

To Evaluate the Role of MRI in Assessment of Myelopathy.

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Abstract

Background: Myelopathy is a disease of spinal cord which can lead to significant neurological morbidity. MRI because of better soft tissue differentiation, not only helps in determining the cause of myelopathy but also plays significant role in their management. **Subjects and Methods:** A cross sectional study was done on 80 patients in our institute between June 2017 to October 2018. All patients having complaints of pain in the back with sensory or motor dysfunction or both, having suspicion of myelopathy, referred to the department of Radio-diagnosis for MRI spine evaluation. MRI scan performed on 1.5 Tesla PHILIPS ACHIEVA machine, standard surface & body coils were used for acquisition of images. **Results:** In this study out of 80 patients of myelopathy, 65% were due to cord compression and 35% were due to non compressive causes. Involvement of cervical spine was more common than the thoraco-lumbar spine. Most common condition associated with compression of the cord was Degenerative spine disease followed by Potts spine and trauma. Under the causes of Non compressive myelopathy- Myelitis was the most common cause, which was seen most in Isolated cases followed by associations with Multiple sclerosis and Neuromyelitis optica. **Conclusion:** MRI is the mainstay modality to diagnose spinal cord pathologies. Degenerative spine disease was the most common cause of compressive myelopathy and Myelitis was the most common cause of Non compressive myelopathy. Early diagnosis using MRI improves the prognosis of myelopathy.

Keywords: MRI, Myelopathy.

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Introduction

Myelopathy is the disease of the spinal cord which clinically is diagnosed by neurological localisation only; hence imaging plays a crucial role in its diagnosis. Myelopathy can be grossly categorised in two broad categories of Non compressive and Compressive myelopathy. Majority of these conditions are managed medically, some cases of compressive myelopathy are managed surgically and at times even in emergency. Hence early and efficient diagnosis of these myelopathies is important. Our present study is aimed at familiarising the clinicians in general and the radiologist in particular about the etiological assessment of myelopathies by MRI.

Subjects and Methods

A cross sectional study was done on 80 patients in our institute between June 2017 to October 2018. All patients having complaints of pain in the back with sensory or motor dysfunction or both, having strong suspicion of myelopathy, those referred from other departments to the department of Radio-diagnosis for MRI spine evaluation were included in study. MRI was done on 1.5 Tesla PHILIPS ACHIEVA machine, standard surface and body coils, were used for cervical, thoracic and lumbar spine for acquisition of

images. Conventional spin echo sequences T1WI, T2WI, Sag, STIR sag, T1WI, T2WI axial & FFE axial, and post contrast T1W axial, Sag and coronal. Gadolinium contrast was used. CE MRI -Orbit & Bain were also included in the study S.O.S. Images were analysed by expert radiologists and were categorised as compressive and Non-compressive, also were classified as per etiologies based on MRI findings.

Results

In our study 24 patients were female and 56 patients were male. Maximum patients belonged to the age group of >40 years (85%). In this study compressive causes were the most common cause of myelopathy (Table-1) with predominant involvement of cervical more than thoraco-lumbar spine [Figure 1]

Table 1: Etiological distribution of myelopathy cases

	Etiology	Total patients	Percentage
Compressive	Degenerative disc disease	32	40
	Trauma	9	11.25
	Potts spine	6	7.5
	Syrinx	4	5
	Spinal A-V Malformation	1	1.25
Non compressive	Myelitis (Isolated or associated with MS, ADEM, NMO, SACS etc)	26	32.5
	Others	2	2.5

In the present study, 32 cases of compressive myelopathy due to degenerative causes were having cervical & lumbar spondylosis and showed high intramedullary signal intensity on T2W images. In our study 60% patients of degenerative disc disease developed myelopathy due to disc osteophyte complexes, 20% patients had diffuse disc bulges and 10% patients had other causes of myelopathy (diffuse disc bulges with osteophytes, ligamentum flavum hypertrophy etc). Second most common condition associated with compression of the cord was Trauma, seen in 11.25% of our patients, followed by Potts spine [Table 1].

In our study 28 cases were categorised under the causes of Non compressive myelopathy- Myelitis was the most common cause, which was seen most in Isolated cases (50% patients) followed by associations with MS, NMO, Sub acute combined degeneration of spinal cord and ADEM. In our study we had 4 patients (14%) of SADC, who had increased T2 signal intensity in the spinal cord predominantly in the dorsal column, which showed no post contrast enhancement. In our study we had 3 cases of MS (11%), 2 cases of ADEM (7%) and 3 cases of NMO (11%) who had myelopathy diagnosed on MRI.

