Cauda Equina Syndrome Following Decompression for Spinal Stenosis

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Abstract

Cauda equina syndrome following decompression for spinal stenosis appears to occur more commonly than the literature suggests. A large series of spinal stenosis decompressions was reviewed. Based on these findings, a theory is put forth as to the cause of this complication. One hundred seventy-five cases of decompression for spinal stenosis done over a 2.5-year period were reviewed. Follow-up was 1 year to 2 years and 4 months. There were 14 cases of postoperative urinary retention, for an incidence of 8%. Of those, five were ultimately diagnosed with cauda equina syndrome, for an incidence of 2.8%. Of the nine cases that were not diagnosed as cauda equina syndrome, five resolved spontaneously over 2 to 6 weeks. The remaining four were diagnosed as having mechanical urinary problems (e.g., prostate or prolapsed bladder) that required surgical treatment. Anal sphincter monitoring at the time of surgery was not predictive in those patients who developed cauda equina syndrome. All patients who developed cauda equina syndrome improved over 3 to 9 months, but none completely resolved. Three cases underwent further decompression with no apparent improvement. Cauda equina syndrome occurs in 2.8% of decompressions for spinal stenosis.

Keywords

- cauda equina syndrome
- spinal stenosis
- decompression
- postoperative complications

The cauda equina is a collection of dorsal and ventral lumbar and sacral nerve roots, typically L1–L5 and S1–S5. Cauda equina syndrome (CES) is usually characterized as an acute compressive neuropathy with a symptom complex that commonly presents as perineal (saddle) numbness, urinary retention or incontinence, and bilateral leg pain and weakness,¹ typically following lumbar disc herniation.

CES is also a known postoperative complication of lumbar surgery. The literature cites the incidence of postoperative CES as being rare, between 0.08% and 0.2%.^{1–4} Typically, the onset of CES as a postoperative complication has most often been reported to follow lumbar discectomy for herniated lumbar disc.^{1–5} However, the literature regarding the incidence of CES following decompression for spinal stenosis is sparse and typically in the form of case reports.^{1,6–8}

Therefore, the objective of the current investigation is to document the actual incidence and natural history (once the

problem occurs) and to document problems of diagnosis by reviewing a large series of spinal stenosis decompressions. Based on these findings, a theory is put forth as to the cause of the complication.

Methods

The medical records of a consecutive series of patients who had undergone a decompressive laminectomy procedure for spinal stenosis from January 2007 to March 2010 by a single fellowship-trained spine surgeon were reviewed. This review involved detailed review of hospital charts, operative reports, consultations, and preoperative/postoperative office notes. When this review revealed cases of postoperative urinary retention, personal interviews of patients were then conducted. The diagnosis of postoperative CES was made when the following were present: saddle numbness, loss of anal

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Table 1 Demographics of Patients with Postoperative Cauda Equina Syndrome

Case No.	Sex	Age (y)	Decompressed Level	Comorbidity
1	F	38	L5–S1	Diabetes
2	F	60	L3–L5	Hypertension, coronary artery disease
3	M	75	L4–S1	Hypertension
4	F	84	L2–L5	Antiphospholipid, hypertension, coronary artery disease
5	F	49	L4–L5	Fatty liver

sphincter tone (although not necessarily bowel incontinence), urinary retention, and abnormal cystometrogram showing neurogenic bladder.

From each patient record, the following data were obtained: age, gender, date of birth, preoperative diagnosis, surgical procedure, date of procedure, level(s) of decompression and/or fusion, postoperative urinary retention, consultation findings, progress notes, and preoperative questionnaire.

All patients received a preoperative magnetic resonance imaging study (sagittal and axial T₁- and T₂-weighted sequences) and/or computed tomography, which demonstrated a spinal stenosis.

Results

During the study period, 175 cases of decompression for spinal stenosis were reviewed. Follow-up was 1 year to 2 years and 4 months.

A total of 14 cases of postoperative urinary retention were found and of those, five cases of postoperative CES were detected for an incidence of 8.0% and 2.8%, respectively.

In regards to the five postoperative CES cases, one patient was male and four were female, with a range in age from 38 to 84 years (mean age, 61 years). Demographics of CES cases are presented in ▶Table 1. All five patients who developed postoperative CES improved over a 3- to 9-month period, but none completely resolved. Three of these cases underwent further decompression with no apparent improvement.

Of the nine cases of postoperative urinary retention that were not diagnosed as CES, five resolved spontaneously over 2 to 6 weeks; the remaining four were diagnosed as having mechanical urinary problems (e.g., enlarged prostate or prolapsed bladder) that required surgical treatment.

