Journal Homepage: - www.journalijar.com

INTERNATIONAL JOURNAL OF ADVANCED RESEARCH (IJAR)

Article DOI: 10.21474/IJAR01/13939
DOI URL: http://dx.doi.org/10.21474/IJAR01/13939

ISSN NO. 2320-5407

RESEARCH ARTICLE

THE UNDERLYING PATHOPHYSIOLOGY AND CLINICAL APPEARANCE OF LSS, AND THE AVAILABLE DIAGNOSTIC AND THERAPEUTIC OPTIONS

Divya Singh[1], Rahul Singh[2], P.K. Sharma[3] and Sonia Jaiswal[4]

1. Ph.D. Scholar, Department of Anatomy, Era Medical College, Lucknow, India, 2117.
2. Consultant Psychiatrist, Barabanki District Hospital.
3. H.O.D. &Professor, Era's Lucknow Medical College and Hospital.
4. Assistant Professor, Era's Lucknow Medical College and Hospital.

Manuscript Info

Manuscript History
Received: 15 October 2021
Final Accepted: 18 November 2021
Published: December 2021

Key words:-
Lumbar Spinal Stenosis, Back Pain, Diagnosis

Abstract

The term lumbar spinal stenosis (LSS) refers to the narrowing of the spinal canal due to anatomical reasons, and it is linked to a variety of clinical symptoms.The onset of stenosis LSS can be unilateral or bilateral, monosegmental or multisegmental. The stenosis can be characterized as central, lateral, or foraminal anatomically.Neurogenic claudication is the most prevalent symptom of LSS, which is defined as limping or cramping lumbar pain that spreads into the legs primarily during walking.Typical patient symptoms include unilateral or bilateral (exertional) back and leg discomfort that develops over months, if not years.As people live longer and seek a better quality of life, as well as increased knowledge of the condition and the availability of improved imaging techniques, the number of people diagnosed with degenerative LSS has increased.

Introduction:-

The term lumbar spinal stenosis (LSS) refers to the narrowing of the spinal canal due to anatomical reasons, and it is linked to a variety of clinical symptoms. LSS has a documented annual incidence of five instances per 100,000 people, which is four times higher than the incidence of cervical spinal stenosis [1].

Neurogenic claudication, a term coined by Dejerine (1911)2 and described by von Gelderen (1948)3 and later, Verbiest, is the defining sign of LSS (1954). "Localized, bony discoligamentous constriction of the spinal canal that is associated with a complex of clinical signs and symptoms including back pain and stress-related symptoms in the legs," von Gelderen wrote in his paper (claudication) [2-4].

This classification is still used today. Because of the improved quality and availability of radiological imaging, LSS has become the most prevalent indication for lumbar spine surgery [5]. The increased use of LSS surgery reflects the older population's increased demand for mobility and flexibility. Controlled, evidence-based advice for individual treatment decisions is beginning to emerge, propelled by the rising prevalence of this disorder[5–7].

Primary stenosis is produced by a congenital narrowing of the spinal canal[8,9], whereas secondary stenosis is caused by a variety of diseases, the most common of which is chronic degeneration, resulting in a destabilized

Corresponding Author:- Divya Singh
Address:- Ph.D. Scholar Department of Anatomy Era Medical College, Lucknow, India.

vertebral body. Rheumatoid disorders, osteomyelitis, trauma, tumors, and, in rare situations, Cushing illness or iatrogenic cortisone injection are further causes of secondary stenosis [10].

The underlying pathophysiology of LSS, with a focus on degenerative LSS, is discussed in this review, as well as the clinical hallmarks of the ensuing clinical syndrome, paraclinical determinants of the disorder, and the outcomes of interventional trials.

Pathophysiology

The onset of stenosis LSS can be unilateral or bilateral, monosegmental or multisegmental. The stenosis can be characterized as central, lateral, or foraminal anatomically. Central, lateral, and foraminal stenosis can develop alone or in combination, depending on the amount of the degeneration. LSS most commonly affects the L4–5 spinal discs, followed by the L3–4, L5–S1, and L1–2.

Multiple variables can play a role in the development of spinal stenosis, and these factors might work together to exacerbate the illness.

1. Protrusion of the vertebral disc occurs frequently as a result of degeneration, resulting in ventral constriction of the spinal canal (central stenosis).
2. The height of the intervertebral space is lowered as a result of disc degeneration, causing the recess and intervertebral foramina to narrow (foraminal stenosis), putting strain on the facet joints.
3. Increased load can cause facet joint arthrosis, joint capsule hypertrophy, and the development of expanding joint cysts (lateral stenosis), all of which contribute to spinal instability [11].
4. The ligamentaflava forms wrinkles as the segment's height decreases, putting pressure on the spinal dura from the dorsal side (central stenosis).
5. Concurrent instability caused by loosened tendons (such as the ligamentaflava) propagates preexisting hypertrophic alterations in the soft tissue and osteophytes, resulting in the trefoil-shaped narrowing of the central canal [11–16].
6. LSS can also be split into relative and absolute LSS based on the anterior–posterior diameter of the spinal canal—a categorization that has yet to be clinically confirmed. Absolute LSS (spinal canal 10mm in diameter; physiological value is 22–25mm) is often symptomatic and is associated with the absence of free subarachnoid space (as seen on lateral plain X-ray films).
7. Relative LSS (spinal canal 10–12mm in diameter; physiological value is 22–25mm) is usually asymptomatic. If the lateral recess has a diameter of less than 2mm (physiological diameter is 3–5mm), it is deemed stenotic.

From stenosis to claudication

Each of the degenerative mechanisms that contribute to the development of LSS might produce clinical signs on its own, making diagnosis and treatment selection difficult. **Neurogenic claudication is the most prevalent symptom of LSS, which is defined as limping or cramping lumbar pain that spreads into the legs primarily during walking (Figure 1).**

Nerve root compression causes localized inflammation, which changes the excitatory condition of the nerve root [18].

