

CASE REPORT

White cord syndrome following cervical spine surgery: A case report and systematic literature review

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Abstract

White cord syndrome (WCS) is a rare but severe post-operative complication following cervical spine decompression surgery, characterized by acute neurological deterioration and T2-weighted hyperintensity on magnetic resonance imaging (MRI), suggestive of ischemia-reperfusion injury. This study presents a case of WCS following anterior cervical discectomy and fusion (ACDF) and conducts a systematic review to analyze risk factors, management strategies, and outcomes associated with WCS. A systematic literature review was conducted according to preferred reporting items for systematic reviews and meta-analyses guidelines. PubMed, Embase, Scopus, and Cochrane Library were searched for studies reporting WCS following cervical decompression surgery. Inclusion criteria encompassed case reports and case series with documented post-operative neurological deficits and MRI-confirmed T2 hyperintensity. Data extraction included demographics, clinical presentation, treatment strategies, and neurological outcomes. In addition, we describe a case report of WCS following ACDF at C6–C7 with complete neurological recovery after corticosteroid therapy and intensive rehabilitation. A total of 30 articles, including 37 patients, were analyzed. ACDF and posterior cervical decompression and fusion were the most common procedures associated with WCS. Postoperatively, all patients developed acute neurological decline, with 25% achieving full recovery, 44% showing partial improvement, and 31% remaining permanently disabled. Steroid administration and early rehabilitation correlated with improved outcomes, whereas delayed intervention and pre-existing myelomalacia were associated with worse prognoses. WCS is an underrecognized but potentially devastating complication of cervical decompression surgery. Early recognition, aggressive treatment with corticosteroids, blood pressure optimization, and intensive rehabilitation may improve functional recovery. Standardized treatment protocols and multicenter prospective studies are needed to define preventive and therapeutic strategies for this condition.

Keywords: White cord syndrome; Cervical spinal cord injury; Ischemia–reperfusion injury

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1. Background

White cord syndrome (WCS) is characterized by the observation of intramedullary hyperintensity on post-operative T2-weighted magnetic resonance imaging (MRI) sequences in the setting of unexplained neurological deficits after cervical spinal cord decompression.¹ It is an extremely rare complication, following anterior cervical discectomy and fusion (ACDF) or posterior cervical decompression and fusion (PCDF), described for the 1st time in 2013.² Very few cases have been reported in the literature, and the mechanism of acute weakness is not fully understood. The proposed pathogenesis of WCS involves an ischemia-reperfusion injury mechanism, characterized by oxidative stress, endothelial dysfunction, and neuroinflammatory cascades.¹ These mechanisms are well-recognized in other ischemic conditions, such as myocardial infarction and traumatic brain injury, and may similarly underlie the neurological decline observed in WCS.² This acute para- or tetraplegia is associated with a pathognomonic radiographic finding, hence the name that is the presence of hyperintensity on T2-weighted MRI. The cause of the neurological deficit, in the absence of perceptible technical cause, seems related to reperfusion damage of the spinal cord already suffering from a chronic compression.³ It remains a diagnosis of exclusion. Hence, we report a case of WCS following ACDF for C6–C7 cervical disc herniation, where the patient experienced a sudden neurological post-operative tetraplegia. Moreover, a systematic review of the literature was performed to identify and analyze all the potential factors that could determine a reperfusion or ischemic damage of the spinal cord.

2. Case presentation

A 67-year-old patient suffering from cervicobrachialgia lasting for several months, resistant to drug therapy or conservative treatments, presented with a cervical herniated intervertebral disc identified at C6–C7 level on C-spine MRI. The patient did not report pre-operative neurological deficit or claudication. ACDF C6–C7 surgery was performed in November 2021. No complications were reported during the procedure. Immediately after the operation, physical examination revealed acute tetraplegia, and an emergency MRI was performed. On MR images, there were no signs of dural lesions or compression, but there was a T2 high signal myelopathy suspected as reperfusion injury at C6–C7 level (Figure 1). No emergency surgery was performed, but a pharmacological therapy was started immediately according to the NASCIS protocol (National Acute Spinal Cord Injury Study), which led, after an intensive rehabilitation program, to a total recovery of motor and sensory functions in about 30 days.

During pre-operative evaluation, the patient manifested 5/5 motor strength in bilateral upper extremities, no weakness or paresthesia in bilateral lower extremities necessitating crutches, and intact sensory functions in all four extremities. He had a negative Hoffmann's sign in his bilateral upper extremities. A left radial arterial line was placed, and mean arterial pressure (MAP) was maintained >80 mmHg throughout the case with a norepinephrine drip. Intubation was performed with cervical spine precautions, with his neck maintained in the neutral position during intubation using a GlideScope (Verathon Inc., Bothell, WA, USA). Neuromonitoring was not utilized due to the surgeon's preference. The patient underwent C6–7 ACDF with a tantalum cages. Hemostasis was achieved before closure. There were no surgical or anesthetic complications noted intraoperatively.

After 6 months, the patient had fully recovered the motor and sensory functions. He could walk autonomously and had an NDI-Oswestry (neck disability index - Oswestry disability index) of 8%, and an mJOA (modified Japanese Orthopaedic Association scale) of 18. After 12 months of follow-up, the patient did not present any neurological symptoms, and the NDI-Oswestry score was at 4% while the mJOA maintained at 18.

3. Literature review

3.1. Literature review methods

The aim of this systematic review was to identify, analyze, and synthesize all published cases of WCS to better define risk factors, management strategies, and outcomes. In the present study, a systematic literature review according to the preferred reporting items for systematic reviews and meta-analyses guidelines was performed (Figure 2).⁴ MEDLINE through PubMed and Embase, Scopus, Cochrane Library database were searched using the keywords: "White cord syndrome," "Reperfusion injury" AND "Spinal cord" AND "Surgery" and their MeSH terms in any possible combinations using the logical operators "AND" and "OR." The reference lists of relevant studies were screened to identify other studies of interest. The literature search was conducted iteratively and continued until December 31, 2024.

In this review, the full-text articles describing WCS after ACDF or PCDF were considered eligible. Only articles written in English were included. No date limits for publication were set. Expert opinions, studies on animals, unpublished reports, *in vitro* investigations, case reports, letters to the editor, abstracts from scientific meetings, and book chapters were excluded from review.

