Tightening the Screws: How a Routine Lumber Decompression with Posterior Instrumentation Tightening Triggered a Case of Acute Cauda Equina Syndrome

Brian H Goldman1*, Hyrum Judd1, Paul DeVito2, Bryan Wilent3 and Jose Valerio4

1Department of Orthopedic Surgery, Larkin Community Hospital, South Miami, FL, USA
2Holy Cross Orthopedic Institute, Fort Lauderdale, FL, USA
3Department of Quality and Clinical Performance- IONM, Specialty Care, Brentwood, TN, USA
4Department of Neurosurgery, Miami Neuroscience Center, Larkin Community Hospital, South Miami, FL, USA

*Corresponding Author: Brian H Goldman, Department of Orthopedic Surgery, Larkin Community Hospital, South Miami, FL, USA.

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Abstract

Background: Cauda equina syndrome (CES) is a compressive neuropathy that has rarely been described in the peri-operative period. This is the first reported case of acute post-operative CES following tightening of lumbar instrumentation.

Case: A 51-year-old male with severe spinal stenosis undergoing lumbar spine decompression with posterior instrumentation experienced intraoperative cauda equina syndrome.

Conclusion: Surgical tightening of posterior lumbar instrumentation can alter adjacent vertebral levels, creating new onset stenosis and acute CES. Significant intraoperative neuromonitoring changes after instrumentation may be without apparent cause but the potential for evolving injury, such as CES, warrants the "Wake-up" test and magnetic resonance imaging. Once diagnosed, emergent surgical management of CES is recommended to prevent long-term neurological complications.

Keywords: Cauda Equina Syndrome; Posterior Instrumentation; Lumbar Decompression; Neuromonitoring; Wake-up Test

 Abbreviations


Introduction

Cauda equina syndrome (CES) is a compressive neuropathy affecting the lumbosacral and coccygeal nerve roots, and if left untreated, can result in irreversible neurologic damage. CES occurs at an incidence rate of 3.4/1,000,000 and has numerous etiologies, with disc herniation being the most common cause [1]. Less common etiologies have been previously described [2]. Varying symptoms can mark the onset of CES; diminished or absent lower limb reflexes along with a sensory loss in the proximal lower limbs, perineal, or saddle region and proximal lower limb weakness or flaccid paraparesis often signal advancing compression requiring rapid diagnosis and intervention.

CES has rarely been observed as a complication of surgical intervention, but a recent case series reported that surgery was the iatrogenic cause of CES lesions in as many as 15% of their patients, compared to 0 - 6.6% in other studies. These patients were significantly
older and had more severe urinary symptoms compared to patients with different etiologies [3]. Bartleson, et al. report a delayed presentation of CES in a patient with ankylosing spondylitis who underwent instrumentation and fusion [4]. Other case reports have identified hemostatic agents such as Surgicel and Gelfoam, post-operative hematoma, and fat grafts as potential causes of post-operative thecal sac compression [5-7]. Sokolowski, et al. reported a post-operative epidural hematoma rate of 58% in patients undergoing lumbar decompressive surgery, with 28% of these patients experiencing adjacent level compression [8]. Use of laminar hooks in the treatment of scoliosis has also been observed to cause CES, both in the acute intraoperative stage as well as in a delayed manner [9,10].

Urgent management of CES is necessary and complete decompression should be performed unless there is a contraindication. Early time to treatment is paramount as compression can impede both arterial supply and venous return to vital nerve roots, resulting in permanent loss of function [2]. Although available data lacks clear consensus, most studies advocate for emergent management of CES, preferably within 48-hours or even sooner as permitted [11-13]. This case study is the first to report tightening of lumbar instrumentation as a potential cause for CES, but more importantly, it demonstrates that immediate return to the operating room for surgical decompression is critical to avoid the long-term clinical implications of acute post-operative CES.

Case Presentation

A 51-year-old Hispanic male presented to our clinic with intractable lower back pain, bilateral lower extremity paresthesias, and weakness consistent with lumbar radiculopathy. Examination demonstrated lower extremity motor deficit and numbness, left worse than right, along with unsteady gait and left-sided foot drop. Nonoperative management, including epidural injections and course of physical therapy, had been unsuccessful. MRI (magnetic resonance imaging) of the lumbar spine demonstrated central and foraminal stenosis from the level of L2 to S1, most severe at the levels of L4-S1.

