Low Back Pain in Adolescent and Geriatric Populations

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KEYWORDS

• Adolescent • Geriatric • Scoliosis • Low back pain • Spinal stenosis

KEY POINTS

- Adolescents have back pain in increasing numbers as they grow older.
- Back pain unassociated with a specific cause is the most common diagnosis associated with adolescents.
- Lumbar spinal stenosis is becoming an increasingly frequent clinical problem as the number of geriatric individuals increases.
- Consensus exists in regard to historical factors associated with the diagnosis of lumbar spinal stenosis.
- Debate remains concerning the long-term improved outcome of lumbar spinal stenosis patients treated with surgical decompression versus nonsurgical interventions.

INTRODUCTION

As discussed in the American College of Rheumatology Pain Management Task Force report in 2010, pain is the most common symptom of patients with rheumatic disorders.[1] Both acute pain and chronic pain are associated with inflammatory and noninflammatory rheumatic conditions. The generation of this symptom in the spine may have different origins depending on the underlying disorder (**Table 1**). With such a broad range of disorders, all age groups are at risk. Adolescents and older individuals are the subjects of this article.

ACUTE AND CHRONIC PAIN

Acute spinal pain may be generated by most anatomic structures of the spine except the interior of intervertebral discs. Acute injury to tissues results in a local inflammatory process that is recognized by nociceptive peripheral nerves. When peripheral nerves are damaged, a neuropathic component of pain may become manifest. When pain persists with modification of the pain pathways, uncoupled from the signs of injury

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Rheum Dis Clin N Am 47 (2021) 149–163
https://doi.org/10.1016/j.rdc.2020.12.001
rheumatic.theclinics.com

Table 1
Disorders associated with low back pain

Mechanical	*Rheumatologic*
Muscle strain	Ankylosing spondylitis
Herniated intervertebral disk	Reactive arthritis
OA	Psoriatic arthritis
Spinal stenosis	Enteropathic arthritis
Spondylolisthesis	Diffuse idiopathic skeletal hyperostosis
Adolescent/adult scoliosis	Fibromyalgia
Infectious	Polymyalgia rheumatica
Osteomyelitis	*Endocrinologic*
Discitis	Osteomalacia
Pyogenic sacroiliitis	Osteoporosis
Herpes zoster	Parathyroid disease
Neoplastic/infiltrative	Microcrystalline disease
Osteoid osteoma	*Referred pain*
Osteoblastoma	Aortic aneurysm
Osteochondroma	Pancreatitis
Giant cell tumor	Gall bladder disease
Gaucher disease	Kidney
Skeletal metastases	Bladder
Multiple myeloma	Uterus
Chordoma	Ovary
Neurologic/psychiatric	Prostate
Neuropathic arthropathy	*Miscellaneous*
Neuropathies	Paget disease
Psychogenic rheumatism	Vertebral sarcoidosis
Malingering	Retroperitoneal fibrosis
	Subacute bacterial endocarditis

Modified from Borenstein DG, Wiesel SW, Boden SD. Low Back and Neck Pain: Comprehensive Diagnosis and Management, 3rd edition Appendix A Philadelphia: WB Saunders 2004. p. 870 to 889; with permission.

and inflammation, acute pain becomes chronic and a problem unto itself.[2] Under these circumstances, changes in the central nervous system, such as central sensitization and increased psychological response to pain, make treatment of the disorder more difficult. In both adolescents and geriatric patients, peripheral, neuropathic, and central components of pain may be playing roles in their complaints. These considerations are important when prescribing nonpharmacologic and pharmacologic therapies for these patients.[3]

ADOLESCENT SPINE DISORDERS

Epidemiology

Low back pain (LBP) affects upwards of 80% of the world's population. The common cold is the only disorder that occurs more frequently than spinal pain. The incidence of LBP in the United States is estimated at 59 million individuals in a 3-month period.[4]

In regard to adolescents, a cross-sectional survey of 3669 healthy individuals 10 years to 18 years of age demonstrated that 33% reported back pain in the previous year. Of these, 26.3% reported severe pain (defined as pain intensity $\geq$7 on a 0–10 scale). The prevalence increased with age and was above 45% in the 17-year and 18-year age groups. Pain was located most often in the lumbar region (68.9%) followed by the thoracic, sacral, and cervical regions.[5] In this same cohort, 40.9% of adolescents with back pain received some form of treatment, including physical therapy (most frequently, massage chiropractic adjustments) or medication.

