



## Case Report

# White cord syndrome following posterior decompression and fusion for severe OPLL and an acute traumatic cervical injury – A case report and review of literature

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## ABSTRACT

**Background:** White cord syndrome (WCS) refers to the observation of intramedullary hyperintensity due to edema/ischemia and swelling on postoperative T2-weighted MRI sequences in the setting of unexplained neurological deficits after cervical spinal cord decompression. Pathophysiologically, WCS/reperfusion injury (RPI) occurs due to oxygen derived free radicals as a result of acute reperfusion or direct trauma from blood flow itself. Intraoperative neurophysiologic monitoring (IONM) can give early warning and detect neurologic deficits. Here, we are presenting a case of a patient who had a chronic severe ossification of posterior longitudinal ligament (OPLL) of cervical cord, underwent decompressive surgery, and developed quadriplegia postoperatively without any perceptible iatrogenic cord trauma, documented by IONM and postoperative MRI with classical signs of WCS.

**Case Description:** A 63-year-old male presented with low velocity fall at home followed by quadriparesis. X-ray images on presentation showed C6 fracture and local kyphosis. MRI images showed that there is marked spinal canal stenosis from C2 down to C4 due to OPLL with intrinsic signal changes in the cord. On decompression, motor-evoked potential signals were not present below C4. Immediate postoperative MRI was done to rule out any compressive pathology. MRI showed T2 hyperintensity of the cord at C3 level with cord edema. No evidence of epidural hematoma or other compressive lesion was found and the diagnosis of WCS/RPI was established.

**Conclusion:** WCS is essentially a diagnosis of exclusion. Very rarely, patients sustain severe/new neurological deficits postoperatively attributed to WCS. Unless, this is confirmed postoperatively with classical MRI signs of intramedullary hyperintensity, the diagnosis should not be invoked.

**Keywords:** Cervical spine, Diagnosis of exclusion, Ossification of posterior longitudinal ligament, Reperfusion injury of cord, White cord syndrome

## INTRODUCTION

Posterior cervical decompression and fusion (PCDF) is a frequently performed technique for cervical spondylotic myelopathy (CSM). New neurologic deficits are rare complications following spine surgery, occurring at a rate <1%.<sup>[1,2]</sup> Epidural hematoma, inadequate/residual compression, and acute ischemia from hypoperfusion are the most common causes of neurologic complications, apart from implant and instrument related cord injury following posterior decompression for

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CSM. Very rarely, infection with abscess, cerebrovascular insult, or a flare of an undiagnosed demyelinating condition may be the causes.

White cord syndrome (WCS) has been described as neurodeficit that cannot be explained by these things, which can be caused by an acute reperfusion injury (RPI) of the spinal cord. The so called “WCS” is usually a diagnosis of exclusion as it can be diagnosed only if all the other etiologies have been ruled out. Iatrogenic and postoperative compressive pathologies are still the leading causes of neurological deficits postoperatively. Spinal cord RPI is a rare complication and only few cases are reported in the literature. First described by Chin *et al.*,<sup>[4]</sup> WCS refers to the observation of intramedullary hyperintensity due to edema/ischemia and swelling on postoperative T2-weighted MRI sequences in the setting of unexplained neurological deficits after cervical spinal cord decompression.

Ischemia-RPI typically occurs after a cervical decompression surgery, where a chronically compressed spinal cord is injured by reperfusion following decompression. Such an injury occurs due to acute restoration of blood flow to the previously ischemic spinal cord due to long standing compression like ossification of posterior longitudinal ligament (OPLL) in cervical spine. This can occur in various cervical spine surgeries, both anterior and posterior approaches such as anterior cervical decompression and fusion (ACDF), multiple laminectomies, or corpectomy to relieve chronically compressed spinal cord. Pathophysiologically, WCS/RPI occurs due to oxygen-derived free radicals as a result of acute reperfusion or direct trauma from blood flow itself. This, in turn, gives rise to a cascade of reactions such as lipid peroxidation of neuronal membrane and change of architecture of spinal cord resulting in secondary injury.<sup>[5]</sup> Intraoperative neurophysiologic monitoring (IONM) can give early warning and detect neurologic deficits which can allow surgeon to take necessary measures.

