

# Original Article: Fifteen Pearls in treating lumbar disk herniation: A Narrative study

Seyed Reza Mousavi<sup>1</sup> , Keyvan Eghbal<sup>2</sup> , Navid Kalani<sup>3</sup> , Ali Kazeminezhad<sup>4\*</sup> 

<sup>1</sup>Assistant Professor of Spine Surgery, Department of Neurosurgery, School of Medicine, Chamran Hospital, Namazi Teaching Hospital, Shiraz University of Medical Sciences, Shiraz, Iran.

<sup>2</sup>Assistant Professor of Spine Surgery, Department of Neurosurgery, School of Medicine, Namazi Teaching Hospital, Shiraz University of Medical Sciences, Shiraz, Iran.

<sup>3</sup>Research Center for Non-Communicable Diseases, Jahrom University of Medical Sciences, Jahrom, Iran

<sup>4</sup>Department of Neurosurgery, Peymanieh Hospital, Trauma Research Center, Jahrom University of Medical Sciences, Jahrom, Iran



**Citation** S.R. Mousavi, K. Eghbal, N. Kalani, A. Kazeminezhad\*, **Fifteen Pearls in treating lumbar disk herniation: A Narrative study.** *Eurasian J. Sci. Technol.* **2023**, 3(1):10-21.

 <http://dx.doi.org/10.22034/EJST.2023.1.3>



## Article info:

**Received:** 31 May 2022

**Accepted:** 10 June 2022

**Available Online:** 13 June 2022

**Checked for Plagiarism:** Yes

**Language Editor:** Ermia Aghaie

**Editor who Approved Publication:**

**Professor Dr. Ali Nokhodchi**

## Keywords:

Lumbar disc herniation, Surgical method, Positioning technique, Sciatica, Prone position, Anterior versus posterior approach, Contralateral radiculopathy, Incidental dural tearing.

## ABSTRACT

Lumbar disc herniation (LDH) is a common disease in neurospine surgery. Conservative treatment is effective in 80-90% of patients, and a small percentage (10-20%) need an operation. Lumbar discectomy is the most prevalent and easy surgery; however, it may cause serious catastrophic complications. To avoid complications and treat the LDH, some pearls constitute an important guide. These pearls include correct patient selection, the timing of the operation, correct correlation between patients signs and symptoms and level of LDH, right side of the disc, correct imaging study, underlying instability, nondiscogenic sciatica, the correct location of disc, surgical method, differentiating hip pathology, good positioning technique, anesthesia care and pearls, upper versus lower LDH, and incidental dural tear.

## Introduction

The incidence of LDH is about 5-20 cases per 1000 adults per year, with 1-3% in symptomatic LDH, and 15-20% of these patients need surgery. The most

common age range of herniated lumbar disc disease is in the 3<sup>rd</sup> to 5<sup>th</sup> decade of life. It is more frequent in male than female[m:f=2:1]. In 1934, the first operation of LDH was done by Mixter and Barr as laminectomy, durotomy, and discectomy. Later it was done by Semmes as

\*Corresponding Author: Ali Kazeminezhad (kazemimd@msn.com)

hemilaminectomy and discectomy. This technique is the most frequent spinal operation, and still, it has potentially serious complications [1, 2]. The following 15 pearls are important tips in LDH, both in treatment and in complication avoidance.

### *Correct Patient Selection*

Inappropriate patient selection is an essential cause of failed operation in surgical therapy of LDH. Psychosocial factors are important issues in patient selection, and the following factors are influential in the outcome of surgery: compensation issues, litigation issues, and unemployment at the time of operation are risk factors for poor prognosis. The imaging study interpretation is a significant factor in case selection and prognosis. Radiologic reports of small central disc herniation or protrusion are so common and are in favor of an annular bulge and are not to be used for justifying operation. LDH with moderate or severe defects with lateralization are good candidates for surgery with a good outcome of about 93% [2, 3]. Neurological findings confirm the level diagnosed by imaging studies, which are not predictive of outcome [2, 3].

There is a significant association between positive root tension sign and outcome. Restriction of straight leg raise test (SLR) as less than 60 degree is associated with a great outcome. Considering the SLR test as positive, the restriction should be unilateral and restricted by leg pain, not back pain. If there is bilateral SLR limitation, it should be considered skeptically other than an associated large imaging defect or anatomic neurological findings. There is a relationship between outcome and positive reverse lasague test or femoral nerve stretch test in LDH of l23 or l34 levels [2, 3].

### *Timing of Operation*

Sciatica or leg pain is the paramount manifestation of LDH and can be prolonged for days to years. At first nonsurgical therapy is started. With prolonged noxious stimulus, some changes occur in the neural system that maintains pain after removal of stimulus. This is

called central hypersensitivity. With disc herniation, sensitization of neurons in the dorsal horn of the spinal cord and other regions in the somatosensory pathways occurs. Considering this sensitization, the ideal time for surgery in sciatica is between 2 to 12 months from the onset of sciatica; before 2 months is too early and after 12 months frequently leads to poorer outcomes with cases suffering from chronic neuropathic pain and sensory disturbance. In leg pain, the ideal time for surgery is within 6 months from the onset of leg pain and most frequently leads to a good outcome; however, more studies are needed to prove this more conclusively [4].

