Lumbosacral Radiculopathy

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Lumbosacral radiculopathy is one of the most common disorders evaluated by neurologists and is a leading referral diagnosis for the performance of electromyography. Degenerative spondyloarthropathies are the principal underlying cause of these syndromes and are increasingly common with age. The clinical presentation and initial management of lumbosacral radiculopathies of various etiologies are discussed.

Epidemiology

Although precise epidemiologic data are difficult to establish, the prevalence of lumbosacral radiculopathy is approximately 3% to 5%, distributed equally in men and women [1,2]. Men are most likely to develop symptoms in their 40s, whereas women are affected most commonly between ages 50 and 60 [1].

Anatomy

Detailed spinal anatomy is discussed elsewhere in this issue by Devereaux; however, clinically relevant points are reviewed. There are five moveable lumbar vertebrae, five fused sacral vertebrae, and four fused coccygeal vertebrae [3] with intervertebral disks sandwiched between each of the lumbar vertebrae and between the fifth lumbar vertebra and sacrum. The moveable vertebrae are connected by paired facet joints between the articular processes of the pedicles and by the anterior and posterior longitudinal ligaments. The intervertebral foramina are formed by notches in the articular processes.
of adjacent pedicles of two vertebrae; the disk is anterior and medial to the foramen.

In adults, the spinal cord terminates at the L1-2 intervertebral level as the conus medullaris. The nerve roots descend from this point through the spinal canal as the cauda equina and exit eventually through the neural foramina at their respective intervertebral levels. Eleven pairs (five lumbar, five sacral, and one coccygeal) of spinal nerves emerge from the spinal cord in the lumbosacral region. Ventral roots, containing primarily motor fibers, arise from rootlets, which extend from the ventral gray matter of the spinal cord. Dorsal rootlets, carrying sensory information, extend centrally from the dorsal root ganglia that lie outside the spinal cord, within the neural foramen. Just distal to the intervertebral foramen, the dorsal and ventral roots unite to form a mixed spinal nerve, which divides into dorsal and ventral primary rami. The dorsal rami supply the paraspinal muscles and skin overlying the paraspinal region, whereas the ventral rami give rise to the lumbosacral plexus and, eventually, the individual nerves supplying the lower limbs and sacral region. The muscles supplied by a single spinal segment constitute a myotome; the skin region supplied by a single spinal segment is a dermatome.

**History and physical examination**

Performance of a careful history and physical examination is the initial and integral step in the diagnosis and management of lumbosacral radiculopathy. Lesion localization depends on demonstration of a segmental myotomal or dermatomal distribution of abnormalities; a working knowledge of the relevant anatomy is essential. Sciatica, the classic presenting symptom of lumbosacral radiculopathy, is characterized by pain in the back radiating into the leg. Patients variably describe this pain as sharp, dull, aching, burning, or throbbing. Pain related to disk herniation is exacerbated by bending forward, sitting, coughing, or straining and relieved by lying down or sometimes walking [4]. Conversely, pain related to lumbar spinal stenosis characteristically is worsened by walking and improved by forward bending. Pain that is exacerbated by or fails to respond to the recumbent position is a distinctive feature of radiculopathy produced by inflammatory or neoplastic lesions and other nonmechanical causes of back pain. The distribution of pain radiation along a dermatome may be helpful in localizing the level of involvement; when present, the dermatomal distribution of paresthesias is more specific [4].

In the majority of cases, lumbosacral radiculopathy is caused by compression of nerve roots from pathology in the intervertebral disk or associated structures. The differential diagnosis of lesions producing lumbosacral radiculopathy, however, is broad and includes neoplastic, infectious, and inflammatory disorders (Box 1). Important risk factors for serious underlying disease that should be sought in the history include age greater than 50,
### Box 1. Causes of lumbosacral radiculopathy

**Degenerative**
- Intervertebral disk herniation
- Degenerative lumbar spondylosis

**Neoplastic**
- Primary tumors
- Ependymoma
- Schwannoma
- Neurofibroma
- Lymphoma
- Lipoma
- Dermoid
- Epidermoid
- Hemangioblastoma
- Paraganglioma
- Ganglioneuroma
- Osteoma
- Plasmacytoma
- Metastatic tumors
- Leptomeningeal metastasis

**Infectious**
- Herpes zoster (HZ)
- Spinal epidural abscess (SEA)
- HIV/AIDS-related polyradiculopathy
- Lyme disease

**Inflammatory/metabolic**
- Diabetic amyotrophy
- Ankylosing spondylitis
- Paget’s disease
- Arachnoiditis
- Sarcoidosis

**Developmental**
- Tethered cord syndrome
- Dural ectasia

**Other**
- Lumbar spinal cysts
- Hemorrhage
previous history of cancer, unexplained weight loss, and failure to improve after 1 month of conservative therapy [5].

