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Cervical spondylotic myelopathy: Make the difficult diagnosis, then refer for surgery

ABSTRACT

Cervical spondylotic myelopathy is the result of narrowing of the cervical spinal canal by degenerative and congenital changes. Prompt surgical treatment is key, but the diagnosis can be difficult because the signs and symptoms can vary widely and there are no pathognomonic findings.

KEY POINTS

Cervical spondylotic myelopathy is the most common type of spinal cord dysfunction in patients older than 55 years.

The onset is usually insidious, with long periods of fixed disability and episodic worsening. The first sign is commonly gait spasticity, followed by upper extremity numbness and loss of fine motor control in the hands.

Surgery is superior to conservative measures. Strong evidence suggests that performing surgery relatively early (within 1 year of symptom onset) is associated with a substantial improvement in neurologic prognosis.

The choice of a ventral vs dorsal surgical approach depends on the relative location of the abnormality (dorsal vs ventral), the alignment of the cervical spine (lordosis vs kyphosis), and patient-specific spinal biomechanics.

CERVICAL SPONDYLOTIC MYELOPATHY is different than many other problems associated with the spine and the back. While conservative medical management is usually the first treatment option for many of these conditions, early surgery is recommended for cervical spondylotic myelopathy. Evidence strongly suggests that performing surgery within 1 year of symptom onset is associated with a substantial improvement in neurologic prognosis.

The challenge is to make the diagnosis, which is often difficult because of the variety of clinical signs and symptoms and the absence of any pathognomonic clinical findings. The onset of cervical spondylotic myelopathy is invariably insidious and commonly involves gait spasticity, followed by upper extremity numbness and the loss of fine motor control in the hands.

PATHOPHYSIOLOGY

Cervical spondylotic myelopathy is the most common type of spinal cord dysfunction in patients older than 55 years and the most common cause of acquired spastic paraparesis in the middle and later years of life.^{1,2}

First defined in 1952 by Brain et al,³ cervical spondylotic myelopathy is caused by narrowing of the cervical spinal canal due to congenital and degenerative changes.⁴ The primary pathophysiologic abnormality is a reduced sagittal diameter of the spinal canal.

Mechanical factors

White and Panjabi⁵ divide the mechanical factors involved in the pathogenesis of cervical spondylotic myelopathy into two groups: static

