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White cord syndrome: a rare and challenging diagnosis. Illustrative case

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BACKGROUND White cord syndrome (WCS) is a rare and extremely serious complication that can occur following spinal decompression procedures for severe mostly cervical spinal stenosis. It is often reported immediately after surgery or several hours to days postoperatively and is identified via a diagnosis of exclusion based on new-onset sudden motor weakness after a decompression procedure.

OBSERVATIONS The authors report the illustrative case of a 54-year-old female patient with WCS, who was managed with surgical intervention, corticosteroid therapy, and mean arterial blood pressure support. Additionally, the authors systematically reviewed an additional 27 cases of WCS documented in the literature.

LESSONS A relatively favorable clinical outcome was observed in this patient following surgical intervention combined with corticosteroid therapy and mean blood pressure support. Currently, there are no established guidelines for the treatment of WCS; however, in any patient experiencing sudden neurological deterioration after cervical spinal decompressive surgery—especially when a known cause is unidentified—WCS should be considered as a potential diagnosis, and prompt treatment should be initiated to attempt to improve outcomes.

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KEYWORDS white cord syndrome; reperfusion injury; decompression surgery; spine

Cervical spondylosis with spinal canal stenosis is one of the most common neurosurgical pathologies that can lead to neurological symptoms and myelopathy. Selected studies have reported that radiological spondylosis is seen in approximately 95% of patients older than 65 years of age. One of the rarest but most devastating complications after decompression surgery is so-called white cord syndrome (WCS),[1,2] which occurs at a rate of less than 1%.[3,4] The most common reported occurrences of WCS typically happen after cervical decompression procedures.[2] Diagnosis of WCS is often made via a diagnosis of exclusion, based on new-onset sudden motor weakness reported by the patient either immediately or hours to days after decompression surgery. This motor weakness cannot be reported before the procedure, nor can it be caused by damage to the spinal cord (as identified radiologically).

The mechanism of WCS is hypothesized to be ischemia-reperfusion injury via damaged oxygen-derived free radicals after acute reperfusion of chronically ischemic tissue.[2,5] The injury is defined as the sudden postdecompressive expansion of the compressed cord and a rush of blood supply with subsequent edema and swelling. This sudden rush can lead to disruption of the blood-brain and blood–spinal cord barriers and trigger a cascade of events, including hypoxia and inflammation, that may result in acute neurological dysfunction.[5,6] Ironically, the treatment intended to relieve pressure can disrupt spinal cord blood supply and lead to unfavorable outcomes worse than the patient's preoperative neurological status. Cervical spine MRI suggesting WCS is based on the presence of a hyperintense intramedullary area in postoperative T2-weighted MRI studies with associated worsening neurological deficit, mainly motor.[7,8]

Herein, we present a case report of WCS after an anterior cervical discectomy and fusion (ACDF) procedure with a systematic review of additional cases from the literature to raise awareness of this rare but potentially devastating condition in which early identification and treatment are essential. The objective of this systematic review was to demonstrate the authors' experience treating WCS and summarize other known cases of WCS.

ABBREVIATIONS ACDF = anterior cervical discectomy and fusion; MAP = mean arterial pressure; MRC = Medical Research Council; WCS = white cord syndrome.

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Methods

We present our own case of WCS first. The patient provided written informed consent for publication. For the systematic review, we searched the literature for cases of patients diagnosed with WCS from January 1, 2013, to October 31, 2024. We chose 2013 as the start date for our search because the syndrome was first delineated in the literature in 2013.[5] The review was conducted in accordance with the PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) guidelines to ensure methodological rigor and transparency (Fig. 1).[9]

A comprehensive search of the National Library of Medicine's PubMed database was performed in November 2024 using the following search string: ((white cord syndrome) OR (reperfusion injury)) AND spine. We used the term "reperfusion injury" because WCS is sometimes reported as reperfusion injury. Two independent searches were carried out by the authors, and additional references from the studies included were manually searched to identify any further relevant articles.

Inclusion criteria were articles published in the English language and WCS articles that specifically addressed WCS or reperfusion injury related to spinal cord injuries, focused on the clinical presentations of WCS-related neurological deficits, and included radiologically confirmed T2-weighted MRI hyperintense signals of the spinal cord. WCS diagnoses in these articles were made following the exclusion of all other potential diagnoses. Exclusion criteria included review articles and studies that did not provide sufficient detail about the diagnostic protocol or that presented an evident alternative etiology other than WCS for reported neurological deterioration.

After a thorough examination, data extraction was conducted to compile relevant information, including demographics, clinical features, therapeutic interventions, and outcomes for each patient under study.

