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Case Report

Progressive growth of arachnoid cysts with cauda equina syndrome after lumbar spine surgery

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Abstract

Intradural arachnoid cysts are a rare cause of spinal cord compression. In symptomatic cases neuropathic pain, gait disturbance, and paraparesis or quadriparesis are often present. Postoperative arachnoid cysts have rarely been reported. We describe a 56-year-old male who developed progressively enlarging arachnoid cysts with cauda equina syndrome and vertebral body erosion after lumbar surgery. The clinical presentation of the patient, the possible mechanisms of cyst formation, and the management of the disease are discussed with regard to previous literature.

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1. Introduction

Intradural arachnoid cysts are a rare cause of spinal cord compression.¹⁻³ These cystic lesions are located in the middle to lower thoracic spinal cord in 80% of patients and lie mostly dorsal to the spinal cord.⁴⁻⁶ They rarely occur in the lower lumbar or sacral areas. The pathogenesis of the cysts remains obscure. Proposed etiologies include a congenital origin, trauma, or inflammation.⁷ Intradural spinal arachnoid cysts reportedly result from alterations in the arachnoid trabeculae that may be attributable to previous trauma or arachnoiditis, but are mainly idiopathic in origin. Other rare causes may be postoperative complications, spinal procedures such as lumbar myelography, meningitis with adhesions, and epidural hematoma.^{8,9} There are several clinical manifestations when an intradural arachnoid cyst becomes symptomatic.⁸ Neuropathic

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pain, gait disturbance, and paraparesis or quadriparesis are the most common complaints and clearly decrease the quality of life in patients. We report an unusual case of progressively enlarging arachnoid cysts of the lumbosacral spine that presented as cauda equina syndrome after surgery to the lumbar spine.

2. Case report

Approximately 30 years prior to presenting at our hospital Taipei Veterans General Hospital (Taipei, Taiwan), a 56-yearold male had undergone discectomy and posterior decompression with laminectomy for a herniated intervertebral disc at the L4–S1 level at another institute. He suffered from low back pain that began four years previously. As the disease progressed, he developed sciatica with numbness of the left leg. He tried Chinese manipulation and traditional therapy without improvement and then came to our outpatient clinic for assistance. Paresthesia was noted at the left L4 dermatome. His deep tendon reflex was normal at the lower limbs. The patient was able to walk without any assistance.

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Computed tomography (CT) revealed a status postlaminectomy at the L4–L5 level, an L4 pars fracture with grade I lytic spondylolisthesis of L4 on L5, and L4–L5 facet joint hypertrophy with lateral recess stenosis. In addition, a cystic lesion was present in the canal at the L5–S1 level (Fig. 1). Because the patient's symptoms were on the left side, we performed L4–L5 radiculopathy, L4–L5 decompression, instrumentation with pedicle screws, and posterolateral fusion. The intraoperative finding was an expansile dural sac with severe adhesion between the bone and the dura mater. Decompression was performed carefully through the interface of the bone and the dural sac. Because the dural sac lacks elasticity, duroplasty was not performed. The surgery was uneventful, and the patient was discharged 8 days later.

During the postoperative follow-up period, some degree of sciatica persisted on the left side. Three months after the surgery, magnetic resonance imaging (MRI) revealed an expansile irregular dural sac extending from the L3 to S2 levels and showed abnormal enhancement on the surface of the dural sac and in the thecal sac. Arachnoiditis with arachnoid cyst formation was present (Fig. 2). The serum level of C-reactive protein was within normal limits. A conservative course of treatment such as physical therapy was indicated. The patient's walking status was normal, and he went hiking every day as part of his rehabilitation program.

Three years after the surgery, the patient developed right lower leg numbness and sphincter dysfunction with urination and bowel movements. The motor function in his lower extremities was not disturbed and he still hiked daily, although he complained of urine incontinence and impotence. A neurologic examination revealed paresthesia on the right L5 dermatome and perineum region, and decreased ankle reflex on the right side. The lower extremity muscle power was full (5/5). The patient had neither low back pain nor left leg pain. The follow-up radiographs showed solid fusion of the L4-L5 vertebrae with normal implant position (Fig. 3). MRI showed a progressive enlargement of lobulated arachnoid cysts at levels L3 to S2. Nearly one-half of the L4 and L5 vertebral bodies were eroded by the cysts, and the cauda equine was tightly adhered to the cysts (Fig. 4). Surgical interventions such as cyst aspiration or a shunting procedure were considered. The sequelae of leaving the cysts without further intervention were explained to the patient. However, the patient refused further treatment.

