Review Article

Updates in current concepts in degenerative cervical myelopathy: a systematic review

Kiara Thompson[1]^, Hannah Travers[1], Alex Ngan[1,2], Tim Reed[1,3], Shaya Shahsavarani[1], Rohit B. Verma[1,2]

[1]Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, Hempstead, NY, USA; [2]Department of Orthopedic Surgery, North Shore University Hospital-Long Island Jewish Medical Center, Manhasset, NY, USA; [3]Department of Orthopedic Surgery at Huntington Hospital, Huntington, NY, USA

Contributions: (I) Conception and design: RB Verma, K Thompson, H Travers, S Shahsavarani; (II) Administrative support: All authors; (III) Provision of study materials or patients: All authors; (IV) Collection and assembly of data: K Thompson, H Travers; (V) Data analysis and interpretation: K Thompson, H Travers; (VI) Manuscript writing: All authors; (VII) Final approval of manuscript: All authors.

Correspondence to: Dr. Rohit B. Verma, MD. Assistant Professor, Department of Orthopedic Surgery, North Shore University Hospital-Long Island Jewish Medical Center, 300 Community Drive, Manhasset, NY 11030, USA; Donald and Barbara Zucker School of Medicine at Hofstra/Northwell, 611 Northern Boulevard, Suite 200, Great Neck, NY 11021, USA. Email: rverma1@northwell.com.

Background: The incidence of degenerative cervical myelopathy (DCM) has increased over the years due to an increasing aging population, yet there is a dearth of recent comprehensive data evaluating the multiple facets of this degenerative condition. Recent publications have highlighted the biochemistry and biomechanics of DCM, which are paramount to understanding the degenerative nature of the condition and selecting the most optimal treatment options for improved patient outcomes. In addition, there have been recent studies establishing the superiority of surgical to non-surgical treatments for DCM, which until now was a poorly substantiated claim that has permeated the medical field for decades. The authors of this systematic review sought to collect and assess available high quality peer reviewed data to analyze the nature of DCM and gain a better understanding for its treatment choices.

Methods: PubMed and Cochrane Central Register of Controlled Trials were systematically searched on January 19, 2023 with date restrictions of 2015–2023 imposed. For initial data collection, five independent searches were completed using the following keywords: pathogenesis, pathophysiology, and epidemiology of DCM; cervical spondylotic myelopathy (CSM) and DCM recent developments; management and treatment for CSM and DCM; diagnosis and management of DCM; and pathophysiology of DCM. The results were screened for their application to DCM; any study that did not directly address DCM were identified and removed through abstract assessment, such studies included those pertaining to alternative fields including cardiology and psychiatry. Studies found relevant through full-text assessment and those published in English were included in this study and unpublished studies and studies found irrelevant based on titles and keywords were excluded from this study. The 115 articles that met criteria were critically appraised independently by the 2 reviewers and the principles of Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) were applied to assess the quality of evidence from each study.

Results: A total of 352 studies resulted from the original search. There were 71 duplicate articles that were removed and a total of 281 articles were screened. 166 articles were then removed based on the exclusion/inclusion criteria, title, and abstract. Of the 138 articles that remained, a final list of 115 articles was created based on the reporting measures.

Conclusions: DCM is a multifactorial disease that has the potential to impair neurological function and cause significant paralysis. Although the multiple facets of this disease have not been fully elucidated, there have been significant breakthroughs in understanding the mechanisms involved in this disease process. The use of complex imaging modalities, genetic sequencing, biomarkers, and pharmacological agents has

^ ORCID: 0000-0003-2975-1177.

provided insight into the factors involved in the progression of DCM, which has consequently cultivated more refined approaches for diagnosis and treatment of DCM.

Keywords: Cervical stenosis; degenerative cervical myelopathy (DCM); cervical spondylotic myelopathy (CSM)

Submitted Oct 12, 2023. Accepted for publication Feb 20, 2024. Published online May 07, 2024.
doi: 10.21037/jss-23-123
View this article at: https://dx.doi.org/10.21037/jss-23-123

Introduction

Background

The incidence of degenerative cervical myelopathy (DCM) has increased over the years due to an increasing aging population, yet there is a dearth of recent comprehensive data evaluating the multiple facets of this degenerative condition (1,2). DCM is characterized as the chronic compression of the spinal cord between the upper cervical region and the upper thoracic region resulting in a wide range of neurologic disabilities. Classically it has an insidious onset progressing in a stepwise manner with functional decline. Without the necessary medical interventions, patients may progress toward significant loss of function (3). The treatment of DCM is multifactorial; while conservative options exist, surgical intervention yields superior results in improving functional outcomes (4,5). The goal of surgical intervention is decompression of the spinal cord and prevention of symptomatic progression (6). The authors of this systematic review sought to collect and assess available high quality peer reviewed data to analyze the nature of DCM and gain a better understanding for its treatment choices.

Highlight box

Key findings
- The synergistic effect of imaging, genetic sequencing, biological markers, and pharmacology has the potential to favorably shift the prognosis of patients with degenerative cervical myelopathy (DCM).
- Implementing standardized approaches that use individual preoperative characteristics to predict postoperative outcomes can address the discrepancies of postoperative surgical outcomes between patients with DCM.

What is known and what is new?
- DCM is a progressive neurological disorder that can lead to significant impairment in affected individuals. The incidence of DCM has increased over the years due to an increasing aging population, making it a salient area of research.
- This manuscript discusses the biochemistry and biomechanics of DCM, which are paramount to understanding the degenerative nature of the condition and selecting the most optimal treatment options for improved patient outcomes.

What is the implication, and what should change now?
- The field of orthopedic surgery has tremendously evolved with recent technological developments. These advancements have provided detailed insight into the pathophysiology of DCM, which have subsequently enhanced management and treatment options for the disease. This systematic review highlights the importance of increased accessibility to imaging modalities, genetic sequencing, biomarkers, and pharmacological agents for the management of DCM as these may improve patient outcomes.

Rationale and knowledge gap

Recent publications have highlighted the biochemistry and biomechanics of DCM, which are paramount to understanding the degenerative nature of the condition and selecting the most optimal treatment options for improved patient outcomes. In addition, there have been recent studies establishing the superiority of surgical to non-surgical treatments for DCM, which until now was a poorly substantiated claim that has permeated the medical field for decades.

Objective

To consolidate the emerging data surrounding the pathogenesis, pathophysiology, natural history, epidemiology, diagnosis, and treatment of DCM into a systematic review that serves to educate physicians on this disease process including current treatment options available. We present this article in accordance with the PRISMA reporting checklist (available at https://jss.amegroups.com/article/view/10.21037/jss-23-123/rc).

Methods

Search strategy

The initial search was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guidelines (7). The guidelines released by Fehlings *et al.* in 2017 dictated parameters to guide clinician approach to DCM. The aim of this study is to capture updates in clinical knowledge and practices that have occurred in the years leading up to and following the release of these guidelines (8). The date restriction was extended to cover articles released following the updates of the widely cited 2015 release outlining the updates in the diagnosis and treatment of DCM (9). Two electronic full-text databases PubMed and Cochrane Central Register of Controlled Trials were systematically searched with date restrictions of 2015–2023 imposed. For the initial data collection, five (5) independent searches were completed using the following keywords: pathogenesis, pathophysiology, and epidemiology of degenerative cervical myelopathy; cervical spondylotic myelopathy (CSM) and degenerative cervical myelopathy recent developments; management and treatment for CSM and degenerative cervical myelopathy; diagnosis and management of degenerative cervical myelopathy; and pathophysiology of degenerative cervical myelopathy. Study titles and abstracts were filtered against the inclusion and exclusion criteria by two reviewers independently (K.T., H.T.). Full texts of the relevant studies were retrieved, and their references were perused to identify other relevant studies. The articles that met inclusion criteria were blindly assessed by two junior authors independently and the principles of Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) were applied, resulting in each study being assigned a score of high, moderate, low, or very (10). The principal investigator completed the same process independently. Scores from all three assessors were reviewed and it was found authors concurred on the ratings of the studies; scores have been provided in Table S1.

