CERVICAL SPONDYLOTIC MYELOPATHY

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Cervical spondylotic myelopathy is the most common cause of cervical spinal cord compression in adults > 50 years of age. The term reflects the contribution of many structures of the spine that can undergo degeneration with age and results in compression of the spinal cord including disks, facet joints, uncovertebral joints, vertebrae, and ligaments. One fourth of hospitalizations for quadriparesis are due to cervical spondylotic myelopathy.
The onset of symptoms is often gradual and painless, especially in the absence of an accompanying compressive cervical radiculopathy. As the spinal canal narrows, superimposed disk herniation or modest trauma may produce a rapid evolution of myelopathic symptoms and signs. Spasticity of the arms or legs may be associated with bladder incontinence or urinary frequency, gait ataxia, numbness with a sensory level or circumferential numbness of limbs, upper motor neuron weakness in the legs, upper motor neuron weakness in the arms, or lower motor neuron weakness in the arms. A delay in diagnosis often results from the subtle presentation of non-specific symptoms initially that only become apparent when a new symptom (e.g.-urinary incontinence) or functional limitation (compromised gait) is noted by the patient or family. On the other hand, a large herniated cervical disk or neck trauma can produce sudden cervical cord compression and neurologic deficits.
Cervical cord compression can result in simultaneous injury to the corticospinal tracts (upper motor neuron signs in the arms) and anterior horn cells (lower motor neuron signs in the arms). The clinical presentation can be reminiscent of Amyotrophic Lateral Sclerosis (ALS; motor neuron disease). When cervical stenosis is modest or does not produce increased cord signal on T2 weighted images, strong consideration should be given to search for weakness in other body regions (thoracic, lumbar and bulbar). Given that chronic partial reinnervation can keep pace with the slow loss of up to half of motor axons in a nerve before weakness is detected on clinical exam, electrophysiologic (needle EMG) evidence of chronic partial denervation with reinnervation in the limbs, thoracic paraspinal muscles and the tongue should be considered strongly. Abnormal spontaneous activity, long duration motor units or reduced recruitment at site remote from the cervical spine is highly suggestive for motor neuron disease. Patients with early motor neuron disease presenting in the cervical region frequently undergo unnecessary spine surgeries in which they are exposed to surgical risk with no reasonable expectation of benefit. The neurologist plays a pivotal role in separating these patients out from those with compressive spondylotic myelopathy.
The same or a related degenerative process causing cord compression may also damage nerve roots (*myeloradiculopathy*). When sensory loss or radicular pain are present, in addition to myelopathy, the diagnosis is usually straightforward. Progressive painless weakness arising from the cervical region and unaccompanied by sensory loss or bladder dysfunction should raise a yellow flag for possible motor neuron disease. Cervical radiculopathy is covered by Dr. Ralph in this course and will not be considered further here.
The imaging modality of choice to demonstrate the anatomic changes of spinal cord compression is MRI. The most common finding is increased T2 signal within the cord at the affected segmental level. When the increased T2 signal is accompanied by low signal on T1- weighted images, the prognosis for favorable recovery is reduced significantly as the combination suggest some degree of cord necrosis.
The standard of care for rapidly progressive neurologic deficits or sudden neurologic deficits at high risk for further neurologic progression (e.g.-spine instability) in this setting is surgical decompression and spine stabilization. External stabilization of the spine with a hard collar or other neck brace is common practice in this context. The rationale is that a newly unstable cervical spine could make the cord injury acutely worse with even normal movements of the neck.
Exceptions to his rule occur. For example, central cord syndrome is one example of a sudden cervical cord injury produced by hyperextension of the neck (whiplash injury in MVA, athletics) that produces myelopathic deficits and abnormal MR imaging at the affected cervical segment. Assuming the cervical spine MRI shows adequate CSF space around the cord and there is no spine instability, conservative management is probably best. The clinical context of the injury, underlying pathophysiology and prognosis of the specific injurious process combined with the abnormal neurologic exam and imaging findings drive rational management. In other words, there is no single approach to management that fits all circumstances.
As many as 20-60% of those with cervical spondylotic myelopathy and severe neurologic deficits at baseline eventually require surgery. Prognosis is adversely affected by severity of symptoms and signs at diagnosis,

duration of the myelopathy, and age. Other possible adverse prognostic factors include smoking, diabetes, and pre-surgical psychological state.

One rational approach to management of cervical spondylotic myelopathy of slow onset with mild-to-moderate deficits is conservative (my opinion). Many patients with milder myelopathic deficits and mild cord compression may never require surgery. A randomized controlled trial of surgery vs. conservative care for patients with mild cervical spondylotic myelopathy at baseline showed modestly better or equivalent outcomes for patients in the conservative care group. In my own practice, I usually provide long term, albeit infrequent (every 6-12 months), follow-up for these patients. I ask screening questions and perform a brief screening exam for cervical myelopathy at the visit. The patients know to call me if new symptoms develop between appointments. I have found progression of cervical myelopathy and referred one patient for surgical intervention out of 20+ patients followed. The clinical management includes the informed participation in the decision-making process by the patient at each visit.

Ossification of the Posterior Longitudinal Ligament (OPLL) is a cause of cervical spondylotic myelopathy linked to single nucleotide polymorphisms in collagen genes and occurs in a significant minority of patients of Asian ancestry. The calcification spans the posterior margins of the vertebral bodies as well as the disk spaces and increases the distance between posterior margin of the vertebra and the subarachnoid space. The full extent of calcification, from rostral to caudal segmental levels, can be detected by a cervical spine CT scan. The length of the OPLL can vary form a single segment, to multiple segments, or even polysegmental with intervening spared segments. The calcification can become frankly exophytic and frankly narrow the spine canal even further. The dense adherence of OPLL anteriorly to the vertebral bodies renders attempted surgical removal of the exophytic bone impractical and of high risk. A wide posterior decompression (removal of both lamina and the spinous process) is the surgical method of choice at UCSF. Fusion may or may not be necessary as continuous polysegmental OPLL can result in auto fusion of the cervical spine-making surgical fusion unnecessary. Images of OPLL will be reviewed during the discussion

When the spine surgery options are being explored, I instruct the patient to ask the surgeon for several surgical approaches, accompanied by a list of pros and cons for each approach. Two large studies found that the surgical complication rate for cervical spondylosis with myelopathy was 6.5-13.4% and in-hospital mortality was 0.39-0.60%. Increasing age was a negative predictor for outcome. The spine surgeons at my institution are reluctant to operate on any patients over 84 years of age. If cervical spine surgery is likely at some point, they recommend proceeding at an earlier age to lower the risk of surgical complications.

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