Chapter 49

Cervical Disk Disease

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Introduction

The somewhat vague term, cervical disk disease, most often is used to describe degenerative pathology involving the cervical disk itself as well as associated clinical symptoms. So-called cervical disk disease encompasses a broad clinical spectrum in which the symptoms range from minimal axial neck pain to symptoms characteristic of myelopathy. Between these two extremes, patients may have a combination of symptoms, depending on severity of the disease, including debilitating axial pain, loss of motion, radiculopathy, numbness, tingling, gait imbalance, and fine motor dysfunction. For the purpose of treatment decision making, three overlapping clinical categories can be used: axial neck pain, radiculopathy, and cervical spondylotic myelopathy.

Axial Neck Pain

Etiology

Axial neck pain is one of the most common initial symptoms related to the cervical spine. This condition often is self-limiting, and in almost all patients, it can be resolved with minimal intervention. Some patients will have persistent pain, however, and further evaluation and treatment will be required. Axial or referred pain can occur in the paraspinal musculature, the occipital or periorbital region, or the trapezial or interscapular areas. The presence of several potential pain generators in the neck region complicates the ability to provide an accurate and prompt diagnosis. The often vague and subjective nature of symptoms, the lack of physical examination findings, and the paucity of specific tests to diagnose the anatomic source of the axial pain mean that the evaluation can be difficult. Axial pain related to degenerative disease can stem from the disk, the facet joint, the atlantoaxial joint, and/or neurologic compression. Although radicular pain typically radiates into the arm, the most severe pain usually is proximal, and neck pain may be a significant component of radicular symptoms.

The cervical disk is composed of the outer anulus fibrosus and the nucleus pulposus, both of which undergo degeneration with aging. Within the intervertebral disk, metabolic changes, annular tearing, herniation of disk material, dehydration of the nucleus, and loss of disk height can occur. One theory locates the origin of pain in the disk itself, as a result of vascular or neurologic ingrowth into the anulus.[1] Other causes of axial pain probably are directly related to disk degeneration.

Facet joint pain results from spondylotic changes, the action of inflammatory mediators, and increased stress, which presumably occur with progressive degeneration of the intervertebral disk. As the anterior column of the cervical spine becomes less able to support the physiologic load, the posterior structures and facet joints undergo increased stress. With time, the joint can become hypertrophic, arthritic, and painful (**Figure 1**).

Arthritic changes involving the atlantoaxial joint are known to be a cause of axial pain[2] (**Figure 2**). Discomfort primarily occurs with rotation to the affected side, involves the occipitocervical region, and often radiates into the occiput. The diagnosis is easily missed because the focus may be on the subaxial spine.

Evaluation

A valid and reliable test to identify symptomatic degenerative disks remains elusive. Diskography is unreliable and inconsistent. Imaging, including MRI, has a significant false-positive rate because most of the degenerative changes shown on imaging studies are not the source of pain.[3] Conversely, facet joint injections are valuable for elucidating pain and can be guided by clinically distinct pain distributions.[4,5] These injections are both diagnostic and therapeutic.

Treatment

The key issue in treatment decision making is identifying the true pain generator. This task is difficult if the patient has axial pain only. Treatment should begin with nonsurgical intervention. Anti-inflammatory

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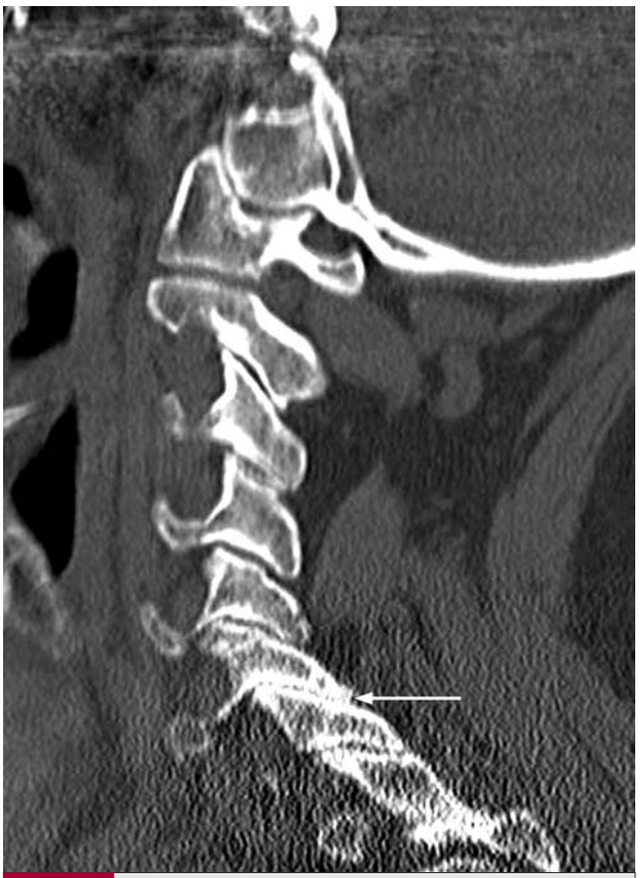

