
Cervical Spondylotic Myelopathy: A Review

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Citation

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Abstract

Cervical spondylotic myelopathy (CSM) is the most common cause of spinal cord dysfunction in older individuals. It results when spondylotic changes lead to cervical cord injury with resulting clinical deficits. Diagnosis is made based on clinical and radiographic features. A patient must have both symptoms and signs consistent with cervical cord injury as well as radiographic evidence of spondylotic cord damaged. MRI is currently the imaging modality of choice for diagnosing CSM. Patients with CSM can be managed conservatively without surgery, however most are currently managed operatively despite the fact that there have yet to be conclusive studies demonstrating the superiority of surgery. Surgery for CSM can be undertaken with either an anterior or posterior approach depending on the clinical situation.

REVIEW

DEFINITIONS

Cervical myelopathy occurs when there is clinically symptomatic dysfunction of the cervical spinal cord. One of the most common causes of cervical myelopathy is extradural compression of the cord, which can occur as a result of spinal trauma, mass lesions, degenerative changes of the spine, or other factors¹. When cord compression is caused by degenerative changes it is referred to as cervical spondylotic myelopathy (CSM), which will be the focus of this article¹.

Cervical myelopathy must be distinguished from the less serious syndromes of cervical radiculopathy and simple neck pain that may also result from spondylosis². Radiculopathy refers to the compression of the spinal nerve roots exiting the spinal canal resulting in pain or paresthesias in the distribution of the compressed nerve^{2,3}. Cervical neck pain occurs when degenerative changes stimulate nociceptors intrinsic to the cervical spine¹. Spondylosis may initially cause neck pain or a radiculopathy that progresses to a myelopathic syndrome when the cord becomes involved, though such progression is rare³.

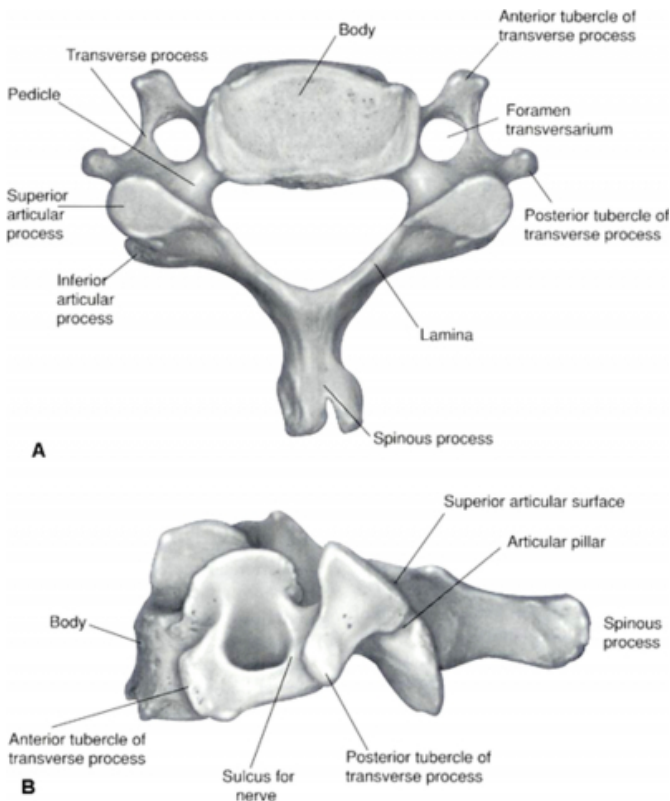
RELEVANT ANATOMY

The cervical spine is uniquely adapted to allow for a wide range of motion including flexion, extension, and movements in the lateral bending planes. It consists of seven vertebrae (see Figure 1) stacked on top of each other, spinal ligaments, and the cervical segments of the spinal cord,

which run within the spinal canal (vertebral foramina). The C1 (atlas) and C2 (axis) vertebrae are anatomically different from the other vertebrae, and these differences allow for much of neck flexion and extension and lateral rotation at the occipital-C1 and C1-C2 joints, respectively. Normally, the cervical spine has a lordotic curvature, i.e. it is concave posteriorly.

Figure 1

Figure 1: Illustration of A. cranial and B. lateral views of a typical cervical vertebra



Vertebrae are made up anterior vertebral bodies, which bear 90% of the load placed on the spine, and posterior vertebral arches. The arch is formed by bony structures called pedicles and lamina. There are five joints between all adjoining cervical vertebrae except C1-C2: an anterior curvilinear disc separating adjacent vertebral bodies, and four synovial facet joints posteriorly, one pair that connects to the vertebra above (superior facets), and one pair that connects to the vertebra below (inferior facets) ⁸. The C1-C2 articulation lacks an intervertebral disc.

Spinal ligaments include the anterior and posterior longitudinal ligaments, which are continuous bands that run along the vertebral bodies, and the ligamentum flavum, a thick band that attaches between the lamina of each vertebra.

