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CERVICAL DEGENERATIVE MYELOPATHY, PANORAMIC REVIEW

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SUMMARY

Introduction: *cervical degenerative myelopathy (DCM) is a general term that entails multiple etiologies. DCM is a group of pathological entities that originate compression of the cervical spinal cord, where spasticity, hyperreflexia, pathological reflexes, loss of manual dexterity, among others, are found.*
Objective: *to detail the current information related to cervical degenerative myelopathy, epidemiology, clinical presentation, role of complementary examinations, pathophysiology, pathogenesis and treatment.*
Methodology: *a total of 32 articles were analyzed in this review, including review and original articles, as well as clinical cases, of which 21 bibliographies were used because the other articles were not relevant to this study. The sources of information were PubMed, Google Scholar and Cochrane; the terms used to search for information in Spanish, Portuguese and English were: cervical degenerative myelopathy, cervical spondylotic myelopathy, cervical pain, ossification of the yellow ligament and degenerative disc disease.*
Results: *cervical myelopathy occurs in almost all individuals with canal stenosis greater than 60%. Spinal cord injury (SCI) is a devastating disorder that influences remarkable neurological morbidity and significant wear and tear on quality of life. The incidence and prevalence of SCI in North America is between 4.1 and 60.5 per 100,000, robust epidemiological data is difficult to obtain because of the multifactorial etiology of the disease process.*
Conclusions: *cervical degenerative myelopathy involves multiple etiologies, all these entities together represent the most common reason for degenerative and non-traumatic alteration of the spinal cord in adult individuals. Degenerative disorders are more common at C5 and C6 or C6 and C7, because of the increased mobility in these sections. It is difficult to obtain solid epidemiological*

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data because of the multifactorial etiology of the disease process. The symptoms and signs presented by the pathology are varied, so an adequate physical examination and a good clinical history should be taken. The pathology can significantly alter neurological function and lead to disability, so early recognition and treatment can prevent further deterioration of affected individuals. Surgical decompression is almost universally indicated with the intention of preventing progression and achieving maximum recovery potential.

KEY WORDS: *myelopathy, cervical, degenerative, pain, spinal, neuralgia.*

INTRODUCTION

Cervical degenerative myelopathy (DCM) is an umbrella term that carries multiple etiologies, among them are cervical spondylotic myelopathy (CSM), ossification of the yellow ligament (OLF), ossification of the posterior longitudinal ligament (OPLL), and degenerative disc disease (DDD). All these entities together represent the most common reason for degenerative and non-traumatic spinal cord impairment in adult individuals(1-3).

The German Herbert von Luschka is credited with the discovery of the changes in the development of the anatomical structures of the cervical spine. Cervical degenerative myelopathy (DCM) is a group of pathological entities that cause compression of the cervical spinal cord, resulting in a clinical syndrome with spasticity, hyperreflexia, pathological reflexes, loss of manual dexterity, gait disturbances and sphincter dysfunction. Some individuals are more likely to form myelopathy in the cervical spine as a result of congenital cervical canal stenosis. Degenerative changes are more common at C5 and C6 or C6 and C7, because of increased mobility in these sections. There are also other factors that contribute to spinal canal stenosis such as hypertrophy of the yellow ligament, listhesis, osteophytosis and facet hypertrophy. Cervical myelopathy occurs in almost all individuals with canal stenosis greater than 60%, considering the sagittal disc space to be less than 6 mm. The pathology has an insidious onset, progressing gradually with reduced function. If the affected person does not receive treatment, it can lead to significant paralysis and loss of function. The level of neurological impairment can be measured using the modified Japanese Association Scale (mJOA) or the Nurick grade. Treatment sometimes requires anterior or posterior decompression surgery of the stenosed surface and sometimes fusion(1,4).

METHODOLOGY

A total of 32 articles were analyzed in this review, including review and original articles, as well as cases and clinical trials, of which 21 bibliographies were used because the information collected was not important enough to be included in this study. The sources of information were Cochrane, PubMed and Google Scholar; the terms used to search for information in Spanish, Portuguese and English were: cervical degenerative myelopathy, cervical spondylotic myelopathy, cervical pain, ossification of the yellow ligament and degenerative disc disease.