Here, we are presenting a case of a patient who had a chronic severe OPLL of cervical cord who underwent decompressive surgery and developed quadriplegia postoperatively without any perceptible iatrogenic technical cord trauma, documented by IONM and postoperative MRI with classical signs of WCS.

## CASE PRESENTATION

A 63-year-old male presented to us with low velocity fall at home followed by quadriparesis. On examination, he had upper limb weakness more than lower limb (Right upper limb power 1/5, the left upper limb power 2/5, and power in both lower limbs 3/5). Sensation to catheter tug presents with intact perianal sensation. The hand dexterity was poor in both upper limbs with the upper motor neuron signs in

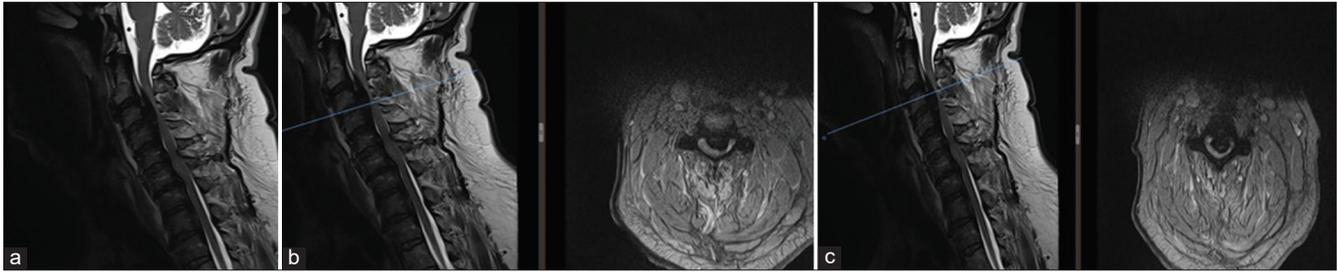
bilateral upper and lower limbs. ATLS protocol was followed when the patient arrived to the emergency department. Cervical spine was immobilized and patient was sent for further investigations like CT scan of whole spine and brain with MRI scan of cervical spine.

The CT scan demonstrated C6 fracture with ossification of the posterior longitudinal ligament in addition to diffuse idiopathic skeletal hyperostosis with anterior osteophytosis. CT picture showed florid OPLL from C2 to C4 with horizontal fracture through the entire width of the C6 vertebral body which extended posteriorly to bilateral facet joints without any dislocation/displacement. There is also calcification of the longitudinal ligament all the way down to T3. However, it was particularly severe from C2 to C4. There was also suggestion of fracture of the ossified PLL at C3 level [Figures 1a-c]. MRI images showed that there is marked spinal canal stenosis from C2 down to C4 due to ossified posterior longitudinal ligament with intrinsic signal changes in the cord. There were also hyperintense fluid signal changes at C2/3 level s/o fracture of PLL at that level. There was also edema of posterior elements and muscles s/o recent fall and injury with evidence of C6 vertebral body fractures [Figures 2a-c].

The patient was initially placed in a cervicothoracic brace for temporary stabilization. The patient’s neurology improved



**Figure 1:** CT images demonstrating C6 fracture with ossification of the posterior longitudinal ligament in addition to diffuse idiopathic skeletal hyperostosis with anterior osteophytosis. There is calcification of the longitudinal ligament from C2/3 down to T3. (a) Sagittal, (b) axial at C3 with island of ossification of posterior longitudinal ligament, and (c) CT scan axial at various levels from C2/3 till C6 #: CT scan axial at various levels from C2/3 till C6.



