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Review article

Transdural herniated lumbar disc disease with muscle patch for closure of durotomy – A Brief review of literature

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ABSTRACT

Purpose: Intradural migration of disc (IDMD) is a rare clinical entity accounting for 0.27–0.33% of all herniated disc diseases. Flimsy or dense adhesion between the ventral dural surface and the opposing posterior longitudinal ligament (PLL) is the principal pathology for intradural migrated disc. The most commonly affected lumbar segments are L4-5 (55%), L3-4 (16%), L5-S1 (10%) and less commonly L2L3 and L1L2. No imaging feature is characteristic and the management protocol of durotomy via an endoscopic method is unclear.

Methods: An L5S1 disc disease was operated by endoscopic method. Difficulty in separating the dural sac from PLL, dense adhesions prompting sharp dissection at this location and a calcified disc are the earliest evidence of intradural migration. MRI features of an intradural location are loss of continuity of posterior longitudinal ligament, beak-like appearance also known as ''Hawk-beak sign'', peripheral enhancement around an intradural disc, fluid-filled intradural cyst. Magnification either by Microscope or Endoscope is of importance when dissecting the intradural disc so as to avoid the nerve root injury. Liberal use of fibrin glue and augmentation with muscle patch was performed.

Results: Ambulated by 48 h and discharged by 5th day. Two and 9 months follow up showed no evidence of pseudomeningocoele.

Conclusion: Autologous muscle patch with fibrin glue for dural rent closure is a simple and effective method which can be performed by endoscopic or minimally invasive approaches. Suturing the dura, being a tedious and cumbersome procedure can be avoided.

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1. Introduction

Dandy identified intradural migrated disc (IDMD) as a complication of lumbar posterior intervertebral disc prolapse (PIVD) in 1942 [1]. IDMD is a rare event occurring in 0.04–0.33% of all lumbar disc herniation [2]. They are seen commonly at L4-5 level, although has been reported at other lumbar levels, cervical and thoracic regions also. Even in this era of advanced imaging techniques, they are hardly identified preoperatively [3]. About 150 reports of this pathology are documented and hence certain radiological features may need to be emphasized for preoperative diagnosis. Masquerading as a simple Lumbar PIVD, cauda equine syndrome, or as a spinal intradural extramedullary lesion [4,5], it can poise a real threat to the nerve roots especially in the hands of a young neurosurgeon. Nevertheless, it could be one of the ''*eureka*'' like moment for any operating neurosurgeon to have safely removed it. We present a case of L5S1 IDMD identified and managed during a routine endoscopic disc surgery along with literature review.

2. Case material

A 42-year-old female patient presented with a 20 years history of low backache and radiation with tingling and numbness in bilateral L5 & S1 nerve roots distribution of 1-year duration. The radicular pain has increased in intensity for the last two months. No antecedent history of trauma was present. On neurologic examination, a positive straight leg raise test on both sides with 20% sensory loss to touch and pain in B/L L5 & S1 distribution was noted. There were no motor weakness or bladder disturbances. X ray showed reduced L5S1 disc space with no obvious listhesis. MRI showed L5S1 right paracentral type PIVD with significant foraminal stenosis (Fig. 1). At no point was an intradural extension or mass-like lesion was observed and hence a gadolinium-enhanced MRI was not performed. Patient was planned for an endoscopic discoidectomy. Under general anesthesia and in prone position, a right paramedian, muscle-splitting incision was made after localization of L5 lamina on C-Arm and a Destandau Endospine® system was inserted. Patient underwent a single surgeon, endoscopic assisted (Destandau Endospine® System, Karl Storz) L5 partial hemilaminectomy by drilling through the working channel endoscope. The lamina was unusually thick during the drilling. This was followed by detachment and excision of calcified ligamentum flavum. While creating a plane between the dural tube and the adjacent vertebrae, dense adhesions were encountered which made further dissection impossible. Hence a possible calcified disc & intradural disc was kept in mind. A dural opening was created along the lateral edge of the dural tube of a length of 1 cm with evident flow of CSF and herniation of the nerve root. On manipulation of the roots, an intradural location of partially calcified disc was identified (Fig. 2A). Entire herniated disc was removed in to after adequate mobilization from the nerve roots (Fig. 2B,C). There were no adhesions between the disc and the roots in our case. With satisfactory decompression, the dura rent was closed with a free muscle patch (1.5 cms × 1.5 cms × 0.3 cms flat sheet of paraspinal muscle prepared after pressing between gauge piece) reinforced with SURGICEL® FIBRILLAR™ and with human fibrin glue (EVICEL® fibrin sealant, Johnson & Johnson) (Fig. 2E,F). Muscle was not sutured to the dura but was just placed to cover all the edges of the defect. We do not advocate use of fibrin glue directly over the defective area as it may lead to adhesions of the underlying roots and may even result in prolapse of roots it the muscle patch give away. Literature however has rare incidences of such untoward complications despite. A disc of 2 cms × 1.5 cm was delivered out (Fig. 2D). Lumbar fascia approximated with vicryl 2-0, and skin incision was closed with interrupted nylon sutures without any drain. Prone nursing for 24 h and absolute bed rest for 48 h was maintained. Patient was ambulated after 48 h. Despite a 1 cm dural incision there was no evidence of post op CSF leak was noted. Incidental durotomy need not be an indication for conversion to open technique as one assumes expertise in endoscopic management of dural rents including suturing. With no post op neurological deficits patient was discharged on day 4. Follow up at 2 months was uneventful with complete resolution of symptoms. MRI done at 9 months follow up shows residual desiccated disc with minimal thecal sac compression. There is no evidence of pseudomeningocoele (Fig. 3, arrow – indicating the region of muscle patch). As patient is completely symptom free, no further procedure was contemplated.

3. Discussion

Intradural migration of disc (IDMD) is a rare clinical entity accounting for 0.27–0.33% of all herniated disc diseases [6,7]. Since Dandy first described the pathology in 1942, over 150 cases have been reported with many review of literature on a pub med search with key words ''intradural herniation''. Majority of reported cases are of lumbar region (92%), followed by thoracic (5%) and cervical (3%) [6]. The most commonly affected lumbar segments are L4-5 (55%), L3-4 (16%), L5-S1 (10%) and less commonly L2L3 and L1L2 [8]. Ruptured herniated disc appear to migrate not beyond the same disc level and very rarely even to the mid vertebral body level indicating a constant attachment with the mother disc at that level. Moreover, intradural migration of disc fragment, to the level of the vertebral body from the level of ruptured intervertebral disc space, is particularly rare [9,10].

3.1. *Etiopathogenesis*

Flimsy or dense adhesion between the ventral dural surface and the opposing PLL resulting from congenital adhesions [8]/posttraumatic [11]/post surgery [12]/chronic inflammatory/degenerative conditions, leads to an ''enmasse rupture'' and migration of the disc following an event of raised intradiscal pressure the subsequent perforation of these firmly adhesive tissues, including the annulus fibrosus [13–15]. Chronic inflammation and erosion process by the herniated disc may itself perforate the dura [13]. Both of this explains the fact that IDMD occurs at L4L5, L5S1 levels, the most event full disc spaces [16]. In our case, there was dense adhesion between dura and the PLL at L5S1 level. Even an ossified PLL

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