Furthermore, at least two interdependent vascular mechanisms are thought to play a role in the development of neurogenic claudication in LSS

1. Reduced arterial blood flow, which causes ischemia, and venous congestion, which causes nerve compression and subsequent perfusion insufficiency[19, 20].
2. Compressive radiculopathy, on the other hand, might result in autonomic dysregulation and poor circulation in the legs[21].

Classification according to etiology

Primary stenosis

1. idiopathic stenosis
2. Achondrodysplasia

Secondary stenosis

1. Degenerative (for example, spondylosis, spondylolisthesis, scoliosis)

2. Ossification of the ligamentumlongitudinaleposterius and ligamentumflavum
3. Metabolic or endocrine causes (for example, epidural lipomatosis, acromegaly)
4. Infections (discitis, osteomyelitis, Pott's disease [tuberculous spondylitis])
5. Neoplastic
6. Rheumatological conditions (for example, Paget disease, spondylosisankylopoetica, rheumatoid arthritis)
7. Posttraumatic or postoperative stenosis (for example, fracture of vertebrae, laminectomy, fusion, fibrosis)

Classification according to anatomy
1. Central stenosis (with or without lateral stenosis)
2. Isolated lateral stenosis
3. Foraminal stenosis

Differential diagnoses
1. Intermittent claudication or vascular claudication
2. Radiculopathies or polyneuropathies
3. intraspinal synovial cyst
4. Disc prolapse
5. Tethered cord or spina bifida
6. Coxarthrosis or arthrosis of the iliosacral joint
7. Abdominal aortic aneurysm
8. Neoplasia (for example, tumor of myelon, spinal roots, meninges, bones or filiae)
9. Inflammatory conditions (for example, spondylodiscitis, meningeosis, arachnoiditis)
10. Dissociative syndromes

Signs and symptoms
Typical patient symptoms include **unilateral or bilateral (exertional) back and leg discomfort** that develops over months, if not years.

Back discomfort is usually centered in the lumbar spine but can radiate to the gluteal region, groin, and legs, forming a pseudoradicular pattern.

Isolated radiculopathy can arise in patients with lateral recess stenosis or foraminal stenosis.

The most specific symptom of LSS is neurogenic claudication, though it is almost always accompanied by other symptoms.

LSS can be divided into grades I–III based on the severity of the symptoms.
Grade I (neurogenic intermittent claudication) is defined by a shortened walking distance (induced by discomfort) and short intermittent sensomotoric impairments that may be unnoticeable at rest but can worsen while walking [23,24].

Classifications of LSS exist because not all individuals with LSS have symptoms that are compatible with neurogenic intermittent claudication. If there is chronic, progressive paresis with partial pain regression, **grade III is reached.**

Occurrence of pain regression following flexion (delordosis) of the spine in neurogenic claudication can be clinically separated from vascular intermittent claudication (for example, while cycling).
1. Unlike vascular claudication, the pain experience in patients with LSS does not improve when they stand.
2. The proportions of low back pain (an indicator of pathology such as simultaneous vertebral instability or facet joint arthrosis) and leg pain (a marker of pathology such as concomitant vertebral instability or facet joint arthrosis) have proven useful for clinical orientation [25].
3. Lasègue testing (a passive leg flexing test) is usually negative in people with LSS, and it is frequently accompanied by a sensation of 'heavy legs,' a symptom of LSS.
4. A variety of common comorbidities, such as peripheral neuropathies, make it difficult to identify LSS [26-28].
5. Approximately 20% of people with LSS have depressive symptoms, and 25% are dissatisfied with their lives prior to surgery—a pattern similar to that seen in individuals with other chronic diseases [29,30].

6. Patients' mood and happiness must be assessed, since these can differ significantly between patients with LSS and healthy controls, influencing diagnostic and treatment options.
7. Patient-reported symptoms, even if they are fleeting, should be taken seriously during the diagnosis process, particularly during early visits.

Diagnosis

As people live longer and seek a better quality of life, as well as increased knowledge of the condition and the availability of improved imaging techniques, the number of people diagnosed with degenerative LSS has increased. However, because the symptoms of LSS might be similar to those of other disorders, it can be difficult to identify. Various comorbidities, which are common in the elderly population, can, on the other hand, cause secondary stenosis or mimic its symptoms. As a result, distinguishing LSS from a variety of other diseases is critical. At rest, clinical symptoms of LSS are generally absent. Furthermore, determining whether pain (and other patient-reported symptoms) is related to LSS or to other causes might be challenging (for example, instability, facet joint arthrosis, osteoporosis, arthritis, or diabetic polyneuropathy). As a result, the only way to diagnose LSS is to use a combination of clinical history, physical examination, and radiological abnormalities [31,32].

Differential diagnoses

Hyposensibility caused by peripheral neuropathies, in contrast to LSS, usually has a bilateral distal stockingshaped pattern, regardless of posture, rest, or physical stress. When standing or walking, iliosacral joint disease might mimic LSS, with low back pain extending to the buttocks and thighs. Unlike LSS, however, iliosacral joint discomfort is accompanied with joint tenderness. Caudaequina syndrome, which includes sacral hypesthesia, loss of tendon reflexes in the lower limbs, and incontinence, is only seen in rare situations as a result of LSS. The sacral nerves are well protected from compression due to their central location within the caudaequina, hence sphincter involvement is uncommon in LSS. Cervical or thoracic myelopathy must be ruled out in patients with vesicorectal voiding and upper motor neuron symptoms (such as Babinski's reflex and hyperreflexia) [33].

