

Back Pain

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Case Presentation

A 48-year-old obese man presents to the Physical Medicine and Rehabilitation (PM&R) clinic with new onset low back pain. He reports that low back pain, for years without clear onset that intermittently radiates into his right and left posterior thighs, is worse when standing and is associated with morning stiffness. However, he states that he had a sudden onset of back pain 3 weeks ago when he was moving furniture in his home. His pain is located at the lower back and radiates through his left leg. He characterizes his low back pain as cramping and aching and characterizes his left leg pain as burning and electric. His leg pain is worse with sitting and his back pain is worse when coughing. He takes an occasional Tylenol, which seems to help but only temporarily.

REVIEW OF SYSTEMS

He reports associated numbness at the plantar aspect of the left foot. He denies weakness. He denies bowel and bladder incontinence. He denies any weight loss, night sweats, or fevers.

Past medical history: He has a history of hypertension (HTN) for which he is taking Losartan 25 mg daily for the past 10 years.

Social history: Works as a lawyer, recently unemployed 6 months ago. He lives with his wife and two children on the fourth floor with an elevator. He ambulates without assistive device and is independent in his activities of daily living. He smokes 1 pack of cigarettes per day for the past 20 years. He does not drink alcohol or use intravenous drugs.

Allergies: No drug or environmental allergies

Medications: Losartan 25 mg daily, Tylenol as needed

BP: 140/70 mmHg, RR: 16/min, PR: 80 per min, Temp: 97° F, Ht: 5'6", Wt: 220 lbs, BMI 35 kg/m²

Head, eyes, ear, nose, and throat (HEENT)-extraocular movements (EOMs) full, no ptosis

PHYSICAL EXAMINATION

General: He is alert and oriented. He is in moderate distress because of left-sided back pain.

MUSCULOSKELETAL AND NEUROLOGIC EXAMINATION:

Lateral bending and extension is limited by pain, flexion of trunk is significantly limited by pain
Diffusely rigid and tender to palpation along middle and lower paraspinal muscles

Motor examination: right and left lower extremities 5/5

Bilateral Achilles tendon reflex and bilateral patella tendon reflex 2+. Plantar reflex is down-going bilaterally. No clonus bilaterally.

Sensory examination: Dull to light touch along the left lateral thigh, lateral calf, and dorsum of foot.

Normal sensation along right lower extremity.

Straight leg test is positive on the left side and negative on the right side

Gait is normal

General Discussion

The approach to a patient with lower back pain (LBP) generally involves categorizing distinct sources of back pain: axial, radicular, and referred pain.¹ Axial lumbosacral pain involves the lumbar region, L1–L5 vertebral segments, and sacral region, S1 to sacrococcygeal region.¹ Axial back pain is commonly used to describe LBP associated with degenerative disc disease without compromise of neural elements. Radicular pain involves radiation of pain that travels through the

TABLE 2.1 ■ Red Flags

Trauma
- Major trauma
- Minor trauma in elderly or osteoporotic patients
Infection/Tumor
- History of malignancy
- New onset back pain age <20 years or age <50 years
- Constitutional symptoms
- Recent infection
- Immunosuppression
- Intravenous drug use
- Pain worse at night
Neurologic compromise
- Severe or worsening sensory or motor deficits
- New bowel or bladder dysfunction
- Saddle anesthesia

leg along a dermatomal distribution consistent with a nerve root, or dorsal root ganglion, level compromise, most commonly secondary to mechanical compression. Referred back pain involves pain that travels from a source in a nondermatome distribution along elements of the same mesodermal origins. Although these terms attempt to simplify etiologies of LBP, LBP is largely a multifactorial condition, including multiple physiologic and psychosocial factors that remain difficult to define, diagnose, and treat.¹

Once categorized, the determination of potentially progressive or unstable etiology of back pain is paramount (Table 2.1). “Red flags” are used to determine such etiologies, including cancer, infection, trauma, and neurologic compromise. These findings include fevers/night sweats/chills, bowel or bladder incontinence, “saddle anesthesia” (decreased sensation around the perineum, groin, and/or medial thighs), thoracic pain, gait ataxia, or prior history of cancer or high impact trauma. Positive findings should prompt emergent evaluation, either via imaging and/or referral to specialists (surgery, oncology, etc.).

