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Case Report

Intradural lumbar disc herniation: case report

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Abstract

Intradural disc herniation is a rare complication that, despite the precision of modern diagnostic tools, is usually confirmed intraoperatively. Therefore, we aimed to present a case of intradural disc herniation, reviewing the incidence, pathogenesis, clinical presentation, diagnostic options, and management.

A 67-year-old man presented with a history of hypertension, rheumatoid arthritis, and previous surgery for a herniated disc at the L4–L5 level. He was suffering from severe low back pain radiating towards the legs for four weeks that was followed by progressive weakness of the lower extremities to the point of inability to walk unassisted. The neurological examination demonstrated weakness of the muscles of both thighs and distal legs, absent knee-jerk and Achilles reflexes bilaterally, hypoesthesia of L4, L5, and S1 dermatomes bilaterally, and inability for plantar and dorsiflexion of the feet, while the bowel and bladder control were preserved. Magnetic resonance imaging (MRI) showed signs suggestive of disruption of the posterior longitudinal ligament at the L3–L4 level, the presence of intradural air, and a ratio between the maximum diameter of the herniated disc and the maximum diameter of the dural sac of 0.75. These findings made us suspect an intradural herniated disc at the L3–L4 level that was confirmed intraoperatively.

MRI findings that detect disruption of the posterior longitudinal ligament, presence of air in the intradural space, and a high ratio between the maximum diameters of the herniated disc and the dural sac are suggestive of the diagnosis, especially in the presence of two or more imaging findings.

Keywords

intradural, lumbar, disc herniation, diagnosis, MRI, surgery

Introduction

Intradural disc herniation (IDH) is a rare condition characterized by protrusion of the nucleus pulposus into the intradural space. It affects more men, usually between the ages of 50 and 60.[1] IDH was first described by Walter E. Dandy in 1942, and to date, just over 150 cases have been described in the English language literature.[2,3]

IDH is a serious condition that can cause severe neurological deficit and presents a significant surgical challenge, as patients are at risk from the herniation itself and from potential iatrogenic complications associated with surgical removal of the fragment.[3]

IDH most commonly occurs in the lumbar region (92%), followed by the thoracic segment (5%) and the cervical spine (3%).[4,5] In 55% of the cases, IDH is localized between the L4–L5 vertebrae, followed by L3–L4 (16%) and L5–S1 (10%), and in isolated cases at the L2–L3 or L1–L2 levels.[4,6]

There are two types of IDH: intra-arachnoid and extra-arachnoid.[7] Extra-arachnoid disc herniation is located between the dura mater and the arachnoid mater, in which

there is no leakage of cerebrospinal fluid, despite the rupture of the dura mater. Mut et al. distinguish between two types of IDH: type A, in which the nucleus pulposus is entirely intradural, and type B, in which the nucleus pulposus penetrates the nerve root's dural sheath.[8]

Case description

The patient is a 67-year-old male with a medical history of hypertension, rheumatoid arthritis, and prior surgical intervention for a herniated disc at the L4–L5 level, which occurred six years prior. After the initial surgery, he had mild persistent chronic lower back pain. Four weeks before his readmission to the clinic, he experienced severe lower back pain that radiated to his legs after physical exertion. Gradually, the patient developed weakness of the lower limbs to the extent of being unable to walk unassisted.

Upon admission to the clinic, he presented with severe lumbar vertebral syndrome with limitation of the movements in the lower back. The neurological examination revealed bilateral weakness of the thighs and distal lower legs, absent knee and Achilles reflexes with hypoesthesia across the L4, L5, and S1 dermatomes, and inability to plantar and dorsiflex the feet (2/5). Bowel and bladder control were preserved.