In our study we had 2 cases (7%) of Spinal artery ischemia syndrome who had underwent cardiac surgeries/stroke in the past.

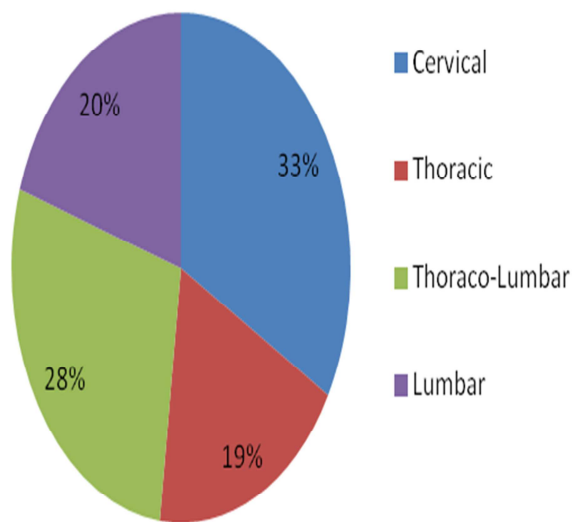
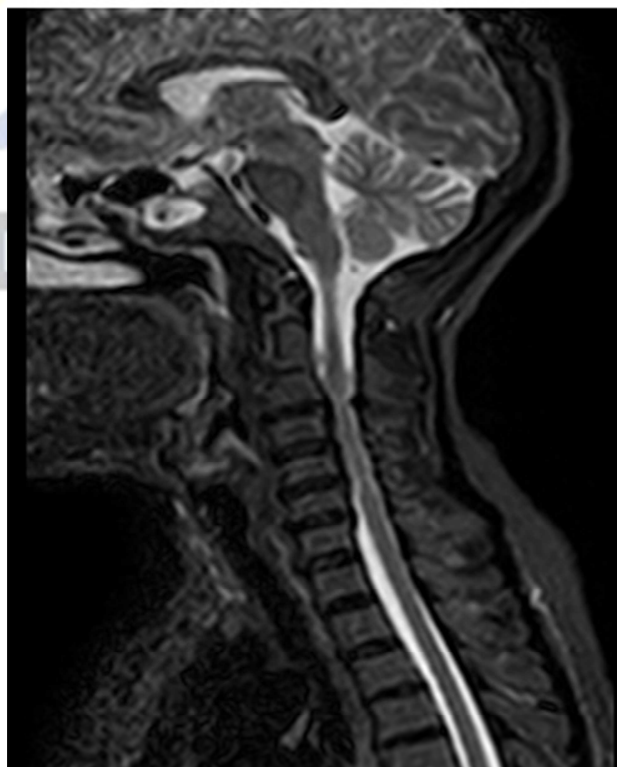


Figure 1: Diagram showing the level of spinal cord involvement.