As with any evaluation, a thorough history and physical examination are essential in diagnosis of lumbosacral radiculopathy. Regarding history, a complete description of pain is necessary, including onset, location, duration, characterization, alleviating and aggravating factors, radiation, timing, and severity. Associated paresthesias, such as numbness, tingling, and weakness, can often be appreciated as well. Of significant importance is associating these symptoms to a specific dermatome and/or myotome level for radicular back pain, requiring focal questioning (Fig. 2.1). Regarding physical examination, a comprehensive neurologic examination is necessary, including assessment of motor strength and evaluation for upper motor findings (Fig. 2.2). In addition, certain special maneuvers can guide to specific etiologies of low back pain as discussed earlier, such as facet loading test (axial back pain, facet arthropathy), straight leg raise and Slump test with reproduction of symptoms (radicular back pain, L4 to S1 radiculopathy), reverse straight leg raise, and Ely test with reproduction of symptoms (radicular back pain, –L4 radiculopathy; Table 2.2).

Differential Diagnosis

1. **Myofascial pain**—most commonly localized to the low back. There may be radiation to the bilateral lower extremities along the posterior buttocks and thighs. This referral of pain is not consistent with a dermatome distribution, and should not be mistaken for radiculopathy.²

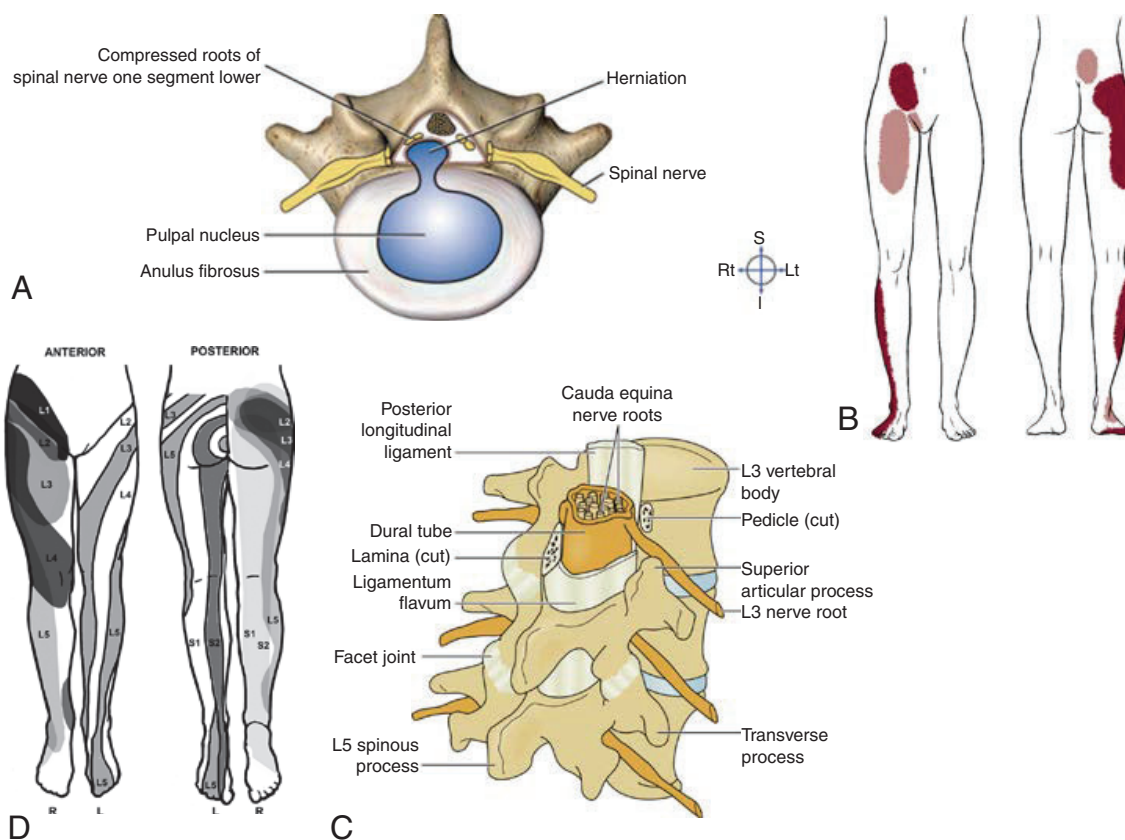


Fig. 2.1 Lumbar radiculopathy. (With permission from: (A) B. Liebgott, *The Anatomical Basis of Dentistry*, 4e, Mosby, Elsevier, 2017; (B) C.C. Goodman, J. Heick, R.T. Lazaro, *Differential Diagnosis for Physical Therapists: Screening for Referral*, 6e, Saunders, Elsevier, 2017; (C) W.R. Frontera, T.D. Rizzo, J.K. Silver, *Essentials of Physical Medicine and Rehabilitation: Musculoskeletal Disorders, Pain, and Rehabilitation*, 4e, Elsevier, 2018; (D) W.S. Bartynski, K.A. Petropoulou, *The MR imaging features and clinical correlates in low back pain related syndromes*. *Magn. Res. Imaging Clin. N. Am.* 15 (2007) 137–154.)

