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Cervical spondylotic myelopathy: Part II: clinical and imaging considerations*

SH Burns, DC, FCCS(C)
SM O'Connor, DC
SA Mior, DC, FCCS(C)

In this, the second of a two part series, we continue to review the recent literature pertaining to cervical spondylotic myelopathy (CSM). Caused by the compromise of the spinal canal resulting from the superimposition of spondylotic changes upon a congenitally narrowed canal, CSM has a predictable radiographic and clinical presentation. The clinical presentation frequently includes both upper and lower motor neuron signs and symptoms. Careful analysis of the plain film images usually reveals a spinal canal measuring 12 mm or less. Additional imaging modalities confirm the diagnosis.

This paper presents the clinical and imaging characteristics underlying CSM and stresses the importance of including CSM in the differential diagnosis of patients complaining of neck and leg dysfunctions.
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KEY WORDS: cervical spondylotic myelopathy, cervical spine, spondylosis, radiology, chiropractic, manipulation.

Dans cette deuxième partie, on poursuit notre révision de la littérature sur la myélopathie vertébrale cervicale. Causée par un canal vertébral trop étroit, lui-même résultant de changements vertébraux dans une région où le canal est déjà compromis, la MRC se caractérise par ses manifestations radiologique et clinique. La manifestation clinique présente fréquemment des signes de lésion aux neurones moteurs supérieurs et inférieurs. Une évaluation attentive de films radiologiques démontrera habituellement un canal vertébral mesurant 12 mm ou moins. D'autres formes d'imagerie confirmeront le diagnostic.

Cette étude présente les caractéristiques cliniques et radiologiques sous-jacentes à la MRC et souligne l'importance d'inclure la MRC comme diagnostic différentiel chez les patients souffrant du cou et des jambes.
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MOTS-CLÉS: myélopathie vertébrale cervicale, colonne cervicale, vertébral, radiologie, chiropratique, manipulation.

Cervical spondylotic myelopathy (CSM) was defined in Part I, as a neurological disease caused by the stenotic encroachment of the cervical spinal cord.[1] The stenosis may contribute to the compression of the cord and its adjacent vascular structures with resulting neuroischemia.

CSM is considered the most common neurological spinal cord disorder after middle age.[2] This paper reviews the literature pertinent to the clinical investigation of this entity. A discussion of the clinical appearance of CSM, its differential diagnosis, as well as imaging techniques useful in its diagnosis is presented.

Clinical appearance of CSM

The clinical appearance of CSM varies, depending upon the degree of impingement and neural damage. In fact, in many geriatrics, mild cervical myelopathy often goes unnoticed, with the signs and symptoms usually being attributed as part of their aging process.[3] Thus, it is imperative to specifically examine for the presence of CSM.

The typical patient is over 50 years of age. There may be a predilection for males, especially those who were manual labourers.[3] Although symptoms are insidious in onset, patients may on occasion, present acutely after some degree of neck trauma. The typical complaint is of unilateral clumsiness and loss of dexterity in the hand, with bilateral numbness in the lower limbs and difficulties with walking or balance. Bowel and bladder signs are rare, except in extreme cases.[2]

The most common presentation is that of an upper motor neuron lesion in the lower limbs co-existing with a lower motor neuron lesion in the upper limbs.[4] However, there may also be upper motor neuron signs in the arms from levels below the cord compression, producing a mixed lesion.[5]

The possibility of a mixed neurological lesion should be kept in mind when examing the upper limb. The most common

* Division of Postgraduate and Continuing Education, Canadian Memorial Chiropractic College, 1900 Bayview Avenue, Toronto, Ontario M4G 3E6.

findings are those of nerve root impingement causing decreased sensation in a dermatomal pattern, with hyporeflexia and some degree of flaccid weakness on the side of nerve root involvement. This may involve any of the C5 through C8 roots, since they are most susceptible to compression from degenerative changes. However, the upper limb reflexes originating from levels below the cord lesion, may be hyperactive so that, for example, there may be hyporeflexia of the biceps but hyperreflexia in the triceps.[5]

Examination of the lower limbs, commonly reveals an ataxic gait, hyperreflexia, spastic muscle weakness and sensory changes. The sensory changes can be quite varied depending upon which sensory pathways are impaired. There may be compression of either the spinothalamic tract, or the posterior columns, or both. These give rise to deficits in contralateral pain and temperature sensation, and ipsilateral position and vibration sense, respectively. The former explains symptoms of decreased pain appreciation, while posterior column destruction accounts for the wide-based ataxic gait.[6]

