Cervical spondylotic myelopathy – natural history and role of surgery

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Cervical spondylotic myelopathy (CSM) is a chronic, progressive degenerative condition. It is the most common cause of spinal cord dysfunction in patients

over the age of 55 years.[1] The prevalence is increasing owing to the ageing global population.

Epidemiology

CSM is more common in men, and increases in prevalence after the 5th decade. Although spondylotic changes of the cervical spine occur in more than half of patients over 50, less than a quarter of these become symptomatic.[2]

The only proven risk factor for developing CSM is a congenitally narrow spinal canal. Canal diameter is assessed using the Pavlov ratio: canal diameter divided by the AP vertebral body diameter in the sagittal plane. (A ratio of less than 0.82 denotes significant risk.[3])

Pathophysiology

CSM occurs as a result of congenital or acquired narrowing of the cervical spinal canal, with subsequent mechanical and ischaemic insults to the cervical spinal cord and roots. Important factors in the pathogenesis of CSM are listed under these two headings in Table 1.[4]

The pathological endpoints include demyelination and gliosis of white matter tracts, with necrosis and cavitation of grey matter. The cell death and cavitation are irreversible.

Natural history

The natural history of CSM is mixed. Some patients will experience a slow, step-wise deterioration. Others will enjoy long periods of quiescence. A subgroup will see spontaneous improvement in function.

Seventy-five per cent of patients with mild symptoms (sensory changes but no functional disturbance) will remain stable over 5 years.[5]

Clinical presentation

The onset of symptoms in CSM tends to be insidious, with long periods of little or no progression punctuated with episodes of more pronounced deterioration. However, patients may also present following an acute event, such as a hyperextension injury after a fall.

Neck pain or stiffness and gait abnormalities are common initial complaints. Numbness and paraesthesia of the fingers, along with hand clumsiness, are also common.

Upper limb

Motor examination may reveal weakness and wasting of the upper limb musculature (particularly the hand intrinsics). Muscle tone tends to be increased but is dependant on the level of stenosis. Hoffmann's sign and an inverted radial reflex sign may be elicited on examination. Sensory examination may reveal loss, which is often not in a dermatomal distribution.

Lower limb

Muscle tone is increased, plantars are upgoing, and clonus is seen with more advanced disease. Flexor compartment (hip and ankle dorsi flexors) weakness may be evident. Gait tends to be spastic, with a broad-based step and jerky movements.

Sensory function (particularly dorsal column function) tends to be affected. A sensory level may be found at varying levels below the level of stenosis.

Bladder and bowel function

Fifty-three per cent of CSM patients complain of lower urinary tract symptoms and 20% will have neurogenic bladders on urodynamic testing.[6]

Differential diagnosis

As the clinical findings in CSM are varied, the differential diagnosis is very broad. The progressive history and involvement of both motor and sensory function as well as the absence of cranial nerve involvement are key features. Table 2 summarises the differential diagnosis of CSM.

Investigation

Plain cervical spine radiographs provide valuable information. Osteophytes, malalignment (sagittal plane deformity) and canal stenosis (AP diameter less than 13 mm) may be seen. X-rays also help to differentiate between osteophytic and ligamentous structures, which is often difficult on MRI.

MRI (Fig. 1) not only provides information on the cord and surrounding (and potentially pathological and compressive) structures, but may also exclude differential diagnoses. MRI features such as cord oedema, cavitation and axial plane morphology are important prognostic factors.[7]

Table 1. Pathophysiological factors in CSM

Mechanical	
Static	Congenital canal stenosis Cervical disc prolapse Vertebral osteophyte formation Hypertrophic ossification of PLL Ligamentum flavum hypertrophy Facet/unconvertable hypertrophy
Dynamic	Repetitive movements Primarily in sagittal plane Poor cord elasticity
Ischaemic	Compression of larger arteries Decreased pia/medullary flow Venous congestion

PLL – posterior longitudinal ligament.

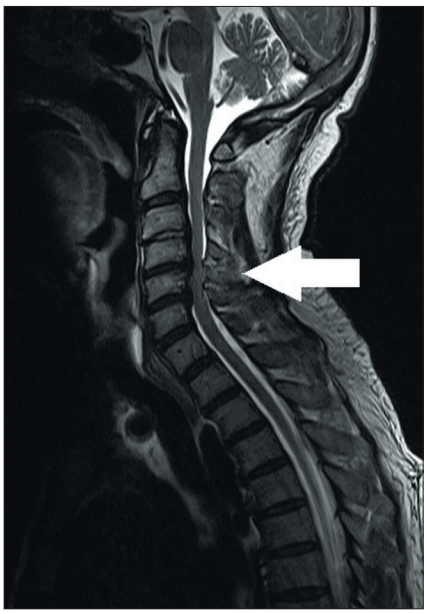

Fig. 1. T2-weighted MR scan of the cervical spine showing significant canal stenosis with cord compression and signal change in CSM.

