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Case Report

Noncommunicating Spinal Extradural Arachnoid Cyst in a Child: A Case Report

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Abstract

Spinal extradural arachnoid cyst is a rare cause of spinal cord compression. The common theory to the formation of this cystic lesion is the existence of dural defects with a communication between the cyst cavity and subarachnoid space. In a few cases, there is a lack of communication. We report a case of noncommunicating spinal extradural arachnoid cyst in a 6-year-old male who admitted with symptoms of spinal cord compression. A laminotomy was performed with complete cyst removal. There is no pedicle between the cyst lesion and the dura was intact. The patient had a complete recovery and the cyst was disappeared without CSF leaking or meningocele postoperatively. We review the literature to highlight the physiopathology of this noncommunicating cyst formation.

Keywords: Arachnoid cyst; Spinal; Noncommunicating; Spinal cord compression

Introduction

Spinal Extradural Arachnoid Cyst (SEAC) is an unusual cause of spinal cord compression with an incidence approximatively around 1% of evolving tumors in the spine [1,2]. The physiopathology is unclear but the common theory suggested the presence of dural defect and contributions of CSF fluctuations in the formation of the cyst [3]. The mainstay of management of symptomatic SEAC is the cyst removal and closure of the dural defect. In very rare cases, the dural defect cannot found and these constitute a noncommunicating SEAC. Here, we report an unusual case of noncommunicating SEAC in a 6-year-old male presented as a spinal cord compression. We review also literature papers to discuss the physiopathology.

Case Presentation

A 6-year-old male was presented with a history of 5 months of progressive gait disorder without back pain. There was no history of trauma, infection, or any previous surgical procedure to the spine. On clinical examination, the patient had no motor weakness but presented a proprioception impairment, Babinski sign, and hyperreflexia in both lower limbs. Bladder and bowel function were normal. Upper extremities and cerebral function were normal. The neurological findings were consistent with thoracic or lumbar spinal cord compression symptoms. The thoracolumbar Magnetic Resonance Imaging (MRI) revealed an elongated well-defined intraspinal posterior epidural cystic lesion from T5 to T10 levels. The lesion was isointense comparing to Cerebrospinal Fluid (CSF) both T1 and T2-weighted images (respectively hypointense and hyperintense). The lesion causes posterior compression of the thoracic medulla against the posterior wall of the vertebral bodies. There are no evidence of cord lesions and a widening of the spinal canal. There is no enhancement after the administration of gadolinium (Figure 1). The patient underwent a T5 to T10 laminotomy in addition to a total removal of the cyst to obtain complete decompression of the spinal cord. Intraoperatively, and under microscope, no dural defect was found after the removal of the walls of the cyst. Valsalva maneuver was not induced CSF leaking. Histological examination revealed fibro collagenous layers without nerve cells. The patient was discharged at home at 5 days postoperatively. A rehabilitation was performed and the patient's gait disorder completely decreased four weeks postoperatively. There are no CSF leaking or infectious complications after surgery. Follow-up MRI at 3 months showed that the cyst lesion had completely disappeared (Figure 2).

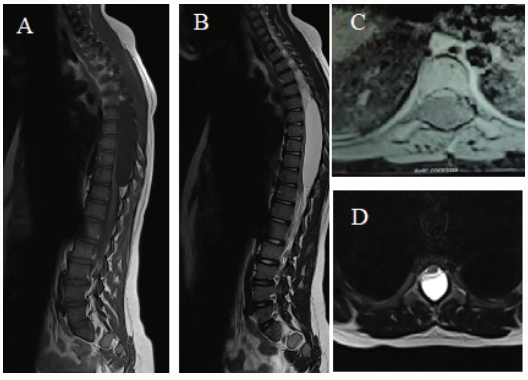

Figure 1: Preoperative magnetic resonance images showed a spinal extradural arachnoid cyst with posterior spinal cord compression. (A) Sagittal T1-weighted image: hypointense lesion at the thoracic region with no enhancement after gadolinium administration. (B) Sagittal T2-weighted image: Hyperintense lesion at the posterior thoracic spinal canal. (C) Axial T1-weighted image and (D) T2-weighted image showing a posterior spinal cord compression.