Authors of relevant studies were not contacted in this process for original data, therefore, all data in the present study was extrapolated from published literature. The last literature search conducted on PubMed and Cochrane Central Register of Controlled Trials was January 19, 2023.

Inclusion criteria

Relevance was classified as any article resulting from the search terms examining the etiology, pathophysiology, epidemiology, diagnosis, or treatment of cervical stenosis or DCM. Studies found to be relevant through abstract screening followed by full-text assessment and those published in English were included in this study.

Exclusion criteria

Unpublished studies and studies found irrelevant based on titles and keywords were not used in this study.

Selection of studies

A total of 115 studies fit the inclusion criteria and a full-text literature review was conducted of each relevant source. After relevant study articles were identified, abstracts and full-texts were assessed independently by the reviewers K.T. and H.T. and evaluated for accuracy. There was no disagreement among the 2 researchers in the determination of study inclusion and data inclusion.

Results

Literature search results

The literature search process is summarized in *Figure 1*. A total of 352 studies resulted from the original search. There were 71 duplicate articles that were removed and a total of 281 articles were screened. 166 articles were then removed based on the exclusion/inclusion criteria, title, and abstract. Of the 138 articles that remained, a final list of 115 articles was created based on the reporting measures.

Quality assessment

The 115 selected studies were assessed for quality of evidence. Of the 115 selected studies, 1 was considered "high quality", 21 were considered "moderate quality", 66 were considered "low quality", and 27 were considered "very low quality" evidence according to the GRADE criteria (10).

Discussion

Etiology

DCM consists of acquired and congenital abnormalities related to the cervical spine (11). The most common cause of DCM in patients of more than 55 years of age is CSM, which is the age-related deterioration of intervertebral

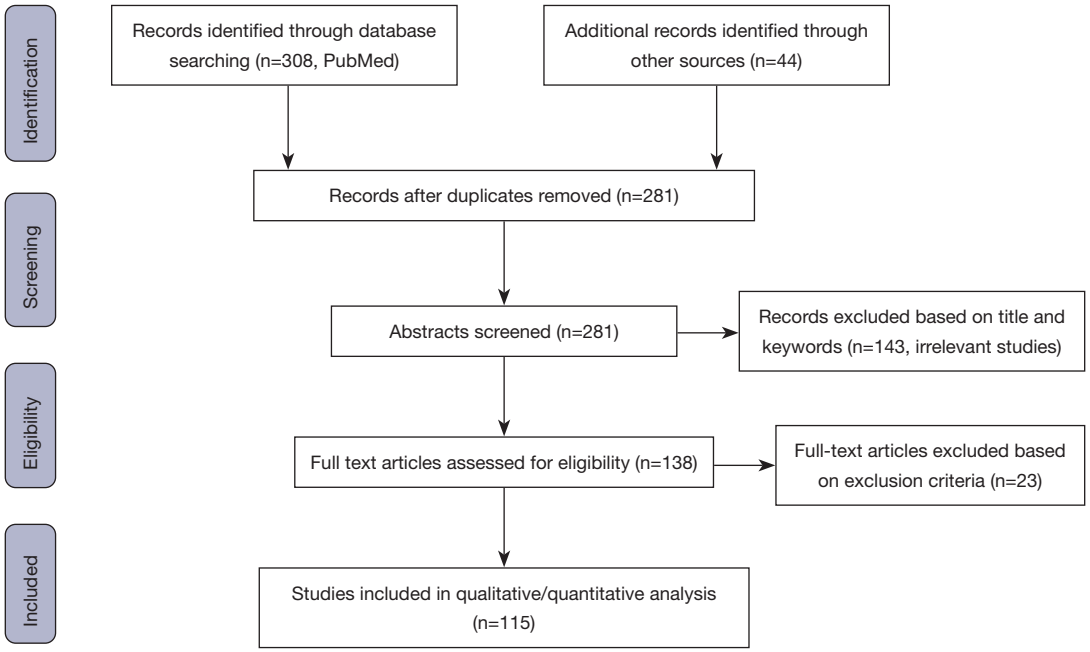

Figure 1 Flow of studies through literature review.

discs (12,13). Intervertebral disc herniation, ossification of posterior longitudinal ligament (OPLL), bony architectural pathologies, ligamentum flavum hypertrophy, canal stenosis or a combination of these account for the remainder of acquired cervical myelopathy cases (4,14). The underlying problem unifying these conditions is injury to the spinal cord due to pathologic compression (1).

Congenital spinal stenosis (CSS) is defined as the "decreased spinal canal diameter at multiple levels of the cervical spine in the absence of degenerative changes" (15). Patients with CSS have pathoanatomical features of their cervical spines that contribute to the narrowing of their spinal canals. Compared to controls, they have significantly smaller AP lateral masses, lamina lengths, lamina-pedicle angles, and larger lamina-disc angles at levels C3 to C7. A decrease in the lamina-pedicle angle with an increase in the lamina-disc angle ultimately leads to a smaller mid-sagittal canal diameter. These anatomic elements devise a right triangle that results in the progressive narrowing of the spinal canal over time, and the eventual onset of DCM (15).

Pathophysiology

There has been increasing interest in the pathophysiology of DCM recently. Studies have shown that the pathophysiology is multifactorial encompassing static factors causing stenosis, dynamic factors resulting in continuous mechanical injury to the spinal cord, and histopathologic factors resulting in ischemia and inflammation (13,16-18). Du *et al.* reports that serum Interleukin-6, a potent proinflammatory cytokine, is significantly elevated in patients with DCM and hypothesizes that its concentration may predict symptom severity (19). Other studies note additional pathophysiological mechanisms of DCM include alterations of the vascular architecture, endothelial cell impairment, disruption of the blood-spinal cord barrier, and oligodendrocyte and neuronal apoptosis (20).

Static factors result from either congenital stenosis or acquired stenosis secondary to spondylosis, disc degeneration, or pathology of the ligamentum flavum (12,21). The degenerative cascade of cervical myelopathy typically begins with the deterioration of an intervertebral disc (16,22,23). As time progresses, the disc annulus weakens and the nucleus pulposus bulges posteriorly, creating a herniated disc and a narrowed spinal canal at that level. Deteriorated discs can also shorten, which causes the spinal column to shorten, resulting in nerve impingement and numbness (13,22,24-26). An additional static factor contributing to the development of DCM is pathology of the ligamentum flavum. The ligamentum flavum lies posterior to the spinal column and as it loses its elasticity and vigor, it thickens and applies mechanical pressure to

the spinal cord. The ligament can also calcify, resulting in a thick and rigid band-like structure impinging the spinal cord (13,14,22,23).

Dynamic factors involve pathologic repetitive movement of the cervical spine, which can aggravate spinal cord compression (SCC) in physiologic and pathologic movements (16). Researchers explain flexion may compress the spinal cord against anterior osteophytes and intervertebral discs while hyperextension might lead to cord pinching between the posterior margins of the vertebral bodies anteriorly and a hypertrophied buckled ligamentum flavum posteriorly (16,17,22,23). Although both flexion and extension can exacerbate myelopathy, there is a significant increase of spinal stenosis in extension more so than in flexion because in extension, the ligamentum flavum folds, which further constricts the spinal canal, exacerbating cord impingement (17,27).

