

REVIEW ARTICLE

Lumbar disc herniation and
radiculopathy: An integrative neurological
review of pathophysiology, diagnosis, and
evolving management paradigms

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Abstract

Lumbar disc herniation (LDH) is a primary cause of radiculopathy, a prevalent and debilitating neurological condition characterized by nucleus pulposus (NP) extrusion from the intervertebral disc (IVD), leading to the compression of nearby neural structures and causing neuropathic pain and neurological deficits. The pathophysiology of LDH involves progressive IVD degeneration, triggered by the accumulation of mechanical stress, inflammatory processes, and oxidative stress, leading to cellular damage, disc cell apoptosis, and extracellular matrix degradation. Clinically, LDH patients typically present with radicular pain, neurological deficits, and symptoms of sciatica, which may vary in intensity depending on the level and extent of the herniation. Diagnosis relies on clinical correlation and imaging studies, with magnetic resonance imaging as the gold standard. Treatment for a herniated NP depends on the symptoms and severity of nerve compression. Without severe or progressive neurological deficits, first-line treatment includes conservative management. Surgery is indicated when there is significant nerve compression, refractory pain, or progressive neurological deficits. This comprehensive review focuses on LDH, aiming to provide a neurologically oriented appraisal. First, it delves into the pathomechanism of LDH, offering updated insights into the biological and biomechanical processes that lead to disc herniation and subsequent nerve involvement. Second, it examines current diagnostic approaches, including advanced imaging techniques and neurological assessment methods, used to accurately identify and characterize LDH. Finally, it explores the evolving spectrum of management strategies, encompassing conservative treatments and various interventional and surgical options, with a particular emphasis on the neurological outcomes and considerations associated with each approach.

Keywords: Microdiscectomy; Lumbar disc herniation; Herniated nucleus pulposus; Lumbar pain; Low back pain; Spine surgery; Neurology

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1. Introduction

A herniated nucleus pulposus (HNP) is defined as the protrusion or extrusion of the gelatinous material from the intervertebral disc (IVD) through the fibrous annulus. Herniation may occur with or without radiculopathy. This process may manifest as either a partial or complete extrusion of disc material, or as a contained protrusion within the fibrous annulus.¹ HNP is a highly prevalent clinical condition, with a lifetime prevalence of up to 43%, and represents one of the leading causes of low back pain and sciatica. The clinical presentation may range from mild discomfort to severe neurological deficits, including leg pain, sensory disturbances, and motor weakness.²

A HNP primarily affects middle-aged adults and is becoming increasingly common due to the aging population and lifestyle factors, which collectively contribute to degenerative spinal processes. Although the majority of cases respond well to conservative treatment, a subset of patients experience persistent symptoms or severe complications. These complications include refractory low back pain, progressive motor weakness, cauda equina syndrome, and debilitating symptoms that significantly impair quality of life. For such patients, surgical intervention—most commonly discectomy—remains the treatment of choice.³

In this context, the present review aims to explore the core principles of microdiscectomy for managing degenerative lumbosacral HNP, based on an overview of anatomy, pathophysiology, clinical presentation, and radiologic-anatomic correlates. By synthesizing recent evidence, this work seeks to provide a comprehensive understanding of the condition and its surgical management, as well as to contribute to the refinement of current practices.

This manuscript is designed to serve as an educational resource for medical students, neurologists, junior spine surgeons, neurosurgeons, and other specialists involved in the care of patients with degenerative spine disorders.

2. Morphofunctional anatomy of the lumbosacral spine

The lumbosacral spine functions as a complex biomechanical pivot, supporting the largest axial loads in the vertebral column while facilitating multidirectional movement. Structurally, it consists of five lumbar vertebrae and the fused sacrum.⁴ For a spine surgeon, the critical anatomical landmarks are those defining the surgical window and the neural relationships. The interlaminar space, covered by the ligamentum flavum, provides the access point for the spinal canal. The neural foramen, a key site for compression, is bounded superiorly and inferiorly

by the pedicles, anteriorly by the IVD and vertebral body, and posteriorly by the facet joints and ligamentum flavum.^{5,6} Through each neural foramen passes the same-numbered spinal nerve root, recurrent meningeal nerves, and radicular blood vessels. The components of a lumbar vertebra are presented in [Figure 1](#).

Lumbar vertebral bodies are large and robust, almost square, with kidney-shaped bodies that are wider from side to side than from front to back. L4 and L5 vertebral bodies are taller in front than in back, contributing to the lordosis of the lumbar region. Up to 90% of the compressive load received by the vertebral bodies is carried by the inner cancellous bone, with the remaining 10% borne by the outer cortical shell of the vertebral bodies. Lumbar pedicles are longer and wider, ranging from 10 to 18 mm. The spinous processes are horizontal; L4 and L5 spinous processes are shorter than the other lumbar spinous processes and are difficult to palpate. The tips of the transverse processes of the lumbar vertebrae are located approximately 5 cm lateral to the midline and usually are not palpable.

Lumbar facets are oriented in a nearly sagittal plane, with the concave superior articulating processes facing medially and somewhat posteriorly, and the inferior articulating processes, lying medial to the superior processes, correspondingly face laterally and somewhat anteriorly. Specifically, lumbar facet joints are oriented more parallel to the sagittal plane at L2–L3 and L3–L4, allowing limited rotation and favoring flexion and extension movements; and the lower lumbar facet joints L4–L5 with increased coronal angulation facilitate greater rotational movements.⁷

Historically, the L4 spinous process was considered to be in a horizontal plane with the supracristal plane; however, more recent evidence shows that the supracristal plane is located at the level of L3–L4 in nearly 75% of the normal population, and in the remainder, this plane is found at either L2–L3 or L4–L5. Anteriorly, the vertebral level of the umbilicus is typically at the level of L3, but this varies depending on body type and weight.

2.1. The IVD and ligaments

The IVD comprises the central nucleus pulposus (NP)—a hydrated, gelatinous matrix rich in type II collagen and aggrecan—surrounded by the annulus fibrosus, a resilient ring of Type I collagen. Biomechanically, the Anterior Longitudinal Ligament (ALL) acts as a robust tension band during extension. In contrast, the Posterior Longitudinal Ligament (PLL), while reinforcing the annulus fibrosus, is significantly narrower (narrows caudally) and weaker. Its tapering width creates a specific zone of anatomical vulnerability in the posterolateral region, predisposing this