The spinal canal houses the spinal cord and is surrounded anteriorly by the vertebral bodies, intervertebral discs, and the posterior longitudinal ligaments; laterally and posteriorly by the bony vertebral arch; and posteriorly by the ligamentum flavum. At the C1 level the spinal cord occupies just one half of the canal. It occupies three quarters of the canal at the C5-C7 levels, however, which helps to explain why CSM predominately occurs in the lower cervical spine ⁴

The spinal cord is made up of anterior gray matter, lateral white matter, and posterior gray and white matter. Lower motor neuron bodies make up the anterior gray matter, while the descending corticospinal tracts and the pain and temperature fibers of the spinothalamic tract run in the lateral white matter. Cell bodies of first order sensory neurons make up the dorsal gray matter while the dorsal white matter contains some of the axons of proprioceptive, vibratory, and touch sensory neurons. Cervical nerve roots exit the canal perpendicularly through the neuroforamina ⁸. Generally speaking, anterior cord damage leads to motor dysfunction whereas posterior cord damage causes sensory deficits.

The vascular supply of the cord consists mainly of two small dorsolateral arteries and a larger anterior spinal artery (ASA), the latter of which supplies 60-75% of cord blood flow. The midsagittal position of the ASA, along with the fact that it has fewer segmental medullary feeders compared with the dorsolateral arteries, makes the anterior spinal cord particularly susceptible to ischemia ⁵.

PATHOPHYSIOLOGY

Cervical spondylosis is the most common cause of cervical spinal cord dysfunction in individuals older than 55 ⁵. By age 30 virtually all individuals will have at least microscopic degenerative changes in their cervical spine; by age 40, most will have plain radiographic evidence of degenerative changes ⁶.

Spondylotic changes often begin in the lower segments of the cervical spine (especially C4-C7), but they may be present at all levels, particularly in the elderly ⁷. The process typically begins in the intervertebral disc, probably as a result of the loss of water content in the disc that occurs secondary to the loss of proteoglycan matrix with age ^{1,5,8}. The dehydrated disc becomes stiff and begins to shrink, leading to partial or complete collapse. Sometime the disc may herniate acutely, causing symptoms. Even if this does not happen, the disc space narrows and reactive and compensatory changes occur: osteophytes develop in the vertebral bodies, facet joints, and arches, and the ligamentum flavum thickens and ossifies ⁸. In some individuals thickening and ossification of the posterior longitudinal ligaments (OPLL) takes place as well ⁹. As the vertebrae becoming increasingly damaged, the cervical spine may lose its normal lordotic curvature.

When cervical spondylosis progresses, neural structures may be damaged. This occurs most commonly at the C5-C7 levels where spondylosis is often most advanced^{5,14}. Most commonly, nerve roots exiting the canal are injured. In a small number of individuals, the cord itself may be damaged, and a myelopathic syndrome may result¹⁰. It is important to keep in mind that cord damage is not always symptomatic, however (see Radiologic Assessment and Other Studies)¹¹.

Spondylosis may lead to cord damage in three ways. First, there may be static-mechanical compression of the cord by osteophytes, spinal ligaments, or disc material, which encroach upon the canal space resulting in stenosis of the canal. Those with a congenitally narrowed spinal canal (10-13 mm) are especially vulnerable to static-mechanical compression^{1,5}. Cord compression can be from any direction, but most commonly is along the AP axis. Anterior compression is caused by osteophytes on the posterior surface of the vertebral bodies, disc material, or rarely a thickened or ossified posterior longitudinal ligament. Loss of normal cervical lordosis exacerbates anterior compression because the cord is forced anteriorly within the spinal canal. Lateral and posterior cord compression is due to osteophytes from the vertebral arches or a thickened ligamentum flavum⁵. Patients commonly become symptomatic when the cord is compressed by 30% or more, however this varies considerably from patient to patient⁵. Patients with severe canal stenosis are also at considerably increased risk of a major spinal cord injury with trauma¹².

Dynamic-mechanical cord compression can also occur. This happens when normal flexion and extension of the neck aggravates cord damage. Neck flexion reduces the AP diameter of the spinal canal by 2-3 mm, which leads to AP compression by osteophytes¹³. Neck extension can cause the ligamentum flavum to pinch the cord against anterior osteophytes¹⁴. Lateral neck movements may lead to nerve root compression, causing radicular symptoms⁵.

Finally, spondylotic changes may impair the circulation within the cord, leading to cord ischemia and a resultant myelopathy¹⁴. Osteophytes can compress the ASA or a critical medullary feeder, or can compress venous drainage, leading to a neuroischemic myelopathy usually affecting the anterior cord⁵.

CLINICAL HISTORY

As noted above, CSM is the most common cause of

myelopathy in those middle-aged and older¹⁴. While the prevalence of CSM is unknown, one quarter of the patients with tetraparesis or paraparesis at a regional neuroscience center in the United Kingdom was found to have CSM¹⁵. Symptoms range from mildly uncomfortable to completely disabling¹.

CSM often presents insidiously with short periods of symptomatic progression followed by longer intervals of relatively stable symptoms¹⁶. Symptoms may develop suddenly, however, especially with acute disc herniation or following a flexion or extension injury. CSM may occasionally develop in patients with spondylotic cervical neck pain or radiculopathies^{1,4}.

