Transdural lumbar disc herniation: experiences often ten cases with review of literature

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Background: Intradural or intraradicular lumbar disc herniations are uncommon presentations of a relatively frequent pathology, representing less than 1% of all lumbar disc hernias.

Methods: The purpose of this study was to analyze the mechanism, clinical, radiological, and the surgical treatment requires a transdural discectomy. Ten cases of intradural and intraradicular disc were analyzed retrospectively. The cases comprised 0.5% of the 620 cases of lumbar disc that underwent surgery from 2000 to 2015. The data described the causes of this pathology, and analyzed it from clinical, diagnostic, and therapeutic perspectives. The difficulties in the preoperative diagnosis issues and the surgical techniques will be interpreted.

Results: The commonest involved site is at level L4–L5. In intradural herniation, most patients reported a chronic history of back pain, complicated later by neurologic signs. In intraradicular extension, the patients were usually complaining of severe sciatica. In the present series, diagnosis was obtained by means of magnetic resonance image. All patients underwent surgery were reporting excellent results in six cases and good results in the other four. Surgery was performed either with laminectomy for intradural extension and fenestration discectomy for intraradicular extension. Dorsal dural was repaired only, with no leakage of the cerebrospinal fluid.

Conclusions: In intradural and intraradicular disc herniations; the diagnosis is mainly intraoperative, but MRI could help, the surgical technique has some special aspects, and the prognosis is good.

Keywords: Lumbar spine; intradural disc herniation (IDH); intraradicular disc herniation; transdural discectomy

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Introduction

Intradural disc herniation (IDH) was first defined by Dandy in 1942 (1) as the intervertebral disc nucleus pulposus herniated into the dural sac. In 2009, the estimated cases of IDH were 140 cases (2). Most cases refer to patients with ventral dura herniation. Dura perforation at the radicular sheath level is caused by fragments of lumbar intervertebral disc which was first documented in 1984 (3). Since then, 22 cases of intraradicular herniation (IRH) have been reported (4). IDH is a rare occurrence. It is constituted less than 1% of all LDHs. Lumbar region is the most frequent affected site (92%) especially at L4–L5 disc spaces (5–7).

The pathogenesis of lumbar IDH is most likely related to dense adhesions between the ventral dura mater and the posterior longitudinal ligament (PLL). The adhesions can apparently result from congenital canal stenosis, repeated minor trauma, or from prior surgery (7,8).

Clinically, the incidence of cauda equine syndrome (CES) is higher in IDH than in extradural herniations (2,5,6). Most of cases are only diagnosed at the surgical field, despite the present advance in neuroimaging techniques (2).

Many patients require surgical intervention (2–4,6). However, Sakai et al., report a case of recurrent intradural lumbar disc herniation in which there was a spontaneous recovery with conservative therapy (9).

The purpose of this paper is representing our experience with ten cases of IDH and IRH discs, review the relevant literature, and discuss the pathogenesis, diagnosis, and treatment.

Methods

From 2000 to 2015, we retrospectively studied in 620 patients suffering from lumbar disc herniation, ten cases of intradural and intraradicular perforation was selected. The details of eight cases of intradural and two cases of intraradicular lumbar disc herniation were described. Detail demographic, clinical and radiological data were described in Table 1.

