Diagnostic pitfalls in spine surgery: masqueraders of surgical spine disease

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Disorders of the spine are common in clinical medicine, and spine surgery is being performed with increasing frequency in the US. Although many patients with an established diagnosis of a true surgically treatable lesion are referred to a neurosurgeon, the evaluation of patients with spinal disorders can be complex and fraught with diagnostic pitfalls. While “common conditions are common,” astute clinical acumen and vigilance are necessary to identify lesions that masquerade as surgically treatable spine disease that can lead to erroneous diagnosis and treatment. In this review, the authors discuss musculoskeletal, peripheral nerve, metabolic, infectious, inflammatory, and vascular conditions that mimic the syndromes produced by surgical lesions. It is possible that nonsurgical and surgical conditions coexist at times, complicating treatment plans and natural histories. Awareness of these diagnoses can help reduce diagnostic error, thereby avoiding the morbidity and expense associated with an unnecessary operation. (DOI: 10.3171/2011.7.FOCUS11114)

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Arguably one of the greatest predictors of surgical outcomes in spinal disease is proper patient selection. With the widespread availability of spinal MR imaging and the prevalence of back pain, the presence of disc bulges or protrusions in people with low-back pain may frequently be coincidental. The anatomical continuum that extends from the “normal” asymptomatic spine, to the spine experiencing changes from physiological wear and tear, and to the spine exhibiting degenerative changes responsible for symptomatic disease is a blurred one, creating an abiding debate of what constitutes a pathological, surgically treatable lesion. Lacking concordance among a patient’s history, neurological examination, and neuroimaging studies, spine surgeons can be led astray by incidental radiological findings and can potentially attribute a patient’s symptoms to an unrelated entity. Furthermore, surgery may not necessarily alter the natural history of the disease processes, even after addressing an implicated lesion(s). In addition to all of these difficult aspects of surgical decision making, one must consider the lesions that masquerade as surgically treatable spine disease. Entities less common than a herniated lumbar disc or spinal stenosis do in fact present to spine surgeons. These entities fall outside the spectrum of “indeterminate to definite” surgical utility in the category of “unadvisable.” A patient’s symptom complexes can be further complicated by the so-called double-diagnostic entities, such as the combination of CTS and cervical radiculopathy—the so-called double-crush syndrome. Because we frequently see patients with these symptoms, accurate diagnosis is necessary to render an excellent surgical evaluation. Conversely, true spinal disease may present to practitioners of different specialties due to the common aspects of presentation.

There are several general categories of diagnostic pitfalls commonly encountered in spinal surgery. The first category involves erroneously attributing a patient’s symptoms to an unrelated radiological finding, resulting in an unnecessary operation that fails to address the true cause of the patient’s distress; the second category entails the failure to diagnose an unusual presentation of a common spinal disorder; and the third category is the misdiagnosis of something as a surgically treatable spinal disorder that is instead a medical or neurological condition mimicking myelopathy or radiculopathy. In the present review, we provide an overview of some of the common conditions that masquerades as surgically treatable spinal disease.

Abbreviations used in this paper: ALS = amyotrophic lateral sclerosis; CTS = carpal tunnel syndrome; EMG = electromyography; TOS = thoracic outlet syndrome.

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Metabolic, Vascular, Inflammatory, and Infectious Masqueraders of Spinal Disease

Metabolic Deficiencies

Malnutrition can be found in all areas of the world and can be the result of deficient nutrient intake, absorption, or utilization. For example, the increasing prevalence of bariatric surgical procedures raises concern for the possibility of increasing prevalence of nutritional deficiencies in this growing population. Micronutrient deficiencies are common in postoperative gastric bypass patients. Hypocupremia, such as found secondary to impaired absorption following bariatric surgery, can mimic significant myelopathy. Spinal cord changes seen in hypocupremia can even affect the brainstem. Supplementation, regardless of the cause, has been effective in correcting the neurological manifestations of copper deficiency. Other nutrient and vitamin deficiencies, such as vitamin B12 and folate deficiency, have characteristic effects on the posterior and lateral columns of the spinal cord. Symptoms of weakness, paresthesia, impaired proprioception, and impaired vibratory sensation can mimic spinal cord disease, especially severe myelopathy, as the condition progressively worsens. These deficiencies can be secondary to poor absorption and malnutrition in the setting of alcoholism and pernicious anemia or as a result of impaired cofactor utilization in rare conditions. Micronutrient deficiency should also be addressed in chronic hemodialysis patients with neurological symptoms attributed to the spine because these individuals are commonly deficient of several vitamins and minerals. Homocysteine is converted to methionine en route to myelin synthesis, impairment in this pathway leads to neurological dysfunction and can be corrected with supplementation, not surgery.