Figure 1 CT of the cervical spine showing significant facet joint narrowing and arthritic changes (arrow).

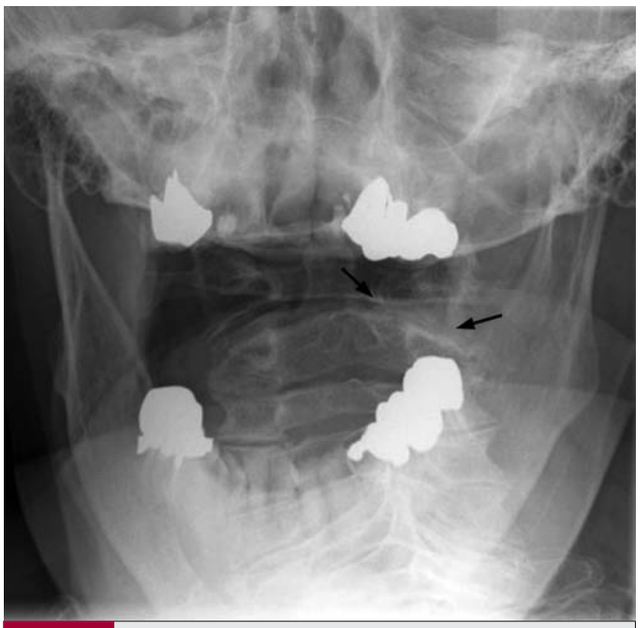

Figure 2 Open-mouth odontoid radiograph showing bilateral C1-C2 joints with unilateral arthritic changes (arrows).

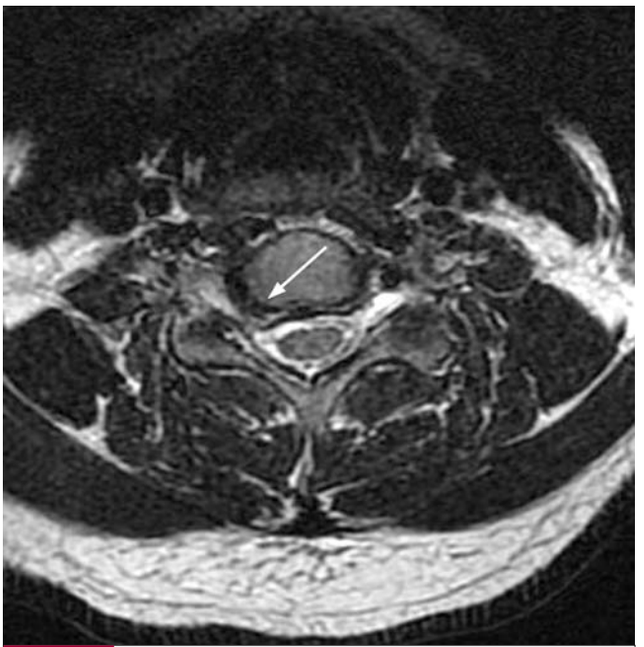

Figure 3 Axial T2-weighted MRI of the cervical spine showing a soft disk herniation causing significant neurologic impingement (arrow).

medication, activity modification, and a short course of immobilization are of potential benefit. Moderate-quality evidence supports the use of physical therapy for chronic cervical pain.[6] Injection of local anesthetic with or without a steroid can provide benefit after a pathologic diagnosis is made. Surgical fusion is the last recourse for treating axial neck pain. Although there is controversy and a lack of level I evidence, the surgical treatment of axial neck pain has provided clinical benefit.[7,8]

Radiculopathy

Etiology

Radiculopathy is caused by nerve root pathology with etiologies including mechanical compression, ischemia, and inflammation. The signs and symptoms of radiculopathy include pain in a dermatomal pattern, myotomal weakness, hyporeflexia, and paresthesias. The factors determining the response of the nerve are not entirely understood. However, it is well known that relieving the nerve from any offensive lesion often alleviates the radiculopathy. The nerve rarely sustains permanent damage.

