

TABLE 22-3 CAUSES OF BACK OR NECK PAIN

Lumbar Disk Disease
Degenerative Spine Disease
Lumbar spinal stenosis without or with neurogenic claudication
Intervertebral foraminal or lateral recess narrowing
Disk-osteophyte complex
Facet or uncovertebral joint hypertrophy
Lateral disk protrusion
Spondylosis (osteoarthritis) and spondylolisthesis
Spine Infection
Vertebral osteomyelitis
Spinal epidural abscess
Septic disk (diskitis)
Meningitis
Lumbar arachnoiditis
Neoplasms—Metastatic, Hematologic, Primary Bone Tumors
Fractures
Trauma/falls, motor vehicle accidents
Atraumatic fractures: osteoporosis, neoplastic infiltration, osteomyelitis
Minor Trauma
Strain or sprain
Whiplash injury
Metabolic Spine Disease
Osteoporosis—hyperparathyroidism, immobility
Osteosclerosis (e.g., Paget's disease)
Congenital/Developmental
Spondylolysis
Kyphoscoliosis
Spina bifida occulta
Tethered spinal cord
Autoimmune Inflammatory Arthritis
Other Causes of Back Pain
Referred pain from visceral disease (e.g., abdominal aortic aneurysm)
Postural
Psychiatric, malingering, chronic pain syndromes

nerve roots within the spinal canal. Clinical manifestations of specific nerve root lesions are summarized in Table 22-2.

The differential diagnosis covers a variety of serious and treatable conditions, including epidural abscess, hematoma, fracture, or tumor.

Fever, constant pain uninfluenced by position, sphincter abnormalities, or signs of spinal cord disease suggest an etiology other than lumbar disk disease. Absence of ankle reflexes can be a normal finding in persons older than age 60 years or a sign of bilateral S1 radiculopathy. An absent deep tendon reflex or focal sensory loss may indicate injury to a nerve root, but other sites of injury along the nerve must also be considered. For example, an absent knee reflex may be due to a femoral neuropathy or an L4 nerve root injury. A loss of sensation over the foot and lateral lower calf may result from a peroneal or lateral sciatic neuropathy or an L5 nerve root injury. Focal muscle atrophy may reflect injury to the anterior horn cells of the spinal cord, a nerve root, peripheral nerve, or disuse.

A lumbar spine MRI scan or CT myelogram is necessary to establish the location and type of pathology. Spine MRIs yield exquisite views of intraspinal and adjacent soft tissue anatomy. Bony lesions of the lateral recess or intervertebral foramen are optimally visualized by CT myelography. The correlation of neuroradiologic findings to symptoms, particularly pain, is not simple. Contrast-enhancing tears in the annulus fibrosus or disk protrusions are widely accepted as common sources of back pain; however, studies have found that many asymptomatic adults have similar findings. Asymptomatic disk protrusions are also common and may enhance with contrast. Furthermore, in patients with known disk herniation treated either medically or surgically, persistence of the herniation 10 years later had no relationship to the clinical outcome. In summary, MRI findings of disk protrusion, tears in the annulus fibrosus, or hypertrophic facet joints are common incidental findings that, by themselves, should not dictate management decisions for patients with back pain.

The diagnosis of nerve root injury is most secure when the history, examination, results of imaging studies, and the EMG are concordant. The correlation between CT and EMG for localization of nerve root injury is between 65 and 73%. Up to one-third of asymptomatic adults have a lumbar disk protrusion detected by CT or MRI scans.

Management of lumbar disk disease is discussed below.

Cauda equina syndrome (CES) signifies an injury of multiple lumbosacral nerve roots within the spinal canal distal to the termination of the spinal cord at L1-L2. Low back pain, weakness and areflexia in the legs, saddle anesthesia, or loss of bladder function may occur. The problem must be distinguished from disorders of the lower spinal cord (conus medullaris syndrome), acute transverse myelitis (Chap. 456), and Guillain-Barré syndrome (Chap. 460). Combined involvement of the conus medullaris and cauda equina can occur. CES is commonly due to a ruptured lumbosacral intervertebral disk, lumbosacral spine fracture, hematoma within the spinal canal (e.g., following lumbar puncture in patients with coagulopathy), compressive tumor, or other mass lesion. Treatment options include surgical decompression, sometimes urgently in an attempt to restore or preserve motor or sphincter function, or radiotherapy for metastatic tumors (Chap. 118).

DEGENERATIVE CONDITIONS

Lumbar spinal stenosis (LSS) describes a narrowed lumbar spinal canal and is frequently asymptomatic. Typical is *neurogenic claudication*, consisting of back and buttock or leg pain induced by walking or standing and relieved by sitting. Symptoms in the legs are usually bilateral. Unlike vascular claudication, symptoms are often provoked by standing without walking. Unlike lumbar disk disease, symptoms are usually relieved by sitting. Patients with neurogenic claudication can often walk much farther when leaning over a shopping cart and can pedal a stationary bike with ease while sitting. These flexed positions increase the anteroposterior spinal canal diameter and reduce intraspinal venous hypertension, resulting in pain relief. Focal weakness, sensory loss, or reflex changes may occur when spinal stenosis is associated with neural foraminal narrowing and radiculopathy. Severe neurologic deficits, including paralysis and urinary incontinence, occur only rarely.

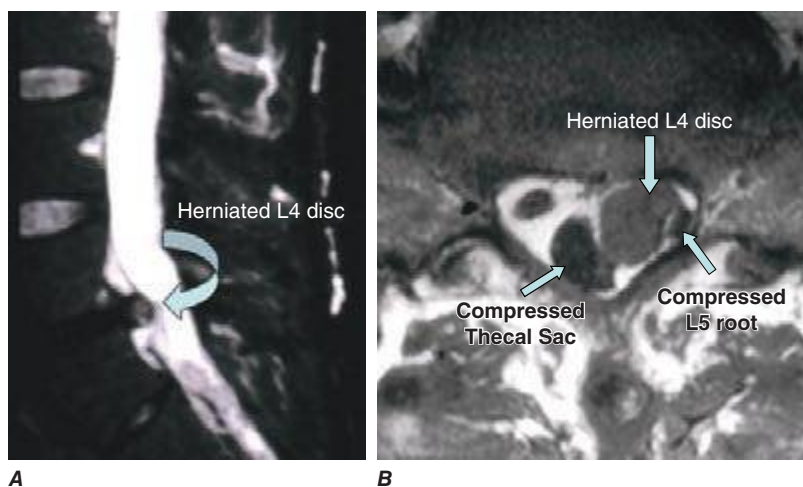


FIGURE 22-4 Left L5 radiculopathy. **A.** Sagittal T2-weighted image on the left reveals disk herniation at the L4-L5 level. **B.** Axial T1-weighted image shows paracentral disk herniation with displacement of the thecal sac medially and the left L5 nerve root posteriorly in the left lateral recess.