Classification of intradural herniated disc in relation to ventral dura (IDH) or lateral near the root’s axilla (IRH) divided into these two groups. Ten cases of IDH and IRH perforation were selected. For intradural approach, first we used a small interlaminar fenestration and the exposure of the dural sac, the diagnosis of intradural herniation was confirmed in surgery by the following: (I) absence of a significant herniated fragment in the extradural site; (II) abnormal swelling of the dural sac and root; (III) impossibility of moving both the sac and the root because of adhesions between the disc capsule, PLL, and dura mater which represented as a single mass, and (IV) double-checking the disc level by X-ray, and empty insignificance disc material. For neural safety, we decided to perform laminectomy to the affected level. Under magnification loop and using microsurgical techniques, we separated the nerve rootlets and found the IDH fragments. After fragment removal, we found a free communication between the dural sac, PLL, as well as the disc space. After decompression, the anterior ventral dural sheath tear left open. The dorsal dural incision was sutured with continuous stitches Prolene 5–0; the soft tissues were closed ordinarily in a continuous sutures manner. After closure of the dorsal dura, a valsalva was negative up to 11 times and made sure that there was no cerebrospinal fluid (CSF) leakage. In case 2, 3, and 6, we found that the disc fragments were extensively adhering to the dura and PLL and became a thin one layer and transdural (ventral dura) resection was done. Lumbar sheath was closed in continuous sutures manner also. In case of IRH, we first do fenestration and removal of disc. Suspicion for IRH was estimated by the following: (I) abnormal swelling of the dural root; (II) difficulties in moving both the sac and the root on the lesion side, and (III) double-checking the root for missed fragment in the shoulder or the axilla. Dural opening was done in the last case and not in the first IRH case (case 10).

Lumbar drains and bed rest for 3 days after surgery were employed. Most patients were allowed to ambulate on the 4th day if there were no evidence of CSF leakage. A stay suture was prepared during operation and used to close the drain opening with drain removal on the 5th day. Patient was presented with persistent neurological deficit, physiotherapy was immediately initiated. During following up, the patient outcome was assessed using Mac nab scale (10).

Results

Intradural and IRH disc comprise 0.5% of the 620 cases of lumbar disc that underwent surgery from 2000 to 2015. The detail of each patient was presented in Tables 1,2.

IDH was observed in eight cases (Table 2), five males and three females in the age range of 28–50 years. As to the length of neurologic symptoms, most of the IDH cases symptoms are extended for years. In contrast with for radicular
Table 1 Descriptive clinical and radiological summary of the cases

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (years)</th>
<th>Sex</th>
<th>Work</th>
<th>Symptoms duration</th>
<th>Sensory changes</th>
<th>Motor deficit</th>
<th>Ankle reflexes</th>
<th>Sphincter affection</th>
<th>Clinical diagnosis</th>
<th>MRI affected level</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>50</td>
<td>Male</td>
<td>Driver</td>
<td>4 years</td>
<td>Hypoesthesia, perianal, right L5, S1</td>
<td>Weak foot dorsiflexion and planter flexion</td>
<td>Lost right side</td>
<td>Urinary retention 1 day before surgery</td>
<td>Cauda equine syndrome</td>
<td>L4/5</td>
</tr>
<tr>
<td>2</td>
<td>40</td>
<td>Female</td>
<td>Housewife</td>
<td>10 years</td>
<td>Sever pain last 2 months along left S1</td>
<td>Subjective planter flexion weakness</td>
<td>Lost left side</td>
<td>–</td>
<td>Claudicating canal stenosis</td>
<td>L5–S1, less at L3–4 and L4–5</td>
</tr>
<tr>
<td>3</td>
<td>32</td>
<td>Female</td>
<td>Housewife</td>
<td>8 years</td>
<td>Sever pain 20 days along right S1</td>
<td>Weak dorsiflexion</td>
<td>–</td>
<td>–</td>
<td>Radicular syndrome</td>
<td>L4–5</td>
</tr>
<tr>
<td>4</td>
<td>49</td>
<td>Male</td>
<td>Fisherman</td>
<td>1 year</td>
<td>Pain along left L5 and perianal hypoesthesia</td>
<td>Bilateral weakness plantar flexion last 3 days</td>
<td>Lost bilateral</td>
<td>Urination difficulties but no retention</td>
<td>Early cauda equine syndrome</td>
<td>L4–5</td>
</tr>
<tr>
<td>5</td>
<td>38</td>
<td>Male</td>
<td>Cruise ship worker</td>
<td>2 years</td>
<td>Hypoesthesia, right L5, S1</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>Radicular syndrome</td>
<td>L4–5 and less at L3–4</td>
</tr>
<tr>
<td>6</td>
<td>48</td>
<td>Male</td>
<td>Driver</td>
<td>6 years</td>
<td>Sever pain last 1 month along L5 bilateral</td>
<td>Bilateral weakness plantar flexion last 3 days</td>
<td>Lost bilateral</td>
<td>Urinary retention and constipation 3 days</td>
<td>Cauda equine syndrome</td>
<td>L4/5</td>
</tr>
<tr>
<td>7</td>
<td>28</td>
<td>Female</td>
<td>Housewife</td>
<td>2 months</td>
<td>Sever pain 20 days along right L5</td>
<td>Complete foot drop</td>
<td>Lost right side</td>
<td>–</td>
<td>Radicular syndrome</td>
<td>L4–5, less L5–S1</td>
</tr>
<tr>
<td>8</td>
<td>31</td>
<td>Male</td>
<td>Driver</td>
<td>4 months</td>
<td>Sever pain 20 days along right L5, S1</td>
<td>Weak foot dorsiflexion and planter flexion</td>
<td>Lost right side</td>
<td>Early retention of urine</td>
<td>Cauda equine syndrome</td>
<td>L4–5, less L5–S1</td>
</tr>
<tr>
<td>9</td>
<td>36</td>
<td>Male</td>
<td>Mechanical technician</td>
<td>4 months</td>
<td>Sever pain along right L5</td>
<td>Subjective dorsiflexion weakness</td>
<td>Lost right side</td>
<td>–</td>
<td>Radicular syndrome</td>
<td>L4–5, less at L5–S1</td>
</tr>
<tr>
<td>10</td>
<td>32</td>
<td>Male</td>
<td>Fisherman</td>
<td>4 months</td>
<td>Sever pain along right S1</td>
<td>Subjective plantar flexion weakness</td>
<td>Lost right side</td>
<td>–</td>
<td>Radicular syndrome</td>
<td>L5–S1, less at L3–4 and L4–5</td>
</tr>
</tbody>
</table>