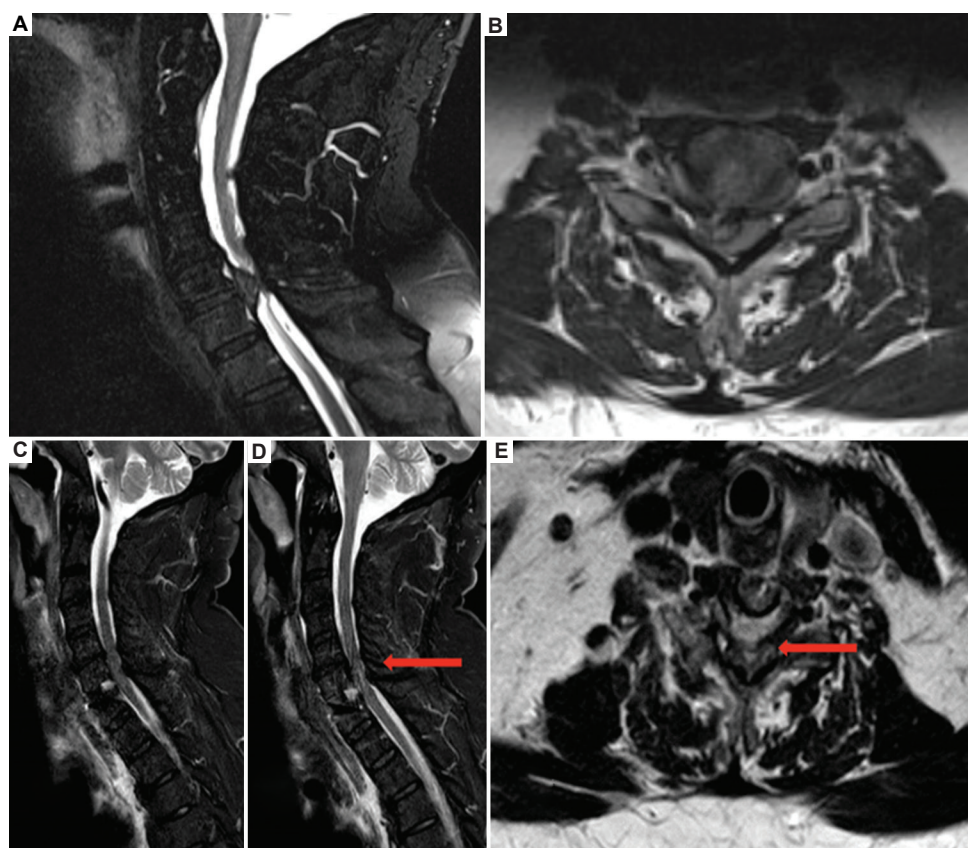


Figure 1. Pre-operative magnetic resonance imaging (MRI) scans showing sagittal (A) and axial views (B), demonstrating a herniated disc with significant spinal cord compression. Postoperatively, urgent MRI scans in sagittal (C, D) and axial views (E) reveal the characteristic T2 hyperintensity of white cord syndrome, indicating spinal cord edema without residual mechanical compression (red arrow).

Two independent authors (C.V. and M.I.B.) searched and collected data from the included studies. Any discordances were solved by consensus with a third author (A.P.). The following data were extracted: Demographic features, diagnosis, comorbidities, possible complications, clinical outcomes and follow-up, pre-operative Nurick grade. Risk of bias assessments and quality assessments of included studies was checked using the Cochrane risk of bias tool (Figure 1).⁵ Numbers software (Apple Inc., Cupertino, CA) was used to tabulate the obtained data. Categorical variables are presented as frequency and percentages. Continuous variables are presented as means and standard deviations. Only one decimal digit was reported and was rounded up.

3.2. Literature review results

The electronic literature research yielded 139 articles, which were screened by title and abstract after duplicate removal. Only 30 articles were included for full-text revision and were considered for eligibility. Full-text papers of these articles were downloaded and carefully reviewed, while clinical, surgical, and radiological data were collected as

described below. The studies included in the review are level of evidence IV.

3.2.1. Patient characteristics and assessment

WCS has been reported in various cases following cervical decompression surgery. The mean age of affected patients in this review is 57.1 years, with cases spanning a broad age range, including a rare pediatric case.

One of the earliest reported cases comes from Chin *et al.*,² who described a 59-year-old male undergoing ACDF. Initially, he had no significant motor deficits, but immediately postoperatively, he developed incomplete tetraplegia. MRI revealed T2 hyperintensity at C6, characteristic of WCS. Despite early corticosteroid administration, recovery was only partial, with persistent neurological impairment (Nurick 4) at 2 months follow-up. Similarly, Giammalva *et al.*⁶ reported a 64-year-old male who underwent ACDF at C3–C4 and C5–C6. Immediately postoperatively, he developed sudden tetraparesis, which rapidly progressed to complete paraplegia. MRI confirmed non-compressive spinal cord edema. No further surgery was performed, and despite steroid therapy, the patient

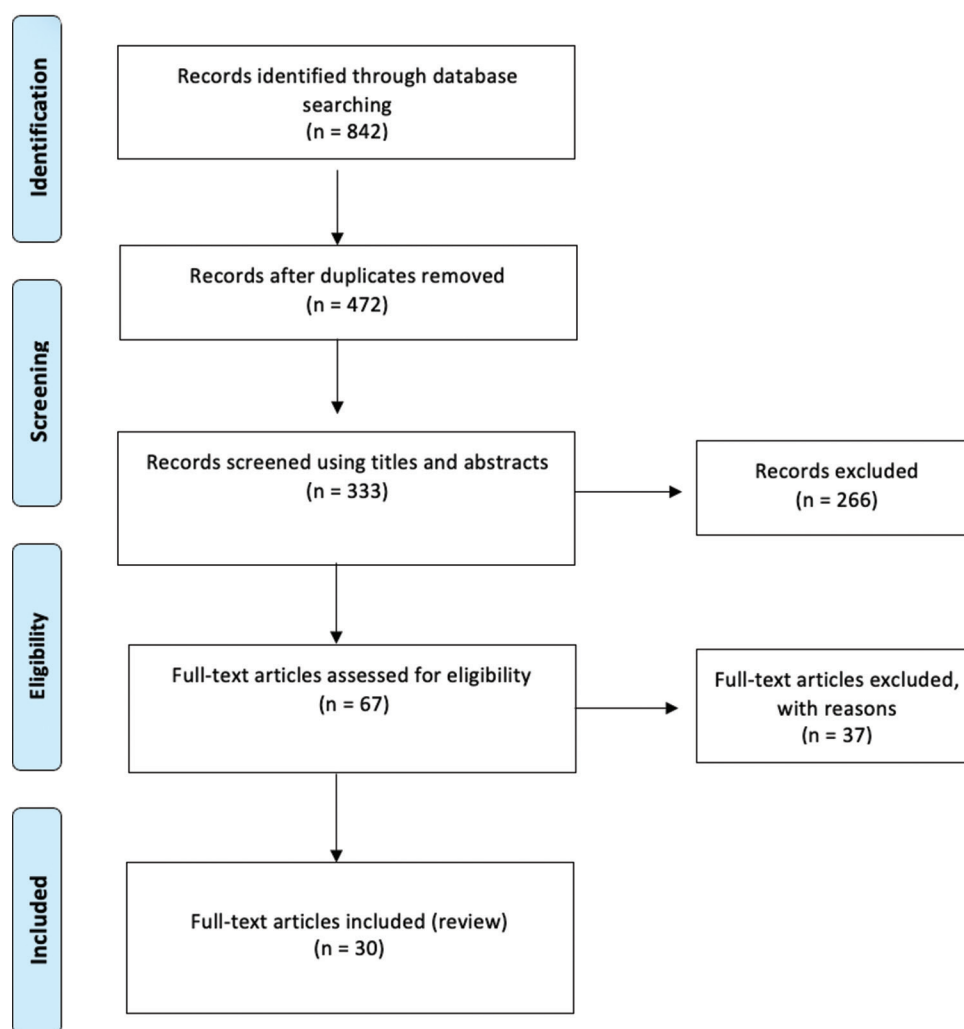


Figure 2. Preferred reporting items for systematic reviews and meta-analyses flowchart

