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Case Report

# Spinal Epidural Arachnoid Cyst: Case Report and Literature Review

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## ABSTRACT

**Background:** Spinal epidural arachnoid cysts (SEACs) are rare, accounting for ~1% of all spinal tumors. While often incidental, large cysts may compress nerve roots or the spinal cord, leading to motor or sensory deficits. The etiology and pathophysiology of these conditions remain unclear, and no standardized management guidelines exist. Surgical treatment is indicated for symptomatic cases, with dural defect repair essential to prevent recurrence.

**Case Description:** A 25-year-old female presented with paraparesis and intermittent leg pain of one month's duration. Thoracolumbar magnetic resonance imaging revealed a cystic lesion extending from T11 to L1 with T2 hyperintensity similar to cerebrospinal fluid (CSF). She underwent hemilaminectomy with complete cyst resection and dural defect repair. Postoperatively, the patient demonstrated progressive neurological recovery without complications.

**Conclusion:** SEACs are rare lesions that may cause significant neurological symptoms if untreated. For symptomatic cases, complete cyst excision with dural repair is the preferred management to prevent recurrence and achieve favorable outcomes.

**Keywords:** Arachnoid cyst, Spinal intradural arachnoid cyst, Spinal cord compression, Spinal cord Neoplasms, Spinal cord diseases

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## Highlights

- SEACs are rare lesions that can cause progressive spinal cord/root compression.
- MRI is the key diagnostic tool, typically demonstrating a CSF-intensity extradural cystic lesion.
- Cyst enlargement is often explained by a one-way valve CSF mechanism through a dural defect.
- Surgical excision with dural defect repair is the preferred treatment for symptomatic cases to prevent recurrence.
- Early surgery provides good neurological recovery, as shown in the thoracolumbar SEAC case in our study.

## Introduction

Spinal epidural arachnoid cyst (SEAC) is a rare lesion, accounting for approximately 1% of all spinal tumors [1, 2]. It may be discovered incidentally or present with cord compression leading to progressive neurological symptoms, such as weakness and sensory disturbance [3]. Although SEAC can occur throughout the spinal axis, the thoracic region is the most common site of occurrence [2]. It etiology is thought to be predominantly congenital, though trauma and iatrogenic factors have also been implicated [1]. The mechanisms underlying cyst enlargement remain unclear despite several proposed hypotheses. Magnetic resonance imaging (MRI) is the diagnostic modality of choice, typically demonstrating a cystic mass with signal intensity similar to cerebrospinal fluid (CSF) [4]. Identification of an associated dural fistula can be challenging and often requires adjunctive modalities, such as computed tomography or myelography. The optimal surgical strategy has yet to be established, highlighting the need for further study [5]. Here, we present our experience in the diagnosis and management of SEAC.

## Case Presentation

A 27-year-old female presented with a history of paraparesis that began three years earlier and had progressively worsened over the past two months. She reported no pain, paresthesia, or sphincter disturbance. There was no history of trauma, prior surgery, or infection. Thoracolumbar MRI demonstrated an extradural cystic lesion extending from T11 to L1, hypointense on T1-weighted and hyperintense on T2-weighted images, consistent with CSF signal (Figure 1). The lesion was located dorsolaterally, more prominent on the left side, with encroachment on the T12 nerve root. Myelography revealed communication between the cyst and the subarachnoid space at the T12 level, suggesting an arachnoid origin (Figure 1). Surgical resection was performed, and the patient recovered uneventfully without complications (Figure 2). Histopathological examination showed a cyst wall composed of fibrous connective tissue, vascular structures, and attenuated epithelial lining, without evidence of malignancy, consistent with an arachnoid cyst (Figure 3).

### Surgical technique

The patient was positioned prone, and a midline incision was made over T11–L1. A left-sided subperiosteal dissection was performed, preserving the contralateral fascia and musculature. Left hemilaminectomy from T11 to L1 was carried out using a high-speed drill and rongeur, exposing a thin-walled, transparent cyst. The cranial and caudal poles of the cyst were identified at T11 and L1, respectively. At T12, the cyst extended laterally into the neural foramen, encroaching on the nerve root, necessitating a foraminotomy for adequate visualization. The cranial and caudal poles were carefully dissected and mobilized toward the midline. Upon further dissection, a dural defect was identified at the dorsolateral aspect of T12. The cyst was completely resected, and the dural defect was repaired with primary suturing reinforced with fibrin glue (Figure 2).