SCC can lead to histopathologic changes such as ischemia and inflammation secondary to vascular compression. Chronic cord compression can lead to neuronal cell loss, excitotoxicity, degeneration of the posterior columns and anterior horn cells, and endothelial damage resulting in a compromised blood-spinal barrier, which all accumulate to the demise of the patient (16,17,22).

The known risk factors for developing DCM are age, smoking, male gender, and genetics. Recent studies have identified certain conditions that are significant risk factors for developing DCM such as Klippel-Feil and Down syndromes. As technology becomes readily available, there has been an interest in the potential diagnostic utility for genetics in the future evaluation of CSM. Single nucleotide polymorphisms have been tracked to specific features of the disease including inflammatory levels in intervertebral discs (28). Genes implicated in the processes of inflammation, bone metabolism, and survival in the cell cycle were correlated to features of CSM including susceptibility to, severity of symptoms, and clinical progression of CSM (28). Other studies have noted that genetic polymorphisms may be associated with increased risk of DCM and can even affect postoperative outcomes (28,29). The genes involved are primarily related to bone pathophysiology pathways and include: osteopontin (OPN), vitamin D receptor (VDR), bone morphogenetic protein-4 (BMP-4), collagen IX tryptophan, apolipoprotein E (APOE), hypoxia-inducible factor a (HIF-1a) (30-37). Given the known importance of vitamin D in the metabolism and development of bone, studies queried VDR's role in CSM, finding that specific polymorphisms related to disc degeneration and severity of disease on magnetic resonance imaging (MRI) (31,32). Collagen IX acts as a bridge between proteins in tissues, and specific alleles of collagen IX (specifically Trp2) increased the risk of CSM, similar to findings with BMP4 which induces *de novo* cartilage formation and bone formation (33,34). BMP4 was also found to be useful in the assessment of postoperative outcomes (33). The specific allele APOE ε4, previously known to impact outcomes in other neurologic disorders, has been implicated in increased likelihood of development of DCM and was an independent predictor of CSM occurrence in patients with SCC and outcomes in operative management (29,35,36). HIF-1α expression in the nucleus pulposus plays a critical role in the survival of disc cells, with polymorphisms affecting susceptibility to CSM and expression associated with clinical features in affected patients (37). Ultimately, associations in specific gene loci may lead to the availability of genetic testing for the evaluation of DCM. In a 2021 study, MR T-2 hypersensitivity and CSS were determined risk factors for rapid progressive CSM (38).

Epidemiology

Cervical stenosis appears to be common and is present in 4.9% of the adult population, 6.8% of the 50-year-old or older population, and 9% of the 70-year-old or older population (1,39). The number of patients with CSM, specifically, has increased due to the increase in the aging population (12). Most patients with CSM are diagnosed in their 50s, as it is uncommon to present before the age of 40 years (40).

Although DCM is the most common cause of spinal cord injury in the developed world, the epidemiology of DCM remains poorly characterized (1). In the Netherlands, the prevalence of surgically treated CSM cases was reported at 1.6 per 100,000 people, though the actual prevalence is likely significantly higher (41-43). In a North American study, the incidence and prevalence of DCM were estimated at a minimum of 41 and 605 per million in North America, respectively (43). In Taiwan, the incidence of CSM-related hospitalizations was reported at 4.04/100,000 person-years (1,43,44). Published studies also report that males are more commonly affected than females. However, it is anticipated that these are underestimated as they are based on operative incidence and fail to account for underdiagnosis (1).

Diagnosis

Incidental cervical SCC is a frequent finding in the age of technological advancement. Individuals with asymptomatic SCC tend to go undetected until they encounter an unrelated health issue requiring an imaging modality, reasserting the epidemiology of DCM is poorly understood, partly due to undetected/asymptomatic individuals (13,43). In a series of randomly selected volunteers aged 40–80 years, incidental cervical cord compression was detected on MRI in 59% of individuals, yet only two individuals reported related symptoms (40).

The preliminary diagnosis of a patient with spinal stenosis often begins with a detailed history of symptoms and physical exam, focusing on sensation, motor strength, reflexes, and gait. Confirmatory diagnoses can be made with X-ray, CT, or MRI imaging modalities, but there is drastic variability in these techniques and they each have their own strengths and limitations (20,45,46). Plain radiographs are of limited value although it provides an exceptional contrast between the vertebrae and surrounding structures. Plain radiographs also permit various patient positionings, allowing evaluation of spinal alignment and instability under physiological conditions. Similar to plain radiographs, CT offers an exceptional visualization of bony structures with an even greater level of detail. CT can be especially useful in distinguishing bony sources of compression [e.g., ossified disc herniations, osteophytes, ossification of the posterior longitudinal ligament (OPLL)] (47). Furthermore, CT myelography involving the use of contrast dye offers a pristine view of spinal cord contour and potential compressive surrounding structures. The limitation of its use, however, is the significant exposure to ionizing radiation and the restriction to supine positioning. MRI is considered the gold standard to diagnose DCM as it provides a detailed visualization of soft tissue structures such as the spinal cord and surrounding structures (4,47-49). MRI can detect the extent of pathologic changes in the intervertebral disk, hypertrophy or buckling of the ligamentum flavum, amount of decrease in spinal canal diameter, severity of SCC, and abnormal signal intensity of the intramedullary lesion (50). Researchers strongly advise visiting a specialist if CSM, a subset of DCM, is suspected as early intervention is paramount for a better prognosis (4,51,52).

DCM frequently leads to severe neurologic disability, but is still frequently underdiagnosed. One explanation may be the variability of the symptoms presented by the patients, from paresthesia to quadriplegia, making it a great masquerader (53). While currently available imaging modalities can detect DCM in many cases, the sensitivity could be improved with the recent advances of technology. For instance, there is future promise for the use of functional MRI (fMRI), MR spectroscopy, myelin imaging and diffusion tensor imaging (DTI) techniques to detect metabolic and microstructural abnormalities in patients before conventional MRI can detect differences. Such techniques may be more effective in predicting outcomes (42,48,46,54,55). Banaszek *et al.* remarks, "DTI provides quantitative information about the myelopathy severity that could help select patients who would benefit most from surgical decompression" (18). DTI has even been named as a valuable diagnostic tool in identifying patients who may benefit most from surgical interventions (56). Rajan *et al.* concurs and adds perfusion imaging and positron emission tomography (PET) are additional promising techniques in the diagnosis of DCM-all of these modalities precisely assess the physical structure, biochemical composition, perfusion, and intrinsic connectivity of the spinal cord, aiding in the diagnosis of DCM (50). They further that MR spectroscopy, which utilizes MRI to measure the concentration of different chemical components within tissues, can aid in the detection of DCM as studies have shown that individuals with DCM have lower N-acetylaspartate/creatine ratio and lactate peaks compared to age-matched controls (47,57-61).

Emerging research on the use of serum biomarkers and T2-weighted MRI in the diagnosis and staging of DCM has the potential to innovate the field of medicine. In 2019, researchers published an article comparing the level of microRNAs in OPLL patients' plasma or serum to that of normal and patients with intervertebral disc degeneration to assess the accuracy of OPLL-specific microRNAs in identifying OPLL (62). Experts found that miR-10a-5p, miR-563, and miR-210-3p showed high accuracy and significance in discerning OPLL from other groups individually, however an index condensing these miRNAs achieved the highest accuracy among these individual miRNAs (62). The discovery of these OPLL- disease specific microRNAs can possibly aid in early detection of OPLL and with timely interventions halt progression to DCM.