Pathological reflexes are usually only present in severe and long-standing myelopathy. L'hermitte's sign may be present, giving a sensation of electrical shock in the upper or lower limbs.[7] The plantar response is usually down-going. Hoffman's reflex in the fingers is variable; since it is indicative of upper motor neuron involvement, its presence depends on the level of cord compromise.[5]

Additional symptoms may be present, given the complexity of the neurological pathways in the cervical spine. The spondylotic changes that contribute to CSM may also be the underlying cause of vertebral arterial insufficiency, causing dizziness, nystagmus and nausea during the Wallenburg provocation test.[5] Occasionally patients complain of diplopia, tinnitus, dysphagia, or feelings of separation from their surroundings. These are thought to be secondary to involvement of the sympathetic chain.[2] Table 1 summarizes the clinical signs and symptoms associated with CSM.

Differential diagnosis

CSM must be differentiated from several other diseases which can present with similar symptoms. These include amyotrophic lateral sclerosis, which also commonly has combined upper and lower motor neuron signs, as well as multiple sclerosis, spinal cord tumours, cerebrovascular disease and ossification of the posterior longitudinal ligament (OPLL). Cerebrospinal fluid analysis, as well as magnetic resonance imaging may be helpful in differentiating these entities.[5]

Natural history

The natural history of CSM is important from a prognostic and intervention standpoint. In reviewing the work of Clarke and Robinson,[8] Lees and Turner,[9] Nurick[10] and others, LaRocca[11] has outlined the typical course of CSM. He concludes that most cases progress in a series of episodes with aggregate signs and symptoms. The clinical course is typically slow, with periods of partial, but never complete remission. Patients may remain functional for several years but occasionally deteriorate rapidly,

TABLE 1 SUMMARY OF CLINICAL SIGNS AND SYMPTOMS IN CSM

	SYMPTOM	SIGN	MECHANISM
UPPER LIMBS	– hand numbness or clumsiness	– decreased light touch	– root or cord impingement
	– weakness in arm/hand	– *flaccid or spastic weakness	– root or cord compression (LMN or UMN)
	– hand 'restless' or 'tight'	– hyperreflexia	– descending inhibition decreased
	– hand/arm feels 'dead'	– hyporeflexia	– loss of motor power
LOWER LIMBS	– trouble with walking/balance	– stooped, wide-gait	– posterior column damage
	– decreased appreciation of pain, hot, cold	– decrease to pinprick or temperature	– spinothalamic tract damage
	– legs restless or 'tight' but weak	– hyperreflexia	– loss of descending inhibition of corticospinal tract
OTHER	– tinnitus, diplopia	– usually none	– cervical sympathetic chain involvement
	– nausea, dizziness, TIAs dysphasia	– nystagmus and increase in S/S with Wallenburg provocation position	– VBI secondary to spondylotic compression of vertebral artery

especially if cervical trauma intercedes. It is difficult to predict the rate and extent of progression in any given patient, except to say that those with mild and insidious symptoms will likely remain functional longer than those with rapidly appearing or debilitating deficits. There are few objective criteria on which to identify those who will continue to deteriorate, although increased cervical mobility has been shown to be an indicator of poor prognosis.[12]

Imaging CSM

The imaging modalities utilized in the evaluation of cervical spinal stenosis include plain film x-ray, tomography, myelography, computed tomography (CT) scanning (with or without contrast medium) and magnetic resonance imaging (MRI). Recently, Alker[13] has reviewed these modalities and concurs with others, that CT myelography is the single most important imaging method at present. The CT scan in the axial plane depicts the size and direction of osseous encroachment; while the contrast medium (usually iopamidol or ioherol) demonstrates the neural contents in detail by outlining the cord. Penning, Wilmik, and van Woerden[14] have used CT myelography to measure the cross-sectional area of the spinal canal in subjects with clinical signs and symptoms of CSM. They found that the spinal cord can sustain compressions which reduce its size by up to 30% before symptoms become apparent. The role of MRI will undoubtedly increase in imaging stenotic myelopathies as it becomes more readily available.