Symptoms most of cases are extended for months. For intradural cases, most of patients were developed exacerbation of symptoms from a day up to three months before surgery. They may present with various symptoms from early cauda (cases 1, 4, 6–8), or claudicating pain (case 2), or radicular syndrome (cases 3, 5, 7, 8). All the IRH cases (cases 9 and 10) had only radicular symptoms. Nearly 75% (6/8) of all cases of IDH were L4–5, while IRH disc cases were L4–5 and L5–S1. Case 9 IRH disc had calcified disc intra-operative.

Magnetic resonance imaging (MRI) was used in all cases: in the beginning, it allowed good visualization of
the herniation. In cases 1, there was an intradural mass mimic a tumor (Figure 1). In cases 3–5, a characteristic beak likes the appearance of the MRI denotes intradural disc (11) (Figures 2–4). In case 7, a characteristic beak likes appearance presented in the sagittal plane clearly (Figure 5). In cases of 2, 6, and 8, a huge median and paramedian disc with PLL adhesion with cephalic orientation (Figures 6-8). They represented interdural extension. In cases 4, and 5, 8 MRI myelography showed complete block at L4–5 (Figures 3D, 4D, 8D). In early cases, as in case 9, MRI myelography was inapplicable (Figure 9). In case 10 of IRH (Figure 10), MRI myelography revealed root absent at the S1 root of the affected side (Figure 10E).

All patients underwent surgery, and laminectomies were performed in all intradural cases. In cases 1, 4–5, 7, and 8, true intra canal disc fragments were seen. In cases 2, 3, and 6, the ventral dural was forming a dome shape inside the canal with a thin layer (Figure 11A). The disc was firmly attached to the ventral dural and the absence of a significant herniated fragment in the extradural site. It was impossible to move both the sac and the root, because of dense adhesions where the disc capsule, PLL, dura mater, and disc fragment representing one mass. In case 2, and after ventral opening, some rootlets were sagging in the ventral opening with no compression or entrapment.

