Case Report

Intradural arachnoid cyst associated with thoracic spinal compression fracture: 7-year follow up after surgery

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Study design: A case report with long-term follow after a surgical procedure.

Objectives: To describe a case of intradural arachnoid cyst secondary to a compression fracture in the thoracic spine and to report long-term results after surgical treatment with hemilaminectomy.

Setting: Osaka, Japan.

Methods: A 68-year-old man who had a traumatic intradural arachnoid cyst following an adjacent compression fracture of T5 underwent surgery. Intraoperatively, after recognition of intradural arachnoid cyst with an echogram following hemilaminectomy, the dural sac was incised and the arachnoid cyst was resected under microscopic observation.

Results: At 7 years after the operation, the low intensity within the vertebral body of the compression fracture had resolved and the spinal cord remained in its normal shape and position. No progression of kyphotic deformity was detected.

Conclusion: A compression fracture of the thoracic spine can be associated with an intradural arachnoid cyst. Microscopic resection via hemilaminectomy for the cyst showed a good result in a 7-year follow up.

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Keywords: arachnoid cyst; intradural; surgical treatment; long-term follow up

Introduction

Intradural arachnoid cyst is a relatively rare condition. These cysts are congenital or acquired. Acquired cysts can be induced by trauma or operation. Even in the cases where cysts develop, usually their cause can not be determined. In this case paralysis of the lower extremities developed 6 months after compression fracture in the thoracic spine induced by a fall from the loading platform. MR images revealed an intradural arachnoid cyst adjacent to the compression fracture. This study describes the 7-year follow up after resection of the traumatic intradural arachnoid cyst.

Case report

A 63-year-old man fell from the platform of a truck and suffered a compression fracture of the fifth thoracic vertebra and was hospitalized for 1 month. Six months after trauma he gradually began to develop difficulty in walking.

On admission to our hospital neurological examination disclosed spastic paraparesis. There was muscle

weakness in both lower extremities and diminished response to pin prick, temperature and vibration below the level of T4. Neurogenic bladder was not observed. According to the American Spinal Injuries Association and International Medical Society of Paraplegia (ASIA/IMSOP) standards, the motor score showed 90 points pinprick score 76 points and light touch score 76 points. The ASIA impairment scale was D. A MRI study revealed an alteration of intensity within the vertebral body of T5, and just proximal to the site the spinal cord was displaced anteriorly, and the subarachnoid space posterior to the spinal cord was enlarged at T2–T4 (Figure 1). CT Myelography demonstrated that the spinal cord itself became flat and the contrast medium was collected dorsally (Figure 2). Based on these findings, we diagnosed the lesion as an intradural arachnoid cyst. Due to the development of gait disturbance, surgery was performed.

Following hemilaminectomy, we attempted to determine the extent of the cyst using an echogram. It demonstrated hypertrophy and adhesion of the arachnoid membrane at the T4 level and just proximal

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Figure 1 Pre-operative MR imaging demonstrates anterior displacement of the spinal cord and enlargement of the subarachnoid space at T2-T4, just proximal to the compression fracture of T5

Figure 2 CT myelography demonstrates flatness of the spinal cord and contrast medium collecting dorsally to the spinal cord

to the adhesive lesion, CSF was congested (Figure 3). After recognition of the precise location of the arachnoid cyst with the echogram, the dural sac was incised. The arachnoid membrane was adherent and proximal to the adhesion, the arachnoid was dilated (Figure 4). The adherent lesion of the arachnoid membrane was excised under microscopic observation. After improvement of CSF flow had been recognized, the dura mater was closed with 5-0 nylon sutures.

Histological examination of the cyst wall revealed hypertrophy of the arachnoid membrane. The patient's postoperative course was uneventful and improvement in muscle strength was observed 1 month after the operation. A postoperative MRI study demonstrated normalization of the subarachnoid space and showed the formerly displaced portion of the spinal cord had returned to normal (Figure 5A). Seven years after

Intraoperative Echogram

Figure 3 Sagittal view of intraoperative echogram demonstrates hypertrophy and adhesion of the arachnoid membrane at T4. Proximal to the adhesive lesion, the CSF is congested and the spinal cord is displaced anteriorly

Figure 4 Following incision of the dural sac, adhesion of the arachnoid membrane was recognized. In the proximal portion of that adhesion, the arachnoid membrane is dilated showing the cystic lesion

surgery, no abnormal neurological findings except for disturbance of deep sensation were observed. MRI at the time showed normal intensity within the vertebral body at the site of compression fracture and no deterioration of thoracic hypophysis. The spinal cord was located in its normal position in the dural sac, in spite of indentation of the spinal cord due to re-adhesion of the arachnoid membrane to the posterior aspect of the cord (Figure 5B).

Discussion

Intradural arachnoid cyst producing progressive myelopathy is a relatively rare condition.¹⁻⁹ Although these cysts can be detected throughout the spine, the thoracic spine is the most frequent location of intradural arachnoid cyst. These cysts may occur sporadically or result from trauma or intraspinal hemorrhage.²⁻⁴ To our knowledge, the first case of

Figure 5 (A) Post-operative MRI reveals normalization of the previously dilated region of the subarachnoid space and normal position of the displaced portion of the spinal cord. (B) MRI 7 years after the procedure shows the normal intensity of the compressed vertebral body and the normal position of the spinal cord despite of slight indentation of the cord

arachnoid cyst of traumatic origin was described by Teng *et al*¹⁰ in 1966. Since then, the number of reports of cysts of a traumatic origin is increasing, consistent with the development of MRI.¹¹ However, previous reports that demonstrated an apparent relationship between trauma and cyst formation are few.^{12–14} In the present case we suspected that the intradural arachnoid cyst might have resulted from trauma, since there was a history of trauma and the site of cyst formation was adjacent to the compression fracture. We considered the mechanism of cyst formation as follows. Trauma induced bleeding and consequent adhesion of the arachnoid membrane. The smooth flow of CSF was disturbed and the subarachnoid space subsequently became dilated. The dilation of the arachnoid space gradually increased and finally resulted in a cystic lesion. As for surgical treatment, total laminectomy is usually undertaken for the resection of the lesion within the spinal canal. However, when total laminectomy is performed, kyphotic deformity already existing at the site of compression fracture has a possibility to deteriorate. On the other hand, hemilaminectomy should not result in postoperative spinal deformity and instability.^{15,16} The rationale for choosing a unilateral approach is to preserve the midline ligamentous structure and the posterior supporting structures on the contralateral site. Hemilaminectomy combined with microsurgical techniques should be given priority over standard laminectomy in the surgical management of extramedullary lesions arising in the spinal canal.^{17,18} For this reason we selected hemilaminectomy which was wide enough to permit incision of the dura mater and resection of the intradural cyst. In a 7-year follow-up

of this case, a normal intensity of the compressed vertebral body and no deterioration of the kyphotic deformity was observed. In spite of slight re-adhesion of the arachnoid membrane, the spinal cord can maintain in its normal position because of no progression of the kyphosis.

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