Although sophisticated imaging techniques have largely replaced plain film x-ray in the analysis of spinal canal stenosis, these procedures remain expensive and relatively inaccessible to the vast majority of Canadian chiropractors. However, CSM can be suspected from less expensive plain film analysis. Most authors point out that lateral cervical spine radiographs continue to be of value, as a first line assessment of spinal stenosis.[15,16] Not only are they cost effective, but they are easily available to and interpretable by the non-specialist. The role of CT scanning and MRI may be more important in planning surgical approaches, while plain film, combined with clinical findings, is often sufficient to confirm the original impression.

Plain film imaging

In the mid 1950's, Clarke and Robinson[8] specifically identified degenerative joint and disc disease as an etiologic factor in cervical myelopathy. This coincided with Pallis'[17] and Arnold's[18] reports of the significance of cervical canal diameters. Since then, many investigators have attempted to correlate the diameter of the cervical spinal canal, as measured on lateral radiographs, with clinically evident myelopathies.[19,20,21]

Boijsen[22] examined the lateral cervical spine radiographs of 200 generally healthy persons, reporting an average anterior-posterior (AP) diameter of 18.5 mm at the C4 to C7 vertebral levels. The measurements were taken from the midpoint of the back of the vertebral body to the nearest point of the lamina of the same segment. (See Figure 1) This is still the commonly

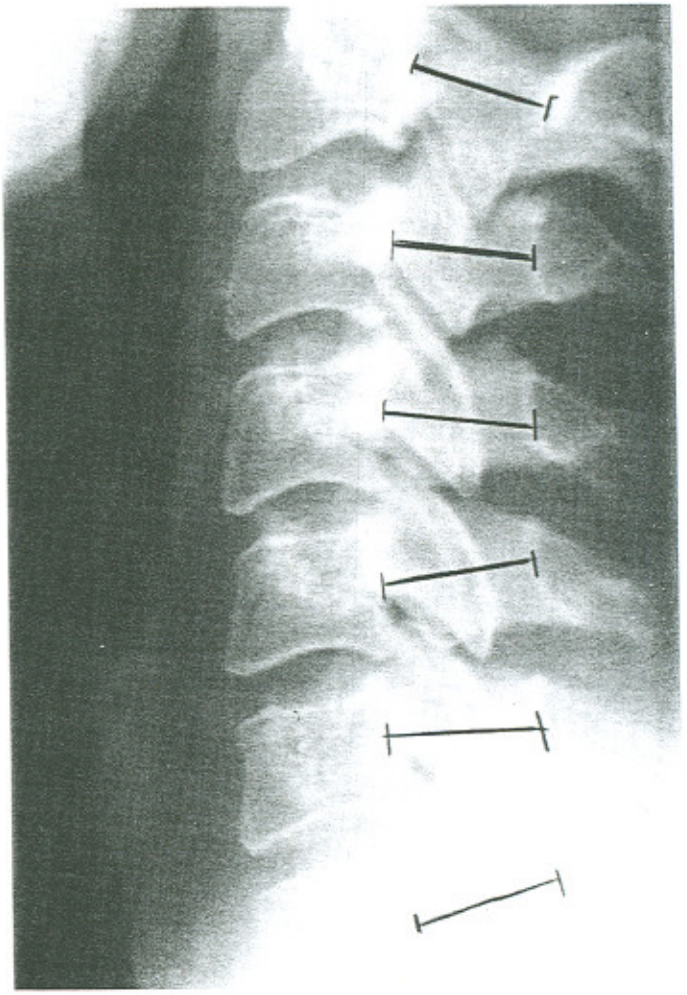

Figure 1 Measurement of the cervical anteroposterior (AP) canal diameter on a neutral lateral projection.

used method. Wolf et al.,[23] in 1956, reported the normal sagittal depth as being 17 mm. The anode-to-film distance used by Wolf, however, was 72 inches, whereas Boijsen used 60 inches. Thus, the reason for the discrepancy was secondary to magnification distortion. The consensus amongst investigators is that the normal sagittal diameter of the spinal canal in the lower (C4–C7) segments in subjects free of bony stenosis is 17 mm plus or minus 3 mm, when using anode-to-film distance of 72 inches.[23,24]

Most authors agree that 14 mm is the low end of normal and that canals measuring less than 13 mm are at risk of developing cord compression.[23] These high risk patients may insidiously develop clinical signs and symptoms later in life subsequent to degenerative changes or present acutely after hyperextension injuries to the neck.[25]

