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Intraradural Herniation of Intervertebral Disc at the Level of Lumbar 1-Lumbar 2

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ABSTRACT

Intradural disc herniation is a serious and rare complication of intervertebral disc rupture. The preoperative diagnosis of intradural disc herniation is still difficult despite new neuroradiologic investigation possibilities including computerized tomography and magnetic resonance imaging and it is usually diagnosed by during surgery. Here we present an intradural disc herniation case at the level of L1-L2 with accompanying significant myelopathic neurologic deficits. A 50-year-old female patient was admitted to the hospital with pain and weakness in both legs. Her neurological examination revealed paraparesis. Magnetic resonance imaging showed an extruded disc hernia of central localization at the L1-L2 level. She underwent total laminectomy at the level of L1-L2 and her intradural disc fragment was extirpated by microsurgical methods.

KEY WORDS: Lumbar Disc Herniation, Intradural Disc, Magnetic Resonance Imaging.

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INTRODUCTION

Intradural herniation of intervertebral disc is a very rare complication of spinal degenerative process and comprises 0.26-0.30% of all disc herniations (1,2). It was first defined by Dandy in 1942 (3). It is frequently seen in the 5th decade and most of the reported cases (76%) are males (1). Intradural disc herniations (IDH) are seen later in life compared with extradural disc herniation. 92% of reported IDH's are seen in the lumbar, 5% in the thoracal and 3% in the cervical region. 0.04-0.33% of disc protrusions in the lumbar region are located intradurally (1,2). The site most frequently affected is L4-5 (55%), followed by L3-4 (16%) and L5-S1 (10%) (4). Although preoperative computed tomography (CT), myelography and magnetic resonance imaging may help identification of the lesion, it is difficult to identify the lesion prior to surgery (2).

We present here an IDH case at the level of the L1-L2 with myelopathic neurological deficit diagnosed during surgery. Her symptoms resolved significantly after the operation.

CASE REPORT

A 50-year-old female patient presented to the Physical Treatment and Rehabilitation Outpatient Clinic with lumbago and pain in her lower extremities of 4 years duration as well as accompanying weakness of 10 days duration in both legs. Her neurological examination revealed paraparesis with no Lasègue's sign at 90°. She denied urinary and bowel incontinence but had significant lumbago intensified with motion. She had undergone traction with physical treatment and rehabilitation at another center for her lumbago 2 years ago.

Degenerative osteoarthritic alterations at all levels, decreased disk height at L1-L2 level, vacuum phenomenon and outburst of the disc to all directions were seen in non-contrast MRI. Extruded disc hernia of central localization, 12x15 mm in size, isointense with the intervertebral disc and extending to posterior part of spinal canal in T1- and T2-weighted images was seen at the same level (Figure 1A-B). T2-weighted axial images showed the mass lesion at the L1 level (Figure 2). Contrast-enhanced MRI did not reveal contrast enhancement at the extruded disc but the surroundings were contrast enhanced (Figure 3).

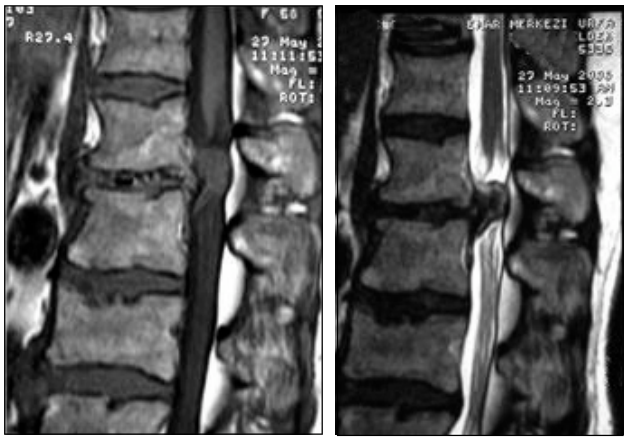

Figure 1: Image of extruded disk hernia of central localization, 12x15 mm in size, isointense with the intervertebral disk, extending to the posterior part of spinal canal at the level of L1-L2 in T1- (Figure 1A) and T2-weighted (Figure 1B) images.

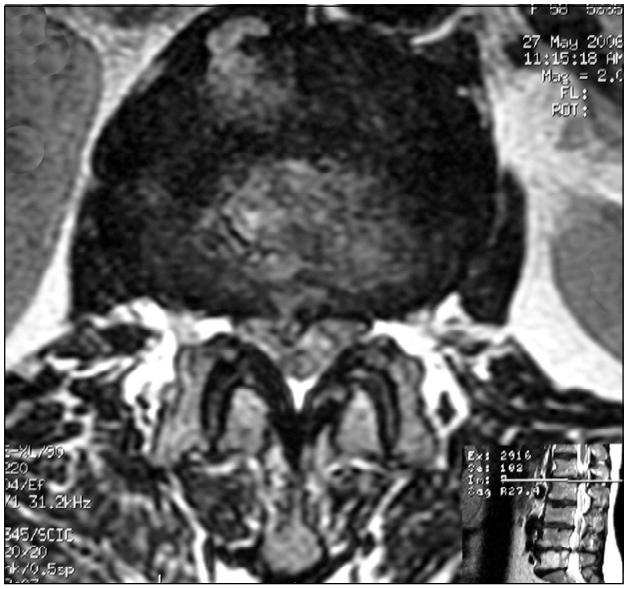

Figure 2: T2-weighted axial image shows the mass lesion at the level of the L1-L2 compressing the cauda equina.