Mechanical compression can occur directly in the presence of a soft or hard disk herniation, osteophyte, or facet capsule infolding, or it can occur indirectly through foraminal narrowing or instability (**Figures 3** and **4**). Ischemic changes can result from direct compression and possibly from inflammation of the root. Inflammation occurs as a result of exposure to disk material, joint irritation, or mechanical instability.

Evaluation

Detection of radicular pathology often is fairly straightforward and is based on a complete history, a detailed neurologic examination, and correlation with imaging and diagnostic studies. The history and physical examination are crucial. Classic dermatome distributions and provocative testing of muscles and reflexes involving the upper extremities are shown in Figures 5 and 6.

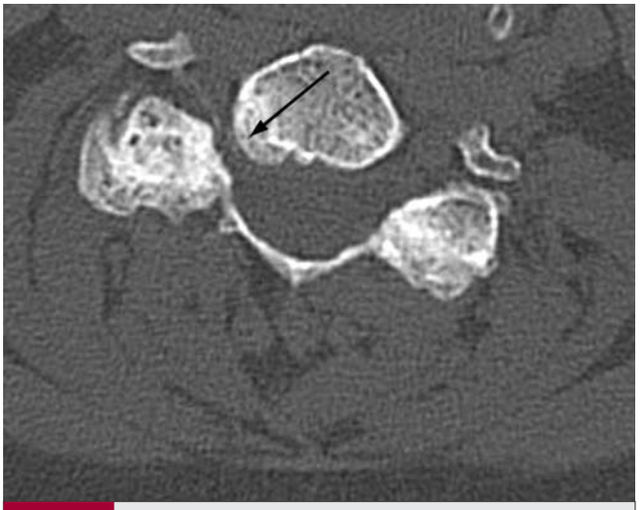

Figure 4 Axial CT of the cervical spine showing foraminal stenosis (arrow).

The Spurling maneuver, in which the patient extends the neck and rotates the head toward the side of typical pain, can help differentiate a true cervical root etiology from other potential sources of pain. In a positive Spurling sign, the maneuver re-creates or enhances the patient's typical pain. During the examination, the clinician must keep in mind the need to rule out other sources of upper extremity pain, such as the shoulder or a peripheral nerve.

The use of imaging studies (plain radiographs, MRI, and CT myelogram) and diagnostic selective nerve root injections should be primarily confirmatory in nature. Imaging studies may reveal the pathology, though the results can be equivocal or show multiple abnormalities. Selective nerve root injections can be used to precisely identify the symptomatic area if the imaging findings are equivocal.[9] The placement of the selective nerve root injection is guided by the history and examination findings. Electromyography and nerve conduction testing can be used to identify nonradicular pathology, such as peripheral neuropathy.

Treatment

Treatment should begin with nonsurgical measures because most cervical radicular pain will improve over time. During the acute phase, the patient can be given steroids, NSAIDs, and a short course of narcotic medication.[10] Typically, narcotic medications have a limited ability to control nerve-related pain and should be used sparingly. The key is to break the cycle of acute pain so

