

LUMBAR CANAL STENOSIS

Abstract

The clinical consequence of this compression is neurogenic claudication and varying degrees of leg and back pain. Degenerative lumbar spinal stenosis is a major cause of pain and impaired quality of life in the elderly. The natural history of this condition varies; however, it has not been shown to worsen progressively. Nonsurgical management consists of nonsteroidal anti-inflammatory drugs, physical therapy, and epidural steroid injections. If nonsurgical management is unsuccessful and neurologic decline persists or progresses, surgical treatment, most commonly laminectomy, is indicated.

Recent prospective randomized studies have demonstrated that surgery is superior to nonsurgical management in terms of controlling pain and improving function in patients with lumbar spinal stenosis

Relevant anatomy

Cauda Equina

Cauda Equina: Neural elements are organized in a well defined pattern. I.e., at 4-5 disc: S 2-5 is most posterior and S1 in the centre and L5 is more anterior.

The motor component of each nerve root is anterior and sensory posterior in a spinal nerve root. Within the neuroforamen lie the dorsal root ganglion, which consists of small motor component anteriorly, and a larger sensory component, containing neural fibers and cell bodies, posteriorly.

Lateral canal [3 Zones]

a. The entrance zone

Is the most cephalad part,

Located underneath the SAF

The anterior wall is the posterior surface of the disc, and the posterior wall is the facet

b. The mid zone

Located under the pars interarticularis

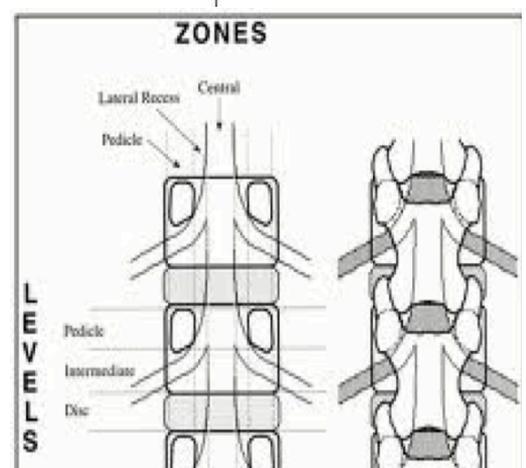
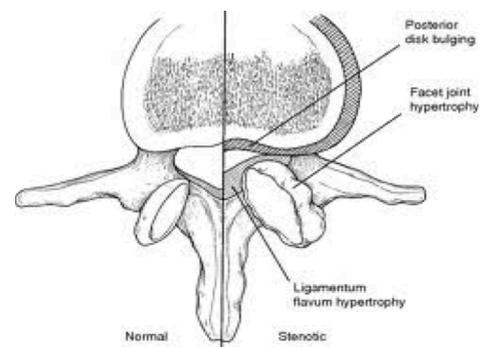
The anterior border of this zone is the posterior aspect of the vertebral body.

The posterior border is the pars interarticularis; the lateral border is the pedicle

This zone contains the dorsal root ganglion

c. The exit zone

Is the area surrounding the IVF foramen. The posterior border is the lateral aspect of the facet joint of the lower level; and the anterior border is the disc of the lower level



Definition: Lumbar spinal stenosis is a reduction in the volume of the central spinal canal, the lateral recesses, and/or neuroforamina that decreases the space available for the exiting nerve roots.

Pathology

Collagen: NP Type II (help to provide high level of hydration)

AF Type Equal amounts of I and II

Age related changes:

Type I collagen in AF increases with age.

KSO₄ increase by age

Water concentration decreases

Proteoglycan: Responsible for Disc compressibility. With age Pg content decreases

Degeneration starts at 20 years and by 50 most have some degree of degeneration

Commonest site is L4-5, L5-1

Because of biomechanical changes in the disc, excessive stresses are transmitted to facet joint leading to degeneration of the facet joint. As a result, hypertrophy of the facet joint, arthritis, synovial cyst form as well as thickness of ligamentum flavum, bulging of the annulus. These changes collectively cause spinal canal stenosis.

