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Traumatic spinal extradural arachnoid cyst—A case report

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ABSTRACT

Spinal extradural arachnoid cysts (SEACs) can be congenital or acquired and are assumed to result from dural defects. Communication between the cysts and the intradural subarachnoid space is reported in nearly all cases of SEACs. The mainstay of the current treatment is resection of the cyst wall followed by obliteration of the communicating hole. Despite its clinical importance, the location of the dural defect is often elusive before operation. We report a case of a patient presenting with an extradural arachnoid cyst as a sequel of a major blunt trauma of the lower back approximately 20 years ago. The 56-year-old man presented with progressive paraparesis and back pain. Radiographic images of the lumbar spine showed scalloping of the L1–2 vertebrae. Magnetic resonance imaging of the spine revealed an SEAC at the T10–L2 levels. During the operation, an ovoid dural defect was identified at the L1 level, which coincided with the area where the most severe vertebral scalloping was observed. We postulate that delayed-onset posttraumatic extradural arachnoid cysts should be considered during the differential diagnosis of intraspinal cysts, and vertebral scalloping can be used as a sign to locate dural defect before surgery.

1. Introduction

A spinal extradural arachnoid cyst (SEAC) causes spinal cord or nerve root compression, and enlarging the spinal canal is rarely feasible [1]. The cause of these cysts is not definitively known, although they most probably have a congenital origin. They may be caused by trauma, infection, or inflammation [2]. A traumatic extradural arachnoid cyst is a rare entity. The late occurrence of paraparesis due to the formation of an extradural arachnoid cyst as a sequel of blunt abdominal trauma is extremely rare. We report a case of a patient with symptomatic SEAC that may be due to trauma experienced approximately 20 years ago.

2. Case report

A 56-year-old man presented with a 2-year history of lower back pain and occasional numbness of bilateral lower extremities. Gradually, motor weakness of bilateral legs developed and walking became progressively difficult in 6 months. He had sustained severe back contusion and pelvic fracture in a motorcycle accident approximately 20 years ago, which caused him to be bedridden for 2 months.

Physical examination on admission revealed bilateral proximal weakness in the lower extremities graded at 3/5 on the Medical Research Council scale. Hyperactive knee and ankle jerk reflexes were noted. The area of hypoesthesia was not clearly consistent with the dermatome.

Radiographic images of the lumbar spine showed scalloping of the L1–2 vertebrae. Magnetic resonance imaging (MRI) of the spine revealed long segmental cystic lesion of cerebrospinal fluid signal intensity at the dorsal extramedullary space of the T10–L2 level, suggesting an arachnoid cyst with diffuse cord compression (Fig. 1).

The cyst was exposed through a T10–L2 laminectomy. The posterior wall of cyst was thin and tightly adhered to lanina, so the wall was torn during laminectomy and the CSF leakage was noted. The anterior wall of cyst was also tightly adhered to the dura. On the basis of these finding, we believed that the complete resection of the cyst would be difficult. On exposure, a small ovoid dural defect was identified at the right side of the dorsolateral aspect of the dural sac between nerve root sleeves at the L1 level. The dural defect was packed with a piece of muscle and sealed with fibrin glue (Tisseel) (Fig. 2). A laminoplasty and spinal fusion or instrumentation was not performed due to the thin lamina and, besides, facet joints were grossly preserved. Histologic examination of the cyst wall confirmed the pathological diagnosis as an arachnoid cyst. After the operation, the patient's symptoms alleviated gradually, and follow-up MRI (Fig. 3) at 7 months after the operation showed no residual cyst.