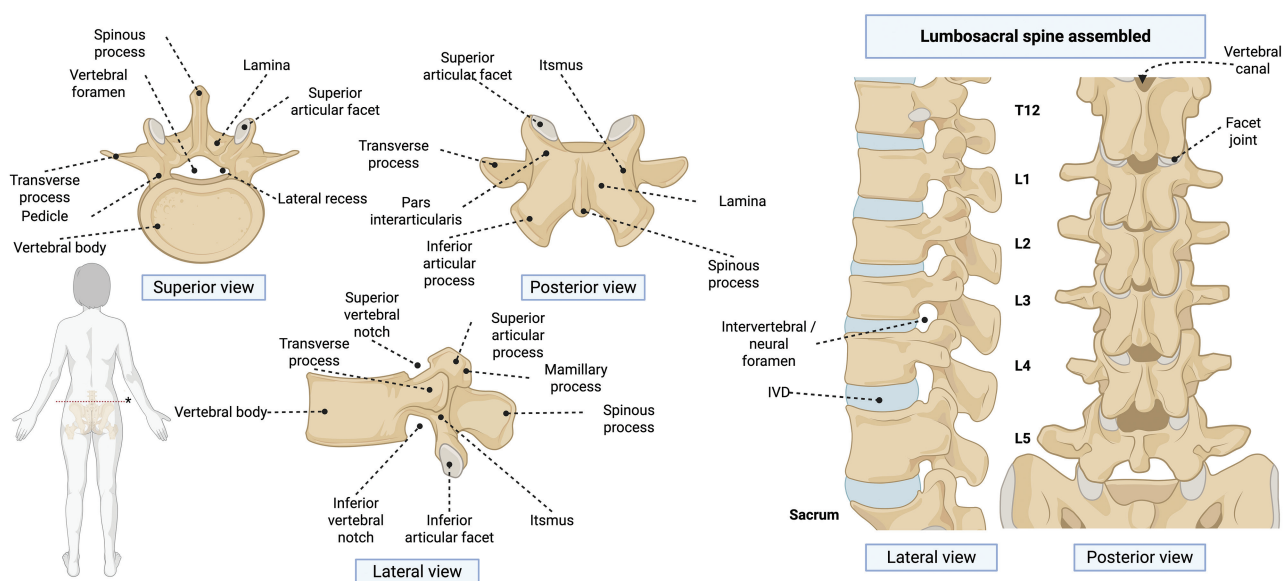


Figure 1. Superior, posterior, and lateral views of a lumbar vertebra, and lateral and posterior views illustrating the osseous anatomy of the lumbosacral spine. Asterisk (*) and the red line represent the supracristal plane (Tuffier line), an important body surface landmark for the lumbar spine. Image created by the authors.

Abbreviation: IVD: Intervertebral disc.

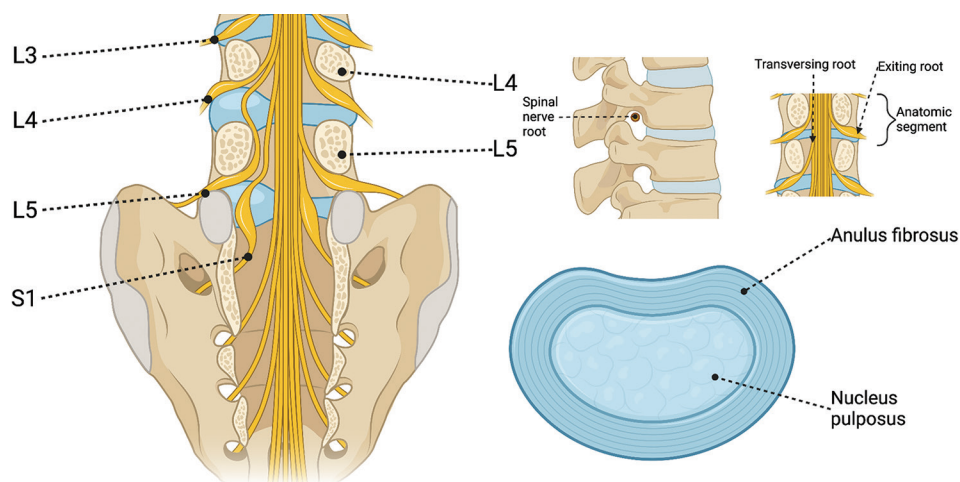


Figure 2. Lumbosacral nerve root course/arrangement in the spinal canal. Lumbar nerve root branches exit the dural sac one vertebral level above their respective foramen. The exiting nerve root and traversing root are shown at the upper-left corner. The traversing nerve root becomes the exiting nerve root of the anatomic segment below. Image created by the authors.

area to herniation during flexion-rotation stresses.⁴ While a healthy NP is avascular and aneural, the outer annulus contains nociceptive fibers, which play a significant role in the genesis of discogenic pain.⁸⁻¹² Figure 2 provides a superior perspective of the IVD structure.

2.2. Musculature and biomechanics

Stability is maintained by the intrinsic paraspinal muscles (multifidus and longissimus), which act as dynamic stabilizers during extension. The lumbar spine allows for

flexion, extension, and lateral bending, with the L4–L5 and L5–S1 segments providing the majority of sagittal motion, making them the most common sites for degeneration and herniation.¹³⁻¹⁶ More specific details regarding these movements and their respective ranges can be found in Table 1.

2.3. Neural anatomy and root relationships

The spinal cord typically terminates at the T12–L1 level, tapering into the conus medullaris and continuing as the

cauda equina. A precise understanding of the nerve root trajectory is paramount for avoiding iatrogenic injury. In the lumbar spine, nerve roots exit the dural sac one level above their respective neuroforamina. For example, at the L4–L5 disc level, the L4 nerve root is the exiting root (leaving the canal via the L4–L5 foramen), while the L5 nerve root is the traversing root (passing across the disc space to exit at the level below). Consequently, a paracentral disc herniation at L4–L5 typically compresses

the traversing L5 root, whereas a far-lateral or foraminal herniation affects the exiting L4 root.^{4,13,17} Throughout its course, the lumbosacral plexus and the sciatic nerve emit branches that provide sensory and motor innervation to the hip joint, thigh, leg, and foot. [Figure 3](#) illustrates the corresponding reflexes, dermatomes, and myotomes of the lower limb.

3. Physiopathology of lumbar disc degeneration and herniation

The pathophysiology of lumbar disc degeneration and herniation involves a complex interplay of structural and biochemical changes in the IVDs, disrupting their normal anatomy and influencing potential treatment strategies.¹⁸ In [Figure 4](#), the mechanisms involved in the pathophysiology of degenerative disc disease and IVD herniation are presented.

Disc cells regulate their extracellular matrix by synthesizing new components and breaking down existing ones through the production and activation of degradative enzymes, such as matrix metalloproteinases and a disintegrin and metalloproteinase. These anabolic

Table 1. Ranges of motion of the lumbar spine and key risk factors for mobility

Movement	Degree of mobility	Risk factors for mobility
Flexion	55.4±12.4°	Asymmetry between right and left lumbar facet joints
Extension	23.4±10.1°	Disc degeneration
Right lateral bending	16.4±7.2°	Obesity
Left lateral bending	18.3±5.7°	Connective tissue disorders
Right axial rotation	7.5±4.5°	Genetic predisposition
Left axial rotation	9.2±7.3°	Others

