



Diagnosis and Management of Cervical Myelopathy

ABSTRACT

Cervical myelopathy is a degenerative disease that occurs secondary to direct spinal cord compression and compromise of spinal vasculature through a process of gradual spinal canal narrowing. Patients generally present with signs and symptoms of long tract compromise. Once myelopathy is suspected on clinical grounds, MRI is the test of choice to confirm canal stenosis and cord injury. Treatment involves surgical decompression, anteriorly and/or posteriorly of the spinal. Despite optimal management in this patient population, outcomes may be poor and are usually limited to halting progression of the disease rather than relieving deficits already present.

KEYWORDS: Cervical myelopathy, cervical stenosis, degenerative spine disease, spondylosis



CME

Pre-test Quiz



Introduction

Myelopathy is a term used to describe the clinical syndrome present when a disease process in or around the spinal cord interrupts the normal transmission of information along the ascending and descending tracts contained within it. The syndrome can be differentiated based on the level of pathologic involvement, which can generally be determined by the extent of involvement of the extremities as documented on physical examination. Cervical myelopathy occurs when the disease process affects the cervical spinal cord, and can manifest as symptoms in all four extremities. As implied in the definition, a number of pathological processes can give rise to this clinical syndrome, but the most common cause is canal stenosis secondary to degen-



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erative changes commonly seen with aging, termed ‘cervical spondylotic myelopathy’.¹

Pathophysiology

Cervical spondylotic myelopathy (CSM) is a syndrome of spinal cord

MYELOPATHY TYPICALLY PRESENTS WITH DEFICITS IN THE LATERAL ASPECT OF THE CORD, WITH ANTERIOR AND POSTERIOR FUNCTIONS SPARED UNTIL LATE IN THE DISEASE PROCESS.

dysfunction secondary to spinal cord compression, resulting from spinal canal narrowing via both static and dynamic mechanisms, with the underlying cause being slow mechanical degeneration. The normal anterior-posterior (AP) canal diameter in the cervical spine has been reported as 17 to 18 mm.² However, in patients with CSM, the AP diameter decreases, and signs of myelopathy begin to show when the diameter is decreased to 14 mm or less.³ The average diameter at which myelopathy occurs is 12 mm,⁴ and this has been accepted as the absolute diameter below which myelopathy is very likely to be present. These studies, based on measurement from plain x-rays, noted that myelopathic findings are present across a range of canal diameters, and this is highly individual, lead-

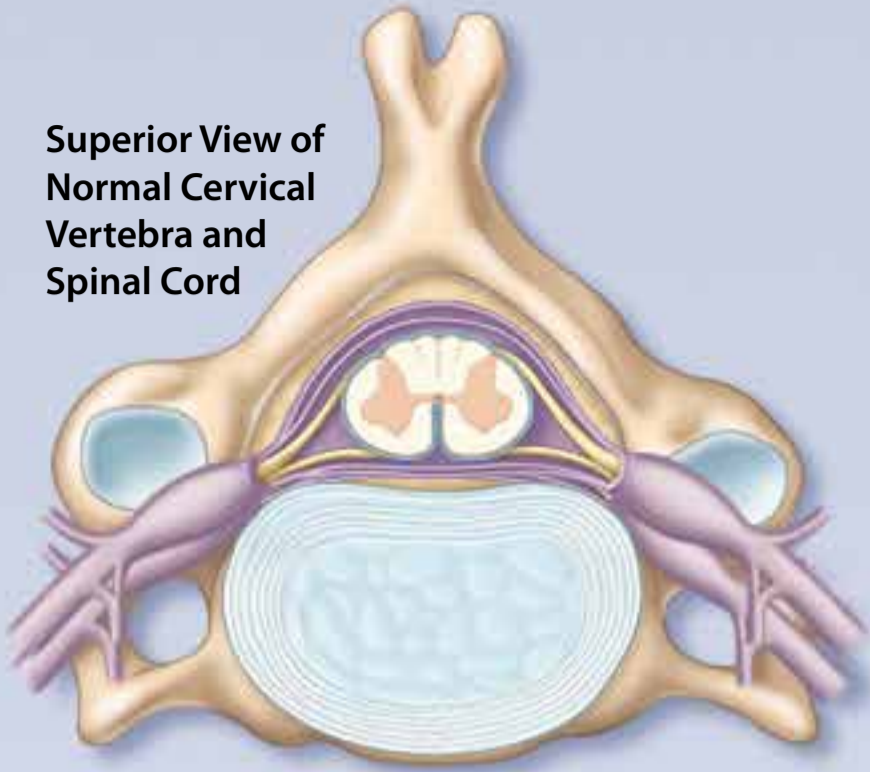
ing to the argument that cord compression alone does not generate myelopathy. Subsequently, cadaver studies have demonstrated arterial filling defects in spinal arteries with neck motion,⁵ thus leading to the hypothesis that cord ischemia, as opposed to direct compression, is the primary mechanism by which cord damage occurs.⁴ Ischemia may result from occlusion of the penetrating arteries to the spinal cord by direct compression, or from narrowing of arteries due to distortion from spinal cord displacement. As the spine becomes flattened by anterior or posterior compression, the cord bulges out laterally, which places the vessels running laterally on the cord under stretch, thus reducing their diameter and limiting the flow of blood to the lateral columns of the cord.⁶ It is for this reason that myelopathy typically presents with deficits in the lateral aspect of the cord, with anterior and posterior functions spared until late in the disease process, as outlined below.