The choice of bibliography exposes elements related to cervical degenerative myelopathy, epidemiology, clinical presentation, role of complementary examinations, pathophysiology, pathogenesis and treatment.

DEVELOPMENT

Epidemiology

The incidence and prevalence of DCM in North America is between 4.1 and 60.5 per 100,000, solid epidemiological data are difficult to obtain because of the multifactorial etiology of the disease process. There are authors who estimate that degenerative diseases of the spine are among 59% of non-traumatic spinal cord injuries in Japan, 54% in the United States, 31% in Europe, 22% in Australia and 30% in Africa. It is further stated that the area incidence per million was 76 in North America, 26 in Europe and 6 in Australia. Although these data are not limited to cervical spinal cord impairment and several individuals with minor symptoms were excluded from multiple studies included in the review, it could be inferred that since DCM is one of the most common origins of non-traumatic cervical spinal cord injury, it is a major drawback for the North American population(1-3).

A clinical trial comparing magnetic resonance imaging (MRI) data from asymptomatic individuals showed that about 25% of individuals under 40 years of age had radiological findings compatible with cervical spondylosis. The incidence of these findings was approximately 60% among individuals older than forty years(5,6).

Clinical Presentation

Spinal cord injury (SCI) is a devastating disorder that results in marked neurological morbidity and significant impairment of quality of life.

The presentation of the affected individual presents with a wide range of symptoms ranging from barely perceptible dysfunction, through numbness or loss of motor dexterity, to severe dysfunction, such as tetraparesis and sphincteric impairment. It should be noted that paresthesias in the limbs are usually the first sign and because they are mild, they are often not recorded by affected individuals and health personnel(7).

The following clinical signs and symptoms may generally be present in individuals with DCM:

- Motor deficits.
- Numbness of the hands.
- Hoffmann's sign.
- Tenar atrophy.
- Ankle clonus.
- Inverted brachioradialis reflex.
- Hyperreflexia.
- Babinski's sign.
- Spasticity.
- Romberg's sign.
- Lhermitte's phenomenon.
- Impaired gait.

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- Incontinence.
- Clumsy hands.
- Weakness.
- Paresthesias(1).

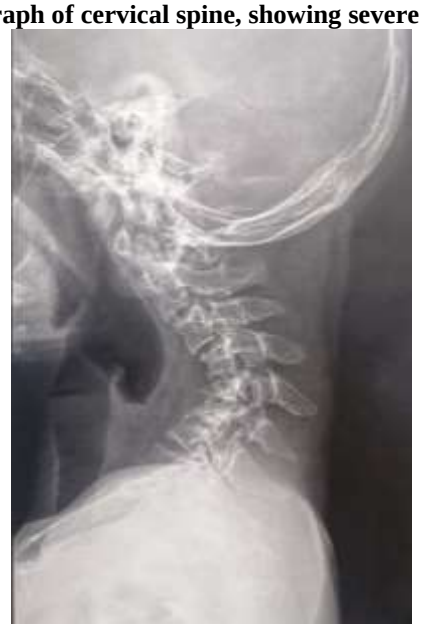
The myelopathic signs in the physical examination present different sensibility and specificity: hyperreflexia comprising between 72 and 43 percent, showing the biceps as the most sensitive and the brachioradialis the most specific, Hoffmann's sign comprising between 59 and 84 percent, brachioradialis reflex comprising between 51 and 81 percent, clonus comprising between 13 and 100 percent and Babinski comprising between 13 and 100 percent(8).

Role of Complementary Tests

The diagnosis of cervical degenerative myelopathy is complex. Somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP) are usually used to find objective evidence of functional abnormalities of the spinal cord(5).

Evaluation of cervical degenerative myelopathy usually includes plain radiographs. Lateral views allow assessment of spinal canal narrowing, disc height, presence of ossification of the posterior longitudinal ligament (OPLL), cervical sagittal alignment and subluxation. In individuals with cervical degenerative myelopathy, increased C2-C7 Cobb angles, superior C7 slopes, inferior C7 slopes and superior T1 slopes are usually shown(5,9).