CSM presents in a highly variable manner. It usually manifests as one of five clinical syndromes depending on the anatomical location of the cord damage and the extent of disease. These are: brachialgia and cord syndrome, central cord syndrome, anterior cord syndrome, Brown-Sequard syndrome, and transverse lesion syndrome (see table 1)⁶.

Figure 2
Table 1

Syndrome	Clinical	Anatomical basis of injury
Brachialgia and cord syndrome	- stabbing pain in shoulder/UE; a dull "achy" feeling in the arm - Sensory: possible numbness or tingling in the hands - Reflexes: may have hyperreflexia below lesion, especially in UE	Degenerative changes impinging on nerve roots causes brachialgia and mild sensory changes; may have hyperreflexia from early corticospinal tract involvement
Central cord syndrome	- Motor: UE > LE weakness below lesion ("clumsy hands") - Sensory: contralateral pain and temperature loss below lesion, relative sacral sparing - Reflexes: hyperreflexia below lesion	Degenerative changes cause central cord compression, damaging the corticospinal tracts at one or more levels; medial cervical fibers preferentially damaged over lateral thoracic/lumbar/sacral fibers leading to UE > LE dysfunction
Anterior cord syndrome	- Motor: weakness at level of lesion - Sensory: pain and temperature loss below lesion - Reflexes: hyporeflexia at level of lesion	Degenerative changes, often by causing vascular compromise (ASA impingement), damage the anterior cord including lower motor neuron nuclei and desiccating pain and temperature fibers at one or more levels
Brown-Sequard syndrome (hemicord)	- Motor: contralateral weakness below lesion, ipsilateral weakness at level of lesion - Sensory: contralateral loss of pain and temperature below lesion, ipsilateral loss of proprioception, vibration, and touch below lesion - Reflexes: ipsilateral hyporeflexia at level of lesion, contralateral hyperreflexia below lesion	Degenerative changes damage on one side of the cord including the corticospinal and spinothalamic tracts and posterior columns at one or more levels
Transverse lesion syndrome	- Motor: weakness at and below lesion - Sensory: loss of pain and temperature and proprioception, vibration, and touch below lesion - Reflexes: hyporeflexia at level of lesion, hyperreflexia below lesion	Degenerative changes damage full diameter of cord leading to damage of corticospinal and spinothalamic tracts and posterior columns at one or more levels

In general patients with CSM will experience deep aching or burning around the upper extremities (brachialgia) and will commonly have neck pain, stiffness, and crepitus with movements early in the course of their disease ¹⁷. Upper extremity weakness and impaired dexterity, especially of the hands, often develop soon afterwards followed by lower extremity dysfunction. Motor dysfunction may be unilateral or bilateral depending on the extent and location of cord damage patients also commonly report sensory symptoms including upper extremity numbness, pain, and paresthesias initially, followed by lower extremity sensory changes. Sensory loss develops below the level of the lesion, and typically affects pain and temperature sensation more than proprioception, vibration, and touch ¹. Since CSM most commonly affects the C5-C7 region of the cord, it is the dermatomes and muscles groups corresponding to these levels, as well as those below, that are most commonly affected (see table 2).

Figure 3

Table 2

Cord Level	Dermatome	Muscle Innervated	Groups
C1	None	Neck flexion/extension	
C2	Occiput and posterior ear	Neck flexion/extension, diaphragm	
C3	Upper 1/3 of neck	Shoulder elevation, diaphragm	
C4	Lower 2/3 of neck	Shoulder elevation, diaphragm	
C5	Anterior shoulder and arm	Shoulder abduction	
C6	Lateral arm and thumb	Elbow flexion, wrist extension	
C7	Posterior arm, 2 nd and 3 rd digits	Elbow extension, wrist flexion	
C8	Medial arm, 4 th and 5 th digits	Thumb extension, ulnar deviation	

Gait abnormalities and bowel and bladder instability represent some of the most bothersome symptoms in patients with CSM. Gait defects result from the involvement of long tracts in the cord, often causing patients to have a broad-based, hesitant, shuffling gait ²⁵. Likewise, bowel and bladder symptoms develop because of long tract involvement, resulting in reduced sphincter control and sometimes frank incontinence, and affect between 15%-50% of patients ^{1,26}.

Several studies have suggested that the most common initial presentation of CSM is a minor gait disturbance followed soon after by progressive weakness of the hands ¹⁸.

It is important to distinguish the symptoms of myelopathy from those of cervical spondylotic pain or radiculopathy that may occur without cord involvement. In brief, cervical spondylotic neck pain is often felt in the posterior neck over

the paraspinous musculature (cervicalgia). Patients may also experience occipital headaches, interscapular pain, and neck stiffness, but symptoms should be limited to these areas ^{1,3}. Cervical radiculopathy is manifested by proximal pain and distal paresthesias, often in an overlapping dermatomal distribution. Weakness may be present, however there should be no lower extremity or bowel or bladder symptoms ^{1,2,3,8}. Symptoms of cervical spondylotic neck pain and radiculopathy may coexist with, or rarely progress to, CSM ^{1,4}.

