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Case Report

Extradural spinal cyst in a pediatric patient: A case report

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ABSTRACT

Background: Spinal extradural arachnoid cysts comprise <1% of all spinal lesions and are rare findings in pediatric patients. The pathogenesis of spinal extradural arachnoid cysts is not well known but is thought to most commonly be due to congenital dural defects. Other origins include trauma, inflammation, or infection, such as arachnoiditis. Spinal magnetic resonance imaging is the gold standard for diagnosis, showing a fluid-filled space dorsal to the spinal cord with signal intensity akin to cerebrospinal fluid (CSF) and often the site of dural defect with CSF leak. While most spinal extradural arachnoid cysts are asymptomatic, large cysts can compress the spinal cord or nerve roots, leading to myelopathy, radiculopathy, or focal pain symptoms. In such cases, surgical management is indicated.

Case Description: Here, we present a case of a 15-year-old female who presented with lower back pain radiating to her bilateral posterior thighs and knees, with imaging indicating a thoracolumbar spinal extradural arachnoid cyst. After failed conservative treatment, surgical intervention in the form of laminectomy, fenestration of the arachnoid cyst, and repair of the dural defect was required, resolving the patient's symptoms with no recurrence of the cyst.

Conclusion: Complete resolution of pain in our patient following surgical management of spinal arachnoid cyst suggests that treatment of the arachnoid cyst can be achieved through minimal exposure to the site of the CSF leak to fenestrate the cyst and repair the leak.

Keywords: Case report, Dural defect, Laminectomy, Spinal arachnoid cyst

INTRODUCTION

Spinal arachnoid cysts are rare lesions of the spinal cord. They comprise only about 1–3% of all spinal masses.[13,19] Along the length of the spinal cord, spinal arachnoid cysts could be intradural, extradural, or perineural in location.[8] Two studies that analyzed 35 patients with spinal arachnoid cysts demonstrated that intradural arachnoid cysts are more common than extradural arachnoid cysts, with the prevalence of each ranging between 50–54% and 27–38%, respectively.[2,4,5] In fact, extradural spinal cysts encompass <1% of all spinal epidural lesions.[2]

Spinal extradural arachnoid cysts (SEACs) consist of fluid-filled spaces within protrusions of arachnoid through a dural defect.[2,9] Most SEACs communicate with the underlying subarachnoid space such that the cyst is composed of cerebrospinal fluid (CSF), although some

cases have reported no communication between the cyst and subarachnoid space.[2,19]

Spinal extradural arachnoid cysts most commonly present within the 2nd–5th decades of life and are especially rare in the pediatric population.[2,18,19] Thoracic extradural cysts mainly appear in the adolescent population, while lumbar and sacral cysts are seen mostly in adults.[9,17] While a majority of SEACs are asymptomatic, compression of the spinal cord and nerve roots can lead to a variety of neurologic symptoms ranging from myelopathy to focal manifestations.[2,3,19] In such cases, surgical management is often indicated.

Here, we present a case of a 15-year-old female with symptoms of lower back pain, bilateral proximal leg pain and numbness as a result of spinal cord and nerve root compression by a spinal extradural arachnoid cyst.

CASE DESCRIPTION

A 15-year-old female presented with three months of lower back pain radiating into her bilateral posterior thighs and knees. She had no history of trauma, infection, connective tissue disease, or previous spine condition. She had no weakness in her upper and lower extremities, bowel or bladder incontinence, saddle anesthesia, or difficulty ambulating. Twice daily ibuprofen and physical therapy for two months did not improve or resolve her symptoms. Thoracic and lumbar spine magnetic resonance imaging (MRI) demonstrated a large dorsal extradural meningeal cyst at T11–L2 with associated neural foraminal extension and bony expansion [Figure 1]. Imaging also showed ventral displacement and compression of the distal spinal cord and nerve roots of the cauda equina. On imaging, there is a flow void noted at L1 on the left side, thought to be the site of a dural defect leading to the cyst [Figure 2]. The patient's bilateral lower extremity symptoms were due to the extension of the large cyst into the bilateral neuroforamina from T11 to L2 and associated ventral compression and displacement of the distal spinal cord and cauda equina nerve roots.

