International Journal of Research and Review
[Indexed, Peer-Reviewed Journal]
Volume 13; Issue: 4; April 2026
Website: www.ijrrjournal.com
Review Paper *E-ISSN: 2349-9788; P-ISSN: 2454-2237*

Lumbar Spinal Canal Stenosis: A Review Article

I Putu Lari Sandy[1], I Gusti Ngurah Paramartha Wijaya Putra[2]

[1]Resident of Orthopaedic and Traumatology Department, Faculty of Medicine,
Udayana University/Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Bali, Indonesia
[2]Orthopaedic and Traumatology Department, Faculty of Medicine,
Udayana University/Prof. Dr. I.G.N.G. Ngoerah General Hospital, Denpasar, Bali, Indonesia

Corresponding Author: I Putu Lari Sandy

DOI: https://doi.org/10.52403/ijrr.20260401

ABSTRACT

Lumbar spinal stenosis is a degenerative disorder that results in progressive narrowing of the spinal canal and compression of neural and vascular structures. It most frequently affects individuals above sixty years of age and manifests as persistent low back pain, radiating leg pain, numbness, and neurogenic claudication. These symptoms are typically aggravated by standing or walking and relieved by forward bending or sitting. The underlying mechanisms include disc degeneration, facet joint arthrosis, ligamentum flavum thickening, and osteophyte formation that collectively reduce canal space and compromise neural function. Diagnosis is established through clinical assessment supported by magnetic resonance imaging, which allows precise evaluation of the spinal canal, neural elements, and soft tissue changes. Management begins with conservative measures such as anti-inflammatory medication, physical rehabilitation, and epidural injections to control pain and maintain mobility. Surgical intervention is recommended for patients who experience persistent pain, neurological deficits, or functional decline despite conservative care. Decompressive laminectomy with or without fusion remains the standard procedure, and minimally invasive approaches have demonstrated similar effectiveness with faster recovery and fewer complications. Early diagnosis, timely decompression, and structured rehabilitation are associated with favorable outcomes, while multilevel involvement or delayed treatment predicts less satisfactory results. Comprehensive understanding of the pathophysiology, diagnostic features, and evidence-based management strategies is essential to improve function and quality of life in patients with lumbar spinal stenosis.

Keywords: *lumbar spinal stenosis, degenerative spine disease, neurogenic claudication*

INTRODUCTION

Lumbar spinal canal stenosis (LSCS) is defined as a narrowing of the spinal canal caused by mechanical compression of the spinal nerve roots [1,2] The term “spinal stenosis” refers to an anatomical diagnosis whose frequency increases with age and may also be present in asymptomatic individuals. The exact mechanism leading to the onset of symptoms in LSCS remains unclear. The presence of both symptomatic and asymptomatic cases is thought to be related to individual variations in compensatory mechanisms against spinal canal narrowing [3]

EPIDEMIOLOGY

The prevalence of lumbar spinal canal stenosis (LSCS) varies by diagnostic

method. Clinically, it is about 11% in the general population, while imaging studies report rates from 7% to 23%, and in some reports up to 53%. Prevalence rises sharply with age up to 47% in people aged 70–79 years compared to around 12% at age 40. LSCS is among the leading causes of spinal surgery in adults over 65, contributing to substantial healthcare costs and socioeconomic burden.

ANATOMY

Lumbar Vertebral Structure

The lumbar spine consists of five vertebrae, composed of the vertebral body anteriorly and the neural arch posteriorly. The vertebral canal is triangular, with lateral recesses forming the nerve root canals. Pedicles are short with slight medial angulation, increasing in width from L1 to L5. The laminae are thick, vertically oriented, and divided into cephalic and caudal portions, with the pars interarticularis being a common site of stress fractures. The superior articular facets are concave, while the inferior facets are convex. Spinous processes are nearly horizontal, quadrangular, and thickened along their posterior and inferior margins.

Joint and Ligament Structure

The intervertebral disc and paired facet (zygapophyseal) joints are key components of the lumbar spine, allowing flexion, extension, and lateral bending. The disc consists of a central nucleus pulposus, a surrounding annulus fibrosus, and cartilaginous end plates. The nucleus pulposus is gelatinous and composed of 70–90% water, but its water content decreases with age, leading to disc degeneration. The annulus fibrosus is a laminated collagen structure, thinner posteriorly than anteriorly, with vertically oriented fibers that predispose to posterior or posterolateral herniation. Lumbar disc herniation most commonly occurs at L4–L5 and L5–S1 levels.