While serum biomarkers are an exciting new focus, improvements in imaging technology, namely high-resolution computed tomography (hrCT) have improved identification of pathology *in vivo* (62). Despite being the gold standard imaging technique used in diagnosis, hrCT

use is limited clinically by concern for radiation exposure and lower resolution when viewing soft tissue structures compared to MRI. New quantitative magnetic resonance imaging (qMRI) has the potential to allow physicians to view specific properties of the spinal cord in DCM, including microstructures (63). State of the art protocols leveraging MRI technology have the potential to be transformative in the assessment of DCM before development of severe clinical symptomatology (64). Furthermore, in the imaging space, T2-weighted MR imaging (T2WI) can act as a biomarker detecting white matter injury (62-64). On MRI, T2-hyperintensitivity is a nonspecific marker for spinal cord injury, and demonstrates variability between patients requiring normalization. MR T2-hyperintensity was found to be a risk factor in progression of CSM (38). T2WI was found to provide strong diagnostic accuracy in identifying subclinical white matter degeneration correlated with features of DCM. Fine tuning of this tool for clinical use may improve sensitivity in the diagnosis of cord injury in DCM (63). Before these tools can be adopted clinically, more work is required to fine tune protocols and more prospective, randomized, group studies with long term follow-up are required to validate the implementation of T2WI.

Presentation and progression

Cervical stenosis does not necessarily cause symptoms. Cervical stenosis is a chronic condition that is generally well compensated, however an acute traumatic event can trigger the onset of neurological deficits (65). These deficits may be due to radiculopathy, defined as the compression of a nerve root or myelopathy, compression of the spinal cord and even then, presentation is contingent on the levels of spinal cord that are impinged (12,14). Lannon & Kachur report that no pathognomonic sign exists for DCM, thus, clinicians must be knowledgeable on the array of manifestations associated with this disease process (66). Marie-Hardy & Pascal-Moussellard further state that the severity of symptoms can greatly differ from one person to the next, earning it the title "the great masquerader" (53). Interestingly enough some patients may not experience neurological impairment at all despite SCC (50).

Though patients with DCM have heterogeneous presentations, the typical signs and symptoms include pain in the neck or back, ataxia, abnormal gait, lack of coordination, muscle weakness, rhythmic muscle spasms, stiff muscles, muscular atrophy, reduced sensation, bladder dysfunction and paresthesia in the extremities (17,40). Most patients' symptoms worsen over the years; however, deterioration can occur rapidly and is sometimes irreversible (67). Generally, the neurological examination of patients with DCM appears either normal or with minor abnormalities such as brisk deep tendon reflexes, extensor plantar response, positive Romberg's sign, positive Hoffman's sign, Babinski reflexes, and hyperactive reflexes, including clonus (68-70). In addition, there may be decreased and painful range of motion, Lhermitte sign, and upper motor neuron reflexes (70,71). The manifestations of this disease are localized to pathology within the spinothalamic and corticospinal tracts as they are impacted first due to their location in the lateral aspect of the spinal cord (70-72).

The progression of DCM can be monitored with advanced imaging modalities and self-report assessments evaluating functionality. qMRI is capable of detecting features characteristic of DCM including microstructural processes including tissue changes, axonal injury, demyelination, and atrophy (73). The modified Japanese Orthopaedic Association (m-JOA) scale is a self-report tool used to assess neurological function in patients with cervical myelopathy and includes the following categories: upper and lower extremity motor function, upper extremity sensory function and sphincter function. Patients can then be stratified into mild, moderate, and severe disease based on their mJOA score with higher scores denoting greater neurological function (74). As the disease progresses, patients' m-JOA scores decline denoting neurological deterioration (75).

The natural course of DCM is one of progressive functional neurological decline and surgery is often recommended for patients with moderate and severe disease. For mild cases, surgery is not necessarily required, clinicians must exercise their clinical judgment and assess patients on a case-by-case basis, considering the various factors that influence the disease course. Factors such as medical comorbidities, level of inflammatory markers, congenital and genetic conditions, neurophysiological states, mJOA scores, and duration of symptoms should be assessed for each individual patient as these factors influence the decision to opt into surgical or nonsurgical treatment options (76).

Treatment

The criteria for surgical intervention vary amongst surgeons,

but the general threshold consists of severe compression of the spinal cord, progressive signs of neurological impact, or a diagnosis of CM for ≥6 months (8,77). Prior to pursuing surgical intervention in patients with minor, nonprogressive symptoms or when considering patients who are not suited for surgical options, conservative measures may be implemented and evaluated for efficacy. These measures include immobilization with a cervical collar, physical therapy, analgesics, and anti-inflammatories (78). Despite the widespread use of anti-inflammatory agents, a retrospective cohort study conducted in 2021 found that there is an overall increased odds of surgery after cervical injections (79). Historically, conservative treatment has not been recognized as a feasible approach for prevention of chronic disease progression or complete symptom relief (80). Zárate-Kalfopulos *et al.* and Gulati *et al.*, remark that early surgical management is vital in achieving better neurological outcomes (81,82). Likewise, Choi & Kang report that surgical management will not only suppress progression, but also improve quality of life (52). The decision to pursue surgical treatment, however, does depend on a variety of factors including severity of symptoms, etiology of symptoms, comorbidities, compression ratio of the cervical spinal cord and surgical options (8,52,53,83). Kadaňka *et al.* described in a 10-year retrospective study that there was no significant difference in outcomes for patients treated conservatively versus surgically for mild to moderate spondylotic cervical myelopathy (CSM) (6,84). Conversely, other studies have demonstrated that surgical intervention yields superior outcomes when compared to conservative intervention for improvement of functional outcomes (4,5). Similarly, it has been described that the majority of patients undergoing conservative measures showed worsening in the ability to undergo their daily activities, without any evidence of halted progression, and increased risk of hospitalization (85,86). It is important to consider, however, that spinal surgery comes with significant risk including infection, non-fusion, and reoperation (8,77,78). In addition, the point of irreparable damage to the spinal cord can be reached in severe cases; in such situations, surgery is done to arrest the progression, but may not improve functionality (87). Fehlings *et al.* composed a clinical guideline practice to determine appropriate interventions for patients with DCM. For patients with moderate and severe DCM, surgical interventions are recommended. In contrast, patients with mild DCM are recommended for surgical intervention or supervised rehabilitation services. In the event neurological function deteriorates while pursuing a nonsurgical approach, surgical intervention is recommended (8,77). It can be argued that conservative treatments such as cervical collar, muscle relaxers and analgesics should be offered to patients with mild DCM, before surgery is indicated. In progressive cases, however, pain and an intramedullary signal change on MRI make surgery more salient. Nonetheless, these factors should not be analyzed in a vacuum, they should be considered a conglomerate of a patient's story (88).

The goal of surgical intervention is to achieve decompression of the spinal cord and to prevent progression (6,53). Conventionally, there are 3 approaches to decompression of the spinal cord: anterior, posterior, and combined. Selection of approach depends on the disease state (i.e., number of involved spinal levels), nature of the underlying disease, surgeon specific preference, and patient specific factors (i.e., surgical history) (6,20,89). Farrokhi *et al.* recently devised a stepwise approach considering patient characteristics such as sagittal balance, number of levels affected, age, etc. to select the most optimal surgery for patients with CSM (90). However, a body of evidence has been generated over the past two decades comparing anterior to posterior approaches, without naming either as a definitively superior as a standard of care (7,8,14,69,91-94).

