Intradural Lumbar Disc Herniation: Review of Literature with a Case Report

Dr. Hitendra Uike¹, Dr. Surendra Jain¹, Dr. Devendra Purohit², Mch, Dr. Sanjeev Chopra³*, Mch, Dr. Somnath Sharma⁴, Mch

¹Mch Resident, Department of neurosurgery, S.M.S. Medical College and attached hospital, Jaipur, Rajasthan, India
²Professor and unit head, Department of neurosurgery, S.M.S. Medical College and attached hospital, Jaipur, Rajasthan, India
³Associate professor, Department of neurosurgery, S.M.S. Medical College and attached hospital, Jaipur, Rajasthan, India
⁴Assistant professor Department of neurosurgery, S.M.S. Medical College and attached hospital, Jaipur, Rajasthan, India

*Corresponding author: Sanjeev Chopra

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Abstract

Intradural lumbar disc herniation (IDH) is a rare pathology representing less than 1% (0.26-0.30%) of all herniated disc [2]. The pathogenesis of intradural lumbar disc is not known clearly [3]. Surgical and pathological findings indicate a dense adhesion between the ventral dura, posterior longitudinal ligament (PLL) and annulus fibrosus. These adhesions result from either repeated minor trauma or from prior surgery which would facilitate the nucleus pulposus herniation into the dural sac [1, 6]. We report a case of intradural disc herniation and discuss various pathophysiologies, radiological feature and review available literature.

Keywords: Intradural disc herniation (IDH), posterior longitudinal ligament (PLL).

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INTRODUCTION

Intradural disc herniation was first described by dandy in 1942 [4] and more than 150 cases of intradural disc herniation are reported in literature so far. Rupture of disc material into the intradural space is rare but must be considered in the differential diagnosis of mass lesion causing nerve root compression and cauda equina syndrome (CES) [3]. Intradural disc herniation (IDH), 5% are found in thoracic, 3% in cervical and 92% in lumbar region of which L4-L5 (55%) disc is most frequently followed by L3-L4 (16%), and L5-S1 (10%) [4,5,8]. Despite the advancement of neuroradiology techniques still IDH is not yet possible to diagnose accurately. The diagnosis is only confirmed at surgical field in most cases [1].

CASE REPORT

A 37 yrs old female admitted to our neurosurgery department with complain of low backache for 4 yrs and pain and numbness radiating to medial aspect of left lower limbs since 4 months with sudden exacerbation of symptom for 2 days prior to admission. The patient was able to ambulate normally but required analgesics for pain control but pain not relieved by medication. There was no history of trauma or spine surgery in the past. Straight leg raising test was positive on the left side and left L3-L4 hypoesthesia present. MRI suggested voluminous L3-L4 disc herniation hypointense in both T1 and T2 images, occupying great portion of vertebral canal (Fig. 1). Surgery was performed and L3 and L4 laminas were removed. The dura was tense and bulged, immobile at L3-L4 level with no disc was found extradurally and so a dural incision was given. On opening the dura a large disc was seen in the centre compressing and displacing the nerve roots laterally (Fig-2). It was a glistening white, firm disc having continuity with the L3-L4 intervertebral disc space. Careful microsurgical excision of the disc material was done; a small rent in the midline ventral dura was seen. Primary repair of dural defect done with vicryl 4-0. Post-operative period was uneventful and there was no CSF leak, patient was symptom free and discharge on 7th day. On follow up patient doing well.
DISCUSSION

Intradural disc herniations are rare, but important cause of radiculopathy and cauda equina compression (CES). There are several causes that are responsible for intradural disc herniation. Congenital or acquired adhesions between the ventral dural sac and posterior longitudinal ligament have been accepted as predisposing factor [6, 13]. The adhesions are loose in most of the cases but in some cases the adhesion may be extremely dense and unable to separate by blunt dissection [6, 9]. Some believe that patient with a degenerative disc would present as chronic inflammatory process which would favour the occurrence of these adhesions that causing erosion of adjacent dural sac leading to intradural disc herniation [1,8,9]. Yildizhan et al. [9] suggested prenatal adhesion formation between the PLL and dural sac in the cadaver study. However, the congenital union as the cause of IDH is controversial because it is clinically difficult to prove the existence of a congenital union in patients with IDH. Acquired adhesion to previous surgery or chronic herniated disc may be accepted as the main cause of intradural disc herniation [8, 10, 11]. The hypothesis of chronic inflammation due to a pre-existing disc herniation is most favourable in patients with no history of surgery because many patients with herniated disc had several-months to year history of lower back pain [8] as in our case. There are no differences in clinical signs between extradural and IDH [14], although CES is more common in the presence of IDH [6, 9]. CES occurs in approximately 0.5–1% of lumbar disc herniations in general [1, 12], while it is described in up to 30% of the lumbar IDH [5].

Various neuroimaging techniques have been used to locate more accurately the position of the disc herniation, but despite advance and refinement of present techniques, the final diagnosis of IDH is mostly made during the intraoperative period [1] as in our case. MRI finding is important for IDH preoperative identification. Choi et al. [13] presented loss of continuity of PLL and a sharp, beak-like appearance on T2-weighted image for suspicion of intradural disc
herniation. The usual image of a disc herniation occupying most part of the vertebral canal, with hypointense both in T1 and T2. Following characteristics may be associated to IDH, the loss of continuity of the PLL shown in sagittal section and the “hawk-beak” sign in axial T2 section, which show a triangular aspect of the herniated disc compressed laterally by the cartilaginous edges of the annulus fibrosus [1, 8, 13]. Rim enhancement of the herniated disc was seen on the contrast MRI. This enhancement is caused by the granulation tissue and its neovascularization that originated at the edge of the herniation. However, the IDH imaging can be indistinguishable from extradural ones. Other spinal pathologies must be considered in the differential diagnosis, such as neurofibroma, lipoma, other spinal tumors, arachnoid cyst, arachnoiditis, and metastasis [1, 15]. A contrast MRI is not routinely performed and acute herniated disc shows no rim enhancement [1]. Therefore, we think the usefulness of contrast MRI is limited in the differential diagnosis of intradural disc herniation when intradural lesions are suspected on non-enhanced MRI. As in our case we didn’t performed contrast MRI because plain MRI showed herniated disc and patient had acute symptoms of herniated disc. During surgery of IDH the common problem is difficult to dissect anterolateral portion of dural sheath. In our reported case, we found dense adhesions between the disc space and the dural sac during intraoperative period. Aydin and Lee [3] have also found these difficulties, describing the dural sac as being harsh, tense and motionless. As in our case dural sac was tense, bulged, and immobile and we performed a midline dural incision followed by IDH removed using microscope and microscope is good for minimizing injury to nerve roots for better delineation. The anterior dural sheath should be repaired when there is no risk to surrounding nerve roots, we primary repaired dural defect and advice for 24 hrs. Bed rest, patient had no CSF leak and other complications. The patient presenting with radicular pain, L3-L4 hypoesthesia preoperatively and patient has complete recovery after the surgical procedure without neurological deficits suggesting that the gentle manipulation of the rootlets using microscope does not cause neurological dysfunction.

CONCLUSION

During herniated disc surgery, surgeon must be careful and aware of some characteristic of this rare pathology mainly dissecting anterolateral aspect of the dural sac that densely adheres with herniated disc otherwise it could be disastrous for the patient.

REFERENCES