A Danish birth cohort study, including 46,726 children, using a definition of pain combining both frequency and intensity of pain, reported that 14.1% of the participants had severe (3.7%) or moderate (10.4%) pain. For both categories, girls reported more LBP than boys. In terms of impact, approximately 6% of the cohort reported greater than 2-times daily-life consequences by means of a composite measure based on questions about school absenteeism, physical activity restrictions, and health care utilization.[6]

Overall, children and adolescents seem to tolerate back pain better than their adult counterparts but quality of life (QOL) is reduced in subjects reporting multiple painful regions[7] or whole-body pain.[8]

Adolescents self-reporting LBP and whole-body pain showed a decreased QOL but also had more health problems (unrelated to the spine) and more life events (unrelated to health) than adolescents who were free of pain or those who reported pain limited to the lower back.[8]

Causes

Psychological factors are stronger predictors of incident LBP than mechanical factors in adolescent populations. In these individuals, an organic etiology for back pain cannot be found despite thorough investigation. A large study analyzed the data of 215,592 American adolescents presenting with LBP from 2007 to 2010. During 1 year after the initial presentation, patients were tracked for imaging obtained and eventual subsequent spinal pathology diagnoses. More than 80% of patients had no identifiable diagnosis at follow-up.[9] Family structure also may play a role in the frequency of back pain as a complaint. The frequency of a weekly complaint of back pain in male adolescents ranged from 8.5% among boys with joint physical custody to 15.5% in single-parent paternal homes. Among female adolescents, the values ranged from 19.1% in girls living in a nuclear family to 28.4% for those in a step family.[10]

As with older adults, mechanical disorders do cause LBP in adolescents.[11] A spectrum of these disorders, as listed in **Table 2**, include muscle strain, disk herniation, spondylolysis, spondylolisthesis, Scheuermann disease, and scoliosis.

Early-onset scoliosis is defined as lateral curvature of the spine greater than 10° with onset before the age of 10 years. The category includes several types: congenital (i.e., structural abnormalities of the spine or thorax), neuromuscular, miscellaneous (i.e. any other syndrome excluding the previous 2 types), and idiopathic. In terms of age, scoliosis is named infantile (ie, onset from birth to 2 years of age), juvenile (ie, onset from 3 years to 9 years of age), or adolescent (ie, onset from 10 years to 18 years). Most adolescents with nonprogressive idiopathic scoliosis can be seen by a primary care physician or rheumatologist and do not require active treatment.[19] Characteristics of adolescent scoliosis are listed in **Box 1**.

Inflammatory illnesses, including spondyloarthritis, discitis, tumors (both benign and malignant), and neurologic neoplasms occur less frequently than mechanical disorders.[23]

Table 2
Adolescent low back pain studies

Reference	Study Design	Study Population	Study Findings	Comments
Ramirez et al,[12] 2019	Retrospective LBP patients with whole-spine MRI Patients with spondylolysis excluded	N = 388 Women—270 Men—118 Age 10–18 y	158 abnormal MRI Disk disease 122 Syringomyelia 4 Spinal cord tumor 4 Tethered cord 2 Paraspinal cystic mass 2 Bone edema 1	Incidental findings —Schmorl nodes Ovarian/renal cysts Hemangiomas Liver mass Facet joint disease
Yamashita et al,[13] 2019	Retrospective LBP athletes—diagnosis by GO vs SS	N = 69 Women—15 Men—54 Age 9–19 y (15.2 y ± 2.3 y)	GO vs SS Lysis 47 vs 51 Disk[a] 7 vs 11 Facet arthritis 1 vs 4 Apophyseal ring Fx 1 vs 1 Unidentified 13 vs 1 Articular process Fx 0 vs 1	SS provided a second opinion to patients—ordered more STIR-MRIs and functional blocks than GO
Brooks et al,[14] 2018	Retrospective Birth to 18 y old Pediatric emergency room Back pain chief complaint 1-year period	N = 232 encounters 177 study subjects Women—103 Men—74 Age <4 y—2.8%; 4–12 y—37.9%; >12 y—59.3%	Discharge diagnoses Nonspecific LBP—76.8% Other noninfectious 12.4% Other infectious 8.5% Radiology performed 37.9% Abnormal findings 16.9% Laboratory tests 35%	Plain radiographs—not CT or MRI associated with pathology 21% had problems unrelated to spine (abdominal, GU, GYN)
Yang et al,[9] 2017	Retrospective National insurance database 2007–2010 Consults for LBP Followed for 1 y	N = 215,592 Women—57% Men—43% Age 10–14 y—35% Age 15–19 y—65%	Database diagnosis LBP unspecified—80.3% Spasm—8.9% Scoliosis—4.7% Degenerative disk—1.7% Disk herniation—1.3% Spondylolysis, olisthesis, infection, tumor, fracture <1%	84%—no imaging