Facet Joint

Facet joints consist of adjacent articular processes covered with hyaline cartilage, allowing gliding movements of the spine. The articular capsule is thin, with an inner synovial and outer fibrous layer attached peripherally. With aging, joint space narrows, cartilage thins, and subarticular cortical bone hypertrophy develops in the lumbar region.

Ligaments

The stability of the lumbar spine is maintained by several ligaments, including the anterior and posterior longitudinal ligaments, the ligamentum flavum, and the interspinous and supraspinous ligaments. The anterior longitudinal ligament extends along the anterior aspect of the vertebral bodies and discs from the skull to the sacrum, functioning primarily to limit spinal extension. In contrast, the posterior longitudinal ligament attaches to the posterior aspect of the vertebral bodies, broad in the cervical region but narrower in the thoracic and lumbar regions, and stabilizes the spine during flexion. The ligamentum flavum, composed of elastic yellow fibers, connects adjacent laminae and merges laterally with the facet joint capsule, forming part of the posterior wall of the spinal canal. The interspinous and supraspinous ligaments connect neighboring spinous processes, while the intertransverse ligaments span between transverse processes, particularly in the lumbar region.

Neural Structures

The spinal cord extends from the T12 intervertebral disc to the L1–L2/L3 level and terminates as the conus medullaris, a cone-shaped structure, followed by the cauda equina, a bundle of nerve roots arranged in an orderly pattern. A lumbar enlargement is present between the L1 and S3 segments, supplying the lower extremities. The spinal cord is enveloped by three membranes: the dura mater, arachnoid mater, and pia mater. The dura mater is the outermost dense fibrous layer, separated

from the spinal canal by the epidural space containing fat, loose connective tissue, and a venous plexus. The arachnoid mater forms the middle delicate layer, with the subdural space containing serous fluid. The innermost pia mater, rich in blood vessels, gives rise to denticulate ligaments that protect the cord during spinal movements. The subarachnoid space, located between the arachnoid and pia mater, contains cerebrospinal fluid.

Spinal Nerves

Spinal nerves arise from ventral and dorsal roots entering and exiting the spinal cord. The ventral roots contain motor neurons from the anterior horn, while the dorsal roots carry sensory axons from cell bodies in the dorsal root ganglia. In the lumbosacral region, there are 11 pairs of spinal nerves, five lumbar, five sacral, and one coccygeal. After leaving the spinal canal, each nerve divides into a small dorsal ramus and a larger ventral ramus. The dorsal rami supply the spinal ligaments, muscles, and skin of the back, while the ventral rami extend inferolaterally to form the lumbar and sacral plexuses. The lumbar plexus, formed by the ventral rami of L1–L4, innervates the lower abdominal wall and part of the lower limb, with the femoral nerve as its largest branch. The sacral plexus supplies the gluteal region, perineum, and lower limb, with the sciatic nerve being its largest branch, exiting through the greater sciatic notch to innervate the leg and foot.

Blood Vessels

The lumbar spine and spinal cord are supplied mainly by segmental arteries branching from intercostal and lumbar arteries. These vessels enter via the intervertebral foramina and form anastomoses with adjacent spinal arteries. The spinal cord receives blood from the single anterior spinal artery, paired posterior spinal arteries, and radicular arteries, the largest being the artery of Adamkiewicz, which is crucial for lower spinal cord perfusion. Disruption of these vessels, such as by osteophytes or disc herniation, increases the risk of ischemic injury.

Muscles

Lumbar spinal muscles are divided into posterior, lateral, and anterior groups. Posterior muscles include superficial thoracolumbar fascia, intermediate serratus posterior inferior, and deep erector spinae (iliocostalis, longissimus, spinalis). These muscles stabilize posture and allow extension, rotation, and lateral bending. Lateral muscles include iliopsoas and quadratus lumborum, while anterior and lateral groups assist in lumbar flexion and rotation. Innervation is from dorsal and ventral rami of spinal nerves, with segmental arterial supply.

