Extradural cyst causing spinal cord compression in osteoporotic compression fracture

Case report

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Intraspinal cystic lesions with different pathogeneses have been reported to cause neurological deficits; however, no one has focused on the intraspinal extradural cysts that develop after osteoporotic compression fracture. The reported case features a 66-year-old woman presenting with progressive neurological deficit, back pain, and no history of additional trauma after undergoing conservative treatment for an osteoporotic fracture of L-1. The authors present serial radiographs and MR images demonstrating an epidural cyst successfully treated via a single posterior approach. This appears to be the first such case reported in the literature. (http://thejns.org/doi/abs/10.3171/2013.4.SPINE121101)

Key Words  • neurological deficit  • extradural cyst  • osteoporotic compression fracture

OSTEOPOROTIC compression fractures, described as stable spinal injuries, are common in the elderly and in most cases are well managed with nonoperative treatment or percutaneous bone cement augmentation. Neurological compromise, mostly caused by delayed collapse of the vertebral body that results in kyphosis, is not uncommon in elderly osteoporotic patients.

Several types of intraspinal cysts with different pathogeneses have been reported, such as perineural cysts, synovial cysts, arachnoid cysts, ganglion cysts, ligamentum cysts, and perimembranous hematoma. To our knowledge, however, no studies have described the neurological deficits that develop as the result of an intraspinal extradural cyst after OCF.

We describe the case of a patient presenting with neurological deficits after OCF and analyze the characteristics, radiographic appearance, and histological findings of an intraspinal cyst in the ventral extradural space that caused the deficits. The possible reasons for the neurological deficits and intraspinal cysts are explored based on a review of the literature.

Case Report

History and Examination. A 66-year-old postmenopausal woman presented with progressive paraparesis and backache lasting a month. She had been treated at another institute with a custom-made plastic thoracolumbosacral orthosis for an L-1 OCF after incurring an injury while sitting down 3 months earlier. Plain radiographs taken at the time revealed anterior wedging of the L-1 vertebral body and degenerative changes of the lumbar spine. Magnetic resonance imaging showed anterior wedging with bone marrow edema at the same level (Fig. 1). Dual-energy radiograph absorptiometry showed osteoporosis of -2.9 SDs on the lumbar spine.

Two months later, radiating pain developed in the left lower extremity and became aggravated. The patient was transferred to our institute with neurological deficits including sensory changes and weakness in the lower extremities. She was no longer able to stand without support. She had no history of other trauma, chronic illness, or addiction. Plain radiographs demonstrated progression

Abbreviation used in this paper: OCF = osteoporotic compression fracture.
of regional kyphosis due to collapse of the anterior and midcolumn of the injured vertebra (Fig. 2A). Magnetic resonance imaging showed a fluid collection with resorption of the cancellous bone adjacent to the inferior endplate, spinal canal encroachment with cortical disruption, cord compression by the cystic lesion on the left paracentral posterior wall, an extradural cyst location, and homogeneous high signal intensity on T2-weighted images at the level of the injured vertebra of L-1 (Fig. 2B).

**Operation.** Because the kyphotic deformity was progressive over time and causing neurological deficit, the patient underwent posterior fusion with anterior column reconstruction and complete resection of the cyst. During the surgical procedure, water-soluble nonionic contrast material was injected under fluoroscopic guidance to reveal the integrity of the cystic lesion. Contrast material was delivered after placing the needle in the T12–L1 and L1–2 discs via a transforaminal approach and the L-1 vertebral body via the pedicle. After injecting the contrast agent, lateral fluoroscopic imaging revealed spread of the opacification of the vertebral body into a round, homoge-

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**Fig. 1.** Sagittal T2-weighted (A and B) and axial (C) MR images obtained 3 months earlier, immediately posttrauma, showing a 10% compression fracture without any bony encroachment or dural sac compression.