MacDonald et al,[15] 2016	Retrospective Pediatric sports clinic 242 encounters (71 initial) 1-y duration	N = 93 Women—50 Men—43 Age 14.1 y ± 2.3 y	Nonspecific LBP 148 Scoliosis 17 The numbers refer to visits.	Micheli Functional Scale Validation study Positive correlation Oswestry Disability Index
Gennari et al,[16] 2015	Retrospective Single-center 2009–2014	N = 116 (LBP in 69) Age 13.6 y Tumors and dysraphism excluded	Nonspecific LBP 32 Scoliosis 31 Scheuermann 23 Spondylolysis 13 Spondylolisthesis 5 Osteoid osteoma 1 Eosinophilic granuloma 1	Low numbers for a 5-y study
Ramirez et al,[17] 2015	Prospective Single-center 2 y duration Systematic approach to chief complaint—LBP	N = 261 (8.6% of all visits) Women—177 Men—84 Age 4–18 y, mean age 13.9 y Diagnostics yield with history, physical examination, plain radiographs (8.8%); bone scan (22%); MRI (36%)	34% identifiable pathology Scoliosis—90 (Cobb angle >25° in 20 patients)	Scoliosis not included as a source of pain
Miller et al,[18] 2013	Retrospective 8-y duration	N = 2846 Women—63% Men—37% Mean age 14.3 y	Nonspecific LBP 2159 Spondylolysis 136 Spondylolisthesis 59	No mention of scoliosis

Abbreviations: FX, fracture; GO, general orthopedists; GU, genitourinary; GYN, gynecologic; SS, spine surgeons; STIR, short tau inversion recovery.

[a] In the article by Yamashita and colleagues, the numbers of disk herniation and discogenic pain are presented separately. The 2 categories have been pooled together in this table.

Box 1
Characteristics of adolescent scoliosis

1. Adolescent scoliosis has onset at age greater than 10 years and less than 18 years.[20]
2. In terms of etiology, a vast majority of cases are idiopathic (adolescent idiopathic scoliosis).
3. The overall prevalence ranges from 0.47% to 5.2%.
4. Taking into account the prevalence of backache in adolescents (general population), back pain is NOT frequently a relevant problem in teenagers with adolescent idiopathic scoliosis.
5. The previous statement does NOT apply to scoliotic adults who frequently report significant back pain secondary to degenerative changes in the spine.
6. Adolescents' back pain correlates better with patients' self-perception of their image than with number of abnormal spinal biomechanical variables.
7. Pain catastrophizing has been reported to be an important construct in adolescent idiopathic scoliosis–related pain and should be evaluated.[21]
8. Skeletally mature patients with curves less than 40°–45° should be observed if there is no pain, no progression, and no imbalance.[22]
9. The main predictors of outcome are the magnitude of the curve (Cobb angle), the stage of skeletal maturity (different methods of evaluation exist), and the remaining growth potential.

Clinical Evaluation

History/physical examination

As with adults, characteristics of the onset, quality, duration, location, and radiation of pain may be helpful identifying an underlying pathology. For example, for pain radiating to the groin, particularly in a female adolescent, unrecognized hip dysplasia may be the cause.

A study attempted to determine the sensitivity, specificity, and likelihood ratios of constant pain, night pain, and abnormal neurologic examination to predict the presence of an underlying positive finding (based on magnetic resonance imaging [MRI]) as a cause of back pain. In this series, 388 patients (mean age 14.5 years $\pm$ 2.6 years) underwent MRI, which showed any pathologic condition in 158 (40.8%). Of these, 122 (31.4% of the whole sample) presented disk disease, 3.6% had other pathologic findings, and 5.8% had findings considered incidental. An abnormal neurologic examination (in only 2% of cases) appeared to be the strongest predictor for the presence of any underlying pathologic condition, with very low sensitivity (0.05) and good specificity (0.95).[12]

