Cervical Radiculopathy: Diagnosis and Nonoperative Management

Marc J. Levine, MD, Todd J. Albert, MD, and Michael D. Smith, MD

Abstract

Cervical radiculopathy presents as pain in a dermatomal distribution. This frequently represents compression of an exiting cervical nerve root by either a herniated disk or a degenerative cervical spondylotic change. Most patients will improve with nonoperative treatment, and a small percentage will require further diagnostic evaluation and ultimately surgical intervention. An understanding of the normal anatomy and the pathologic changes in cervical radiculopathy will improve the understanding of diagnosis and decision making regarding nonoperative interventions. An algorithmic approach for decision making and a review of nonoperative management are presented.

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Cervical radiculopathy is a clinical diagnosis based on a sclerotomal distribution of motor and/or sensory changes or complaints. Any process that causes impingement of exiting cervical nerve roots can lead to a radicular disorder. Impingement may be brought about by acute pathologic changes or by degenerative changes consistent with cervical spondylosis. Retropulsed disk material, zygoapophyseal joint hypertrophy, neurocentral joint hypertrophy, and other soft-tissue abnormalities all may cause compression of an exiting nerve root. Chemical irritation of the nerve root due to neurohumoral factors has also been described.

The accurate diagnosis of cervical radiculopathy begins with a history and physical examination. An appreciation of cervical spine anatomy and the pathophysiology of radicular pain will greatly enhance the diagnostic skills of the physician. Differentiating between radiculopathy and peripheral nerve compression is a common diagnostic dilemma. Once a preliminary diagnosis has been made, appropriate imaging modalities should be utilized to determine the source of impingement. If any question remains as to the cause of sclerotomal changes, nerve-conduction studies may be useful. This review will address these issues as well as the nonoperative management of cervical radiculopathy.

Epidemiology

In a recent study,[1] the records of over 550 patients seen between 1976 and 1990 with complaints of radiculopathy (average follow-up period of almost 5 years) were reviewed. The average annual incidence of cervical radiculopathy in this population in Rochester, Minnesota, was estimated to be 83.2 cases per 100,000 population, with a higher rate for males than females. An age-specific peak (202.9 cases per 100,000) was seen in individuals aged 50 to 54 years. The radiculopathy was caused by a confirmed disk protrusion in 21.9% of the patients; spondylosis, with or without disk herniation, was responsible in 68.4%. Forty-one percent of the patients had a previous history of lumbar radiculopathy. The most common presentation was C7 monoradiculopathy, followed by C6 monoradiculopathy. During the 4.9-year period of surveillance, 31.7% of the patients had a recurrence of the condition, and roughly 25% of the symptomatic cohort underwent surgery. At last evaluation, 90% of the patients who were not treated surgically were asymptomatic.

Dr. Levine is in private practice in St. Petersburg, Fla. Dr. Albert is Assistant Professor of Orthopaedic Surgery, Thomas Jefferson University, and Attending Surgeon, The Rothman Institute, Philadelphia. Dr. Smith is Attending Surgeon, Minnesota Spine Center, Minneapolis.

Reprint requests: Dr. Albert, The Rothman Institute, 800 Spruce Street, Philadelphia, PA 19107.

Anatomy

Cervical radiculopathy is largely secondary to mechanical compression of exiting nerve roots. An appreciation of both the osseous anatomy and the neuroanatomy in this region enhances diagnostic and therapeutic acumen. This brief overview is restricted to the subaxial cervical spine.

Each subaxial cervical spine motion segment consists of five articulations (Fig. 1). Anteriorly, the intervertebral disk functions as a joint, allowing motion in multiple planes. Two neurocentral (uncovertebral) joints lie along the posterolateral aspect of the vertebral body and provide articulation through osseous projections extending to the vertebral body above. These joints of Luschka lie between the disk and the nerve-root canal. The facet joints located posteriorly are angled 30 to 50 degrees to the transverse plane.[2]

The intervertebral foramina are bounded anteriorly by the vertebral body, the uncinate process, and the disk; posteriorly by the facet joints; and cranially and caudally by the pedicles. The subaxial cervical foramina are approximately 9 to 12 mm in height and 4 to 6 mm in width. The foramina are aligned obliquely 45 degrees to the sagittal plane.[3]

Projecting laterally from each vertebral body is a rudimentary rib, or costal process, that ends in the anterior tubercle. An embryologic transverse process extends from the lateral masses to the posterior tubercle. These two osseous elements fuse laterally to form the true transverse process of the cervical spine. A groove, known as the costotransverse lamella, transmits the ventral ramus of each exiting spinal nerve and is bounded posteriorly by the transverse process and the posterior tubercle and anteriorly by the vertebral artery and the anterior tubercle. The facet joint and the base of the lamina constitute the lateral boundary, and the vertebral body is the medial boundary. Because of the relationship of these structures, osteophyte formation, cervical instability, disk protrusion, or congenital deformities may lead to compression and subsequent radiculopathy.

The neuroanatomy of the cervical spine is unique to this region and is unlike that of either the thoracic or the lumbar spine. Each cervical root exits above the pedicle for which it is named except C8, which exits above the T1 pedicle. For instance, the C3-4 disk space or foramen transmits the C4 nerve root.

