Case Report

"White Cord Syndrome" of Acute Tetraplegia after Posterior Cervical Decompression and Resulting Hypoxic Brain Injury

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Abstract

White cord syndrome (WCS) is a rare case of severe neurological deterioration after surgical decompression for cervical myelopathy. It was proposed to be secondary to an ischemia/reperfusion injury. An association of WCS with a hypoxic brain injury (HBI) has not been documented. A 63-year-old man presented to us with progressive symptoms of cervical myelopathy. Computed tomography scan and magnetic resonance imaging (MRI) scan findings were suggestive of an ossified posterior longitudinal ligament with cord atrophy and myelomalacia changes. He was managed surgically by decompression and fusion through a posterior approach. During the surgery, there was a sudden loss of neuromonitoring signals after laminectomy, and wake-up assessment revealed neurological deterioration. Immediate postoperative imaging revealed adequately placed screws and adequate cord decompression. A high dose of intravenous steroids was given. Repeat MRI scan on the 3rd postoperative day suggested cord edema over a large area on T2-weighted images. He was diagnosed as WCS and managed conservatively. He had persistent abdominal distension postoperatively, and a diagnostic endoscopy was advised. At the start of the procedure, the patient had a sudden-onset loss of consciousness. Electrocardiogram suggested bradyarrhythmias with hypotension. The patient was resuscitated, intubated, and shifted to intensive care unit. He was diagnosed to have a HBI. He was managed with multidisciplinary rehabilitation and discharged at 4 months' postoperatively with stable vitals. There was no improvement in the neurology or his consciousness. Spine surgeons have to be aware of this potentially disastrous complication of WCS. One should take adequate postoperative care to avoid preventable complications like HBI associated with it.

Keywords: *Cervical myelopathy, complications, decompression, deterioration, neurology, surgery, white cord syndrome*

Introduction

Cervical myelopathy is a common cause of neurological dysfunction in the elderly.[1] Surgical decompression of the cervical spinal cord through an anterior or a posterior approach is an effective treatment option for cervical myelopathy and aims to halt the progression of neurological deficit. Severe spinal cord injury after posterior cervical decompression and fusion is rare, with an incidence of 0.18% documented in the literature, the common causes being epidural hematoma, and inadequate decompression.[2] White cord syndrome (WCS) is a term used to describe the appearance of cord edema over a large area at the surgical site on sagittal T2-weighted magnetic resonance imaging (MRI) images.[3] It is a rare cause of acute onset severe neurological deficit after cervical decompression. It was proposed to be secondary to ischemia/reperfusion injury due to oxygen-derived free radical damage.[3] An association of WCS with hypoxic brain injury (HBI) has not been documented in the literature.

Case Report

A 63-year-old man was referred to our hospital with complaints of numbness in both upper limbs, lower limbs, and trunk for 6 months and gait imbalance for 3 months. The symptoms rapidly progressed in the past 3 months. He had no history of any trauma, and there were no constitutional symptoms. His medical history included diabetes for the past 10 years, which was well-controlled with medications. The family history was insignificant. The patient

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walked with the support of a walker. On examination, there was no neck tenderness, but the movements of the cervical spine were terminally restricted. Muscle power was reduced in both upper limbs. The limbs were hypertonic with signs of an upper motor neuron lesion. The Japanese Orthopaedic Association score was 10 points on a scale of 17 points. MRI and computed tomography (CT) scan of the cervical spine suggested cervical canal stenosis with extensive ossified posterior longitudinal ligament and cord atrophy with myelomalacia changes [Figure 1]. Surgical decompression by laminectomy and fusion by a posterior approach was planned under neurological monitoring guidance.

