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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 pseudomeningocele.

**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 pose 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 discectomy. 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

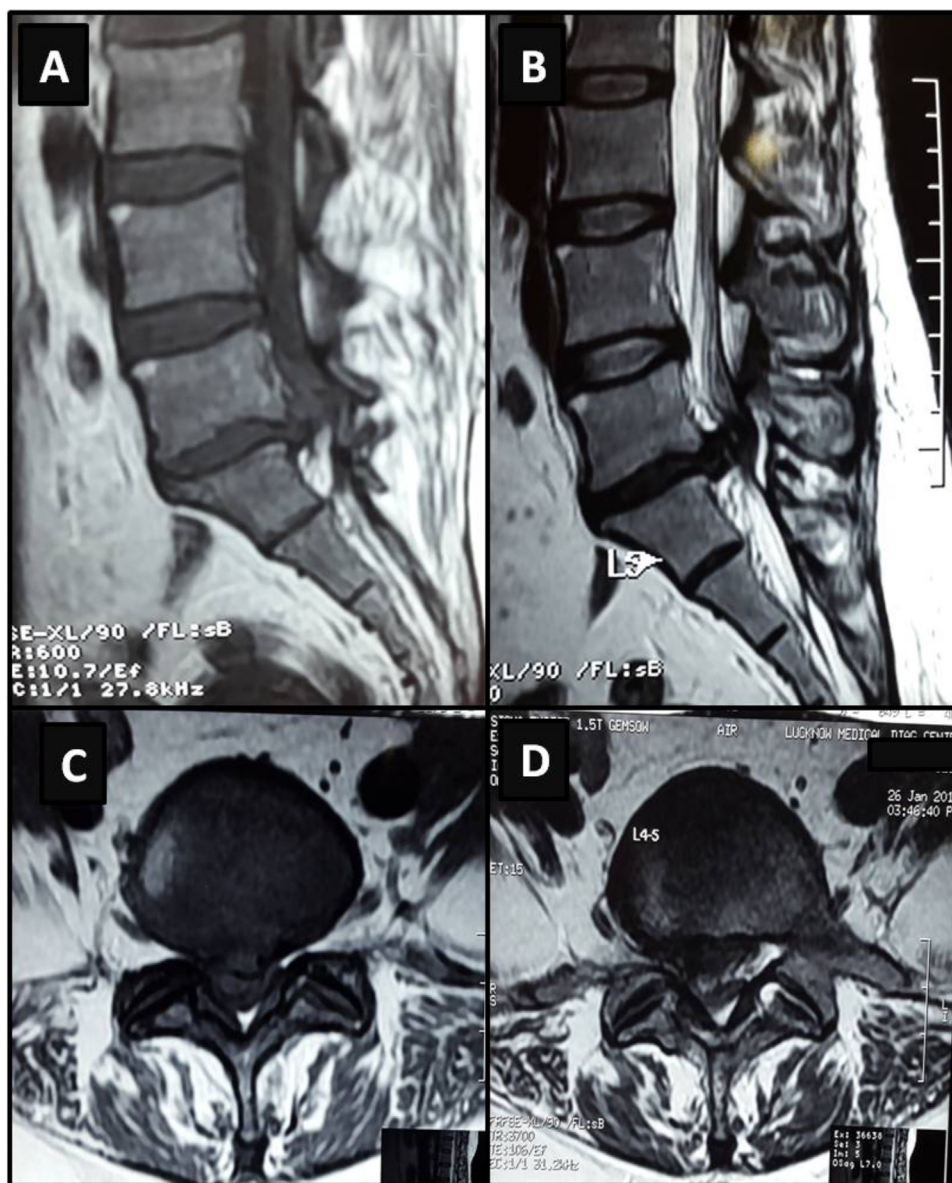
(1.5 cms × 1.5 cms × 0.3 cms flat sheet of paraspinal muscle prepared after pressing between gauge piece) reinforced with SURGICEL® FIBRILLAR™ and with human fibrin glue (EVICEL® fibrin sealant, Johnson & Johnson) (Fig. 2E,F). Muscle was not sutured to the dura but was just placed to cover all the edges 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 if 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 pseudomeningocele (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



**Fig. 1 – (A) MRI T1W1 and (B) MRI T2W1 sagittal image showing herniated disc with loss of continuity of PLL at the disc space with no obvious CSF space around the disc. (C) T1W1 and (D) T2W1 axial image with an obvious disc fragment. PLL also appears to be discontinuous which is one of the indicator of intradural disc fragment.**

may perforate the dura and may lead to migration of fragments into the intradural compartment following a subtle trauma in a simple degenerative disc disease [17,18].