2. Degenerative spine disease

Discogenic pain: Most commonly associated with low back pain worse with flexion, sitting, twisting, and increased abdominal pressure (coughing, sneezing).³

Facet arthropathy: Most commonly associated with extension and lateral bending.

Classically associated with facet-loading test positive; however, studies have shown it is unreliable in diagnosing facet arthropathy mediated pain alone.⁴

3. **Lumbosacral radiculopathy**—most commonly associated with leg pain, oftentimes will supersede lumbar pain, such that patients will experience leg pain greater than back pain.⁵ This pain usually radiates in a dermatomal fashion, associated with the pathologic nerve root.
4. **Lumbar stenosis**—can present with low back pain, neurogenic claudication (discomfort, pain, numbness or weakness in the calves, buttocks, or thighs that is precipitated in lumbar extension and relieved in lumbar flexion), sensory disturbances in dermatomal and nondermatomal fashion, motor weakness, and pathologic reflexes.⁶
5. **Lumbar postlaminectomy syndrome**—commonly described as “failed back surgery syndrome”; it is defined by International Association for the Study of Pain as “lumbar spinal pain of unknown origin either persisting despite surgical intervention or appearing after surgical intervention for spinal pain originally in the same topographic location.”⁷ Comprehensive history should include evaluation of preoperative risk factors (psychosocial factors, smoking, obesity), intraoperative risk factors (operating at a single level, operating at the wrong level), and postoperative risk factors.⁸

