

An Overview on Management of the Lumbar Spinal Stenosis

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Abstract:

Background: Lumbar spinal stenosis is most commonly caused by age-related degenerative changes that result in narrowing of the spinal canal and compression of the cauda equina or nerve roots. The condition is a leading cause of pain and functional limitation in adults over the age of 60. Although the natural history of LSS varies, many patients experience progressive symptoms over time. Management decisions depend on the severity of symptoms, neurological findings, and patient comorbidities, with the goal of improving function and quality of life while minimizing risks.

Keywords: Lumbar spinal stenosis, neurogenic claudication, conservative management, decompressive laminectomy, minimally invasive surgery, spinal canal narrowing.

Introduction:

Lumbar spinal stenosis (LSS) is a clinical syndrome resulting from a reduction in the space available for the neural and vascular elements in the lumbar spine, most often due to degenerative changes. It is a leading cause of pain, functional impairment, and reduced mobility in the elderly population (1).

The pathophysiology of LSS typically involves a combination of degenerative disc disease, facet joint hypertrophy, and thickening of the ligamentum flavum, which together reduce the cross-sectional area of the spinal canal. These changes can lead to compression of the cauda equina and nerve roots, resulting in neurogenic claudication, back pain, and radicular symptoms (2).

Diagnosis is based on a combination of clinical history, physical examination, and imaging studies such as MRI, which remains the gold standard for confirming spinal canal narrowing and identifying the underlying anatomical causes (3).

Management of LSS follows a stepwise approach, starting with conservative treatments such as physical therapy, nonsteroidal anti-inflammatory drugs, and epidural steroid injections. Surgical decompression, including open laminectomy or minimally invasive techniques, is indicated for patients with persistent or progressive symptoms despite adequate conservative management (4).

Conservative treatment versus surgical decompression:

It can be stated as a rule that only symptomatic patients should be treated. A “pathological” radiological finding is not, in itself, an indication for treatment. Direct comparisons of the indications of conservative and surgical treatment are further complicated by the fact that patients with mild stenosis generally undergo the former, while those with severe stenosis generally undergo the latter (5). Chou et al., in a review of this topic, concluded that moderately good evidence indicates the superiority of surgical over conservative treatment in the first two years of symptoms (6).

Atlas et al. prospectively followed patients for eight to ten years and found better results in the first four years in the patients who had undergone surgery. At the end of the follow-up period, however, the two

groups no longer differed with respect to low back pain or overall satisfaction, while all patients had a marked reduction of the leg-pain component (7).

A functionally disabling neurological deficit is an indication of surgical treatment. The indication is urgent if the deficit is acute and severe, particularly if there is a disturbance of bladder and/or bowel function (5).



Conservative treatment:

Conservative treatment is mainly aimed at alleviating the major clinical manifestation of degenerative instability and may, in fact, bring some patients long lasting relief. There has been criticism of the long-term use of non-steroidal anti-inflammatory drugs and muscle relaxants, the use of steroids and antidepressants, and the use of long-acting opioids. The main pillars of conservative treatment, above all in older people, are intensive physiotherapy with muscle-relaxing techniques in the acute phase, and strengthening of the back muscles in the later course to preserve function and mobility. Local injections (facet infiltrations, epidural injections of local anesthetic and/or steroids) may be helpful in individual cases but are not supported by any hard scientific evidence, even though multiple randomized trials have been performed (5).

Hormone calcitonin has been shown to alleviate pain and improve performance in patients with lumbar spinal stenosis. Calcitonin is known to have an analgesic effect, although its mechanism of action is still unknown. It is thought that calcitonin acts by raising the level of endogenous opioid metabolites (beta-endorphin) in blood, thus relieving pain (8).

Epidural steroid injection may provide some modest relief of inflammation related to radicular pain. In addition to their anti-inflammatory effects, steroids may inhibit pain via their ability to suppress ectopic discharges from injured nerve fibers and depress conduction in normal unmyelinated C fibers (9).

Prostaglandin E1 (PGE 1) is a potent vasodilator and inhibitor of platelet aggregation and is of interest as a therapeutic agent for LSS with intermittent claudication. The prostaglandin E1 derivative, limaprost alfadex, improves leg pain, leg numbness, and intermittent claudication in patients with LSS. However, the use of limaprost has limitations, such as the requirement for careful administration to patients with a bleeding tendency and patients receiving antiplatelet agents, thrombolytic agents, or anticoagulants, making it difficult to use limaprost in such patients (10).



