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StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-.

Spinal Stenosis And Neurogenic Claudication

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Last Update: February 12, 2022.

Continuing Education Activity

Approximately 90 percent of the population will present with low back pain at some point in their lifetime. Spinal stenosis is a condition that is caused by the narrowing of the central canal, the lateral recess, or neural foramen. It can cause significant discomfort, interfere with activities of daily living, and in some cases, may result in progressive disability. This activity reviews the evaluation and effective management of symptomatic lumbar canal stenosis and highlights the role of the interprofessional team in utilizing clinical and radiological markers to formulate appropriate therapeutic protocols.

Objectives:

- Describe the etiology of lumbar stenosis.
- Identify the region of the lumbar spine most commonly affected by lumbar stenosis.
- Summarize the differential diagnosis for lumbar stenosis.
- Explain the need for a well-integrated, interprofessional team approach to improve care for patients with lumbar stenosis.

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Introduction

Approximately 90% of the population will present with low back pain at some point in their lifetime. Spinal stenosis is a condition that is caused by the narrowing of the central canal, the lateral recess, or neural foramen. This condition can cause significant discomfort, interfere with activities of daily living, and may result in progressive disability.[1][2][3] With increasing longevity of humankind, degenerative diseases of the spine and its sequelae are bound to have an immense negative impact on the global front.[4]

It is important to appreciate that spinal stenosis is part of the aging process, and predicting who will develop symptoms is not always easy. There is no clear cut association between spinal stenosis and the presence of symptoms. While the degenerative process is not 100% preventable, it can be slowed down with exercise diet or changes in lifestyle. Overall, spinal stenosis is most common in the lumbar and cervical regions.

Etiology

Spinal stenosis most commonly is caused by degenerative osteoarthritis of the spine or spondylosis and occurs most frequently at the L4 to L5 level, followed by L5 through S1 and L3 to L4. Additional risk factors include obesity or a family history of this condition. Other factors such as disc protrusion or bulging (for example, caused by progressive disc degeneration with aging or trauma), loss of disc height, facet joint arthropathy, osteophyte formation, or ligamentum flavum hypertrophy can all lead to encroachment on and narrowing of the central canal and neural foramina.[5][6]

Spondylolisthesis, the translation of one vertebral body anteriorly or posteriorly relative to an adjacent vertebral body, may also exacerbate spinal canal narrowing.

Additional acquired causes of spinal stenosis include space-occupying lesions such as synovial or neural cysts, neoplasms, or lipomas; traumatic or postoperative changes such as fibrosis; and skeletal diseases such as ankylosing spondylitis, rheumatoid arthritis, or Paget disease.

Congenital or developmental causes of spinal stenosis include dwarfism, namely achondroplasia, Morquio syndrome, and spinal

dysraphism such as spina bifida, spondylolisthesis, and myelomeningocele.

Epidemiology

Spinal stenosis occurs most frequently in individuals over the age of 60. In adults over the age of 65 years undergoing spinal surgery, lumbar spinal stenosis remains the leading pre-operative diagnosis. Many conditions correlate with the development of spinal stenosis, but symptomatic spinal stenosis tends to occur most frequently in the setting of degenerative changes.

The majority of spinal stenosis tends to occur in lower lumbar levels as dorsal root ganglion diameter tends to be increased in this region, causing greater encroachment of the neural foramina. The lower lumbar segments also tend to have a greater incidence of spondylosis and degenerative disc disease, leading to an even greater predisposition to spinal stenosis and nerve root impingement.

Though the majority of individuals over the age of 60 have some degree of spinal stenosis, most of these patients are also asymptomatic. The exact incidence of spinal stenosis is, therefore, difficult to determine.

Pathophysiology

Neurologic symptoms such as claudication associated with spinal stenosis occur most commonly as a result of ischemia or mechanical compression of nerve roots.

Contributing factors disc herniation and bulge, facet joint and ligamentum flavum hypertrophy and buckling and concomitant spondylolisthesis.[7]

Increased intrathecal (subarachnoid space) compression as a result of narrowing of the spinal canal also can lead indirectly to mechanical compression of nerve roots and cause venous congestion, diminished arterial blood flow, and resultantly decreased impulse conduction at the nerve roots. However, narrowing at multiple spinal levels may be necessary to elicit such complications.

Symptoms of spinal stenosis are caused by and become most prominent when there is a reduction of the interlaminar space; this occurs naturally with prolonged standing when the spine is in an erect position. Extension of the spine causes the overlapping of laminar edges of adjacent vertebral bodies, with resultant relaxation and inward buckling of the ligamentum flavum along with the movement of the superior facets in a rostral-anterior direction. Walking may additionally exacerbate symptoms as the increase in oxygen demands of the spinal nerve roots may exceed the available blood flow, especially in the case of elevated pressures in the intrathecal (subarachnoid) space.