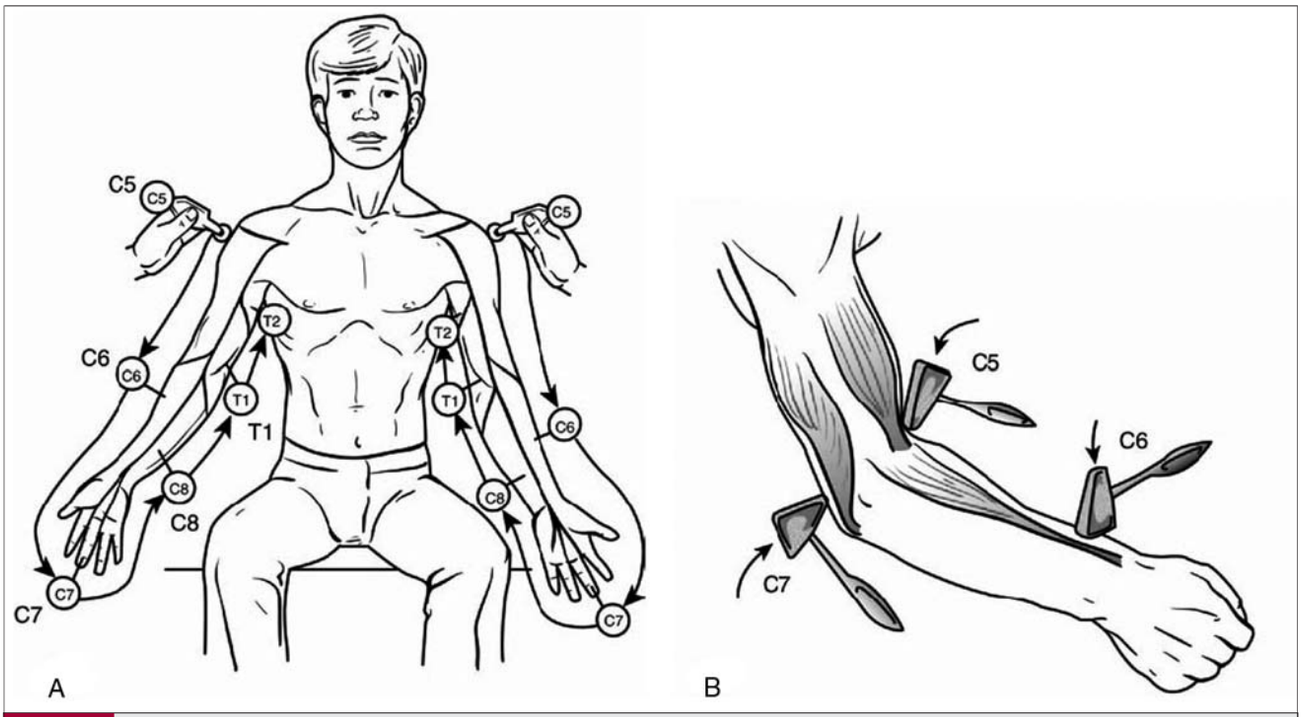

Figure 5 Schematic drawings showing the upper extremity dermatome distribution (**A**) and reflex examination (**B**). (Reproduced from Grauer NJ, Beiner JM, Albert TJ: Cervical disk disease, in Vaccaro AR, ed: *Orthopaedic Knowledge Update*, ed 8. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2005, pp 527-534.)

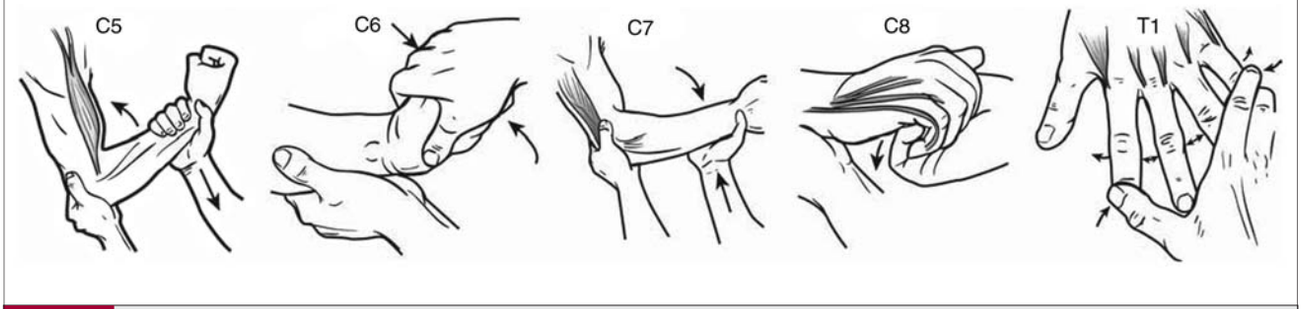

Figure 6 Schematic drawing showing the upper extremity motor examination. (Reproduced from Grauer NJ, Beiner JM, Albert TJ: Cervical disk disease, in Vaccaro AR, ed: *Orthopaedic Knowledge Update*, ed 8. Rosemont, IL, American Academy of Orthopaedic Surgeons, 2005, pp 527-534.)

that the patient can tolerate gradual healing of the radiculopathy, with resolution of paresthesias or weakness over a period of months. To avoid exacerbating the discomfort, a structured physical therapy program is delayed until the pain has been alleviated.