Imaging

Plain radiographs remain the best screening examination for adolescents with back pain. The anteroposterior view demonstrates vertebral body alignment. Lateral view reveals disk space narrowing, end-plate irregularities, and bony modifications. Oblique views are not needed because spondylolysis is identified on the anteroposterior and/or lateral view.[18]

As with adults, MRI identifies anatomic abnormalities in asymptomatic adolescents. Asymptomatic pediatric subjects examined by MRI show frequent incidental findings, mainly disk related. The prevalence of different findings ranges from 2.9% for disk herniation or protrusion to 51.6% for abnormal nucleus shape. Degenerative disk disease occurs in 19.6% and disk space narrowing in 33.7%.[24]

A systematic review and meta-analysis reported the prevalence rates in nonathletes without LBP, in athletes with LBP, and in athletes without LBP. The pooled prevalence rates were, respectively, 22%, 44%, and 22% for disk degeneration; 1%, 38%, and 13% for herniated discs; 5%, 22%, and 11% for end-plate changes; and 0%, 30%, and 6% for pars fractures.[25]

Feldman and colleagues[26] evaluated their algorithmic approach on a group of 87 adolescents (mean age 13.4 years). Specific diagnoses were obtained in 21 cases with initial radiographs. Of the 66 subjects with negative radiographic findings, MRI was obtained in 19 cases of patients who reported having constant pain, night pain, or radicular pain and/or who had an abnormality on neurologic examination. Ten of the 19 patients had MRI findings that were positive for a specific diagnosis. Overall, of 31 patients with a specific diagnosis, radiographs already showed the pathology in 21 cases whereas MRI was necessary in 10 additional cases. The usefulness of the main clinical variables were summarized as follows: sensitivity ranged from 15% (for thoracic pain) to 67% (lumbar pain), specificity from 54% (lumbar pain) to 100% (radicular pain or abnormal neurologic examination), positive predictive value from 17% (thoracic pain) to 100% (radicular pain or abnormal neurologic examination), and negative predictive value from 56% (thoracic pain) to 75% (lumbar pain).[26] In a French cohort of 116 adolescents evaluated over a 5-year period, the largest group was 32 individuals with a diagnosis of nonspecific LBP. The other groups included 31 with scoliosis, 23 with Scheuermann disease, 13 with spondylolysis, 5 with spondylolisthesis, 8 with transitional vertebral abnormalities, 2 with disk herniations, 1 with osteoid osteoma, and 1 with eosinophilic granuloma.[16]

Management

Most of the published series of adolescent patients show a majority of individuals have mechanical causes for LBP and consequently a majority are treated conservatively.[11] Nonsurgical therapy may include activity modification for a period of rest. Nonsteroidal anti-inflammatory agents may be useful. For children with more chronic symptoms, core strengthening and improved flexibility are helpful. Exercise treatment has been found effective in 4 studies for the treatment of LBP, with an average improvement of almost 3 on a pain visual analog scale over the previous month, but has no effect of reducing the prevalence of LBP in adolescents.[27]

The use of opioids for therapy for LBP and chronic pain in general remains controversial. A Cochrane systematic review has highlighted the complete absence of studies eligible for the review, preventing the investigators from commenting about the efficacy or harm of opioids in adolescents.[28] The need for more research has been highlighted in a study of effect the impact of prescription opioids had on 140 of the 283 patients aged 18 years to 23 years followed at a tertiary-care pain clinic[29] The impact of pain-related interference with activities of daily living along with the use of opioid drugs impeded the expected transition to young adulthood with age-appropriate development of cognition, emotion, behavior, and stress responses needed to cope with a chronic condition.

A determination in regard to nonsurgical or surgical therapy depends on the natural history of the malady and its potential impact on adult QOL. The aim of treatment is to resolve pain in adolescence, if possible, to forego the chronicity of the process as an adult.[29]

Health Care Transition

The transition from pediatric, parent-supervised health care to more independent, patient-centered adult care is not an automatic process.[30] Adult rheumatologists

must be aware that this transition may be difficult for an adolescent with a chronic condition like LBP. A vast majority of US adolescents do not receive any transition preparation. A major barrier, for example, is difficulty in leaving their pediatric clinician with whom they have had a long-standing relationship. Communication between the pediatric and adult rheumatologists can help bridge the transition of care.