Neurogenic claudication results from central canal stenosis, whereas radiculopathy is the sequelae to lateral recess encroachment.[7]

History and Physical

The most common symptom associated with spinal stenosis is neurogenic (or pseudo) claudication.[8]

This presentation is attributable to the sequence of Porter concept of two-level stenosis, vascular compromise due to central stenosis and the compression of a nerve root due to degenerative pathology due to lateral stenosis.[8]

A key feature of neurogenic claudication is its relationship to the patient's posture where lumbar extension increases, and flexion decreases pain, thereby attributing a specific "simian stance" seen among these subsets of patients. The same phenomenon is accountable for better tolerance to climbing uphill compared to downhill walking.[8] Pain is exacerbated by walking, standing, or upright exercises. Pain relief occurs with sitting or forward flexion at the waist such as involved with squatting, leaning forward, or lying down. Many patients are asymptomatic when inactive. Extending the back while standing leading to the development of symptoms which promptly resolve by subsequently leaning forward 20 to 40 degrees at the waist a classic presentation.

Additional symptoms of spinal stenosis, generally as a result of spinal nerve root involvement within the lumbar spinal canal, may include general discomfort, weakness in the legs, numbness, or paresthesias. Most patients typically experience bilateral symptoms, though in some cases the symptoms may be asymmetric in their complaint. Either case usually involves the entire leg rather than just one portion.

Although most patients often have a normal neurological exam, and some may have neurological signs or symptoms reflecting multiple lumbosacral radiculopathies in addition to the more typical symptoms of spinal stenosis. There may be evidence of focal weakness, absent deep tendon reflexes, or sensory loss.

Bilateral extensor digitorum brevis wasting is a reliable clinical bedside marker while assessing for underlying lumbar canal stenosis.[9]

The five repetitive sit to stand test (5R-STST) wherein a patient with the ability to perform the test in around 10 seconds does not rate as having a significant functional impairment.[10]

Evaluation

Neuroimaging is necessary if a patient presents with new-onset symptoms, or there are signs or symptoms of radiculopathy or spinal stenosis. Magnetic resonance imaging (MRI) is the diagnostic modality of choice for spinal stenosis as it allows for visualization of both soft tissues and neural structures. Thus, MRI confirms the presence of anatomic narrowing of the spinal canal or the presence of nerve root impingement. Though MRI is preferred, computed tomography (CT) may better show bony structures when clinically indicated, and CT myelography is an option in the setting of MRI contraindications in individual patients.

Computerized tomogram revealing trefoil appearance and MRI findings of positive sedimentation signs are radiological hallmarks of underlying canal stenosis.[11]

CT myelography is an adequate test to confirm the presence of narrowing of the spinal canal or nerve root impingement.[12][13][14]

Electrodiagnostic evidence of fibrillation potentials and the absence of tibial H-wave may aid in further confirming the diagnosis of the lumbar canal stenosis.[15]

Treatment / Management

Initial treatment can include both conservative and nonsurgical methods. These methods include physical therapy such as stretching, strengthening, and aerobic fitness to improve and stabilize muscles and posture; anti-inflammatory and analgesic medications; and epidural steroid injections. In addition to these methods, patients with lumbar spinal stenosis should be advised to avoid aggravating factors such as downhill ambulation and excessive lumbar extension.

Surgery is for only those who fail repeated nonoperative treatments.[16] In most cases, surgical treatment of spinal stenosis is elective, aimed at improving symptoms and function rather than preventing neurologic complications, and merits consideration only after attempting nonsurgical modalities, or if a patient's symptoms result in disability. If a patient presents with rapidly progressive neurological deficits or if there is the presence of bladder dysfunction, urgent surgery is necessary. This situation may present in cases of cauda equina syndrome, conus medullaris syndrome, trauma, or an intraspinal canal tumor. The surgical approach is multilevel decompressive laminectomy with or without lumbar fusion. Lumbar fusion is generally reserved for patients with spondylolisthesis.[17][18][19][20]

Patients with symptomatic spinal stenosis treated surgically maintain substantially greater clinical improvement than those treated nonsurgically.[21] For patients with lumbar stenosis without spondylolisthesis, a decompression alone is recommended.[22] The Spine Patient Outcomes Research Trial (SPORT) provided level II evidence indicating laminectomy and fusion did provide better results than nonoperative approaches.[23]

