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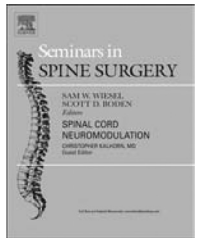

Pathophysiology and clinical presentation of lumbar stenosis

Shahbaaz A. Sabri, Venkatanarayanan Ganapathy*, and Chase Kluemper

Department of Orthopaedics, University of Tennessee College of Medicine, 979 East 3rd Street, Suite C-430, Chattanooga, TN 37403, United States

A B S T R A C T

Originally described by Kirkaldy–Willis, the degenerative cascade leading to anatomic LSS occurs in a predictable pattern, beginning with altered biomechanics of the intervertebral disk (Yong-Hing and Kirkaldy-Willis, 1983). Further studies have elucidated several theorized mechanisms responsible for the varied clinical manifestations experienced by patients with similar radiographic evidence of LSS; however, a single explanation for these finding has yet to be identified (Olemarker et al., 1996; Rydevick et al., 1991; Pedowitz et al., 1992). Obtaining thorough patient history and performing a complete physical exam is critical to ensure an accurate diagnosis. Although neurogenic claudication symptoms are classic for LSS, patients frequently present with varied and often mixed symptomatology (Buckland et al, 2017; Lesher et al., 2008). Consistent screening for myelopathic symptoms, identifying "red-flag" symptoms suggesting systemic illness, and awareness of potential extraspinal pathology is necessary to improve patient outcomes (Devin et al, 2012; Golob et al, 2006; Berger et al, 2017).

1. Pathophysiology

Lumbar spinal stenosis (LSS) describes the anatomic narrowing of the spinal canal (Fig. 3)[18]; its classification is traditionally based on both etiology and location.[1] The anatomic features of LSS may arise due to congenital or developmental abnormalities, acquired secondary to degenerative changes, or occur as a combination of both etiologies.[2] Although the initial vertebral anatomy may differ between developmental and degenerative LSS, the pathophysiology ultimately leading to the clinical manifestation of LSS is similar.[5] Acquired causes are far more common than congenital or developmental etiologies and symptoms typically present later in life.[3,16]

Kirkaldy–Willis et al initially described the degenerative cascade leading to symptomatic lumbar stenosis.[5] Occurring in a predictable manner, the cascade begins with intervertebral disc degeneration (Fig. 1). Subsequent disc height loss due to desiccation alters its ability to tolerate mechanical load, leading to progressive microtrauma of the disc, specifically the annulus fibrosis. As disc degeneration continues, the posterior longitudinal ligament bulges posteriorly towards the cauda equina (Figs. 2 and 3).

The authors proposed viewing the vertebral unit as a "tripod" consisting of the intervertebral disc and the two facet joints. At this point in the cascade the biomechanics of the anterior limb of the tripod, the intervertebral disc, is altered. Increased physiologic mechanical load is shifted dorsally and transferred to the facet joints; thus, triggering degeneration. As the facet joints settle, osteophyte formation and joint hypertrophy occurs. The combination of disc degeneration, facet hypertrophy along with effusion in the facet joints can lead to degenerative spondylolisthesis (Fig. 2). Finally, ligamentum flavum thickening and buckling causes further stenosis of the lumbar spinal canal. As the degenerative cascade continues, neural compression eventually occurs, and neighboring vertebral levels become involved.[5]

* *Corresponding author.*
E-mail address: drganapathy08@gmail.com (V. Ganapathy).