3. Discussion

Intradural arachnoid cysts can be defined as spaceoccupying lesions containing fluid that is similar to cerebrospinal fluid and consisting of membranous arachnoid matter.¹⁰



Fig. 1. (A) Preoperative computed tomography reveals a L4 pars fracture with grade I lytic spondylolisthesis of L4 on L5. (B) A cystic lesion is in the canal at the L5–S1 level.

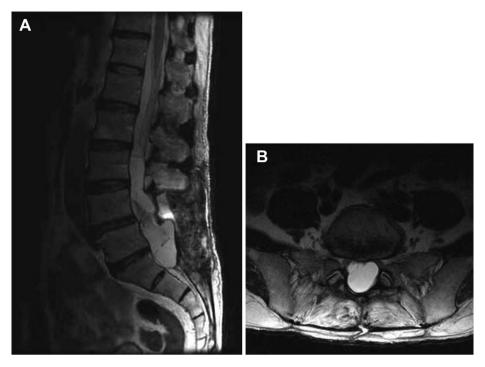


Fig. 2. Postoperative magnetic resonance imaging at 3 months. (A) The arachnoid cysts extend from levels L3 to S2. (B) Axial view of the L5 vertebrae.



Fig. 3. Postoperative plain radiographs at 3 years show solid fusion and proper implant position. (A) Anteroposterior view. (B) Lateral view.

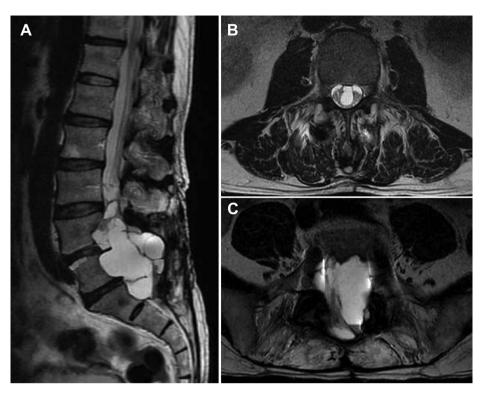


Fig. 4. Postoperative magnetic resonance imaging at 3 months. (A) The progression of multiple lobulated arachnoid cysts and bony erosion of the L4 and L5 vertebral bodies. (B) Axial view at the L3 level. (C) Axial view at the L5 level shows cysts extending to the muscle layer and severe vertebral body erosion.

These cysts are typically situated on the dorsal side of the middle to lower thoracic spinal cord.^{5,6,11,12} According to the statistics, dorsal cysts on average extend the length of 3.7 vertebral bodies. Typical intradural arachnoid cysts present in adolescence or early adulthood.⁵ Spinal intradural arachnoid cysts, which cause neurologic symptoms, rarely appear after spinal surgery. Valls et al¹³ report a patient who underwent lumbar laminectomies at the L2 to L5 levels. Although the procedure was uneventful, intradural arachnoid cysts occurred 36 hours later at the T4 to T10 levels. Ford reports five cases of arachnoid cysts that occurred after dural tears during lumbar disc surgery.¹⁴ Nottmeier et al¹⁵ also report two cases of arachnoid cysts occurring as a complication of dural tearing during lumbar laminectomy. In the present case, it is unknown if a dural tear occurred during the lumbar surgery he had undergone 30 years earlier. Preoperative CT showed an arachnoid cyst at the L5-S1 levels. A dural tear complication could presumably cause an arachnoid cyst.