Instead of a traditional approach with multi-level laminectomy and given the identification of the likely site of the dural defect at L1, a minimally invasive approach in the form of L1–L2 laminectomy, fenestration of the arachnoid cyst, and L1 dural defect repair was selected to reduce the potential morbidity associated with a more extensive approach. Intraoperatively, the extradural cyst wall was located dorsally [Figure 3]. A microdissection of the cyst was completed. A portion of the cyst wall was bipolared, cut and removed and sent to pathology for the permanent specimen. A dural defect at the L1 nerve root was confirmed [Figure 4]. Primary repair was not possible

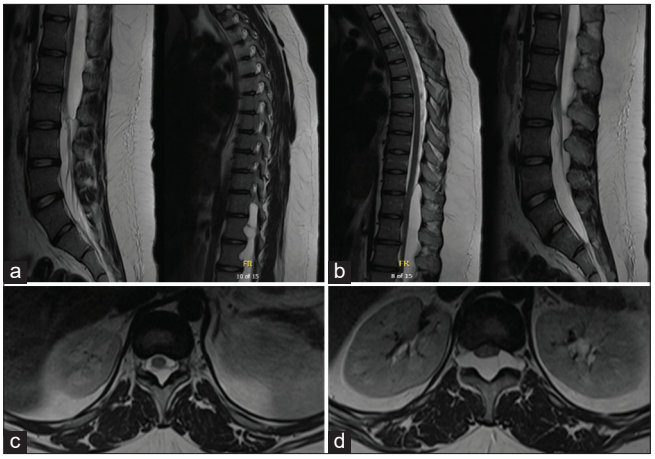

Figure 1: (a) Preoperative T2-weighted parasagittal thoracic and lumbar magnetic resonance imaging (MRI). (b) Preoperative T2-weighted midsagittal thoracic and lumbar MRI. (c) Preoperative T2-weighted axial MRI T11–T12. (d) Preoperative T2-weighted axial MRI L1.

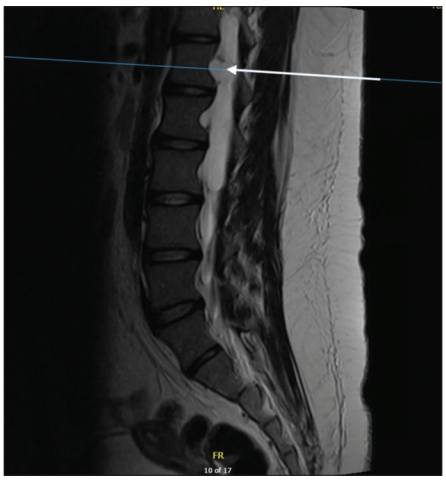

Figure 2: Preoperative T2-weighted sagittal lumbar magnetic resonance imaging, arrow pointing at L1 dural defect.

as the dural edges were too far apart, and the dural opening was near the axilla of the nerve root. The secondary repair was completed by sealing the dural defect with a fibrin sealant, harvesting the fascial graft, and adding another layer of fibrin sealant. Valsalva demonstrated no evidence of a CSF leak, and the incision was closed in standard fashion. Histological findings of the spinal cyst wall showed benign fibrovascular tissue, consistent with a spinal arachnoid cyst.

The patient had an uneventful postoperative period. Repeat imaging four months postoperatively demonstrated no residual extradural meningeal cyst [Figure 5]. The patient's lower back pain, bilateral thigh pain, and paresthesias completely resolved.

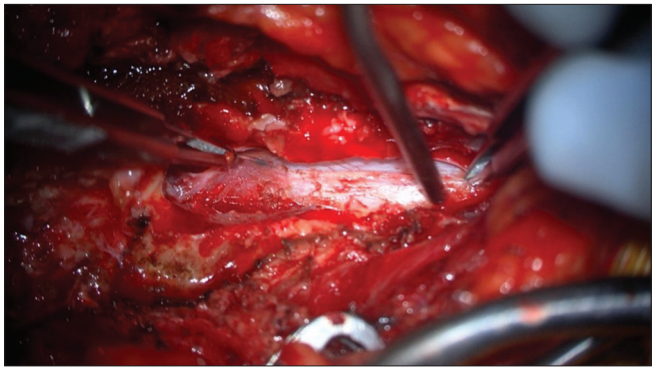

Figure 3: Extradural cyst.

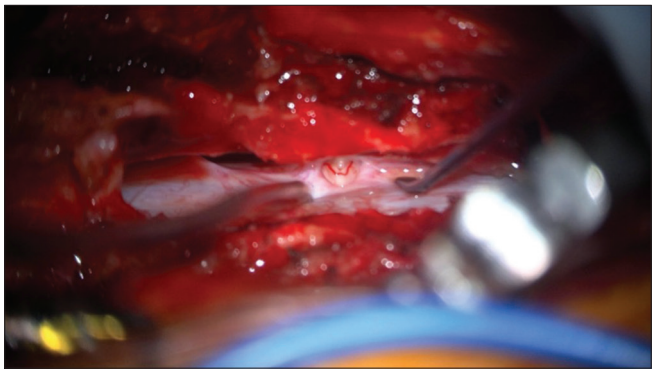

Figure 4: Nerve root dural defect.