Ventral spinal cord compression

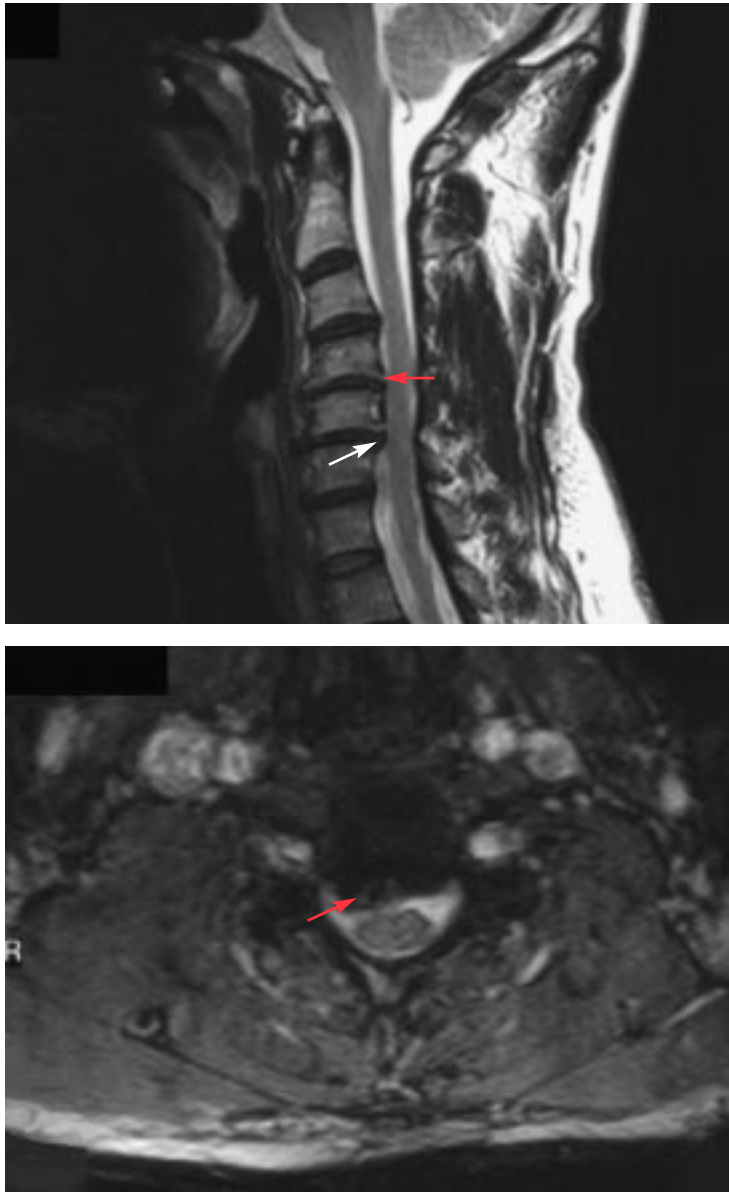


FIGURE 1. Top, sagittal magnetic resonance imaging (MRI) of the cervical spine showing ventral spinal cord compression from disc herniation (white arrow) and vertebral body osteophytes (red arrow). Bottom, axial MRI of same spine showing large right-sided herniated disc (arrow) with reduction in cervical spinal canal diameter.

and dynamic.

Static factors include:

- Congenital spinal canal stenosis (< 13 mm anterior-posterior diameter)
- Disc herniation (**FIGURE 1**)
- Osteophyte formation in the vertebral bodies

- Degenerative osteophytosis of the uncovertebral and facet joints
- Hypertrophy of the ligamentum flavum and posterior longitudinal ligaments.

Dynamic factors are abnormal forces placed on the spinal column and spinal cord during flexion and extension of the cervical spine under normal physiologic loads. An example would be the trauma caused to the spinal cord by repetitively being compressed against an osteophytic bar during normal flexion and extension of the cervical spine.

Ischemia

Mechanical compression of neural elements is only one of the pathologic mechanisms that lead to cervical spondylotic myelopathy. Another is spinal cord ischemia, which happens when degenerative elements compress blood vessels that supply the cervical spinal cord and proximal nerve roots.

Ischemia may result from three mechanisms: direct compression of larger vessels such as the anterior spinal artery, overall reduced flow in the pial plexuses and the penetrating small arteries that supply the cord, or impairment of venous flow, leading to venous congestion.

Pathologic findings that indicate that a vascular mechanism is the cause of cervical spondylotic myelopathy include spinal cord necrosis and cavitation in gray matter. The region of the spinal cord with the highest frequency of cervical spondylotic myelopathy (ie, C5 through C7) is also the area in which the vascular supply is the most tenuous.^{4,6–8}

■ SIGNS AND SYMPTOMS ARE PROTEAN

Cervical spondylotic myelopathy can cause a variety of signs and symptoms, and no one finding is pathognomonic.

The onset is invariably insidious. In the series reported by Brain et al,³ the duration of symptoms ranged from 1 week to 26 years, and almost half of the patients had symptoms for more than 1 year at the time of presentation.

Symptoms of cervical spondylotic myelopathy are protean and can include:

- Pain in the neck, subscapular area, or shoulder.



- Numbness or paresthesias in the upper extremities. These are usually nonspecific, although dermatomal specific sensory complaints can occur from a coexisting radiculopathy.

- Sensory changes in the lower extremities. These are common and typically involve the dorsal columns.

- Motor weakness in the upper or lower extremities.

