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# Central, Lateral Recess, or Foraminal? A Narrative Review of MRI Stenosis Compartments and Lower-Limb Radiculopathy Patterns

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## ABSTRACT

**Background**: Degenerative lumbar canal stenosis (LCS) is a leading cause of low-back pain, neurogenic claudication, and lower-limb radiculopathy in older adults. Although magnetic resonance imaging (MRI) is the preferred modality for anatomical assessment, clinical interpretation often remains challenging because stenosis can occur in distinct compartments—central canal, lateral recess (subarticular zone), and neural foramen—each with different neuroanatomical targets.
**Objective**: This narrative review summarizes compartment-based lumbar stenosis anatomy, degenerative mechanisms, MRI evaluation and commonly used grading systems, and the typical symptom patterns associated with central, lateral recess, and foraminal stenosis, with emphasis on how compartment dominance may shape radiculopathy distribution.
**Methods**: A narrative synthesis of key concepts from anatomical, radiological, and clinical literature on degenerative LCS was performed, focusing on mechanisms of narrowing (disc height loss, facet hypertrophy/osteophytes, ligamentum flavum thickening/buckling), MRI-based localization, and compartment-specific clinical manifestations.
**Results**: Central canal stenosis commonly involves multiroot compression and is frequently associated with bilateral symptoms and neurogenic claudication. Lateral recess stenosis typically affects traversing nerve roots and more often produces unilateral dermatomal pain, while foraminal stenosis compromises exiting roots and may present with distal-predominant radiculopathy influenced by posture. MRI grading systems provide standardized morphological descriptors; however, static MRI may not fully capture dynamic compression and vascular/venous contributors to symptoms.
**Conclusion**: A compartment-based approach improves clinical reasoning in degenerative LCS and supports symptom-relevant MRI interpretation. Direct evidence linking dominant stenosis compartment to specific lower-limb radiculopathy patterns remains limited, highlighting a need for targeted clinicoradiological studies.

***Keywords:*** Degenerative lumbar canal stenosis; Magnetic resonance imaging;

Central canal stenosis; Lateral recess stenosis; Foraminal stenosis

## INTRODUCTION

Degenerative lumbar canal stenosis (LCS) is a prevalent spine disorder and a major contributor to low-back pain, lower-limb symptoms, and functional limitation in older adults. The condition is increasingly encountered with population aging and is frequently implicated in activity restriction, reduced walking tolerance, and diminished quality of life, resulting in a substantial clinical burden.[1]

Degenerative LCS represents the cumulative effect of progressive structural changes involving the intervertebral disc, facet joints, and ligamentous complex. Disc height loss redistributes axial load toward the posterior elements, promoting facet hypertrophy and osteophyte formation, while reduced disc tension contributes to ligamentum flavum buckling and thickening. [2] These processes converge to reduce the space available for neural structures within anatomically distinct compartments, namely the central canal, the lateral recess (subarticular zone), and the neural foramen, each with different boundaries and neuroanatomical targets.[2,3]

Clinically, LCS is best understood as a syndrome rather than a single lesion, requiring integration of typical symptoms, physical/neurological examination, and imaging findings for diagnosis and treatment planning. [4] Symptom patterns range from low-back pain and radicular pain to paresthesia and motor deficit, and may include neurogenic claudication. Importantly, symptom distribution and laterality may vary by the dominant stenosis compartment: central canal compromise often relates to multiroot involvement and neurogenic claudication, while lateral recess compromise more closely relates to traversing root compression, and foraminal compromise to exiting root pathology.[1,3]

Magnetic resonance imaging (MRI) is the preferred non-invasive modality to evaluate stenosis location, level, and side, and to visualize relationships between degenerative structures and neural elements.[1,4] Structured MRI grading systems are commonly used to standardize morphological descriptions; for example, Schizas-based qualitative grading is frequently applied for central canal stenosis on axial T2-weighted images.[5] Additionally, contemporary radiology reviews have summarized the Lee grading framework for neural foraminal stenosis and related compartment-based MRI assessment.[6] Nevertheless, MRI interpretation has recognized limitations, including potential underestimation of stenosis severity compared with computed tomography (CT) in certain contexts, which may influence surgical planning in severe cases.[7]