The patient was positioned prone with appropriate padding of all bony prominences. The Mean arterial pressure was maintained above 90 mm Hg throughout the procedure. A posterior midline approach was used with subperiosteal dissection of the affected region. Pedicle screws were placed at bilateral C2 and T1 levels, and lateral mass screw fixation was done at bilateral C3, C4, and C5 levels. A high-speed burr was used to create troughs bilaterally at the lamina-facet junction, and the laminae removed *en bloc*. There was a complete loss of motor-evoked potentials (MEPs) immediately after the laminectomy. The anesthetist was alerted, and the necessary measures were taken. Still, there was no improvement in the MEP signal. Wake up test was performed, and the patient could not move his limbs. Rods were connected bilaterally, and the wound was closed over a negative suction drain after adequate hemostasis. Except for the loss of the MEP signal, the surgical procedure was uneventful with no cerebrospinal fluid leak, increased blood loss, or cord handling.

The immediate postoperative period assessment revealed complete tetraplegia. Postoperative MRI and CT scan showed appropriately placed screws and adequate fixation of the implant with no evidence of bony compression or epidural hematoma [Figure 2a]. The patient was put on intravenous methylprednisolone according to the National Acute Spinal Cord Injury Study–II protocol. Repeat MRI scan on the 3rd postoperative day was suggestive of a WCS, and he was managed conservatively [Figure 2b]. Since the patient had persistent abdominal distension postoperatively, a diagnostic endoscopy was advised on the 4th postoperative day. At the start of the procedure, the patient had a sudden loss of consciousness with hemodynamic collapse, and electrocardiogram revealed bradyarrhythmias. Resuscitative measures were taken, and the patient intubated. CT and MRI scans of the brain suggested no evidence of infarct or hematoma. He was diagnosed to have a possible HBI. He was managed with multidisciplinary rehabilitation and discharged after 4 months with stable vitals. MRI scan of the cervical spine at the time of discharge suggested resolution of edema [Figure 2c]. There was no improvement in the neurology or his Glasgow Coma Scale score.

Discussion

WCS is a rare entity with four cases (two posterior surgeries and two anterior surgeries) reported in the literature to date.[3-6] Among the two posterior surgeries, there was a loss of MEPs on one side after the closure of suprafascial tissues in one case and the other reported weakness 24 h after the surgery. In our case, the loss of MEPs was noticed immediately after the laminectomy. A sudden decompression with the restoration of flow may have triggered the ischemia-reperfusion cascade. Complete tetraplegia, like in our case, was not reported in previous cases. All the previously reported cases were treated with high-dose steroids similar to our case and showed remarkable clinical improvement. The hypoxic insult to the brain secondary to cardiac bradyarrhythmias may have contributed to poor prognosis in our case. Bradyarrhythmias

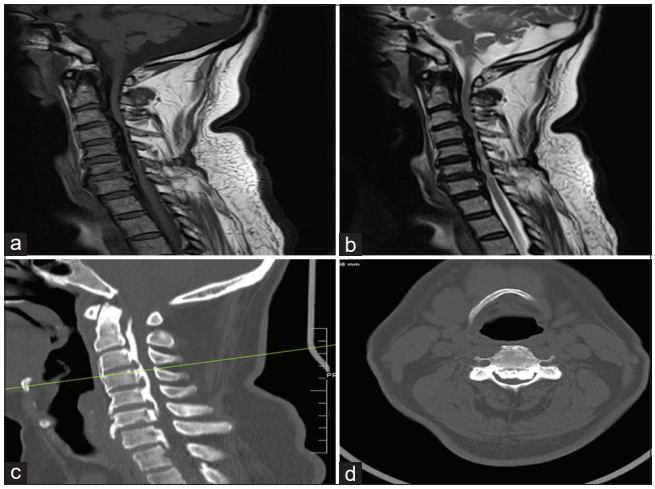

Figure 1: Preoperative sagittal T1 and T2 magnetic resonance imaging images of the cervical spine (a and b) show a multilevel ossified posterior longitudinal ligament with cervical canal stenosis and cord compression with myelomalacic changes. Preoperative computed tomography scan images (c and d) suggest ossified posterior longitudinal ligament with severe canal stenosis