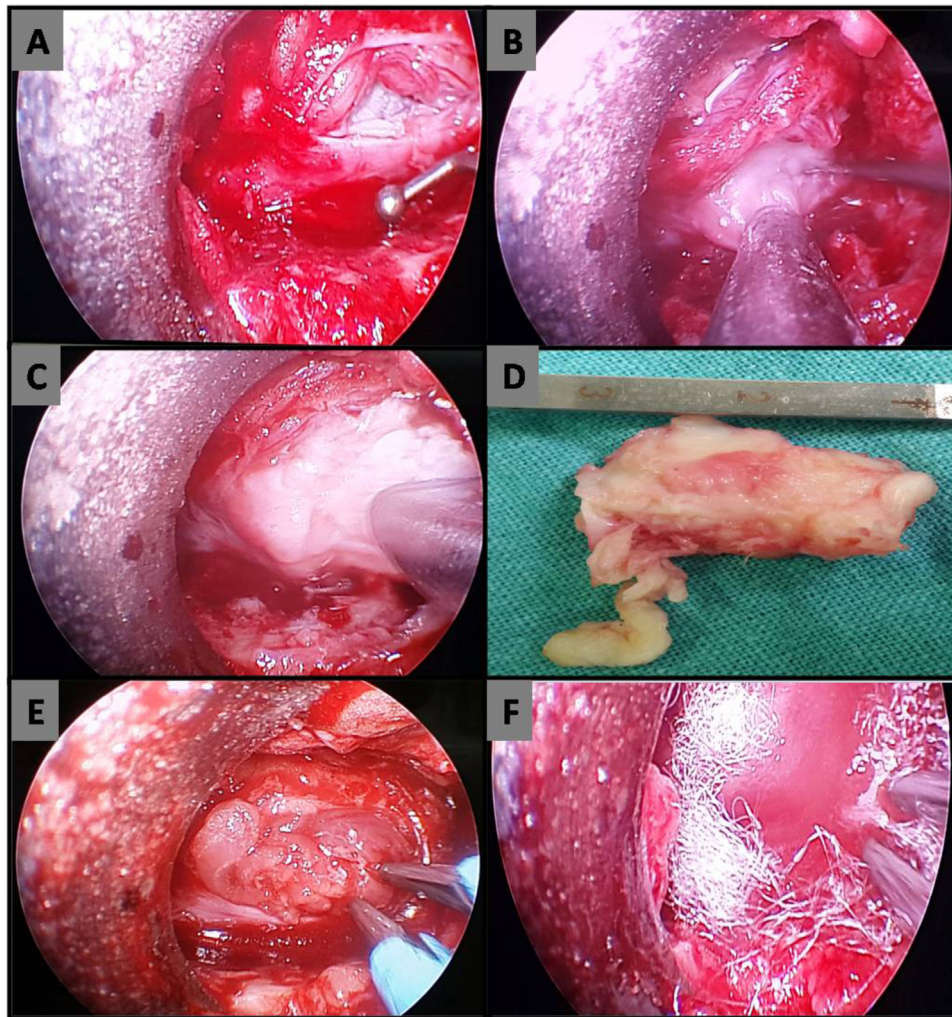
### 3.2. Symptomatology

30% of IDMD present with cauda equine syndrome (CES) (0.5–1% in early PIVD) [19,20]. Rest present mainly as a chronic lumbago, or radiculopathy [16]. Mechanical compression theory, ischemic theory, chemical irritative theories have been elucidated in the literature for explaining the possible events resulting in CES [2]. Our patient also had a chronic lumbago of 20 years duration and hence was prone for events of degeneration, calcification, adhesions, rupture and migration despite any trauma. Rapoport et al. have reported a case of

cranial neuropathy secondary to intracranial hypotension and B/L abducent nerve palsy arising from CSF leak at the site of intradural herniation of disc fragment with post surgical complete resolution of symptoms [21]. Agarwal et al. used autologous blood to seal the dural rent with intradural disc detected by CT myelography with complete resolution of symptoms [22].

