COMPARATIVE STUDY OF FUNCTIONAL OUTCOME ANALYSIS AND EXTENT OF PARASPINAL MUSCLE DAMAGE BETWEEN LUMBAR SPINOUS PROCESS SPLITTING DECOMPRESSION AND CONVENTIONAL MIDLINE DECOMPRESSION FOR LUMBAR CANAL STENOSIS

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

in partial fulfillment of the regulation for the award of

M.S. DEGREE IN ORTHOPAEDIC SURGERY

BRANCH II

TIRUNELVELI MEDICAL COLLEGE

THE TAMILNADU Dr. M. G. R. MEDICAL UNIVERSITY

CHENNAI – 600 032

APRIL 2015

CERTIFICATE

This is to certify that the work entitled **"COMPARATIVE STUDY OF FUNCTIONAL OUTCOME ANALYSIS AND EXTENT OF PARASPINAL MUSCLE DAMAGE BETWEEN LUMBAR SPINOUS PROCESS SPLITTING DECOMPRESSION AND CONVENTIONAL MIDLINE DECOMPRESSION FOR LUMBAR CANAL STENOSIS"** which is being submitted for M.S Orthopaedics, is a bonafide work of **Dr.S.NALLA KUMAR,** Post Graduate student in the department of Orthopaedics, Tirunelveli Medical College, Tirunelveli.

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Tirunelveli Medical College,
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He has completed the necessary period of stay in the Department and has fulfilled the conditions required for submission of this thesis according to the university regulations. The study was undertaken by the candidate himself and observations recorded have been periodically checked by us.

Recommended and forwarded

Prof. ELANGOVAN CHELLAPPA,
Prof. & HOD, Department of Orthopaedics,
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DECLARATION

I solemnly declare that the dissertation titled **"COMPARATIVE STUDY OF FUNCTIONAL OUTCOME ANALYSIS AND EXTENT OF PARASPINAL MUSCLE DAMAGE BETWEEN LUMBAR SPINOUS PROCESS SPLITTING DECOMPRESSION AND CONVENTIONAL MIDLINE DECOMPRESSION FOR LUMBAR CANAL STENOSIS"** was done by me from JUNE 2013 onwards under guidance and supervision of **Prof. ELANGOVAN CHELLAPPA, M.S Ortho., D.Ortho.**

This dissertation is submitted to the **Tamilnadu Dr. MGR Medical University , Chennai** towards the partial fulfillment of the requirement for the award of **M.S. Degree in Orthopaedics (Branch II)** to be held in April 2015.

Place:

Date: **Dr. S. NALLA KUMAR**

ACKNOWLEDGEMENT

The most pleasant part of writing a thesis is acknowledging one's gratitude to all those who have helped in its completion.

It is my proud privilege to express my sincere thanks to **Dr. L.D. Thulasiraman, M.S.ortho**, Dean, Tirunelveli Medical College for permitting me to utilize the clinical materials of this hospital.

I take this opportunity to express my deep sense of gratitude, although I find words inadequate to express the greatness of my teacher **Prof. Elangovan Chellappa, M.S. Ortho., D.Ortho**., Professor and Head of the Department of Orthopaedics, Tirunelveli Medical College for permitting me to use all the clinical materials and for his valuable advice and encouragement in conducting the study. He has been a pillar of discipline, courage and immense kindness and who was instrumental in guiding me throughout the course of this thesis. I consider myself fortunate and privileged to work under his affectionate guidance, excellent supervision and sustained support.

I express my sincere and hearty gratitude to **Prof. N. Manikandan., M.S. Ortho** for his constant advice and guidance provided throughout this study.

My sincere thanks and gratitude to **Prof.Thanigaimani, M.S.Ortho: Prof. Thannappan., M.S.Ortho**., for his excellent support and advice.

My profound and immense thanks to **Dr .S. Mageswaran** , Asst. Prof of Orthopaedics, who has been my guide, provided constant advice and excellent support for the successful completion of this study.

I humbly acknowledge and express my sincere thanks to **Dr.M.SenthilKumar, Dr. M.S. Abul Kasim, Dr .A. Suresh Kumar**, **Dr. R. Sundarapandian** , **Dr. Arokia amalan** for their excellent encouragement in the completion of this study.

My sincere thanks **to Dr.V. Chandrasekar, Dr. Abraham James** and **Dr. Babu aloy**, for their suggestion and help during my study.

I thank my colleagues; CRRI's and Staff nurses who have been a source of constant help. I am very much indebted to **all my patients** who lent themselves for this study.

TABLE OF CONTENTS

INTRODUCTION

Lumbar spinal canal stenosis is a clinical syndrome of back or leg pain with characteristic provocative and palliative features, which occurs due to narrowing of spinal canal, nerve root canal and the intervertebral foramen. Lumbar spinal canal stenosis has been regarded as “the forgotten spinal disease” for more than 100 years. This neglect occurred because of the association between herniated intervertebral discs and sciatica received most of the attention after it was discovered by Mixter and Barr (1) in 1934. However, Lumbar spinal canal stenosis was not widely understood until Verbiest (2) in 1954 described the classic finding of this syndrome. It occurs in middle aged and older adults with back pain and lower extremity pain precipitated by standing and walking and aggravated by hyperextension. The secondary degenerative changes that further narrow the lumbar spinal canal precipitated symptoms. Lumbar spinal canal stenosis now is an accepted clinical entity. The degenerative lumbar spinal canal stenosis is due to thickening of interspinous ligament, ligamentum flavum and facet joint hypertrophy.

Lumbar spinal canal stenosis cause signs of intermittent neurogenic claudication, and it can lead to decreased quality of life. Conservative measures provide relief from symptoms for a shorter period only, but finally surgical decompression of the neurovascular structures will be needed (3).

At present, various surgical options are available. The surgical options include midline decompression by laminectomy , different kinds of unilateral and bilateral fenestrations and partial or full hemilaminectomies. Nowadays, it is not very clear which of the techniques is the most favourable and their long term results are inconclusive.

Since the patients suffering from degenerative lumbar spinal canal stenosis are elderly patients and its incidence increases considerably. And the elderly patients have associated co-morbid conditions compared to younger generatio problems regarding various surgical procedures need to be addressed.

Such choices of proedure are important because greater invasiveness associated with higher mortality, greater complications but generally similar clinical benefits use. So risk versus benefit ratio carefully weighed before choosing surgical procedure.

Standard conventional laminectomy is the commonly performed surgical treatment for degenerative lumbar canal stenosis (4,5). This method involves damage to the integrity of posterior complex of spine and elevation of paraspinal muscles from the spinous processes. Which results in paraspinal muscle atrophy, spine extensor weakness, Iatrogenic instability of spine and possibly, ”Failed back syndrome (6,7) .

Lumbar spinal stenosis decompression by spinous process splitting laminectomy method thought to avoid paraspinal muscle damage and extensor weakness by preserving muscle and ligamentous attachments to the spinous processes(8). We present the prospective randomized control study comparing the outcome of Lumbar spinous process splitting decompression and conventional midline decompression (CMD) by laminectomy in 20 patients who underwent surgery for lumbar spinal canal stenosis.

REVIEW OF LITERATURE

In the 18th century the concept of lumbar canal stenosis was describrd by portal in the year1803. And the awareness of lumbar canal stenosis bought by various authors. Finally in the year 1954 Dutch surgeon Verbiest gives the classical description of Lumbar canal stenosis , he understands the clinical significance and need for surgical treatment. After that various surgical treatment modalities evolved. Since the classical description of Lumbar canal stenosis (LCS) by Verbiest in 1954 (Fig-1), our understanding of the disease and its treatment modalities have evolved tremendously(2). In general the initial treatment of Lumbar canal stenosis is non-operative, surgical decompression remains the mainstay of treatment in patients refractory to conservative treatment methods. Standard midline decompression by conventional laminectomies are regarded as an effective surgical treatment for degenerative lumbar spinal stenosis.

Fig-1: **Henk VERBIEST(**1909–1997**)**

In a randomized controlled study by Amundsen et al, of 100 patients with symptomatic spinal canal stenosis, 19 patients with severe symptoms were treated operatively, 50 patients with moderate symptoms were treated conservatively and 31 patients were randomly separated to receive operative and non-operative treatment. Regardless the treatment pain relief was noted after 3months in most of the patients, but took 12 months in a few patients. Results in non-operatively treated patients deteriorate over time. 80% patients treated operatively still had good results at the end of 4 years(23).

Weinstein et al. showed significant improvement in patients treated operatively when compared to those treated conservatively(23).

These authors concluded that non-operative treatment is appropriate for patients with moderate pain and those who had 50% pain relief in less than 3 months duration, but operative treatment is probably indicated in whom conservative treatment fails and in patients with severe pain .

Kalbarczyk et al. performed a midline decompression by conventional laminectomy in 70% of their patients with degenerative lumbar spinal stenosis(15).

Cirak et al. concluded that in patients with degenerative lumbar spinal stenosis, decompression by laminectomy and extensive foraminotomy without instrumentation have good outcomes(16).

Turner and colleagues(18) reported only a 65% a success rate after wide laminectomies. Due local tissue trauma and postoperative spinal instability, resulting from a wide decompression, have been frequently attributed to the unsatisfying results of this technique.

It is important in treatment of degenerative lumbar spinal canal stenosis to achieve adequate decompression with maintaining the spinal integrity. The preservation of posterior ligamentous and muscle complex associated with less invasive surgery could minimize the risk of developing post operative changes in the spinal alignment and acceleration of disc and facet joint degeneration.

The best alternative would be an adequate decompression without disturbing the stability of the spine. Various methods have been developed to decrease the incidence of paraspinal muscle atrophy and preservation of posterior musculoligamentous structures.

Weiner et al described spinous process osteotomy decompression for degenerative lumbar spinal canal stenosis, where the spinous process is osteotomised at its base and retracted to opposite side with unilateral elevation of paraspinal muscle (24, 25)

Lin et al. Described chimney sublaminar decompression in which spinous process bisected with high speed burr until its junction with lamina. Further decompression done with kerroson's rongeurs(27).

Cho et al. described a technique called ‘Marmot operation’ where the spinous process splitting done and interspinous ligaments are retracted, exicision of the hypertrophied ligamentum flavum and facetal undercutting is done. Reports of safety and efficacy are lacking(28).

.

Watanabe et al. in described a new technique called spinous process splitting laminectomy. In this method spinous process burred till their base and osteotomised, followed by minimal dissection of muscles from the lamina and then laminectomy. The supraspinous and interspinous ligaments incised longitudinally are resutured later(28).

Lee et al. reported a series of 25 patients undergone spinous process splitting laminectomy. Outcome measures include VAS for pain and Oswestery disability index(ODI). At one year follow up VAS and ODI observed was 65.2% and 52.2% respectively and concluded that this procedure yielded good clinical outcome(29).

NATURAL HISTROY

Although symptoms of lumbar canal stenosis may arise from narrowing of the spinal canal, not all patients with narrowing will develop symptoms. The natural history of most forms of spinal canal stenosis is insidious development of symptoms. But , there can be an acute onset of symptoms precipitated by trauma or heavy activity. Many patients have significant radiological findings with minimal complaints or clinical findings. Johnsson, Rosén, and Udén reported 19 (70%) of 27 patients with moderate, untreated spinal stenosis (≥11 mm anteroposterior canal diameter) remained unchanged after 4 years of continuous observation; four (15%) improved, and four deteriorated. Johnsson et al. found that 11 of 19 (58%) untreated patients were remain unchanged at 31-month follow-up, six were improved, and only two went worse. In a prospective study design to compare operative and conservative treatment of stenosis, Atlas et al. found that 50% of patients treated conservatively reported improved back and leg pain after 8 to 10 years.

In a prospective, randomized controlled study by Amundsen et al. of 100 patients with symptomatic spinal canal stenosis, 19 patients with severe symptoms were treated surgically, 50 patients with moderate symptoms were treated conservatively, and 31 patients were randomly assigned to receive conservative (18) or operative (13) treatment. Pain relief was noted after 3 months in most patients regardless the treatment, but it took 12 months in few

patients. Results in non operatively treated patients deteriorated over time, at 4 years were excellent or fair in 50% of patients treated nonoperatively; 80% of patients treated operatively had good results. Results were not worse if surgery was done 3 years after failed conservative line of management, and significant deterioration did not occur during the 6 years of follow-up in any of the three groups. These authors concluded that conservative treatment is appropriate for patients with moderate pain, but operative treatment is indicated for patients with severe pain and patients in whom conservative treatment fails(22).

Reported studies suggest that most patients with spinal stenosis, a stable course can be predicted, with 15% to 50% showing some improvement with conservative treatment. Worsening of symptoms despite conservative treatment is an indication for surgery.

DEFINITION:

Lumbar Canal Stenosis is defined as narrowing of the lumbar spinal canal, nerve root canal or intervertebral foramina with decrease in the cross sectional area of dural sac <75 mm^2 or < 10mm in sagittal plane of spinal cord and causing clinical symptoms of pain in the back, buttock and leg with provocative and palliative features(8).

APPLIED ANATOMY:

The lumbar spine is consists of five vertebra. Each vertebra is made up of vertebral body, pedicles, superior and inferior facets, pars interarticularis, laminae, spinous process and the transverse process.

JOINTS:

Each vertebra has three joints between the superior and inferior vertebrae, one anterior and two posterior joints. The anterior joint or the intervertebral joint comprises of the adjacent surface of the vertebral bodies along with the intervertebral disc. The posterior joints are the facet joints. The facet joints are the synovial joints between superior facet of the lower vertebra and inferior facet of the upper vertebra. The superior facet is anterolaterally situated as compared to the inferior facet of the upper vertebra. They are concave and convex reciprocally. so it permits some rotation movements in addition to flexion and extension.

THE BONY LUMBAR CANAL:

The conus medullaris ends at the level of lower border of L1 vertebra. Beyond which the dural sheath contains only the cauda equine.three basic shapes in the lumbar spinal canal round,trefoil and oval(fig-2). The shape of bony lumbar canal varies from L1 to L5. At the level of L1 it is almost round. At L5 level it is trifoliate. The well developed lateral recesses is due to this transformation at L4 & L5 vertebrae. Any pathology in the Lateral recess can be maximally seen in these two vertebrae. The normal sagittal diameter of this canal varies from 15 to 25 mm. A canal of 20 mm is capacious and canal diameter between 12 and 15mm are suggestive of small canal and below 12 mm the canal is narrow causing spinal canal stenosis. Acquired stenosis is more at the level of L4 -L5 and L3 –L4 .

As the nerve root leaves the dural sac, it passes through the lateral recess or nerve root canal. Each nerve root is intimately related to the medial and the inferior aspect of the corresponding pedicle.

It is necessary to distinguish between the terms spinal canal, nerve canal and intervertebral foramen.

THE NERVE CANAL:

The spinal nerve root leaves the dural sac through the lateral part of spinal canal by an oblique passage called nerve canal. The nerve canal ends where the nerve root emerges from the intervertebral foramen.

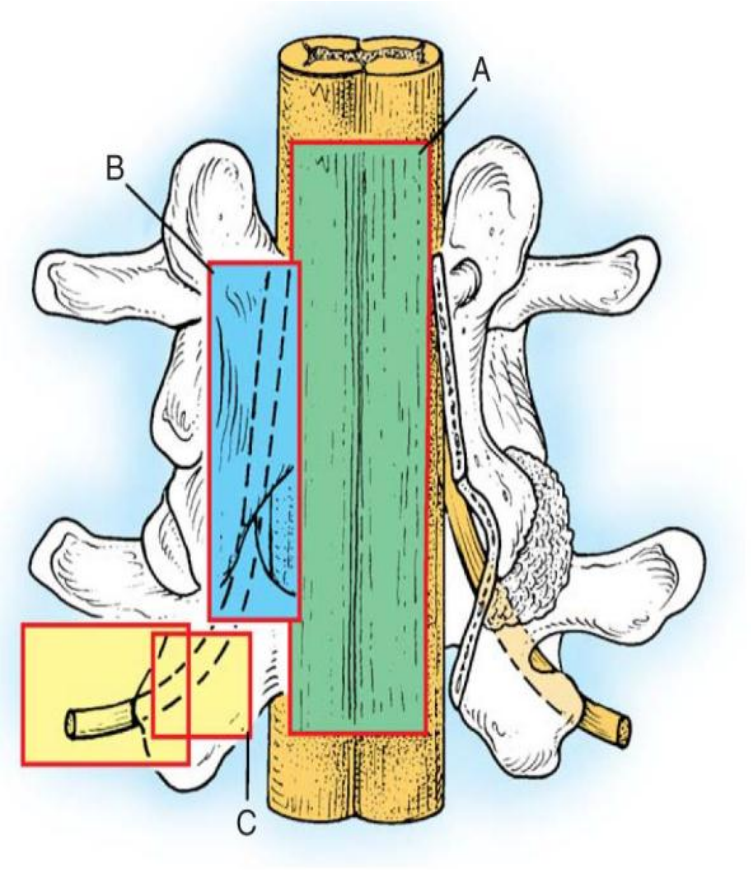


Fig-2: Lumbar canal

A – Bony lumbar canal

B – Nerve canal

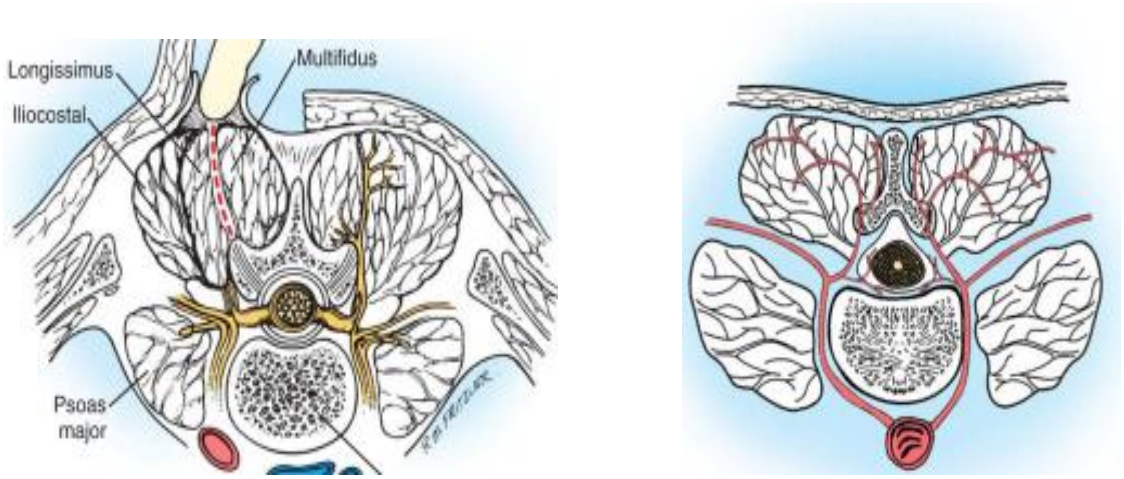
C – Intervertebral foramen

PARASPINAL MUSCLES:

On either side of the spinous process is the convex column of muscle known as paraspinal muscles, which are collectively called as erector spinae or sacrospinalis(fig-3). It consists of three muscles namely multifidus, longissimus and iliocostalis muscles. The main function of sacrospinalis muscle is to maintain the spine erect . Subperiosteal resection of the paraspinal muscles should be carried out in order to maintain the blood supply and its musculature to be handled to the minimum.

Lazzennec had done extensive MRI studies on cross section of spinal musculature after surgery and has demonstrated weakening in the muscles following fibrosis due to operative procedures(32).

Fig -3: Paraspinal muscles



LIGAMENTS:

A) LIGAMENTUM FLAVUNM

These are strong, yellow, elastic ligaments which unites the adjacent lamina(fig-4). They are short and limited on either side by articular process. Along with the lamina it forms the smooth posterior surface. With aging these ligaments losses its elastic nature and the collagen hypertrophies which buckle and encroach the thecal sac and cause spinal canal stenosis

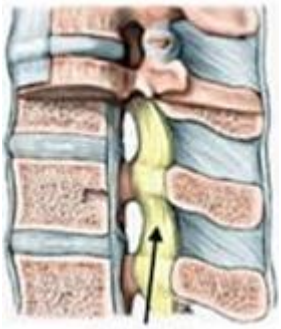


Fig-4: Ligamentum flavum

B) INTERSPINOUS LIGAMENTS

A thin ligament passes posteriorly from ligamentum flavum and unites with the posterior margin of spine and it continuous posteriorly with a strong supraspinous ligaments.this ligament attached to the spine throughout the length of the length of thoracic and lumbar vertebral coloumn. This ligaments are sufficiently elastic to allow flexion movement of the spine..

C) ANTERIOR LONGITUDINAL LIGAMENT

It stretches from atlas to sacrum. It firmly attaches to the vertebral bodies and intervertebral disc.the lateral margin fades into the periosteum.

D) POSTERIOR LONGITUDINAL LIGAMENT

It lies within the vertebral canal. It attaches to the posterior margin of intervertebral disc and adjacent margins of vertebra. The ligaments are narrowed to allow basivertebral veins (Fig-5).

