Cervical spondylotic myelopathy

This article outlines the key points in history taking and examination that can lead to the diagnosis of cervical spondylotic myelopathy and the principles of surgical management.

Introduction

Cervical myelopathy is the characteristic pattern of neurological symptoms and signs that occurs as a result of damage to the cervical spinal cord. The underlying pathology that causes damage can be anything from trauma, viral infection, inflammatory or autoimmune disorders, tumours or degenerative processes such as spondylosis and intervertebral disc herniation.

Both spondylosis and intervertebral disc herniation are pathologies dealt with by neurosurgeons, primarily because they cause compressive damage to the spinal cord, which can be treated with surgical decompression procedures.

Cervical spondylosis is a term used to describe the age-related, degenerative changes that affect the cervical spinal column. Radiological evidence of spondylosis is present in 85% of asymptomatic patients above the age of 60 years (Matsumoto et al, 1998). When spondylosis is symptomatic, the classical presentations are of neck pain, radiculopathy or myelopathy.

Cervical myelopathy secondary to spondylosis is a common cause of poor mobility and falls in the elderly population. However, it is often a delayed diagnosis because of its insidious onset. As a result, patients often have severe and permanent disability by the time that they are referred to neurosurgical services. Cervical spondylotic myelopathy is the commonest cause of spinal cord injury worldwide and it is the most common cause of non-traumatic paraparesis or tetraparesis in adults (Moore and Blumhardt, 1997).

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Incidence and prevalence

The exact incidence and prevalence of cervical spondylotic myelopathy is unknown but it commonly occurs in individuals between the ages of 40 and 60 years. The average age of diagnosis is 64 years (Fehlings et al, 2013). It has a higher prevalence in men (3:2) and the most frequently affected spinal levels are C5/6 followed by C6/7 and C4/5. The minimum prevalence of surgically treated cervical spondylotic myelopathy is thought to be around 1.6 per 100 000 (Boogaarts and Bartels, 2015).

Pathophysiology

As the spine is subsequently exposed to increased physiological loads throughout life, this can result in hypertrophy of the facet joints, posterior longitudinal ligament and ligamentum flavum. Osteophytes and bony spurs also develop and vertebral subluxation can occur. The diameter of the cervical canal is reduced and static compression of the spinal cord occurs. Localized areas of narrowing can occur, which may cause compression of a particular nerve root and, therefore, radicular pain.

Dynamic spinal cord compression is also described, whereby normal flexion and extension movements of the neck result in repetitive damage to the spinal cord. The spinal cord stretches in flexion of the neck and thickens in extension (Brieg, 1960). In the context of a narrow cervical canal, such movements may subject the spinal cord to higher intrinsic pressures.

Static and dynamic compression do not entirely explain the mechanism of neuronal damage in cervical spondylotic myelopathy, particularly when patients with equivalent degrees of radiological compression appear to have very different severities of neurological deficit.

Research suggests that a multitude of factors and mechanisms is involved in the pathophysiology of cervical spondylotic myelopathy (Karadimas et al, 2014). Some suggest that the blood supply to the spinal cord is affected as feeding arteries are stretched and compressed by osteophytes. The subsequent ischaemia compounds neuronal damage. The number of small vessels also appears to be reduced, as are endothelial cells. It is felt that the latter propagates inflammation as endothelial cells are the key constituents of the blood– spinal cord barrier and a breakdown in its integrity allows peripheral inflammatory cells to invade the spinal cord parenchyma.

Presentation

Spondylosis manifests as episodes of neck pain and stiffness in the majority of the general population at some point in their lives. When neural compression occurs, this may be that of a single nerve root, therefore causing radicular pain, which in the arms is referred to as brachalgia. This is a characteristic, lancinating pain that radiates into the dermatome supplied by the compressed nerve root. Over time, a lower motor neuron pattern of weakness will arise in the affected myotome with wasting of the affected muscle(s) and a reduced deep tendon reflex.

Patients with myelopathy report a characteristic pattern of symptoms affecting the limbs. In the legs, they describe unsteadiness with subsequent falls at an early stage with progressive difficulty in walking. In those with more advanced disease, a spastic ataxic gait becomes apparent. On direct questioning, patients will admit to their legs jerking spontaneously at night or while sedentary during the day. Upper limb symptoms include numbness in the fingertips along with paraesthesias in the arms. The numbness may progress to a burning

sensation as well as motor deficit, which is often described as poor grip strength or difficulty with fine motor tasks such as doing up buttons or writing. The combination of the motor and sensory hand symptoms often leads to reports that they frequently drop objects such as mugs and pens.