Static spinal cord degeneration can occur over an extended period of time, and significant stenosis must occur before symptoms are recognized due to redundancy in canal diameter.⁷ The process begins with degeneration and bulging of the intervertebral discs. Disc degeneration causes bony hypertrophy in adjacent endplates, and the resulting dorsally-



Causes of Spinal Cord Ischemia in Cervical Myelopathy

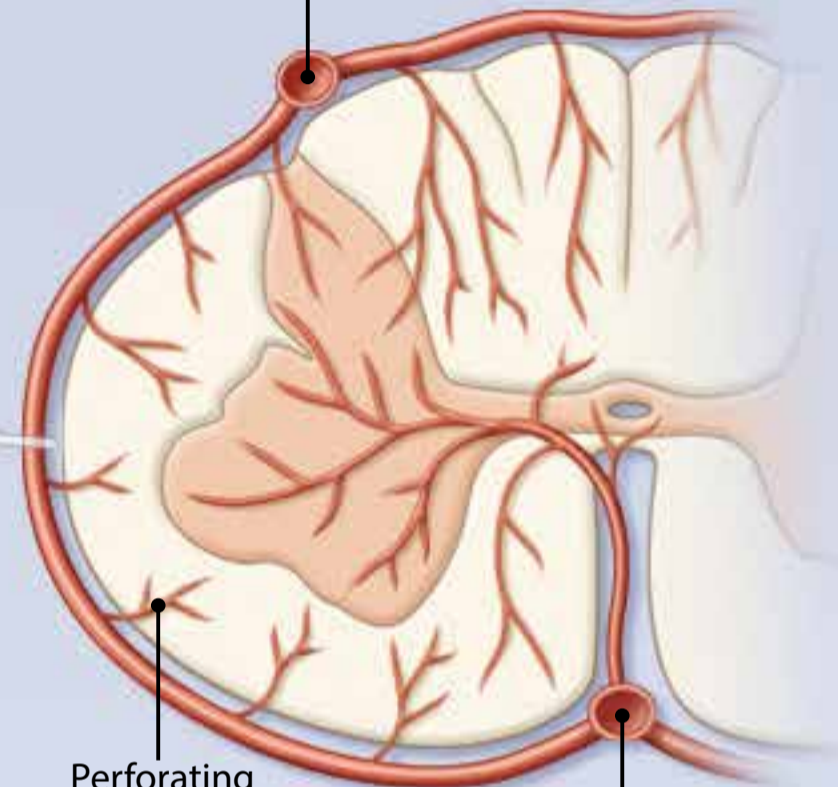
Superior View of Normal Cervical Vertebra and Spinal Cord



