

Spinal Intradural Arachnoid Cysts : MR Imaging Features¹

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We describe 3 cases of surgically proved spinal intradural arachnoid cysts. Two cases were developed after antecedent tuberculous infection of the central nervous system and in 1 case the cause was uncertain but presumed to be an acquired condition. The location of the cysts were cervical, cervicothoracic, and thoracic spine level respectively. On the magnetic resonance(MR) images, the lesions were presented as widening of subarachnoid space with spinal cord compression and atrophy. The cysts were slightly hyper-(case 1 and 2) or hypo-(case 3) intense to the cerebrospinal fluid on T1-weighted images and isointense with internal heterogeneous dark portion on T2-weighted images. In 2 cases which had previous history of tuberculosis, accompanying syringomyelias were noted. Precise localization and diagnosis were obtained with MR pre-operatively.

Index Words : Spine, MR
Spine, Cysts

INTRODUCTION

Extramedullary intradural arachnoid cysts(IDACs) are uncommon causes of spinal cord compression(1). They can be present as congenital lesions or as the sequelae of trauma or chronic infection(2, 3). Prior to the advent of magnetic resonance(MR) imaging, radiologic diagnosis of IDACs could be obtained by myelography or computed tomography(CT)-myelography (3), but sometimes it was difficult to get a precise preoperative diagnosis(4). With the MR imaging, the diagnosis can be made more accurately and non-invasively(3, 5-7).

We present MR imaging features of three cases of surgically proved IDACs, two of which were accompanied by syringomyelias and developed several months to years after spinal or intracranial tuberculous infection, and another one case of unknown origin but presumed to be acquired condition.

CASE 1.

A 43-year-old male patient was admitted due to quadriplegia which developed five years ago. He had had a tuberculous meningitis 16 years ago. On the neurological examination, right side motor weakness was more prominent than that of the left side. Tones in both lower extremities were increased and slightly rigid. Cervical MR images demonstrated widening of anterior subarachnoid space from C1 to C3 vertebral level and the cord was displaced posteriorly and compressed at the same level. Multiple syrinx cavities were noted from C4 to C7 vertebral body level. On axial T1-weighted images the shape of the widened subarachnoid space was ovoid and convex toward the spinal cord and the signal intensity was slightly higher than that of the cerebrospinal fluid(CSF). The dorsally displaced spinal cord was thin and crescent shaped. The signal intensity of the cystic lesion on T2-weighted images was the same as that of the CSF(Fig. 1). C2 and C3 laminectomies were performed and ventrally located intradural arachnoid cyst was found. When it was punctured, high pressure clear CSF gushed out. After removal of the membrane, the cord restored its volume and position. His symptoms improved postoperatively.

CASE 2.

A 28-year-old male was first admitted due to low back pain. Lumbar MR images revealed features of tu-

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berculous spondylitis and arachnoiditis. Brain CT showed features of tuberculous meningitis. He was operated on the lumbar spine and the diagnosis of spinal tuberculosis and tuberculous arachnoiditis was confirmed. Two months after the operation, voiding difficulty and gait disturbance developed and persisted since then. Neurologic examination revealed diminished pinprick sense below C5 level, and diminished vibration sense below the anterior superior iliac spine level. Both lower extremities showed motor weakness of grade III to IV. CSF cytology and chemistry were not remarkable. MR images obtained on the last admi-

ssion revealed focal widening of ventral subarachnoid space at C5-T1 vertebral level. On axial T1-weighted images the signal intensity of the lesion was slightly higher than that of the CSF. At the level of the thoracic spine, multiple beaded appearance of syringomyelia was noted(Fig. 2). Laminectomy of C6 vertebra was performed and thickened fibrotic changes with arachnoid septations were noted. When opening the cyst, clear fluid gushed out with moderate pressure. Fibrous arachnoid membrane was partially removed and fenestration was done. At the level of the syringomyelia, total laminectomy of T12 vertebra and syringo-suba-