- Gait difficulties. Many patients with cervical spondylotic myelopathy have a “spastic gait” that is broad-based, hesitant, and jerky,¹ compared with the smooth rhythmic normal gait.

- Myelopathic or “upper motor neuron” findings (ie, spasticity, hyperreflexia, clonus, Babinski and Hoffman signs, and bowel and bladder dysfunction). These often occur within a confusing framework of:

- “Lower motor neuron” findings (eg, upper extremity hyporeflexia and atrophy). The upper motor neuron signs predominate typically below the level of the clinically expressed lesion.

Gorter⁹ reviewed 1,076 cases of cervical spondylotic myelopathy and concluded that a subtle gait disturbance is the most common presentation. Spastic gait occurred first, followed temporally by upper extremity numbness and loss of fine motor control of the hands. Lundsford et al¹⁰ confirmed this presentation pattern in their series as well.

Numbness, decreased vibratory sense, and decreased fine motor control in the hands are common in patients with cervical spondylotic myelopathy. Epstein et al¹¹ found that 55% of their patients with cervical spondylotic myelopathy over age 65 had such symptoms. No specific spinal level accounts for this hand involvement, although it is believed to be associated with dysfunction above the C6-C7 level.¹

Bowel and bladder dysfunction is also common. In a series of 269 patients,¹² bowel dysfunction was observed in 15% and bladder dysfunction was observed in 18%. Epstein et al¹¹ found that 20% of their patients with cervical spondylotic myelopathy over age 65 had bladder dysfunction, mostly associated with urinary retention. Likewise, Lundsford et al¹⁰ found that 50% of their patients had bowel or bladder dysfunction or both.

■ DIAGNOSIS

Keep three facts in mind when contemplating the diagnosis of cervical spondylotic myelopathy^{13–17}:

- It can cause a vast array of signs and symptoms
- There are no pathognomonic findings
- The onset is insidious, with long periods of fixed disability and episodic worsening.

Differential diagnosis is broad

The differential diagnosis of cervical spondylotic myelopathy is quite broad.

It is important to exclude both **multiple sclerosis** (a central demyelinating process with a tendency to cause both motor and sensory abnormalities) and **amyotrophic lateral sclerosis** (which affects both upper and lower motor neurons), as their clinical presentations are similar to that of cervical spondylotic myelopathy. Cervical spondylotic myelopathy does not affect the cranial nerves or the normal jaw jerk reflex, whereas these other disorders may. In addition, amyotrophic lateral sclerosis is a pure motor disease; therefore, sensation is not affected.² Cervical spondylotic myelopathy may have motor findings similar to those of amyotrophic lateral sclerosis, in addition to sensory findings such as numbness or paresthesias in the upper extremities.

Other disorders in the differential diagnosis include spinal cord tumors, syringomyelia, subacute combined degeneration, cerebral hemisphere disease, and peripheral neuropathy. Normal pressure hydrocephalus, with its gait and bladder involvement, should also be considered.

Radiographic studies: MRI most valuable

The diagnostic workup of cervical spondylotic myelopathy often includes cervical radiographs, which may demonstrate osteophyte formation, kyphosis, or subluxation.

The most valuable tool, however, is MRI. Along with the ability to rule out a tumor or syrinx (a slit-like cavity in the spinal cord), MRI allows for specific evaluation of the spinal cord, intervertebral discs, vertebral osteophytes, and ligaments.⁴

Signal changes on T2-weighted MRI images at the level of spinal compression are

The most common presentation of cervical spondylotic myelopathy is a spastic gait

often increased in patients with cervical spondylotic myelopathy. Such findings are thought to represent edema, inflammation, ischemia, myelomalacia, or gliosis. Several studies assessed preoperative and postoperative MRIs in patients with cervical spondylotic myelopathy and correlated the degree of T2-weighted signal changes with subsequent postoperative improvement.^{18–20}

■ ARGUMENTS FOR SURGERY

Strong arguments suggest that, to treat cervical spondylotic myelopathy, surgery is better than medical management such as collar immobilization and traction.