PHYSICAL EXAM

A full physical exam with a complete neurological assessment is needed for any patient with suspected CSM, although as previously noted, CSM presents in a highly variable manner and many exam findings are not particularly specific. Cranial nerves should be intact unless there is coexisting pathology above the foramen magnum. On motor exam, mixed upper and lower motor neuron findings may be present in the upper extremity depending on the level of cord damage (see table 2). Flaccid weakness due to upper motor neuron damage might be present at the level of the lesion whereas spastic weakness would be expected below the lesion ¹. In the common setting of cord compression at the C5-C6 level, for example, biceps and suppinators are flaccidly weak whereas triceps (C7) exhibit spastic weakness ¹⁹. Weakness of hand muscles is also common in CSM, and the fifth digit may abduct spontaneously due to intrinsic muscle weakness ²⁶. Atrophy may be present if the myelopathy is long-standing. When weakness is present in the lower extremity, spastic weakness is expected due to corticospinal tract damage.

CSM may cause decreased sensation of any or all modalities depending on the anatomic location of compression, but affects pain and temperature sensation most commonly ¹. Sensory loss may not follow a clear dermatomal distribution if neurons corresponding to more than one dermatome are damaged (see table 2). Proprioceptive, vibratory, and touch sensation may be impaired on the side of the body ipsilateral to the lesion whereas pain and temperature sensation will be impaired on the contralateral side because the latter fibers decussate upon entering the cord. It is important to remember that other peripheral neuropathies such as diabetic neuropathy may have sensory findings that mimic those of CSM ²⁶.

Reflexes in the upper extremity may be hypo- or hyperreflexive because both upper and lower motor neurons

may be damaged. A hyperactive pectoralis reflex suggests a high cord compression in the C2-C4 region ²⁰. In a lower compression at the C5-C6 level, hyporeflexia of biceps and supinators and hyperreflexia of triceps will be seen ²⁹. Hyperreflexia of the lower extremity may be present in advanced disease. Jaw jerk should be tested by tapping the open jaw and watching for the normal masseter reflex contraction to exclude pathology above the foramen magnum. The Hoffman's sign, elicited by tapping the nail on the third or fourth finger and watching for involuntary flexion of the end of the thumb and index finger is indicative of upper motor neuron damage. It is moderately sensitive for detecting CSM when present along with other upper motor neuron findings ²¹. Lhermitte's sign, in which neck flexion results in paresthesias and weakness, is a sensitive sign of posterior column compression, though it is not specific for CSM ²². Clonus of the upper or lower extremities may be present as well. A positive babinski sign suggests advanced disease.

Finally, gait abnormalities, typically a broad-based, hesitant, stiff or spastic gait, secondary to upper motor neuron disease and proprioceptive loss, are common, especially late in the disease course ²⁶.

While many of the findings present in CSM may also be present with cervical spondylotic neck pain or radiculopathies, in the latter conditions upper motor neuron signs should not be present and all findings should be limited to the neck and upper extremities ^{1,3,8}.

RADIOLOGIC ASSESSMENT AND OTHER STUDIES

A diagnosis of CSM can be made only if a patient is both symptomatic and has radiographic evidence of spinal cord impingement or compression. Thus radiographic assessment is essential. The correlation between radiographic evidence of spondylotic cord damage and clinically significant CSM is not perfect, however ²³.

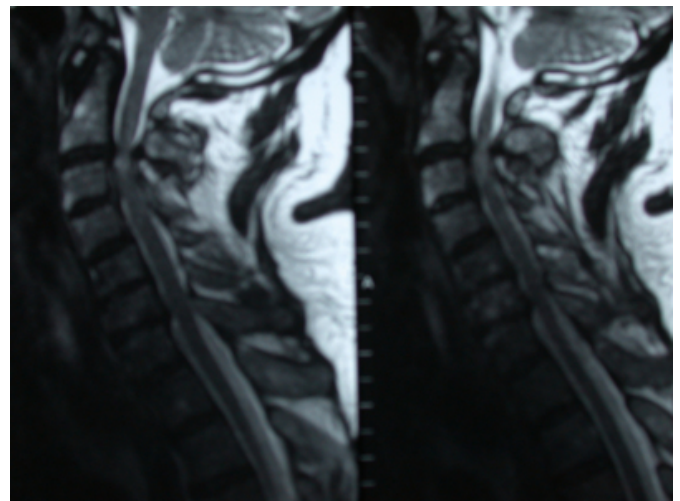
Plain radiography may be useful in an initial evaluation for visualizing the extent of cervical spondylotic changes including disc space narrowing, osteophytosis, kyphosis, joint subluxation, and stenosis of the spinal canal. A CT scan is helpful in assessing canal stenosis, and may show osteophytes better than plain radiography. It is also especially good at defining the neural foramina, and is useful in diagnosing OPLL ^{10,13,35}.