Aside from assessment of potential serious disease, the history is geared toward establishing the involvement of nerve roots and their anatomic level. Similarly, the aim of the physical examination is to elucidate motor, sensory, or reflex abnormalities in a radicular distribution relevant to the suspected clinical level. Sciatic nerve tension signs may provide supporting evidence of L5-S1 radiculopathy; however, they may be present with lesions of the lumbosacral plexus, sciatic nerve, or hip joint and in mechanical lower back pain [6–8]. With the patient supine and one hand on the iliac crest of the affected side, an examiner passively elevates the heel slowly while keeping the knee straight; the angle at which pain or paresthesias are produced and the distribution are noted. Dorsiflexion of the foot may increase symptoms. The straight leg raise test is positive if symptoms are produced between 30° and 70° [6]. In a similar fashion, the femoral nerve stretch test produces tension on the L3, L4 nerve roots. With a patient lying on the asymptomatic side and the lower limb flexed at the hip and knee, the symptomatic knee is extended passively at the hip [9]. Pain radiating into the anterior thigh with this maneuver suggests L3 or L4 radiculopathy.

Clinical presentation of monoradiculopathies

L1 radiculopathy

Disk herniation at this level is rare; consequently, L1 radiculopathy is extremely uncommon. The typical presentation is one of pain, paresthesias, and sensory loss in the inguinal region, without significant weakness. Infrequently, subtle involvement of hip flexion is noted. Muscle stretch reflexes (MSRs) are normal. Differential diagnostic considerations include ilioinguinal and genitofemoral neuropathies. Physical examination may help distinguish between these conditions, but imaging of the lumbosacral spine or pelvis often is required (Table 1).

L2 radiculopathy

Also rarely caused by disk herniation, L2 radiculopathy produces pain, paresthesias, and sensory loss in the anterolateral thigh. Weakness of hip flexion may occur; MSRs are normal. Lateral femoral cutaneous neuropathy (meralgia paresthetica) may mimic L2 radiculopathy; the presence of hip flexor weakness suggests radiculopathy rather than meralgia. Femoral neuropathy and upper lumbar plexopathy may present similarly.

L3 radiculopathy

Although more common than with higher lumbar roots, disk herniation is an uncommon cause of L3 radiculopathy. Pain and paresthesias involve
the medial thigh and knee, with weakness of hip flexors, hip adductors, and knee extensors; the knee jerk may be depressed or absent. L3 radiculopathy may be confused with femoral neuropathy, obturator neuropathy, diabetic amyotrophy, or upper lumbar plexopathy. Combined weakness of hip adduction and hip flexion differentiates L3 radiculopathy from femoral and obturator mononeuropathies.

**L4 radiculopathy**

Unlike the higher lumbar levels, L4 radiculopathy is produced most commonly by disk herniation. Spinal stenosis frequently involves this nerve root in conjunction with roots at adjacent spinal levels. Sensory symptoms involve the medial lower leg in the distribution of the saphenous nerve. As with L3 radiculopathy, knee extension and hip adduction may be weak; additionally, foot dorsiflexion weakness uncommonly may be observed. When present, ankle dorsiflexion weakness generally is less prominent than in L5 radiculopathy. The knee jerk may be depressed or absent. Lumbosacral plexopathy is the main differential diagnostic consideration; saphenous neuropathy also is a possibility in pure sensory syndromes.