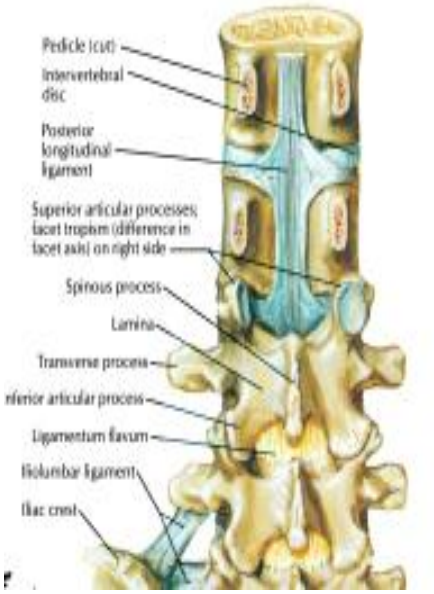


Fig-5: posterior longitudinal ligament

The pathology of Lumbar canal stenosis can be caused by dynamic as well as a structural component. Degeneration of the intervertebral disc occurs with narrowing of disc space and subsequent ligamentous redundancy which compromises spinal canal. Instability may occur. This relative hypermobility leads to the formation of facet overgrowth and ligamentous hypertrophy. The ligamentum flavum may be markedly thickened into the lateral recess where it attaches to facet capsule causing nerve root compression.

Spinal canal stenosis in this region usually caused by protruded disc, bulging annulus, osteophyte, thickened or buckled ligamentum flavaum.resulting in narrowing of both the central and lateral canals. This can occur alone or in combination to create the symptom complex characteristic of spinal stenosis.

The earliest change in the facet joint is probably synovitis. Later cartilage destruction and capsular laxity lead on to instability and subluxation. Eventually, osteophytes formation and enlargement of articular process in order to provide stability, which results in encroachment of central and lateral canals. Tropism or proliferative osteoarthritis of facet joint is due to abnormal and excessive external stimulation which is related to too much of static stress on the facets from an abnormally immobile motion segment. The superior facets are enlarged usually and may nearly bisect the spinal canal horizontally with inner border almost approaching the midline. Loss of disc space height results

from internal disruption and disc resorption. Later osteophytes form at the back of the vertebral bodies. Disc may also losses its height as a result of infection, excision or herniation.

The combination of changes in the inter vertebral disc and posterior joints results in subluxation of the facetal joints , as a result the superior articular process moves upward and forward to encroach on the nerve canal. The nerve canal also narrowed by bulging annulus, osteophytes at the back of the vertebra, and subluxated superior articular process. Central spinal stenosis results from enlargement of inferior articular process.

Junghan's in 1932 introduced concept of motion segment.

THE MOTION SEGMENT

Intervertebral disc

Intervertebral foramen

Superior and inferior facets

Interlaminar space

Ligamentum flavum

Inter and supraspinous ligaments

Any change in the intervertebral disc height produce change in the whole motion segment.

Motion segment should be preserved to a maximum extent in any operative procedure.

The changes in the disc space are not the only factors that would change the function of the motion segment. The effect of each component in the motion segment can be influenced by the other. If the supraspinous or interspinous ligament is removed, then there is an increased range of motion in flexion and to a lesser extent in extension of the lumbar spine. This would produce alteration of the loading point.

This concept is utilised in decompression by spinous process splitting. The spinous process and associated ligaments are maintained intact and only the pathology causing lateral recess stenosis is tackled. The venous congestion and hypertension likely to be responsible for symptom complex known as intermittent claudication.

CLASSIFICATION:

Based on etiology and anatomic location.

Van Akkerveeken has classified canal stenosis(12).

Primary stenosis:

It is rare, it is approximately 9%, due to congenital malformations or developemental defects(achondroplasia).

Secondary stenosis:

Mainly due to acquired conditions.

1.Degenerative

Central canal

Lateral recess, foramen

Degenerative spondylolisthesis

Degenerative scoliosis

2.Iatrogenic

Postlaminectomy

Postfusion

Spondylolytic

Posttraumatic

3.MISCELLANEOUS

Paget’s disease

Fluorosis

Diffuse idiopathic skeletal hyperostosis(DISH)

Hyperostotic lumbar spinal stenosis(Forestier disease)

Pseudogout

Oxalosis

DEGENERATIVE SPINAL STENOSIS:

The most common type of type of spinal canal stenosis is degenerative arthritis of spine. The disc degenerates and losses its elasticity and height, the annulus bulges into the canal. Similarly the vertebral body and facet approach each other with the formation of osteophytes at the margin. The nerve root emerges through the intervertebral foramen got caught between the facet and pedicle(Fig-6). The degenerative process mostly localised to the facet joints and ligamentum flavum.

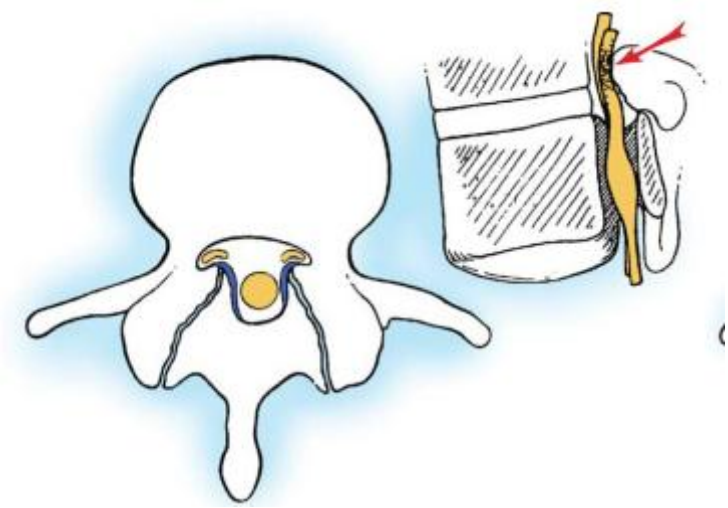


Fig-6: DEGENERATIVE SPINAL STENOSIS

CONGENITAL OR DEVELOPMENTAL STENOSIS:

There is uniform narrowing of the canal, usually it is central canal stenosis. Idiopathic congenital narrowing will have decreased AP diameter due to short pedicles. In achondroplasia, the neural canal got compressed due to diminish in the interpedicular distance.

IATROGENIC STENOSIS:

In iatrogenic stenosis the mechanism is unclear the probable reasons are,

- Incomplete treatment of stenosis
- Hypertrophy of posterior bone graft
- Infolding of ligamentum flavum just superior to the fusion mass
- Epidural scar formation.

ANATOMIC CLASSIFICATION:

Central spinal stenosis:

It denotes the involvement of area between the facet joints, which is occupied by the dura & its contents.

Causes:

1. Protrusion of disc
2. Bulging annulus
3. Osteophytes
4. Buckled or thickened ligamentum flavum.

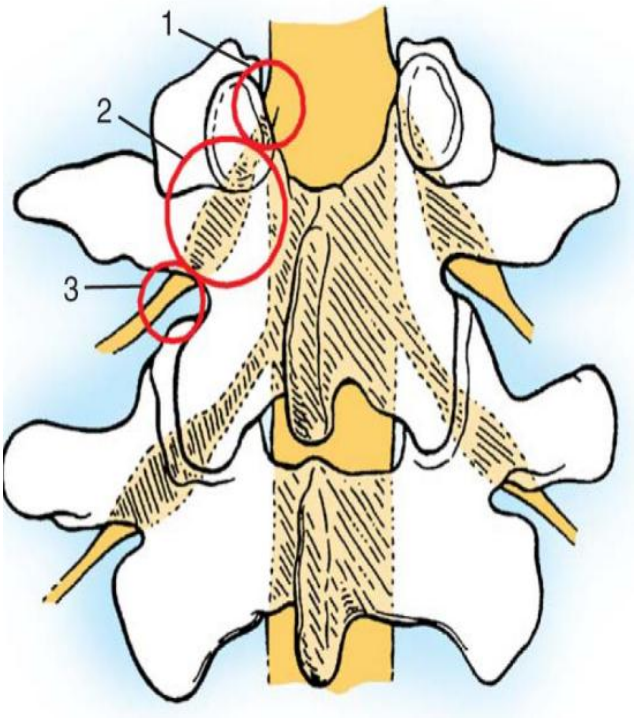
Most common symptom: neurogenic claudication

Lateral canal stenosis:

Lateral to dural sac is the lateral canal which contains nerve roots.

Most common symptom by root compression is the radiculopathy.

Fig-7:Zones of nerve canal



Lee's entrance zone (lateral recess or lateral canal stenosis)	Lee's mid zone (foraminal stenosis)	Lee's exit zone
Borders: Medial-central canal. Lateral-pedicle. Dorsal -superior facet. Ventral - disc & posterior ligamentous complex .	**Borders**: Medial-lateral recess. Lateral-lateral border of pedicle. Ventral- Posterior vertebral body & disc. Dorsal –pars interarticularis.	Is the area lateral to the facet joint.

Causes:	**Causes:**	**Cause:**
Facet arthritis, vertebral body spur,protruded disc,etc.,	pars fracture with proliferative fibro cartilage, lateral disc herniation.	"far lateral" disc, spondylolisthesis,facet arthritis.

Table-1: Zones of nerve canal

CLINICAL PRESENTATION OF LUMBAR CANAL STENOSIS:

1. Common in older individuals.
2. Back pain predominant in 95% of the cases.
3. Leg pain approximately in 90% of the cases which maybe unilateral or bilateral.
4. Neurogenic claudication defined as poorly localized pain, numbness and cramping pain in one or both lower extremities of a neurologic origin, which is more on walking and relieved by sitting, frequently accompanies Lumbar canal stenosis.
5. Patient may complaint of numbness involving the dermatome of compressed nerve roots

6. In rare instances, patients may present as acute cauda equine syndrome with the involvement of bowel and bladder disturbance.
7. In patients with central canal stenosis, symptoms usually are bilateral which involve the buttocks and posterior thighs in a nondermatomal fashion.
8. Lateral canal stenosis, symptoms usually are dermatomal due to the compression of specific nerves.
9. Patients with lateral canal stenosis may have more pain during night and at rest, but more walking tolerance than patients with central stenosis.
10. Neurogenic claudication should be distinguished from vascular claudication,which has a different etiology and clinical features and the differences are given below

Evaluation	**Neurogenic claudication**	**Vascular claudication**
Back pain	Common	Occasional
Walking distance	Variable	Fixed
Walking uphill	Painless	Painful
Bicycle test	Negative	Positive(painful)
Palliative factors	Sitting/bending	Standing

Provocative factors	Walking/standing	Walking
Back motion	Limited	Normal
Weakness	Occasional	Rare
Pain character	Numbness,aching-proximal to distal	Cramping-distal to proximal
Skin changes	Present	Absent
Pulses	Present	Absent

Table-2: Difference between Neurogenic claudication and vascular claudication

11) The differences between the spinal canal stenosis and lumbar disc herniation are given below

	Lumbar canal stenosis	Lumbar disc herniation
Age	>50	<50
Onset	Insidious	Acute
Pain	Referred/diffuse	Radicular/dermatomal
Provocative factors	Standing/walking	Sitting
Palliative factors	Sitting	Standing
Weakness	Uncommon	Common
Sensory changes	Uncommon	Common
Tension sign	Rare	Present

Neurological findings	Rare	Present

Table-3: Differences between the spinal canal stenosis and lumbar disc herniation

CLINICAL EXAMINATION:

1. Straight-leg raise test is positive in approximately 50% of patients.
2. Symptoms are aggravated by extension and weight bearing, relieved by flexion and non weight bearing postures.
3. Associated sensory and motor deficit may be present.

In most of the cases, patients seeks medical attention when the walking distance progressively diminished due to Neurogenic claudication.

PATHOPHYSIOLOGY OF SPINAL STENOSIS:

Stage of transient dysfunction:

In the beginning the complaints of the patients are mild and not very frequent. Physical signs are subtle or even absent. Radiological investigations are normal. Conservative treatment is highly successful. Surgical intervention has no place in the management at this stage.

Stage of instability:

As the disease progresses, there is abnormal movement at the facet joints due to laxity in the ligaments. There is also abnormal motion at the level of intervertebral disc. Following subluxation the superior facet moves upwards and forwards and causes stenosis. Complaints became more severe and more frequent. X ray shows abmormality. Conservative treatment usually helps but symptoms persist. Surgical decompression and fusion is indicated.

Stage of fixed deformity:

As the result of severe degenerative changes with osteophyte formation, the instability gives rise to restricted mobility and fixed deformity with entrapment of nerve roots. Usually surgical intervention is needed.

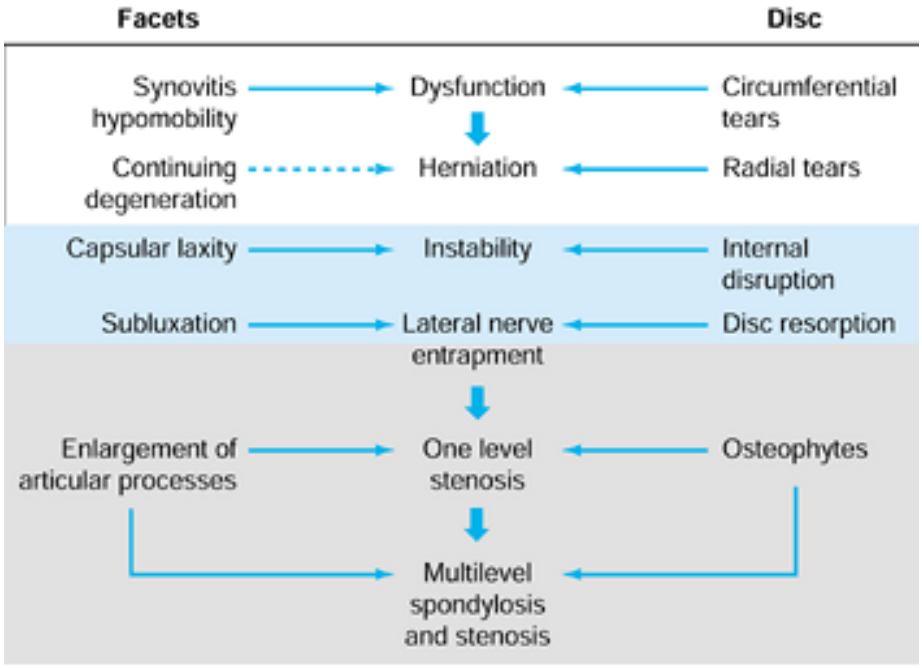


Table-4: Pathophysiology Of Spinal Stenosis

RADIOLOGICAL EVALUATION:

The imaging modalities available for diagnosing Lumbar Canal Stenosis are

a)Plain X-ray lumbo-sacral spine shows,

1. Fecetal joint hypertrophy.
2. Distance between the pedicle and the level of facet joint reduced.

3. Reduced distance between the posterior border of the vertebral body and anterior border of the superior facet.
4. Short, stout spinous process and laminae with reduced distance between the pedicles of adjoining vertebrae.
5. Associated features narrowing of intervertebral disc space, posterior osteophytes.
6. Decrease in inter laminar space.
7. Irregular and laterally arranged facets.

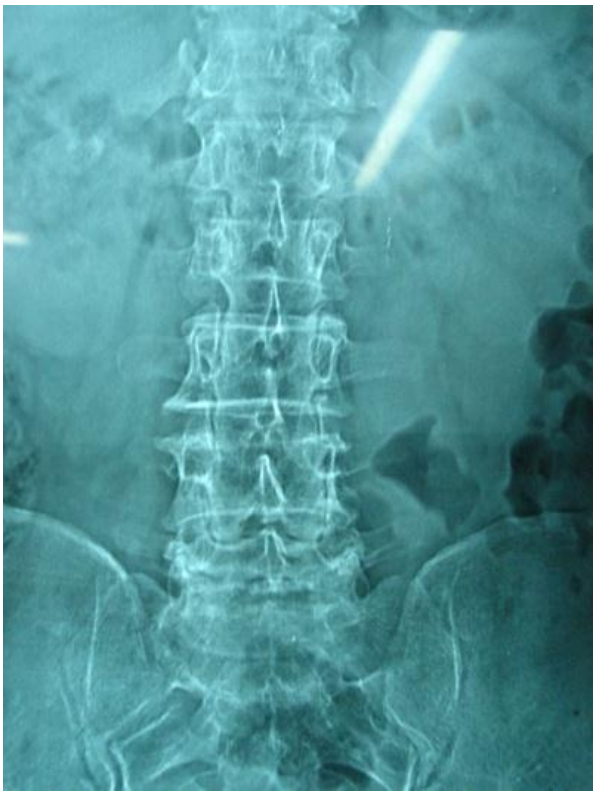


Fig. 8: Antero posterior view

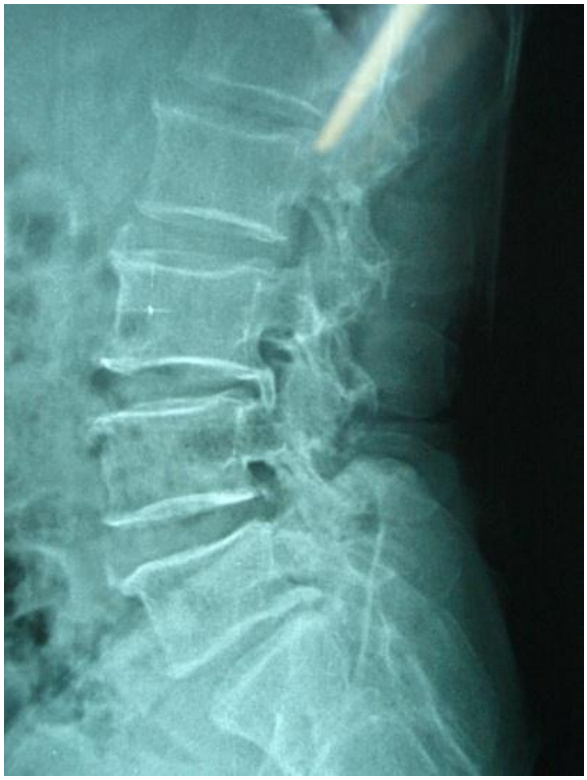


Fig.9: lateral view

MYELOGRAPHY:

Myelography was the gold standard one for diagnosis of Lumbar canal stenosis in earlier days. The anterior-posterior (AP) diameter of spinal canal on myelography was used as a reference(13).

AP diameter Normal = 15 mm

Relative stenosis = 10 to 12 mm

Spinal stenosis = < 10 mm

Advantages :

1. Visualisation of entire extent of lumbar spine

2. To know the narrowing during movement of spine.

Disadvantages:

1. Adverse effects to contrast agents.
2. Difficulty in identifying lateral stenosis pathology.

Now **Computed tomography (CT) and magnetic resonance imaging (MRI)** are the most commonly used diagnostic modality for Lumbar canal stenosis. Diagnosis of Lumbar canal stenosis using CT or MRI is based on the sagittal diameter of the spinal canal or the cross-sectional area of the dural sac.

Schonstrom et al. reported the cross-sectional area of the dural sac to be a more reliable diagnostic measure and have defined cross-sectional area of more than 100 mm^2 at the narrowest point as normal, 76 mm^2 to 100 mm^2 as moderately stenotic, and less than 76 mm^2 as severely stenotic(11,12,13).