PATHOGENESIS

Lumbar Spinal Canal Stenosis (LSCS) may be congenital or acquired, with the degenerative form being the most common. Age-related degeneration of the intervertebral disc nucleus, recurrent spinal microtrauma, and axial muscle atrophy with fatty infiltration lead to disc dehydration and collapse. This instability shifts axial load to posterior elements facet joints, interspinous ligament, and ligamentum flavum causing hypertrophy, synovial cyst formation, osteophytes, and ligament thickening, which together narrow the spinal canal [8-10]

Ligamentum flavum thickening and vertebral slippage may further exacerbate stenosis. Genetic predisposition also plays a role, influencing canal size and clinical outcome, explaining wide prevalence variation of moderate (24–78%) and severe (8–30%) stenosis in adults over 40 years [8-10]

Clinically, central LSCS is associated with axial back pain, neurogenic claudication, and possible motor or sensory deficits, most often at L4-5, followed by L3-4 and L5-S1. Two main theories explain neurogenic claudication: the ischemic theory (nerve root hypoperfusion) and the venous stasis theory (impaired capillary oxygenation with metabolite buildup in the cauda equina)

[11-13]

Lateral and foraminal LSCS produce unilateral radiculopathy. Lateral stenosis compresses roots via hypertrophied facet joints and subarticular ligaments, while foraminal stenosis may result from scoliosis, lateral disc herniation, or synovial cysts, affecting spinal nerves or sensory ganglia [14-16]

ETIOLOGY

Lumbar Spinal Canal Stenosis (LSCS) results from narrowing of the spinal canal that compresses neurovascular structures, producing lower extremity pain with or without low back pain [17] It may arise from congenital or acquired causes, with the latter being more common. Degenerative changes such as facet joint hypertrophy, spondylolisthesis, trauma, iatrogenic procedures, and systemic conditions including Paget's disease, ankylosing spondylitis, and acromegaly contribute to acquired LSCS [18] More specifically, the pathogenesis involves (1) disc degeneration leading to protrusion and narrowing, (2) osseous reactions such as facet hypertrophy causing further constriction, and (3) reactive instability related to disc degeneration and capsuloligamentous laxity. [19]

Herniated Disc

Herniated disc represents a degenerative process in which annular tears permit the displacement of the nucleus pulposus, potentially compressing nerve roots or the spinal cord and resulting in pain and neurological dysfunction. The condition is typically classified into four stages. Grade I, or bulging, involves slight outward displacement with minimal nerve compression and is often asymptomatic. Grade II, or protrusion, is characterized by a more pronounced bulging that may produce radicular symptoms. Grade III, or extrusion, occurs when the nucleus pulposus ruptures through the annulus fibrosus, leading to significant neural compression accompanied by severe pain, weakness, or sensory deficits. The most advanced stage, Grade IV, or sequestration, is defined by a free disc fragment migrating within the spinal canal, which can cause persistent nerve compression and frequently necessitates urgent intervention. Magnetic resonance imaging (MRI), particularly axial T2-weighted sequences, plays a crucial role in distinguishing protrusion, which appears as a broad-based extension, from extrusion, which presents as a narrow-based extension.

Disc Space Narrowing

Disc space narrowing occurs when degeneration of the nucleus pulposus reduces intradiscal pressure, leading to collapse of the intervertebral space and approximation of vertebral bodies. It is defined as a reduction in disc height compared with adjacent normal levels and can be assessed using lateral X-ray or sagittal T2-weighted MRI. Measurements below 10.5 mm indicate narrowing, while values above 10.5 mm are considered normal. The Hurxthal method, which evaluates mid-disc height on sagittal MRI, is commonly applied.

Ligamentum Flavum Hypertrophy

Ligamentum flavum hypertrophy is a critical contributor to lumbar spinal canal stenosis, as thickening of this structure narrows the canal and causes radiculopathy [20] MRI studies demonstrate that loading the spine increases ligamentum flavum thickness, which decreases by 50–85% in the supine position [21] Degenerative changes are strongly correlated with ligamentum flavum thickness, with significant associations between disc degeneration and ligament hypertrophy [22] A thickness below 3.1 mm is considered normal, whereas ≥3.1 mm indicates hypertrophy. This parameter can be measured on axial T2-weighted MRI.

Level of Stenosis

Lumbar spinal canal stenosis may occur at a single or multiple levels, reflecting the extent of vertebral segment involvement. Multilevel stenosis is more often associated with neurogenic claudication and less

frequently with radicular pain distribution, whereas single-level stenosis commonly involves L4–5, followed by two levels at L3–5, and three levels at L2–5. Clinical outcomes after surgery differ significantly between single- and multi-level stenosis, and MRI sagittal T2-weighted imaging remains the standard modality for determining stenotic levels.