Sectional View of Artery



Posterior Spinal Artery



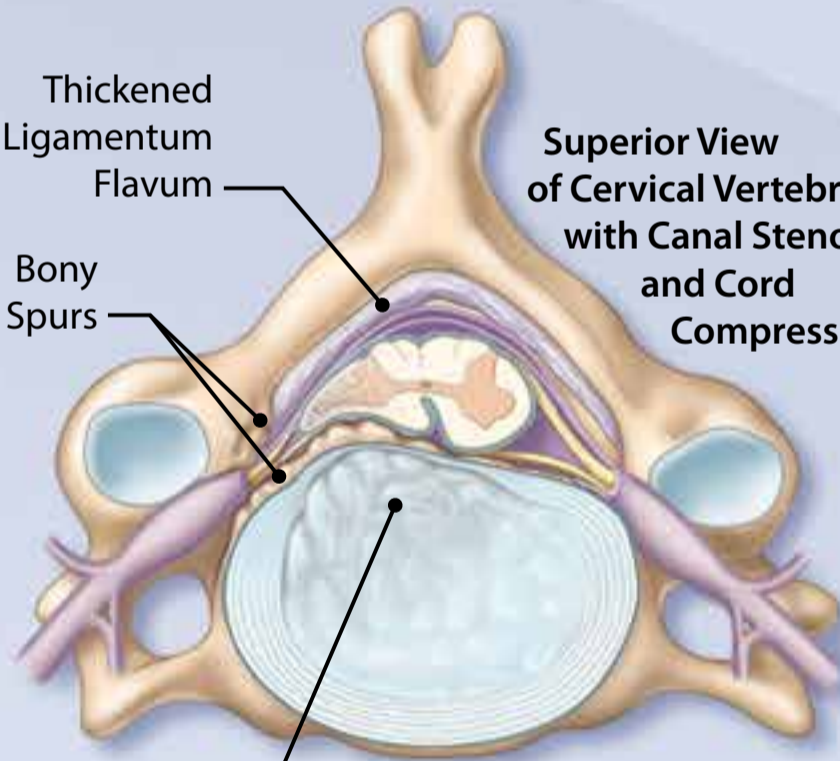
Perforating Arteries

Anterior Spinal Artery

Thickened Ligamentum Flavum

Bony Spurs

Superior View of Cervical Vertebra with Canal Stenosis and Cord Compression



Herniated Disc

Posterior Compression

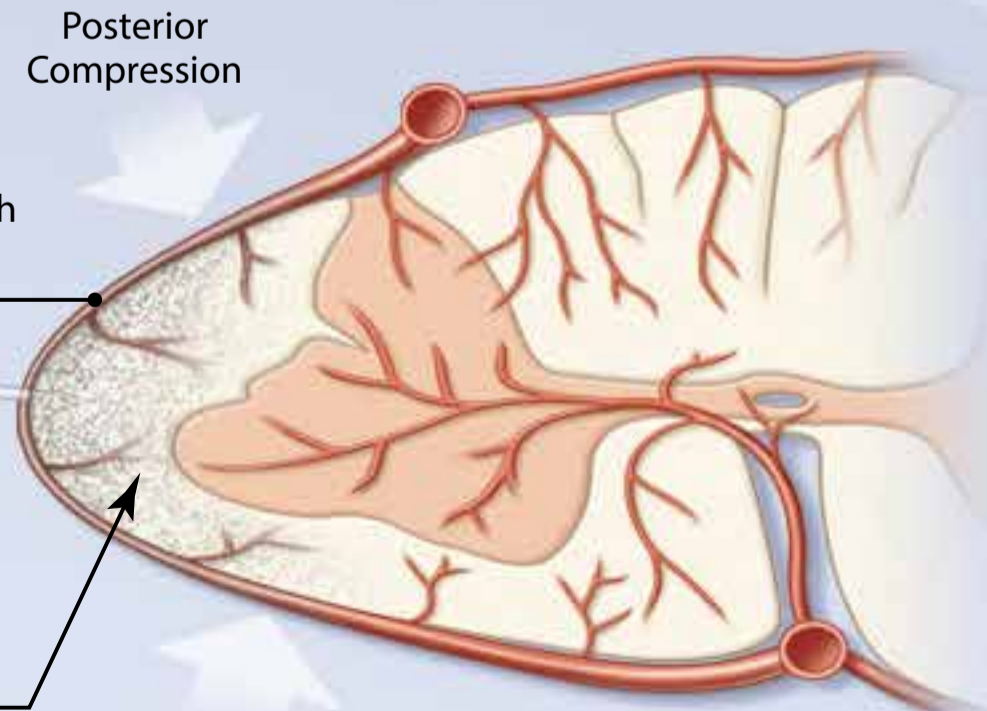
Arteries under Stretch (Reduced Diameter and Blood Flow)

Sectional View of Artery



Ischemic Region (Lateral)

Anterior Compression



oriented bony spurs lead to further canal compromise. Concurrently, end-plate proximity from loss of disc height leads to over-riding and destruction of the uncovertebral joints, which in turn causes reactive hyperostosis and extension of bony and soft tissue into the canal and proximal foramina. These changes decrease inter-body mobility and cause disuse hypertrophy of the posterior elements, namely the facet joints and ligamentum flava, which further encroach upon the canal. Loss of mobility at a single segment causes increased strain and hypermobility at adjacent segments, hastening the degenerative process and perpetuating the disease.

In addition to the static degeneration, dynamic instability becomes a problem later in the disease process, and is perhaps the factor that leads to the development of clinical myelopathy. With degeneration, hypermobility may be experienced at adjacent spinal cord levels, and lead to further canal narrowing with flexion or extension of the neck.⁸ Even normal motion of a stenotic canal can cause further narrowing by approximating anterior hypertrophic tissue to posterior tissue (i.e. extension bringing a posterior bony spur into proximity with a hypertrophied ligamentum flavum or facet joint). This effect is compounded in a hypermobile segment of the spine that has

occurred due to adjacent degenerative fusion. Degeneration can also cause the vertebrae to slide anteriorly or posteriorly relative to one another during neck motion, and this can further contribute to dynamic stenosis.⁹ The effects of both static and dynamic degeneration are more likely to cause symptomatic myelopathy in a congenitally narrowed spinal canal.²

Epidemiology

Not all patients with degenerative changes on imaging will exhibit the signs and symptoms of CSM. In fact, the incidence of degenerative changes reported in the literature is higher than the incidence of clinical CSM. Cadaver studies have demonstrated rates of stenosis at 4.9% in the adult population,¹⁰ with higher rates being found in older individuals, reaching 9% in the 8th decade. In radiographic studies, 95% of asymptomatic male patients (70% of women) in their 7th decade have degenerative abnormalities on plain films.¹¹ Even in patients under 40, MRI has demonstrated degenerative changes in up to 14%.¹² These data indicate: that CSM resulting from canal stenosis is a slow process, that not all patients with degenerative spine disease will develop clinical features and that myelopathy is a clinical diagnosis not a radiographic one.

The incidence and prevalence of clinical CSM is not well reported



in the literature. Wu *et al.*, (2013) reported an incidence of CSM-related hospitalization at 4.04 per 100,000 person years in an eastern Asian cohort.¹³ As CSM is the result of a degenerative process, the incidence is higher among the older

DEGENERATION CAN ALSO CAUSE THE VERTEBRAE TO SLIDE ANTERIORLY OR POSTERIORLY RELATIVE TO ONE ANOTHER DURING NECK MOTION, AND THIS CAN FURTHER CONTRIBUTE TO DYNAMIC STENOSIS.

population, with rates approaching 30% by the 8th decade of life.¹³ In a British cohort, the mean age at time of diagnosis was reported as 64 years.¹⁴ Symptomatic disease is more often seen in men, with a male to female ratio of 2.7:1. Overall, CSM has been found as the cause of symmetric limb paresis in almost 26% of non-traumatic cases.¹⁶ While cervical spondylosis typically affects multiple levels, the most commonly involved level is C5-C6.¹⁴ It should be noted that patients presenting with CSM can often be found to have stenosis in the lumbar region as well, with some studies quoting rates of up to 59% (Laroche *et al.*, 1991). This is an important consideration when working up patients with complaints of gait difficulties and lower

extremity symptoms. Overlooked upper extremity symptoms/signs could lead to a lumbar imaging study, which may direct treatment towards lumbar stenosis, whereas the more appropriate level to treat is the cervical spine.