Anterior approaches including anterior cervical discectomy with fusion (ACDF) and anterior decompression with fusion (ADF) have been considered the "gold standard" for many years and is one of the most frequent spinal surgeries performed in the U.S (95). Such anterior approaches can be contrasted with cervical laminoplasty (LAMP), cervical laminectomy, and posterior decompression with fusion (PDF). Anterior approaches are specifically useful in cases of significant kyphosis and OPLL (4). The traditional ACDF procedure consists of generating a wide anterior exposure to allow for decompression. A widely accepted technique for performing ACDF follows the Smith-Robinson anterior cervical approach to ensure proper exposure and verification of the appropriate vertebral levels (96). Historically, ACDF is associated with more technical difficulty as compared to LAMP. As such, there are drawbacks to ACDF, including the concern for long operative time, respiratory complications, loss of mobility, and dysphagia (97-99). Dysphagia is a particular concern in the early postoperative period for ACDF patients, but can persist. There has been investigation into the etiological factors of dysphagia to attempt to decrease the prevalence, with limited success (100). To prevent complications seen in anterior

surgeries, a retrospective case study conducted in 2017 attempted to identify modifiable preoperative risk factors but concluded that awareness, early recognition, and appropriate management were of great importance in mitigating complications (97).

In contrast, LAMP is used to provide posterior decompression to the spinal cord in DCM. It was proposed in the early 1970's by Japanese surgeons to treat OPLL and congenital cervical stenosis (95,101,102). The procedure involves the incision of the skin and division of the nuchal ligament followed by a removal of the spinous process, with preservation of surrounding architecture. While the goals of LAMP and ACDF are similar: to decompress the spinal cord, they differ in that LAMP generally better preserves motion and ACDF creates fusion (85). A 2015 systematic review found that patients with moderate and severe CSM may benefit from either a LAMP or laminectomy and fusion approach opposed to anterior approaches (103). Common concerns for postoperative complications with LAMP include C5 palsy and axial neck pain.

The combined approach combines the LAMP or laminectomy with ACDF or anterior cervical corpectomy fusion (ACCF) approaches. The technique is used to resect both ventral and dorsal compressions directly in one operation to provide decompression bidirectionally. Theoretically this procedure may be most effective for severe cases of multilevel DCM, but is associated with higher risk of complication (53). This approach is also associated with a high technical level of skill compared to simpler traditional approaches and is reserved for severe cases (1). An alternative hybrid surgical approach makes use of both ACDF and cervical disc arthroplasty (CDA) to preserve segmental motion and prevent adjacent degeneration. While this approach also shows theoretical promise in multilevel cervical disc degeneration, further investigation is required to verify the generalizability of the results (3,104,105).

Despite the lack thereof clarity on the standard of care for DCM, surgical intervention is widely employed and varies between surgeons, institutions, and regions. While specific patient metrics including findings of radiological studies, physiological curvature of the cervical spine, and number of levels involved can be used to direct the selection of one procedure over the other, often times surgeon preference plays a large role in technique selection (14,53,106). To improve surgical outcomes in patients with DCM, Jannelli *et al.* developed an approach incorporating the clinical, radiological, and electrophysiological components of each patient's disease process to predict those more likely to benefit (this approach is also said to diagnose DCM early) (107). Generally, younger, nonsmoking patients with fewer comorbidities and milder preoperative myelopathy tend to see sooner and superior outcomes following treatment (78,85,108,109). Despite this trend, a retrospective study found that after controlling for comorbidities and demographics, compared with patients 60–69 and 70–79 with CSM, octogenarian patients with CSM did not have different surgical outcomes (110). Likewise, Madhavan *et al.* in 2016 and Zhang *et al.* in 2017 report that despite the higher risks involved in operating on older patients, there is no difference in the incidence of postoperative complications compared to younger patients (111-113), while Wilson *et al.* demonstrates that frailty has a greater effect size and a higher discriminative value to predict complication than age alone (114). Lastly, a 2021 research study found that patients who are ApoE4^{+} displayed poorer progress after decompression surgery, reinforcing individual characteristics influence surgical outcomes (115).

The future of DCM treatment is quite promising. Researchers are evaluating the use of pharmacological agents as adjuncts to surgical interventions. In 2012, researchers conducted a multicenter, double-blind, placebo-controlled, randomized trial to investigate the effects of riluzole, a glutamate antagonist, on patients with DCM undergoing decompression surgery. Although riluzole had no impact on m-JOA scores, it was found to substantially reduce neck pain 6 and 12 months post-decompression surgery compared to the placebo (64,116). The results of this study may have profound implications for treatment and management of DCM and can potentially spark the emergence of similar studies.

Conclusions

DCM is a multifactorial disease that has the potential to impair neurological function and cause significant paralysis. Although the multiple facets of this disease have not been fully elucidated, there have been significant breakthroughs in understanding the mechanisms involved in this disease process. The use of complex imaging modalities, genetic sequencing, biomarkers, and pharmacological agents has provided the necessary insight to cultivate better approaches for diagnosis and treatment of DCM in addition to understanding the factors involved in the progression of DCM. To address the discrepancies of postoperative

surgical outcomes between patients, researchers have developed standardized approaches to predict postoperative outcomes based on individual preoperative characteristics. DCM is an active area of research, and the technological advancements that have been fundamental in understanding the structural and biochemical mechanisms involved in its onset, progression, treatment, and postoperative outcomes may significantly enhance patient outcomes.

Acknowledgments

Funding: None.

Footnote

Reporting Checklist: The authors have completed the PRISMA reporting checklist. Available at https://jss.amegroups.com/article/view/10.21037/jss-23-123/rc

Peer Review File: Available at https://jss.amegroups.com/article/view/10.21037/jss-23-123/prf

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at https://jss.amegroups.com/article/view/10.21037/jss-23-123/coif). The authors have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

References

1. Nouri A, Cheng JS, Davies B, et al. Degenerative Cervical Myelopathy: A Brief Review of Past Perspectives, Present Developments, and Future Directions. J Clin Med 2020;9:535.
2. The World Bank. DataBank: Population estimates and Projections. Available online: http://databank.worldbank.org/data/reports.aspx?source=health-nutrition-and-population-statistics:-population-estimates-and-projections# (2019)
3. Donnally CJ 3rd, Patel PD, Canseco JA, et al. Current Management of Cervical Spondylotic Myelopathy. Clin Spine Surg 2022;35:E68-76.
4. McCormick JR, Sama AJ, Schiller NC, et al. Cervical Spondylotic Myelopathy: A Guide to Diagnosis and Management. J Am Board Fam Med 2020;33:303-13.
5. Johansen TO, Vangen-Lønne V, Holmberg ST, et al. Surgery for degenerative cervical myelopathy in the elderly: a nationwide registry-based observational study with patient-reported outcomes. Acta Neurochir (Wien) 2022;164:2317-26.
6. Rodrigues-Pinto R, Montenegro TS, Davies BM, et al. Optimizing the Application of Surgery for Degenerative Cervical Myelopathy AO Spine RECODE-DCM Research Priority Number 10. Global Spine J 2022;12:147S-58S.
7. Page MJ, Moher D, Bossuyt PM, et al. PRISMA 2020 explanation and elaboration: updated guidance and exemplars for reporting systematic reviews. BMJ 2021;372:n160.
8. Fehlings MG, Tetreault LA, Riew KD, et al. A Clinical Practice Guideline for the Management of Patients With Degenerative Cervical Myelopathy: Recommendations for Patients With Mild, Moderate, and Severe Disease and Nonmyelopathic Patients With Evidence of Cord Compression. Global Spine J 2017;7:70S-83S.
9. Lebl DR, Bono CM. Update on the Diagnosis and Management of Cervical Spondylotic Myelopathy. J Am Acad Orthop Surg 2015;23:648-60.
10. Guyatt GH, Oxman AD, Vist GE, et al. GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. BMJ 2008;336:924-6.
11. Badhiwala JH, Ahuja CS, Akbar MA, et al. Degenerative cervical myelopathy - update and future directions. Nat Rev Neurol 2020;16:108-24.
12. Nishida N, Kato Y, Imajo Y, et al. Biomechanical analysis of cervical spondylotic myelopathy: the influence of dynamic factors and morphometry of the spinal cord. J Spinal Cord Med 2012;35:256-61.
13. Milligan J, Ryan K, Fehlings M, et al. Degenerative cervical myelopathy: Diagnosis and management in primary care. Can Fam Physician 2019;65:619-24.
14. Asher AL, Devin CJ, Kerezoudis P, et al. Comparison of Outcomes Following Anterior vs Posterior Fusion Surgery

