Saudi Journal of Medicine and Public Health

https://saudijmph.com/index.php/pub

https://doi.org/10.64483/202522370

Lumbosacral Disc Injuries: A Physical Therapy–Centered Framework for Assessment and Rehabilitation

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Abstract

Background: Lumbosacral disc injuries are a leading cause of low back pain and radiculopathy, primarily affecting the L4–L5 and L5–S1 levels due to high mechanical demands. These injuries result from complex interactions between biomechanical stress, genetic predisposition, and inflammatory processes rather than isolated mechanical overload.

Aim: To present a physical therapy–centered framework for assessment and rehabilitation of lumbosacral disc injuries, emphasizing evidence-based evaluation and conservative management strategies.

Methods: This review synthesizes current literature on anatomy, pathophysiology, epidemiology, and clinical assessment, integrating findings from imaging studies, genetic research, and rehabilitation trials. It outlines diagnostic principles, red-flag screening, and treatment pathways, including exercise-based interventions and adjunctive measures.

Results: Evidence indicates that most patients (>90%) improve with conservative care, including structured physical therapy emphasizing trunk control, graded strengthening, and patient education. Imaging findings often lack correlation with symptoms, underscoring the need for clinical reasoning over reliance on MRI. Genetic factors account for approximately 34% of disc degeneration variability, while mechanical loading contributes minimally. Surgical intervention offers faster relief for select cases with persistent radicular symptoms but does not guarantee superior long-term outcomes compared to rehabilitation.

Conclusion: Physical therapy plays a pivotal role in managing lumbosacral disc injuries through individualized, function-oriented programs. Emphasis on movement confidence, graded exposure, and interprofessional coordination enhances recovery and reduces chronicity.

Keywords: Lumbosacral disc injury, physical therapy, radiculopathy, conservative management, rehabilitation, genetic predisposition.

Introduction

The lumbosacral region, spanning the junction between the lumbar vertebrae and the sacrum, represents a biomechanical transition zone that is inherently vulnerable to injury. This susceptibility arises because the lumbar spine is designed to permit substantial mobility—particularly flexion, extension, and rotation—whereas the sacrum functions as a comparatively rigid structure optimized for load transfer to the pelvis. The shift from a highly mobile segment to a more fixed base alters force distribution and increases mechanical demand at the lower lumbar motion segments, especially during activities that combine axial loading with bending or twisting. For physical therapists, this anatomic and mechanical context is central to understanding why lumbosacral disc injuries are so prevalent and why symptoms frequently emerge during common

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Receive Date: 28 November 2025, Revise Date: 23 December 2025, Accept Date: 24 December 2025

occupational and daily tasks. Between adjacent vertebral bodies lie the intervertebral discs—specialized fibrocartilaginous structures that contribute to spinal stability, permit controlled motion, and participate in load sharing. Discs function as viscoelastic cushions that attenuate compressive forces and distribute pressure across the vertebral endplates. Structurally, they are composed of two principal components: an inner nucleus pulposus and an outer annulus fibrosus. The nucleus pulposus is relatively hydrated and gelatinous, enabling it to deform under load and help disperse compressive stress. In contrast, the annulus fibrosus is formed by concentric layers of collagen-rich lamellae that provide tensile strength, limit excessive motion, and constrain the nucleus within the disc space. The integrity of this annulus is therefore essential for normal disc mechanics and for maintaining segmental control during functional movement. [1][2][3]

When the annular architecture is compromised—through repetitive microtrauma, sustained adverse loading, age-related changes, or acute overload—the disc may undergo degenerative alterations or structural failure. Annular fissuring can reduce the disc's capacity to resist shear and torsional forces, and it can permit displacement of nuclear material toward the periphery. As the nucleus migrates or protrudes through weakened annular fibers, it may encroach upon adjacent neural tissues. Depending on the direction and magnitude of the disc displacement, this process can contribute to mechanical compression and chemical irritation of a nerve root, producing pain, sensory disturbance, and potential weakness in a radicular distribution. In some cases, symptoms are driven not only by direct compression but also by inflammatory mediators associated with disc disruption, which may amplify neural sensitivity and pain experience. Epidemiologically and clinically, lumbosacral disc pathology is concentrated at the lower lumbar levels. Slightly more than 90% of disc herniations occur at the L4–L5 or L5–S1 interspaces, reflecting the high loads and frequent end-range demands placed upon these segments during lifting, gait, and transitional movements. This distribution is especially relevant for rehabilitation planning because deficits in trunk control, hip mobility, and load-management strategies often influence symptom provocation at these levels. While many disc-related presentations improve with conservative care, progression to significant neurologic compromise or substantial limitation in activities of daily living may necessitate surgical intervention aimed at decompression and stabilization. In the absence of motor deficits, however, a non-operative course is typically recommended for several months, incorporating analgesia, graded activity modification, and—when appropriate—image-guided injections to facilitate pain control and functional restoration. [4]