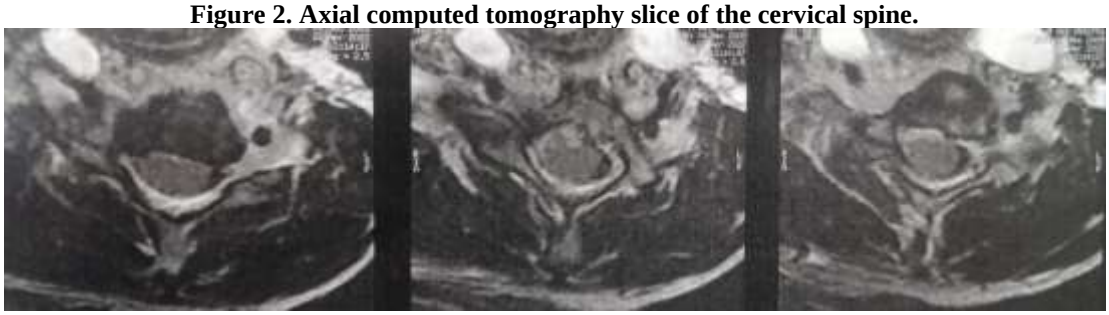
Figure 1. Lateral radiograph of cervical spine, showing severe cervical spine deformity.



Source: The Authors.

A study showing the correlation between preoperative computed tomography (CT) myelograms and clinical outcomes following the surgical procedure showed that individuals with greater spinal cord cross-sectional area at the degree of maximum compression had better outcomes. Other studies show that kinematic CT scans have limited potential to demonstrate myelopathy or correlate findings with clinical outcomes of DCM. Research projects based on tomography play a fundamental role in the diagnosis of diseases such as OPLL (10,11).

Figure 2. Axial computed tomography slice of the cervical spine.



Source: The Authors.

MRI can provide direct evidence of spinal cord compression, playing an important role in the choice of appropriate treatment, as well as predicting outcomes. Magnetic resonance imaging allows the observation of soft tissue structures such as intervertebral discs, showing early signs of degeneration, as well as spinal ligaments and other structures that are not easily visualized. At the moment there is no certainty as to whether there is a direct relationship between the amount of degeneration and changes in the cord signal independent of canal stenosis. MRI scans can find modifications in the signal intensity of the vertebral plaques. When related to disc degeneration, they are named Modic endplate changes (MEC).

There are 3 subtypes described in the literature according to MRI. Some bibliographies showed that type 2 modifications were the most common, mostly at C5-6 and C6-7 levels.

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However, SCMs are a dynamic phenomenon. Other studies show that the natural history of cervical degenerative myelopathy in 426 individuals with neck pain and the prevalence of cervical degenerative myelopathy type 1 increased from 7.4% to 8.2% after 2.5 years of follow-up. Likewise, the prevalence of type 2 increased from 14.5% to 22.3%. In addition, 12 segments with type 1 converted to type 2 during follow-up and no conversions from type 2 to type 1 were seen. MRI also gives the possibility to assess spinal canal stenosis(5,12,13).

Pathophysiology and Pathogenesis

Typically, degeneration of the cervical spine occurs over time as a result of structural loading, repetitive microtrauma and age-related modifications in the physiology of the bones, muscles and intervertebral discs. The degenerative process usually begins with disc wear, which acts to distribute pressure forces evenly across the vertebral plates and facet joints. Because of the decrease in proteoglycans and water, the discs lose their elastic and supporting capacity, beginning to create unequal pressure forces on adjacent vertebrae, triggering the formation of osteophytes. In addition, the vertebrae progressively lose their height and gradually widen. The completion of these processes converges in the form of spinal canal stenosis leading to chronic compression of the spinal cord and subsequent development of myelopathy. Thus, individuals with a narrow spinal canal or a large spinal cord or imbalance between the canal and the spinal cord are at increased risk of developing DCM over the course of their years. In addition to static injury, these significant morphologic changes can lead to increased mobility or both stable and unstable spondylolisthesis. Being unstable, increased range of motion may result in dynamic injury and repetitive minor trauma. From a pathophysiological point of view, spinal cord injury over time modifies the blood-spinal cord barrier, causing neuroinflammation, ischemia and apoptosis, which together allow demyelination, astrogliosis and axonal degeneration. This will later end with the manifestation of symptomatic myelopathy with its characteristic clinical findings(14-17).