In the two cases of IRH, case 9 had a calcified disc and

<table>
<thead>
<tr>
<th>No.</th>
<th>Sex</th>
<th>Operated level</th>
<th>Operation</th>
<th>Disc material</th>
<th>Result</th>
<th>Complication</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Male</td>
<td>L4/5</td>
<td>Laminectomy</td>
<td>Intradural</td>
<td>Excellent</td>
<td>No</td>
</tr>
<tr>
<td>2</td>
<td>Female</td>
<td>L5–S1</td>
<td>Laminectomy</td>
<td>Transdural</td>
<td>Good</td>
<td>Urinary retention 1 month, sever constipation 3 months</td>
</tr>
<tr>
<td>3</td>
<td>Female</td>
<td>L5–S1</td>
<td>Laminectomy</td>
<td>Transdural</td>
<td>Excellent</td>
<td>No</td>
</tr>
<tr>
<td>4</td>
<td>Male</td>
<td>L4–5</td>
<td>Laminectomy</td>
<td>Intradural</td>
<td>Excellent</td>
<td>No</td>
</tr>
<tr>
<td>5</td>
<td>Male</td>
<td>L4–5</td>
<td>Laminectomy</td>
<td>Intradural</td>
<td>Excellent</td>
<td>Urinary retention, constipation 1 week</td>
</tr>
<tr>
<td>6</td>
<td>Male</td>
<td>L4/5</td>
<td>Laminectomy</td>
<td>Transdural</td>
<td>Good</td>
<td>Urinary retention, sever constipation 5 months. Persist low dermatomal sensation along L5</td>
</tr>
<tr>
<td>7</td>
<td>Female</td>
<td>L4–5</td>
<td>Laminectomy</td>
<td>Intradural</td>
<td>Good</td>
<td>Persistent foot drop</td>
</tr>
<tr>
<td>8</td>
<td>Male</td>
<td>L4–5</td>
<td>Laminectomy</td>
<td>Intradural</td>
<td>Good</td>
<td>Mild gait disturbance</td>
</tr>
<tr>
<td>9</td>
<td>Male</td>
<td>L4–5</td>
<td>Fenestration discectomy, foraminotomy</td>
<td>Intraradicular</td>
<td>Excellent</td>
<td>No</td>
</tr>
<tr>
<td>10</td>
<td>Male</td>
<td>L5–S1</td>
<td>Fenestration discectomy, foraminotomy</td>
<td>Intraradicular</td>
<td>Excellent</td>
<td>No</td>
</tr>
</tbody>
</table>

**Table 2 Operative data and clinical outcome**

**Figure 1** A T2-weighted sagittal image in case 1 axial (A) and sagittal (B) demonstrates an intradural mass suspicious for intradural extension of disc fragments at the L4–L5 level. It showed caudal migration.
immobilized large root. We were unable to open the root in this early experience and just did foraminotomy and the patient was improved (Figure 9). In the second case, the root dura seemed to be divided into two layer and fragment in-between. This finding was described before elsewhere (4) (Figure 10).

Once the intradural hernia was removed, a discharge of CSF was noted in all cases, except no 9 and minimal in case 10. We do nothing for ventral dura, and dorsal dura was closed with Prolene 5-0 with no postoperative CSF for all cases. We used gelatin compressed sponge (gel foam) in all cases after dura repair as fibrin glue was not available.

All patients underwent surgery, reporting excellent results in six cases and good results in the four cases. Excellent results mean the normalization of neurologic examination at discharge; while good results mean the improvement of patient’s pain and neurologic deficit (10,12).

The mean follow-up has been 8 years. It ranges from a minimum of 2 years up to a maximum of 14 years in case 1. Cases 2, 5, 6 and 8 exhibited urinary retention; severe constipation, and saddle area hypoesthesia from one week up to 3 months. But all cases were improved later on. Case 7 had persistent right foot drop, and case 8 had mild gait disturbance due to weak gluteal muscle in the right side (Table 2).