After discussing potential treatment options, the patient agreed to surgical intervention. The patient was taken back to the operating room where a laminectomy with decompression and posterior instrumentation and fusion from the level of L4-S1, including L4-L5 transforaminal interbody fusion was then performed (Figure 1).

Figure 1: (A) Post-operative instrumentation/fusion lateral and (B) posterior anterior radiographs show posterior instrumentation/fusion, and L4-L5 transforaminal interbody fusion.
Intraoperatively, once all polyaxial screws were in place, each screw was stimulated to ensure that more than 12 volts were required to produce EMG activity for each associated muscle group. This was found to be satisfactory, so we proceeded with tightening of the closure tops. Somatosensory evoked potentials (SSEP) and motor evoked potentials (MEP) initially remained stable up until ten minutes later when the amplitude of the cortical SSEPs to the right posterior tibial nerve decreased by greater than 50%, and the surgeon was alerted (Figure 2). MEPs were acquired again 1 minute after the SSEP alert, at which time there was a bilateral loss of responses from the abductor hallucis muscles (Figure 3). MEPs from all other muscles remained stable. However, a “wake-up” test revealed that the patient was unable to move both lower extremities and had bilateral lower extremity paresthesia in the L2 to S1 dermatomal distribution.

**Figure 2:** Left panel shows the cortical SSEPs to right posterior tibial nerve stimulation, middle panel shows MEPs obtained from left leg muscles, and the right panel shows MEPs obtained from the right leg muscles.

The patient was immediately transferred to the MRI suite for emergent imaging. MRI of the lumbar spine demonstrated severe adjacent level compression at the L3-L4 interspace. It appeared that tightening of the implants and restoration of spinal alignment had caused

**Figure 3:** (A) Post-operative instrumentation/fusion sagittal and (B) axial T2-weighted MRI cuts demonstrating severe central compression at L3-L4.

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a critical increase in the mild pre-existing spinal stenosis at this level (Figure 3). The patient was then brought back to the operating room immediately to undergo L3-L4 bilateral laminectomy with foraminotomy. In the immediate post-operative evaluation, the patient had residual weakness with 3/5 motor strength on the right and 4/5 motor strength on the left, along with mixed sensory disturbances of the right leg and resolved left foot drop. Within a week, all of the patient’s neurological deficits had resolved.

Discussion

Multiple peri-operative causes of CES have been described, but to the best of our knowledge, this is the first study to report implant tightening as a causative factor. It appears that tightening of closure tops to the standard torque of 90 in-lbs (10.2 N-m) using appropriate technique can worsen adjacent level compression that was previously noncritical. This case illustrates specifically the integral role of intraoperative neuromonitoring in recognizing CES; MEP and SSEP alerts noted after pedicle screw and curved rod placement that were crucial to the diagnosis. The continued use of neuromonitoring along with advanced imaging can systematically identify and treat peri-operative neural compression in a prompt manner.

Vauzelle first described the usage of a “wake-up” test in the 1970s when neurologic compromise was suspected during scoliotic correction surgery. Today, intraoperative neuromonitoring (IONM) provides an ongoing assessment of neurologic function and has greatly obviated the need for “wake-up” tests. However, the “wake-up” test remains a useful complement to IONM in certain situations, especially when faced with IONM changes that are consistent with evolving cauda equina or spinal cord dysfunction where a further clinical examination is needed to pinpoint the etiology [14]. While MEPs have excellent sensitivity for evolving motor dysfunction, false positives changes are not uncommon [15]. When there is suspicion for false positive MEP changes, but the pattern of neuromonitoring changes is disconcerting, the next prudent course of action is to perform a “wake-up” test. The role of the test is to confirm if the MEP change is indeed clinically significant. This case highlights the usefulness of neuromonitoring when combined with “wake-up” testing.