<table>
<thead>
<tr>
<th>Root level</th>
<th>Pain</th>
<th>Sensory loss (paresthesias)</th>
<th>Motor abnormalities or weakness</th>
<th>Muscle stretch reflex abnormalities</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1 Inguinal region</td>
<td>Inguinal region</td>
<td>None</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>L2 Groin, anterior thigh</td>
<td>Anterolateral thigh</td>
<td>None</td>
<td>None</td>
<td></td>
</tr>
<tr>
<td>L3 Anterior thigh to knee, anterior leg</td>
<td>Medial thigh and knee</td>
<td>Quadriceps, iliopsoas, hip adductors</td>
<td>Knee jerk</td>
<td></td>
</tr>
<tr>
<td>L4 Medial foreleg</td>
<td>Medial lower leg</td>
<td>Tibialis anterior, quadriceps, hip adductors</td>
<td>Knee jerk</td>
<td></td>
</tr>
<tr>
<td>L5 Lateral thigh and lower leg, dorsum foot</td>
<td>Lateral lower leg, dorsum foot, great toe</td>
<td>Toe extensors and flexors, ankle dorsiflexor, everter and inverter, hip abductors</td>
<td>Internal hamstrings</td>
<td></td>
</tr>
<tr>
<td>S1 Posterior thigh, calf, heel</td>
<td>Sole, lateral foot and ankle, lateral two toes</td>
<td>Gastrocnemius, hamstrings, gluteus maximus, toe flexors</td>
<td>Ankle jerk</td>
<td></td>
</tr>
<tr>
<td>S2-4 Medial buttocks</td>
<td>Medial buttocks, perineal, perianal region</td>
<td>None unless S1-2 involved</td>
<td>Bulbocavernousus, anal wink. Ankle jerk if S1 involved</td>
<td></td>
</tr>
</tbody>
</table>

Table 1: Neurologic examination findings in monoradiculopathies
**L5 radiculopathy**

The most common cause of L5 radiculopathy is intervertebral disk herniation. Foot drop is the salient clinical feature, with associated sensory symptoms involving the anterolateral leg and dorsum of the foot. In addition to weakness of ankle dorsiflexion, L5 radiculopathy commonly produces weakness of toe extension and flexion, foot inversion and eversion, and hip abduction. Common peroneal neuropathy closely mimics and must be distinguished from L5 radiculopathy. Physical examination is helpful in localization as weakness of foot eversion (mediated by the L5/peroneal-innervated peroneus muscles) in conjunction with inversion (mediated by the L5/tibial-innervated tibialis posterior) places the lesion proximal to the peroneal nerve. Lumbosacral plexopathy and sciatic neuropathy are important differential diagnostic considerations. The involvement of hip abductors (gluteus medius and minimus) indicates a lesion proximal to the sciatic nerve but does not differentiate L5 radiculopathy from lumbosacral plexopathy. Although there is no classic MSR abnormality associated with L5 radiculopathy, an asymmetric internal hamstring reflex can support its presence.

**S1 radiculopathy**

S1 radiculopathy also is caused commonly by intervertebral disk herniation, with associated weakness of foot plantar flexion, knee flexion, and hip extension. Subtle weakness of foot plantar flexion may be demonstrated by having patients stand or walk on their toes. Sensory symptoms typically involve the lateral foot and sole. The ankle jerk is depressed or absent. Sciatic neuropathy and lower lumbosacral plexopathy may mimic S1 radiculopathy. Both of these conditions, however, also are expected to affect L5-innervated muscles.

**Lumbosacral polyradiculopathy and cauda equina syndromes**

Multiple, contiguous nerve roots may be involved by compressive lesions affecting several individual nerve roots, either in the vertebral canal or the neural foramina; less frequently, infiltrating or inflammatory processes spreading along the meninges produce similar clinical syndromes. Lesions involving the cauda equina should be considered when nerve roots at more than two neighboring levels are involved, developing acutely or gradually. Acute cauda equina syndrome most often is the result of compression of the lower lumbar and sacral nerve roots by a large, central disk herniation, usually at L4-5. Sacral nerve roots, which lie medially in the cauda equina, often are affected disproportionately, leading to sacral polyradiculopathy with prominent bowel and bladder dysfunction and characteristic saddle anesthesia. Lumbar nerve roots also may be involved, resulting in leg weakness that can progress to paraplegia, depending on the extent of nerve root...
compromise. A true neurologic emergency, prompt recognition of cauda equina syndrome is necessary to preserve sphincter function and ambulation.

A more insidious manifestation of cauda equina dysfunction results from central spinal stenosis, creating a clinical syndrome of intermittent neurogenic claudication (discussed in Chad’s article, elsewhere in this issue). The neurologic and electrodiagnostic examinations in these cases may demonstrate patchy lumbosacral polyradiculopathy but often are normal.