Table 2. Differential diagnosis of CSM

Congenital	Congenital canal stenosis, Chiari malformation, syringomyelia, kyphosis, os odontoideum
Acquired	
Infective	TB, empyema, syphilis, post-viral, AIDS-related myelopathy, parasitic cyst, vertebral osteomyelitis
Inflammatory	Transverse myelitis, amyotrophic lateral sclerosis, multiple sclerosis, Guillain-Barré syndrome
Traumatic	Fracture, spinal shock, epidural haematoma, basal skull trauma
Tumour	Intramedullary, intradural extramedullary, and extradural spinal tumours (both primary and metastatic), paraneoplastic syndrome
Endocrine	Cushing's syndrome, Paget's disease, obesity
Metabolic	SCDC, local anaesthetic
Degenerative	Spondylosis, OPLL
Vascular	AVM, haematoma (sub- or epidural), spinal cord infarction, radiation necrosis

SCDC – subacute combined degeneration; OPLL – ossification of the posterior longitudinal ligament; AVM – arteriovenous malformation.

Treatment

Conservative versus surgical

Management decision-making should be based primarily on the clinical picture and functional status, rather than on the imaging features. There is no role for prophylactic surgery in the asymptomatic patient.

Non-operative management may be appropriate in the setting of mild CSM symptoms, such as sensory abnormalities involving the hands. While symptoms are unlikely to improve with conservative care, surgery has not been shown to guarantee better outcomes in mild CSM.[8]

Medical treatment comprises cervical spinal immobilisation in a hard collar, anti-inflammatory medication and avoidance of high-risk activities. Patients require ongoing clinical surveillance to detect any neurological deterioration timeously.

Surgical management is recommended in response to functional disturbances such as hand weakness or gait abnormalities. In this setting, surgery has been shown to improve functional status, decrease neurological symptoms and improve overall pain.[9]

Surgery aims to decompress the neurological structures and stabilise any instability. The operative approach is based on the number of vertebral levels involved and the spinal sagittal alignment.

Negative predictors of surgical outcome may include duration of symptoms, advanced age, neurogenic bladder and pre-operative neurological function.[10]

Conclusions

- CSM is the commonest cause of spinal cord dysfunction in the elderly.
- The clinical picture may range from mild hand symptoms to significant functional deficits of both upper and lower limbs.
- Mild CSM has a relatively benign course and may be treated conservatively.
- CSM with functional impairment is best treated surgically.

References

1. Fehlings M, Babak A. Editorial: Surgical management of cervical degenerative disease: The evidence related to indications, impact and outcome. J Neurosurg Spine 2009;11:97-100. [http://dx.doi.org/10.3171/2009.5.SPINE09210]
2. Nagata K, Yoshimura N, Muraki S, et al. Prevalence of cervical cord compression and its association with physical performance in a population-based cohort in Japan. Spine 2012;37(22):1892-1898. [http://dx.doi.org/10.1097/BRS.0b013e31825a2619]
3. Singh A, Tetreault L, Fehlings MG, et al. Risk factors for development of cervical spondylotic myelopathy: Results of a systematic review. Evidence-Based Spine-Care Journal 2012;3(3):35-42.
4. White AA, Panjabi MM. Biomechanical considerations in the surgical management of cervical spondylotic myelopathy. Spine 1988;13(7):856-860. [http://dx.doi.org/10.1097/00007632-198807000-00029]
5. Sumi M, Miyamoto H, Suzuki T, et al. Prospective cohort study of mild cervical spondylotic myelopathy without treatment. J Neurosurg Spine 2012;16(1):8-14. [http://dx.doi.org/10.3171/2011.8.SPINE11395]
6. Fukuda K, Ozaki T, Tsumura N, et al. Neurogenic bladder associated with pure cervical spondylotic myelopathy. Spine 2013;38(2):104-111. [http://dx.doi.org/10.1097/BRS.0b013e318267af02]
7. Chatley A, Kumar R, Jain VK, et al. Effect of spinal cord signal intensity changes on clinical outcome after surgery for cervical spondylotic myelopathy. J Neurosurg Spine 2009;11(5):562-567.
8. Yonenobu K. Is surgery indicated for asymptomatic or mildly myelopathic patients with ossification of the posterior longitudinal ligament? Spine 2012;37(5):E315-E317. [http://dx.doi.org/10.1097/BRS.0b013e318239ccbd]
9. Fehlings MG, Jha NK, Hewson SM, et al. Is surgery for cervical spondylotic myelopathy cost-effective? A cost-utility analysis based on data from the AO Spine North America prospective trial. J Neurosurg Spine 2012;17(Suppl):89-93.
10. Holly LT, Matz PG, Anderson PA, et al. Clinical prognostic indicators of surgical outcome in cervical spondylotic myelopathy. J Neurosurg Spine 2009;11(2):112-118. [http://dx.doi.org/10.3171/2009.1.SPINE08718]