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Small Thoracic Disk Herniation without Spinal Stenosis Presenting with Acute Myelopathy: Three Case Reports

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Abstract

We herein describe three patients with thoracic disk herniation (TDH) that presented with acute myelopathy at the Tokyo Metropolitan Neurological Hospital between 2014 and 2021 (age range, 45-76 years; male/female ratio = 1:2), with a focus on the mechanisms underlying their development. All patients had sudden-onset gait disturbance due to acute nontraumatic paraparesis. The specialties of the doctors at the first hospital were neurology and orthopedic surgery. TDH was overlooked at the first hospital, and the patients were referred to our hospital. The TDH in all cases was of the central type; however, since they were small, no spinal stenosis was observed. The key feature of all three cases is the small anterior deformation of the spinal cord, making a vascular etiology for the symptoms more plausible than a compressive etiology. After a follow-up of several months or years, two out of three patients underwent surgery with the use of the transfacet pedicle-sparing approach due to residual symptoms. Intraoperative ultrasonography showed that the spinal cord was anchored to TDH by the dural attachment of dentate ligaments. The physical relationship between the dentate ligaments and TDH may be associated with the vascular cause of the symptoms of small TDH.

Keywords: thoracic disk herniation, acute myelopathy, anterior spinal artery syndrome

Introduction

Symptomatic thoracic disk herniation (TDH) has a significantly lower prevalence than cervical or lumbar disk herniation.[1] Slowly progressive myelopathy with truncal and leg pain is a well-known TDH presentation.[2-4] Acute myelopathy is rarer in symptomatic cases, in which the spinal cord is severely compressed by large lesions with spinal stenosis.[4,5] We herein present three patients with TDH that encountered diagnostic challenges because they presented with acute paraparesis despite having small TDH without spinal stenosis.

Case Reports

Case 1

Approximately 7 years before presenting to our hospital, a 76-year-old woman developed acute paraparesis with paresthesia of the trunk and right leg while walking without traumatic episodes. Paraparesis spontaneously improved within 2 days, enabling the patient to walk again. At the neurology department of another hospital, magnetic resonance imaging (MRI) was performed, and a small TDH was overlooked. The patient also underwent spinal angiography; however, spinal vascular lesions were not detected. After a 7-year follow-up, the patient was referred to our hospital.

Neurological examination revealed monoparesis of the right leg (Manual Muscle Testing [MMT] score of 4/5), sensory paresthesia of the trunk (T10/12 level on the right side) and right leg, and hyperreflexia of the right patellar tendon. The Japanese Orthopaedic Association (JOA) score of the leg, trunk, and bowel function was 6/11 (leg 3, 0; trunk 0; bowel function 3).

Myelographic computed tomography (CT) showed a central-type TDH at the T4/5 level with mild deformation of the spinal cord; however, because it was small, spinal stenosis was not observed, and the dorsal subarachnoid space was preserved (Fig. 1). The spinal canal occupation ratio was 20%. An abnormal T2 high signal change of the