In an attempt to better quantify the sagittal canal diameter,

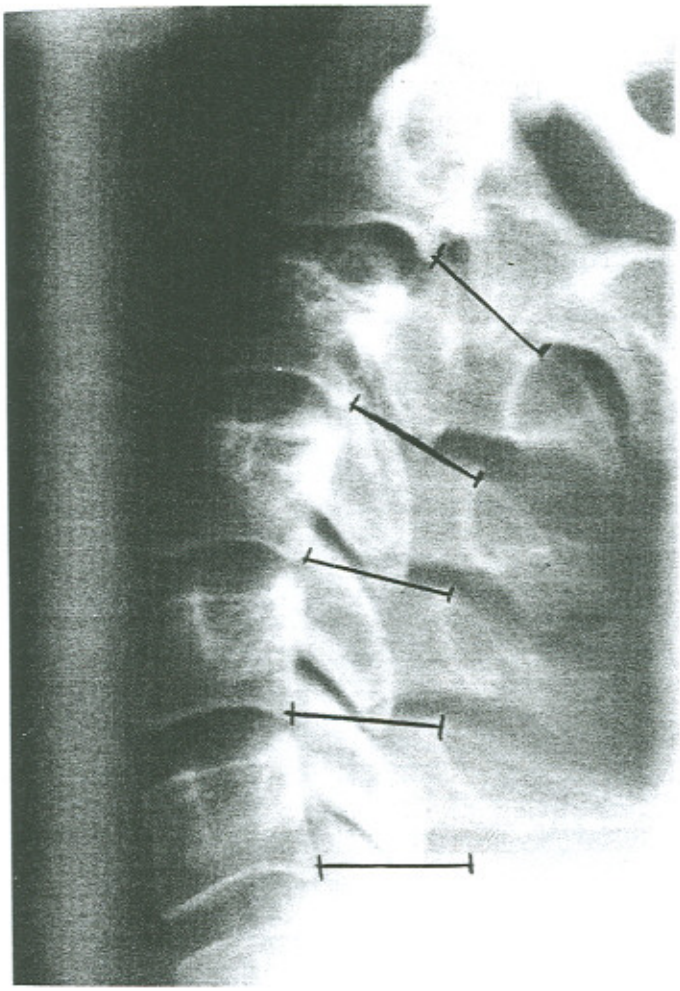

Figure 2 Measurement of the cervical AP canal diameter with the neck in extension.

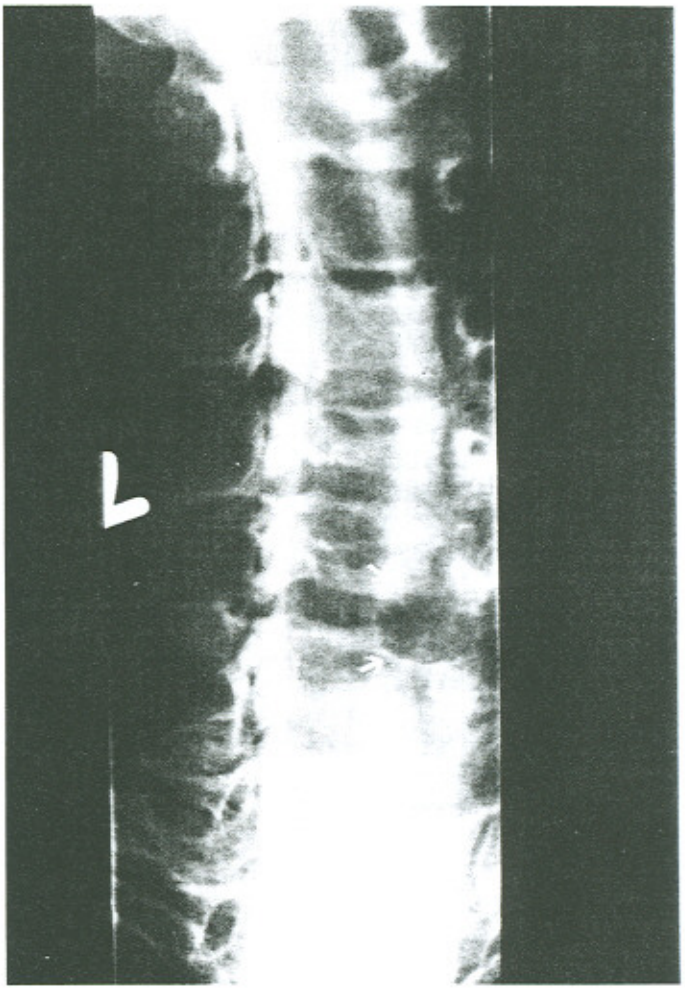

Figure 3 Oblique view of a cervical myelogram using water-soluble contrast material. Note the impression on the cord (arrow) at the C6–C7 interspace.