### *Correct Correlation between Patient's Manifestations and the level of LDH*

There is a difference in the level of diagnostic reliability and accuracy of clinical manifestations of l5s1 and l45 disc herniation [DH], with more reliability and accuracy of l5s1 than l45 DH presentations. Because of the lower cross-sectional area of the spinal canal at the 4th level compared with 5<sup>th</sup> level, double root compression is more frequent in 4<sup>th</sup> disc herniation than in 5<sup>th</sup> disc herniation. In lower LDH, clinical presentation for level diagnosis is particular but relatively insensitive [5].

Foot drop is another clinical presentation of concern. Foot drop [FD] can be central or peripheral. If there is intact active ipsilateral hip abduction, it indicates intact ipsilateral L5 nerve root and helps to differentiate a central from a peripheral cause; electrophysiologic testing is also confirmatory. In L5 nerve root lesion as a cause of FD, there is the weakness of dorsiflexion of the foot, paresis of foot inversion, and paresis of foot eversion in contrast to common peroneal nerve lesion with FD that presents with paresis on eversion with intact foot inversion. For detection of the location of these lesions, we can use electromyography and nerve conduction studies [6-13].

### *Correct Side*

As a rule, lumbar disc herniation at any side, right or left, causes the same side signs and symptoms. However, this is not always right, and

rarely does LDH cause contralateral neurological deficit and radiculopathy. The probable causes of contralateral neurological deficits and radiculopathy are: friction radiculitis, migrated epidural fat, nerve root anomaly, or venous congestion, and the more popular cause is traction force generated on the contralateral nerve root or contralateral lateral recess stenosis. Electrodiagnostic findings are particular in these situations, but MRI is very sensitive. Because of this, if EMG findings favor definitive or suspicious contralateral lateral recess stenosis on the contralateral side, then bilateral decompression is needed. If there is no contralateral lateral recess stenosis or radiculopathy on EMG on the symptomatic side, bilateral decompression may not be necessary because in this situation, traction more than direct compression is a probable cause of contralateral symptoms [14, 15]. Further studies should be done to prove this more precisely.

### *Correct Imaging Study*

The most common ordered test for evaluating patients with sciatica is MRI. MRI is very sensitive in lumbar disc herniation, far lateral discs, and reoperation. We usually describe MRI findings based on the sagittal T1W & T2W images and the correlation of these findings with the transverse T2W images. For the reliable following of the whole nerve tract in MRI, we must take continuous slices with the exact angulation parallel to the level where nerve compression is suspected. MRI with different angulation in multiple levels is not reliable for following the whole nerve tract [16]. It is not recommended to use a saturation band on the anterior side or a rectangular field of view (RFOV).

Before operation, take a lumbar X-ray in all patients. Lumbar x-ray, especially with flexion and extension views, can be a helpful adjunct to other imaging evaluations [17].

Because of good clarification of bony structures in, CT-myelography is better than MRI in the following situation; CT-myelography may be done: in patients before reoperation and in patients with severe spondylotic changes [17].

### *Underlying Instability*

The stability of the motion segment changes with degenerative disc disease and facet joint osteoarthritis. Suppose there is a 3mm or more anterior translation in comparison between MRI and dynamic x-ray of lumbar spine in patients with LBP. In that case, it favors disc degeneration and facets joint disease [18]. A sagittal translation (ST) of  $\geq 4$  mm or  $\geq 8\%$  and sagittal rotation (SR) of  $\geq 10^\circ$  in L1-5 and  $\geq 2^\circ$  in L5-S1, respectively, are frequently accepted as a radiologic sign of pathologic mobility and seem to have a strong clinical impact. A low-grade spondylolisthesis with ST of less than 5 mm, compared with flexion-extension X-ray in a standing position, no longer seems to represent a criterion for fusion surgery [18-24].

In patients with annular tears or traction spurs dynamic x-ray as flexion-extension view should be taken. Conventional supine MRI is the technique of choice in acute and chronic LBP for detecting lumbar spine instability. Still, supine position MRI cannot answer a clinical question in one of three cases. Suppose the patient has clinical symptoms in favor of surgical intervention without any sign of cauda equina or lumbar nerve roots compression in supine MRI. In that case, it is better to repeat imaging in an upright position with the addition of flexion and extension to pick up the underlying instability [18-24].

Nondynamic imaging methods in the supine position can only recognize indirect radiologic signs of instability and some direct signs (malalignment of vertebral bodies). However, to detect changes in intersegmental motion, upright and positional MRI is needed [18-24].

### *Nondiscogenic Sciatica*

Any pathology in the sciatic nerve pathway from its origin (lumbosacral nerve root) to its bifurcation into tibial and common peroneal nerves can cause solastalgia. Sciatalgia can be discogenic or nondiscogenic. The presence of varicose vein in the limited epidural space is a challenging cause of discogenic sciatica. There are infrequent reports of sciatica due to idiopathic epidural varicosities. Secondary

epidural varicosis can occur in the followings: inferior vena caval anomalies (hypoplasia, aplasia), obstruction of inferior vena cava (in pregnancy or thrombosis), portocaval HTN, compressive lesions in the spinal cord, and the herniated disc itself. MRI is ideal diagnostic imaging. Patent large flowing vessels display decreased signal on T1W&T2W, whereas a thrombosed vein has increased signal intensity on both images. Although some authors proposed coagulative ablations in idiopathic and symptomatic epidural varicosis, the preferred approach is surgical resection of the varicose vein [25, 26].

