Radiology Case Reports 20 (2025) 3231–3235

Available online at www.sciencedirect.com

ScienceDirect

journal homepage: www.elsevier.com/locate/radcr

Case Report

Radiographic changes in delayed white cord syndrome from postsurgical cervical myelopathy: A case report and review of the literature ☆,☆☆

*Serena Sheen, Volney Sheen**

Department of Neurology, Beth Israel Deaconess Medical Center and Harvard Medical School, Boston, MA 02115, USA

ARTICLE INFO

Article history:
Received 20 January 2025
Revised 16 March 2025
Accepted 17 March 2025

Keywords:
Case report
Cervical myelopathy
Delayed
White cord syndrome

ABSTRACT

White cord syndrome (WCS), also referred to as reperfusion injury of the spinal cord, is a rare condition involving sudden neurological deterioration following cervical spinal decompression. The syndrome is diagnosed in the absence of an iatrogenic cord injury or perioperative complications. Both loss of neurophysiological signaling during intraoperative monitoring and the appearance of hyper-intensity on T2-weighted magnetic resonance imaging are hallmarks of WCS. We present a report of a female patient who presented with the condition and followed her imaging studies longitudinally. Imaging studies showed prolonged and persistent contrast enhancement over a year and a half postsurgery. Such findings have not previously been reported with WCS. We provide a brief review of the literature, highlighting the main radiologic findings.

Introduction

White cord syndrome (WCS), also known as spinal cord reperfusion injury, refers to sudden neurological deterioration following cervical decompression surgery. Loss of neurophysiological signaling during surgery and the appearance of an increase in signal intensity on T2-weighted magnetic resonance imaging (MRI) radiographically are hallmarks of this disorder [1–4]. Moreover, an increase in the extension of the hyperintensity on T2-weighted images is often seen in a previously compressed cord with compressive myelopathy. Here we present a case of WCS, which showed persistent contrast enhancement on T1-weighted imaging in the effected cervical cord region for over 19 months. Additional evaluation for causes of enhancement excluded other diagnostic possibilities. In general, contrast enhancement is not immediately seen after WCS, albeit no other studies have actually looked at the extended later time points described in this case report.

☆ Competing Interests: The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

☆☆ Acknowledgments: This work was supported by NIH 1 R01 HD109794-01 to V Sheen.

* Corresponding author.
E-mail address: vsheen@bidmc.harvard.edu (V. Sheen).

https://doi.org/10.1016/j.radcr.2025.03.062

These observations add to the radiological features associated with WCS.

Case report

A 48-year-old female with no significant past medical history presented with bilateral hand pain and numbness for one month and gait disturbance for 3 weeks. Pain and numbness primarily resided in the bilateral first 2 digits, although the pain intermittently radiated up her arms. Orthopedic surgery evaluation and injection for carpal tunnel syndrome provided no relief. Gait issues developed while traveling for work with patient endorsing generalized weakness as if all her limbs felt fatigued. Neurological examination was notable for diffuse upper extremity weakness, left greater than right, and intact reflexes and sensation. MRI of cervical spine showed severe spinal canal stenosis at the cervical 4-5 (C4-5) and C5-6 levels with associated abnormal spinal cord T2-hyperintensity signal at these levels (Fig. 1A). No contrast enhancement was seen on T1-weight MRI at this time. She underwent C3-7 laminectomy with C3-6 posterior fusion decompression. The procedure was notable for loss of neurophysiological signals that did recover to near baseline over the course of the surgery with the exception of some diminished motor evoked potentials in the left hand. No surgical complications were noted, and appropriate positioning of the hardware was confirmed by cervical spine radiographs (Fig. 2A,B). Postoperatively, the patient had worsening left upper weakness and underwent a course of steroids as well as ICU management to maintain mean arterial pressure (MAP) > 85 mm Hg with some improvement.