The ventral (anterior) motor nerve root consists of six to eight nerve rootlets exiting the spinal cord. The dorsal (posterior) sensory nerve root consists of six to eight nerve rootlets entering the spinal cord. The two unite to form the cervical spinal nerve root, which passes at an angle of 45 degrees to the coronal plane and inferiorly at 10 degrees to the axial plane. The cervical nerve root then passes directly laterally to the corresponding cervical disk and over the corresponding pedicle to enter the neuroforamen. The ventral motor nerve root lies anteroinferiorly in close proximity to the uncovertebral joint. The dorsal sensory nerve root lies near the superior articular process. As the nerve root enters the neuroforamen, it is located medially at the level of the tip of the superior articular process. It then courses laterally and inferiorly (Fig. 2). In the distal aspect of the neuroforamen, the cervical nerve root (both the anterior and posterior portions) forms the dorsal root ganglion. Just distal to the dorsal root ganglion and outside the neuroforamina, the anterior and posterior nerve roots join to form the spinal nerve. The spinal nerve then divides into ventral and dorsal rami. As mentioned previously, the costotransverse lamella transmits the ventral ramus.

In the normal spine of a young person, the cervical nerve root occupies approximately one third of the available space in the neuroforamen. This proportion may increase with age and degenerative changes. In addition, this proportion may increase in an extended neck because of the relative decrease in foraminal size in this position.[4] (Henceforth, the terms "cervical nerve" and "cervical root"

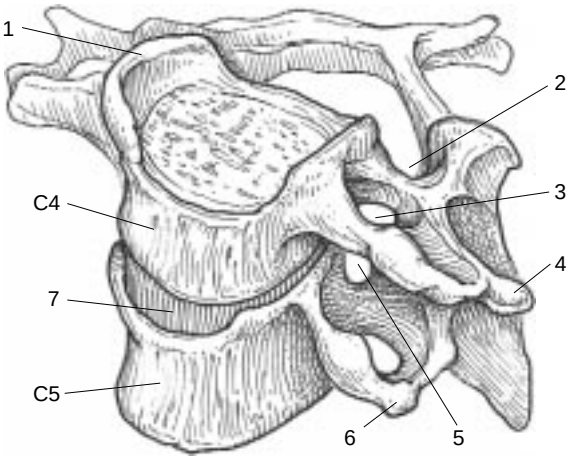

Fig. 1 Superior oblique view of C4 and C5. 1 = uncinate process; 2 = superior intervertebral notch; 3 = foramen transversarium; 4 = posterior tubercle of transverse process; 5 = spinal nerve foramen; 6 = anterior tubercle of transverse process; 7 = C4-5 disk.

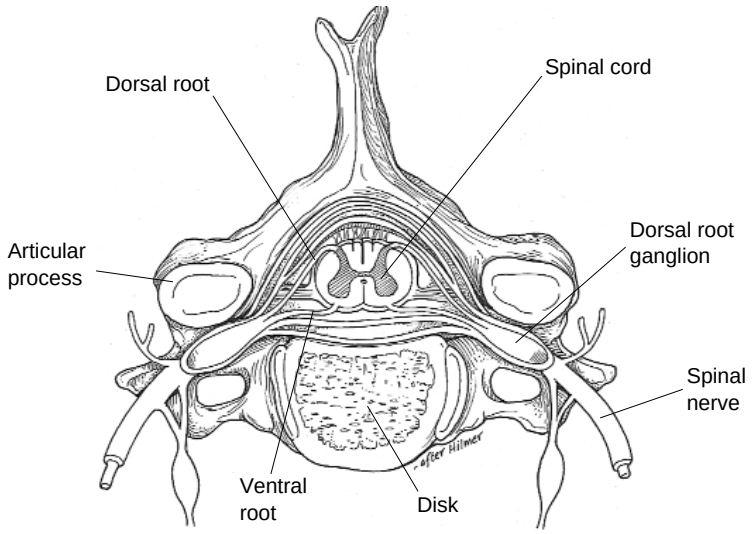

Fig. 2 Cross-sectional view of the neural structures of the cervical spine.

will be used to mean the spinal nerve at the cervical level unless otherwise specified.)

Pathophysiology

The mechanical nature in which cervical nerves become compressed has been well studied both clinically and radiographically. However, the mechanism by which this compression elicits pain is, as yet, poorly understood. Cornefjord et al[5] used a pig model to study the effects of chronic nerve root and dorsal ganglia compression. The concentrations of the neuropeptides substance P and substance VIP were measured in compressed roots. The authors found a significantly increased concentration of substance P in compressed nerve roots after 1 week of compression but not after 4 weeks. Numerous other chemical mediators of pain have been implicated as contributing to radicular neck pain. These chemical mediators are largely involved in the inflammatory response to compression.[6]

The vascular response to compression was studied by Olmarker et al[7,8] in a porcine model involving the cauda equina. These investigators found that blood flow to some venules stopped with 5 to 10 mm Hg of pressure, although the venular occlusion pressure ranged from 5 to 60 mm Hg. The study also suggested that intraneural edema occurred more readily in nerve roots than in peripheral nerves. In addition, edema formation was more pronounced in nerves that were rapidly loaded than in nerves exposed to a slower rate of compression. Similarly, there was a more profound effect on nutritional status in roots loaded rapidly than in those loaded more slowly.

Natural History

Although the natural history of cervical radiculopathy has not been as well studied as that of lumbar radiculopathy, it has been estimated that slightly more than half of the adult population will experience neck and radicular symptoms at some time during their lifetime.[9,10] Lees and Turner[11] reviewed the natural history of cervical radiculopathy and found that the condition rarely progressed to a myelopathic state. In patients treated nonoperatively, however, long-term follow-up revealed persistent symptoms in 66% of the population. In two other studies,[12,13] 23% of the patients with persistent neck or radicular pain were unable to return to their original occupation. It is unclear from these studies whether there were specific variations between the treatment received by the patients who were able to return to work and the treatment received by those who were not.