Summary—Adolescent Spine Disorders

The prevalence of LBP increases with age from childhood and approaches the values found in adults by the end of adolescence. Specific diagnoses are identified more frequently in juveniles than in adults, but nonspecific LBP remains the most frequent diagnosis in adolescents. Combining an extensive clinical evaluation and imaging studies leads to the identification of an increased number of spinal pathologies. Nevertheless, the results of imaging studies in asymptomatic subjects show an important number of incidental findings. Adolescents with nonspecific LBP usually can be managed with some form of nonsurgical therapy, including exercise.

GERIATRIC SPINE DISORDERS (SPINAL STENOSIS)

Epidemiology

In 2010, a Global Burden of Disease study ranked LBP the highest of the 291 conditions studied in terms of years lost to disability.[31] In 2015, an update to that study estimated that 266 million individuals (3.63%) worldwide had lumbar degenerative spine disease.[32] Based on population sizes, low-income and middle-income countries had 4-times as many cases as high-income countries; 39 million individuals (0.53%) worldwide had spondylolisthesis, 403 million (5.5%) individuals had symptomatic disk degeneration, and 103 million (1.41%) had spinal stenosis annually.

Cause

The first manifestations of aging in the spine occur in the intervertebral discs. The nucleus pulposus loses its resistance to compressive forces and the annulus fibrosus fissures, resulting in degeneration of fibers. With an inadequate annulus fibrosus, the nucleus pulposus protrudes or herniates. The result of this process is an intervertebral disk that is narrower at that interspace.

Secondary to disk space narrowing, increased pressure is placed on apophyseal joint cartilage. The severity of facet joint osteoarthritis (OA) is related directly to the degree of disk space narrowing. The converse does not occur, so disk degeneration is the initiating factor in facet arthritis.[33] The resulting biomechanical insufficiency from disk degeneration, including loss of paraspinous muscle mass, transfers forces posteriorly to the ligaments and facet joints. Disk degeneration affects women and men equally and increases with age. Like other osteoarthritic joints in the body, the presence of modification of joint anatomy is not related directly to the presence of pain.[34] Localized back pain occurs when alterations in facet joint alignment and pressure results in articular pain.

In an attempt to decrease pressure on painful joints, the lumbar lordosis may flatten. Placing pressure on the anterior components of the vertebrae decompresses the facet joints but places increasing tension in the supporting muscles, which may fatigue and become painful.

The growth of facet osteophytes, protrusion of intervertebral discs, and redundancy of the ligamentum flavum reduce the space in the spinal canal or neural foramen. With decreased volume, the vasa nervorum is compressed. With decreased blood flow, neurogenic claudication occurs with associated pain in the corresponding nerve

distribution. With reversal of the compression, blood flow is restored and pain is relieved. The longer the duration of the vascular compromise, the more persistent and total becomes the neural dysfunction. The clinical correlate of this pathophysiology is radicular pain followed by numbness and muscular weakness in the lower extremities.

Clinical Evaluation

History/physical examination

Neurogenic claudication is the most common symptom associated with lumbar spinal stenosis (LSS). Pain that is associated with standing or walking occurs in the buttock, thigh, or lower leg. The patterns of back pain and/or leg pain vary with the patients with the disorder. Most patients have back pain and leg pain. A smaller proportion of patients have leg pain alone. Some patients have bilateral leg pain. The distribution of leg pain may be different in each extremity. Multiple dermatomes may be affected. In the setting of widespread distribution of symptoms, ascribing compression to a single nerve root lesion is difficult. In addition to pain, patients may have paresthesias, numbness, or weakness. Neurogenic claudication is relieved by flexing at the waist, lying down, or sitting.

A total of 279 musculoskeletal physicians participated in a Delphi method to reach a consensus concerning the historical factors associated most closely with LSS.[35] The most important history items for diagnosis of LSS included leg pain or buttock pain while walking, flexing forward to relieve symptoms, feeling relief when using a shopping cart or bicycle, motor or sensory disturbance while walking, normal and symmetric foot pulses, lower extremity weakness, and LBP. The presence of 6 of these characteristics is associated with an 80% certainty of LSS diagnosis.