Pavlov and Torg proposed that the ratio of the AP diameter of the vertebral body to the corresponding AP diameter of the canal may be used.[15] They observed that the vertebral body diameter alone is not related to risk of myelopathy. They state that the body-to-canal depth ratio should be 1:1, and canals measuring 80% of the body depth or less are at risk of spondylotic or traumatic myelopathy. The authors point out two advantages of using this ratio method of analysis. First, false positives do not result from subjects with small necks. Second, and more importantly, no correction factor is necessary for potential image magnification and distortion caused by differences in target distance and object to film distance.[15]

Ehni,[26] studying developmental variations in the cervical spine, confirmed the above findings. He concluded that if a canal was ony 85–90% as deep as the vertebral body, spondylotic myelopathy is a possibility; if the canal is only 80% that of the body, spondylotic myelopathy was probable; and if shallower (50–75%) it was a near certainty.

Considering that the depth of the cervical canal is narrowest in extension, Hayashi et al.[27] emphasize the importance of measuring the sagittal diameters on extension films. However, their method of measurement is somewhat different than that conventionally used. They recognize that the shortest sagittal diameter is usually from the posterior-inferior margin of the vertebra to the superior arch of the next inferior vertebra. (See Figure 2) They recommend that this critical measurement be used as the functional diameter of the canal.[16] Parke[29] supports using this method, not only on the extension view but also on the neutral lateral view.

Myelography

Although plain film radiography is excellent for the depiction of

bone detail and degenerative changes, it does not provide sufficient information about the underlying soft tissue structures. The injection of intrathecal contrast material will demonstrate the dural sac surrounding the spinal cord, as well as the nerve root sleeves. Any compression on the cord due to osteophytes, bulging discs or hypertrophied ligaments will appear as a focal area of thinning of the contrast column. (See Figure 3) Lack of the nerve root sleeve visualization indicates compression on these structures.[28] These changes may become more pronounced with extension of the neck as the ligamentum flavum buckles and compresses the cord even further.[28]

Computed tomography

Computed tomography in the axial plane provides excellent information about the dimension and shape of the spinal canal. Any narrowing of the canal due to posterior osteophytes or hypertrophied facet joints and ligaments can be visualized in detail. Stanley et al.[30] studied the cervical CT images of 52 normal subjects and 80 subjects with neck pain. They concluded that the lower limit of normal for the AP diameter of the cervical spinal canal is 10 mm. This figure is, however, smaller than the 13 mm 'lower limit' for plain films because of the magnification factors with CT.

CT scans of the cervical spine without the use of contrast medium are of limited use, however, as they do not demonstrate the cord and other soft tissue structures to the degree necessary for the evaluation of patients with spinal canal stenosis.[13] The spinal cord is less clearly seen and the nerve roots are not well outlined.

Computed tomography with myelography (CTM) has proven to be invaluable to the assessment of patients with spinal canal stenosis. The CT allows visualization of the bony details, whereas the addition of intrathecal contrast demonstrates the soft tissue in greater detail. (See Figure 4a,b) With this, the radiologist can quantify the degree of stenosis present, as well as qualify which tissue structures are compromised. According to Alker,[13] the concentration of contrast material needed for CTM is less than that needed for conventional myelography and is thus a safer procedure.

Magnetic resonance imaging

Magnetic resonance imaging (MRI) provides detailed information, not only on cord compression, but also on pathology within the cord, such as syrinx, tumour, infarcts and contusions. The AP diameter of the thecal sac and bony spinal canal can be determined from the sagittal images. The MRI is helpful in differentiating bulging or herniated discs, osteophytes, hypertrophied ligaments and degenerative facets by their differing sagittal intensities. With the use of T2 weighted images, the MRI can achieve a myelographic effect, outlining the spinal cord and nerve root.

The benefits of using MRI over CTM are many. CTM requires the use of intrathecal contrast which is an invasive procedure and not without side effects.[30] There are no known adverse biologic risks with MRI. Also, the CT scan exposes the patient to 4.8 to 7 Rads of ionizing radiation, whereas MRI gives no ionizing radiation.[31]

It is apparent that the role of MRI in the evaluation of CSM will increase in the near future. However, at this time MRI remains a relatively expensive procedure and is not readily available in smaller communities.

