BACK HEALTH

Symptomatic Lumbar Canal Stenosis— A Review and Primer on Surgical Decision Making

ABSTRACT

Lumbar canal stenosis is an anatomical term used to describe narrowing of the spinal canal either congenitally or from age-related degenerative changes. It refers to a structural finding that may or may not be symptomatic. A decrease in canal diameter can lead to compression of the neural components, causing a constellation of symptoms. Family physicians should familiarize themselves with the various presentations of canal narrowing and the available diagnostic and treatment options.

KEYWORDS: lumbar spinal stenosis, neurogenic claudication, back pain, radiculopathy





Introduction and Background:

Narrowing of the lumbar spinal canal, lumbar canal stenosis (LCS), is an inevitable result of the degenerative changes associated with aging. It varies in degree and may or may not become symptomatic. When symptoms do arise, they can present as low back pain, lumbar radiculopathy or neurogenic claudication.¹ Rapid and severe compression can cause cauda equina syndrome, a surgical emergency, that is not discussed here. This article covers the pathogenesis, clinical presentation, and imaging recommendations for patients presenting with neurogenic claudication or lumbar radiculopathy. We present basic concepts in decision making for the array of surgical management strategies aimed at symptomatic lumbar spinal stenosis.





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Figure 1: Diagram showing the anatomy of a normal spinal canal, and a canal with spinal stenosis in both the axial and sagittal views.



Pathogenesis:

The progressive narrowing of the spinal canal in the lumbar region results from a variety of degenerative changes, including disc herniation, vertebral body and facet joint osteophyte formation and hypertrophy of the ligamentum flavum (Figure 1).

Lumbar canal stenosis may remain asymptomatic but, as it grows more intrusive, can lead to neural compression with resultant clinical manifestations. Stenosis can be classified as central, lateral recess, foraminal, or extra-foraminal (Figure 2).

Stenosis commonly affects the central canal and lateral recesses causing mechanical compression and intermittent ischemia of the cauda equina and nerve roots. This impairs nerve conduction, leading to symptoms such as neurogenic claudication or radiculopathy.² Due to the greater biomechanical forces exerted on the lower lumbar spine, the most affected levels are L4-5 and L5-S1.³



Clinical Presentation:

The history and physical examination should focus on identifying pain producing movements, location of the dominant pain sites, areas of numbness, motor weakness and bowel or bladder function. There are several findings which help confirm the diagnoses of neurogenic claudication or lumbar radiculopathy.

Neurogenic claudication is seen most often in people over 60 years old. Symptoms include an aching pain or heaviness in both legs pain, transient weakness or numbness. Variations of these complaints are present in 93% of patients who suffer symptoms from LCS.⁴ Walking and standing for prolonged periods typically exacerbate the symptoms.⁵ Patient's may also describe gait unsteadiness related to leg weakness or numbress of the feet. Pain relief is commonly achieved by sitting or leaning forward, which increases the volume of the neural foramina and decreases pressure on the spinal nerves.^{5,6}

Radicular symptoms are less common and manifest as pain distributed in a specific dermatomal pattern with or without associated sensory or motor disturbances. The patient may present with weakness in a specific myotomal distribution. Common positive radicular findings for nerve root irritation/compressions are the straight leg raise sign (SLR) for the lower lumbar nerve roots (L4, L5, S1)and the femoral nerve stretch, for the upper roots (L2, L3, L4).

Both neurogenic claudication and radicular symptoms are frequently associated with activityrelated lower back pain.

The most ominous findings of LCS are bowel or bladder symptoms. Bladder dysfunctions include urinary incontinence, detrusor over or underactivity and frequent urinary tract infections that significantly impact the quality of life.^{7,8} Bowel symptoms such as constipation and numbness in the saddle area are less frequent.⁹

Additional aspects of history include constitutional symptoms. The presence of depressive symptoms in LCS patients is associated with poorer surgical outcomes, highlighting the importance of identifying psychological factors.¹⁰

Eliciting deep tendon reflexes in the lower limbs are an important part of the examination. The presence of concurrent stenosis in both the cervical and lumbar spine is 11%.¹¹ While LCS can produce either diminished or absent patellar and Achilles tendon reflexes, cervical stenosis with spinal cord compression results in hyperreflexia, a marker of cervical myelopathy.

There are several pathologies that can masquerade as neurogenic claudication or radiculopathy resulting from LCS. Conditions which may be confused with LCS include peripheral vascular disease, osteoarthritis of the hip or knee, trochanteric bursitis, sacroiliitis and peripheral neuropathies.