In addition to nutritional deficiencies, the chronic condition of impaired glucose utilization (diabetes mellitus) is another widespread disease with secondary neurological effects. Although not yet fully elucidated, our working understanding of the pathophysiology of diabetic neuropathy is that it stems from chronic hyperglycemia, which leads to a dysfunction of primary hemostasis and increased activity of the coagulation system, ultimately reducing endoneurial blood flow. Increased oxidative stresses in turn increase the activity of the nuclear factor–κ B, as well as the production of vasoactive factors and cytokines leading to demyelination and/or programmed cell death (apoptosis). It is not known why different patients have a predilection for the development of neuropathy in various locations. Diabetic neuropathy that mimics spinal disease can be classified into 3 categories: sensory mononeuropathy, proximal motor neuropathy, and distal sensory neuropathy. An example of sensory mononeuropathy is CTS, a condition that can mimic cervical radiculopathy. Outcomes after surgical release of the carpal tunnel in patients with diabetes indicate that release is an effective treatment. Proximal motor neuropathy can present as weakness, pain, and atrophy of the proximal lower extremities and should not be mistaken for lumbar spinal stenosis. The most common type of neuropathy in diabetic patients, distal sensory neuropathy, typically affects the distal lower extremities first with paresthesia, numbness, or pain. In progressive disease, these symptoms progress proximally and can commonly be confused with or confounded by lumbar spinal stenosis. Because of diabetes’ widespread prevalence, it is important to determine preoperatively if this disease is present. In addition to establishing a differential diagnosis of surgically and nonsurgically treatable spinal conditions, it is also useful in the surgical evaluation of patients. In the Spine Patient Outcomes Research Trial, diabetic patients with spinal stenosis and degenerative spondylolisthesis were shown to benefit from surgery, whereas patients with disc herniations and diabetes generally did not benefit from surgical intervention. Hence, even traditionally viewed surgically treatable lesions in diabetic patients may in fact be masqueraders of surgically correctable lesions.

Infectious Diseases

Infection of the spine can be classified as intradural infectious disease, extradural infectious disease, and vertebral column infectious disease. The first and last of these are generally treated nonsurgically, whereas the second often represents a surgical emergency. (A caveat to this would be the rare case of subdural empyema.) Intradural infectious disease can produce symptoms attributable to a mass lesion within the spinal cord itself or even mimic radiculopathy. Infections, such as tuberculosis, have been reported to infiltrate the spinal cord and are treated effectively with antitubercular agents. Although tuberculosis is more common in developing countries, immunocompromised patients in any geographic location should raise suspicion for infectious conditions. In a review of 55 patients with AIDS and associated spinal infections or neoplasms, cytomegalovirus polyradiculitis was the most common cause of intradural extramedullary disease, with herpes radiculitis and tuberculous infection being less common. HIV itself can cause direct infiltration of the spinal cord, resulting in a condition known as vacuolar myelopathy. Spirochete infection can also infiltrate the spinal cord and cause inflammation associated with neurological manifestations. The mainstay of these treatments is nonsurgical.

Infectious disease of the disc space and vertebral body are causes of severe back pain and can mimic surgical causes of radiculopathy, myelopathy, and sensory loss. Unlike the disc space, excellent clinical outcomes are typically achieved nonsurgically when the structural integrity of the vertebral body is maintained. Risk factors for osteomyelitis of the spine include immunocompromised status, intravenous drug abuse, and the presence of a right-to-left cardiac shunt.

Autoimmune, Neurodegenerative, and Inflammatory Disease

Imaging and pathomorphological studies in multiple sclerosis suggest that axonal injury and axonal loss play a crucial role in individuals with persistent disability and long-standing disease. Demyelinating plaques and myelitis, particularly in the cervical spinal cord, can produce a variety of neurological conditions that are shared with surgical spine lesions. These include weakness, radiculopathy, and myelopathy.
Diagnostic pitfalls in spine surgery

Other examples of diseases with insidious onset that commonly present for spine surgery evaluation include ALS and Parkinson disease. In a study of 344 patients with Parkinson disease, 6% were initially referred to a surgeon for spinal or gait-related problems. Also, early ALS can mimic radiculopathy, myelopathy, mononeuropathy, and arthropathy. In a review of 260 consecutive patients with ALS, 55 had had surgery within the 5 years prior to diagnosis of ALS. A remarkable 34 of these 55 underwent surgery for symptoms and signs that retrospectively were attributable to early manifestations of ALS.

