

A Case Report: White Cord Syndrome Following Posterior Cervical Decompression: Importance of Timely Diagnosis and Management

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1. Abstract

A rare complication following cervical decompression the so-called "white cord syndrome" was depicted for first time in 2013 [1]. This delineation was made on the basis of neurological deterioration following cervical decompressive surgery associated magnetic resonance finding of intramedullary high-signal intensity on T2-weighted series without structural compression [2-5]. To our knowledge, there are only few cases of white cord syndrome having been published. Therefore we share our experience of a case with this syndrome and management. A 54-year-old female patient complained about neck pain and numbness radiated to right shoulder and upper limb and cervical stenosis secondary to ossification of posterior longitudinal ligament and herniated intervertebral disc over C2-C5 was diagnosed. The patient was referred to our institution and received posterior decompression from C2 to C5 plus C3-5 lateral mass screw fusion. Within the few hours following surgery, she developed left limbs weakness with dysesthesia. Cervical MRI disclosed high signal intensity in T2-weighted sequences at C2-C4 levels and white cord syndrome was impressed. Intravenous mega-dose methylprednisolone was prescribed and also hyperbaric oxygen therapy. The patient's neurologic outcome was better than the preoperative status but left upper limb weakness persisted.

2. Case Presentation

A 54-year-old woman with neck pain and numbness radiated to right shoulder and upper limb was referred to our neurosurgical

team. Cervical MRI demonstrated moderate to severe cervical stenosis secondary to ossification of posterior longitudinal ligament and herniated intervertebral disc at C2-C5 levels with bilateral neuroforaminal narrowing at C4-C6 levels secondary to multilevel degenerative changes (Figure 1). The evaluation before surgery revealed she had full range of motion of the cervical spine, 5/5 in strength in all muscle groups of the upper and lower extremities, and grossly intact sensation dermatomes except right C5, C6. The patient had negative Hoffman and Lhermitte signs and negative Spurling maneuvers. She was assessed as Nurick grade 1 with subtle evidence of cervical myelopathy based on history but not obviously clinical presentation.



Figure 1: Cervical MRI demonstrated moderate to severe cervical stenosis secondary to ossification of posterior longitudinal ligament and herniated intervertebral disc at C2-C5 levels with bilateral neuroforaminal narrowing at C4-C6 levels secondary to multilevel degenerative changes.

The patient underwent C2-C5 posterior decompression with laminectomy, lateral mass screw-rod arthrodesis, C3-5 and the exiting nerve roots were decompressed with foraminotomies. We didn't use somatosensory evoked potentials or motor evoked potentials as monitor but there were no concerns of iatrogenic manipulation throughout the surgical procedure. Within the few hours following surgery, she developed left limbs weakness, 3/5 in strength with dysesthesia without hyperreflexia and abnormal left plantar reflex on physical examination. Postoperative sagittal cervical MRI disclosed high signal intensity in T2-weighted sequences at C2-C4 levels (Figure 2). This patient was managed with high-dose steroid protocols and showed mild improvement in neurologic deficits. After steroid was tapered, this patient received following steroid therapy and rehabilitation at outpatient department. Cervical MRI after 6 months revealed resolved high signal intensity in T2-weighted sequences except minimal signal change over C3-4 level (Figure 3). Now, this patient recovered in limbs strength but mild numbness and dysesthesia over left upper limb were still complained.



Figure 2: Postoperative sagittal cervical MRI disclosed high signal intensity in T2-weighted sequences at C2-C4 levels.



Figure 3: Cervical MRI after 6 months revealed resolved high signal intensity in T2-weighted sequences except minimal signal change over C3-4 level

3. Discussion

Current indications for laminectomy include cervical myelopathy or myeloradiculopathy secondary to OPLL, cervical spondylosis, congenital stenosis, multilevel disk herniation, and traumatic central cord syndrome. Although posterior cervical decompression surgery has been considered safe and effective and has been refined as a surgical technique, the common complications, such as C5 nerve palsy, axial neck pain, and loss of lordosis sometimes occurred. White cord syndrome is a rare complication of decompression surgery of the spinal cord and it's diagnosed based on newly intramedullary lesion on T2-weighted magnetic resonance imaging accompanied newly neurologic deficits. There are no definite risk factors or pathogenesis has been identified but several mechanisms have been speculated, such as ischemia-reperfusion injury, and altered perfusion due to internal recoil of the spinal architecture or biochemical mediators following decompression. Suddenly increased perfusion result in a condition of oxidative stress in the decompressed areas of the spinal cord. Theoretically, free radicals further cause damage to the phospholipid-rich membranes of the neurons. The clinical improvement of neurologic function after high-dose steroids confirms the former speculated mechanism, inhibition of lipid peroxidation after spinal cord injury. However, it should be aware, that methylprednisolone use still has no consistent Class I medical evidence. Clinical presentations and postoperative MRI suggest that certain impairment due to acute diffuse spinal cord edema limited to the gray matter occurred. White cord syndrome should be differentiated with C5 nerve palsy, which is thought nerve root dysfunction and deduced extradural tethering effect as the mechanism. Literatures review revealed several reported cases of paresis in the absence of known intraoperative direct cord injury with an etiology of reperfusion injury. The first two cases describing this phenomenon in the literature as "white cord syndrome" was in a patient with neck pain radiating to the shoulder underwent ACDF for severe cervical compression. Spinal cord reperfusion injury is a speculated etiology for newly neurological deficits after decompressive surgery of spinal cord without intraoperative trauma or manipulation. It is also confirmed given the absence of any postoperative mass-effect lesions such as epidural hematoma, malposition of graft and instrumentation or incomplete decompression. Intraoperative hypotension could lead to spinal cord hypoperfusion and it should be avoided. We recommend a sequence of intraoperative neuromonitoring and postoperative image to exclude other causes. This allows the surgeon to reasonably approach the diagnosis of exclusion of spinal cord reperfusion injury. Early diagnosis and intervention, as we have mentioned in this case, preserves neurologic function and leads to a good prognosis. Exploring the pathophysiology of such phenomenon has great potential for future management of spinal cord reperfusion injury and animal models or clinical trial should be

established. Physical therapy is also reported and prevailingly accepted as a treatment option for the patients with acute or chronic spinal cord injury. Finally, we proposed the combination of high-dose steroids and postoperative physical therapy should continue to be the mainstream of treatment.

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