The preoperative MRI revealed suspicious data for a rupture of the posterior longitudinal ligament at the L3–L4 level, the presence of an intradural focus with air intensity, and a ratio between the maximum diameter of the disc herniation and the maximum diameter of the dural sac of 0.75 ***(Figs 1, 2)****. These findings let us consider an intradural median disc herniation at the L3–L4 level.*

Surgery was indicated given the clinical symptoms and MRI findings. After laminotomy at the L3–L4 level, a tense dura mater was encountered. We performed a midline durotomy, and after arachnoid and nerve root microsurgical dissection, we discovered that the intradural disc fragment was tightly attached to the cauda equina's roots. The IDH was completely removed by meticulous microsurgical technique ***(Fig. 3)****. Histological examination confirmed degenerative nucleus pulposus tissue. Postoperatively, we documented substantial reduction in pain syndrome and improvement of the lower limb motor weakness. The patient's one-year follow-up revealed mild hypesthesia along the L4 and L5 dermatomes on both sides, as well as residual motor weakness in the feet (4/5). He was able to walk unassisted.*

Discussion

IDH is more common in men with a mean age ≥60 years, a finding that aligns with the presented case. Nonetheless, instances of IDH have been documented in younger patients.[9,10]

The pathogenesis of IDH is not completely understood, although some predisposing anatomical factors are known. [11] First of all, these are existing adhesions between the annulus fibrosus, the posterior longitudinal ligament, and the dura mater. At the L4–L5 and less so at the L5–S1 levels, the posterior longitudinal ligament is firmly connected to the annulus fibrosus, which facilitates the perforation of the dura mater by the herniated disc.[12] Adhesions can be congenital, as in cases of congenital lumbar spinal stenosis and smaller epidural space, or acquired as

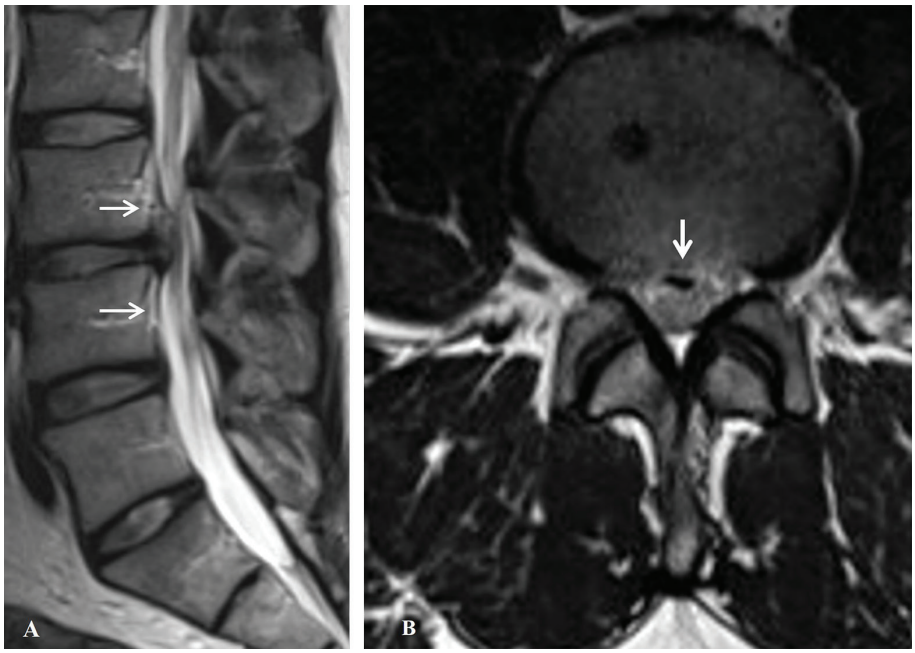

Figure 1. Preoperative T2-weighted MRI: **A**. Sagittal view at level L3–L4 shows suspected rupture of the posterior longitudinal ligament at the L3–L4 level (arrows); **B**. Axial projection at level L3–L4 shows the presence of an intradural focus with air intensity (arrow).