At 2 months postoperatively, the patient continued to have persistent left hand weakness (3/5 in hand intrinsic muscles) and hyperesthesia. MRI of the cervical spine at this time (Fig. 1B) showed postsurgical changes related to posterior decompression, instrumented fusion at C3-C6 and no spinal cord encroachment. T2-weighted hyperintensity was seen most prominently at the C4-C5 level, which was improved over the left aspect of the spinal cord compared to the preoperative scan. At 4 months, the patient began feeling better, strength improved in the left hand (4/5 in hand intrinsic muscles), although residual paresthesia continued in the first 2 digits of the left hand.

At 5 months postoperatively, symptoms worsened with extension of the pain and paresthesia to the right hand. At eleven months, she conveyed these symptoms during Neurosurgical clinic, and exam was notable for the same weakness and sensory issues in the left hand but also sensory change in the right first 3 digits, with positive Tinel's sign and Phalen's test. Repeat MRI of cervical spine was obtained to exclude adjacent segment disease or any other compressive etiology. Imaging showed increased T2 hyperintensity along the central cord (Fig. 1C), and focal enhancement along the periphery of the right hemi-cord at C4-5, which was not previously seen prior to surgery. Given the enhancement, the patient underwent further diagnostic evaluation. MRI brain with and without contrast was normal. Her white blood cell count was normal (3.8 k/μL), lumbar puncture was bland (1 TNC 0 RBC, normal protein and glucose, negative gram stain and culture, and negative cytology). MRA of C spine showed no evidence of early draining vein to suggest arteriovenous fistula. EMG revealed mild, chronic right C5-7 radiculopathies and mild, chronic left C5-6 radiculopathies. There was no evidence for a median neuropathy. Repeat imaging at thirteen and 19th months postsurgery demonstrated a progressive increase in T2 and FLAIR hyperintensity signal within the cervical cord and persistent but slow decrease in enhancement (Fig. 1D, E). Her examination continued to improve with minimal weakness in the hand, and the primary complaint of difficulty with fine coordination tasks. Sensory changes persisted.

Discussion

WCS is a very rare condition that occurs following decompression of a chronically compressed spinal cord. First described by Chin et al.[1], it is a diagnosis of exclusion and occurs in the absence of any iatrogenic cord injury or perioperative complications. Clinically, there is sudden neurological deterioration either during or immediately after surgery, although deterioration has been reported 1 month postsurgery [2–5]. Radiographically, WCS is uniformly associated with new hyperintense changes on T2-weighted imaging post surgically [1–3]. On intraoperative monitoring, loss of neurophysiological signaling has been reported [3]. Presumptive reperfusion and/or ischemic injury with associated edema of the spinal cord is thought to be the underlying pathological mechanism [1–3].

The diagnosis of WCS derives from exclusion of other causes. Iatrogenic injury from intraoperative trauma should be apparent at time of the procedure. Cord compression from a postsurgical hematoma is seen on imaging. Demyelinating, infectious, and malignant etiologies would be apparent on imaging and evaluation of the cerebral spinal fluid. A vascular malformation can be evaluated by vessel imaging of the cord. Finally, ischemia by hypoperfusion may be examined with diffusion-weighted imaging sequences. In this patient, no iatrogenic injury was experienced. Imaging of the cervical spine vessels did not reveal any vascular malformation. While diffusion-weighted imaging was not performed post surgically, vascular imaging showed no large vessel occlusion. Finally, the persistence of contrast in the cervical cord over many months to a year would be highly unusual for spinal cord ischemia or even traumatic injury at the time of the surgery.

Several radiographic features have been reported in WCS. Uniformly all cases are associated with T2-weighted MR changes prior to surgery and new hyperintensity on T2-weighted MR imaging in the region of the previously effected spinal cord after surgery [1–5]. An increase in the extension of the hyperintensity on T2-weighted imaging in the area of the previously compressed cord has been reported [6]. On T1-weighted MR imaging, the lesion shows a hypointense signal. Contrast enhancement following the administration of gadolinium has not been seen in WCS, albeit imaging with contrast performed months after surgery has not been re-