History and Physical Examination

The presentation of cervical radiculopathy varies greatly among patients. Presenting complaints can include pain, paresthesias, and motor weakness in different combinations and proportions. Classically, most patients complain of significant radicular pain and referred trapezial and periscapular pain.[13] Sensory disturbances associated with nerve compression often do not follow a strict dermatomal pattern. In fact, in a review of the data on more than 840 patients, Henderson et al[14] found that only 55% of patients with a nerve-root compression had pain in a strictly radicular pattern. Other studies have shown that motor deficits are present in 60% to 70% of patients with radiculopathy and that roughly 70% have reflex changes.[15]

Patients often describe symptoms that correlate with various head positions. Many will find relief with decreased neck motion when pain is due to acute cervical-

disk compression. Patients often report exacerbation of symptoms with neck hyperextension, particularly when the head is tilted toward the affected extremity. This position decreases the size of the neuroforamina. Conversely, patients report relief with a slight amount of neck flexion.

A number of maneuvers can be used during physical examination to support the diagnosis of cervical radiculopathy. As already described, a combination of neck extension and head tilt toward the affected limb (the modified Spurling maneuver) may reproduce radicular complaints, as can a Valsalva maneuver. The shoulder abduction relief sign, as described by Davidson et al,[16] has been shown to be specific for radiculopathy caused by soft disk herniations.[17]

A confusing presentation complex may arise in men or women who complain of breast pain while being evaluated for breast lesions or anginal symptoms. In one study,[18] 18 women had breast pain that was relieved with therapeutic measures directed at documented C6 and C7 radiculopathies. Breast pain is a much less common primary presenting complaint, but should be considered when other manifestations of cervical radiculopathy are absent. Cardiac workups have also been performed for this radiculopathic presentation of "cervical angina." Brodsky[19] reviewed the data on 438 patients with such symptoms, 88 of whom he had treated surgically.

Full descriptions of the various nerve-specific radicular symptoms are available in many textbooks, but a brief presentation of typical complaints for specific nerve roots (Fig. 3) will be presented here. Radiculopathies above C2 are extremely rare. They can cause jaw pain and occipital headaches, but no motor deficit is seen.

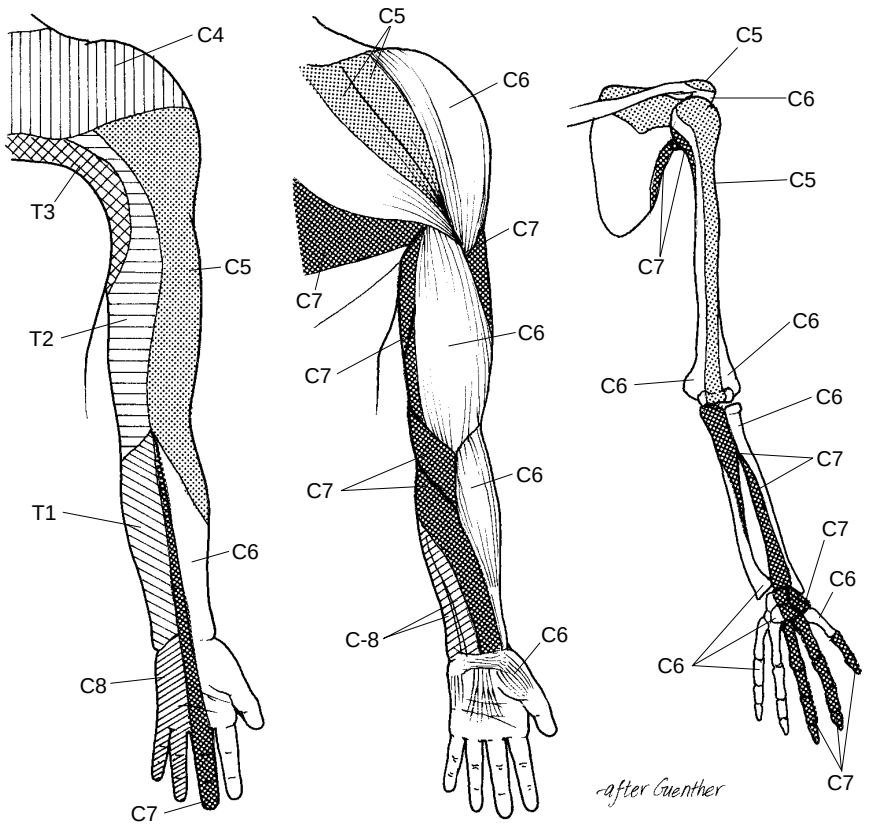

Fig. 3 Radicular patterns of distribution for the C4 through T3 dermatomes.

C3 radiculopathy, most often caused by disk disease at C2-3, is not common. Presenting complaints include headaches and pain along the posterior aspect of the neck that extends to the posterior occipital region and occasionally to the ear. There are no motor deficits, which may make differentiation from tension headaches difficult.

Disk protrusions at C3-4 typically involve the C4 spinal nerve root and most commonly present with neck and trapezial pain. There are no motor deficits, and diaphragmatic involvement has not been well documented. Patients occasionally complain of numbness and pain at the base of the neck that extends to the shoulder and scapular region.

Compression of the C5 nerve root classically produces pain and/or numbness in an "epaulet" pattern that includes the superior aspect of the shoulders and the lateral aspect of the upper arm. Deltoid motor function is often weakened, as in an intrinsic shoulder disorder; the diagnosis of radiculopathy at this site is crystallized by observing the absence of impingement signs or pain with passive shoulder motion. Patients may complain of difficulties with activities of daily living if there is involvement of the supraspinatus, infraspinatus, or elbow flexors. Depression of the biceps reflex is an inconsistent finding.

