CERVICAL SPINAL STENOSIS

John Engstrom, MD
San Francisco, California

Cervical spinal stenosis is defined as an abnormal narrowing of the cervical spinal canal. The essential clinical challenges with cervical spinal stenosis are the following: 1) Narrowing of the cervical spinal canal may or may not result in altered cervical spinal cord function. 2) Narrowing of the cervical spinal canal may or may not be associated with cervical radiculopathy. 3) Narrowing of the cervical spinal canal may be unassociated with a separate pathologic process affecting the spinal cord or other portions of the nervous system.

Cervical spinal stenosis can result from any process that narrows the canal. It can be congenital and usually produces no clinical disease in childhood. When degenerative conditions of the spine are superimposed on congenital stenosis, the adults may begin to show symptoms or signs of degenerative spine disease prematurely (in the 30s and 40s).

We will review the important clinical anatomy of cervical spinal stenosis by reviewing the normal imaging appearance of the cervical spine and then considering the more common contributors to spinal canal narrowing. The T2-weighted sagittal views of the normal cervical spine reveal a very small distance between the posterior margin of the cervical vertebrae and the subarachnoid space. Similarly, the distance from the posterior spinal canal to the bony elements of the posterior spine should also be narrow. Any process extending posteriorly from the vertebral body can focally narrow the spinal canal. Common acquired anterior extradural processes that can narrow the spinal canal include disk herniation, disk-osteophyte complex, or ossification of the posterior longitudinal ligament (OPLL). A posterior spinal process that may narrow the column of CSF posteriorly is calcification of ligamentum flavum. Less common but dangerous causes of acquired spinal stenosis include extension of tumor from the vertebral body posteriorly to compress the spinal cord, epidural abscess, or displaced vertebral fractures. Focal cervical spine instability from other causes will be addressed separately.

When considering the possible contribution of cervical spinal stenosis to cervical myelopathy it is helpful to consider all the anatomic locations that can affect spinal cord function using an “outside-in” approach. In other words, what are the pathological processes that occur in one of three anatomic compartments: 1) Extradural, extra-axial space (outside the cord and dura); 2) Intradural, extra-axial space (outside the cord but inside the dura); 3) Intra-axial space (inside the cord). Many of these anatomic relationships are easily defined by modern MR or CT cervical spine imaging. However, it is often the questionable cases that end up being evaluated by the neurologist.

There are several potentially problematic clinical scenarios in which the neurologist is asked to give an opinion: 1) Cervical stenosis without myelopathy by imaging and no myelopathic findings on neurologic examination; 2) Cervical spinal stenosis with findings of myelopathy by neurologic examination but not abnormal cord signal on MR imaging; 3) Cervical spinal stenosis with abnormal cord signal and no findings on neurologic examination; 4) Cervical spinal stenosis with both abnormal cord signal and findings on examination. The first and last categories are usually the most straightforward, but exceptions exist.

Cervical stenosis without myelopathy by imaging and no myelopathic findings on examination There is no compelling clinical evidence to support prophylactic surgery for asymptomatic cervical spinal stenosis unaccompanied by either abnormal clinical neurologic examination findings supporting the presence of myelopathy or abnormal spinal cord findings on MR imaging. The one potential exception to this rule occurs when there is spinal instability at the stenotic cervical segment. *Dynamic instability* refers to movement of one vertebra upon another during flexion or extension of the neck. If the patient has postural neck pain, a prior history of whiplash injury or other spine/head injury, a Lhermitte sign, or the appearance of listhesis at the stenotic segment on cervical plain x-rays, MRI, or CT, then it can be helpful to obtain cervical flexion-extension x-rays to look for dynamic instability at the stenotic segment. The appearance of listhesis alone, unaccompanied by a change in cervical vertebral alignment during flexion and extension, is not grounds for surgical intervention. An example of cervical spine dynamic instability in the setting of cervical stenosis will be shown and discussed during the session.

Cervical spinal stenosis with findings of myelopathy by neurologic examination but not abnormal cord signal on MR imaging In this scenario, the certainty of anatomic localization to the cervical cord must be questioned and consideration given to a possible brain, brainstem, or thoracic cord localization. The possibility of dynamic instability, as discussed above, should be considered with a low threshold to get cervical spine flexion-extension

x-rays. The possibility of a medical myelopathy not defined by imaging should be entertained. Examples of such medical myelopathies include genetic (e.g.-hereditary spastic paraparesis), infectious (HTLV I/II infection), metabolic (e.g.-B12 deficiency), multiple sclerosis (with bilateral brain plaques and not cervical cord plaques), and connective tissue disorders.