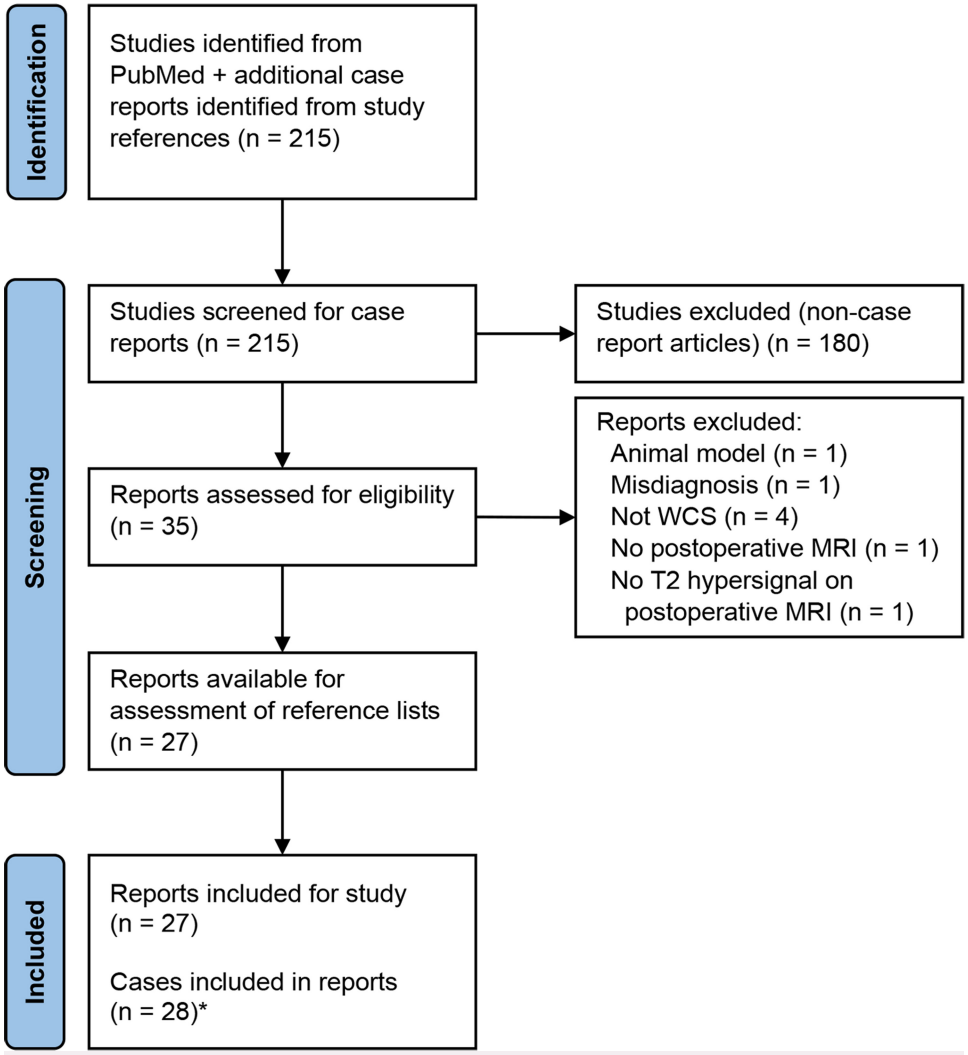

FIG. 1. PRISMA flow diagram. Data added to the PRISMA template (from Moher D, Liberati A, Tetzlaff J, Altman DG, The PRISMA Group. Preferred Reporting Items for Systematic Reviews and Meta-Analyses: The PRISMA Statement. *PLoS Med.* 2009;6[7]:e1000097) under the terms of the Creative Commons Attribution (CC BY-NC 2.0) License (https://creativecommons.org/licenses/by/2.0).

Illustrative Case

Case Presentation

A 54-year-old woman was admitted to the Department of Neurosurgery at the Clinical Center University of Sarajevo for scheduled ACDF surgery. Her initial presentation included chronic neck stiffness and pain, as well as right hand weakness and pain and a loss of fine motor skills. These symptoms were present in the 6 months prior to surgery. Conservative treatment prior to surgery failed.

The patient reported no weight loss, night fever, or other systemic complaints. On examination, we observed a limited and painful range of motion in her neck. The muscle strength in her right hand was weakened, scoring 3/5 on the Medical Research Council (MRC) scale. She also exhibited evident tremor in her right hand when raised and dysmetria during the finger-nose test on the right side. Lhermitte's sign was positive, indicating an electric shock–like sensation when the neck was flexed. However, a Hoffmann's test produced negative results. Her right leg was also weakened, scoring 4/5 on the MRC scale. A finger-nose proprioception test on the right side showed dysmetria, while her tandem gait was normal.

MRI of the cervical spine showed a herniated disc from C3 to C5 with findings of C3–4 grade I spondylolisthesis and C4–5 disc herniation with signs of myelopathy (Fig. 2).

Operation

The C3–5 ACDF procedure was performed. Intraoperatively, we observed that the dura somewhat adhered to the posterior longitudinal ligament. The remaining aspects of the surgery proceeded without complication. Subsequently, the patient was awakened but presented immediately with motor deficits, including weakness affecting the upper and lower extremities, while retaining intact speech and cranial function.

Postoperative Course

The patient was admitted to the neurosurgical intensive care unit to maintain optimal blood pressure, which required a continuous administration of dobutamine and noradrenaline to maintain a systolic blood pressure above 90 mm Hg. Despite these measures, the patient did not demonstrate any improvement in neurological function approximately 2 hours postsurgery. Brain and cervical spine CT imaging was performed to investigate a cause for the patient's clinical status (Fig. 3A); however, the CT did not reveal any clear morphological evidence to explain the patient's condition. Following the scan, the patient was brought back to the operating room for revision surgery for a possible hematoma or compression of the hemostatic material.