C6 involvement may present as pain or sensory abnormalities

extending from the neck to the biceps region, down the lateral aspect of the forearm to the dorsal surface of the hand, between the thumb and index finger, and including the tips of these fingers. The brachioradialis reflex may be depressed, and wrist extensor weakness is usually present. The infraspinatus, serratus anterior, triceps, supinator, and extensor pollicis muscles may also be affected.

The C7 nerve root is commonly involved. Pain and sensory abnormalities extend down the posterior aspect of the arm and the posterolateral aspect of the forearm and typically involve the middle finger, which is rarely affected in C6 disorders. Absence of the triceps reflex is common, and triceps weakness is almost always present. The wrist flexors, wrist pronators, finger extensors, and latissimus dorsi may also be affected.

C8 radiculopathy is least likely to be associated with pain. Sensory changes are usually restricted to below the wrist; motor involvement of the interossei makes differentiation from ulnar neuropathies and intrinsic hand disorders difficult. The rarity of C8 radiculopathy should make one search persistently to rule out an intrinsic hand problem or compressive neuropathy as the cause of ulnar symptoms. Myelopathy must also be ruled out.

Cervical Disk Herniation and Degenerative Spondylosis

Acute radiculopathies are typically secondary to disk herniations. More insidious symptoms occur as a result of degenerative changes. Acute disk herniation is more common in the younger population and is referred to as a soft disk herniation. Three types of soft disk herniation have been described by Stookey[20] and by Rothman and Marvel[21] (Fig. 4). Intraforaminal herniation is the most common and is often evidenced by radicular symptoms in a dermatomal distribution. Posterolateral herniation results in predominantly motor symptoms, including weakness and atrophy. Midline herniation may result in myelopathy.

Unlike the lumbar spine, where three joints are involved in each motion segment, in the cervical spine each motion segment involves five articulations. The term "cervical spondylosis" describes the continuum of degenerative changes that involve the disk, the two neurocentral joints, and the two facet joints. Uncovertebral osteophytes cause radiculopathy by compressing the nerve root anteriorly. Less commonly, osteophytes extending from the ventral portion of the superior articular process can cause compression by neuroforaminal narrowing. Neuroforaminal narrowing also occurs as a result of degenerative disk disease and the associated decrease in disk height.[22,23] The term "hard disk" is used to describe osteophytes that arise due to degenerative spondylosis and that may compress the spinal cord or nerve root. The remainder of this article will address radiculopathies caused by soft and hard disks.

Differential Diagnosis

Clinical evaluation requires the physician to rule out a number of processes that can mimic cervical radiculopathy. While the management and etiology of these disorders are beyond the scope of this discussion, we will present a brief overview and discuss pertinent differences on clinical examination.

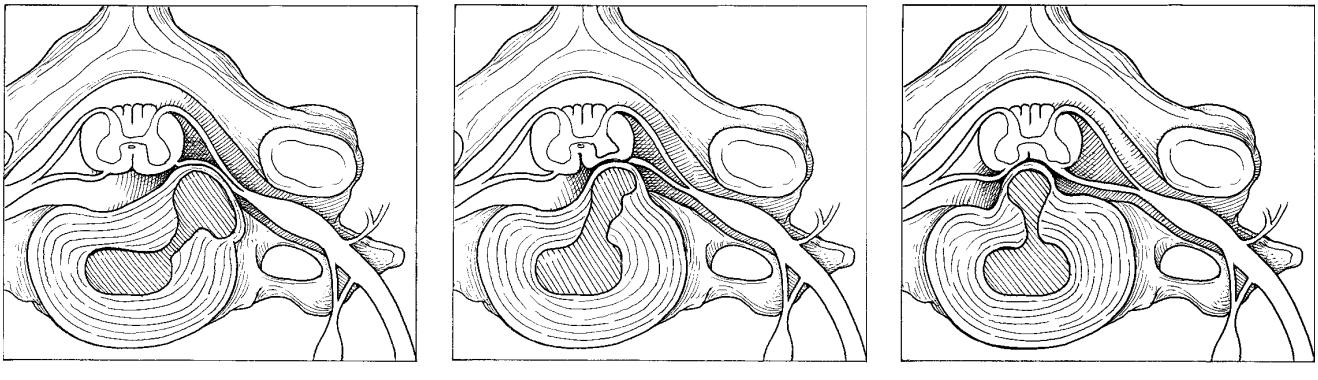

Fig. 4 Types of soft disk herniation. Intraforaminal **(A),** posterolateral **(B),** and midline **(C)** protrusions are usually associated with different clinical presentations.

Myelopathy

Cervical myelopathy is a distinct entity from both a diagnostic and a treatment perspective. Patients with cervical myelopathy may present with complaints of bowel and bladder changes, sexual dysfunction, gait disturbance, and difficulty with fine-motor function of the hand (writing, buttoning, handling change). Clinical examination is remarkable for upper motor neuron findings including, but not limited to, crossed and inverted radial reflexes, clonus, a Babinski sign, and a finger-release sign (Hoffman sign). While peripheral radiculopathic symptoms may accompany myelopathy, the presence of upper motor neuron changes requires a different treatment protocol. It must be remembered that a small number of patients with myelopathy may present with only hand dysfunction as an initial complaint.

Entrapment Syndromes

A number of entrapment syndromes can mimic cervical radiculopathy. Diabetes, smoking, alcohol consumption, rheumatoid arthritis, and hypothyroidism are generally considered risk factors for peripheral nerve entrapment.[24]

Peripheral median neuropathies may be confused with cervical radiculopathies. The pronator syndrome may mimic a C6-C7 radiculopathy with sensory involvement of the radial three and a half fingers and median nerve–innervated muscles. The muscles affected in the pronator syndrome include the pronator teres and the flexor carpi radialis; the radial nerve–innervated muscles of the C6 and C7 dermatomes (wrist extensors and triceps) are spared.