remained severely disabled at follow-up (Nurick 4). A particularly severe case was reported by Vinodh *et al.*⁷ in a 51-year-old female who underwent PCDF from C2 to C5. She became tetraplegic at the C3 level immediately after surgery. High-dose steroids (NASCIS III protocol) were administered over 48 h, but she never regained significant neurological function and required long-term ventilatory support. Antwi *et al.*¹ described a 68-year-old male who underwent PCDF from C4 to C7. During surgery, there was a sudden loss of motor evoked potentials (MEPs) in the left hand and leg, prompting an urgent intraoperative MRI. Suspecting a hematoma, the team performed an emergency revision surgery, preventing further neurological decline. This patient showed significant improvement following rehabilitation, achieving near-complete functional recovery (Nurick 4). Papaioannou *et al.*⁸ described a 79-year-old male who underwent PCDF from C3 to C6 and developed post-operative paraparesis. Unlike previous

cases, his neurological decline was delayed, occurring several hours postoperatively. MRI confirmed cord edema without compression. A second decompression procedure was performed, but neurological deficits persisted at 18 months follow-up (Nurick 4). A catastrophic case was reported by Kalidindi and Sath⁹ in a 63-year-old male who underwent PCDF from C2 to C7 *en bloc*. He developed complete tetraplegia immediately postoperatively, which was non-responsive to steroids. His neurological function remained unchanged (Nurick 6), marking one of the worst outcomes reported in WCS. A series of cases was presented by Fathalla *et al.*,¹⁰ who documented six cases of WCS following PCDF at various levels (C3–C7). All six patients developed acute post-operative tetraplegia, with MRI revealing spinal cord edema without residual compression. None underwent additional surgery, and treatment was limited to corticosteroids. Outcomes varied: Two patients showed moderate recovery (Nurick 3–4);

three patients remained severely impaired (Nurick 4–5); one patient had no neurological recovery (Nurick 6). Busack and Eagleton¹¹ described a 63-year-old male with cervical stenosis who underwent PCDF at C3–C6. He developed acute tetraparesis with paraplegia but showed partial improvement with steroids (Nurick 4 at follow-up). A notable pediatric case was reported by Sepulveda *et al.*¹² in a 12-year-old male who underwent PCDF from C2 to C4. He developed dysautonomia and motor deficits. High-dose corticosteroid therapy resulted in significant neurological recovery (Nurick 6). A different presentation was observed by Jun *et al.*¹³ in a 49-year-old female who underwent ACDF at C6–C7. She developed paraplegia rather than tetraplegia and was treated with laminoplasty (C4–C7) and steroids, achieving complete neurological recovery (Nurick 0). Mathkour *et al.*¹⁴ described a 79-year-old male who developed right-sided hemiparesis after PCDF at C3–C5. He received dexamethasone (4 mg every 6 h) and showed partial recovery at 4 months. Malinovic *et al.*¹⁵ reported a 46-year-old male who underwent PCDF from C2 to T2 and developed acute tetraplegia postoperatively. He was treated with dexamethasone and hypertonic saline boluses but failed to regain any motor or sensory function, marking one of the worst recorded outcomes in WCS. Goyal *et al.*¹⁶ presented a 50-year-old male who underwent ACDF for myelopathy and developed post-operative tetraparesis. Despite early steroid administration, his neurological function did not fully recover. Wiginton *et al.*³ reported a 41-year-old male who underwent C1 bony decompression for cervical spondylotic myelopathy. Intraoperatively, he experienced a complete loss of motor and somatosensory evoked potentials (MEPs and SSEPs), which returned before surgery completion. Postoperatively, he developed quadriplegia, with MRI showing hyperintensity in the central cervical cord, characteristic of WCS. Treatment with corticosteroids and MAP optimization led to full functional recovery. Liao *et al.*¹⁷ described a 58-year-old male who underwent ACDF at C3–C5 and developed delayed-onset tetraparesis. MRI revealed extensive spinal cord edema without residual compression. Despite high-dose steroids and supportive therapy, the patient remained with persistent neurological deficits (Nurick 4). Acharya *et al.*¹⁸ presented a 63-year-old male who developed acute tetraplegia following PCDF at C4–C7. The patient showed only mild improvement despite aggressive steroid therapy and rehabilitation. Segal *et al.*¹⁹ documented a 55-year-old male who underwent posterior decompression and experienced severe neurological deterioration postoperatively. MRI showed diffuse hyperintensity in the spinal cord. Despite maximal medical therapy, his neurological function remained significantly impaired. Singh *et al.*²⁰ described two cases of WCS in patients

undergoing posterior decompression. Both developed sudden-onset motor deficits postoperatively, and while one patient demonstrated partial recovery, the other remained severely impaired despite steroid treatment. Carter *et al.*²¹ reported a 67-year-old female who developed paraplegia post-ACDF. Corticosteroid therapy and rehabilitation led to moderate improvement over 6 months. Lei *et al.*²² documented a case of a 50-year-old male with post-operative tetraparesis after C4–C7 ACDF. Despite steroid therapy, his functional recovery remained limited. Dahpute *et al.*²³ reported a 48-year-old male who underwent posterior decompression at C2–T1 and developed acute WCS, showing only partial neurological improvement. So *et al.*²⁴ described a 53-year-old female who became paraplegic following ACDF at C5–C7, with MRI showing ischemia-reperfusion injury. Despite early intervention, only minimal recovery was observed. Algahtani *et al.*²⁵ presented a 61-year-old male who underwent ACDF and developed post-operative quadriplegia. Neurological deficits persisted despite steroid administration. Tanaka *et al.*²⁶ reported a 72-year-old male who became paraplegic following decompression surgery at C3–C6. Aggressive therapy resulted in partial recovery. Ranjan *et al.*²⁷ described a case of a 59-year-old male who developed WCS post-ACDF, with persistent neurological deficits at 1-year follow-up. Yen Hsin *et al.*²⁸ presented a 47-year-old female who developed delayed paraplegia postoperatively, showing limited improvement with conservative treatment. Chatzikomninos *et al.*²⁹ described a 60-year-old male with acute tetraplegia following cervical decompression, with MRI findings consistent with WCS. Despite therapy, deficits persisted. Jain *et al.*³⁰ documented a 55-year-old male who developed post-operative WCS and showed moderate recovery after corticosteroid treatment. Zach *et al.*³¹ presented a 51-year-old male with complete paraplegia following decompression at C3–C7. His condition remained unchanged despite treatment. All data are summarized in [Table 1](#).