Differential diagnosis

The majority of lesions causing lumbosacral radiculopathy are compressive in nature and result from disk herniation or spondylosis with entrapment of nerve roots. It is important, however, to recognize a variety of other lesions that may produce lumbosacral radiculopathy, including several neoplastic, infectious, and inflammatory disorders (see Box 1).

Degenerative spine disorders

Acute disk herniation

Intervertebral disk herniation is the most common cause of lumbosacral radiculopathy in patients under age 50 [7]. At birth, the boundary between the gelatinous nucleus pulposus and the tough, surrounding annulus pulposus is distinct; with increasing age, the concentration of collagen in the disk increases and water content decreases [7]. As a result, clefts and fissures form in the disk and disruption of annular fibers occurs, predisposing to herniation of the nucleus pulposus [7]. Acute disk herniation produces symptoms by direct compression of the nerve roots and by inflammatory and ischemic mechanisms involving the roots and dorsal root ganglia [8]. The intervertebral disks affected most frequently are L4-5 and L5-S1, leading to L5 or S1 radiculopathies. Pain characteristically is of abrupt onset and intense, often precipitated by bending over or lifting. Patients may report of sciatica without back pain. Aggravation of pain with movement, particularly forward or lateral bending, or with Valsalva’s maneuver is typical; usually, pain is relieved with recumbency. In addition to pain, patients frequently report paresthesias in the involved dermatome. Cauda equina syndrome with prominent bowel and bladder involvement may be the presenting syndrome with large, central disk herniations.

Often, a diagnosis of acute disk herniation may be made on clinical grounds. MRI and EMG, however, may be helpful in establishing the diagnosis and distinguishing disk pathology from other causes of lumbosacral radiculopathy. Although MRI is sensitive, lumbar disk herniations are identified in 30% to 40% of asymptomatic subjects by MRI and in an equivalent number at autopsy with CT and with myelography [7]. Initial treatment is aimed at pain control and identification of patients who require urgent surgical consideration to prevent permanent neurologic deficits.
Degenerative spondylosis

After age 50, acute disk herniation is a less common cause of lumbosacral radiculopathy, and chronic lesions related to degenerative spinal arthropathy predominate [7]. With advancing age, intervertebral disks desiccate and flatten, transferring increasing axial load to the facet joints, with resultant facet joint hypertrophy, osteophyte formation, and thickening of the ligamentum flavum [7,10]. These changes contribute to narrowing of the central spinal canal, lateral recesses, and neural foramina. L4-5 and L5-S1 levels in particular are affected [7]. Chronic radiculopathy may result from entrapment of nerve roots in the lateral recess, intervertebral foramen, or central canal, involving single or multiple nerve roots. Clinical syndromes of radicular pain involving buttock, hip, or posterior thigh and intermittent neurogenic claudication are more common than back pain [10].

MRI frequently is performed in the evaluation of these lesions, although bony pathology is demonstrated better by CT. Because degenerative changes are commonplace in older patients, electrodiagnostic studies frequently are necessary to establish the relevance of neuroimaging abnormalities. Initial management involves pain control with analgesic medications and physical therapy to strengthen supporting musculature and improve postural mechanics. Surgical decompression is considered for progressive or recalcitrant symptoms or worsening neurologic deficits.

Neoplasms

Radiculopathy may result from tumor in various locations within the spinal canal; usually, these lesions are extramedullary. Primary tumors tend to be intradural, whereas metastatic lesions are extradural. Furthermore, primary lesions tend to be solitary (neurofibromatosis type 1 being a notable exception), whereas metastatic lesions frequently are multiple.

Primary tumors

Primary nerve root tumors are a rare cause of lumbosacral radiculopathy. Most primary spinal tumors are benign and slow growing, and their clinical manifestations may be difficult to distinguish from more common causes of radiculopathy, such as disk herniation [11]. Both are characterized by back pain; however, the nature of pain related to tumor is distinctive, as it becomes increasingly severe over time and is worse when lying down, often interfering with sleep. Primary tumors producing lumbosacral radiculopathy most frequently are neurofibromas (often associated with neurofibromatosis type 1) and ependymomas; less common are schwannomas (in neurofibromatosis type 2), meningiomas, lipomas and dermoids, and lipomas [11]. Ependymomas and neurofibromas typically affect the filum terminale, producing a cauda equina syndrome [11]. Diagnosis of primary tumors is established by MRI, and their definitive treatment is surgical.
Epidural and vertebral metastases