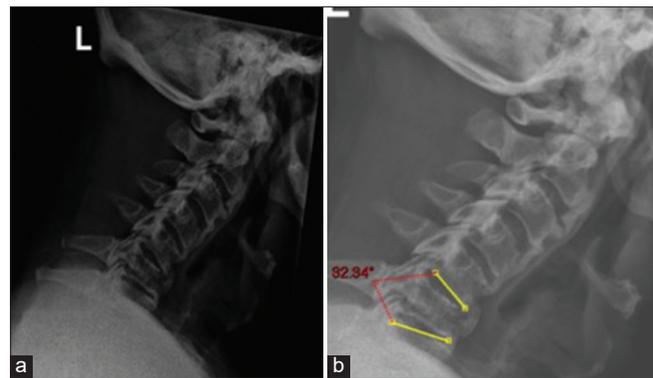
**Figure 2:** MRI scan images showing that there is marked spinal canal stenosis from C2 down to C4 due to ossified posterior longitudinal ligament with intrinsic signal changes in the cord. (a) Sagittal scan of cervicothoracic spine, (b) images showing severe cord compression at C3/4 levels, and (c) at C2/3 level. The blue bar in figures 2b and 2c indicates the axial level. 2b axial at C3/4 disc level and 2c axial at C3 vertebral body level. This also corresponds to the area of maximal stenosis and cord compression.

after the initial injury, and hence, the decision was made to continue the brace to allow his neurology to improve further and settle at a baseline after multidisciplinary team (MDT) discussion. The plan was to operate him on an elective basis after 7–10 days to allow for complete assessment. However, subsequent standing AP and lateral X-rays showed 32° of local kyphosis without brace with reduction in the width of C6 body through the fracture line [Figures 3a and b]. Due to progressive kyphosis which was suggestive of instability and concern that patient may require a much bigger surgery if fracture gets fused in kyphosis, it was decided to intervene surgically at that point of time.

The plan was to proceed with a posterior cervical decompression from C2 to C7 and instrumented fusion from C2 to T2 under navigation and neuromonitoring. The case was discussed in the local MDT and there was overall agreement about the decision to proceed with surgery and the surgical plan. Despite the fact in the UK use of spinal cord monitoring in cervical myelopathy is not commonly used, we had recognized that the risks were significantly higher in the presence of severe pre-existing OPLL and an unstable fracture in this particular case.

Intraoperative monitoring (IONM) was undertaken using a 32 channels and multimodal XLTEK Protektor intraoperative monitoring recording system. Intraoperative recording modalities consisted of transcranial motor-evoked potentials (MEPs), free running electromyography, and upper limb and lower limb somatosensory-evoked potentials (SSEPs). SSEPs were recorded for all four limbs using stainless steel corkscrew electrodes supine MEP baselines that were recorded before the patient was transferred to the surgical table. The patient was transferred to a prone position and MEP responses repeated and set as baseline.

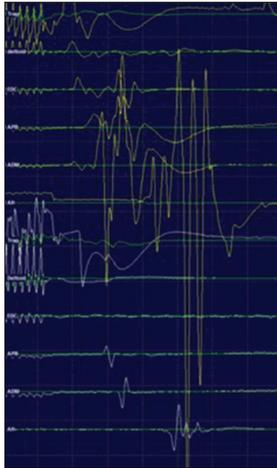
Transcranial MEP amplitudes remained stable during placement of screws from C2 to T2 under navigation. Posterior cervical decompression was carried out with bone cuts using bone scalpel for laminectomy. During decompression, after the laminectomy at C3/4 was completed, transient amplitude changes were noted for the right extensor



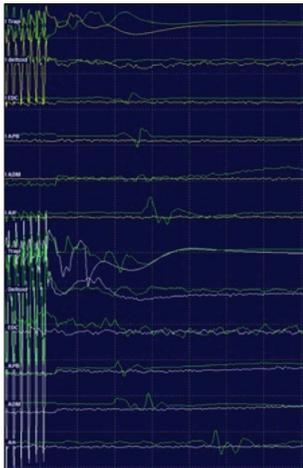
**Figure 3:** (a and b) Standing X-ray 10 days after admission showing progressive kyphosis of 32° at C5/6 level. The yellow line in the figure indicates the lower end plate of superior and upper end plate of inferior vertebral bodies to measure Cobb's Angle.

digitorum communis (EDC), right abductor pollicis brevis (APB), and right abductor digiti minimi (ADM). When the lamina was removed, MEP responses initially augmented for the right trapezius, right deltoid, right EDC, right APB, right ADM, and right abductor hallucis [Figure 4] in association with an augmented body jerk from the patient. An alert was given to the surgeon and the patient retested four min later. On retest, MEPs were abolished below spinal level C4 [Figure 5] with preservation of trapezius MEPs.