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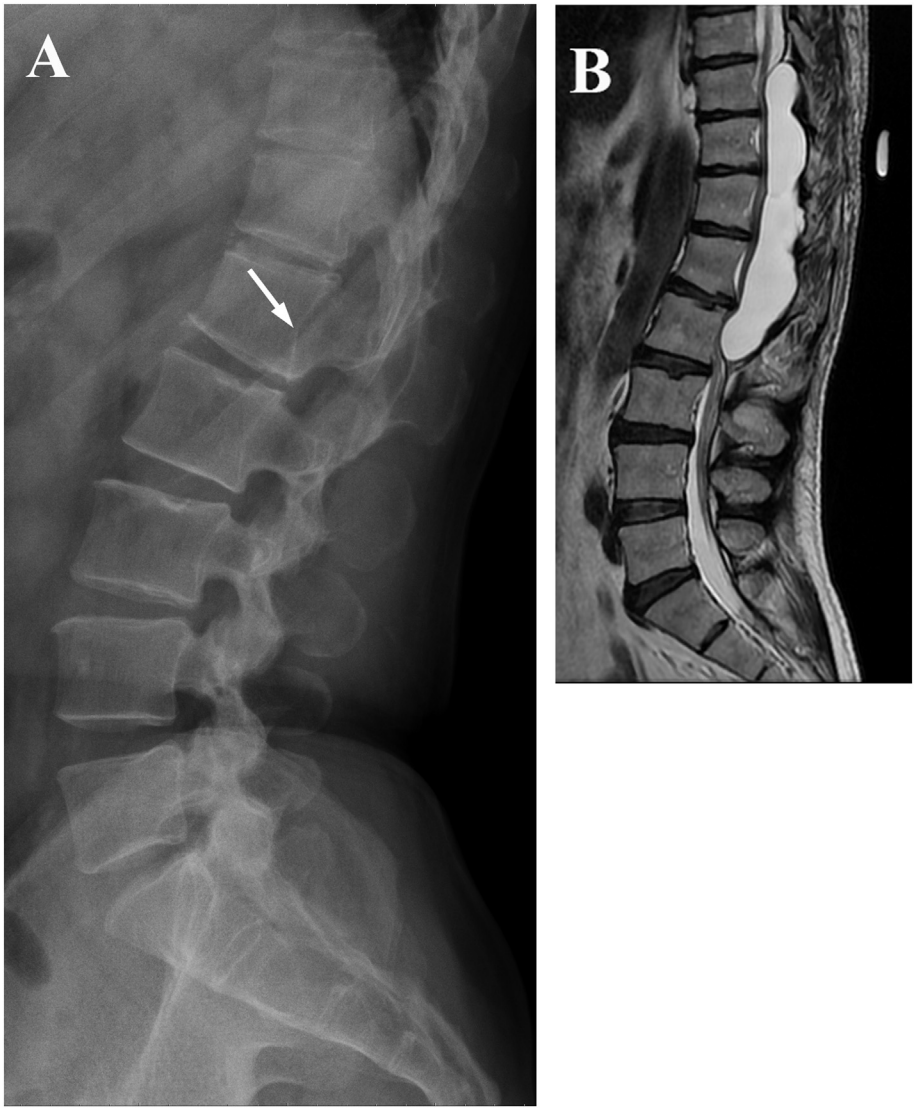

Fig. 1. (A) Lateral radiographic image of the lumbar spine showing the scalloping of L1 and L2 vertebrae, which is the most severe at the L1 level (white arrow). (B) Preoperative sagittal T2-weighted magnetic resonance image of the thoracolumbar spine showing an extradural cyst posterior to the dural sac at the T10–L2 levels with severe cord compression.

3. Discussion

SEACs are an uncommon cause of myelopathy secondary to spinal cord compression [3,4]. The etiology of SEACs remains unclear and can be congenital or acquired. SEACs are assumed to result from dural defects. Communication between cysts and the intradural subarachnoid space has been reported in nearly all cases of arachnoid cysts [5,6]. Congenital origin is proposed, involving either congenital diverticula of the dura or herniation of arachnoid through a congenital dural defect [7]. Furthermore, several theories of acquired origin, such as following spinal surgery, trauma, chronic arachnoiditis, parasitic infection, or degenerative spondylosis, have also been reported [7,8]. The mechanism of enlargement of initial asymptomatic cysts leading to spinal cord compression is debatable. Pulsatile cerebrospinal fluid dynamics, osmotic gradient between the subarachnoid space and cysts, and the valve-like mechanism between the cyst and the subarachnoid space may play a crucial role in cyst expansion [7].

Most cases of SEACs are idiopathic and those of traumatic origin are especially rare [4,7]. In cases of SEACs of congenital origin, most patients are presented with SEACs in adolescence or in early adult life, and the dural defect tends to occur at central regions [9]. In our patient, the dural defect was observed at the junction of the sleeve and thecal sac on the right L1 level. In the present case, there was a clear history of blunt back and pelvic trauma, and we postulated that the cyst was of traumatic origin. Probably an initial tiny tear occurred at the right dorsolateral aspect of the L1 dura when the patient was injured, and the intact arachnoid bulged out through the opening later. The arachnoid out-bulging enlarged slowly and gradually to form an epidural arachnoid cyst over 20 years and thus lead to vertebral scalloping (L1, L2).