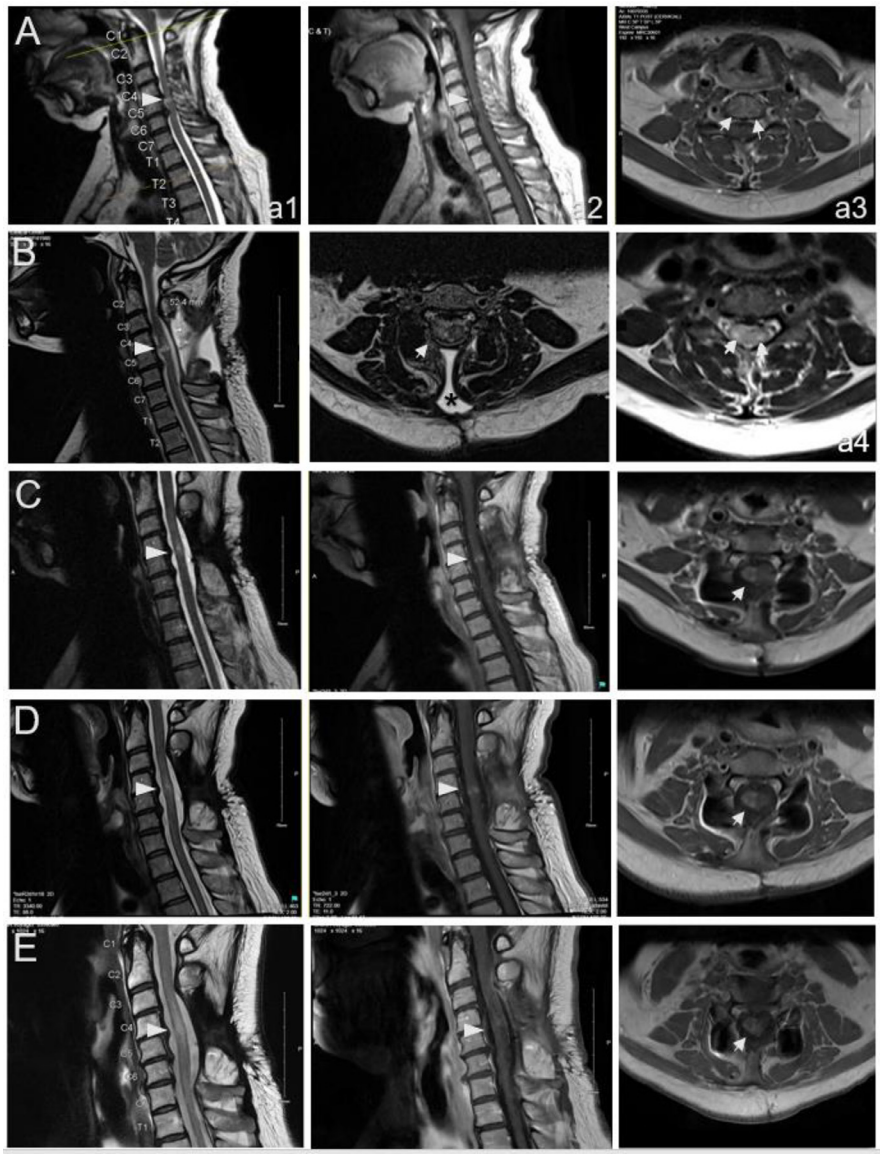

Fig. 1 – Progression of T2-weighted changes and persistence of enhancement on cervical MRI of the spine. (A) Spinal MRI prior to surgery showed intervertebral disc bulge with flattening of the spinal cord at multiple levels of the cervical spine, worst at C4-C5 and C5-C6. T2-weighted, sagittal image shows hyperintensity in the cord at this level (arrowhead in a1), with no apparent enhancement following contrast administration on T1-weighted sagittal (arrowhead in a2) and axial (arrows in a3). Corresponding T2-weighted axial image shows right greater than left hyperintensities (arrows in a4). (B) Two months later, spinal MRI showed postsurgical changes related to posterior decompression and instrumented fusion at C3-C6 and re-demonstration of right greater than left T2-weighted hyperintensity in the spinal cord at C4-C5 level on sagittal (arrowhead) and axial (arrow) images. The hyperintensity at this level was not significantly changed in the right aspect of the spinal cord (arrow), however it appeared improved in the left aspect of the spinal cord compared to the preoperative MRI. Postsurgical changes with seroma formation (asterisk) were also appreciated. No contrast scan were obtained at this time. (C) Eleven months later, sagittal T2-weighted spinal MRI showed hyperintensity of the cord spanning C3 through C5 (arrow). To the right, sagittal (arrowhead) and axial (arrow) postcontrast T1-weighted images showed focal enhancement along the periphery of the right hemicord at C4-C5, which was new since the previous contrasted pre-surgical examination and was thought to reflect enhancing scar tissue and/or revascularization. (D) Thirteen months later, sagittal T2-weighted spinal MRI showed similar intramedullary cord signal abnormality spanning C3-C6 but most prominent at the C4-C5 intervertebral disc level (arrow), likely reflecting progression of myelomalacia. Heterogeneous postcontrast enhancement centered at C4-C5 (arrow) was still seen on sagittal (arrowhead) and axial (arrow) postcontrast T1-weighted images. (E) Nineteen months later, sagittal T2-weighted spinal MRI showed continued progression and increase in the spinal cord signal hyperintensity (arrow). To the right, the postcontrast enhancement on T1-weight sagittal (arrowhead) and axial (axial) images at the C4-5 level persisted despite no spinal cord encroachment, but was improved from prior.

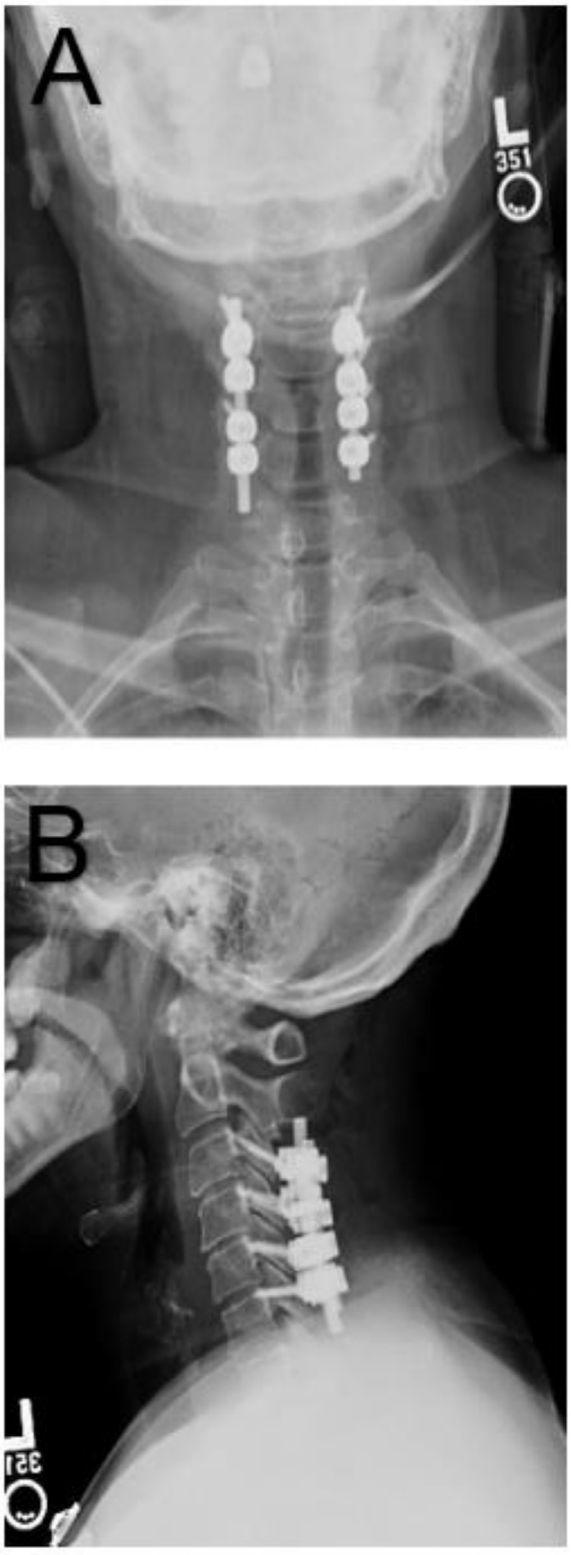

Fig. 2 – Cervical spine radiographs, AP and lateral views. Images show intact posterior fusion hardware associated with recent laminectomies and posterior fusions from C3 through C6 with bilateral pedicle screws and vertical struts. No prevertebral soft tissue swelling. No spondylolisthesis. Mild background narrowing of C4-C5 is unchanged with small anterior osteophytes along several levels.