If the rate of facet joint alteration (erosion/capsular laxity) exceeds that of disc, anterior subluxation occurs.

Although degeneration is universal, only in some individuals, it may be symptomatic.

The symptoms and signs of neurogenic claudication are not solely related to the underlying compressive changes. Compression alone does not independently cause pain but can cause neurology

In a normal spinal canal, the nerve moves as much as five mm within the neuroforamen.

If normal movement obstructed, the internal tension in the nerve increase. Inflammation of the nerve in this situation causes pain. Therefore pain is due to combination of inflammation, mechanical pressure, venous congestion and ischemia.

Factors for stenosis

- a. Pre-existing congenital or developmental narrowing of the lumbar spinal canal [short pedicle]
- b. Shape of the canal: Trefoil more susceptible than round canal
- c. Degenerative changes reducing canal size:
 - Ligamentum flavum hypertrophy, Facetal and capsular thickening, annular bulge, osteophytes and synovial cyts.
- d. Always occurs in the mobile segment [in case of transitional vertebra: L4/5 is more commonly involved]

Classification

- I Congenital Achondroplasia [short pedicles in 20% of cases]
- II Developmental Idiopathic
- III Dysplasia Osteopetrosis
- IV Acquired Degenerative: Central
Lateral
Degenerative listhesis
- V Traumatic
- VI Iatrogenic Post fusion
Post laminectomy
- VII Miscellaneous: Acromegaly, Paget, and Fluorosis

Natural History

1. Prevalence of degenerative lumbar spondylosis in the general population ranges from 20% to 25% and increases with age >50 years.
 2. Johnsson : untreated patients with spinal stenosis over 49 months. Symptoms of neurogenic claudication remained unchanged in 22 patients (70%), symptoms improved in 5 patients (15%), and symptoms worsened in 5 patients (15%).
- Acute presentation frequently occurs in patients with large central disk herniation, whereas delayed presentation often occurs in patients with chronic spinal stenosis.

Diagnosis

1. Vague bizarre complaints: back ache and leg pain
2. Pain more on standing and walking
3. Cycling, bending forward, leaning on a trolley or stooped posture help pain.
4. Neurogenic claudication
5. Degenerative spondylolisthesis: 6 times more common in women and most of them have sagittal orientation of facet joint. Slip rarely exceed 30%
6. In the rare case of a progressive neurologic deficit or cauda equina syndrome, urgent surgical decompression is indicated.
7. In most cases, the natural history of degenerative lumbar stenosis is variable and does not follow a progressively deteriorating course.
8. Variable weakness, numbness
9. Night muscle cramps

Neurogenic claudication is described as pain or numbness or weakness in the leg or thigh or buttock while standing as well as walking capacity. This is observed in 94% of patients Whereas numbness (63%), or weakness (43%).

Ability to walk distances can be increased by ambulating with the spine in a flexed forward posture such as that used when pushing a shopping cart.

Typically, spinal extension narrows the spinal canal and worsens neurogenic symptoms, whereas

spinal flexion and sitting increases the diameter of the spinal canal, partially alleviating symptoms.

Neurogenic Vs Vascular claudication

	Vascular	Neurogenic
Type	Sharp/cramping	Vague, heaviness
Location	Calf	Radicular or diffuse
Radiation	None	Proximal to distal
Aggravation	Walking	Walking/standing
Relief	Standing still	Bend/squat
Relief	Quick	slow
Neurological symptoms	Absent	Present
Claudication distance	Constant	Variable
Walking uphill, bike	Claudication present	Absent
SLR	Negative	May be positive

Radiographic Evaluation

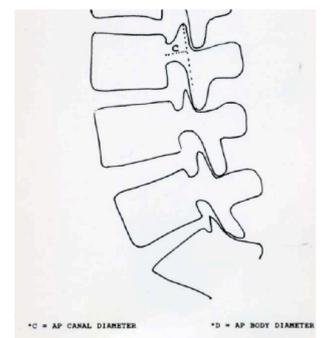
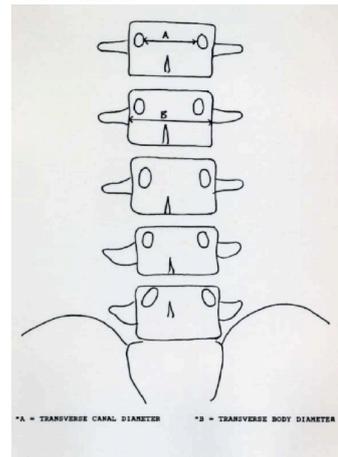
1. Plain X ray