The presence of bowel and bladder dysfunction in cervical spondylotic myelopathy is exceedingly rare and usually only manifests in the most severe cases. Cranial nerve examination should be performed in order to rule out brain or brainstem pathology.

Lhermitte's sign is a sharp, 'electric shock' like pain that runs down the spine and into the limbs on flexion of the neck and is also an occasionally reported symptom.

For the most part, onset and progression is rather subtle and insidious which results in patients and doctors dismissing the early symptoms and signs. As such, greater consideration should be given to cervical spondylotic myelopathy as a cause of falls and poor mobility in the elderly population.

Clinical examination findings are dependent on the extent of disease progression. The general picture is that of an upper motor neuron pattern of weakness below the level of cord compression, i.e. spasticity, clonus, brisk reflexes and upgoing plantars. Sensory and proprioceptive deficits are variable. Romberg's, Hoffman's and the inverted supinator sign may be positive. Lower motor neuron signs can be evident at the level of compression. For example, a C4/5 disc prolapse could result in wasting of the deltoid and a reduced biceps reflex with long tract signs in the lower limbs.

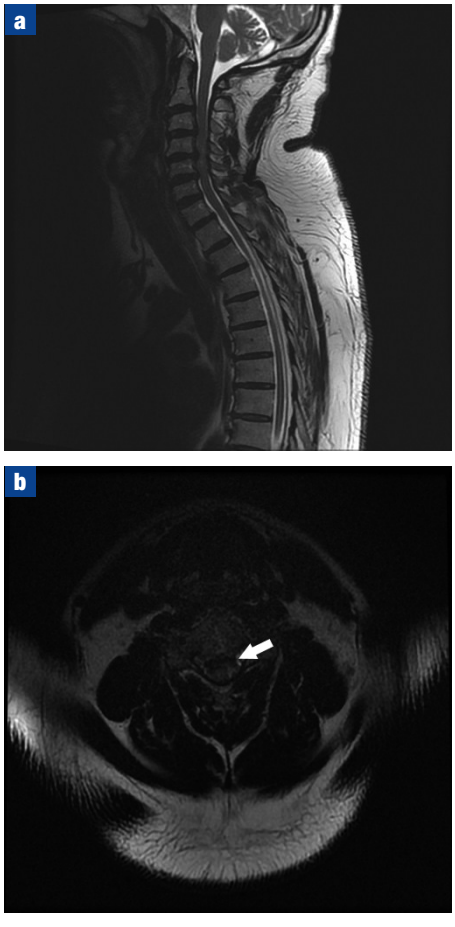
Risk factors

The most significant risk factor identified for the development of cervical spondylotic myelopathy is that of a congenitally narrow spinal canal (Singh et al, 2012). A measure of this is the Torg/Pavlov ratio, which is the ratio of the sagittal diameter of the cervical canal divided by the corresponding diameter of the vertebral body. A ratio of 0.82 or less is indicative of canal stenosis. However, this has little value in clinical practice, as it does not confer any predictive value.

Diagnosis

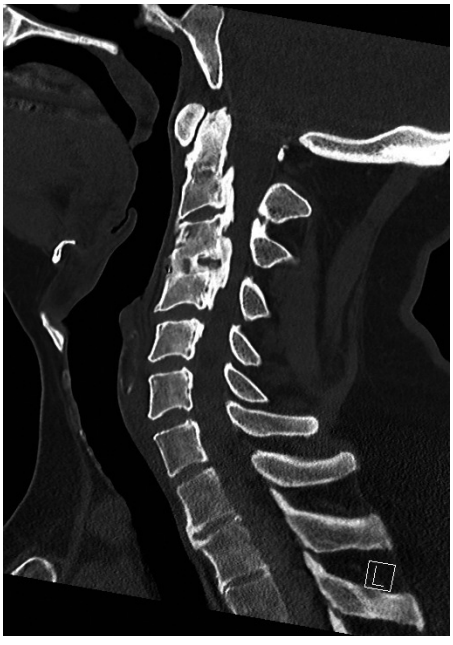
Cervical spondylotic myelopathy is essentially a clinical diagnosis with cervical cord compression being confirmed with magnetic resonance imaging. T1- and T2-weighted sagittal and axial images are obtained and these enable visualization of disc prolapses, facet joint and ligamentum flavum hypertrophy as well as cord oedema (*Figure 1*) (Lebl et al, 2011).