The spinal cord at risk protocol was initiated. A bolus of the decompression was extended higher and lowers including the whole of posterior elements of C2–C7. The mean arterial pressure (MAP) was kept above 85 mmHg for the whole procedure. To assess for any ongoing compression and hardware malposition, we performed an intraoperative CT which showed that the decompression was satisfactory and no evidence of spinal subluxation or screw misplacement [Figure 6]. The left C7 screw looked to breach the medial wall and was removed. After rod placement, AP fluoroscopy confirmed adequate metalwork placement. Intermittent MEP monitoring was performed every 5 min using maximum stimulation parameters (500 Hz, train of 6 pulses, 0.05 ms,



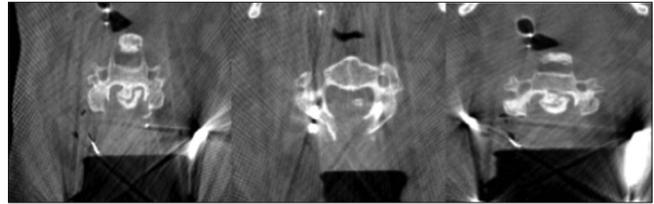
**Figure 4:** Augmented motor-evoked potentials from the right (yellow) cross-over noted to the left (white).



**Figure 5:** Motor-evoked potential (MEP) abolished below C4, MEPs remained present bilaterally from trapezius, right (yellow), left (white) and abolished below from deltoid, extensor digitorum communis, abductor pollicis brevis, adductor digiti minimi, and abductor hallucis. Prone baseline MEPs are displayed in green.

and 500 volts). Persistent spontaneous prolonged discharges were noted bilaterally from trapezius post lamina removal and the surgeon was informed.

Five minutes from losing MEPs, the right upper limb SSEPs showed a 50% decrease in amplitude from baseline values



**Figure 6:** Intraoperative CT scan images showing satisfactory wide decompression of cord at various levels.

with an increase in the sensory threshold (50 mA, 1.0 ms). The left upper limb SSEPs remained with intraoperative baseline values. Bilateral lower limb SSEPs remained stable and within intraoperative baseline values. MEPs remained absent below trapezius during rod placement.

Following the placement of rods and completion of the surgical procedure, MEPs remained absent below trapezius. The right upper limb SSEP showed minimal N20 amplitude reduction (<50%) from intraoperative baseline values, the left upper limb SSEPs were within baseline values, the right lower limb SSEPs remained within intraoperative baseline values, and the left lower limb SSEPs showed a reduction in P37 amplitude of 50% from intraoperative baseline values.