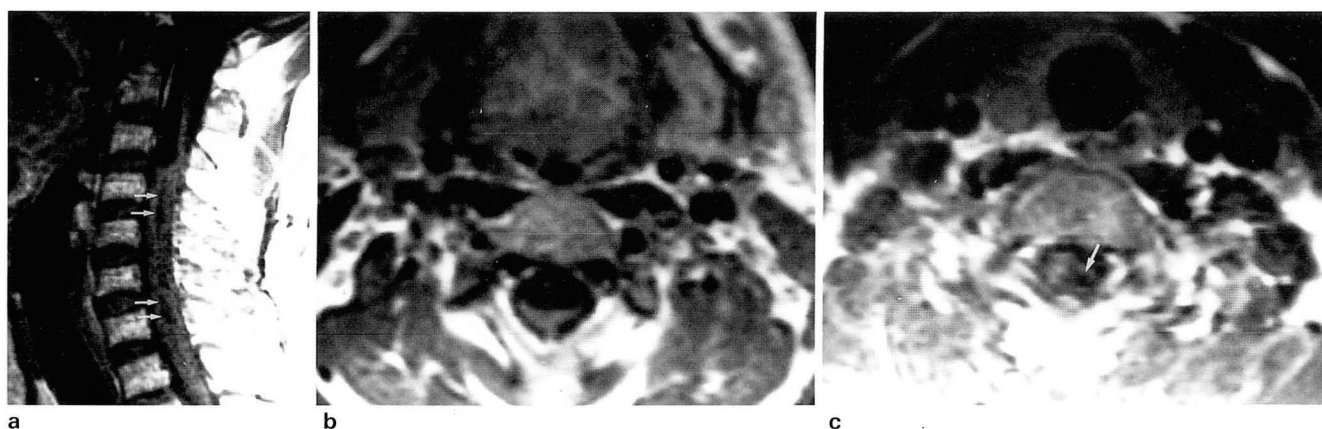


Fig. 1. Case 1.

- a. Sagittal T1-weighted image shows postero-inferiorly convex enlarged CSF space in the ventral spinal canal extending from C1 to C3 level with compressed, displaced spinal cord. Note multiple syrinx cavities in the enlarged cord at C4-C7 level(white arrows).
- b. Axial T1-weighted image at C2 level shows thin, crescent shaped, dorsally displaced spinal cord compressed by a ventrally located cystic lesion. The signal intensity of the cyst is slightly higher than that of CSF and somewhat heterogeneous.
- c. Axial T1-weighted image at C7 level shows eccentrically located syrinx in the irregularly deformed spinal cord(white arrow).



Fig. 2. Case 2.

- a. Sagittal T1-weighted image shows focal widening of ventral subarachnoid space at C5-C7 level and thin, dorsally displaced spinal cord. Note syrinx cavities at C7-T2 vertebral level.
- b. Axial T1-weighted image at C6 level shows widened ventral subarachnoid space with central ovoid increased intensity(white arrows). This may represent pulsation or flow artifacts or increased protein content in the loculated fluid.
- c. Sagittal T1-weighted image at lower thoracic level shows beaded appearance of syrinx cavities within the enlarged spinal cord.

