Thoracic Myelopathy

Myelopathy secondary to spondylosis is less common in the thoracic spine as compared with the cervical or lumbar spines.

Over the last 30 years, 28 patient case reports and 1 cohort of 132 patients with thoracic spondylotic myelopathy have been reported, all of whom were treated surgically.

Barnett et al suggest that compressive myelopathy resulting exclusively from stenotic narrowing of the spinal canal from degenerative spondylosis in the thoracic region is rare in patients not also suffering from a generalized rheumatologic, metabolic, or orthopedic disorder or a history of trauma, and describes 6 such cases. However, Marzluff et al suggest that the condition is not as rare as previously thought, although these authors conceded that thoracic myelopathy is less prevalent than that in the cervical or lumbar regions.

Without further epidemiological research of the disorder, it is difficult to estimate the number of patients that may present with this condition regardless of health care setting. Therefore, an understanding and appreciation of this condition are relevant for chiropractors because (1) Americans use chiropractic services primarily for ameliorating spine-related symptoms, particularly chronic pain, and (2) patients with thoracic myelopathy may present with symptoms that can mimic other types of musculoskeletal disorders and can vary greatly.

Although described as uncommon, thoracic myelopathy is a potential diagnosis for patients with abnormal neurologic findings.

The variable symptoms and general low suspicion level complicate diagnosing thoracic myelopathy, potentially leading to delays in treatment and additional diagnostic tests. In this case report, the neurologic evaluation performed by the chiropractic student intern identified ongoing abnormal neurological signs and, when combined with the patient's history, accurately established an initial working diagnosis of myelopathy. However, a more region-specific diagnosis was not made until after neurological referral and magnetic resonance imaging. Although the sequence of events resulted in an accurate diagnosis, generally speaking, establishing a more precise working diagnosis that includes location is desirable, thus enabling diagnostic imaging to confirm or refute the diagnosis. In this case report, myelopathy may have been localized to the thoracic spine with additional maneuvers, thus establishing a more specific diagnosis.

A stepwise approach that considers the combination of presenting neurological findings can increase the precision of localizing the lesion in spinal disease, thus leading to more accurate diagnosis and treatments. Firstly, clinical findings of myelopathy must be recognized, which may include involvement of one or more neurologic levels, unilateral or bilateral complaints, sensory disturbances, motor weakness, disuse atrophy, spastic tone, hyperreflexia, wide-based gait, poor balance, presence of abnormal reflexes, and absence of superficial reflexes.

Secondly, establish the presence or absence of neurological findings in particular regions. For example, a cranial nerve examination revealing intact cranial nerves with the absence of corticobulbar signs and absent jaw jerk reflex may reinforce the suspicion that the lesion lies below the foramen magnum.

The presence of a scapulohumeral reflex is highest among cervical spine disorders affecting the high cervical region and above, with the reflex center presumed to be located between C1 and C3.16 Thus, the absence of the scapulohumeral reflex may further assist localizing the lesion to below the C1-3

cord segments.

Furthermore, the absence of both Hoffman sign and motor, sensory, and reflex abnormalities in the upper extremities may further imply that the lesion exists below the cervical spine. Moving into the thoracic region, the absence or markedly asymmetrical superficial abdominal reflex response may indicate an interruption in the pathway between the brain and the spinal cord at the thoracic level. The abdominal reflex divides the abdomen into quadrants; stimulation of the skin in each quadrant is performed tangentially toward the umbilicus. An abdominal muscle contraction results in a brisk motion of the umbilicus toward the stimulated quadrant. The absence or marked side-to-side asymmetry above the umbilicus indicates a problem at T8-T10 or above, whereas absence or marked asymmetry below the umbilicus indicates a lesion at T11-T12 or above.

Finally, spondylotic changes in the lower lumber region are not likely to create the upper motor neuron signs seen with myelopathy because the spinal cord terminates in the upper lumber spine in the majority of people. Instead, these changes are likely to result in lower motor neuron signs. Motor weakness and sensory disturbances are common in both situations; however, the myotatic reflexes and plantar response can differentiate the conditions. Hyperreflexia and the presence of an “up-going” toe are associated with upper motor neuron lesions, whereas hyporeflexia and the absence of an “up-going” toe are seen in lower motor neuron lesions.

Future clinical research should investigate the potential role that nonpharmacological, nonsurgical treatments may serve in the management of this condition. Despite having similar etiologies, treatment guidelines for spondylotic myelopathy vary depending of the location in the spine. Nonoperative, nonpharmacological treatment with careful regular monitoring for neurological deterioration is considered appropriate for patients with mild or subtle myelopathy.

Current treatment recommendations for thoracic myelopathy include only surgical approaches. There are no nonsurgical recommendations or case reports for conservative management of thoracic myelopathy. Future research should look to address the gap in clinical information surrounding the appropriate role of nonsurgical treatments for spondylotic thoracic myelopathy.