Clinical Vignette: Case 1

This 63-year-old man presented to our clinic with neurological decline following anterior cervical disectomy and fusion for cervical myelopathy. He had developed worsening gait unsteadiness, spasticity, and hand clumsiness (Fig. 1). He underwent posterior laminectomy, and his symptoms initially improved. Three years later, however, he began ambulating slower, taking short steps, and he had a stooped posture. Consideration was given to possible decompression of adjacent levels that were stenotic (Fig. 2). On examination, he had now developed increasing left-sided stiffness with spasticity and a new left-sided tremor. He was referred to a movement disorder neurologist who diagnosed Parkinson disease. Neurological symptoms were treated successfully with dopamine agonist therapy; further surgery was avoided.

Clinical Vignette: Case 2

This 63-year-old woman presented with progressive, painless bilateral arm weakness. The weakness was greater on the right side and affected the proximal muscles more prominently (deltoid muscle Grade 3/5 strength; remaining muscle groups Grade 4/5 strength). Magnetic resonance imaging demonstrated a prominent C6–7 disc resulting in severe stenosis at that level (Fig. 3). The patient’s neurological findings did not correlate with the imaging findings, and electromyography demonstrated positive sharp waves, fibrillations, and long-duration and high-amplitude motor unit action potentials throughout right arm, right thoracic paraspinal areas, and right leg, consistent with the electro-diagnostic criteria for ALS. Surgery was averted as her symptoms were not attributable to the radiographic lesion and could be explained by her systemic diagnosis.

A variety of other systemic inflammatory diseases such as Behçet disease and sarcoidosis can also mimic disease of the spine treated surgically. While uncommon, they must be recognized as nonsurgical entities. In a study of patients treated for spinal cord sarcoidosis, the nonsurgical group had better outcomes than the surgical group.

Clinical Vignette: Case 3

This 37-year-old man presented with axial cervical and thoracic spine pain. There were no neurological findings such as weakness or abnormal reflexes. Flexion and extension of his neck was limited secondary to pain. There was no lymphadenopathy, and his serum laboratory studies were normal except for an elevated serum angiotensin-converting enzyme level. Plain chest radiography and abdominal CT scanning showed findings within normal limits. Spinal MR imaging demonstrated several cervical, thoracic, and lumbar vertebral bodies with abnormal, increased signal intensity (Fig. 4). Biopsy of a skin rash demonstrated sarcoidosis. After 6 months of steroid therapy, all the patient’s symptoms resolved, and repeat MR imaging revealed normal finding (Fig. 5).

More localized inflammatory conditions can also mimic disorders of the spine that are amenable to surgery, such as brachial neuritis (Parsonage-Turner syndrome). Patients with this condition often experience a sudden onset of severe pain in or about the shoulder girdle, followed by weakness of at least one shoulder muscle. This can
easily be confused with a number of disorders, such as cervical radiculopathy (particularly in patients with significant preexisting spondylosis). The diagnosis should be suspected, especially when the pain decreases spontaneously and weakness subsequently develops. Electromyography can confirm the neuropathy once weakness is apparent.

**Peripheral Vascular Disease**

The differentiation of neurogenic claudication (resulting from lumbar spinal stenosis) from lower-extremity vascular claudication is not always easy from a patient’s history of lower-extremity cramping on exertion. Peripheral arterial disease is typically also associated with impotence in men, dystrophic skin changes, foot pallor, and decreased peripheral pulses. Typically, patients with neurogenic claudication experience relief with changes in posture (bending forward, sitting, and so on) as opposed to vascular claudication that merely improves with rest regardless of position. Ankle/brachial indices, Doppler ultrasound, and vascular surgery consultation may also be helpful in reaching a diagnosis. Ultimately, like several of the conditions described in this review, these conditions may occasionally coexist in the same patient.

**Inherited Neurological Diseases**

Certain inherited neurological diseases have variable penetrance and delayed onset of expression. Thorough assessment must be undertaken to identify discrepancies in a patient’s history and examination, particularly when radiographic and physical findings are discordant.