Neuroradiological assessment

Some intrinsic issues with imaging of the lumbar spinal canal must be recognized while doing radiological examination of LSS. For starters, imaging of symptomatic patients is complicated by the fact that degenerative changes in the lumbar spine are common in the asymptomatic population: 20% of persons over 60 years old will show symptoms of LSS. Second, imaging has a tendency to overestimate significant degenerative alterations in the spinal canal and their implications[33].As a result, radiologically diagnosed LSS frequently detects more segments of the body than clinically suspected. A tentative diagnosis of LSS can be made in the vast majority of instances based on the clinical appearance of the disorder and the patient's medical history. When any type of interventional or surgical therapy is being considered, imaging is often used selectively. Specifically, imaging is most commonly employed in patients with moderate to severe LSS symptoms [34].The goal of imaging in presurgical patients with LSS symptoms is to confirm the existence or absence of LSS, rule out other diagnoses, link congesting symptoms to osseous and discoligamentous structures, and pinpoint the specific location of LSS for precise presurgical planning [35]. As previously stated, LSS is primarily caused by degenerative disc disease, at least at first. The various imaging modalities used can visualize morphological abnormalities such as loss of disc height, disc signal, bulging discs, disc herniations, reactive endplate and bone marrow changes, and spondylophytes to varying degrees. Hypertrophic facet degeneration, as well as inwardly buckled and hypertrophied ligamentaflava, result from increased load on the facet joints. Central, lateral, or foraminal stenosis can result from these modifications.

MRI

For the radiological examination of LSS, MRI is the chosen imaging modality. When compared to other imaging modalities, this approach gives higher soft-tissue contrast, has multiplanar imaging capabilities, and does not emit ionizing radiation. However, MRI is contraindicated or impossible to perform in individuals with pacemakers, some other forms of metal implants, or claustrophobia. T1 and T2 weighted images are frequently orthogonal in MRIs of patients with LSS (sagittal and axial). The use of a fat-suppressed T2 weighted sequence appears to enable for more precise detection of related degenerative bone marrow alterations. 'Myelography-like' pictures of the thecal sac, intrathecal and intraforaminal nerve roots and the spinal cord can be generated noninvasively using T2-weighted imaging and the inherent signal intensity of cerebrospinal fluid. LSS can be monosegmental or manifest itself on numerous levels. MRI, like CT imaging, can identify the osseous and discoligamentous structures that contribute to LSS. Despite the fact that the anatomy of the spine is depicted in great detail, research on the clinical utility of the information obtained by MRI has yielded mixed results[36-38]. According to the findings of a study conducted by

modic and colleagues in patients with radiculopathy, low back pain, and sciatica, changes detected by MRI offer little or no therapeutically meaningful information to clinical assessment alone in terms of prognosis and predicting surgery outcome [37,39].Unless previous surgery has been performed and fibroid scar tissue must be detected by its contrast enhancement, gadolinium-based contrast media are not generally required for imaging of LSS [40,41]. However, other investigations suggest that contrastenhancedmrI may have a superior role in LSS patients with neurogenic claudication, as augmentation of compressed nerve roots can be seen in a minority of these patients[42–44]. This improvement could be due to clogged periradicular veins, showing venous stasis, or a breakdown of the blood–nerve barrier, indicating chronic compressive radiculitis. Magnetic resonance (mr) myelography can be conducted noninvasively and without contrast administration using strongly T2 weighted fat suppressed sequences. Despite the ability of this technique to accurately display the thecal sac, investigations on the efficacy of this sequence have produced mixed findings [45,46]. As a result, the use of mrmyelography is only recommended as a supplement to conventional MRI. Open MRI is currently the sole technology that allows for a functional assessment of spinal flexion and extension while axial loading is applied, or even in the supine position [47].

CT scanning

CT scans are quick and accurate, allowing for detailed examination of the spinal canal and discrimination between disc, ligament, and bony structure compression. In terms of the latter, this method outperforms MRI. CT is currently performed with a spiral multislice approach, which acquires isotropic data enabling multiplanar reformatting in any desired plane and threedimensional reconstructions. Multisegmental imaging in one plane is thus conceivable even with significant torsion scoliosis, which is not possible with MRI. Because these structures have similar densities to the cerebrospinal fluid, CT cannot image intrathecal nerve roots or the spinal cord. The use of CT myelography may be able to solve this problem. CT myelography is a procedure that involves spiral CT imaging after inthecal iodine delivery, which is usually done under fluoroscopic guidance. Lumbar puncture can be done under CT guidance in rare cases of significant degenerative or postsurgical alterations. Even in patients who cannot be examined by other means, CT-guided puncture of the thecal sac is a reliable approach. In patients for whom MRI is contraindicated, MRI results are inconclusive, or clinical complaints do not correspond well with MRI findings, CT and CT myelography may be recommended[47]. Furthermore, CT techniques may be employed for presurgical planning in circumstances when good depiction of bone anatomy is required.

Conventional X-rays and myelography

The use of routinely collected plain radiographs in the first assessment of individuals with LSS has been called into question[48,49]. Indeed, the rules for the agency for Health Care Policy and Research no longer include the gathering of such radiographs. Many patients, on the other hand, have traditional radiography performed as part of their initial evaluation because it is a low-cost and simple procedure. Conventional radiographs may be useful in determining the contribution of bone degeneration to LSS and the alignment of the vertebral bodies in the lateral and coronal planes, but only to a limited extent. This approach can also be used to rule out traumatic alterations or other unexpected findings as differential diagnoses (for example, Paget disease, spondylodiscitis, or scoliosis). Plain radiographs taken 50 days following surgery are helpful in establishing the integrity and proper position of fusion material, as well as detecting evidence of loosening of implanted fixating plates and/or screws. Plain radiographs were reported to have a sensitivity and specificity of 66 percent and 98 percent, respectively, when it came to the contribution of bony alterations to central spinal stenosis. Additional lateral radiographs in flexion and extension positions (so-called functional radiographs) are not usually necessary to rule out segmental instability since symptoms of segmental instability can be seen with adequate accuracy on conventional lateral radiographs [51]. Furthermore, according to a recent study, these new perspectives provided no significant benefits. 50 Even in patients who were expected to have segmental instability, the diagnostic significance of lateral radiographs in flexion and extension could not be proven conclusively [52].Traditional functional myelography has long been the gold standard for diagnosing LSS and is still useful for determining the impact of hyperextension and hyperflexion on the stenosis's extent. This technique may still be the only standard way for determining the morphological correlates of a functioning, posture-dependent, symptomatic LSS. Furthermore, for patients with spinal metallic implants, which can generate artifacts on mrI and CT, it is the sole correct imaging approach. Furthermore, standard functional myelography allows the lumber spine to be assessed while standing, and thus under normal body weight stress. Conventional myelography is an intrusive procedure that requires the introduction of an iodinated contrast agent intravenously. As a result, it is linked to side effects include postpunctional headaches and rare life-threatening consequences such allergic responses and spinal infections. Conventional myelography, like other imaging procedures, frequently finds abnormalities that were not detected clinically. The block of contrast flow, which is a good predictor of the favorable outcome of decompression surgery, is one of the few solid prognostic indicators