Compression of the spinal cord by degenerative changes may also result from enlargement and ossification of the ligaments of the spinal canal, especially the posterior longitudinal ligament (PLL) and the yellow ligament (LF). Ligamentous enlargement may occur as a result of bulging of the disc into the canal resulting in enlargement of the PLL, which may progress to reactive ossification of the PLL, or loss of disc height, which usually results in buckling and infrequently ossification of the LF and compression of the spinal cord from the posterior aspect. In addition, genetic factors have been implicated in the formation of ossification of these spinal ligaments, so it is possible that these individuals do not have clear degenerative findings. Affected individuals from East Asia have been shown to be uniquely compromised by OPLL.

In the pathogenesis of the disease, 3 main factors can be observed: static, dynamic and histopathological.

Static Factors

These are structural elements of the spine that cause the narrowing of the spinal canal. The degenerative cascade of DCM commonly begins with deterioration of the intervertebral disc that gradually collapses and protrudes into the spinal canal, reducing its caliber. The reduction in disc height causes the spine to shorten and exhibit abnormal biomechanics. The yellow ligament can also cause posterior spinal cord compression by thickening and proliferation. The ossification of the posterior longitudinal ligament can lead to cervical degenerative myelopathy by direct compression of the spinal cord anteriorly. All these alterations cause stiffness of the cervical structures involved. To compensate for the reduction of motion at the affected sites, the adjacent regions of the spine become hypermobile.

Dynamic Factors.

Abnormal repetition of cervical spine motion during flexion and extension causes irritation and compression of the spinal cord. Flexion can compress the spinal cord anteriorly against osteophytes and intervertebral discs. Hyperextension may result in narrowing of the spinal cord between the posterior edges of the vertebral body anteriorly and the hypertrophied yellow ligament posteriorly.

Histopathological Factors.

Mechanical compression of the spinal cord leads to vascular modifications causing ischemia and inflammation. Chronic compression of the spinal cord can result in cell loss, degeneration of the posterior columns, anterior horn cells and endothelial damage due to dysfunction of the blood-brain barrier of the affected spinal cord, generating continuous functional deterioration(7).

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Figure 3. Surgical procedure for symptom improvement in cervical spine.

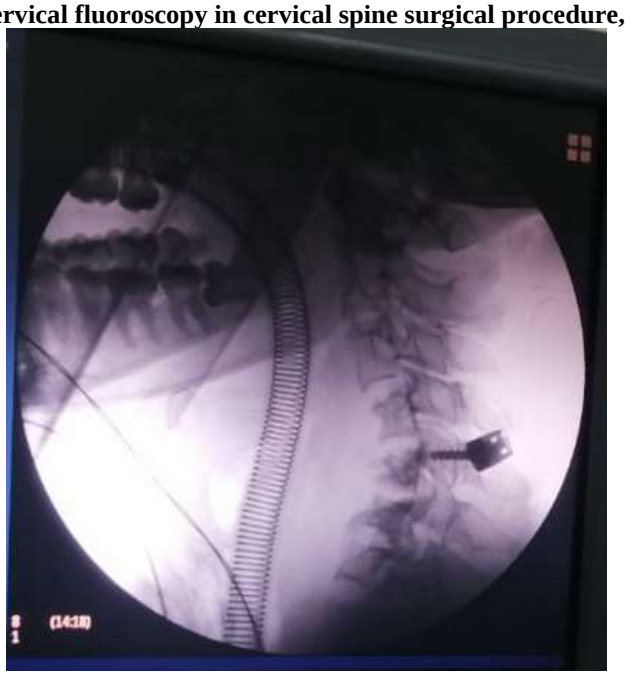
Source: The Authors.

Treatment

Cervical degenerative myelopathy is most often considered a surgical problem, with 20-62% of individuals worsening between 3 and 6 years of follow-up when management is expectant. In addition, in individuals who have asymptomatic cord compression, the incidence of symptomatic myelopathy formation is about 8% per year and almost 23% at 4 years of follow-up. A published article also shows improvement in all age groups treated surgically, with significant recovery within 1 week and 6 months after surgery. Surgical decompression is almost universally indicated with the intention of preventing progression and achieving maximum recovery potential.