### *Correct Location*

The most prevalent level of lumbar disc herniation is at L<sub>5</sub>S<sub>1</sub> level, and 95% of cases of LDH occur at L<sub>4</sub>L<sub>5</sub> and L<sub>5</sub>S<sub>1</sub> levels. LDH has 4-different locations: [27, 28]

**Far lateral location** -The area in the lateral part of the superior and inferior pedicles is the far lateral compartment. The far lateral disc represents 7-12% of all LDH. These far lateral discs are free fragments of a disc situated at the superolateral part of the disc space of origin. In far lateral disc herniation, compression of the superiorly exiting nerve root and ganglion occurs, culminating in superior nerve root syndrome. As far lateral disc herniation of L<sub>1</sub>L<sub>2</sub> → L<sub>1</sub> nerve root, L<sub>2</sub>L<sub>3</sub> → L<sub>2</sub> nerve root, L<sub>3</sub>L<sub>4</sub> → L<sub>3</sub> nerve root, L<sub>4</sub>L<sub>5</sub> → L<sub>4</sub> nerve root, L<sub>5</sub>S<sub>1</sub> → L<sub>5</sub> nerve root. Most frequently occur at L<sub>3</sub>L<sub>4</sub> or L<sub>4</sub>L<sub>5</sub> followed by L<sub>5</sub>S<sub>1</sub>.

**Foraminal location** -This location has the same presentation as far lateral with a different anatomic location of disc herniation relative to far lateral. In fact, far lateral disc herniation may be purely far lateral or extraforaminal or include intraforaminal components.

**Postrolateral location** - Disc protrusion is located at the posterolateral part of the disc space of origin and usually compresses the next lower nerve root. Hence posterolateral disc herniation of L<sub>5</sub>S<sub>1</sub> affects S<sub>1</sub> and L<sub>4</sub>L<sub>5</sub> affects L<sub>5</sub> nerve root.

**Central location** - It is less frequent. Central disc herniation above the level of L<sub>2</sub> can cause spinal cord compression or equine cauda syndrome. However, lower lumbar can cause S<sub>1</sub> radiculopathy [27, 28].

### *Surgical Method*

Conservative treatment is responsive in 90% of cases of acute sciatica, so in symptomatic lumbar disc herniation, the treatment is nonoperative unless the patient has refractory pain or an acute or progressive neurological deficit. Indications of operation are as followings: altered bowel and bladder function, progressive neurological deficits, and refractory radicular pain after 2-3 months of nonoperative therapy. Lumbar discectomy has no absolute contraindications. However, we must consider the following factors before decision making on lumbar discectomy: clinical radiologic discrepancy, primarily back pain, inadequate conservative therapy (2-3 months of conservative therapy and at least 6wk of physical therapy). The most common and routine surgical approach for lumbar discectomy is the posterior approach [29, 30].

### *Differentiating Hip Pathology*

In most cases, the hip and lumbar spine pathologies occur in combination. The common presentations of these patients are buttock, groin, and possibly knee pain. These patients have a challenging diagnosis and treatment [31].

### *History*

In history, the following symptoms are essential and must be considered [31-36]:

-Nocturnal pain may reflect a tumor or an infection.

-Groin pain is in favor of hip pathology

- Buttock and back pain favor lumbar spine pathology, but overlap exists.

-Hip pathology is usually associated with difficulty getting in and out of a car.

-Burning pain or pain with electric character favors lumbar spine pathology.

-Inability to lie on the side because of pain is in favor of trochanteric bursitis.

-Pain in motion of the hip or clicking or snapping in hip motion are in favor of intra-articular hip pathology.

-C-sign favors hip pathology [hip pain description with grasping the lateral aspect of the hip with their thumb and index finger in the groin].

-Postural pain in the groin and thigh is in favor of psoas pathology

-Postural low back pain is in favor of spinal instability

-Startup pain is a pain that usually starts with walking and resolves after 5 to 10 steps, and then gradually returns. Startup groin pain is in favor of a loose total hip arthroplasty (THA), and startup back or buttock pain is in favor of spinal instability.

### *Physical Examination [31-36]*

For detection of the primary source of pain complete physical examination must be done, including some common provocative tests that help in differentiating hip and lumbar spine pathologies as followings:

-Straight leg raise test (SLR): Leg raising with knee extension is done on this test. If pain elicited from 30 degrees to 60 degrees, it is in favor of lumbar (lower lumbar) radiculopathy.

-Contra-lateral SLR: Contra-lateral leg raising with knee extension is done on this test. If pain occurs in the contralateral leg from 30 to 60 degrees, it favors lumbar (lower lumbar) radiculopathy.

-Femoral nerve stretch test: Hip extension and knee flexion in a supine position is done on this test. If there is any pain, it is in favor of lumbar (upper lumbar) radiculopathy.

-Thomas test: Grasping one knee and flexing it to the chest in the supine position, on the unaffected side, is done on this test. If the contralateral leg does not fully extend, the test is positive and is in favor of hip flexion contracture of the examined leg.

-Ober test: With knee and hip flexion at 90 degrees while the patient is lying laterally on the unaffected side, the symptomatic hip and leg are brought from abduction to adduction if the leg remains in the abducted position and does not fall to the table and patient experience lateral knee pain the test is positive and in favor of iliotibial band and tensor fascia lata tightness.