Symptomatic SEACs are primarily treated with complete resection of the cyst wall and complete obliteration of the communicating hole between the cyst and the subarachnoid space after laminectomy of the affected vertebrae [7,8]. Alternatively, some reports have noted that partial cyst wall resection and closing of the communicating hole yield excellent results in terms of avoiding spinal instability and malalignment after complete cyst resection [1,5]. Accurate preoperative identification of the location of the communicating hole is necessary to successfully close the hole with minimal laminectomy [5]. Several studies have used cine-MRI, time-spatial labeling inversion pulse MRI, and digital subtraction cystography to locate the defect between the cyst and the subarachnoid space [10]. In our case, the dural defect was observed at the L1 level during the operation, which was at the area

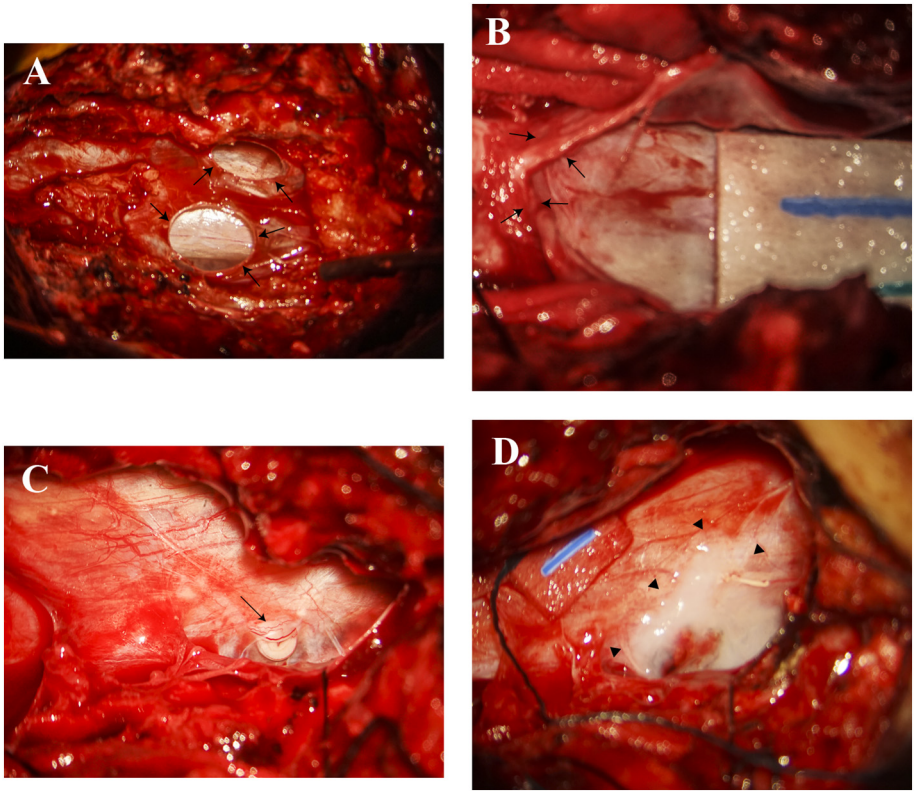

Fig. 2. (A) The posterior wall of cyst was thin and it was torn during laminectomy. (arrows) (B) The upper margin of the cyst showed the anterior wall tightly adhered to the dura. (arrows) (C) Intraoperative photograph showing a 4 × 2-mm ovoid dural defect (arrow) at the right side of the dorsolateral aspect of the dural sac between nerve root sleeves at the L1 level. (D) Microsurgical repair of the dural defect was performed through muscle packing and sealing with fibrin glue (Tisseel) (arrow heads).

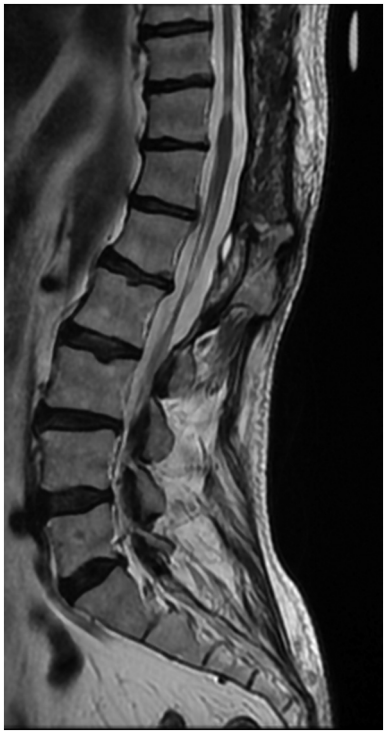

Fig. 3. Postoperative sagittal T2-weighted magnetic resonance image 7 months after the surgery, demonstrating that the cyst had disappeared and the spinal cord was well decompressed.

where the most severe vertebral scalloping was observed. Thus, vertebral scalloping may be used as a sign to located dura defect before surgery.

Conflict of interest

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

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