CT CHANGES IN SPINAL STENOSIS

Herniated disc

Disc protrusion

Vacuum disc sign

Hypertrophy of posterior articular processes

Osteoarthritis of apophyseal joints

Osseous proliferations of nonarticular aspects of superior apophyseal joint

Osseous proliferations of nonarticular aspects of inferior apophyseal joint

C/O of posterior longitudinal ligament

C/O of yellow ligament

C/O of supraspinal ligament

Anterior C/O of posterior articular capsule

Posterior C/O of posterior articular capsule

Anteroposterior diameter of spinal canal

Transverse diameters of spinal canal

(C/O, calcification or ossification or both)

Advantage:

1. Central and lateral canal can be directly visualised and measured.
2. Soft tissue pathology can be identified.

MRI FEATURES:

1. Waist-like narrowing of dural tube at the level of facet joint .
2. Indentation of the dural tube by prolapsed disc.
3. Axial CT OR MRI cuts demonstrates hypertrophy.
4. Lateral recess narrowing in lateral cuts of MRI .
5. Reduced mid sagittal distance in saggittal cuts

MRI :

Fig-10: SAGITTAL SECTION

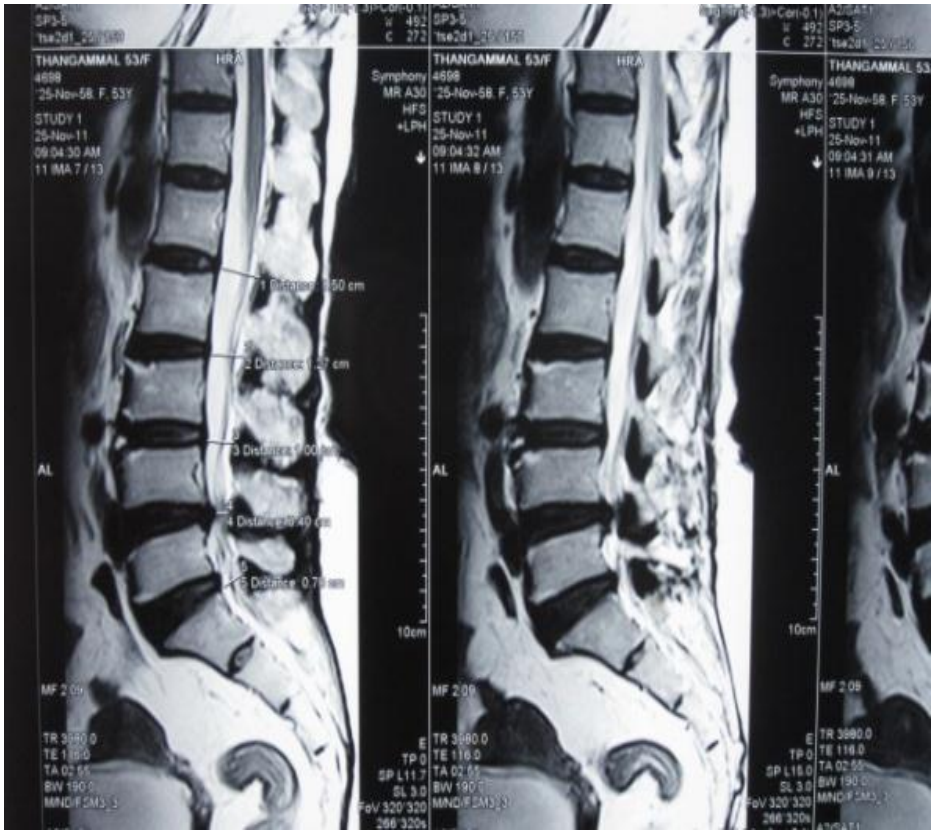
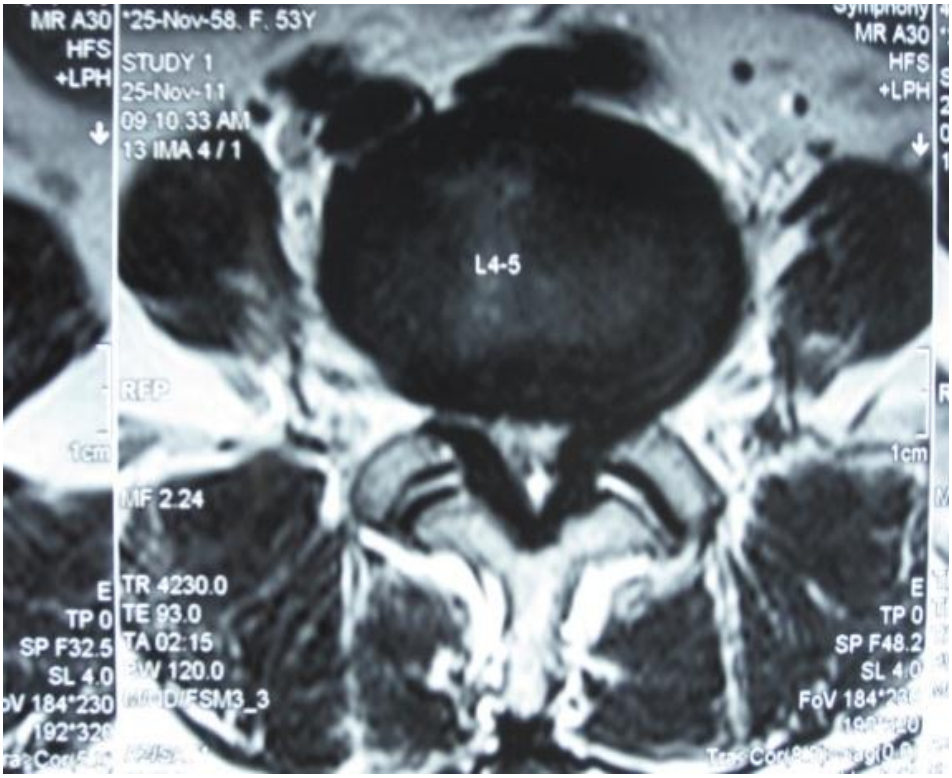


Fig-11: CORONAL SECTION



MEASUREMENT OF LATERAL RECESS (CIRIC INDEX)

Normal - 5mm

Suggestive - 3mm

Diagnostic - 2mm

OTHER DIAGNOSTIC STUDIES:

Electrodiagnostic studies should be used if the diagnosis of neuropathy is uncertain, especially in patient with diabetes mellitus.

Electromyography (EMG) and nerve conduction velocity (NCV) have been recommended as useful adjuncts to diagnosis in patients with peripheral neuropathy from Lumbar canal stenosis.

MANAGEMENT OF LUMBAR CANAL STENOSIS

NONOPERATIVE TREATMENT:

Symptoms of spinal canal stenosis usually respond well to conservative management.

Conservative measures should include

1. Bed rest not exceeding for 2 days.
2. Pain management with NSAIDS and acetaminophen.
3. Trunk stabilization with braces and exercise program along with good aerobic fitness and abdominal muscle strengthening exercise
4. Traction has no proven benefit in adult lumbar spinal stenosis.
5. Stationary cycling

Onel and colleagues proposed a program of flexion exercises and infrared heating modalities to reduce pain and spasm and improve flexibility, because of the structural narrowing of the spinal canals produced by extension.

Epidural Steroid Injection

1) Spinal canal stenosis cause mechanical compression of neural elements, which leads to structural and chemical injury to the nerve roots.
2) Edema and venous congestion of nerve roots can lead to further compression and ischemic neuritis.
3) This result in the leakage of neurotoxins, such as phospholipase and leukotriene B, which lead to increased inflammation and edema.
4) Corticosteroids are potent antiinflammatory agents, which decreases the leukocyte migration, the inhibition of cytokines and decrease edema.
5) These actions provide the rationale for the use of epidural steroid injections in spinal canal stenosis.
6) Although epidural steroid injections have been used for many years, no scientifically validated long-term outcomes have been reported to substantiate their use..

The technique of placement- Caudal, translaminar and transforaminal with fluoroscopy.

COMPLICATIONS:

Epidural hematoma, temporary paralysis, retinal hemorrhage, epidural abscess, chemical meningitis, dural puncture and headache.

Suitable candidates:

Patients with acute radicular symptoms or neurogenic claudication unresponsive to traditional analgesics and rest.

SURGICAL TREATMENT:

1. The primary indication for surgery in patients with spinal canal stenosis is increasing pain that is resistant to conservative methods.

2. Patients with severe back and leg pain with significant limitation in walking tolerance

3. Acute cauda equina syndrome

4. Rapidly deteriorating neurological deficits.

In general, surgeries give good relief of claudicatory leg pain with variable response to back pain.

Prognostic factors with better results in associated

1. Disc herniation
2. Stenosis at single level
3. Weakness < 6 weeks duration
4. Monoradiculopathy
5. Age < 65 yr

PRINCIPLES OF SPINAL STENOSIS SURGERY:

Decompression is the treatment of choice for lumbar canal stenosis. Fusion is required if excessive bony resection compromises spine stability or if isthmic or degenerative spondylosis , scoliosis or kyphosis is present.

Laminectomy is preferred in older individuals with multiple level stenosis and fenestration procedures in younger patients with intact disc especially done through a minimally invasive approach.

During surgery specific attention should be directed to symptomatic area, which may result in less extensive decompression than would normally be done with the pain source unconfirmed. If radical decompression of one root is necessary, additional stabilisation is unnecessary. The removal of more than one complete facet joint usually requires fusion with or without instrumentation. Position the patient with the abdomen hanging free to minimize bleeding. If fusion is likely, the hips should be extended to prevent positional kyphosis.

The lateral recess and foramen dissection may require a small, sharp osteotome, which allows the surgeon to thin the bone sufficiently to allow removal with angled curets. In contrast to disc surgery, decompression the lateral recess is best seen from the opposite side of the table. The operating surgeon may switch sides during the operation to view the nerve roots better. Blunt probes with increasing diameters are useful for determining adequate foraminal enlargement.

A good approach is to start with decompression at the point of lesser stenosis and work towards the area of more severe stenosis. This frees the neural structure enough to make the final decompression easier and decrease the risk of damage to duramater and the nerve root.

It is generally agreed that surgery for lumbar canal stenosis is more effective in relieving radicular symptoms and that the presence of low back pain alone is rarely an indication for surgery.

LAMINECTOMY

The gold standard surgical procedure for lumbar canal stenosis is conventional midline laminectomy. This procedure involve the removal of lamina and ligamentum flavum on both sides of the stenotic level and the lateral recess. Decompression starts from distal extent of neural compression and proceeds in a proximal direction. Perform decompression sequentially, from medial to lateral.

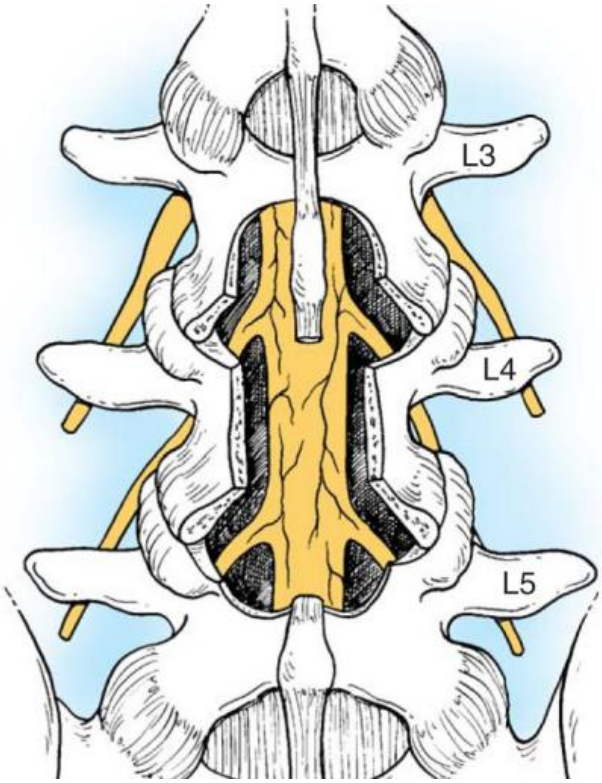


Fig-12: Laminectomy

HEMILAMINECTOMY

Hemilaminectomy involves unilateral removal of lamina and ligamentum flavum. The spinous processes, interspinous ligaments, and supraspinous ligaments are preserved. So, less risk of development of postoperative instability. Preserve the pars interarticularis laterally in order to minimize risk of postoperative instability . Hemilaminectomy is appropriate for patients with unilateral symptoms from stenosis. A disadvantage of this procedure is the difficulty of performing contralateral decompression

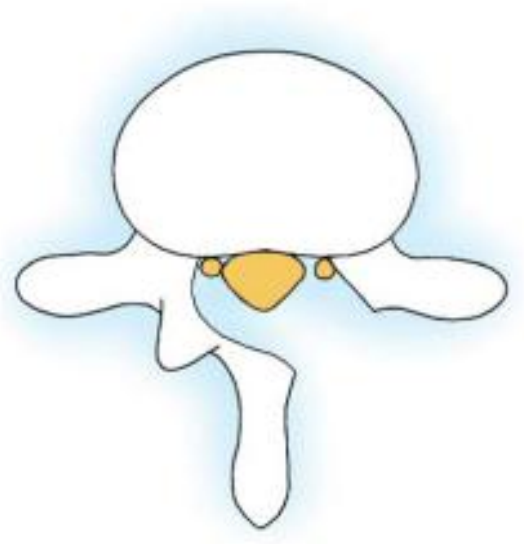


Fig-13: Hemilaminectomy

HEMILAMINOTOMY:

Hemilaminotomy involves removal of only the ligamentum flavum and adjacent portions of two hemilaminae responsible for neural compression. This procedure is more commonly performed in younger patients. Extensive laminectomy carries the risk of instability.

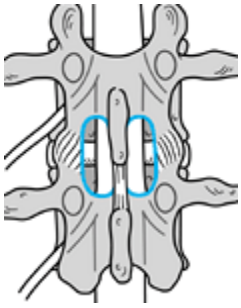


Fig-14: Hemilaminotomy

WIDE FENESTRATION:

Wide fenestration is done for central stenosis in which only the medial portion of the inferior facets and adjacent ligamentum flavum is removed . Preserve the interspinous or supraspinous ligament complex and spinous processes, which form the midline stabilizing structures.

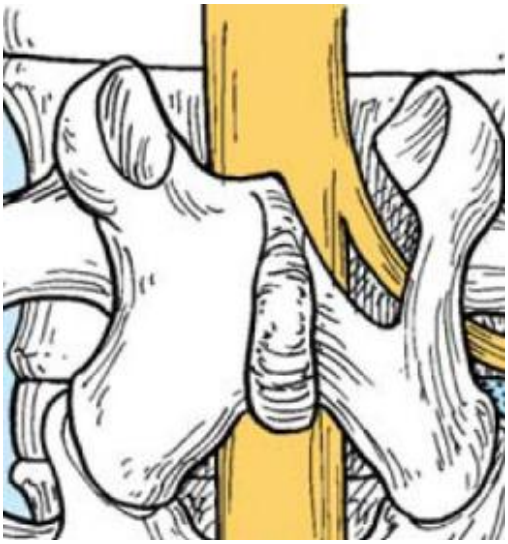


Fig-15: Fenestration

SPINOUS PROCESS OSTEOTONY DECOMPRESSION

The spinous process is ostetomised at its base and retracted to opposite side. Removal of lamina and ligamentum flavum are done. Complete laminectomy is recommended for severe stenosis or congenital stenosis involving the anatomical zones (central, lateral recess, and foraminal zones). A minimally invasive technique allows decompression of compressing anatomy, while preserving paraspinal muscles, the spinous processes, and intervening supraspinous and interspinous ligaments.

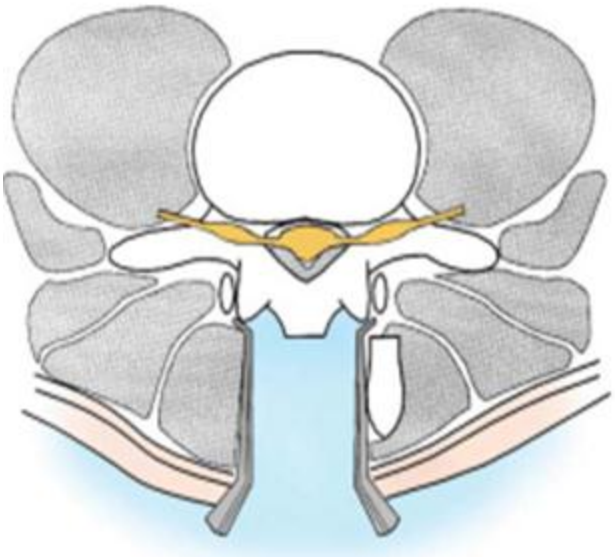


Fig-16: Spinous process osteotomy

LUMBAR SPINOUS PROCESS SPLITTING DECOMPRESSION:

The spinous process was identified and burred down until its base. The interspinous ligaments and supraspinous ligaments were cut longitudinally in line with the spinous processes. Using osteotome the spinous process split into two halves, the split halves of the spinous processes were osteotomized at the base and separated it from the lamina. The split halves of the spinous process along with the paraspinal muscles were then retracted on either side to expose the laminae. Decompression then done according to the conventional laminectomy method. The spinous process and paraspinal muscles are reapproximated with each other.

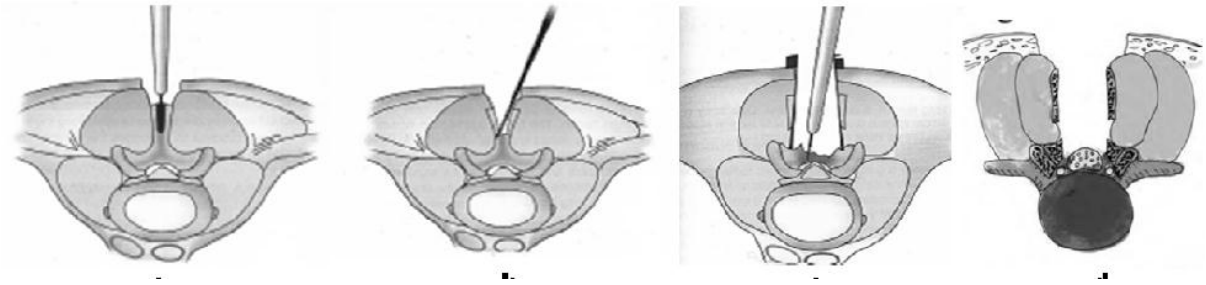


Fig-17: Lumbar spinous process splitting

COMPLICATIONS:

1) Intra operative and
2) Post operative complications

These complications include inadequate neural decompression, recurrent stenosis, incidental dural tear, neural injury, epidural hematoma, neural compression from either fat grafts or other barriers to scar formation, vascular injury, and late instability.

INADEQUATE NEURAL DECOMPRESSION

Sites of compression include central compression of the cauda equina and lateral compression, either within the lateral recess, within the neural foramen, or extraforaminally. It is important to identify all clinically significant sites of neural compression and to decompress those levels adequately. Inadequate decompression leads to failure in symptomatic relief of radicular leg pain. The surgery should be continued until the offending neural compression is found. It is important for the surgeon to recognize and look for additional sites of neural compression that may account for inadequate relief following decompression of only one site. This condition is sometimes referred to as a double crush phenomenon and is thought to be at least partially due to venous congestion of

the neural segment located between the two sites of compression resulting in a compartment syndrome like condition of the intervening segment,which may leads to inadequate neural decompression.

RECURRENT STENOSIS

Distinguishing between neural compression and scar formation in recurrent symptoms is difficult. It requires a precise history and high-quality radiographic imaging. Failure to obtain even temporary pain relief following decompression suggests either inadequate neural decompression, irreversible neural damage at the time of surgery, or a nonspinal cause for the pain. A pain-free interval of less than 6 months suggests development of scar formation as the cause of recurrent pain. Recurrence of pain following a free interval of more than 6 to 12 months suggests a new process such as a recurrent disc herniation or recurrent stenosis.

DUROTOMY

It is the incidental injury of the dura during surgery. The incidence is 0.3% to 13%. The injury is noted immediately by the sudden appearance of cerebrospinal fluid (CSF) within the wound or later by the clinical appearance of persistent spinal headache, the presence of CSF drainage from the wound.

Repair of Durotomy

- The patient is placed in a slightly head-down (Trendelenberg) position to minimize the amount of CSF in the field. This provides a drier operative field and minimizes the tendency for the individual roots of the cauda equina to float on the surface, which can result in injury during dural repair.
- For large tears, place a small cottonoid patty over the exposed nerve roots for the initial portion of the repair and then remove it just before dural closure
- For tears associated with loss of tissue, or tears in difficult locations, an autologous fat graft, a piece of autograft fascia (thoracolumbar fascia or fascia lata) may be required to close the defect
- Perform a watertight closure.
- Drain is often not employed in order to minimize risk of development of a CSF fistula.
- Keep the patient on bed rest for 3 to 5 days.

If a postoperative dural leak is suspected due to persistent spinal headache or a pseudomeningocele, confirm the diagnosis by myelography or MRI.

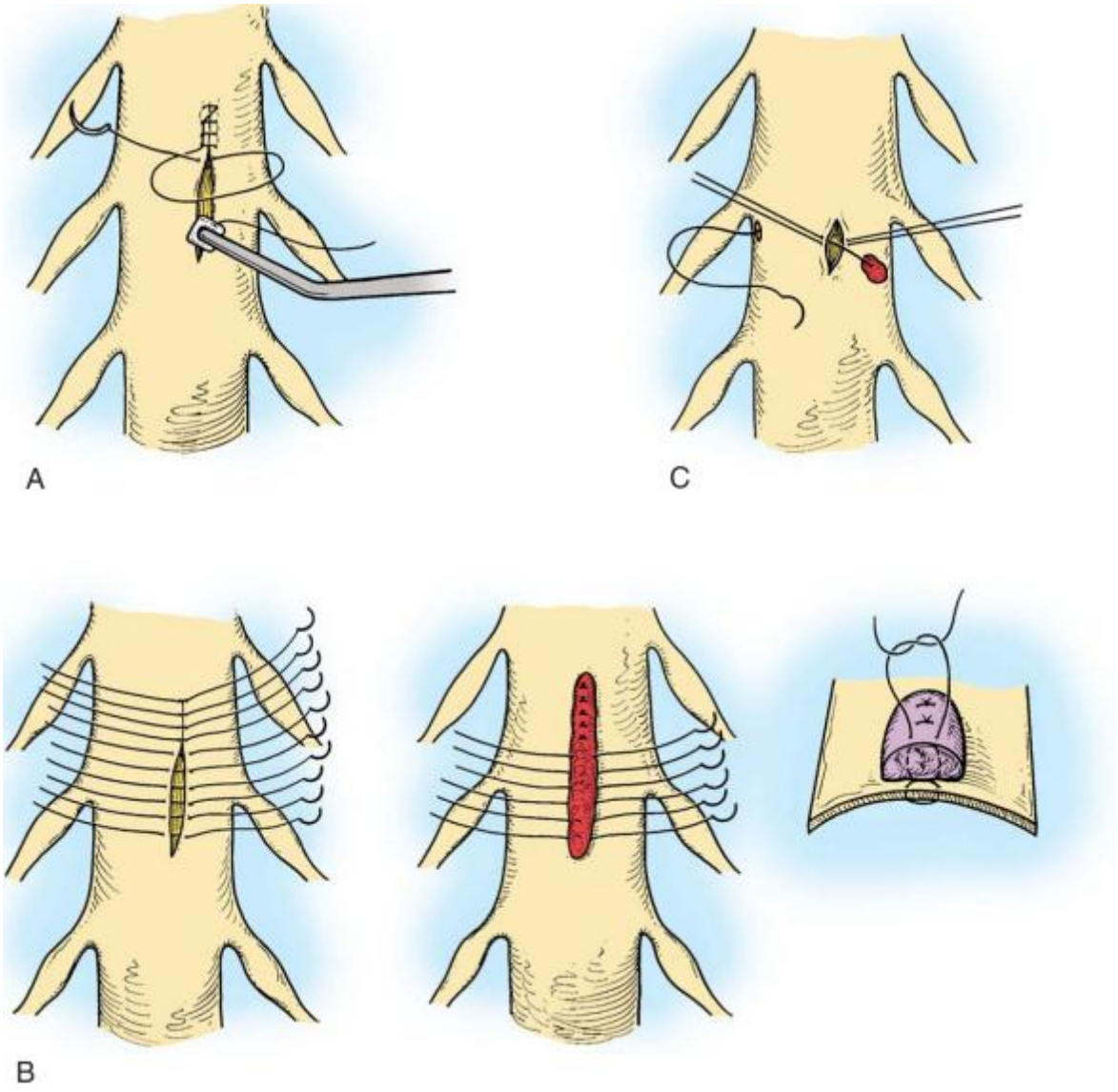


Fig-18: Dural repair

NERVE ROOT INJURY

Neural injury may occur due to direct trauma to the nerve root during surgery. This injury may occur due to excessive neural retraction, contusion, laceration, or electrocauterization. The incidence of neurologic complications has been estimated to be 0.2%..Note the preoperative presence of spina bifida occulta or

a pre-existing laminectomy defect on the preoperative radiograph in order to minimize risk of damage to underlying dura and nerve roots. It is important to visualize the lateral edge of the nerve root during surgical decompression to aviod inadvertent exposure in the axilla of a nerve root where accidental dural laceration and neural injury could occur . In revision lumbar surgery dissection performed usually lateral to the root along the lateral edge of the bony canal to avoid a potentially dangerous midline scar. While performing lateral nerve root decompressions,work parallel, rather than perpendicular, to the long axis of the nerve root in order to minimize risk of cutting across a root.