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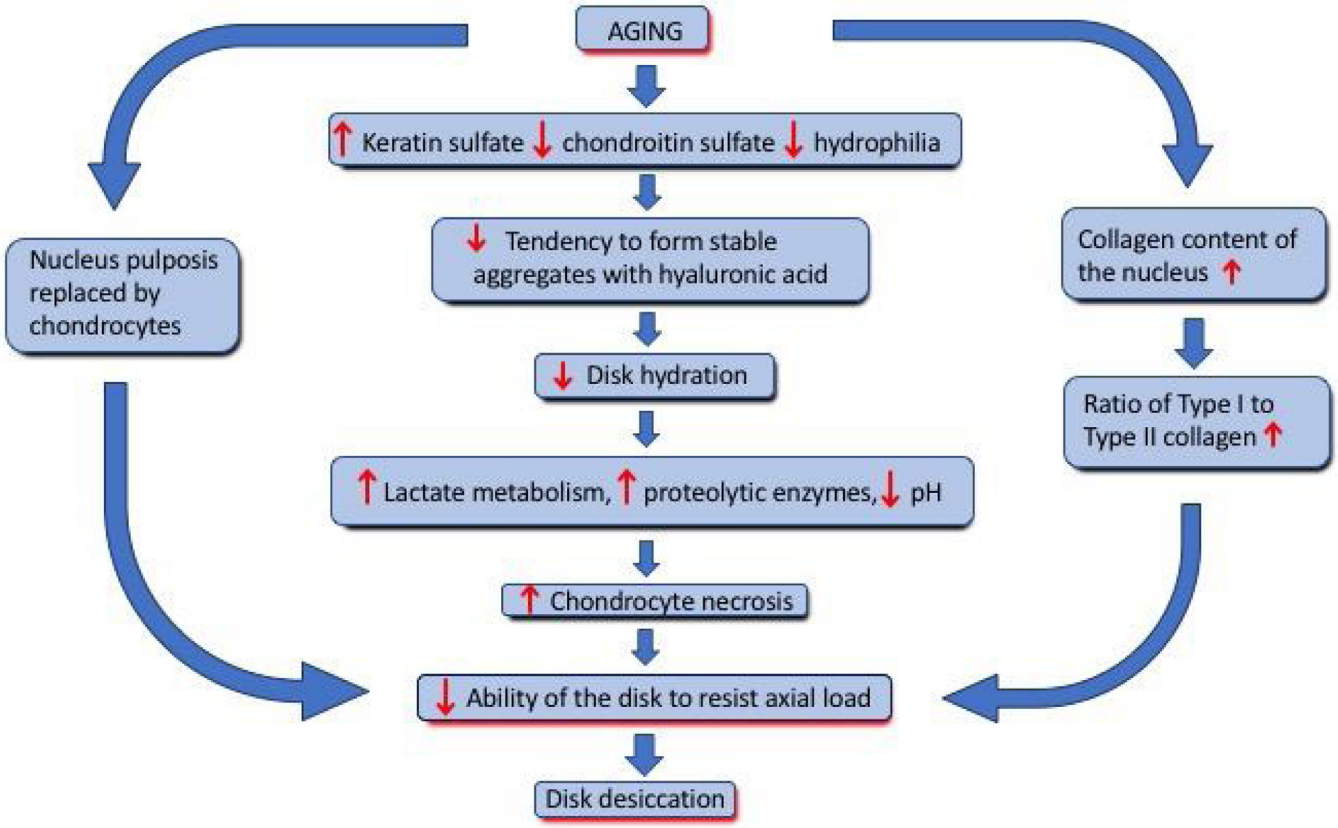

Fig. 1 – Effect of aging on disk health.

A single explanation has yet to be identified as the root cause for the varied clinical presentations in those with similar radiographic evidence of neural compression in the lumbar spine.[11] Historically, three predominant mechanisms have been proposed[6,7]: microvascular changes as a result of sustained neural compression, nerve damage from direct compression, inflammatory biochemical mediated nerve root irritation.

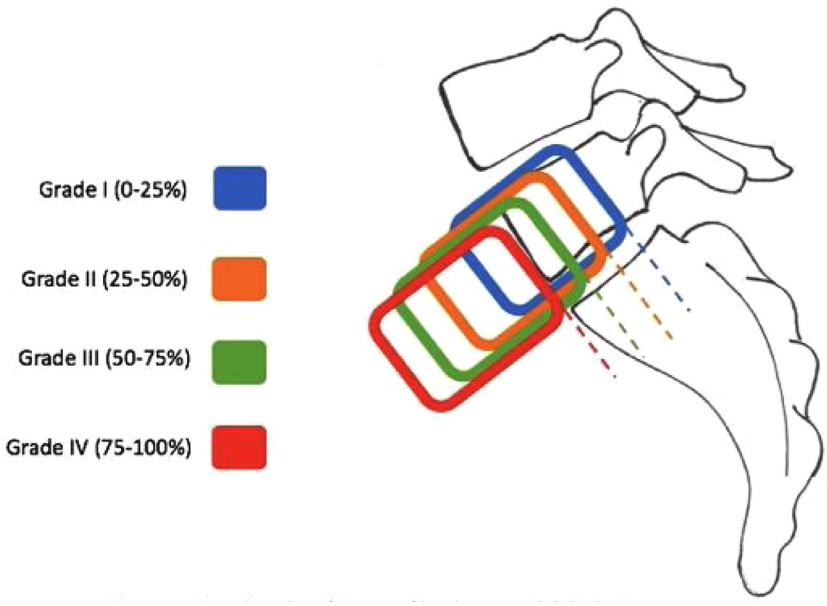

Fig. 2 – Myerding classification of lumbar spondylolisthesis.