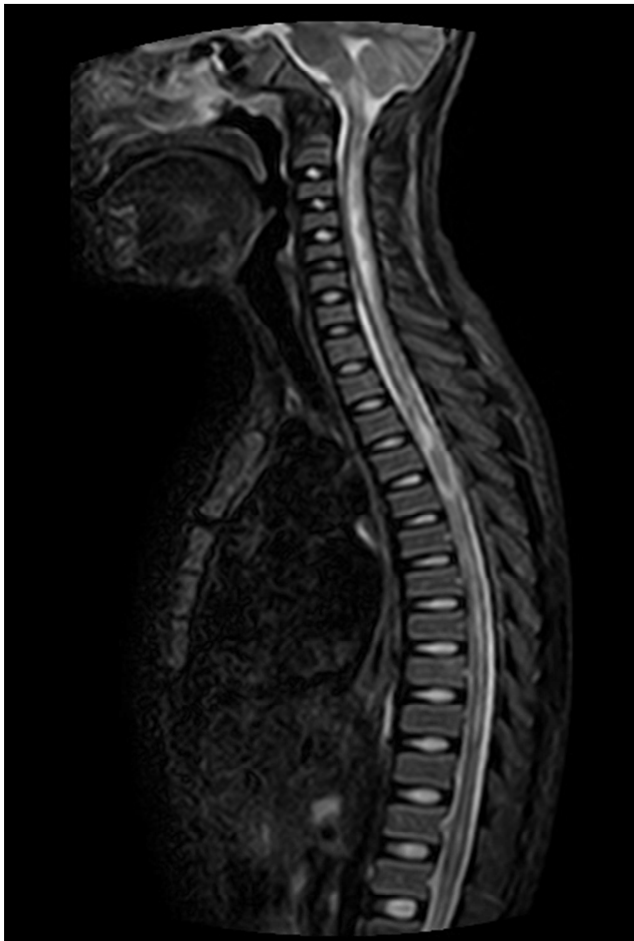


A. T2W sagittal

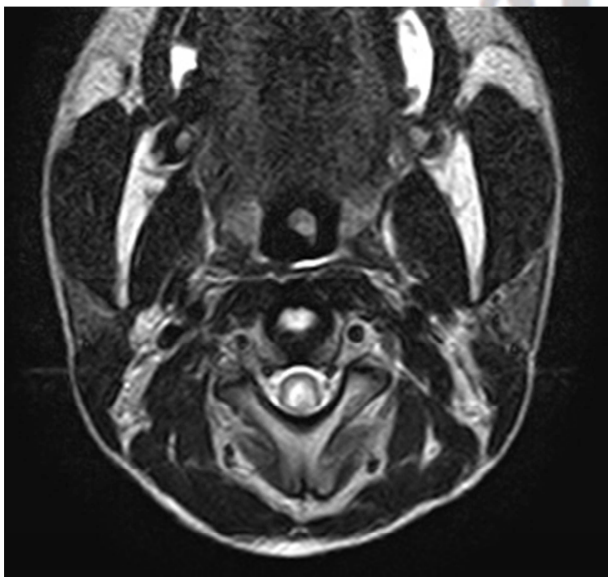


B. T2W-STIR sagittal

Figure 2: A 65 year old female having complaints of bilateral upper and lower limb weakness since 1 month, on MRI cervical spine had disc osteophyte complexes with ligamentum flavum hypertrophy was seen at multiple levels in the cervical spine causing compression of the cord and resultant T2 (a) and STIR (b) hyperintensity within the cord on sagittal images – Compressive Myelopathy.



A. T2W-STIR

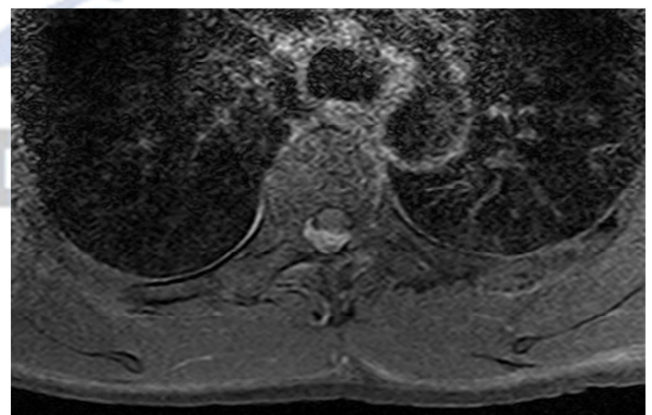


B. T2W axial

Figure 3: A 16 year old male having sudden onset bilateral lower limb weakness since 8 days. On MRI long segment T2 hyperintensity is noted in the spinal cord from the CV junction upto the D10 level (a, b) was diagnosed to have Acute transverse myelitis



A. T2W-SAG



B. T1 Axial-Post contrast

Figure 4: A 42 year old male, K/c/o Pulmonary Tuberculosis-took incomplete medication had bilateral lower limb weakness since 15 days. On MRI T2 hyperintensity in the spinal cord from the D3 to D5 level (a), secondary to compression of the cord by adjacent paraspinous abscess (b).