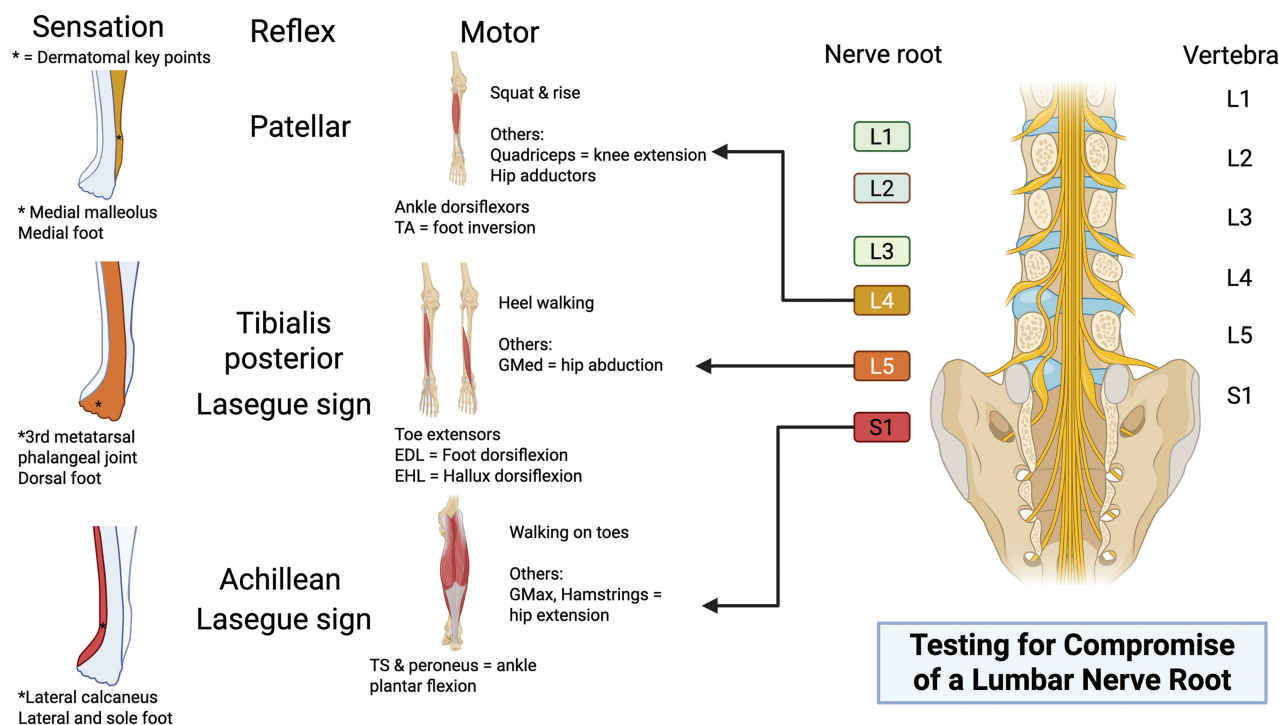


Figure 3. Principal lower extremity myotomes, dermatomes, and reflexes by lumbar nerve root. In high lumbar radiculopathies of L2 or L3, hip flexion may be impaired, making climbing stairs difficult or impossible. Middle lumbar (L2, L3, and L4) radiculopathies may affect knee extension, leading to reduced stability and a claudicating gait often accompanied by frequent falls. In L4 impairment, dorsiflexion and inversion of the foot may be compromised, sometimes resulting in a clubfoot gait in severe cases. In L5 radiculopathy, dorsiflexion and eversion of the foot may be impaired, causing reduced ankle stability. In S1 radiculopathy, plantar flexion is affected, which can lead to an inability to run. Image created by the authors.

Abbreviations: EDL: Extensor digitorum longus; EHL: Extensor hallucis longus; GMax: Gluteus maximus; GMed: Gluteus medius; TA: Tibialis anterior; TS: Triceps sural.

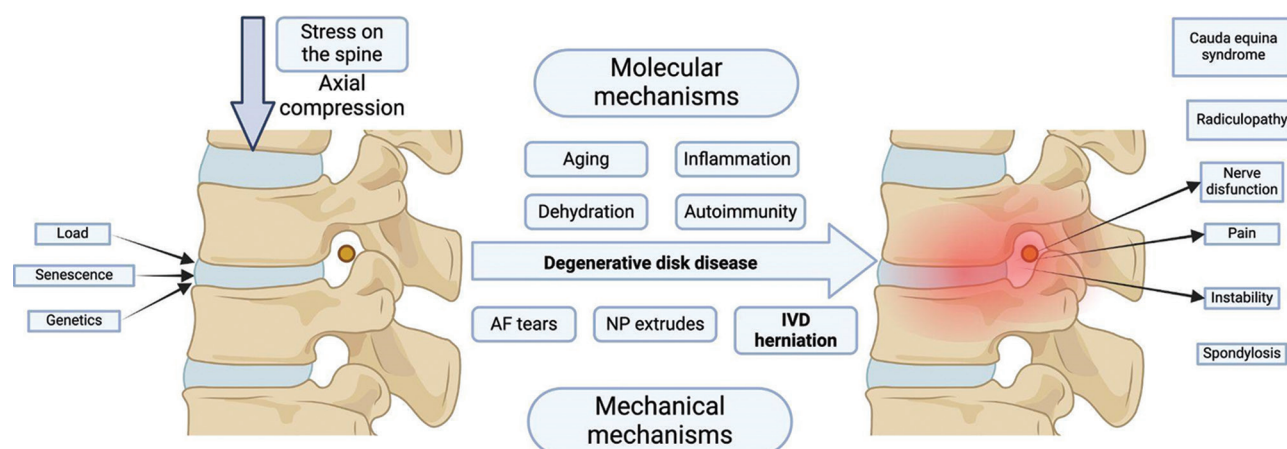


Figure 4. Mechanisms and consequences of degenerative disc disease leading to intervertebral disc herniation, including mechanical, molecular, and environmental factors. Image created by the authors.

Abbreviations: AF: Annulus fibrosus; NP: Nucleus pulposus.

and catabolic processes are critical in the initiation and progression of disc degeneration.¹⁹ Several growth factors, including bone morphogenetic protein-2 (BMP-2), BMP-7, growth differentiation factor-5, transforming growth factor- β (TGF- β), and insulin-like growth factor-1, stimulate matrix synthesis, while interleukin-1 (IL-1), IL-6, IL-10, IL-4, IL-8, and tumor necrosis factor- α (TNF- α) promote matrix degradation.^{9,10,20,21}

Degenerative changes in the annulus fibrosus, often associated with aging, contribute to HNP. These changes include desiccation, fissures, disc narrowing, mucinous degeneration, intradiscal gas formation, osteophyte formation, inflammation, and subchondral sclerosis. Fissures in the annulus compromise its structural integrity, permitting migration of disc material beyond its boundaries.²²

While mechanical, biochemical, and age-related damage are significant contributors, genetic predisposition plays a critical role in disc degeneration. Polymorphisms in genes related to cytokines, matrix components, oxidative stress, nutritional status, and risk factors such as obesity, infections, smoking, and metabolic disorders have been implicated.²³⁻²⁶ Matrix disruption in disc degeneration results from abnormalities in matrix component production, elevated matrix-degrading mediators, or reduced levels of tissue inhibitors of metalloproteinases.¹⁰ Senescent cells exacerbate this process by producing increased cytokines and matrix-degrading enzymes, exerting paracrine effects.²⁷ Unlike most tissues, where healing progresses from the inside outward, IVD repair occurs from the outside inward.²⁸ This ultimately results in a vicious cycle of damage, with the release of cytokines and antigens from the herniated disc, triggering more inflammation and further release of free radicals, thus perpetuating the injury.²⁹

Painful IVD exhibits distinct characteristics compared to asymptomatic degenerative discs. In pathological discs, the highly hydrated NP is replaced by fibrous tissue. Additional changes include the presence of inflammatory vascular granulation tissue and extensive innervation along tears in the posterior annulus fibrosus. These features are accompanied by elevated expression of growth factors such as basic fibroblast growth factor, TGF- β , and connective tissue growth factor, which collectively promote aberrant innervation and angiogenesis.^{28,30-32}

Blood vessels and nerves are primarily confined to proteoglycan-depleted areas of damaged tissue, particularly within annular fissures.³³ The inflammatory cascade, while facilitating resorption of herniated discs, also contributes to symptoms. Although uninjured IVDs lack significant innervation, exposure to the inflammatory cascade often results in nerve ingrowth, as evidenced by their presence in surgically removed herniated discs.^{34,35}

Chronic pain may arise even in the absence of herniation or nerve root compression, as degenerated discs can cause discogenic pain by releasing inflammatory cytokines that irritate nearby nerve fibers.³⁶ In addition, nociceptive nerve growth and vascularization into the disc are hallmarks of discogenic pain. Nerve fibers from the sinuvertebral nerve penetrate deeper and are denser in the endplates of degenerated discs compared to normal discs, further contributing to pain pathology.^{37,38}

3.1. Pathophysiology of nerve root neuropathic pain: Peripheral and central sensitization

Radiculopathy—neuropathic pain arising from the nerve root—is a complex chronic condition resulting from a lesion or disease of the somatosensory nervous system.

