Cauda equina syndrome as a postoperative complication of lumbar spine surgery

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Object. The term “cauda equina syndrome” (CES) has been used to describe the signs and symptoms in patients with compressive neuropathy of multiple lumbar and sacral roots. This syndrome is well known as an indication for surgical intervention in treating lumbar spine disease, but relatively unknown as a postoperative complication following surgery for disease. In this study the author describes two cases of CES that occurred following uneventful lumbar spine procedures—one microdiscectomy and one decompressive laminectomy.

Methods. Preoperative, operative, and postoperative management is discussed and the relevant literature reviewed. One patient suffered perineal numbness and bowel and bladder difficulty following a decompressive laminectomy. Postoperative imaging studies were negative for residual lesion and the treatment goal pursued was partial long-term resolution of symptoms. The second patient had progressive numbness and weakness in the lower extremities. Results of urgent postoperative magnetic resonance imaging studies were inconclusive and repeated exploration was performed within hours of the initial procedure. The patient made a full recovery, although the intraoperative findings did not reveal a clear cause of the patient’s symptoms.

Conclusion. Postoperative symptoms of partial or complete CES represent a medical emergency, especially if they are progressive. It is necessary to perform urgent postoperative imaging in patients, but the results are not always helpful. Surgical exploration is warranted if a mass lesion is demonstrated on imaging studies or if symptoms progress and the disease origin is not clear based on available information.

KEY WORDS • cauda equina syndrome • lumbar spine surgery • postoperative complication

The term “cauda equina syndrome” refers to the signs and symptoms in patients with compressive neuropathy of multiple lumbar and sacral nerve roots. The syndrome is characterized by bilateral sciatica, lower-extremity weakness, saddle-type hypesthesia, and bowel and bladder dysfunction. It is usually caused by extradural compression from tumors, trauma, infection, spinal stenosis, or disc herniation. Note that this syndrome has also occurred as a postoperative complication as reported in large series of lumbar spine procedures. In this paper I describe two illustrative cases and review the pertinent literature concerning postoperative CES.

ILLUSTRATIVE CASES

Case 1

This 67-year-old woman had a 2-year history of progressive symptoms consistent with neurogenic claudication. An MR imaging series demonstrated stenosis at L4–5 and L5–S1. The patient underwent an L4–S1 laminectomy with bilateral foraminotomies. The procedure was complicated by an incidental durotomy. Attempts were made to repair this primarily, but a fat graft patch was ultimately used. Postoperatively, the patient regained full strength in her lower extremities, but she also experienced numbness in the perineum. The following day, after a Foley catheter had been removed, she suffered from urinary retention. Postoperative imaging revealed only normal postoperative changes. She was treated with the aid of intermittent catheterization. Two years later, she continues to have incomplete recovery of bladder function, but is otherwise neurologically intact.

Case 2

This 37-year-old woman had a 2-year history of a left S-1 radiculopathy that was unresponsive to conservative measures. An MR image demonstrated a focal L5–S1 herniated disc impinging on the left S-1 nerve root (Fig. 1 left and center). The patient underwent an uncomplicated left L5–S1 microdiscectomy (Fig. 1 right). Postoperatively, she was neurologically intact on extubation and initially in the recovery room. Approximately 30 minutes after the procedure, the patient began to experience decreased sensation in her left leg, followed by similar symptoms in the right leg. Within the next hour, she complained of heaviness in her legs, which progressed to a weakness in both dorsiflexion and plantar flexion. After the patient reported saddle anesthesia, a Foley catheter was placed and 1200 ml residual urine was noted. According to spinal cord protocol, Solu-Medrol was administered (32 mg/kg loading
dose followed by 5.4 mg/kg/hr). An emergent MR imaging study was completed, and although the results made us suspect the occurrence of an epidural hematoma, there was no definitive evidence of a compressive lesion (Fig. 2). The patient was returned to the operating room for repeated exploration. Because no specific source of her neurological deficits was found, a complete S-1 and L-5 laminectomy was performed (Fig. 3). In the recovery room, the patient demonstrated complete resolution of her symptoms. She was discharged home with no further complication.