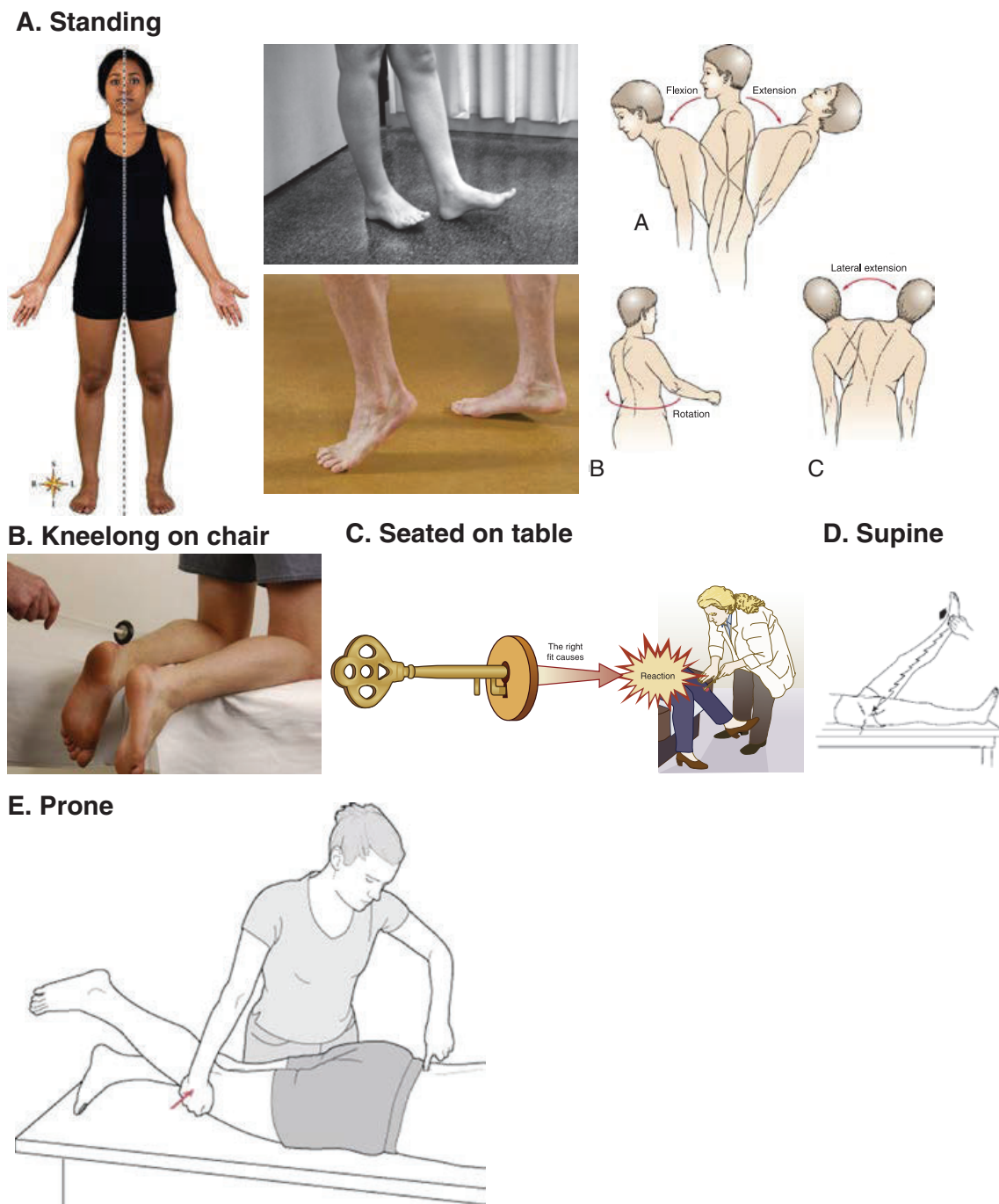


Fig. 2.2 Lumbar radiculopathy. (With permission from: (A) K. Patton, G. Thibodeau, *Structure & Function of the Body*, 16e, Elsevier, St Louis, 2020, Fig. 1-3; T. Cueco, *Essential Guide to the Cervical Spine: Volume One: Clinical Assessment and Therapeutic Approaches*, Elsevier, Philadelphia, 2016, Fig. 8-63; R.C. Evans, *Illustrated Orthopedic Physical Assessment*, 3e, Mosby, Philadelphia, 2009, Fig. 8-77; M.H. Swartz, *Textbook of Physical Diagnosis: History and Examination*, 8e, Elsevier, Philadelphia, 2021, Fig. 20-22; (B) N.J. Talley, S. O'Connor, *Clinical Examination Volume One: A SysGuide to Physical Diagnosis*, 8e, 2018, Fig. 28-11; (C) A. Guerra, K. Davis, *Mosby's Pharmacy Technician: Principles and Practice*, 5e, Churchill Livingstone, Australia, 2019, Fig. 17-5; (D) V.J. Devlin, *Spine Secrets*, 3e, Elsevier, Philadelphia, 2021, Fig. 1-3; (E) L. Chaitow, J. DeLany, *Clinical Application of Neuromuscular Techniques: Volume 2: The Lower Body*, 2e, Churchill Livingstone, London, 2012, Fig. 10-39.)