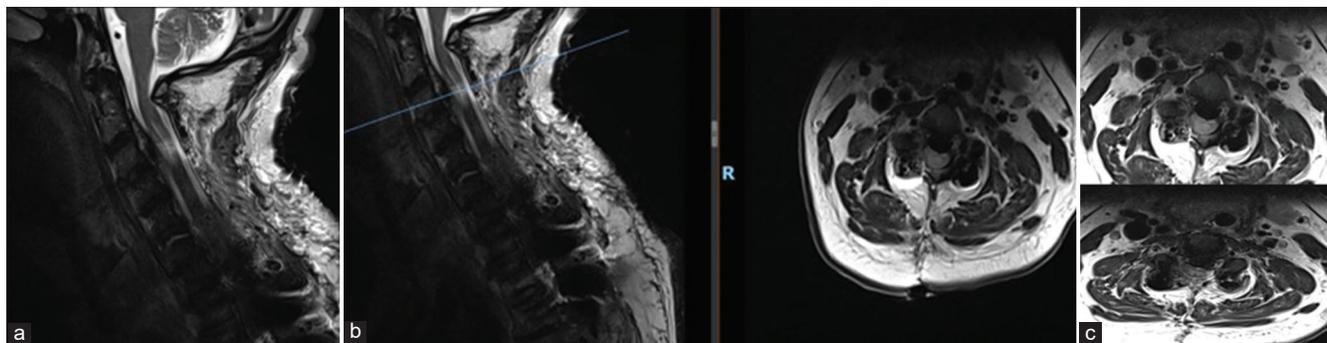
The patient was transferred to ITU intubated with ventilator support, MAP >85, and to continue dexamethasone 8MG twice daily. Postoperatively, he had quadriplegia with C5 sensory level. Immediate postoperative MRI was done to rule out any compressive pathology.

MRI showed T2 intramedullary hyperintensity of the cord at C3 level which appeared expanded with a central syrinx. Cord edema was noted at the C3 level. No evidence of epidural hematoma or other compressive lesion was noticeable at the operative site [Figures 7a-c] and the diagnosis of WCS/RPI was established based on postoperative MRI findings as there were no other discernible causes of Quadriplegia.

Steroids were tapered over a period of 1 week. Tracheostomy was performed and gradual weaning from ventilator support was started. The patient was transferred to inpatient rehabilitation center 6 weeks after surgery with still no improvement in motor strength.

## DISCUSSION AND REVIEW OF LITERATURE

WCS is a rare complication of surgery of the spinal cord. This ischemic edematous lesion of the cord is attributed to injury due to immediate reperfusion of the newly-decompressed cord intra- or postoperatively. In the literature, we could find very few cases of WCS/RPI following anterior/posterior cervical decompressive surgeries. The immediate postoperative MRI findings in all such cases were consistent with the RPI. All such case reports/series involved similar postoperative clinical pictures and MRI images as above.



**Figure 7:** Immediate postoperative MRI was done to rule out any compressive pathology. (a) MRI showed T2 intramedullary hyperintensity of the cord at C3 level which appeared expanded with a central syrinx. (b) Cord edema was noted at the C3 level. No evidence of epidural hematoma or other compressive lesion at the operative site indicating reperfusion injury. The blue bar in the figure indicates the axial images with maximal intramedullary hyperintensity of the cord and the reperfusion injury which corresponds to C3 level. (c) Axial at C3 and C3/4 Levels showing whitening of the cord (White Cord Syndrome).

Chin *et al.*<sup>[4]</sup> reported 1<sup>st</sup> case of WCS after C45 C56 ACDF. There was loss of MEP intraoperatively and patient woke up with incomplete quadriplegia. Postoperative MRI was suggestive of residual compression behind C5 body compressing spinal cord. Hence, the patient underwent urgent surgery for C5 corpectomy with fusion with intraoperative IV hydrocortisone infusion and an acute spinal cord injury steroid protocol with IV dexamethasone taper and acute inpatient rehabilitation postoperatively. There was partial improvement after 16 months postoperatively. However, in this case, there was MR documented residual compression for postoperative neurodeficit which rules out RPI/WCS.

Giammalva *et al.*<sup>[6]</sup> reported the second case of WCS. It was a case of C3/4 C5/6 ACDF for disk herniation with C4–6 severe cord compression in a 64-year-old man. The patient developed acute tetraparesis after an uncomplicated surgery. The patient was immediately started on a high-dose methylprednisolone (National Acute Spinal Cord Injury Studies III protocol). There was partial recovery after 3 days of surgery.