Discussion

Lumbar disc herniation complicated by IDH is a rare incidence and constitutes less than 1% of all LDHs (2,4-6). In this series, it represented 0.6%.

Males’ predominance reported in most of cases (2,4,5,7), thus sex distribution is similar to that of the uncomplicated
lumbar disc. In our series, male prevalence is estimated 0.7%, the highest incidence occurred between the 4th and the 5th decades. The incidence is much lower than reported in the literature, 6th to 7th decades (2,3,12), may be due to heavy work habit by patients in this study. The site most frequently involved by dural perforation is L4–L5, followed by L5–S1 (11,12).

Mut et al. in 2001 suggested the following classification for intradural disc. Type A: herniation of a disc into the dural sac; type B: herniation of a disc into the nerve root (8). The herniation is usually in the preganglionic region (13). However, in this paper, we found that type A was further subdivided into intra and inter dural as in cases 1, 4, 5, 7, 8 and 2, 3, 6 respectively. Sasaji et al. described “Y-sign” in the lumbar spine (14). He described a state of IDH fragment that peeled the arachnoid from the dura. It is called extra-arachnoid disc herniation. The one line which representing the combined dura and arachnoid was subdivided into two lines. The branching of the ventral dura line of the disc fragment appeared as a “Y-sign”. This finding is also confirmed by Wang et al. (15). In our statement, we represented a different appearance where the fragment extensively adhesive and maximally stretched the dura rather than peeling dura from the arachnoid. It is finally looking like if it is a thin arachnoid (Figure 11A).

Type B was named IRH, which is much less frequent: about 22 cases were previously reported (4). Ozer et al. suggested the term of “pseudointraradicular” disc herniation when the disc herniation only penetrated the outer dural layer of the nerve root (16). To be lodged between the internal and external dura layers, so the term of interdural disc herniation is preferable. In this article, this finding was confirmed in case 10.

The pathogenesis of dural sac perforation caused by

Figure 3 A T1- and T2-weighted axial image in case 3 axial (A,B) and sagittal T2 (C,D) demonstrates a wedge-shaped disc herniation to the right suspicious for intradural extension of disc fragments at the L4–L5 level (a sharp beak-like appearance of intradural disc herniation, see white arrows). MRI showed marked facet degeneration on such side.
herniated disc fragments is not well known. According to Dandy (1), dural perforation was caused by an acute and sudden pressure of the herniated disc. Blikra (17) carried out an anatomic study of 40 cadavers, which revealed anatomic adhesions between the ventral dural sac and the PLL, particularly at the L4–L5 level. Nowadays, many reports confirmed such finding (2,6,11,12,14).

It has been hypothesized that degeneration of the nucleus pulposus secretes an inflammatory substance. These substances act as a “foreign body” in the spinal canal, which activate an autoimmune process (18). The congenital fusions between the PLL and the dura have also been postulated (19). In this series, all cases represented chronic inflammation and adhesion. Cases 2 and 6 represented canal stenosis, case 7 represented traumatic left heavy object, and case 8 had previous surgery.

For radicular interdural lumbar disc herniation, as described by Akhaddar et al. (4), the predisposing conditions stated in the literature are also congenital reduction of dural thickness, vertebral canal congenital stenosis (7), and previous surgeries (2). Due to younger age in radicular cases, we thought there may be congenital reduction of the dural thickness.

Seventy-nine percent of cases reported in literature (20) had been suffering from low-back pain or sciatica for more than a year. Back pain may extend up to 30-year histories (2,4,17,20). Our patients presented with low back pain, extended from 1 to 10 years, with increasing pain intensity just before admission, comparable to 3–4 months in cases of radicular discs. Up to 33% of cases described in literature (2,7,12,20) had been previously operated on for intraradicular disc herniation. In this thesis, it represented...
20%. As most patients as in this series had a history of low back pain or chronic sciatica suddenly complicated by neurologic deficit causing often a cauda equina syndrome (CES) or sciatica (2,4,6,7,12).