- **Patients treated medically show continual progressive neurologic deterioration.** In a series of 1,355 patients with cervical spondylotic myelopathy treated conservatively, Epstein et al¹⁴ found that 64% showed no improvement and 26% deteriorated neurologically.

In a series reported by Clark and Robinson,²¹ approximately 50% of patients with cervical spondylotic myelopathy treated medically deteriorated neurologically.

Using a disability scale to assess functional status, Symon and Lavender¹⁶ found that 67% of their patients with cervical spondylotic myelopathy experienced progressive deterioration in function.

Similarly, Roberts¹⁷ found that in a series of 24 patients with cervical spondylotic myelopathy, 70% either showed no improvement or deteriorated neurologically with conservative measures. This deterioration was graded by degree of motor disability, measured by the ability to perform daily activities.

- **Patients with cervical spondylotic myelopathy are at an increased risk of spinal cord injury from relatively mild traumatic events.** If the anterior-posterior diameter of the cervical spinal canal is decreased, the spinal cord has limited room to move.⁴ Many traumatic cervical injuries are due to hyperextension, which results in maximal narrowing of the spinal canal.

Epstein et al²² evaluated 200 patients with severe cervical canal stenosis (< 13 mm) admitted over a 4-year period to a spinal cord trauma center. Twenty-three patients had no fracture or dislocation, and in this subgroup there was a direct relationship between smaller

anterior-posterior diameter of the spinal cord and more severe myelopathy after trauma.

Similarly, Firooznia et al²³ described three patients with severe cervical canal stenosis who all became quadriplegic after minor spinal trauma without any fracture or dislocation.

- **Early surgery can improve prognosis.** Montgomery and Brower² found that the prognosis after surgery was better for patients with less than 1 year of symptoms, young age, fewer levels of involvement, and unilateral motor deficit.

Phillips²⁴ examined 65 patients treated surgically and found that symptoms of less than 1 year's duration significantly correlated with benefit from treatment.

Similarly, Ebersold et al²⁵ evaluated several possible predictors of outcome in 84 patients treated surgically. Using the Nurick functional grade, they found that the only significant variable predictive of outcome was how long the symptoms had lasted before surgery.

■ A DORSAL VS VENTRAL APPROACH

There are two surgical options for patients with cervical spondylotic myelopathy: a dorsal approach (ie, cervical laminectomy) or a ventral approach (ie, either discectomy at one or more levels with interbody fusion or one or more corpectomies with interbody fusion). Corpectomy typically involves cervical plating to provide stability until fusion occurs.

Since no clinical study has demonstrated a significant difference in the outcomes of the dorsal vs ventral approaches,^{12,25–31} the choice is based on the surgeon's preference. However, two factors guide this decision:

- **The relative location of the stenosis (ie, dorsal vs ventral).** For patients with cervical spine stenosis that primarily results from dorsal compression, cervical laminectomy (ie, the dorsal approach) is better. This includes patients with dorsal spinal cord impingement from the buckling or enfolding of the ligamentum flavum or from facet arthropathy or both.¹³

For patients with ventral disc herniations and osteophytes, however, cervical laminectomy alone does not allow sufficient access to the ventral spinal cord. These patients benefit more from a ventral decompression and fusion procedure.

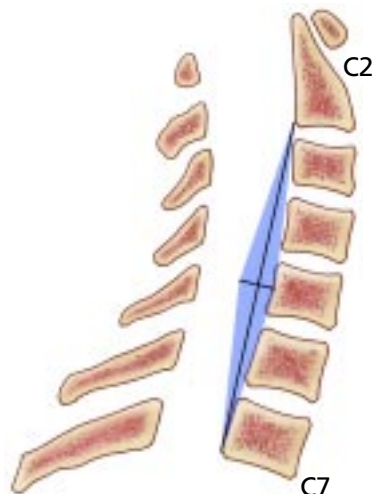
Most patients with cervical spondylotic myelopathy do not improve without surgery