DISCUSSION

Cauda Equina Syndrome Following Disc Prolapse

Cauda equina syndrome is manifested by saddle anesthesia, loss of bladder function (flaccid neurogenic bladder with overflow incontinence), loss of bowel function (flaccid external sphincter with fecal retention and overflow incontinence), and loss of sexual function (loss of genital sensation, reflex erection, and ejaculation). Patients presenting with CES following acute lumbar disc herniation is well documented.\textsuperscript{11,20} Most patients have presented with perineal numbness, urinary retention or incontinence, progressive motor weakness, and progressively decreasing sensation in the lower extremities.\textsuperscript{11} At times, urinary retention has been reported to occur in patients with no pain, motor, or sensory symptoms, but with a “protruded lumbar disk.”\textsuperscript{17} Other researchers have described a “hemi-CES,” which is defined as a combination of unilateral leg pain, unilateral sensory loss in dermatomes S1–5, and sphincter paralysis as manifested by either urinary retention or incontinence.\textsuperscript{1} Urinary retention is the most severe complication and carries the worse prognosis. Return of motor function portends a better prognosis. In any case, urgent decompression seems to translate into the best return of function.\textsuperscript{7,11,16,20}

Cauda Equina Syndrome Following Lumbar Spine Surgery

A careful review of published series of lumbar spine procedures indicates that CES is a rare (0.08–0.2% incidence) but potentially disastrous complication of lumbar intervertebral disc prolapse.\textsuperscript{3,16,19,20} In patients undergoing routine one- or two-level discectomies without fusion or instrumentation at community hospitals, postoperative CES occurred in 23 (0.08%) of 28,395 patients.\textsuperscript{19} In a similar series, Spangfort\textsuperscript{21} reported five (0.2%) of 2504 patients with postoperative CES among a population with a broad range of lumbar disc problems and levels of surgical complexity. McLaren and Bailey\textsuperscript{13} reported six cases of CES in patients presenting immediately after uneventful lumbar discectomy from among a series of 2842 procedures performed during a 10-year period. None of these patients had neurological deficits preoperatively. Four patients developed postoperative numbness in the perineum and decreased rectal tone. Motor weakness was detected in all patients. Five patients developed symptoms in the recovery room, and one patient developed symptoms 4 days after surgery; all of these patients required catheterization for urinary retention (four patients) or incontinence (two patients). Decreased sensation starting in the “perineum, sacral strips, and the feet was progressive” and was noted in all patients. Voiding dysfunction and urodynam-ic function can be worsened by lumbar spine surgery compared with preoperative baseline function.\textsuperscript{3,10} Bear in mind
however, that transient urinary retention is a common postoperative complication of both cervical and lumbar spine surgery, but is not necessarily a result of direct nerve injury.2

**Origin of Postoperative CES**

Factors that might precipitate postoperative CES include inadequate decompression, nerve root swelling, hematoma, retained disc fragments, Gelfoam, intradural masses, or vascular insufficiency (tenuous vascular supply of cauda equina). Some authors have asserted that patients with underlying spinal canal stenosis who have undergone discectomy via the “keyhole interlaminar approach” are more susceptible to postoperative CES.13,19 This does not seem to hold true, however, given the absence of an increase in postoperative occurrence rates of CES despite the facts that lumbar microdiscectomy has gained widespread acceptance and become the most common method of treating lumbar disc herniation.

Two cases of postoperative CES following the placement of a free epidural fat graft have been reported.18 Both cases involved routine lumbar discectomies, and the patients developed symptoms on postoperative Day 2 that included urinary retention, motor weakness, and saddle hypesthesia. Both patients had undergone imaging studies whose results demonstrated spinal canal compromise.