Figure 1. **a.** A sagittal T2-weighted image of the cervical spine demonstrating multi-level disc prolapses at C3/4, C4/5 and C5/6. The C4/5 disc prolapse appears to be the most significant, causing compression of the spinal cord with consequent high signal intensity. Effacement of the surrounding CSF (white) and a milder degree of cord compression is seen at the adjacent levels. **b.** An axial T2-weighted image is used in conjunction to assess the extent of spinal cord compression at the C4/5 level. The disc prolapse protruding posteriorly and causing spinal cord compression is highlighted (white arrow).



Anterior-posterior and lateral cervical spine X-rays are often performed to assess spinal alignment and detect ossification of the posterior longitudinal ligament. In the latter condition, the posterior longitudinal ligament becomes calcified and causes cervical canal stenosis (*Figure 2*). This is important to detect as it influences the surgical approach (i.e. anterior or posterior)

Figure 2. A sagittal computed tomography scan of the cervical spine which shows ossification of the posterior longitudinal ligament. Significant narrowing of the spinal canal is apparent and this results in spinal cord compression at this level.



that the surgeon takes to decompress the spinal cord. Patients in whom ossification of the posterior longitudinal ligament is suspected would go on to have a computed tomography in order to confirm the extent of calcification. However, a computed tomography scan is not a routine investigation in the management of cervical spondylotic myelopathy.

In addition, plain X-rays are used by some surgeons to plan the location of their incision in the neck by looking at where the targeted disc or vertebral body is in relation to the hyoid, thyroid and cricoid cartilage.

Flexion and extension lateral cervical spine X-rays are useful to detect any dynamic compression that may be affecting the spinal cord. Dynamic compression is a consequence of hypermobility in the spine, which may arise in segments adjacent to the stiff spondylotic regions. In cases where there is hypermobility and dynamic compression, the surgeon may adjust his/her surgical approach with the addition of an anterior plate as well as decompression or posterior instrumentation.

A computed tomography myelogram may be performed in patients who have contraindications to magnetic resonance imaging.

Natural history

The natural history of cervical spondylosis is highly variable and it is therefore difficult to predict those patients who will go on to develop myelopathy. In addition, it is equally impossible to know whether patients presenting with relatively mild symptoms of myelopathy will progress to a more severe clinical picture.

A study of 120 patients with cervical spondylotic myelopathy undertaken in 1956 suggested that 75% of patients experienced a step-wise deterioration whereas 20% reported a more steady progression of symptoms (Clarke and Robinson, 1956). In 5%, there was an acute decline followed by a plateau for some time.

In 1963, Lees and Turner looked at long-term follow up for 'myelopathic' and 'non-myelopathic' patients with cervical spondylosis. They summarized that patients presenting with signs and symptoms of myelopathy generally had variable periods of clinical stability interspersed with episodes of decline. The non-myelopathic patients did not go on to develop myelopathy but 15% of patients were lost to follow up (Lees and Turner, 1963).

In 2013, a systematic review of patients with radiological compression without clinical myelopathy suggested that 8% of patients at 1 year and 23% at a median of 44 months would develop clinically evident myelopathy (Wilson et al, 2013).

Most spinal surgeons would agree that patients presenting with even mild myelopathy should be offered surgery as it cannot be predicted when the decline in their symptoms will arise. For patients without myelopathy, factors such as the degree of compression, symptom profile and age of the patient need to be considered. From experience, patients often report symptoms suggestive of myelopathy before the development of clinical signs and with significant radiological compression a discussion should be had with the patient regarding the timing of surgical intervention. For the most part, outpatient follow up or re-referral at the time of developing symptomatic myelopathy would be advised.

Treatment

Non-operative management in the form of analgesia is an option for patients with cervical spondylosis but no symptoms and signs of myelopathy. Physiotherapy aims to improve paraspinal muscle tone and therefore spinal stability but it is not clear whether it affects the progression of spondylosis. In overtly myelopathic patients, surgery should be offered and the severity and speed of progression would determine the timing of intervention. In those with mild symptoms and signs, a discussion should be had with the patient regarding the timing of surgery.

The aim of surgery is to decompress the spinal cord and prevent further neurological damage (*Figure 3*). Therefore, patients should be warned that surgical intervention might not improve their current level of disability.

