Dorsal Extradural Lumbar Disc Herniation Causing Cauda Equina Syndrome: A Case Report and Review of Literature

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INTRODUCTION
A lumbar disc sequestration is penetration of the posterior annulus and the posterior longitudinal ligament (PLL) and migration of intervertebral disc within the spinal epidural space. The majority of ruptured or sequestrated disc fragments migrate in upward, downward, or lateral directions owing to anatomic properties of the anterior epidural space. Dorsal extradural sequestration of disc herniation is very rare, and there are few reported cases of the migration of lumbar disc herniation to the dorsal surface of the thecal sac. We present an unusual case of dorsal extradural sequestration of lumbar disc herniation. We review the literature and discuss the difficulties in the diagnostic evaluation of such a migrated disc fragment.

CASE REPORT
A 73-year-old man presented with severe leg pain, acute low back pain, and urinary incontinence. The patient could not extend his low back due to severe radicular leg pain. Reflexes were absent at the knee and ankle, and motor strength of ankle dorsiflexion and great toe dorsiflexion were 3/5 in both legs. There was bilateral lumbo-sacral hypesthesia in the S1, S2, and S3 dermatomes. Rectal tone was decreased but the patient had no bowel bladder disturbances.

Magnetic resonance (MR) imaging showed a large isointensity lesion at the L4-L5 level on the T2 axial image (Fig. 1), indenting circumferentially the thecal sac from lateral to posterior of the thecal sac. We present an unusual case of dorsal extradural sequestration of lumbar disc herniation. We review the literature and discuss the difficulties in the diagnostic evaluation of such a migrated disc fragment.

KEY WORDS: Lumbar disc herniation · Dorsal · Intradural · Migrated.
of the next below the level. T2-weighted sagittal image (Fig. 2A) demonstrated that a low intensity lesion within the thecal sac compressed film terminale. It seemed to be intradural disc herniation. Gadolium enhanced T1-weighted sagittal image (Fig. 2B) revealed the lesion well defined with marginal enhancement. On MR myelography (Fig. 3), there was a complete block of the signal at the L4-L5 level. On the axial computed tomography (CT) scan, at the L4-L5 disc level, there was an iso-intense lesion that appeared to compress the thecal sac from the right side (Fig. 4). The radiological scans gave a probable diagnosis of either an intradural disc herniation or less likely, a posterior extradural disc herniation. The authors planned bilateral laminotomy and if needed, durotomy. The surgery was performed under neural intraoperative monitoring (NIM). Intra-operatively, after removing the ligamentum flavum, we noticed some irregular and hard material encased in veins; it was enveloping the thecal sac from behind (Fig. 5). After removing the material slowly with the help of a nerve hook, we found it to be the herniated disc, which was covered in veins. After the removal, the thecal sac was relatively free and fluctuating again.

After surgery, the patient was relieved of the pain, and discharged without further complications. The patient felt the strength of the lower extremities improving at 3 months follow-up.
DISCUSSION

Sequestrated disc fragments account for 28.6% of all symptomatic disc herniations. Herniated disc fragments are known to migrate within the spinal canal in many directions, including cranial, caudal, and lateral. However, posterior epidural migration of an extruded disc in the lumbar region is relatively rare because the PPL at the level of the concave vertebral bodies forms the anterior epidural space between itself and the periosteum of the vertebral bodies; this is separated in the midline by the septum posticum, which prevents the movement of the herniated disc from one side to the other. At the level of the disc, the PLL is firmly adherent to the posterior annulus and is attached to the lateral membrane, also called peridural membrane, which extends medially from the lateral edge of the PLL to the lateral wall of the spinal canal; it limits the movement of the extruded disc fragment beyond the postero-lateral corner of the dural sac. This makes it difficult for a disc to herniate posterior to the dural sac.

Due to the general nature of disc herniation being in either upward or downward direction, there are very few reports of posterior extradural disc migration since it is not the first impression that is taken into account. The earliest report of such an occurrence was by Lombardi in 1973.

It is difficult to diagnose posterior extradural disc herniation. MR imaging is now accepted as the gold standard for the evaluation of lumbar disc herniation and should be done as a baseline investigation to diagnose the herniation and the migration if present. In the case of an extradural herniation the herniated disc fragment will appear hypo-intense on the T1-weighted image and hyperintense on T2 images, which was similar to our case. The posterior compression of the dural sac may sometimes lead to an impression of either an epidural abscess or a benign epidural tumor. If diagnosis is uncertain, a Gadolinium-enhanced MR imaging scan should be performed if possible.

Pathologically, when a disc herniates posteriorly, it squeezes along with it epidural fat; the epidural fat being vascular, the vessels are concentrated and encircle the herniated fragment. Inflammation due to vasoactive factors released by the herniated disc can develop hypervascularization around the fragment. These phenomena lead to hyperhydration of the fragments, and signal changes that are similar, but not identical to, the cerebral spinal fluid (CSF), thus leading to rim enhancement findings after Gadolinium injection. Our literature review supports these MR imaging findings in most cases. The enhanced signal on Gadolinium-injected scans may sometimes result in a misleading differential diagnosis.

The differential diagnosis of an enhanced lesion can be: arthro-synovial cyst, meningeal cyst, ligament cyst, perineural cyst, abscess, benign tumor, or hematoma. However, meningeal cyst doesn’t show Gadolinium enhancement and shows a similar signal to CSF. Synovial cyst from the facet joint is centered on the facet, and rim enhancement is often seen. Tumorous conditions have different patterns of enhancement, showing no rim enhancement.

In our case, the indicating factor towards a disc herniation was that the patient had a sudden development of symptoms and otherwise his general health was good, apart from his pathological reports being normal. All of these factors pointed towards the less likely possibility of epidural tumor or abscess. The literature review shows 20 cases of cauda equina syndrome associated with dorsal extradural lumbar disc herniation since the first report by Lombardi in 1973. All of these cases exhibited rim enhancement of the extruded disc. A reason for such a herniation could be due to what Kuzeyli et al. has suggested, that heavy labor, traction, spinal manipulation, and conditions of hypermobility may predispose the disc, to posterior migration of the fragments. As seen by us in our review of similar cases most of them have history of some trauma before the onset of symptoms, which support this hypothesis. Also in all cases, prompt surgery by an open procedure with complete decompression provided good results.
CONCLUSION

We reported a case of dorsal extradural lumbar disc herniation mimicking intradural disc. Determining the location of lumbar disc herniation, either extradural or intradural, is a diagnostic challenge. Where there is concern about the diagnosis, appropriate scans and different diagnostic modes should be utilized. Any compressive lesion causing cauda equina syndrome should be decompressed on an urgent basis.

• Acknowledgements

This study was supported by a grant from the Wooridul Spine Foundation.

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