Treatment

There are several methods of treating patients with CSM, that can be broadly categorized as conservative or surgical approaches. In either case, the goals of treatment are to decrease the patients painful symptoms and to arrest or delay progression of the disease. Long-standing neurological deficit is not amenable to either mode of treatment.

Several factors should be kept in mind when deciding the best method of treatment. CSM is a slowly progressive disease with long intervals of partial remission during which the patient may be relatively confortable and functional.[11] Therefore, non-invasive therapy is the usual first approach. Only in those cases with rapidly increasing neurological deficit or in those involving a recent and sudden increase in signs and symptoms, should surgery be considered.

The objectives of conservative therapy are primarily to provide palliative and preventive care. Modalities such as TENS, short wave diathermy and cervical traction (with the neck in slight flexion), have all been shown to give temporary relief to the radicular and neck pains in some patients.[32] Since many of the transient signs and symptoms in CSM patients are brought on by either flexing or extending the neck, a hard cervical collar is often prescribed.[33] The intention is to restrict movement, thereby limiting the dynamic component of cord compression. Patients managed this way must be monitored from time to time for any change in their neurological or functional status. This is especially true if they suffer any injurious events involving the cervical spine. Occasionally, the older patient with underlying CSM may decline rapidly, following seemingly trivial flexion/extension neck injuries.

Surgical treatment may be unpredictable and frustrating for both the surgeon and the patient. Poor results, even at the hands of a skilled neuro-orthopaedic surgeon, may result from unrealistic expectations based on an over simplified model of the pathogenesis of CSM.[32] CSM involves more than simple mechanical compression from spondylotic changes. Neuroischemia also plays an important role and may cause widespread and diffuse areas of cord necrosis. Necrotic patches are unaffected by surgery and it is unlikely that the operation will have any beneficial affect on the vascular integrity. In fact, it has been proposed that surgery can actually cause increased ischemia by accidentally damaging radicular arteries during the operation.[32]

Keeping in mind the limited ability to influence some aspects of CSM's multi-factorial pathology, surgery has two main objectives: stabilization and decompression. The operation may