Surgical approaches to decompress the spinal cord can be done by eliminating the causative compressive pathology, expanding the spinal canal through removal or manipulation of the posterior lamina of the vertebrae. Anterior approaches are preferred in individuals with cervical kyphosis and in individuals with large anterior pathology, while posterior approaches are indicated when you have multilevel cervical compression or OPLL. The literature suggests that adjunctive pharmacologic treatment may be used, addressing glutamate-induced excitotoxicity with riluzole, accompanied by surgical decompression. Conservative treatment may be indicated when it is the choice of the affected individual or if surgical risk is unacceptable. Conservative treatment options include structured and careful physiotherapy, the use of a soft collar, massage therapy, analgesics and antineuralgic drugs, however there is limited evidence regarding efficacy(7,14,18-21).

Figure 4. Cervical fluoroscopy in cervical spine surgical procedure, first stage.



Source: The Authors.

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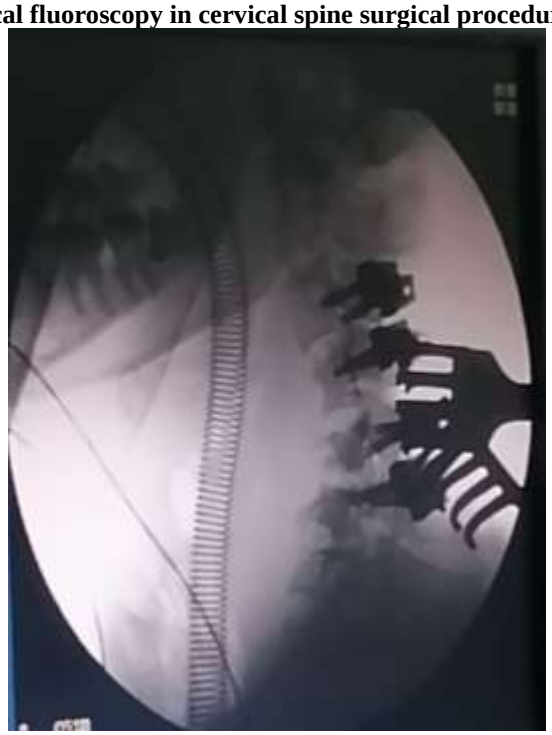
Figure 5. Cervical fluoroscopy in cervical spine surgical procedure, second stage.



Source: The Authors.

CONCLUSIONS

Cervical degenerative myelopathy involves multiple etiologies, all these entities together represent the most common reason for degenerative and non-traumatic alteration of the spinal cord in adult individuals. Degenerative disorders are more common at C5 and C6 or C6 and C7, because of the increased mobility in these sections. It is difficult to obtain solid epidemiological data because of the multifactorial etiology of the disease process. The symptoms and signs presented by the pathology are varied, so an adequate physical examination and a good clinical history should be taken. The pathology can significantly alter neurological function and lead to disability, so early recognition and treatment can prevent further deterioration of affected individuals. Surgical decompression is almost universally indicated with the intention of preventing progression and achieving maximum recovery potential.