Surgical Management (decompression):

The goal of surgery is to decompress the nerve roots, dura mater, and vessels without impairing segmental stability. It must first be determined whether the patient is suffering from spinal stenosis alone or from segmental instability in addition (11).

Since Mixter and Barr developed laminectomy in 1934, the procedure has been widely used in the treatment of lumbar stenosis. Laminectomy decompression is effective although associated with significant blood loss, postoperative wound pain, prolonged hospital stay and impaired lumbar stability requiring fusion or stabilization. Modification to the original technique to reduce the morbidity without affecting its effectiveness include less invasive surgery such as partial laminectomy or bilateral laminotomy decompression, foraminotomy with medial facetectomy as well as microdiscectomy (12).

Indications for surgery: (13):

- Moderate to severe claudication symptoms.
- Significant interference with lifestyle.
- Progressive neurological deficits (rare).
- Cauda equina syndrome (very rare).

With the exception of a cauda equina syndrome or progressive neurologic deficits, the indication for surgery remains relative and is dominated by the subjective interference with the patients' quality of life (13).

Types of surgery:

A.Laminectomy (Figure 1):

The objective of decompression is to create more space for the cauda equina and nerve roots by liberating the neural structures from compressing soft tissues (disc herniation, hypertrophied flavum, thickened facet joint capsules) and osseous structures (hypertrophied facet joints, osteophytes). Until the last decade, total laminectomy was the standard method of decompression in central spinal stenosis. However, the recognition that total laminectomy may increase or cause segmental instability has led to a more conservative approach, preserving the lamina and only removing those parts which actually cause the stenosis (14).

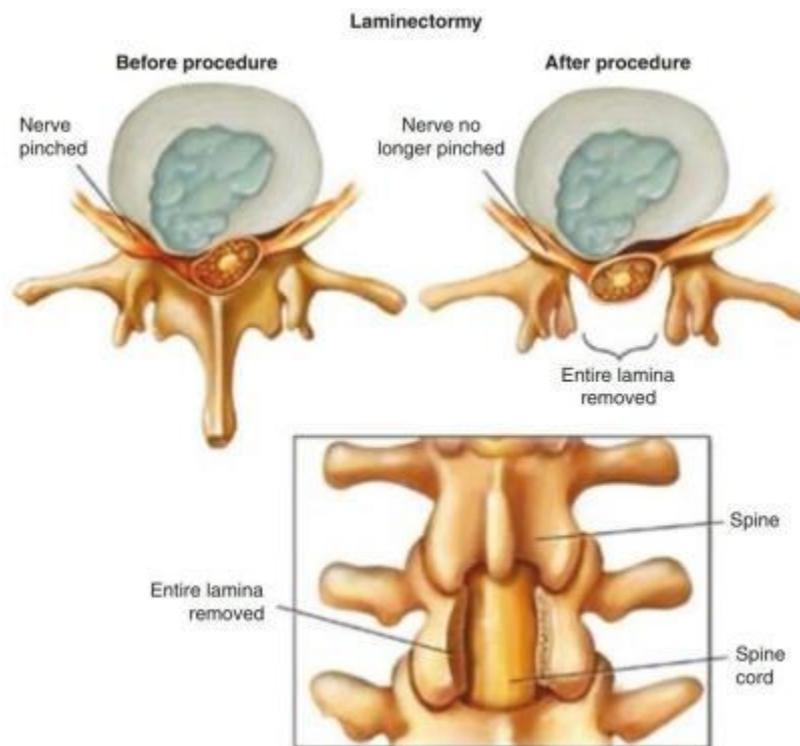


Figure (1): Extent of decompression in wide laminectomy procedure (15).

B.Fenestration "Laminotomy" (Figure 2)

The Fenestration "Laminotomy" Procedure means to make an opening in the lamina while preserving the interspinous and supraspinous ligaments. On the other hand, the ligamentum flavum is to be excised. This allows the surgeon to decompress the spinal canal & perform discectomy whenever indicated. The bone of the lower feature of the cephalad lamina and, to a small extent, from the superior feature of the inferior lamina should be resected, and following flavectomy is done to expose the canal. The medial feature of the facet joint can be trimmed to expand the lateral recess whenever indicated as in cases of lateral recess stenosis (16).