Cervical spinal stenosis with abnormal cord signal and no findings on neurologic examination *The* abnormal cord signal should be juxtaposed to the stenotic segment and not at a different cervical spine level in order to be postulated as related to the spinal stenosis. The possibility of dynamic instability should be considered associated with early injury to the cord that is insufficient to produce neurologic exam findings. The possibility of a process unrelated to the spinal stenosis should be considered. The lack of clinical neurologic exam findings can suggest some other processes: 1) Edema or gliosis may be present following a whiplash injury or demyelinating process. The history may yield a prior history of a car accident, other trauma, or other demyelinating events that resolved over time. A brain MRI will likely show other demyelinating lesions. 2) An infiltrative process separating fiber tracts such as a primary cord glial tumor may be present. Tumors are often associated with increased cord caliber or contrast enhancement at the affected segment. 3) Mass lesions that may not be destructive to the cervical cord, such as sarcoidosis, may occur. Up to 40% of CNS sarcoidosis occurs in the presence of asymptomatic pulmonary sarcoidosis. A chest CT is frequently highly suggestive of the diagnosis and presents the opportunity for a pulmonary tissue diagnosis, possibly obviating the need for a spinal cord biopsy

Cervical spinal stenosis with both abnormal cord signal and findings on examination While this can be a straightforward scenario leading to decompressive cervical spine surgery, exceptions do occur. For example, there may be myelomalacia with abnormal cord signal in the setting of a chronic injury. The subarachnoid space around the cord can expand to “fill the space” left by the atrophic cord. No cord decompression is necessary as the atrophy has already decompressed the cord!

Other clinical issues need to be added to management considerations in this setting including the presence or absence of progressive neurologic deficits, the degree to which symptoms and signs intrude on the patient’s activities of daily living or quality of life, medical comorbidities and other surgical risks, and the degree to which surgical intervention can be expected to arrest progression or enhance functional improvements.

The role of the ligamentum flavum in posterior cervical canal stenosis is less well described than in the lumbar and thoracic spine. Traumatic injury to the cervical spine may injury the ligamentum flavum such that infolding or chronic ossification of the ligament may produce a dark signal on T2-weighted sagittal images posterior to the spinal cord. Alternatively, a dark wedge posterior to the cord can also occur with posterior osteophyte formation.

Are there other diagnostic modalities useful in the setting of cervical stenosis? There are instances in which it would be beneficial to “see” abnormal signal in the cervical cord to go along with abnormal examination findings and apparent spinal stenosis. We frequently obtain CSF flow and diffusion studies. The flow study is based on the pulsatile flow of CSF and the fact that tight stenosis can produce a functional spinal block. The impairment of the imaging equivalent of CSF flow confirms the severity of the block. We also use diffusion which can be abnormal in the setting of cytotoxic injury that is not apparent on a routine cervical spine MRI scan. The location of the signal abnormality also shows the location of the cord injury.

There is interest in using dynamic imaging to reflect the fact that patients often have worse symptoms in a specific posture. Flexion-extension plain x-rays of the cervical spine are a good, inexpensive, and easily obtained study that can show movement of one vertebra on another (listhesis). The limitation of plain cervical spine x-rays is that they don’t image changes with movement in the soft tissues of interest (e.g.-disk, ligaments). For example, there are recent studies that show common infolding of the ligamentum flavum on flexion-extension cervical spine MRI scans. In nearly all instances, the utility of these dynamic imaging studies is hampered by the lack of normal data to tell us what range of anatomic change in these soft tissue spine structures is normal, especially when corrected for age. Expense of the MRI, as opposed to plain x-rays, is also a limiting factor to widespread use of this technique.

The prognosis of cervical spinal stenosis depends upon the function of the patient at the time of diagnosis and upon the underlying etiology. Don’t forget to consider cervical spine flexion-extension x-rays

If there is significant spinal instability seen on cervical spine flexion-extension x-rays, then surgical stabilization is the standard of care. Exclusion of coincident non-compressive causes of abnormal cord signal, with or without myelopathic neurologic exam findings, is the unique job of the neurologist. We owe it to out surgical colleagues, as well as our patients, to “keep them out of trouble” by not sending to spine surgery patients who have no reasonable expectation of benefit!

References

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