SCAR TISSUE

It has been implicated as a potential cause for continued pain following spinal surgery. Postoperative scar tissue may be located either intradurally (arachnoiditis) or extradurally (epidural fibrosis). Its etiology is often unclear, but it has been associated with oil-based myelographic contrast agents, prior surgery or dural laceration in which blood gains entry into the dural sac and mixes with neural elements.. When postoperative pain exists, the primary differential diagnosis is between scar and recurrent disc herniation. Radiographic distinction is best made with gadolinium-enhanced MRI or post-contrast CT .

Postoperative scar prevention: Delicate surgical technique, meticulous hemostasis and drainage, and the use of some form of an interposition

membrane. These barriers include a thin layer of fat or synthetic agents such as an absorbable gelatin sponge. The use of a free fat graft has been considered the gold standard interposition membrane.

EPIDURAL ABSCESS

Epidural abscess, is one of the most feared complications of spinal surgery because of its risk of paresis or frank paralysis. It is a rare occurrence. Patients have significant fever, back pain, and often present with neurologic findings such as nuchal rigidity and weakness or paralysis of the lower extremities. Both the WBC and acute phase reactants are elevated. MRI is the diagnostic imaging modality of choice and clearly visualizes the abscess . Treatment of an epidural abscess: surgical evacuation of the abscess and any adjacent necrotic tissue, followed by parenteral antibiotics.

EPIDURAL HEMATOMA

The clinical feature of epidural hematoma is the presence of severe pain that appears out of proportion to what is normally expected. This is usually associated with a progressive neurologic deficit. Depending on the extent and location of the hematoma, the neurologic deficit may be focal and unilateral, or it may be widespread. Confirm the diagnosis with MRI, myelography, or CT.

Once the diagnosis is confirmed immediately return the patient to the operating room for decompression and drainage of the hematoma.

The risk of this complication can be minimized by meticulous attention to preoperative, intraoperative, and postoperative detail. check the prothrombin time (PT), partial thromboplastin time (PTT), bleeding time, platelet count, and platelet function. Intraoperatively, position the patient with the abdomen hanging freely in order to minimize epidural venous congestion. Keep the blood pressure below 100 mm Hg systolic, in order to minimize bleeding. Control epidural bleeding with bipolar electrocoagulation. At the end of the surgery, after removing the deep paraspinal muscle retractors check the muscle walls for persistent bleeding, because prolonged retraction may temporarily occlude but significant muscle bleeders that could begin bleeding after muscle layer closure.

SUPERFICIAL WOUND INFECTION

Postoperative spine infections may be divided into superficial or deep infections. Superficial wound infections are located beneath the dermis but superficial to the deep thoracolumbar fascia and are characterized by tenderness and localized erythema. They associated drainage and fluctuance, although in

milder cases consisting only of cellulitis these may be absent. Patients may be febrile. Laboratory data show elevation of the erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP).

Treatment of superficial wound infections consists of surgical debridement of necrotic tissue , local wound care with short-term parenteral antibiotics.

DEEP WOUND INFECTIONS

Symptoms include disproportionate back pain or leg pain. The patient look ill and may exhibit generalized malaise. Fever is often present but may be deceptively low grade. The ESR and CRP are usually elevated. MRI provides the best and most useful information by revealing both the presence and extent of a deep abscess. If MRI is not available, diagnosis may be confirmed radiographically by the presence of a circumscribed area of fluid density visualized by CT. If a deep abscess is suspected, diagnosis may be confirmed by aspiration, with culture and sensitivity of any fluid obtained.

Treat deep wound infection with surgical debridement of all necrotic tissues, followed by appropriate parenteral antibiotics, remove any fat graft or absorbable gelatin sponge material. After the removal of infected or suspicious tissues, thoroughly irrigate the wound with pulsatile lavage. Place a drain and close the wound meticulously. Tightly close the deep fascia with interrupted absorbable suture with a continuous running stitch. Use of suction-irrigation tubes for a few days may be considered.

POSTOPERATIVE INSTABILITY

Instability following surgical decompression can occur in either the anteroposterior plane (spondylolisthesis) , in the mediolateral plane (lateral listhesis and scoliosis), or in both planes simultaneously. The risk of postoperative instability can be minimized by maintaining the integrity of atleast one facet joint at the level decompressed. If a total of more than one facet is removed, consider prophylactic fusion of that level. When decompressing a stenotic level associated with a degenerative spondylolisthesis, concomitant fusion should generally be performed because surgical outcome has been shown to be better with fusion than with decompression alone. This reduces risk of a subsequent increase in the slip.

FUNCTIONAL EVALUATION:

Various scoring systems are available to assess low back function. Japanese Orthopaedic Association Score (JOA Score) has two components subjective symptoms assessed in first section (maximum 9 points) and the clinical signs in second section (maximum 6points). The Scores from the two sections are added to form a JOA Score. The Score of -6 represents poorest function, and negative points being incorporated for bladder symptoms. A score of 15 represents an asymptomatic and fully functional subject.

JOA recovery rate can be calculated using the formula of Hirabayashi.

$$\text{Recovery rate}(\%) = \frac{(\text{postoperative score} - \text{preoperative score})}{(15 - \text{preoperative score})} \times 100$$

JAPANESE ORTHOPEDIC ASSOCIATION SCORE(JOA Score) FOR LOW BACK DISORDERS

Symptom/sign Points

I. Subjective symptoms (9 points)

A. Low back pain
- a. None 3
- b. Occasional mild pain 2
- c. Frequent mild or occasional severe pain 1
- d. Frequent or continuous severe pain 0

B. Leg pain and/or tingling
- a. None 3
- b. Occasional slight symptoms , 2
- c. Frequent slight or occasional severe symptoms 1
- d. Frequent or continuous severe symptoms 0

C. Gait
- a. None 3
- b. Able to walk farther than 500 m although it results in pain, tingling, and/or muscle weakness 2
- c. Unable to walk farther than 500m owing to pain, tingling, and/or muscle weakness 1
- d. Unable to walk farther than 100 m although it results in pain, tingling, and/or muscle weakness 0

II. Clinical signs (6 points)

A. Straight-leg-raising test (including muscle weakness)
- a. Normal 2
- b. 30°–70° 1
- c. less than 30° 0

B. Sensory disturbance
- a. None 2
- b. Slight disturbance 1
- c. Marked disturbance 0

C. Motor disturbance (MMT)
- a. Normal (grade 5) 2
- b. Slight weakness (grade 4) 1
- c. Marked weakness (grade 0–3) 0

D. Bladder function
- a.normal 0
- b.mild -3
- c.severe dysuria -6

possible range of scores -6 to 15

Max score-15 (full function); Min score -6(poorest function)

Table-5

Neurogenic claudication Outcome score(NCOS):

Neurogenic claudication Outcome score(NCOS) is a questionnaire designed mainly for assessing the severity of neurogenic claudication symptoms. The questionnaire consists seven sections of questions pertaining to activities of daily living and a section of visual analog scale for pain. These scores are added to form the NCOS score. A score of 0 represents poorest function while a score of 100 represents full function.

Table 2. Neurogenic Claudication Outcome Score and How to Calculate It

1. How far can you walk before having to stop and rest?
   a) <100 yards (0 points) b) Between 100 yards and ½ mile (2 points) c) Between ½ and 1 mile (4) d) Greater than 1 mile (6)
2. How long can you stand still before looking for a place to sit down?
   a) <5 min (0) b) 5 to 15 min (2) c) 15 to 45 min (4) d) as long as I want (6)
3. Once your symptoms arise, you have:

	None	Mild	Moderate	Severe
Back pain	(6)	(4)	(2)	(0)
Leg pain	(6)	(4)	(2)	(0)
Numbness/tingling	(6)	(4)	(2)	(0)
Heaviness/weakness	(6)	(4)	(2)	(0)

4. How much do your symptoms affect the following activities?

	Not at All (can do easily)	Mildly (symptoms, but can do)	Moderately (difficult, but can do)	Severely (impossible to do)
Sports or activities	(6)	(4)	(2)	(0)
Household or odd jobs	(6)	(4)	(2)	(0)
Walking	(6)	(4)	(2)	(0)
Standing	(6)	(4)	(2)	(0)
Sitting	(6)	(4)	(2)	(0)
Sex life	(6)	(4)	(2)	(0)

5. How long must you rest before the symptoms resolve?
   a) <5 min (6) b) between 5 and 10 min (3) c) >10 min (0)
6. How frequently do you take pain medicine for your back/leg pain?
   a) Never (6) b) Occasionally (4) c) Daily (2) d) Frequently (0)
7. How frequently do you see a doctor for your back/leg pain?
   a) Never (6) b) Rarely (4) c) Monthly (2) d) Frequently (0)
8. Please indicate the level of your pain on the following scale: (scored as 10–X)

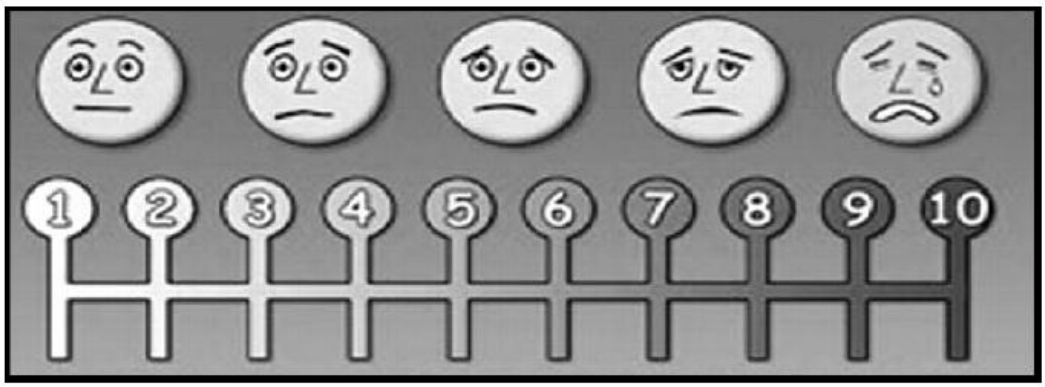
0	1	2	3	4	5	6	7	8	9	10
No pain										Worst pain possible

Note: Total points possible = 100 (asymptomatic, full function).

Table -6

Visual Analog Scale(VAS):

The typical visual analog scale is a rectangular strip with pictorial representations of human faces with varying degrees of pain which are numbered from zero (smiling face, no pain) to 10(worst imaginable pain). Patient is asked to choose the face that most closely represents his/her pain. This can be used to assess the back pain as well as the claudication pain.



Visual Analog Scale for Pain

Paraspinal muscle injury

Elevation of paraspinal muscles during spine surgery shows muscle atrophy. The magnitude of the muscle damage depends on the extent of detachment of paraspinal muscles from the posterior elements and time of retraction by retractors. This is thought to lead to atrophy due to ischemic insult to the muscles as well as denervation. Several studies have correlated muscle dysfunction and persistent low back pain. So surgical methods involving lesser

damage to the paraspinal muscles are more likely to have a good functional outcome.

1) **Creatine Phosphokinase**

Creatine Phosphokinase muscular isoenzyme(CPK-MM) originates from skeletal muscle and which is a reliable indicator of skeletal muscle damage during surgery. CPK-MM normal value ranges from 45- 230 U/L. CPK-MM levels in blood rises immediately after surgery and plateaus off after first postoperative day. The enzyme level starts to decrease one to two weeks after surgery. Measurement of CPK-MM levels in the postoperative period can be used to determine the amount of paraspinal muscle damage.

2) **C- Reactive Protein**

C- Reactive Protein also related to skeletal muscle damage and inflammation in many studies.

Comparison between the preoperative and postoperative levels of these protein is an indicator of the invasiveness of the procedure and the extent of paraspinal muscle injury .

PREAMBLE

In this era of modernisation and sophisticated investigation technologies, degenerative lumbar canal stenosis has come to the fore as a cause of low back pain in the elderly with more cases diagnosed and more surgeries done, it is imperative to find a comparison between surgical modalities. In this study, we have made an attempt to identify the best surgical modality for lumbar canal stenosis.

AIM OF THE STUDY

This prospective Randomised Control Study compares **the the functional outcome and extent of paraspinal muscle damage between Lumbar spinous process splitting decompression (LSPSD) and Conventional Midline Decompression(CMD) by laminectomy surgical approaches in degenerative lumbar canal stenosis** and their aim was whether

1) Lumbar spinous process splitting decompression (LSPSD) approach provide sufficient decompression.

2) Preserve posterior musculoligamentous complex and reduces associated morbidity.

STUDY DESIGN:

Prospective randomised Control study

MATERIALS AND METHODS

This randomized prospective control study was approved by the medical ethics committee of the Institutional Review Board in our hospital. Patients meeting the following inclusion criteria were enrolled for the study after obtaining written informed consent. 20 patients with degenerative lumbar canal stenosis are randomly divided into two groups and recruited into the study based on the following criterias

INCLUSUION CRITERIA:

- Degenerative LCS affecting 3 or less levels,
- Typical neurogenic claudication symptoms,
- Magnetic resonance image demonstrating good clinical correlation
- Failure of conservative methods of treatment for a minimum period of 6 months.

EXCLUSION CRITERIA:

- Spondylolisthesis with slip grade 2 or greater (Meyerding grade).
- Instability at the level of stenosis (as defined by >3-mm translation or >10° angular change on flexion extension lateral radiographs)
- Associated symptomatic cervical or thoracic stenosis.
- Multiple level canal stenosis.
- Spinal canal stenosis due to congenital, traumatic , iatrogenic causes.
- Presence of spinal disorders(ankylosing spondylitis, neoplasm)
- Comorbidities (such as cardiopulmonary insufficiency, peripheral neuropathy, peripheral vascular disease, prior lumbar spine surgery, and severe hip or knee disease).

Preoperative evaluation:

-Patient history and neurological examination

- Preoperative clinical evaluation of the patients was made by

1) Japanese Orthopaedic Association (JOA) score

2) Neurogenic claudication outcome score (NCOS).

3) Visual analogue score for back pain and neurogenic claudication (VAS)

- Baseline C -reactive protein (CRP) and

CPK-MM levels.

Radiography of lumbosacral spine

a) Antero –posterior view

b) Lateral view

c) Dynamic flexion-extension lateral view

d) MRI of the lumbo sacral spine.

Surgical Technique

For either procedure patient was placed prone knee chest position under general anaesthesia and surgical level was confirmed using fluoroscopic image prior to incision

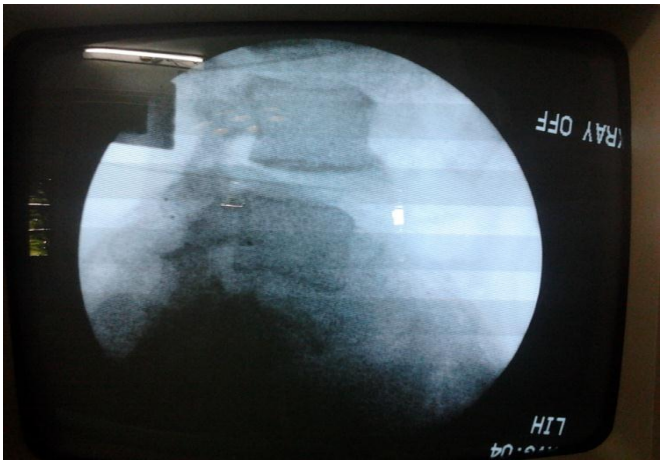


Fig-19: Level identification with C-arm

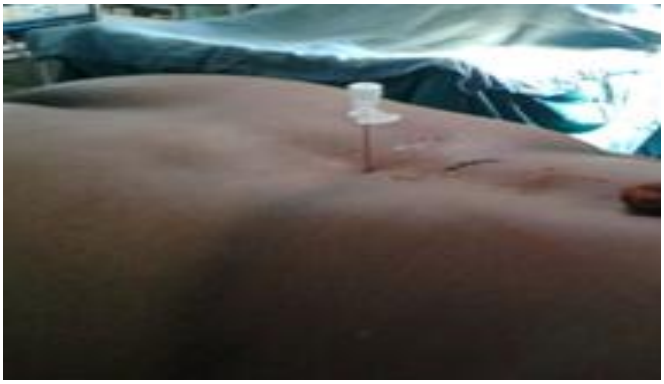


Fig-20: Level marking

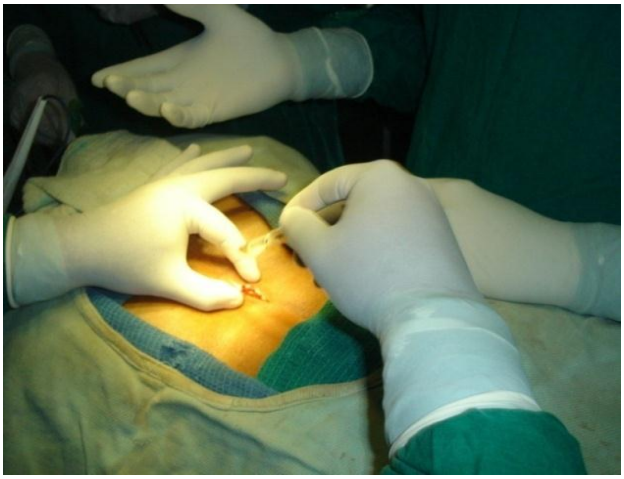


Fig-21: Skin incision

CONVENTIONAL MIDLINE DECOMPRESSION

- About 5to8 cm skin incision was made in the midline centered over the level of stenosis

- The deep fascia incised in the midline and the either side paraspinal muscles were elevated subperiosteally from the spinous process and lamina.

- Identify and remove the spinous process, the interspinous and the supraspinous ligaments of the level to be decompressed.

-Remove the lamina upto the insertion of ligamentum flavum.

- Once the of ligamentum flavum has been identified it can be removed from the lamina.

- Remove the lamina upto medial border of the pedicle, it can be helpful in decompressing thelateral canal.

- Adequate decompression of the dura and nerve root by probing the foramen .

-Identify disc space by c-arm control and gross herniations are removed and no discectomy in cases with minimal disc bulge.

-The paraspinal muscles were approximated in the midline using absorbable sutures and the subcutaneous tissue and skin were closed.

