C.2. Diagnosis and Management of Non-Traumatic Neck and Back Problems

Introduction

Degenerative diseases of the spine provide the typical neurosurgeon with the bulk of his or her practice. Low back pain is the most common specific complaint generating a physician visit, and is the most often cited reason for missed work. It is estimated that 75% of people will visit a health care professional at some time in their lives for back pain. Estimates of prevalence suggest that at any given time 1 in 5 adults in the US will complain of low back discomfort if asked.

The direct and indirect economic impact of low back complaints is enormous. Direct costs of treatment are estimated at $20-30 billion a year. Some 200 million person-days of work are lost per year due to low back problems. Low back pain has recently ranked 6th in hospital-bed days per year. Clearly, efficient and effective management of degenerative conditions of the spine is crucial, both at the level of the primary care provider, and at the level of the subspecialty spine surgeon.

The purpose of this chapter is to give the student an overview of how patients present with degenerative complaints of the lumbar and cervical spine. The focus will be on identifying symptom complexes that point to specific pathological entities that are surgically treated. Special attention will be paid to identifying signs and symptoms that are suggestive of serious pathology that may threaten life or neurologic function and require urgent attention.

a. Categories of Symptoms:

In order to have an orderly approach to the patient with spine related complaints, the physician needs to be familiar with the broad categories of symptoms with which patients will present. The differential diagnosis of spine related pain is extensive. Although the vast majority of patients have benign, non-surgical problems, very serious medical illness can present with the chief complaint of back, neck, or extremity pain. With experience, common presenting syndromes emerge, and allow more rational triage of patients into groups who need further evaluation, and perhaps surgical referral, and those who are best managed without extensive testing and referrals.

i. Axial Spine Pain

Pain in the spine with or without radiation into the extremities is the most common presenting compliant related to spinal disease. Initial evaluation should center on obtaining a detailed history of the patient’s complaints and previous medical history. A history of recent trauma should raise the suspicion of a fracture. Character, severity, frequency, inciting and relieving events should be thoroughly explored. Inquiry about associated symptoms, such as fever and visceral pain can suggest diagnoses and avenues of investigation. The timing of pain can also lead to appropriate investigation. Morning stiffness and pain that relents as the day progresses suggests an inflammatory disorder, where nocturnal pain associated with recumbency is a much more ominous symptom, being seen with malignant, destructive lesions.

Activity-related back pain without neurologic symptoms or findings is quite common. In the absence of other worrisome symptoms or findings, most patients

can be successfully managed with a brief reduction in activity level and analgesics. The optimum duration and content of non-operative treatment is controversial. Some practitioners advocate strict bed rest, while others avoid it. Reduction of general activity level while avoiding any specific inciting events, coupled with non-steroidal agents, is the mainstay of most clinicians' first line therapy.

Evaluation for pure axial spine pain that does not resolve with conservative measures can include plain radiographs, magnetic resonance imaging, computed tomography, myelography, electrophysiologic studies, and discography. A set of plain films can exclude fracture, and identify serious bony pathology such as a destructive tumor. However, for soft tissue imaging the MRI is the study of choice. CT can give detailed information about bony anatomy. Myelography is sensitive to canal and foraminal pathology, and when paired with a post-myelogram CT scan, can be very useful in the patient in whom an MRI is not an option. Discography is controversial, and will not be discussed at length.

ii. Radiculopathy

Referred pain into the upper or lower extremities often accompanies back or neck pain. Referred pain can be the initial symptom of a compressed nerve root by a ruptured disc or neural foraminal stenosis from osteophytes. Radicular pain is usually described as sharp or even shock-like, and may be associated with certain activities or positions. The distribution of the pain may not always be classic, and often doesn't respect dermatomal distributions.

Sensory changes are also often seen, with complaints of tingling and numbness being very common. On examination decreased sensation to pinprick and light touch are found in a dermatomal distribution in many patients. It is interesting that areas of referred pain and sensory loss often are different. Making determinations of level of nerve root compression solely from pain or sensory distribution is often difficult.

Motor weakness is also seen in nerve root compression syndromes. Muscle innervation is more constant and has less overlap than sensory innervation and is better at predicting level of pathology. Motor deficits that are of a more long-standing nature can have significant wasting. Hyporeflexia in the appropriate distribution is also seen.

aa. Cervical

Cervical radiculopathy can present acutely, as with a traumatic ruptured disc, or can be of a more chronic and intermittent nature, as is seen in foraminal narrowing from osteophytes. Typically, the inferior nerve root is affected (e.g. C5-6 disc abnormalities affect the C6 nerve root). C5-6 and C6-7 are the most commonly affected segments.