An ACDF revision procedure was undertaken that involved removal of the previously placed pretracheal drain, skin sutures, titanium plate, fixation screws, and cages. Additional exploratory procedures were conducted. Notably, there was no evident compressive effect noted on the dura, and the ventral dura appeared to be adequately decompressed. The cages were subsequently reinserted, and a titanium plate was secured following the same technique as in the initial operation. Hemostasis, lavage, and subfascial drain placement were performed next, followed by closure of tissue layers with suturing.

Following the procedure, the patient again experienced a prolonged emergence from anesthesia, spontaneous respiration, and marginal improvement in the mobility of the lower extremities (minimal toe wiggling). Her blood pressure was continuously targeted above 90 mm Hg. Methylprednisolone was administered intravenously at a dose of 30 mg/kg as a bolus, followed by a continuous infusion of 5.4 mg/kg/hr for a duration of 23 hours. The following day, the patient's quadriparesis persisted and somewhat worsened. MRI of the cervical spine was conducted on the next day, which revealed an area of modified signal intensity within the cervical spinal cord, specifically in the C3–5 region, and within the medulla as observed on the T2 sequence (Fig. 3).

Because of the presence of edema of the spinal cord, the patient underwent a third operation: a posterior decompressive unilateral hemilaminectomy procedure at the C3–5 level. The patient was sedated for 24 hours after surgery. Following this sedation period, the patient regained consciousness and exhibited quadriparesis again, including paraplegia, while maintaining stable vital signs. Physical therapy was initiated, wound healing progressed uneventfully, and sutures were removed. Inotropic support was discontinued, resulting in hemodynamic stability.

The patient was transferred to the Department of Physical and Rehabilitation Medicine. At the time of discharge 3 months after

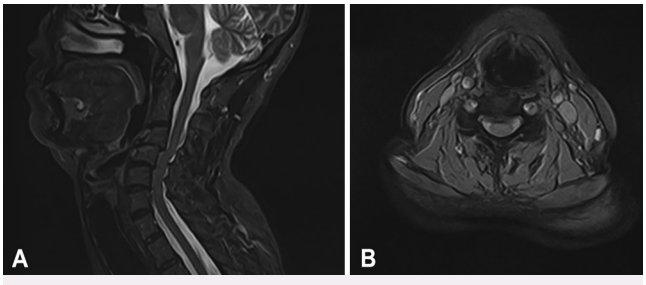

FIG. 2. A: Preoperative sagittal T2-weighted MR image of the cervical spine, showing marked cervical lordosis with visible degenerative changes of the definitive spondylosis type and discrete spondylarthrosis on the right side. Degenerative retrolisthesis of the vertebral bodies at the level of C3–4 and C4–5 was more pronounced in the proximal part. **B:** Axial T2-weighted MR image of the C4–5 level, showing disc herniation at the C4–5 level accompanied by patient-demonstrated clinical signs consistent with myelopathy, as evidenced by a significant reduction in spinal canal diameter measuring up to 7.4 mm.

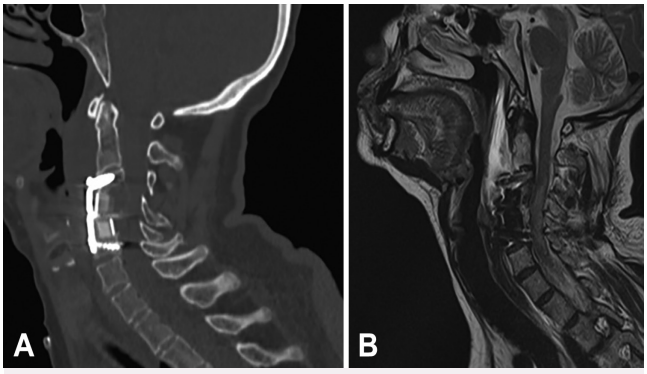

FIG. 3. A: Postoperative cervical CT scan obtained immediately after the first surgical attempt. The sagittal scan with "bone window" shows appropriate anterior instrumentation of the cervical spine with the correction of listhesis and no worsening of preoperative stenosis. **B:** Postoperative sagittal T2-weighted MR image of the cervical spine, showing marked cervical lordosis with visible degenerative changes of the spondylosis deformans and discrete spondylarthrosis on the right. Worsening myelopathy was also noted.

surgery, the patient was communicative and hemodynamically stable with left arm strength graded at 4/5, right arm strength at 3/5, and both legs at 3/5 with assistance for standing. After another month, both hand strengths improved to 4/5, while leg strengths improved to 3+ to 4/5. The patient was able to achieve a seated position with minimal assistance, maintaining balance independently. With assistance, the patient was able to achieve an upright position and take a few steps. A second follow-up MRI study of the cervical spine 4 months after the surgeries revealed regression of the previously observed hyperintense region in the spinal cord, corresponding to the previously diagnosed WCS (Fig. 4). At the 1-year follow up, the patient could walk with assistance and was able to lean better on her left leg (with stable motor strength).