Clinical Presentation

As previously stated, the degree of canal stenosis required to produce myelopathic symptoms is varied, and highly individual. Likewise, the sequence of symptoms is also variable among individuals with the disease.¹⁷ Some patients with degenerative disease of the cervical spine will present with only mild symptoms related to the degeneration, such as neck pain or possibly radicular symptoms. These patients, by definition, do not suffer from cervical myelopathy. In many patients, the earliest sign of myelopathy is gait stiffness or trouble with balance, and may lead them to seek medical attention.¹⁸ A number of other symptoms can be seen,¹⁹ including paresthesias in a non-dermatomal distribution, weakness or paresis (more commonly affecting fine motor movements in the hands;¹⁷), spasticity or sphincter disturbance and incontinence. Motor involvement of the lower extremities tends to be more pronounced proximally rather than distally, and bowel and bladder dysfunction is not often seen until the disease has progressed in severity. About half of patients pre-



Table 1: Modified Japanese Orthopedic Association (JOA) Score For Cervical Myelopathy²⁶

Score		Description
Upper extremity (UE) motor dysfunction		
0		Unable to feed self
1		Unable to use knife and fork; can eat with spoon
2		Can use knife and fork with much difficulty
3		Can use knife and fork with slight difficulty
4		None (normal)
Lower extremity (LE) motor dysfunction		
0		Unable to walk
1		Can walk on flat surface with walking aid
2		Can walk up and/or down stairs with handrail
3		Lack of smooth and stable gait
4		None (normal)
Sensory deficit		
0	UE	Severe sensory loss or pain
1		Mild sensory loss
2		None (normal)
0	LE	Severe sensory loss or pain
1		Mild sensory loss
2		None (normal)
0	trunk	Severe sensory loss or pain
1		Mild sensory loss
2		None (normal)
Sphincter dysfunction		
0		Unable to void
1		Marked voiding difficulty (retention)
2		Some voiding difficulty (urgency or hesitation)
3		None (normal)



compression at C5-C6 (the most commonly involved level) will yield brisk reflexes in the triceps and lower extremities, while potentially sparing the biceps and brachioradialis reflexes. However, an inverted radial reflex can be present (this occurs when the efferent arc of the brachioradialis reflex results in stimulation of the forearm finger flexors). Other pathologic signs may also be present. Hoffmann's sign is present when a downward flick of the distal phalanx of the middle finger results in reflexive flexion of the other fingers and thumb of the ipsilateral hand. This sign may be present in myelopathic patients, but the sensitivity and positive predictive value are low (58% and 62% respectively).²¹ Clonus may be present at the ankles, consistent with hyper-reflexia. The Babinski sign, dorsiflexion of the large toe with a noxious stimulus to the plantar surface of the foot, may be present in up to 80% of patients with severe myelopathy, but is less consistent for patients with milder disease.²² Fine motor movements may be decreased on exam, and can manifest as difficulty performing rapid grip and release or difficulty with brisk finger movements.¹⁷ Muscle wasting may also be seen, and wasting of the hand intrinsic muscles with spasticity gives rise to the characteristic "myelopathy hand," reported as being present in 90% of patients with CSM.²³

Examination of the sensory system should be carried out, as patients commonly present with sensory findings as well. A sensory level may be found with pinprick testing, and posterior column dysfunction is usually a sign of severe long-standing myelopathy.¹⁷ If the posterior columns are involved, patients may also display a positive Romberg test, losing balance when the eyes are closed with the arms abducted slightly from their side. This test, however, has poor sensitivity for CSM and is positive in only 44% of patients with cord signal change.²⁴

Multiple scores have been developed in an attempt to classify patients according to myelopathy-related deficits. The most widely cited of these, the Japanese Orthopedic Association Scale²⁵ has been modified to the North American population (Table 1).²⁶ These scores are used clinically to determine which patients have severe enough symptomatology to be treated surgically, and to track progression or improvement post-operatively. However, many of these scales suffer from a lack of consistency,²⁷ and should therefore be interpreted with this in mind.

Diagnosis

While CSM is the most common cause of myelopathy, especially in older patients, many disorders can also cause myelopathy and must



be kept in the differential on initial workup (Table 2).²⁸ A focused history and physical exam can direct the clinician toward the correct diagnosis, but the signs and symptoms typically associated with CSM are not highly sensitive or specific,²⁹ and various clinical tests are generally required to differentiate between CSM and its numerous mimics. These tests, combined with a good clinical evaluation and routine imaging, including MRI, can often identify the specific diagnosis.

When CSM is suspected, plain x-rays of the cervical spine may be obtained, and will often show signs of degeneration. Loss of disc height may be seen at levels of disc degeneration and, if chronic, posterior disc-osteophyte complexes may be seen intruding on the canal. In cases of long-term CSM, facet joint ossification and fibrosis, as well as ligamentous calcification, may be seen. Dynamic changes, such as listhesis (or slippage) of vertebral

bodies, can also be identified with plain films. CT can be used as an adjunct when plain films do not adequately denote bony anatomy. Estimates of canal diameter can also be completed with these imaging modalities, and compared to those typically listed as consistent with myelopathy (see above). The Pavlov ratio, which can be calculated on plain films as the ratio of the canal diameter to the sagittal width of the vertebral body, provides a reliable method for predicting deficits from canal stenosis when the calculated ratio is <0.8 .³⁰ However, with the increased availability of MRI, the utility of bony imaging modalities as a first study in the clinical setting of cervical myelopathy is questionable.