On a preoperative questionnaire, all of the patients denied urinary problems prior to the spine surgery.

Surgical Technique

The same operation technique was used in all five CES patients, and there was no trend noted in the specifics of the surgery. The patients were placed in the prone position on the Jackson table. The affected levels of the spine were exposed through a longitudinal midline incision. The spinous processes, laminae, facet joints, and transverse processes

were exposed. A large self-retaining retractor was placed. The spinous processes of the affected levels were removed. Bilateral laminectomy and foraminotomy were performed at the effected levels. At the conclusion of decompression, each nerve root was followed out to the foramen and appeared adequately decompressed. At this point, facet joints at the affected levels were removed with a rongeur. The transverse processes of affected levels were decorticated with a rongeur, and bone from the bone bank was packed along the decorticated areas across the facet joints into the gutters between the affected levels. Spinal cord and nerve monitoring showed no changes at the end of the procedure for all five patients. Anal sphincter monitoring at the time of surgery was not predictive in those patients who developed CES.

Discussion

In the current study, five patients developed postoperative CES after decompression surgery for spinal stenosis. Previously, the cause of postoperative CES after decompression surgery has been shown to be caused by inadequate decompression, unsuspected stenosis at a more proximate level, malpositioning of a fat graft, a retained surgical sponge, and a compressive hematoma.^{1,6,9} These were not the causes of postoperative CES in the current study.

CES is traditionally accepted to be the result of a compressive neuropathy. Yet, in the situation where CES arises in a postoperative setting where no compressive source is identified, as in the current study, the compressive neuropathy definition can fall short. Previously, it has been put forth that tension, not compression, and the subsequent ischemic effects on spinal cord microcirculation are a more descriptive analysis of the precursors to CES.^{10,11} In 1977, Murphy emphasized that the extrusion of disc material in the lumbar region puts tension, rather than compression, on the nerve root, which can likely precipitate CES.¹¹

Furthermore, it has been shown that there is an area of hypovascularity below the tip of the conus medullaris, which would be very vulnerable to dural tension of nerve roots at mid to lower lumbar levels, thus potentially inducing root ischemia, precipitating CES.^{1,12} Dural tension affecting this area of hypovascularity could be caused by spinal stenosis, as well as retropulsion of disc material in mid to lower lumbar levels.

Figure 1 (A) Preoperative diameter (arrow) of dura on cross-sectional magnetic resonance imaging. (B) Postoperative diameter (large arrow) of dura on computed tomography.

We propose that, in the five cases of postoperative CES presented herein, that the source of dural tension on lumbosacral nerve roots, as described above, was expansion of the decompressed dura. The tension on the lumbar sacral nerve roots would further compromise an area that already is hypovascular, inducing root ischemia and ultimately CES (►Figs. 1 and 2). This would also explain why further decompression in three of the five patients was not effective at improving their symptoms of CES and would also explain the presence of postoperative CES where no other compressive etiology could be elucidated. This hypothesis would also explain why anal sphincter monitoring was not predictive of CES in these patients because we believe that the syndrome occurs after surgery.

It is also possible that these patients were predisposed to root ischemia secondary to dural tension of lumbosacral nerve roots. ►Table 1 shows that four of the five patients with postoperative CES had comorbidities that can compromise microvasculature, such as diabetes, hypertension, and antiphospholipid syndrome.

Finally, it is important to note that a majority of the patients in this study who did experience postoperative urinary retention (including the subgroup of those who were diagnosed with CES) did not initially disclose to their physician or surgeon their symptoms. Their symptoms were only elucidated after extensive follow-up contacts with the patients. Thus we feel that these symptoms of postoperative urinary retention and other symptoms of CES are likely

Figure 2 Intraoperative image. Note stretch of dura above S1.

underreported by patients in general and is likely reflective of why the incidence in the current literature is lower than what was found in the current study.

Conclusions

Our study demonstrates that postoperative CES as a complication of decompression surgery for spinal stenosis occurs at an incidence 2.8%, which is more common than the literature suggests. It is theorized that the cause of postoperative CES after decompressive surgery for spinal stenosis, where no other compressive source can be identified, is the result of dural tension on lumbosacral nerve roots, secondary to dural expansion following decompression, resulting in root ischemia. This would explain why further decompression appeared to not be effective in these patients. Unfortunately, our study demonstrates that once this complication occurs, though it may improve, it will not fully resolve. In this setting, anal sphincter monitoring was not predictive of this complication.

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