Systematic Review

A comprehensive search of the PubMed database resulted in 215 articles available for screening. Following initial assessment, abstracts were screened for case reports, resulting in the exclusion of 180 articles. The remaining 35 papers underwent a detailed review to ascertain their relevance to WCS, ultimately yielding 27 applicable papers and abstracts. Furthermore, after a meticulous manual examination of the reference lists of these papers, we discovered an additional 5 articles that were subsequently included in the study for a total of 27 articles (Fig. 1) featuring 28 cases (the study by Singh et al. reported 2 cases[10]).

The inaugural case of WCS was reported by Chin et al. in 2013 following ACDF surgery, marking the first documented reference to "white cord syndrome."[5] In a systematic review conducted by Mathkour et al. in 2020, only 6 cases of WCS were discussed.[1] Subsequent work by Tanaka et al. identified an additional 7 cases,[11] culminating in a total of 13 documented cases by 2021, with the case of an 81-year-old patient subsequently published in 2023.[11] All patients reported were older than 18 years of age, with two notable pediatric instances. Specifically, Carter et al.described a case involving a 3-year-old patient who developed WCS after resection of a thoracic spine tumor,[8] while Sepulveda et al. reported on a 12-year-old patient who exhibited WCS following the resection of an anterior cervical arachnoidal cyst.[12]

The mean age of the affected individuals was 54.76 years, with 7 patients younger than 50 years, including the aforementioned pediatric cases. Among these, a notable case reported by Chatzikomninos et al. described a 33-year-old male patient with Klippel-Feil syndrome.[13] The youngest patient in the cohort was 3 years of age, whereas the oldest patient was 81 years. A demographic analysis revealed that 20 (71%) of the patients were male.

Surgical approaches were predominantly posterior (53.6%), and intraoperative neuromonitoring was documented in 15 (53.6%) cases. Additionally, 8 (28.6%) cases reported measurements of middle arterial perfusion during surgery. The most prevalent form of neurological deterioration after surgical intervention was tetraplegia, which was observed in 17 (60.7%) cases. Among the 28 cases reviewed, 7 patients required additional surgical procedures, and nearly all received corticosteroid treatment following the onset of neurological deterioration, with only 1 article reporting otherwise. Some patients had full recovery, but some did not recover at all, as shown in Table 1.

Informed Consent

The necessary informed consent was obtained in this study.

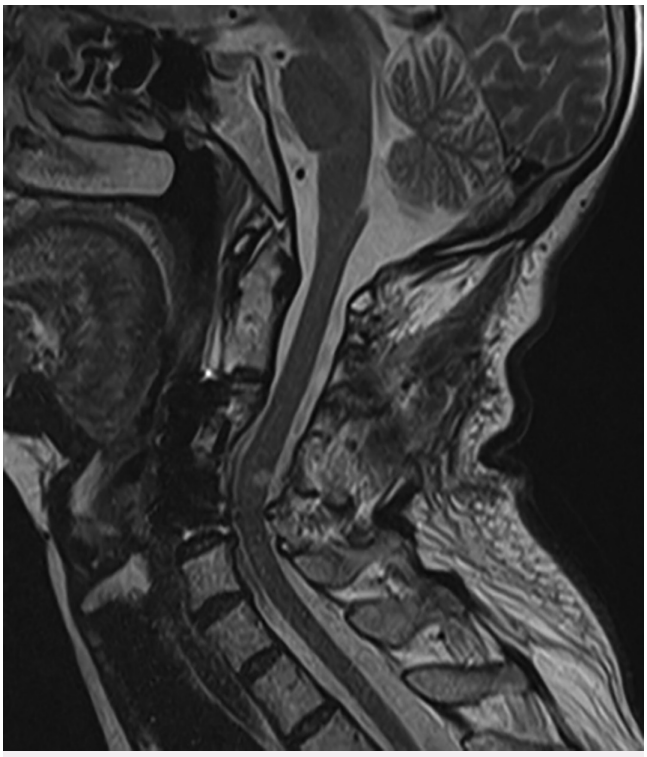

FIG. 4. Postoperative sagittal T2-weighted MR image of the cervical spine obtained 5 months after surgeries, showing regression of the previously observed hyperintense region of the spinal cord.

Discussion

Observations

This report illustrates the case of 54-year-old woman with WCS after ACDF. We performed 2 consecutive operations within 24 hours to support neurological improvement and prevent respiratory arrest. Support to maintain blood pressure above 90 mm Hg was initiated immediately after surgery. Methylprednisolone was immediately administered intravenously at a dose of 30 mg/kg as a bolus, followed by a continuous infusion of 5.4 mg/kg/hr for a duration of 23 hours. During the postoperative period, physical therapy was aggressively maintained as it has been reported to be a beneficial nonsurgical treatment for WCS. Six months postsurgery, our patient was able to walk with assistance and could perform activities of daily living independently.

