

White Cord Syndrome after Cervical Decompression: A Narrative Review and Proposed Management Algorithm

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Abstract:

➤ *Background:*

White Cord Syndrome (WCS) is a rare but devastating complication of cervical decompression, defined by acute postoperative neurological deterioration with intramedullary T2 hyperintensity on MRI, in the absence of compressive pathology. It is increasingly recognized as a manifestation of ischemia–reperfusion injury of the chronically compressed spinal cord.

➤ *Methods:*

A targeted review of the literature (2010–2025) was performed, including case reports, series, and systematic reviews. Data on clinical presentation, pathophysiology, management, and outcomes were synthesized.

➤ *Results:*

Most patients improved with high-dose steroids and hemodynamic augmentation. Approximately 25–30% underwent additional posterior decompression (laminectomy/laminoplasty). Early surgical rescue was associated with better outcomes than delayed intervention. Controversies remain regarding steroid efficacy, optimal mean arterial pressure (MAP) targets and duration, and timing of surgical escalation.

➤ *Conclusions:*

Medical therapy remains the cornerstone, but urgent posterior decompression may be warranted in refractory cases, especially in multilevel stenosis or ossification of the posterior longitudinal ligament (OPLL). We present an updated synthesis of evidence, highlight controversies, and propose a pragmatic management algorithm.

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I. INTRODUCTION

Postoperative neurological deterioration is among the most feared complications in cervical spine surgery. Common causes include epidural hematoma, graft malposition, and incomplete decompression. However, in rare cases, deterioration occurs without any compressive lesion. In 2013, Chin et al. first described such an event following anterior cervical discectomy and fusion (ACDF), coining the term White Cord Syndrome (WCS) [1].

The defining feature is acute or subacute postoperative neurological worsening, with new or worsened intramedullary T2 hyperintensity on MRI. This “white cord” represents a presumed ischemia–reperfusion injury: chronic cord compression produces hypoperfusion and neuronal

adaptation; sudden decompression restores blood flow but provokes oxidative stress, excitotoxicity, and edema [2–4].

Although rare, WCS may be underdiagnosed. Reasons include overlap with hematoma or residual compression, diagnostic uncertainty in patients with preexisting T2 changes, and variable severity. Recent systematic reviews identified an increasing number of cases [5], reflecting growing recognition as decompressions are performed in patients with severe myelopathy and OPLL.

Most patients improve with steroids and MAP augmentation [6,7]. However, several reports describe urgent posterior decompression as salvage in refractory cases, sometimes with dramatic recovery [8,9].

- Gap in Knowledge: Prior reviews, largely before 2023, lack integration of recent cases and do not define criteria for escalation from medical therapy to surgical intervention.
- Objective: To synthesize evidence (2010–2025), highlight controversies, and propose a pragmatic management algorithm for WCS after cervical decompression.

II. METHODS

A targeted search of PubMed, Scopus, and Embase was conducted (2010–2025) using the keywords: “white cord syndrome”, “reperfusion injury”, “cervical decompression”, “anterior cervical discectomy and fusion”, “laminectomy”, and “laminoplasty”.

- Inclusion Criteria: English-language case reports, series, and systematic reviews describing postoperative WCS with MRI confirmation.
- Exclusion Criteria: Neurological deterioration explained by hematoma, graft displacement, or infection.
- Data Extracted: patient demographics, procedure type, timing of deterioration, MRI findings, management (medical vs surgical), and outcomes. Given heterogeneity and rarity, data were synthesized narratively.

III. DISCUSSION

➤ Pathophysiology

WCS is best explained by ischemia–reperfusion injury. Chronic cord compression results in hypoperfusion, endothelial dysfunction, and metabolic adaptation. Rapid decompression triggers oxidative stress, inflammatory infiltration, blood–spinal cord barrier disruption, and neuronal apoptosis [2,3].