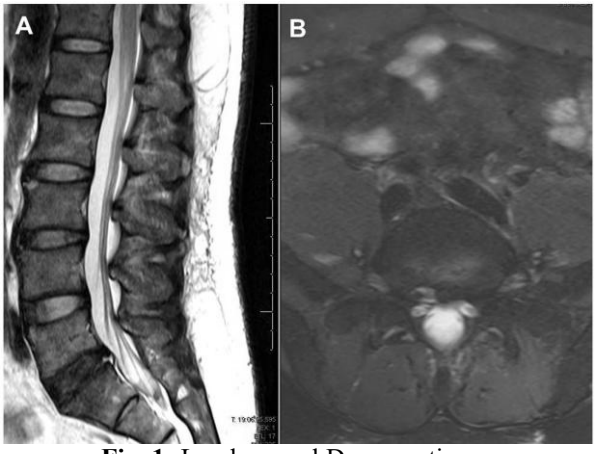

Fig. 1: Lumbosacral Degeneration.

Etiology

The etiology of lumbosacral disc injuries is multifactorial and extends beyond the traditional biomechanical narrative that has historically emphasized repetitive forward flexion and heavy manual loading as primary causal mechanisms. Although flexion-based postures and lifting tasks can acutely provoke symptoms and may contribute to transient increases in intradiscal pressure, contemporary evidence does not support a clear dose–response relationship between cumulative physical loading and the occurrence of disc injury. In other words, while mechanical exposures may influence symptom expression and may interact with individual vulnerability, the available literature has not demonstrated that progressively greater quantities of physical loading reliably translate into proportionally higher rates of disc damage. This distinction is particularly important for physical therapists, as it cautions against overly deterministic explanations that attribute disc pathology solely to "poor posture" or repeated bending, and it encourages a more nuanced, biopsychosocial interpretation of risk. Moreover, earlier occupational studies that linked intervertebral disc disease to heavy physical work are increasingly recognized as potentially confounded. Specifically, associations between high-load occupations and disc degeneration may reflect correlated factors such as socioeconomic status, healthcare access, comorbid health behaviors, and lifestyle variables that cluster within certain occupational groups. These confounders can create the appearance of a direct mechanical causation when, in reality, disc-related outcomes may be influenced by broader contextual determinants. Thus, the relationship between occupation and disc injury is likely more complex than a simple mechanical exposure model, and the interpretation of occupational risk requires careful attention to social and behavioral covariates rather than a narrow focus on spinal loading alone. [5]

A parallel and increasingly influential line of evidence highlights a substantial genetic contribution to disc degeneration and susceptibility to disc injury.

Twin studies, particularly those involving monozygotic twins who share nearly identical genetic material, have been instrumental in isolating genetic effects from environmental exposures. Such research suggests that inherited factors may significantly shape disc composition, collagen integrity, inflammatory responses, and the capacity for tissue repair—all of which can affect how discs respond to mechanical stress across the lifespan. Supporting this view, imaging-based investigations of monozygotic twins have demonstrated that a meaningful proportion of variability in disc degeneration is attributable to genetic influences. For instance, in a study analyzing magnetic resonance imaging in 115 monozygotic twin pairs, genetics accounted for approximately 34% of the variability in disc degeneration at the L4–S1 region, whereas physical loading explained roughly 2% and age approximately 7%. These findings reinforce the concept that biological predisposition can play a dominant role in disc health, while mechanical and chronological factors may contribute more modestly or operate primarily as modifiers of an underlying genetic risk profile. For clinical practice in physical therapy, these etiologic insights support a balanced approach to patient education and management. Emphasizing genetic and multifactorial influences can reduce fear-based beliefs about movement, improve adherence to graded activity, and shift rehabilitation toward modifiable contributors such as conditioning, movement confidence, and load tolerance, rather than assigning disproportionate blame to specific postures or isolated activities [4][5].