**Fig. 2.** A: Serial plain full-spine radiographs revealing a kyphotic angle of +0.3° increasing to +9.9° over 3 months. The sagittal positive imbalance was 48 mm. B: High signal intensity on T2-weighted images (*center and right*) and low signal intensity on a T1-weighted image (*left*) showing a round to oval mass lesion compatible with a liquid-containing cyst, a relatively large ventral extradural space-occupying lesion.
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neous cystic lesion on the posterior wall of L-1 (Fig. 3). Resection of the cyst was performed via a single posterior approach under somatosensory and motor evoked potential monitoring. Pedicle screws were inserted 2 levels above (T-11 and T-12) and 1 level below (L-2) the injured vertebra and did not include the injured vertebra. After laminectomy and foraminotomy were performed on the left side of the injured vertebra to decompress neural elements, the transverse process was removed to expose the lateral wall of the pedicle. Meticulous subperiosteal dissection was performed down to the lateral wall of the vertebral body. Using direct visualization, we moved the left pedicle of L-1 to expose the cyst (Fig. 4). The dural sac and left L-1 root were compressed by a well-circumscribed cyst located in the left anterior extradural space of the vertebral canal, with no adhesions to the nerve root and dural sac but some to the peridural membrane. The cyst was excised, and the posterior part of the L-1 vertebral body was exposed. After removing the L-1 vertebral body, the kyphotic deformity was slightly improved by gravity while the anterior gap widened. For an anterior column reconstruction, a titanium expandable cage was inserted. Then, the remaining local bone was impacted into a space between the cage and the preserved vertebral cortex. Final evaluation of the spinal canal should be performed to confirm that there is no residual compression or translation at the resected margins. After the resection and anterior fusion procedures, rods precontoured to the normal sagittal alignment were seated into screws and locked under compression over the resected margins.

Postoperative Course. Postoperative plain radiography demonstrated −15.9° of local kyphotic angle (Fig. 5), and sagittal alignment was nearly restored (9 mm ante-

![Fig. 3. Lateral fluoroscopic image showing contrast material extending from the L-1 vertebral body (empty arrow) to the ventral extradural cyst (filled arrow). However, this cyst did not communicate with the intervertebral disc. Tips of the spinal needles were placed at the T12–L1 disc, L-1 vertebral body, and L1–2 disc.](image)

rior to the C-7 plumb line). Therefore, 25.8° of correction had been achieved. The patient was out of bed with a thoracolumbosacral orthosis after 3 days. Immediately after surgical treatment, the pain radiating to her lower extremities resolved, and she was able to walk without support 6 weeks after surgery. At the 2-year follow-up evaluation, sagittal correction was maintained and there was no evidence of a recurrence. Histology confirmed the diagnosis of a multicellular intraspinal cystic lesion lined with well-differentiated fibrous connective tissue and containing fibrin and red blood cells (Fig. 6).

![Fig. 4. After left pedicle (dotted circle) subtraction, the cyst (empty arrow) was observed at the ventral side of the dural sac and the L-1 root (filled arrow).](image)

Discussion

Intraspinal space-occupying lesions are common causes of low-back pain and neurological deficit. Advances in radiographic technology, including MRI, have allowed identification of a variety of intraspinal space-occupying lesions. The most common of these lesions is lumbar disc herniation. But some lesions, such as intraspinal cysts, are difficult to differentiate from lumbar disc herniation based on clinical symptoms.