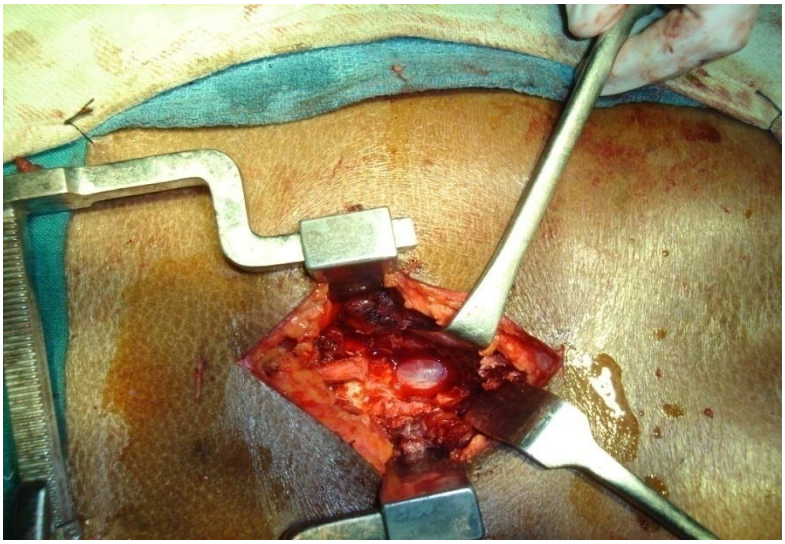


Fig-22: Removal of spinous process & interspinous ligaments

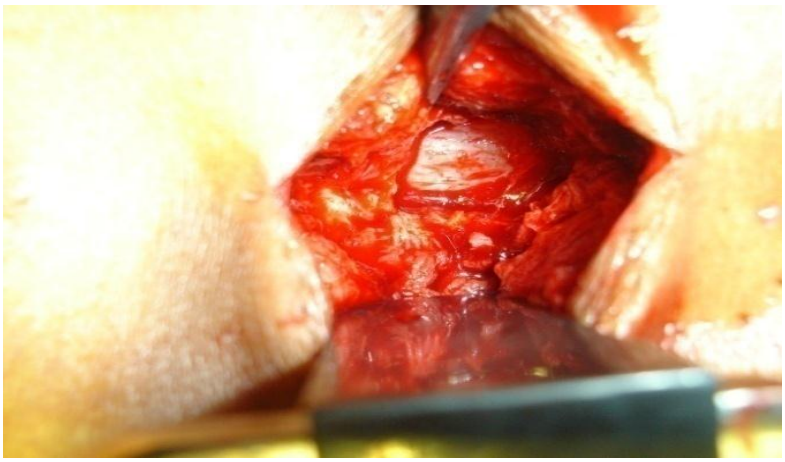


Fig-23: Cord decompression

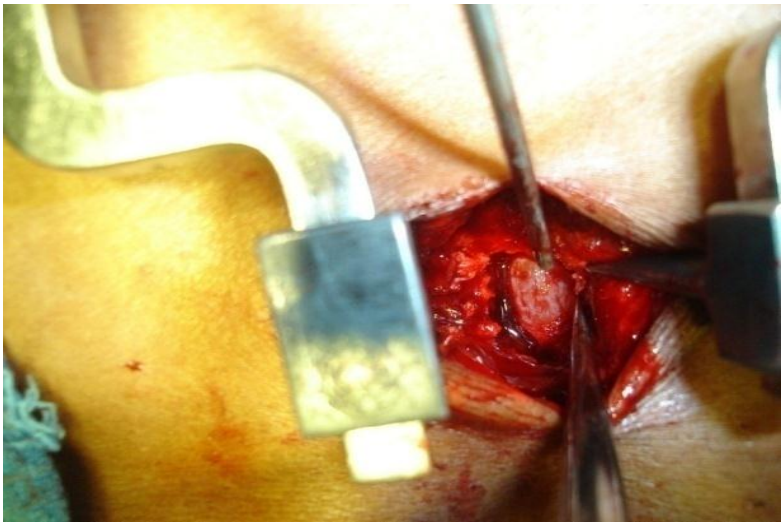


Fig-24: Root decompression

LUMBAR SPINOUS PROCESS SPLITTING DECOMPRESSION(LSPSD)

- The positioning of the patient and anaesthesia techniques are similar to the standard midline decompression technique.

- A 3 to 5 cm long incision was made over the proximal spinous process (e.g., L3 spinous process in case of L3–L4 decompression

- The posterior surface of the L3 spinous process was identified and using a high-speed 2-mm burr, the spinous process was burred down until its base.

-The proximal and distal interspinous ligaments and supraspinous ligaments were cut longitudinally in line with the spinous processes.

- Using an 5mm osteotome, the split halves of the spinous processes were osteotomized at the base and separated it from the lamina .

-The split halves of the spinous process along with the paraspinal muscles were then retracted on either side to expose the laminae. Decompression then done according to the conventional laminectomy method.

-Holes were made in the center of the split spinous processes to facilitate easy closure

- After adequate decompression of the dural sac and the roots, the split halves of the spinous process were sutured with no.1 Vicryl .

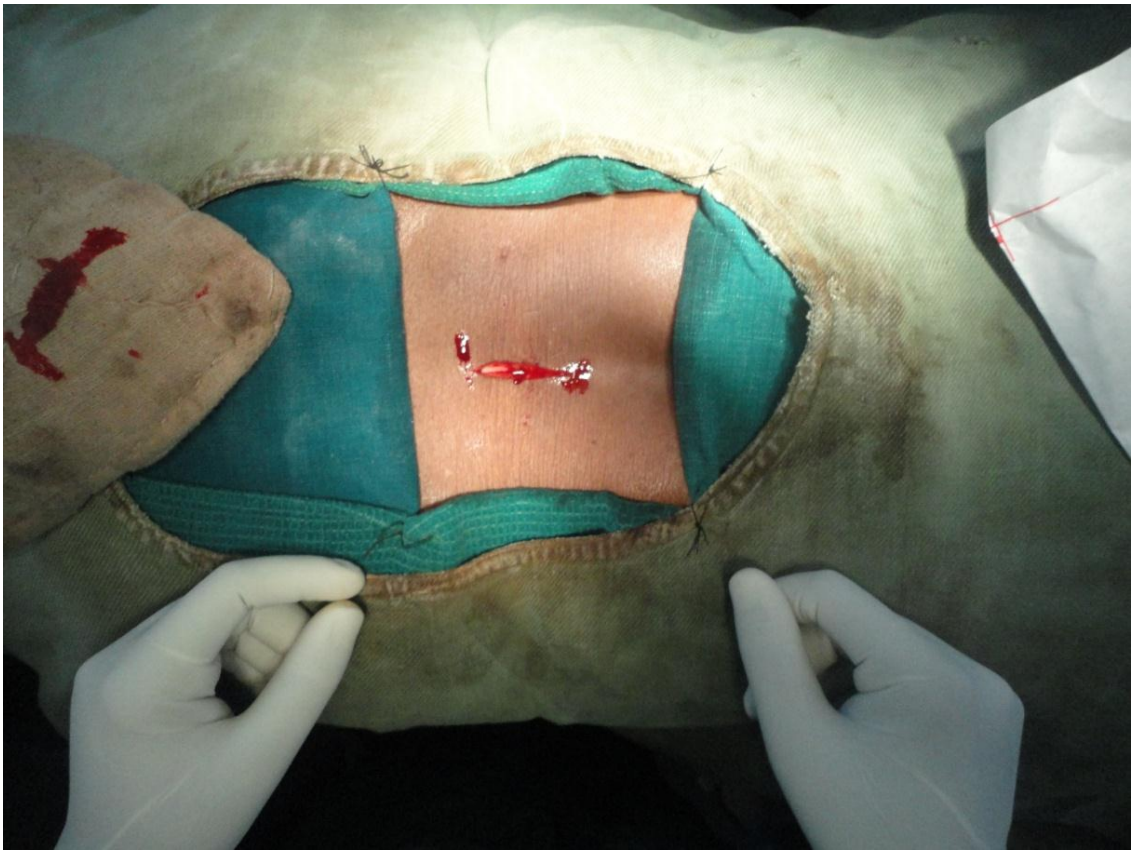


Fig-25: skin incision

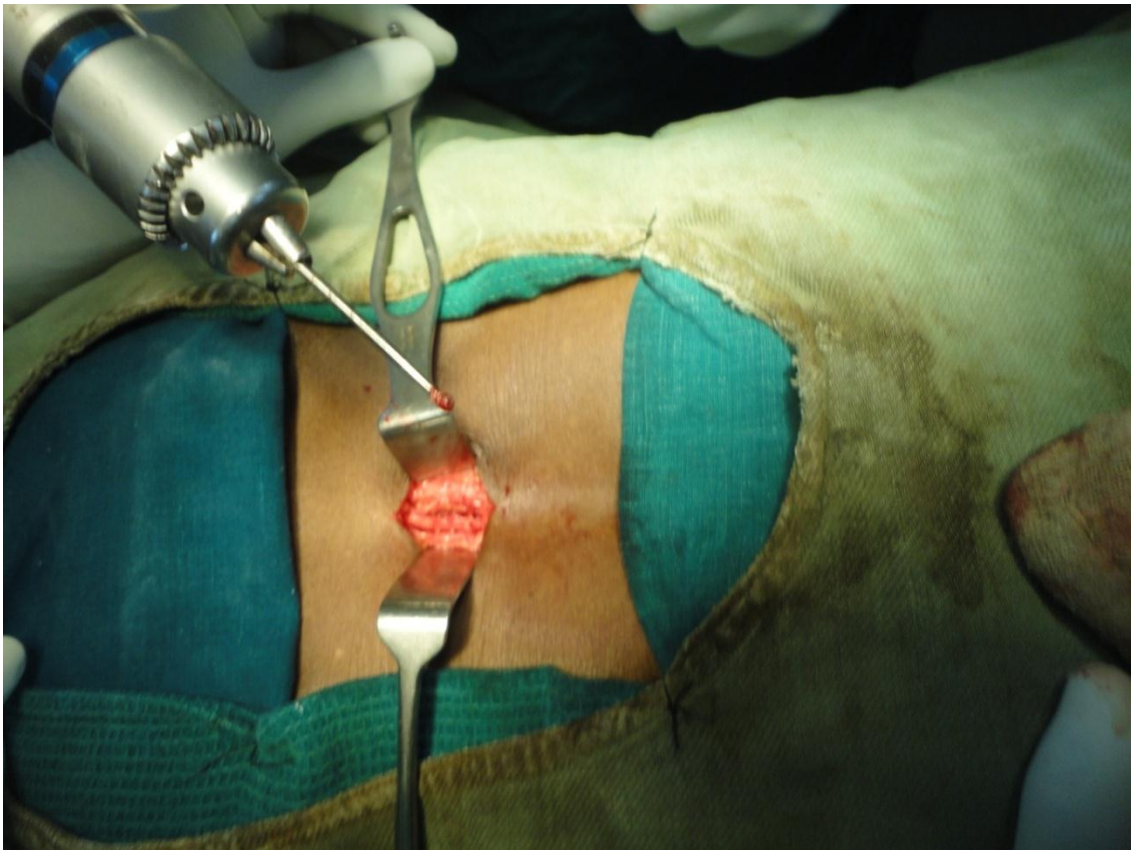


Fig-26: Spinous process splitting with burr

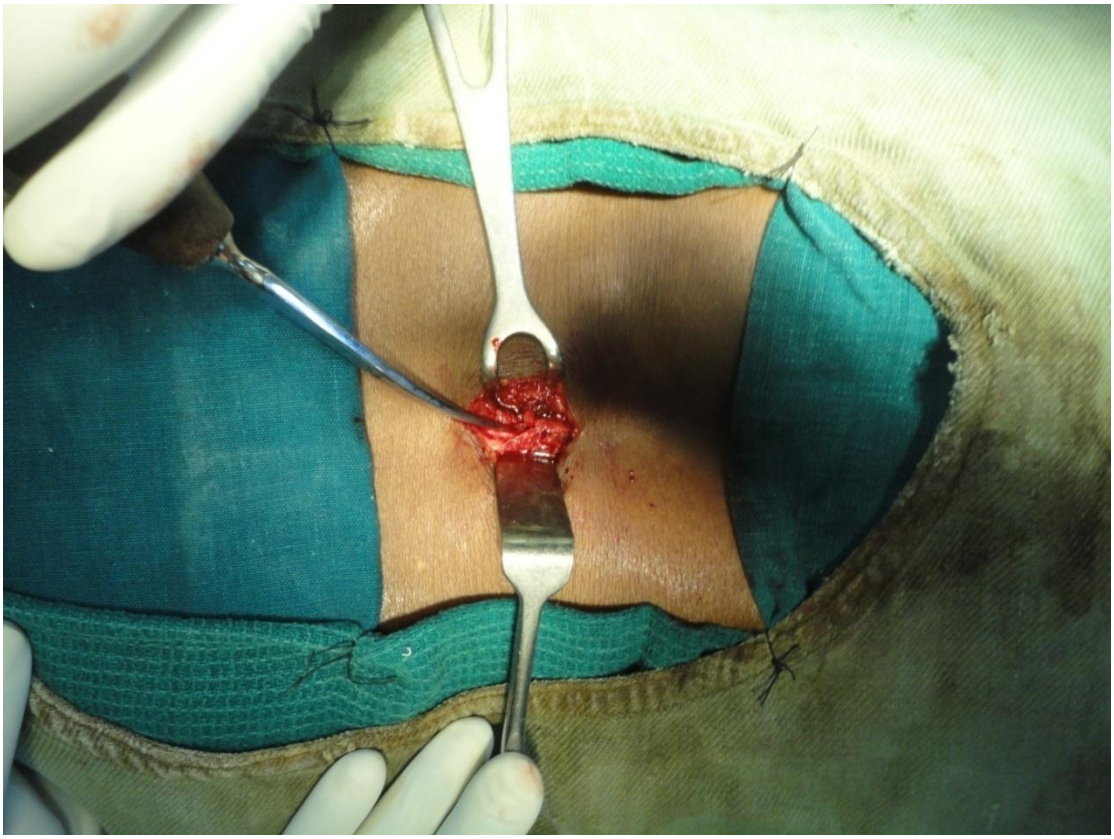


Fig-27: Splitting with osteotome

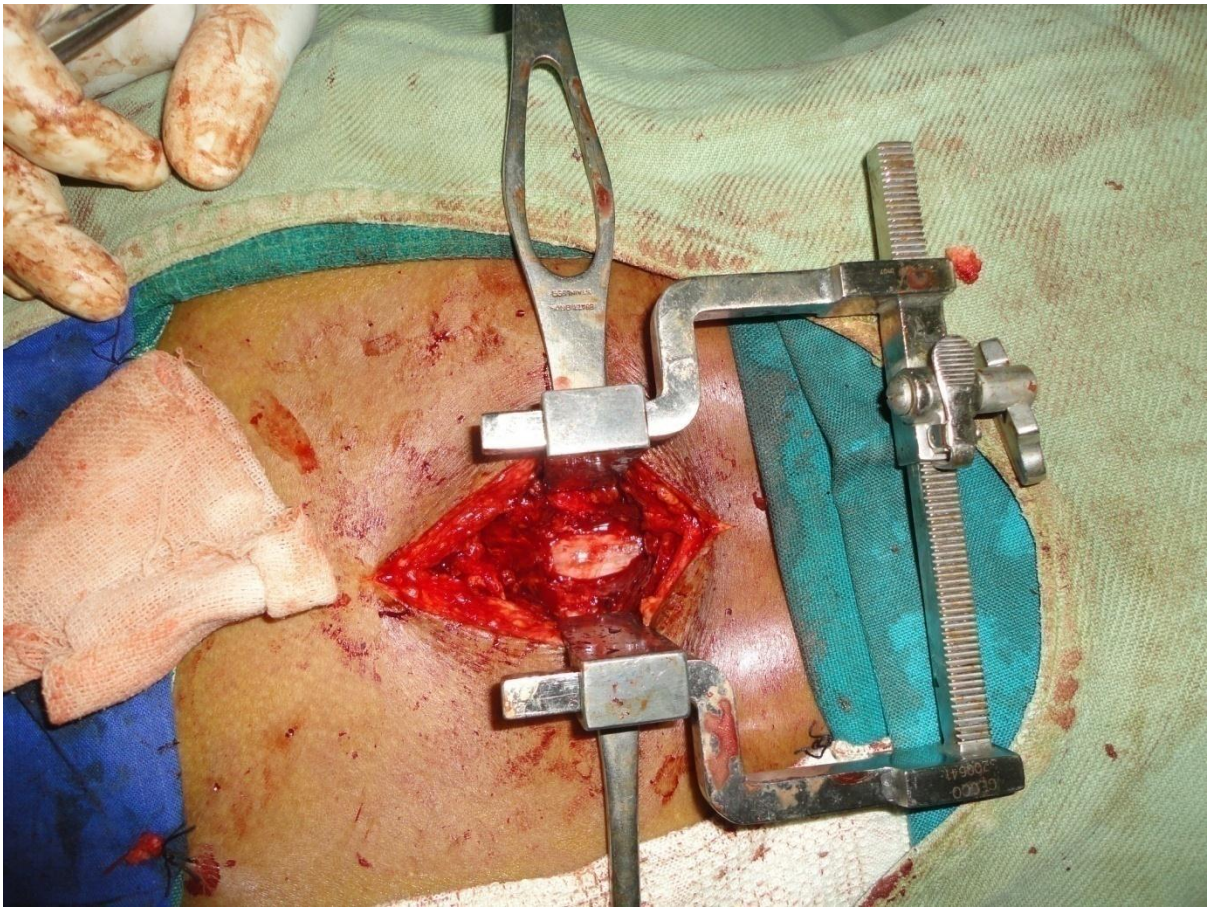


Fig-28: Decompression of spinal canal

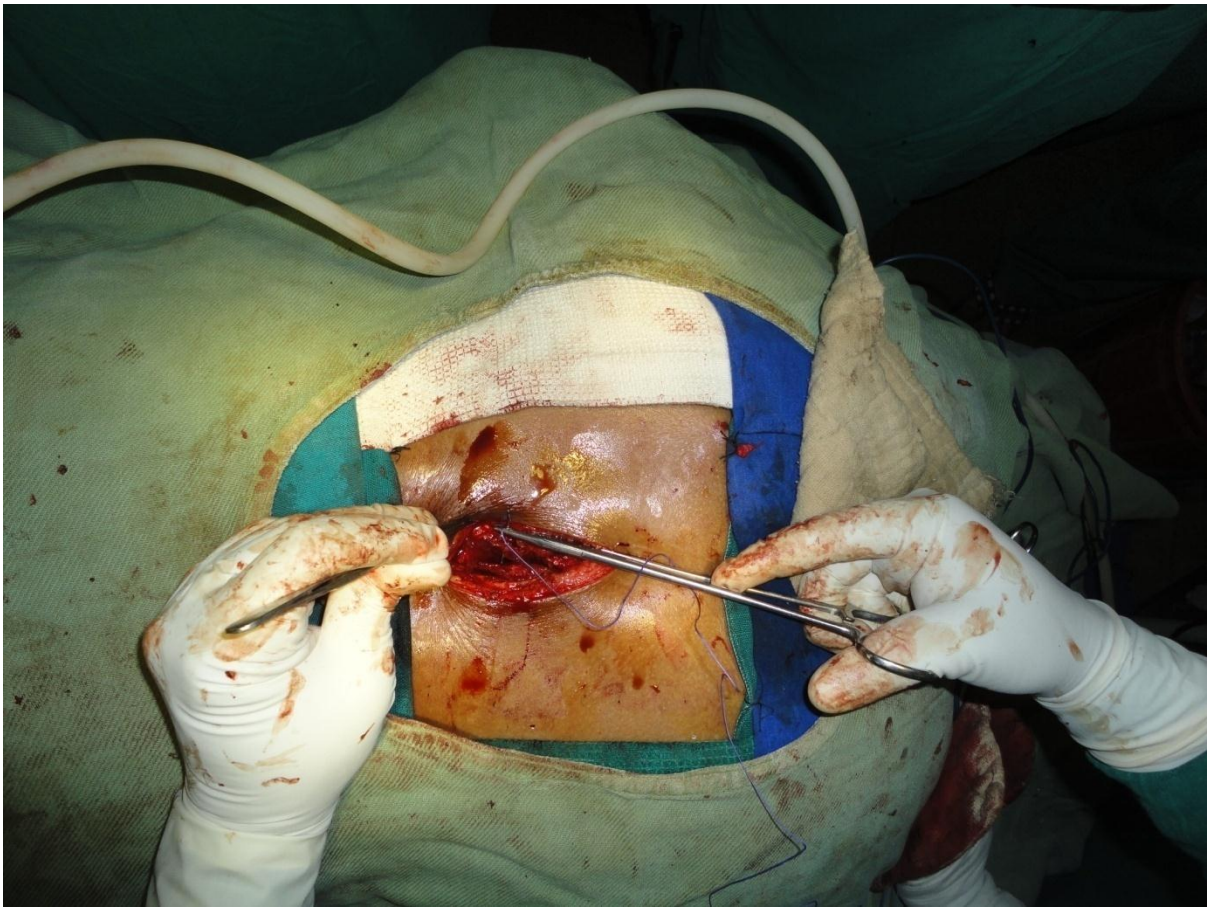


Fig-29: Suturing of spinous process

Intra operative assessment:

- Operative time

- Blood loss and transfusion

- Operative morbidity like dural tear and neural injury

POSTOPERATIVE PROTOCOL

-Patients allowed to turn to sides in the bed on the day of surgery and were encouraged to sit as early as possible once pain subsides.

-Patients were encouraged to walk as soon as is comfortable.

-Postoperative day on which patient is able to ambulate was noted, the day of the surgery being counted as day 0. Patients were discharged on the tenth day or when comfortable.

- Serum CRP and CPK-MM values were assessed on postoperative day 1 and day 3

- Patients were put on a program of spinal flexion exercises 3 weeks after surgery.

- Patients were followed-up regularly every month during the first three month and thereafter every 3 months upto 12 months and assessed using JOA and NCOS scores.VAS for back pain and neurogenic claudication.

COMPLICATIONS

No major intra operative complications were observed in both groups. One patient of Lumbar spinous process splitting decompression group and one patient of Conventional midline decompression group had intraoperative dural tear which were managed without repair. There were no cases of new neurological deficits due to surgical intervention. One patient in the Conventional midline decompression group and one patient in Lumbar spinous process splitting decompression group developed wound dehiscence and was treated with resuturing of the wound. Two patients of Conventional midline decompression group developed UTI during the post operative period and two patients Lower respiratory tract infections treated with appropriate antibiotics. One patient in conventional midline decompression had instability at 9 month follow up.