Discussion

Myelopathy is more common in older age group (age >40 years). Male population is predominantly affected. Spinal cord pathologies manifest in their severe most form as para or quadriplegia &/or severe sensory deficits. Myelopathy can be divided into two broad categories: Compressive and Non-Compressive. Compressive causes usually have chronic course,

rarely re-occur and also are the most common cause of myelopathy in geriatric patients^[1]. Increased T2W signals in the cord can be seen in degenerative changes, trauma, vascular malformations, tumors etc. Age related changes occur in the form disc bulges, disc protrusions/ herniation and end plate osteophytes which end up compressing the spinal cord resulting in changes within it. MRI in these cases can evaluate increased T2W signal in the cord as explained by the study of Aria Nouri et al.^[2] In our study degenerative causes were the most common cause of myelopathy and the findings of the study are in concordance with study of Navya Sindhu .V. et al^[3] .In patients with trauma, compression of the cord may occur secondary to compression by the retropulsion of the fracture fragment or compression by the epidural hematoma. Chance fractures are usually associated with cord transection. Retropulsion of the fracture fragment was the most common cause of myelopathy amongst the trauma patient in our study seen in 7 patients (64%), followed by epidural hematoma seen in 4 patients (36%) with traumatic myelopathy. In developing countries TB Spine is still one of the important cause of myelopathy according to the study of N.K. Kadam et al.^[4] However, in our study trauma was the more common cause of myelopathy than TB. Tuberculosis of spine occurs usually through spread by haematogenous route via the Batson's plexus^[5]. The thoracolumbar spine being the most frequent site of involvement.^[6] On MRI endplates irregularities with marrow edema and dural, marrow and subligamentous discal enhancement on post contrast study are the usual findings.^[4] In our study, degenerative cause (Cervical spondylosis) was the most common cause of myelopathy, followed by caries spine. These findings are in concordance with the study of Sreeramulu et al^[7] in 2015. Increased intramedullary signal intensity (T2W) in the cervical spine indicates poor prognosis^[8] Non compressive myelopathy - Once the causes of compression are ruled out , the clinical history and careful clinical examination of the patient are needed to look for other causes spinal cord pathologies like transverse myelitis, Acute Disseminated encephalomyelitis etc. Acute transverse myelitis is an inflammatory condition in which both the halves of cord are involved. It has a rapid sensory, motor and autonomic dysfunction which is mostly progressive in nature. On MRI , more than three segments of the cord (Long segment) show increased T2W signal intensity, occupy more than 2/3rd of the area(cross sectional) of the cord and show variable post contrast enhancement with no restricted diffusion^[9]. In our study 14 patients (50%) had long segment increased T2W signal intensity in the cord and showed variable post contrast enhancement. Acute disseminated encephalomyelitis (ADEM) develops within days to weeks, in patients post vaccination or viral illness^[10] .Patients complaints of headache, acute hemiparesis, seizures, nuchal rigidity, cerebellar ataxia, cranial nerve palsies and altered sensorium^[11]. Brain is the most commonly

involved in ADEM patients with spinal cord disease in about a quarter of patients.^[11] ADEM is usually seen in children of <10-years^[12], but can occur in all age groups. It can be monophasic, multiphasic – that is the second event occurred in some different area, or recurrent – where the second event occurs in the same area after 3 months from the first event^[10]. About of quarter of ADEM patients subsequently develop Multiple Sclerosis^[13] .In ADEM of spinal cord, poorly marginated T2W increased signal intensity is noted in the spinal cord for a length of two to three vertebral bodies, which rarely show post contrast enhancement. Similar MRI findings were noted in 2 patients (7%) of ADEM in our study. Multiple sclerosis (MS) is a chronic relapsing demyelinating disease involving the central nervous system. After trauma , Multiple sclerosis is the second most common cause of neurological impairment in young adults^[11]. According to the revised McDonald criteria – more than or equal to five T2W hyperintensities, one brainstem lesion and more than equal to two periventricular lesions ; have increased chances of diagnosis in patients more than eleven years without having features of ADEM^[13,15] . Oligoclonal bands are one of the most specific finding in patients of MS, which is seen in more than 90 per cent patients^[16]. Plaques of Multiple sclerosis seen in the spinal cord are usually smaller (than the lesions of Transverse myelitis), poorly marginated, patchy, and mostly involve the posterolateral aspect of the cord. Similar MRI findings were noted in 3 patients (11%) of Multiple sclerosis in our study. Neuromyelitis optica (NMO), or Devic's disease is considered by some, as a triad of long segment myelitis, optic neuritis and presence of Aqua-porin4 antibodies. In paediatric age group, it is more often seen in non white and children with some systemic autoimmune disease, such as SLE^[17] . Optic neuritis usually presents as vision loss & pain in the eyes and bilateral optic neuritis is more frequently seen in children than adults^[18,19]. The most specific marker for this condition is the NMO-IgG (Neuromyelitis optica immunoglobulin G), who's presence can help in early diagnosis^[20] . On MRI increased signal intensity is noted in the cord on T2W images for more than three segments which shows enhancement on post contrast study in acute lesions. Swelling within the cord may be seen. Similar MRI findings were noted in 3 patients (11%) of Neuromyelitis optica in our study. Sub-acute Combined Degeneration of spinal cord is seen in patients with vitamin B12 deficiency. On MRI increased signal intensity on T2W images is seen in the posterior column with no enhancement on post contrast study^[21]. Clinical improvement usually correlates with the imaging findings.^[22] Similar MRI findings were noted in 4 patients (14%) of SACS in our study. Spinal Artery Ischemia syndrome-Acute spinal cord ischemia syndrome is seen only in 5-8% of all acute myelopathies^[23,24] and <1% of all stroke patients^[25]. Patients usually present with severe back pain the most commonly (60-70%), followed by loss