The inflammatory and mechanical insult to the nerve root initiates a cascade of maladaptive changes in both the peripheral and central nervous systems.

3.1.1. Peripheral sensitization

Injury or chemical irritation (e.g., from NP exposure or inflammatory cytokines released by the degenerated disc) to the nerve root and the dorsal root ganglion (DRG) leads to the hyperexcitability of primary afferent sensory neurons. This is driven by molecular changes, including the abnormal expression and trafficking of voltage-gated ion channels within the DRG and nerve terminals. This channelopathy facilitates the spontaneous generation of ectopic action potentials, perceived clinically as sharp, shooting, or burning pain.³⁹

Recent studies have highlighted the role of neuro-immune interactions in the periphery, with the release of pro-nociceptive mediators such as TNF- α and the activation of immune cells contributing to the lowered activation threshold of nociceptors.⁴⁰

3.1.2. Central sensitization

Persistent afferent input from the sensitized nerve root to the dorsal horn of the spinal cord is the main catalyst for central sensitization, which is considered the essential mechanism for maintaining chronic neuropathic pain. Central sensitization involves the increased excitability of second-order neurons, characterized by enhanced synaptic efficacy, particularly through the upregulation and sustained activation of N-methyl-D-aspartate and α -amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid receptors.⁴¹ A key feature is the involvement of non-neuronal cells (glia, especially microglia and astrocytes) in the spinal cord. These cells become activated and release inflammatory and neurotrophic factors that amplify pain signaling, weakening descending inhibitory controls. Clinically, central sensitization manifests as allodynia (pain from a non-painful stimulus) and hyperalgesia (exaggerated response to a painful stimulus).⁴²

4. Clinical features of low back pain with radiculopathy

The most common etiology of lumbar radicular pain is lumbar disc herniation (LDH). It is speculated that a large percentage of patients remain asymptomatic despite having an abnormal disc on magnetic resonance imaging (MRI).⁴³ In a study conducted on individuals without back pain, it was determined that approximately 52% of the subjects had a bulging disc, 27% had a protrusion, and 1% had an extrusion.⁴⁴ In symptomatic patients, it generally affects middle-aged individuals, with a predominance in males.⁴³

The pain is typically located in the lower back and associated with symptoms in the lower extremities, classically described as sharp and shooting pain radiating from the back to the buttocks, groin, or leg. Patients may also experience sensory symptoms, such as tingling, and in more severe cases, motor symptoms such as weakness, fatigue, or cramps may appear. The clinical presentation may vary depending on the affected level.^{1,43} The pain is triggered by activities requiring forward torso flexion, such as bending, coughing, getting up, and sitting, as these activities increase pressure on the IVD and, consequently, on the nerve root.^{43,45}

Patients with radicular pain due to a herniation can initially be diagnosed through medical history and physical examination, but confirmation requires imaging. However, red flag symptoms, including trauma, unexplained weight loss, neurologic findings, age >50 years, fever, intravenous drug use, steroid use, and history of cancer, summarized in the acronym TUNA FISH, must be ruled out, which would require imaging and appropriate referral.^{43,46} One high-risk condition is cauda equina syndrome. It is characterized by lumbosacral nerve root dysfunction (bowel, bladder, or sexual dysfunction), saddle anesthesia, asymmetric sensorimotor changes (diminished or absent sensation, decreased tone, and second motor neuron symptoms with paresis Medical Research Council power force $\leq$ M3 of lower extremity), and low back pain. Clinically, it is crucial to distinguish between incomplete CES (CESI) and complete CES (CESR). CESI is defined by variable lower extremity deficits and altered urinary sensation (e.g., hesitancy, loss of urge) but with preserved voluntary bladder and bowel control. In contrast, CESR is characterized by established painless urinary retention and overflow incontinence accompanied by anal atonia. The transition from CESI to CESR carries a worse prognosis; therefore, early recognition is vital. Treatment requires urgent spine decompression by decompressive laminectomy. Surgical outcomes are time-dependent. Current evidence and consensus guidelines recommend decompression as soon as possible, ideally within 24 hours and certainly within 48 hours of symptom onset, to optimize neurological and functional recovery, particularly regarding bladder and bowel function. Patients should undergo decompression immediately once the diagnosis is confirmed, as delays beyond 48 hours are consistently associated with poorer outcomes.⁴⁷⁻⁴⁹

The physical examination should include a comprehensive neurological assessment, evaluating reflexes, muscle strength, sensation, and a range of movement in both lower extremities (Figures 3 and 5).⁴⁶ A detailed examination may provide clues about the affected spinal cord site, but cannot determine the exact level.⁴⁸ Further details regarding the exploratory maneuvers are

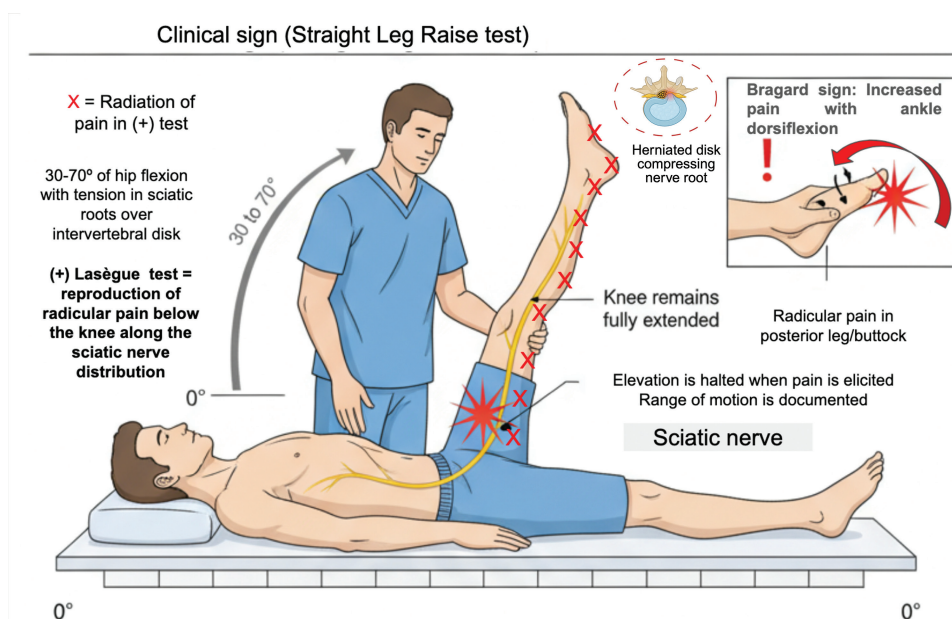


Figure 5. The Straight Leg Raise test (Lasègue's sign) for lumbar radiculopathy. The maneuver is performed with the patient in the supine position, passively raising the leg with the knee extended to stretch the L5 and S1 nerve roots over the IVD (hip in flexion and adduction, knee in extension). A positive test consists of the reproduction or marked worsening of the patient's initial radicular pain, radiating from the buttock to below the knee, typically occurring when the leg is elevated between 30 and 70°. This nocifensive response must be distinguished from hamstring and gluteal tightness, which elicits more diffuse discomfort and allows for higher elevation if performed slowly. While the sensitivity of the Straight Leg Raise (SLR) test for disc herniation is approximately 90%, its specificity is low. However, sensitivity may be increased by adding dorsiflexion of the foot (Bragard's sign) to further augment dural tension or by performing cervical flexion. It has high specificity and sensitivity in L4–L5 herniations. It has low specificity and sensitivity in L3–L4 herniations and migrated and sequestered disc herniations. Nerve bias of SLR includes the sciatic nerve and the tibial nerve. Image created by the authors.

provided in Table 2. Diagnostic criteria for acute radicular syndrome sciatica due to an HNP are presented in Table 3.

