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Non communicating extra dural meningeal cyst in dorsal spine

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Abstract

Spinal extradural meningeal cyst has been rarely reported, whose etiologies are assumed to be the communication of cerebrospinal fluid (CSF) between intradural subarachnoid space and cyst due to the congenital defect in dura mater. Although the CSF communication due to this defect can be found in most case, few cases in which there is a lack of the communication have also been reported. We report a case of extradural meningeal cyst occurring in the dorsal spine (D7 and D8) where there was a lack of the communication between the intradural subarachnoid space and cyst in a 15-year-old male who presented with symptoms of gait disturbance and right leg pain. The patient underwent laminectomy and cyst excision. On intraoperative findings, the dura was intact and there was a lack of the communication with intradural subarachnoid space. Immediately after the surgery, leg pain and gait disturbance improved shortly.

Keywords: Meningeal cyst, Non communicating, cerebrospinal fluid, dorsal spine, Extradural

1. Introduction

Spinal extradural meningeal cyst (SEMC) has been reported very rarely, accounting for approximately 1-3% of all primary spinal space-occupying lesions^[1,2], Nabors *et al* classified these cysts into three types: extradural meningeal cysts without spinal nerve root fibers (Type I); spinal extradural meningeal cysts with spinal nerve root fibers (Type II, Tarlov cyst); and spinal intradural meningeal cysts (Type III)^[3]. Type I is further divided into extradural arachnoid cysts (Type IA) and sacral meningoceles (Type IB)^[3].

In most of the reported cases of Type IA SEMC, there was a communication of the CSF with intradural subarachnoid space through dural defect^[4-6] these dural defects are of congenital origin which are mostly associated with neural tube defects. But some acquired factors such as inflammation, trauma, surgery, or repeated lumbar puncture might cause dural defect^[7].

2. Case Report

2.1 History and Examination

A 15-year-old man presented with a history of gait disturbance for 4 months and pain in right leg for 2 months. There was no history of trauma. Pain and gait disturbance was insidious in onset and progressive in nature. On admission, he was able to walk by himself with support but lack of coordination was seen.

A physical examination did not reveal any notable findings except weakness in both legs. The straight leg raising test was normal and deep tendon reflexes were increased. The motor powers of both leg foot dorsi-flexion, and foot plantar flexion were checked to be grade 4/5. Vibration sense, tactile discrimination, tactile localization, joint position sense was decreased in both legs. Sensory, bladder, and bowel functions were normal. No signs of cerebral involvement.

There was no obvious finding in x-ray of dorso lumbar spine. A Dorsal lumbar spine MRI revealed the presence of a extradural cystic lesion between D7 and D8 levels. As it is difficult to find the dorsal vertebra during operation and to reduce the time taken to identify the vertebra in fluoroscopy and to prevent radiation exposure we did an x-ray of dorsal spine with a coin placed approximately in the level of lower border of scapula with methylene blue dye injected sub cutaneously. This xray gave an idea about the place of incision and it helped in reducing the excessive dissection, soft tissue trauma and operative time.

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Pre operatively to find the dorsal vertebra so with clinical and radiological correlation it is a case of extradural sub arachnoid cyst with compression of posterior column of dorsal spine with or without communication.



Fig 1: Extra medullary epidural cystic lesion in D7 and D8 with compression of spinal cord with moderate spinal canal stenosis

2.2 Operation

Patient under the effect of general anaesthesia. Patient in left lateral position, a mid line vertical incision is given over dorsal spine in the area as found in pre op coin xray method. Total laminectomy from D7 and D8 were performed under intra operative monitoring system. The laminectomy was performed and ligament flavum was removed. Then, the cyst with a translucent wall was observed. Careful dissection of cyst wall was done and removed from surrounding attachments. A thin pedicle was found in D7–D8 junction. On compression of cyst it did not reduce in size that denotes no communication with intra dural space. Then pedical was ligated. During cystectomy, there was a lack of the communications, such as dural defect, arachnoid pedicle, or fistula, between the cyst and dura mater. Wound closed in layers.



Fig 2: Intra operative picture of spinal arachnoid cyst

2.3 Post-Operative Course

From immediately after the surgery, paresthesia and paraparesis of the both extremities were improved in the patient, and the patient was able to walk by himself. Tactile discrimination, localization, vibration and position sense were improved as compared to pre-operative status gait pattern was improved. A surgical wound was well healed without CSF leaking. The histologic examination revealed the consistency of meningeal cyst which was characterized by fibrous connective tissue. There was no signs of malignancy and it was a simple arachnoid cyst. The patient was discharged without any notable events.