3.2.2. Surgical technique

Among the 16 cases, ACDF was performed in three patients (19%), while the majority (13 patients, 81%) underwent PCDF. The most commonly decompressed levels were C3–C7, with some cases involving multilevel decompression extending to C2 or T2. Intraoperative neuromonitoring was not routinely utilized, and in some cases, a sudden loss of MEPs was noted intraoperatively, prompting early post-operative imaging. A few patients underwent extended decompression or hematoma evacuation in revision surgeries, but most were managed conservatively with high-dose corticosteroids (NASCIS protocol or dexamethasone regimens).

Table 1. Demographic and epidemiological data of patients included in this systematic review

Author	Year	Age (years)	Gender	Comorbidities	Pathology	Surgical treatment	Level decompressions	Imaging MRI/CT
Chin <i>et al.</i> ²	2013	59	M	-	-	ACDF (PEEK)	C4–C5, C5–C6	MRI, CT
Giammalva <i>et al.</i> ⁶	2017	64	M	-	-	ACDF	C3–C4, C5–C6	
Vinodh <i>et al.</i> ⁷	2018	51	F	-	Extramedullary metastatic ductal carcinoma with extradural and intradural lesions	PCDF	C2–C5	MRI, CT
Antwi <i>et al.</i> ¹	2018	68	M	-	Cervical stenosis, spondylolisthesis	PCDF	C4–C7	MRI, CT
Papaioannou <i>et al.</i> ⁸	2019	79	M	Hypertension, fibrillation	C4–C6 stenosis	PCDF	C3–C6	MRI after 2 nd surgery
Kalidindi and Sath ⁹	2020	63	M	-	Cervical stenosis, OPLL,	PCDF	C2–C7 <i>en bloc</i>	MRI, CT
Busack and Eagleton ¹¹	2020	63	M	-	Cervical stenosis	PCDF	C3–C6	
Sepulveda <i>et al.</i> ¹²	2020	12	M	Encephalopathy	Arachnoid cyst cervical canal from C2 to C4	PCDF	C2–C4	MRI (worse)
Jun <i>et al.</i> ¹³	2020	49	F	-		ACDF	C6–C7	MRI
Mathkour <i>et al.</i> ¹⁴	2020	79	M	Hypertension	C3–C6 spondylosis with canal stenosis	PCDF	C3–C5 laminectomy with C2–C6 instrumented fusion	MRI
Wiginton <i>et al.</i> ³	2020	41	M	-	C1 stenosis	ACDF	C1 laminectomy	MRI
Liao <i>et al.</i> ¹⁷	2020	51	M	-	C2–C4 OPLL	PCDF	C3–C4 posterior laminectomy decompression and C2–C5 fusion	MRI, CT
Fathalla <i>et al.</i> ¹⁰	2020	-	5 M, 2 F	-	-	4 cases underwent cervical laminectomy only and 3 cases underwent laminectomy and fusion	3 (C3–C7), 3 (C3–C6), 1 (C3–C5),	MRI, CT
		62	M	Hypertension, diabetes	-	C3–C7 laminectomy and fusion		MRI
		65	M	Hypertension	-	C3–C6 laminectomy and fusion		MRI
		70	M	Hypertension, diabetes	-	C3–C7 laminectomy and fusion		CT
		61	M	Hypertension, diabetes	-	C3–C5 laminectomy and fusion		MRI
		63	M	Hypertension	-	C3–C6 laminectomy and fusion		MRI

(Cont'd...)

Table 1. (Continued)

Author	Year	Age (years)	Gender	Comorbidities	Pathology	Surgical treatment	Level decompressions	Imaging MRI/CT
		69	F	Hypertension, diabetes	-	C3–C6 laminectomy		MRI
		65	F	Hypertension	-	C3–C7 laminectomy		MRI
Acharya <i>et al.</i> ¹⁸	2021	49	M	-	C2–C3 to C7–T1 OPLL	PCDF	C3–C7	MRI
Segal <i>et al.</i> ¹⁹	2021	55	M	-	-	PCDF		MRI
Malinovic <i>et al.</i> ¹⁵	2021	46	M	Achondroplasia, club foot, obstructive sleep apnea, scoliosis	-	PCDF	C2–T2	MRI
Singh <i>et al.</i> ²⁰	2021	59	F	Hypertension, ype 2 diabetes mellitus, parathyroidectomy	C3–C6 stenosis, C5–C6 OLF	Posterior decompressive laminectomy	C3–C6	MRI, CT
		66	F	Hypertension	C3–C5 stenosis, OPLL	C4 corpectomy with placement of an expandable cage, ACDF C6–C7	C3–C7	MRI
Carter <i>et al.</i> ²¹	2021	3	F	Mediastinal neuroblastoma partially resected 2 years prior	T4–T8 compression from a mass of ganglioneuromatous tissue	T5–T7 laminoplasty	T5–T7	MRI, CT
Lei <i>et al.</i> ²²	2022	54	M	-	Degeneration and herniation of the C4–C7 discs	ACCF	C4–C7	MRI
Dahpute <i>et al.</i> ²³	2022	63	M	-	OPLL from C2 to C4, C6 body fracture, spinal canal stenosis C2–C4	PCDF, decompression from C2 to C7, instrumented fusion from C2 to T2	C2–C7	MRI, CT
Goyal <i>et al.</i> ¹⁶	2022	39	F	Type 2 diabetes mellitus	Prolapsed inter-vertebral disc at C5–C6 and loss of cervical lordosis	ACDF	C5–C6	MRI
So <i>et al.</i> ²⁴	2022	61	M	None	C4–C5 stenosis due to bone spury, OPLL	ACDF	C4–C7	MRI
Algahtani <i>et al.</i> ²⁵	2022	66	M	Hypertension, ischemic heart disease	C5/6 stenosis due to bulge osteophyte complex	ACDF	C5–C6	MRI
Tanaka <i>et al.</i> ²⁶	2023	81	M	-	Spinal canal stenosis due to an OPLL of C3–C5	C3–C6 double-door laminoplasty, C5 laminectomy	C3–C6	MRI

(Cont'd...)