[53].The inability of conventional myelography to detect the etiology of block or compression, as well as to visualize extrathecal nerve root compression, limits it. As a result, this approach is frequently paired with a CT scan conducted after myelography (postmyelographic CT), which compensates for the limitations of this technique. Preoperative surgical planning may need myelography mixed with postmyelographic CT to assess the thecal sac and the bone condition of the operating area. In conclusion, recent investigations have revealed that traditional myelography, CT myelography, and mrI have similar diagnostic and predictive values [54]. When evaluating imaging modalities, keep in mind that the radiological degree of LSS, both before and after surgery, does not always correlate with the severity of clinical signs and symptoms [54–58].

Additional diagnostics

Selective diagnostic injections may be effective in some patients to quantify the contribution of various pain components to the patient's overall health, particularly in the context of chronic pain psychology.

1. If vascular etiology is suspected, noninvasive diagnostic approaches include calculating the ratio of systolic blood pressure in the ankles to that in the arms (ankle–brachial index), which is abnormal when the result is less than 0.5.
2. Routine duplex Doppler angiography, contrast-enhancedmr angiography, and—in rare circumstances before intervention—digital subtraction angiography can also be used to assess whether vascular genesis is involved in generating pain. An electrophysiological study is only indicated to rule out other illnesses, especially if the distribution of pain and numbness is atypical, due to the limited practical value of classical electromyograghy and nerve conduction investigations in diagnosing LSS (for example, suspicion of peripheral polyneuropathy or myopathy, which might both occur concomitantly with LSS)[55,56]. Although this approach is not yet prevalent in regular practice, walking on a treadmill is an acceptable provocation test for such assessments[55,56]. Routine laboratory tests can be used to diagnose comorbidities such as diabetes or diabetic polyneuropathy (through glucose and Hba1c detection) and infections like spondylodiscitis (by C-reactive protein measurement).

Therapy

Because degenerative LSS is a progressive disorder, entirely curing it is improbable; hence the major goal of any treatment is to minimize the severity of the symptoms. Pain (bothersome indices) and physical function have been the key end targets of contemporary interventional techniques[57]. In the majority of patients, the indications for intervention are not absolute. However, if you have caudaequina syndrome or related paresis, you should get medical help right once.The selection of a suitable method is challenging due to the significant pathological and clinical heterogeneity of LSS, the lack of treatment guidelines, and the huge number of different therapies. 4 Prospective, randomized trials comparing different therapy are desperately needed[58, 59]. The significant economic impact of low back pain, which is projected to surpass uS$100 billion, reflects the need for effective therapy for LSS, with lost productivity at work accounting for the bulk of the overall expenses [60].

Natural disease course

LSS is a degenerative illness that progresses slowly over time, and the neurological abnormalities are minor during the majority of the disease's clinical course. LSS is typically diagnosed in adults over the age of 50 for these reasons. There are, however, no prospective long-term studies that track the normal progression of symptoms over time[58]. This complicates the beginning and selection of a specific therapy; as such decisions should ideally be based on an assessment of the condition's natural course. In several intervention trials, only limited data is available from untreated individuals in subgroups. The Spine Patient Outcomes Research Trial (SPorT) found that most patients in the conservatively treated control group saw no worsening of symptoms over the course of two years. Another study indicated that in 20% of untreated instances, the intensity of symptoms increased, while another study concentrating on pain development over almost 5 years found that 70% of patients' clinical symptoms reached a plateau, 15% suffered pain aggravation, and 15% spontaneously recovered [59]. Given the commonality of long-term clinical stability in LSS, acute exacerbations of symptoms should not be mistaken for a shift in the patient's trajectory.

Conservative therapy

Physical therapy, ergotherapy, behavioral therapy, delordosing orthopedic devices, girdles, acupuncture, manual therapy, and pharmaceutical intervention are among the conservative treatments for LSS [60]. Few studies have been completed to establish the efficacy of conservative therapy in the treatment of LSS, although those that have done so have claimed success rates of up to 70%[61–63]. However, none of the trials available give enough evidence to establish the superiority, or even efficacy, of any of the several conservative therapies [64].In the

absence of evidence-based treatment recommendations, a multidisciplinary, intensive approach should be prioritized above a single therapy [64, 65]. Flexion, distraction, neural mobilization, and alleviation of the afflicted segments are the goals of appropriate physiotherapy and manual therapy procedures, as well as improvements in paravertebral muscle tone through the use of stabilizing exercises[61, 66]. Bed rest is not indicated in the treatment of chronic or acute pain, according to most experts. 64 Individualized advice is critical in LSS, especially in instances with minor symptoms, because a modest change in one's behavior might be enough to stabilize or improve the disease. The pharmaceutical component of conservative therapy is equivalent to the drug used to treat a disc protrusion and is used to ease painful nerve root disorders (herniation). NSAIDs, various peripheral analgesics, steroids, muscle relaxants, opioids, antidepressants, and, in the most severe instances when quality of life is compromised, neuroleptics are used to treat LSS. Weekly therapeutic injections, in addition to oral medicine, can provide short- to medium-term relief. In epidural, deep paravertebral, pararadicular, and facet joint injections, steroids are frequently used with local anesthetics. Invasive operations, on the other hand, are linked to an increased risk of infection. The effectiveness of pharmacological regimens has only been studied in a few trials, as has the case with all conservative treatment options. Long-term use of NSAIDs and muscle relaxants, as well as the usage of steroids, antidepressants, and long-acting opioids, cannot be recommended based on evidence [65]. Similarly, there is no evidence for the effectiveness of therapeutic injections for LSS [67–69].