Etiology and Cases

In this systematic review, we found 28 cases of patients with a diagnosis of WCS, including our case. One of the most serious and devastating complications following cervical spinal surgery resulting in neurological motor dysfunction is quadriplegia, which is hypothesized to occur due to mechanical injury of the spinal cord, inappropriate fixation, hematoma formation, dural tear, edema, and/or reperfusion injury, known as WCS.[7]

The first case of WCS was described in 2013 after ACDF surgery.[5] Currently, there are no established guidelines to diagnose WCS, which is typically diagnosed by excluding other etiologies. A diagnosis of WCS is made via postoperative T2-weighted MRI sequences that demonstrate a hyperintense intramedullary signal.[1–8] The pathophysiological mechanism underlying WCS is not fully understood; however, it is proposed that reperfusion of the spinal cord following decompression leads to a sudden increase in blood flow and elevated levels of oxygen free radicals. These free radicals can damage the spinal cord

TABLE 1. Reported cases of WCS

Case No.	Authors & Year	Age, yrs/Sex; Comorbidities	Pathology (symptom duration)*	Approach	Intraop Monitoring	Neurological Deficit	Reported BP	Reop	Steroids	Outcomes (later FU)
1	Chin et al., 2013[5]	59/M	Herniated disc C5–6 (7 mos)	ACDF C4–6	Complete loss SSEPs & MEPs	Postop incomplete tetraplegia	—	C5 corpectomy, decompression	Hydrocortisone 100 mg (intraop) & dexamethasone postop	Improvement in standing & walking; required an assistive frame to get around house & wheelchair outside (16 mos)
2	Giammalva et al., 2017[24]	64/M	Herniated disc C3–4 & C5–6	ACDF C3–4 & C5–6	SSEPs & MEPs decreased in voltage during closure time of superficial planes	Postop tetraplegia	—	—	High-dose dexamethasone	Partial recovery in rt hand prehensile strength (3/5), partial rt arm recovery in flexion (2/5), flexion of both legs on thighs (2/5) (day 3)
3	Antwi et al., 2018[2]	68/M	Cervical stenosis w/ CSM C4–6 (2–3 yrs)	Pst decompression C4–7 via laminectomy & C3–7 instrumentation	SSEPs & MEPs; loss of MEPs during closure of suprafascial tissues	Postop lt-sided hemiparesis	MAPs 88–105 mm Hg throughout case; MAPs >113–115 mm Hg after loss of MEPs	Wound reopened & explored & laminectomy at C7, screws checked	Solumedrol infusion for 24 hrs followed by dexamethasone taper	Pt improved over 3 days; pt was ambulatory w/ a walker (day 3)
4	Vinodh et al., 2018[6]	51/F; cervical ductal carcinoma	Cervical extramedullary metastatic ductal carcinoma (1 mo)	Pst laminectomy C2–5; rods w/ pedicle screws used to fuse C1, C2, C5, & C6	—	Postop tetraplegia & required ventilator support	—	—	High-dose dexamethasone	Neurological status never improved
5	Wiginton et al., 2019[25]	41/M	Severe cervical stenosis C1 w/ severe cord compression (6 mos)	Pst laminectomy C1 & partial C2	SSEPs from hands gone, followed by all extremities; loss of MEPs in upper extremities, after laminectomy	Tetraplegia	MAP >85 mm Hg during op; >95 mm Hg after SSEP & MEP loss; MAP >90 mm Hg for 5 days	—	IV dexamethasone 10 mg every 6 hrs	Shortly after recovering from anesthesia, patient was able to bend knees bilaterally against gravity; hand grip became 4/5 bilaterally

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TABLE 1. Reported cases of WCS

Case No.	Authors & Year	Age, yrs/Sex; Comorbidities	Pathology (symptom duration)*	Approach	Intraop Monitoring	Neurological Deficit	Reported BP	Reop	Steroids	Outcomes (later FU)
6	Mathkour et al., 2020[1]	79/M; hypertension	Cervical stenosis secondary to spondylosis C3–6 w/ CSM (2–3 mos)	Pst laminectomy C3–5 w/ C2–6 instrumentation	While closing fascia, SSEPs were 50% of baseline amplitude, MEPs were normal	Worsened rt-sided hemiparesis	MAP >85 mm Hg	—	Dexamethasone 4 mg every 6 hrs for 5 days	Ambulatory w/ use of a walker; rt lower extremity showed full strength; rt upper extremity strength continued to show improvement; exam showed equal sensation bilaterally (4 mos)
7	Papaioannou et al., 2019[22]	79/M; hypertension, atrial fibrillation, previous ACDF	Pst stenosis C4–6 w/ CSM (2 yrs)	Pst decompression C3–6 & pst fusion w/ lat mass screws C2–7 (double-level ACDF w/ ant plate & cage performed 2 yrs prior)	Neuromonitoring; no intraop neurological worsening	24 hrs postop tetraplegia	—	Extended pst decompression	High-dose steroid	3/5 strength in rt upper extremity, 4/5 strength in lt upper extremity, 2/5 strength in rt leg, 3/5 strength in lt leg (18 mos)
8	Jun et al., 2020[26]	49/F	Extruded disc C6–7	ACDF C6–7	—	Paraplegia lower extremity	—	Laminoplasty C4–5 to C6–7	Methylprednisolone 30 mg/kg/15 mins, 5.4 mg/kg/23 hrs	Fully recovered
9	Liao et al., 2020[27]	51/M	OPLL C2–4 w/ spinal stenosis (6 mos)	Pst laminectomy decompression C2–4 & C2–5 instrumentation	—	Lt hemiplegic paralysis	—	—	Methylprednisolone bolus (500 mg), intraop methylprednisolone (30 mg/kg) over 15 mins, followed by 45-min rest & 23-hr maintenance infusion (5.4 mg/kg/hr) after op	Lt limb muscle strength recovered to grade 4/5, which was same as preop level (day 5)
10	Sepulveda et al., 2020[12]	12/M; VP shunt, chronic encephalopathy, neonatal meningitis, spastic tetraparesis, epilepsy	Cyst in intradural extramedullary space C2–4, ant	Pst cervical decompressive op & arachnoid cyst fenestration	—	Postop monoplegia; symptoms of dysautonomia 1 mo 9 days later	—	—	Dexamethasone	Good improvement

