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White cord syndrome—an unforeseen complication and diagnosis of exclusion: a case report and review of management

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Abstract

Background White cord syndrome is an unexpected and dreaded complication of decompression surgery in cervical myelopathy with an unforeseeable prognosis. Ischemic reperfusion injury has been advocated as an underlying pathophysiology. Not only must the surgeon be acquainted with this complication, but the patient should also be counseled before surgery regarding the expected outcome and worsening of neurological status after surgery in severe chronic compressive myelopathy.

Case presentation A 48-year-old female presented with difficulty walking and numbness in all four limbs. The clinical and radiological assessment was suggestive of multilevel cervical cord compression. Posterior cervical decompression with C3–C7 lateral mass screw was performed. The patient developed a worsening neurological status in the immediate postoperative period. The patient was shifted to the intensive care unit and managed with high-dose dexamethasone along with monitoring and maintaining mean arterial pressure above 85 mm Hg. A partial improvement in neurology was noted during the in-hospital stay. The patient was attached to the local rehabilitation clinic. At the one-year follow-up, the patient was ambulatory with some residual numbness and weakness in the upper limbs.

Conclusions White cord syndrome following spinal decompression in chronic cervical myelopathy is a rare, unfortunate complication and a diagnosis of exclusion with variable outcomes. An increase in signal hyper-intensity on T2-weighted MRI imaging in this entity is the most consistent finding. This report reviewed our current knowledge on the management of white cord syndrome based on our present experience.

Keywords White cord syndrome, Cervical myelopathy, Surgical decompression, Reperfusion injury, Case report

Background

White cord syndrome (WCS) is referred to as hyperintense T2-weighted signal intensity on MRI and is observed in cases of ischemic reperfusion injury following spinal cord decompression surgery. It clinically manifests as a transient neurological deficit that is not attributable to any direct or indirect injury to the spinal cord [1]. There are only a few studies that elucidate this unusual condition, which affects patients with neurological deficits after spinal decompression. In comparison with cervical decompression surgery, thoracic posterior decompression has a greater documented rate of ischemic reperfusion injury [2, 3]. Although the mechanism of the injury is not fully understood, it has been hypothesized that increased blood flow after decompression surgery disrupts the blood-spinal cord barrier [4]. The rise in oxygen-derived free radicals and pro-inflammatory cytokines like tumor necrosis factor-α (TNF-α) and interleukin-β (IL-β) with lipid peroxidation

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is thought to mediate the neuronal damage in ischemic reperfusion injury [5]. None of the various suggested interventions for treating ischemia reperfusion injury, including tapering doses of methylprednisolone, maintaining MAP > 85 mm of Hg, and antioxidants, have yielded immutable outcomes [6]. We hereby present our experience with a patient of white cord syndrome with quadriparesis and review the literature to understand and expand our knowledge regarding pathophysiology, risk factors, and management strategies for this devastating perioperative complication.

Case presentation

A 48-year-old female patient presented with difficulty walking and numbness in all four limbs for one year. The patient was not diabetic or hypertensive and had grade II obesity (BMI 35). She had Asia Impairment Scale (AIS) motor power of 4/5 in both upper and lower limbs. She had reduced sensation below the C7 dermatome. Deep tendon reflexes were brisk. Hoffmann's sign and plantar reflex were suggestive of an upper motor neuron lesion. A cervical spine radiograph was suggestive of degenerative changes with loss of cervical lordosis. Magnetic resonance imaging (MRI) indicated multilevel cervical cord compression from C3–C6 (Fig. 1). A clinical diagnosis of Nurick grade III cervical myelopathy was made. The Japanese Orthopedic Association cervical myelopathy score was 9. Patient had no improvement with conservative treatment and neurological deficit progressed over the months. This patient had multilevel degenerative cervical myelopathy, necessitating posterior cervical laminectomy and fixation from C3 to C7.

