pulled toward the neck of the sac while, inferiorly, nerve roots (small arrowheads) are emerging from the neck. Note the fluid level (large arrowheads) of the Pantopaque column. **Right:** Anteroposterior view. The conus medullaris (C) is deviated toward the neck of the cavity located on the right side of the body of the vertebra (Fig. 3). Note erosion of the right pedicle (P) by the sac (S). Entering (A) and emerging (B) nerve roots are clearly seen.

FIG. 5. Myelogram with Pantopaque 8 weeks after surgery. **Left:** Lateral view. The sac (arrowheads) is no longer filled with Pantopaque. A few droplets of Pantopaque anterior to the theca within the sac were not in communication with the theca on fluoroscopy. The conus medullaris (C) is now posteriorly located. Compare with Fig. 4 left. **Right:** Anteroposterior view showing the bone sac (arrowheads) free of Pantopaque except for fixed droplets. Although the conus medullaris (C) remains deviated toward the right, it is now no longer in proximity to the neck of the sac as shown by the lateral view (left).

equina were dipping into the cavity, others coursed around the neck of the cavity. The nerve roots entering the cavity were acutely angulated at the bone neck and appeared thinned and flattened. The conus medullaris was deviated toward the opening of the cavity in the body of the vertebra. The roots of the cauda equina coursing through the cavity were brought out. Some rootlets adhered tightly to the bone, and there was no obvious membrane between the neural tissue and the bone.

After all the neural elements were brought out of the cavity in the body of the L-1 vertebra, the cavity was filled with a large plug of fat removed from the subcutaneous tissue. A piece of lumbar fascia was placed on the fat plug, anterior to the nerve roots and the lower part of the conus, thus reconstructing the anterior wall of the dural sac.

**Postoperative Course.** The right lower extremity was considerably weaker as compared to the preoperative status, but over the next 6 months it returned to its preoperative level. There was no neurological deficit in the left lower extremity, and bladder and bowel sphincter control was normal. A postoperative myelogram 2 months after surgery (Fig. 5) showed adequate obliteration of the pseudomeningomyelocele.

Over the next 27 months, the patient's leg pain markedly diminished and she had progressive improvement in muscle power in all groups of affected muscles. She still had residual sensory deficit as before surgery. She was readmitted on December 26, 1979, for metrizamide lumbar myelography (Fig. 6) which demonstrated the reappearance of a small part of the cyst, presumably due to shrinkage of the fat plug and scarring. However, the conus medullaris remained well posterior to the cavity in the bone. As she has remained neurologically stable, and the major part of the cyst is obliterated, no further surgical procedure is planned at present.

**Discussion**

Congenital symptomatic and asymptomatic arachnoid diverticula in the spinal column have been reported.\(^{1,2,25,26,29,32,33}\) Posttraumatic arachnoid diverticula, for example after cervical nerve root avulsion, have been described.\(^{8,14,16-18,23,24,36,38,39,40}\) More extensive forms of these outpouchings extending out of the spinal canal into the thoracic cage have infrequently been found.\(^{4,8,19,20,38,42}\) Lumbar pseudomeningoceles after root avulsion or trauma to bone structures have also been reported.\(^{1,2,6,7,9,12,21}\) Iatrogenic pseudomeningoceles or pseudocysts after spinal surgery are not uncommon.\(^{3,27,34,39}\) A search of the literature has failed to reveal a case similar to the one reported here. Wortzman, *et al.*\(^{40}\) described the case of a 63-year-old man with progressive neuro-
logical deficit due to herniation of the ventral part of the spinal cord into the body of the T-7 vertebra. The cause of this herniation was not obvious, but the authors did not think that the herniation was related to trauma. Hoffman, et al.,\textsuperscript{18} reported a case of a 12-year-old boy with paraparesis progressing over a period of 5 years after craniospinal trauma. They mention “loss of pedicle” at the level of the lesion in the x-ray report. At surgery, a large arachnoid diverticulum dorsal and lateral to the dura mater and communicating with the subarachnoid space at C-5 was discovered and excised with excellent postoperative neurological recovery. It is quite likely that the loss of the pedicle noted on the x-ray films was caused by a continued pulsatile effect of the diverticulum which was in communication with the subarachnoid space. The mechanism of formation of the lobulated erosive cavity in our case was most likely the pulsatile pressure of the subarachnoid CSF over the course of many years.

It is tempting to draw a comparison between the formation of such a lesion and the so-called “enlarging fracture” of the skull. Goldstein, et al.,\textsuperscript{18} presented experimental evidence that, in addition to the open dura, a tear in the arachnoid is also necessary. Their experiments on dogs indicated that the formation of a cyst or pouch in the separated margins of bone increases the incidence of enlarging craniotomy lines as compared to simple opening of the dura and the arachnoid. Taveras and Ransohoff\textsuperscript{31} postulated a similar mechanism in their experiments.

In our case, fracture of the body of L-1 was, in all likelihood, associated with a tear in the ventral dura. A pseudocyst in communication with the lumbar subarachnoid space resulted. It is conceivable that the type of fracture of the L-1 body in our case helped in the formation of such a pseudocyst. It appears that the fracture extended vertically in the posterior surface of the body without involving the anterior and lateral cortical bone. The possibility of this type of fracture having occurred is supported by the plain films and tomograms which show no compression of the anterior part of the body of L-1, and indicate that the normal height of the body was maintained. There was no evidence of the healed fracture extending to the anterolateral cortex of the body.

The unique nature of the vertical fracture of L-1 in our case, involving only the posterior part of the body of the vertebra without fragmentation of the body, appears to have provided the necessary prerequisite for this pseudocyst formation and further enlargement by transmitted CSF pulsations.

We believe that the fracture line in the body of L-1, with the break in the pedicle, opened and immediately closed at the time of impact due to the molding force exerted by the intact anterolateral cortical shell, much like the closing of a partially cracked walnut. The ventral dura was most likely torn on impact. The dura and arachnoid were probably trapped in the fracture line, forming a “pouch” in communication with the subarachnoid space. This set the stage for normal pulsations of CSF to “enlarge” the fracture over many years. The “pouch” or “pseudocyst” progressively enlarged into the body and eroded the pedicle. The neural elements herniated into this sac. Progressive stretch caused the continued increase in neurological deficit over many years.

The rarity of enlarging fractures, even in the skull, is still unexplained. Perhaps the “ball valve mechanism” with arachnoidal enlargement, as postulated by Taveras and Ransohoff,\textsuperscript{31} may help to explain why the fracture line “enlarges” in rare cases.

It is our belief that in our case, by obliteration of the cavity of the body of L-1 with fat and reconstruction of the anterior dura with fascia, further herniation of neural elements into the cavity and increase in neurological deficit has been halted to a major extent. The patient is being followed clinically, and in the event of increase in signs and symptoms secondary to further enlargement of the cystic pouch, further repair,
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probably with muscle and fascia, may have to be considered.

References


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