Bayley *et al.*<sup>[2]</sup> performed C67 ACDF in a 30-year-old male with large C67 disk herniation who presented with the left leg weakness and left upper limb numbness. C67 ACDF was performed and the patient developed quadriplegia immediately after surgery. MRI done immediately postoperatively was suggestive of ongoing compression with marked intramedullary hyperintensity/cord swelling at the C6–C7 level. Hence, 2<sup>nd</sup> surgery (posterior laminectomy and fusion) was performed to decompress the cord. Patient neurology started improving 48 h after 2<sup>nd</sup> surgery and normal function was regained after 3 months. However, similar to the case of Chin *et al.*, in this case, there was MR documented residual compression for postoperative neurodeficit which rules out RPI/WCS. However, postoperative deficits in two patients who were attributed to MR-documented extrinsic

disease (respectively, stenosis, and OPLL) which required another surgery, therefore cannot be attributable to RPI/WCS.

Jun *et al.*<sup>[7]</sup> presented a case of WCS in a 49-year-old woman with C67 herniated disk, for which C67 ACDF was performed. Immediately after the operation, physical examination revealed paraplegia and emergency MRI showed T2 high-signal myelopathy suspected as RPI at C6–7 level, and emergency surgery (C4–7 laminoplasty) was performed under diagnosis of WCS. Two weeks after 2<sup>nd</sup> surgery, motor power and sensory deficit of bilateral lower extremity were fully recovered.

Antwi *et al.*<sup>[1]</sup> reported the 1<sup>st</sup> case of WCS after posterior cervical surgery in a 68-year-old man affected by C4–6 severe cervical stenosis, who underwent C4–7 posterior decompression surgery and fusion. SSEPs and MEPs were monitored throughout the procedure. There were no concerns throughout the surgical procedure until closure of the wound when there was loss of MEPs in the left hand and leg. After the loss of MEPs, the patient was started on 100% oxygen and his MAPs were maintained above 113–115 mmHg to ensure adequate perfusion of the spinal cord. The wound was reopened and explored to exclude expanding hematoma, CSF leak. After awakening, the patient had left sided hemiplegia. The MRI T2-weighted scans showed an increased hyperintense signal at the left aspect of the spinal cord at the C5–C6 levels concerning for acute spinal cord ischemia. The patient was started on a high-dose steroid protocol (IV solumedrol infusion for 24 h followed by a dexamethasone taper). The patient improved in the subsequent days and ambulating with a walker by postoperative day 3.

Busack and Eagleton<sup>[3]</sup> reported a case of 63-year-old male with cervical myelopathy (Nurick grade 3) and severe cervical stenosis from C2–C3 to C5–C6 with associated hyperintense intramedullary signal change at C5–C6 planned for a C3–C6 laminectomy and C2–T1 posterior fusion. During surgery

after the laminectomies were completed, there was loss of MEPs and diminished SSEPs in the bilateral upper and lower extremities. Postoperatively, the patient had lack of sensation below the T3 level and 0/5 motor strength of all muscle groups except 4/5 strength of bilateral deltoids. No MRI was done postoperatively. The patient was started on IV dexamethasone therapy and completely recovered 30 days postoperative.

Vinodh *et al.*<sup>[11]</sup> reported a patient with cervical stenosis secondary to metastatic tumor in the intradural and extradural compartments with the lower limb paraparesis. She underwent an uneventful tumor excision with PCDF. Postoperatively, she was quadriplegic and postoperative MRI scan showed focal hyperintensity on the T2-weighted image suggestive of spinal cord edema extending into the lower brain stem.

Wiginton *et al.*<sup>[13]</sup> presented a CSM with MRI showing severe cord compression at C1 and partial hyperintense signal. Intraoperatively, after C1 bony decompression, the patient experienced a complete loss of both SSEPs and MEPs with an eventual return to baseline before completing the operation. The patient had acute quadriplegia after surgery and an immediate postoperative MRI revealed a more pronounced hyperintensity in the central cervical cord on T2-weighted sequences. The patient was started on dexamethasone and increased MAP which resulted in the patient regaining full strength over a period of months.