CLINICAL MANIFESTATIONS

Lumbar spinal canal stenosis is more common in women and typically presents with leg heaviness, numbness, paresthesia, and aching discomfort [1,2] Leg pain, paresthesia, or weakness triggered by walking or standing and relieved by forward flexion (e.g., sitting or squatting) occurs in over 90% of patients [23] Although axial low back pain may be present, most patients report greater discomfort in the lower extremities. Symptoms usually appear after standing or walking for 5–10 minutes and improve with sitting, squatting, or leaning forward, giving rise to the term "spinal claudication." Patients may prefer uphill walking, which flexes the spine and increases canal capacity, compared to downhill walking, which extends it. Foraminal stenosis may cause unilateral symptoms, and some patients have a history of disc prolapse, chronic back pain, or prior spine surgery. [17]

PHYSICAL EXAMINATION

Physical examination is essential to exclude alternative causes of back and leg pain such as hip osteoarthritis, peripheral arterial disease (PAD), or greater trochanteric pain syndrome, which can mimic pseudoclaudication. Elderly patients with stenosis may also have concurrent hip or vascular disease, termed "hip-spine syndrome." Neurologic exams are often normal, but distal pulses and absence of trophic skin changes help differentiate spinal from vascular claudication. In vascular claudication, symptoms resolve after a short rest or even while standing, whereas spinal stenosis requires flexion for relief. Vascular claudication worsens with uphill walking or cycling. Differential diagnosis should also consider hip joint pathology and peripheral neuropathy. [1,2] Pain assessment often uses the Visual Analog Scale (VAS), a psychometric tool widely applied in both research and daily practice. Although VAS is sensitive and reproducible, it is subjective and not specific for lumbar spinal canal stenosis [24]

RADIOGRAPHIC FINDINGS

Plain radiographs, including anteroposterior lumbar, lateral, and pelvic views, are considered first-line imaging. These help assess spinal alignment, disc degeneration, spondylolisthesis, and fractures. Dynamic lateral X-rays may reveal instability with >5 mm translation, while oblique views can demonstrate pars defects. Pelvic radiographs are useful for evaluating the hip and sacroiliac joints. MRI remains the gold standard for lumbar spinal canal stenosis, being non-invasive and safe, with superior soft tissue differentiation. It identifies disc degeneration, prolapse, facet arthrosis, and effusion. CT myelography, indicated in cases with MRI contraindications or metallic implants, may provide better visualization of dynamic stenosis or severe scoliosis. [1,2]

GRADING OF CANAL STENOSIS

Canal stenosis is graded using cerebrospinal fluid (CSF) distribution on axial T2-weighted MRI, based on the visibility and arrangement of nerve rootlets within the dural sac [25] Grade A denotes no or minor stenosis, with visible CSF but variable rootlet distribution (A1–A4 depending on rootlet location and dural sac occupancy). Grade B indicates moderate stenosis, where rootlets fill the dural sac but remain distinguishable, with residual CSF. Grade C represents severe stenosis with no identifiable rootlets, producing a homogeneous gray dural sac signal with posterior epidural fat. Grade D denotes extreme stenosis, with no rootlets or posterior epidural fat visible. Ligamentum

flavum thickness can also be measured on axial MRI using T1W or T2W sequences, typically along the facet joint line.

MANAGEMENT

Non-Operative

The primary goal of non-operative management in lumbar spinal canal stenosis (LSCS) is pain reduction and functional improvement. Initial treatment typically involves a multimodal approach combining anti-inflammatory medications, physical therapy, and epidural injections [26] Nonsteroidal anti-inflammatory drugs (NSAIDs) are considered first-line therapy, while opioids and muscle relaxants may provide temporary relief but are less effective and carry higher risks, particularly in older patients [27] Physical therapy focuses on strengthening, flexibility, and stabilization, though strong evidence supporting its efficacy as monotherapy is lacking. However, the SPORT trial subgroup analysis suggested that early physical therapy within six weeks reduced the likelihood of surgery at one-year follow-up. Epidural injections may provide short-term pain and functional relief, though their long-term benefit remains limited. The addition of steroids to local anesthetics has not consistently demonstrated superior outcomes, making epidural injections an option only for selected patients with severe, disabling symptoms [26]