Consistent with these complexities, clinicoradiological correlation is often imperfect. Morphological stenosis may be present even among individuals without concordant symptoms, and MRI severity does not always translate into symptom burden.[5,8] Furthermore, in surgically confirmed lateral lumbar spinal canal stenosis, MRI-based lateral stenosis severity may show limited association with symptoms and walking capacity, underscoring the need for cautious symptom-relevant interpretation.[9]

In Indonesia, comprehensive epidemiological data remain limited; however, a hospital-based report from Dr. M. Djamil Central Public Hospital documented 697 spinal stenosis cases between 2018 and 2022, including 418 lumbar spinal stenosis cases, with the most frequent level reported at L4–L5.[10] These findings align with typical degenerative loading patterns but also highlight the need for context-specific evidence to support clinical decision-making.

Therefore, a compartment-based framework may strengthen diagnostic reasoning by mapping MRI stenosis dominance (central, lateral recess, or foraminal) to expected lower-limb symptom patterns. However, direct evidence specifically linking dominant stenosis compartment on MRI

with the distribution pattern of lower-limb radiculopathy remains limited, supporting the rationale for targeted clinicoradiological studies in degenerative LCS.

## LITERATURE REVIEW

### ANATOMY OF LUMBAR SPINE

The lumbar spine is located between the lower thoracic region and the sacrum and typically consists of five vertebrae from L1 to L5. It is structurally adapted to bear substantial axial loads while allowing flexion and extension with limited rotation, thereby enabling effective transmission of forces from the trunk to the pelvis and lower extremities.[11]

The anterior components of the lumbar spine are primarily formed by the vertebral bodies and intervertebral discs, supported by the anterior longitudinal ligament and posterior longitudinal ligament. Each intervertebral disc comprises a central nucleus pulposus and a surrounding annulus fibrosus. Together with the vertebral endplates, the disc functions as a load sharing and shock absorbing unit while contributing to segmental mobility and alignment.[11]

Posterior elements include the vertebral arch, which is formed by paired pedicles and laminae, and the transverse processes and spinous process that serve as attachment sites for paraspinal musculature and ligamentous structures. The superior and inferior articular processes form the facet joints, which guide motion and contribute to segmental stability, particularly during flexion and extension. These posterior structures also play a protective role by enclosing the spinal canal and supporting neuromuscular control of lumbar posture.[11,12]

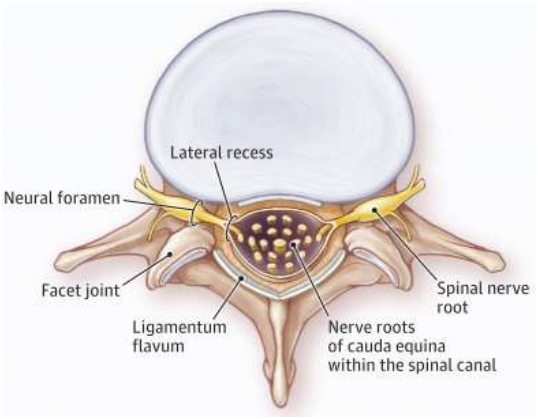


**Figure 1. Normal lumbar vertebral anatomy[1]**

The spinal canal is bounded anteriorly by the vertebral body and intervertebral disc and posteriorly by the laminae and ligamentous tissues. In adults, the spinal cord typically terminates as the conus medullaris around the L1 to L2 level, and the cauda equina continues caudally as a bundle of descending nerve roots within the thecal sac.[13] This arrangement is clinically important because symptoms may arise from compromise of neural structures at different points along the nerve root course.[13]

Lumbar nerve roots travel within the canal and exit through the intervertebral foramina. Two anatomical corridors are particularly relevant for compartment-based interpretation in degenerative disease. The lateral recess is the region where traversing nerve roots course before exiting, and the neural foramen is the passage where exiting nerve roots travel in close relationship with perineural fat and adjacent disc and bony structures. Because these spaces provide limited anatomical reserve, relatively small degenerative changes of the disc, facet complex, or ligamentum flavum can reduce available room for nerve roots and help explain compartment specific patterns of radiculopathy in degenerative lumbar canal stenosis.[11,12]