of bladder (60%) and bowel control (40%)^[23,24]. The anterior spinal cord is usually the most commonly involved, with sensory symptoms (60%)^[23], first to be noticed. Involvement of the anterior and central portion of the spinal cord (mostly bilaterally), giving the so-called owl eye appearance^[26] on axial T2W images are seen in the anterior spinal artery ischemia. In our study we had two such patients having the similar appearance on T2W images.

Management of myelopathy depends on the cause of myelopathy. Non surgical management of myelopathy includes application of braces, physical therapy and medical management. Role of surgery in the management of myelopathy is in the conditions where compression of the spinal cord is there due to one or the other cause (e.g. compression due to herniated discs or by bony spurs), in such conditions decompression surgery is the mainstay procedure for removal of the pressure symptoms. Laminoplasty is done in cases where spinal canal stenosis is involved^[27]. In patients where laminoplasty is not indicated then decompression with vertebral body fusion is also a method of management. However delayed decompressive surgeries can also worsen the myelopathy.^[28]

Conclusion

MRI is the mainstay modality to diagnose spinal cord pathologies. Myelopathy is usually seen in age above 40 years & is more common in cervical spine; with degenerative disease being most common cause of compressive & Myelitis being the most common cause of non-compressive myelopathy, respectively. However, TB spine is still an important cause of myelopathy in developing countries. Prognosis of the reversible causes of myelopathy can be greatly improved with the use of MRI in diagnosing these conditions in their early stages.