A C5 radiculopathy typically presents with pain in the shoulder and the upper part of the lateral arm. Paresthesias are often seen in the more distal part of the affected dermatome. Deltoid weakness is seen commonly with a C5 radiculopathy. Biceps or brachioradialis weakness can be seen with a C6 radiculopathy along with the appropriate hyporeflexia. Paresthesias and frank sensory loss are more distal, and can extend into the hand. Root compression at C7 produces triceps weakness and a

decreased triceps reflex. Pain extending into the distal forearm or hand is common. Sensory loss is commonly seen in the hand.

bb. Lumbar

Sciatica is a classic syndrome of lower lumbar nerve root compression. Low back pain, that may or may not have been associated with some sort of trauma, is commonly antecedent to the onset of leg pain by days to a few weeks. Pain tends to be more proximal, and in a slightly different distribution than sensory changes. Motor weakness is also seen, but can be missed if dynamic testing is not done. All patients should be asked to stand on their toes and heels, as confrontational testing will miss subtle motor deficits in the lower extremities. As in the cervical spine, the pathologic level usually affects the caudal nerve root (e.g. L5-Sl disc produces an S1 radiculopathy). L5-S1 and L4-5 are overwhelmingly the most common levels affected. The upper lumbar spine is affected less frequently.

The classic S1 radiculopathy results in pain down the back of the leg and into the heel or foot. Sensory loss is usually over the lateral aspect of the foot. Plantar-flexion weakness is seen, but can be subtle. A loss of the Achilles reflex is also fairly specific to S1. The L5 radiculopathy produces similar pain, but the sensory symptoms tend to be over the dorsum of the foot. Weakness in dorsiflexion of the foot (or more specifically extensor hallicus longus) is the motor finding associated with L5. There is not a reliably reproducible reflex associated with L5.

iii. Cauda Equina Syndrome

The cauda equina syndrome is important to recognize, as prompt surgical attention may be necessary in cases of acute pathology. Patients typically present with low back pain and diffuse lower extremity complaints. Minimal or absent leg pain is seen, in contrast to the predominance of extremity pain seen in the radicular syndromes. Bowel, and, more commonly, bladder dysfunction, are also seen. In many patients this goes unrecognized until bladder distention leads to overflow incontinence. Because of the vague nature of the complaints, and the common lack of severe pain associated with a large disc herniation producing a cauda syndrome, delay in diagnosis is not uncommon. Patients with back pain and complaints of lower extremity weakness should be carefully examined to rule out a cauda equina syndrome. Weakness can be diffuse, and vary from subtle to paraplegia. Sensory findings are variable, but often found in the perineal area. Checking a post void residual can give a quick initial assessment of bladder function, and should be done prior to placing a foley. Reflex changes are variable, but in general reveal diffuse hyporeflexia. Patients in whom the diagnosis is suspected should undergo urgent imaging with MRI or a myelogram.

Recovery from a severe cauda equina syndrome, even with prompt surgical management, can be very slow and incomplete. Bladder function is often the slowest symptom to improve. This is another reason to keep a high index of suspicion for this uncommon entity in patients presenting with back pain and complaints of lower extremity weakness.

iv. Myelopathy

Myelopathy is the clinical presentation of pathology affecting spinal cord function. The differential diagnosis for causes of myelopathy is large and includes trauma,

metabolic, degenerative, inflammatory, toxic, infectious, and neoplastic etiologies. Degenerative conditions of the spine may produce the symptoms of myelopathy. In many instances the onset of the myelopathy is insidious, and symptoms and signs subtle. Longstanding myelopathy, unfortunately, is rarely reversible. Early identification of patients with progression of myelopathy is essential to prevent permanent loss of neurologic function. Therefore, in patients who present with neck or thoracic spine pain, the history and physical exam should be tailored to exclude myelopathy.