## Discussion

SEACs are rare lesions, accounting for approximately 1% of all spinal tumors, with a male predominance and a median age of diagnosis around 30 years [6, 7]. They occur most frequently in the thoracic region, particularly the mid-to-lower thoracic spine (65%), but may also be observed in the lumbar (30%), cervical (15%), and, rarely, sacral regions (3%) [2, 8]. SEACs are typically located posteriorly or posterolaterally to the spinal cord, although anterior presentations have also been reported [7, 9, 10]. The relatively narrow thoracic canal may contribute to earlier symptom progression and the higher prevalence of thoracic involvement compared to other regions.

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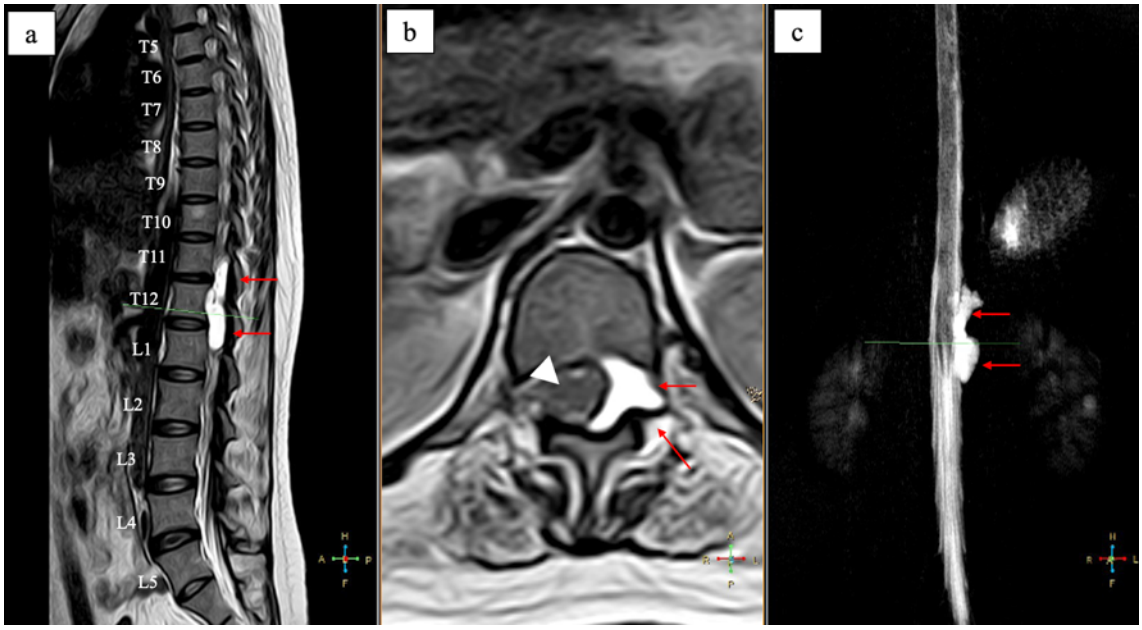


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**Figure 1.** A) Pre-operative sagittal T2-weighted MRI of the thoracolumbar spine at T11–L1 demonstrated a well-circumscribed extradural cystic lesion exhibiting high signal intensity (red arrows), B) Axial T2-weighted image at the corresponding level showed the cystic lesion (red arrows) exerting mass effect on the spinal cord (white arrowhead), C) Myelography revealed CSF extravasation into the cyst cavity (red arrows), confirming communication with the subarachnoid space