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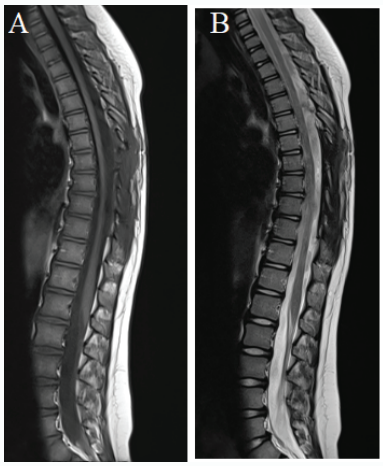

Figure 2: Postoperative magnetic resonance images showed a complete resolution of the spinal extradural arachnoid cyst. (A) Sagittal T1-weighted image and (B) Sagittal T2-weighted image.

Discussion

SEAC is more common in the posterolateral aspect of the thoracic and thoracolumbar spine [4,5]. Diagnosis is made by progressive signs and symptoms caused by spinal cord compression [6]. The more common presenting symptoms are radiculopathy, pain, lower extremity weakness, and gait instability [2,5]. The gait instability in our patient was caused by the direct compression of the gracile fasciculus named also tract of Burdach that is a sensory pathway in the spinal cord and conveys proprioception from the skin and joints.

The mainstay hypothesis to explain the formation of SEAC is the existence of the dural defect [3,7]. A dural dissection cyst was proposed as an accurate term to define arachnoid cyst [7]. The exact etiology is unclear but two considerations can be supported to dural defect: congenital origin or acquired. In acquired origin, several factors are associated such as trauma, arachnoiditis, surgery [3,8,9]. In the absence of inflammation or traumatic causes, most authors believes in congenital origin [10]. Hydrostatic pressure of CSF and pulsations of the CSF flow explain the enlargement and the growth of the cyst.

MRI is the modality of choice for diagnosis and the imaging findings are well-demarcated lesion with the isointense signal comparing to CSF signal [6]. There is no contrast enhancement of the walls. MRI and myelography can demonstrate the pedicle site of the pedicle that communicates the subarachnoid space and the cyst cavity. Garg et al. [2] reported a proportion of 14% of patients with combining MRI and myelography in a case series that were nonconclusive to determine the communicating channel.

The absence of the communicating channel was reported by several authors [4,11,12]. In our illustrative case, there are no dural defects after the complete removal of the cyst wall adherent to the dura. The pathophysiology remains unclear. In our opinion, there are pre-existing communication between the cyst and subarachnoid space that can explain the formation of the cyst. We thought that the communicating channel was obstructed secondary with the effect of cyst pressure. Kim et al. [13] supported that according to the Laplace law, the closure of the communication occurs under the action of the cyst force on the neck of the cyst. We adhere to this hypothesis and thought also that an essential condition is existing of thin dural defect in favor of spontaneous obstruction. According to depletive lumbar puncture used to manage CSF leaking after spine surgery, the mechanisms are reducing the subarachnoid fluid pressure and increasing the epidural space pressure that stop CSF leak [14]. The theory of osmosis in SEAC formation had been rejected according to the fact that the cyst content similar fluid as that of CSF [13,15,16].

Excision of the cyst removal and closure of the dural defect is the treatment of choice [6] and contributes to restoring the CSF flow. The transforaminal approach was also reported in the successful management of SEAC [17]. The authors performed resection of the cyst wall with closure of the ostium. The major risk of surgery of spinal arachnoid cyst is postoperative CSF fistula and recurrence of the cyst. A research of dural defect and his closure with cyst removal is the mainstay of the treatment. Liu et al. [11] reported a successful management of noncommunicating arachnoid cyst treated by complete excision without dural repair. Isolated excision of the cyst can be performed in noncommunicating SEAC with complete recovery without recurrence or CSF leaking. Fibrin glue can be used intraoperatively in the site of surgery to prevent CSF leaking and improve the results. Postoperatively, regular clinical and radiological follow-up could be performed to diagnose CSF leaking or recurrence of the cyst.

Conclusion

Spinal extradural arachnoid cyst is an unusual cause of spinal cord compression. In our case, there was a lack of communication channels with SEAC. Cyst removal was safety performed to treat it with complete recovery and the cyst completely disappeared.

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