Operative

Surgical intervention is considered for patients with persistent symptoms, progressive neurological deficits, or functional limitations despite adequate conservative therapy. Decision-making is guided by clinical evaluation of motor weakness, radicular symptoms, and the anatomical distribution of neural compression, whether central, lateral, or foraminal [28] The most common surgical procedure is decompressive laminectomy, performed with or without spinal fusion depending on the presence of instability. Fusion, with or without instrumentation, may improve stability and promote fusion in compromised segments [29]

Postoperative Care and Rehabilitation

Postoperative management and rehabilitation play a crucial role in optimizing recovery, restoring function, and enhancing quality of life. Early postoperative care includes monitoring vital signs, pain control, prevention of complications, wound care, fluid and nutritional management, and medication adjustment. Rehabilitation aims to restore mobility, independence, and functional capacity through physiotherapy, occupational therapy, and psychological support. Physiotherapy focuses on improving muscle strength, flexibility, and range of motion, while occupational therapy helps patients regain daily living skills. Psychological support addresses pain-related anxiety, coping strategies, and lifestyle adjustments. Patient education, adherence to care plans, and coordinated support from the healthcare team are essential to achieving successful recovery.

COMPLICATIONS

The diagnosis of lumbar spinal canal stenosis (LSCS) is primarily based on subjective symptoms, which typically manifest during standing or walking and are absent at rest. Surgical treatment has been shown to result in greater long-term patient satisfaction (63%) compared to non-surgical management (42%) at 4-year follow-up. Moreover, surgical outcomes are strongly correlated with the severity of preoperative stenosis [30]

Early Complications

Despite adequate decompression, some patients may remain unable to return to occupations requiring heavy physical activity. Chronic radicular neurological deficits associated with muscle atrophy are unlikely to recover fully. In addition, low back pain arising from underlying degenerative arthritis, rather than entrapment-related radiculopathy, is the

symptom least likely to improve following decompressive surgery. In-hospital mortality within six weeks after discharge has been reported at 0.8% for patients under 75 years, 1.1% for those aged 75–79 years, and 2.3% in individuals over 80 years [31]

Late Complications

Late postoperative complications of lumbar surgery include vascular events such as deep vein thrombosis (DVT), pulmonary embolism, postoperative hematoma, and catastrophic vascular injury. The overall reported incidence of DVT is 2.2%, while pulmonary embolism occurs in approximately 0.3%. Among patients undergoing elective posterior lumbar decompression with or without fusion, the risk of DVT without prophylaxis is estimated at around 5% based on venographic or clinical diagnosis. Studies have suggested that mechanical prophylaxis alone may be insufficient for patients undergoing combined anterior/posterior spinal fusion. Untreated LSCS can result in chronic pain or even paralysis. Prolonged pain often leads to immobility, predisposing patients to further complications such as pneumonia, muscle atrophy, urinary dysfunction, and decubitus ulcers [32]

Recurrence Rate

Recurrent lumbar spinal stenosis (LSS) remains a concern despite surgical treatment, as stenosis may reappear at the index, adjacent, or distant levels. Reported revision rates vary, with 0–13% of patients requiring reoperation, most commonly within 16–35 months after the initial procedure. The risk of recurrence is strongly related to time since surgery, the type of procedure performed, and patient-specific factors.

PROGNOSIS

Lumbar spinal stenosis typically becomes symptomatic after the sixth decade, progressing gradually with neurogenic claudication as the hallmark feature. Prognosis is favorable with early and adequate decompression, preserved facet stability, and proper rehabilitation, but worsens with multilevel disease, neurological deficits, delayed surgery, failed prior procedures, or chronic systemic comorbidities.

DIFFERENTIAL DIAGNOSIS

Differential diagnoses of lumbar spinal canal stenosis (LSCS) include several degenerative, inflammatory, traumatic, and infectious conditions. Osteoarthritis can cause facet joint degeneration and ligamentum flavum hypertrophy leading to pain. Ligamentum flavum cysts, though rare, may result from degeneration or microtrauma and often occur at L4–L5. Ankylosing spondylitis involves axial inflammation, new bone formation, and facet ankylosis that impair mobility. Spinal gout may present with mild symptoms or severe complications, including neurological deficits from compression. Trauma, especially thoracolumbar fractures and facet dislocations, represents the most common spinal injury. Infections, such as septic arthritis of the facet joint, usually spread hematogenously or after interventions and must be distinguished from conditions like herniated disc, radiculopathy, spondylosis, rheumatoid arthritis, sacroiliac dysfunction, or fibromyalgia.