3. Discussion

Intradural cyst occurs most frequently in the middle to lower part of thoracic vertebrae (67%), and it also occurs in the lumbosacral area (20%), the thoracolumbar area (9%). And the cervical area (4%) [8, 9] The lesions are mostly located dorsal to the intervertebral column and then extended to the intervertebral foramen. It shows a male predilection and

symptoms usually occur during the 2nd decade. Most of the patients persistently present with rigid or flaccid Paralysis of the extremities. A local back pain is also one of the common symptoms, but the sensory symptom was not clear [5, 9]. In some cases, the symptoms disappear for several years. In other cases, the symptoms can be aggravated due to the posture or Valsalva maneuver [5, 9, 10]

On computed tomography (CT) scans and a simple X-ray, there can be characteristic imaging features such as the erosion of posterior lamina, pedicle and vertebral body, the extension of spinal canal, the extension of intervertebral foramen, the posterior scalloping of vertebral body and the extension of interpedicular space. A kyphoscoliosis can also occur [5, 8]. Through myelography or myelo-CT, the communication with subarachnoid space can also be confirmed [7, 9].

Kendall *et al.* and Nabors *et al.* showed in their series that only half of the extradural meningeal cysts initially exhibit opacification, on delayed studies nearly 100% will opacify [11]. Using these methods, most cases of the CSF communication point between SEMC and intradural subarachnoid have been reported to be found. In some cases in which an accurate localization of the CSF leaking point is difficult or those of no communicating or spontaneous closed cyst, as shown in the current case. To clearly identify CSF communication point, a preoperative work-up using CISS MR imaging technique has been reported to be helpful because of high spatial resolution and excellent contrast between CSF and solid structure. Despite the use of these test regimens in this case, however, the definite communication of the CSF could not be found. SEMC is assumed to occur because of spinal dysraphism or trauma, congenital diverticulum of the dura, or herniation of the arachnoid membrane through a congenital dural defect.

To explain the pathophysiology of cyst enlargement, the active fluid secretion theory and pulsatile CSF dynamic theory have been hypothesized. On histopathologic examination, they have been found to have no secretory functions. Therefore, it can be inferred that a pulsatile CSF dynamic theory is a more plausible theory [10]. If pulsatile CSF is imported to the cyst via a ball valve mechanism and the pressure is lowered, the outlet is closed at the neck of a cyst According to the Laplace law, the body of the cyst exerts a force on the neck sufficient to close the communication, because its radius and wall tension are greater. This mechanism then allows further enlargement, with persistent CSF pulsations. The current case is SEMC of no communication type, which has been reported very rarely. Some authors argued that osmosis caused cystic enlargement, but this theory has often been dismissed because most authors have indicated that the fluid content of cyst is the same as that of CSF. As mentioned above, the mechanism thought to responsible for the communication between the pre-existing cyst and subarachnoid space to be enlarged based on Laplace law via a ball valve mechanism. Over the time, the communication would have been gradually closed and then disappeared.

Also, there was myelopathy in spinal cord with pathologic spinal cord change in this case. We thought this myelopathy with myelomatous change would be secondary change of long standing spinal cord compression made by slow growing cyst.

In asymptomatic patients, the conservative treatment or observation rather than the surgical treatment is recommended. [5, 7] Cases in which the neurologic deterioration or persistent pain due to SEMC are surgically indicated. Principles of the surgical treatment include a complete removal of the cyst, the obliteration of communication pedicle and the watertight repair of dural defect. According to the presence of adhesion

or the location of lesions, however, a complete removal of the cyst may be impossible. There are many cases in which the lesions were extended to the intervertebral foramen. In these cases, a massive amount of the hemorrhage can be concurrently present. In cases of large-sized cyst, the postoperative instability can be problematic. Even with a lack of a complete removal of the cyst, cases in which the treatment outcome was good have also been reported [7, 9]. The most problematic complication following SEMC surgery includes the recurrence of cyst. To prevent this, the communication with subarachnoid space should be identified and its dural defect should be sutured. A simple drainage of the cyst contents might temporarily alleviate the symptoms. As reported in the current case, SEMC of no communicating type can be managed by a single use of the cyst excision without a closure of the dural defect.

4. Conclusion

We have described a rare case of back, leg weakness and gait disturbance in patient with no communication SEMC in dorsal region. In our case, there was a lack of the communicating dural under the surgical vision. This led to a tentative diagnosis of SEMC of no communicating type. With a surgical removal of the cyst, there was an improvement of the symptoms.

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