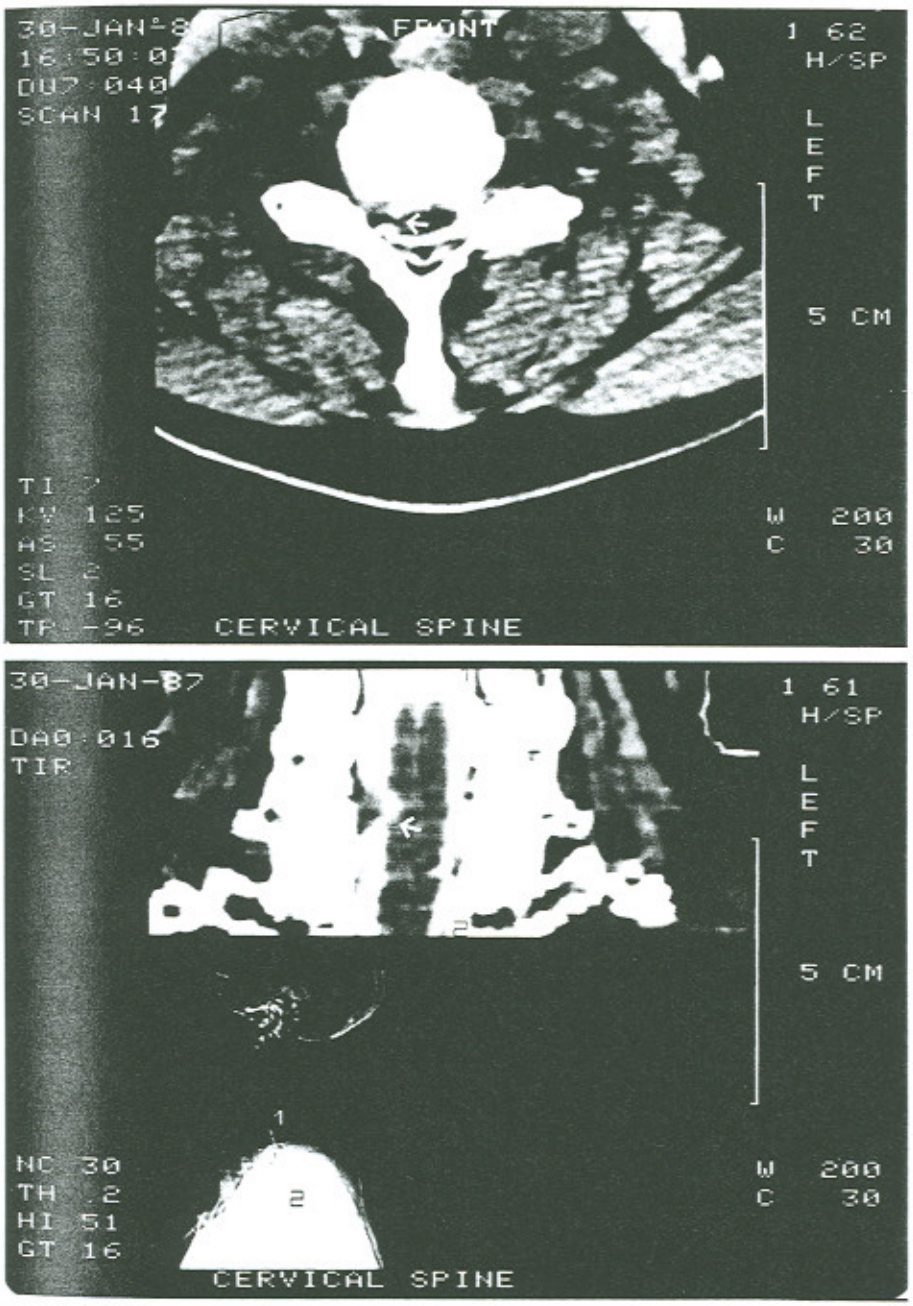

Figure 4 **A** Axial and **B** Coronal computer tomographic images with contrast enhancement (CTM) showing flattening and compression of the thecal sac (arrows) secondary to disc herniation at C6–C7.

attempt to decompress the cord and nerve roots by several methods including foraminotomy, laminectomy, or discetomy. Since the appearance of symptoms is often related to neck movements, and patients with increased cervical mobility have a poorer prognosis,[12] interbody fusion is often performed in an attempt to stabilize the spine and limit progression.

In assessing the success of any mode of treatment one must keep in mind the natural history of CSM, as well as the degree of pre-intervention involvement and the requirements of the patient. An older patient, who is only moderately active and has mild signs and symptoms, may appear to do quite well regardless if he chooses to be or not to be treated. Conversely, a younger and more active patient with high expectations but with more severe or progressive involvement may be unsatisfied with the results of even the most aggressive treatment.

Summary

In summary, CSM is a neurological disease characterized by the compromise of the cervical spinal cord from the narrowing of the spinal canal. This narrowing is frequently the result of the combination of spondylotic changes superimposed upon congenitally narrowed canals. The resultant spinal cord symptoms arise from mechanical and/or vascular dysfunctions. These dysfunctions have predilections for specific areas in the cervical spinal cord. Such affected areas manifest themselves in a clinical picture that is consistent with both upper and lower motor neuron lesions. Careful attention to details in the clinical history and examination will provide the clues that may direct the clinician to consider CSM as a diagnosis. This may then be confirmed by appropriate imaging modalities.

In considering the increasing middle age population and that CSM is the most common neurological spinal cord disorder affecting this age group, the significance of CSM to the chiropractor should be self-evident. However, it is still common to attribute the gait and sensory changes noted in a middle aged patient to 'normal aging' and not seek further explanations. It is hoped, by reviewing the etiology and the clinical presentation of CSM, that due consideration will be given to this disorder.