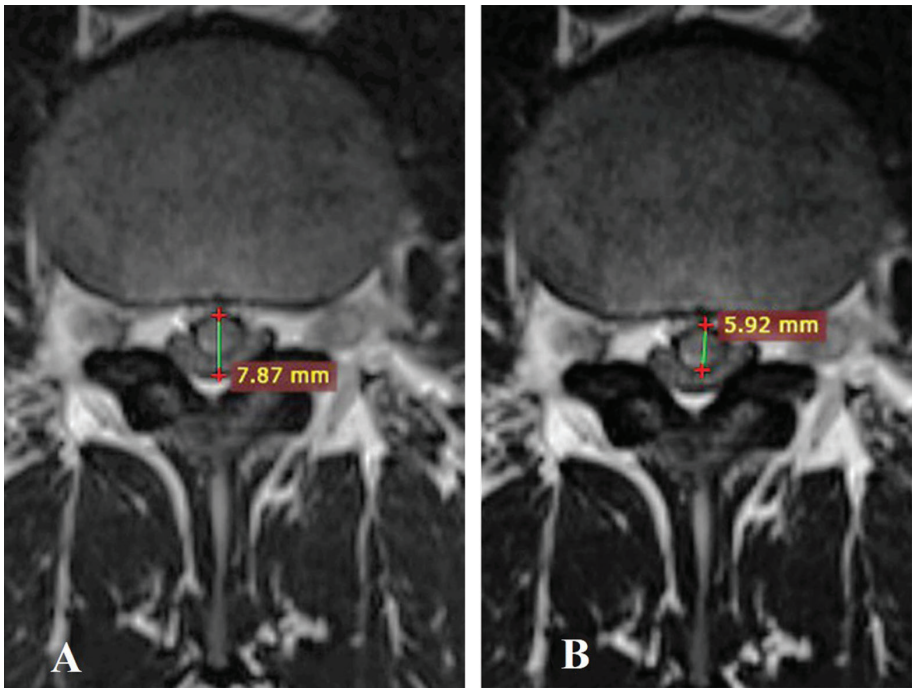

Figure 2. Preoperative T2-weighted MRI: **A**. Axial projection – shows the maximum diameter of the dural sac, and **B**. the maximum diameter of the disc herniation.

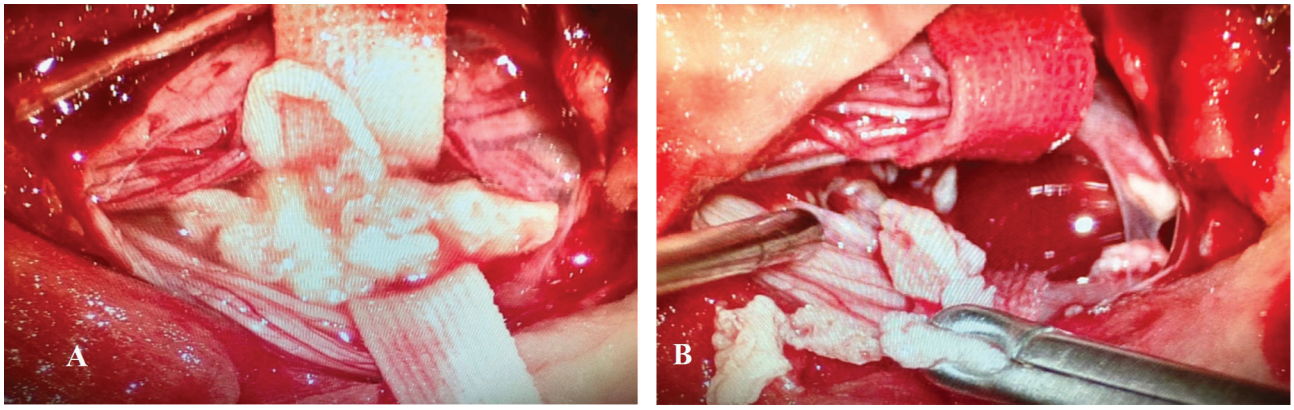

Figure 3. Intraoperative images: **A**. Intradural disc fragment between the cauda equina nerve roots was visualized; **B**. Stage of intradural fragment removal.