MRI is the most sensitive test for identifying spinal cord compression and injury in the setting of myelopathic symptoms. MRI can demonstrate the extent of degenerative disc herniation and other soft tissue hypertrophy (e.g., facet joint capsule, ligaments) better than plain films or CT (Figure 1, Figure 2). CSF space around the cord can be visualized, and if this space is absent in the axial plane, severe stenosis is present. It will also provide a more accurate measure of canal diameter as soft tissues that cannot be visualized by x-ray or CT

Figure 1: MRI showing signal change within the cord (myelomalacia)



Figure 1A

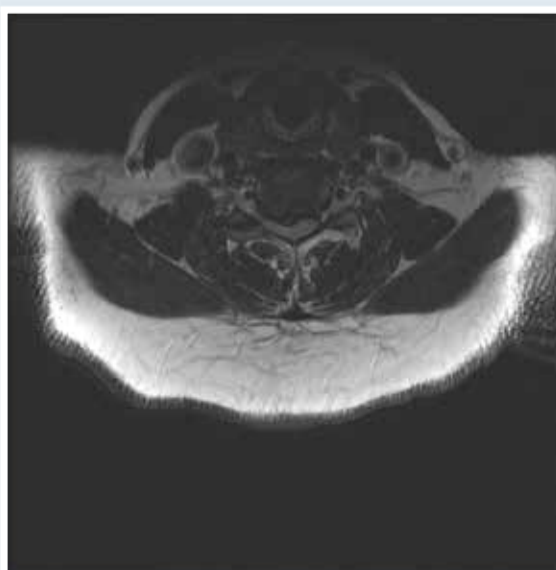


Figure 1B



Table 2: Diagnoses that can present similar to CSM, divided based on etiology²⁸

Compressive Myelopathies

CSM

Spondylosis

Disc herniation

Ossification of the posterior longitudinal ligament (OPLL)

Subluxation

Cord compression by spinal tumour

Spinal epidural abscess

Spinal epidural hematoma

Hematomyelia

Chiari malformation

Trauma

Non-compressive myelopathies

Spinal cord infarction

Inflammatory and immune myelopathies

Multiple sclerosis

Rheumatoid arthritis

Neuromyelitis optica

Systemic lupus erythematosus

Post-infectious myelitis

Acute infectious myelitis

Radiation myelopathy

Infection (disc space or osteomyelitis)

Amyotrophic lateral sclerosis (ALS)

Chronic myelopathies

Vascular malformations of the cord or dura (dural AV fistula)

Retrovirus-associated myelopathies (HTLV-1, HIV)

Syringomyelia

Subacute combined degeneration (vitamin B deficiency)

Hypocuric myelopathy

Tabes dorsalis

Other

Congenital anomalies of the atlantoaxial joint (dwarfism, Down syndrome, odontoid hypoplasia)

Malformations of the occipital bone (basilar invagination)

Klippel-Feil syndrome

Os odontoideum

Familial spastic paraplegia

Adrenomyeloneuropathy



can be accounted for. In addition, MRI provides good visualization of the spinal cord itself, and spinal cord injury can be visualized. Signal change within the cord (myelomalacia) is an indication of damage (Figure 1), and is most diagnostic when T1 hypointensity correlates with T2 hyperintensity.²⁸ Finally, MRI can also distinguish between CSM and other soft-tissue causes of myelopathy, such as intradural tumours, vascular lesions, multiple sclerosis or syringomyelia.

Other investigations are available to help support the diagnosis of CSM, although their utility is somewhat redundant, especially in the setting of a clinically myelopathic patient with an MRI indicative of cervical canal stenosis with concurrent cord injury. Nerve conduction studies or EMG can help to differentiate CSM from some of the neurodegenerative diseases like ALS that present similarly. Lumbar puncture and analysis

of cerebrospinal fluid (CSF) can indicate infectious, neoplastic, or inflammatory conditions as a cause of myelopathic symptoms. Specialized imaging tests such as positron emission tomography (PET) and angiography can also differentiate neoplastic or vascular causes. The utility of these investigations increases in the setting of clinical myelopathy and an MRI that demonstrates little or no spinal cord compression.

Management

There has been debate in the literature over the best way to manage patients with CSM. It has been noted that the natural history of patients with early CSM is not always one of rapid deterioration. Some studies have shown that patients tend to have deterioration to a certain point, and then plateau and remain stable for years, with truncated deterioration periodically.^{31,32} However, a recent review

of the literature on the natural history of CSM estimated that 20–60% of patients will deteriorate over time if treated conservatively, and a greater extent of spine compression predicts a higher chance of deterioration.³³ It was also pointed out that a large number of patients who initially elect to undergo conservative manage-