Epidemiology

The epidemiology of lumbosacral disc injuries is complicated by two interrelated realities: the definition of what constitutes a "disc injury" is not uniform across clinical and research settings, and the widespread availability of magnetic resonance imaging (MRI) has substantially expanded the detection of disc-related structural changes, many of which are incidental. As MRI became more accessible and routinely employed for back pain and radicular symptoms, clinicians and investigators began identifying disc signal changes, bulges, and other morphologic variations at a frequency that exceeded the prevalence of clinically meaningful symptoms. This shift has altered both the perceived incidence of disc pathology and the interpretation of imaging findings, with important implications for physical therapy practice, patient education, and healthcare utilization. A major epidemiologic challenge is that most intervertebral disc degenerations are asymptomatic. Structural changes within the disc—such as reduced hydration, annular fissuring, or contour irregularities—can be present in individuals who report no pain, no functional limitation, and no neurologic symptoms. Consequently, establishing the "true" prevalence of disc injury is inherently difficult: prevalence estimates rise dramatically when the case definition is based on imaging morphology alone, yet they may be far lower when restricted to symptomatic presentations with concordant clinical findings. This distinction is clinically consequential because it underscores the risk of over-attribution, wherein an incidental MRI finding is presumed to explain symptoms without adequate correlation to the patient's history, physical examination, and functional impairments. In addition, the lack of uniform definitions for disc degeneration and disc herniation undermines cross-study comparability. Different investigations use varying criteria to classify disc bulges, protrusions, extrusions, and degenerative changes, and they may employ different grading systems for disc height loss, signal intensity reduction, or annular disruption. As a result, synthesizing prevalence estimates across multiple studies is challenging, and reported rates can appear highly variable even when populations are similar. These methodological inconsistencies contribute to broad epidemiologic ranges and can complicate guideline development, especially when clinicians attempt to interpret imaging prevalence data for patient counseling [4][5].

This variability is evident in a meta-analysis of 20 studies that evaluated MRI findings in asymptomatic individuals. In that analysis, disc abnormalities at any spinal level were common, with reported prevalence ranging from 20% to 83% for reduced signal intensity, 10% to 81% for disc bulges, 3% to 63% for disc protrusion (compared with 0% to 24% for disc extrusion), 3% to 56% for disc narrowing, and 6% to 56% for annular tears. These wide ranges likely reflect differences in study populations, imaging protocols, and diagnostic thresholds, but the overarching message is consistent: incidental disc "abnormalities" are frequently observed even in people without symptoms. [6] For physical therapists, the epidemiologic implication is that imaging findings should be interpreted cautiously and contextualized within the patient's clinical presentation. The presence of disc degeneration or herniation on MRI is not synonymous with pathology requiring specialist referral. Rather, this evidence supports the position that incidental disc disease, in the absence of pain, neurologic deficit, or meaningful functional limitation, should not automatically prompt escalation of care. Instead, emphasis should be placed on clinical correlation, patient reassurance, and rehabilitation strategies directed toward function and symptom behavior rather than imaging labels alone [5][6].

Pathophysiology

The characteristic radiation of low back pain associated with lumbosacral disc pathology is most commonly explained by irritation or compression of

neural elements within the spinal canal and lateral recesses. When disc material bulges or herniates posteriorly or posterolaterally, it may encroach upon adjacent nerve roots, producing radicular pain that follows a dermatomal distribution and may be accompanied by paresthesia, weakness, or reflex changes. Importantly, neural compromise in disc disease is not solely attributable to the disc itself. Degenerative remodeling of surrounding spinal structures can contribute to a narrowed canal or foraminal space and thereby amplify the mechanical and inflammatory burden on the nerve root. Hypertrophy or infolding of the ligamentum flavum, osteoarthritic changes in the facet joints, and segmental instability with secondary tissue thickening can all reduce the available space for neural tissues, creating a multifactorial substrate for nerve root irritation. In clinical practice, this helps explain why symptom severity may not correlate neatly with a single imaging feature and why some individuals experience prominent radicular symptoms even when disc protrusion appears modest, while others remain relatively asymptomatic despite more conspicuous anatomic changes. Beyond purely mechanical compression, contemporary models also emphasize biochemical and neurophysiologic mechanisms. Disc disruption can expose neural tissues to inflammatory mediators that sensitize nociceptors and heighten mechanosensitivity, contributing to pain amplification with otherwise tolerable movement or loading. This sensitization may manifest as pain provoked by low-intensity activities, altered tolerance to sustained postures, or disproportionate symptom responses to minor perturbations. In this framework, the clinical syndrome of “sciatica” reflects an interplay between structural proximity, local inflammation, and the nervous system’s dynamic sensitivity, rather than a simple one-to-one relationship between a disc contour abnormality and pain intensity [6].

Epidemiologic observations regarding symptom onset also challenge common assumptions about the pathophysiology of acute radicular presentations. In a 2010 study by Suri and colleagues involving 154 consecutive patients presenting with new lumbar disc herniation, the majority reported that symptoms began spontaneously rather than following a distinct mechanical event. Specifically, 62% described spontaneous onset, while 26% linked symptom onset to a specific household task or a seemingly routine, non-lifting activity. Notably, fewer than 8% reported that their acute sciatica followed heavy lifting or physical trauma. These findings are clinically instructive because they suggest that acute radicular pain frequently emerges without an identifiable “injury moment,” supporting the notion that underlying disc and degenerative changes may reach a threshold where even ordinary movements—or no clearly recalled trigger—can precipitate symptoms. For physical therapists, this reinforces the value of patient education that de-emphasizes fear-based narratives around isolated movements and instead focuses on graded exposure, load management, and restoration of function within an evidence-informed biopsychosocial approach [6].