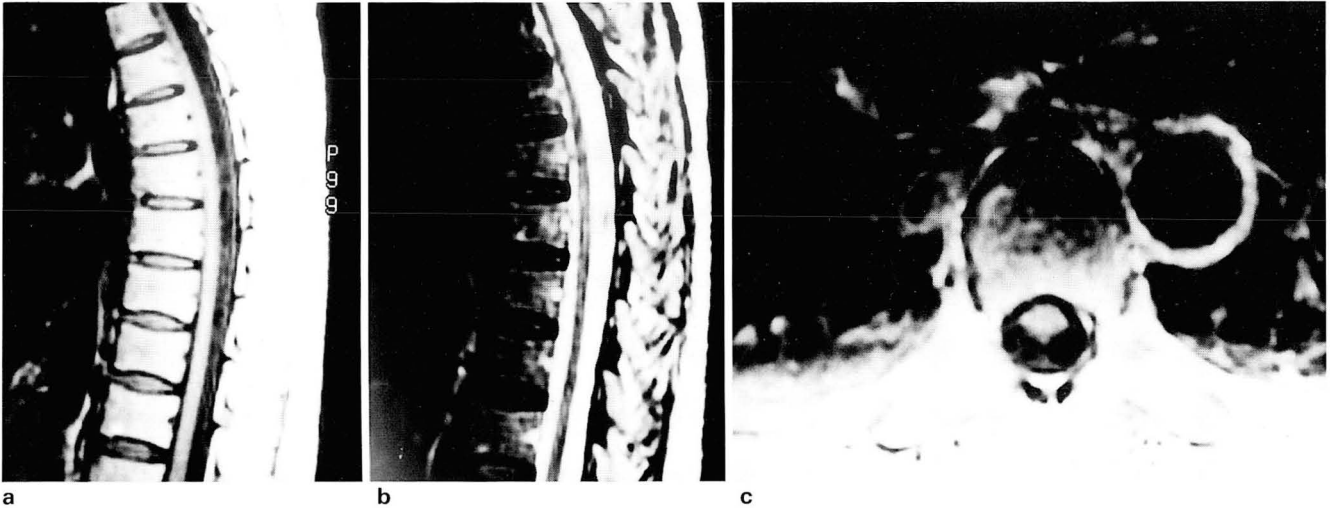


Fig. 3. Case 3

a,b. Sagittal T1(a)-, and T2(b)-weighted images show wide posterior subarachnoid space with heterogeneous signal intensities at mid- to lower- thoracic spine.

c. At T6 vertebral level, axial T1-weighted image revealed wide posterior subarachnoid space with multiple compartments showing variable signal intensity.

arachnoid shunt were performed. His symptoms were stationary after the operation.

CASE 3

A 57-year-old female patient was admitted due to chronic constipation and urinary difficulty developed 4 years ago. She also had both lower extremity paresthesia. Twenty days prior to admission, low back pain started and paresthesia worsened. On the neurologic examination, left lower extremity tone was slightly spastic and hypesthesia was noted below T6 dermatome. MR images showed widening of posterior subarachnoid space at mid- to lower T-spine level with irregular posterior margin of the spinal cord. On T1-weighted axial images, irregular septum-like structure was noted in the posterior subarachnoid space (Fig. 3). On T2-weighted axial images, low signal lesions of variable shape were interspersed in the high signal subarachnoid space but the shape did not correspond with the septum-like low signal intensity structures noted on T1-weighted images. CT myelography failed to reveal this septum-like structure. Laminectomies were performed at T8 and T9 vertebrae and an arachnoid cyst was found from T6 to T12 vertebral levels. The cyst was compartmentalized. Fenestration of the cyst was done. On cyst puncture, fluid gushed out with high pressure and in pulsating nature. Pathologic specimen showed dense connective tissue. Her sensory symptoms improved after the operation.

DISCUSSION

IDACs can occur as congenital lesions or can be caused by inflammatory adhesions of subarachnoid space after infection, trauma, surgery, or blood or con-

trast medium in the CSF(1–3, 8, 9). Most of the congenital IDACs occur in the dorsal subarachnoid space of the thoracic spine and some authors suggested the congenital arachnoid cysts to develop from the septum posticum, which is thin irregularly lamellated membranous partition dividing the posterior subarachnoid space longitudinally(1, 10, 11), but the precise mechanism of development of the arachnoid cysts are not known yet. The arachnoid cysts originating from chronic arachnoiditis are found in either anterior or posterior subarachnoid space(3). The acquired cysts are suggested to be caused by formation of granulation tissue within the subarachnoid space and collection of fluid in irregular compartments(3). In our case 1 and 2, the arachnoid cysts developed as sequelae of antecedent tuberculous meningitis with or without spinal tuberculous arachnoiditis. In case 3, we believe it was an acquired lesion because there were multiple compartments in the cyst and pathological specimen revealed connective tissue proliferation, although no definite predisposing episode was found in her past medical history.

Most of the arachnoid cysts communicate with spinal subarachnoid space through ostiums(1), but sometimes the arachnoid cysts may expand due to check valve mechanism of the ostium or may not communicate with the subarachnoid spaces and cause spinal cord compression and atrophy(4). This makes the myelographic features variable. The myelographic features are block of the contrast medium column, filling defect, delayed filling of the cyst, or only displacement or atrophy of the cord(1, 4, 8, 10–12). Occasionally, a small communicating arachnoid cyst has been missed in a metrizamide study as metrizamide mixes rapidly with the CSF both inside and outside of the thin cyst

wall(4). With CT-myelography, arachnoid cysts could be identified as an intradural extramedullary fluid collection, sometimes of slightly higher density than the remainder of the subarachnoid space, and with compression of the cord(3). Delayed filling of the non-communicating cysts may be seen by diffusion of the contrast material(12). The myelography or CT-myelography has limitations in diagnosing arachnoid cysts in case of block or non-filling of the cysts(3).