-Anterior impingement test (FADIR test): The patient is in the supine position, and hip flexion, internal rotation, and adduction are done. If anterolateral hip region pain is elicited, it is positive and in favor of hip impingement (femoroacetabular impingement), hip labral tearing, hip loose bodies, hip chondral lesion

-Posterior impingement test (FABER test): Patient is in the supine position, and hip flexion, abduction, and external rotation are done. If sacroiliac joint pain is elicited, it favors sacroiliac joint disorders. With elicited groin pain, it is in favor of iliopsoas strain or intraarticular hip pathology; with elicited posterior hip pain, it is in favor of posterior hip impingement.

- Active piriformis contraction test: Patient is in a sitting position and pushes the heel down into the table or ground on the unaffected side and abducting, flexing, and externally rotating hip on the symptomatic side with the patient forward bending as the examiner monitors the piriformis. Pain and weakness may indicate sciatic nerve entrapment.

- Trendelenburg test: Patient is standing on one leg, the opposite hemipelvis drops and is in favor of weakness of gluteus medius on the standing leg.

### *Diagnostic Tests [31-36]*

-Plain radiography:

.Pelvis x-ray as anteroposterior and lateral view in patients suspected of hip OA.

.Spine x-ray as anteroposterior and lateral view with flexion-extension X-ray.

.36-inch anteroposterior and lateral standing X-ray for detecting spinal malalignment if there is any suspicion of spinal malalignment.

-MRI and CT can differentiate pathologic conditions of the hip from the lumbar spine, and in patients whose MRI is contraindicated CT scans of the lumbar spine in combination with CT myelograms are useful.

-Electrophysiologic studies are helpful for differentiation of radiculopathy from peripheral nerve disorders if other diagnostic tests are equivocal. Normal findings of these studies do not rule out the possibility of radiculopathy.

-Vascular studies are helpful in the identification of peripheral vascular disorders. In Leriche syndrome with internal iliac artery stenosis, buttock and thigh pain can occur. In vascular claudication patients, symptoms are aggravated with walking and improve with standing [31-36].

### *Anesthesia Care and Pearls*

The critical factor in anesthetic technique for the prone position is the correct placement and security of non-kinking ETT for providing a secure and reliable airway and ventilation. Swelling of the tongue causing macroglossia can occur due to its dependent position. Turning the patient from the supine to the prone position causes a fall in a cardiac index secondary to a decrease in stroke volume; thus, there is increased systemic and pulmonary resistance to maintain arterial blood pressure. Physiologic issues are significant in high-risk patients and possibly in the very obese when using the prone position. However, more recent studies have found that the prone position during general anesthesia doesn't negatively affect respiration; instead, it improves lung volume and oxygenation [37].

### *Good Positioning Techniques*

The prone position is commonly used for lumbar discectomy. Three types of complications have been associated with the prone position. The first type arises directly as a result of the method of positioning. This includes crush injuries to limb muscles resulting in myoglobinuria, pressure necrosis of skin, peripheral pressure nerve and positional brachial plexus palsies, and blindness.

The 2<sup>nd</sup> type arises when the operating site is above the level of the patient's heart allowing for potential venous air embolism.

The third type is associated with anesthesia in the prone position [37]. The most frequent prone position-associated complications with lumbar discectomy are nerve palsies and compression injuries. Simple padding with cushions, sheets, blankets, or egg crate padding prevents these complications. Excessive abduction of the shoulders can cause brachial plexus injury [38].

### *Upper versus Lower LDH*

Spondylosis, disc degeneration, and herniated discs are less common in the upper lumbar spine. Commonly in upper LDH, non-specific symptoms can occur as: lower back pain, buttock pain, and posterior thigh pain, with the typical clinical symptom as anterior thigh pain or inguinal pain. Ill-defined poly-radiculopathies occur following upper LDH because of the smaller cross-sectional area of the upper lumbar spinal canal. The femoral stretch test is frequently favorable in upper LDH. The femoral nerve mainly originates from L2, L3, and L4 spinal nerve roots, and because of this, in symptomatic upper LDH, the chance of a positive femoral stretch test is more than in lower LDH. Because of the low incidence and delayed diagnosis of upper LDH, treatment of upper LDH is complex and challenging. The outcome of the operation of LDH in upper lumbar levels is less reasonable than lower lumbar levels [39-44].

Unique anatomy of the upper lumbar spinal canal can cause the followings: polyradiculopathy or conus medullaris involvement may occur in upper LDH because of the narrower upper lumbar spinal canal, and/or direct cord compression may occur for shorter lengths of the lamina. Because of this particular anatomy, selecting a surgical approach is challenging and selecting a suitable surgical approach, anterior versus posterior, is a significant factor. Factors that are important in the selection of surgical approach are disc size, disc location, the severity of calcification, surgeon's experience, degree of spinal cord deformation, and the general medical condition

of the patient. If the disc herniation is primarily anterior to the spinal cord anterior approach can be used. Still, the preferable approach is the posterior approach because of the familiarity with this approach. However, with a small, focal, soft, and laterally located upper LDH in the spinal canal posterior approach is a suitable approach. So presurgical detection of the nature and amount of disc material is important. For the operation of upper LDH, we can do unilateral laminectomy in patients with focal, unilateral, and soft disc herniation or bilateral laminectomy in patients with bilateral symptoms and corresponding bilateral radiological evidence. Because medial facetectomy as 30% medial facetectomy in the lumbar spine and 50% in the cervical spine does not affect postoperative stability, we can do this to remove ruptured discs. If there is a large, central, or broad-based LDH with severe involvement of neural elements, the posterior transdural approach can be an excellent alternative. The postoperative outcome of upper LDH is less favorable than lower LDH [39-44].