Laboratory tests are not routine for chronic back pain unless a more serious underlying condition is suspected; if so, general and specific tests should be requested as indicated by the clinical findings or presurgical evaluation.⁴⁶ Regarding imaging, it is not often used unless red flags are present. In the absence of red flags, imaging is recommended only when medical treatment is not effective after 4–6 weeks, with lumbar spine MRI being the imaging modality of choice.^{1,43} Moreover, multiple differential diagnoses should be considered, both neurological and extra-neurological, as listed in Table 4.

5. Complementary examinations

5.1. Lumbar spine MRI

MRI is the gold standard for detecting IVD diseases and for identifying the etiology of radicular syndrome caused by HNP. It offers excellent soft tissue contrast, allowing for more precise visualization of disc changes and nerve roots.⁵⁸ MRI can confirm suspected LDH with 97% accuracy and demonstrates a high correlation between neuroradiological interpretation and intraoperative findings.⁵⁹ However, MRI

is not indicated for routine use; it is primarily reserved for patients with lumbar pain persisting for less than six weeks when associated with neurological motor deficits or suspected cauda equina syndrome.⁶⁰

While MRI is the undisputed gold standard, clinical screening for contraindications is mandatory. In patients with non-compatible cardiac pacemakers, ferromagnetic aneurysm clips, metallic intraocular foreign bodies, or severe claustrophobia, MRI is precluded. In such scenarios, CT myelography serves as a valid alternative diagnostic modality to delineate nerve root compression.^{62,63}

5.2. Lumbar spine computed tomography

Lumbar spine computed tomography (CT) is used when an MRI is not feasible and can also confirm LDH. CT detects bone or calcified lesions and offers high spatial resolution and contrast, even with metal implants. However, it is invasive, requires a skilled radiologist, and carries risks from radiation exposure.^{60,61}

5.3. Functional/Dynamic radiographs

It is recommended to perform posteroanterior and lateral images, as well as include flexion and extension sequences

Table 2. Provocative tests for lumbosacral radiculopathy

Function test	Description	Accuracy	References
Lasègue test	Also known as the Straight Leg Raise test. The patient should be positioned supine, with the head extended and the hips and legs maintained in a neutral position, avoiding abduction, adduction, or rotation. The affected leg is passively and gradually elevated by holding the ankle, ensuring that the knee remains fully extended throughout the maneuver. When pain is elicited, the elevation is halted, and the range of motion is carefully documented. The test is considered positive if it induces radicular pain below the knee and/or if the pain occurs when the leg is elevated between 30° and 60°–70° from horizontal. (The “complete” test involves, once pain is reproduced, slightly flexing the knee to relieve pain, then re-stretching the nerve via ankle dorsiflexion to confirm the return of radicular pain).	Sen.: 72–97% Spe.: 11–66%	49,50
Crossed-over Lasègue test (Fajersztajn test)	The same maneuver as the Lasègue test is performed on the contralateral (asymptomatic) leg. During elevation, the patient reports pain on the affected side. This finding is typically indicative of severe nerve root compression or a central disc prolapse.	Sen.: 23–42% Spe.: 85–100%	49
Bragard Test	This test is a sensitizing maneuver for the Lasègue test. After a positive Lasègue test, the examiner lowers the patient's leg below the pain threshold and performs a dorsiflexion maneuver to further stretch the roots. When positive, it indicates pain due to sciatic nerve irritation.	Sen.: 69.3% Spe.: 67.4%	51
Kemp test	The patient may be positioned either seated or standing. They are instructed to maximally extend the spine, followed by rotation and lateral flexion toward the side of the pain. The examiner may apply axial pressure to increase the stress, although this step is optional. The test is considered positive if it reproduces lumbar or radicular pain.	Sen.: 31–100% Spe.: 11.6–67.3%	52
FABER test	The patient is positioned supine on the examination table and instructed to flex, abduct, and externally rotate the hip on the affected side. The examiner stabilizes the pelvis by placing one hand over the contralateral anterior superior iliac spine while applying downward pressure on the affected extremity, thereby generating tension across the anterior sacroiliac joint.	Sen.: 82% Spe.: 44%	53
Slump test	With the patient seated on a pedestal, with their knees in contact with the edge, they are instructed to relax the spine into maximum passive flexion while the examiner gently maintains cervical flexion. The patient is then asked to sequentially perform ankle dorsiflexion, ipsilateral knee extension, and cervical extension before returning to the initial position. The procedure is repeated on the contralateral side. The test is considered positive if it elicits neurological symptoms such as pain, paresthesia, or a burning sensation.	Sen.: 91% Spe.: 70%	54
Femoral Stretch Test	The patient should be placed in a prone position. The examiner passively flexes the knee toward the thigh. When positive, it indicates a high disc herniation (L2–L4).	Sen.: 50% Spe.: 100%	1,55

Notes: Other provocative tests include the Bowstring Test and Christodoulides Test. Specific bedside maneuvers can be helpful in determining whether symptoms are radicular in nature. The combination of Lasègue/Bragard, Slump, and Bowstring tests significantly improves the sensitivity and specificity of the physical examination for sciatica compared to relying on any single test.

Abbreviations: Sen.: Sensitivity; Spec.: Specificity.

Table 3. Criteria for the diagnosis of sciatica due to a herniated nucleus pulposus

No.	Description
1	Leg pain is the dominant complaint compared with back pain
2	Specific neurologic symptoms (paresthesia in a typical dermatomal distribution)
3	Significant Straight Leg Raise (SLR) changes
a	SLR <50% of normal
b	Bowstring discomfort
c	Crossover pain
4	Neurologic signs: weakness, wasting, sensory loss, or reflex alteration

Notes: 3 of 4 of these criteria must be present, the only exception being young patients who are very resistant to the effects of nerve root compression and thus may not have neurologic symptoms (Criteria 2) or signs (Criteria 4). Differential diagnosis includes central or lateral spinal stenosis, facet syndrome, sacroiliac articulation dysfunction, axial pain, peripheral neuropathy, and systemic diseases.⁵⁶

Table 4. Etiologies of sciatic neuropathies^{57]}

Etiologies of sciatic neuropathies	Percentage
Trauma	25
Iatrogenic (surgery)	25 ^a
Vascular	13
Prolonged compression and immobilization	11
Idiopathic progressive	7
Idiopathic non-progressive	5
Presumed postviral	3
All other etiologies	1

Note: ^aMostly orthopedic.

to assess spinal instability as a contributor to symptoms.⁵⁸ Findings may include decreased disc height, associated formation of syndesmophytes in the adjacent vertebral body, facet hypertrophy, and spondylolisthesis.⁶⁰

5.4. Electromyography (EMG), nerve conduction studies, and other electrodiagnostic studies

Electrodiagnostic studies—comprising nerve conduction studies, needle EMG, H-reflexes, and somatosensory evoked potentials (SSEPs)—provide critical functional and physiological data on the peripheral nervous system. Their primary utility lies in confirming the presence of radiculopathy, localizing the specific level of nerve root involvement, assessing severity and chronicity of the lesion, and excluding mimicking conditions such as peripheral neuropathy or plexopathy, particularly when clinical and radiological findings are discordant.⁴⁶

Nerve conduction studies can help differentiate a pre-ganglionic radiculopathy (where sensory nerve action potentials remain normal) from a more distal post-ganglionic lesion (excluding peripheral neuropathies and entrapment syndromes that may mimic radicular symptoms).