References

- Ghezzi A Baldini SM, Zaffaroni M. Differential diagnosis of acute myelopathies. *Neurol Sci.* 2001;22(Suppl 2):S60-4.
- Nouri A, Martin AR, Mikulis D, Fehlings MG. Magnetic resonance imaging assessment of degenerative cervical myelopathy: a review of structural changes and measurement techniques. *Neurosurgical focus.* 2016 Jun;40(6):E5.
- Navya Sindhu et al.2017, Role of Mri In The Evaluation of Compressive Myelopathy. *Int J Recent Sci Res.* 8(4), pp. 16396-16403. DOI: <http://dx.doi.org/10.24327/ijrsr.2017.0804.0141>
- Kadam NK, Gehlot K. A Study Of Role Of Magnetic Resonance Imaging In The Evaluation Of Compressive Myelopathy. *Int Res Pub Med Sci.* 2015;1(3):52-56.
- Burrill J, Williams CJ, Bain G et-al. Tuberculosis: a radiologic review. *Radiographics.* 27 (5): 1255-73. doi:10.1148/rg.275065176 - Pubmed citation
- Spinal Tuberculosis: A Study Of The Disease Pattern, Diagnosis And Outcome Of Medical Management In Sri Lanka BMGD
- Yasaratne1, SNR Wijesinghe2 and RMD Madegedara [Indian J Tuberc 2013; 60: 208-216]
- Sreeramulu Diguvinti, Chennakesavulu Dara, et al. Clinico-Mri Correlation of Compressive Myelopathy. *IJAR* 2015; 1(7): 60-64
- Shin JJ. Intramedullary high signal intensity and neurological status as prognostic factors in cervical spondylotic myelopathy. *Acta Neurochir (Wien)* 2010;152(10):1687-1694. doi: 10.1007/s00701-010-06928. [PubMed] [CrossRef]
- TRANSVERSE MYELITIS: PATHOGENESIS, DIAGNOSIS AND TREATMENT Chitra Krishnan 1 , Adam I. Kaplin 2 , Deepa M. Deshpande 1 , Carlos A. Pardo 1 and Douglas A. Kerr . [Frontiers in Bioscience 9, 1483-1499, May 1, 2004]
- Krupp LB, Banwell B, Tenenbaum S. Consensus definitions proposed for pediatric multiple sclerosis and related disorders. *Neurology.* 2007;68(16 Suppl 2):S7-12.
- Barkovich AJ, Raybaud C. *Pediatric neuroimaging.* 5th ed. New York, NY: Raven Press; 2011.
- Banwell B Ghezzi A, Bar-Or A, et al. Multiple sclerosis in children: Clinical diagnosis, therapeutic strategies, and future directions. *Lancet Neurol.* 2007;6:887-902.
- Callen DJ, Shroff MM, Branson HM et al. MRI in the diagnosis of pediatric multiple sclerosis. *Neurology.* 2009;72:961-967.
- Sarbu N, Shih RY, Jones RV et-al. White Matter Diseases with Radiologic-Pathologic Correlation. *Radiographics.* 2016;36 (5): 1426-47. doi:10.1148/rg.2016160031 - Pubmed citation
- Sadaka Y, Verhey LH, Shroff MM, et al. 2010 McDonald criteria for diagnosing pediatric multiple sclerosis. *Ann Neurol.* 2012;72:211-223.
- Tortori-Donati P, Rossi A. *Pediatric Neuroradiology.* New York: Springer; 2005:1716.
- McKeon A., Lennon VA, Lotz T et al. CNS aquaporin-4 autoimmunity in children. *Neurology.* 2008;71:93-100.
- The clinical profile of optic neuritis. Experience of the optic neuritis treatment trial. *Optic neuritis study group. Arch Ophthalmol.* 1991; 109:1673-1678.
- Wilejto M, Shroff M, Buncic JR, et al. The clinical features, MRI findings, and outcome of optic neuritis in children. *Neurology.* 2006; 67:258-262.
- Lotze TE, Northrop JL, Hutton GJ, et al. Spectrum of pediatric neuromyelitis optica. *Pediatrics.* 2008;122: e1039-1047.
- al Deeb SM, Yaqub BA, Bruyn GW, et al. Acute transverse myelitis. A localized form of postinfectious encephalomyelitis. *Brain.* 1997;120:1115-22
- DeSanto J, Ross JS. Spine infection/inflammation. *Radiol Clin North Am.* 2011;49:105-27.
- Nedelchev K, Loher TJ, Stepper F et-al. Long-term outcome of acute spinal cord ischemia syndrome. *Stroke.* 2004;35 (2): 560-5. *Stroke* - doi:10.1161/01.STR.0000111598.78198.EC
- Vargas MI, Gariani J, Sztajzel R et-al. Spinal cord ischemia: practical imaging tips, pearls, and pitfalls. *AJNR Am J Neuroradiol.* 2014;36 (5): 825-30. doi:10.3174/ajnr.A4118
- Novy J, Carruzzo A, Maeder P et-al. Spinal cord ischemia: clinical and imaging patterns, pathogenesis, and outcomes in 27 patients. *Arch. Neurol.* 2006;63 (8): 1113-20. doi:10.1001/archneur.63.8.1113
- Masson C, Pruvo JP, Meder JF et-al. Spinal cord infarction: clinical and magnetic resonance imaging findings and short term outcome. *J. Neurol. Neurosurg. Psychiatr.* 2004;75 (10): 1431-5. *J. Neurol. Neurosurg. Psychiatr.* (full text) - doi:10.1136/jnnp.2003.031724
- Central Decompressive Laminoplasty for Treatment of Lumbar Spinal Stenosis : Technique and Early Surgical Results *J Korean Neurosurg Soc.* 2014 Sep; 56(3): 206-210. Published online 2014 Sep 30. doi: [10.3340/jkns.2014.56.3.206] PMID: PMC4217056 . PMID: 25368762
- Delayed decompression exacerbates ischemia-reperfusion injury in cervical compressive myelopathy *JCI Insight.* 2017 Jun 2; 2(11): e92512. Published online 2017 Jun 2. doi: [10.1172/jci.insight.92512]

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