Histopathology

At the tissue level, the pain and neurologic sequelae associated with lumbosacral disc pathology are closely linked to microvascular compromise and inflammation within the perineural environment. The spinal cord and nerve roots are enveloped by a dense network of small-caliber blood vessels that are essential for maintaining neural homeostasis. These microvessels deliver oxygen and metabolic substrates, remove waste products, and support local signaling through the transport of chemomodulators that influence immune activity and nociceptive processing. Because nerve roots have high metabolic demands and limited tolerance for reduced perfusion, even partial compromise of this microcirculation can produce clinically meaningful dysfunction. When disc bulge, herniated disc material, or hypertrophic degenerative tissues encroach upon the spinal canal or intervertebral foramen, they may compress not only the nerve root itself but also its accompanying microvasculature. The immediate consequence of this compression is impaired venous outflow and reduced capillary perfusion, which can precipitate a localized ischemic effect. Ischemia alters axonal transport, disrupts ionic gradients, and increases susceptibility to ectopic neural firing, thereby contributing to pain and neurologic symptoms. Clinically, this process can manifest as radiating pain along the distribution of the affected nerve root, paresthesia, and, in more advanced cases, weakness or reflex alterations. Importantly, this pattern of pain is not simply the direct result of mechanical pressure; rather, it reflects the downstream biologic consequences of reduced perfusion and metabolic stress imposed on neural tissues [6][7].

Compression-induced ischemia is rapidly accompanied by an inflammatory cascade within the epidural and perineural compartments. Mechanical deformation of nerve root tissues and ischemic stress can trigger endothelial activation and increased vascular permeability, setting the stage for infiltration of immune cells and amplification of nociceptive signaling. A key feature of this response is an upregulation of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-alpha), which has been implicated in sensitizing nociceptors and promoting neurogenic inflammation. Elevated cytokine activity can increase the excitability of dorsal root ganglion neurons and contribute to the clinical phenomenon of heightened mechanosensitivity, where relatively minor movements or low-load activities provoke disproportionate symptoms. In parallel, macrophage recruitment is a hallmark of the inflammatory response. Macrophages migrate to the site of tissue

stress, where they participate in phagocytosis, cytokine production, and modulation of local repair processes. While macrophage activity can be beneficial in clearing damaged tissue and contributing to remodeling, it can also sustain inflammatory signaling and perpetuate pain, particularly when the mechanical driver of compression persists. From a rehabilitation perspective, this histopathologic model supports an understanding of radicular symptoms as the product of both ischemic and inflammatory mechanisms. It helps explain why symptom behavior may fluctuate with posture and loading, why pain can persist even when gross mechanical compression appears limited, and why interventions that reduce mechanical irritation while promoting gradual neural and functional tolerance can be clinically effective [7].

History and Physical

A high-quality history is foundational to the evaluation of suspected lumbosacral disc injury and should be structured to clarify symptom onset, symptom distribution, and functional impact while simultaneously screening for features that suggest neurologic compromise or non-mechanical pathology. The clinician should first determine the temporal profile of symptoms, including whether onset was sudden or gradual, whether pain has progressed or fluctuated, and whether there was any identifiable inciting event such as a twist, fall, or lifting episode. Although many patients report an activity-related onset, disc-related symptoms may also begin without a clear mechanical trigger, and the absence of a discrete injury should not reduce clinical suspicion when radicular features are present. Particular attention should be paid to the presence or absence of radicular symptoms, including leg-dominant pain, paresthesia, numbness, or weakness, as this constellation often indicates nerve root involvement and carries different prognostic and management implications than isolated axial low back pain. A thorough subjective assessment should explore postural and movement-specific influences on symptoms. Patients should be asked whether flexion, extension, prolonged sitting, lying supine, or transitions such as sit-to-stand meaningfully alter pain intensity or distribution. These symptom-modifying patterns can provide clinically useful information about mechanical sensitivity and directional preference, inform activity modification, and guide initial rehabilitation decisions. Functional tolerance should be quantified with practical anchors, such as the distance or duration the patient can ambulate before symptoms emerge, whether symptoms force them to stop or change posture, and whether they can negotiate stairs. A history of prior episodes, previous injuries, or surgeries is also essential, as recurrence patterns, prior imaging findings, and surgical history can shape differential diagnosis and influence expectations. In addition, clinicians should explicitly inquire about weakness and sensory symptoms, including numbness and tingling, as these may indicate nerve root compromise requiring urgent medical assessment if progressive [7].