Surgery

If a diagnosis of LSS has been made based on consistent findings from the clinical history, physical examination, and radiological evaluation, conservative treatment should be used for 3–6 months with the goal of obtaining adequate symptom relief. Surgical intervention is advised in individuals who have severe symptoms and functional impairment, unless this technique is contraindicated for other reasons. Clinicians should also keep in mind that some patients, while matching these criteria, simply may not want surgery, while others have false expectations of what surgical techniques can accomplish[70]. In LSS, all surgical treatments are designed to release the entrapped neural components while maintaining the segment's integrity. Such decompression operation frequently results in pain reduction in the legs and, to a lesser extent, in the low back [71].Even if pressure on nerve roots, dura, and blood vessels is properly relieved, the time and amount of recovery is uncertain. Laminectomy and hemilaminectomy, hemilaminotomy, fenestration, foraminotomy, and the implantation of interspinous distraction devices are all decompressive surgical treatments [72,73]. Decompression surgery has a complication rate of 14 percent (during and after the surgical treatment) 57 percent to 35 percent or more [74–76]. Fusion surgery, which is more intrusive than decompression surgery and is utilized in situations of instability, is linked to a greater likelihood of complications. Dura vascular lacerations, epidural hematomas, insufficient decompression with severe residual stenosis, instability, and reossification are all common consequences of both decompression and fusion surgery. All of these issues lead to a re-compression of the nerves [75–78]. Reoperation rates following decompressive surgical operations have been found to vary from 10% to 23% after ten years. In one research [71,79] extra fusion surgeries reduced the incidence of reoperation. Because instability can produce spinal root congestion, additive fusion surgery may be required in situations of instability (vertebral body mobility >3 mm), spondylolisthesis (>5 mm forward movement of a lumbar vertebra relative to one below)80, or scoliosis (lateral curvature of the spine) >20°,81. In the literature, success rates for decompression surgery in instances with LSS range from 40 to 90 percent, depending on a number of criteria such as the kind of decompression, the length of follow-up, the age of the patients, and comorbidities[82–88]. According to one study, individuals who had a laminotomy were more likely to have a significant improvement in lumbago than those who had a laminectomy. At one year after surgery, a randomized experiment found that bilateral laminotomy provided higher clinical benefit than unilateral laminotomy or laminectomy in individuals with LSS.Another research compared the 1-year outcomes of tissue sparing—also known as undercutting decompression—with those of the more invasive laminectomy operation and found no statistically significant difference. 91 percent of patients who had a unilateral foraminotomy for degenerative foraminal stenosis reported a reduction in leg discomfort, albeit one-third of them also experienced an increase in lumbago. Although a 90-degree excision of the pars interarticularis does not appear to produce segment instability, it may increase the incidence of lumbalgias [89-91].For central LSS, laminoplasty is indicated since two-thirds of patients recover after six years [92].Laminoplasty can give satisfactory outcomes, similar to those obtained with additive fusion, even in situations of moderate degenerative spondylolistheses (complicated stenosis)[86]. There is currently no consensus on whether simply the symptomatic level and side should be decompressed, or if additional nonsymptomatic—but clearly confirmed—neighboring stenosesshould be decompressed as well [93–96]. In general, as with preoperative diagnosis, the paraclinical evaluation of a successful surgical intervention is complicated by the lack of correlation between imaging data and clinical presentation.

Conclusions:-

LSS is becoming a more common diagnosis, and clinicians are treating it in a variety of ways. Clinical experience and developing guidance from trial-based data, which is beginning to meet stringent evidence-based medicine requirements, are used to make treatment decisions. Surgery is frequently indicated for severe LSS with growing neurological impairments and severe neurogenic claudication, although the choice to operate is based on clinical experience rather than scientific data. By suggesting surgery exclusively for the most severe instances of LSS, the available data have been skewed, making the choice between conservative therapy and surgery more difficult. Several metaanalyses have been conducted to compare the efficacy of conservative therapy against surgery. Until recently, metaanalyses (including Cochrane reviews) of surgical treatments for spinal stenosis found insufficient evidence to recommend surgery over nonsurgical therapy. With the release of the maine Lumbar Spine Study, which published the 8–10 year outcome results for conservative versus surgical LSS therapy, the importance of diligent follow-up became clear.For individuals with LSS, short-term (1 year) to mid-term (4 year) data revealed that surgery was more helpful than conservative care. Regardless of the original treatment modality, about half of the patients reported a reduction in low back pain after 8–10 years. The maine Lumbar Spine Study has been criticized for using nonrandomly assigned patients, which has an impact on the degree of evidence obtained by trials. After 4 and 10 years of follow-up, a prospective study discovered a superior clinical outcome following surgery than in a control group receiving conservative therapy. However, because the research was only partially randomized (31 out of 100 patients) and 20% of the enrolled patients were lost to follow-up, the findings can only be classified as level 2b evidence. The authors advocated conservative treatment in the first instance because there was no difference in clinical result between patients who were operated on soon after being diagnosed and those who underwent surgery after first receiving physiotherapy.