CONCLUSION

Patients with lumbar spinal stenosis may present with symptoms such as back pain, radiating pain, and neurogenic claudication. While long-term outcomes of surgical and non-surgical treatments are often comparable, surgery offers advantages including short-term symptom relief and reduced fall risk. Surgical management typically involves decompression, with or without adjunct procedures depending on the extent of decompression and spinal instability. Recently, minimally invasive surgical approaches have shown excellent outcomes in treating lumbar spinal stenosis. Therefore, a comprehensive approach that

integrates pathophysiological understanding with appropriate treatment strategies is essential to achieve more effective management.

Declaration by Authors

Ethical Approval: Not applicable
Acknowledgement: None
Source of Funding: None
Conflict of Interest: No conflicts of interest declared.

REFERENCES

1. Blom A, Warwick D and Whitehouse M (2017a) Apley & Solomon's System of Orthopaedics and Trauma. 10th editi. New York: CRC Press.
2. Blom A, Warwick D and Whitehouse M (2017b) Apley & Solomon's System of Orthopaedics and Trauma. 10th editi. New York: CRC Press.
3. Garfin S, Eismont F, Bell G, et al. (2017) Rothman-Simeone and Herkowitz's The Spine, 2 Vol Set. 7th ed. Elsevier.
4. Fortin M, *et al*. (2017) Association between paraspinal muscle morphology, clinical symptoms and functional status in patients 56 with lumbar spinal stenosis. European Spine Journal. DOI: 10.1007/s00586-017-5228-y.
5. Kalichman L, *et al*. (2009) Spinal stenosis prevalence and association with symptoms: the Framingham Study. Spine Journal. DOI: 10.1016/j.spinee.2009.03.005.
6. Schroeder GD, *et al*. (2016) Lumbar spinal stenosis: How is it classified? Journal of the American Academy of Orthopaedic Surgeons. DOI: 10.5435/JAAOS-D-15-00034.
7. Yoshida M, *et al*. (1992b) Hypertrophied ligamentum flavum in lumbar spinal canal stenosis: Pathogenesis and morphologic and immunohistochemical observation. Spine. DOI: 10.1097/00007632- 199211000-00015.
8. Battié MC, *et al*. (2014) Brief report: Lumbar spinal stenosis is a highly genetic condition partly mediated by disc degeneration. Arthritis and Rheumatology. DOI: 10.1002/art.38823
9. Parker SL, Godil SS, Mendenhall SK, et al. (2014) Two-year comprehensive medical management of degenerative lumbar spine disease (lumbar spondylolisthesis, stenosis, or disc herniation): A value analysis of cost, pain, disability, and quality of life: Clinical article. Journal of Neurosurgery: Spine. DOI: 10.3171/2014.3.SPINE1320.
10. Yabe Y, Hagiwara Y, Ando A, et al. (2015) Chondrogenic and Fibrotic Process in the Ligamentum Flavum of Patients with Lumbar Spinal Canal Stenosis. Spine 40(7): 429–435. DOI: 10.1097/BRS.0000000000000795.
11. Lurie J and Tomkins-Lane C (2016) Management of lumbar spinal stenosis. BMJ (Online). DOI: 10.1136/bmj.h6234.
12. Siebert E, Prüss H, Klingebiel R, et al. (2009a) Lumbar spinal stenosis: Syndrome, diagnostics and treatment. Nature Reviews Neurology. DOI: 10.1038/nrneurol.2009.90.
13. Tomkins-Lane CC, Battié MC, Hu R, et al. (2014a) Pathoanatomical characteristics of clinical lumbar spinal stenosis. Journal of Back and Musculoskeletal Rehabilitation. DOI: 10.3233/BMR-130440
14. Choi Y-S (2009) Pathophysiology of Degenerative Disc Disease. Asian Spine Journal. DOI: 10.4184/asj.2009.3.1.39.
15. Siebert E, Prüss H, Klingebiel R, et al. (2009b) Lumbar spinal stenosis: Syndrome, diagnostics and treatment. Nature Reviews Neurology. DOI: 10.1038/nrneurol.2009.90
16. Tomkins-Lane CC, Battié MC, Hu R, et al. (2014b) Pathoanatomical characteristics of clinical lumbar spinal stenosis. Journal of Back and Musculoskeletal Rehabilitation. DOI: 10.3233/BMR-130440.
17. Harry Herkowitz, Steven Garfin, Frank Eismont, et al. (2011a) RothmanSimeone The Spine - 6th Edition.
18. Miller MD and Thompson SR (2016) Miller's Review of Orthopaedics. 7th ed. Philadelphia, PA: Elsevier.
19. Thomé C, Börm W and Meyer F (2008) Degenerative Lumbar Spinal Stenosis – Current Strategies in Diagnosis and Treatment. Deutsches Aerzteblatt Online. DOI: 10.3238/arztebl.2008.0373.
20. Yoshida M, Shima K, Taniguchi Y, et al. (1992a) Hypertrophied ligamentum flavum in lumbar spinal canal stenosis. Spine 17(11): 1353–1359. DOI: 10.1097/00007632-199211000-00015.
21. Hansson T, Suzuki N, Hebelka H, et al. (2009a) The narrowing of the lumbar spinal canal during loaded MRI: the effects of the disc and ligamentum flavum. European