The most common clinical manifestations of symptomatic arachnoid cysts are neuropathic pain, paraparesis, hypoesthesia or dysesthesia, and occasionally incontinence.¹⁶ Rare manifestations include headache as a result of cervical cysts and angina as a result of thoracic cysts.¹ Paraparesis and severe gait disturbances may lead to falling easily. Spinal cord compression induces nearly all clinical symptoms, including weakness.⁴

The arachnoid cysts in the present patient were located in the lumbosacral spine and caused symptoms that affected bladder, bowel, and sexual functions. The patient complained of paresthesia over the right lower limb, although he did not exhibit any muscle weakness and was able to go hiking every day. Even with MRI, the relationship between the nerve root and the arachnoid cyst could not be identified clearly. The main lesion was presumably the tight adhesion of the S2–S4 root to the dural sac. His motor function was not involved.

The exact mechanisms leading to the formation of intradural arachnoid cysts remain unknown. Possible mechanisms in adults can be classified into five categories,^{1,4,10,12,17} as follows: (1) congenital origin; (2) arachnoid adhesions, followed by viral, spirochetes, or bacterial inflammation; (3) arachnoiditis resulting from a subarachnoid hemorrhage, contrast media, spinal anesthetics, meningitis, fibrin glue, or bone dust; (4) trauma to the vertebral column resulting from a diagnostic lumbar puncture, spinal anesthesia, or intradural surgery; and (5) idiopathic origin.

Hypothetical explanations regarding the mechanism of cyst enlargement have been proposed, including secretions from cells on the cystic wall,^{1,10} the presence of a unidirectional valve,^{1,13} and a diverticulum resulting from the pathological distribution of the arachnoid trabeculae.^{1,18} Some scholars speculate that degeneration of trabecular cells increase the osmotic effect of the cysts; this then stimulates fluid movement into the cells, thereby causing enlargement. The formation of the cysts leads to the abnormal flow of cerebrospinal fluids, which engorge the epidural vein and enlarge the cysts, or decrease the cerebrospinal fluid buffer between the spinal cord and the cysts. All of these factors could contribute to spinal cord compression.

In the present patient, the arachnoid cyst enlarged rapidly after the second surgery and exacerbated the neurologic symptoms of the cauda equine syndrome. We believe that a unidirectional valve may have caused enlargement of the cystic lesion. The second spinal surgery may have further stimulated the inflammatory course, leading to an aberrant flow of cerebrospinal fluid. The fluid and pressure may have accumulated in the cyst and compressed the cauda equina. An alternative explanation is that inflammation inside the cyst may have induced adhesion to the cauda equina and stretched the nerve root. Either mechanism could have caused the clinical symptoms to progress. The pressure and inflammation of the cystic lesion may have led to vertebral body erosion-a condition that is rarely reported in the literature. MRI showed that nearly one-half of the L4 and L5 vertebral bodies had been destroyed. We predict that complete vertebral body destruction could occur in the near future if the condition were not treated.

Treatment options for intradural arachnoid cysts include surgical resection, fenestration of the cyst wall, and percutaneous drainage or shunting to the peritoneum, the atrium, or the pleura.^{6,12} Many authors recommend the total removal of the cyst as the preferred treatment.⁴ Preoperative aspiration under CT or MRI guidance to identify the communication between the cyst and subarachnoid space has also been suggested.¹⁹ However, if symptoms recur after aspiration, communication is likely to still exist. For removing a dorsal cvst, a posterior approach with laminectomy has been suggested. If ventral cysts are present, a fenestration can be made by posterolateral laminectomy. In patients with multiple cysts that extend over many vertebral levels, laminectomies at all levels are not appropriate because this can affect stability. In this situation, a limited excision of the cyst at the levels of maximum pressure and the fenestration of smaller cysts may be considered.⁴ The outcomes of cyst excision are excellent. Cyst-to-peritoneum or atrium shunting is an additional option.²⁰ A simple needle aspiration and drainage has reportedly been used in patients with cysts that cannot be resected.¹⁹ In the present patient, the cysts were lobulated at the lumbosacral levels and the cysts were strongly adhered to the cauda equina. If cystic resection had been performed, it may have caused irreversible nerve injury. Therefore, we recommend a shunting procedure or simple needle aspiration with drainage as the optimal treatment option for this patient.

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