### DEGENERATIVE LUMBAR CANAL STENOSIS

#### Definition

Degenerative lumbar canal stenosis refers to a reduction in the available space for neural

and vascular structures within the lumbar spinal canal, the lateral recess, and or the intervertebral foramen that occurs secondary to age related degenerative changes of the intervertebral disc, facet joints, and ligamentum flavum.[14,15] This narrowing may result in compression or irritation of the cauda equina and lumbar nerve roots and can produce lower limb symptoms such as neurogenic claudication and radiculopathy.[14,15] Because radiological narrowing can also be present in asymptomatic individuals, degenerative LCS is best considered a clinical syndrome, and diagnosis requires correlation of characteristic symptoms and neurological findings with imaging features rather than reliance on imaging alone.[14,15]

**Epidemiology**

Degenerative lumbar canal stenosis is predominantly a condition of older adults and its reported frequency varies widely because epidemiologic estimates depend on how stenosis is defined (clinical syndrome, imaging based narrowing, or both).[14,18] In population based screening using a validated diagnostic support tool, the estimated prevalence increases with age, rising from about 1.9 percent in adults aged 40 to 49 years to 10.8 percent in those aged 70 to 79 years.[16] Consistent with this age related pattern, lumbar canal stenosis is a major contributor to back and leg symptoms in later life and represents a substantial health care burden, including large volumes of surgical treatment in aging populations.[14] Global estimates suggest a very large worldwide burden of degenerative lumbar spine disease and spinal stenosis, reinforcing its public health relevance.[14,17] Data from Indonesia remain limited, but a single center descriptive study from Dr. M. Djamil Central Public Hospital (2018 to 2022) reported that most patients with lumbar spinal stenosis were aged 60 to 69 years and the most frequent level involved was L4 to L5, supporting the common observation that lower lumbar levels are most often affected.[19]

**Etiopathophysiology**

Degenerative lumbar canal stenosis develops through a progressive degenerative cascade involving the intervertebral disc, facet joints, and ligamentous structures, resulting in reduction of the available space for neural elements within the central canal, lateral recess, and neural foramen.[14,20] Early disc degeneration is characterized by reduced disc hydration and loss of disc height, which alters segmental biomechanics and shifts a greater proportion of axial load to the posterior elements.[14,20] This posterior load transfer accelerates facet joint arthropathy, including cartilage wear, capsular thickening, hypertrophy of the facet complex, and osteophyte formation, all of which contribute to encroachment on adjacent neural corridors.[14,20]

Ligamentum flavum changes are another key driver of stenosis. With disc height loss, the ligamentum flavum may lose its normal tension and progressively fold inward, while chronic mechanical stress can be associated with thickening.[14,20] Together, facet hypertrophy, osteophytes, and ligamentum flavum thickening and folding narrow the central canal and lateral recess.[14,20] Degenerative spondylolisthesis and segmental instability may further reduce available space, and symptoms can be amplified by dynamic narrowing during extension and axial loading, even when supine static imaging appears less severe.[14,20]

From a compartment based perspective, central canal stenosis reflects narrowing that compromises the dural sac and may affect multiple cauda equina roots, whereas lateral recess stenosis preferentially compromises traversing nerve roots as they course toward their exit.[14,20] Foraminal stenosis arises from combined effects of reduced disc height, endplate and osteophyte changes, and facet joint remodeling that reduce foraminal dimensions and increase contact pressure on the exiting nerve root, often with loss of protective perineural fat.[14,20] These compartment specific anatomical mechanisms provide a structural basis for

differences in symptom laterality and distribution.

Symptom generation in degenerative lumbar canal stenosis is not explained by mechanical compression alone. Proposed mechanisms include impaired microvascular perfusion of nerve roots, venous congestion with reduced clearance of metabolic byproducts, and inflammatory and mechanical irritation that varies with posture and motion.[14,15,20] The frequent discordance between imaging severity and clinical symptoms therefore likely reflects a combination of static anatomical narrowing, dynamic biomechanical factors, and neural and vascular susceptibility that differs between individuals.[14,15,20]