for Patients With Degenerative Cervical Myelopathy: An Analysis From Quality Outcomes Database. Neurosurgery 2019;84:919-26.
15. Jenkins TJ, Mai HT, Burgmeier RJ, et al. The Triangle Model of Congenital Cervical Stenosis. Spine (Phila Pa 1976) 2016;41:E242-7.
16. Baptiste DC, Fehlings MG. Pathophysiology of cervical myelopathy. Spine J 2006;6:190S-7S.
17. Lebl DR, Hughes A, Cammisa FP Jr, et al. Cervical spondylotic myelopathy: pathophysiology, clinical presentation, and treatment. HSS J 2011;7:170-8.
18. Banaszek A, Bladowska J, Podgórski P, et al. Role of Diffusion Tensor MR Imaging in Degenerative Cervical Spine Disease: a Review of the Literature. Clin Neuroradiol 2016;26:265-76.
19. Du S, Sun Y, Zhao B. Interleukin-6 Serum Levels Are Elevated in Individuals with Degenerative Cervical Myelopathy and Are Correlated with Symptom Severity. Med Sci Monit 2018;24:7405-13.
20. Fehlings MG, Tetreault L, Hsieh PC, et al. Introduction: Degenerative cervical myelopathy: diagnostic, assessment, and management strategies, surgical complications, and outcome prediction. Neurosurg Focus 2016;40:E1.
21. Wilson JR, Tetreault LA, Kim J, et al. State of the Art in Degenerative Cervical Myelopathy: An Update on Current Clinical Evidence. Neurosurgery 2017;80:S33-45.
22. de Oliveira Vilaça C, Orsini M, Leite MA, et al. Cervical Spondylotic Myelopathy: What the Neurologist Should Know. Neurol Int 2016;8:6330.
23. Fehlings MG, Skaf G. A review of the pathophysiology of cervical spondylotic myelopathy with insights for potential novel mechanisms drawn from traumatic spinal cord injury. Spine (Phila Pa 1976) 1998;23:2730-7.
24. Kalsi-Ryan S, Karadimas SK, Fehlings MG. Cervical spondylotic myelopathy: the clinical phenomenon and the current pathobiology of an increasingly prevalent and devastating disorder. Neuroscientist 2013;19:409-21.
25. Kato S, Fehlings M. Degenerative cervical myelopathy. Curr Rev Musculoskelet Med 2016;9:263-71.
26. Toledano M, Bartleson JD. Cervical spondylotic myelopathy. Neurol Clin 2013;31:287-305.
27. Meyer F, Börm W, Thomé C. Degenerative cervical spinal stenosis: current strategies in diagnosis and treatment. Dtsch Arztebl Isnt 2008;105:366-72.
28. Wang G, Cao Y, Wu T, et al. Genetic factors of cervical spondylotic myelopathy-a systemic review. J Clin Neurosci 2017;44:89-94.
29. Pope DH, Davies BM, Mowforth OD, et al. Genetics of Degenerative Cervical Myelopathy: A Systematic Review and Meta-Analysis of Candidate Gene Studies. J Clin Med 2020;9:282.
30. Wu J, Wu D, Guo K, Yuan F, Ran B. OPN polymorphism is associated with the susceptibility to cervical spondylotic myelopathy and its outcome after anterior cervical corpectomy and fusion. Cell Physiol Biochem 2014;34:565-74.
31. Song DW, Wu YD, Tian DD. Association of VDR-FokI and VDBP-Thr420Lys polymorphisms with cervical spondylotic myelopathy: A case-control study in the population of China. J Clin Lab Anal 2019;33:e22669.
32. Wang ZC, Chen XS, Wang da W, et al. The genetic association of vitamin D receptor polymorphisms and cervical spondylotic myelopathy in Chinese subjects. Clin Chim Acta 2010;411:794-7.
33. Wang D, Liu W, Cao Y, et al. BMP-4 polymorphisms in the susceptibility of cervical spondylotic myelopathy and its outcome after anterior cervical corpectomy and fusion. Cell Physiol Biochem 2013;32:210-7.
34. Wang ZC, Shi JG, Chen XS, et al. The role of smoking status and collagen IX polymorphisms in the susceptibility to cervical spondylotic myelopathy. Genet Mol Res 2012;11:1238-44.
35. Setzer M, Vrionis FD, Hermann EJ, et al. Effect of apolipoprotein E genotype on the outcome after anterior cervical decompression and fusion in patients with cervical spondylotic myelopathy. J Neurosurg Spine 2009;11:659-66.
36. Setzer M, Hermann E, Seifert V, et al. Apolipoprotein E gene polymorphism and the risk of cervical myelopathy in patients with chronic spinal cord compression. Spine (Phila Pa 1976) 2008;33:497-502.
37. Wang ZC, Hou XW, Shao J, et al. HIF-1α polymorphism in the susceptibility of cervical spondylotic myelopathy and its outcome after anterior cervical corpectomy and fusion treatment. PLoS One 2014;9:e110862.
38. Zhong W, Wang L, Huang T, et al. Risk factors for rapid progressive neurological deterioration in patients with cervical spondylotic myelopathy. J Orthop Surg Res 2021;16:75.
39. Lee MJ, Cassinelli EH, Riew KD. Prevalence of cervical spine stenosis. Anatomic study in cadavers. J Bone Joint Surg Am 2007;89:376-80.
40. Davies BM, Mowforth OD, Smith EK, et al. Degenerative cervical myelopathy. BMJ 2018;360:k186.
41. Boogaarts HD, Bartels RH. Prevalence of cervical spondylotic myelopathy. Eur Spine J 2015;24 Suppl