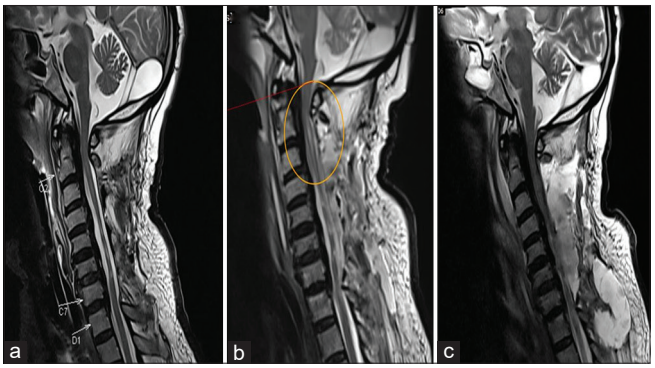

Figure 2: Immediate postoperative sagittal T2 magnetic resonance imaging image (a) suggest adequately decompressed cord with no epidural hematoma or bony compression. Third postoperative day sagittal T2 magnetic resonance imaging images of the cervical spine suggest white cord syndrome with edema extending over a long segment. Postoperative sagittal T2 magnetic resonance imaging images (c) at the time of discharge show the resolution of edema in the cervical spine

are known to occur after spinal cord injury, and vagal stimulation during nasotracheal suction or invasive procedures may precipitate the episodes.[7,8] The endoscopic intervention may be the triggering event in our case. Revision surgery by further decompression was attempted in three of the previous cases, but the outcomes were worse. Since there was no clear evidence of cord compression, we did not attempt a revision surgery. The sudden rush of blood flow after decompression of chronically ischemic tissue can lead to disruption of the blood-spinal cord barrier with an increase in the permeability of the inflammatory mediators and release of free radical oxygen species with resulting neuronal apoptosis.[9,10] Removing lamina from C2 to C7 *en bloc* may have added to the insult resulting from the sudden expansion of the cord. We suggest that a more gradual laminectomy by removing individual lamina at a time may reduce the intensity of the assault.

The differential diagnoses in our case included epidural hematoma, cerebrovascular insult, or iatrogenic trauma to the spinal cord. Imaging findings could rule out epidural hematoma and cerebrovascular insult. Although iatrogenic trauma could not be ruled out, there was no/minimal handling of the cord intraoperatively, and the loss of MEPs immediately after laminectomy suggests a reperfusion injury as a possible mechanism.

WCS is a disastrous complication after cervical spine surgery. One has to be aware of this potentially morbid complication while managing the patient postoperatively and the necessary measures taken to avoid complications.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot bechrological order guaranteed.

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Conflicts of interest

There are no conflicts of interest.

References

1. Lawrence BD, Jacobs WB, Norvell DC, Hermsmeyer JT, Chapman JR, Brodke DS. Anterior versus posterior approach for treatment of cervical spondylotic myelopathy: A systematic review. Spine (Phila Pa 1976) 2013;38:S173-82.
2. Currier BL. Neurological complications of cervical spine surgery: C5 palsy and intraoperative monitoring. Spine (Phila Pa 1976) 2012;37:E328-34.
3. 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.
4. Giammalva GR, Maugeri R, Graziano F, Gulì C, Giugno A, Basile L, *et al*. White cord syndrome after non-contiguous double-level anterior cervical decompression and fusion (ACDF): A "no-reflow phenomenon"? Interdiscip Neurosurg. 2017;7:47-9.
5. 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;113:33-6.
6. 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.
7. Lehmann KG, Lane JG, Piepmeier JM, Batsford WP. Cardiovascular abnormalities accompanying acute spinal cord injury in humans: Incidence, time course and severity. J Am Coll Cardiol 1987;10:46-52.
8. Gilgoff I, Davison-Ward S, Hohn S. Cardiac pacemaker in high spinal cord injury. Arch Phys Med Rehabil 1991;72:601-3.
9. Lee JY, Choi HY, Ahn HJ, Ju BG, Yune TY. Matrix metalloproteinase-3 promotes early blood-spinal cord barrier disruption and hemorrhage and impairs long-term neurological recovery after spinal cord injury. Am J Pathol 2014;184:2985-3000.
10. Sharma HS. Pathophysiology of blood-spinal cord barrier in traumatic injury and repair. Curr Pharm Des 2005;11:1353-89.