#### 3.2.1. Imaging

An incidental IDMD was identified during discography by Benyamin et al. [23] Conventional Myelography and CT myelogram may show a complete blockage of the contrast medium, but their accuracy at localizing the IDMD is still doubtful [12,24]. Although studies by Schisano et al. and Sarlieve et al. demonstrate the utility of myelography, the



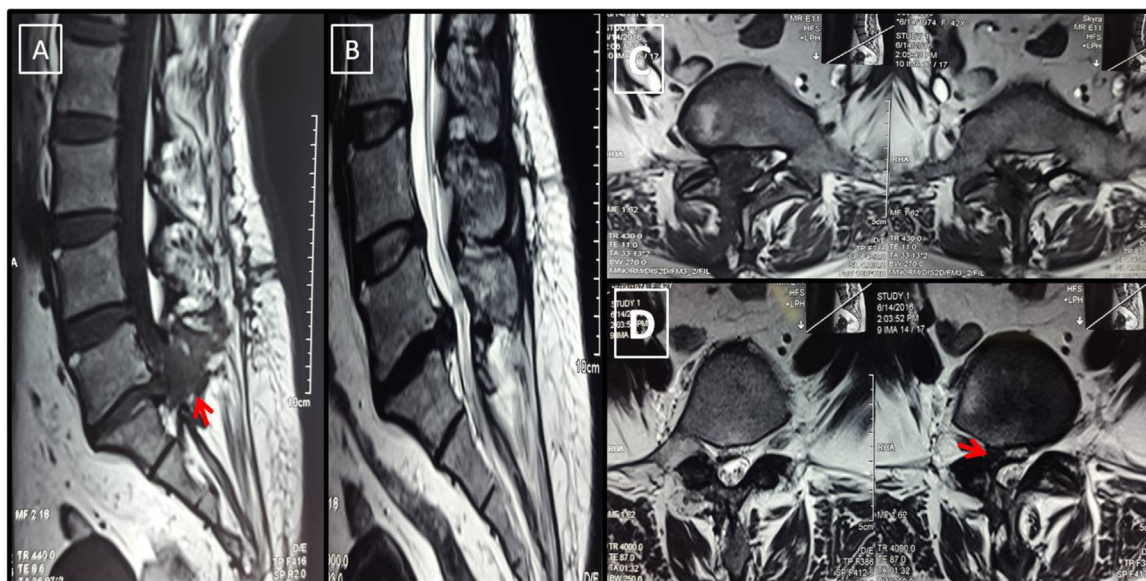
**Fig. 2 – (A) Endoscopic view of the dural incision with visible intradural disc and lumbar nerve rootlets. (B) Disc being mobilized from the adjacent nerve roots. (C and D) Entire disc of 2 × 1.5 cms being delivered out. (E and F) endoscopically placed intradural muscle patch with fibrillar and fibrin glue.**

suspicion of intradural location based on noninvasive MRI imaging is difficult to contemplate. There are no reliable findings on NCCT and CECT in identifying the intradural location [24,25]. A study by Hidalgo et al. showed an association of intradural gas on CT scan with intradural migration of disc in 5 patients [2]. Non-contrast MRI is the gold standard investigating modality in disc diseases. GAD MRI has no additional features in simple disc disease and hence is seldom done. Few of the imaging features in the literature which may feature an intradural location are loss of continuity of posterior longitudinal ligament, beak-like appearance also known as “Hawk-beak sign” [25], peripheral enhancement around an intradural disc like lesion attributed to inflammatory granulation tissue is typical of disc fragment [26–29], fluid-filled intradural cyst [30].