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**Fig. 2.** Postoperative gradient echo (upper left), T2-weighted (upper right), and T1-weighted (lower left) midsagittal MR images obtained after development of CES while the patient was in the recovery room following an uneventful microdiscectomy, demonstrating postoperative changes and possible epidural hematoma. Axial T2-weighted image (lower right) obtained through postoperative levels, demonstrating postoperative changes.
from the fat graft. This was followed by surgical exploration and decompression. Fortunately, both of these patients made good recoveries. The patient in Case 1 in the present report also required a fat graft for repair of an incidental durotomy; the graft was not believed to have contributed to the patient’s symptoms, however, because it was unremarkable on postoperative imaging studies. Furthermore, this graft was placed at the L4–5 level and the patient’s motor function was intact, thus indicating that compression from this graft did not contribute to her symptoms. In another case report, authors describe incarceration of the cauda equina through a small dural defect unrecognized at the time of a first operation. This defect was reexplored, the nerves were replaced into the dural sac, and the durotomy was repaired with complete resolution of symptoms. This case supports repeated operation in patients with postoperative CES following lumbar spine surgery.

In patients with symptoms consistent with CES but no demonstrable compressive lesion, a vascular origin might be indicated. The vascular supply to the cauda equina, in particular its roots, is tenuous. In the study by Parke, et al., vascular injection of perinatal cadavers demonstrated a regional hypovascularity below the level of the conus medullaris. This result provides an anatomical rationale for the suspected neuroschemic manifestations concurrent with CES. Even in patients with a compressive lesion, secondary ischemia could be considered a common underlying mechanism. Histological examination performed in a study of CES in a dog model producing CES revealed arterial narrowing and venous congestion of the nerve roots and the dorsal root ganglia. In the second case in the present study, the patient experienced a progressive, fairly rapid onset of symptoms and, at the time of repeated operation, did not have a definitive compressive lesion. This set of circumstances coupled with the fact that she had complete resolution of symptoms in the recovery room immediately following surgery indicates a potential ischemic origin of her symptoms.

**Indications for and Timing of Repeated Surgery**

Altered bowel and bladder function associated with acute disc protrusion is commonly accepted as an indication for emergency surgery. Decompression of an acute disc protrusion in less than 48 hours offers the best chance of recovery. Improvement in function after late decompression has also been reported, but the chance for recovery in this event is decreased.

McLaren and Bailey reported on six cases of CES in patients presenting immediately after uneventful lumbar discectomy. Four patients underwent urgent myelography followed by surgery. Two patients were treated nonsurgically with bed rest and steroid agents. Functional bladder and bowel recovery was achieved in the four patients who had undergone early decompressive laminectomy, whereas altered sphincter function was not recovered with late decompression. Recovery of motor weakness was incomplete in the two patients who had severe paraparesis prior to decompression. The authors asserted that if compressive pathophysiological findings are not demonstrated, surgical decompression is not indicated. In both cases in the present study, no specific compressive pathophysiology was revealed on imaging studies. The patient who did not undergo surgery continues to have urinary symptoms, whereas the patient who underwent repeated exploration is symptom free. Therefore, it seems reasonable to recommend repeated exploration in any patient with postoperative CES and in whom there is no reasonable explanation for existing neurological deficits.

**Prognosis Following CES**

The prognosis for preoperative CES seems to be heavily weighted by a patient’s preoperative neurological condition. Patients with severe motor deficit prior to decompression tend to have less chance of motor recovery after decompression compared with that in patients with minor deficits. Nevertheless, motor function is much more likely to return after surgery than is bladder function. Recovery of urinary retention and bowel incontinence is much more variable but occurs in approximately 70% of patients treated within 48 hours. Some authors assert that the most important indicator for the prognosis of bladder function is the severity and extent of sensory disturbances in the so-called saddle areas. In fact, recovery of bladder function seems to parallel the return of saddle area sensation. This can be a very slow process, requiring an interval of several months to years. In patients with postoperative CES, the numbers are obviously much smaller for making a prediction of recovery. As mentioned previously, repeated surgery appears to provide for the best recovery, with approximately 80% of patients making either a complete or a delayed partial recovery and 10 to 20% making no recovery.
CONCLUSIONS

Cauda equina syndrome is a compressive neuropathy involving multiple nerve roots affecting motor, sensory, bowel, bladder, and sexual function. The syndrome has a variety of origins, but can occur as a postoperative complication of lumbar spine surgery. In any case, urgent neuroimaging studies of the lumbar spine are advised. Surgical decompression is necessary if a compressive lesion is found. In the case of postoperative CES, surgical exploration and further decompression are recommended in all situations.

References


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