MRI remains the imaging modality of choice for CSM, even

in an initial evaluation, because of its superior ability to show pathology of neural structures ²⁴. MRI allows for clear visualization of cord impingement or compression, and can be used to accurately measure space within the spinal canal (see Figure 2) ²⁶. Signal intensity may be increased at the level of cord damage, particularly on T2-weighted images, due to inflammation, edema, ischemia, gliosis, or myelomalacia ²⁵. In fact, signal changes have been shown to be a fairly reliable indicator of irreversible cord damage ²⁶. Diffusion-weighted MR imaging, in particular a technique known as diffusion tensor imaging, may enhance sensitivity for detecting CSM. Because of its ability to detect significant cord damage before the appearance of symptoms it is currently being evaluated for its potential prognostic value (see Natural History and Surgical Indications) ²⁷. Finally, MRI can be used to evaluate for non-spondylotic causes of myelopathy both intrinsic and extrinsic to the cord.

Figure 4

Figure 2: MRI showing cervical spondylotic myelopathy



MRI findings are not completely specific for clinically significant CSM, however. In one study, spinal cord impingement was seen on MRI in 16% of asymptomatic patients under 64 and in 26% of asymptomatic patients 64 and older ³³.

Myelography, which involves plain radiographic or CT imaging after intrathecal injection of contrast, may have some use in patients who cannot tolerate an MRI because of pain, claustrophobia, or implanted metal objects ^{10,15}. Myelography has been shown in some older studies to have a sensitivity and specificity similar to that of MRI for evaluating cord pathology, and has the added benefit of allowing for simultaneous CSF analysis ²⁸. In general, however, MRI is superior because it is better at detecting

bony pathology, it is not contraindicated when elevated intracranial pressure is a concern, and it is more comfortable for patients^{36,37}. Myelographic studies may be limited as well when a spinal subarachnoid block is present.

Finally, electromyography, nerve conduction studies, and motor and somatosensory evoked potentials have a role in some cases in differentiating CSM and nerve root compression from peripheral neuropathies and myopathies. EMG may show evidence of muscular denervation in cases in which CSM, radiculopathy, or another peripheral neuropathy has been present for an extended period of time, but will show evidence of intrinsic muscle pathology in myopathies²⁹. Nerve conduction studies will usually be normal in CSM and radiculopathies except with extensive axonal or upper motor neuron damage because the distal part of the peripheral nerve is normal in these conditions. In contrast, conduction studies may show slowed conduction with many peripheral neuropathies or with a peripheral nerve entrapment⁴⁶. Motor and somatosensory evoked potentials measured with scalp electrodes will be abnormal in a wide array of patients with neurological dysfunction including many patients with CSM and radiculopathies, and may help in the assessment of disease severity³⁰.

In summary, radiologic evidence of spondylotic cord damage is necessary for a diagnosis of CSM, and MRI is generally the study of choice. Still, radiologic imaging is not always completely accurate for making a diagnosis.

DIFFERENTIAL DIAGNOSIS

Establishing a diagnosis of CSM early is very difficult yet essential. Unfortunately, an incorrect diagnosis is often made, and in one study it was found that 14.3% of patients operated on for CSM actually were misdiagnosed³¹.

When faced with a patient who has clinical symptoms or signs worrisome for CSM, first it is important to decide whether the patient has a myelopathic condition, i.e. whether there is actual spinal cord pathology. Several conditions can mimic a myelopathy including cervical spondylotic neck pain or a radiculopathy, injuries or arthritis of the upper extremity, peripheral neuropathy or nerve injury, myopathies, vascular disease, drug intoxication or withdrawal, autoimmune diseases, and metabolic abnormalities³².

Additionally, myelopathies have various etiologies other than spondylosis. Besides spondylosis, three of the most common causes of myelopathy due to cord compression are

hemorrhage, abscess, and tumor³³. Other causes of myelopathy include vascular disease, infections like syphilis or encephalitis, inflammatory conditions, drug use, congenital conditions such as syringomyelia, autoimmune or acquired diseases like MS or ALS, trauma, normal pressure hydrocephalus, or metabolic abnormalities like vitamin B-12 deficiency³⁴. Depending on the clinical situation, it may be important to rule out certain of these conditions.

NATURAL HISTORY

In order to know how best to treat CSM, it is essential to have an understanding of the natural history of the disease. Before doing this, it is important to note that most studies of the natural history of CSM, which will be discussed below, were carried out more than two decades ago before modern radiologic diagnostic methods were available. Some of the patients presumed to have CSM in these studies may have been misdiagnosed, possibly leading to inaccurate results. Furthermore, nonsurgical therapy (see Nonoperative Management below) has been used to treat CSM for several decades. Most of the patients in these studies were treated with such therapy, which may alter the natural history of the disease compared to no therapy at all. Now, most patients with CSM are treated with surgical therapy, and it is becoming increasingly difficult to study the natural history of CSM.