## Clinical Manifestation Patterns of Degenerative Lumbar Canal Stenosis

Degenerative lumbar canal stenosis does not have a single diagnostic symptom or sign and the clinical spectrum is broad. Patients may report low back pain, unilateral or bilateral leg pain, paresthesia, numbness, and or weakness, with symptom intensity influenced by the degree and compartment of neural compromise.[14,15] Importantly, anatomical stenosis on imaging can be present in individuals without symptoms, while some patients with clinically significant limitations may show only moderate narrowing on static imaging.[14,15]

A structured clinical assessment should define the dominant symptom location and distribution in the lower limb, including buttock, thigh, calf, and foot involvement, and should document laterality, dermatomal character, sensory disturbance, and motor deficits[14,15] This symptom mapping is essential because compartment dominant stenosis affects different neural targets. Central canal compromise more often involves multiple cauda equina roots, lateral recess compromise affects traversing roots before their exit, and foraminal compromise affects exiting roots within the foramen.[14,20]

Central canal stenosis commonly presents with neurogenic claudication. Patients describe leg discomfort, heaviness, or pain that is provoked by standing and walking and relieved by sitting or lumbar flexion, often accompanied by bilateral symptoms because multiple roots may be affected simultaneously.[14,20] In more advanced cases, patients may report diffuse sensory symptoms and weakness that can involve more than one myotome, reflecting multiroot compromise.[14,20]

Lateral recess stenosis more often produces unilateral radicular pain with a dermatomal distribution corresponding to the traversing nerve root at the affected level.[14,20] Symptoms may include paresthesia or numbness in the same distribution, and objective deficits may follow the expected myotomal pattern, depending on the severity and chronicity of compression.[14,20]

Foraminal stenosis typically compromises the exiting nerve root and may present with radicular pain that is often posture dependent and can be more distal predominant.[14,20] Patients may describe pain provoked by extension or lateral bending toward the affected side, consistent with reduced foraminal reserve, while sensory and motor findings may align with the exiting root level.[14,20]

Overall, clinical patterns in degenerative lumbar canal stenosis are shaped by the compartment involved, the level and side of narrowing, and physiological factors such as dynamic compression and vascular susceptibility.[14,15,20] A compartment-based framework therefore supports more symptom relevant interpretation of imaging and more consistent localization during clinical decision making.[14,15]

## Influence of Age and Sex on the Clinical Presentation of Degenerative Lumbar Canal Stenosis

Age is a key determinant of both the development and the clinical expression of degenerative lumbar canal stenosis. Progressive degeneration of the intervertebral disc, facet joints, and ligamentum flavum accumulates with aging, increasing the likelihood of central canal, lateral recess, and foraminal narrowing.[14,15]

However, age related structural narrowing does not translate linearly into symptom severity, as a substantial proportion of older individuals demonstrate radiographic stenosis without corresponding complaints, highlighting the need for clinicoradiologic correlation when interpreting MRI findings.[22]

Large scale imaging-based analyses also suggest that age influences not only the frequency but also the anatomic distribution of stenosis. In a natural language processing study of 43,255 lumbar MRI reports, the prevalence of clinically relevant spinal canal and neural foraminal stenosis increased with age across levels, with the largest age associated effect at upper lumbar levels from T12 to L3.[21] These findings support the concept that age may modify the dominant level and pattern of stenosis, which can subsequently shape the distribution of lower limb symptoms. At the same time, an MRI study in asymptomatic individuals demonstrated that stenosis risk increases from upper to lower lumbar levels, and that older females showed the highest proportion of borderline and high risk narrowing at L4 to L5 and L5 to S1, despite the absence of clinical signs, again emphasizing that anatomy alone is insufficient to define clinical disease.[22]

Sex related differences further complicate symptom interpretation. Imaging data indicate that moderate or greater central canal and neural foraminal stenosis may be more prevalent in males across lumbar levels.[21] Nevertheless, symptom related disability and pain reporting can be greater in females even after accounting for age and imaging severity. In a study evaluating pain sensitivity among patients with symptomatic degenerative lumbar spinal stenosis, women reported higher pain and disability scores, and the observed sex difference in symptom severity was partly explained by higher pain sensitivity.[23] Similarly, a clinical study using standardized disability and psychological questionnaires found comparable symptom severity between sexes, yet higher disability scores and higher depression measures among female patients, suggesting that pain perception and psychological factors may mediate sex dependent differences in functional impact.[24]