Steroids should be used sparingly, if at all, because of the potential adverse effects.[11] Although the use of oral steroids can quickly and dramatically diminish radicular pain, no good data have shown their long-term effectiveness for treating radicular symptoms. If the patient's symptoms are not relieved by oral medical therapy, a selective nerve root injection may be beneficial. The therapeutic benefit of selective nerve root injection is a topic of controversy, but it certainly has diagnostic value.[9] A patient who continues to have incapacitating nerve root pain, has undergone at least 6 weeks of nonsurgical treatment, or has a progressive neurologic deficit may be a candidate for surgical treatment.[12]

Several surgical options are available for treating radicular pathology. The goal is to remove the agent causing the radiculopathy. Standard anterior procedures include anterior cervical diskectomy and fusion (ACDF) and anterior cervical disk arthroplasty (ACDA).

ACDA is a relatively new surgical option for symptomatic cervical disk disease after unsuccessful nonsurgical treatment. Although long-term data are lacking, the early results are promising compared with those of ACDF. A 4-year follow-up study of ACDA using the Bryan Cervical Disc implant (Medtronic Sofamor Danek) found outcomes significantly superior to those of ACDF.[13] The measures included the Neck Disability Index, the Medical Outcomes Survey 36-Item Short Form physical component, scales for neck and arm pain, and overall success. The potential for long-term complications after ACDA, as related to durability and wear, requires further study.

Posterior pathology can be treated through laminoforaminotomy, which has been well described elsewhere.[14,15]

Cervical Spondylotic Myelopathy

Etiology

The all-encompassing term, myelopathy, is used to describe spinal cord dysfunction with its associated signs and symptoms. Myelopathy in cervical degenerative disease is believed to directly result from static or dynamic spinal cord compression. Spondylotic changes include loss of disk height, disk bulging or herniation, osteophyte development, pathology of the posterior longitudinal ligament, buckling of the ligamentum flavum, instability, and loss of lordosis. All of these anatomic and physiologic conditions can lead to compression of the spinal cord (**Figure 7**). Congenital narrowing of the spinal canal can predispose the spinal cord to compression. It is not entirely clear how compression of the spinal cord leads to clinical dysfunction. Theories include ischemia, inflammation, edema, gliosis, and demyelination.[16,17] It is most likely a combination of these factors that result in cervical spondylotic myelopathy.

Evaluation

The signs and symptoms of clinically diagnosed cervical spondylotic myelopathy depend on the severity of the disease, chronicity, levels of involvement, and factors that are not fully understood.[18] A thorough history and physical examination are crucial but may not be as straightforward as with radicular pathology.

Patients typically describe difficulty in hand and other fine motor functions, including writing and gait, as well as diffuse weakness or numbness. If the condition is severe, the patient may have disturbance in bowel and bladder function. Examination findings may include both upper and lower motor neuron abnormalities. Long tract signs include hyperreflexia, clonus, the Babinski sign, the Hoffman sign, the Lhermitte sign, and an inverted radial reflex. With concomitant nerve root compression, concurrent lower motor neuron findings may be seen in the upper extremities. These classic findings are completely absent in approximately one fifth of patients with cervical spondylotic myelopathy, however.