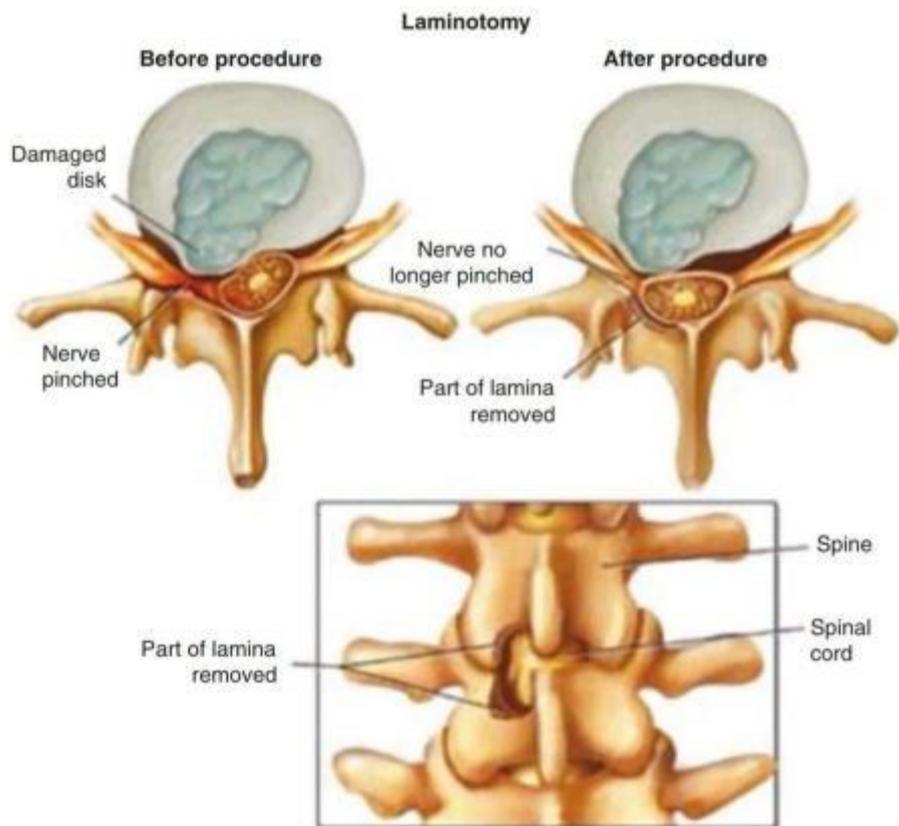


Figure (2): Extent of decompression in laminotomy procedure (15).

C. Minimally invasive surgery (MIS)

Minimally invasive surgery (MIS) methods have rapidly developed to minimize damage to the adjacent structures while still providing the same clinical outcomes. Microscopic decompression has become one of the most common procedures offered, as it reduces blood loss and postoperative pain, aids in early ambulation, and leads to shorter hospital stays. Nonetheless, microscopic approaches also have pitfalls, as paravertebral muscles still need to be disrupted to some degree, and there is a limited intraoperative view (17).

More recently, uniportal and biportal endoscopic decompressive approaches have been reported in the literature, with safe and outstanding clinical outcomes. One of the primary benefits of endoscopic methods is reducing even further the disruption of the surrounding soft tissue and providing direct visualization of the pathologic process. The uniportal full- endoscopic system involves a single portal containing the optical device and a working channel. Meanwhile, the biportal endoscopic system includes separate endoscopic viewing and working channels (17).

D. Instrumentation in addition to decompression:

Lumbar spinal fusion was introduced approximately 70 years ago and has evolved as a treatment option for symptomatic spinal instability, spinal stenosis, spondylolisthesis, and degenerative scoliosis. Broader applications including use as a treatment of chronic low back pain and recurrent radiculopathy have resulted in a dramatic increase in the rates of lumbar fusion procedures within the last decade. Lumbar spinal fusion is often performed after a posterior decompressive procedure when there is evidence of preoperative lumbar spinal deformity or instability that could worsen after laminectomy alone (18). White and Panjabi defined criteria for diagnosing instability from flexion-extension radiographs as:

- Sagittal plane translation greater than 4.5 mm or greater than 15% of the vertebral body width

- Sagittal plane rotation of greater than 15° at L1/L2, L2/L3 or L3/L4, greater than 20° at L4/L5, or greater than 25° at L5/S1 (19).