Several types of intraspinal cysts with different pathogeneses have been described in the past, including perineural cysts arising from the myelin sheath,\(^14\) synovial cysts originating from the facet joint (which may be caused by mucoid degeneration of the connective tissues due to repetitive dynamic loads exerted on the facet joint capsule or the posterior longitudinal ligament),\(^10\) and arachnoid cysts of mainly congenital origin.\(^8\) Various additional cysts, such as those caused by gas produced in an extensively degenerated disc,\(^9\) ligamentum flavum cysts,\(^2\) lumbar epidural varices,\(^10\) and premembranous hematomas,\(^9\) have been discussed as well. Our patient presented with an intraspinal extradural cyst that was contiguous with the peridural membrane and extended into the adjacent vertebral body, and fluoroscopy-guided contrast injection revealed a communication between the vertebral body and the cyst (Fig. 3). The cyst contained fibrin and red blood cells without a synovial lining; histopathology...
of the intraspinal cyst demonstrated a cell lining negative on cytokeratin immunostaining (Fig. 6C). These features are contrary to those of other cystic lesions in the spine. Wiltse et al. described the “peridural membrane” as a fibrous membrane lying between the posterior surface of vertebral bodies and the posterior longitudinal ligament. This membrane is approximately one-half the toughness of the dura mater and is not attached to the posterior longitudinal ligament, except at the disc space, where it blends with the annulus fibrosus. The veins of the Batson plexus lie on the dorsal surface of this peridural membrane and perforate through the membrane at multiple locations to lie ventral to the membrane and the vertebral body. Although an epidural hematoma has been considered to originate from the arterial system, bleeding from tearing of the fragile epidural veins lying within the premembranous space, possibly due to an abrupt change in venous pressure after blunt trauma, is the most commonly accepted source. In reporting a strong coincidence between a premembranous epidural hematoma and an underlying disc disruption (annular tear or herniation), Gundry and Heithoff postulated that spontaneous epidural hematomas resulted from tearing of fragile epidural veins lying adjacent to the displaced annulus or nucleus. Chiba et al. mentioned that an unknown mechanism, such as mucous degeneration at the surface of a hematoma, may disrupt absorption of a hematoma and produce a cyst. However, in our case, no disc herniation or any disc abnormality was detected, and the intraspinal extradural cyst causing cord compression did not communicate with the intervertebral disc (Fig. 3).

Although multiple pathogeneses may be considered as possible causes of intraspinal cysts, 2 hypotheses for the formation of the cyst in our patient are suggested. Posttraumatic epidural hematomas are rarely associated with spine fractures, with an incidence ranging from 0.5% to 7.5%, and the hematoma formed by cancellous bone bleeding from a fractured vertebral body may

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**Fig. 5.** The patient underwent posterior L1 vertebral column resection and posterior instrumentation of T11–L2. Plain radiograph obtained 1 week after surgery (A), showing good kyphosis correction. At 2 years after surgery (B), the kyphotic angle was −14.1°, and the sagittal positive imbalance improved to 28 mm. Magnetic resonance images (C) obtained 2 years after surgery showed no recurrence. POD = postoperative day.

**Fig. 6.** A: Photomicrograph showing a multilocular cystic lesion containing fibrin and red blood cells. The cystic wall was composed of fibrous connective tissue. H & E, original magnification ×12.5. B: The cystic wall was lined by 1–2 layers of flattened cells and supported by a capsule of well-differentiated fibrous connective tissue. H & E, original magnification ×400. C: Immunohistochemical analysis revealed negative reactivity of the cell lining. Cytokeratin stain, original magnification ×400.
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be located on the ventral side of the epidural space. Such posttraumatic epidural hematomas may be resorbed and organized to form a cyst and develop spinal cord compression as an intraspinal space-occupying lesion.

On the other hand, such a cyst may originate from a vertebral body, the mechanism of which involves a load imposed on the fracture body and its high internal pressure. The load is transmitted through a crack of the frac-
tured vertebral body and exerts kinetic stresses on the intraspinal tissues, such as the posterior longitudinal ligament and the peridural membrane. There is a possibility that this load may play a role in the development of the cyst.

Conclusions

In summary, the intraspinal cyst communicating with the vertebral body occurred during a compression fracture and compressed spinal cord. An intraspinal cyst can occur during a compression fracture, and such an intraspinal space-occupying lesion may cause neurological deficits. Thus, when neurology is observed after the diagnosis of a compression fracture, additional precise tests, including MRI, are necessary even if abnormal findings are not found on plain radiographs. When a definite lesion is observed on MRI, physicians must be careful, as the case may require surgical treatment.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following: Conception and design: all authors. Critically revising the article: all authors. Reviewed submitted version of manuscript: all authors. Approved the final version of the manuscript on behalf of all authors: JH Lee.

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