Entrapment of the anterior interosseous nerve (a motor branch of the median nerve) may present with pain in the proximal forearm and weakness of the flexor pollicis longus, pronator quadratus, and flexor digitorum profundus of the index finger, but no sensory deficits. These findings may be confused with those of C8 radiculopathy except for the absence of a sensory deficit. From a motor standpoint, true C8 radiculopathies are characterized by weakness in all ulnar nerve–innervated muscles.

The classic carpal tunnel syndrome also mimics a C6-C7 radiculopathy from a sensory standpoint. However, the triceps and wrist extensor muscles are not weakened, as they are innervated above the carpal tunnel. The thenar motor weakness associated with carpal tunnel syndrome may suggest T1 radiculopathy except that other T1 nerve–innervated muscles, including the hypothenar and ulnar nerve–innervated dorsal interosseous muscles, are normal.

Diagnosis of entrapment of a palmar cutaneous nerve is based on the absence of motor deficits despite sensory changes in the C6 distribution. Electrodiagnosis (measurement of nerve-conduction velocities) is useful in identifying a peripheral neuropathy.

Diagnostically, peripheral ulnar neuropathies can also be difficult to differentiate from true radiculopathies. Cubital tunnel syndrome typically presents with weakness of ulnar nerve–innervated muscles distal to the elbow, with corresponding sensory changes, and may be confused with a C8 or T1 radiculopathy. The flexor carpi ulnaris, flexor digitorum profundus to the ring and little fingers, interosseous, and hypothenar muscles are affected in both this entrapment syndrome and C8 or T1 radiculopathy. Muscles that are not affected in cubital tunnel syndrome but are affected in C8 or T1 radiculopathy are the flexor pollicis longus, the thenar musculature, and the median nerve flexors to the index and long fingers. Compression of the ulnar nerve in Guyon's canal typically affects the superficial and deep branches, leading to a sensory deficit along the volar portion of the ulnar one and a half digits. Dorsal sensation in these digits remains normal in this syndrome because the nerves to this region do not pass through the canal. Motor deficits are consistent with involvement of the muscles of the deep motor branch of the ulnar nerve.

A true C8 or T1 radiculopathy is characterized by sensory disturbances on both the volar and the dorsal surface and causes motor deficits of median nerve–innervated muscles, such as the T1-dependent thenar muscles. Remembering that C8 and T1 radiculopathies are remarkably rare and using electrodiagnosis judiciously should ensure the appropriate diagnosis of a peripheral compression syndrome.

The radial nerve is commonly compressed at the elbow by a number of structures. Usually, only the motor branch (posterior interosseous nerve) is involved, affecting the extensor digitorum communis, extensor carpi ulnaris, abductor pollicis longus, and extensor pollicis longus. This is also consistent with C7 radiculopathy, but unlike that condition, there is no sensory change and no involvement of the triceps or wrist flexor musculature.

Thoracic Outlet Syndrome

Symptoms of thoracic outlet syndrome often involve the contributions of the lower cervical roots to the brachial plexus and present as changes in median and ulnar distribution. Either vascular or neurogenic causation is possible. On physical examination, the presence of vascular bruits, asymmetric

pulses, or thenar muscle wasting greater than interosseous muscle wasting is more consistent with thoracic outlet syndrome than radiculopathy. Radiographs showing cervical ribs also implicate thoracic outlet syndrome rather than C8 or T1 radiculopathy.

Other disease processes that can mimic cervical radiculopathy include reflex sympathetic dystrophy, herpes zoster, brachial neuritis, and rotator cuff and shoulder girdle injuries.[21]

Other Causes of Compression

While most nontraumatic cervical radiculopathies are caused by acute disk disease and degenerative changes, there are other, less common causes of compression. A thorough history and physical examination and the judicious use of imaging modalities can expedite effective diagnosis and treatment.

Both intraspinal and extraspinal tumors can cause radicular complaints by direct compression or secondary to structural collapse of bone elements. In general, most malignant tumors will cause myelopathic symptoms bilaterally. Unilateral radicular changes may be seen with osteochondromas extending from the posterior elements. Schwannomas that arise from the nerve sheath also cause unilateral radiculopathies, which often progress to myelopathy. These lesions are more commonly intradural and may be exacerbated by the Valsalva maneuver. Extraspinal radiculopathies may be caused by direct extension of thyroid, esophageal, and pharyngeal tumors. Pancoast tumors have eroded through the pedicles of C7 and T1, causing C8 radiculopathies. Cervical radiculopathies have also been caused by soft-tissue compression secondary to sarcoidosis and arteriovenous malformations.

Evaluation

An algorithmic approach is justified for the logical workup and treatment of cervical radiculopathy (Fig. 5).

Imaging Modalities

The role of plain radiography is somewhat limited in the evaluation of the nerve roots. It remains an important initial study to rule out instability or pathologic changes in the bone. Oblique views of the cervical spine can show narrowing of the neuroforamina secondary to degenerative changes. Cervical instability may be visualized with dynamic flexion and extension films.

Plain radiography may be used as an initial study for evaluating neck pain associated with radiculopathy as long as the sensitivity and specificity of this test are understood. Friedenberg and Miller[25] showed that by the fifth decade 25% of their asymptomatic patients had evidence of degenerative changes; by the seventh decade, this number rose to 75%. When 92 asymptomatic patients were compared with a group of matched symptomatic patients, the only radiographic difference was a higher rate of degenerative changes at the C5-6 and C6-7 spaces in the symptomatic population. Plain radiographs should be obtained only after conservative management for 4 to 6 weeks has failed.

