MRI Diagnosis Of Intradural Lumbar Disc Herniation. Report Of Three Cases With Review Of Literature.

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Abstract

With recent improvements in magnetic resonance imaging (MRI) it has become possible to preoperatively diagnose intradural migration of disc fragments. Apart from the classical ring enhancement of the intradural fragments on gadolinium contrast MRI, there are several other imaging features that may help in diagnosis of this rare condition. We report three cases of intradural lumbar disc herniation and review literature with emphasis on MR diagnosis.

INTRODUCTION

Intradural disc herniation is a rare complication of intervertebral disc hernia. Since the first description by Dandy in 1942, over hundred cases have been reported in literature. The diagnosis of intradural disc herniation in most case reports has been an intra-operative finding. However with recent improvement in magnetic resonance imaging, mainly in terms of higher resolution, it has become possible to preoperatively diagnose intradural migration of disc fragments thus limiting the chance of negative surgical exploration. We report three cases of intradural disc herniation with a special emphasis on the preoperative MRI findings and its differential diagnosis.

MATERIALS

CASE 1

A 70 years old patient presented with a sudden onset acute flaccid paraplegia of six hours duration. He had experienced a sharp stabbing pain in the mid-dorsal spine that radiated along the lower ribs, while attempting to pick up an object from the ground. The intensity of pain had spontaneously and significantly reduced over six hours. He had never received treatment for significant back pain. The patient was unable to move both lower limbs voluntarily. There was significant hypoesthesia below the groin crease. The deep tendon reflexes were absent in both lower extremities. He was catheterized on admission to decompress an insensate and distended bladder.

A magnetic resonance imaging (non contrast) was performed ten hours after the episode. The T2 sagittal image (Figure 1a) showed a ruptured posterior annulus at D12-L1 level with extruded nuclear material into the spinal canal. The PLL showed an abrupt interruption at the level of the disc suggesting an intradural location of the disc fragment. T2 axial image (Figure 1b) showed a right paracentral fragment lying within the dural sac. The conus was pushed to the left side. Cord edema was noticed as a hyper intense signal within the cord substance on the T2 weighted MRI image (Figure 1a).
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Figure 1
Figure 1: Case 1 – a) T2 sagittal image showing ruptured posterior annulus at D12-L1 level with extruded nuclear material. The PLL showed an abrupt interruption at the level of the disc. b) T2 axial image showing a right paracentral fragment lying within the dural sac.

CASE 2
A 58 years old man presented with cauda equina syndrome of 10 days duration with retention of urine, constipation and severe left lower limb radiculopathy. He had suffered from low back pain and multiple episodes of left lower limb radiculopathy for the past 2 years for which he had taken conservative treatment. He had no motor deficit but had sensory hypoesthesia in the L5 root and perianal distribution. Knee jerk was normal and bilateral ankle jerks were absent. Local spinal exam was normal except for a paraspinal muscle spasm.

MRI (non contrast) study was performed. The sagittal images (Figure 2a) showed a disc herniation at L4-5 level with a rupture of posterior annulus fibrosus. The disc material was seen to be migrated in the cephalad direction. The image also clearly demonstrated a continuity of the fragment with the intradiscal nuclear material. The continuity of the PLL was disrupted at the disc level without its actual elevation. c) T2 axial and d) T1 axial images showed the disc material completely enclosed by the dural sac indicating an intradural location of the disc hernia.

CASE 3
A 68 years old man presented with left lower limb radiculopathy with an ankle dorsiflexor weakness of 1 month duration. The patient had a high steppage gait with grade 1 power in the left ankle dorsiflexors. Sensory hypoesthesia was found in L4 root distribution. Left knee jerk and Achilles jerk were depressed. Patient had normal bowel and bladder function. Local exam revealed a spinal list on the left side.

MRI gadolinium (Gd-MRI) contrast study was performed (Figure 4b). The findings which suggested the disc herniation was intradural were: 1) Mass lesion in front of the L3-4 disc 2) No lifting up of the PLL 3) Peripheral enhancement of the fragment.
Magnetic resonance imaging confirmed the diagnosis preoperatively in all three cases. The key features, which clinched the diagnosis of intradural disc herniation, were:

1) Fragment located in front of the disc space

2) Ruptured posterior annulus fibrosis with continuity of the herniated material with intradiscal contents.

