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Cervical Myelopathy: Pathophysiology, Diagnosis, and Management

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Abstract

The natural history of cervical myelopathy involves both static and dynamic factors which trigger a cascade of reactions resulting in cord compression and ischemic dysfunction of the spinal cord. Conservative management is an option in those with mild Cervical Spondylotic Myelopathy (CSM), though close clinical and radiological follow-up is important. Operative management remains the standard of treatment for more symptomatic CSM, however, indications must be carefully considered with each individual patient. A randomized, double-blind, placebo-controlled multi-centre study, assessing whether Riluzole (a neuroprotective drugs) has any benefits as a treated for surgical decompression in those with CSM, is being carried out. Here we review the pathophysiological, diagnostic and treatment aspects of CSM.

Keywords: Degenerative cervical myelopathy; Non-surgical treatment; Surgical approach

Introduction

Myelopathy, or dysfunction of the spinal cord, can commonly be caused by a host of conditions including congenital stenosis, degenerative changes, rheumatoid arthritis, and trauma. This debilitating condition is called cervical spondylotic myelopathy (CSM), which reflects that the myelopathy is often associated with and caused by the normal osteoarthritic changes of the axial spine [1]. CSM is part of a range of cervical spine degenerative disorders which include; neck pain syndromes, radiculopathy and myelopathy. These conditions may present with a variety of clinical features, some of which overlap. Cervical spondylotic myelopathy is the most common cause of cervical spinal cord dysfunction in individuals older than 55 [2]. By the age of 40, most will have degenerative changes evident on radiographs [3]. In fact, by age 60-65 years, 95% of asymptomatic men and 70% of asymptomatic women show degenerative changes on plain films [4]. Degenerative changes often begin in the lower segments of the cervical spine, C4-7, but they may present at all levels, especially in the elderly [5]. The current article reviews the evaluation and management of patients who have CSM.

Pathophysiology

The pathophysiology of CSM largely involves compressive forces on the spine, due to a combination of static factors (such as ossification of the posterior longitudinal ligament causing stenosis) and dynamic factors (such as hypermobility causing repetitive trauma). In healthy adults, the intervertebral discs in the cervical spine have a similar structure to that of the lumbar spine, consisting of the annulus fibrosis and nucleus pulposus [6]. Intervertebral discs lose elasticity and hydration, due to loss of proteoglycan matrix, with age which predisposes the disc to collapse as a result of biomechanical incompetence [7]. This may or may not result inherniation of the annulus, causing symptoms. The resulting cervical cord stenosis may be accentuated by hypertrophy and ossification of the posterior facet joints, of the ligamentum flavum, and of the posterior longitudinal ligament (OPLL) [7-9]. Several other changes occur during degeneration such as hypertrophy of the uncinate process and facet hypertrophy which compromise the ventrolateral and dorsolateral portions of the foramen, respectively [10]. Marginal osteophytes begin to develop which can be exacerbated by trauma or additional stresses.

Patients with a congenitally narrow canal (<13 mm) are at a higher risk for developing clinical features from static-mechanical compression [11]. A narrowed spinal canal is thought to cause compression of the spinal cord, leading to local tissue ischaemia, neural cell injury and neurological impairment. Patients tend to become symptomatic if is greater than 30% cord compression, though this can vary between patients [2].

One study showed that cervical myelopathy is strongly suspected when the dynamic canal space during extremes of flexion or extension is (<11 mm) [11]. In conjunction with a pre-existing cervical canal stenosis, there is increased strain and shear pathological forces applied on the spinal cord, which can potentially cause localised and widespread axonal injury [12]. One study measured changes in disc bulge, ligamentum flavumbuldge, and anteroposterior canal diameter in response to tension-compression forces and combined loading forces in the lower cervical spine (C4-7) in five human cadavers [13]. From tension to compression, the average disk bulge changed

1.13 mm or 10.1% of the original canal diameter. The ligamentum flavum bulge changed 0.73 mm or 6.5% of the canal diameter. From flexion to extension the average disk bulb changed 1.16 mm or 10.8% of the canal diameter, whereas the ligamentum flavum bulge changed 2.68 mm or 24.3% of the canal diameter. These results show that neck flexion reduces cord compression by increasing the sagittal diameter whereas neck extension in the presence of a ligamentum flavum bulge will exacerbate stenosis [13].