Unilateral groin pain, knee pain, pain that decreases with continued walking, and pain that occurs immediately with walking or standing are more indicative of hip arthritis.¹² A limp, painful range of hip motion and groin pain with internal rotation of the leg all indicate hip pathology and speak against LCS as the source of the problem.¹³ Local pain produced by direct palpation over the greater trochanter suggests trochanteric bursitis. A diagnostic ultrasoundguided injection of local anesthetic can help resolve an ambiguous situation. A similar approach can be utilized for sacroiliitis with palpation of the sacroiliac joint and the use of a diagnostic injection.

Arterial insufficiency in peripheral vascular disease (PVD) is a common mimic of neurogenic claudication (Table 1). Patients with arterial insufficiency will typically walk for a fixed distance before needing to rest whereas patients with neurogenic claudication can walk variable distances.¹⁴ Patients with PVD find relief when they stop walking, even if they remain standing, whereas pain relief from neurogenic claudication requires sitting or squatting to flex the spine. Pain that comes on predominantly in the buttocks and above the knees is more likely from lumbar canal stenosis while pain that appears below the knees and in the calves is more commonly PVD.¹⁴ Vascular pathology in the lower limbs may be indicated by poor or absent peripheral pulses or abnormal arterial bruits.¹⁵ Values less than 0.9 on the Ankle-Brachial Index (ABI) suggest the presence of **PVD.**¹⁶

Peripheral neuropathy producing numbness, tingling, or weakness in the lower limbs is another condition with features that can mimic neurogenic claudication or lumbar radiculopathy. A history of lower limb trauma could suggest peripheral nerve damage. The

Table 1: Symptoms to help differentiate pain from lumbar canal stenosis versus peripheral vascular disease.	
Neurogenic Claudication	Vascular Claudication
Pain worsens with walking variable distance	Pain worsens with walking same distance
Pain relieved by sitting	Pain relieved by standing
Pain relieved by leaning forward	No specific body position relieves pain
Pain predominantly above the knees	Pain predominantly below the knees
Peripheral pulses normal	Weak/Absent peripheral pulses

symptoms of a peripheral nerve entrapment may be reproduced by tapping over the anatomical area of the compression; an examination finding that rules against lumbar canal stenosis.¹³ Patients with diabetes or vitamin B12 deficiencies may present with numbness or pain in the lower limbs. Examining for femoral lateral cutaneous nerve compression, peroneal compression, and tarsal tunnel compression can help the diagnosis when suspicions arise. Examination may reveal loss of sensation in a stocking distribution which is not the result of LCS. Nerve conduction studies or EMG may aid in localizing the pathology.¹⁷

Imaging for Lumbar Canal Stenosis:

When the history and physical examination strongly suggest significant symptoms of lumbar canal stenosis, imaging is usually indicated. It is important to realize that positive magnetic resonance imaging (MRI) findings can be found in up to 68% of asymptomatic individuals above the age of 55.^{18,19} As such, imaging findings alone can be misleading and prompt unnecessary investigations, referrals or treatments.

MRI can provide a clear anatomic view of the soft tissues and nerves within the lumbar spine and visualize the degree of degeneration of the discs, facet joints and ligamentum flavum with a sensitivity ranging from 81% to 97%.²⁰ Images can display intervertebral disc degeneration, disc bulges or herniations, hypertrophied facets or ligamentum flavum, vertebral osteophytes or ligament ossification. These degenerative changes may be superimposed on an already congenitally narrowed canal, identified by the short pedicles. Asymmetric degenerative changes can lead to degenerative scoliosis.

MRI can identify degenerative spondylolisthesis, a shift in the alignment of a vertebral body relative to the one adjacent that can produce a dramatic amount of canal narrowing. Flexion-extension x-rays establish the degree of instability; the radiologic definition of instability is greater than 3 mm dynamic sagittal translation and/or 10 degrees of angulation.^{21,22}

There are instances when an MRI may be contraindicated: metallic foreign bodies close to the eye or other vital structures, first trimester pregnancy, patients with pacemakers or patients who have had prior spinal surgery with metal implants. In these cases, a CT myelogram may be substituted to obtain the needed information.^{23,24}

Non-Operative Management of Lumbar Spinal Stenosis

The initial management of patients presenting with symptomatic lumbar canal stenosis, either neurogenic claudication or radicular pain involves non-operative modalities including physiotherapy, lifestyle modifications, patient education, medication and, in a few cases, image-guided injections. There is moderate quality evidence that this multimodal approach, which may also include manual therapy and exercise, is safe and effective.²⁵ The same study concluded that epidural steroid injections are not effective for managing symptoms of neurogenic claudication while spinal manipulation and acupuncture have low quality evidence for their use.25 A high quality randomized controlled trial compared glucocorticoid plus lidocaine injection versus lidocaine injection alone for patients with symptomatic lumbar spinal stenosis. At 6-week follow up, there was no difference in pain or disability outcomes.²⁶ Although epidural steroid injections are largely ineffective for treatment, they may aid in diagnosis and the localization of pathology.