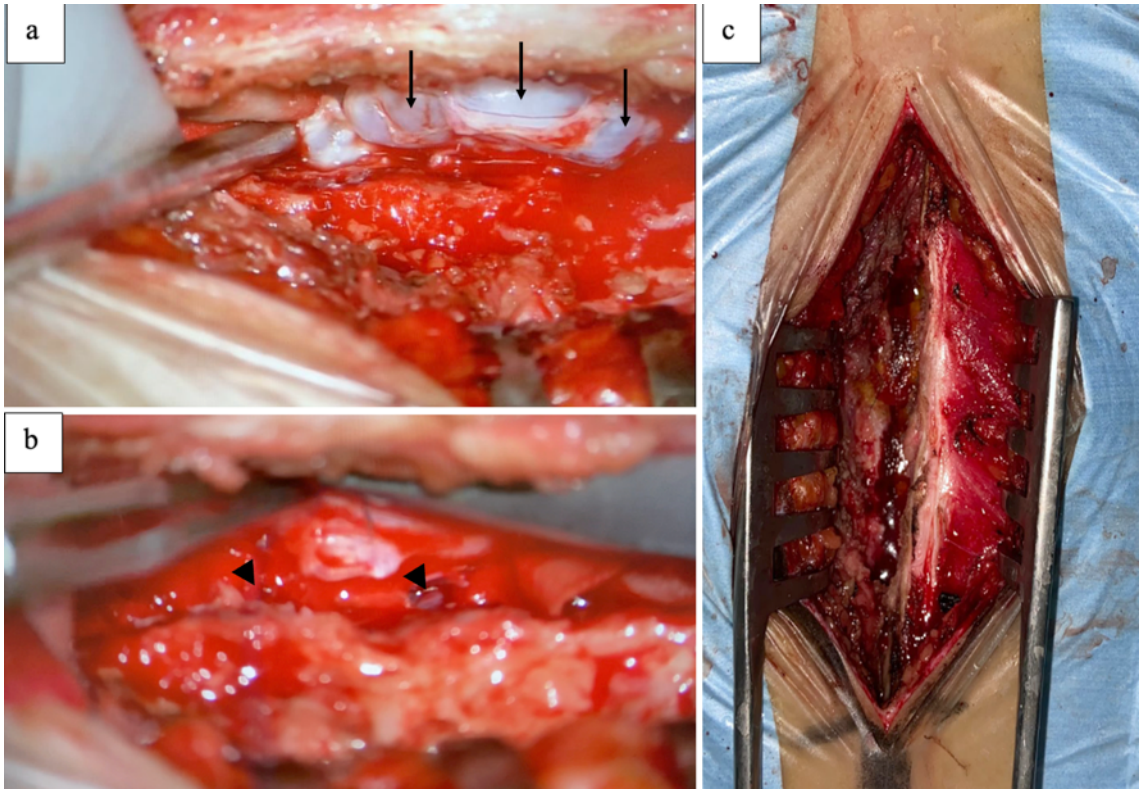


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**Figure 2.** A) Intraoperative view following laminectomy, demonstrating an epidural cystic lesion (black arrows) adherent to the dorsal dura, B) After partial cyst resection, a dural fistula is visualized at the dorsolateral aspect of the spinal cord (black arrowheads), indicating communication with the subarachnoid space, C) Operative field after complete cyst removal and dural reconstruction, showing restoration of dural integrity

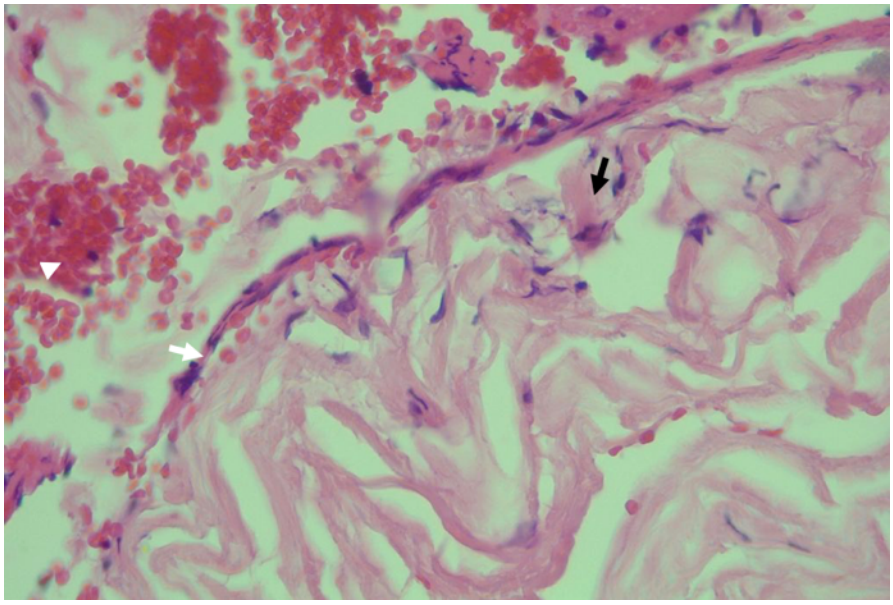

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**Figure 3.** Histopathological section (H&E stain) demonstrating fibrous tissue (black arrow)