FOLLOWUP AND RESULTS

20 patients were followed up for 6-18 months with mean average follow up of 11.4 months. Data of 10 patients (5 men and 5 women) in the lumbar spinous process splitting decompression group and 10 patients (4 men and 6 women) in the Conventional Midline Decompression group were included in the final analysis. The mean age was 58.9 (range 54-65) yrs for the lumbar spinous process splitting decompression group and 60.4 (range 55-65) yrs for Conventional Midline Decompression group. Mean number of decompressed levels were 1.30 for Conventional Midline Decompression group and 1.20 for lumbar spinous process splitting decompression.

PRE OPERATIVE PARAMETERS:

S.NO	CONTENTS	CONVENTIONAL MIDLINE DECOMPRESSION (LAMINECTOMY)	LUMBAR SPINOUS PROCESS SPLITTING DECOMPRESSION
1.	No. of patients	10	10
2.	Average age	60.4	58.9
3.	Male: Female	4:6	5:5
4.	Mean No. of decompressed levels	1.30	1.20
5.	Associated protruded disc removal	6	7
6.	Average duration of follow up	11.6 months	11.2 months

Table-7

INTRA OPERATIVE PARAMETERS:

S.no	Contents	Conventional Midline Decompression (Laminectomy)	Lumbar spinous process splitting decompression
1.	Average duration of procedure	71.23 min	80.25 min
2.	Average blood loss	126 ml	130 ml
3.	No.of blood transfusion	2	1
4.	Dural Tear	1	1
5.	Iatrogenic Neurologic Deficit	–	–

Table - 8

S.no	Contents	Conventional Midline Decompression (Laminectomy)	Lumbar spinous process splitting decompression
1.	Average ambulation Time	6.52 days	4.45 days

- **POST OPERATIVE PARAMETERS:**

2.	Wound complications	1	1
3.	Urinary Tract Infections	2	-
4.	Lower Respiratory Tract Infection	2	_
5.	Instability	1	_

Table - 9

FUNCTIONAL OUTCOME SCORES:

Japanese Orthopaedic Association Score (JOA Score):

In the Lumbar spinous process splitting decompression group JOA score improved from preoperative mean 5.4 to 12.50 at the last follow up. In the Conventional Midline Decompression the score improved from preop mean 5.3 to 11.3 at the last follow up. The mean JOA recovery rate was 73.96% for the Lumbar spinous process Decompression group and 61.86% for the Conventional Midline Decompression group. There was no statistically significant difference between the two groups.

Parameter	Lumbar spinous process splitting decompression (LSPSD)	Conventional Midline Decompression (Laminectomy)	Significance (P<0.05)
Preop JOA score	5.4	5.3	P>0.05
JOA score at last follow up	12.5	11.9	P>0.05
Change in JOA Score	7.1	6.6	P>0.05
JOA Recovery rate (%)	73.96	68.05	P>0.05
N =	10	10	

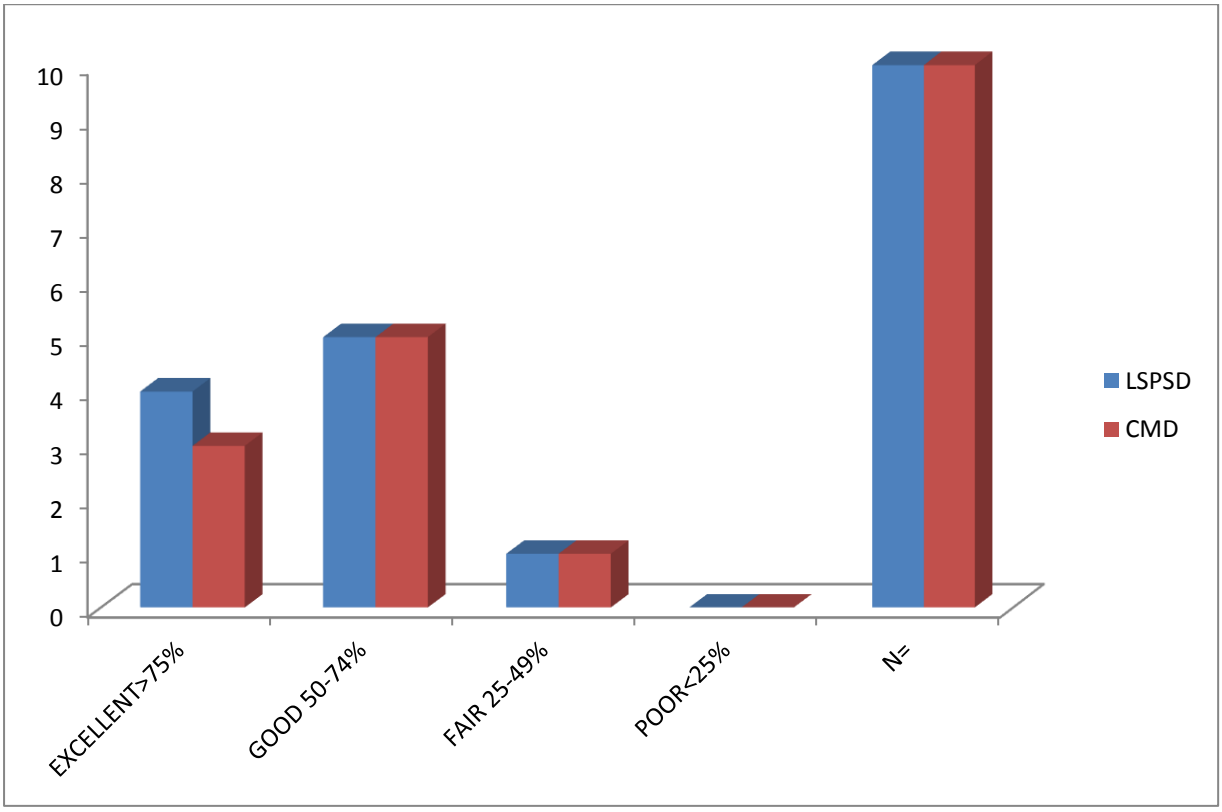
Table - 10

Notably 70% of Conventional Midline Decompression group had good or excellent outcome while 100% of Unilateral Decompression group had good or excellent outcome.

Outcome(JOA score Recovery rate) at final follow up	Lumbar spinous process splitting decompression (LSPSD)	Conventional Midline Decompression (Laminectomy))
Excellent(≥ 75%)	4	3
Good(50-74%)	5	5
Fair (25-49%)	1	2
Poor(≤24%)	0	0
N=	10	10

Table - 11

Fig-30: JOA RECOVERY RATE Score



Neurogenic Claudication Outcome Score(NCOS)

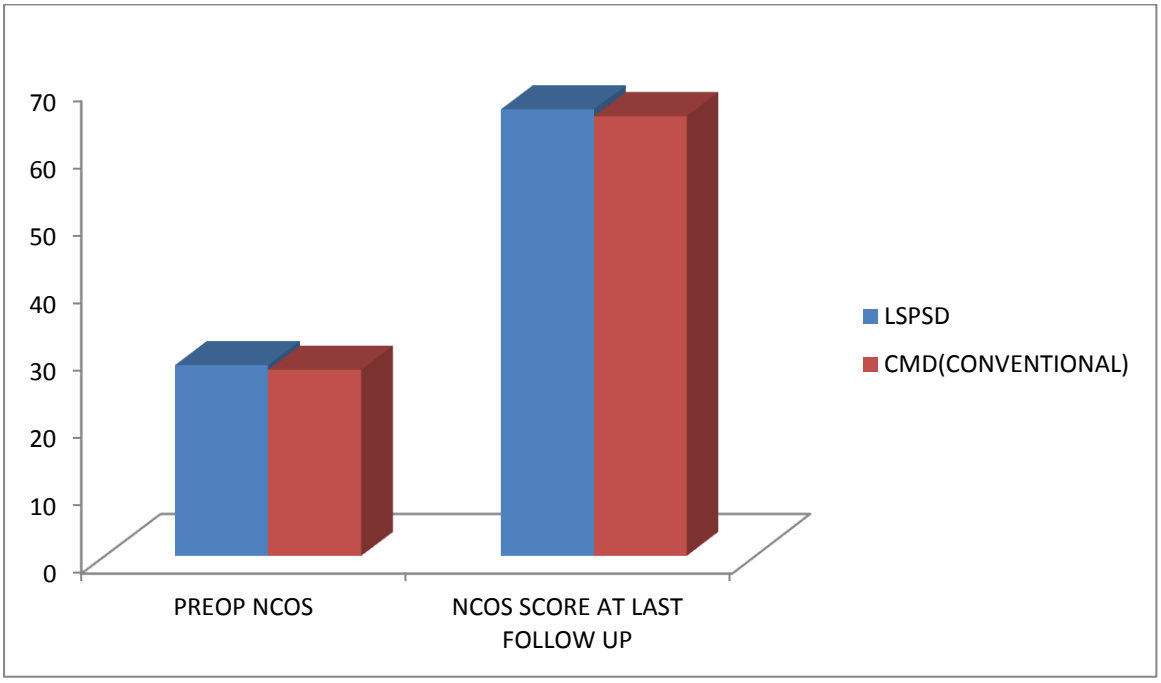
NCOS score improved from a mean preoperative score of 28.30 to 66.10 at last follow up in the Lumbar spinous process Decompression group, and from 27.60 to 65.10 in the Conventional Midline Decompression group . Statistical analysis did not reveal any significant difference between groups.

	Lumbar spinous process splitting decompression (LSPSD)	**Conventional Midline Decompression (Laminectomy)**	**Significance (P<0.05)**
Preop NCOS score	28.30	27.60	(P>0.05)
NCOS score at last follow up	66.10	65.10	(P>0.05)
Change in NCOS Score	37.80	37.50	(P>0.05)

N =	10	10	

Table - 12

Fig-31:Neurogenic Claudication Outcome Score(NCOS)



Visual Analog Scale for Back Pain (BPVAS):

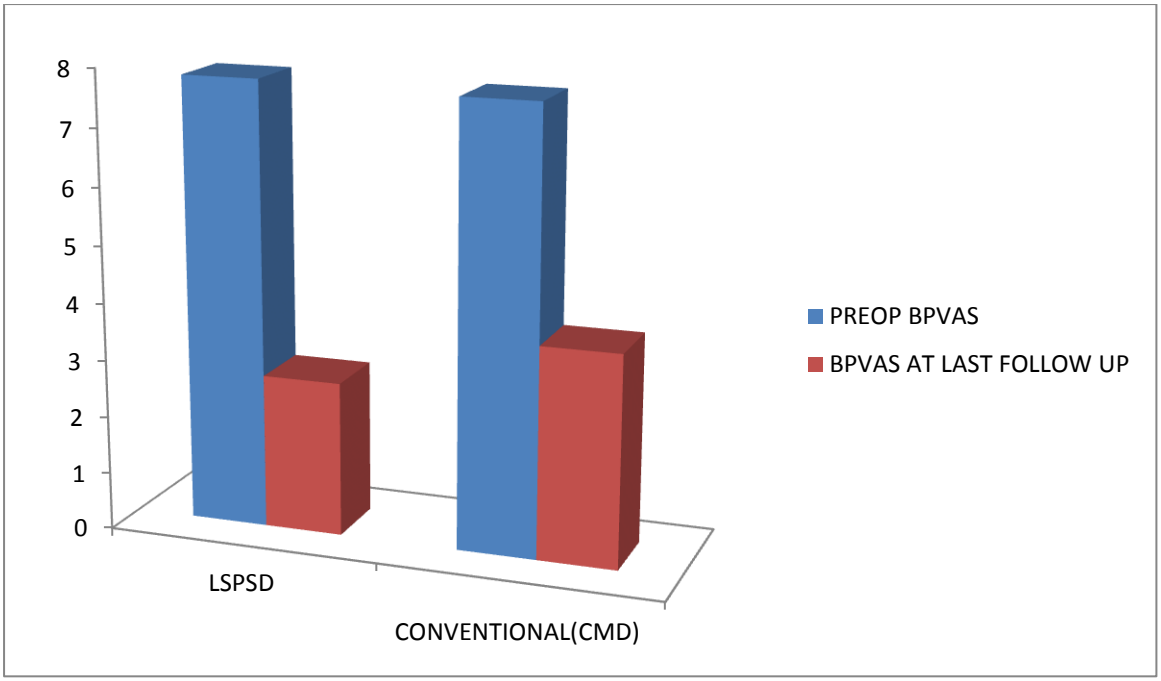
At the last follow up the mean BPVAS score for the Lumbar spinous process Decompression group was 2.7 and for Conventional Midline Decompression group it was 3.70. Statistical analysis revealed significant difference between two groups.

Parameter	**Lumbar spinous process splitting decompression (LSPSD)**	**Conventional Midline Decompression (Laminectomy)**	**Significance (P<0.05)**
Preop BPVAS	7.8	7.7	(P<0.05)
BPVAS score at	2.7	3.7	(P<0.05)

last follow up			
Change in BPVAS	5.1	4.0	(P<0.05)
N =	10	10	(P<0.05)

Table - 13

Fig-32:Visual Analog Scale for Back Pain (BPVAS):



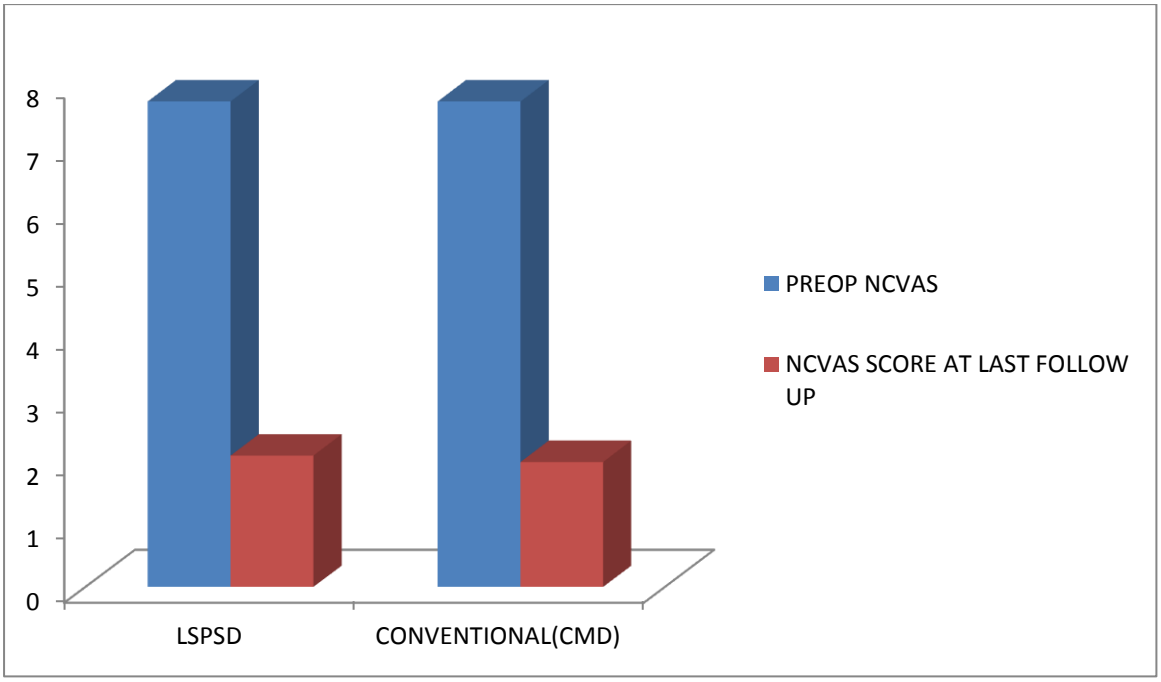
Neurogenic Claudication(NCVAS):

Mean NCVAS score at last follow up was 2.10 for Lumbar spinous process Decompression group and 2.0 for Conventional Midline Decompression group. There was no significant difference between the two groups

Parameter	Lumbar spinous process splitting decompression (LSPSD)	Conventional Midline Decompression (Laminectomy)	Significance (P<0.05)
Preop NCVAS	7.70	7.70	(P>0.05)
NCVAS S score at last follow up	2.10	2.0	(P>0.05)
Change in NCVAS	5.60	6.70	(P>0.05)
N =	10	10	

Table - 14

Fig-33:Neurogenic Claudication(NCVAS):



Biochemical markers for paraspinal muscle damage

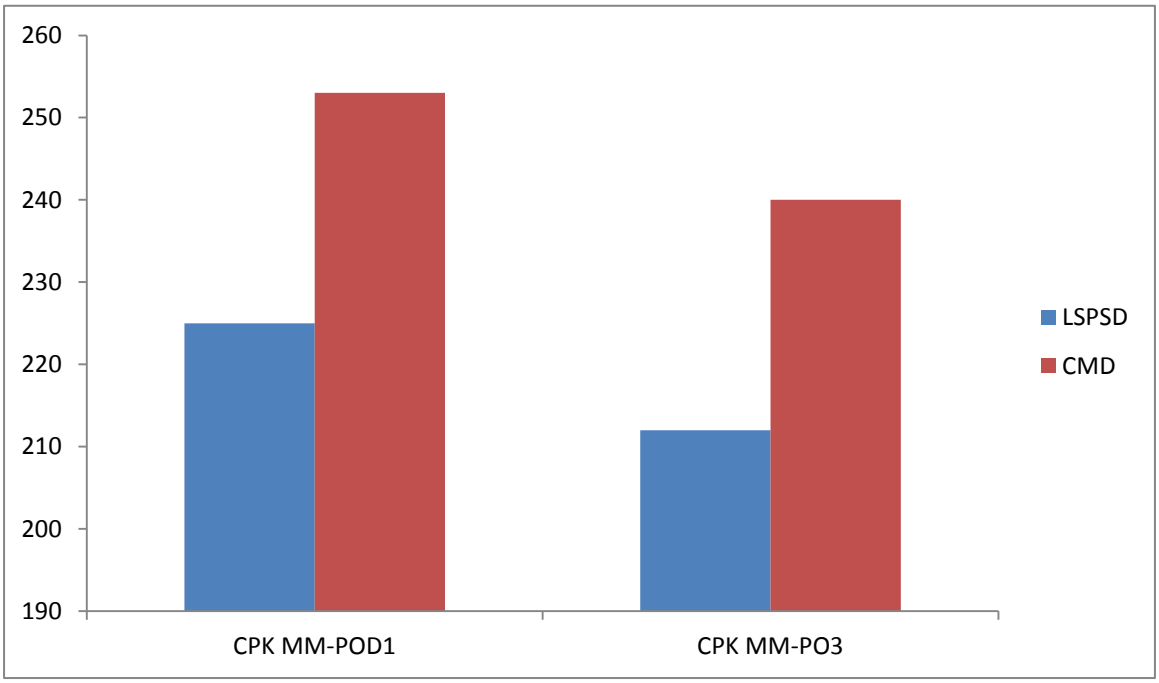
1) Creatine Phosphokinase (CPK-MM)

Rise of CPK-MM on postoperative day 1 and day 3 were estimated. CPK MM-1 (POD1-Preop)mean(U/L) for LSPSD was 225U/Land for CMD was 253U/L.CPK-MM-3 (POD3-Preop)mean (U/L) for LSPSD was 212 and for CMD was 240.Although the mean values on POD1 and POD3 for the CMD group were higher than the LSPSD group signifying more muscle damage, the difference was not statistically significant(p>0.05)

Parameter	**LSPSD**	**Conventional (CMD)**	**Significance (P<0.05)**
CPK MM-POD1	225	253	(P>0.05)
CPK MM-POD3	212	240	(P>0.05)
N =	10	10	

Table - 15

Fig-34: Creatine Phosphokinase (CPK-MM)



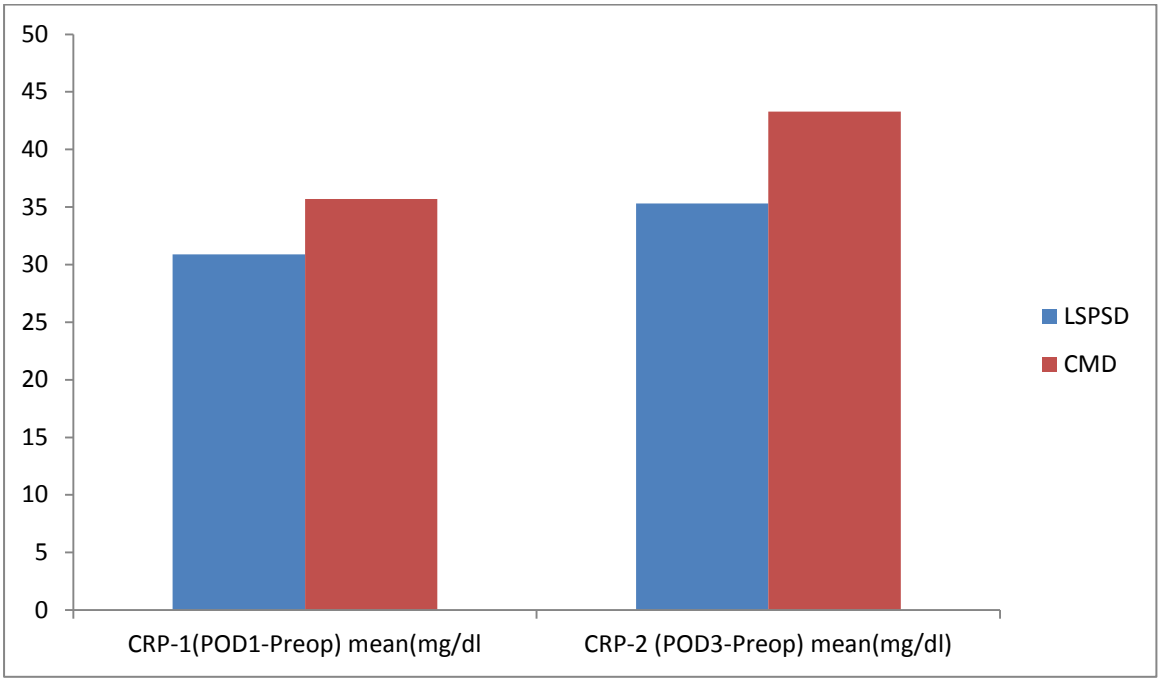
2) C-Reactive Protein (CRP)

Rise of CRP(mg/dL) on postoperative day 1 and day 3 were estimated. CRP-1 (POD1-Preop)mean(mg/dl) for LSPSD was 30.9 and for CMD was 35.7. CRP-3 (POD3-Preop)mean (mg/dl) for LSPSD was 35.3 and for CMD was 43.4.There was no statistically significant difference between the groups.