Although metastatic tumor is the most common type of neoplasm involving the spinal canal, it is rare in the general population. In one series of 1975 patients who had low back pain, 13 (0.7%) had a malignancy to account for this problem [5]. These lesions chiefly are seen in patients who have a known malignancy and, in a small percentage, are the presenting feature. Approximately 30% of epidural metastases occur in the lumbar spine, and radicular pain is an initial symptom in approximately half [12,13]. Metastases typically invade the spinal column and extend from there into the epidural space [14]. Metastases seed the vertebrae by way of Batson’s venous plexus (which drains the vertebrae and anastomoses with veins draining the viscera). Less commonly, paravertebral lesions spread directly to nerve roots through the intervertebral neural foramina [14].

The three most common cancers involving the lumbosacral spine are breast, lung, and prostate cancer, each accounting for approximately 10% to 20% of cases [14,15]. Virtually all cancers, however, may produce metastatic spinal cord compression, and in 20%, spinal cord compression is the initial feature [12]. Tumors of the pelvic region, including colon and prostate, preferably metastasize to the lumbosacral region. Back pain is the most common initial complaint and, as with primary tumors, is unremitting and characteristically worse with recumbency; radicular pain is more variable. Percussion tenderness at the site of the lesion is noteworthy. Bowel and bladder disturbances occur in a minority of patients at onset but tend to be more common as disease progresses [13,14].

Contrast-enhanced MRI is the procedure of choice in the evaluation of suspected spinal metastases [16]; as lesions frequently are multiple, scanning of the entire spine is indicated [17]. Corticosteroids and external beam radiation are the mainstays of treatment [15]. The neurologic prognosis of patients who have radiculopathy as the sole symptom of metastatic disease is good; most patients likely maintain ambulation after treatment with radiotherapy [15,18]. Prognosis is correlated closely with the degree of neurologic dysfunction at diagnosis, so early recognition is crucial.

Leptomeningeal metastases/meningeal carcinomatosis

Cancer cells may infiltrate the leptomeninges and subarachnoid space diffusely, leading to a syndrome reflecting involvement of cranial nerves, spinal nerve roots, and brain. Manifestations include radiculopathy, cranial polyneuropathy, headache, memory loss, seizures, and gait disturbances [15]. Radicular discomfort is the most common presenting symptom, usually involving lumbosacral levels resulting from involvement of the cauda equina [19]. Although all cancers have the potential to produce this condition, the most likely primary tumors to do so are leukemia, lymphoma, and breast carcinoma [15,19,20]. Other tumors that may produce leptomeningeal metastasis include melanoma, lung cancer, gastrointestinal cancers, and sarcoma [15,20,21]. Initial cerebrospinal fluid (CSF) examination reveals
a mildly increased cell count in approximately half, elevated protein in a large majority, and low glucose in approximately 25% [15,20]. Positive cytologic examination is seen in half of initial lumbar punctures and 90% after three lumbar punctures [15]. MRI may reveal contrast enhancement of the meninges in a diffuse or nodular pattern [15].

Infections

Herpes zoster

Primary infection with varicella-zoster virus produces chickenpox, usually in children, after which it lies dormant in dorsal root ganglia and may be reactivated decades later, producing acute Herpes zoster (HZ) or shingles [22,23]. A common disorder, it is prevalent especially in immunocompromised and elderly populations. HZ usually affects a single dermatome and is accompanied by intense neuralgic pain reflecting the level of infection; pain often precedes the classic vesicular eruption. Ophthalmic and thoracic dermatomes are affected most commonly, whereas lumbosacral zoster accounts for approximately 20% of cases [23,24]. In the presence of rash, the diagnosis of HZ is obvious, but a minority of patients may present with zoster sine herpete, dermatomally distributed pain without rash [25]. Approximately 5% of patients may develop a local neuritis of the spinal nerve, which subsequently affects the motor axons, producing a segmental zoster paresis [26]. The pain of acute HZ, frequently overwhelming at the outset, gradually subsides as the vesicles crust over in most patients. Approximately 10% to 15% suffer from chronic pain, or postherpetic neuralgia (PHN), however, despite treatment with antiviral agents [27]. Complete resolution of motor deficits occurs in 50% to 70% of those who have segmental zoster paresis [24].