Figure 2: MRI demonstrating the extent of degenerative disc herniation



Figure 2A

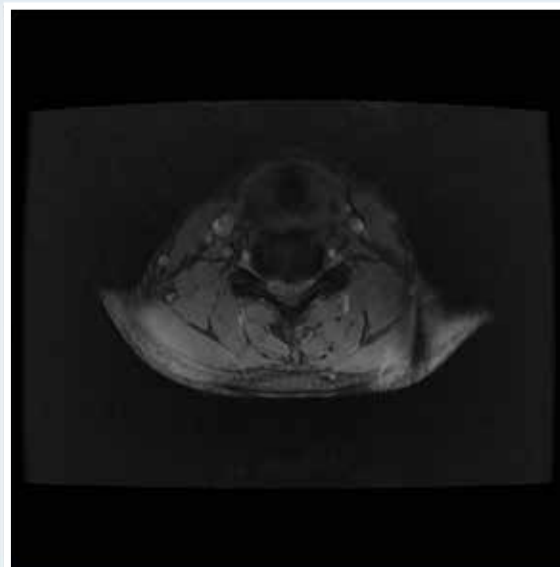


Figure 2B



ment will eventually deteriorate enough to require surgery, with increased vertebral mobility and loss of lordosis predicting a need for surgery. Thus, it has been recommended that patients be given a surgical option, even in cases of mild myelopathy or asymptomatic spondylosis, given the unpredictability of deterioration and the possibility of deficits remaining despite surgery once they have appeared.³⁴

The surgical treatment for CSM is focused on decompression of the spinal cord to prevent further damage from occurring. As mentioned, decompression will not always reverse symptoms that are already present, and this must be kept in mind when recommending surgery to patients. If there is inherent or predicted instability following decompression, then fusion procedures may be required to restore stability. Decompression and fusion can be accomplished both anteriorly and posteriorly, with each approach have its benefits and drawbacks.

Anterior cervical decompression involves removal of intervertebral discs and/or vertebral bodies, and fusion across the removed structures with either the patient's own bone or cadaveric bone and instrumentation with a plate and screws (Figure 3). A meta-analysis of studies looking at anterior surgical treatment for CSM found that benefits were maintained for up to 15 years,³⁵ especially for

Figure 3: MRI showing anterior decompression involving removal of intervertebral discs



Figure 3A



Figure 3B



Figure 3C

This figure is the same case as Figure 1.



patients suffering from more severe myelopathy. In cases of multi-level compression, disc removal with plate fixation was equivalent to removal of the intervening vertebral body and plate fixation with regards to fusion rates.³⁶ A small percentage of patients will deteriorate immediately after surgery, ranging from 2 to 5% in the literature. A cause of deterioration is not always identified, but when present, it is usually due to graft failure or retraction injury.³⁷

When approaching posteriorly, decompression can be achieved by removing the laminae and posterior ligaments that form the roof of the spinal canal (Figure 4). This opens up the dorsal portion of the canal, giving the spinal cord more room to avoid anterior compression, and directly removes any sources of posterior compression present. This expansion can be achieved via laminectomy or laminoplasty. Fusion is not always necessary if the decompression is limited to one or two levels, the spine maintains a lordotic curvature and the facet joints are left intact, but the risk of post-operative spondylolisthesis or kyphosis may still be as high as 43% when fusion is not done.³⁸ Some authors have advocated decompressing two levels above and below the level of stenosis,³⁹ but this is not always done in practice today. There is a very low incidence of neurologic worsening with the pos-

Figure 4: MRI demonstrating that decompression can be achieved by removing the laminae and posterior ligaments



Figure 4A



Figure 4B



Figure 4C

This figure is the same case as Figure 2.





SUMMARY OF KEY POINTS

Cervical spondylotic myelopathy is a degenerative disease that results from compression of the spinal cord with subsequent cord injury and impaired conduction along the tracts contained within it.

Myelopathy is a clinical diagnosis based on signs and symptoms of spinal cord dysfunction and should not be used to refer to isolated imaging findings of spinal cord degeneration or stenosis.

MRI is the most sensitive test to identify cervical canal stenosis and injury to the cord and should be arranged when myelopathy is found on clinical evaluation to identify a specific diagnosis and guide management.

Surgical decompression can prevent progression of cervical spondylotic myelopathy, and in some patients improve gait and hand function.

terior approach, but the incidence or radicular symptoms following surgery approach 7%,⁴⁰ and is generally attributed to C5 palsy from retraction on the C5 nerve roots as the cord moves posteriorly following decompression. These symptoms are generally self-limiting, but patients with more severe deficits pre-operatively may take longer to recover.⁴¹

There has been long-standing debate over which approach is superior in the treatment of multi-level CSM. In general, no approach has been shown to be superior over the other to date. A recent systematic review conducted by Lawrence *et al.* (2013) has upheld this finding, and recommended tailoring surgical approach to the individual patient's pathology.⁴² In general, anterior disease is treated from an anterior approach when three or fewer levels are affected, in part due to the finding of higher rates of late deformity

in patients undergoing laminectomy.³⁶ The posterior approach is often reserved for patients with extensive disease (> 3 levels) or with primarily posterior pathology, such as ligamentous hypertrophy and infolding. However, the lack of high quality studies makes firm recommendations impossible, and a randomized controlled trial is currently underway to attempt a conclusive answer.⁴³

Outcomes following decompressive surgery in CSM can be disappointing, particularly for patients who maintain an expectation of complete recovery. Rates of neurologic improvement are low, and the goal of surgery is often to prevent progression of myelopathy rather than to reverse findings already present. Worse outcomes will often be seen in patients of advanced age, in those who have had symptoms for a greater duration of time, and in patients with a higher