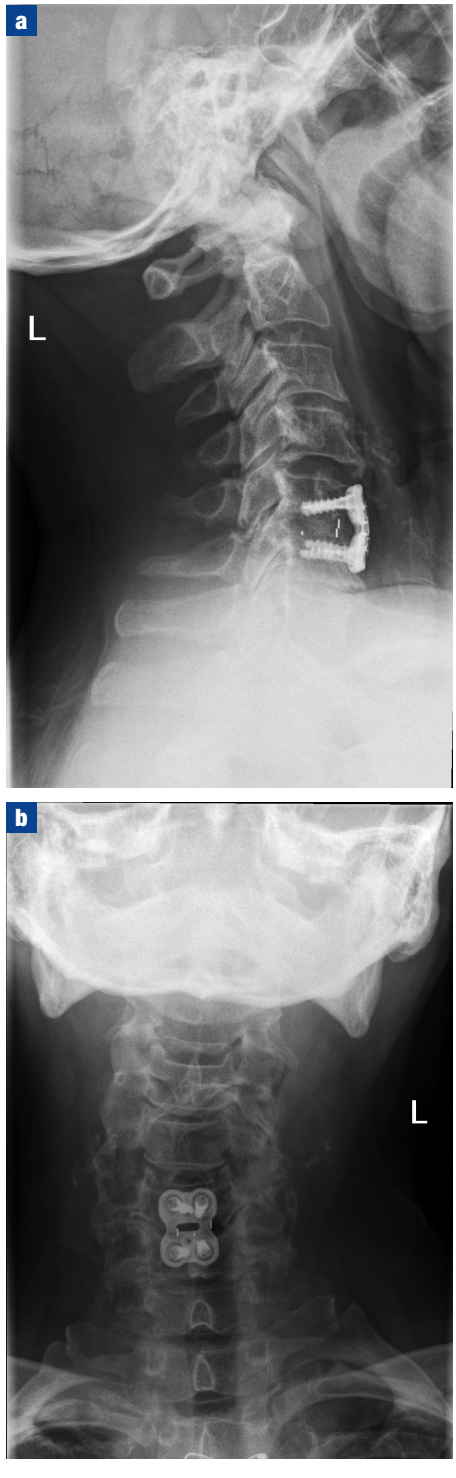
Surgical approaches can be categorised as either anterior or posterior and the chosen approach is dependent upon several factors. The direction of compression is a key factor in decision making. For example, anterior cord compression secondary to disc prolapse or ossification of the posterior longitudinal ligament would prompt an anterior approach to remove the disc (anterior cervical discectomy) or vertebral body (anterior cervical corpectomy). In these latter operations, artificial cages (made of polyether ether ketone, carbon fibre or titanium) are placed in the disc space to maintain height and these can sometimes be packed with bone graft. This will encourage bony fusion to occur over the ensuing months.

However, if ligamentum hypertrophy is a key component of compression a posterior approach in the form of a laminectomy (excision of the lamina) or laminoplasty (the lamina is cut and hinged to an 'open' position) is advised. Posterior instrumented fusion is advised in patients with dynamic instability on preoperative flexion or extension X-rays.

Other factors considered are the patient's age and comorbidities; laminectomy requires prone positioning but is generally regarded as a quicker procedure enabling a shorter duration of general anaesthesia. There is less operative risk compared to an anterior approach where surgery is performed between the carotid artery laterally and the oesophagus and larynx medially. It also possible to achieve a relatively quick, multi-level decompression via the posterior approach. This is often required and preferred in the elderly population.

Anterior and posterior approaches have similar efficacy in the treatment of cervical spondylotic myelopathy (Fehlings et al, 2013), although in this study the anterior approach was preferentially chosen for younger patients. This is often the case because of the potential for post laminectomy kyphosis that can occur following a posterior

Figure 3. **a.** A lateral cervical spine X-ray following an anterior cervical discectomy of the C5/6 disc space with cage and plate fixation. The white lines in the disc space are marking on the cage that has been placed. Note that the anterior plate is curved to maintain normal cervical lordosis. **b.** An anterior-posterior cervical spine X-ray view of the cage and plate.



decompression. This is essentially a form of chronic spinal instability, which can cause late neurological deterioration in up to 47% of patients (McAllister et al, 2012). It is thought to be the result of the disruption caused to the posterior spinal muscles and ligaments in the surgical approach.

In the anterior approach, some surgeons opt to use a metal plate, which is screwed into the cortical bone above and below the excised disc. This improves the rates of fusion and reduce the incidence of cage subsidence (Song et al, 2010).

Following an anterior cervical discectomy and fusion, degenerative changes can be accelerated above or below the operative level in 3% of patients per year (Hilibrand et al, 1999). This is known as adjacent segment disease. Therefore, in patients reporting a new deterioration in their symptoms, a repeat magnetic resonance imaging scan should be performed. Artificial disc replacements were deemed to be a potential solution, but there is no evidence to support their superiority over standard discectomy and fusion (Saavedra-Pozo et al, 2014).