Table 1. (Continued)

Author	Year	Age (years)	Gender	Comorbidities	Pathology	Surgical treatment	Level decompressions	Imaging MRI/CT
Ranjan <i>et al.</i> ²⁷	2023	64	F	-	C5–C6 tubercular lesion with extradural compression	ACCF, titanium mesh filled with autogenous bone graft harvested from the iliac bone placed between C4 and C7	C5–C6	MRI, CT
Yen Hsin <i>et al.</i> ²⁸	2023	61	M	Previous decompressive surgery 2 years prior for C6–C7 neural foramne stenosis	Severely narrowed left C6–C7 neural exit canal	ACDF	C6–C7	MRI
Chatzikomninos <i>et al.</i> ²⁹	2024	33	M	Klippel–Feil syndrome	Kyphotic malformation of the cervical spine	1 st stage ACDF C4–C6, C5 corpectomy; 2 nd stage laminectomy of C5–C7 and posterior fusion of C5–T1; 3 rd stage occipitocervical fusion	C4–T1	MRI
Jain <i>et al.</i> ³⁰	2024	63	M	Hypertension, type 2 diabetes mellitus	C2–T1 OPLL and further HLF from dorsal 1 st to 4 th spine	Posterior instrumented decompression	C2–T5	MRI, CT, X-ray
Zach <i>et al.</i> ³¹	2024	49	M	Hyperlipidemia, hypertension, lipoma resection	C3–C5 stenosis, spinal cord compression and myelomalacia	ACDF	C3–C5	MRI
Velluto <i>et al.</i> [This paper]	2021	65	M	Hypertension	-	ACDF	C6–C7	MRI, X-ray
Author	Pre-operative	Neuromonitoring (MEPs, SSEPs)	MEPs, SSEPs	ASIA motor score	Clinical findings	Steroid	Treatment	Outcome
Chin <i>et al.</i> ²	3	1	-	B (post-operative), C (discharge), D (2 months), D (16 months)	C6 incomplete tetraplegia	Postoperative	C5 corpectomy, hydrocortisone 100 mg intraoperatively, intravenous dexamethasone	Improvement al 2 months (Nurick 4, ASIA D)
Giammalva <i>et al.</i> ⁶	3	1	At closure	-	Tetraparesis with complete paraplegia and severe motor weakness to upper limbs with diffuse spastic hypertonia.	Postoperative	Neck collar	Improvement, Nurick 4, JOA 6

(Cont'd...)

Table 1. (Continued)

Author	Pre-operative Nurick	Neuromonitoring (MEPs, SSEPs)	MEPs, SSEPs	ASIA motor score	Clinical findings	Steroid	Treatment	Outcome
Vinodh <i>et al.</i> ⁷	-	0	-	-	Tetraplegic from C3 level	Postoperative	NASCIS III – 2 days of steroids	Weaned off ventilation, neurological status never improved
Antwi <i>et al.</i> ¹	1	1	At closure	-	Loss of MEPs in the left hand and left leg	Intraoperative during revision procedure, post-operative	Exploration hematoma after MEPs loss, Steroid protocol	Improvement, Nurick 4 at discharge
Papaoiannou <i>et al.</i> ⁸	3	1	None	B (post-operative)	Paraparesis	Post-operative	Extended decompression	Persistent weakness (18 months), Nurick 4
Kalidindi and Sath ⁹	5	1	Complete loss after laminectomy (<i>en bloc</i>)		Tetraplegia	Post-operative	NASCIS II	No improvement, Nurick 6
Busack and Eagleton ¹¹	3	1	Loss of MEP and diminished SSEPs after laminectomy		Tetraparesis with complete paraplegia	Pre-operative, intraoperative, post-operative		Improvement, Nurick 4
Sepulveda <i>et al.</i> ¹²	6	0	-	-	Dysautonomia	Post-operative	High-dose steroids	Improvement, Nurick 6
Jun <i>et al.</i> ¹³	1	0			Paraplegia	Post-operative	Laminoplasty C4–C7, high-dose steroid	Full recovery, Nurick 0
Mathkour <i>et al.</i> ¹⁴	4	1	At closure	-	Right hemiparesis	Post-operative	Dexamethasone 4 mg every 6 h	Partial recovery (4 months)
Wiginton <i>et al.</i> ³	1	1	During surgery	-	Acute tetraplegia	Post-operative	C2 decompression	Not mentioned
Liao <i>et al.</i> ¹⁷	2	0	-	-	Left hemiparesis	Post-operative	Further surgery	Recovery to preoperative level; re-exploration at 2 months for continued CSF leakage
Fathalla <i>et al.</i> ¹⁰	-	0	-	-	-	Post-operative	No further surgery	Six months after surgery, all the cases showed partial improvement of their neurological status
	2	0	-	-	-	Post-operative	No further surgery	4
	3	0	-	-	-	Post-operative	No further surgery	4
	3	0	-	-	-	Post-operative	No further surgery	4
	1	0	-	-	-	Post-operative	No further surgery	3
	3	0	-	-	-	Post-operative	No further surgery	4
	2	0	-	-	-	Post-operative	No further surgery	4

(Cont'd...)

Table 1. (Continued)

Author	Pre-operative Nurick	Neuromonitoring (MEPs, SSEPs)	MEPs, SSEPs	ASIA motor score	Clinical findings	Steroid	Treatment	Outcome
	2	0	-	-	-	-	No further surgery	3
Acharya <i>et al.</i> ¹⁸		0	-	-	Acute tetraplegia	Post-operative	No further surgery	Recovered (8 weeks)
Segal <i>et al.</i> ¹⁹	6			-	Acute tetraplegia	Post-operative	No further surgery	
Malinovic <i>et al.</i> ¹⁵	1	0	-	-	Complete tetraplegia	Post-operative	Dexamethasone and hypertonic saline boluses	Failed to regain any further motor or sensory function
Singh <i>et al.</i> ²⁰	3	0	-	C (pre- operative), A (post- operative)	Muscle strength decrease, hypotension and bradycardia, severe tetraparesis	Post-operative	High-dose dexamethasone and hemodynamic support	Grade 4 muscle strength in all limbs 6 months after discharge
	3	0	-	C (pre- operative), B (post- operative)	Tetraparesis	Post-operative	Dexamethasone	Partial recovery, unable to walk unassisted at 3-month follow-up
Carter <i>et al.</i> ²¹	3	1	None	D (pre- operative), C (post- operative)	Paraplegia	Post-operative	No further surgery, high-dose steroids for 7 days	Recovered (4 months)
Lei <i>et al.</i> ²²	1	0	-	D (pre- operative), C (post- operative)	Upper limb strength of 3/5 and leg strength of 3/5 according to the Medical Research Council scale	Post-operative	Methylprednisolone combined with mannitol and neurotrophic drug mecobalamin for 10 days; posterior cervical decompression; hyperbaric oxygen therapy to decrease spinal cord edema and improve reperfusion injury	Neurological function not deteriorated at 7-month follow-up
Dahpute <i>et al.</i> ²³	1	1	After the laminectomy at C3/4, MEP initially augmented, at the end of surgery, MEPs remained absent below trapezius	B (post- operative)	Low-velocity fall at home followed by quadriparesis	Post-operative	No further surgery	Inpatient rehabilitation center 6 weeks after surgery with no improvement in motor strength

(Cont'd...)