Parameter	LSPSD	Conventional (CMD)	Significance (P<0.05)
CRP-1(POD1-Preop) mean(mg/dl)	30.9	35.7	(P>0.05)
CRP-2 (POD3-Preop) mean(mg/dl)	35.3	43.4	(P>0.05)
N=	10	10	

Table - 16

Fig-35: C-Reactive Protein (CRP)



ANALYSIS OF DATA

The data was analysed by an independent observer to find any statistical difference between two groups in terms of Japanese Orthopedic Association Score and Neurogenic Claudication Outcome Scores,. Student's t test and chi-square tests were used.

At 1 year follow up the results were classified according to JOA score recovery rate as

Excellent - >75% or more

Good - 50- 74%

Fair - 25-49%

Poor - 24% or less

COMPARISON OF PILOT STUDY AND MY STUDY:

S.NO	CONTENTS	CMD		LSPSD	
		Pilot study	My study	Pilot study	My study
1.	No. of patients	16	10	18	10
2.	Average age (years)	69	60.4	71	58.9
3.	Male: Female	8:8	4:6	10:8	5:5
4.	Mean No. of decompressed levels	1.5	1.30	1.4	1.20
5.	Associated protruded disc removal	10	6	12	7
6.	Blood loss (ml)	103	126	119	130
7.	Time (min)	69	71.23	82	80.25
8.	JOA Recovery rate	74%	63.69%	75%	69.52%

Table - 9

DISCUSSION

With 20 patients, we have presented the prospective randomized control study comparing short term functional outcome of Lumbar Spinous Process Splitting Decompression with Conventional midline decompression by laminectomy.

The two groups of our study were comparable to each other in terms of patient characteristics like age and sex. The patients in our study (mean age 59.65 years) which parallels the average life expectancy in India (65 years), degenerative canal stenosis affects more females than males.

The average intra operative blood loss incurred in the Lumbar Spinous Process Splitting Decompression group (130 ml) is more than that in the Conventional Midline Decompression by laminectomy group (126 ml).

.

In our study the complications were few and were comparable between groups.

Other complications like dural tear (one patient 10%) and wound dehiscence (one patient 10%) were observed equal in frequency in both the groups. Conventional Midline Decompression by laminectomy group, as also the post operative morbidity like UTI, LRI (14.3%) etc .The average ambulation time in Lumbar Spinous Process Splitting Decompression (4.45 days) is less when compared to Conventional Midline Decompression by laminectomy (6.52 days). Post operative radiological evaluation to assess the instability was not routinely performed and when the clinical symptoms and signs of back pain and claudication persist, X-rays of lateral view, flexion and extension view was taken to rule out post operative instability. One patient developed instability in the last follow up in Conventional Midline Decompression group, later posterior fusion and pedicle screw instrumentation were done. .

The complications are in the expected frequency. No case of new neurological deficit was observed following surgery in both the groups. Hence Lumbar Spinous Process Splitting Decompression appears to have safety profile comparable with Conventional Midline Decompression in terms of early

mobilisation and decreased back pain VAS due to preservation of posterior musculoligamentous complex.

Japanese Orthopaedic Association (JOA) Score and recovery rate:

The two (LSPSD and CMD) groups were comparable in terms of the preoperative JOA scores (5.4 and 5.3). The postoperative JOA scores at last follow up (12.5 and 11.5 respectively) and change in JOA score (7.1 and 6.0 respectively) did not show any statistically significant difference.

However a closer analysis reveals that although the Conventional Midline Decompression group was marginally more symptomatic than the Lumbar Spinous Process Splitting Decompression group preoperatively, at the final follow up, the Conventional Midline Decompression group fared better in terms of absolute values of JOA score and JOA recovery rate which is statistically insignificant.

Analysis of the recovery rate showed that 70% of patients in the Conventional Midline Decompression group had good or excellent outcome while the Lumbar Spinous Process Splitting Decompression group fared better with 100% patients experiencing good or excellent outcome. These findings demonstrate a marginally better outcome for the Lumbar Spinous Process Splitting Decompression Although the difference is not statistically significant, it does represent a clinically important observation.

Neurogenic Claudication Outcome Score (NCOS):

Analysis of the NCOS revealed that the Lumbar Spinous Process Splitting Decompression group fared slightly better than the Midline Decompression group in terms of mean change in NCO Score (37.80 & 37.50 respectively). However the difference was statistically insignificant. NCO Score is purely symptom oriented and no importance is given for the clinical findings. This is in contrast to the JOA score where both clinical findings and symptoms are given equal importance.

Back Pain Visual Analog Scale:

There was a significant difference between two groups at the last follow up in the Visual analog score for Back Pain (BPVAS).This signifies that the Lumbar Spinous Process Splitting Decompression technique have comparable outcome with regard to back pain.

Claudication Visual Analog Scale:

No significant difference was observed between Lumbar Spinous Process Splitting Decompression group and Conventional Midline Decompression groups in the Visual analog score for neurogenic claudication (NCVAS) at the last follow up.

Creatine Phosphokinase(CPK MM)

We measured the rise in CPK MM values on postoperative Day 1 and Day 3 compared to thepreoperative values. The average rise on POD1 and POD3 were higher in CMD (253 and 240U/L) compared to LSPSD (225 and 212 U/L).

Levels of CRP showed similar trends on POD1 and POD3. However the differences between groups were not statistically significant. CPK MM levels are dependent on the paraspinal muscle damage due to elevation from the posterior bony elements as well as the duration and pressure of retraction. Our findings suggest more paraspinal muscle damage with CMD than LSPSD which may forerun paraspinal muscle atrophy in the long term. However long-term follow up is required to substantiate this assumption.

This signifies that both techniques have comparable outcome with regard to leg pain.

There was no statistically significant difference between the 2 different surgical techniques regarding the postoperative results.

A concern about Lumbar Spinous Process Splitting Decompression is the difficulty in decompressing the lateral recess and foramen due to difficulty in retracting the paraspinal muscles along with spinous process in single level decompression. We didn't use the visual analog scale to assess radicular symptoms. The improvement in neurological symptoms following surgery was assessed in all cases. In 70% of the patients who underwent Lumbar Spinous Process Splitting

Decompression neurological symptoms improved. Less invasive surgery causes less soft tissue trauma, results in a shorter hospital stay, and there is less need for analgesics (NSAIDS). Hospital-related complications such as infections, pneumonia, and urinary retention also occur less frequently.

Major improvement was noted regarding the increase in the postoperative walking distance and decrease in back pain. However long-term follow up is required to substantiate this assumption.

The main advantages of Lumbar Spinous Process Splitting Decompression are the preservation of posterior musculoligamentous complex and bony structure which prevents surgically induced instability. Only the hypertrophied and compressive medial parts of the facet joints are resected. Midline ligamentous structures are preserved.

CONCLUSION

In our study, Lumbar Spinous Process Splitting Decompression provides minimal exposure for decompression in lumbar canal stenosis while preserving musculoligamentous attachments of the posterior elements of spine and good postoperative results after one year with favourable outcomes of atleast 70% on the Japanese orthopaedic association score and Neurogenic claudication outcome score. With both these surgical techniques, a significant improvement in the outcome after surgical decompression could be demonstrated. There was no significant difference between the Lumbar Spinous Process Splitting decompression and Midline decompression by laminectomy techniques regarding the later outcome.

But Lumbar Spinous Process Splitting decompressive approach is not suitable for cases with bilateral intervertebral disc protrusion and bilateral fac et joint arthritis with hypertrophy causing degenerative lumbar canal stenosis and foraminal stenosis.

However, minimal invasive procedure seems to be more favourable in elderly patients in the early post operative period. However, long term results still need to be evaluated further.

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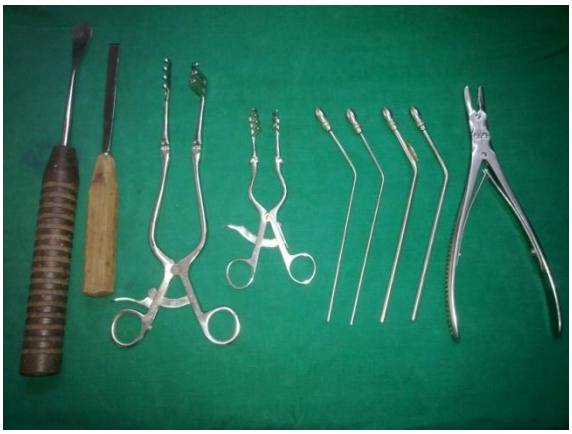
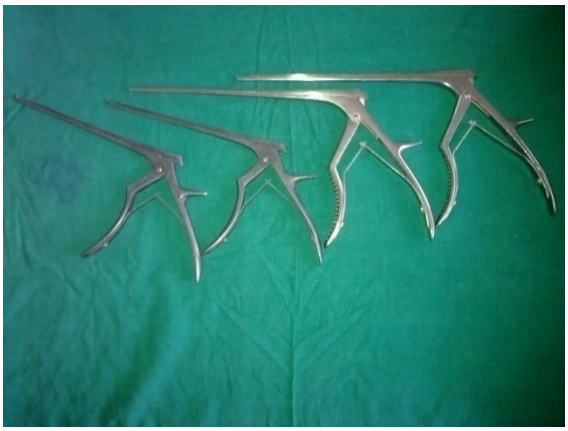
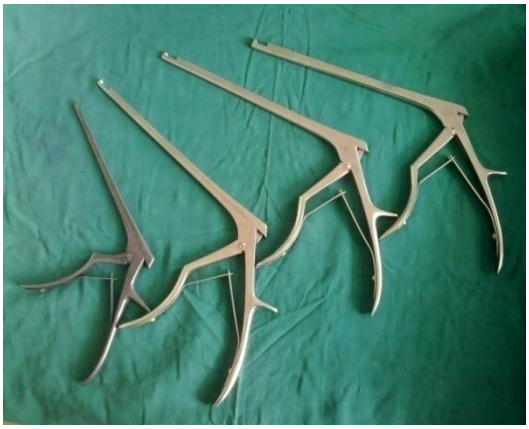
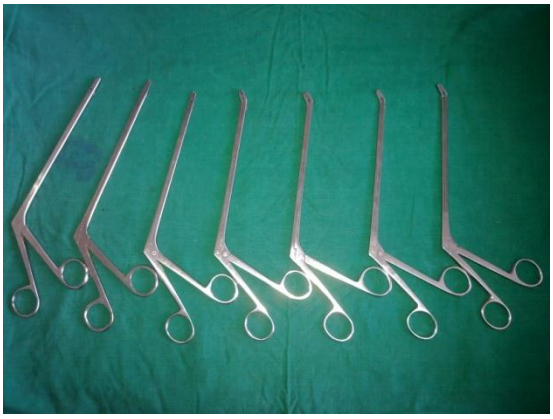
ILLUSTRATION:

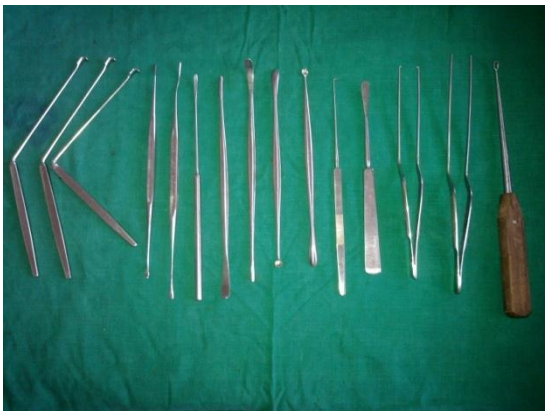
INSTRUMENTS





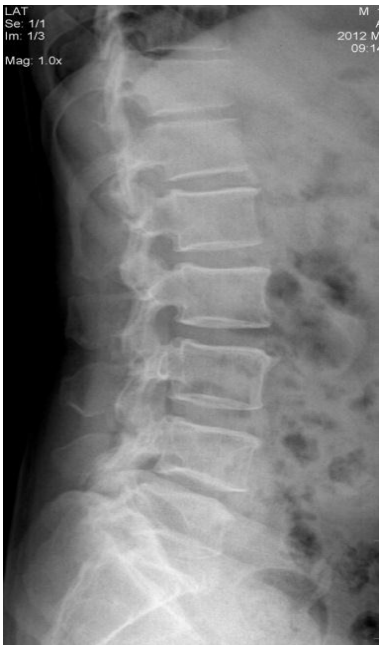




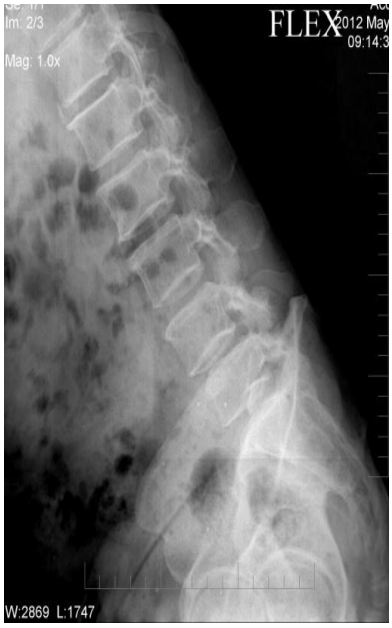


CONVENTIONAL MIDLINE DECOMPRESSION (LAMINECTOMY)