TABLE 2.2 ■ Diagnosis of Lumbar Radiculopathy

Nerve Root	Pain Radiation	Gait Deviation	Motor Weakness	Sensory Loss	Reflex Loss
L3	Groin and inner thigh	Sometimes analgic	Hip flexion	Anteromedial thigh	Patellar (variable)
L4	Anterior thigh or knee, or upper medial leg	Sometimes analgic Difficulty rising onto a stool or chair with one leg	Knee extension, hip flexion and adduction	Lateral or anterior thigh, medial leg, and knee	Patellar
L5	Buttocks, anterior or lateral leg, dorsal foot	Difficulty heel walking; if more severe, then foot slap or steppage gait Trendelenburg gait	Ankle dorsiflexion, foot eversion and inversion, toe extension, hip abduction	Posterolateral thigh, antero-lateral leg, and mid-dorsal foot	Medial hamstring (variable)
S1	Posterior thigh, calf, plantar foot	Difficulty toe walking or cannot rise on toes 20 times	Foot plantar flexion	Posterior thigh and calf, lateral and plantar foot	Achilles

(With permission from M.J. Ellenberg, M. Ellenberg, Lumbar Radiculopathy, In: W.R. Frontera, J.K. Silver, T.D. Rizzo (eds), Essentials of Physical Medicine and Rehabilitation, 4e, Elsevier, 2019, pp257–263.

6. Other causes of low back pain:

- a. Cauda equina syndrome: Constellation of symptoms indicating neurologic compromise related to dysfunction of the ropelike nerve fibers at the distal spinal cord, most commonly caused by large lumbar disc prolapse with compression.⁹ These symptoms include, but are not limited to, saddle anesthesia, sexual dysfunction, fecal incontinence, bladder dysfunction, and lower limb weakness. Magnetic resonance imaging (MRI) is the choice of imaging, and treatment is urgent surgical decompression unless medically contraindicated.⁹
- b. Tumor: The strongest risk factor for back pain secondary to bone metastasis is a history of cancer.¹⁰ Those cancers associated with bone metastasis include breast, lung, renal cell, and prostate cancers.¹¹
- c. Infection: A comprehensive history of infection related to low back pain includes recent fevers, malaise, spinal injections, epidural catheter placement, intravenous drug use, and immunosuppression.¹²
- d. There are numerous other causes of nonspine-related back pain, including fibromyalgia, piriformis syndrome, hip osteoarthritis, and aortic aneurysm

Case Discussion

Our patient presents with acute on chronic low back pain. In evaluating this patient, it is important to delineate his separate pain complaints. On one hand, he describes insidious onset of low back pain, which suggests chronicity. Given his chronic description of aching pain in the low back without dermatomal radiation, risk factors of obesity and smoking, physical examination findings of paraspinal tenderness and rigidity, this patient likely has axial back pain. Therefore differential diagnoses should include those associated with degenerative spine changes, such as discogenic disease, facet arthritis, and spondylosis.

On the other hand, he describes clear onset of pain after an inciting incident, which suggests acuity. Along with history of pain and numbness along L5 dermatome and physical examination findings of positive straight leg test, this patient likely has lumbosacral radiculopathy as well. Therefore differential diagnoses should include space occupying lesions resulting in compression of the L5 nerve root from the central canal to the neural foramen, including disc herniation, degenerative spondylosis, neural foraminal stenosis, and fractures.

DIAGNOSTIC TESTING

In the absence of red flags, trauma, prior spinal surgery, and refractory back pain to conservative management, diagnostic testing is not indicated in the majority of patients with low back pain. Initial treatment should be geared toward conservative management.¹³

Presently, there is no one type of imaging that shows a clear advantage over others. X-rays are a simple, cost-effective method of evaluation of bony anatomy to reveal gross bony abnormalities commonly associated with degenerative spine disease (such as disc space narrowing, osteophyte formation, neuroforaminal stenosis, facet arthropathy), misalignment (spondylolisthesis), and trauma (vertebral fractures, pars interarticularis fractures). X-rays are commonly used for chronic, persistent low back pain with acute pain associated with new red flags secondary to trauma, including fracture or instability. Flexion and extension views are recommended to evaluate symptomatic spondylolisthesis.⁵

In patients with refractory radicular pain syndrome (radicular low back pain lasting 4–6 weeks after conservative management), MRI is considered the gold standard for imaging given its sensitivity for soft tissue evaluation, including disc, tumor, muscle, and nerve involvement. Of note, it is important to evaluate MRI with a high pretest probability, garnered from a precise clinical suspicion based on history and physical examination. Lumbosacral MRI examinations are more likely to be “abnormal” by age 40 years in asymptomatic individuals, and herniated discs are not infrequently found in asymptomatic young adults.⁵

For patients who cannot undergo MRI, computed tomography (CT) remains an alternative option. Although routine CT is not recommended for acute, subacute, or chronic nonspecific or radicular low back pain, CT is recommended for patients with refractory radicular pain who are in consideration for epidural steroid injections. If these patients are in consideration for surgical discectomy or have a history of prior spinal surgery with hardware, CT myelography is recommended.