The vascular aspect of the spinal cord plays an important role in the pathophysiology of CSM. Considerable evidence exists to support ischaemia as a major underlying pathologic event contributing to myelopathy. Anterior compression compromises perfusion through the transverse arterioles arising from the anterior sulcal arteries, while posterior cord compression compromises perfusion through the intramedullary branches of the central gray matter [14]. Oligodendrocyes, cells responsible for myelination of axons, respond poorly to ischaemia and this may explain the demyelination that occurs with chronic cervical myelopathy [15,16].

Clinical manifestations

Cervical spondylotic myelopathy can be divided into distinct syndromes based on clinical presentation **(Tables 1 and 2)** [17,18]. A thorough history, clinical examination and supporting radiological findings are essential in making the clinical diagnosis of CSM.

Table 1 Crandal and Batzdorf classification of CSM.

Syndrome	Clinical features
Brachial cord	Motor (paralysis) and sensory deficits (and pain) in upper extremities
Central cord	Motor and sensory deficits in upper extremities more than in lower extremities
Anterior cord	Spasticity
Brown-Squared syndrome	Ipsilateral motor deficits with contralateral sensory deficits
Transverse lesion	Corticospinal, spinothalmic, and posterior cord tracts are all involved

Table 2 Ferguson and Caplan classification of CSM.

Syndrome	Clinical features
Medial	Long-tract symptoms
Lateral	Radicular symptoms
Combined	Combined medial and lateral syndromes
Vascular	Vascular insufficiency causing a rapidly progressive myelopathy

In the early stages of myelopathy, patients typically complain of classic 'spastic gait' disturbances, which may be stooped, wide-based or jerky [19,20]. Pain is a less frequently noted complaint and its absence may often lead to a delay in diagnosis. It has been strongly suggested that subtle gait disturbance is the most common presentation, followed by loss of fine motor control of the hands with associated numbness [20]. Other authors have also reported similar findings [21]. Numbness or paraesthesia in the upper extremities is usually non-specific. With certain motions of the neck (usually flexion and extension), the patient may describe electric shock-like sensation that extends throughout the body (the Lhermitte sign). Bladder and/or bowel incontinence and quadriparesis usually signify late stage myelopathy and develop because of long tract involvement, resulting in reduced sphincter control, and affect between 15% to 50% of patients [7,22].

It is important to distinguish the symptoms of cervical myelopathy similar clinical presentations such as cervical spondylotic pain or cervical radiculopathy. Cervical spondylotic neck pain is often felt in the posterior aspect of the neck and patients may complain of associated occipital headaches and neck stiffness [7]. Cervical radiculopathy is manifested by proximal pain and distal paraesthesia and may be associated with weakness, however, there should be no lower limb or bladder/bowel symptoms [7].

Examination

Examination of patients who have CSM typically reveals lower motor neuron signs at the level of cervical lesions (weakness and hyporeflexia) and upper motor neuron signs below the lesions (spasticity, hyperreflexia, clonus, Hoffman sign and Babinski sign). Since, CSM most commonly affects the lower cervical spinal cord (C5-7), it is the dermatomes and

muscle groups corresponding to these levels that are most commonly affected **(Table 3).**

Table 3 Clinical examination findings at corresponding cervical levels.

Cord level	Dermatome (sensory)	Action (motor)
C1	None	Neck extension/flexion
C2	Occiput and posterior ear	Neck extension/flexion
C3	Supraclavicular fossa	Neck extension/flexion
C4	Over acromioclavicular joint	Spontaneous respiration
C5	Radial side of antecubital fossa	Shoulder abduction and elbow flexion
C6	Thumb	Elbow flexion and wrist extension
C7	Middle finger	Elbow flexion and wrist extension
C8	Little finger	Finger flexion

A hand dexterity test should be performed such as the '15-second test' where patients are asked to grip and release their fingers as rapidly as possible for 15 seconds [23]. This may prove difficult in those with CSM. The "myelopathy hand" in cervical spondylosis includes findings of localised wasting and weakness of the extrinsic and intrinsic muscles of the hand [24]. The 'finger-escape sign' may also be evident (spontaneous abduction of the little finger due to weak intrinsic muscles).

