Spinal extradural meningeal cyst

Case report

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The case of a 30-year-old man with a spinal extradural meningeal cyst in the thoracolumbar region is reported. Operative findings revealed a dural defect that allowed communication between the extradural cyst cavity and the subarachnoid space. Application of the Valsalva maneuver made the cerebrospinal fluid flow into the cyst cavity; however, reverse flow did not occur. These findings indicate that a valvelike mechanism developed in the enlarging cyst. Surgical resection of the cyst wall and closure of the dural defect provided a favorable result.

KEY WORDS • spinal cyst • extradural cyst • meningeal cyst • etiology

Spinal extradural meningeal cysts are relatively uncommon1–3,11 and have been described as “arachnoid cysts,” “pouches,” and “diverticula.”6,8,11 Although their classification has proved to be somewhat complicated, Nabors, et al.7 have simplified the classification of spinal meningeal cysts into three major categories: extradural cysts without nerve root fibers (Type I); extradural cysts with nerve root fibers (Type II); and intradural cysts (Type III).

Spinal extradural meningeal cysts are most often located in the mid- to lower thoracic area,1–3,11 are found predominantly in males,1,3,11 and tend to be symptomatic during the second decade of the patient’s life.1,3,11 Patients usually present clinically with progressive spastic or flaccid para- or quadriparesis.1–3,11 Approximately 10% of the patients present with monoparesis.1 Sensory deficits are less prominent.11 The clinical symptoms develop over months,2 although partial relief may occur in more than one-third of the patients.3 In some cases, some cases are associated with a long-term remission extending for years.10 Among these cases, reciprocal obstruction and recanalization are thought to mediate remission and relapse.5,10

Nontraumatic spinal extradural meningeal cysts are believed to be congenital.1–3,7,8 Proposed causes of cyst expansion are active secretion from the internal cell lining,2 an osmotic spinal gradient between the subarachnoid space and cyst,3,5 pulsatile cerebrospinal fluid (CSF) dynamics,6,8 and valvelike mechanisms.1,3,4,6,7,9 However, active secretion by the inner cell lining has never been demonstrated and this hypothesis has been discredited.1,3 Here, we present a case of a Type I meningeal cyst that appears to support the hypothesis of a valvelike mechanism as the cause of cyst enlargement.

Case Report

This 30-year-old man was referred to our clinic with a 6-month history of progressive right leg weakness. Six years prior to presentation, he had experienced intermittent weakness of both legs with spontaneous remission. He had no other prior illness or trauma except for a lumbar bruise that occurred when he was 14 years old.

Examination. On examination, the patient had no evidence of phacomatosis. Neurologically, the muscle strength of his right leg was reduced slightly to Grade 4+/5. There was moderate atrophy of his right leg. Deep-tendon reflexes of the legs were increased. Clonus was present at the right patella and ankle. The plantar response was extensor on the right side. Hypoesthesia was noted in a small midthigh area bilaterally and on the dorsal side of the left foot. Bowel and bladder functions were normal.

Laboratory and Radiological Examinations. Routine laboratory tests yielded normal results. Thoracolumbar vertebral roentgenograms revealed an enlarged interpedicular space. Magnetic resonance (MR) imaging demonstrated an extradural cystic lesion extending from the T10–L2 level. This lesion was visualized best as a hyperintense T2-weighted signal associated with lateral pouches (Fig. 1). It compressed the thecal sac and spinal cord to the ventral
Side. The results of a nerve conduction study were normal. Electromyography and a sensory evoked potential study suggested myelopathy. A myelogram disclosed a complete block at the level of L-1. However, a delayed computerized tomography (CT) myelogram showed the communication between the spinal subarachnoid space and the cyst cavity at 3 hours (Fig. 2).