Bone scans can be used to evaluate for osteomyelitis, occult fractures, and inflammatory arthropathy. Single-photon emission CT (SPECT) imaging has also been used to evaluate inflammatory arthropathy, specifically that of the sacroiliac (SI) joint; however, SPECT imaging is not currently recommended for low back pain evaluation.⁵ Although discography, when paired with MRI or CT, can provide anatomic information for surgical decisions regarding discectomy for significant radiculopathy, the lack of standardization in discography leads to low predictive value and is currently moderately not recommended for evaluation of acute, subacute, and chronic low back and radicular pain.¹⁴

Electrodiagnostic studies (EDX), primarily electromyography (EMG), can be used to evaluate radicular pain syndromes. It can be useful to determine if neurologic compromise is present, chronicity of symptoms, and/or aggravation of preexisting injury.¹⁵ However, it is important to note that EDX tests only motor axonal loss or conduction block, and would not yield abnormalities affecting the sensory nerve root. Therefore EDX is not recommended for low back pain without radicular pain symptoms. In addition, EMG represents a high specificity, low sensitivity test for radiculopathy, serving as a good way to confirmatory test rather than a screening test. Therefore it is important to use EDX studies as a supplement to clinical decision making and EDX results are interpreted in the realm of prevalence of suspected pathology.¹⁶

Case Discussion

Our patient presents with radicular low back pain syndrome. Although he has not completed any conservative management, he does have a history of an inciting, traumatic event. Therefore imaging with X-rays would not be unreasonable.

Because our patient does not exhibit constitutional symptoms, neurologic compromise on examination, or red flags, further diagnostic testing is not indicated. Although MRI would not be necessary at this time, it could yield important information regarding further treatment options, such as interventional management.

Objective Data

X-ray lumbosacral spine—mild lumbar disc space narrowing suggested at L3–S1 without significant change in position of vertebral bodies in alignment upon flexion and extension. Mild diffuse spondylosis is seen without visualized compression fracture. Mild to moderate facet arthropathy is noted in the lower lumbar spine. The pedicles and vertebral heights are intact. The SI joints are patent and symmetric bilaterally.

MRI lumbar spine without contrast—degenerative changes and diffuse ligamentum flavum hypertrophy throughout causing mild to moderate spinal stenosis and mild bilateral neural foraminal stenosis at L3 through S1. Most pronounced, left sided L4–L5 posterolateral disc protrusion.

Laboratory work—complete blood count (CBC) within normal limits. Erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) within normal limits.

On comprehensive review of our patient, he presents with acute L5 radiculopathy on chronic axial low back pain. Regarding his axial back pain, imaging reveals multiple possible etiologies, including degenerative discs, facet arthropathy, and spondylosis. Given his history of pain with extension and morning stiffness, his axial back pain is likely related to facet arthropathy. Regarding his radicular pain, imaging reveals two possible etiologies, including disc herniation and neural foraminal stenosis. Although both processes could cause radiculopathy, the acuity of his presentation suggests that disc herniation is the likely etiology. Diffuse neural foraminal stenosis is likely the result of ongoing degenerative spine disease, and is likely not the primary etiology of his radiculopathy.