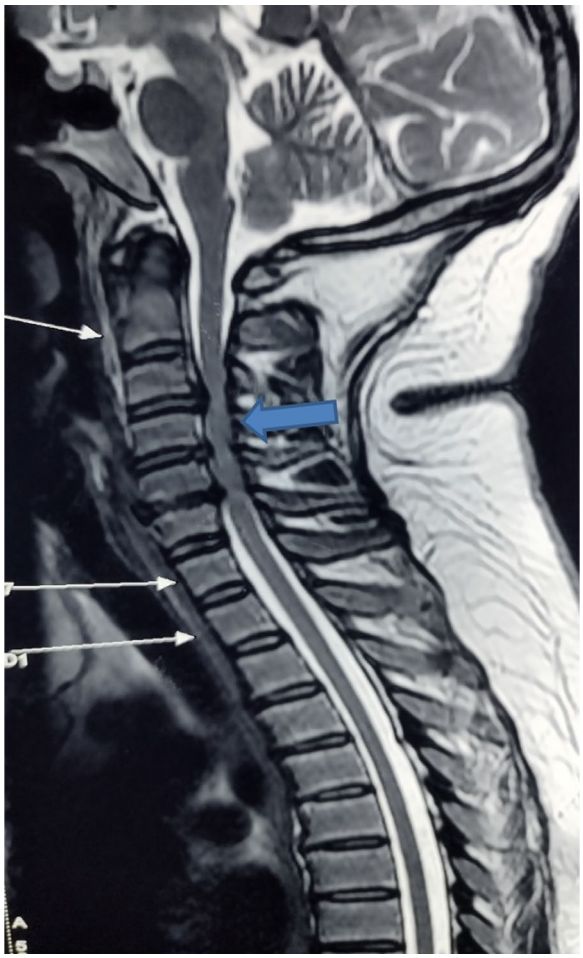

Fig. 1 Preoperative T2-weighted sagittal MRI image suggestive of severe cervical cord compression at C3-C6 with preoperative cord changes, marked by blue arrow

Under general anesthesia, the patient was turned prone, and a posterior cervical midline skin incision was made. Posterior cervical laminectomy was performed for spinal decompression along with lateral mass screw fixation from C3 to C7 (Fig. 2). The mean arterial pressure (MAP) of 70 to 80 mm Hg was maintained during the entire surgery, but at the time of surgical wound washing, the MAP dropped to 45. Intraoperative hypotension is one of the potential causes of neurophysiological abnormalities during spine surgery that could be indicative of spinal cord injury. Dexamethasone plays a role in acute spinal cord injury. In light of these, we administered a stat dose of dexamethasone following the fall of MAP; the rest of the surgery was uneventful. The total duration of surgery was around 70 min, and blood loss was 100 ml. The patient was shifted to the intensive care unit (ICU) in the immediate postoperative period and propped up.

After one hour of surgery, the patient was reexamined; she had quadriplegia with motor power AIS grade 0/5 in all four limbs. Sensations decreased below C6. Injection of dexamethasone started at a tapering dose, and MAP was maintained above 85 mm Hg via the arterial line. After 4 h of surgery, the patient regained partial improvement; motor power was AIS grade 2/5 in both upper limbs and 3/5 in bilateral lower limbs. The patient was shifted for cervical and brain MRI after approximately 6 h of surgery. High signal intensity was noted on T2-weighted imaging in cervical MRI, suggesting white cord syndrome (Fig. 3). The patient was managed with physiotherapy and tapering steroid dosages during the hospital stay and attached to a rehabilitation center after discharge from the hospital. At three months of follow-up, the neurological status remained the same. At one-year follow-up, the patient was able to walk with support with residual weakness and paresthesia in the upper limbs.

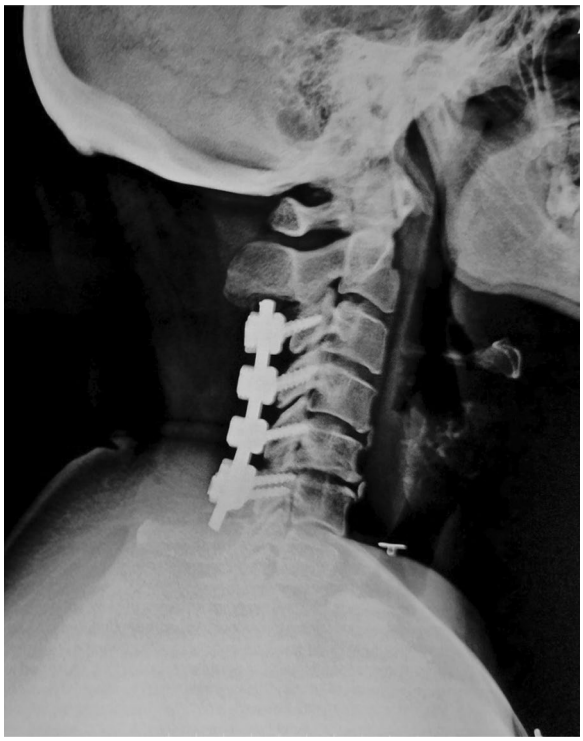

Fig. 2 Intra-operative imaging showing lateral screw fixation from C3–C7 with well-aligned implant pointed by blue arrow

