Case Report

Spinal Extradural Arachnoid Cyst

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Spinal extradural arachnoid cyst (SEAC) is a rare disease and uncommon cause of compressive myelopathy. The etiology remains still unclear. We experienced 2 cases of SEACs and reviewed the cases and previous literatures. A 59-year-old man complained of both leg radiating pain and paresthesia for 4 years. His MRI showed an extradural cyst from T12 to L3 and we performed cyst fenestration and repaired the dural defect with tailored laminectomy. Another 51-year-old female patient visited our clinic with left buttocck pain and paresthesia for 3 years. A large extradural cyst was found at T1-L2 level on MRI and a communication between the cyst and subarachnoid space was illustrated by CT-myelography. We performed cyst fenestration with primary repair of dural defect. Both patients' symptoms gradually subsided and follow up images taken 1-2 months postoperatively showed nearly disappeared cysts. There has been no documented recurrence in these two cases so far. Tailored laminotomy with cyst fenestration can be a safe and effective alternative choice in treating SEACs compared to traditional complete resection of cyst wall with multi-level laminectomy.

Key Words: Arachnoid cyst · Cerebrospinal fluid · Thoracolumbar spine.
The cyst wall was severely adherent to the dural sac and there was moderate bleeding on dissecting the cyst. We only fenestrated the cyst. Some portion of the cyst wall was removed and sent to pathologic department. Pathologic findings were compatible to arachnoid cyst (Fig. 4).

Her symptoms relieved gradually and follow up MRI taken at 2 months after the operation showed much decreased cyst and there was no evidence of cord compression due to residual cyst (Fig. 5).

DISCUSSION

SEAC is a rare disease entity accounting for 1% of all spinal tumors. These cysts may originate in protrusion of arachnoid membrane through a dural defect and may be enlarged by CSF accumulation. When enlarged, they may compress the root or cord and result in symptoms such as pain or weakness. Besides the increase in cyst size, pressure changes in extradural space as well as in arachnoid cyst might cause spinal cord compression and result in such symptoms.

Pathogenesis and classification

The etiology of SEACs remains still unclear and can be congenital or acquired. SEACs are assumed to be the result of dural defects. Communication between the cysts and the intradural subarachnoid space has been reported in nearly all cases of arachnoid cysts.

The cause of dural defect can be congenital or acquired. Trauma, arachnoiditis or iatrogenic cause can result in small dural tear and subsequent CSF accumulation to develop SEACs. Some reports demonstrated an association with dural ectasia or Marfan syndrome. In this condition, a primary defect in the organization of collagen with decreased tensile strength weakens the ligamentous structures and other supporting tissues. Dural stretching can lead to dural thinning to such an extent that it becomes ectatic and even deficient in areas. Although there is still debate in determining the etiology of SEACs, the theory of congenital dural defect is widely accepted.

Dural defect is often found around the nerve root sleeves. One possible explanation is that tension across the movable dural sac and relatively fixed roots can predispose such dural tears. If patients have underlying structural abnormality such as Marfan
syndrome, the probability of such tears may be further increased. In our experience, both patients had no history of trauma, arachnoiditis or previous spine operation. No specific underlying disease was documented. Most authors believe that the cyst can be enlarged by pulsatile CSF dynamics and this theory can explain symptom fluctuation with exercise or valsava maneuver related to CSF dynamics. There is limitation of understanding the pathogenesis of cyst development with osmotic gradient because there is lacking evidence of any difference of cystic fluid compared to CSF. If in case of no communication, authors explained that the cyst developed by pulsatile CSF dynamics via a dural defect and the communication had been gradually closed over time and then disappeared. On histopathologic examination, SEACs had no secretary function and the pathogenesis of active secretion by the cysts is invalid for explaining enlargement of the cysts.

Nabors et al. categorized SEACs in three major groups of meningeal cyst, non-meningeal epidural cysts, and neurenteric cysts. Meningeal cysts are further classified in 3 subgroups: 1) type 1: extradural meningeal cyst that contains no neural tissue, 2) type 2: extradural meningeal cyst that contains neural tissue, 3) type 3: intradural meningeal cyst. Type 1 meningeal cysts consist of extradural arachnoid cysts (type Ia) and sacral meningoceles (type Ib).

The second case in current study is similar to the type 2 meningeal cyst categorized by Nabors et al. However, we propose a different lesson from the case when assuming the pathogenesis of cyst development. An trapped rootlet was found around the dural defect and we assumed it might contribute to cyst development. CSF leaks out via the dural defect and the rootlet caught in the defect interrupts its return into the subarachnoid space. This valve-like mechanism was previously described in several studies and the second case in this study can be another evidence supporting this mechanism. The initial formation of the dural defect is assumed to be idiopathic.

**Clinical outcome and surgical treatment**

Signs and symptoms of SEACs are due to chronic cyst expansion and compression of the neural structures and thus, SEACs need surgical treatment. Diverse surgical techniques have been introduced and complete microsurgical resection of SEACs with meticulous repair of dural defect has been advocated as treatment of choice for SEACs.

Alternatively, one report describes a selective interlaminar fenestration at the transdural communication site seen as pulsating flow voiding on preoperative cine-MRI at T12-L1 in a cyst from T1- to L3, with clipping of the dural rent and this resulted in subsequent cyst regression and improvement in clinical outcome. This technique has an apparent advantage of limited laminotomy but depends on precise preoperative planning of clip position at the communication site.

Lee et al. demonstrated that the recurrence of the SEAC was related to the repair of the dural defect and not to the complete-
ness of SEAC excision. To minimizing the extent of laminectomy is important to avoid postoperative complications such as kyphosis.

With improved diagnostic tools, we can verify the location of communication or dural defect preoperatively and it can help planning tailored laminotomy to avoid multi-level laminotomy and subsequent increased risk of complications. Currently many surgeons favor the tailored laminotomy and cyst fenestration with meticulous dural repair rather complete resection of SEACs to reduce the complication related to long-level laminectomy [6,11,13].

We performed tailored laminectomy for cyst fenestration and repair of the dural defect and had good clinical and radiologic outcome.

CONCLUSION

We have experienced two cases of SEACs and performed tailored laminectomy and cyst fenestration with dural repair in these cases. Clinical and radiologic outcome was excellent in both cases and there has not been any evidence of cyst recurrence till now.

In the second case, a loop of single rootlet was found got stuck within the dural defect and it might contribute to develop the cyst by inhibiting spontaneous dural closure. It is a rare case and can be considered as another etiology of SEACs.

References