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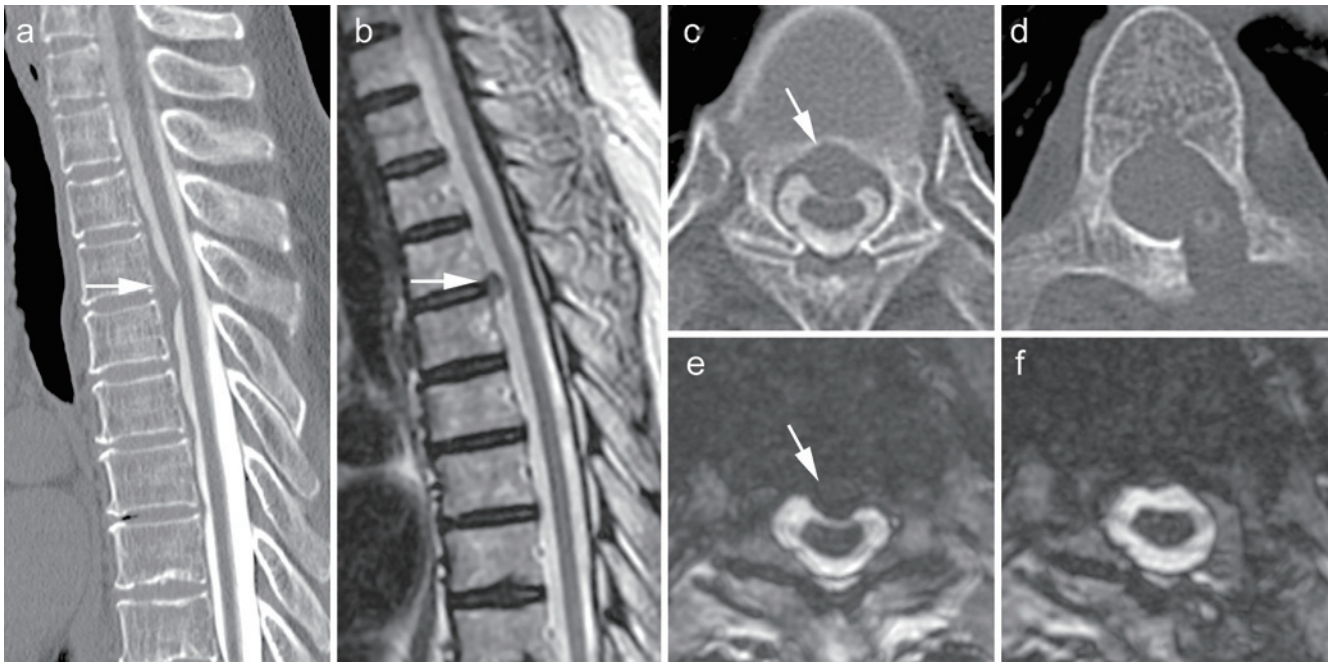

Fig. 1 Case 1.
Preoperative sagittal (a) and axial (c) myelographic CT images showing a noncalcified central-type disk herniation (arrows) compressing the spinal cord at T4/5. Note that spinal stenosis was not evident and the subarachnoid space around the spinal cord was preserved.
Preoperative sagittal (b) and axial (e) T2-weighted MR images showing compression of the spinal cord by the disk herniation (arrows). Postoperative CT (d) showing the transfacet pedicle-sparing approach via hemilaminectomy. Postoperative MR image (f) showing complete removal of the disk herniation and decompression of the spinal cord.

spinal cord and vascular flow voids were not observed on MRI. In the initial examination, the TDH was overlooked as it was only examined on axial, not sagittal, images. The patient was considered for surgical indication due to residual myelopathic symptoms after a 7-year follow-up.

The patient underwent T4 hemilaminectomy and T4/5 medial facetectomy. The TDH was removed from the axilla of the left T4 thoracic root under the guidance of O-arm navigation. The TDH was solid and was removed piece by piece using microsurgical forceps. Spinal fixation was not required as the lateral half of the facet joint remained intact. Postoperative MRI revealed the disappearance of the TDH and decompression of the spinal cord. Motor paresis of the right leg and paresthesia of the trunk improved following surgery. The JOA score also increased from 6 to 9. No recurrence was noted in the 4-year follow-up.

Case 2

A 68-year-old woman presented with acute nontraumatic paraparesis with paresthesia of the bilateral legs when waking up in the morning. Paraparesis spontaneously improved within 3 h, enabling the patient to walk again. At the neurology department of our hospital, the patient underwent thoracic MRI, on which the TDH was identified; however, it was unclear whether it was a corresponding lesion owing to its small size. The patient was referred to the neurosurgical department.

Neurological examination revealed monoparesis of the left leg (MMT score of 5–/5), sensory paresthesia of the bilateral legs, hyperreflexia of the left patellar tendon, and occasional urinary incontinence. The JOA score was 8/11 (leg 2, 1; trunk 2; bowel function 3).

Myelographic CT revealed a central-type TDH at the T9/10 level with deformation of the spinal cord and no spinal stenosis (Fig. 2). The spinal canal occupation ratio was 15%. An abnormal T2 high signal change of the spinal cord and vascular flow voids were not observed on MRI. The patient was considered for surgical indication due to residual myelopathic symptoms after a 4-month follow-up.

The patient underwent T9 laminotomy and T9/10 medial facetectomy. Intraoperative ultrasonography showed that the spinal cord was anchored to the TDH by the dural attachment of dentate ligaments (Fig. 3). The TDH was removed from the axilla of the left T9 thoracic root under the guidance of O-arm navigation and ultrasonography. The TDH was solid and was removed piece by piece using microsurgical forceps. Postoperative MRI revealed the disappearance of TDH and decompression of the spinal cord. Motor paresis of the left leg, paresthesia of the bilateral legs, and urinary incontinence improved following surgery. The JOA score also increased from 8 to 10. No recurrence was noted in the 1-year follow-up.