Magnetic resonance (MR) imaging has had a significant impact on the radiographic evaluation of cervical radiculopathy. Soft-tissue visualization is unsurpassed by that possible with any other modality. The physician should recognize that visualization of foraminal stenosis may not be appreciated as well on this study as on computed tomography (CT) performed with or without a contrast medium.

An MR imaging study should, at a minimum, include both T1- and T2-weighted sequences. The T1 sagittal examination provides an excellent survey of the cervical spine, which is another valuable asset of this modality. The axial T1 images provide insight into both intraspinal and extraspinal disorders, as well as the intrathecal nerve root anatomy. On T1 images, a hypointense signal is common for herniated degenerative disks, calcified ligaments, and bone spurs, making differentiation of these structures more difficult. The T2-weighted sequence or variants thereof may provide a "myelographic" view of the cervical spine. Unlike MR imaging of the lumbar spine, imaging of the cervical spine may be less accurate because of problems with motion artifact. Other sequences (proton-density and fat-suppressed fast spin-echo images) may add further information.

Numerous studies have compared the accuracy of myelography, CT (with and without contrast-material enhancement), and MR imaging.[26,27] Modic et al[28] prospectively compared the accuracy of MR imaging, myelography, and CT-myelography for evaluation of cervical radiculopathy. Magnetic resonance imaging was as sensitive as CT-myelography for identifying a diseased segment but was less accurate for identifying the exact disease process. The authors concluded that an MR imaging study accompanied by nonenhanced CT provides excellent visualization of the cervical spine. In a prospective study of 100 patients,[29] MR imaging was shown to be as accurate as postmyelography CT in the evaluation of cervical radiculopathy. In another prospective study, Neuhold et al[30] correlated MR imaging findings with intraoperative pathologic findings and

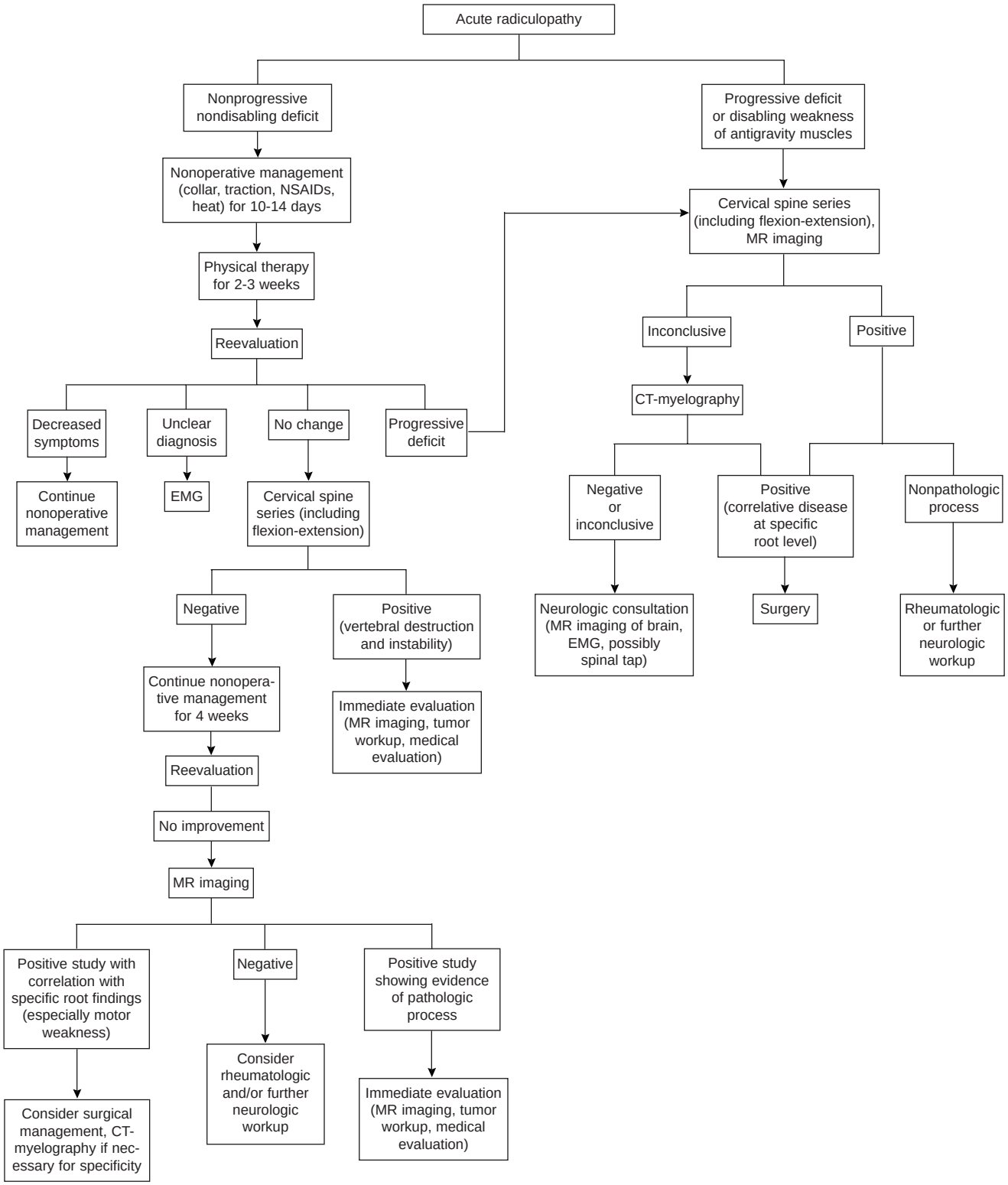

Fig. 5 Algorithm for temporal sequence of diagnosis and nonoperative management of acute cervical radiculopathy. CT = computed tomography; EMG = electromyography; MR = magnetic resonance; NSAIDs = nonsteroidal anti-inflammatory drugs.

found MR imaging to be a viable alternative to postmyelography CT. They suggested the use of MR imaging as the initial diagnostic modality when cervical disk disease is suspected.