Tricyclic antidepressants, gabapentin, pregabalin, opioids, and topical lidocaine patches are effective in the treatment of acute herpetic neuralgia (grade A evidence) [28]. Amitriptyline usually is effective, in a dosage of 75 to 100 mg per day, in the treatment of pain related to PHN (grade A) [29]. Gabapentin, in doses between 1800 and 3600 per day, also is effective in relieving the symptoms of PHN (grade A) [30] and may be preferred to amitriptyline because of a lower incidence of side effects. If begun within 72 hours of development of the rash, famciclovir, valacyclovir, or acyclovir reduces the pain of acute HZ but may not be effective in the prevention of PHN (grade A) [23]. Of these antiviral agents, valacyclovir (1000 mg 3 times a day for 7 days) is preferred for its more rapid resolution of neuralgia symptoms, shorter duration of PHN, and smaller pill burden [31]. Corticosteroids by themselves do not alter the course of PHN but, in combination with an antiviral agent, may improve pain [23].

Spinal epidural abscess

Spinal epidural abscess (SEA) most commonly involves the thoracic and lumbar spine. Risk factors for development of SEA include diabetes
mellitus, history of intravenous drug abuse, spinal surgery, spinal or paraspinal injection, epidural catheter placement, and immunocompromised status [32,33]. Severe back pain, often with a radicular component, is the presenting complaint [34]. Fever is a common, but not universal, sign. Leukocytosis and elevation of the erythrocyte sedimentation rate are typical and in the presence of fever and back pain, the diagnosis should be straightforward. Only 20% of patients, however, have the classical clinical triad of fever, back pain, and neurologic deficits, so a high index of suspicion should be maintained [35]. The diagnostic test of choice is contrast-enhanced MRI.

Treatment of SEA must be initiated urgently with surgical débridement generally the treatment of first choice. There is increasing evidence, however, that management with 6 to 8 weeks of intravenous antibiotics with or without oral antibiotics may result in similar outcomes (grade B) [36]. Antibiotic treatment should be directed to treat the most common infecting organisms, which include *Staphylococcus aureus*, other gram-positive cocci, gram-negative rods, and anaerobes [33]. Close monitoring is necessary, and urgent surgical decompression must be considered strongly if neurologic compromise develops.

**Polyradiculopathy in HIV and AIDS**

Polyradiculopathy secondary to HIV infection is uncommon, accounting for only 2% of HIV-related neurologic consultations [37]. The majority of patients have an AIDS-defining illness before the development of radiculopathy, and the CD4 count is less than 100 cells per μL in almost all patients [38,39]. Polyradiculopathy in AIDS tends to involve the lumbosacral nerve roots, producing a rapidly progressive cauda equina syndrome with severe low back pain [37–40]. Cytomegalovirus accounts for most HIV-related radiculopathy. Other causes of HIV-radiculopathy include herpes simplex virus, lymphomatous meningitis, mycobacteria, *Cryptococci*, and treponemal infection [38,39].

Examination of CSF demonstrates pleocytosis, with polymorphonuclear predominance and, in some patients, decreased glucose [38–40]. A positive CSF polymerase chain reaction for cytomegalovirus also is supportive of the diagnosis. Recommended treatment includes intravenous ganciclovir, foscarnet, or both for 3 to 6 weeks (grade B) [41]. Development of polyradiculopathy in AIDS generally portends a poor prognosis, with minimal functional recovery after treatment and a median survival time of 2.7 months [39].

**Lyme radiculopathy**

Lyme disease is transmitted by the bite of *Ixodes* ticks infected with the spirochete *Borrelia burgdorferi* [42]. The classic rash of erythema migrans develops in 50% to 90% of patients [43]. In addition to a flu-like illness, hematogenous dissemination may affect the heart, joints, and nervous system [42,43]. Acute Lyme radiculoneuropathy is seen most commonly in the first
2 months of infection and mimics structural disk herniation; a minority of cases affects the lumbosacral nerve roots [43]. Radicular signs and symptoms usually occur in conjunction with cranial neuropathies and lymphocytic meningitis.