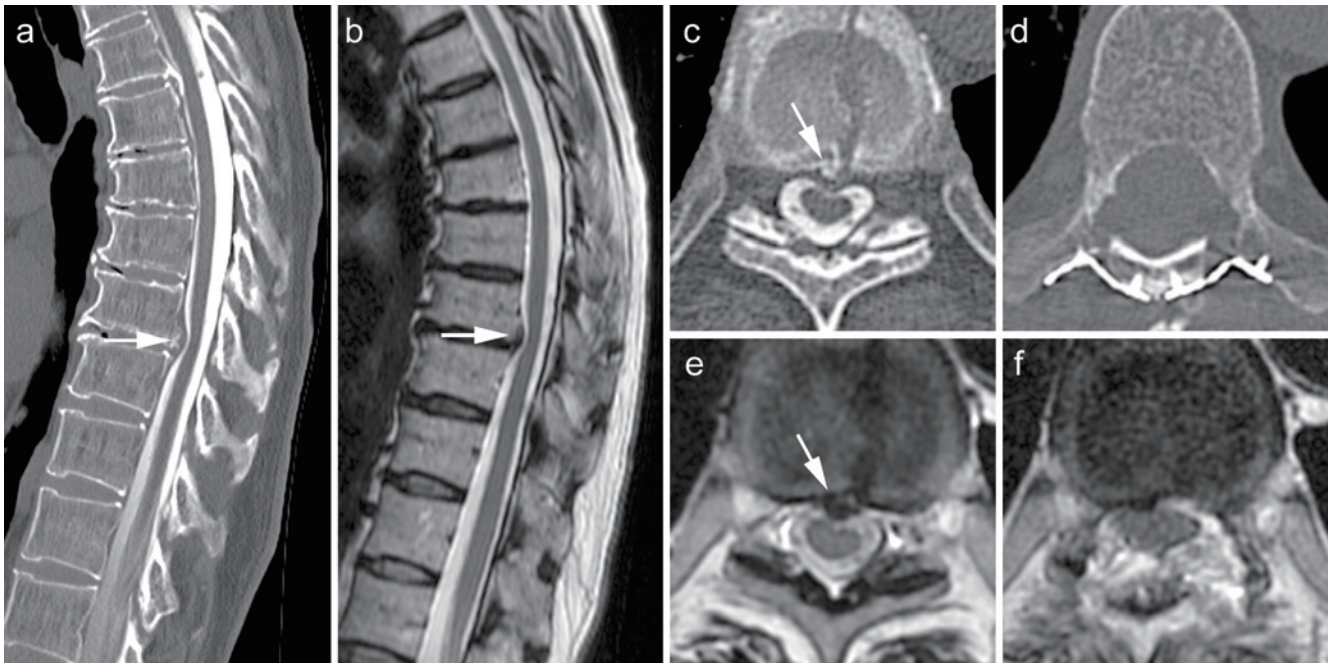

Fig. 2 Case 2.
Preoperative sagittal (a) and axial (c) myelographic CT images showing a calcified central-type disk herniation (arrows) compressing the spinal cord at T9/10. Note that spinal stenosis was not evident and the subarachnoid space around the spinal cord was preserved.
Preoperative sagittal (b) and axial (e) T2-weighted MR images showing compression of the spinal cord by the disk herniation (arrows). Postoperative CT (d) showing the transfacet pedicle-sparing approach via laminotomy. Postoperative MR image (f) showing complete removal of the disk herniation and decompression of the spinal cord.

Case 3

A 45-year-old man presented with acute nontraumatic paraparesis with back pain and paresthesia of the right leg when working. Neurological examination showed monoparesis of the right leg (MMT score of 4/5), sensory paresthesia of the trunk and right leg (T7/S1 level on the right side), hyperreflexia of the right patellar tendon, and urinary urgency. The JOA score was 5/11 (leg 1, 0; trunk 2; bowel function 2). MRI revealed a central-type TDH at the T5/6 level with deformation of the spinal cord and no spinal stenosis, which was similar to the other two cases. The patient did not undergo surgery as neurological symptoms improved spontaneously within 3 months.