Systemic screening is equally important, particularly in the presence of atypical features. The clinician should investigate systemic symptoms, recent illnesses, unintentional weight loss, or recent travel that could raise suspicion for infectious, inflammatory, or neoplastic processes. These “red flag” considerations do not diagnose a specific condition in isolation, but they contextualize risk and help determine when referral or urgent imaging may be warranted. From a prognostic standpoint, presentations dominated by radiating leg pain are generally more anatomically localizable and may have more predictable outcomes with surgical decompression than nonspecific low back pain syndromes, which are often multifactorial and may reflect muscle fatigue, strain, or broader sensitization processes. Similarly, a strongly mechanical pattern—where pain is provoked reliably by specific movements or positions—may suggest segmental instability or a degenerative defect such as a pars interarticularis lesion at L5, particularly when symptoms worsen with extension-based loading. The physical examination should be organized and systematic, with the explicit goal of determining functional limitation, identifying neurologic deficits, and distinguishing radicular syndromes from other sources of pain. Observing gait is critical because it provides a direct window into daily functional impact. A practical sequence includes asking the patient to rise from a chair, ambulate normally, and then perform heel walking and toe walking. Heel walking challenges ankle dorsiflexion and can reveal L4–L5 or L5 involvement, whereas toe walking challenges plantarflexion and can unmask S1 weakness. After this functional screen, the patient can be positioned on the examination table for targeted testing of strength, sensation, reflexes, and neural tension [5][6][7].

A complete examination should include a neurologic screen of the extremities and, when indicated by history, an assessment of bladder and bowel function to identify urgent conditions such as cauda equina syndrome. The key elements are not limited to strength testing; sensation and reflex integrity are equally important, and subtle asymmetries may carry diagnostic significance. The clinician should also inspect the skin over the spine for bruising, rash, or prior surgical scars and palpate for focal tenderness, including tenderness to compression, which may suggest bony involvement or localized inflammatory pain. Neural tension testing is commonly incorporated, particularly the straight leg raise. In this test, the patient lies supine while the clinician passively elevates the fully

extended leg from approximately 0 to 80 degrees. Reproduction of radiating leg pain, particularly when it follows a recognizable dermatomal pattern, supports nerve root irritation and is consistent with disc herniation or canal compromise. While the description is sometimes framed in relation to stenotic canal symptoms, the clinical interpretation should be integrated with the overall examination and symptom behavior rather than treated as a standalone diagnostic endpoint. At the L5–S1 level, disc herniation may produce overlapping patterns depending on the direction of displacement. A far-lateral herniation encroaching into the neural foramen can compress the L5 nerve root, potentially producing weakness in hip abduction musculature, ankle dorsiflexion (anterior tibialis), and extension of the great toe (extensor hallucis longus). By contrast, a central herniation into the canal may compress the S1 nerve root, leading to weakness in ankle plantarflexion, typically mediated by the gastrocnemius-soleus complex. Recognizing these patterns helps physical therapists map impairment findings to plausible neural involvement, guide targeted functional testing and determine when symptoms warrant expedited medical evaluation [7].

Evaluation

Evaluation of suspected lumbosacral disc injury in physical therapy practice should integrate clinical reasoning, targeted neurologic testing, and judicious use of imaging in a manner that aligns structural findings with symptom behavior and functional limitation. Because low back and leg pain syndromes are heterogeneous, the objective is not merely to "confirm" a disc lesion, but to determine whether symptoms are consistent with nerve root compromise, to identify signs that warrant urgent medical referral, and to guide an evidence-informed plan of conservative management when appropriate. Specific clinical tests can enhance diagnostic accuracy when interpreted within a coherent examination framework. Neural tension testing, particularly the straight leg raise (SLR), remains a cornerstone for identifying lumbosacral radicular involvement. Evidence suggests that combining neurologic signs can improve sensitivity for lower lumbar nerve root impingement. In a 2011 study by Suri and colleagues, the combination of positive findings on the SLR and the Achilles reflex test demonstrated a sensitivity of 79% for low lumbar nerve root impingement, indicating that clustered findings may better capture clinically relevant nerve root compromise than any single test alone. They also reported that a positive ipsilateral SLR can be supplemented by a contralateral SLR, which increases specificity for lower lumbar disc herniation, with reported specificity improving from 84% to 96% when contralateral symptoms are provoked. These observations support the clinical value of test clusters and side-to-side comparisons, particularly when the history indicates leg-dominant pain and neurologic symptoms. [7][8][9]