Discussion

The emergence of postoperative neurological deficits in spine surgery is not very common. Epidural hematoma, vascular compromise, graft dislodgment, iatrogenic cord injury, and incomplete decompression have been enumerated as possible causes of neurological deficits [7]. An indecipherable neurological deficit thought to be due to ischemic reperfusion injury was initially reported by Chin KR et al. in 2013 in a 59-year-old male after anterior cervical decompression and named it "White Cord Syndrome," alluding to the characteristic T2 hyperintensity seen on MRI [1]. Few reports have been published after it, citing neurological deficits owing to white cord syndrome after both anterior and posterior cervical decompression in long-term severe spinal stenosis [4, 8]. Most of the reports have documented WCS after cervical spine decompression surgery, as in the present case, but cases have also been reported after thoracic decompression [9]. Sudden spinal cord expansion after a rush of blood in decompression surgery culminates in ischemic reperfusion injury; the pathophysiology is thought to be a breach in the blood-spinal cord barrier and extravasation of cytokines like TNF to its saturation level [1, 10]. Wu et al., using an experimental rat model postulated the potential role of free radicals and lipid peroxidation-mediated neuronal injury in chronic severe spinal cord compression. They proposed mitochondrial-dependent apoptosis, inflammatory reactions, and phospholipid signaling cascades to possibly have a role in white cord syndrome [11]. Micro-thrombi interrupting vascular supply in the watershed region of the thoracic spine and cord recoil have been suggested as other possible causes of transitory neural deficits after thoracic decompression [12].

Similar to the present report, previously mentioned cases developed neurological deficits either during surgery or in the early postoperative period, but late presentations have also been reported [13], although the degree of neurological deficit differed among different cases. The appearance of new or enlarging T2 signal hyper-intensity on magnetic resonance imaging is a widely accepted radiological sign of this entity, which got its name "White Cord Syndrome" [8, 9]. This must be differentiated from preexisting neuronal demyelination, which gives an identical MRI picture [1]. Kalb et al. reported five patients of cervical spinal cord infarction after cervical spine decompressive surgery, who had acute onset neurological deficit in postoperative period. Although the clinicoradiological profile of these patients was similar to that of "White Cord syndrome," most of them had preoperative vascular compromise or hypotension [14]. There is no such protocol in the management of ischemic reperfusion injury owing to the scanty literature and the absence of good-quality studies. Steroids are known to reduce oxidative stress and thus have a role in the management of ischemic reperfusion injury in conjunction with physical rehabilitation, but no coherence has been seen among various case studies in view of dose and duration of steroid use, which varied from the NASCIS III protocol to tapering doses [1, 4, 8–13]. Mean arterial pressure (MAP) has been thought to affect the outcome of acute spinal cord injuries and is proposed to be kept above 85–90 mm Hg for 5–7 days for a better outcome [15]. On a similar principle, MAP management has been followed by some authors setting values above 85 mm of Hg for around a week, but this is not unequivocal [16]. The role of potent antioxidants in the management of white cord syndrome by scavenging oxygen free radicals is limited by their clinical utility [12, 17]. Previous research has suggested certain preventive measures to lessen the incidence of white cord syndrome, including the use of propofol, remote ischemic preconditioning, and intraoperative monitoring of motor and sensory evoked potentials. Monitoring intraoperative motor and sensory evoked potentials warns of any direct or ischemic injury and compression. Remote ischemic preconditioning protects the neural tissue from ischemic damage by lowering the levels of neuron-specific enolase and S-100B. Propofol diminishes the expression of nuclear factor-κB, thus reducing the