Mean AP diameter: 12 mm (Absolute <10)

Jones Index: 1:2 to 1:4.5

Spondylolisthesis, disk space narrowing, end plate sclerosis, osteophytes, and facet hypertrophy.

Jones index



2. CT Minimal cross section area:

In normal, it is 100 mm

In stenosis, <77 mm

May show trefoil-shaped spinal canals



3. MRI because it can demonstrate disk degeneration or herniation, hypertrophy of the ligamentum flavum and facet capsule, and narrowing of the central canal and lateral recess

At the site of stenosis: Abnormal intrathecal enhancement on Gadolinium enhanced MRI



4. Myelographic [hardly performed these days]

Defect increases with extension

Filling defect of the dural sleeve

Redundant nerve root



Differential Diagnosis

	<i>Lumbar disc herniation</i>	<i>Lumbar spinal stenosis</i>
Age (years)	<50	>50
Sex	Male	Female
Onset	Acute	Insidious
Pain	Radicular/dermatomal	Referred/diffuse
Provocative factors	Sitting	Standing/walking
Palliative factors	Standing	Sitting
Weakness	Common	Uncommon
Sensory changes	Common	Uncommon
Tension sign	Present	Absent
Neurological findings	Present	Absent

Nonsurgical

1. NSAIDS may temporarily and partially alleviate pain.

2. Epidural steroid injections can also be used to provide temporary relief of radicular symptoms in select patients. In a prospective study of 34 patients [*Am J Phys Med Rehabil* 2002;81(12):898-905.] with radicular pain used by degenerative lumbar spinal stenosis. Botwin reported that 75% of patients noted at least a >50% reduction in pain scores, 64% had improved walking tolerance, and 57% had improved standing tolerance at 1-year.

Surgical

Indication

1. Intractable pain,

2. Altered quality of life, or substantially diminished functional capacity

3. Significant Neurogenic claudication,

Compression may be isolated or may extend to multiple levels and can involve the central or lateral nerve roots, those in the foramen, or a combination of all three. Radicular symptoms or motor weakness along specific nerve root distributions require careful assessment of the lateral recess and foramen.

How to decide level of decompression

1. Clinical assessment – Nerve root and confirm with imaging
2. Function diagnosis: Root block, EMG and SNAP
3. No need to decompress each every level shown in the MRI or decompress all level.
Decompress the adjacent area.

Simple spinal stenosis:

Stenosis without radiographic evidence of instability Or with less than grade I degenerative spondylolisthesis Or less than 20 of degenerative scoliosis or no previous surgery.
These patients may be treated with decompression surgery only.

Complex spinal stenosis,

>grade I, or degenerative scoliosis with curve exceeding 20, post laminectomy junctional stenosis.

These cases often need decompression and fusion with or without instrumentation. When fusion is indicated, an instrumentation is better. However, no correlation with clinical outcome and pseudarthrosis.

Fraser 30% transition syndrome at 10 years.

Laminectomy

After resection of the spinous processes and superficial dorsal lamina, central decompression is performed with removal of the midline laminae and underlying ligamentum flavum. Partial medial facetectomies is performed. The exiting nerve roots, which may be decompressed using Kerrison rongeurs. The decompression is completed checking the nerve root canals.

The decompression should begin away from the area of maximal stenosis and should be carried out from caudad to cephalad direction. The lamina can be removed safely out to the most medial portion of the articular facets.

If dura, poor pulsations, or tight lateral recess, further decompression is indicated in the lateral direction, usually requiring resection of the medial portion of the superior facets.

3 mm Probe is passed. If it is tight in the recess, it means mid zone is tight and need complete excision of facet.