### *Incidental Dural Tear*

The accidental dural tear is a relatively rare complication in lumbar decompressive operation. The rate of accidental dural tear is between 1-17%. The rate of dural tears is increased with an increased rate of reoperation and the advanced spinal degenerative changes with ossified yellow ligament among older adults undergoing operation [45, 46].

There are two main mechanisms of dural tearing as follows:

### *Intraoperative mechanisms*

Causes of dural tearing during primary procedures are dural erosion, thin dura, dural adhesions and fibrosis, dural redundancy, aggressive nerve root traction, implantation of instruments, and failure to recognize spina bifida occulta presurgically [45, 46].

### *Postoperative mechanisms*

These mechanisms are less common than intraoperative ones. Postoperative causes of the

dural tear are residual bone spikes or increased cerebrospinal fluid pressure [such as coughing, violent awakening from anesthesia, or postoperative seizures] that may disrupt a dural repair. Intraoperatively for detection of cerebrospinal fluid (CSF) leakage, we can do Valsalva maneuver (45,46).

Most cases of dural tear and CSF leakage are recognized intraoperatively. However, some cases cannot be recognized intraoperatively. Postoperative detection of unrecognized intraoperative dural tear and CSF leakage may be more complex, and the followings are helpful in postoperative detection of dural tear:

**=Clinical history.** Clinical presentation of unrecognized or unrepaired dural tear and CSF leakage are postural headache, nausea, vomiting, pain or tightness in the neck or back, dizziness, diplopia due to VI cranial nerve paresis, photophobia, and tinnitus.

**=Physical examination.** When there is an early clinical sign as a fluid collection at the operation site or watery discharge from the wound, a cerebrospinal fluid leak must be considered.

**=Laboratory tests.**  $\beta$ -2-transferrin immunofixation electrophoresis is a test for the detection of cerebrospinal fluid, and if there is any question about the diagnosis of CSF leakage, it can be done. Beta-2-transferrin is present only in CSF and perilymph and is produced by cerebral neuraminidase.

### *Imaging Studies*

**-Magnetic resonance imaging (MRI)**-MRI is very sensitive for CSF collection and pseudomeningoceles. MRI consistently shows a focal collection of extrathecal clear or blood-stained fluid.

**-High-resolution computed tomography (HRCT)**-HRCT detects bony defects and may accurately delineate fluid collections adjacent to bony lesions.

If there is any suspicion of CSF fistula, but MRI is negative newer imaging techniques are helpful:

**-Contrast myelography**-In contrast myelography, contrast material is injected into

the thecal sac with a following X-ray in a different patient's position.

**-CT-Cisternography**-Contrast myelography is often used with CT that, is named CT myelography and sometimes called CT cisternography. Disadvantages of CT-Cisternography are invasive, time-consuming, necessitates ionizing radiation, sensitive to detecting actively draining fistulous tracts at the time of the study, and is contraindicated in patients with intracranial mass lesions.

**-MR Cisternography**-It is a new examination method with a higher sensitivity for detecting csf fistula than ct cisternography.

### *Intraoperative Repair*

**Dural injury with possible primary repair**-For possible primary repair the dural tear must be adequately exposed and visualized. First, the laminectomy site is extended to create a suitable space for dural repair. With a Penfield Number 4 dissector site of the dural tear is probed, and extension of the dural tear is identified. For falling of nerve roots to anterior and relaxation of dural edges, gentle suction of CSF leakage must be done, and facilitating this Trendelenburg position is effective. For creating a dry surgical field, we can use a very small cottonoid in the defect during the early phase of the closure. The needle for suturing must be flexible, thin, and malleable and may need to bend it gently [45, 46]. Repair must be started a few millimeters above the proximal edge of the leak and tear to a few millimeters below the distal part of the tear. At this stage, Trendelenburg position is reversed, and the anesthesia team does the induced Valsalva maneuver. Placement of a small piece of double-layered blood-soaked Surgicel over the repair may enhance the dural rent seal [45,46].

After dural repair, the second layer that is very important is the fascial layer which must be sutured and closed with nonabsorbable suture material. Removal of the spinous processes and mobilizing the paraspinal muscle can facilitate such closure. Suppose approximation of the paraspinal muscles can not be made. In that case, lateral relaxing incisions are made bilaterally to

allow one to lift and pull the muscle toward the midline over the defect. For wound drains, there are two choices no wound drains or keep drains for 48hours [45, 46]. Postoperatively after dural repair following must be considered:

- for wound drains there are two choices no wound drains or keep drains for 48hours

- smooth reversal of anesthesia

- laxatives are prescribed, and narcotic usage must be avoided

- a urinary catheter should be left in place 3 days in the hospital and for 7 days at home after discharge from the hospital. If it is removed, the patient is allowed out of bed during the first 10 postoperative days only for bathroom privileges and meals [45, 46].

**Dural injury without possible primary repair**-There are two types of dural tearings without possible primary repair:

- Inaccessible dural tearings- Laterally situated dural lacerations are inaccessible. At this instance, the lateral patch technique must be used. A small piece of muscle or fat is tied to a suture, and a second midline durotomy is created. The graft is then inserted into the thecal sac and pulled through the lateral defect from the inside out, effectively plugging the tear.

- Accessible dorsal dural lesions- If these lesions cannot be closed primarily may be amenable to a fascia patch graft of autologous fascia from the fascia lata or the paravertebral muscles [45-47].