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Case No.	Authors & Year	Age, yrs/Sex; Comorbidities	Pathology (symptom duration)*	Approach	Intraop Monitoring	Neurological Deficit	Reported BP	Reop	Steroids	Outcomes (later FU)
11	Kalidindi & Sath, 2020[19]	63/M; DM	Cervical canal stenosis w/ extensive OPLL & cord atrophy w/ myelomalacia (6 mos)	Pst laminectomy C2–T1 & C2, C3, C4, C5 & T1 instrumentation	Complete loss of MEPs after laminectomy	Tetraplegia	MAP >90 mm Hg	—	Methylprednisolone	No improvement (coma) (4 mos)
12	Singh et al., 2021[10] (case 1)	58/F; hypertension, type 2 DM, parathyroidectomy	Spinal stenosis C3–6 w/ OLF T2 hyperintense signal C3–4 & C5–6 (2 mos)	Pst decompressive laminectomy C3–6	—	Almost tetraplegia POD 4	Hypotension (40/20 mm Hg) POD 4; MAP >100 mm Hg	—	Initial dexamethasone 10 mg, followed by 4 mg QID for 6 days	Able to stand independently & grade 4 muscle strength in all limbs (6 mos)
13	Singh et al., 2021[10] (case 2)	66/F; hypertension	Spinal stenosis C3–4 & C4–5; T2 hyperintense signal C2–3 to C6–7; OPLL C3–4 & C4–5; deformity correction (3 mos)	C4 corpectomy w/ placement of expandable cage; ACDF C5–7 w/ plate fixation C3–7	—	Paralysis in lt arm; grade 4 muscle strength rt arm; grade 3–4 muscle strength rt leg; grade 4 muscle strength lt leg; POD 8	—	—	Dexamethasone 2 mg BID for 3 days	Pt improved but was unable to walk unassisted (3 mos)
14	Carter et al., 2021[8]	3/F; mediastinal neuroblastoma partially resected 2 yrs prior	Ganglioneuromatous tissue, causing thoracic cord compression T4–8 (5 days)	Laminoplasty T5–7 for microsurgical debulking of thoracic mass	No motor or SSEP responses in bilat lower extremities present at any point in case	Paraparesis POD 1	—	—	Steroids for 7 days	Ambulating well & 5/5 strength in all muscle groups, w/ no neurological deficits (4 mos)
15	Segal et al., 2021[28]	55/M	CSM	Pst cervical decompression & instrumentation	—	Acute tetraplegia	MAP >90 mm Hg	—	Dexamethasone	Markedly improved clinical exam
16	So et al., 2022[14]	61/M	OPLL C3–6 w/ ruptured disc at C6–7 w/ cord signal change C5–6	ACDF C4–5 & C6–7	—	Near-complete tetraplegia	MAP 60–85 mm Hg during 1st op; MAP 76–93 mm Hg during 2nd op	Laminectomy C4–6; subtotal laminectomy C7 & lat mass screw fixation C4–6	Methylprednisolone	Motor weakness fully recovered (2 mos)
17	Goyal et al., 2022[29]	39/F; DM	Prolapsed intervertebral disc C5–6	ACDF C5–6	—	Tetraplegia	—	—	8 mg dexamethasone stat, followed by 6 mg QID, later tapered	Power improved to 4+/5 in all 4 limbs; ambulating w/o support (17 mos)