Note: The squamous epithelial lining (white arrow) appears attenuated/partly denuded, and a small vessel is indicated (white arrowhead).

SEACs were first described by Schlesinger in 1893 as part of the broader entity of spinal arachnoid cysts [1]. Various terms have since been used, including "arachnoid diverticula" and "serous spinal meningitis". Nabors et al. later proposed a classification of spinal meningeal cysts into three types: Type I, extradural cysts without nerve root involvement; type II, extradural cysts with root involvement; and type III, intradural cysts [11]. Thoracic cysts are more commonly reported in adolescents, whereas lumbar and thoracolumbar cysts tend to present later, typically in the fourth decade of life [2, 12]. Clinically, pain is the most frequent presenting symptom, often radicular in nature, followed by myelopathic features, such as lower extremity weakness and bladder dysfunction [7]. Epstein described a syndrome associated with intracranial hypotension, likely caused by CSF leakage into the cyst, manifesting as postural headache with myelopathy or radiculopathy [6]. Additional symptoms may include gait disturbance, paresthesia, and neuropathic pain [2, 3]. Cervical cysts may present with spastic tetraparesis, whereas thoracic and lumbar cysts are more often associated with radiculopathy and bladder involvement [2]. Some SEACs are discovered incidentally, while in pediatric cases, symptoms may be overshadowed by coexisting central nervous system anomalies [3].

The exact etiology of SEACs remains unclear and is likely multifactorial. Both congenital and acquired mechanisms have been proposed. Acquired cysts may arise spontaneously or secondary to trauma, iatrogenic procedures, or inflammation of the spinal meninges [2, 3, 6, 13]. Congenital theories include the presence of a dural defect or diverticulum leading to arachnoid herniation, supported by reports of associated congenital anomalies in some cases [1]. Up to 68% of children with spinal arachnoid cysts have concurrent central nervous system malformations, frequently linked to neural tube defects [14]. Genetic predisposition has also been suggested: Ogura et al. identified FOXC2 gene mutations as a cause of congenital and syndromic SEACs [15]. Perret et al. proposed that dorsal spinal arachnoid cysts may originate from the septum posticum, a midline structure at the dorsal cord [16]. Another hypothesis involves the entrapment of arachnoid granulations, resulting in localized CSF sequestration at sites of least resistance [17].

The mechanism underlying cyst enlargement remains debated, with several theories proposed. The most widely accepted is the one-way valve mechanism, in which a narrow communication develops between the cyst and the subarachnoid space. Increased subarachnoid pressure opens this passage, permitting CSF to enter the cyst; however, reverse flow is prevented, leading to progressive enlargement [3, 10]. As pressure decreases, the dis-

tended cyst wall may compress its pedicle, further limiting outflow due to the greater tension and radius of the cyst compared with the pedicle [2]. Other mechanisms have been suggested, including active secretion of fluid by the cyst lining and osmotic gradients between the subarachnoid space and the cyst cavity [18]. Histologically, the cyst wall consists of fibrous connective tissue with an inner arachnoid lining, which may be absent in some cases. Pathological findings may include single-layer squamous epithelium, thin-walled dilated vessels, and collagen fibers [19]. Not all cysts communicate with the subarachnoid space, as indicated by the absence of dural defects in some intraoperative observations [2].