**Clinical Vignette: Case 4**

This 44-year-old man presented with upper- and lower extremity neurologic symptoms...
lower-extremity weakness and muscular atrophy. He was found to have multilevel cervical stenosis and elected to undergo multilevel cervical laminectomy and foraminotomy. Postoperatively, he experienced continued loss of function, despite adequate radiographically documented decompression. Profound atrophy of the right trapezius, supraspinatus, infraspinatus, deltoïd, and biceps muscles was present (Fig. 6). While there was initially concern for residual cervical root compression, further workup revealed spinal muscular atrophy Type IV. Further surgery was not indicated as a causative diagnosis had been reached.

Musculoskeletal Masqueraders of Surgical Spinal Disease

Because upper- and lower-extremity musculoskeletal problems are common, the spine surgeon must frequently evaluate a patient who has both a spinal condition and an appendicular source of pain. In a review of patients referred for electrodiagnostic testing, for example, 21% of the patients with a lumbosacral radiculopathy also had a musculoskeletal disorder such as myofascial pain, trochanteric bursitis, or iliotibial band syndrome.8

Shoulder Pathology

Patients with shoulder disease can present with pain and weakness, and this pathological entity needs to be differentiated from cervical radiculopathy. Occasionally, a patient with a rotator cuff tear undergoes MR imaging of the cervical spine and is referred for nerve root decompression, usually the C-5 nerve. Attention to the possibility of shoulder pathology in the examination of a patient with suspected C-5 nerve dysfunction could avoid a diagnostic error. In C-5 radiculopathy, there can be weakness of shoulder abduction, flexion, internal and external rotation, and elbow flexion. The pain is often provoked by movement of the neck, and it radiates down the neck. The pectoral and the biceps reflexes are affected. There can be decreased range of motion of the shoulder due to weakness, but the movement is generally not painful unlike what patients can experience with rotator cuff tears. With rotator cuff tears, activity often produces pain that is diffuse and can resemble C-5 radiculopathy (especially when performing overhead movements). Rotator cuff tears produce weakness of shoulder abduction and external rotation. When a tear has been present chronically, it is associated with atrophy of the supra- and infraspinatus musculature. If a rotator cuff tears is suspected, an MR image of the shoulder should be obtained, as the modality is both sensitive and specific.9 A shoulder examination, including palpation, range of motion, strength testing, and provocative tests, is usually diagnostic, and its effectiveness has been reviewed recently.40 Electromyography and selective nerve root blocks can be used for further diagnostic elucidation.40

Another shoulder condition, acromioclavicular arthritis, often presents with localized pain over the anterosuperior aspect of the shoulder and tenderness and is uncommonly mistaken for cervical radiculopathy. The cross body adduction test is frequently positive in acromioclavicular arthritis. Additionally, adhesive capsulitis often presents with pain and a reduction of range of motion. The pain is often diffuse, can encompass the deltoid re-
gion, and can resemble a C-5 radiculopathy. Radiographic studies are generally negative. Passive range of motion is significantly limited, unlike that in a patient with radiculopathy. A diagnosis is more complicated if both spine and shoulder conditions are present.

Clinical Vignette: Case 5

This 78-year-old man, with a known supraspinatus tear and shoulder pain, presented with progressive weakness of external shoulder rotation. His internist suspected a superimposed radiculopathy, and cervical imaging demonstrated severe C-4 and C-5 foraminal stenosis (Fig. 7); EMG revealed evidence of an ipsilateral C-5 radiculopathy. He was appropriately referred for surgical decompression and improved following selective foraminotomy.

Clinical Vignette: Case 6

This 66-year-old man had previously undergone a cervical laminoplasty for cervical stenosis. He presented with new-onset bilateral triceps weakness after a mechanical fall. Cervical MR imaging demonstrated residual stenosis of the C-7 nerve roots. He was referred by a neurologist for consideration of reoperative decompression. Physical examination revealed features consistent with bilateral triceps tendon rupture, and the patient underwent bilateral repair with improved strength postoperatively. Spinal reoperation was not necessary.

The diagnostic features distinguishing triceps tendon rupture from a cervical radiculopathy are based on the physical examination. They include the presence of tenderness at the site of possible rupture, a palpable defect in the tendon, and the absence of weakness in other C-7 myotomes, such as the wrist and finger extensors.

Hip Arthropathy

Patients with hip pathology can present with symptoms that mimic spinal stenosis or lumbar radiculopathy. Hip and spine degenerative disease are common, and their incidence increases with advancing age. It is therefore not surprising to encounter each condition in the same patient. A comprehensive review of the incidence
of hip osteoarthritis places the mean incidence at 8%. Lumbar stenosis studies place its prevalence higher than 19% in individuals over 60 years of age.