2:139-41.
42. Tu J, Vargas Castillo J, Das A, et al. Degenerative Cervical Myelopathy: Insights into Its Pathobiology and Molecular Mechanisms. J Clin Med 2021;10:1214.
43. Nouri A, Tetreault L, Singh A, et al. Degenerative Cervical Myelopathy: Epidemiology, Genetics, and Pathogenesis. Spine (Phila Pa 1976) 2015;40:E675-93.
44. Wu JC, Ko CC, Yen YS, et al. Epidemiology of cervical spondylotic myelopathy and its risk of causing spinal cord injury: a national cohort study. Neurosurg Focus 2013;35:E10.
45. Tetreault L, Kalsi-Ryan S, Benjamin Davies, et al. Degenerative Cervical Myelopathy: A Practical Approach to Diagnosis. Global Spine J 2022;12:1881-93.
46. Ellingson BM, Salamon N, Holly LT. Advances in MR imaging for cervical spondylotic myelopathy. Eur Spine J 2015;24 Suppl 2:197-208.
47. Rajan PV, Pelle DW, Savage JW. New Imaging Modalities for Degenerative Cervical Myelopathy. Clin Spine Surg 2022;35:422-30.
48. Martin AR, Tadokoro N, Tetreault L, et al. Imaging Evaluation of Degenerative Cervical Myelopathy: Current State of the Art and Future Directions. Neurosurg Clin N Am 2018;29:33-45.
49. Tracy JA, Bartleson JD. Cervical spondylotic myelopathy. Neurologist 2010;16:176-87.
50. Choi SH, Kang CN. Degenerative Cervical Myelopathy: Pathophysiology and Current Treatment Strategies. Asian Spine J 2020;14:710-20.
51. Bakhsheshian J, Mehta VA, Liu JC. Current Diagnosis and Management of Cervical Spondylotic Myelopathy. Global Spine J 2017;7:572-86.
52. Tetreault L, Wilson JR, Kotter MRN, et al. Is Preoperative Duration of Symptoms a Significant Predictor of Functional Outcomes in Patients Undergoing Surgery for the Treatment of Degenerative Cervical Myelopathy? Neurosurgery 2019;85:642-7.
53. Marie-Hardy L, Pascal-Moussellard H. Degenerative cervical myelopathy. Rev Neurol (Paris) 2021;177:490-7.
54. Martin AR, Aleksanderek I, Cohen-Adad J, et al. Translating state-of-the-art spinal cord MRI techniques to clinical use: A systematic review of clinical studies utilizing DTI, MT, MWF, MRS, and fMRI. Neuroimage Clin 2016;10:192-238.
55. Bhosale S, Ingale P, Srivastava S, et al. Diffusion tensor imaging as an additional postoperative prognostic predictor factor in cervical myelopathy patients: An observational study. J Craniovertebr Junction Spine 2019;10:10-3.
56. Rindler RS, Chokshi FH, Malcolm JG, et al. Spinal Diffusion Tensor Imaging in Evaluation of Preoperative and Postoperative Severity of Cervical Spondylotic Myelopathy: Systematic Review of Literature. World Neurosurg 2017;99:150-8.
57. Taha Ali TF, Badawy AE. Feasibility of 1H-MR Spectroscopy in evaluation of cervical spondylotic myelopathy. Egypt J Radiol Nucl Med 2013;44:93-9.
58. Kowalczyk I, Duggal N, Bartha R. Proton magnetic resonance spectroscopy of the motor cortex in cervical myelopathy. Brain 2012;135:461-8.
59. Salamon N, Ellingson BM, Nagarajan R, et al. Proton magnetic resonance spectroscopy of human cervical spondylosis at 3T. Spinal Cord 2013;51:558-63.
60. Holly LT, Freitas B, McArthur DL, et al. Proton magnetic resonance spectroscopy to evaluate spinal cord axonal injury in cervical spondylotic myelopathy. J Neurosurg Spine 2009;10:194-200.
61. Mohey N, E-H MA. Cervical spondylotic myelopathy: Can MR spectroscopy be helpful? Med J Cairo Univ 2019;87:2431-6.
62. Xu C, Zhang H, Zhou W, et al. MicroRNA-10a, -210, and -563 as circulating biomarkers for ossification of the posterior longitudinal ligament. Spine J 2019;19:735-43.
63. Martin AR, De Leener B, Cohen-Adad J, et al. A Novel MRI Biomarker of Spinal Cord White Matter Injury: T2*-Weighted White Matter to Gray Matter Signal Intensity Ratio. AJNR Am J Neuroradiol 2017;38:1266-73.
64. Moghaddamjou A, Badhiwala JH, Fehlings MG. Degenerative Cervical Myelopathy: Changing Frontiers. World Neurosurg 2020;135:377-8.
65. Gangavalli AK, Malige A, Sokunbi G. Multilevel critical stenosis with minimal functional deficits: a case of cervical spondylotic myelopathy. Spinal Cord Ser Cases 2018;4:104.
66. Lannon M, Kachur E. Degenerative Cervical Myelopathy: Clinical Presentation, Assessment, and Natural History. J Clin Med 2021;10:3626.
67. Naito K, Yamagata T, Ohata K, et al. Management of the Patient with Cervical Cord Compression but no Evidence of Myelopathy: What Should We do? Neurosurg Clin N Am 2018;29:145-52.
68. Parthiban J, Alves OL, Chandrachari KP, et al. Value of Surgery and Nonsurgical Approaches for Cervical Spondylotic Myelopathy: WFNS Spine Committee Recommendations. Neurospine 2019;16:403-7.
69. Iyer A, Azad TD, Tharin S. Cervical Spondylotic Myelopathy. Clin Spine Surg 2016;29:408-14.

70. Kane SF, Abadie KV, Willson A. Degenerative Cervical Myelopathy: Recognition and Management. Am Fam Physician 2020;102:740-50.
71. Bhattacharyya S. Spinal Cord Disorders: Myelopathy. Am J Med 2018;131:1293-7.
72. Bican O, Minagar A, Pruitt AA. The spinal cord: a review of functional neuroanatomy. Neurol Clin 2013;31:1-18.
73. Martin AR, De Leener B, Cohen-Adad J, et al. Monitoring for myelopathic progression with multiparametric quantitative MRI. PLoS One 2018;13:e0195733.
74. Hejrati N, Moghaddamjou A, Marathe N, et al. Degenerative Cervical Myelopathy: Towards a Personalized Approach. Can J Neurol Sci 2022;49:729-40.
75. Martin AR, Kalsi-Ryan S, Akbar MA, et al. Clinical outcomes of nonoperatively managed degenerative cervical myelopathy: an ambispective longitudinal cohort study in 117 patients. J Neurosurg Spine 2021;34:821-9.
76. Houten JK, Shahsavarani S, Verma RB. The Natural History of Degenerative Cervical Myelopathy. Clin Spine Surg 2022;35:396-402.
77. Fehlings MG, Tetreault LA, Riew KD, et al. A Clinical Practice Guideline for the Management of Degenerative Cervical Myelopathy: Introduction, Rationale, and Scope. Global Spine J 2017;7:21S-7S.
78. Williams J, D'Amore P, Redlich N, et al. Degenerative Cervical Myelopathy: Evaluation and Management. Orthop Clin North Am 2022;53:509-21.
79. Manzur MK, Samuel AM, Vaishnav A, et al. Cervical Steroid Injections Are Not Effective for Prevention of Surgical Treatment of Degenerative Cervical Myelopathy. Global Spine J 2023;13:1237-42.
80. Tetreault LA, Rhee J, Prather H, et al. Change in Function, Pain, and Quality of Life Following Structured Nonoperative Treatment in Patients With Degenerative Cervical Myelopathy: A Systematic Review. Global Spine J 2017;7:42S-52S.
81. Zárate-Kalfopulos B, Araos-Silva W, Reyes-Sánchez A, et al. Hybrid Decompression and Fixation Technique for the Treatment of Multisegmental Cervical Spondylotic Myelopathy. Int J Spine Surg 2016;10:30.
82. Gulati S, Vangen-Lønne V, Nygaard ØP, et al. Surgery for Degenerative Cervical Myelopathy: A Nationwide Registry-Based Observational Study With Patient-Reported Outcomes. Neurosurgery 2021;89:704-11.
83. Hilton B, Tempest-Mitchell J, Davies BM, et al. Cord compression defined by MRI is the driving factor behind the decision to operate in Degenerative Cervical Myelopathy despite poor correlation with disease severity. PLoS One 2019;14:e0226020.
84. Kada ka Z, Bednařík J, Novotný O, et al. Cervical spondylotic myelopathy: conservative versus surgical treatment after 10 years. Eur Spine J 2011;20:1533-8.
85. Tetreault L, Kopjar B, Côté P, et al. A Clinical Prediction Rule for Functional Outcomes in Patients Undergoing Surgery for Degenerative Cervical Myelopathy: Analysis of an International Prospective Multicenter Data Set of 757 Subjects. J Bone Joint Surg Am 2015;97:2038-46.
86. Rhee J, Tetreault LA, Chapman JR, et al. Nonoperative Versus Operative Management for the Treatment Degenerative Cervical Myelopathy: An Updated Systematic Review. Global Spine J 2017;7:35S-41S.
87. Tomii M, Mizuno J. Clinical Characteristics and Management of C3-4 Degenerative Cervical Myelopathy. Neurosurg Clin N Am 2018;29:153-8.
88. Koyanagi I. Options of Management of the Patient with Mild Degenerative Cervical Myelopathy. Neurosurg Clin N Am 2018;29:139-44.
89. Lawrence BD, Shamji MF, Traynelis VC, et al. Surgical management of degenerative cervical myelopathy: a consensus statement. Spine (Phila Pa 1976) 2013;38:S171-2.
90. Farrokhi MR, Ghaffarpasand F, Khani M, et al. An Evidence-Based Stepwise Surgical Approach to Cervical Spondylotic Myelopathy: A Narrative Review of the Current Literature. World Neurosurg 2016;94:97-110.
91. Wang MY, Shah S, Green BA. Clinical outcomes following cervical laminoplasty for 204 patients with cervical spondylotic myelopathy. Surg Neurol 2004;62:487-92; discussion 492-3.
92. Kato S, Nouri A, Wu D, et al. Comparison of Anterior and Posterior Surgery for Degenerative Cervical Myelopathy: An MRI-Based Propensity-Score-Matched Analysis Using Data from the Prospective Multicenter AOSpine CSM North America and International Studies. J Bone Joint Surg Am 2017;99:1013-21.
93. Piazza M, McShane BJ, Ramayya AG, et al. Posterior Cervical Laminectomy Results in Better Radiographic Decompression of Spinal Cord Compared with Anterior Cervical Discectomy and Fusion. World Neurosurg 2018;110:e362-6.
94. Lin JH, Chien LN, Tsai WL, et al. Reoperation rates of anterior cervical discectomy and fusion versus posterior laminoplasty for multilevel cervical degenerative diseases: a population-based cohort study in Taiwan. Spine J 2016;16:1428-36.
95. Joaquim AF, Ghizoni E, Tedeschi H, et al. Management of