Imaging should be approached with similar nuance. In many care pathways, initial evaluation of low back pain—especially when symptoms are persistent or when bony pathology is a concern—may include anterior–posterior (AP) and lateral radiographs of the involved spinal region. Radiographs can assist in identifying gross structural abnormalities such as fractures, significant degenerative changes, alignment abnormalities, or spondylolisthesis. When segmental instability is suspected based on mechanical symptom behavior, history of recurrent episodes, or examination findings suggesting aberrant motion, flexion–extension radiographs may be added to assess for dynamic translation. From a physical therapy perspective, the value of these images lies primarily in ruling in or out red-flag structural concerns rather than confirming disc injury, as discs themselves are not well visualized on plain radiographs. In the presence of red flags, advanced imaging becomes more urgent and clinically justified, both to clarify diagnosis and to support possible surgical planning. Magnetic resonance imaging is typically the modality of choice when serious pathology is suspected or when progressive neurologic compromise is present. Examples of red flags include symptoms consistent with cauda equina syndrome—such as difficulty controlling bowel or bladder function or difficulty initiating urination—where immediate escalation is required due to the risk of permanent neurologic injury. Suspicion of infection should be heightened in individuals with intravenous drug use, a history of fever, or nocturnal chills, as spinal infections can present with back pain and systemic features. Malignancy should be considered when there is a known history of cancer or unexplained new-onset weight loss. Significant trauma, including falls, assaults, or collisions, increases concern for fracture or destabilizing injury and warrants imaging aligned with injury mechanism and clinical findings [8][9].

Equally important is recognizing when imaging is unnecessary. Most patients presenting with symptoms consistent with acute paraspinal or low lumbar muscle strain do not require radiographic evaluation. Even in suspected acute disc herniation, MRI is not recommended at initial presentation in the absence of red flags, because many patients improve with conservative care, and early MRI can lead to incidental findings that do not correlate with symptoms and may increase fear, unnecessary referrals, and healthcare utilization. Instead, a structured conservative trial—often framed as approximately six weeks of physical therapy—may be appropriate, emphasizing symptom-guided activity modification, progressive loading, and functional restoration. If symptoms persist despite appropriate rehabilitation, if neurologic deficits develop or progress, or if clinical course deviates from expected

recovery, MRI can then be obtained. When MRI is pursued for suspected disc-related neural compromise, attention is typically directed to T2-weighted sagittal and axial sequences, which best illustrate hydration changes, disc contour, and potential compression of neurologic elements, thereby informing decisions about ongoing conservative care versus referral for interventional or surgical consultation. Finally, imaging findings must be interpreted cautiously in relation to prognosis. Over time, both symptomatic and asymptomatic disc herniations often decrease in size on MRI, reflecting the dynamic nature of disc material resorption and remodeling. Moreover, the presence of disc degeneration or herniation on MRI does not reliably predict chronic pain or future need for surgery. For physical therapists, this reinforces a central principle of evidence-informed practice: imaging should support clinical decision-making when it changes management, but it should not replace functional assessment, symptom behavior analysis, and patient-centered rehabilitation planning [8][9].

Treatment / Management

Management of lumbosacral disc injuries within physical therapy practice is anchored in the strong natural history of improvement and the central role of conservative care. Clinically, the majority of patients—often cited as more than 90% with L5–S1 disc-related presentations—will improve without surgical intervention, particularly when treatment is structured to restore function, modulate symptoms, and progressively rebuild load tolerance. This favorable prognosis supports an initial nonoperative approach in most cases, provided that red flags and progressive neurologic deficits have been excluded. For physical therapists, the early phase of care emphasizes symptom-informed activity modification, patient education, and a rehabilitation program that balances protection of irritable tissues with avoidance of unnecessary deconditioning. A commonly implemented conservative pathway includes an approximately six-week course of physical therapy with emphasis on trunk and lumbopelvic control, graded strengthening, and mobility work. “Core strengthening” in this context is best conceptualized not as isolated abdominal bracing alone, but as improving coordination and endurance of the trunk, hip, and pelvic musculature to enhance spinal stability during functional tasks. Exercise selection is individualized based on symptom behavior and movement tolerance and typically progresses from low-load isometrics and motor-control strategies to more demanding functional strengthening and conditioning. Stretching and mobility interventions may be incorporated to address contributing restrictions in the hips, thoracic spine, or neural tissues, though these should be prescribed in a manner that respects symptom irritability and avoids aggressive end-range loading when radicular symptoms are easily provoked. Education is equally central: patients benefit from clear messaging that disc-related symptoms are common, often improve, and can be managed through graded return to activity rather than fear-based avoidance [8][9][10].

Adjunctive non-surgical measures frequently complement rehabilitation, particularly during periods of high pain intensity. Activity modification may include temporary reduction of movements or positions that exacerbate symptoms—such as prolonged flexion, sustained sitting, or repeated heavy lifting—paired with strategies to maintain general activity and preserve aerobic conditioning. Pharmacologic management, commonly with non-steroidal anti-inflammatory drugs (NSAIDs), may reduce pain and inflammation sufficiently to improve participation in therapy and daily activities. In selected cases, epidural steroid injections are used as an additional symptom-modulating intervention. Evidence suggests that epidural injections can provide moderate, short-term relief for radicular pain associated with disc herniation, potentially facilitating engagement in rehabilitation and accelerating functional gains. However, the literature is less definitive regarding the effectiveness of injections for chronic, non-radiating low back pain, where drivers may include multifactorial degenerative change, sensitization, and psychosocial contributors rather than focal nerve root inflammation. When imaging indicates that facet-mediated inflammation may be a prominent contributor—such as MRI T2 signal changes consistent with localized inflammatory activity—interventions may also be directed at the L5–S1 facet region, illustrating how imaging can occasionally refine interventional targeting in complex presentations. [10][11][3]