A cervical myelogram outlines the spinal cord and exiting nerve roots with radiopaque dye. The study is performed by introducing dye into one of two areas. The water-soluble agent may be injected via the C1-2 interval, allowing the dye pool to gravitate caudally. This expeditious means of visualization may be complicated by the inherent risks of introducing a needle at this interspace. If dye is introduced into the lumbar region, the patient must be placed in a position that forces the dye pool cranially. Although needle placement is less critical in this region, a delay in visualization, dilution of the dye load for cervical imaging, and the risk of dye passing through the foramen magnum detract from the attractiveness of this protocol. As with all imaging modalities, clinical correlation is imperative in formulating a treatment plan. Hitselberger and Witten[31] reported a 21% incidence of cervical filling defects in 300 asymptomatic patients who underwent myelography for evaluation of acoustic tumors.

The accuracy of cervical myelography alone has been estimated to range from 67% to 92%.[26] For this reason, cervical myelography is often accompanied by CT. Axial images of dye-enhanced neural elements offer excellent visualization of nerves in relation to surrounding osseous structures. The combination of myelography and postmyelography CT provides important details required for preoperative planning after failure of nonoperative management.

In summary, we believe that after the initial observation period of 4 to 6 weeks, plain radiography, including flexion and extension views, should be the initial diagnostic imaging modality used to evaluate the possibility of cervical radiculopathy and to rule out instability or pathologic changes in osseous structures. If the clinical findings support a diagnosis of cervical radiculopathy due to nontraumatic soft disk or hard disk changes, MR imaging should be performed. If surgical treatment becomes necessary and the MR images are nonspecific (especially in a case of multilevel spondylosis), myelography and postmyelography CT can be pursued for greater specificity.

Electrodiagnosis

Nerve compression may lead to motor, sensory, and/or autonomic changes. In cases of polyradiculopathy or difficult clinical diagnoses, the use of modalities that utilize electrical stimulation may be necessary to help differentiate radiculopathy from peripheral compression syndromes.

The usefulness of electromyography (EMG) and nerve-conduction velocity studies is dependent on their ability to detect motor changes occurring as a result of nerve compression. In radiculopathy, abnormalities in sensory-nerve action potentials (SNAPs) are uncommon. Typically, compression that leads to cervical radiculopathy occurs proximal to the dorsal root (sensory) ganglion. Unless the dorsal root ganglion at the distalmost aspect of the neuroforamen is involved, the SNAPs will remain normal. In the case of a compressive brachial plexopathy, the SNAPs are routinely abnormal unilaterally because the encroachment is distal to the sensory ganglion. Bilateral SNAP changes are suggestive of peripheral polyneuropathy.

Compound-muscle action potentials show a decrease in amplitude proportional to muscle atrophy. Significant alterations may be seen in polyradiculopathies with multiple muscle involvement. Dramatic changes are more commonly seen in lumbar stenosis but may occur in cases of severe cervical spondylosis.

Nerve-conduction velocity and latency changes are not typically found in cervical radiculopathies unless there is extreme demyelination of axons. Because the lesion is proximal to the region tested, the usefulness of peripherally oriented studies is limited.[32]

Another alternative for electrically evaluating cervical radiculopathies is cervical root stimulation (CRS). With this technique, cervical roots are stimulated by placing monopolar needles in the paraspinal muscles, and compound-muscle action potentials are recorded in the biceps, triceps, and abductor digiti minimi muscles.

Electromyography has historically been the modality of choice for differentiating cervical radiculopathies from more peripheral disturbances. Electromyographic changes represent a continuum that begins with a decrease in motor-unit potentials and progresses to fibrillation potentials of multiple muscles. Many of the changes seen with chronic radiculopathies are not unique to radiculopathy and require careful interpretation. Electromyography has been shown to correlate better with clinical symptoms than does plain radiography. In a retrospective review of 108 patients,[33] the disk height and neuroforaminal size were of little use in predicting clinical findings, in contrast to EMG.

The literature currently favors CRS over EMG for accurate differentiation of cervical radiculopathy. Berger et al[34] compared CRS with conventional EMG and evaluation of nerve conduction and late

responses in 34 patients. Of the 18 patients with clinical evidence of radiculopathy, 11 had abnormal EMG studies, and all 18 had abnormal responses to CRS. Of the 16 patients with symptoms but no signs of radiculopathy, 5 had abnormal EMG studies, and 9 had abnormal responses to CRS.

In a more recent study,[35] the authors compared the CRS, EMG, and nerve-conduction velocity findings in 32 patients with both clinical signs and symptoms suggestive of cervical radiculopathy. Conventional EMG was positive in a little over half of the patients, while CRS was abnormal in more than 75%. The CRS study was positive in 25 patients. Thirteen of the 25 patients subsequently underwent surgery, which documented intraoperative findings consistent with radiculopathy. Only 10 of the 13 had a positive EMG study.

Overall, the role of electrodiagnostic testing is to assist in difficult diagnostic situations and to rule out peripheral neuropathies, but not to be an additional test for confirmation of a clear monoradiculopathy, which is a clinical diagnosis. Whether the physician chooses EMG or CRS, the temporal sequence remains constant. These studies should follow plain radiography and a period of conservative management and should precede more complex imaging modalities. In practice, we rarely order electrodiagnostic testing in a workup other than for patients with unusual presentations or diabetes or to rule out a peripheral compression syndrome.