Patients with hip arthritis present with pain in the inguinal region, an antalgic gait, and a decreased hip range of motion. The groin pain can radiate widely and commonly affects the anterior and lateral thigh. It can also involve the buttock and can occasionally radiate below the knee. A study of referred pain patterns in patients undergoing hip intraarticular injections demonstrated that the buttock was a site of referral in 71% of patients compared with the thigh (57%) and the groin (55%). An upper lumbar radiculopathy can also cause radiating pain to the inguinal region and anterior thigh. Usually, a careful examination can clarify the diagnosis. The absence of a sensory disturbance, the provocation of pain on internal rotation of the hip joint, and an antalgic gait with an abbreviated strike phase on the affected side should alert one to the presence of a hip problem. In fact, a retrospective study of patients presenting to an orthopedic clinic found that patients with a limp and limited internal rotation of the hip were 7 times more likely to have a hip problem than a spinal diagnosis. In another study of patients presenting to a spine clinic, 12.5% of patient had a diagnosis referable to a hip joint. Plain radiographs of the hip are readily obtainable and can reveal a decreased joint space, the presence of subchondral cysts, sclerosis, and the formation of osteophytes.

Occasionally a double diagnosis exists with a single pain being dominant. In this situation, it is advisable to discuss with the patient which symptoms are attributable to each region and to decide whether an operation at one or both sites is warranted.

Clinical Vignette: Case 7

This 41-year-old man had symptoms of neurogenic claudication and right thigh pain when ambulating. He was referred for a lumbar laminectomy by a neurologist after an MR imaging revealed L3–4 and L4–5 central canal stenosis. His neurological examination was unrevealing. Physical examination demonstrated a markedly reduced and painful range of motion of the right hip. He ambulated with a limp. Plain radiography and MR imaging (Fig. 8) confirmed the diagnosis of hip arthropathy, and the patient elected to undergo a hip replacement and a lumbar decompression because he had symptoms attributable to both areas. Postoperatively, he experienced resolution of both the stenosis and hip-related symptoms.

Peripheral Nerve Diseases and Entrapment
Masquerading as Compressive Radiculopathy

Patients with entrapment neuropathies can present with weakness, pain, and sensory loss. When they occur in a patient with concurrent spondylosis or stenosis, they can lead to an unnecessary spinal operation. In general, the key to the diagnosis is in the characterization of factors that exacerbate the symptoms and in the distribution of the sensory and/or motor abnormality.

Thoracic Outlet Syndrome Mimicking Cervical Radiculopathy

Thoracic outlet syndrome can present with symptoms resembling cervical radiculopathy. It can be divided into neurogenic TOS, vascular TOS, and disputed TOS. Vascular TOS occurs less commonly than neurogenic TOS and presents with venous stasis, arterial insufficiency, and even embolic stroke. It is not typically diagnostically confused with cervical radiculopathy. Neurogenic TOS, however, generally presents with an inferior brachial plexus compression. As it courses to the arm, the plexus traverses...
undergo cervical imaging and can be referred to a surgeon in patients with CTS. In a study of diagnostic tests performed for TOS, patients with suprascapular nerve entrapment exhibit denervation in the supraspinatus and infraspinatus muscles, with sparing of other C-5–innervated muscles such as the deltoid and biceps. If it is caused by a ganglion cyst, ultrasound or an MR imaging will demonstrate the cyst. However, in cases of impingement at the level of the transverse ligament, imaging will typically be negative.

**Axillary Nerve Entrapment Mimicking C-5 Radiculopathy**

The axillary nerve emerges from the posterior cord of the brachial plexus and traverses the quadrilateral space before giving rise to the teres minor and deltoid nerves. It traverses the quadrilateral space with the posterior circumflex humeral artery. Entrapment of the axillary nerve in the quadrilateral space was first described in 1983. It usually occurs spontaneously in young individuals. It presents with pain over the quadrilateral space, anterior aspect of the shoulder, and arm paresthesias. The presentation resembles rotator cuff tendinitis, acromioclavicular pathology, and C-5 radiculopathy. It is a rare entrapment and is therefore likely underdiagnosed. The results of neurological examination are often normal, although there may be deltoid weakness. Magnetic resonance imaging can reveal atrophy of the teres minor. Electrodiagnostic testing can demonstrate deltoid denervation. If positive, the EMG findings narrow the diagnosis because the observed denervation is limited to the deltoid muscle. In contrast, C-5 radiculopathy is also associated with denervation of the paraspinal musculature, the supraspinatus, and the biceps musculature. Angiography can demonstrate occlusion of the posterior circumflex humeral artery with abduction and external rotation of the arm.