The most general signs of myelopathy are those of upper motor neuron dysfunction. Subtle symptoms include difficulty with fine motor control of the hands and fingers, gait problems and instability, and numbness. Hyperreflexia, increased tone, and weakness are the hallmarks of the clinical exam. Abnormal plantar response and Hoffman's sign are frequent abnormal reflexes seen in patients with myelopathy. Urinary dysfunction, such has hesitancy, frequency, and incontinence, is also seen, but tends not to be severe.

aa. Chronic

Progression of the myelopathy can be very slow and gradual, or stepwise. It is not uncommon for the onset to be so insidious that patients are quite disabled before they seek medical evaluation for their symptoms. Careful history and examination can direct the level of suspicion. In general, symptoms that affect the hands and upper extremities should prompt cervical evaluation, while isolated lower extremity symptoms and a trunk sensory level are more suspicious for a thoracic lesion. Often in chronic myelopathy the distinction can be difficult. In patients with chronic myelopathy an MRI is the study of choice to evaluate the spinal canal. In patients who are unable to have an MRI, myelography and CT myelography is adequate and gives good information about the spinal canal.

bb. Acute

Degenerative disease may lead to acute onset of myelopathy. Acute disc herniation can be seen in the setting of trauma, but also without significant injury. Patients who present with the acute onset of myelopathic symptoms deserve urgent evaluation with MRI or myelography. If pathology such as an acutely ruptured disc causing spinal cord compression is found, surgical evaluation should be sought. Unfortunately, patients with complete spinal cord injuries only infrequently make full recoveries.

Acute worsening of cervical myelopathy in the setting of cervical stenosis can be seen in the face of fairly minor trauma. In patients with acute myelopathy without obvious fracture, but significant degenerative disease, cervical stenosis should be suspected.

b. Specific Conditions

i. The Herniated Disc

The central portion of the intervertebral disc is the nucleus pulposus. Under certain pathologic conditions it may rupture through the annulus fibrosis and into spaces occupied by neurologic structures. Central, large disc rupture causing compression of the spinal cord or cauda equina may be seen. It is, however, much more

common to find a posterior-lateral rupture producing nerve root compression and radiculopathy.

Mechanisms producing radicular pain are poorly understood. Direct compressive effects certainly play a role. The dorsal root ganglion appears especially sensitive to compressive effects. Recent animal models have suggested a role for biochemical factors leading to inflammation. Increasingly, experimental evidence suggests that the mechanisms leading to pain generation are more complex than once thought.

aa. Lumbar

Lumbar radiculopathy is commonly known as sciatica. The classic presentation is in younger patients who will present with a history of back pain followed in a few days to weeks by intense leg discomfort, paresthesias, and radicular weakness as described in the previous section.

Soft disc herniations in the lumbar spine leading to radicular complaints are seen most often in the 3rd through 6th decade of life. Estimates of prevalence vary widely in the literature, from as low as 2% to high as 40%.

Non-surgical therapies for symptoms due to lumbar disc herniation are plentiful. The natural history of radiculopathy is one of improvement in many individuals. A variety of therapies are successful in helping patients get through very painful periods. Oral or epidural steroids can be quite successful in managing lumbar radiculopathy, although the results are often temporary. Physical therapy, chiropractic manipulation, and a host of other devices and regimens are used and promoted. Definitive evidence on the superiority of any particular approach to non-surgical therapy is lacking. All patients without severe neurologic deficit should undergo a trial of non-surgical therapy. The duration of non-surgical therapy is not set, and is often driven by the patient's ability to continue to tolerate their symptoms. Frequently 4 weeks, and, preferably 2-3 months of non-surgical treatment are recommended., It is, however, common for patients with severe pain or neurologic deficit to be operated upon more quickly.

The lumbar laminectomy for discectomy is one of the most widely performed spinal procedures. Despite this, indications continue to be debated. Except for patients with a cauda equina syndrome, non-operative therapy is always an option. There is prospective data from Weber that would suggest that patients that undergo discectomy improve more quickly over the short term. This benefit appears to dissipate by 4 years. With those disclaimers, most spine surgeons would agree that reasonable operative indications would include 1) large midline disc herniation with resulting cauda equina syndrome; 2) nerve root compression with pain and significant motor and sensory deficit; 3) nerve root compression with or without neurologic deficit and incapacitating pain that fails to improve with non-surgical measures; 4) recurrence of incapacitating episodes of LBP and sciatica that prevent the patient from leading a normal life.

In patients with clinical radiculopathy and concordant imaging findings a successful surgical outcome can be expected in 80-95% of patients. Recurrence rates are reported at 2-12%. The incidence of serious complications is very low (<2%).

bb. Cervical

Cervical radiculopathy from nerve root compression can be caused by a herniated disc or from foraminal narrowing from osteophytes. The root compression syndromes produced by these conditions have been described above. Neck pain associated with degenerative disc disease and osteophytes will improve in the majority of people without invasive treatment; although there is certainly a group that will go on to have chronic symptoms.