Reference:-

1. Johnsson, K. e. Lumbar spinal stenosis. A retrospective study of 163 cases in southern sweden. ActaOrthop. Scand. 66, 403–405 (1995).
2. Dejerine, J. intermittent claudication of the spinal cord [French]. Press Med. 19, 981–984 (1911).
3. vanGelderen, C. An orthotic (lordotic) cauda syndrome [German]. ActaPsychiatr. Neurol. 23, 57–68 (1948).
4. verbiest, H. A radicular syndrome from developmental narrowing of the lumbal canal. J. Bone Joint Surg. 36-B, 230–237 (1954).
5. Lurie, J. D., Birkmeyer, N. J. &weinstein, J. N. rates of advanced spinal imaging and spine surgery. Spine 28, 616–620 (2003).
6. Atlas, s. J. &Delitto, A. spinal stenosis: surgical versus nonsurgical treatment. Clin. Orthop. Relat. Res. 443, 198–207 (2006).
7. Deyo, r. A., Ciol, M. A., Cherkin, D. C., Loeser, J. D. &Bigos, s. J. Lumbar spinal fusion. A cohort study of complications, reoperations, and resource use in the Medicare population. Spine 18, 1463–1470 (1993).
8. singh, K. et al. Congenital lumbar spinal stenosis: a prospective, control-matched, cohort radiographic analysis. Spine J. 5, 615–622 (2005).
9. Tubbs, r. s. & Oakes, w. J. An unusual presentation of achondroplasia. Case report. J. Neurosurg. 103 (Suppl.), 170–171 (2005).
10. Fogel, G. r., Cunningham, P. Y. 3rd &esses, s. i. spinal epidural lipomatosis: case reports, literature review and meta-analysis. Spine J. 5, 202–211 (2005).
11. schulte, T. L. et al. Lumbar spinal stenosis. Orthopade [German] 35, 675–692 (2006).
12. DiMaio, s., Marmor, e., Albrecht, s. & Mohr, G. Ligamentumflavum cysts causing incapacitating lumbar spinal stenosis. Can. J. Neurol. Sci. 32, 237–242 (2005).
13. Park, J. B., Lee, J. K., Park, s. J. &riew, K. D. Hypertrophy of ligamentumflavum in lumbar spinal stenosis associated with increased proteinase inhibitor concentration. J. Bone Joint Surg. Am. 87, 2750–2757 (2005).
14. sairyo, K. et al. Pathomechanism of ligamentumflavum hypertrophy: a multidisciplinary investigation based on clinical, biomechanical, histologic, and biologic assessments. Spine 30, 2649–2656 (2005).
15. Yayama, T. et al. Pathogenesis of calcium crystal deposition in the ligamentumflavum correlates with lumbar spinal canal stenosis. Clin. Exp. Rheumatol. 23, 637–643 (2005).
16. Kawaguchi, Y. et al. spinal stenosis due to ossified lumbar lesions. J. Neurosurg. Spine 3, 262–270 (2005).
17. rydevik, B., Brown, M. D. &Lundborg G. Pathoanatomy and pathophysiology of nerve root compression. Spine 9, 7–15 (1984).
18. Kobayashi, s. et al. effect of lumbar nerve root compression on primary sensory neurons and their central branches: changes in the nociceptive neuropeptides substance P and somatostatin. Spine 30, 276–282 (2005).

19. Kobayashi, s. et al. Blood circulation of caudaequina and nerve root [Japanese]. Clin. Calcium 15, 63–72 (2005).
20. Porter, r. w. spinal stenosis and neurogenic claudication. Spine 21, 2046–2052 (1996).
21. Chosa, e., sekimoto, T., Kubo, s. & Tajima, N. evaluation of circulatory compromise in the leg in lumbar spinal canal stenosis. Clin. Orthop. Relat. Res. 431, 129–133 (2005).
22. Danielson, B. &willén, J. Axially loaded magnetic resonance image of the lumbar spine in asymptomatic individuals. Spine 26, 2601–2606 (2001).
23. Takahashi, K. et al. Changes in epidural pressure during walking in patients with lumbar spinal stenosis. Spine 20, 2746–2749 (1995).
24. Takahashi, K., Miyazaki, T., Takino, T., Matsui, T. & Tomita, K. epidural pressure measurements. relationship between epidural pressure and posture in patients with lumbar spinal stenosis. Spine 20, 650–653 (1995).
25. sekiguchi, M., Kikuchi, s. & Myers, r. r. experimental spinal stenosis: relationship between degree of caudaequina compression, neuropathology, and pain. Spine 29, 1105–1111 (2004).
26. ito, T. et al. rho kinase inhibitor improves motor dysfunction and hypoalgesia in a rat model of lumbar spinal canal stenosis. Spine 32, 2070–2075 (2007).
27. Goh, K. J., Khalifa, w., Anslow, P., Cadoux-Hudson, T. &Donaghy, M. The clinical syndrome associated with lumbar spinal stenosis. Eur. Neurol. 52, 242–249 (2004).
28. Hufschmidt, A. &Lücking, C. H (eds) Neurologie Compact (Thieme, stuggart, 2006).
29. sinikallio, s. et al. Depression is associated with poorer outcome of lumbar spinal stenosis surgery. Eur. Spine J. 16, 905–912 (2007).
30. sinikallio, s. et al. somatic comorbidity and younger age are associated with life dissatisfaction among patients with lumbar spinal stenosis before surgical treatment. Eur. Spine J. 16, 857–864 (2007).
31. Cahill, P. et al. Lumbar spinal stenosis, part i and ii. Cont. Spine Surg. 5, 56–68 (2004).
32. Boden, s. D., Davis, D. O., Dina, T. s., Patronas, N. J. &wiesel, s. w. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J. Bone Joint Surg. Am. 72, 403–408 (1990).
33. Amundsen, T. et al. Lumbar spinal stenosis. Clinical and radiologic features. Spine 20, 1178–1186 (1995).
34. Bigos, s. et al. Acute Low Back Problems in Adults. Agency for Health Care Policy and research, Public Health service. Clinical Practice Guideline No. 14. report No. 95–0642 (2004).
35. saifuddin, A. The imaging of lumbar spinal stenosis. Clin. Radiol. 55, 581–594 (2000).
36. North American spine society (NAss). Diagnosis and treatment of degenerative lumbar spinal stenosis [online], http://www.guideline.gov/ summary/summary.aspx?doc_id=11306#top (2007).
37. Modic, M. T. et al. Acute low back pain and radiculopathy: Mr imaging findings and their prognostic role and effect on outcome. Radiology 237, 597–604 (2005).
38. Carragee, e. J. & Kim, D. H. A prospective analysis of magnetic resonance imaging findings in patients with sciatica and lumbar disc herniation. Correlation of outcomes with disc fragment and canal morphology. Spine 22, 1650–1660 (1997).
39. Modic, M. T. &ross, J. s. Lumbar degenerative disk disease. Radiology 245, 43–61 (2007).
40. Hueftle, M. G. et al. Lumbar spine: postoperative Mr imaging with Gd-DTPA. Radiology 167, 817–824 (1988).
41. ross, J. s. et al. Mr imaging of the postoperative lumbar spine: assessment with gadopentetatedimeglumine. Am. J. Roentgenol. 155, 867–872 (1990).
42. Jinkins, J. r. Gd-DTPA enhanced Mr of the lumbar spinal canal in patients with claudication. J. Comput. Assist. Tomogr. 17, 555–562 (1993).
43. Jinkins, J. r. et al. spinal nerve enhancement with Gd-DTPA: Mrcorrelation with the postoperative lumbosacral spine. AJNR Am. J. Neuroradiol. 14, 383–394 (1993).
44. . Kobayashi, s. et al. imaging of caudaequina edema in lumbar canal stenosis by using gadolinium-enhanced Mr imaging: experimental constriction injury. AJNR Am. J. Neuroradiol. 27, 346–353 (2006).
45. O'Connell, M. J., ryan, M., Powell, T. &eustace, s. The value of routine Mrmyelography at Mri of the lumbar spine. ActaRadiol. 44, 665–672 (2003).
46. eberhardt, K. e., Hollenbach, H. P., Tomandl, B. &Huk, w. J. Three-dimensional Mrmyelography of the lumbar spine: comparative case study to X-ray myelography. Eur. Radiol. 7, 737–742 (1997).
47. Alyas, F., Connell, D. &saifuddin, A. Upright positional Mri of the lumbar spine. Clin. Radiol. 63, 1035–1048 (2008).
48. Katz, J. N. & Harris, M. B. Clinical practice. Lumbar spinal stenosis. N. Engl. J. Med. 358, 818–825 (2008).
49. scavone, J. G., Latshaw, r. F. &weidner, w. A. Anteroposterior and lateral radiographs: an adequate lumbar spine examination. AJR Am. J. Roentgenol. 136, 715–717 (1981).