Conclusions

Spondylotic cervical myelopathy is a common cause of falls and poor mobility in the elderly population. As such, a considered history and examination could lead to early surgical decompression and prevent patients from being rendered immobile. Younger patients are also susceptible to such a clinical presentation secondary to an acute disc prolapse. The key investigation is a magnetic resonance imaging scan of the cervical spine in those for whom it is not contraindicated. **BJHM**

Conflict of interest: none.

KEY POINTS

- Cervical myelopathy is a clinical syndrome which may result from a variety of causes.
- Cervical spondylotic myelopathy specifically describes cervical cord compression secondary to degenerative changes in the spine.
- History and examination findings should prompt a magnetic resonance imaging scan of the cervical spine.
- The speed of surgical intervention is governed by the rapidity of symptom progression.
- Surgical decompression may be performed by an anterior or posterior approach depending on the direction of pathology as well as several patient-specific factors.

Boogaarts HD, Bartels RH (2015) Prevalence of cervical spondylotic myelopathy. *Eur Spine J* **24** (Suppl 2): 139–141. https://doi.org/10.1007/s00586-013-2781-x

Brieg A (1960) *Biomechanics of the Central Nervous System. Some basic, normal and pathologic phenomena.* The Year Book Publishers Inc, Chicago

Clarke E, Robinson PK (1956) Cervical myelopathy: a complication of cervical spondylosis. *Brain* **79**(3): 483–510.

Fehlings MG, Barry S, Kopjar B et al (2013) Anterior versus posterior surgical approaches to treat cervical spondylotic myelopathy: outcomes of the prospective multicentre AOSpine North America CSM study in 264 patients. *Spine (Phila Pa 1976)* **38**(26): 2247–2252. https://doi.org/10.1097/BRS.0000000000000047

Hilibrand AS, Carlson GD, Palumbo MA, Jones PK, Bohlman HH (1999) Radiculopathy and myelopathy at segments adjacent to the site of a previous anterior cervical arthrodesis. *J Bone Joint Surg Am* **81**(4): 519–528.

Karadimas SK, Gatzounis G, Fehlings MG (2014) Pathobiology of cervical spondylotic myelopathy. *Eur Spine J* **24**(2): 132–138. https://doi.org/10.1007/s00586-014-3264-4

Lebl DR, Hughes A, Cammisa FP Jr, O'Leary PF (2011) Cervical spondylotic myelopathy: pathophysiology, clinical presentation and treatment. *Hosp for Spec Surg* **7**(2): 170–178. https://doi.org/10.1007/s11420-011-9208-1

Lees F, Turner JW (1963) Natural history and prognosis of cervical spondylosis. *BMJ* **ii**(5373): 1607–1610. https://doi.org/10.1136/bmj.2.5373.1607

Matsumoto M, Fujimura Y, Suzuki N et al (1998) MRI of cervical intervertebral discs in asymptomatic subjects. *J Bone Joint Surg Br* **80**(1): 19–24.

McAllister BD, Renholz BJ, Wang JC (2012) Is posterior fusion necessary with laminectomy in the cervical spine? *Surg Neurol Int* **3**(3): 225–231. https://doi.org/10.4103/2152-7806.98581

Moore AP, Blumhardt LD (1997) A prospective survey of the causes of non-traumatic spastic paraparesis and tetraparesis in 585 patients. *Spinal Cord* **35**: 361–367.

Saavedra-Pozo FM, Deusdara RA, Benzel EC (2014) Adjacent segment disease perspective and review of the literature. *Ochsner J* **14**(1): 78–83.

Singh A, Tetreault L, Fehlings MG, Fischer DJ, Skelly AC (2012) Risk factors for development of cervical spondylotic myelopathy: results of a systematic review. *Evid Based Spine Care J* **3**(3): 35–42. https://doi.org/10.1055/s-0032-1327808

Song KJ, Taghavi CE, Hsu MS, Lee KB, Kim GH, Song JH (2010) Plate augmentation in anterior cervical discectomy and fusion with cage for degenerative cervical spinal disorders. *Eur Spine J* **19**(10): 1677–1683. https://doi.org/10.1007/s00586-010-1283-3

Wilson JR, Barry S, Fischer DJ et al (2013) Frequency, timing, and predictors of neurological dysfunction in the nonmyelopathic patient with cervical spinal cord compression, canal stenosis, and/or ossification of the posterior longitudinal ligament. *Spine (Phila Pa 1976).* **38**(22-1): S37–54. https://doi.org/10.1097/BRS.0b013e3182a7f2e7