LSS and hip OA occur in older individuals. Symptoms and signs of these 2 conditions may overlap. A group of 51 musculoskeletal physicians participated in a series of surveys to differentiate hip OA from LSS.[36] Eight symptoms favoring hip OA over LSS included groin pain, knee pain, pain that decreases with continued walking, pain that occurs immediately with walking, pain that occurs immediately with standing, pain getting in/out of a car, pain with dressing the symptomatic leg, and difficulty reaching the foot of the symptomatic leg while dressing. Three symptoms favoring LSS over hip OA included pain below the knee, leg tingling and/or numbness, and some pain in both legs. Symptoms that did not discriminate included decreased pain with using a shopping cart, back pain, weakness and/or heaviness of a leg, buttock pain, poor balance, increased pain with weight bearing on painful leg, and stair walking.

Patients with LSS may have no findings on physical examination in a seated position. Abnormalities may appear only after stressing a patient with walking until leg pain appears.[37] Sciatica caused by LSS is distinct from radiculopathy associated with an intervertebral disk in that objective neurologic abnormalities like asymmetric reflexes are found in a minority.[38] Also of utility is checking for the presence of foot and ankle pulses to identify those individuals who may be at risk for vascular claudication.

Seven physical findings favoring hip OA over LSS include limited weight bearing on painful leg when standing, observed limping, and pain or restricted motion with 5 hip maneuvers. Neurologic deficits favored a diagnosis of LSS over hip OA.[36]

Imaging

Many radiographic techniques exist to evaluate LSS patients.[39] The least sensitive but most available is plain radiographs of the lumbar spine. Identifiable abnormalities

include end-plate sclerosis, disk-narrowing, facet-joint hypertrophy, spondylolisthesis, and neural foraminal osteophytes. Soft tissues and neural elements are not visible. Radiographic abnormalities are compatible, but not diagnostic, of spinal stenosis because similar radiographic abnormalities are noted in asymptomatic individuals. MRI can identify bony anatomy, neural elements, vascular structures, and other soft tissues like ligamentum flavum and paraspinous muscles. MRI is the radiographic technique with the greatest potential for identifying anatomic abnormalities associated with LSS. Specific measurements, however, indicative of a definitive diagnosis of LSS are yet to be determined[40] (**Fig. 1**).

Computed tomography (CT), with or without myelography, is a technique using larger exposure of radiation to identify the osseous structures in the spine. This technique is used when patients are unable to have an MRI because of claustrophobia, pacemakers, or other contraindications to MRI.[41]

Also of note is the lack of benefit of early radiographic evaluation in older adults without radiculopathy with an improved outcome at 1 year.[42] The degree of disability was the same in those who had radiographs (Roentgenograms or MRI) versus those treated without the benefit of imaging.

Diagnosis of LSS remains a clinical one because no specific set of clinical, radiographic, or interventional tests is definitive. Therefore, the patient with LSS is characterized by specific historical and physical findings and confirmed, but not diagnosed, by radiographic techniques documenting the compression of neural structures.

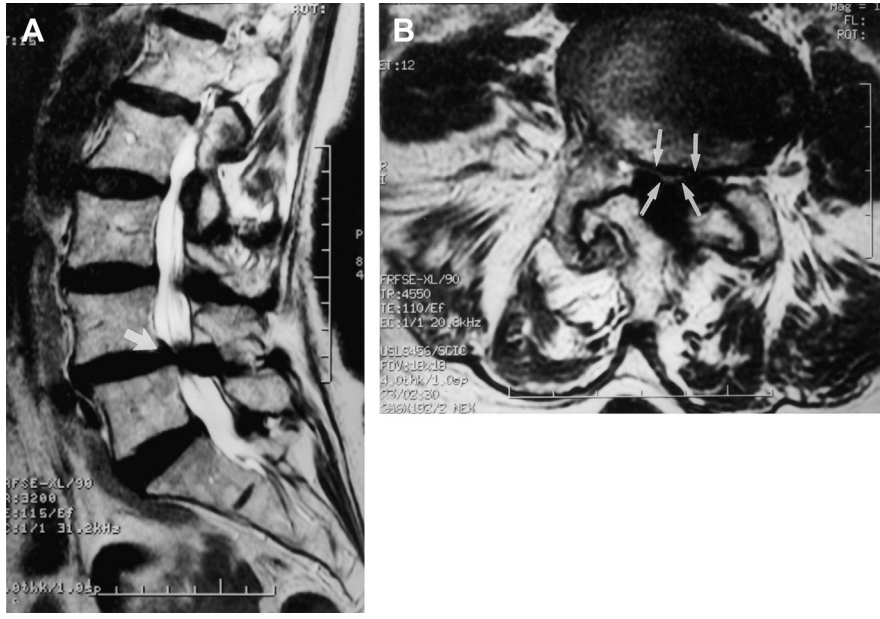

Fig. 1. MRIs of the lumbar spine of a 92-year-old woman with leg numbness and difficulty ambulating. Sagittal (*A*) and axial (*B*) views demonstrating severe stenosis at L4-L5 (*arrows*) caused by facet joint osteophytes, a protruding intervertebral disk, and redundant ligamentum flavum. (*From* Borenstein DG, Wiesel SW, Bowden SD. Low Back and Neck Pain: Comprehensive Diagnosis and Management 3rd edition pg. 272; with permission.)