Spine configuration dictates surgical approach

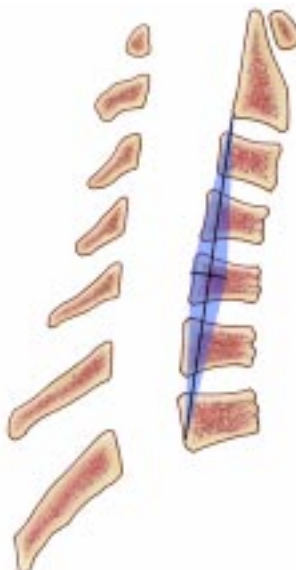
Lordosis

(dorsal approach indicated)



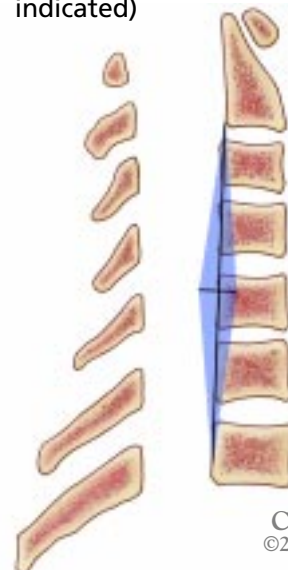
Kyphosis

(ventral approach indicated)



Straight

(dorsal or ventral approach indicated)



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FIGURE 2. Patients with cervical spondylotic myelopathy may present with one of three spinal alignments, which affects the choice of approach for surgical decompression. Using a sagittal magnetic resonance image of the cervical spine, the surgeon may draw a line from the dorsocaudal aspect of the vertebral body of C2 to the dorsocaudal aspect of the vertebral body of C7 and then add a kite-shaped zone, the width of which depends on his or her biases and preferences. If the kite is completely dorsal to the vertebral bodies, the spine is in lordosis, and a dorsal surgical approach is indicated (**left**). If the kite is completely ventral to the dorsal aspects of the vertebral bodies, the spine is in kyphosis and a ventral approach is indicated (**middle**). If the kite is partly dorsal to the dorsal aspects of the vertebral bodies, the spine is considered straight, and either approach is appropriate (**right**).

- **The alignment of the cervical spine** (ie, kyphosis vs lordosis). Effective cervical kyphosis is defined as an alignment of the cervical spine in which any part of the dorsal aspect of any of the C3-C7 vertebral bodies crosses a line drawn in the midsagittal plane (on radiography or sagittal MRI) from the dorsocaudal aspect of the vertebral body of C2 to the dorsocaudal aspect of the vertebral body of C7. In effective cervical lordosis, no part of the dorsal aspect of any of the C3-C7 vertebral bodies crosses this line (**FIGURE 2**).

For patients with effective cervical kyphosis, dorsal decompression is associated with a high probability of failure.^{32,33} In these patients, cervical laminectomy can worsen the ventral spinal cord compression by tethering the dural sac and its contents over ventral osteophytes, which leads to neurologic deterioration.⁴ A dorsal approach in this situation may also lead to progressive kyphotic deformity

and instability requiring repeat surgery and stabilization.

For patients with effective lordosis, a dorsal approach is often optimal for spinal cord decompression, especially when there is dorsal compression. It should be noted that when compression is ventral (eg, herniated nucleus pulposus), the decompression is optimally performed from a ventral approach, even if the cervical spine is configured in lordosis.

Between kyphosis and lordosis is a “gray zone” in which the surgical approach is chosen on the basis of the biases and clinical judgment of the surgeon (**FIGURE 2**).³²

Patient-specific biomechanics should also be considered. Patients should be individualized in regards to the surgical approach chosen. For example, one may consider a dorsal fusion in addition to a large multisegment ventral decompression (corporectomy), even if the spine is not in effective lordosis.





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