A comprehensive review of the pharmacological management of low back pain, neurogenic claudication or radiculopathy is beyond the scope of this manuscript and readers are referred to the many published guidelines on the subject.²⁷⁻³⁰

Surgical Management of Lumbar Spinal Stenosis

Surgical management of lumbar spinal stenosis may be indicated when the symptoms substantially limit the patient's lifestyle and following the failure of 6 months of non-operative treatment. Acute Cauda Equina Syndrome demands urgent intervention while a progressive neurological deficit requires close observation and possible early surgery. Some conditions such as stenosis associated with a deformity are unlikely to respond to nonoperative approaches. In general, multiple large scale randomized controlled trials have demonstrated the superiority of surgical management over prolonged conservative care.³¹⁻³³

The surgical algorithm typically includes an assessment of the extent of the neural element decompression required and the need for subsequent stabilization and fusion of the involved segments.

Decompression of the lumbar spine typically involves removal of the dorsal elements including the spinous process, lamina, and/ or facets. The procedures may include a laminectomy or hemilaminectomy, foraminotomies or discectomy. The exact areas for decompression are determined by the location of the neural compression (central, paracentral, or foraminal), whether the symptoms are unilateral or bilateral. and if more than one nerve root is involved. Significant foraminal compression may necessitate resection of an entire facet joint. There may be the option of an indirect decompression. Restoring the disc height with an interbody implant

SUMMARY OF KEY POINTS

- 1. Lumbar spinal stenosis is commonly caused by age-related degenerative changes involving the intervertebral discs, ligamentum flavum and facet joints.
- 2. Patients with lumbar spinal stenosis may present with neurogenic claudication or radiculopathy.
- 3. The primary care provider needs to distinguish between symptomatic lumbar spinal stenosis and other common mimics
- 4. Surgical treatment is principally decompression of the neural elements with the possible addition of fusion of the affected levels.

preserves the bony elements while increasing the size of the adjacent foramina to provide sufficient space for the nerve roots to exit unimpeded.

Once the extent of decompression has been established, the next consideration is whether stabilization is required. An extensive decompression may render the spine unstable and pedicle screws and rods may be required to restore spinal integrity. Historically, stabilization has been considered an important adjunct in managing degenerative spinal conditions, based on the theory that spinal degeneration is a result of micro-instability in the joints and discs. Instrumentation reduces or eliminates movement. Although fusion has become increasingly common in North America over the past two decades, it must be noted that, in the absence of iatrogenic instability, spondylolisthesis or deformity, large scale randomized control trials have not demonstrated any significant benefit to

the addition of instrumentation to a simple decompression.^{32,34-36} Proponents of fusion point to data suggesting improved outcomes and lower rates of revision surgery.^{37,38} The decision to stabilize the spine after decompression remains subject to surgeon preference and patient considerations.

Instrumented stabilization is not the same as fusion, a fact often misunderstood. The surgical implants provide immediate immobility that promotes bone growth but without subsequent maturation of that biological connection to fuse the two structures into one, the temporary mechanical bridge will fail. Spinal fusion may involve autogenous bone grafts or osteobiologic agents to promote growth at the target levels. The approach may be posterior, fusing through the facets and transverse processes, or lateral using a miniretroperitoneal or an oblique lateral approach. Implants and bone grafts can be placed anteriorly into the disc spaces. Regardless of the approach used, studies show similar overall



1. Degenerative changes in the lumbar spine can lead to various symptoms such as low back pain, lumbar radiculopathy, neurogenic claudication, and cauda equina syndrome.

2. Imaging of the lumbar spine should be ordered when there is a high clinical suspicion of lumbar spinal canal stenosis based on the history and physical examination.

3. Initial management of patients presenting with lumbar canal stenosis involves non-operative modalities like pharmacological therapy, physiotherapy, lifestyle modifications, patient education and image-guided injections.

4. Surgical decompression for symptomatic lumbar spinal stenosis, with or without fusion, is generally indicated when symptoms significantly interfere with daily activity and non-operative treatment has failed after 3-6 months.

outcomes.³⁹⁻⁴¹ For patients with osteoporosis or osteopenia, bone cement can be injected through fenestrated pedicle screws to enhance the hardware construct and prolong its stability.⁴² The goal is to maintain the construct long enough for bony fusion to occur.

Conclusion:

Patients with symptomatic degenerative lumbar canal stenosis may present with features of neurogenic claudication or radiculopathy. Careful evaluation of the patient will distinguish symptoms related to the lumbar spine from other conditions such as osteoarthritis of the hip, peripheral vascular disease or peripheral neuropathy. MRI can identify the neurological area(s) of interest. Family physicians should be familiar with both the non-operative management and the elements of surgical decision making.

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Post-test Quiz

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