Journal of
Clinical Neurology and Neurosurgery

Case Report Open Access

Spinal Extra Dural Arachnoid Cyst: A Rare Cause of Compressive Myelopathy

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Abstract

Background: Spinal extradural arachnoid cysts (SEAC) are rare cause of myelopathy. They account for 1-3 % of spinal tumors. Etiology and optimum treatment still remains unclear.

Clinical case: A 50 years male patient presented with features of spastic paraparesis for 6 months duration. His MRI revealed well defined cystic lesion of size 7.3 × 2.3 cm in posterior epidural space extending from D5 to D8. Laminoplasty (D5 to D8) and complete excision of cyst was done and dural defect was repaired. Patient's lower limb power was improved significantly after surgery.

Conclusion: Surgery is indicated for symptomatic patients. Complete excision of cyst with repair of dural defect remains standard procedure and provides cure.

Keywords: Spinal extradural arachnoid cysts; Spinal tumors; Symptomatic patients

Introduction

Spinal arachnoid cysts are mostly intradural spinal extradural arachnoid cyst (SEAC) is very rare condition accounting for only 1% cases of spinal tumors [1-3]. SEAC is mostly found in males in their second to fifth decades [3]. Most common location of SEAC is thoracic spine.

SEAC results from tiny Dural defect through which out pouching herniation of arachnoid membrane takes place. Etiology of SEAC still remains unclear but can be congenital, post traumatic and post infective. SEAC may enlarge with time and can exert mass effect over cord and/or root and produce myelopathy and radiculopathy [1-5]. These cysts enlarge during exercise or Valsalva maneuvers as these cysts communicates with subarachnoid space [1,6]. Small, asymptomatic cysts can be observed but symptomatic cysts require surgery. Various surgical techniques have been described but still there is no consensus [1,2,7]. Here a case report of SEAC is presented with review of relevant literature.

Case Report

A 50 years old male patient was presented with upper, mid backache with progressive spasticity and weakness of both lower limbs for 6 months duration. He could walk with support only. He was evaluated with MRI and other relevant investigations (Figure 1). His MRI revealed a well-defined cystic lesion of size 7.3 × 2.3 cm in posterior epidural space from D5 to D8 level with compression of the underlying thecal sac and spinal cord. Cyst was extending within the D6-7 and D7-8 neural foramina bilaterally. Patient was operated. D5 to D8 laminoplasty was performed (Figure 2). D5 to D8 laminae along with spinous process and inter spinous ligament were removed in a single piece. Cyst wall was excised completely. Tiny dural defect was present at the posterolateral aspect of dural sac at D7 level. Dural defect was repaired with 4-0 proline. Intact laminae with spinous processes and attached interspinous ligaments were replaced and secured in place. Wound was closed in layers. Patient did not develop any complication. After surgery, patient showed dramatic improvement and in follow up after 2 weeks patient can walk without any support. Histopathological findings were consistent with arachnoid cyst.

Discussion

SEAC is a rare cause of compressive myelopathy, which accounts for only 1% of all spinal tumors. It is more common in males in their 20s. Thoracic spine is most common location [1-3].

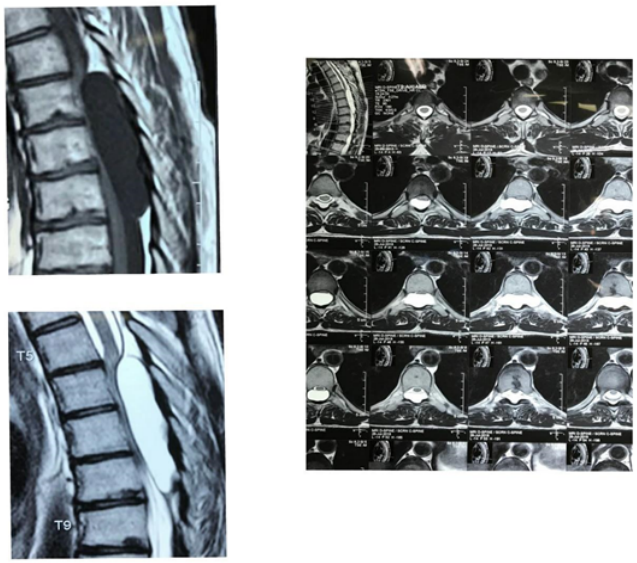

Figure 1: Preop MRI showing arachnoid cyst extending from D5 to D8.