50. Hammouri, Q. M., Haims, A. H., simpson, A. K., Alqaqa, A. &Grauer, J. N. The utility of dynamic flexion-extension radiographs in the initial evaluation of the degenerative lumbar spine. Spine 32, 2361–2364 (2007).
51. Pitkänen, M. T. et al. segmental lumbar spine instability at flexion-extension radiography can be predicted by conventional radiography. Clin. Radiol. 57, 632–639 (2002).
52. Pearcy, M., Portek, i. & shepherd, J. The effect of low-back pain on lumbar spinal movements measured by three-dimensional X-ray analysis. Spine 10, 150–153 (1985).
53. Herno, A., Airaksinen, O., saari, T. &Miettinen, H. The predictive value of preoperative myelography in lumbar spinal stenosis. Spine 19, 1335–1338 (1994).
54. Moon, e. s. et al. Comparison of the predictive value of myelography, computed tomography and Mri on the treadmill test in lumbar spinal stenosis. Yonsei Med. J. 46, 806–811 (2005).
55. Bal, s., Celiker, r., Palaoglu, s. &Cila, A. F wave studies of neurogenic intermittent claudication in lumbar spinal stenosis. Am. J. Phys. Med. Rehabil. 85, 135–140 (2006).
56. Adamova, B., vohanka, s. &Dusek, L. Dynamic electrophysiological examination in patients with lumbar spinal stenosis: is it useful in clinical practice? Eur. Spine J. 14, 269–276 (2005).
57. weinstein, J. N. et al. surgical versus nonsurgical therapy for lumbar spinal stenosis. N. Engl. J. Med. 358, 794–810 (2008).
58. Haarmeier, T. &stolke, D. spinaleengesyndrome. inTherapie und VerlaufneurologischerErkrankungen [German], 5th edn (eds Brandt, T., Dichgans, J. &Diener, H. C) 1203–1220 (Kohlhammerverlag, 2007).
59. Johnsson, K. e., rosén, i. &Udén, A. The natural course of lumbar spinal stenosis. Clin. Orthop. Relat. Res. 279, 82–86 (1992).
60. Dagenais, s., Caro, J. &Haldeman, s. A systematic review of low back pain cost of illness studies in the United states and internationally. Spine J. 8, 8–20 (2008).
61. Murphy, D. r., Hurwitz, e. L., Gregory, A. A. & Clary, r. A non-surgical approach to the management of lumbar spinal stenosis: a prospective observational cohort study. BMC Musculoskelet. Disord. 7, 16 (2006).
62. vo, A. N. et al. rehabilitation of orthopedic and rheumatologic disorders. 5. Lumbar spinal stenosis. Arch. Phys. Med. Rehabil. 86 (Suppl. 1), s69–s76 (2005).
63. simotas, A. C. Nonoperative treatment for lumbar spinal stenosis. Clin. Orthop. Relat. Res. 25, 153–161 (2001).
64. vanTulder, M. w., Koes, B. &Malmivaara, A. Outcome of non-invasive treatment modalities on back pain: an evidence-based review. Eur. Spine J. 15 (Suppl. 1), s64–s81 (2006).
65. vanTulder, M. w., Koes, B., seitsalo, s. &Malmivaara, A. Outcome of invasive treatment modalities on back pain and sciatica: an evidence-based review. Eur. Spine J. 15 (Suppl. 1), s82–s92 (2006).
66. wunschmann, B. w., sigl, T., ewert, T., schwarzkopf, s. r. &stucki, G. Physical therapy to treat spinal stenosis [German]. Orthopade 32, 865–868 (2003).
67. Nelemans, P. J., deBie, r. A., devet, H. C. &sturmans, F. injection therapy for subacute and chronic benign low back pain. Spine 26, 501–515 (2001).
68. Dvorak, J. &Grob, D. epidural injections. what is certain [German]? Orthopade 33, 591–593 (2004).
69. Armin, s. s., Holly, L. T. &Khoo, L. T. Minimally invasive decompression for lumbar stenosis and disc herniation. Neurosurg. Focus 25, e11 (2008).
70. Toyone, T., Tanaka, T., Kato, D., Kaneyama, r. &Otsuka, M. Patients' expectations and satisfaction in lumbar spine surgery. Spine 30, 2689–2694 (2005).
71. Atlas, s. J., Keller, r. B., wu, Y. A., Deyo, r. A. & singer, D. e. Long-term outcomes of surgical and nonsurgical management of lumbar spinal stenosis: 8 to 10 year results from the Maine lumbar spine study. Spine 30, 936–943 (2005).
72. Chiu, J. C. interspinous process decompression (iPD) system (X-sTOP) for the treatment of lumbar spinal stenosis. Surg. Technol. Int. 15, 265–275 (2006).
73. Kim, D. H. & Albert, T. J. interspinous process spacers. J. Am. Acad. Orthop. Surg. 15, 200–207 (2007).
74. Benz, r. J., ibrahim, Z. G., Afshar, P. &Garfin, s. r. Predicting complications in elderly patients undergoing lumbar decompression. Clin. Orthop. Relat. Res. 384, 116–121 (2001).
75. Mayer, H. M., List, J., Korge, A. &wiechert, K. Microsurgery of acquired degenerative lumbar spinal stenosis. Bilateral over-the-top decompression through unilateral approach [German]. Orthopade 32, 889–895 (2003).
76. Thome, C. et al. Outcome after less-invasive decompression of lumbar spinal stenosis: a randomized comparison of unilateral laminotomy, bilateral laminotomy, and laminectomy. J. Neurosurg. Spine 3, 129–141 (2005).
77. Ng, L. C., Tafazal, s. & sell, P. The effect of duration of symptoms on standard outcome measures in the surgical treatment of spinal stenosis. Eur. Spine J. 16, 199–206 (2007).