Table 1. (Continued)

Author	Pre-operative Nurick	Neuromonitoring (MEPs, SSEPs)	MEPs, SSEPs	ASIA motor score	Clinical findings	Steroid	Treatment	Outcome
Goyal <i>et al.</i> ¹⁶	4	0	-	C (pre-operative), D (post-operative)	Tetraplegia	Postoperative	Dexamethasone, baclofen	Recovered (17 months)
So <i>et al.</i> ²⁴	1	0	-	B (pre-operative), D (post-operative)	Tetraplegia	Post-operative	Total laminectomy of C4–C6, subtotal laminectomy of C7 and lateral mass screw fixation of C4–C6; methylprednisolone	Recovered (2 months)
Algahtani <i>et al.</i> ²⁵	4	0	-	C (pre-operative), D (post-operative)	Acute tetraplegia	Post-operative	Dexamethasone	Recovered (3 days); a new severe spinal canal stenosis at level C4/5 at 6 months, posterior cervical decompressive laminectomy at C3–C6
Tanaka <i>et al.</i> ²⁶	2	1	During surgery	-	Quadriplegia	-	No further surgery	Recovered after rehabilitation in hospital and discharged to home 6 months later
Ranjan <i>et al.</i> ²⁷	1	0	-	C (pre-operative), A (post-operative), C (7 th day postoperatively), D (2 nd month follow-up), E (1 year follow-up)	Tetraplegia	Post-operative	High doses of methylprednisolone and rehabilitation therapies	Recovered
Yen Hsin <i>et al.</i> ²⁸	1	1	None	C (pre-operative), D (post-operative)	Bilateral C8 numbness	Post-operative	No further surgery, prednisolone and amitriptyline	Right hemisensory loss, Lhermitte's and Hoffman's tests positive at 6 weeks postoperatively, bilateral lower limb radiculopathy at 8 weeks; treated with pregabalin and referred to rehabilitation

(Cont'd...)

Table 1. (Continued)

Chatzikomninos <i>et al.</i> ²⁹	2	1	During 3 rd surgery	C (pre-operative), B (postoperative)	Tetraplegia	Postoperative	Methylprednisolone and mannitol	Tracheostomy and remained quadriplegic
Jain <i>et al.</i> ³⁰	4	0	-	D (pre-operative), C (postoperative)	Paraparesis	Post-operative	High-dose solumedrol dosage and then dexamethasone on tapering doses for the next 7 days	3/5 power in upper limb, mobilized in a wheelchair
Zach <i>et al.</i> ³¹	3	1	None	C (pre-operative), D (post-operative)	Left leg monoplegia	Post-operative	No further surgery, steroids for 72 h	Tibialis anterior strength of 3/5 in the left leg at 15-month follow up
Velluto <i>et al.</i> [This paper]	1	-	-	-	Complete tetraplegia	During operation and post-operative	NASCIS protocol	Recovered (6 weeks)

Abbreviations: ACCF: Anterior cervical corpectomy and fusion; ACDF: Anterior cervical discectomy and fusion; ASIA: American spinal injury association; CSF: Cerebrospinal fluid; CT: Computed tomography; F: Female; HLF: Hypertrophied ligamentum flavum; JOA: Japanese Orthopaedic Association Scale; M: Male; MEPs: Motor evoked potentials; MRI: Magnetic resonance imaging; OPLL: Ossification of posterior longitudinal ligament; PCDF: Posterior cervical decompression and fusion; PEEK: Polyetheretherketone; SSEPs: Somatosensory evoked potentials.

3.2.3. Clinical, functional, and radiological outcomes

Postoperatively, all patients developed acute neurological deterioration, ranging from incomplete paresis to complete tetraplegia. The majority exhibited T2 hyperintensity in the spinal cord without mechanical compression, confirming the diagnosis of WCS. Recovery patterns were highly variable: Four patients (25%) achieved full neurological recovery within weeks. Seven patients (44%) experienced partial neurological improvement but had residual deficits. Five patients (31%) remained permanently disabled, with persistent tetraparesis or paraplegia at final follow-up. Steroid administration appeared to correlate with better neurological outcomes, though the response was inconsistent. Patients who required additional surgical interventions (*e.g.*, corpectomy, extended decompression) generally had poorer recovery trajectories. At 6–12-month follow-ups, functional scores (NDI-Oswestry and mJOA) were significantly improved in survivors, but some remained with long-term deficits.

4. Discussion

4.1. Pathophysiology

WCS represents a severe post-operative neurological deterioration following cervical spinal anterior or posterior decompression surgery, characterized by an acute onset motor deficits despite adequate decompression. The leading hypothesis attributes WCS to ischemia-reperfusion injury,

a phenomenon observed in various ischemic pathologies, including myocardial infarction and cerebral ischemia.^{1,2} Theoretically, a chronic compression of the spinal cord leads to vascular insufficiency, metabolic adaptation, and ischemic preconditioning. On decompression, the abrupt restoration of blood flow seems to trigger a cascade of oxidative stress, endothelial dysfunction, and inflammatory responses, culminating in neuronal injury.^{6,16} Several mechanisms have been reported in literature in WCS pathogenesis, such as microvascular dysregulation due to a sudden decompression, which can lead to capillary leakage, endothelial edema, and microvascular thrombosis, exacerbating ischemic damage.¹⁰ Another etiological factor could be the oxidative stress and free radicals. It has been reported that a quick reperfusion generates reactive oxygen species, disrupting neuronal homeostasis and leading to apoptosis.⁹ In addition, neuroinflammation increases leukocyte infiltration, platelet aggregation, and complement activation contribute to blood-spinal cord barrier dysfunction, exacerbating spinal cord edema.⁸ Recent evidence also suggests that microglial activation plays a crucial role in the early phase of central nervous system injury, acting as a potential link between acute trauma and the development of neurodegenerative processes.³² Furthermore, the rapid influx of calcium ions post-reperfusion can lead to glutamate-mediated excitotoxicity, further aggravating neuronal injury.¹⁴ Another factor reported could also be the direct trauma

from the rush of blood coming back into the cord. The immediate influx of blood may cause mechanical stress or injury to the spinal cord tissue.³³ A similar mechanism of such complication could be traced also in brain ischemia, in which reperfusion may exacerbate the injury producing a so-called “cerebral reperfusion injury.” This complication may share some pathophysiological pathways with WCS: In fact, multiple pathological processes are involved, including leukocyte infiltration, platelet and complement activation, post-ischemic hyperperfusion, and breakdown of the blood-brain barrier.¹⁷ Reperfusion injury can also be subacute (until 24 h), especially in elderly populations with atherosclerotic vessels, chronic hypertension, and other comorbidities.²⁵ Furthermore, elderly patients are subject to a physiological condition called “inflammaging.”³⁴ Aging has been recognized as an additional factor influencing neuroinflammation. This persistent inflammatory state, characterized by increased levels of pro-inflammatory cytokines (e.g., Interleukin-6, Tumor necrosis factor- α , and C-reactive protein), may exacerbate the vulnerability of the spinal cord to ischemia-reperfusion injury. In elderly patients undergoing cervical decompression, inflammaging may impair microvascular autoregulation, delay resolution of edema, and hinder neuronal repair mechanisms, potentially worsening WCS outcomes.^{20,27}

4.2. Clinical presentation and diagnostic considerations

The hallmark of WCS is acute onset tetraplegia or paresis immediately following surgery, typically in the absence of intraoperative complications. However, some cases exhibit a delayed-onset neurological decline, usually within 24 h postoperatively.^{7,12} MRI remains the gold standard for diagnosis, consistently demonstrating T2-weighted hyperintensity within the spinal cord without evidence of residual mechanical compression.³ Given the unpredictability of WCS onset, continuous neuromonitoring in the early post-operative period is crucial for timely recognition and intervention.