Nonoperative Management

Initial nonoperative management is appropriate in almost all cases of cervical radiculopathy caused by soft or hard disks. The exception to this generalization is a progressing neurologic deficit or a deficit that disables the patient (severe deltoid or wrist extensor weakness). Compression secondary to trauma, infection, tumor, or other pathologic changes in the soft tissues is excluded from this discussion. The efficacy of treatment modalities is often related to the pathophysiologic processes that cause the pain. The acuity of the presenting symptoms is a factor in the selection of appropriate treatment protocols.

Acute neck pain due to cervical radiculopathy can be treated initially with a short course of cervical immobilization in a soft collar. Patients with acute pain typically present to their physician within 2 weeks after the onset of symptoms.[36] Immobilization serves to decrease the acute inflammatory response and helps to decrease pain. Prolonged immobilization should be avoided, however, because the cervical musculature atrophies rapidly. The duration of immobilization should not exceed 10 days to 2 weeks and should be followed by gradual weaning. During the weaning period, the paraspinal muscles can be strengthened with isometric exercises.[37]

A review of the literature disclosed that the efficacy of soft collars in treating cervical radiculopathy is still unclear. While some have reported benefits from their use, others have found cervical collars of little value.[38-40] While many patients are comfortable with the higher part of the collar anteriorly, the extension this necessitates aggravates the condition in other patients, who prefer to reverse the collar to encourage neck flexion.

A very short course of bed rest can also serve as a form of cervical immobilization and has the benefit of eliminating axial forces caused by gravity. The inverted-V pillow arrangement can further relieve radicular symptoms. With this pillow arrangement, the head is flexed slightly, and the shoulders are internally rotated. The neck flexion serves to enlarge restricted neuroforamina, and the internal rotation of the shoulders decreases the stretching of the cervical nerves. This position may be specifically suited for patients with radicular symptoms.[41] There are commercially available cervical pillows that simulate the inverted-V pillow arrangement.

Home traction devices that attach to door frames have provided relief for some patients with radicular symptoms. Traction forces of 8 to 12 lb are generally applied for 15- to 20-minute periods. Theoretically, traction forces relieve pressure from compressed nerves. Some believe that traction may increase blood flow and decrease ischemia while flushing out inflammatory by-products. The angle of traction application has been studied by Colachis and Strohm.[42] The maximum intervertebral distance was achieved with traction forces applied at an angle of approximately 24 degrees of flexion. The application of traction should not be initiated until muscle spasms have been alleviated. This technique is usually contraindicated in patients with myelopathic or long-tract signs.[41] Care should be taken that traction does not hyperextend the neck, thereby compressing the foramina. A survey of the literature reveals conflicting reports regarding the benefits of cervical traction.[38,43]

The pharmaceutical management of cervical radiculopathy can be divided into three categories of medication. Narcotic analgesics can be used in the acute setting but should be used cautiously because of the addictive and depressive side effects. Drugs directed at muscle spasm also may serve a role

in the acute setting. Spasms occur as a result of increased muscle tension at insertion sites, which leads to avascularity and buildup of anaerobic metabolic by-products. [41] Antispasmodic agents disrupt the cycles of repetitive spasm.

Nonsteroidal anti-inflammatory drugs decrease pain brought about by the inflammatory process. These agents prevent the formation of various substances in the cyclooxygenase pathway, which have been implicated as contributing to the pain response in cervical radiculopathy. Systemic monitoring, particularly of the liver and the gastrointestinal system, is important with any long-term usage.

Oral corticosteroids are generally not recommended for cervical radiculopathies because of the associated risk factors. Olmarker et al[44] used a porcine model to explore the effects of intravenous administration of methylprednisolone on nucleus pulposus–induced nerve root injury. Their results suggested that high doses of this agent may reduce nerve root damage secondary to compression by disk material if administered in the first 24 to 48 hours.

Epidural administration of corticosteroids has been shown to be most beneficial in patients with both signs and symptoms of a radicular disorder.[45] Recommended injections include lidocaine and methylprednisolone acetate or triamcinolone diacetate. The anti-inflammatory effects of cervical epidural injections can be repeated with multiple injections; however, the risks of needle placement should be weighed.

Appropriate physical therapy protocols require a coordinated effort between the physician and the therapist. Patients with initial acute radicular symptoms may benefit from immobilization, followed by heat and cold therapy. Electrical stimulation applied by the therapist can help break spasm cycles. During the weaning period from use of a cervical collar, isometric neck-strengthening protocols are introduced. Stretching exercises can also be instituted at this time. If the patient is free of pain after 6 weeks, more aggressive exercise regimens can be introduced to build up the paraspinal muscles and protect the neck from further attacks.

It should be noted that none of the above-mentioned nonoperative approaches to managing cervical radiculopathy has been subjected to prospective randomized efficacy trials to gauge efficacy, nor have they been compared with observation alone. Until this occurs, we will continue to treat patients on the basis of available retrospective information and anecdotal experience.

Summary

Diagnosis of cervical radiculopathy requires the physician to appreciate not only the cervical anatomy but also the numerous disease processes that can mimic cervical radiculopathy. Initial plain radiographs are useful as a preliminary study to check for instability and gross structural change. Conservative management protocols should be started almost immediately in an effort to control the inflammatory process. Electrodiagnostic tests can be used in particularly confounding cases but should not be considered part of the routine workup. Imaging modalities are useful in confirming clinical diagnoses and identifying the cause of compression when initial nonoperative protocols fail. More aggressive therapy should be tailored to the patient's symptoms and the chronicity of pain. When nonoperative management is ineffectual or disabling weakness, progressing radiculopathy, or myelopathy is present, consideration of surgical intervention is warranted.

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