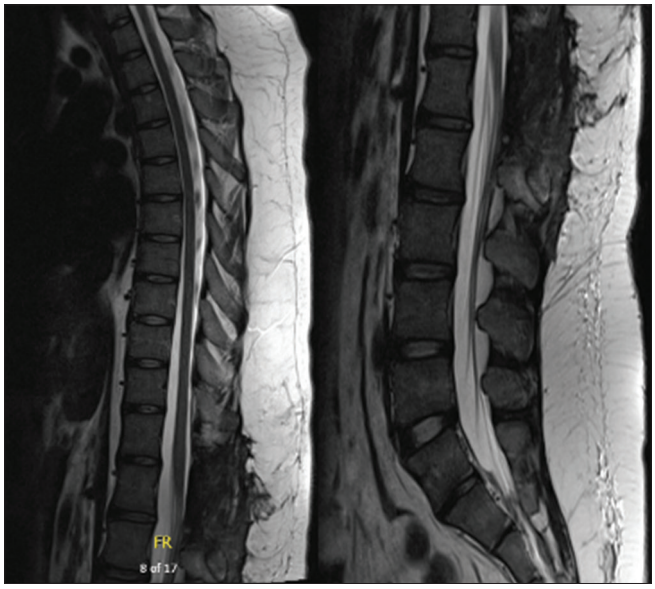

Figure 5: Postoperative T2-weighted midsagittal thoracic and lumbar magnetic resonance imaging.

DISCUSSION

Nabors *et al*.[10] categorized spinal arachnoid cysts into three groups: spinal extradural meningeal cysts without nerve root fibers (type I), spinal extradural meningeal cysts with nerve root fibers (type II), and spinal intradural meningeal cysts (type III). Type I cysts are further classified into spinal extradural arachnoid cysts (type IA) and occult sacral meningocele (type IB).[9,12] Type IA lesions are rare subtypes, as described in this case. A majority of spinal extradural arachnoid cysts occur within the thoracic region of the spinal cord (65%) and are less likely in the lumbar and lumbosacral (13%), thoracolumbar (12%), sacral (7%), and cervical regions (3%).[6,8] Lesions are more commonly located dorsally resulting in displacement of the spinal cord anteriorly.[3,8,13,19] As in this case, SEACs commonly extend over several spinal segments, ranging between 2 and 13, with a mean of 5.04 vertebrae.[2,9]

The pathogenesis of spinal arachnoid cysts is not well understood. Congenital dural defects are the most probable cause of SEAC, and identified small dural tears are often congenital in origin.[2,19] Dural defects are most commonly located within the dural sleeve of the nerve root or at the dural sleeve and thecal sac junction. Less often, dural tears can be present at the dorsal midline of the involved thecal sac.[9] SEACs communicate with the underlying subarachnoid space through a pedicle at these regions.[13] Possible etiologies of SEACs include both congenital and non-congenital causes, such as trauma, infection, or inflammation that increases CSF flow and leads to herniation of the arachnoid membrane.[9] As our patient did not present with any history of trauma or illness, she most likely had a congenital form of the lesion. SEAC could also result from mechanical stress on the spine due to diffuse idiopathic skeletal hyperostosis, wedge deformity of the vertebral body, or disc herniation.[2]

SEACs are usually asymptomatic.[2] When SEACs enlarge, thought to be due to intermittent pressure surges within the subarachnoid space, they can lead to compression of the spinal cord or nerve roots, resulting in radiculopathic or myelopathic findings or focal pain and weakness.[2,19] Presentations of motor weakness are more common than sensory findings, and the most common symptoms include back pain and leg weakness.[8,9] In pediatric patients, symptoms vary between back pain, sensory changes, and weakness with muscle atrophy or paraparesis to quadriplegia, saddle anesthesia, spasticity, and gait abnormalities. Bowel and bladder dysfunction more commonly occur in spinal lesions of the lumbar and sacral regions.[3]

The gold standard for diagnosis of SEAC is spinal MRI without contrast. Spinal MRI is useful for localizing the lesion, identifying the extent of the lesion, and demonstrating its relation to the dura and nerve root.[18] SEACs appear hypointense in T1 imaging and hyperintense in T2 imaging, akin to the signal enhancement of CSF, and do not enhance with gadolinium.[2,9,17] MRI imaging can also identify contour changes in the spinal cord that demonstrate spinal cord displacement at the site of the lesion.[5] In some cases,

MRI alone may not show cystic communication with the subarachnoid space; therefore, MRI flow studies can be done to detect the site of CSF leakage.[2] Computed tomography myelography is another radiographic imaging that can detect the site of a dural defect with underlying subarachnoid fluid communication, especially in instances of MRI contraindications.[2,9] Other findings on radiologic imaging include osseous changes of the spine, such as widening of the interpedicular distance, erosion and scalloping of the posterior vertebra, foraminal enlargements, widening of the spinal canal, and kyphoscoliosis due to long-standing hydrostatic pressure within the cyst.[9,12] Similarly, our patient presented with foraminal and bony expansion due to the chronic mass effect of an enlarged cyst.