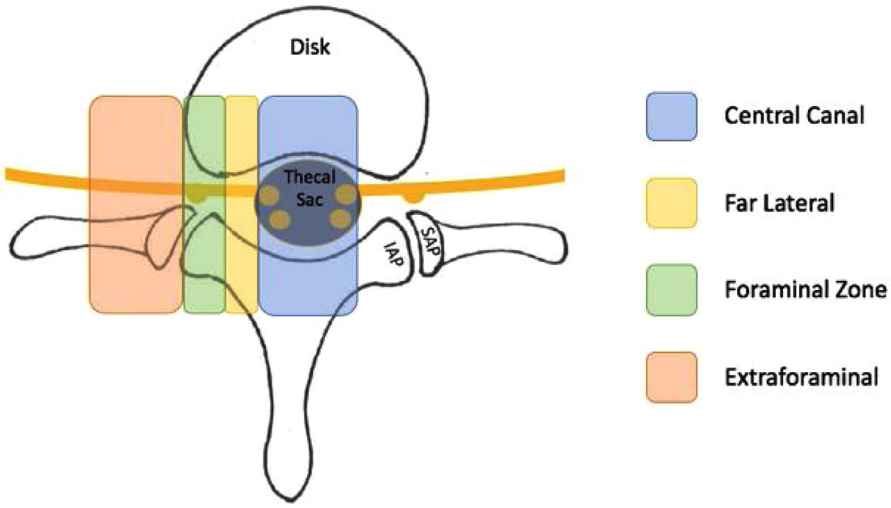

Fig. 3 – Axial cut of lumbar spine herniation zones.

A thorough understanding of the anatomy of nerve roots as they exit the spinal cord and form peripheral nerves is critical to conceptualize the interplay of mechanisms. As the dorsal and ventral nerve roots exit the cord within the dural sheath, they approach the intervertebral foramen. The dorsal root ganglion arises from the dorsal root and is usually located near the central part of the intervertebral foramen; however, its position is not constant. The DRG may be positioned within the canal, foramen, or extra-foraminal. Additionally, its size varies from level to level with the largest typically noted in the L5-S1 region. At this level, the DRG averages 6.40 ± 0.91 mm and 11.58 ± 2.25 mm in width and length, respectively.[4] Continuing distally, the dorsal and ventral roots join to form the spinal nerve and subsequently form peripheral nerves.[7] Well-developed layers of connective tissue consisting of endo-, peri-, and epineurium function to protect peripheral nerves from compression and tension.[9] On the contrary, nerve roots lack a defined perineurium and have a less organized stroma of epineurium. This anatomic difference has historically been theorized to subsequently render nerve roots more susceptible to compression.[7,9] However, it is important to note that nerve roots are surrounded by cerebrospinal fluid which does provide mechanical protection when compression is encountered.[10]

Rydevik et al. investigated the critical acute pressure necessary to induce functional changes in a porcine cauda equina model. A threshold of 50–75 mmHg resulted in changes of both afferent and efferent nerve root conduction.[10] Further studies by Pedowitz et al. reported on the significance of prolonged duration of continuous nerve compression along with the increasing magnitude of compressive force. The authors concluded the increasing negative effect of pressure-time thresholds may be related to various biochemical and microvascular abnormalities of the neural tissue under compression.[10,11]

Olmarker et al. reported on the structural changes in spinal nerve roots following exposure to autologous nucleus pulposus. Using a porcine model, epidural application of nucleus pulposus tissue without any compressive pressure resulted in significant ultrastructural changes of spinal nerve roots evaluated under electron microscopy.[8] Kang et al. further characterized the biochemical effect of specific mediators. They evaluated herniated lumbar discs along with non-herniated controls subjected to biochemical analysis for a variety of known inflammatory mediators. Herniated lumbar discs showed significantly increased amounts of IL-6, prostaglandin E2, and nitric oxide as chemical irritants to nerve roots.[15] As previous studies suggested, biochemical mediators arising from the inflammatory response to intervertebral disc material within the spinal canal may potentiate neural irritability.[13–15]

2. Clinical presentation

Classically, patients with clinical manifestations of lumbar spinal stenosis will describe symptoms consistent of neurogenic claudication. The term “claudication” originates from the Latin word *Claudus* or *claudicare,* and is defined as “to limp.” Literature reports range from 62% to 96% for this occurrence in those diagnosed with LSS.[22] Patients predominantly complain of leg pain rather than low back pain; however, both are common.[22] Hall et al. reported on a series of 68 patients with surgically confirmed LSS. Clinical neurogenic claudication was diagnosed in 94% and bilateral symptoms were seen in 68%.[17,18] If a specific nerve root is subjected to significant lateral recess compression, patients may describe a dermatomal pain distribution more clearly.