Various neuroimaging techniques have been analyzed in order to try to locate more precisely the position of the herniated disc. In myelographic examination, IDH of the lumbar region usually shows as a complete block (4,17). Hodge et al. (21) suggested that a myelographic view of IDH is characterized by an irregular filling defect. Also, vacuum phenomenon inside the canal could be indicative of intradural herniation. Anda et al. (22) stated that the presence of gas in intradiscal space (vacuum phenomenon) might be indicative of intradural perforation. Chowdray et al. (23) showed the passage of contrast medium into the dural rupture is caused by the herniated disc. Computerized tomography myelogram (CTM) could identify the intradural component (24), visualize of the extra- and intradural portions of the disc fragment (7,12,22).

Benyamin et al. (25) reported that incidentally during fluoroscopically guided injection into L4–5 that contrast was not contained within the disc and spread intrathecally with a myelographic appearance.

MRI is the gold standard neuroimaging study. The first description of intradural lumbar disc herniation with MRI is by Epstein et al. (6). The two following characteristics may be associated to IDH and raise the suspicion of IDH: the loss of continuity of the PLL shown especially in the

Figure 5 A axial T1- and T2-weighted sagittal image in case 5 (A,B) sagittal T2 (C), demonstrates a wedge-shaped disc herniation to the right of intradural extension of disc fragments at the L4–L5 level (a sharp beak-like appearance of intradural disc herniation with no canal, see white arrows). MRI myelogram (D) revealed complete irregular amputation of the myelogram medium at the L4–L5 level.
sagittal acquisitions as in cases 1, 2, 6, and 8, second the “hawk-beak” sign in axial T2 acquisitions in cases 3–5, and 7, which show a triangular aspect of the herniated disc compressed laterally by the cartilaginous edges of the annulus fibrosus (11). MRI with gadolinium showed ring enhancement of the IDH caused by the granulation tissue and its neovascularization at the edge of the herniation in chronic condition (2,7). An IDH as in cases 1 and 8 may mimic other intradural spinal pathologies including; neurofibroma, menigioma, epidermoid tumor, lipoma, arachnoid cyst, and metastasis (5). All cases in this study did not have MRI with contrast, as it is not routinely performed during lumbar exam.

From the analysis of neuroimaging and intra operative finding; cases no 1 and 8 had true intradural mass with caudal migration, cases 4, 5, and 7 had a characteristic “hawk beak” perforation of the dura with intradural fragment. But cases no 2, 3, and 6 had different presentation; they were interdural rather than intradural with relative cranial migration. The dura was partially perforated with thin layer

Figure 6 The T1- and T2-weighted axial cuts (A,B) demonstrate a huge compressing lesion to the dural sac with mild cranial migration at the L4–5. On a sagittal T1 (C), and T2 (D), revealed there is an abrupt loss of the continuity of the posterior longitudinal ligament. MRI myelogram (E) revealed complete irregular amputation of the myelogram medium at the L4–L5 level.

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covering the disc. The term intradural remains erroneous. Transdural would be the better term for herniation that pierces the dura. However, there is a conventional understanding of the term in the literature (11). During the surgery, there is a tense dura together with intradural mass during palpation (26) (Figure 11A-C).

However, despite the advances in the earlier neuroimaging techniques, the final diagnosis of IDH is often made during the intraoperative period (2).

In this study, as with other studies, L4–5 was the most frequent disc (2,5,12,20). In all intradural cases, the dura was open in the dorsal midline for an easy closure, even if the content was a paramedian. The ventral dural is left open. The study exhibited no single CSF leakage cases. The most suitable explanation for such statement was due to firm adhesion between the ventral dura, PLL, and disc capsule. For intraradicular, delivery of the root disc material was attempted in the second case between the dural layers. Minor CSF leakage was noticed with free muscle graft and gel foam.

In literature, it has been mentioned that the radicular sheath should not be opened routinely; however, this procedure should be undertaken in cases where after the removal of the intradural fragment, a tense or dilated radicular sheath is found, probably indicating the existence of another fragment (27) (Figure 11D).

