Spinal Stenosis Clinical Presentation

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History

The primary clinical manifestation of spinal stenosis is chronic pain. In patients with severe stenosis, weakness and regional anesthesia may result. Among the most serious complications of severe spinal stenosis is central cord syndrome. Central cord syndrome is the most common incomplete cord lesion. The presentation commonly is associated with an extension injury in a patient with an osteoarthritic spine. In hyperextension injury, the cord is injured within the central gray matter, which results in proportionally greater loss of motor function of upper extremities than loss of motor function of lower extremities, with variable sensory sparing.

Patients with spinal stenosis become symptomatic when pain, motor weakness, paresthesia, or another neurologic compromise causes distress. Spinal stenosis of the thoracic spine is more likely to directly affect the spinal cord because of the relatively narrow thoracic spinal canal.

Spinal stenosis of the cervical and thoracic regions may contribute to neurologic injury, such as development of a central spinal cord syndrome following spinal trauma. Spinal stenosis of the lumbar spine is associated most commonly with midline back pain and radiculopathy. In cases of severe lumbar stenosis, innervation of the urinary bladder and the rectum may be affected, but lumbar stenosis most often results in back pain with lower extremity weakness and numbness along the distribution of nerve roots of the lumbar plexus.

Spinal canal size is not always predictive of clinical symptoms, and some evidence suggests that body mass may play a role in limitations of function in this population.[26]

Severe radiologic stenosis in otherwise asymptomatic individuals suggests inflammation, not just mechanical nerve root compression. Specific inflammation generators may include herniated nucleus pulposus (HNP), ligamentum flavum, and facet joint capsule.

Metastatic and infectious processes that affect the spine may present with both regional pain and signs of central spinal canal narrowing. The regional pain may result from pathologic fractures or nerve root compression by the tumor or abscess. Long tract findings may result from bone fragments, a hemorrhage, an abscess, or a tumor compressing the spinal cord.

Cervical stenosis

Stenosis of the cervical spine causes the clinical syndrome of cervical spondylotic myelopathy (CSM). Initial symptoms may be subtle loss of hand dexterity and mild proximal lower extremity weakness, often without neck or arm pain. With progression, spastic quadripareisis results. Pathologic reflexes such as the Hoffman sign, clonus, and/or the Babinski reflex may augment the diffuse hyperreflexia. Some patients also have associated ataxia from compression of spinocerebellar tracts.[4, 10, 11, 27, 28]

If associated cervical root impingement exists, patients may experience sharp radicular pain into the affected arm, with associated paresthesias and weakness referable to the compressed root. Depending on the level, some upper extremity reflexes (biceps, triceps, brachioradialis) may be depressed or absent in such patients. Males older than 55 years most commonly are affected. Up to two thirds of patients with myelopathy have deteriorating or unchanging conditions. They are also at increased risk of spinal cord injury in the setting of minor trauma.
Lumbar stenosis

Katz and colleagues report that the historical findings most strongly associated with lumbar spinal stenosis (LSS) include advanced age, severe lower extremity pain, and absence of pain when the patient is in a flexed position.[29] Fritz and colleagues contend that the most important elements involve the postural nature of the patient’s pain, stating that absence of pain or improvement of symptoms when seated assists in ruling in LSS.[22] Conversely, LSS cannot be ruled out when sitting is the most comfortable position for the patient and standing/walking is the least comfortable.

Patients with significant lumbar spinal canal narrowing report pain, weakness, numbness in the legs while walking, or a combination thereof. Onset of symptoms during ambulation is believed to be caused by increased metabolic demands of compressed nerve roots that have become ischemic due to stenosis. This is the hallmark of neurogenic claudication. The pain is relieved when the patient flexes the spine by, for example, leaning on shopping carts or sitting. Flexion increases canal size by stretching the protruding ligamentum flavum, reduction of the overriding laminae and facets, and enlargement of the foramina. This relieves the pressure on the exiting nerve roots and, thus, decreases the pain. The most common nerve affected is the L5, with associated weakness of extensor hallucis longus.

LSS classically presents as bilateral neurogenic claudication (NC). Unilateral radicular symptoms may result from severe foraminal or lateral recess stenosis. Patients, typically aged more than 50 years, report insidious-onset NC manifesting as intermittent, crampy, diffuse radiating thigh or leg pain with associated paresthesias. Indeed, leg pain affects 90% of patients with LSS.