Mixed upper and lower motor neuron findings may be present in the upper extremities **(Table 1)**. Muscular atrophy may also be present. Sensation may be impaired depending on the level of cord damage. Pain and temperature changes are most commonly noted [7]. Proprioception, vibration, and touch sensations may be impaired on the ipsilateral side to the lesion, whereas pain and temperature sensation will be impaired on the contralateral side due to cord decussation. Reflexes in the upper extremity may be either hypo reflexive or hyper reflexive.

Many disease severity classifications have been used such as the European Myelopathy Score, Nurick's Functional Scale, Ranawat Classification of Neurological Deficit, and the modified Japanese Orthopaedic Association scoring system **(Table 4)** [25]. Although they are helpful in determining severity, they do have limitations.

Table 4 Nurick classification system for myelopathy.

Grade	Level of neurological impairment
I	Normal gait
II	Mild gait involvement. Still employable
III	Gait abnormality prevents employment
IV	Walk with assistance
V	Wheelchair bound or bedridden

Investigations

Plain radiographs (anteroposterior and lateral) of the cervical spine are routine in the assessment of symptomatic cervical spondylosis. Narrowing of the disc space, bone spurs, osteophytes, joint subluxation, facet joint arthrosis, spondylolisthesis, and ossification of posterior longitudinal ligament may be visualized on standard films. An 'absolute stenosis' has been defined as a sagittal canal diameter <10 mm and a 'relative stenosis' with a narrowed spinal canal of less than 13 mm. Flexion-extension views can be used in the radiographic evaluation of subtle cervical instability. Narrowing of the spinal canal (seen on oblique views) can be calculated using Pavlov's ratio. This can be calculated by dividing the AP diameter of the spinal canal by the AP diameter of the vertebral body. A ratio if <0.8 is suggestive of stenosis.

Magnetic Resonance Imaging (MRI) is the imaging modality of choice [26] One study showed cord impingement in 16% of asymptomatic patients under age 64 years and in 26% of asymptomatic patients aged greater than 64 [27]. In those where MRI is contraindicated or not tolerated, contrast-enhanced CT may be used which is just as useful in characterizing compression of the spinal cord.

Electromyography (EMG) and Somatosensory Evoked Potentials (SSEPs) are diagnostic investigations which are infrequently used to exclude differential diagnoses such as

multiple sclerosis, amyotrophic lateral sclerosis, and peripheral neuropathy [28].

Treatment

Conservative

Conservative management is usually for patients with mild CSM or for patients who are unfit for surgery due to other medical comorbidities or personal choice. Treatment options include lifestyle changes, physiotherapy, analgesia, and neck braces. Mild symptoms warrant observation with close clinical and radiological follow-up.

Lifestyle changes involves avoiding activities that exacerbate symptoms, for example, avoidance of heavy lifting [7]. Physiotherapy may involve traction, heat and ultrasound therapy, and may be of use in those who can tolerate it.

Regular analgesia, such as gabapentin or pregabalin, should be considered for patients with pain from radicular symptoms or be used an adjunct to simple analgesics such as NSAIDs. The use of systemic and epidural steroids is controversial.

Despite little evidence for its efficacy as immobilisation, cervical collars or neck braces are commonly used. Immobilisation, however, does not seem to change long-term outcomes [29,30]. In addition, potential adverse effects must be considered with prolonged collar immobilisation such as muscle wasting and reduced range of motion.

Surgical

It is generally accepted that surgical intervention should be offered to patients with progressive disease, intolerable symptoms, or when conservative management is unsuccessful [31]. The primary goal of surgery is cord decompression thus achieving adequate expansion of the cervical canal allowing for improvement or preservation of neurological function. One study showed that two-thirds of patients with a Nurick score of 5 who were either bedridden or in wheelchairs at the time of diagnosis, improved at least one degree on the Nurick scale after surgical management [32].