The prognosis of IDH is not as those with no complicated disc herniations (2,7,20,24,27,28). D’Andrea et al. (7) reported satisfactory result in 67% of cases, with patients always able to go back to work normally, while in 33% of cases, there is a residual neurologic deficit. The prognosis is related to the duration of symptoms, the kind of symptoms (radiculopathy versus CES) and the presence of previous surgery (2,7,11,12,20). In the presented study, all cases of cauda improved due to early surgical intervention. The patients presenting only radicular pain preoperatively had completed recovery after surgery. In case 7 due to chronic of foot drop, the patient did not improve. In case 8, the patient had improved

Figure 7  A T1- (A) and T2-weighted image in axial (B,C) and sagittal T2 (D,E) demonstrates a wedge-shaped disc herniation to the right suspicious for intradural extension of disc fragments at the L4–L5 level (a sharp beak-like appearance of intradural disc herniation, see white arrow). It is more prominent in sagittal than in the axial plane. She had a small disc at L5–S1.
except for mild gait disturbance, and case 5 who developed some caudal symptoms post-operative that improved after one week.

**Conclusions**

Lumbar IDH is very rare pathologies. The pathogenesis is not well known. But those adhesions are due to a result of traumatic irritation such as; a herniated disc or previous surgery. MRI with gadolinium is still the gold standard neuroimaging study. The final diagnosis of IDH is often made during the intraoperative period. During the surgery, transdural would be the better term for herniation that pierces the dura according to the invasion stages.

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**Figure 8** A T1- (A,B) and T2-weighted sagittal image in axial (C,D) demonstrates a voluminous intradural huge mass suspicious for intradural extension of disc fragments at the L4–L5 level. It is continuous with the disc (see white arrows). Disc fragment crossed dura seen clearly in sagittal T2 (E,F). Scar of previous surgery noticed along right L5 side (F). Other discs were noticed at L5–S1 with preserved continuity of the posterior longitudinal ligament. MRI myelogram (G) revealed completely irregular amputation of the myelogram medium at the L4–L5 level.
Dorsal midline incision of the dura is safe, and ventral dural can leave open with no complication. Root invasion can be left, if there is adequate decompression of the root. It usually lies between two dural layers. The prognosis of IDH is not as easy as any complicated disc herniation. Early intervention, especially for CES carried good prognosis.

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**Footnote**

*Conflicts of Interest:* The authors have no conflicts of interest to declare.

*Ethical Statement:* This study was approved by the Ethical Committee of the Department of Neurosurgery at Suez Canal University. All patients studied in this paper had a consent during admission that they were enrolled in a scientific research. The consent form was accepted during neurosurgery meeting committee.

**Figure 9** A T1- and T2-weighted sagittal image in case 9 axial (A,B) and sagittal T2 (C) demonstrates a wedge-shaped disc herniation to the right suspicious for intraradicular extension of disc fragments at the L4–L5 level. The patient had multilevel degenerated discs. Intra operative the disc is calcified.

**Figure 10** A T1-, and T2-weighted axial (A,B) and sagittal (C,D) images demonstrate a cone shape voluminous disc herniation into the right S1 nerve root and continuous with it. An intraradicular extension of disc fragments at the L5–S1 level. The patient had multilevel degenerated disc. MRI myelogram (E) revealed complete irregular amputation of the right S1 root in myelogram medium at the L5–S1 level.
Figure 11 Transdural disc herniation stages presented in axial and sagittal view. (A) Transdural large disc perforates PLL and dura with remaining thin sheet of the dura. Herniated disc usually directed cranially; (B) beak-like hard disc perforates PLL and dura, with no dura sheet. It had a tendency for caudal migration; (C) large intradural disc fragment usually presented with caudal migration; (D) intraradicular disc herniation in the preganglionic region. Large and tense right root compared to the left one. PLL, posterior longitudinal ligament.

References

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