The natural history of cervical radiculopathy is not as well characterized as that of cervical myelopathy from degenerative disease. Radiculopathy will improve with time in many patients. However, it is impossible to define strict rules on the length of non-surgical therapy to undertake before surgery should be considered. Cervical radiculitis from a soft disc herniation may be less likely to improve spontaneously as that due to osteophytes. Non-surgical therapy can include oral or epidural steroids, cervical traction, physical therapy, bracing, and many others.

In carefully selected patients with radicular symptoms and evidence of nerve root compression on their imaging studies, more than 90% can expect a favorable outcome with careful surgical management. Serious complications are rare (<1%).

ii. Spinal Stenosis

Spinal stenosis is the narrowing of the cross-sectional diameter of the spinal canal to such an extent that neurologic symptoms or signs are produced. The syndromes produced by lumbar and cervical stenosis are quite distinct and will be discussed separately.

aa. Lumbar

Lumbar stenosis classically produces neurogenic claudication. Neurogenic claudication is leg pain produced by walking or standing that is typically relieved by a change of position such as squatting, leaning over or sitting down. Leg pain can be in a variety of distributions, and becomes quite debilitating. Patients often report associated paresthesias. Neurologic examination may be normal at rest, though sensory deficits and hyporeflexia are sometimes seen. When motor weakness is found it can be associated with wasting, as stenosis is usually a slowing progressive disease. Approximately 2/3 of patients with symptomatic spinal stenosis will present with some variety of the classic picture of neurogenic claudication.

Acquired spinal stenosis is caused by advanced degenerative disease of the disc, facets and ligaments. The hypertrophy of the facets and associated ligaments, such as the ligamentum flavum, combine with bulging discs to produce both central and lateral narrowing. Most patients with acquired lumbar stenosis are in their 6th to 7th decade or beyond.

Surgical treatment for lumbar stenosis involves decompressive laminectomy and may require medial facetectomies for lateral recess stenosis and foraminal stenosis. More recently, surgeons have been exploring the role of lumbar fusion in the treatment of spinal stenosis in the older population. The role of fusion and instrumented fusion in this setting is yet to be fully determined. The reader is referred to the suggested readings for more on this topic.

Early improvement after surgery for lumbar spinal stenosis is the rule (>90%) in patients with a postural component to their pain. However, late progression of symptoms is not uncommon. A large review by Turner et al. suggests that good results are maintained in approximately 64% of patients over time.

bb. Cervical

Cervical spondylotic myelopathy (CSM) is the clinical entity produced by cervical stenosis. CSM usually progresses slowly, in a stepwise fashion. This myelopathy can be quite subtle in the early stages, and some patients will have significant disability before seeking appropriate medical care. The most common presenting complaints include neck pain, gait difficulties, and hand numbness and clumsiness. Loss of bowel and bladder control is uncommon early in CSM. Occasionally patients will present with acute and profound spinal cord injury after mild trauma (usually a hyperextension injury). More common is a stepwise decline in spinal cord function.

The typical patient with CSM is older than 50 and male. Men are seen nearly twice as often as women. Myelopathic findings dominate the physical examination of patients with CSM. Increased reflexes in both the upper and lower extremities with lower extremity spasticity are common. Pathologic reflexes such as Babinski and Hoffman are also often positive. Lhermitte's sign (electric, shock-like pain radiating down the spine on neck flexion) is classically described, but occurs in a small minority of patients. Complicating the clinical picture in CSM is the lower motor neuron findings that can be seen secondary to nerve root compression. Wasting, fasciculations, and hypoactive reflexes can be seen in the upper extremities due to nerve root compression.

The differential diagnosis of CSM includes multiple sclerosis, syringomyelia, spinal cord tumor, subacute combined degeneration, and normal pressure hydrocephalus. Special care should be taken in patients with both upper and lower motor neuron signs, as amyotrophic lateral sclerosis and CSM can be difficult to distinguish.

Surgical decompression of the cervical spinal cord will be recommended by most neurosurgeons in the setting of any signs of myelopathy and significant cervical canal stenosis. Deficits acquired by patients with CSM are rarely completely corrected by surgery, so most surgeons will tend to offer decompression as early as possible. In patients with significant cervical stenosis without signs or symptoms of myelopathy operative indications are less clear. The role of fusion in the treatment of CSM is debated, and is beyond the scope of this chapter.

Surgical results in the large series available suggest that in 75-90% of cases the myelopathy can be stabilized or improved. The incidence of worsening of myelopathy with surgery is low (<1%). Other complications are approach- related and the reader is referred to the suggested readings.