Clark and Robinson were the first to study the natural history of CSM in 1956³⁵. They looked at 44 CSM patients with symptoms ranging from mild (not affecting everyday activities) to severe (unable to walk without assistance) and followed these patients for one to 32 years. Onset of symptoms in these patients ranged from age 31 to 80. They found that about 75% experienced a waxing and waning disease course characterized by short periods of symptomatic worsening followed by long intervals of relatively stable disease. In about 20% of patients, CSM showed a slow, steady, progressive course, and in about 5% the disease did not seem to progress after the initial appearance of symptoms. While sensory and sphincter function sometimes improved with time, motor function and gait abnormalities usually persisted or worsened, and complete disease regression never occurred. Slightly more than half were severely disabled at some point during the period in which they were followed, however none died directly from CSM.

Several others have also studied the natural history of CSM³⁶. Like the patients followed by Clark and Robinson, the

patients in these studies presented initially with a disability level ranging from mild to severe and rarely if ever had complete disease regression after symptoms began. Some patients remained relatively stable while others deteriorated considerably from their initial presenting condition. There has been considerable discordance among these studies regarding the exact percentage of patients who deteriorated, however. At the extremes, a study by Lee and Turner suggested that only a third of patients worsen considerably from their presenting condition while Symon and Lavender found that about two thirds of patients do ^{58,62}. There still is no consensus on this important issue.

Since outcomes vary so widely, with some patients remaining relatively stable and others deteriorating, much effort has been put into finding parameters that can help predict the clinical course of a given patient ³⁷. No one has found clear evidence that clinical parameters such as patient age, type of clinical presentation (acute versus subacute), or anatomic level of disease can reliably predict which patients will deteriorate ^{57, 63}. Radiographic imaging is currently being evaluated for its prognostic value, however. As noted above, signal intensity change on T2 weighted MRI suggests irreversible cord damage, which may mean that patients with considerable signal change are less likely to regain neurological function with treatment ^{39,41,42}. Recently, it has been suggested that diffusion weighted MRI may have some utility in identifying patients likely to have progressive disease ^{38,43}, however this is not yet well established (see Surgical Indications).

Thus, it can be said that once a diagnosis of CSM has been made, it may be very difficult to predict the disease course in a given patient. Somewhere between one third and two thirds of patients will deteriorate. The rest will stabilize, though significant improvement is rare. Clinical and radiographic parameters have shown limited ability to predict which patients are likely to deteriorate and which are likely to stabilize.

NONSURGICAL MANAGEMENT

Nonsurgical therapy provides symptomatic relief in patients with CSM, and may slow disease progression to some extent, though the latter statement has not been proven ³⁸.

First, and probably most importantly, patients who are being nonoperatively managed should avoid activities that exacerbate symptoms ^{1, 64}. This means not participating in activities requiring neck movements or with the potential to

cause neck trauma because patients with CSM are highly susceptible to further cord damage with movement or trauma. Patients should also be instructed to rest as much as possible when symptoms flare.

Long term cervical immobilization with a cervical collar or neck brace is commonly used, though there is little evidence for its efficacy ³⁹. Neck motion is not appreciably limited with most commercial neck collars and braces, and immobilization most likely does not change long-term outcomes ⁴⁰.

Systemic and epidural steroids have been proposed to treat spondylosis ⁴¹. No studies have looked at the effects of steroids on outcomes in patients with CSM, however. Experimental treatments to reverse existing cord damage include antibodies to block axonal growth inhibitors, gangliosides to augment neurite growth, 4-aminopyridine to enhance axonal conduction through demyelinated fibers, and stem cell therapy ⁵². Unconventional healing methods have been used as well, though the evidence to support these treatment options is scant ⁴². NSAIDs, tricyclic antidepressants, muscle relaxants, and opioids can be used for pain relief at the patient's and physician's discretion.

Many patients with CSM are treated surgically (see Operative Indications). Some patients may not be surgical candidates due to other medical morbidities or may elect for nonoperative management. Any patient being nonoperatively managed, however, should be referred for a discussion of the surgical options.

OPERATIVE INDICATIONS

Many patients with CSM are treated surgically in the hopes of preventing neurological deterioration or even achieving some recovery of function ⁴³. Studies comparing nonsurgical management with surgical management are limited, however ⁴⁴.

Probably the best evidence that surgery is an effective treatment for CSM is that patients who are operated on early (within a year of the onset of symptoms) have better outcomes than those who are operated on more than a year after the onset of symptoms ⁴⁵. A study by Ebersold et al. found that the only factor correlated with postoperative functional status is duration of symptoms preoperatively ⁷⁷, and a study by Phillips found that patients operated on within a year of the onset of symptoms were more likely to have favorable surgical outcomes than those who were not ⁶¹. Furthermore, several studies have shown that many patients

treated surgically have good outcomes. A prospective study by Mann of 50 CSM patients with rapidly deteriorating neurological status showed that more than half of the patients stabilized or improved after surgery, however the outcomes were not compared with a control group managed nonsurgically ⁴⁶.