When an adequate conservative course fails to produce acceptable improvement, patients commonly confront three broad trajectories: persistence of pain and limitation, substantial avoidance of symptom-provoking activities with attendant participation restriction, or consideration of surgical intervention. Shared decision-making becomes essential at this stage, because the “failure” of conservative care is not defined solely by imaging findings but by persistent functional impairment, unacceptable symptom burden, or progressive neurologic compromise. One of the most frequently cited sources comparing operative and nonoperative care for lumbar disc herniation is the Spine Patient Outcomes Research Trial (SPORT). In those reports, patients who elected surgery demonstrated better outcomes at three months, two years, and four years compared with patients managed without surgery, particularly with respect to relief of radicular symptoms and functional measures. These findings are clinically meaningful for therapists because they underscore that while many patients improve without

surgery, a subset may experience faster or more substantial relief with operative management, especially when leg-dominant symptoms are prominent. Surgical options and techniques continue to evolve, yet the broader literature suggests that traditional open discectomy and microdiscectomy are both effective and broadly similar in outcomes for appropriately selected patients. Surgical nuance also includes the extent of disc removal. A limited discectomy may yield greater pain relief and satisfaction than subtotal discectomy, but it may carry a higher risk of recurrent herniation, illustrating the trade-off between tissue preservation and recurrence risk. Notably, patient outcomes and satisfaction following repeat or revision microdiscectomy have been reported as similar to those after initial discectomy, a point that can be reassuring when counseling patients who fear that recurrence necessarily implies poor long-term outcome. Hospitalization requirements vary; while some patients require overnight monitoring, many contemporary procedures are designed as outpatient interventions. Across both conservative and surgical pathways, a critical counseling point is that outcomes are generally more predictable for radicular pain than for isolated axial low back pain. Surgical decompression tends to address nerve root compression and the inflammatory milieu that drives leg symptoms, whereas nonspecific back pain often reflects multifactorial contributors not fully resolved by structural intervention alone. For physical therapists, this reinforces the importance of functional rehabilitation and education regardless of whether surgery occurs, with care plans tailored to restore movement confidence, graded capacity, and long-term self-management [10][11][3].

Differential Diagnosis

The differential diagnosis for lumbosacral disc–related symptoms must remain broad, particularly when presentation includes severe pain, atypical symptom patterns, systemic features, or neurologic compromise. Although disc injury and degenerative change can produce low back pain with or without radicular symptoms, several urgent or non-spinal conditions can mimic these presentations and require immediate medical evaluation. In older adults presenting with abdominal discomfort and back pain, clinicians should consider serious visceral and vascular etiologies rather than assuming a musculoskeletal origin. Acute aortic dissection, for example, may present with back or flank pain and can be catastrophic if missed; sudden onset, severe intensity, hemodynamic instability, or associated chest symptoms should prompt emergent referral. Nephrolithiasis is another common mimic that can produce acute flank or back pain with radiation and may be associated with urinary symptoms, nausea, or restlessness, distinguishing it from posture-dependent mechanical patterns. Neurologic emergencies must also be considered. Cauda equina syndrome represents a high-stakes diagnosis characterized by bowel or bladder dysfunction, saddle anesthesia, and progressive neurologic deficits; any concern for these features warrants urgent escalation. Epidural infections, including spinal epidural abscess, may present with back pain and evolving neurologic findings and are more likely in individuals with fever, immunosuppression, intravenous drug use, or recent invasive procedures. Similarly, osteomyelitis of the spine can generate persistent back pain, often with systemic symptoms or elevated inflammatory markers, and may not respond to typical conservative measures. Herpes zoster can produce severe neuropathic pain preceding the characteristic rash, and early recognition is important because the pain may be mistaken for radiculopathy or mechanical back pain prior to cutaneous findings. Constipation, while generally benign, can produce referred discomfort to the lower back, especially in older adults or those with reduced mobility, and should be considered when pain coincides with bowel habit changes. Hematologic conditions such as sickle cell anemia may also present with pain crises involving the spine or pelvis, requiring a different medical pathway than musculoskeletal rehabilitation. Finally, mechanical back pain from muscular strain, facet irritation, or ligamentous overload remains common and may coexist with imaging-detected disc abnormalities, reinforcing that the presence of disc findings does not automatically establish causation. A careful synthesis of symptom behavior, neurologic status, systemic screening, and red-flag assessment is therefore essential to avoid misclassification and delayed referral [11][12].