Review of Proposed Pathology and Pathobiomechanics

The majority of back pain is caused by myofascial low back pain, which is usually caused by back sprain or lumbago. Myofascial pain is likely a result of stress applied to muscle or ligament resulting from primary trauma or secondary continuous postural instability. Myofascial pain may be associated with trigger points, defined as taut muscle bands secondary to chronic contractions.²

The most widely accepted theory of degenerative spine disease is described in a three-phase model:¹⁷

Phase 1: Dysfunctional phase, characterized by repetitive microtrauma causing annular fissures and tears in the disc, leading to predispositions to disc herniation, inability to retain water, and loss of disc height

Phase 2: Instability phase, characterized by progressive disc tears and disc height loss, leading to additional mechanical stress on the facet joints

Phase 3: Stabilization phase, characterized by further disc height narrowing, fibrosis, and osteophyte formation

This degeneration of the disc cascades stress-induced osteophyte formation throughout the lumbosacral spine is known as spondylosis. Areas of the spine that are commonly associated with degenerative changes include the facet joints and sacroiliac joints. Degenerative changes in the facet joint, also known as facet arthropathy, can also be seen with paraspinal muscle rigidity and weakness. Spondylosis results in osteophyte production that can become either primary pain generators in axial back pain or secondary pain generators in radicular back pain caused by compression on neural elements. Lumbosacral radiculopathy or stenosis is described as a condition in which the spinal cord and nerve roots are entrapped in one of three anatomic locations: (1) central canal, (2) neural foramen, (3) lateral recess. Etiologies of central canal and neuroforaminal stenosis include disc herniation, ligamentum flavum hypertrophy, articular facet hypertrophy, posterior longitudinal ligament ossification, and vertebral body subluxation (spondylolisthesis).¹⁸ Lumbar radiculopathy is most commonly caused by lesions of discs and

TABLE 2.3 ■ General Physical Therapy Program for Low Back

Lumbar stabilization:

1. Local segmental stability: pelvic tilting exercises (most commonly accentuating posterior pelvic tilt to decrease mechanical stress on lumbar spine), core stabilization exercises (strengthening abdominal wall)
2. Closed chain stability exercises: focus on strengthening weight-bearing muscles with gravity-dependent stretch (gluteus maximus/gluteus medius/abdominal wall) with squats, balance board
3. Open chain stability exercises: assisted stretching and strengthening of specific muscles as necessary to assist proper closed chain stability exercises (hip flexor stretch, such as iliopsoas and quadriceps, hamstring stretch, or gluteus medius strengthening)

degenerative disease of the spine. Most incidences are self-limiting and resolve within 1 to 2 weeks (50% of cases) or 6 to 12 weeks (90% of cases).¹³ Lumbar radiculopathy and lumbar stenosis share similar pathologic mechanisms with regard to mechanical compression. Another theory regarding lumbar stenosis is that of vascular compression, a compression of local vascular structures that leads to transient ischemia of the spinal cord from arterial insufficiency and venous stasis.

Treatment Strategies

Low back pain treatment depends on its etiologies, presence of radiculopathy, and physical or radiologic findings.¹⁹ Based on general consensus, conservative management is considered first-line management. Conservative management consists of multiple modalities, including physical and rehabilitation therapy, pharmacologic treatment, psychological treatment, and complementary and alternative medicine approaches.¹

Physical therapy has been shown to have a greater improvement on acute low back pain than chronic low back pain. Stretching exercise is most associated with pain reduction, whereas strengthening exercise is most associated with functional gains.¹ Strengthening exercise is aimed toward the multifidus muscles, transverse abdominis muscles, and deep muscles of the spine that aid in lumbar stability. Lumbar stabilization is commonly used for disc herniation treatment to improve proprioceptive senses of the tissues in surrounding joints.²⁰ Early enrollment of physical therapy in acute low back pain leads to decreased health services utilization and improved pain outcomes compared with late physical therapy.²¹

McKenzie method is a widely accepted physiotherapy technique for low back pain, based on the premise of correlating patient preference in directional movement with exercise prescription to decrease pain. McKenzie method is assumed to be an extension-based therapy, but it is meant to be a highly individualized prescription based on specific patients. However, there is still little efficacy for use of McKenzie method for chronic low back pain.²²

Multidisciplinary therapy (physical and psychological therapy combined), acupuncture, massage, and spinal manipulation have small to moderate effects on pain and function in chronic, nonradicular low back pain. Although many other modalities are commonly used in practice, such as ultrasound, electrical nerve stimulation, lumbar supports, and taping, a lack of randomized control trials lead to inadequate data to confirm benefit of use in low back pain.

See [Table 2.3](#) for general low back physical therapy program protocol.