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Case No.	Authors & Year	Age, yrs/Sex; Comorbidities	Pathology (symptom duration)*	Approach	Intraop Monitoring	Neurological Deficit	Reported BP	Reop	Steroids	Outcomes (later FU)
18	Dahapute et al., 2022[30]	63/M	Cervical OPLL C2–4 w/ intrinsic signal changes in cord; C6 fracture	Pst cervical decompression C2–7 & instrumented fusion C2–T2	MEPs abolished below C4 w/ preservation of trapezius MEPs; rt upper limb SSEPs showed 50% decrease	Tetraplegia	MAP >85 mm Hg	—	Dexamethasone 8 mg BID; steroids tapered over 1 wk	No improvement
19	Hasan et al., 2022[20]	62/M; hypertension, DM w/ resultant chronic renal disease	Canal stenosis & spinal cord compression w/ increased T2 signal intensity changes C5–6 & C6–7 (2 days)	Ant discectomy C3–4, C5–6, & C6–7 w/ integrated plate/cage	Neuromonitoring	Tetraplegia POD 1	—	—	Methylprednisolone therapy at a dose of 8 mg BID for 4 days	Gradual improvement in muscle power in both extremities was noted at discharge
20	Lei et al., 2023[21]	54/M	Herniated discs C4–7 w/ CSM	ACCF C4–7	—	Tetraplegia POD 7	—	PCD day 11 after ACCF	Methylprednisolone (80 mg, IV BID)	Limb muscle strength recovered to grade 4/5; leg strength recovered to grade 5/5 (7 mos)
21	Yen et al., 2023[7]	61/M; previous decompressive op 2 yrs prior for lt C6–7 neural foramen stenosis	Narrowed lt C6–7 neural exit canal & moderate spinal canal stenosis at C3–4 & C4–5	ACDF C6–7	Intraop neuromonitoring did not show any alert	POD 6, new-onset bilat C8 numbness; postop 6 wks, rt triceps wasting w/ rt hemisensory loss; postop 8 wks, bilat lower limb radiculopathy	—	—	No steriods; treated with pregabalin and physiotherapy	Not obtained by authors
22	Ranjan et al., 2023[17]	64/F	Pathological lesion (tubercular infection) w/ extradural compression & cord edema C5–6 (3 mos; weakness in previous 3 days)	Cervical decompression (corpectomy C5–6) w/ instrumentation via ant approach	—	Pt could not be extubated for 4 hrs; tetraplegia	—	—	Methylprednisolone (30 mg/kg body weight over 15 mins, followed by 5.4 mg/kg body weight over next 23 hrs)	Normal neurology (1 yr)
23	Tanaka et al., 2023[11]	81/M	Canal stenosis due to OPLL C3–5; intramedullary T2 high signal (~5 yrs)	C3–6 double-door laminoplasty	MEPs showed reduction in amplitude	Tetraplegia	—	—	Steroid	Brunnstrom stage 2 upper rt & lower lt limbs & 3 for upper lt limb (6 mos)

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24	Present study	54/F; dyslipidemia	Cervical disc herniation; spondylolisthesis w/ CSM (6 mos)	ACDF C3–5	—	Tetraplegia	—	Ant revision op & pst decompression/ hemilaminectomy C3–5	Methylprednisolone (30 mg/kg body weight over 15 mins, followed by 5.4 mg/ kg body weight over next 23 hrs)	Ambulatory w/ assistance; leaned better on lt leg (lt 4−/5, rt 3/5); rt hand strength better (4/5, lt 3/5) (1 yr)
25	Chatzikomninos et al., 2024[13]	33/M; Klippel-Feil syndrome	Kyphotic malformation; CSM (2 ops prior)	1st op: ant spinal fusion C4–6 w/ C5 corpectomy; 2nd op: laminectomy C5–7 & pst fusion of C5–T1; 3rd op: 3rd occipitocervical fusion w/ C1–5 laminectomy	At 3rd op after implanting rods in situ, complete nullification of intraop neurophysiological control	Tetraplegia & tracheotomy	—	—	IV intraop methylprednisolone	No improvement (2 mos)
26	Zach et al., 2024[31]	49/M; mild traumatic brain injury 5 mos prior, hyperlipidemia, hypertension, lipoma resection	Stenosis w/ compression & myelomalacia C3–4 & C4–5	ACDF C3–4 & C4–5	Neuromonitoring	Lt leg monoplegia w/ diffuse numbness	—	—	Steroids 72 hrs	Tibialis ant strength 3/5 in lt leg, indicating improvement (15 mos)
27	Jain et al., 2024[32]	63/M; hypertensive	OPLL C2–T1 (2 yrs)	Pst instrumented decompression C2–T5	—	Upper limbs 0/5, tracheostomized	—	—	High-dose solumedrol followed by tapering doses of dexamethasone for next 7 days	Decannulated, power in upper limbs improved to 3/5; mobilized in a wheelchair (6 mos)
28	Mahamid et al., 2024[33]	52/M	Herniated discs C2, C3, C4, C6, & C7, OPLL (3 wks)	Laminectomy C3–6 & cervical fixation C3–7	Neuromonitoring	Weakness in rt hand & leg	—	—	Steroids	Improvement in rt leg motor function; no change was observed in rt hand (4 wks)

ACCF = anterior cervical corpectomy and fusion; ant = anterior; BID = twice a day; BP = blood pressure; CSM = cervical spondylotic myelopathy; DM = diabetes mellitus; FU = follow-up; IV = intravenous; MEP = motor evoked potential; OLF = ossification of ligamentum flavum; OPLL = ossification of posterior longitudinal ligament; PCD = posterior cervical decompression; PCLF = posterior cervical laminectomy and fusion; POD = postoperative day; pst = posterior; pt = patient; QID = four times a day; SSEP = somatosensory evoked potential; TB = tuberculosis infection; VP = ventriculoperitoneal; — = no data/information.
* Included when reported in the case.