BIBLIOGRAPHY

1. *Gibson J, Nouri A, Krueger B, Lakomkin N, Nasser R, Gimbel D, et al. Degenerative Cervical Myelopathy: A Clinical Review. Yale J Biol Med. 2018 Mar;91(1):43–8.*
2. *New PW, Cripps RA, Bonne Lee B. Global maps of non-traumatic spinal cord injury epidemiology: towards a living data repository. Spinal Cord. 2014 Feb;52(2):97–109.*
3. *Nouri A, Tetreault L, Singh A, Karadimas SK, Fehlings MG. Degenerative Cervical Myelopathy: Epidemiology, Genetics, and Pathogenesis. Spine. 2015 Jun 15;40(12):E675-693.*
4. *Pinto EM, Teixeira A, Frada R, Atilano P, Oliveira F, Miranda A. DEGENERATIVE CERVICAL MYELOPATHY: A REVIEW OF CURRENT CONCEPTS. Coluna/Columna. 2020 Dec;19(4):302–7.*
5. *Tu J, Vargas Castillo J, Das A, Diwan AD. Degenerative Cervical Myelopathy: Insights into Its Pathobiology and Molecular Mechanisms. J Clin Med. 2021 Mar 15;10(6):1214.*
6. *Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine in asymptomatic subjects. A prospective investigation.: J Bone Jt Surg. 1990 Sep;72(8):1178–84.*
7. *Yurac R, Matamala JM, Zamorano JJ, Harrop JS, Davies BM, Nouri A, et al. Mielopatía cervical degenerativa: una patología cada vez más frecuente y que requiere diagnóstico y manejo precoz. Rev Médica Chile. 2022 Mar;150(3):339–52.*
8. *Rhee JM, Heflin JA, Hamasaki T, Freedman B. Prevalence of physical signs in cervical myelopathy: a prospective, controlled study. Spine. 2009 Apr 20;34(9):890–5.*
9. *Scheer JK, Tang JA, Smith JS, Acosta FL, Protopsaltis TS, Blondel B, et al. Cervical spine alignment, sagittal deformity, and clinical implications: A review. J Neurosurg Spine. 2013 Aug;19(2):141–59.*
10. *Abiola R, Rubery P, Mesfin A. Ossification of the Posterior Longitudinal Ligament: Etiology, Diagnosis, and Outcomes of Nonoperative and Operative Management. Glob Spine J. 2016 Mar;6(2):195–204.*
11. *Blease Graham Iii C, Wippold Ii FJ, Bae KT, Pilgram TK, Shaibani A, Kido DK. Comparison of CT Myelography Performed in the Prone and Supine Positions in the Detection of Cervical Spinal Stenosis. Clin Radiol. 2001 Jan;56(1):35–9.*
12. *Gao X, Li J, Shi Y, Li S, Shen Y. Asymmetrical degenerative marrow (Modic) changes in cervical spine: prevalence, correlative factors, and surgical outcomes. J Orthop Surg. 2018 Dec;13(1):85.*
13. *Modic MT, Steinberg PM, Ross JS, Masaryk TJ, Carter JR. Degenerative disk disease: assessment of changes in vertebral body marrow with MR imaging. Radiology. 1988 Jan;166(1 Pt 1):193–9.*
14. *Fengbin Y, Deyu C, Xinwei W, Yu C, Jinhao M, Xinyuan L, et al. Trauma-induced spinal cord injury in cervical spondylotic myelopathy with or without lower cervical*

instability. J Clin Neurosci Off J Neurosurg Soc Australas. 2013 Mar;20(3):419–22.

15. *Nouri A, Montejo J, Sun X, Virojanapa J, Kolb LE, Abbed KM, et al. Cervical Cord-Canal Mismatch: A New Method for Identifying Predisposition to Spinal Cord Injury. World Neurosurg. 2017 Dec;108:112–7.*
16. *Matsunaga S, Kukita M, Hayashi K, Shinkura R, Koriyama C, Sakou T, et al. Pathogenesis of myelopathy in patients with ossification of the posterior longitudinal ligament. J Neurosurg. 2002 Mar;96(2 Suppl):168–72.*
17. *Morishita Y, Naito M, Hymanson H, Miyazaki M, Wu G, Wang JC. The relationship between the cervical spinal canal diameter and the pathological changes in the cervical spine. Eur Spine J Off Publ Eur Spine Soc Eur Spinal Deform Soc Eur Sect Cerv Spine Res Soc. 2009 Jun;18(6):877–83.*
18. *Fehlings MG, Tetreault LA, Wilson JR, Skelly AC. Cervical spondylotic myelopathy: current state of the art and future directions. Spine. 2013 Oct 15;38(22 Suppl 1):S1-8.*
19. *Zhang RJ, Shen CL, Zhang JX, Zhang XJ, Dong FL, Tao H, et al. Clinical features and surgical outcomes of cervical spondylotic myelopathy in patients of different ages: a retrospective study. Spinal Cord. 2018 Jan;56(1):7–13.*
20. *Fehlings MG, Wilson JR, Kopjar B, Yoon ST, Arnold PM, Massicotte EM, et al. Efficacy and safety of surgical decompression in patients with cervical spondylotic myelopathy: results of the AOSpine North America prospective multi-center study. J Bone Joint Surg Am. 2013 Sep 18;95(18):1651–8.*
21. *Fehlings MG, Wilson JR, Yoon ST, Rhee JM, Shamji MF, Lawrence BD. Symptomatic progression of cervical myelopathy and the role of nonsurgical management: a consensus statement. Spine. 2013 Oct 15;38(22 Suppl 1):S19-20.*

Conflict of Interest Statement

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