degenerative cervical myelopathy - An update. Rev Assoc Med Bras (1992) 2016;62:886-94.
96. Smith GW, Robinson RA. The treatment of certain cervical-spine disorders by anterior removal of the intervertebral disc and interbody fusion. J Bone Joint Surg Am 1958;40-A:607-24.
97. Tasiou A, Giannis T, Brotis AG, et al. Anterior cervical spine surgery-associated complications in a retrospective case-control study. J Spine Surg 2017;3:444-59.
98. Ganau M, Holly LT, Mizuno J, et al. Future Directions and New Technologies for the Management of Degenerative Cervical Myelopathy. Neurosurg Clin N Am 2018;29:185-93.
99. Ghogawala Z. Anterior Cervical Option to Manage Degenerative Cervical Myelopathy. Neurosurg Clin N Am 2018;29:83-9.
100. Villavicencio AT, Rajpal S, Nelson EL, et al. Local Retropharyngeal Space Anesthetic for Dysphagia Reduction after Anterior Cervical Discectomy and Fusion Surgery: A Single-Center, Prospective, Randomized, Double-Blinded, Placebo-Controlled Clinical Trial. World Neurosurg 2021;146:e1377-83.
101. Satomi K, Nishu Y, Kohno T, et al. Long-term follow-up studies of open-door expansive laminoplasty for cervical stenotic myelopathy. Spine (Phila Pa 1976) 1994;19:507-10.
102. Seichi A, Takeshita K, Ohishi I, et al. Long-term results of double-door laminoplasty for cervical stenotic myelopathy. Spine (Phila Pa 1976) 2001;26:479-87.
103. Kiely PD, Quinn JC, Du JY, et al. Posterior surgical treatment of cervical spondylotic myelopathy: review article. HSS J 2015;11:36-42.
104. Grasso G, Sallì M, Torregrossa F. Does Hybrid Surgery Improve Quality of Life in Multilevel Cervical Degenerative Disk Disease? Five-Year Follow-up Study. World Neurosurg 2020;140:527-33.
105. Yilmaz M, Yucesoy K, Erbayraktar RS, et al. Anterior hybrid construction of multilevel cervical disc disease and spondylotic spinal stenosis: surgical results and factors affecting adjacent segment problems. J Orthop Surg Res 2021;16:298.
106. Sakai K, Yoshii T, Hirai T, et al. Impact of the surgical treatment for degenerative cervical myelopathy on the preoperative cervical sagittal balance: a review of prospective comparative cohort between anterior decompression with fusion and laminoplasty. Eur Spine J 2017;26:104-12.
107. Jannelli G, Nouri A, Molliqaj G, et al. Degenerative Cervical Myelopathy: Review of Surgical Outcome Predictors and Need for Multimodal Approach. World Neurosurg 2020;140:541-7.
108. Tetreault L, Ibrahim A, Côté P, et al. A systematic review of clinical and surgical predictors of complications following surgery for degenerative cervical myelopathy. J Neurosurg Spine 2016;24:77-99.
109. Khan I, Archer KR, Wanner JP, et al. Trajectory of Improvement in Myelopathic Symptoms From 3 to 12 Months Following Surgery for Degenerative Cervical Myelopathy. Neurosurgery 2020;86:763-8.
110. Vonck CE, Tanenbaum JE, Bomberger TT, et al. Short-term outcomes following posterior cervical fusion among octogenarians with cervical spondylotic myelopathy: a NSQIP database analysis. Spine J 2018;18:1603-11.
111. Madhavan K, Chieng LO, Foong H, et al. Surgical outcomes of elderly patients with cervical spondylotic myelopathy: a meta-analysis of studies reporting on 2868 patients. Neurosurg Focus 2016;40:E13.
112. Gibson J, Nouri A, Krueger B, et al. Degenerative Cervical Myelopathy: A Clinical Review. Yale J Biol Med 2018;91:43-8.
113. Zhang RJ, Shen CL, Zhang JX, et al. Clinical features and surgical outcomes of cervical spondylotic myelopathy in patients of different ages: a retrospective study. Spinal Cord 2018;56:7-13.
114. Wilson JRsF, Badhiwala JH, Moghaddamjou A, et al. Frailty Is a Better Predictor than Age of Mortality and Perioperative Complications after Surgery for Degenerative Cervical Myelopathy: An Analysis of 41,369 Patients from the NSQIP Database 2010-2018. J Clin Med 2020;9:3491.
115. Desimone A, Hong J, Brockie ST, et al. The influence of ApoE4 on the clinical outcomes and pathophysiology of degenerative cervical myelopathy. JCI Insight 2021;6:e149227.
116. Satkunendrarajah K, Nassiri F, Karadimas SK, et al. Riluzole promotes motor and respiratory recovery associated with enhanced neuronal survival and function following high cervical spinal hemisection. Exp Neurol 2016;276:59-71.

Cite this article as: Thompson K, Travers H, Ngan A, Reed T, Shahsavarani S, Verma RB. Updates in current concepts in degenerative cervical myelopathy: a systematic review. J Spine Surg 2024;10(2):313-326. doi: 10.21037/jss-23-123