Management

LSS management requires judgment that matches the severity of functional impairment with benefits and risks of interventions. In older adults, determination of their most severe limitation is essential. Is cardiac, pulmonary, or vascular disease their most significant physical limitation? Or is LSS with the development of neurogenic claudication their greatest disability?

The underlying pathophysiology of LSS is the compression of vascular supply to the neural elements. The goal of therapy is to maximize the space in the spinal canal by expanding volume (flexion of the spine) and shrinking inflamed, swollen tissues.

The options include education, weight reduction, exercises, smoking cessation, pharmaceuticals, injections, and surgery. No one therapy works for all patients. A combination of options may be necessary to control symptoms. Surgical decompression is an appropriate choice for individuals who have not responded to nonsurgical interventions or have neurologic compromise that severely impairs function.[43]

Pharmacologic therapies in the forms of nonsteroidal anti-inflammatory medications, acetaminophen, gabapentin, pregabalin, and opioids all have potential toxicities that limit their full potential as therapeutic agents. Duloxetine has an indication for the treatment of chronic LBP but not for the treatment of LSS.[44]

Epidural corticosteroid injections are the most commonly performed outpatient procedures for the treatment of spinal pain.[45] Epidural injections are given in a series of 3. For the pathophysiology of LSS, the injections are delayed until symptoms recur because injections can be given no sooner than every 2 months. The benefits of epidural steroids are time-limited.[46,47]

Surgical decompression is indicated in individuals who have failed medical therapy and are physically incapacitated by spinal stenosis. The goal of surgery is to obtain adequate decompression without causing instability. The difficulty for the spine surgeon is identifying the most symptomatic level and determining the extent of the decompression. The need for fusion and instrumentation remains a controversial decision. It is not clear that individuals who have a fusion necessarily have an improved outcome.[48]

For older adults who are not candidates for decompressive surgery, interspinous spacers are placed with less invasive techniques. The placement of an interspinous spacer in an LSS individual without spondylolisthesis may improve the vertical space in the foramen and decompress the corresponding spinal nerve. Compared with decompressive surgery, spacing devices have fewer complications but higher rates of revision surgery.[49]

Conservative Versus Surgical Treatment

Different studies have followed patients with LSS treated with conservative management versus surgical decompression for variable durations. Some studies have suggested the short-term benefit of decompression whereas long-term follow-up suggests similar outcomes for those treated with either regimen.[43,50,51]

Summary—Geriatric Spine Disorders

The frequency of LSS will increase as a clinical problem as the geriatric population ages. Neurogenic claudication will become a cause of significant disability in those without comorbidities that limit function. Historical factors can help differentiate those patients with LSS from individuals with hip joint arthritis. MRI is the best radiographic technique to reveal those with anatomic findings of nerve compression but is not specific in identifying those who are symptomatic. The aim of therapy is to relieve

compression on spinal nerves. Whether surgical decompression or medical therapy is the best method to achieve that end remains to be determined.

CLINICS CARE POINTS

- A systematic, complete, and adapted clinical history and physical examination for adolescents are good guides to the additional investigations needed to choose appropriate management.
- Rheumatologists need to be cognizant of the risks of indiscriminate imaging studies in adolescents that result in overtreatment.
- A critical evaluation of response to therapy is necessary to avoid overlooking specific pathologies that require more aggressive approaches.
- LSS is an increasingly common clinical problem as the population ages.
- Spinal stenosis and hip OA are differentiated with specific historical and physical findings.
- The pros and cons of surgical management have to be weighed carefully in terms of risks and benefits in deciding on appropriate therapy for neurogenic claudication.

DISCLOSURE

D.G. Borenstein, MD—research grant, Abbvie. F. Balagué, MD—the author has nothing to disclose.

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