Surgical interventions can be considered in two anatomical areas; the upper (C0-C2) and lower (C3-C7) cervical spine, and three general approaches; the anterior approach, the posterior approach, and the combined anterior and posterior approach. When considering approach, multiple factors should be addressed, for example; site of compression, number of levels involved, deformity, instability, radiological characteristics, age, comorbidities, lifestyle (smoking), and patient wishes and expectations. Positioning of the patient and documentation of preoperative cervical range of motion is paramount as a tight cervical canal increases the risk of severe neurological injury if excessive extension is achieved during intubation. Thus, discussion between the surgeon and the anesthetist will allow for safe intubation and positioning.

The four surgical procedures commonly performed to treat CSM are: anterior cervical discectomy and fusion (ACDF); anterior cervical corpectomy and fusion (ACCF); laminectomy; and laminoplasty. Regardless of surgical technique, the primary goal remains the same–cord decompression.

Posterior approach

The posterior approach to surgically manage CSM is the most common; this includes two common procedures; laminectomy and laminoplasty. This approach is useful in those with predominantly dorsal compression due to ligamentum flavum infoldings and in those with multi-level disease. There is risk of damage to the spinal cord and vertebral arteries if this approach is used.

Laminectomy has been linked to deterioration of spinal cord function. In addition, post-laminectomy kyphosis has also been reported as a potential complication following posterior decompression at multiple spinal levels [33,34].

Laminoplasty can be a favourable option in lordotic spines in younger patients where fusion is undesirable. The two main laminoplasty techniques are the 'French Door' and the 'Open Door' techniques. One study reported their findings which showed laminoplasty is not an effective treatment for axial neck pain and that axial symptoms may worsen post-procedure [35].

Anterior approach

Historically, the anterior approach, was used for resection of inflammatory pannus in patients with rheumatoid arthritis, although recent evidence suggests that inflammatory pannus resorbs with rigid posterior stabilization. Despite this, it is still relevant to surgery for tumour of the upper cervical spine and is recommended for patients with fixed cervical kyphosis.

The Smith-Robinson approach is the most common anterior approach used to perform a discectomy. This approach carries multiples risks to nerves (recurrent laryngeal, superior laryngeal), arteries (vertebral) and structures (esophagus, trachea, grafts) [36]. For adequate decompression, resection of the posterior longitudinal ligament (PLL) and posterior osteophytes is required. A corpectomy should be performed if there is significant compression.

The aid of posterior stabilization may address the issue of suboptimal stability especially when extensive resection occurs [37]. Autologous bone graft harvested from the iliac crest has been the standard procedure for many years when providing graft or fusion procedures. Due to increased morbidity of such procedures, synthetic bone grafts with a cage (composed of titanium or polyether-ether ketone (PEEK) mesh) are used which allows disc space to be maintained or restored.

Combined approach

In complex cases, especially when there is compression from both anterior and posterior structures, both approaches may be utilized in conjunction with one another.

Medical

A multi-centre, double-blind, randomized, placebo controlled clinical trial is being carried out which evaluates if a neuroprotective drug (Riluzole, a sodium glutamate antagonist) used both pre-and post-surgery would have any benefits in those undergoing surgical decompression for CSM.

As aforementioned, CSM involves both static and dynamic factors with trigger cell ischemia and cell death due to sodium influx and glutamatergic excitotoxicity. Riluzole is currently FDA-approved for the treatment of amyotrophic lateral sclerosis, which have similar clinical features to CSM.

Discussion and Conclusion

Cervical spondylotic myelopathy is a debilitating condition that commonly affects the elderly and occurs as a result of degenerative changes leading to cord compression. It is important to appreciate its pathophysiology, clinical presentation and investigation when considering optimal management. Conservative treatment remains a role in the management of mild CSM but established surgical treatment options appear to yield improvement in neurological function in those with progressive disease or intolerable symptoms. Anterior surgery is often beneficial in patients with stenotic disease limited to few segments whereas posterior surgery allows for decompression of multiple segments.

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