Taken together, age and sex should be considered important covariates when linking dominant stenosis location on MRI to the distribution of lower limb radiculopathy. They influence the epidemiologic burden of stenosis, the likely anatomic distribution of degenerative narrowing, and the way symptoms are perceived and reported.[14,15,21,24]

## Magnetic Resonance Imaging in Degenerative Lumbar Canal Stenosis

Role of magnetic resonance imaging in evaluating stenosis location Magnetic resonance imaging is generally regarded as the preferred first line modality for evaluating degenerative lumbar canal stenosis because it provides multiplanar visualization of the central canal, lateral recess, and neural foramina, while also characterizing the intervertebral disc, facet joints, and ligamentum flavum, which are key contributors to neural compression.[14,15] In clinical practice, MRI helps determine the most clinically relevant level and side of compression and supports treatment planning by correlating anatomic narrowing with neurologic findings.[14,15] Importantly, MRI can demonstrate morphologic stenosis even in individuals without symptoms, so imaging findings should be interpreted in conjunction with the patient's symptom pattern and physical examination to reduce the risk of overdiagnosis and wrong level intervention.[14,15]

For central canal stenosis, commonly used quantitative parameters include an anteroposterior canal diameter less than 10 mm and a cross sectional canal area less than 70 $mm^2$, although thresholds vary across studies and are not fully standardized.[15] This variability is consistent with evidence showing substantial heterogeneity in semiquantitative and qualitative radiologic criteria and their

reliability for lumbar spinal stenosis diagnosis, highlighting the need for structured interpretation rather than a single universal MRI definition. [7]

For foraminal stenosis, sagittal sequences are used to assess perineural fat effacement and nerve root morphology, with T1 weighted images often facilitating visualization of fat planes. [28] For lateral recess stenosis, axial images are essential to evaluate the subarticular zone where the traversing nerve root is vulnerable to compression from disc pathology, facet hypertrophy, and ligamentum flavum thickening. [15,7]

Although MRI is highly useful, its diagnostic performance is not perfect. A major review reported high sensitivity for lumbar spinal stenosis, while specificity is more limited, reinforcing the principle that MRI should support rather than replace clinical judgment. [15] In addition, interobserver variability remains an issue, particularly when grading lateral recess and foraminal narrowing. [7,28]

A practical limitation is that MRI may underestimate the true bony canal dimensions compared with computed tomography in some patients with severe stenosis. In a comparative study of surgical lumbar spinal stenosis cases, MRI underestimated spinal canal cross sectional area by an average of 15.3 percent relative to CT, with moderate agreement between MRI only evaluation and surgical findings. [27] These findings suggest that multimodal assessment can be considered in selected cases, especially when surgical planning depends heavily on bony constraints or when MRI findings and symptoms are discordant. [27]

Severity assessment and grading systems

Beyond measurements, several morphologic grading systems are widely used to describe stenosis severity in a clinically communicable way. For central canal stenosis, the Schizas classification grades severity based on dural sac morphology on axial T2 weighted images, emphasizing the visibility of cerebrospinal fluid and the arrangement of nerve rootlets. [14] For foraminal stenosis, the Lee grading system is commonly applied using MRI based assessment of perineural fat obliteration and nerve root deformation. [26,28] For lateral recess stenosis, qualitative systems based on the relationship between the traversing nerve root and surrounding structures are often used, including schemes that describe progressive subarticular narrowing and nerve root compromise. [7]

These grading approaches improve reporting consistency and facilitate research comparisons, but they do not guarantee a one-to-one correspondence with symptom severity. Therefore, severity grading should be integrated with symptom distribution, neurologic deficits, and functional limitation when determining the dominant symptomatic level and the optimal management strategy. [14, 15]

**Table 1. Central canal stenosis grading using Schizas[21]**

| Grade | MRI appearance on axial T2 weighted images |
|---|---|
| **A** | Cerebrospinal fluid is visible within the dural sac and nerve rootlets are separated |
| **B** | Nerve rootlets occupy most of the dural sac, individual roots remain identifiable, minimal cerebrospinal fluid may persist |
| **C** | Individual roots are not identifiable and cerebrospinal fluid is not visible, posterior epidural fat may still be present |
| **D** | Roots are not identifiable and posterior epidural fat is not visible |