3) Abrupt interruption of the PLL at the disc level. In extradural disc hernia the PLL is lifted off the posterior aspects of the adjacent vertebral bodies.

4) On axial images the disc fragments are completely contained within the limits of the dural sac.

5) Peripheral enhancement of the herniated material on Gd-MRI.

All patients underwent surgery. Laminectomy was performed at D12, L4 and L3 level in Case 1, 2 and 3 respectively. The intraoperative findings (common in all three cases) were as follows: 1) Absence of extradural herniated fragments 2) Swollen dura 3) Non retractile thecal sac due to the large herniated fragment inside the dura. In case 1, in addition a CSF leak was found arising from the anterior aspect of the dura. In case 2, the dural sac was transparent enough to visualize the floating disc fragment intrathecally.

In all three cases intentional durotomy was done to excise the disc fragments. In case 1, the ventral dural rent had ragged edges and was adherent to the posterior annulus and the posterior longitudinal ligament. No attempt was made to repair it. The ventral dural rents were not found in case 2 and 3. A watertight closure of the durotomy was done after complete removal of the disc fragments. The fragments were confirmed to be disc material on histopathology examinations.

RESULTS

CASE 1

At four years follow-up the patient had normal sensations and bladder dysfunction in the form of urge incontinence. Motor recovery started in the first post-operative week in the right knee extensors but did not reach an ambulatory status (grade 2/5) at the final follow-up. This case has been reported in the year 2008. Readers are requested to read the article for further clinical details [1].

CASE 2

The patient was relieved of the radiculopathy. Bladder dysfunction required clean self-intermittent catherization for 5 months after which the patient was able to void with abdominal pressure. At the latest follow up of 2 years patient complains of intermittent back pain, poor stream while micturition and constipation.

CASE 3

The patient was relieved of the radiculopathy with improvement in the muscle power to grade 4. Gait normalized and at the latest follow up of 1.5 years has no complaints.
DISCUSSION

Herniated intervertebral disc presenting as an intradural migration is rare with an incidence up to 0.33% [2]. Average age of presentation is usually in the fifth and sixth decade. Males represent 76% of the reported cases [3].

In majority of cases, the lumbar spine is affected and the commonest levels being L3-4 and L4-5. The lumbosacral region has lower incidence of these complications which can be due to narrower canal at higher lumbar regions [4]. Cervical and thoracic disc herniations have produced compressive myelopathy [3] [4].

The pathogenesis of intradural herniation is unclear. In most instances the dural erosion may be precipitated by intradiscal calcification, previous herniation, adhesions between the dura and the posterior longitudinal ligament [5], spinal canal stenosis [6]. Adhesions between dura and PLL were found in all three cases presented here. None of the patients had a previous lumbar spine surgery.

There are no differentiating clinical signs between an extradural and intradural disc herniation although certain clinical findings are more commonly found in the latter. The severity of neurological deficit is higher in intradural disc herniation [6,7]. Cauda equina syndrome occurs significantly more frequently (30%) as compared to higher level extradural herniation (<1%) [7]. Almost all authors report several-year history of chronic back or leg symptoms prior to the episode of acute exacerbation [8]. In our series 2 patients (Case 2 and 3) had a chronic history of back pain and radiculopathy before they presented with severe deficits. Past history of lumbar spine surgery is also a risk factor for developing this complication as previous surgery predisposes to scarring and adhesions between the PLL and ventral dura. DAndrea et al reported an incidence of 33% of previous surgery in their series of 7 patients [8].

Positive diagnosis of intradural disc herniation could be obtained only through surgery before routine use of advanced imaging [9]. There are no pathognomonic signs on myelogram, CT and non-contrast MRI for the diagnosis of this lesion. However, authors have reported a striking association of intradural disc herniation and the presence of gas within the spinal canal on CT scan [10,11]. The presence of air within the spinal canal on CT scans, and of an intradural mass on MRI, is almost diagnostic of a herniation rather than a tumor [10].