The progression of IONM changes, in this case, was somewhat confounding but ultimately proved valuable in identifying CES. SSEP monitoring was stable for some time after the final surgical maneuver, and a significant change in SSEPs was not noted until 10 minutes later. In addition, MEP changes in bilateral feet were also not tested until 12 minutes after final tightening, since these were being monitored only intermittently at designated time intervals. There are a few potential reasons for the apparent latency in potentials. When a mechanical force is applied to neural structures, there is a force dependent delay before a functional compromise is seen electrophysiologically [16]. CES likely is an evolving injury and insult to the spinal cord which can be progressive [17]. Additionally, changes in SSEPs often trail changes in MEPs by 5 minutes or more, so this could explain the delay following final instrumentation tightening [18]. The interval between final tightening and the next MEP acquisition probably accounted for the majority of the short delay in diagnosis in this case. There are some clinical considerations, such as patient movement and electrocautery use, which can impact the frequency at which MEPs are obtained but obtaining MEPs within a few minutes after final tightening may have diagnosed the dysfunction earlier.

The addition of MEPs and the combined changes that were seen likely influenced the decision to perform a “wake-up” test during this procedure. Dimopoulos, et al. reported on two cases with post-operative CES in which neuromonitoring with SSEPs, but not MEPs, was utilized [17]. In both patients with post-operative CES, there were SSEP changes noted during the procedure, but there was no obvious intraoperative event to explain the changes. Thus, no “wake-up” test or immediate intervention was performed. This proved to have detrimental consequences, as symptoms in one patient were slowly progressive and clinical signs of CES were not recognized until nearly 1.5 hours after the procedure.

Emergent MRI imaging was conducted within one hour of the initial neurologic changes during this case, which likely played a central role in complete post-operative recovery. With severe stenosis being recognized so quickly, the patient was emergently decompressed resulting in complete neurological deficit reversal. The exact timing of when to order an MRI and address suspected CES with surgery is not entirely clear. Even greater debate exists when post-operative CES is encountered as a complication of surgical intervention. Patients with new onset urinary symptoms in the setting of lumbar back pain should undergo an urgent MRI preferably by the following morning in hospitals where the MRI suite is closed overnight [19]. Other studies propose that clinical symptoms are only reliable for diagnosing CES at late irreversible stages, and emergent MRI should be performed on all patients suspected of having CES within 1 hour [20].

Many authors advocate for surgical decompression within 48-hours following the onset of signs of CES, but some studies report that early versus delayed surgery can result in improved neurological outcomes [21]. However, a recent meta-analysis revealed no significant
improvement in patients treated within 48-hours versus over 48-hours regarding sensory and motor deficits as well as urinary and rectal function [11]. Guidelines from the British Association of Spine Surgeons (BASS) state that "nothing is to be gained by delaying surgery and potentially much to be lost" [12].

It is unknown if other aspects of the surgical technique were relevant to the development of CES in this patient, but the changes in neuromonitoring very closely accompanied the final tightening maneuver. One previous study has recommended that CES lesions can be avoided with a surgical technique whereby a high-speed drill is used instead of a Kerrison rongeur in patients with severe spinal stenosis [4]. Slow and methodical correction of spinal alignment can likely also contribute to neurologic stability throughout surgery. However, clinicians must be wary of adjacent level stenosis during surgical planning, where longer constructs may be needed to avoid post-operative CES, especially if greater correction is planned or multiple spinal levels are being addressed at the time of surgery. In the setting of acute post-operative CES, it is our recommendation to emergently perform both MRI imaging and surgical decompression within several hours of the index procedure. However, further studies are needed to elucidate the proper timeframe with which to perform emergent decompression for acute post-operative CES.

**Conclusion**

This case report is significantly important and relevant to today’s spinal surgeon who may encounter acute post-operative CES following a routine surgical procedure and instrumentation practice. Spine surgeons need to be aware of the potential for posterior lumbar instrumentation exacerbating adjacent level stenosis after tightening is performed. Once diagnosed clinically and with MRI, emergent surgical management of CES is recommended to prevent long term clinical sequelae and medicolegal implications.

**Conflict of Interest**

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

**Bibliography**


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