Extent of width and length of decompression is not clear. ie whether to decompress is determined by the investigation or clinical finding

How to tackle foramen stenosis

1. Remove inferior facet
2. Interbody fusion to increase the height
3. Immobilise the segment.

Outcome

a. Surgery

Most authors reported an outcome of 85% good to excellent results after decompression

Verbiest suggested 68% complete relief with residual symptoms.

Hansraj reported 95% good-excellent of the 103 cases

Kartz suggested 23% requiring revision surgery at 7- to 10-year

b. Surgery Vs Non-operative

More recently, prospective randomized studies have rigorously evaluated surgical management of spinal stenosis. Amundsen reported on the results of 100 patients Randomization for operative and non-operative at 4-year follow-up, excellent or fair results were observed in 9 of 18 patients in the nonsurgical group and 10 of 13 patients in the surgical group.

Poor outcome after surgery.

Female gender, compensation or litigation, negative preoperative diagnostic nerve root block, previous surgery, obesity and smoking

Minimally Invasive Surgical

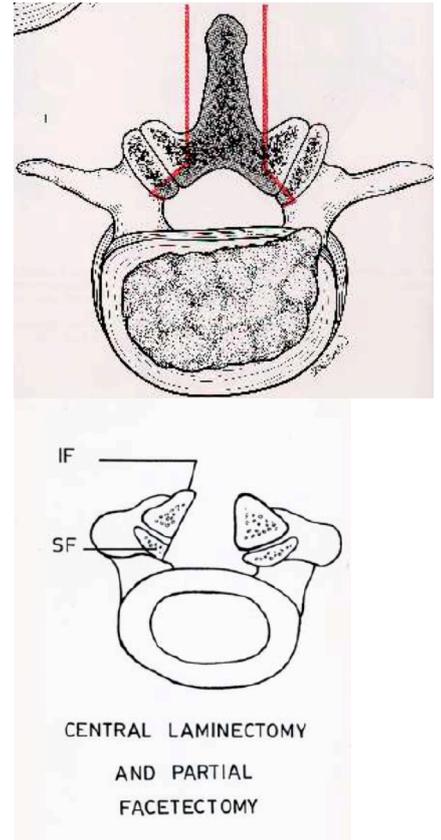
Compared with traditional open techniques, minimally invasive surgery (MIS) for management of spinal stenosis results in better preservation of posterior musculature, diminished intraoperative bleeding, and requires less recovery time. MIS requires further investigation.

Laminotomy

Laminotomy involves removal of a small portion of one side of the lamina. Resection of the distal half of the superior hemilamina is often necessary to identify and remove the insertion site of the ligamentum flavum. This technique is limited because of the difficulty in accessing stenosis in the lateral canal and foramen.

Microendoscopic Laminotomy

A drawback of this technique is that the ipsilateral lateral recess is difficult to access without



extensive facetectomy.

Interspinous Process Devices

Recently, interspinous process devices have been used to manage lumbar spinal stenosis. These implants block spinal extension at the level of the facet joint and limit canal narrowing associated with spinal extension.

In a prospective study: 91 patients were treated nonsurgically and 100 were treated with the X-STOP interspinous implant. At 2-year follow-up, symptom scores improved by 45% over baseline in patients treated with the X-STOP implant compared with 7% improvement in the control

Indication for arthrodesis

Summary

Degenerative lumbar spinal stenosis is a major cause of impaired quality of life and diminished functional capacity in the elderly.

Patients commonly present with neurogenic claudication and diminished standing and walking tolerance; however, their ability to walk distances can be increased by ambulating with the spine in a flexed-forward posture.

Back pain or lower extremity symptoms can often be elicited with lumbar extension.

MRI can best demonstrate the degree of narrowing in the central canal and lateral recess.

Surgical management is indicated following failure of nonsurgical measures.

Recent prospective randomized controlled studies have demonstrated a definite advantage of surgery over nonsurgical treatment

Decompressive laminectomy is the most common surgery

Fusion is not indicated except in the setting of concomitant spondylolisthesis, scoliosis, or iatrogenic instability.

References

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