• Risk Factors:

- ✓ Preoperative intramedullary T2 signal change (marker of chronic injury).
- ✓ Multilevel stenosis or OPLL [5,6].
- ✓ Older age and vascular comorbidities [6].

➤ Clinical Spectrum

- Timing: Most cases present immediately post-op, though delayed onset up to 7 days has been reported [10].
- Severity: Ranges from mild weakness to complete tetraplegia [1,4].
- Imaging: New or expanded intramedullary T2 hyperintensity extending beyond surgical site [7].

➤ Medical Therapy: First-Line

• Steroids

- ✓ Most cases used methylprednisolone, based on spinal cord injury protocols [4–6].
- ✓ Aim: reduce lipid peroxidation and secondary injury.
- ✓ Controversy: evidence remains anecdotal; no RCTs exist.

• MAP Augmentation

- ✓ MAP >85–90 mmHg for 5–7 days recommended [7].
- ✓ Derived from traumatic SCI guidelines.

• Supportive Neuroprotection

- ✓ Oxygenation, normothermia, strict glucose control.

• Adjunctive Therapies

- ✓ Edaravone (free-radical scavenger) has been reported in Japan/Korea [8].
- ✓ Hypothermia and other neuroprotectants remain experimental.

➤ Posterior Decompression: Rescue

~25–30% of reported WCS cases underwent posterior decompression (laminectomy/laminoplasty) [5].

• Indications:

- ✓ Persistent or worsening deficits despite steroids + MAP.
- ✓ Multilevel stenosis/OPLL with inadequate anterior decompression.
- ✓ MRI suggesting residual cord compromise.

• Timing:

- ✓ Early posterior decompression (within 24–72 h) associated with better recovery [9].
- ✓ Delayed intervention often yields incomplete recovery.

➤ Controversies

- Steroid efficacy: Widely used but unproven.
- MAP augmentation: Duration varies from 24 h to 7 days.
- Posterior decompression timing: No consensus — immediate vs delayed.
- Adjunct therapies: Edaravone, hypothermia, antioxidants require trials.
- Prognosis: Outcomes variable — from full recovery [1] to persistent tetraplegia [10]. Predictors remain unclear.

➤ Proposed Algorithm

(Figure 1: deterioration → urgent MRI → exclude hematoma → steroids + MAP → reassess → if no improvement/progression in 24–48 h, especially with OPLL/multilevel stenosis → posterior decompression).

➤ Clinical Take-Home Messages

- Urgent MRI is mandatory for new neurological deficits.
- WCS diagnosis = T2 hyperintensity without compressive cause.
- First-line: steroids + MAP $\geq$ 85 mmHg.
- Most single-level ACDF patients recover medically.
- Consider posterior decompression in refractory/progressive cases.
- Early rescue surgery is better than delayed.

- Future need: multicenter registries and advanced imaging biomarkers.

IV. CONCLUSION

WCS is a rare but increasingly recognized complication of cervical decompression. Medical therapy (steroids + MAP) remains the mainstay, but urgent posterior decompression should be considered in selected patients with refractory deficits, multilevel stenosis, or OPLL. Until prospective data emerge, individualized decision-making guided by clinical course and imaging remains essential.

Table 1 Summary of Reported WCS Cases (2013–2025)

Author (Year)	Procedure	Onset of WCS	Initial Management	Additional Surgery	Outcome
Chin et al. (2013) [1]	Single-level ACDF	Immediate post-op	Steroids + MAP	No	Gradual neurological recovery
Busack et al. (2020) [4]	ACDF (C5–C6)	Immediate post-op	Steroids + MAP	No	Transient tetraplegia; recovery by 6 weeks
Lei et al. (2022) [10]	ACDF (C4–C6)	Post-op day 7 (delayed)	Steroids → persistent deficit	Yes (posterior laminectomy)	Gradual recovery over months
Dahapute et al. (2022) [9]	Posterior decompression (laminectomy)	Immediate post-op	Steroids	Yes (laminoplasty in refractory cases)	Partial recovery by 14 days
Bagherzadeh et al. (2024) [5]	Systematic review (26 cases pooled)	Immediate–delayed	Steroids ± MAP	~25–30% underwent posterior decompression	Variable outcomes (better if early surgery)
Buciuc et al. (2025) [6]	Case series, cervical surgery	Immediate/delayed	Steroids + MAP	Some required additional decompression	Outcomes heterogeneous
Epstein & Agulnick (2025) [7]	Review (2013–2020 cases)	Immediate–delayed	Steroids ± MAP	Several underwent posterior decompression	Heterogeneous outcomes; timing critical