Study reported by Seichi *et al.*<sup>[10]</sup> followed 114 patients with MRI 3 weeks post laminoplasty to look for postoperative abnormal expansion of T2 high-signal intensity areas in the spinal cord. Seven patients (6.1%) had postoperative abnormal expansion of the T2 high-signal intensity area, of which 3 (43%) were asymptomatic. Spinal cord enlargement with abnormal expansion of the T2 high-signal intensity area was strongly related with distal and diffuse type of postoperative paresis of the upper extremity without deterioration of the lower motor function.

Mathkour *et al.*<sup>[9]</sup> described a similar case as our case report. The patient had severe compression of the spinal cord from C3 to C6 with myelopathic changes. He had undergone a posterior cervical decompression and instrumented fusion from C2 to C6 with IONM available intraoperatively. The patient suffered from a RPI with mild decline from his baseline neurology. The patient though regained his neurology with physical therapy and intensive rehabilitation program.

In our case, the patient had a severe cervical cord compression due to OPLL at C2–4 level. There was a documented fall in MEPs and SSEPs few minutes after decompression without any iatrogenic injury to the cord. There was no demonstrable cause which can cause neurodeficit like implant position and adequate decompression was checked with intraoperative O arm. Furthermore, postoperative MRI was done to exclude any residual compression which demonstrated the classical

intramedullary hyperintensity of the cord at the site of maximal cord compression.

Various theories have been postulated for the WCS in the absence of mechanical cord injury, which includes ischemia-RPI, microthrombi, and altered perfusion following decompression. Antwi *et al.* hypothesized that areas of the cord that are chronically compressed can become ischemic from small artery occlusion.<sup>[1]</sup> The most popular cause of injury is the ischemia-RPI that occurs when a chronically compressed cord is decompressed. This allows the return of blood with subsequent damage due to rush-in perfusion of blood, either by direct trauma from blood flow itself or by the oxygen free radicals which damage spinal cord axons through oxidative stress. The damage occurs due to long-term compression which alters the blood-brain barrier (BBB) resulting in influx of oxidative particles and free radicals with subsequent damage and cytokine mediated inflammation which increases the permeability of the blood vessels. Due to long-term compression, both mechanical and ischemic, there is accumulation of excitatory neurotransmitters particularly glutamate which also crosses the BBB.

Another mechanism or the secondary mechanism of injury is due to the lipid peroxidation of the neuronal membrane with mitochondrial mediated apoptosis of the neuronal cells. In rats, an experimental study was made by mechanical compression of the cord for 6 weeks and MRI was performed pre- and post decompression to look for changes in the cord. This study showed that, after decompression of the spinal cord, increased inflammatory markers, tumor necrosis factor  $\alpha$ , and interleukin-1  $\beta$  were observed in conjunction with the reperfusion period.<sup>[8]</sup> Specifically, cord sections of rat had increased 8-oxoG DNA on nuclear staining, a DNA adduct that results from an increase in reactive oxygen species and participates in DNA damage repair.

At present, IONM is not a routine of practice. However, in our case, it did give us an alert as there was drop in MEP and SSEP to which we acted promptly by doing further decompression until C2, increasing MAP, steroids, and performing intraoperative imaging with O-arm to make sure adequate implant position and decompression. Furthermore, the signal changes happened post decompression which effectively rules out implant/instrument related cord injury which can play a role medicolegally as well. Unfortunately, it did not make a difference in prognosis of the patient, but it did give us a timely alert to act promptly.

## CONCLUSION

WCS is essentially a diagnosis of exclusion. Very rarely, patients sustain severe/new neurological deficits postoperatively attributed to WCS. Unless, this is confirmed postoperatively with classical MRI signs, the diagnosis

should not be invoked. IONM can help us detect such an event occurring intraoperatively and help us take appropriate measures in a timely manner. IONM should be the standard of care in such cases of chronic compression and also, it provides a source of permanent record of what exactly happened intraoperatively to prevent medicolegal complications. Understanding WCS/RPI is a valid diagnosis in such circumstances that would be helpful in detailed consenting processes and help prevent an additional decompression surgery for the patient which might, further, increase the risks of mortality and morbidity.

#### Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Nil.

#### Conflicts of interest

There are no conflicts of interest.

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