a result of degenerative changes or chronic inflammatory diseases, such as rheumatoid arthritis or postoperative adhesions.[4,13] Large herniations of the nucleus pulposus, especially if calcified, acquired spinal stenosis, and congenital reduction in dura thickness are other predisposing factors for the development of IDH.[14] Long-term mechanical compression of the dura mater is also a pathogenetic factor for the development of IDH.[15] The pressure of the ossified posterior longitudinal ligament and the presence of osteophytes on the dura mater cause its thinning and reduce its resistance, making it prone to perforation.[16] The history of previous surgical intervention for a herniated disc 6 years ago, the presence of rheumatoid arthritis, and the long-term compression of the dura mater in our patient are most likely the contributing factors for the intradural penetration of the herniated nucleus pulposus, which confirms the pathogenetic factors for the occurrence of IDH reported in the literature.[4,13,15] The clinical presentation of disc herniations depends on their location, with no significant difference in symptoms between intradural and extradural disc herniations.[4,13,14] In most reported cases of IDH, there is a higher incidence of cauda equina syndrome, reaching up to 30% of the cases, compared to 0.5–1% in extradural disc herniations.[4,14] The clinical symptoms that are most frequently reported in the literature are low back pain, sensory disturbances, varying degrees of motor disturbances, and urinary incontinence, but there are also reported cases with minimal symptoms.[17-19] The clinical symptomatology of our case coincides with that described in the literature and includes long-standing chronic pain in the lumbar region

that substantially intensified after physical load, followed by immediate progression of sensory and motor deficit in the legs.

Despite the great advancement in neuroimaging techniques, preoperative diagnosis of IDH is rarely achieved. In their literature review, D'Andrea et al. found that only 6.5% of the cases had an accurate preoperative diagnosis.[4] Similar results were reported by Matsumoto et al., who found that only 13% of cervical and 7% of lumbar IDH were diagnosed preoperatively.[15] The inaccuracy of preoperative diagnosis is determined both by the insufficiency of neuroimaging studies and by the low frequency of IDH, which is usually not suspected by surgeons.[7] In most cases, the accurate diagnosis is confirmed intraoperatively, but this may be associated with difficulties during the surgical intervention due to poor planning.[7] Most authors who have reported cases of patients with IDH focus their efforts on searching for specific imaging findings that allow for a preoperative diagnosis of this complication. Myelography, CT, CT-assisted myelography, and MRI have historically been used to diagnose IDH. Myelography most often shows a filling defect or complete block of contrast material, but it cannot always determine whether the compression is extra- or intradural, and its use has been abandoned due to its invasive nature and low specificity. CT also cannot provide a specific imaging feature of IDH. According to Mercier et al., the presence of non-specific tissue in the intradural space, with a density similar to that of the intervertebral disc (64 HU), which does not enhance after contrast media application, suggests intradural disc herniation.[20] Anda et al. reported that the presence of gas in the intradural space is also suspicious for IDH.[21] MRI is currently considered the gold standard for the diagnosis of intradural disc herniation. Several MRI findings have been reported in the literature that are highly suggestive of the IDH diagnosis. Choi et al. defined the so-called "hawk-beak sign," which is characterized by a sharp, beak-shaped compressive lesion in the dural sac on T2-weighted axial images.[22] On sagittal T2-weighted images, disruption of the posterior longitudinal ligament is also suggestive of IDH.[22]

Sasaji et al.[23] identified a characteristic image in cases of intradural extra-arachnoid disc herniation, which is characterized by separation of the arachnoid from the dura mater, which they called a "Y" sign **(Fig. 4)**.

According to Ducati et al., the contrast agent in enhanced MRI sequences is captured in the nucleus pulposus margin due to the presence of granulation tissue at the nucleus pulposus periphery and its neovascularization.[14] This sign is useful for differentiating IDH from intradural schwannomas and meningiomas.[14] The presence of air-intensity zone in the intradural space also suggests disruption of the integrity of the dura mater and possible IDH[24] **(Fig. 1B)**.