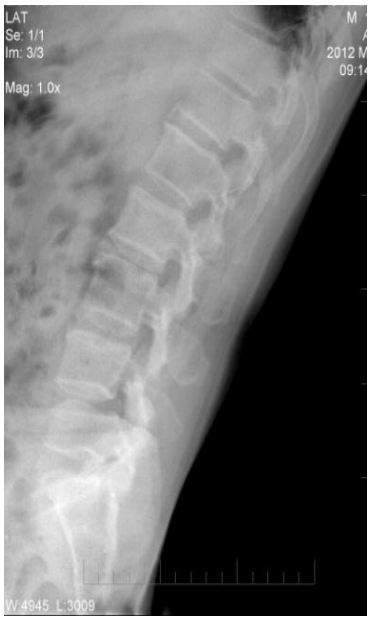
CASE 1: kannammal 66 yr Female



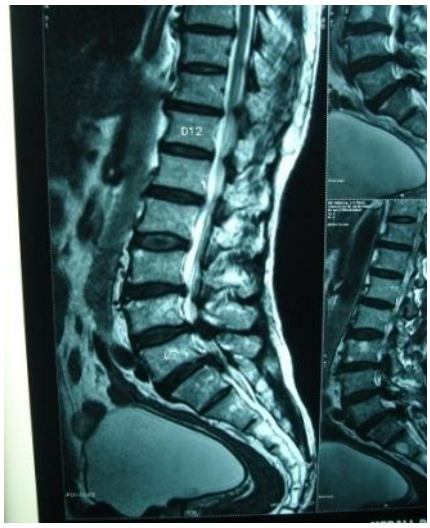
Lateral view



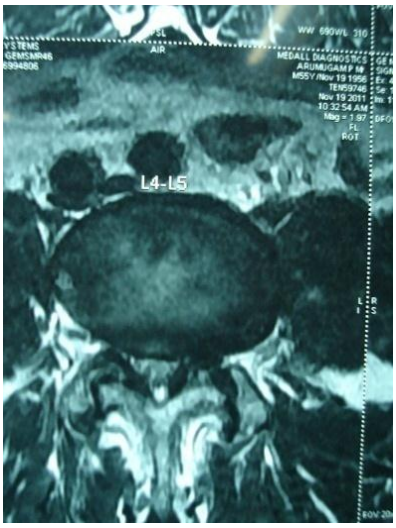
Flexion view



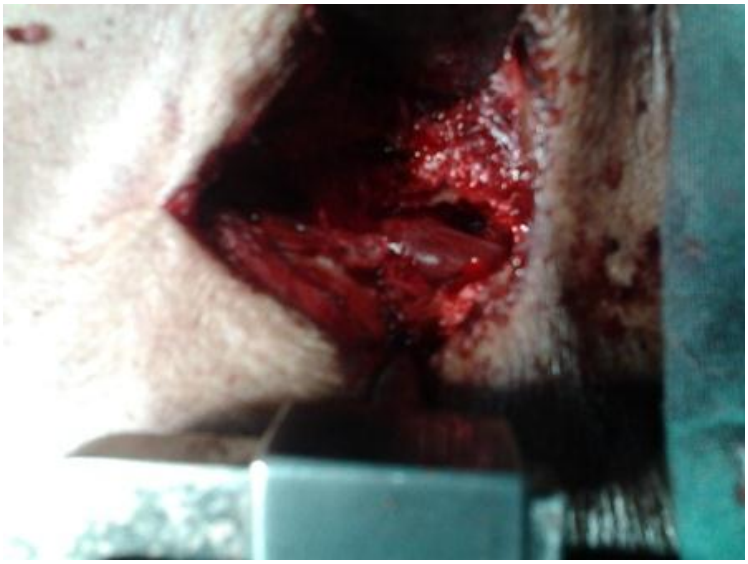
Extension view



MRI- Sagittal view



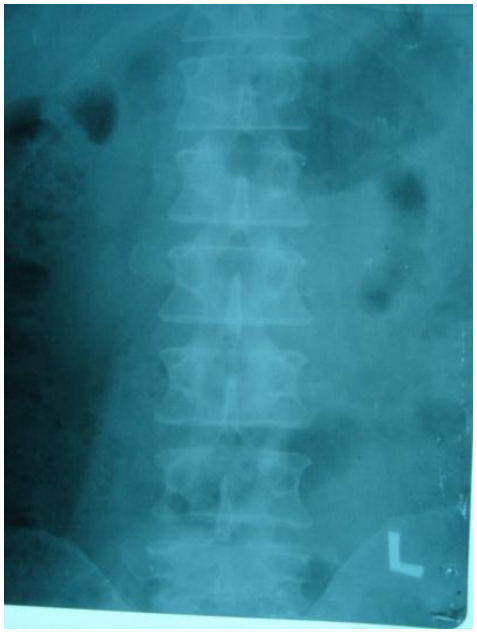
Coronal section

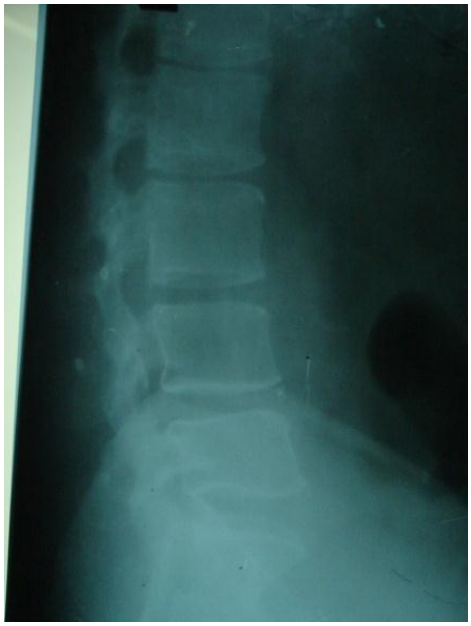


` **Cord decompression**

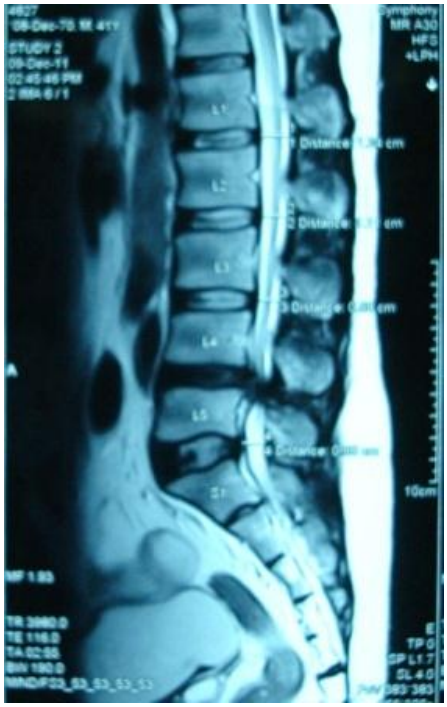
Post operative – Day 10

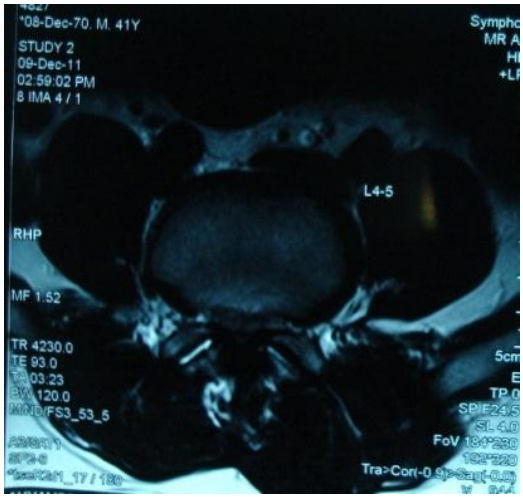
CASE 2: Subbammal 55 yr Female



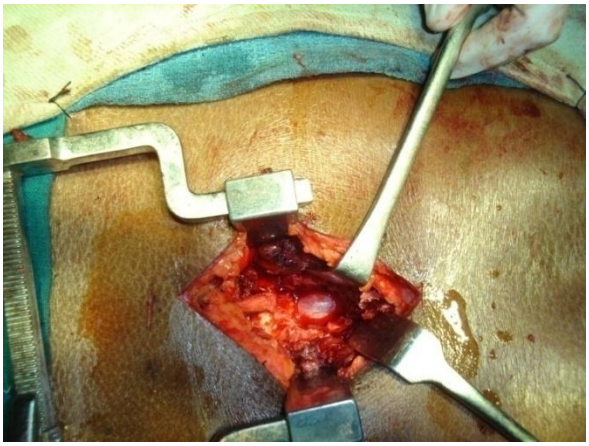


X rays

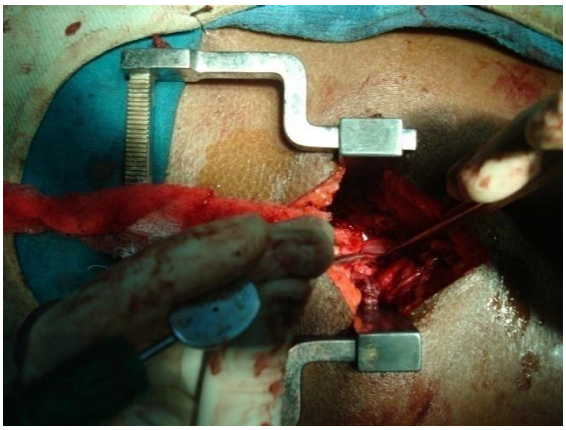




MRI



Cord decompression

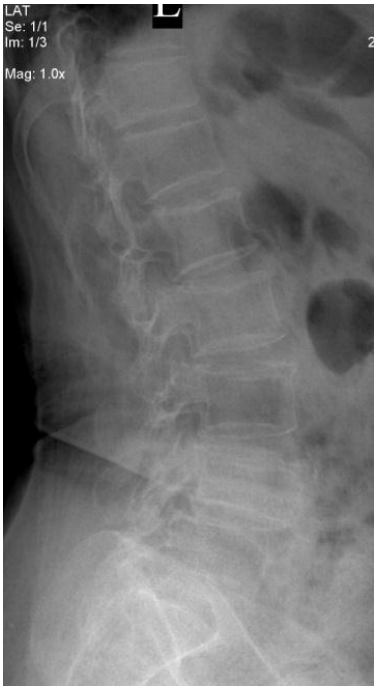


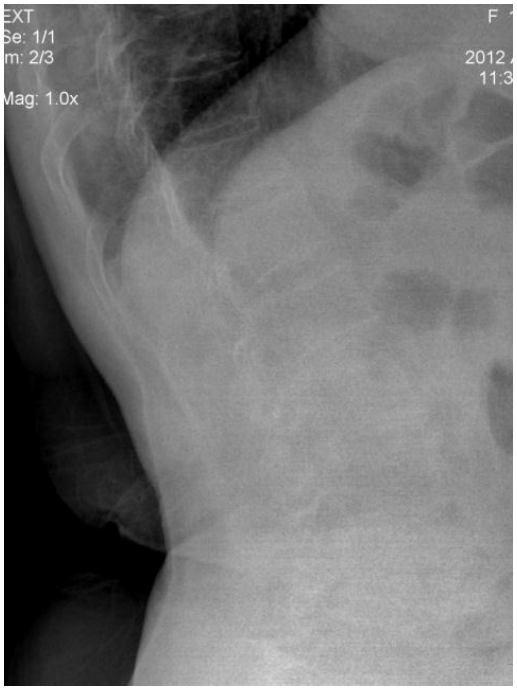
Root decompression

8th post operative day

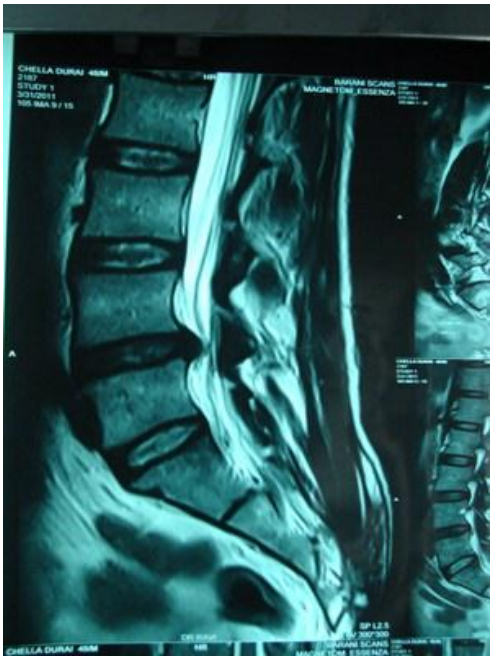
DECOMPRESSION BY LUMBAR SPINOUS PROCESS SPLITTING :

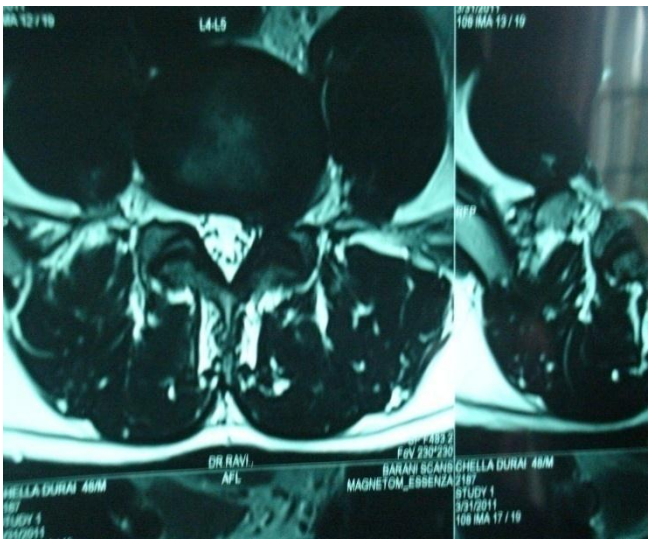
CASE 1: Mr. Kuppaiyandi 57/M



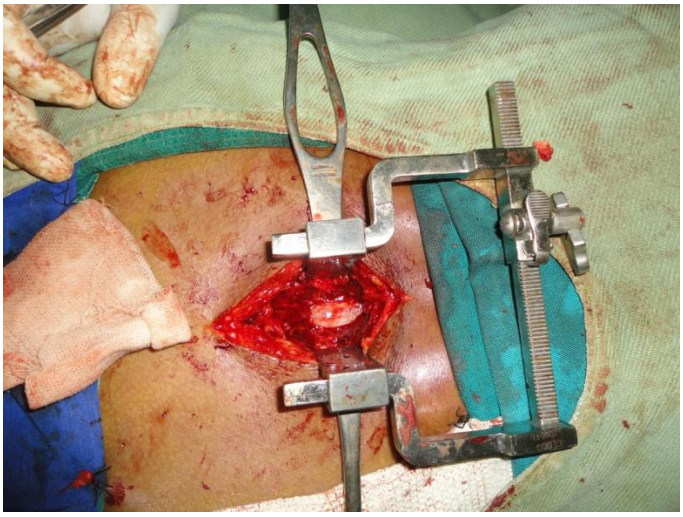


X Rays

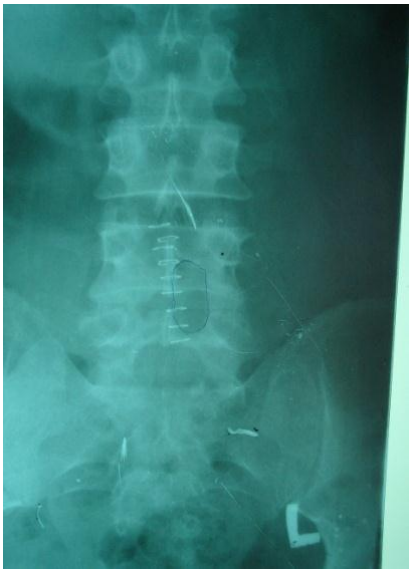




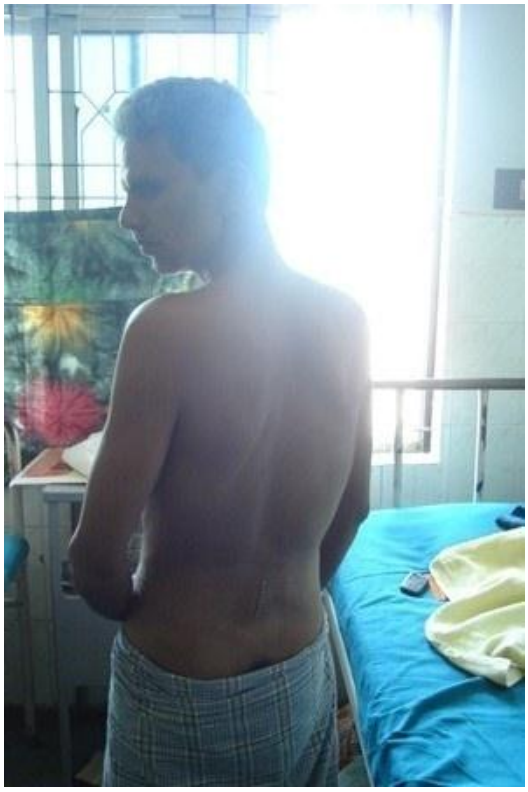
MRI

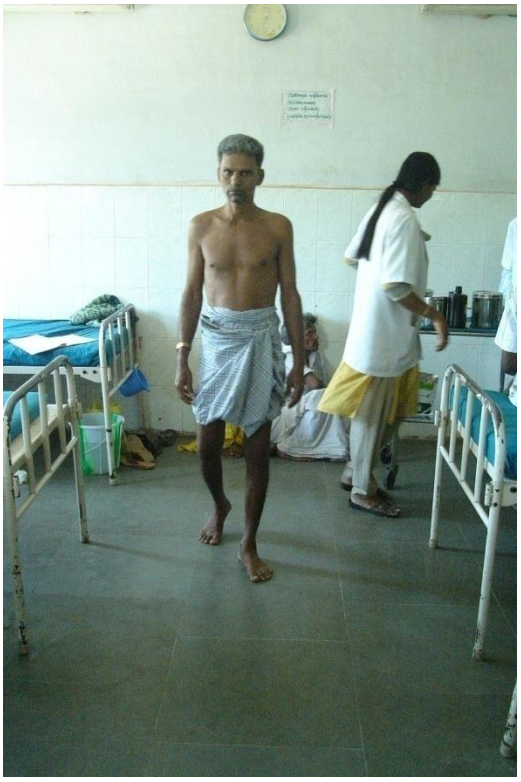


Cord decompression



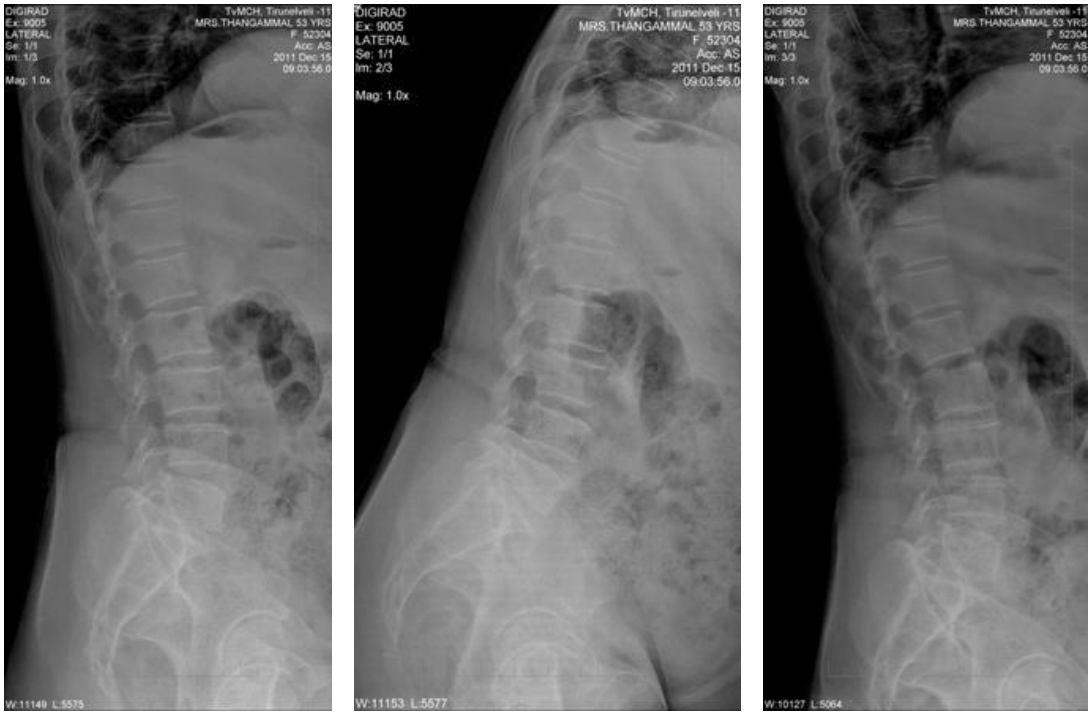
Post op X ray



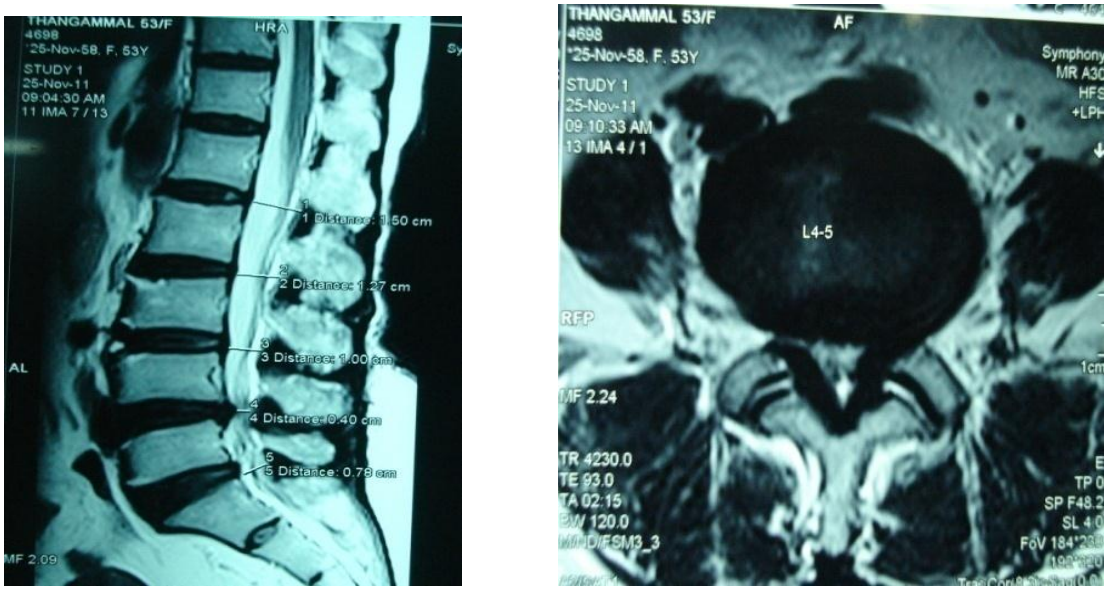


Post operative day 5- Mobilization

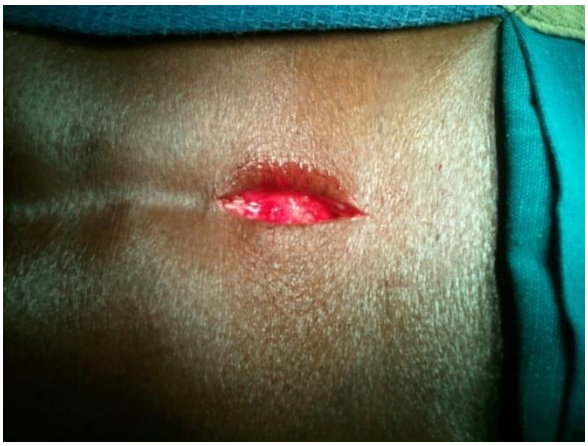
CASE 2:Mrs. Chitra 58/F



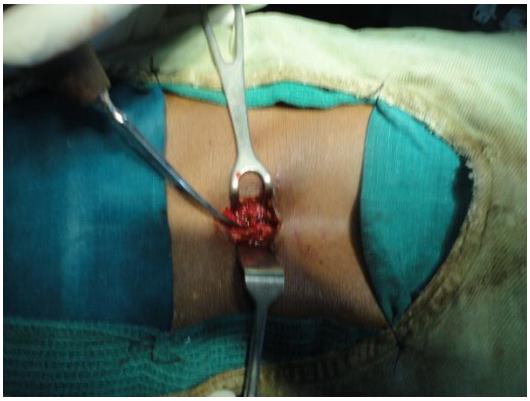
X rays



MRI



Skin incision



spinous process splitting

Post operative day 1

Neurological examination

Post operative day 4

Patient ambulation

MASTER CHART

LUMBAR SPINOUS PROCESS SPLITTING DECOMPRESSION (LSPSD)

S.NO.	NAME	AGE/ SEX	DIAGNOSIS	JOA			NCOS			BPVAS		NCVAS		JOA RECOVERY RATE	RESULTS
				PRE OP	POST OP	CHANGE IN JOA	PRE OP	POST OP	CHANGE IN NCOS	PRE OP	POST OP	PRE OP	POST OP		
1	Radha	60/F	Degenerative disc disease L4-5/L5-S1	6	12	6	32	60	32	7	2	8	2	66.67%	Good
2	Kuppaiyandi	57/M	LCS L3-4/L4-5	5	10	5	26	68	42	7	3	8	2	50%	Fair
3	Petchiyammal	65/F	LCS with Lt radiculopathy L4-5	6	13	7	29	68	39	8	3	7	2	77.77%	Excellent
4	Lakhshmanan	60/M	LCS with neurogenic claudication	5	13	8	24	69	45	9	4	9	2	80%	Excellent
5	Joseph	55/M	LCS with B/L neurogenic claudication, IVDP L4-5	3	11	8	33	71	38	8	3	7	1	66.67%	Good
6	Essakiammal	54/F	Degenerative disc disease L4-5	7	13	6	22	65	43	7	2	8	3	75%	Excellent
7	Ganesan	63/M	LCS with neurogenic claudication	5	13	8	32	64	32	7	2	8	2	80%	Excellent
8	Chitra	58/F	Degenerative disc disease L4-5	5	12	7	30	60	30	8	2	7	2	70%	Good
9	Lakshmi	55/F	LCS with neurogenic claudication	6	12	4	26	70	44	9	3	8	3	66.67%	Fair
10	Madasamy	62/M	Degenerative disc disease L4-5	7	12	7	29	57	28	8	3	7	2	62.5%	Good

MASTER CHART

CONVENTIONAL MIDLINE DECOMPRESSION (LAMINECTOMY)

S.NO.	NAME	AGE/ SEX	DIAGNOSIS	JOA			NCOS			BPVAS		NCVAS		JOA RECOVERY RATE	RESULTS
				PRE OP	POST OP	CHANGE IN JOA	PRE OP	POST OP	CHANGE IN NCOS	PRE OP	POST OP	PRE OP	POST OP		
1	Kannammal	60/F	LCS with neurogenic claudication, , IVDP L4-5	6	13	7	30	65	35	8	5	7	2	77.77%	Excellent
2	Muthaiah	63/M	LCS with B/L Radiculopathy,L3-4/L4-5	4	12	8	28	69	41	7	4	7	2	72.73%	Good
3	Chelladurai	58/M	LCS with neurogenic claudication	5	13	8	21	72	51	8	4	7	2	80%	Excellent
4	Thangapandi	63/M	LCS with neurogenic claudication	4	12	8	27	70	43	8	4	8	2	72.73%	Good
5	Sundari	65/F	Degenerative disc disease L4-5	7	10	3	28	65	37	7	4	8	3	37.5%	Fair
6	Subbammal	55/F	LCS with neurogenic claudication	6	12	6	32	64	32	8	3	8	2	66.67%	Good
7	Parvathy	57/F	LCS with Rt Radiculopathy	6	10	4	31	79	48	8	3	8	2	44.44%	Fair
8	Rajakili	60/F	Degenerative disc disease L4-5/ L5-S1	7	13	6	26	83	57	9	4	8	2	75%	Excellent
9	Karpagam	59/F	LCS with neurogenic claudication	5	11	6	26	68	42	7	3	8	1	60%	Good
10	Mahalingam	64/M	Degenerative disc disease L4-5, L5-S1	3	9	6	29	46	19	7	3	8	2	50%	Good