**Carpal Tunnel Syndrome Mimicking C-6 Radiculopathy**

Cervical radiculopathy and CTS are both common and can coexist in the same patient. In a series of 12 patients who initially were diagnosed with CTS and who underwent ineffective operations, 3 were ultimately diagnosed with cervical radiculopathy. The prevalence of CTS is approximately 125 per 100,000. Patients with CTS present with hand numbness, paraesthesias, and pain that can radiate into the forearm and, rarely, into the arm. Although patients frequently describe the symptoms as affecting the entire palm of the hand, the distribution of sensory symptoms most closely resembles the C-6 dermatome.

There are a number of key distinguishing features between CTS and cervical radiculopathy. The symptoms of CTS are often worse after use of the hand and at night. Although the pain and paresthesias can radiate up the
forearm and arm, there is a sensation that they originate in the palmar region of the hand. The symptoms of cervical radiculopathy, unlike those of CTS, can often be provoked by neck movement. A careful sensory examination can differentiate the 2 conditions. In CTS, inspection of the musculature may reveal an asymmetry in the thenar eminence. Any loss of motor dysfunction is limited to the thenar muscles. In a C-6 radiculopathy, the biceps, triceps, wrist flexors, and deep finger flexors can all be affected. In CTS, the loss of sensation occurs distal to the wrist crease and spares the proximal palmar aspect of the hand, due to the forearm takeoff point of the palmar sensory nerve. In a cervical radiculopathy, the loss of sensation can be pronounced at the level of the fingers because of their rich innervation, but it extends into the forearm.

**Ulnar Nerve Entrapment at the Elbow Mimicking C-8 Radiculopathy**

This is not a common diagnostic pitfall because the 2 conditions present differently. In general, C-8 radiculopathy is uncommon. The key to distinguishing these 2 entities is a careful examination. Percussion of the ulnar nerve at the elbow often elicits a Tinel sign. Keeping the elbow flexed for 3 minutes often provokes the symptoms. In severe C-8 radiculopathy, there is weakness of thenar muscles, which are carried with the median nerve and are therefore spared in severe ulnar neuropathy. In ulnar nerve entrapment at the elbow, the sensory examination is characterized by hypesthesia over the fourth and fifth digits, extending over the palm and dorsal aspect of the hand medially. In C-8 radiculopathy, the sensory loss extends proximally over the medial forearm.

**Peroneal Nerve Palsy Mimicking L-5 Radiculopathy**

An L-4 or L-5 radiculopathy can result in weakness of ankle dorsiflexion. At times a patient with a foot drop may be referred for foraminal decompression when, in fact, the problem is peripheral. Lumbosacral plexopathies, sciatic neuropathies, and peroneal nerve palsies can all present with weakness of ankle dorsiflexion and a steppage gait. Because the peroneal nerve arises from fibers of the L-4, L-5, and S-1 nerve roots, radiculopathy affecting these roots necessarily also affects the peroneal nerve. There are a number of important differences on motor and sensory examination. As with any attempt at differentiating potential nerve root pathology from a peripheral nerve entrapment, one must attempt to identify a muscle both supplied by the suspected root and uninverted with a potential entrapment. In peroneal nerve palsy, the motor weakness involves the ankle and toe dorsiflexors and the ankle extensor muscles. In L-5 radiculopathy, there is weakness of ankle inversion (tibialis posterior muscle), which is carried by the posterior tibial nerve, in addition to the weakness of dorsiflexion and toe extension. An L-4 radiculopathy presents not only with dorsiflexor and inversion weakness, but commonly there is also frequent weakness of the quadriceps muscle and a diminished patellar reflex. On sensory testing, differences can also be characterized. In peroneal nerve palsy, the sensory abnormality can limit itself to the first web space if it involves only the deep branch of the peroneal nerve. If the entrapment affects both the deep and superficial peroneal nerves, the sensory abnormality will also involve the top of the foot and lateral aspect of the leg but will not be as proximal in extent as an L-5 radiculopathy.

**Conclusions**

Meticulous evaluation is necessary to identify lesions that masquerade as surgically treatable spine disease that can lead to erroneous diagnosis and treatment. Key aspects of the historical presentation, diagnostic and provocative examination of the patient, and anatomical understanding should be considered of primary importance in the evaluation of select patients given the high prevalence rates of radiographically documented spinal abnormalities.

**Disclosure**

The authors report no conflict of interest concerning the materials and methods used in this study or the findings specified in this paper.

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