The first descriptions of intradural disc herniation [5,12] with MRI reported the presence of an intradural fragment corresponding the disc space. Such presentation though suggestive, is not specific for an intradural disc herniation. Konishi et al have reported a cauda equina tumor (Schwannoma) mimicking an intradural disc [13]. Disc bulges and protrusions are frequent findings on MRI and therefore coexistence of intradural masses next to the disc herniation is not enough to diagnose intradural disc herniation.

Wasserstrom first demonstrated peripheral disc enhancement of intradural disc herniation on Gd-MRI in 1992 [9]. The rim enhancement was attributed to granulation tissue around the avascular fragment, which was confirmed on histopathology. Since then many authors have demonstrated ring enhancement of intradural disc fragments differentiating it from a variety of intradural tumors [14-17].

Ring enhancement has similarly been reported in extradural herniated discs. Extradural disc that show ring enhancement have been shown to spontaneously disappear or reduce in size in 75 to 100% of cases [18]. Similarly, authors have reported spontaneous resorption of enhancing intradural disc fragments [11,19]. The possible mechanism proposed is infiltration of peripheral blood vessels, migration of phagocytes and finally, phagocytosis of disc material [11,19]. However, it is common for intradural disc herniations to present with severe pain and deficits due to which conservative management usually is not an option available.

Any of the intradural cauda equina tumors can mimic the appearance of intradural disc herniation. The differential diagnosis includes schwannoma, meningioma, ependymoma, epidermoid and dermoid tumors. In contrast to intradural disc herniation, these lesions present with gradually progressive leg pain, back pain or neurological deficits as they are slowly growing lesions. Gd-MRI helps to establish a differential diagnosis. Schwanommas and meningioma are the most frequent intradural tumors. Schwanommas are isointense on T1 and hyperintense on T2 whereas meningiomas are isointense on both T1 and T2 wt images. These lesions demonstrate a homogenous enhancement pattern in Gd-MRI. Ependymomas are hyperintense on T2 (unlike disc material) and enhance markedly. Although unlikely, there have been two reports of intradural disc herniation demonstrating increased signal intensity on T2-weighted images [20,21]. Epidermoid and dermoid tumors do not enhance on contrast imaging.
Non-enhancing intradural mass however does not rule out an intradural disc herniation. Granulation tissue that forms around the disc fragment is a chronic process and is not seen in acute disc herniation. Therefore in acute presentation of the intradural disc herniation, MRI is unlikely to reveal ring enhancement [9,15]. In such situation the differential diagnosis is narrowed down to a possibility of epidermoid or dermoid tumor.

However there are certain MRI findings on non-contrast imaging which suggest an intradural disc herniation rather than a tumor. Ruptured posterior annulus with continuity of the intradural herniated material with the intradiscal contents is an indicator of intradural herniation. Authors have reported abrupt interruption of PLL at the level of the herniation as a sign of intradural migration of disc fragment [17]. Sometimes, in cases of migrated intradural fragments the herniated material can be seen to be completely enclosed in the dural sac. In spite of these MRI findings, the preoperative diagnosis is difficult due to limited use of Gd-MRI and rarity of intradural herniation.

In the absence of definitive imaging findings on non-contrast MRI, intradural disc fragments can be easily overlooked during surgery. If the intraoperative findings do not match the extent of compression seen on the MRI, a suspicion of intradural migration of disc fragment should be entertained and a radiograph is advisable to rule out wrong level surgery Intraoperatively a tense swollen dura in the absence of extradural herniation is the only tell-tale sign. CSF leak from the ventral aspect of the dura is sealed due to adhesions and may become apparent only on probing [22]. In Case 2 the intradural fragment was actually seen floating in the dural sac (figure 3a). Some authors have recommended intraoperative ultrasonography to help identify loose intradural fragments [23].

Chronicity of the disease and severity of the neurological compromise are two adverse prognostic factors Delay in surgical decompression and presence of cauda equina syndrome seems to lead to poor outcomes [8,24].

Preoperative imaging especially Gd-MRI can be helpful in the diagnosis of intradural disc herniation. However we believe that a high index of suspicion for retained intradural fragments, in the presence of a tense, non-retractile dura and a negative extradural exploration, will help to avoid a missed diagnosis.

References

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