The golden rule in performing these procedures has its basis in the concept that underperforming leads to failed back syndrome, whereas over-doing leads to instability.[4] Laminectomy accounts for significant blood loss, surgical site pain, prolonged hospital stay, and weakening paraspinal muscles, thereby leading to possible spinal instability.[24] To minimize this outcome, surgeons rely on newer surgical techniques such as laminoplasty, hemilaminectomy, laminotomy, and undercutting laminotomies. However, no statistically significant differences appear in the literature between laminectomy and laminotomy in terms of clinical outcomes.[24] Preservation of the posterior elements or the "posterior tension band" is the most important factor in preventing instability.[7] However, during minimal invasive approaches such as endoscopic interlaminar approach and bilateral laminotomy, there is an increased risk for neural injury.[25][26] However, following a learning curve period, data shows the risk of dural tears to be substantially reduced (5 to 15% in laminectomy vs. 2 to 6% in laminotomy).[26]

Another major issue in the minimally invasive spine (MIS) approaches shows in higher rates of reoperation for residual stenosis not adequately addressed in the initial operation.[7] On the other hand, the Spinal Laminectomy versus Instrumented Pedicle Screw (SLIP) study provided level I evidence supporting decompression with fusion since one-third of patients undergoing standalone laminectomy developed instability within four years.[27] The key issue is identifying those subsets of patients who are at risk of postoperative spinal instability in cases of grade I spondylolisthesis.[27]

Lumbar fusion was associated with meaningful improvement in overall physical health-related quality of life than laminectomy alone.[28] Paradoxically spinal fusion surgeries account for the highest aggregate hospital costs of any surgical procedure performed in U.S. hospitals.[28] Advocating the same guidelines for patients in the middle and low-income nations is justifiable only if they provide durable clinical benefit.[27] There have been few alternative solutions such as the use of interspinous distractors; however, though researchers observed fewer complications in these procedures, there were higher risks for redo surgery.[16][29]

Recently, posterior fixation surgery with facet distraction, without decompression has shown to have good clinical outcomes among similar patients.[30]

Differential Diagnosis

The differential diagnosis includes vascular claudication and multiple level lumbar disc protrusions.

Prognosis

Spinal stenosis has significant morbidity and affects the quality of life. With time it can lead to chronic pain and muscle weakness. In some cases, it may lead to cauda equina syndrome. Patients with central spinal stenosis may have difficulty walking and have gait disturbances. While some patients may improve with time, the majority have a progression of the condition, leading to disability. The cost of managing spinal stenosis is enormous, and for patients, it can lead to high healthcare bills.

Complications

Underperforming predisposes patients to a failed back syndrome, whereas over-doing may result in instability in the patients.[4]

Similarly, minimally invasive surgeries add up the risk of neural injury.[26]

Other complications include:

- Lower extremity radicular pain
- Muscle wasting
- Disability
- Spinal deformity
- Pulmonary embolism

Postoperative and Rehabilitation Care

Facet joint tropism and asymmetry of paraspinal muscle volume can be used as reliable adjuncts in monitoring for postoperative spinal instability.[31]

The rehabilitation program has not been found to be superior to the simple recommendation to stay active.[32]

Deterrence and Patient Education

The concept of 'shared decision making' can help in framing judicious and effective treatment plans.[33]

Enhancing Healthcare Team Outcomes

The management of spinal stenosis is best with an interprofessional team that includes a surgeon, neurologist, physical therapist, primary caregiver, and nurse practitioner. Patients with only low back pain should receive conservative management; those with signs and symptoms of nerve compromise need spinal decompression. All patients should receive education on the benefits of exercise, discontinuation of smoking, become physically active, and maintaining healthy body weight. Patients who lead a sedentary lifestyle tend to have relapses of low back pain that can be disabling.[34]

The pharmacist should counsel the patient on how to ease the pain and refrain from taking too many controlled medications, along with performing medication reconciliation to prevent any potential drug interactions. A pain consultation should be obtained. The physical therapist should participate in educating patients about the importance of body weight and exercise. Nursing staff can be instrumental in providing post-operative care, medication administration, monitoring the patient's condition following the procedure. The pharmacist, physical therapist, and nurse should alert the treating physician if they note any concerns.

Gold standard criteria for both its diagnosis and treatment remain elusive and should be guided by the conjoint aids of the neurological examination and radiological analysis.[35]

Lumbar canal stenosis now is a social and economic issue with the upraise in the utility of fusion surgery for preventing spinal instability.[35]

The hallmark for effective management in the symptomatic lumbar canal stenosis is determining the risk of postoperative spinal instability following decompression and thereby opting for fusion surgery. Efforts should focus on safeguarding the posterior osteo-ligamentous complex and targetting for hypertrophic facet joints and ligamentum flavum, the main factors contributing to the stenosis.

Open communication and collaboration among an interprofessional team that includes primary care clinicians, specialists, nursing,

pharmacy, and therapists are necessary to improve patient outcomes. [Level V]

Review Questions

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