Needle EMG serves as the most specific electrodiagnostic tool for detecting radiculopathy by identifying a topographic distribution of muscular denervation corresponding to a single affected nerve root. EMG can identify patterns of active denervation (e.g., fibrillation potentials and positive sharp waves) and chronic reinnervation (e.g., large, polyphasic motor unit potentials with reduced recruitment) in a myotomal distribution. For an L5 radiculopathy, for example, this would involve findings in muscles such as the tibialis anterior, extensor hallucis longus, and peroneus longus, while typically sparing the paraspinal muscles if the lesion is intradural. The short head of the biceps femoris is a key muscle, as its involvement (innervated by L5–S1 via the sciatic nerve) helps distinguish a radiculopathy or sciatic neuropathy from a common peroneal neuropathy at the fibular head.⁶² EMG findings correlate more strongly with muscle weakness and functional impairment than MRI findings. An EMG-confirmed radiculopathy is associated with more severe pain and better clinical outcomes following surgical or interventional treatment.^{63,64}

The H-reflex is a monosynaptic spinal reflex that serves as an objective electrophysiological marker of the S1 nerve root. The compression of the S1 root, typically at the L5–S1 level, leads to reduced H-reflex amplitude and prolonged latency. Normalization of H-reflex occurs rapidly following effective decompression. Intraoperative monitoring has demonstrated a marked and sustained increase in its amplitude following the removal of an acute L5–S1 disc herniation, coinciding with clinical recovery.⁶⁵

The SSEPs assess the functional integrity of sensory pathways traversing the nerve root and spinal cord, providing

data often missed by conventional EMG. Dermatomal SSEPs demonstrate high sensitivity for detecting the level and severity of nerve root involvement, with reported concordance rates of up to 83% compared to imaging and operative findings.^{66,67} Additionally, SSEPs are especially valuable when EMG is non-diagnostic; surface electrode techniques have identified parameters such as the potentials elicited from L3/L4 (NL3) score and those elicited from T12/L1 (NT12) amplitude, which reflect acute and chronic neural impairment, respectively. The improvement in SSEP parameters following surgery confirms the functional impact of decompression on the sensory pathways.^{66,68}

6. Anatomoradiologic diagnosis

LDHs are classified based on MRI findings into different categories according to their morphology (protrusion and extrusion) or based on clinical relevance (contained and uncontained).^{69,70}

6.1. Protrusion

Protrusion occurs when the maximum distance between the edges of the disc material extending beyond the disc space is smaller than the distance between the edges of the base, within the same plane. The base is defined as the cross-sectional area of the disc material at the outer margin of the disc space of origin.

Protrusions can be classified as either “focal” or “broad-based,” depending on whether they involve less than or more than 25% of the disc circumference in the axial plane, respectively.

6.2. Extrusion

Extrusion occurs when the distance between the edges of the disc material beyond the disc space exceeds the distance between the edges of the base, in any plane. It can be further subdivided into:

- Sequestration: If there is no continuity between the extruded disc material and the parent disc
- Migration: If disc material is displaced away from the opening in the annulus through which the material was extruded, regardless of whether sequestration is present.

The term intravertebral herniation may be used to describe a type of herniation in which disc material extends through a rupture in the vertebral body endplate in the craniocaudal direction.^{69,70}

6.3. Contained

Refers to disc material that extends beyond the disc space but remains enclosed by the outer annulus. It typically has a smooth, regular margin on CT and MRI scans.

6.4. Uncontained

The disc material extends beyond the disc space without any surrounding containment. It often has irregular margins on CT and MRI scans due to the penetration of the outer annulus and the posterior longitudinal ligament by the disc material.

6.5. Location classification

Another classification is based on the herniation's location in the axial plane relative to the vertebral canal. This can be categorized as central, paracentral, subarticular, foraminal, or extraforaminal, depending on the anatomical region to which the herniation extends. Additional descriptions can be made based on volume and composition (MSU Classification).⁷¹⁻⁷³

Despite the importance of MRI findings, these classifications should be interpreted in the context of the patient's clinical presentation (clinical–radiological correlation), as surgery is indicated in symptomatic cases where the disc herniation is large enough that surgical removal is likely to alleviate nerve root compression.⁷¹ Thus, the correlation between MRI changes, clinical symptoms, and findings at the neurological examination is crucial for selecting the appropriate candidates for surgery and the surgical technique, thereby achieving a successful outcome.^{71,72}

7. Treatment of symptomatic lumbosacral disc herniation

Conservative management is recommended as first-line therapy for symptomatic lumbosacral disc herniation in the absence of red-flag features. Initial treatment aims to relieve pain, maintain function, and support spontaneous symptom resolution through patient education, activity modification, and multimodal non-operative care. Regarding activity restriction, strict bed rest—historically prescribed—is now discouraged based on Level I evidence. A Cochrane review comparing bed rest with other therapies concluded that it yields no benefit for low back pain and may potentially result in harmful physiological deconditioning. Therefore, current guidelines advocate for maintaining physical activity within pain tolerability limits rather than immobilization.⁷⁵ Invasive interventions are reserved for persistent disabling symptoms or progressive neurologic deficits.

7.1. Pharmacological therapy

Pharmacological therapy should be complemented with education on self-care and physical activity. Medications provide short-term pain relief but have no effect on the progression of disc degeneration. Pharmacologic treatment should be individualized.⁶⁰

- Acetaminophen: First-line treatment for both acute and chronic pain, though ineffective for severe pain. Preferred as a first-line treatment due to few side effects.⁷³
- Non-steroidal anti-inflammatory drugs: Anti-inflammatory and analgesic.⁷³ Effective in acute radicular pain, which is predominantly nociceptive, but less effective for chronic pain with a neuropathic component, where other therapies such as antidepressants are preferred.^{60,74}
- Glucocorticoids: Systemic glucocorticoid therapy is useful for acute episodes but has proven effective only in the short term.⁶⁰
- Opioids: Commonly used for chronic lumbar pain; however, their use is controversial due to addiction potential. Low doses are recommended only for carefully selected patients, where they can relieve pain and improve functionality without causing tolerance, dependence, or other side effects.⁷⁴
- Muscle relaxant: A class of drugs that relax skeletal muscles. The most commonly used muscle relaxant is cyclobenzaprine.⁷⁵ These drugs can be used for acute lumbar pain treatment. However, they have side effects such as gastrointestinal issues, dizziness, and headaches. Their therapeutic use is mainly for muscle stiffness related to spinal cord injuries, cerebral palsy, or stroke.⁷³
- Injections: These may involve local anesthesia, corticosteroids, or a combination of both, administered into the interlaminar space, neural foramen, or facet capsule. They reduce the inflammatory response of the herniated disc and edema, alleviating neural compression. They are recommended for patients who are unable or unwilling to undergo surgical treatment.⁶¹

7.2. Non-pharmacological therapy

7.2.1. Physical therapy

Up to 80% of patients with a prolapsed IVD respond to conservative therapy within 4–6 weeks.⁶⁰ Physical therapy implemented in the early stages of chronic pain helps relieve pain, increases paraspinal muscle strength, and reduces disability, medication doses, and the need for imaging or invasive therapies.⁴⁶

7.2.2. Behavioral counseling

Cognitive-behavioral therapy has shown better results compared to other therapies, improving pain intensity, disability, and quality of life. Mindfulness-based therapy yields similar outcomes.⁴⁶ Several risk factors have been identified as predictors of HNP recurrence, including smoking, younger age, male sex, obesity, and diabetes.^{76,77}