MR seems to play an important role in the diagnosis of the arachnoid cysts. In our case 1 and 2, the diagnosis could be made on the basis of MR imaging without further invasive studies such as myelography or CT-myelography. In case 3 in which both MR and CT-myelography were done, MR was more informative than CT-myelography. On the MR images, the presence of the arachnoid cysts is suggested by focal widening of the subarachnoid space, mass effect on the cord, or indentation and irregularity of the cord surface (2, 3, 6, 7). The signal intensity of the arachnoid cysts is usually isointense to CSF(6, 7). In our cases, the signal intensity was variable. They showed slightly higher signal intensity than that of the CSF on T1-weighted images in case 1 and 2. In case 3, lesion showed heterogeneous dark signal on T1-weighted axial images with internal septations. The lesion showed high signal on T2-weighted images with internal irregular signal voids. The variable signal intensity may be due to differences in CSF pulsation within the cyst relative to the subarachnoid space(13) or due to flow in the cysts(3). Other explanation for the variable signal intensity is as a consequence of the loculation of the arachnoid cysts with resulting increased protein content of the cysts (12). In addition to direct visualization of the cysts, MR could reveal cord atrophy, abnormal cord signal, or other associated abnormalities of the spinal cord and spinal canal. Differentiation should be made from dilatation of anterior subarachnoid space by posterior adhesion of the cord or focal cord atrophy. The distinction between these lesions lies in the morphology of the margin of the cord adjacent to the lesion. The adjacent margin of the cord is convex toward the CSF space in case of contralateral cord adhesion or cord atrophy. In case of arachnoid cysts, the cord margin appears concave toward the cysts(14).

The co-existing syringomyelias in our case 1 and 2 also seemed to be the sequelae of the pre-existing tuberculous arachnoiditis. However, other MR features of active arachnoiditis such as enhancement of the thecal sac or leptomeninges(15) were absent and the cytological and chemical studies of CSF were normal. This suggests that the active inflammation had subsided and the arachnoid cysts and syringomyelias were late sequelae of the antecedent arachnoiditis. Syringomyelia associated with arachnoid cyst can also occur after spinal cord trauma or spinal surgery(3, 6).

The exact pathophysiology of development of syr-

ingomyelia in arachnoiditis is unknown, but several explanations were made. One possible explanation is strangulation of blood supply within the arachnoiditis causing ischemia and necrosis and funneling of the CSF into the spinal cord(16). Another explanation is cord fixation by adhesion and squeezing of the necrotic pulp or fluid filled cavity(16).

Treatment of the arachnoid cysts is removal of the cysts or fenestration. The reported results were fairly good(1, 4, 10). However, often the symptom improved little in postinfectious cases(3). In our case 2 the symptom did not improve after surgery. The reason could be explained by the pre-existing severe arachnoiditis, co-existing syringomyelia or atrophy of the cord.

In summary, MR was useful in detecting intradural arachnoid cysts and could reveal the exact location and extent of the lesion. The MR manifestations of the lesion were focal segmental widening of the subarachnoid space with mass effect and indentation of the spinal cord. The cord surface was irregular in acquired IDACs reflecting antecedent arachnoiditis. MR also displayed lesions of the cord such as syringomyelia, atrophy or abnormal signal intensity.

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척추 경막내 지주막낭종: 자기공명영상소견¹

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척추경막내 지주막 낭종은 척수를 압박할 수 있는 드문 질환으로 선천적, 또는 감염 또는 수술 후 후천적으로 발생할 수 있다. 저자들은 중추 신경계 결핵 감염후 발생한 2예와 원인이 분명치 않으나 후천적으로 발생한 것으로 생각되는 1예 등 수술로써 확진된 3예의 척추경막내 지주막낭종의 자기공명영상 소견을 보고하고자 한다. 낭종의 위치는 경추, 경-흉추 경계부, 흉추에 각각 있었고 지주막하강내에서 2예는 복측, 1예는 배측에 있었다. 자기공명영상에서 병변은 척수를 압박하는 국소성 지주막하강의 확장 소견으로 관찰되었고 해당부위 척수는 위축을 보였다. 낭종내 신호강도는 T1강조영상에서 뇌척수액보다 약간 높거나 낮았고, T2강조영상에서는 뇌척수액과 비슷했으나 불균일하였고 내부에 인공물로 생각되는 낮은 신호부위를 내포하고 있었다. 1예에서는 내부에 중격들이 보였다. 결핵감염의 기왕력이 있는 2예에서는 병변 하부 척수에 지주막 낭종위 치와는 떨어져 다방성의 척수 공동증을 보였다. 조영제 주입후 조영 증강되는 부위는 없었다. 전례에서 자기공명영상소견으로 수술전 진단이 가능하였다.

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