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References

1 Burns SH, Mior SA, O'Connor SM. Cervical spondylotic myelopathy: part I: anatomical and pathomechanical considerations. J Can Chiropr Assoc 1991; 35(2):82–88.
2 Simeone FA, Rothman RH. Cervical disc disease. The Spine. Philadelphia: WB Saunders Company, 1982: 440–476.
3 Jeffreys E. Disorders of the cervical spine. London: Butterworth, 1980: 90–105.
4 Ferguson RJL, Caplan LR. Cervical spondylotic myelopathy. Neurologic Clinics 1985; 3:373–382.
5 Clark C. Cervical spondylotic myelopathy: history and physical findings. Spine 1988; 13:847–849.
6 Nurick S. The pathogenesis of the spinal cord disorder associated with cervical spondylosis. Brain 1972; 95:86.
7 Epstein N, Epstein J, Carras R. Cervical spondylosis, stenosis and myeloradiculopathy in patients over 65: diagnostic techniques and management. Neuro-Orthopedics 1988; 6:13–23.
8 Clarke E, Robinson PK. Cervical myelopathy: a complication of cervical spondylosis. Brain 1956; 79:483.
9 Lees F, Turner JWA. Natural history and prognosis of cervical spondylosis. Br Med J 1963; 2:1607–1610.
10 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.
11 LaRocca H. Cervical spondylotic myelopathy: natural history. Spine 1988; 13:854–855.
12 Barnes MP, Saunders M. The effect of cervical mobility on the natural history of cervical spondylotic myelopathy. J Neurol Neurosurg Psychiatry 1984; 47:17–20.
13 Alker G. Neuroradiology of cervical spondylotic myelopathy. Spine 1988; 13:850–853.
14 Penning L, Wilmink JT, vanWaerden HH, Knol E. CT myelographic findings in degenerative disorders of the cervical spine: clinical significance. Am J Neuro Rad 1986; 7:119.
15 Pavlov H, Torg JS, Rolic B, Jabre C. Cervical spinal stenosis: determination with vertebral ratio method. Radiology 1987; 114:771–775.
16 Hayashi H, Okada K, Hashimato J, Tuda K, Ueno R. Spondylotic myelopathy in the aged patient: a radiographic evaluation of the aging changes in the cervical spine and etiological factors of myelopathy. Spine 1988; 13618–625.
17 Pallis C, Jones AM, Spillane JD. Cervical spondylosis: incidence and implications. Brain 1954; 17:274–284.
18 Arnold JG. The clinical manifestations of spondylochondrosis (spondylosis) of the cervical spine. Ann Surg 1955; 141:870–889.
19 Burrow GH. The sagittal diameter of the spinal canal in cervical spondylosis. Clin Radiol 1963; 14:77–86.
20 Chrispin AP, Lees F. The spinal canal in cervical spondylosis. J Neurol Neurosurg Psychiatry 1963; 26:166–170.
21 Hashimoto I, Tak Y. The true sagittal diameter of the cervical spine canal and its diagnostic significance in cervical myelopathy. J Neurosurg 1977; 47:12–16.
22 Borjsen L. Cervical spinal canal in intraspinal expansive processes. Acta Radiol 1954; 42:101–115.
23 Wolf BS, Khilnami M, Malis L. Sagittal diameter of the bony cervical spinal canal and its significance in cervical spondylosis. Mt Sinai Hosp 1956; 23:283–292.
24 Payne EG, Spillane JD. The cervical spine. An anatomical pathological study of seventy patients. Brain 1957; 70:38.
25 Starshak RJ, Kass GA, Samaraweena RN. Developmental stenosis of the cervical spine in children. Pediatric Radiology 1987; 17:291–295.
26 Ehni G. Developmental variations, including shallowness of the cervical spinal canal. In: Post MJ (ed). Radiographic evaluation of the spine. New York: Masson, 1980: 469–474.
27 Sayashi H, Okada K, Hamada M, Tada K, Ueno R. Etiologic factors of myelopathy. A radiographic evaluation of the aging changes in the cervical spine. Clinical Orthopaedics and Related Research 1987; 214:200–209.
28 Bland JH. Disorders of the cervical spine: diagnosis and medical management. Toronto: WB Saunders Co., 1987: 64–78.
29 Parke WW. Correlative anatomy of cervical spondylotic myelopathy. Spine 1988, 13:831–837.
30 Stanley JH, Schabel SI, Frey GD, Hangerford GD. Quantitative analysis of the cervical spinal canal by CT. Neuroradiology 1986; 28:139–143.
31 Schnebel B, Kingston S, Watkins R, Dillis W. Comparison of MRI–T1, contrast CT in the diagnosis of spinal stenosis. Spine 1989; 14:00–00.
32 Ehni G. Cervical arthrosis. Diseases of the cervical motion segments. Chicago: Year Book Med Pub 1984: 197–283.
33 Adam CBT, Logue V. Studies in cervical spondylotic myelopathies: III, some functional effects of operations for cervical spondylotic myelopathy. Brain 1971; 94:587–594.