Among these, obesity has been recognized as a strong and independent predictor of recurrent HNP following lumbar microdiscectomy. Consequently, surgeons are encouraged to incorporate weight-loss counseling into preoperative discussions with patients.⁷⁸ However, the relationship between obesity and HNP remains controversial.⁷⁹

7.2.3. Multidisciplinary biopsychosocial rehabilitation

Multidisciplinary biopsychosocial rehabilitation focuses on addressing multiple factors shown to worsen chronic low back pain outcomes. It incorporates physiotherapy, cognitive-behavioral therapy, and education. There is still no consensus on the definition of multidisciplinary biopsychosocial rehabilitation.⁴⁶

7.2.4. Other alternative therapy

Osteoking is a traditional Chinese medicine used for degenerative musculoskeletal conditions. It has shown promising effects in LDH; however, the mechanism of action remains unclear. Available data suggest that its active components may target key inflammatory pathways affecting the lumbar disc at the molecular level. The most active components are kaempferol, quercetin, and β -sitosterol.⁸⁰ However, the clinical application of these therapies remains a controversial topic.

7.3. Surgical treatment

Surgery is indicated in cases of serious spinal disease, after ruling out red-flag conditions, and when nerve root dysfunction results from herniation. Imaging

can confirm LDH, but it does not always correlate with nerve root dysfunction. Therefore, clinical findings must be considered. Surgical decompression is recommended for patients with severe, disabling sciatica, persistent radicular pain lasting over four weeks without improvement or with extreme progression, and objective evidence of specific nerve root dysfunction with confirmed HNP at the corresponding level and collateral findings on imaging.^{81,82} There are no standardized protocols in the literature for referral to surgical treatment. Nonetheless, Puerto-Vásquez *et al.*⁸³ developed a calculator for this purpose, based on age, radiographic changes, the Lasègue sign, and Waddell's non-organic signs. Further studies are still needed to assess its applicability in other populations.

The main surgical options for HNP include invasive procedures (e.g., open discectomy, laminectomy, spinal fusion, and artificial disc replacement) and minimally invasive techniques (e.g., endoscopic spine surgery and percutaneous discectomy). Anatomical classification of HNP defines the surgical technique approach.

LDH resection by minimally invasive approaches is associated with better clinical outcomes, less blood loss, and shorter hospital stays.^{58,84-88} On the other hand, microdiscectomy is a variation of standard discectomy, assisted by the use of a surgical microscope, which allows for improved visualization and a smaller skin incision.^{89,90} It allows paramedian unilateral approaches with reduced bone and ligament resection. Microdiscectomy remains the

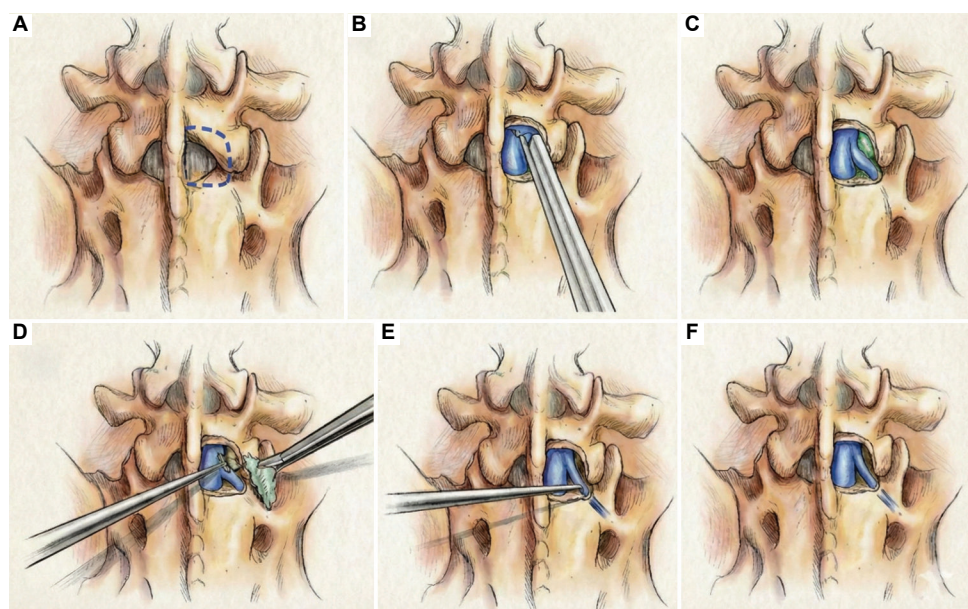


Figure 6. (A-F) Surgical steps involved in a single-level lumbar microdiscectomy for a posterolateral lumbosacral right L5-S1 herniated nucleus pulposus. Figures adapted with permission from Bartleson & Deen.¹⁰⁶

gold standard for surgical treatment for HNP (especially for central and lateral HNPs through interlaminar surgical approach).^{90,91} However, it still involves greater muscle injury than minimally invasive approaches.⁹⁰

For foraminal HNP, a medial partial facetectomy and partial laminectomy are important to allow a wide opening of the neuroforamen. For extraforaminal HNP, an intertransverse or Wiltse transmuscular access is used with a partial lateral facetectomy.

8. Single-level lumbar microdiscectomy as a surgical technique

Lumbar microdiscectomy is the gold-standard surgical technique and is generally safe and efficient, typically lasting 45–60 min. Performed under general or spinal anesthesia with the patient prone, proper positioning is crucial to prevent complications.^{93–95} Figure 6 presents the surgical steps.^{96–103}

Microdiscectomy is considered the gold standard for surgical treatment of HNP (especially for central and lateral HNPs through interlaminar approach). It is assisted by a surgical microscope, allowing for improved visualization, smaller skin incisions, and less tissue trauma compared to standard open approaches. At level L5–S1 (Figure 6), the pathology typically involves the S1 traversing nerve root shoulder. The clinical presentation most likely includes S1 radiculopathy, characterized by pain radiating down the posterior thigh and calf to the lateral aspect of the foot and heel, sensory changes in the S1 dermatome, plantar flexion weakness (difficulty walking on tiptoes), and a diminished or absent Achilles reflex.

Figure 6A presents the exposure and operative planning. Following fluoroscopic confirmation of the level and a standard midline posterior incision (2.5–4 cm), subperiosteal dissection of the paraspinal musculature is performed using a Cobb elevator to expose the relevant hemilamina and facet joint capsules. The dashed blue outline indicates the intended limits of the partial hemilaminectomy and medial facetectomy necessary to access the spinal canal without destabilizing the segment.

Figure 6B presents the bony resection and access (laminotomy and flavectomy). A high-speed drill (burr) is often used to thin the lamina, and a Kerrison rongeur (as depicted) completes the laminotomy. The bony resection typically extends cephalad to the pars interarticularis and caudad to include approximately 3 mm of the inferior lamina. Bony elements are carefully removed to expose the underlying ligamentum flavum. The underlying ligamentum flavum is carefully separated and excised (flavectomy), potentially including a limited medial facetectomy (approximately

3 mm) to release adhesions and widen the neuroforamen. This exposes the thecal sac (dural sac, shown in blue).

Figure 6C presents the visualization of neural elements and pathology. On entering the epidural space, the anatomy is delineated. Epidural bleeding is controlled (e.g., with bipolar cautery and hemostatic agents). A dissector (e.g., Penfield 4) is used to identify the traversing nerve root located laterally. In this view after medial retraction of the traversing nerve root, an extruded fragment of NP (shown in green) is exposed, causing significant compression and posteromedial aversing nerve root.