Spine Journal 18(5): 679–686. DOI: 10.1007/s00586- 009-0919-7.
22. Munns JJ, Lee Joe Y. B., Espinoza Orías AA, et al. (2015) Ligamentum Flavum Hypertrophy in Asymptomatic and Chronic Low Back Pain Subjects. PLOS ONE Chen T (ed.) 10(5): e0128321. DOI: 10.1371/journal.pone.0128321
23. Katz JN, Dalgas M, Stucki G, et al. (1995) Degenerative lumbar spinal stenosis diagnostic value of the history and physical examination. Arthritis & Rheumatism. DOI: 10.1002/art.1780380910
24. Abou-Al-Shaar H, Adogwa O and Mehta AI (2018) Lumbar Spinal Stenosis: Objective Measurement Scales and Ambulatory Status. Asian Spine Journal 12(4): 765–774. DOI: 10.31616/asj.2018.12.4.765
25. Schizas C, Theumann N, Burn A, et al. (2010) Qualitative grading of severity of lumbar spinal stenosis based on the morphology of the dural sac on magnetic resonance images. Spine. DOI: 10.1097/BRS.0b013e3181d359bd
26. Covaro A, Vilà-Canet G, de Frutos AG, et al. (2016) Management of degenerative lumbar spinal stenosis: An evidence-based review. EFORT Open Reviews. DOI: 10.1302/2058-5241.1.000030
27. Kreiner DS, *et al*. (2013) An evidence-based clinical guideline for the diagnosis and treatment of degenerative lumbar spinal stenosis (update). Spine Journal. DOI: 10.1016/j.spinee.2012.11.059.
28. Bagley C, *et al*. (2019) Current concepts and recent advances in understanding and managing lumbar spine stenosis [version 1; referees: 3 approved]. F1000Research. DOI: 10.12688/f1000research.16082.1.
29. Shah JM, Gritsenko K and Wahezi SE (2017) Degenerative lumbar spinal stenosis. In: Pain Medicine: An Essential Review. DOI: 10.1007/978-3-319- 43133-8_122.
30. Weber C, *et al*. (2017) Surgical management of lumbar spinal stenosis: a survey among Norwegian spine surgeons. Acta Neurochirurgica. DOI: 10.1007/s00701-016-3020-0.
31. Ciol MA, *et al*. (1996) An Assessment of Surgery for Spinal Stenosis: Time Trends, Geographic Variations, Complications, and Reoperations. Journal of the American Geriatrics Society 44(3): 285–290. DOI: 10.1111/j.1532-5415. 1996.tb00915. x.
32. Garfin S, *et al*. (2017) Rothman-Simeone and Herkowitz's the Spine, 2 Vol Set. 7th ed. Elsevier.

How to cite this article: I Putu Lari Sandy, I Gusti Ngurah Paramartha Wijaya Putra. Lumbar spinal canal stenosis: a review article. *International Journal of Research and Review*. 2026; 13(4): 1-9. DOI: *https://doi.org/10.52403/ijrr.20260401*

******