For augmentation of dural repairs, we can use fibrin glue (45-47). When fibrin glue uses for reinforcement of identical suture methods. With fibrin glue usage, epidural scarring and fibrosis are inhibited [45-48]. For testing the integrity and strength of the dural repair, we must test it intraoperatively with Valsalva maneuver to detect any persistent CSF leak or repair weakness. In dural repair and CSF leak, the most critical layer for effective repair is the fascial layer, which must be tightly repaired and sealed. After repair, the patient must be on bed rest with or without a drain [49-51].

### *Postoperative Repair*

For treatment of postoperative CSF leak, there are two options as following:

#### *Nonoperative Treatment*

**Subarachnoid drain-** An epidural catheter that is attached to a blood collection bag is inserted into the subarachnoid space away from the leakage site for 4 days with CSF drainage of 200 to 300 ml per day. Drainage volume of CSF can be adjusted by changing the height of the collection bag. This drainage system leads to the healing of dural tearing, if this technique fails, surgical treatment still is possible [52-54].

**Epidural blood patch-** For doing an epidural patch, after taking 20cc of blood from the patient's antecubital vein, it is injected into the epidural space near the fistulous tract. This may be a valuable tool in small persistent CSF leaks [55-57].

**Percutaneous fibrin glue-**We can use fibrin glue for postoperative treatment. After aspiration of CSF under CT guidance, a cryoprecipitate solution, calcium chloride, and thrombin solution are injected simultaneously; CT imaging confirms the fibrin adherence [58].

#### *Operative Treatment*

As operative therapy for intraoperative incidental dural tearing.

#### *Spontaneous Regression of LDH*

Since the first report, spontaneous regression of LDH can occur and has become broadly available in the literature. It is more common in large-sized and sequestered LDH than other LDH subtypes and can be partial or complete. Spontaneous regression of LDH most commonly occurs at L4-L5 level, which is also where LDH occurs more commonly [59-61].

In spontaneous regression of LDH the presence of transligamentous extension is more important than the initial size of the herniation. Suppose disc herniations show little to no signs of regression after 12 months. In that case, the

probable causes are the patients' younger age, the abundance of collagen fibers, and chondrocyte-like cells from the nucleus pulposus in these discs. However, why is the spontaneous regression of LDH more common in large, large, and sequestered disc? For followings:

- no more extended nutrient supplement from the parent disc, so dehydration and shrinkage with the following resorption occur.

- inflammatory response of sequestered discs. Inflammation is an obligatory factor in spontaneous regression of LDH and is an excellent prognostic indicator and should not be suppressed. This inflammation with increased blood flow causes rim enhancement in sequestered disc fragments in MRI with contrast [62-69].

Because of the higher probability and faster resolution rate in sequestered LDH compared to the other LDH subtypes, the first first-line management in these subtypes of LDH is conservative. The candidates of earlier operation are cases with intractable pain, neurological deficit, or bowel or bladder dysfunction. However, at present, it is difficult to predict which patients with DH conservative therapy have a higher possibility for spontaneous regression of disc [70].

### **Conclusion**

LDH is a common disease of the lumbar spine, and lumbar discectomy is an easy and straightforward operation in the lumbar spine but can cause adverse or catastrophic complications. Other than correct patient selection as the most essential factor for preventing these complications, some critical pearls and tips must be considered. The present article is discussing these tips and pearls.

### **Acknowledgment**

We would like to thank the Clinical Research Development Unit of Peymanieh Educational and Research and Therapeutic Center of Jahrom

University of Medical Sciences for providing facilities for this work.

### Funding

This research did not receive any specific grant from public, commercial, or not-for-profit funding agencies.

### Authors' contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

### Conflict of Interest

There are no conflicts of interest in this study.