MRI is the modality of choice for evaluating SEACs, as it delineates the cyst and its relationship with the spinal cord, nerve roots, and adjacent structures [2, 12]. The cyst typically exhibits signal intensity similar to CSF. Additional radiological features may include vertebral body scalloping, neural foraminal widening, and residual epidural fat at the cyst's poles [2]. Dorsal cysts are more common than ventral ones, with a reported ratio of 1.2:1 [16]. To identify the site of communication with the subarachnoid space, myelography may be performed [20], while cine-MRI provides a less invasive alternative [4, 12]. MRI or CT myelography may also reveal characteristic findings, such as cord intussusception, the "double cord" sign, or patterns resembling arachnoiditis [6]. Liu et al. recommended serial CT myelography, with the first performed immediately and a second study 3–8 hours later, to enhance visualization of dural fistula filling [2]. Nevertheless, identification of the communication site may remain challenging, even intraoperatively [1].

Asymptomatic, incidentally discovered SEACs may be managed conservatively with clinical observation and serial imaging to monitor for cyst enlargement or symptom progression [2, 12, 21]. In contrast, patients presenting with neurological deficits require early surgical intervention, ideally within 24 hours of symptom onset, as emphasized by Ozdemir et al., who reported favorable outcomes with prompt surgery [6, 22].

Surgical strategies vary. Complete cyst excision with dural defect repair is generally considered the mainstay of treatment [1, 8, 21, 23]. Marsupialization has been advocated for large, multilevel cysts, whereas aspiration alone is regarded as inadequate due to the high risk of immediate recurrence. Partial excision with closure of the dural defect has also been described [10]. Hatashita et al. suggested that dural reconstruction may not be necessary if total excision is achieved, as some cysts lack a demonstrable dural communication and do not recur postoperatively [24]. However, Bond et al. reported recurrence even after complete excision, highlighting the importance of careful follow-up [3].

For recurrent or refractory cases, cystoperitoneal shunting may be considered [13, 25]. Boody et al. described a case of lumbar SEAC requiring a lumboperitoneal shunt despite prior cyst resection and dural repair [26]. Additionally, syringomyelia may develop either before or after surgery, sometimes necessitating placement of a syringo-subdural shunt [27].

The optimal extent of laminectomy required for adequate cyst resection remains debated [19]. Complete resection of large, multilevel cysts may necessitate extensive laminectomy, which carries risks, such as postoperative instability and kyphotic deformity [5, 28]. To mitigate this, some authors have advocated closure of the dural communication without complete cyst excision, thereby limiting the surgical exposure [7, 29]. Accurate preoperative identification of the communication site is essential for this approach and may be achieved using myelography, cine-MRI, or MRI with constructive interference in steady state (CISS), which has been reported as particularly useful [4, 30]. However, limited laminectomy focusing solely on dural closure may be insufficient in cases of multiple cysts [4].

Minimally invasive alternatives, including endoscopic fenestration of the cyst wall, have also been described, with recurrence rates comparable to partial open resections [29, 31]. In our case, a hemilaminectomy was performed, as the cyst was eccentric to one side. This approach preserved spinal stability while allowing complete cyst dissection, dural defect identification, and reconstruction, thereby preventing recurrence. Our outcome aligns with the experience reported by Jian et al., who achieved favorable results with this strategy [5]. The cyst was completely resected, and the patient's symptoms resolved without recurrence during follow-up. The interval between symptom onset and surgery has been associated with surgical outcome, with durations exceeding one year linked to poorer recovery [27]. Similarly, cysts extending over more than five vertebral levels have been correlated with less favorable outcomes [23].

## Conclusion

SEAC is a rare entity that may lead to disabling symptoms if untreated. For symptomatic cases, complete cyst resection with dural defect repair is essential to prevent recurrence.

## Ethical Considerations

### Compliance with ethical guidelines

However, written informed consent was obtained from the patient to publish this case report and accompanying images.

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### Authors contributions

Conceptualization and supervision: Rohadi Muhammad Rosyidi and Bambang Priyanto; Methodology: Rohadi Muhammad Rosyidi and Bambang Priyanto; Data collection: Made Bhuwana Putra, Tjokorda GB Mahadewa, I Wayan Niryana, and Nyoman Golden; Data analysis and Interpretation: Made Bhuwana Putra, Sri Maliawan, and Maria Monica; Resources: Tjokorda GB Mahadewa, I Wayan Niryana, Nyoman Golden, and Sri Maliawan; Investigation and writing: All authors.

### Conflict of interest

The authors declared no conflict of interests.

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