**Table 2. Foraminal stenosis grading using Lee[22]**

| Grade | MRI appearance, perineural fat and nerve root morphology |
|---|---|
| **0** | No stenosis, perineural fat surrounds the root |
| **1** | Perineural fat obliterated in two opposing directions without root deformity |
| **2** | Perineural fat obliterated in four directions without root deformity |
| **3** | Clear nerve root collapse or morphological deformity due to compression |

## Correlation and Concordance Between Clinical Findings and Magnetic Resonance Imaging in Degenerative Lumbar Canal Stenosis

Despite MRI being central to the anatomic assessment of degenerative lumbar canal stenosis, the concordance between imaging severity and clinical presentation is often limited.[14,15] Degenerative changes and morphologic narrowing can be frequent in individuals without symptoms, and the presence of stenosis on MRI does not necessarily indicate that the imaged level represents the primary pain generator.[29] This high background prevalence of imaging abnormalities supports the principle that MRI findings must be interpreted within the context of symptom distribution, neurologic examination, and functional limitation.[14,15,29]

Evidence from population based and clinical studies demonstrates that associations between MRI severity and typical stenosis symptoms are variable. In a cross sectional analysis of 239 community volunteers, only extremely severe central stenosis showed a strong association with typical lumbar spinal stenosis symptoms, yet even among subjects with severe central stenosis, typical symptoms were not uniformly present.[30] These findings suggest that central canal narrowing increases the likelihood of symptomatic disease primarily at the most advanced grades, while mild to moderate morphologic stenosis may have limited discriminatory value for symptom attribution.[30]

Quantitative approaches have also produced mixed results. In a case control study of patients with suspected lumbar spinal canal stenosis, smaller central canal and lateral recess cross sectional areas were reported to have diagnostic utility, and neurogenic claudication showed the strongest relationship with radiologic markers; however, the study design and symptom heterogeneity highlight that radiologic thresholds are not sufficient to predict symptom distribution or clinical impact at the individual level.[31] Similarly, among patients with surgically confirmed lateral lumbar spinal canal stenosis, MRI graded severity correlated with electromyography abnormalities, but relationships between MRI findings and patient reported symptoms or walking capacity were not demonstrated, supporting a multifactorial model of symptom generation that extends beyond static structural narrowing.[31]

Collectively, these data indicate that MRI is essential for defining anatomy, yet the clinical relevance of a given stenosis pattern is not consistently captured by severity grading alone.[14,15] A clinically important knowledge gap remains the limited direct evidence linking the dominant compartment of stenosis on MRI, central canal, lateral recess, or neural foramen, to the specific distribution pattern of lower limb radiculopathy. Addressing this gap is necessary to improve symptom-based localization, reduce discordance between imaging and clinical decision making, and strengthen the rationale for selecting the most relevant level and side for intervention.[14,15]

## CONCLUSION

Degenerative lumbar canal stenosis is a common age associated condition that arises from a progressive degenerative cascade involving the intervertebral disc, facet joints, and ligamentum flavum, leading to narrowing of the central canal, lateral recess, and neural foramen. This compartment-based anatomy provides a clear structural rationale for typical symptom patterns, including neurogenic claudication in central canal compromise and dermatomal radiculopathy in lateral recess or foraminal compromise. Magnetic resonance imaging remains the preferred modality to define the level, side, and dominant compartment of stenosis and to support grading of severity; however, imaging abnormalities are also frequent in asymptomatic individuals and MRI severity does not consistently predict symptom burden. Age and sex further influence the distribution of stenosis and the way symptoms are expressed and reported, reinforcing the need to interpret MRI findings through a clinical lens.

Overall, the evidence supports an integrated clinicoradiologic approach in which symptom mapping and neurological examination guide interpretation of MRI, rather than relying on morphologic narrowing alone. A clinically important gap persists regarding direct evidence that links the dominant stenosis compartment on MRI to the specific distribution pattern of lower limb radiculopathy. Addressing this gap may improve diagnostic precision, strengthen level selection for intervention, and enhance patient specific decision making in degenerative lumbar canal stenosis.

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