ported in such patients [1–6]. It is important to also note that T2 signal changes have been reported in 6.1% of cervical spine cases following laminoplasty (from a cohort of 114 subjects) and these radiographic changes are not necessarily associated with any focal deficits on examination [7]. In our case, increased T2 signal is seen postoperatively and actually continued to progress on serial imaging over an extended time period of some 19 months. Imaging in our case was not performed immediately after surgery, so it is unclear whether contrast enhancement was present or absent. However, focal enhancement was seen in this patient and more surprisingly, was the persistence of such a signal over an extended period of time postprocedure.

Contrast enhancement seen in WCS bears some resemblance the radiographic changes seen in cervical spondylotic myelopathy (CSM) [8,9]. In CSM, a characteristic transverse band is seen at a level just below the maximum stenosis. T2-hyperintensity is generally greatest at the area of maximum stenosis with spindle like extension vertically up and down the spinal cord. Moreover, circumferential or peripheral enhancement is seen on axial imaging, sparing the gray matter and no pial enhancement is observed. Postcontrast enhancement can be seen for many months in CSM. Each of these same characteristics is seen in WCS, where there is an apparent transverse band of enhancement, which spares the spinal cord gray matter, just below the area of maximum stenosis. In this patient, the enhancement persists for upwards of 19 months. However, no enhancement is seen prior to the decompression, and the development of spindle-like T2-hyperintensity and the circumferential postcontrast enhancement, which spares the gray matter becomes apparent months after the surgery.

The shared radiographic features of CSM and WCS may suggest a common pathophysiology. Contrast enhancement is typically seen on T1-weighted MR imaging following gadolinium administration and indicates a compromise in the blood-spinal barrier within the re-perfused area, high vascularity or leakage into the lymphatic system [10,11]. Prolonged and persistent enhancement in the spinal cord due to CSM can be seen at the level of ongoing or worsening compression [8,9] and is thought to relate to focal breakdown of the blood-spinal cord barrier, formation of new vasculature secondary to injury, and consequent increased vascular permeability from venous hypertension [12,13]. Subarachnoid scarring would also alter cerebral spinal fluid dynamics and contribute to edema [14]. In WCS, prior case reports have raised the possibility that the syndrome develops from an acute reperfusion injury on a chronically compressed cord [1–4]. The chronically compressed cord becomes susceptible to ischemia, either from vascular occlusion or edema from an increase in vascular permeability and venous congestion following decompression [1–4]. A consequence of this vascular insufficiency would potentially be breakdown of the brain-spinal cord barrier and the delayed radiographic changes seen in this patient. Moreover, the prolonged contrast enhancement is consistent with the prolonged duration of recovery in patients with WCS, typically months [1–6]. Presumably, as the integrity of vasculature and the blood vessel-spinal cord barrier recovers, the contrast en-

hancement diminishes. Such radiographic changes appear to coincide with gradual improvement clinically.

Conclusion

WCS is reportedly an extremely rare entity, brought on by decompression of a chronically compressed cord. Radiographically, T2-weighted hyperintensity in the cervical cord is seen prior to surgery, suggestive of myelomalacia. Following decompression, extension of T2-weighted hyperintensity changes occurs within the spinal cord. Clinically, patients have abrupt neurological deterioration. This case report is the first to demonstrate the observation of prolonged post contrast T1-weighted enhancement in WCS and its persistence but gradual resolution over 18 months. The patient clinically improves slowly over these same many months. Such findings suggest both an acute and more chronic phase of recovery for this disorder.