Studies directly comparing long term outcomes in patients who received surgical and nonsurgical treatment for CSM are sparse and have limited scope ⁷⁶. Only two prospective studies have compared conservative and surgical treatment of CSM, only one of which was a randomized study ⁴⁷. Kadanka et al. ⁸⁰ randomized 68 patients with mild or moderate CSM to receive conservative treatment (35 patients) or surgery (33 patients). Clinical outcomes were evaluated by both objective measures of physical disability and subjective assessment by patients themselves at six, 12, 24, and 36 months. At all time intervals, the patients managed surgically and nonsurgically had similar outcomes with minimal deterioration in objective or subjective functioning. The sample size in this study was small, however, and it is not clear if it was adequately powered to detect a small difference in outcomes. Furthermore, the study only evaluated patients with mild or moderate disease.

Sampath et al. ⁸¹ compared outcomes in 43 patients with CSM for whom surgery was offered, 20 of whom elected for surgery and 23 of whom chose nonoperative management. Outcomes were assessed based on a variety of measures of functional status and overall pain with a follow-up period of eight to 13 months. Surgically treated patients had a slight statistically significant improvement in functional status and overall pain while conservatively treated patients had a small but significant worsening in their condition. Patients were not randomized in this study, however. Furthermore, the magnitude of benefit with surgery relative to conservative management was relatively small.

Several retrospective studies comparing surgery with conservative therapy have had conflicting results as well, and are limited because of confounding factors and small sample size ⁴⁸.

Because surgery has not yet clearly been shown to be beneficial in all patients with CSM, many have suggested ways to identify patients most likely to benefit from surgery. For example, patients likely to deteriorate clinically might be good surgical candidates since prophylactic surgery might be able to protect the cord before permanent damage occurs. As noted above, clinical parameters have not been shown to

effectively predict disease course, however radiographic parameters may have some utility that is not yet well established. Patients with considerable signal intensity changes on T2 weighted MRI could, for example, be told that they are less likely to recover neurological function with surgery ^{39,41,42}. Such patients might still be good surgical candidates, however, if they are likely to deteriorate further. More helpful would be radiographic studies that could identify patients likely to deteriorate. If diffusion weighted MRI is shown to be able to do this accurately, patients with findings suggestive of progressive disease could be encouraged to undergo surgery ^{38,43}.

Despite the limitations of our current knowledge, many patients with CSM are treated surgically. There is some evidence that surgery improves long term outcomes, at least in some situations, but more work will be needed to show this definitively. If surgery is shown to be beneficial, more work is also needed to identify which patients are most likely to benefit from surgery. At this time, most patients with CSM should be offered surgical treatment as an option; however nonoperative management is appropriate for those who elect for it. For those who elect for surgical treatment, surgery should occur as soon as possible, particularly in patients with noticeably deteriorating neurological status, since patients operated on early have better outcomes.

OPERATIVE MANAGEMENT

Surgical treatment of CSM is aimed at decompressing the spinal cord in the hopes of preventing further spinal cord damage. Sometimes, some neurological function can even be recovered with surgery, however often neurological damage is irreversible.

Decompression can be accomplished by removing bone, disc, or ligamentous material that is encroaching on the space within the spinal canal. A posterior or anterior approach to the spine may be used. Adjacent vertebrae may need to be fused together to prevent neck instability ⁴⁹.

A posterior approach allows for dorsal decompression of the cord with a laminectomy (see Figure 3) or laminoplasty. Until 20 years ago, a posterior laminectomy was the mainstay of surgical therapy for CSM. In a laminectomy, the laminal arch along with the ligamentum flavum are removed to create room within the spinal canal ⁶³. Often the affected vertebrae are fused ⁵⁰. Because the lamina are removed, a kyphotic deformity can develop postoperatively ⁵¹. In a laminoplasty the lamina are reconstructed in a way that creates more room within the spinal canal but are not

removed⁶³. While a laminoplasty is a more complicated procedure than a laminectomy, patients may have somewhat improved neck stability and mobility and less kyphosis after a laminoplasty because the structural integrity of the vertebrae are maintained⁵². The issue of whether a laminectomy or laminoplasty is superior is still a matter of debate, however⁵³. An anterior approach is used mainly for discectomy and corpectomy operations (see Figure 4)⁵⁴.

Figure 5

Figure 3: Illustration of a cervical laminectomy procedure, A. shows a lateral view and B. shows a cranial view