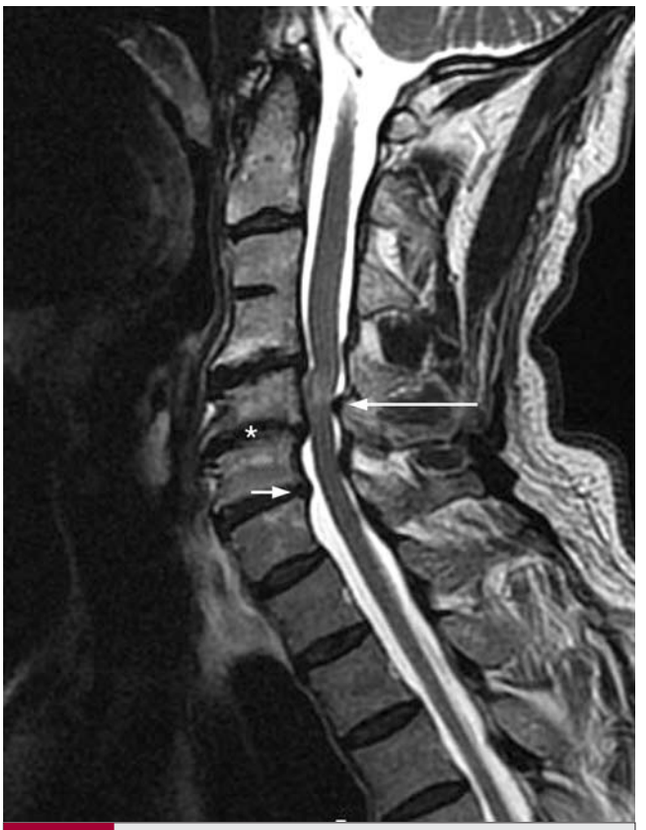

Figure 7 Sagittal T2-weighted MRI of the cervical spine showing spondylotic changes, including disk height collapse (*), disk bulging (small arrow), and buckling of the ligamentum flavum (large arrow), with intrinsic cord changes just anterior.

Plain radiographs, CT myelogram, and MRI are useful for diagnosing and evaluating pathology associated with cervical spondylotic myelopathy. Plain radiographs are used to assess gross spondylotic changes and overall alignment of the cervical spine. CT myelograms are superior for assessing bony pathology, such as osteophytes or ossification of the posterior longitudinal ligament, as well as indirect compression of the neurologic structures. MRI is superior for evaluating the neurologic structures (the spinal cord and nerve roots) and soft-tissue structures, such as the disk, ligamentum flavum, and posterior longitudinal ligament. MRI can be used to detect the extent and cause of compression as well as intrinsic changes within the spinal cord (**Figure 7**). Changes in signal intensity on T2-weighted (hyperintense) or T1-weighted (hypointense) sequences are believed to indicate cord pathology. There is no consensus on the meaning of these changes;however, theories include ischemia, inflammation, edema, gliosis, and demyelination.[16,17]

Electrodiagnostic studies are used to diagnose spinal cord dysfunction. Motor-evoked and sensory-evoked potentials can show abnormalities in central conduction patterns. Patients with subclinical cervical spondylotic myelopathy (with subtle or no physical signs) may have electrophysiologic changes. Motor-evoked potentials have proved to be more sensitive than sensory-evoked potentials.[19,20]

Treatment

The natural history of cervical spondylotic myelopathy is not well understood, although the disease appears to progress slowly over time with variable periods of quiescence and gradual stepwise decline.[21] Rapid neurologic decline is the exception and requires that the patient be treated with surgical decompression. There is much debate as to the best treatment of patients with subclinical myelopathy.

Nonsurgical treatment of cervical spondylotic myelopathy is limited to activity modification, anti-inflammatory medications, and an orthosis. These modalities do not affect the overall condition and may be detrimental to a patient's condition by masking further decline and leading to a delay in surgical treatment.[22]

Surgical treatment entails decompression of the neurologic elements. There is debate as to whether an anterior (ACDF, ACDA, corpectomy), a posterior (laminectomy, fusion laminoplasty), or a combined procedure is most efficacious for treating cervical spondylotic pathology.

An anterior procedure can directly treat anterior pathology and improve lordosis, with fusion of the involved levels providing stability to the spine and spinal cord. Stability is believed to provide an optimal environment for recovery of the neurologic elements; however, this belief may be unfounded. One indication for ACDA may be single-level myelopathy with cord compression caused by a large retrodiscal fragment. The choice of multilevel ACDF or corpectomy should be based on the location of the pathology as well as the patient's healing potential because multilevel ACDF requires fusion of more surfaces than corpectomy.