SEAC results from herniation of arachnoid membrane through the tiny Dural defect [1,2]. These cysts communicate with subarachnoid space through which CSF accumulates [1,6]. Rarely, the cyst does not have any communication with subarachnoid space [8]. Etiology of these dural defects still remains unclear. These defects can be congenital or acquired [1,2,4]. Acquired causes can be trauma, infection or inflammation [1,2,4]. It may be associated with dural ectasia, Marfan syndrome [1]. In this condition, organization of collagen is defective, which results in decreased tensile strength of ligamentous structures and other supporting tissues [9]. Dural stretching can lead to dural

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Received September 03, 2018; **Accepted** October 09, 2018; **Published** October 16, 2018

Citation: Verma S, Soni R (2018) Spinal Extra Dural Arachnoid Cyst: A Rare Cause of Compressive Myelopathy. J Clin Neurol Neurosurg 1: 105.

thinning to such an extent that it becomes ectatic and may be deficient in areas [9]. Although there is still debate in determining the etiology of SEACs, the theory of congenital dural defect is widely accepted [9]. Dural defect is often present near the nerve root sleeves. Proposed reason is that tension across the movable dural sac and relatively fixed roots can predispose such dural defects [2]. Outpouching of arachnoid takes place through these small defects [1,2]. These herniations become enlarge with time during exercise or Valsalva [1,6]. It explains the symptomatic fluctuation during exercise and Valsalva maneuvers. Based on this, pulsatile CSF flow dynamic theory was proposed by many authors to explain enlargement of cyst [1,2,6]. These defects may act like valve as defects are small and arachnoid herniates beyond their margins. Rootlets may also get trapped and it again act like a valve [2]. As enlargement continues, a SEAC can aggravate spinal cord compression or nerve root compression, which leads to myelopathy or radiculopathy [2-4]. Nabors et al. [10] classified in to 3 categories:

Type 1: Extradural cyst without nerve tissue.

1A-Extradural meningeal cyst, 1B- Sacral meningocele.

Type 2: Extradural cyst containing nerve tissue.

Type 3: Spinal cyst.

In our case, there was extradural cyst without any nerve tissue so it was type 1A.

MRI is the most useful tool to diagnose a SEAC. Radiological studies report that a SEAC appears similar to cerebrospinal fluid [2,3,7]. A CT myelography is also a useful diagnostic tool as it can more reliably detect the anatomical location of the cyst. In addition, a myelogram and CT myelography can help locate the Dural defective site [2,6]. CSF flow MRI can identify the pulsating turbulent flow void of a defective site.

Symptomatic cyst needs surgical treatment [1,2,7]. There is a consensus among surgeons to repair the Dural defect in the treatment of a SEAC. However, there is still disagreement regarding the treatment

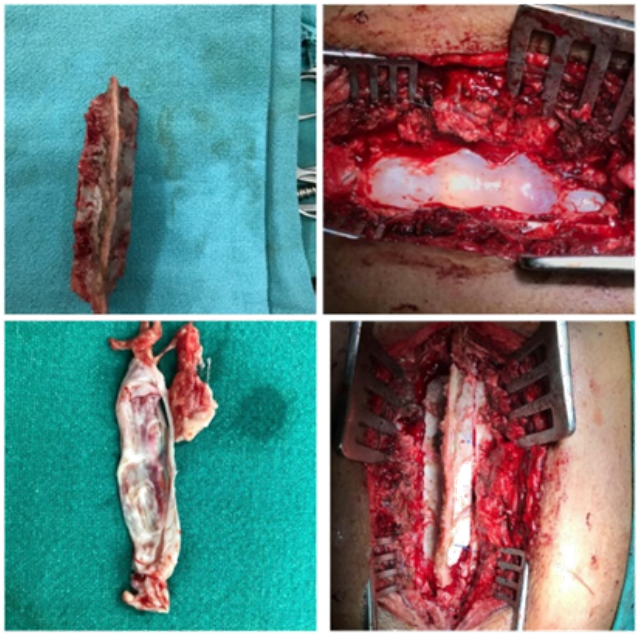

Figure 2: Intra operative pictures showing laminoplasty, cyst and excised wall.