The current recommendation for serologic testing is to use a two-step approach, in which a positive-screening ELISA is confirmed by a Western blot [42]. In addition to serologic analysis, lumbar puncture may be helpful in establishing a diagnosis, with CSF analysis typically showing a mildly increased protein and a lymphocytic pleocytosis of up to a few hundred white cells per mm³ [42,43]. Recommended treatment of acute Lyme radiculopathy is ceftriaxone, 2 g daily for 14 to 28 days (grade B) [44]. Intravenous penicillin G, 18 to 24 million units daily divided every 4 hours, and cefotaxime, 2 g IV every 8 hours, are alternatives (grade B) [44]. In patients intolerant of penicillin and cephalosporins, doxycycline, 100 mg bid per day in two divided doses, is preferred (grade B) [44]. Although the prognosis of acute Lyme radiculopathy generally is excellent, axonal regeneration and resolution of neurologic symptoms may require several months [43].

Chronic Lyme radiculoneuropathy is differentiated from acute Lyme radiculopathy by its development, on average, 8 months after the symptoms of the acute illness, a milder clinical course, and the absence of CSF pleocytosis [45]. The condition can develop despite successful treatment of the acute illness [45]. Treatment regimens are similar to those used for acute Lyme radiculopathy. A small number of nonrandomized patients have been followed up; 6 months after treatment with intravenous ceftriaxone, improvement was reported in 9 of these 12 patients [45]. This improvement, however, usually was incomplete and noted weeks to months after completion of therapy, not during the course of treatment [45].

**Diabetes (diabetic amyotrophy)**

Diabetes may cause a syndrome of severe lower extremity pain and weakness, commonly referred to as diabetic amyotrophy. This syndrome usually involves multiple lumbosacral nerve roots but rarely presents as a monoradiculopathy [46]. Patients typically have well controlled type 2 diabetes and are middle aged or older [47]. In some patients, the neurologic impairment heralds the onset of diabetes [47]. Sudden onset, unilateral lower extremity pain variably involves the groin, anterior thigh, and lower leg; weakness follows shortly. Proximal muscles, in particular quadriceps, tend to be affected first and most conspicuously, but the majority of patients also develop distal and bilateral symptoms [48]. Weight loss is a frequent accompanying symptom [47]. The precise pathophysiology of diabetic radiculopathy is controversial, with nerve ischemia, inflammation, and metabolic causes implicated [47–50].

EMG is helpful in diagnosing diabetic amyotrophy. There is evidence of subacute polyradiculopathy with prominent denervation changes involving
limbs and multiple, bilateral paraspinal regions. Underlying axonal polyneuropathy may also be present. CSF protein is elevated without pleocytosis.

Diabetic radiculopathy is a monophasic illness that improves with time [46,47]. Improvement, however, often is incomplete and prolonged, with motor symptoms slower to resolve than sensory symptoms [47]. Pain control in the early stages can be challenging. Standard agents for neuropathic pain, such as anticonvulsants or tricyclic antidepressants, are beneficial, but narcotic analgesics also may be needed temporarily [47]. Physical therapy and orthoses should be provided as indicated [47]. Up to 20% of patients may suffer a recurrence on the same side [47,51].

**Spinal cysts**

Cystic lesions in the sacral spine are common, with an incidence ranging from 4.6% to 17% on imaging studies [52,53]. Most sacral meningeal cysts are dural diverticula (Tarlov cysts) produced by fluctuations in CSF pressure [52]. There is little to differentiate the presentation of meningeal sacral cysts from other causes of lumbosacral radiculopathy [52]. Radicular pain often is relieved or disappears when patients are recumbent and is aggravated by Valsalva’s maneuver [52]. Because they are common and not necessarily the cause of symptoms, establishing a cyst as the cause of lumbosacral radiculopathy involves eliminating other causes first. MRI is the diagnostic procedure of choice for demonstrating lumbosacral cysts; however, clinical relevance of the imaging findings must still be established. Although analgesic medications may reduce pain, relief of symptoms with fluoroscopic-guided aspiration and surgical treatment is definitive [52].

**Spinal hematomas**

Hematomas are uncommon causes of lumbosacral radiculopathy. Epidural and subdural spinal hematomas occur most frequently in patients who have coagulopathies, who are taking anticoagulants, or who recently have undergone epidural injections or instrumentation of the lumbosacral spine [54–56]. Spinal subarachnoid hemorrhage is uncommon. Unlike its intracranial counterpart, spinal subarachnoid hemorrhage is caused most commonly by arteriovenous malformation rupture rather than aneurysmal rupture [57]. Hemorrhage into synovial cysts or the ligamentum flavum also may produce hematomas and lumbosacral radiculopathy [58,59].