### References

- [1] Alexander M. Dydyk, Ruben Ngnitewe Massa, Fassil B. Mesfin. Disc Herniation In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 Jan. 2020 Nov 20.
- [2] F. Omid-Kashani, H. Hejrati, S. Ariamanesh, *Asian Spine J.*, **2016**, *10*, 955-963. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [3] L.D. Herron, J.A. Turner, L.A. Novell, S.L. Kreif, *Clin. Orthop. Relat. Res.*, **1996**, *325*, 148-155. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [4] A.B. Sabnis, A.D. Diwan, *Indian J. Orthop.*, **2014**, *48*, 127-135. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [5] H. Reihani-Kermani, *Ann. Saudi Med.*, **2004**, *24*, 273-275. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [6] M.B. Katirji, A.J. Wilbourn, *Neurology*, **1988**, *38*, 1723-1728. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [7] W.B. Mathews, Clinical features of multiple sclerosis in clinical neurology. *Clin. Neurol.*, **1993**, *2*, 1098.
- [8] J.F. Kurtzke, *Neurology*, **1983**, *33*, 1444-1453. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [9] L. Steinman, *Sci. Am.*, **1993**, *269*, 107-114. [[Google Scholar](#)], [[Publisher](#)]
- [10] F.D. Westhout, L.S. Paré, M.E. Linskey, *J. Spinal. Cord Med.*, **2007**, *30*, 62-66. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [11] Iizuka Y, Iizuka H, Tsutsumi S, et al. *J. Neurosurg. Spine.*, **2009**, *10*, 260-264. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [12] E, Garrido, R.H. Rosenwasser, *Neurosurgery*, **1981**, *8*, 484-486. [[Google Scholar](#)], [[Publisher](#)]
- [13] H. Abdolhoseinpour, M. Mohseni, A. Kazeminezhad, PAINLESS FOOTDROP IN NEUROSURGERY. *Updates in Emergency Medicine*. Retrieved from, **2022**.
- [14] P. Kim, C.I. Ju, H. S. Kim, S.W. Kim, *J. Korean Neurosurg. Soc.*, **2017**, *60*, 220-224. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [15] H.K. Sucu, F. Gelal, *Eur. Spine J.*, **2006**, *15*, 570-574. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [16] R. Smithuis, Lumbar Disc Herniation and other causes of nerve compression, Publication date, 2014.
- [17] K. Sahrakar, Fr. Talavera, B.H. Kopell, M.G. Nosko, M. Melicharek, Lumbar Disc Disease Workup, Updated: Sep 25, 2018, [emedicine.medscape.com](http://emedicine.medscape.com)
- [18] A. Splendiani, L. Patriarca, S. Mariani, E.D. Cesare, M. Gallucci, *OMICS J. Radiol.*, **2015**, *4*, 2. [[Google Scholar](#)], [[Publisher](#)]
- [19] P.R. Dupuis, K. Yong-Hing, J.D. Cassidy, W.H. Kirkaldy-Willis, *Spine.*, **1985**, *10*, 262-276. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [20] F.P. Morgan, T. King, *J. Bone Joint Surg. Br.*, **1957**, *39*, 6-22. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [21] A. Kanemura, M. Doita, K. Kasahara, M. Sumi, M. Kurosaka, T. Iguchi, *J Spinal Disord Tech.*, **2009**, *22*, 479-485. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [22] I.R.A. Posner, A.A. White III, W.T. Edwards, W.C. Hayes, *Spine.*, **1982**, *7*, 374-389. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [23] C. Thome, D. Zevgaridis, O. Leheta, H. Bazner, C. Pockler-Schoniger, J. Wohrle, P. Schmiedek, *J. Neurosurg. Spine*, **2005**, *3*, 129-141. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [24] N.E. Epstein, *J. Spinal Disord.*, **1998**, *11*, 116-122. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [25] F. Omid-Kashani, E.G. Hasankhani, M. Fathi. *Iran. J. Med. Sci.*, **2015**, *40*, 541-543 [[Google Scholar](#)], [[Publisher](#)]

- [26] D.G. Kulcu, S. Naderi, *J. Clin. Neurosci.*, **2008**, *15*, 1246-1252 [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [27] Lucinda hampton, Chelsea Mclene, Andeela Hafeez, Fauve simoens and Liesa Rodet, Disc Herniation, physiopedia
- [28] wheelless textbook of orthopaedics, Disc hrniation
- [29] S. Jeswani, D. Drazin, J.C. Liu, C.H.R.I.S.T.O.P.H.E.R. Ames, F.L. Acosta, 2012. Anterior lumbar interbody fusion: indications and techniques. In Schmidek and Sweet's operative neurosurgical techniques: indications, methods and results (pp. 1955-1961). Philadelphia: Elsevier Saunders. [[PDF](#)]
- [30] R.J. Mobbs, A. Loganathan, V. Yeung, P.J. Rao, *Orthop. Surg.*, **2013**, *5*, 153-163. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [31] A.J. Buckland, R. Miyamoto, R.D. Patel, J. Slover, A.E. Razi, *J. Am. Acad. Orthop. Surg.*, **2017**, *25*, e23-e34. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [32] S.C. Cho, M.A. Ferrante, K.H. Levin, R.L. Harmon, Y.T. So, *Muscle Nerve*, **2010**, *42*, 276-282. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [33] M. Mondelli, A. Aretini, U. Arrigucci, F. Ginanneschi, G. Greco, F. Sicurelli, *Neurophysiol. Clin.*, **2013**, *43*, 205-215. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [34] M. Nadeau, M.P. Rosas-Arellano, K.R. Gurr, S.I. Bailey, D.C. Taylor, R. Grewal, D.K. Lawlor, C.S. Bailey, *Can. J. Surg.*, **2013**, *56*, 372-377. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [35] R.W. Crawford, G.A. Gie, R.S. Ling, D.W. Murray, *J. Bone Joint Surg. Br.*, **1998**, *80*, 279-281. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [36] J.B. Kleiner, R.P. Thorne, J.G. Curd, *J. Rheumatol.*, **1991**, *18*, 422-427. [[Google Scholar](#)], [[Publisher](#)]
- [37] H. Richard winn, youmans neurological surgery, 5<sup>th</sup> edition, vol1, chapter25, page600-604. [[Publisher](#)]
- [38] Lawrence S Chin, Amit Singla, Jorge E Alvernia, Lumbar Discectomy, Updated: Sep 24, 2019. [[Publisher](#)]
- [39] S.J. Bosacco, A.T. Berman, L.W. Rasis, R.I. Zamarin, *Orthopedics*, **1989**, *12*, 275-278. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [40] T.J. Albert, R.A. Balderston, J.G. Heller, H.N. Herkowitz, S.R. Garfin, K. Tomany, et al. *J. Spinal Disord.*, **1993**, *6*, 351-359. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [41] K. Hsu, J. Zucherman, W. Shea, J. Kaiser, A. White, J. Schofferman, C. Amelon, *Spine*, **1990**, *15*, 679-682. [[Google Scholar](#)], [[Publisher](#)]
- [42] S.P. Sanderson, J. Houten, T. Errico, D. Forshaw, J. Bauman, P.R. Cooper, *Neurosurgery*, **2004**, *55*, 385-389. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [43] D.S. Kim, J.K. Lee, J.W. Jang, B.S. Ko, J.H. Lee, S.H. Kim, *J. Korean Neurosurg. Soc.*, **2010**, *48*, 119-124. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [44] K. Abumi, M.M. Panjabi, K.M. Kramer, J. Duranceau, T. Oxland, J.J. Crisco, *Spine (Phila Pa 1976)*, **1990**, *15*, 1142-1147. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [45] S.J. Bosacco, M.J. Gardner, J.T. Guille, *Clin. Orthop. Relat. Res.*, **2001**, *389*, 238-247. [[Google Scholar](#)], [[Publisher](#)]
- [46] S.K. Kalevski, N.A. Peev, D.G. Haritonov, *Asian J. Neurosurg.*, **2010**, *5*, 54-59. [[Google Scholar](#)], [[Publisher](#)]
- [47] F.H. Mayfield, K. Kurokawa, *J. Neurosurg.*, **1975**, *43*, 639-640. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [48] J.E. Cain, R.F. Dryer, B.R. Barton, *Spine*, **1988**, *13*, 720-725, [[Google Scholar](#)], [[Publisher](#)]
- [49] F.J. Eismont, F.W. Wiesel, R.H. Rothman, *J. Bone Joint Surg.*, **1981**, *63A*, 1132-1136, [[Google Scholar](#)], [[Publisher](#)]
- [50] S.D. Hodges, S.C. Humphreys, J.C. Eck, L.A. Covington, *Spine*, **1999**, *24*, 2062-2064, [[Google Scholar](#)], [[Publisher](#)]
- [51] J.C. Wang, H.H. Bohlman, K.D. Riew, *J. Bone Joint Surg.*, **1998**, *80*, 1728-1732. [[Google Scholar](#)], [[Publisher](#)]
- [52] S.H. Kitchel, F.J. Eismont, B.A. Green, *J. Bone Joint Surg.*, **1989**, *71*, 984-987. [[Google Scholar](#)], [[Publisher](#)]
- [53] J. McCallum, J.C. Maroon, P.J. Jannetta, *J. Neurosurg.*, **1975**, *42*, 434-437. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [54] J.L. Stambough, C.R. Templin, J. Collins, *J. Spinal. Disord.*, **2000**, *13*, 39-41. [[Google Scholar](#)], [[Publisher](#)]
- [55] K.K. Lauer, J.D. Haddox, *J. Clin. Anesth.*, **1992**, *4*, 45-47. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]