Nam et al.[25] discovered that cases with a ratio of the maximum diameter of the herniated disc to the diameter of the dural sac greater than 0.6 may indicate intradural disc herniation **(Fig. 2A, B)**. According to Kang et al., in the presence of two or more of the listed imaging findings, the probability of predicting IDH is 86%, and in the presence of one or less, the probability is 9%.[26]

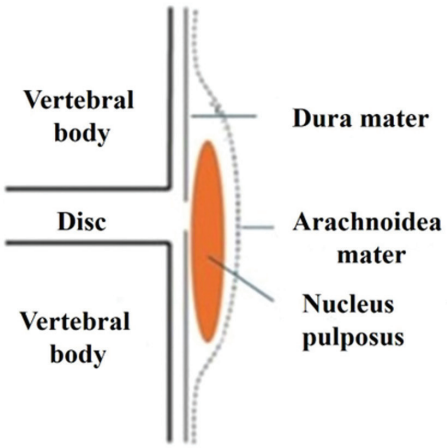

Figure 4. Diagram of the "Y-sign" typical of intradural extra-arachnoid lumbar disc herniation.

Our MRI findings, such as a disruption of the posterior longitudinal ligament, an intradural focus with air intensity, and a high ratio between the maximum diameters of the herniated disc and the dural sac, gave us reason to suggest the diagnosis of IDH, which was confirmed intraoperatively. This confirms the statement of Kang et al. that in the presence of two or more imaging findings typical of IDH, the probability of its occurrence is very high.[26]

The treatment of IDH is surgical, most often consisting of performing a laminotomy, dorsal durotomy, careful microsurgical dissection of the nerve roots from the disc fragment before its extraction, checking for extradural disc fragments, and resealing of both the dorsal and ventral dura, taking care not to cause additional neurological deficits.[7] The use of microsurgical techniques is essential for precise identification of the intradural disc fragment and separation of the adherent nerve roots. When there is tense dura mater and suspected intradural fragment, intraoperative ultrasound is appropriate, which not only identifies the presence of the IDH but also its precise location, which helps to plan the best site for durotomy.[27] After removal of the intradural fragment, it is sometimes advisable to perform a discectomy with intervertebral fusion to completely remove the nucleus pulposus in order to prevent possible recurrence and segmental instability.[28]

Kim et al. used a transforaminal endoscopic technique for removal of IDH, which they reported to give good results, fewer complications, and early mobilization of patients.[29]

With accurate preoperative diagnosis, Choi et al. used an anterior transperitoneal approach, which avoids dissection of the nerve root and the dural sac, minimizes intradural manipulation, and allows for easier repair of the dural defect with fat tissue and adhesive agent.[22]

The methods of ventral dural defect repair vary in the literature. In most cases, direct suturing of the defect is not possible. In these cases, the repair is done with an autogenous fascial or fat graft and adhesive agent.[7,30] After successful removal of the intradural disc fragment, it is advisable to perform dural defect repair, which is the most optimal approach reported in the literature.[25]

The prognosis of IDH treatment depends on the severity and the duration of the clinical symptoms.[9] In patients with cauda equina syndrome, the outcome of treatment depends on the time from the onset of the symptoms to the surgical intervention, which ideally should be performed within the first 24 hours.[31] Timely decompression is the most important factor for favorable neurological recovery.[32] The literature review of the outcomes of surgical treatment done by D'Andrea et al. showed that 62% of the patients with cauda equina syndrome recovered completely within 3 weeks to 32 months.[4] The remaining patients had some sensory disturbances in the legs and perineal area.[22,33]

Conclusion

IDH is a rare complication, and despite the accuracy of modern imaging modalities, the preoperative recognition of this condition is uncertain and is routinely confirmed intraoperatively. MRI findings of "hawk-beak sign," disruption of the posterior longitudinal ligament, the presence of an air-intensity zone in the intradural space, a large ratio between the maximum diameters of the herniated disc and the dural sac, and the capture of contrast material along the periphery of the nucleus pulposus may suggest IDH, especially in the presence of two or more imaging findings. In these cases, careful preoperative planning of the surgical procedure by spinal surgeons is necessary to prevent further complications and improve postoperative outcomes.

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Competing interests

The authors have declared that no competing interests exist.

Author contributions

Ivo Kehayov, Atanas Davarski, Georgi Slavov and Borislav Kitov contributed to the design and implementation of the research, to the analysis of the data, and to the writing of the manuscript.

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