Operation. The cystic lesion was exposed via an en bloc laminotomy extending from T10–L1, and a partial laminectomy of T-9 and L-2 laminae was performed. The cyst wall was white, fibrous, and tense. Following removal of CSF-like fluid by cyst puncture, the cyst reexpanded within a few minutes. The dorsal wall was opened vertically without any bleeding to explore the intracystic cavity. There was a small dural defect adjacent to the right T-10 nerve root. During a forced inflation of the patient’s lungs by the anesthetist using the Valsalva maneuver, the CSF flowed into the cyst cavity via the defect; however, afterward reflux was not observed. The dorsal nerve rootlets herniated into the cyst after the maneuver and acted as a valvelike apparatus. No other defects were noted. The dural defect was closed with a purse-string suture using No. 4–0 nylon. The ventral wall of the cyst could be separated easily from the theca. The lateral pouches were not removed because there was bleeding from the paravertebral venous plexus. The wound was closed in a watertight fashion following the laminoplasty.

Postoperative Course. The patient’s postoperative course was uneventful with no neurological complications.

Pathological Findings. Histopathological study of the cyst wall revealed that it was composed of fibrocollagenous tissue with scattered meningotheelial cells and a focal infiltration of inflammatory cells. There was an inner single-cell lining (Fig. 3). The findings were compatible with that of an arachnoid cyst.

Discussion

Both active and passive fluid-transport mechanisms have been hypothesized to explain enlargement of extradural meningeal cysts. Histopathological findings often fail to reveal an inner single-cell lining of extradural meningeal cysts, and cells with secretory capability frequently are absent. Most investigators now prefer the passive fluid-transport theory to explain the etiology of cyst expansion via pulsatile CSF dynamics and an osmotic gradient with or without valvelike mechanisms. In the early stages, pulsatile CSF dynamics may act as a promoting factor to enlarge the cyst; an osmotic gradient can then facilitate further expansion.

![Fig. 1. Left: A T1-weighted magnetic resonance (MR) image of the thoracolumbar spine showing a hyperintense spinal cord signal and compression of the cord posteriorly by the extrameningeal cyst (arrow). Right: A T1-weighted MR image of a coronal section of the spinal cord clearly demonstrating lateral pouches.](image1)

![Fig. 2. Axial computerized tomography myelogram revealing communication between the cyst and the subarachnoid space.](image2)

![Fig. 3. Photomicrograph of the cyst wall demonstrating a fibrocollagenous layer with an inner single-cell lining. H & E, original magnification × 50.](image3)
Myelography or operative findings demonstrate communication between extradural meningeal cysts and the subarachnoid space in approximately half of the cases. However, a recent study suggests that a communication exists in nearly all cases of meningeal cysts. Histopathologically, the cyst wall has been reported to consist of fibrous connective tissue with or without an inner single-cell lining, which may represent an arachnoid membrane. Long-standing elevation of the osmotic gradient cannot be created without production or active transport of protein or electrolytes into the cyst cavity. However, the question of active secretion and absorption of fluid by the wall of the cyst has not yet been resolved. Many reports have supported a valvelike mechanism to explain continuous enlargement of the cyst. This mechanism would make intracystic CSF pressure greater than normal hydrostatic pressure.

In our case, the cyst deflated immediately after intraoperative puncture but expanded within a few minutes. Our direct inspection of the inner surface of the cyst confirmed that the influx of CSF through the dural defect followed forced inflation of the lungs. Although an osmotic pressure gradient may be partly responsible for cyst enlargement, intraoperative findings strongly supported a valvelike mechanism as reported by Rohrer, et al.

Magnetic resonance imaging is a useful tool for diagnosing intraspinal meningeal cysts. It provided clear anatomical information and demonstration of pathological change in our case. The hyperintense lesion located in the spinal cord was caused by long-standing spinal cord compression by the cyst. Although the prognosis of extradural meningeal cysts is favorable, only one-third of patients attain remission with complete recovery. The persistent T2 signal showing cord hyperintensity in our patient emphasizes the potential in this case for a permanent subclinical lesion.

In previous reports, resection of the cyst wall from the posterior aspect of the thecal sac and closure of the dural defect was performed to eradicate the valvelike mechanism. Because the remaining cystic wall cannot promote recurrence, total resection is unnecessary if the posterior wall of the theca has been explored and the dural rent has been obstructed as was the case with our patient.

References


Manuscript received January 25, 1996.
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