4.3. Current management and strategies

There is no universally accepted treatment protocol for WCS; however, several strategies have been widely employed: First, high doses of corticosteroids. The administration of methylprednisolone (NASCIS protocol) or dexamethasone aims to mitigate inflammation and oxidative stress, although efficacy remains debated.^{11,18} Second, an optimized perfusion pressure seems to be fundamental. In fact, maintaining MAP above 85–90 mmHg is recommended to enhance spinal cord perfusion and minimize ischemic injury. The use of

pressor agents, such as norepinephrine, has been explored as a supportive therapy.¹⁷ Another important factor is neuroprotective pharmacotherapy. The potential role of antioxidants (e.g., edaravone, *N*-acetylcysteine) and calcium channel blockers in mitigating ischemia-reperfusion injury has been suggested but remains investigational.¹⁵ Finally, early mobilization and intensive physical therapy have demonstrated benefits in promoting neurological recovery in select patients,²⁵ and also in our reported case, the physical rehabilitation performed on the patient has allowed and contributed to a complete restoration of lower and upper limb strength. At least, in selected cases, surgical re-exploration should be considered. Revision surgery may be necessary in cases where a compressive lesion (e.g., hematoma, residual stenosis) is suspected. However, the success rate is variable, and many cases remain refractory to surgical intervention.³⁰ The improvement of neurological function after high-dose steroids supports the former hypothetical mechanism, as steroids inhibit lipid peroxidation after spinal cord injury. Indeed, some have suggested the use of potent antioxidants in managing spinal cord ischemia/reperfusion.¹ However, neither risk nor prognostic factors could be found, and only high doses of dexamethasone and early intensive rehabilitation seemed to be a suitable treatment thus far in the case of post-operative WCS.

4.4. Prognosis and long-term outcome

The prognosis of WCS remains highly variable, with outcomes ranging from full neurological recovery to permanent disability. A systematic review of cases revealed that out of 37 patients analyzed: Three patients (8.1%) achieved full neurological recovery; 12 patients (32.4%) showed partial neurological improvement but retained some degree of functional impairment; and 22 patients (59.5%) exhibited persistent neurological deficits at follow-up, with no significant improvement. Several factors have been identified as poor prognostic indicators, including the presence of pre-existing myelomalacia, severe intraoperative signal loss in neuromonitoring, and delayed initiation of treatment.¹⁹ However, cases managed with early aggressive therapy, including corticosteroid administration and MAP optimization, appear to have a more favorable recovery trajectory.²⁶ An ongoing debate concerns the clinical relevance of high signal intensity on T2-weighted MRI images and the potential reversibility of spinal cord edema. Notably, in many cases, increased T2-weighted signal intensity suggestive of myelopathy was already present before decompression surgery, raising the possibility that gliosis rather than acute ischemia-reperfusion injury may contribute to the observed neurological deficits.² The systematic review of

the 30 included studies shows consistent post-operative neurological deterioration across all patients, with highly variable outcomes, thus supporting the role of ischemia-reperfusion injury as the underlying mechanism.

4.5. Future directions and preventive strategies

Despite increased recognition of WCS, effective preventive and therapeutic strategies remain elusive. Future research should focus on identifying early biomarkers, such as investigating cerebrospinal fluid and serum markers of ischemia-reperfusion injury for early risk stratification.²⁸ Another strategy could be finding neuroprotective agents. In fact, evaluating the role of erythropoietin, magnesium sulfate, and anti-inflammatory cytokine inhibitors might reduce secondary injury.²⁷ Finally, advanced intraoperative monitoring, such as the using of MEPs and SSEPs, could be essential to detect ischemic insults in real time and enable immediate intervention.²⁰

4.6. Limitations

This study has several limitations. As a systematic literature review, it is inherently constrained by the quality and heterogeneity of the included studies. Many reported cases are derived from case reports and small retrospective series, limiting the generalizability of findings. In addition, inconsistencies in diagnostic criteria, therapeutic interventions, and follow-up duration among studies introduce potential selection and reporting bias. The absence of prospective, multicenter studies further restricts the ability to establish definitive treatment guidelines. Future research should aim to standardize diagnostic and therapeutic approaches through larger, well-designed prospective studies.

5. Conclusion

Despite its rarity, WCS is a serious post-operative complication of cervical decompression, with potentially severe neurological consequences. Its pathophysiology aligns with ischemia-reperfusion injury, yet key aspects of risk factors, prevention, and treatment remain unclear. Early recognition is crucial for spine surgeons, as a detailed neurological examination and prompt intervention can significantly improve outcomes. The lack of standardized protocols highlights the need for prospective multicenter studies to clarify its mechanisms and develop effective prevention and treatment strategies. Increasing awareness among spinal surgeons will be essential in reducing morbidity and optimizing patient care.

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Conflict of interest

The authors declare that they have no competing interests.

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Ethics approval and consent to participate

Ethical review and approval were waived for this study due to the retrospective nature of the research and the use of de-identified patient data, in accordance with the policies of the Institutional Review Board. Informed consent was obtained from the patient included in the study.

Consent for publication

Informed consent for publication was obtained from the patient included in the study.

Availability of data

The data that support the findings of this study are available from the corresponding author on reasonable request.

References

1. 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;113:33-36.
doi: 10.1016/j.wneu.2018.02.026
2. Chin KR, Seale J, Cumming V. "White cord syndrome" of acute tetraplegia after anterior cervical decompression and fusion for chronic spinal cord compression: A case report. *Case Rep Orthop.* 2013;2013:697918.
doi: 10.1155/2013/697918
3. Wiginton JG 4th, Brazdzionis J, Mohrdar C, Sweiss R, Lawandy S. Spinal cord reperfusion injury: Case report, review of the literature, and future treatment strategies. *Cureus.* 2019;11(7):e5279.
doi: 10.7759/cureus.5279
4. Page MJ, McKenzie JE, Bossuyt PM, *et al.* The PRISMA 2020