Neurogenic claudication symptoms in the lower extremities can be vague but usually described as a diffuse aching, burning, numbness, or heaviness occurring with activity- typically while the lumbar spine is in an extended position. Lumbar flexion relieves these symptoms as anatomic distraction of the posterior elements of the vertebral unit elongate and relieve thecal sac compression. The classic example being the “grocery cart sign” where patients with symptomatic LSS experience relief of lower extremity symptoms as they lean forward over the grocery cart while walking. In contrast to

neurogenic claudication, vascular claudication presents with functional lower extremity pain that is worsened with physical activity and unrelated to position. In vascular claudication, pathology is secondary to lower extremity muscle exertion and inadequate muscular perfusion. Therefore, patients experience relief of lower extremity pain during periods of rest- effectively decreasing oxygen demand of the muscles involved.

Accurately differentiating between these two pathologies is critical. A detailed history should be obtained regarding the patient's activity during onset of lower extremity pain and their maneuvers to alleviate pain in order to differentiate between the two. Further clinical assessment with a detailed vascular examination can help corroborate the clinical suspicion.

For patients presenting with activity related low back, groin, and/or lower extremity pain, the differential diagnose can be extensive.[20] The interrelated symptomatology and often confusing clinical picture seen in patients with overlapping degenerative spine and hip osteoarthritis is known as Hip-Spine Syndrome.[19] Patients often describe buttock or unilateral low back pain as "hip pain;" however, pain from true hip pathology is classically felt in the groin. Lesher et al. reported on referred pain patterns in 51 patients with hip OA. Surprisingly, 22% of patients in this series reported pain distal to the knee that was relieved by a fluoroscopic guided corticosteroid intraarticular hip injection.[21] This finding reinforces the need for clinicians to perform thorough physical examinations on both the spine and the hip in patients with assumed LSS. If positive findings suggest potential hip pathology, appropriate imaging and diagnostic modalities of the involved hip should be employed to ensure an accurate diagnosis.[23]

Symptoms related to LSS do not typically include severe neurologic deficits, bowel or bladder incontinence, profound gait disturbance, or UMN symptoms. First described by Teng and Papatheodorou in 1964, "Tandem Spinal Stenosis" (TSS) is defined as compromised canal diameter in at least 2 distinct regions of the spine, most commonly the cervical and lumbar regions. It often presents as a clinical triad of symptoms of intermittent lower extremity claudication, gait disturbance, and mixed upper and lower motor neuron signs.[27] The prevalence of TSS is estimated from 7.6% to 60%.[12] Lee et al. performed a cadaveric study on 440 skeletally mature skeletons and found the association of cervical and lumbar stenosis statistically significant. They reported stenosis in on area of the spine positively predicts stenosis in the other area 15.3−32.4% of the time.[28] These findings suggest a high degree of association and necessity for clinicians to explore and examine all patients for upper motor neuron involvement. In patients with TSS, many surgeons elect to decompress and stabilize the cervical spine prior to lumbar procedures given to the potential for disability, risk of progressive cord injury and the natural history of cervical myelopathy. However, much debate exists regarding the appropriate staging and protocol in surgical management of this subset of patients.[12]

Gathering history related to the patients overall medical status and obtaining a complete review of systems is essential for all patients presenting to a spine surgeon. Medical comorbidities such as hypertension, coronary artery disease, diabetes, etc. provide additional information to either aid or exclude pain originating from spinal pathology. In patients with symptomatic vascular claudication, Golomb et al. reported the co-prevalence of CAD as high up to 90% in studies using confirmatory angiography.[24] Current treatment guidelines recommend initiation of medical therapy with an anti-platelet agent and statin in symptomatic PAD to reduce risk of MI, stroke, and vascular death.[25,26] Back pain in the presence of constitutional symptoms such as unintentional weight loss, fevers, fatigue, dyspnea, etc. may indicate underlying malignancy and warrant further workup as well. Most importantly, acknowledging a patient's medical comorbidities and overall medical status ultimately determines their surgical risk and helps guide their course of treatment.

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