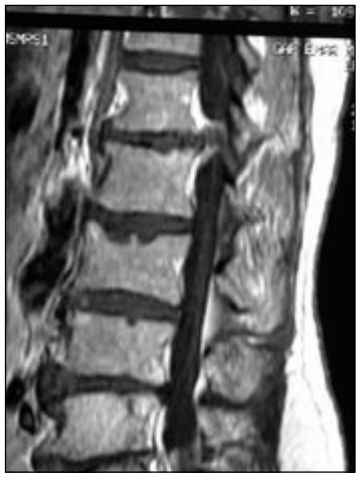

Figure 3: In contrast-enhanced MRI, there was no contrast enhancement in the extruded disk but the surroundings did enhance.

She underwent total laminectomy at the level of L1-L2 at the prone position. There was no significant extradural lesion after laminectomy. A hard mass could be felt along the dura. There was an intradurally positioned mass 1x1 cm in size consistent with an intervertebral disk. This mass pushed the rootlets posteriorly and caused significant traction. The thin arachnoid capsule over the disc mass was incised and the fragment was extirpated by microsurgical methods. There was a tear at the ventral wall of the dura. There was arachnoid membrane over the disc material and the rootlets were attached strictly to this membrane, especially at the right side. The disc material was highly calcified and contained bony particles. Dura was closed water-tight at the end of the operation. Pathological investigation of the specimen revealed degenerated cartilaginous tissue with mixoid degeneration and profuse edema. The patient had no complication and her pain resolved significantly. She was discharged 1 week after the operation.

DISCUSSION

Intradural herniation of intervertebral disc is a rare condition that is generally diagnosed during surgery. The ruptured disc fragment may rarely migrate intrathecally (5). Klop et al. have reported a disc herniation case located between inner and outer dura leaflets (6).

Mut et al. suggested a classification for IDHs based on the spinal dural anatomy as follows: Type A: herniation of disc material into the dural sac

(intradural disc herniation); Type B: herniation of disc material into the dural sheath in the preganglionic region of the nerve root (intraradicular disc herniation) (7).

Perforation of PLL and dura matter is required for IDH to occur. Dandy claimed that acute pressure of the protruded extradural disc may erode and penetrate the anterior wall of the dura mater (3). Lyons and Wise also supported the idea of dural penetration by sustained pressure (8). According to the reported cases in the literature, IDH is found more in areas where there is increased spinal movement. In this case, protrusion of extradural disc may result in chronic irritation and tear of dura matter secondary to vertebral movement. The physiological and pathological features of this entity have not been fully elucidated. Nevertheless, some probable causes have been proposed to contribute IDH occurrence: 1. Adhesions between the annnulus fibrosus, posterior longitudinal ligament, and dura mater, 2. Congenital narrowing of the spinal canal with less epidural space, 3. Congenital and iatrogenic thinness of the dura mater (1,4). The most blamed factor among these is adhesions. These adhesions also serve as a barrier to lateral migration of the fragment, forcing it directly dorsally through the annulus-PLL-dural layer.

The clinical features of lumbar intradural or intraradicular disc herniations generally include long-lasting low-back pain and signs of the cauda equina syndrome (1). Intradural herniation above the conus medullaris seems to lead to neurological dysfunction more rapidly. However, there may be cases where the disc protrudes intradurally to compress a single root and show signs only of root compression (1).

Most of the reported cases in the literature had symptoms of more than 1 year duration. In our patient, the time between symptoms onset and surgery was 4 years. These findings seem to support the theory that interdural sequestration may simply be an intermediate stage in the process of complete transdural migration of a disk fragment. Nevertheless, there are some reported cases that had a symptom duration of 5 weeks from onset to surgery (6). Prognosis is related to the duration of the symptomatology, presence of the cauda equina syndrome and the complete removal of the herniated material. Long duration of symptoms is a negative prognostic factor and surgical intervention must be performed urgently, especially in the presence of the cauda equina syndrome (1,4).

MRI, non-contrast CT, myelography and CT myelography have increased the possibility of making a proper diagnosis (9). Diagnosis of disc herniation results in imaging of the calcified disc within the spinal canal in direct radiography and deformity in techal sac in Pantopaque myelography (10). Lumbar IDH lesions are usually visualized as a complete block in myelography (1,2). Although CT and postmyelogram CT can provide valuable information, they are not able to identify intradural disc rupture reliably. In addition, although there are some reports indicating that CT and myeloCT are more valuable in demonstrating IDH compared to MRI (9), some authors have declared that MRI is the most reliable method for the diagnosis of IDH (11,12). Wasserstrom et al. and Whittaker et al. reported intradural disc herniation with a ring enhancement pattern on MRI with gadolinium (13,14). Our case also demonstrated ring enhancement.

Based on the MRI findings, neurinoma, meningioma, schwannoma, ependymoma, and dermoid should be considered in the differential diagnosis. Lymphoma, meningioma, metastasis, and herniated disc are less likely but also considered in the differential diagnosis. Neurinoma and meningioma both have homogeneous enhancement that is clearly different from the ring enhancement of intradural herniations. Noncontrast MRI has been inadequate to demonstrate these lesions. However, a review of the literature reveals that MRI with gadolinium would be useful in such cases.

Preoperative diagnosis may be difficult due to the variable clinical presentations and radiological appearance of IDH cases. However, the preoperative recognition of an intradural herniation is important as it influences the operative strategy.

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