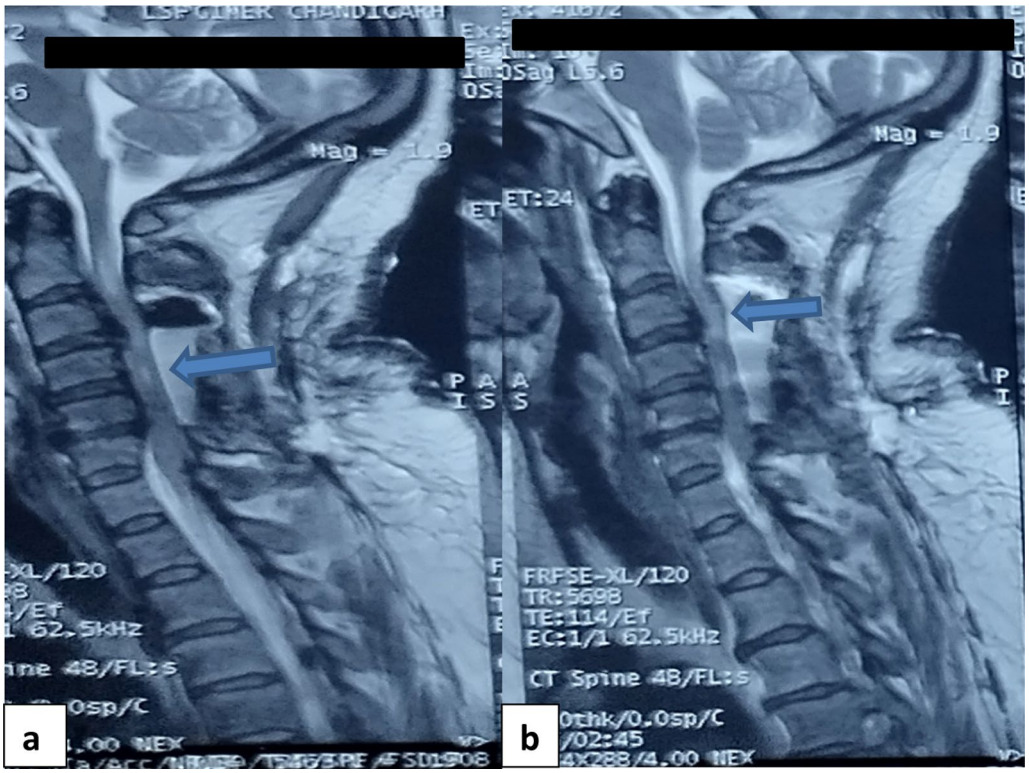

Fig. 3 **a** Postoperative T2-weighted sagittal MRI image showing cervical decompression, pointed by a blue arrow **b** Postoperative T2-weighted mid-sagittal MRI image showing cervical decompression and linear signal changes in the cord suggestive of ischemic reperfusion injury, indicated by the blue arrow

permeability of the blood–brain barrier and providing neuroprotection [18, 19]. It has been observed that neurological deficits show an early partial improvement followed by a steady phase before any improvement is seen. We have a similar observation in our case, where motor strength goes one or two grades up and stays the same till six month follow-up [11–13, 20–24]. At one-year follow-up, the patient was ambulatory with some residual weakness in the upper limbs.

Conclusions

In this review, we have provided an overview of the current literature on white cord syndrome and its management based on our experience. As only a few cases of white cord syndrome have been mentioned in the literature; the explicit mechanism of injury, associated risk factors, and management strategies are open to debate and discussion. Frequently employed modalities of treatment like dexamethasone and MAP maintenance combined with physical therapy have had varying degrees of success. The majority of studies have reported an improvement in motor function after a certain period of time, although this improvement is erratic and unpredictable. Further high-quality studies with more cases are imperative to anticipate this unexplained neurological deficit in vulnerable populations so that proper patient counseling and the preoperative deterrent measure can be taken.

Abbreviations

WCS	White cord syndrome
TNF *α*	Tumor necrosis factor-*α*
IL-*β*	Interleukin-*β*
MRI	Magnetic resonance imaging
BMI	Basal mass index
AIS	ASIA impairment scale
MAP	Mean arterial pressure
ICU	Intensive care unit

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Author contributions

VK and SD analyzed and interpreted the patient data. AR and VK drafted the manuscript. All authors read and approved the final manuscript.

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Availability of data and materials

All data generated or analyzed during this study are included in this article.

Declarations

Ethics approval and consent to participate
Ethical approval not applicable. An informed consent was obtained from the patient to participate in this study.

Consent for publication
An informed consent for publication of this case report was obtained from the patient and attached to her case file.

Competing interests
The authors declare that they have no competing interests.

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