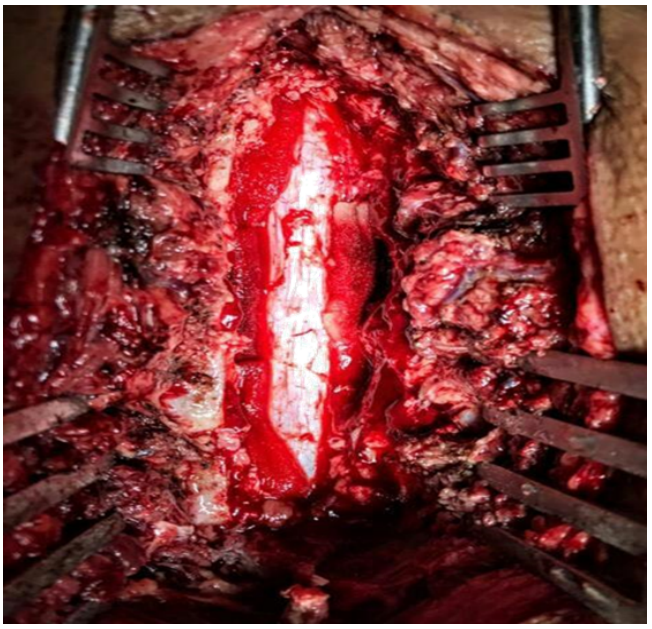

Figure 3: Decompressed thecal sac after excision of cyst.

of the cyst [2-4]. Diverse surgical techniques have been described and complete microsurgical resection of SEACs with meticulous repair of Dural defect has been advocated as treatment of choice for SEACs [2,11]. Long segment laminectomy and complete excision of cyst may be associated with complications like bleeding, post-operative kyphotic deformity and instability (Figure 3) [1,3,4,7,9]. Alternative surgical techniques have been described to avoid these complications. Payer et al. described selective interlaminar fenestration at communication site with repair of Dural tear. Communication site was identified as flow void seen on preoperative cine MRI [11]. This technique has an advantage of minimal laminotomy but requires very precise localization of Dural defect that may not be possible every time. Woo et al. described laminectomy at defect site, penetration of cyst and Dural repair [12]. But they had to extent their laminectomy because of spatial limitation or not finding Dural defect. Won Choi et al. described tailored laminectomy, fenestration and closure of Dural defect [2]. Javier Quillo-Olvera et al. did evacuatory puncture of cyst and concluded that if the patient has mild symptoms, clinical observation is recommended [13]. In our case, cyst was large, extending from D5 to D8 level. We did laminoplasty from D5 to D8. As the laminae became thin because of cyst pressure, it was easy to fracture at lamina-facet junction. Laminae along with spinous processes and attached interspinous ligaments were removed in one piece. Cyst was completely excised and dural defect was repaired. Whole posterior segment was replaced and secured in place. Advantages of our procedure include posterior column remains in place so there are no chances of kyphosis and instability. There is no need for precise localization of dural defect. As the cyst is completely excised so more rapid improvement of symptoms.

Conclusion

SEACs are rare cause of compressive myelopathy but should be kept in mind as they respond very well after surgery. Various surgical techniques have been described. Meticulous repair of dural defect is necessary to prevent recurrence. We performed laminoplasty (to avoid postoperative kyphosis and instability), complete excision of cyst wall (for rapid improvement of symptoms) and repair of Dural defect (to prevent recurrence).

Acknowledgement

The authors have no financial conflicts of interest. The authors have taken permission from the patient for this case report.

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