Figure 6D presents the nerve root retraction and microdiscectomy (sequestrectomy). To safely address the pathology, the traversing nerve root (and common dural sac) is gently retracted medially using a nerve root retractor, protecting it from injury. If not already extruded, a slit incision is made in the annulus fibrosus with a #15 blade. Pituitary, disc, or Caspar rongeurs are introduced to remove the herniated disc fragment and any degenerate NP, as indicated.

Figure 6E presents the exploration of the nerve root trajectory to the neural foramen. Following removal of the primary extruded fragment, the nerve root trajectory and neural foramen are carefully explored with a probe or exploration hook to ensure thorough decompression and that no residual loose fragments remain that could lead to early recurrence.

Figure 6F presents the final decompression. The nerve root has been released from retraction. Note the complete decompression of the traversing nerve root, which is now free of tension and has returned to its normal anatomical course laterally. Finally, the surgical field is irrigated, hemostasis is achieved, and wound closure is performed in layers (fascia, subcutaneous tissue, and skin).

9. Outcomes of microdiscectomy for symptomatic spinal disc herniation

Surgery for HNP aims to relieve nerve compression and alleviate pain, with outcomes generally showing favorable results. However, complications and recurrence rates remain considerations for long-term recovery.

Long-term positive outcomes following open microdiscectomy have been reported in 84% of cases.¹⁰⁷ Research has demonstrated that post-operative exercise programs focused on lumbar stabilization significantly improve outcomes, reducing pain, increasing spinal mobility, and facilitating a quicker return to work.¹⁰⁶ However, it is crucial to determine the optimal time to begin these exercises, as early initiation may lead to the separation of the annulus fibrosus layers before they have fully healed.⁷⁰

The complication rate for open microdiscectomy is reported to be 12.5%, which includes nerve root injury (0.4%), new or worsening neurological deficits (0.6%), hematoma (0.7%), wound complications (1.3%), durotomy (2.8%), recurrent disc herniations (3.3%), and reoperation (6.0%).^{93,94,109} Beyond these common adverse events, surgeons must account for rare but catastrophic intraoperative complications. These include wrong-level surgery and iatrogenic ventral perforation causing retroperitoneal injury to major vessels (e.g., abdominal aorta, inferior vena cava) or bowel visceral injury, which constitute life-threatening emergencies requiring immediate intervention.^{96,109} Other studies have indicated an average recurrence rate of 15%, with a range of 5–18%, influenced by factors such as smoking, heavy labor, increased disc height, continued weightlifting after the initial surgery, and degenerative facet changes.^{73,91,108} An interdisciplinary rehabilitation approach has also been recommended to achieve better outcomes following surgery.^{100,108}

10. Limitations of the technique and areas for development

While microdiscectomy is an effective procedure for relieving the compressive symptoms of HNP, it is considered a “palliative” surgery, addressing the immediate issue of nerve compression rather than the underlying process of disc degeneration. This limitation has spurred interest in advancements in regenerative medicine, aiming to address the root cause of disc degeneration.

A key challenge remains the recurrence of HNPs, with some patients experiencing reherniation or the development of new hernias over time. Moreover, the molecular mechanisms behind disc degeneration are still not fully understood, making it difficult to develop effective therapies to prevent further damage because there are multiple pathways that produce different grades of injury.

Future developments may involve intraoperative interventions that promote healing and help maintain the health of the IVD, potentially offering more sustainable solutions for patients undergoing spine surgery.¹¹

11. Interspinous process device (IPD) implantation and percutaneous full-endoscopic lumbar discectomy (PELD)

A recent study offers one of the first direct comparisons between microdiscectomy, IPD implantation, and PELD for the treatment of simple HNP.¹⁰⁹

The IPD implantation and PELD are distinct minimally invasive surgical techniques used in the management of lumbar spine conditions. IPD implantation involves the insertion of

a device between the spinous processes of the vertebrae. This procedure is primarily used to treat degenerative lumbar spinal stenosis by acting as an extension blocker, which helps to prevent compression of neural elements without the need for direct surgical removal of tissue adjacent to the nerves. The procedure is minimally invasive and aims to preserve spinal stability while providing symptomatic relief. Studies have shown that IPD implantation can yield outcomes comparable to traditional surgical methods, such as microdiscectomy, with similar rates of complications.¹¹² PELD, on the other hand, is a technique used to remove herniated disc material that is causing nerve compression. This procedure is performed using an endoscope, which allows for a minimally invasive approach through either a transforaminal or interlaminar route. The full-endoscopic approach is associated with reduced approach-related morbidity, shorter hospital stays, and fewer perioperative complications compared to traditional open discectomy techniques. Clinical outcomes of PELD have been shown to be similar to those of microsurgical techniques, with the additional benefits of being less invasive.^{111,113}

These findings show that all three procedures achieved significant and comparable improvements in pain relief, functional recovery, and patient satisfaction after one year. However, PELD stood out as the least invasive option, associated with the smallest incision, minimal blood loss, and the shortest hospital stay, translating into faster post-operative recovery and fewer wound-related complications. These results reinforce PELD as a safe and effective minimally invasive alternative to conventional discectomy, though its success largely depends on surgical expertise due to its learning curve and distinct complication profile, particularly regarding dural tears.¹⁰⁹

In contrast, IPD implantation demonstrated unique biomechanical advantages by being the only technique that preserved and even slightly increased disc height, while also showing the lowest rate of post-operative spinal instability requiring fusion.¹⁰⁹ This suggests that IPD may play a valuable role in maintaining segmental stability and reducing long-term degenerative changes. Despite high initial costs, both PELD and IPD provide benefits that may offset their expense through faster recovery or improved spinal preservation. Overall, the study supports a more individualized approach to LDH surgery, highlighting how advancements in minimally invasive and stabilization technologies can complement traditional microdiscectomy to optimize patient outcomes.

12. Conclusion

This review explored the pathophysiology, diagnosis, and management of lumbosacral HNP, with a specific focus

on the role of microdiscectomy in surgical treatment. HNP remains a prevalent cause of low back pain, with a considerable portion of affected patients experiencing persistent or severe symptoms. Although conservative management is effective for most, microdiscectomy provides substantial relief for appropriately selected patients.

Complementary minimally invasive options, including PELD and IPDs, further broaden available strategies for selected patients. Microdiscectomy continues to be the gold standard for surgical treatment of symptomatic LDH, with favorable long-term outcomes. However, recurrence rates, albeit low, and the potential for surgical complications such as nerve injury and durotomy remain important considerations. Post-operative rehabilitation, including lumbar stabilization exercises and interdisciplinary care, plays a crucial role in enhancing recovery and long-term functionality.

Future research should prioritize improving patient stratification for both surgical and non-surgical pathways, refining minimally invasive techniques to reduce complications and recurrence, and evaluating the timing and structure of rehabilitation programs. An integrated framework that considers nerve root compression, inflammatory mechanisms, and nociceptive processing is essential for accurate diagnosis and personalized management. More broadly, emerging biological and regenerative strategies—including stem cell-based approaches and targeted immunomodulation—may promote disc repair and reduce recurrence, thereby offering new treatment avenues. Lifestyle optimization—including weight management, smoking cessation, and structured physical therapy—remains essential for the long-term prevention and reduction of recurrence risk. In summary, effective management of lumbosacral HNP relies on a coherent, neurologically informed, and patient-centered approach that synthesizes advances in surgical practice, rehabilitation science, and biologically targeted emerging therapies.

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Conflict of interest

The authors declare that they have no competing interests.

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