Informed consent

Written informed consent from patients was waived as this is a case report.

Discussion

Our three cases were unique due to an acute myelopathic presentation despite small TDH without spinal stenosis (Table 1). The clinical improvements achieved in these cases support evidence showing that small TDH was attributed to acute myelopathic symptoms.

Clinical characteristics and diagnosis of TDH

Symptomatic TDH is rare, accounting for 0.15% to 4% of disk herniation in surgical cases.[1,4] Awwad et al. compared asymptomatic and symptomatic TDH and did not identify imaging features to classify a disk as asymptomatic or symptomatic TDH.[1] More than 80% of patients with TDH commonly present with slowly progressive myelopathy with truncal and leg pain;[2-4] however, acute myelopathic cases of TDH have rarely been reported.

Our three symptomatic cases of small TDH may be explained by the following three reasons. First, transient arterial flow occlusion associated with the attachments of dentate ligaments may have contributed to the development of acute paraparesis. Kahn reported the importance of the dentate ligaments in the spinal cord compression by the herniated disk.[6] Attachments of the dentate ligaments may prevent equal distribution of pressure in anterior spinal cord compression. The greatest stress is distributed on the anterior part of the spinal cord, whereas secondary stress is distributed on the lateral part. The anterior pressure in the cord would directly act on the anterior spinal artery whereas the lateral pressure on the corticospinal tract (Fig. 3).

The second reason may be thoracic kyphosis. Kim et al. reported that in 4,999 patients who were assessed for pulmonary disease by thoracic CT, thoracic kyphosis (a cobb

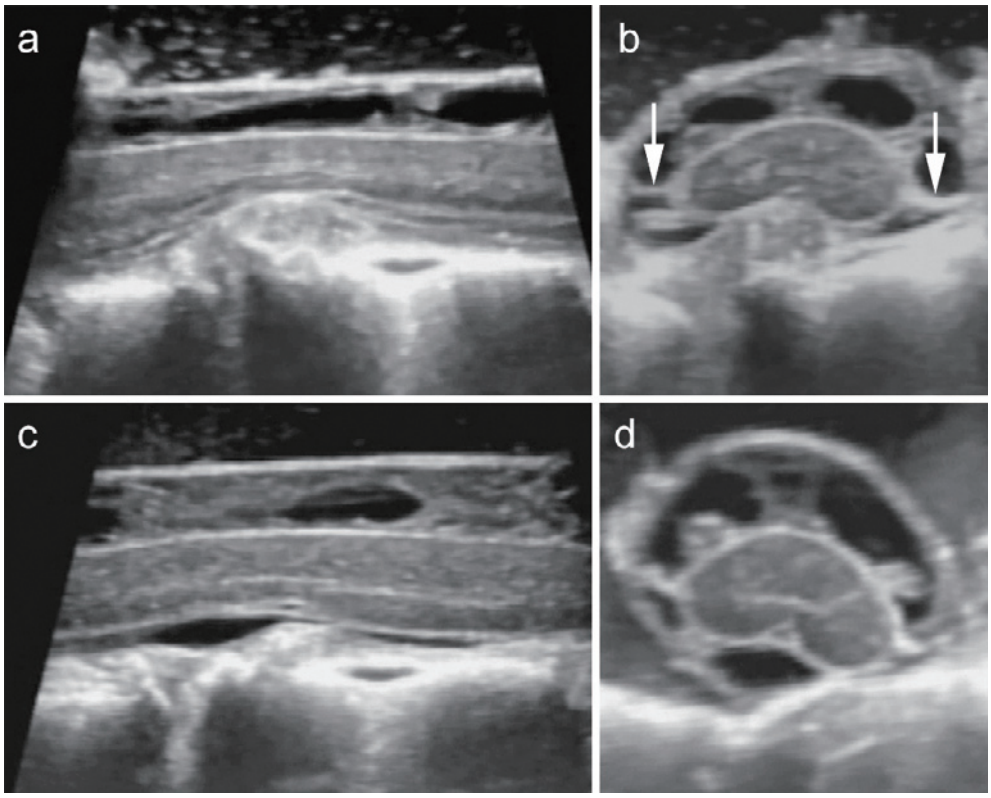

Fig. 3 Case 2.
Sagittal (a) and axial (b) intraoperative ultrasonographic images showing compression of the spinal cord by the disk herniation. Note that the spinal cord was anchored to the disk herniation by the dural attachment of dentate ligaments (arrows in b).
Sagittal (c) and axial (c) ultrasonographic images showing removal of the disk herniation and decompression of the spinal cord.