A posterior procedure can be used to treat posterior pathology directly. Lordosis can be partially corrected with fusion. Both fusion and laminoplasty decrease motion and thereby improve stability. A posterior cervical fusion clearly provides stability, and overall motion is decreased with laminoplasty, which is meant to indirectly decompress the spinal cord and preserve motion. Laminoplasty should be avoided if the cervical spine is kyphotic because the spinal cord will not drift posterior, and progression of the deformity and myelopathy will be allowed to continue. Lordosis or neutral alignment is a necessary prerequisite for a laminoplasty. A posterior laminectomy and decompression should routinely be accompanied by instrumentation and fusion to prevent postlaminectomy kyphosis.

Recent research found that surgical decompression led to significant improvement in the outcomes of patients with mild to severe cervical spondylotic myelopathy, with the greatest improvement in those who had severe myelopathy.[23] This research justifies a strong recommendation for surgical intervention to treat mild or moderate myelopathy and a definite recommendation to treat severe myelopathy.

Surgical Complications

Surgical treatment of cervical degenerative disk disease can lead to complications, the most common of which is dysphagia. Although dysphagia is more prevalent after an anterior procedure, it also can occur with a posterior procedure.[24] The reported incidence of dysphagia is as high as 50% during the first month after anterior surgery, typically with significant improvement over time.[25] The etiology of dysphagia is not entirely clear, but identifiable risk factors include extended length of surgery, wound retraction, a multilevel procedure, and high endotracheal cuff pressure. The assessment of patients with dysphagia is difficult because diagnostic criteria and measurement tools are poorly defined.[26,27] Patients undergoing surgery for cervical degenerative disk disease should be counseled to expect dysphagia, with gradual improvement over time.

Other complications include wound infection, nerve root palsy, epidural hematoma, retropharyngeal hematoma, cerebrospinal fluid leakage, blindness, instrumentation or graft failure, pseudarthrosis, instability, vascular injury, and neurologic injury. Medical comorbidities or cardiopulmonary complications can result from any surgical procedure. Most of these complications are rare, and often they are self-limiting and resolve with time.

Summary

Degenerative changes of the cervical spine present in many ways and vary from axial neck pain to severe myelopathy. Most symptoms related to cervical disk disease are self-limiting and benign; however, indicated surgical treatments result in high success rates. The key to a positive outcome is prompt diagnosis of the problem, development of a treatment plan, and sound execution. Fortunately, with adherence to these principles, complications rarely occur.

Key Study Points

- Atlantoaxial arthritis is an easily missed cause of axial neck pain.
- Arthroplasty using the Bryan Cervical Disc had significantly better outcomes than anterior cervical diskectomy and fusion.
- Cervical spondylotic myelopathy is a clinical diagnosis with a constellation of signs and symptoms, and surgical treatment often is required to halt its progression.

Annotated References

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   A literature review of randomized controlled studies of therapeutic exercise for adult neck pain found moderate evidence of symptom improvement with combined therapies specific to the cervicoscapular region, including stretching and strengthening. Nonspecific upper extremity therapy provided no benefit for neck pain.

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A multicenter prospective randomized study compared the Bryan artificial disk with fusion for treatment of cervical spondylosis. Early and midterm (48-month) results revealed significantly superior results after arthroplasty. Level of evidence: I.

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Cervical degenerative disease results in axial neck pain, radiculopathy, and myelopathy. The treatment of such disease involves nonsurgical and surgical means depending on the severity and symptoms. There are a multitude of successful surgical options according to the type of pathology, including decompression, fusion, laminoplasty, and arthroplasty.

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A prospective study followed 278 patients with mild, moderate, or severe cervical spondylotic myelopathy for 1 year after surgical decompression. Outcome measures included the modified Japanese Orthopaedic Association score, the Nurick Scale, the Neck Disability Index, and Medical Outcomes Study 36-Item Short Form (version 2). On all measures, there was a statistically significant improvement from baseline, and with the exception of the modified Japanese Orthopaedic Association score, the measures showed that improvement was not dependent on the preoperative severity of myelopathy. Level of evidence: II.

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An analysis of outcomes data for 302 patients included adverse events, complications within 30 days of the procedure, and complications 31 days to 2 years after the procedure. Early complications were more likely among patients who were older or had a combined anterior-posterior procedure, a longer surgical time, and greater blood loss compared with other patients. Surgical treatment of cervical spondylotic myelopathy had a low rate of neurologic complications and minimal risk of long-term morbidity.

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