Asymptomatic patients may not require treatment, and only long-term observation is indicated.[8] In the case of large symptomatic SEACs, surgical treatment is indicated, although the type of surgical management varies.[2,3,7,16] The surgical approach in most cases of SEAC removal is laminectomy or laminoplasty at the site of the lesion, as a majority of SEACs are posterior in location. Watertight closure of the dural communication follows to prevent refilling of the cyst.[14] Some authors have argued that complete cyst excision with removal of the cyst wall is necessary to prevent recurrence; however, this remains controversial.[2,3,7] Lee *et al*. proposed that the incidence of cyst recurrence is related rather to repair of the dural defect as opposed to cyst wall excision, demonstrating a 2% recurrence rate in patients with repaired dural defects compared to 10% in patients with completely excised cysts in a retrospective study consisting of 52 patients. Further, large SEACs that extend over multiple vertebrae would require extensive laminectomy for total cyst removal, increasing the risk for complications such as kyphoscoliosis, bleeding, muscle damage, and spinal misalignment.[6,7,11] Instead, selective laminectomy with cyst fenestration and repair of the dural defect has been shown to be an alternative to total cyst excision in large SEACs with good outcomes.[1,6,16,19] Our patient had a cyst extending from T11 to L2, which would have required huge exposure and multi-level laminectomy for total cyst excision and high-risk surgery with the potential for significant postoperative complications. Our approach for selective laminectomy, cyst fenestration, and secondary repair of the dural defect was effective at relieving this patient's symptoms without recurrence. Localization of the dural defect before surgery, in this case, detected as a flow void on imaging, would guide this approach.[11,19] Non-surgical management with cyst aspiration is not an effective long-term treatment, as it only allows for temporary improvement in symptoms and can lead to higher rates of recurrence.[12,13,18] Similarly, shunt placements, including cystopleural, cystoperitoneal, and cystosubarachnoid shunts, have been reported as failed attempts for the treatment of SEAC.[13]

Histological findings of resected SEAC walls do not have defining features for diagnosis. Characteristic findings include collagenous fibers or fibrous connective tissue with an inner single-cell lining.[2,9] This flat, inner cell lining is representative of arachnoid tissue and may or may not be present in histology.[14] In our patient, only fibrovascular tissue was identified within the cyst wall.

Our case presentation demonstrates the treatment of SEAC in a patient who presented only with symptoms of pain in the absence of other neurologic deficits. Conservative measures, including pain medication and physical therapy, failed to improve or resolve this patient's symptoms. Rather, surgical fenestration of the cyst with dural repair resulted in a favorable outcome with the resolution of pain and no recurrence of the cyst. We propose treatment of SEAC with surgical management through this less invasive approach, even before the presentation of neurologic deficits, as opposed to waiting, mainly to provide this young patient with symptomatic relief. Previous studies have indicated that surgery in patients who have a more chronic course of SEAC results in less favorable outcomes and that earlier surgical repair demonstrates better clinical outcomes.[6,15] Therefore, surgically treating SEAC even before conservative treatment may yield an improved clinic course.

CONCLUSION

Spinal arachnoid extradural cysts are rare lesions of the spinal cord that can result in motor, sensory, and bowel or bladder dysfunction due to spinal cord or nerve root compression. This case presents a rare finding in a pediatric patient with complete resolution of symptoms following cyst fenestration, partial resection of cyst wall, and dural repair of nerve root defect. Most cases are a result of congenital lesions and pressure changes in the subarachnoid space that drive fluid into the cystic cavity, which is the most accepted mechanism of cystic enlargement. Neuroimaging is diagnostic for spinal arachnoid cysts, and surgical management is standard for symptomatic treatment and prevention of recurrence. Histological findings demonstrate fibrous connective tissue with or without an inner single-cell arachnoid lining. We demonstrate the resolution of pain symptoms in our case presentation, suggesting that surgical intervention using our minimal approach should be pursued even in the absence of neurological deficit, resulting in the resolution of the cyst and improvement in symptoms.

Ethical approval

The Institutional Review Board approval is not required.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

Use of artificial intelligence (AI)-assisted technology for manuscript preparation

The authors confirm that there was no use of artificial intelligence (AI)-assisted technology for assisting in the writing or editing of the manuscript and no images were manipulated using AI.

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