- statement: An updated guideline for reporting systematic reviews. *BMJ*. 2021;372:n71.
doi: 10.1136/bmj.n71
5. Higgins JP, Altman DG, Gøtzsche PC, *et al*. The Cochrane Collaboration's tool for assessing risk of bias in randomised trials. *BMJ*. 2011;343:d5928.
doi: 10.1136/bmj.d5928
6. Giammalva GR, Maugeri R, Graziano F, *et al*. White cord syndrome after non-contiguous double-level anterior cervical decompression and fusion (ACDF): A "no reflow phenomenon"? *Interdiscip Neurosurg*. 2017;7:47-49.
doi: 10.1016/j.inat.2016.12.001
7. Vinodh VP, Rajapathy SK, Sellamuthu P, Kandasamy R. White cord syndrome: A devastating complication of spinal decompression surgery. *Surg Neurol Int*. 2018;9:136.
doi: 10.4103/sni.sni_96_18
8. Papaioannou I, Repantis T, Baikousis A, Korovessis P. Late-onset "white cord syndrome" in an elderly patient after posterior cervical decompression and fusion: A case report. *Spinal Cord Ser Cases*. 2019;5:28.
doi: 10.1038/s41394-019-0174-z
9. Kalidindi KK, Sath S. "White cord syndrome" of acute tetraplegia after posterior cervical decompression and resulting hypoxic brain injury. *Asian J Neurosurg*. 2020;15:756-758.
doi: 10.4103/ajns.AJNS_240_20
10. Fathalla H, Ragab O, Ashry A. White cord syndrome: the terrible nightmare. *Open J Mod Neurosurg*. 2020;10:275-283.
doi: 10.4236/ojmn.2020.102030
11. Busack CD, Eagleton BE. White cord syndrome causing transient tetraplegia after posterior decompression and fusion. *Ochsner J*. 2020;20(3):334-338.
doi: 10.31486/toj.19.0081
12. 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):2343-2347.
doi: 10.1016/j.radcr.2020.08.047
13. Jun DS, Baik JM, Lee SK. White cord syndrome following anterior cervical discectomy and fusion: Importance of prompt diagnosis and treatment. *BMC Musculoskelet Disord*. 2020;21:157.
doi: 10.1186/s12891-020-3162-3
14. Mathkour M, Werner C, Riffle J, *et al*. Reperfusion "white cord" syndrome in cervical spondylotic myelopathy: Does mean arterial pressure goal make a difference? Additional case and literature review. *World Neurosurg*. 2020;137:194-199.
doi: 10.1016/j.wneu.2020.01.062
15. Malinovic M, Walker J, Lee F. Ischemia-reperfusion injury after posterior cervical laminectomy. *Cureus*. 2021;13(9):e18298.
doi: 10.7759/cureus.18298
16. Goyal N, Chaturvedi J, Kandwal P, Gupta P, Kaushal A, Kumar M. "White cord syndrome": A rare catastrophic complication following anterior cervical discectomy and fusion. *Neurol India*. 2022;70(Suppl):S306-S309.
doi: 10.4103/0028-3886.360940
17. Liao YX, He SS, He ZM. "White cord syndrome", a rare but disastrous complication of transient paralysis after posterior cervical decompression for severe cervical spondylotic myelopathy and spinal stenosis: A case report. *Exp Ther Med*. 2020;20(5):90.
doi: 10.3892/etm.2020.9218
18. Acharya S, Kaucha D, Sandhu AS, Adsul N, Chahal RS, Kalra KL. Misdiagnosis of "white cord syndrome" following posterior cervical surgery for ossification of the posterior longitudinal ligament: A case report. *Surg Neurol Int*. 2021;12:244.
doi: 10.25259/SNI_268_2021
19. Segal DN, Lunati MP, Kukowski NR, Michael KW. White cord syndrome and acute tetraplegia after posterior cervical decompression: A case report. *JBJS Case Connect*. 2021;11(2):e20.00281.
doi: 10.2106/JBJS.CC.20.00281
20. Singh RD, Arts MP, de Ruiter GCW. 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
21. Carter LM, Pelargos PE, Gernsback JE. White cord syndrome after thoracic cord decompression in a pediatric patient. *Pediatr Neurosurg*. 2021;56(5):477-481.
doi: 10.1159/000517757
22. Lei CZ, Gong DJ, Zhou YF. Late-onset white cord syndrome following anterior cervical discectomy and fusion: A case report. *Exp Ther Med*. 2022;25(1):71.
doi: 10.3892/etm.2022.11770
23. Dahpute AA, Balasubramanian SG, Annis P. White cord syndrome following posterior decompression and fusion for severe OPLL and an acute traumatic cervical injury: A case report and review of literature. *Surg Neurol Int*. 2022;13:501.
doi: 10.25259/SNI_692_2022
24. So JS, Kim YJ, Chung J. White cord syndrome: A reperfusion injury following spinal decompression surgery. *Korean J Neurotrauma*. 2022;18(2):380-386.
doi: 10.13004/kjnt.2022.18.e36

25. Algahtani AY, Bamsallm M, Alghamdi KT, Alzahrani M, Ahmed J. Cervical spinal cord ischemic reperfusion injury: A comprehensive narrative review of the literature and case presentation. *Cureus*. 2022;14(9):e28715.
doi: 10.7759/cureus.28715
26. Tanaka S, Yoshida S, Tomio R, Mukasa A, Nishimatsu T. White cord syndrome after cervical laminoplasty in an 81-year-old man. *Cureus*. 2023;15(6):e40386.
doi: 10.7759/cureus.40386
27. Ranjan S, Debbarma S, Skandh A, Reang S. Recovery from white cord syndrome after anterior cervical corpectomy and fusion: A case report. *J Orthop Case Rep*. 2023;13(6):138-143.
doi: 10.13107/jocr.2023.v13.i06.3726
28. Yen Hsin L, Samynathan CVV, Yilun H. White cord syndrome: A treatment dilemma. *Cureus*. 2023;15(4):e38177.
doi: 10.7759/cureus.38177
29. Chatzikomninos I, Pappa E, Zafeiris CP, *et al*. White cord syndrome following cervical surgery in a patient with Klippel-Feil syndrome: A case report. *Cureus*. 2024;16(3):e55353.
doi: 10.7759/cureus.55353
30. Jain M, Tripathy SK, Varghese P, Naik S, Sahoo DR, Singh AK. White cord syndrome following long posterior decompression. *J Orthop Case Rep*. 2024;14(9):14-18.
doi: 10.13107/jocr.2024.v14.i09.4712
31. Zach RV, Abdulhamid M, Valizadeh N, Zach V. Delayed lower extremity monoplegia after anterior cervical discectomy and fusion: A report of a rare case of cervical spinal ischemic reperfusion injury. *Cureus*. 2024;16(7):e65071.
doi: 10.7759/cureus.65071
32. Olczak M, Poniatowski ŁA, Siwińska A, *et al*. Elevated serum and urine levels of progranulin (PGRN) as a predictor of microglia activation in the early phase of traumatic brain injury: A further link with the development of neurodegenerative diseases. *Folia Neuropathol*. 2021;59(1):81-90.
doi: 10.5114/fn.2021.105137
33. Epstein NE. Reperfusion injury (RPI)/white cord syndrome (WCS) due to cervical spine surgery: A diagnosis of exclusion. *Surg Neurol Int*. 2020;11:320.
doi: 10.25259/SNI_555_2020
34. Fülöp T, Larbi A, Witkowski JM. Human inflammaging. *Gerontology*. 2019;65(5):495-504.
doi: 10.1159/000497375