Table 1 Radiological and clinical characteristics of three patients

Case no.	Age	Sex	TDH level	T3-T12 Cobb angle	CO ratio	Spinal stenosis	Calcification on CT	Myelomalacia on MRI	Time between onset to referral	JOA score	Presentation	Treatment
1	76	F	4/5	28°	20%	No	No	No	7 y	6	Paraparesis	Surgery
2	68	F	9/10	56°	15%	No	Yes	No	4 mo	8	Paraparesis	Surgery
3	45	M	5/6	32°	15%	No	NA	No	5 mo	5	Paraparesis	Conservative

CO, canal occupation; JOA, Japanese Orthopaedic Association; THD, thoracic disk herniation

angle between T3 and T12) was found to be 29.7°±8.9° in the normal control group.[7] However, the three patients described previously had greater thoracic kyphosis, 38.7°± 15.1° (Table 1).

The third reason may be the watershed region of the spinal cord. Reynolds et al. reported that the anterior thoracic spinal cord is considered to have a watershed region at T4-T9.[8] As blood supply from rostral and caudal sources may overlap or be insufficient, a watershed region is more vulnerable to ischemic events, which can lead to conditions such as anterior spinal cord artery syndrome.

Reynolds et al. reported a TDH case of anterior spinal artery syndrome, in which MRI showed a small central-type TDH at the T7/8 level, which produced a focal indentation of the spinal cord without cord displacement or spinal canal stenosis.[8] Guest et al. reported a TDH case of transient anterior spinal artery syndrome, in which the artery of Adamkiewicz was compressed by a small protruding lateral-type TDH adjacent to the lateral neural foramen.[9] In the present cases, the TDH was of the central type, not the lateral type; therefore, our cases were compatible with Reynolds' case. We found that the spinal cord was anchored to the TDH by the dural attachment of dentate ligaments when the TDH protruded adjacent to den-

tate ligaments.[6] Transient anterior spinal artery occlusion may occur when the spinal cord is anchored to a central-type TDH.

TDH may cause acute myelopathy in cases of large herniated disks with spinal stenosis, as previously reported. Cornips et al. described eight cases of TDH presenting with acute (<24 h) paraplegia with Frankel grade C or worse, which accounted for 4% of all symptomatic cases.[5] Radiological characteristics included the dominance of lower thoracic lesions (T9/12), a high canal occupation ratio (more than 40%), spinal stenosis (50%), calcification/ossification (75%), and myelomalacia on MRI (75%). Contrarily, the present cases exhibited the dominance of high thoracic lesions, a low canal occupation ratio (20% or less), no spinal stenosis, and no myelomalacia on MRI.

Nakajima et al. described six cases of TDH presenting with acute (<72 h) paraplegia with Frankel grade C or worse.[4] Radiological findings included high thoracic lesions (T1/4) without calcification or ossification. The affected thoracic level matched the upper line of the sternum. These patients were young (mean age of 29 years) and had a high body mass index (BMI) (mean BMI of 32 kg/m^2). Contrarily, the affected thoracic level did not match the upper line of the sternum, and the BMI was not high (mean 21 kg/m^2) in the present cases.

Surgical indications and approaches for TDH

The surgical indications for TDH have not yet been established for symptomatic TDH. Haro et al. reported two symptomatic cases of TDH presenting with myelopathy that spontaneously improved without surgery.[10] Similar to cervical and lumbar disk herniation, we consider it necessary to follow-up symptomatic cases for at least 3 months.[2,3] Among the several surgical approaches reported in the literature, the transfacet pedicle-sparing approach has the fewest surgical complications.[11,12] In our two surgical cases, this approach resulted in good outcomes without minor or major complications. Intraoperative ultrasonography was also useful for confirming complete removal of the TDH and decompression of the spinal cord in the present cases.[11]

Conclusions

It is noteworthy that acute myelopathy may be caused by a small TDH without cord displacement or spinal stenosis. Spinal cord ischemia may occur when the spinal cord is anchored to a central-type TDH by the dentate ligaments.

Conflicts of Interest Disclosure

The authors declare that they have no conflict of interest.

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