CME

Post-test Quiz

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level of pre-operative disability.⁴⁴ Some patients will have neurologic improvement post-operatively, and a small percentage will remain stable for a period of time followed by late deterioration requiring further surgery.⁴⁵ Patients who have undergone anterior decompression are at risk of developing instability and/or degeneration at levels adjacent to fusion, but the incidence of clinical adjacent segment disease requiring re-operation is low⁴⁶ and there is debate as to whether this instability is progression of the disease as opposed to the fusion. Some more recent evidence has suggested improvement in a number of quality of life assessments following both anterior and posterior approaches for CSM, providing some hope that surgery may actually improve the patients experience.⁴⁷

Summary

Cervical spondylotic myelopathy is a degenerative, and is therefore a relatively common cause

of disability in the aging population. Diagnosis of CSM requires and understanding of its presenting features: upper motor neuron findings and sensory disturbances in the extremities, with gait disturbance secondary to spasticity being one of the earliest presenting features. Multiple imaging techniques can be employed to differentiate CSM from other etiologies not related to spinal degeneration, but MRI is the test of choice. Once diagnosed, patients should be given the option to undergo surgical decompression of the spine, even in mild cases, as surgery will not always reverse the deficits of spinal compression and conservative treatment will likely lead to deterioration that may not be reversible. At present, anterior and posterior approaches for surgical decompression are equivalent and can halt progression of myelopathy, but a small number of patients will experience post-operative deterioration regardless.



CLINICAL PEARLS

Cervical myelopathy can be differentiated from radiculopathy on clinical exam by the presence of upper motor neuron signs as a result of injury to the spinal cord, which will be absent in radiculopathy.

MRI is helpful in working up cervical spondylotic myelopathy as it allows visualization of the elements causing compression, provides an estimate of the extent of stenosis through loss of CSF space surrounding the cord, and allows identification of cord injury manifest as hyperintense signal change in the cord on T2 weighted imaging.

Patients with symptomatic cervical myelopathy should be referred to a spine surgeon for evaluation and management.



References

1. Cooper P R. Cervical spondylotic myelopathy. *Contemp Neurosurg* 1997; 19(25): 1–7.
2. Payne E, Spillane J. The cervical spine, an anatomico-pathological study of 70 specimens (using a special technique) with particular reference to the problem of cervical spondylosis. *Brain* 1957; 80: 571–596.
3. Wilkinson H A, LeMay M L, Ferris E J. Clinical-radiographic correlations in cervical spondylosis. *J Neurosurg* 1969; 30: 213–218.
4. Adams C B T, Logue V. Studies in cervical spondylotic myelopathy II: the movement and contour of the spine in relation to the neural complications of cervical spondylosis. *Brain* 1971; 94: 569–586.
5. Breig A, Turnbull I, Hassler O. Effects of mechanical stress on the spinal cord in cervical spondylosis: a study on fresh cadaver material. *J Neurosurg* 1966; 25(1): 45–56.
6. Turnbull, I M. Microvasculature of the human spinal cord. *J. Neurosurg* 1971; 35: 141–147.
7. Parke W W. Correlative anatomy of cervical spondylotic myelopathy. *Spine* 1988; 13(7): 831–837.
8. Bernhardt M, Hyne R A, Blume H W et al. Current concepts review: cervical spondylotic myelopathy. *J Bone Joint Surg Am* 1993; 75(1): 119–128.
9. Fukui K, Kataoka O, Sho T et al. Pathomechanism, pathogenesis, and results of treatment in cervical spondylotic myelopathy caused by dynamic canal stenosis. *Spine* 1990; 15(11): 1148–1152.
10. Lee M J, Cassinelli E H, Riew K D. Prevalence of cervical spine stenosis: anatomic study in cadavers. *J Bone Joint Surg Am* 2007; 89(2): 376–380.
11. Gore D R, Sepic S B, Gardner G M. Roentgenographic findings of the cervical spine in asymptomatic people. *Spine* 1986; 11(6): 521–524.
12. Boden S D, McCowin P R, Davis D O et al. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. *J Bone Joint Surg Am* 1990; 72(8): 1178–1184.
13. Wu J C, Ko C C, Yen Y S et al. Epidemiology of cervical spondylotic myelopathy and its risk of causing cervical spinal cord injury: a national cohort study. *Neurosurg Focus* 2013; 35(1): E10. doi: 10.3171/2013.4.FOCUS13122.
14. Northover J R, Wild J B, Braybrooke J et al. The epidemiology of cervical spondylotic myelopathy. *Skeletal Radiol* 2012; 41: 1543–1546.
15. Laroche M, Moulinier L, Arlet J et al. Narrow cervical canal and lumbar canal. Frequency of the association, role of hyperostosis. *Rev Rhum Mal Osteoartic* 1991; 58: 853–856. Article in French.
16. Moore A P, Blumhardt L D. A prospective survey of the causes of non-traumatic spastic paraparesis and tetraparesis in 585 patients. *Spinal Cord* 1997; 35: 361–367.
17. Emery S A. Cervical spondylotic myelopathy: diagnosis and treatment. *J Am Acad Orthop Surg* 2001; 9: 376–388.
18. Montgomery D M, Brower R S. Cervical spondylotic myelopathy: clinical syndrome and natural history. *Orthop Clin North Am* 1992; 23(3): 487–493.
19. Lunsford L D, Bissonette D J, Zorus D S. Anterior surgery for cervical disc disease part 2: treatment of cervical spondylotic myelopathy in 32 cases. *J Neurosurg* 1980; 53: 12–19.
20. Young W F. Cervical spondylotic myelopathy: a common cause of spinal cord dysfunction in older persons. *Am Fam Physician* 2000; 62(5): 1064–1070.
21. Glaser J A, Curé J K, Baily K L et al. Cervical spinal cord compression and the Hoffmann sign. *Iowa Orthop J* 2001; 21: 49–52.
22. Houten J K, Noce L A. Clinical correlations of cervical myelopathy and the Hoffmann sign. *J Neurosurg Spine* 2008; 9: 237–242.
23. Ono K, Ebara S, Fuji T et al. Myelopathy hand: new clinical signs of cervical cord damage. *J Bone Joint Surg Br* 1987; 69(2): 215–219.
24. Nemani V M, Kim H J, Piyaskulkaew C et al. Correlation of cord signal change with physical examination findings in patients with cervical myelopathy. *Spine* 2014; 40(1): 6–10.
25. Hukuda S, Mochizuki T, Ogata M et al. Operations for cervical spondylotic myelopathy: a comparison of the results of anterior and posterior procedures. *J Bone Joint Surg Br* 1985; 67(4): 609–615.
26. Chiles B W, Leonard M A, Choudhri H F et al. Cervical spondylotic myelopathy: patterns of neurological deficit and recovery after anterior cervical decompression. *Neurosurgery* 1999; 44(4): 762–769.
27. Singh A, Crockard H A. Comparison of seven different scales used to quantify severity of cervical spondylotic myelopathy and post-operative improvement. *J Outcome Meas* 2002; 5(1): 798–818.
28. Kim H J, Tetreault L A, Massicotte E M et al. Differential diagnosis for cervical spondylotic myelopathy. *Spine* 2013; 38(22S): S78–S88.
29. Rhee J M, Heflin J A, Hamasaki T et al. Prevalence of physical signs in cervical myelopathy: a prospective, controlled study. *Spine* 2009; 34(9): 890–895.
30. Pavlov H, Torg J S, Robie B et al. Cervical spine stenosis: determination with vertebral body ratio method. *Radiology* 1987; 164(3): 771–775.
31. Lees F, Turner J W A. Natural history and prognosis of cervical spondylosis. *Br Med J* 1963; 2: 1607–1610.
32. Nurick S. The natural history and the results of surgical treatment of the spinal cord disorder associated with cervical spondylosis. *Brain* 1972; 95: 101–108.
33. Karadimas S K, Erwin W M, Ely C G et al. Role of non-surgical management in cervical spondylotic myelopathy: pathophysiology and natural history of cervical spondylotic myelopathy. *Spine* 2013; 38(22S): S21–S36.
34. Fehlings M G, Wilson J R, Yoon S T et al. Role of non-surgical management in cervical spondylotic myelopathy: symptomatic progression of cervical myelopathy and the role of non-surgical management. *Spine* 2013; 38(22S): S19–S20.
35. Matz P G, Holly L T, Mummaneni P V et al. Anterior cervical surgery for the treatment of cervical degenerative myelopathy. *J Neurosurg Spine* 2009; 11: 170–173.
36. Mummaneni P V, Kaiser M G, Matz P G et al. Cervical surgical techniques for the treatment of cervical spondylotic myelopathy. *J Neurosurg Spine* 2009; 11: 130–141.