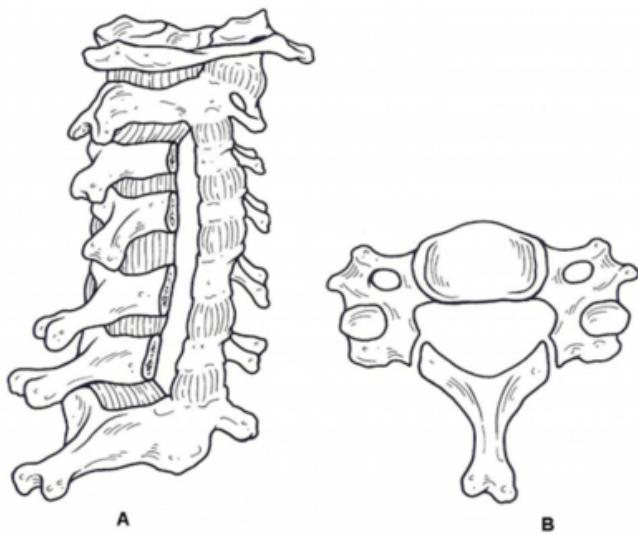
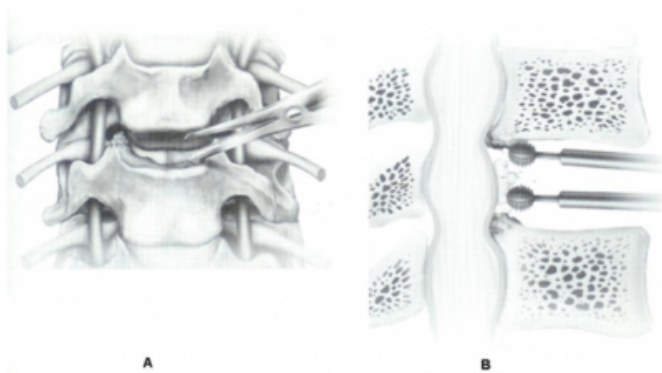


Figure 6

Figure 4: Illustration of an anterior discectomy procedure, A. shows an anterior view and B. shows a lateral view



Anterior discectomy is indicated for single-level and some multi-level disc herniations causing cord damage^{63, 95}. Fusion is almost always performed along with discectomy because spinal instability is common if this is not done⁶³. Corpectomy, in which the vertebral body and disc are removed at one or more levels and the vertebrae above and below the corpectomy are fused, is another option when cord

compression is predominately anterior. It is often favored when three or more cord levels are involved as it may provide superior decompression and a higher rate of successful fusion in these cases^{86, 97}.

Deciding between a posterior or anterior approach depends on a number of factors⁵⁵. A posterior approach generally is favored in cases in which compression is mainly posterior, for example by posterior osteophytes or a thickened ligamentum flavum. Conversely, an anterior approach is preferred in patients with predominately anterior compression from disc material, anterior osteophytes, or a thick or ossified posterior longitudinal ligament^{91, 92, 95}. Either approach can create space within the spinal canal regardless of the anatomical location of compression, however.

Besides anatomical considerations, several factors influence the decision to take a posterior or anterior approach. Posterior approaches are usually used when three or more spinal segments are stenosed regardless of the location of the compression because there is a high rate of pseudoarthrosis (unsuccessful fusion) when an anterior approach is used⁶³. Posterior approaches are less effective in patients with kyphosis, however, because the cord is not able to fall back into the space created by posterior decompression due to the deformity⁵⁶. An anterior approach is usually favored in these cases. A combined posterior and anterior approach may be required when canal stenosis results from both significant anterior and posterior compression⁵⁷. In some cases, it is not clear whether an anterior, posterior, or combined approach is superior and the decision is at the patient's and surgeon's discretion^{63, 92, 91, 98}.

Another area of uncertainty is when to fuse adjacent vertebrae following a laminectomy. (Fusion is usually used in anterior decompressions, and is unnecessary with laminoplasties.) Fusion is accomplished by placing bone graft material between adjacent vertebrae, which stimulates bone growth and ultimately fusion of the vertebrae⁵⁸. This process takes several months, and a patient's neck motion must be limited with cervical immobilization until the fusion is complete. Different types of bone grafting material, autogenic, allogenic, and synthetic, can be used⁵⁹. Sometimes spinal instrumentation such as rods, screws, plates or cages will be put in as well to hold vertebrae together in the hopes of improving the success rate of fusion⁶⁰. While fusion provides improved cervical stability, it limits neck motion, particularly when it is performed at the

occipital-C1 or C1-C2 levels where much of neck flexion and extension and lateral rotation occur, respectively⁶³.

Major complications of spinal cord surgery include damage to neural structures resulting in paralysis, palsies, radiculopathies, and bowel and bladder dysfunction.

Meningitis, stroke, damage to the recurrent laryngeal nerve, vertebral artery damage, and bone graft failure may also occur⁶¹. Major complications, including perioperative death, occur at a rate of approximately 5%⁶². Patients are likely to have reduced range of neck motion after surgery, especially if a fusion is performed.

CONCLUSION

CSM is a common cause of cervical spinal cord injury, particularly in the middle-aged and elderly. Diagnosis is made in patients with neurological deficits consistent with the disease who have radiographic evidence of spinal cord compression. Some patients will deteriorate clinically, however some may stabilize following the development of symptoms. CSM is often managed surgically, however studies comparing long term outcomes in patients managed nonoperatively and operatively are limited and nonsurgical management is appropriate for those who elect for it. More work is needed to determine whether surgery indeed is the treatment of choice for CSM, and if so under which circumstances. To do this, it will be important to identify reliable prognostic factors, such as radiographic findings, that will help predict whether a given patient is likely to deteriorate or stabilize.

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