78. Postacchini, F. &Cinotti, G. Bone regrowth after surgical decompression for lumbar spinal stenosis. J. Bone Joint Surg. Br. 74, 862–869 (1992).
79. Jansson, K. A., Németh, G., Granath, F. &Blomqvist, P. spinal stenosis re-operation rate in sweden is 11% at 10 years—a national analysis of 9664 operations. Eur. Spine J. 14, 659–663 (2005).
80. resnick, D. K. et al. Guidelines for the performance of fusion procedures for degenerative disease of the lumbar spine. Part 9: fusion in patients with stenosis and spondylolisthesis. J. Neurosurg. Spine 2, 679–685 (2005).
81. Ploumis, A., Transfledt, e. e. & Denis, F. Degenerative lumbar scoliosis associated with spinal stenosis. Spine J. 7, 428–436 (2007).
82. Gelalis, i. D. et al. Decompressive surgery for degenerative lumbar spinal stenosis: long-term results. Int. Orthop. 30, 59–63 (2006).
83. ikuta, K. et al. short-term results of microendoscopic posterior decompression for lumbar spinal stenosis. Technical note. J. Neurosurg. Spine 2, 624–633 (2005).
84. Katz, J. N. et al. Predictors of surgical outcome in degenerative lumbar spinal stenosis. Spine 24, 2229–2233 (1999).
85. Mackay, D. C. & wheelwright, e. F. Unilateral fenestration in the treatment of lumbar spinal stenosis. Br. J. Neurosurg. 12, 556–558 (1998).
86. Postacchini, F. surgical management of lumbar spinal stenosis. Spine 24, 1043–1047 (1999).
87. Postacchini, F., Cinotti, G., Perugia, D. &Gumina, s. The surgical treatment of central lumbar stenosis. Multiple laminotomy compared with total laminectomy. J. Bone Joint Surg. Br. 75, 386–392 (1993).
88. spetzger, U., Bertalanffy, H., reinges, M. H. &Gilsbach, J. M. Unilateral laminotomy for bilateral decompression of lumbar spinal stenosis. Part ii: Clinical experiences. ActaNeurochir. (Wien) 139, 397–403 (1997).
89. Delank, K. s. et al. Undercutting decompression versus laminectomy. Clinical and radiological results of a prospective controlled trial [German]. Orthopade 31, 1048–1056 (2002).
90. Tender, G. C., Baratta, r. v. &voorhies, r. M. Unilateral removal of pars interarticularis. J. Neurosurg. Spine 2, 279–288 (2005).
91. Tender, G. C., Kutz, s., Baratta, r. &voorhies, r. M. Unilateral progressive alterations in the lumbar spine: a biomechanical study. J. Neurosurg. Spine 2, 298–302 (2005).
92. Kawaguchi, Y. et al. Clinical and radiographic results of expansive lumbar laminoplasty in patients with spinal stenosis. J. Bone Joint Surg. Am. 87 (Suppl. 1), 292–299 (2005).
93. Amundsen, T. et al. Lumbar spinal stenosis: conservative or surgical management? A prospective 10-year study. Spine 25, 1424–1435 (2000).
94. Herno, A., saari, T., suomalainen, O. &Airaksinen, O. The degree of decompressive relief and its relation to clinical outcome in patients undergoing surgery for lumbar spinal stenosis. Spine 24, 1010–1014 (1999).
95. Fokter, s. K. &Yerby, s. A. Patient-based outcomes for the operative treatment of degenerative lumbar spinal stenosis. Eur. Spine J. 15, 1661–1669 (2005).
96. Herno, A., Airaksinen, O., saari, T. &Luukkonen, M. Lumbar spinal stenosis: a matched-pair study of operated and nonoperated patients. Br. J. Neurosurg. 10, 461–465 (1996).