In a retrospective review of 75 patients with radiographically confirmed LSS, reports of weakness, numbness or tingling, radicular pain, and NC were in almost equal proportions. The most common symptom was numbness or tingling of the legs.[30]

NC pain is exacerbated by standing erect and downhill ambulation and is alleviated with lying supine more than prone, sitting, squatting, and lumbar flexion. Getty and colleagues documented 80% pain diminution with sitting and 75% with forward bending.[31] Lumbar spinal canal and lateral recess cross-sectional area increases with spinal flexion and decreases with extension. Furthermore, cross-sectional area is reduced 9% with extension in the normal spine and 67% with severe stenosis. The Penning rule of progressive narrowing implies that the more narrowed the canal by stenosis, the more it narrows with spinal extension. Schonstrom and colleagues have shown that spinal compressive loading from weight bearing reduces spinal canal dimensions.[32]

NC, unlike vascular claudication, is not exacerbated by biking, uphill ambulation, and lumbar flexion and is not alleviated with standing. Patients with LSS compensate for symptoms by flexing forward, slowing their gait, leaning onto objects (eg, over a shopping cart) and limiting distance of ambulation. Unfortunately, such compensatory measures, particularly in elderly osteoporotic females, promote disease progression and vertebral fracture. Pain radiates downward in NC and, in contrast, upward in vascular claudication. Hall and colleagues note the presence of radiculopathy in 6% and NC in 94% of patients with LSS.[33]

Distinguishing between neurogenic and vascular claudication is important because the treatments, as well as the implications, are quite different. Vascular claudication is a manifestation of peripheral vascular disease and arteriosclerosis. Other vessels, including the coronary, vertebral, and carotid, are also often affected. Further complicating diagnosis and treatment in some patients, neurogenic and vascular claudication may occur together. This is because both conditions frequently occur in the elderly population.

Physical Examination

Patients with cervical stenosis usually present with cervical radiculopathy, with or without myelopathy. Typically, the condition involves the lower cervical spine. Patients frequently complain of radiating arm pain with numbness and paresthesia in the involved dermatomes. Occasionally, associated weakness occurs in the muscles supplied by that nerve root. If the stenosis is severe enough, or if it is positioned centrally in the spine, patients may present with signs and symptoms of myelopathy (spinal cord dysfunction). Typically, these patients complain of finger numbness, clumsiness, and difficulty walking due to spasticity and loss of position sense. In more severe cases, the patients can have bowel and bladder control dysfunction. Upon examination, these patients have "long-tract
signs" such as hyperreflexia and clonus.

Katz and colleagues report physical examination findings most strongly associated with lumbar spinal stenosis (LSS) include wide-based gait, abnormal Romberg test, thigh pain following 30 seconds of lumbar extension, and neuromuscular abnormalities\(^{29}\); however, Fritz and colleagues state physical examination findings do not seem helpful in determining the presence or absence of LSS.\(^{22}\)

Patients with LSS usually present with a constellation of symptoms that include lower back pain, radiating leg pain (unilateral or bilateral), and possible bladder and bowel difficulties. The classic presentation is radiating leg pain associated with walking that is relieved by rest (neurogenic claudication). When patients bend forward, the pain diminishes. Rarely, patients with LSS present with cauda equina syndrome (bilateral leg weakness, urinary retention due to atonic bladder).

Physical examination findings are frequently normal in patients with LSS. Nevertheless, review of the literature suggests diminished lumbar extension appears most consistently, varies less, and constitutes the most significant finding in LSS. Other positive findings include loss of lumbar lordosis and forward-flexed gait. Charcot joints may be present in long-standing disease. Radiculopathy may be noted with motor, sensory, and/or reflex abnormalities. Asymmetric muscle stretch reflexes and focal myotomal weakness with atrophy occur more with lateral recess than central canal stenosis. Some report objective neurologic deficits in approximately 50% of LSS cases. Provocative maneuvers include pain reproduction with ambulation and prone lumbar hyperextension. Pain alleviation occurs with stationary biking and lumbar flexion.

Patients may also have a positive result from the stoop test, which was described by Dyck in 1979.\(^{34}\) This is performed by having the patient walk with an exaggerated lumbar lordosis until NC symptoms appear or are worsened. The patient is then told to lean forward. Reduction of NC symptoms is a positive result and is suggestive of NC.

Negative findings in the physical examination include skin color, turgor, and temperature; normal distal lower extremity pulses; and an absence of arterial bruits.

Importantly, remember the 5 P’s of vascular claudication, as follows:

- Pulselessness
- Paralysis
- Paresthesia
- Pallor
- Pain

The absence of these problems, excluding pain and paresthesias, which are common to neurogenic and vascular claudication, should give the clinician confidence in the diagnosis of NC. If vascular claudication is suspected, referral to an internist for a workup is indicated. This includes a serum cholesterol level, arterial Doppler studies, ankle-brachial index values, and, in some cases, arteriography.

Dural tension signs should be unremarkable. Lumbar segment mobilization often fails to reproduce pain, and palpation locates no trigger points.

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