and blood-brain barrier, inducing mitochondria-dependent apoptosis, production of TNF-α, and specific phospholipid signaling cascades. This mechanism is most commonly observed in older patients with hypertension.[1,5,7,8] A study conducted with a rat model investigated the mechanisms underlying reperfusion spinal cord injury. The researchers proposed that the release of acute spinal cord compression could displace gray matter. They found that rats experiencing severe spinal cord compression were more likely to show a significant decline.[6] The reported incidence of WCS following any type of decompressive spinal surgery, such as ACDF, laminectomy, laminoplasty, or discectomy, is approximately 1%.[14] This low incidence may explain why many cases go unreported or undiagnosed, contributing to the scarcity of reported cases in the literature.

Treatment With Steroids

Most reported cases of WCS appeared to improve with steroid administration alone, although a single case did not receive steroids and another case, that did receive steroids, had a poor outcome.[6,7] Steroids have anti-inflammatory properties and reduce oxidative stress, which is a critical pathological event that can contribute to WCS. They may also help to minimize edema and support blood supply to the spinal cord.[7] However, there is insufficient evidence to advocate for the routine use of steroids in acute spinal cord injuries. A recent meta-analysis concluded that methylprednisolone therapy administered within the first 8 hours after acute spinal cord injury did not lead to significant improvements in overall motor or neurological function compared to patients who did not receive steroids. Furthermore, patients treated with methylprednisolone were more likely to develop pneumonia and hyperglycemia compared to those who did not receive this treatment. Consequently, the routine use of methylprednisolone following spinal cord injuries should be carefully considered in light of these findings.[15]

Surgical Treatment

In some instances, steroids and blood pressure support should not be viewed as the sole treatment options, as demonstrated in our case, in which we performed additional posterior decompression early to allow enough space for the cervical spinal cord and to eliminate the risk of further spinal cord compression. There is no definitive evidence supporting the benefit of additional surgery, as it can lead to spine instability and increase the risk of postoperative complications due to oxidative stress and its by-products. Nonetheless, it is frequently considered. Research on spinal cord injuries, however, suggests that early decompression is a reasonable option that can be performed safely, although no specific evidence supports the use of laminectomy alone. Emerging evidence indicates that intended durotomy followed by extended meningoplasty may improve neurological outcomes in patients with spinal cord injuries with apparent meta-traumatic edema. Nevertheless, the lack of high-quality evidence underscores the need for further research.[16]

Hypotension and Mean Arterial Pressure

Hypotension has been reported in patients with WCS and may be a factor contributing to the lack of neurological recovery. However, there is no strong evidence to suggest that maintaining blood pressure is crucial in spinal cord injury cases. Current guidelines recommend maintaining a mean arterial pressure (MAP) of 85–90 mm Hg for 5–7 days following spinal cord injuries.[17] For patients with WCS, keeping the MAP at or above 85 mm Hg could have positive effects on restoring or improving lost neuromonitoring signals.[10] A decrease in MAP during surgery could be a clinical feature indicating the occurrence of WCS, as observed in our case at the end of the procedure. However, 2 cases from the same study—one with MAP regulation and the other without—both showed neurological improvement.[10] Among the published cases, 10 reported their MAP goals during surgery; of these, 5 indicated goals either below or above 85–90 mm Hg.[2,10,14,18,19] The onset of WCS can be acute, occurring immediately after surgery, as in our case, or delayed, as reported by other authors.[7,20–22]

Intraoperative Neuromonintoring

Intraoperative neuromonitoring is utilized in some centers during cervical spine procedures, as indicated in Table 1. According to guidelines from the Joint Section of the American Association of Neurological Surgeons and Congress of Neurological Surgeons, there is level I evidence supporting the use of intraoperative neuromonitoring for diagnostic detection during spine surgery and level III evidence for its use in therapeutic applications (i.e., reduction of patient deficits) or cost-effectiveness.[23] Regarding the use of intraoperative neuromonitoring in patients with WCS, either signal changes typically occur after decompression of the spinal cord, like during the closure of the fascia,[2,3,6] or there are no signal changes during the surgery (Table 1). Notably, these changes tend to develop over a prolonged period following decompression, making it unlikely that they are related to mechanical injury of the spinal cord. While intraoperative neuromonitoring does not appear to predict the development of WCS reliably, it can be valuable for detecting mechanical trauma to the spinal cord during surgery. Further research is needed to better understand these dynamics and to optimize intraoperative monitoring strategies.

Lessons

WCS is a rare condition following decompressive spinal surgery that can lead to quadriplegia. We present the case of a patient who underwent both anterior revision and posterior decompression surgeries after the occurrence of postoperative quadriplegia. The combination of decompressive surgery, blood pressure support, steroid treatment, and aggressive physical therapy resulted in neurological improvement in this case. We found 28 cases of WCS, including ours, reported in the literature, although we suspect that this postoperative complication has been underreported. Currently, there are no guidelines for the management of WCS; however, early recognition and prompt treatment are crucial steps for improving the patient's neurological deficit.

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Disclosures

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

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