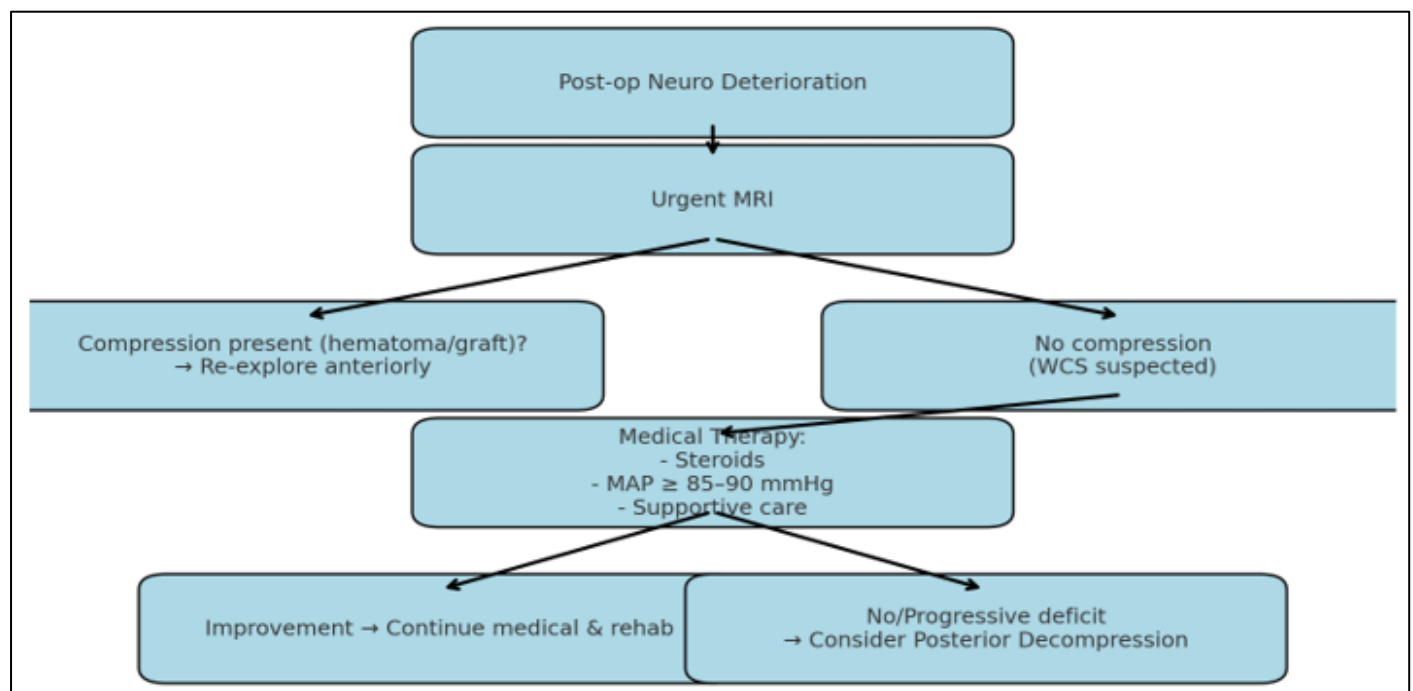


Fig 1 Proposed Algorithm for WCS Management

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