Pedicular screw insertion:

General principles of the insertion of the pedicular screw include the identification of the insertion point (by intersection technique which involves the intersection point of the intertransverse line and a line through the lateral edge of the facet joint), creation of a hole in the outer cortex by a microdrill or a pedicle owl, deepening of the hole into the pedicle and vertebral body by a pedicle probe, examination of the hole walls by a ball-tipped feeler, and insertion of the screw. Fluoroscopy and computed tomography (CT) scan are common modalities used intra-operatively to confirm the correct entry point and trajectory of the screw and reduce the complications caused by the malposition of the screws (20).

Complications of surgery:

◆ **Dural tear:**

Dural tears are not uncommon complications in spine surgery, with an incidence varying from 1.6% to 10%. Although primary repair of a dural tear is generally satisfactory, persistent CSF leakage resulting from incomplete closure can lead to complications such as postural headache, nausea, vomiting, dural cutaneous fistula formation, meningitis and even intracranial hemorrhage. When feasible, the dural tear should be repaired with braided nylon suture, monofilament polypropylene suture and Gore-Tex suture. Larger defects that cannot be closed primarily may require a dural graft such as fat, muscle, or a synthetic substitute. Ventral tears are more challenging as they cannot be directly visualized or primarily closed. Whenever primary closure is not possible, the use of static agents such as fibrin glue may be necessary (21).

◆ **Retroperitoneal structures injury:**

A much more serious complication of the lumbar spine decompression surgery is violation of the anterior longitudinal ligament with injury to the retroperitoneal structures during discectomy. This is usually done during overly aggressive discectomy with a pituitary rongeur. Unexplained egress of irrigation from the disc space should alert the surgeon to a ventral annular tear. It should be noted that the depth of the intervertebral disc averages 3 cm but can be quite varied. Depending on the level of approach, the aorta, inferior vena cava, iliac arteries and veins, abdominal viscera, or ureters may be at risk (22).

◆ **Failed back surgery syndrome:**

Failed back surgery syndrome (FBSS) is a generalized term that is often used to describe the condition where lower back pain persists or appears after spine surgery. The pain may start immediately after surgery or a few weeks or months following the surgery. Although the etiology of FBSS is not clearly understood, several reports are in agreement that its origin is multifactorial and that the causative factors may be categorized into preoperative, operative, and postoperative factors:

✓ **Preoperative:**

- Patient-related factors: psychological, social
- Surgery-related factors: poor candidate selection, revision surgery, improper planning.

✓ **Operative:**

- Inadequate decompression of lateral recesses and foramina
- Instability with excessive decompression
- Incorrect level surgery

✓ **Postoperative:**

- Recurrent disc herniation
- Adjacent segment disease
- Pelvic incidence and lumbar lordosis mismatch
- Battered root syndrome
- Nerve root entrapment syndrome **(23)**.

◆ **Surgical site infection (SSI):**

Postoperative surgical site infection following lumbar decompression is associated with a range of adverse outcomes, including pseudarthrosis, chronic pain, failure of wound healing, sepsis, and neurologic injury. The rate of mortality associated with spinal surgical site infections (SSI) is 0.8-2.3%, relative to a baseline of 0.09% in patients without an SSI. Risk factors include diabetes mellitus, smoking, obesity, and chronic kidney disease. Intraoperative risk factors are prolonged operative time or retraction use and excessive tissue destruction **(24)**.

Wound drainage that persists beyond 10 days after surgery is suggestive of SSI. Pain, hyperemia surrounding the wound, and fever are also suggestive of SSI although nonspecific. If SSI is suspected, serum inflammatory markers can be assessed. Leukocytosis is common after surgery and the presence of leukocytosis alone is not suggestive of SSI. C-reactive protein (CRP) is the most sensitive marker for infection. CRP levels should peak at post-operative day 3 and normalize by week 2. A “second peak” of CRP is suggestive of SSI. MRI with gadolinium infusion is the most sensitive imaging study in the diagnosis of SSI. Factors consistent with infection include marrow signal change (hyperintense on T2 and hypointense on T1), rim enhancement of fluid collection, disc involvement, fluid surrounding the pedicle screw heads, and early osseous destruction. CT can also be useful to identify osseous destruction and lucency surrounding instrumentation **(24)**.

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