37. Yonenobu K, Hosono N, Iwasaki M et al. Neurologic complications of surgery for cervical compression myelopathy. *Spine* 1991; 16: 1277–1282.
38. Arima T. Change of alignment following laminectomy in the cervical spine. *Brain and Nerve Injury* 1969, 1: 71–78.
39. Epstein J A, Janin Y, Carras R et al. A comparative study of the treatment of cervical spondylotic myeloradiculopathy: experience with 50 cases treated by means of extensive laminectomy, foraminotomy, and excision of osteophytes during the past 10 years. *Acta Neurochir* 1982; 61: 89–104.
40. Chang V, Lu D C, Hoffman H et al. Clinical results of cervical laminectomy and fusion for the treatment of cervical spondylotic myelopathy in 58 consecutive patients. *Spine* 2014; 5(S3): S133–S137.
41. Sakaura H, Hosono N, Mukai Y et al. C5 palsy after decompression surgery for cervical myelopathy: review of the literature. *Spine* 2003; 28(21): 2447–2451.
42. Lawrence B D, Jacobs W B, Norvell D C et al. Surgical treatment of cervical spondylotic myelopathy: Anterior versus posterior approach for treatment of cervical spondylotic myelopathy – a systematic review. *Spine* 2013; 38(22S): S173–S182.
43. Ghogawala Z, Benzel E C, Heary R F et al. Cervical spondylotic myelopathy surgical trial: randomized, controlled trial design and rationale. *Neurosurgery* 2014; 75: 334–346.
44. Holly L T, Matz P G, Anderson P A et al. Clinical prognostic indicators of surgical outcome in cervical spondylotic myelopathy. *J Neurosurg Spine* 2009; 11: 112–118.
45. Ebersold M J, Pare M C, Quast L M. Surgical treatment for cervical spondylotic myelopathy. *J Neurosurg* 1995; 82: 745–751.
46. Chung J Y, Kim S K, Jung S T et al. Clinical adjacent-segment pathology after anterior cervical discectomy and fusion: results after a minimum of 10-year follow-up. *The Spine Journal* 2014; 14: 2290–2298.
47. Fehlings M G, Barry S, Kopjar B et al. Anterior versus posterior surgical approaches to treat cervical spondylotic myelopathy. *Spine* 2013; 38(26): 2247–2252.