**Other uncommon causes of radiculopathy**

Sarcoidosis can affect any level of the neuraxis; radiculopathy is an uncommon presentation. Cauda equina syndrome and lumbosacral polyradiculopathy are described as manifestations of sarcoid [60]. Arachnoiditis also may produce lumbosacral radiculopathy. Classically caused by a reaction to intrathecal oil-based contrast dye for myelography,
other causes of arachnoiditis include neurocysticercosis and other infections, blood in the intrathecal space, surgical interventions in the spine, intrathecal corticosteroids, and trauma [61].

Tethered cord syndrome is a developmental malformation characterized by an abnormally low-lying conus medullaris tethered to an intradural abnormality [62]. Uncommonly, this syndrome presents in adulthood, most often as pain centered around the anorectal or inguinal regions, but sometimes diffusely in the legs or in a radicular distribution [63–65]. Approximately 60% to 70% of patients report an inciting traumatic event leading to presentation [65]. A mixed cauda equina and conus medullaris syndrome is seen, without other signs of spinal dysraphism, such as sacral dimples or hair tufts [63–65]. MRI has allowed earlier detection of the syndrome [63,64], with termination of the conus medullaris inferior to the inferior aspect of the L2 vertebral body being diagnostic [63].

**Approach to initial diagnosis and management**

An algorithm for the initial diagnosis and management of lumbosacral radiculopathy is shown in Fig. 1. First, whether or not patients have a disease process that could result in irreversible neurologic dysfunction must be determined. Indications for immediate neuroimaging and surgical evaluation include a cauda equina syndrome, rapidly progressive neurologic deficits, and risk factors for metastatic cancer or epidural abscess.

Provided that none of these indications for urgent evaluation is present, a trial of conservative therapy may be attempted for 4 to 6 weeks. Specific details of conservative and surgical management are discussed by Benzel and colleagues elsewhere in this issue of the Clinics; however, there is little difference among the outcomes of patients treated with bed rest, physical therapy, or continuation of normal activities of daily living, so treatment should be tailored to provide maximum comfort [66,67]. Analgesic medications, including nonsteroidal anti-inflammatory drugs, nonopioid analgesics (eg, tramadol), and, in some cases, narcotic analgesics should be used as indicated.

If 4 to 6 weeks of conservative therapy fails to control painful symptoms or if neurologic deficits progress, further diagnostic studies, including EMG and MRI, are warranted. Needle EMG is the preferred electrodiagnostic technique in the evaluation of radiculopathy and is performed in conjunction with nerve conduction studies to exclude alternative diagnosis, such as neuropathy or plexopathy [68]. EMG is advantageous because it assesses the physiologic integrity of the nerve roots directly and can diagnose compressive and noncompressive radiculopathies. It also provides a measure of severity of radiculopathic disease. MRI is the preferred study to view the structure of the lumbosacral spine and nerve roots. Because of the high prevalence of disk protrusions and degenerative spinal stenosis in older patients, history and physical examination remain paramount in determining the
Fig. 1. Approach to initial diagnosis and treatment of lumbosacral radiculopathy.
relevance of imaging findings [69,70]. EMG also can be used to help provide confirmation of clinically suspected nerve root involvement.

In some cases, further laboratory testing is appropriate based on the history, physical examination, EMG, or imaging findings. Lumbar puncture may be indicated to investigate inflammatory or infectious causes. The direction of the evaluation is guided by patient presentation and subsequent clinical course. As discussed previously, a basic understanding of the diverse disorders producing lumbosacral radiculopathy and those who are at risk for them is the foundation for accurate diagnosis and treatment.

Summary

Lumbosacral radiculopathy is a common neurologic syndrome that is an important source of disability. Although the most common causes are disk herniation and chronic spinal arthropathy, physicians should be mindful of other causes, including neoplasm and infection. Initial evaluation should focus on localization of lumbosacral radiculopathy and exclusion of disorders that may produce irreversible neurologic compromise. Treatment is aimed at providing pain relief and preventing neurologic deficits in addition to appropriate directed therapy based on the underlying cause.

References