Other Issues

A central clinical pearl in lumbosacral care is that the most common cause of low back pain is not disc injury, but rather muscular strain, deconditioning, and fatigue-related overload of spinal and hip musculature. This observation is particularly relevant in physical therapy because it supports an impairment-based approach that prioritizes functional restoration and modifiable contributors over imaging labels. Many patients with low back pain demonstrate movement intolerance, reduced trunk endurance, and suboptimal load distribution during daily activities, which can generate pain even in the absence of discrete structural pathology. Importantly, modifiable factors that contribute to chronic low back pain include sustained poor ergonomics, obesity, physical inactivity, and reduced strength and endurance of key stabilizing muscle groups. The term “core” should be interpreted broadly to include the abdominal musculature, thoracic and lumbar spinal erectors, multifidus, and the oblique muscle system, all of which contribute to trunk stiffness modulation, segmental control, and efficient force transfer between the trunk and lower limbs. Muscular and ligamentous support around the vertebral column can meaningfully offset axial loads and may reduce stress

concentration at the disc level during functional tasks. From a rehabilitation standpoint, this supports programming that combines education on posture and task modification with progressive strengthening and conditioning to improve tolerance to bending, lifting, and prolonged postures. It also reinforces a graded exposure model: rather than prescribing blanket avoidance of flexion or lifting, therapists can progressively reintroduce loads while teaching efficient hip-dominant strategies, breath control, and pacing. Another key issue is the frequent mismatch between imaging and symptoms; disc degeneration and herniation can be present in asymptomatic individuals, and symptom severity does not consistently correlate with the size of a disc bulge. As a result, effective care often hinges on identifying the patient's symptom triggers, improving movement confidence, and restoring participation, rather than attempting to "correct" a structural finding. This reframing can reduce fear, improve adherence, and support long-term self-management [12].

Enhancing Healthcare Team Outcomes

Most patients with suspected lumbosacral disc injury initially present not to physical therapy directly, but to an emergency department, primary care clinician, or nurse practitioner, making interprofessional coordination fundamental to safe and efficient care. While the majority can be managed conservatively, timely referral to an orthopedic surgeon or spine specialist becomes important when neurologic deficits are present, progressive, or functionally significant. From a systems perspective, clear triage pathways help ensure that patients with red flags—such as cauda equina symptoms, suspected infection, malignancy, or significant trauma—are rapidly escalated for appropriate imaging and specialist management, while lower-risk patients receive early education and rehabilitation to reduce disability and prevent chronicity. Primary care teams play a pivotal role in prevention counseling and longitudinal risk modification. Patient education should emphasize strategies that reduce recurrence and chronic pain risk, including weight management, regular physical activity, and avoidance of tobacco use, which has been associated with poorer musculoskeletal health and impaired tissue healing. However, adherence remains a persistent challenge, and relapse of low back pain is common, highlighting the need for consistent messaging across providers and reinforcement of self-management strategies rather than reliance on passive treatments. Physical therapists contribute by translating these preventive goals into actionable programs, monitoring progress, and addressing barriers such as fear-avoidance, low self-efficacy, and unrealistic expectations about imaging and "fixes." When surgery is considered, shared decision-making across the healthcare team is essential. Surgical intervention benefits a select subset of patients—particularly those with clear radicular syndromes and concordant neurologic findings—but it also carries risks and can be associated with serious complications that may result in long-term disability. Therefore, coordinated communication between primary care, rehabilitation, and surgical teams helps ensure appropriate patient selection, realistic expectation setting, and continuity of care before and after any procedure. Comprehensive rehabilitation, whether surgical or non-surgical, is most effective when teams align around consistent goals: protecting neurologic function, restoring mobility and strength, optimizing participation, and equipping the patient with durable self-management skills. [12][13][14]

Conclusion:

Lumbosacral disc injuries represent a multifactorial condition where structural, genetic, and inflammatory mechanisms converge to produce pain and functional limitation. Despite the frequent association of these injuries with mechanical stress, contemporary evidence highlights the dominant role of biological predisposition and the limited predictive value of imaging findings. For physical therapists, this paradigm shift underscores the importance of a biopsychosocial approach that prioritizes functional restoration over structural correction. Conservative management remains the cornerstone of care, with more than 90% of patients achieving meaningful improvement through targeted rehabilitation. Programs should integrate trunk and lumbopelvic control, progressive strengthening, and patient education to reduce fear-avoidance and enhance self-efficacy. Adjunctive measures such as NSAIDs or epidural injections may facilitate participation but should not replace active strategies. Surgical intervention, while effective for select cases with severe radicular symptoms, offers no guarantee of superior long-term outcomes and should be reserved for patients with persistent impairment or progressive neurologic compromise. Ultimately, successful management depends on individualized care, clear communication, and interprofessional collaboration. By focusing on modifiable factors and empowering patients through graded exposure and movement confidence, physical therapy can optimize recovery, minimize disability, and reduce the burden of chronic low back pain.

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