Author contributions

Dr. Sheen, V: Conceptualization, data curation, formal analysis, investigation, methodology, visualization, original draft, writing, editing. Ms. Sheen, S Formal analysis, investigation, methodology, visualization, original draft, writing, editing.

Availability of data and material

Not applicable.

Declaration

The manuscript has not been published previously, is not under consideration for publication elsewhere, is approved by all authors and if accepted, will not be published elsewhere in the same form, in English or in any other language, including electronically without the written consent of the copyright-holder.

Patient consent

Written informed consent has been obtained for publication. The images are anonymized to protect patient identity and privacy.

REFERENCES

[1] Chin K, Seale J, Cumming V. “White cord syndrome” of acute tetraplegia after an anterior cervical decompression and fusion for chronic spinal cord compression: a case report. Case Rep Orthop 2013. doi:10.1155/2013/697918.2013.

[2] Giammalva G, Maugeri R, Graziano F, Guli C, Giugno A, Basile L. White cord syndrome after non-contiguous double-level anterior cervical decompression and fusion (ACDF): a “no reflow phenomenon. Interdisc Neurosurg Adv Tech Case Managt 2017;7:47–9.

[3] Antwi P, Grant R, Kuzmik G, Abbed K. “White cord syndrome” of acute hemiparesis after posterior cervical decompression and fusion for chronic cervical stenosis. World Neurosurg 2018;113:33–6. doi:10.1016/j.wneu.2018.02.026.

[4] Busack C, Eagleton B. White cord syndrome causing transient tetraplegia after posterior decompression and fusion. Ohsner J 2020;20(3):334–8. doi:10.31486/toj.19.0081.

[5] Singh R, Arts M, de Ruiter G. Delayed-onset white cord syndrome after anterior and posterior cervical decompression surgery for symptomatic ossification of spinal ligaments: illustrative cases. J Neurosurg Case Lessons 2021;1(19):CASE2113. doi:10.3171/CASE2113.

[6] Sepulveda F, Carballo L, Carnevale M, Yañez P. White cord syndrome in a pediatric patient: a case report and review. Radiol Case Rep 2020;15(11):2343–7. doi:10.1016/j.radcr.2020.08.047.

[7] Seichi A, Takeshita K, Kawaguchi H, Nakajima S, Akune T, Nakamura K. Postoperative expansion of intramedullary high-intensity areas on T2-weighted magnetic resonance imaging after cervical laminoplasty. Spine (Phila Pa 1976) 2004;29(13):1478–82 discussion 1482. doi:10.1097/01.brs.0000128757.32816.19.

[8] Flanagan EP, Krecke KN, Marsh RW, Giannini C, Keegan BM, Weinshenker BG. Specific pattern of gadolinium enhancement in spondylotic myelopathy. Ann Neurol 2014;76:54–65.

[9] Mustafa R, Passe T, Lopez-Chiriboga A, Weinshenker B, et al. Utility of MRI enhancement pattern in myelopathies with longitudinally extensive T2 lesions. Neurol Clin Pract 2021;11(5):e601–11. doi:10.1212/CPJ.0000000000001036.

[10] Sage M, Wilson A, Scroop R. Contrast media and the brain the basis of CT and MR imaging enhancement. Neuroimaging Clin N Am 1998;8:695–707.

[11] Smirniotopoulos J, Murphy F, Rushing E. Patterns of contrast enhancement in the brain and meninges. Radiographics 2007;27:525–51. doi:10.1148/rg.272065155.

[12] Ozawa H, Sato T, Hyodo H, et al. Clinical significance of intramedullary Gd-DTPA enhancement in cervical myelopathy. Spinal Cord 2010;48:415–22.

[13] Lee J, Koyanagi I, Hida K, et al. Spinal cord edema: unusual magnetic resonance imaging findings in cervical spondylosis. J Neurosurg 2003;99(1 suppl):8–13.

[14] Sasamori T, Hida K, Yano S, et al. Spinal cord swelling with abnormal gadolinium-enhancement mimicking intramedullary tumors in cervical spondylosis patients: three case reports and review of the literature. Asian J Neurosurg 2010;5:1–9.