Pharmacologic treatments are fundamental in acute and chronic low back treatments. Acetaminophen and nonsteroidal antiinflammatory drugs (NSAIDs) are commonly prescribed medications, which are effective in short-term pain relief. Acetaminophen is generally preferred given its safer side-effect profile. When selecting NSAIDs, use of the lowest effective dose for the

TABLE 2.4 ■ Differential Diagnosis, Physical Examination, Physical Therapy Program, Interventions

	Pain Description	Physical Examination	Physical Therapy Program	Interventions
Myofascial	Pain worse at end range of motion, with possible radiation to posterior thighs	Diffuse pain with palpation, ± trigger points	Massage, initial stretching phase followed by strengthening phase	Trigger point injections
Facet-mediated	Pain worse with extension, associated with stiffness	Facet-loading positive, pain with lateral bending	Williams flexion-based therapy	Medial branch nerve blocks and ablation, Facet joint injections
Discogenic	Pain worse with flexion, without radiation, pain with Valsalva	Pain with forward flexion	McKenzie extension-based therapy	Disc biacuplasty, IDET
Disc herniation	Pain that radiates to lower extremities in dermatomal distribution, pain with Valsalva	Straight leg positive, Slump test positive	Posterolateral herniation: extension-based therapy Far lateral herniation: flexion-based therapy	Transforaminal epidural steroid injection, discectomy
Spondylolysis	Pain worse with movement, improved at rest	Pain with flexion or extension	Lumbar stabilization therapy	Bracing, surgery if needed
Sacroiliac Joint	Inferior low back/buttock pain, worse with sitting, intermittent radiation to the posterior thighs	FABER positive, SI distraction	General low back program	Sacroiliac joint steroid injection
Lumbar stenosis	Pain with intermittent radiation into posterior thighs or dermatomal distribution, ± neurogenic claudication	Pain with extension, ± Straight leg or Slump test	General low back program	Epidural steroid injection, decompression (minimally invasive vs. traditional)

FABER, Flexion, abduction, external rotation; *IDET*, intradiscal electrothermal therapy; *SI*, sacroiliac.

shortest duration is recommended given its renal, cardiovascular, and gastrointestinal side effects. Skeletal muscle relaxants have shown short-term relief for analgesic effects; however, there is limited evidence to definitely show differences in efficacy between them. Side-effect profiles to be considered include central nervous system depression and risk for falls. Tramadol and opioids should only be considered as last-line management for low back pain that is refractory to aforementioned therapies. Although potent opioids have shown significant analgesia, their potential for dependence and tolerance discourages any long-term use.¹⁴

Interventional management is reserved for patients with refractory low back pain to conservative measures. There are a number of interventional strategies that are targeted toward etiologies of back pain in conjunction with appropriate conservative management (Table 2.4):

Myofascial pain: trigger point injections

Facet-mediated arthropathy: facet joint injections, medial branch nerve blocks, medial branch nerve ablations⁴

Discogenic: disc biacuplasty, intradiscal electrothermography²³

Stenosis (central or neural foraminal): transforaminal epidural steroid injection, interlaminar steroid injection²⁴

Disc herniation: minimally invasive discectomy, open discectomy²⁵

Spondylolisthesis: decompression (laminectomy) and fusion²⁶

Summary

This patient presented with acute on chronic low back pain, along with radiating symptoms along a dermatome. Given the nature of traumatic event, this patient underwent imaging with x-ray and MRI of the lumbar spine, which revealed degenerative spondylosis, multilevel neuroforaminal stenosis, and left-sided L4–L5 posterolateral disc protrusion. By pairing this patient's history, physical, and imaging findings, he was diagnosed with acute L5 radiculopathy with underlying lumbar spondylosis. The patient was educated on exercise modifications and was referred to physical therapy. This patient underwent a 6-week physical therapy program that consisted of general low back program along with McKenzie extension-based exercises. His back pain is now manageable, and he is able to go to work. He continues to perform exercises at home as instructed during therapy.

Key Points

- Patients with low back pain must be carefully assessed. Red flags should always be addressed during every encounter.
- Imaging can help guide therapy, but should not substitute clinical judgment in making a diagnosis.
- Conservative measures are highly effective if exercises are modified to tailor a specific location of pathology or diagnosis.

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