- [56] N.F. Maycock, J. van Essen, J. Pfitzner, *Spine*, **1994**, *19*, 2223–2225. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [57] B.M. McCormack, S.L. Taylor, S., Heath, J. Scanlon, *Spine*, **1996**, *21*, 2273–2276, [[Google Scholar](#)], [[Publisher](#)]
- [58] M.R. Patel, W. Louie, J. Rachlin, *AJNR Am. J. Neuroradiol.*, **1996**, *17*, 495–500. [[Google Scholar](#)], [[Publisher](#)]
- [59] J. Key, *Ann. Surg.*, **1945**, *121*, 534–539. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [60] J.G. Teplick, M.E. Haskin, *AJR Am. J. Roentgenol.*, **1985**, *145*, 371–375. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [61] M. Macki, M. Hernandez-Hermann, M. Bydon, A. Gokaslan, K. McGovern, A. Bydon, *Clin. Neurol. Neurosurg.*, **2014**, *120*, 136–141. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [62] S.H. Ahn, M.W. Ahn, W.M. Byun, *Spine*, **2000**, *25*, 475–480. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [63] E. Takada, M. Takahashi, K. Shimada, *J. Orthop. Surg.*, **2001**, *9*, 1–7. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [64] T. Henmi, K. Sairyō, S. Nakano, Y. Kanematsu, T. Kajikawa, S. Katoh, V.K. Goel, *J. Med. Investig.*, **2002**, *49*, 40–43. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [65] K.V. Slavin, A. Raja, J. Thornton, F.C. Wagner Jr, *Surg. Neurol.*, **2001**, *56*, 333–336. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [66] R.A. Autio, J. Karppinen, J. Niinimäki, R. Ojala, M. Kurunlahti, M. Haapea, M. Haapea, H. Vanharanta, O. Tervonen, *Spine*, **2006**, *31*, 1247–1252. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [67] S. Kobayashi, A. Meir, Y. Kokubo, K. Uchida, K. Takeno, T. Miyazaki, T. Yayama, M. Kubota, E. Nomura, E. Mwaka, H. Baba, *Spine*, **2009**, *34*, 655–662. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [68] M. Yoshida, T. Nakamura, A. Sei, T. Kikuchi, K. Takagi, A. Matsukawa, *Spine*, **2005**, *30*, 55–61. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [69] C. Cunha, A.J. Silva, P. Pereira, R. Vaz, R.M. Gonçalves, M.A. Barbosa, *Arthritis Res. Ther.*, **2018**, *20*, 251. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]
- [70] C.C. Chiu, T.Y. Chuang, K.H. Chang, C.H. Wu, P.W. Lin, W.Y. Hsu, *Clin. Rehabil.*, **2015**, *29*, 184–195. [[Crossref](#)], [[Google Scholar](#)], [[Publisher](#)]

---

Copyright © 2023 by SPC ([Sami Publishing Company](#)) + is an open access article distributed under the Creative Commons Attribution License(CC BY) license (<https://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.