### 3.3. Operative nuances

Despite all the imaging modalities available, most of the reported cases are a retrospective analysis following an

intraoperative identification of intradural location. Unlike routine disc surgeries there are few caveats which can be kept in mind and can be associated with a possible intradural sequestration. Difficulty in separating the dural sac from anterolateral PLL and dense adhesions prompting sharp dissection at this location are the earliest events [2,31]. The dura at this location is unusually harsh, tense, stiff and non-yielding [10,32]. Disc bulge is hardly visualized due to these findings. In our case too, a difficult dissection and a sense of calcification of disc was noted. Mut et al. has classified this pathology into Type A (dural sac herniation) and Type B (dural sheath around nerve root or intradiscal disc herniation) based on intraoperative finding [33]. Majority of Type B pathology occurs at L5S1 level and Type A occurs at L4L5 level [10,33]. In both the types an adhesion between the disc and the dura is the primary postulated mechanism, but the cause for selective occurrence at L4L5 and L5S1 is unknown. In our case it was Type A variety and decision for incision of dura was made at an earlier stage itself. Magnification either by Microscope or Endoscope is of importance at this stage when



**Fig. 3 – 9 months post op MRI (A – T1W1, B – T2W1–SAGGITAL, C – T1W1, D – T2W1–AXIAL) shows minimal residual disc at L5S1 level with minimal thecal sac compression. No evidence of pseudomeningocele noted in the vicinity of muscle patch (arrows).**

dissecting the intradural disc so as to avoid the nerve root injury. Due to disc, the nerve roots are bound to herniated through the incised dura and their ever presence in the operative field may result in injury. Irrigating the intradural compartment may itself loosen the disc fragment. It is advisable at this stage to clearly see the margins of the disc when delivering it out for certain nerves may be adhered to the disc and get injured. Fragmentation of the disc and its removal may be carried out in case of calcification. Literature does not show any evidence of requirement for further dissection into the disc space in these cases. There are several methods for the repair of dural tear including suturing, tissue sealant, blood patch, tissue grafting, gel foam and glue applications [34].

#### 3.4. Management of durotomy

An incidental durotomy with minimally invasive discectomy can occur in up to 1.6–17.4% of cases [34,35]. Such unintended durotomies have significantly occurred while being operated for a recurrent disease and more so in the lumbar region [36]. Unmanaged durotomies can result in CSF fistula, pseudomeningocele, meningitis, and postural headache [37,38]. Very less information is available in the literature regarding protocols of management of durotomy in endoscopic discectomy cases. Common risk factors associated with inadvertent durotomy are old age, chronic compression with atrophic thinned out dura, OPLL, previous surgeries, associated synovial cysts, multiple level disease, lumbar disease and experience of operating surgeon [34,35,39,40]. Dural rent can be managed by suturing the defect but ensuring that there are no nerve roots and only when it is safe to do so. Albeit the difficulty associated with suturing, it has been done [2,36]. In case series of accidental durotomy during minimally invasive spinal surgery by Ruben et al. [35], Dmitry

et al. [36] and Epstein [37], durotomies were managed by fibrin glue alone for partial-thickness durotomies, blood-soaked Gelfoam with fibrin glue for small full-thickness durotomies, and muscle graft or collagen matrix with fibrin glue to reinforce nonwatertight primary closure of larger durotomies with almost 0% post op leak [36,37]. Usage of Fibrin glue in all cases of durotomy irrespective of additional augmentation procedures like suturing, muscle patch, gel foam has been a norm and has showed very good results [35–37,41] and has been proven beyond doubt to be the best and safe neurosurgical armamentarium in various neurosurgical cases [42–47]. In our case an autologous muscle patch was inserted into the dural sac and was augmented with fibrillar and fibrin glue. Another area of concern is the relative period of absolute rest needed. Most of the studies for open repair advocate rest period of 24 h to up to 7 days, minimally invasive spine surgery has this added advantage of early ambulation (24–48 h) [36]. The techniques of muscle splitting, minimal incision, with less formation of muscular potential spaces around the repaired dura has been a boon for early healing of the wound [48–50].

#### 4. Conclusions

Surgeon's gut-feeling of a difficult dissection and possible calcified disc like impression are the earliest intraoperative findings of an intradural disc location as preoperative imaging is rarely diagnostic. Dissection by magnification, careful disc delivery and dural rent closure is mandatory for avoiding nerve root injury. Durotomy repair using autologous muscle patch with human fibrin glue is a safe and an effective armamentarium to prevent CSF leak and pseudomeningocele formation.

## Conflict of interest

None declared.

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## Ethics

The work described in this article has been carried out in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans; Uniform Requirements for manuscripts submitted to Biomedical journals.

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