A Case Study of Cauda Equina Syndrome

By Dan-Phuong Esther N Ho, MD

Abstract

Primary care and emergency care physicians frequently encounter patients with low back strain and sciatica and must be able to recognize the perilous signs of cauda equina syndrome (CES), a condition usually caused by massive disk herniation. Patients with CES may have peripheral neurologic deficits as well as bowel and bladder dysfunction. Emergent magnetic resonance imaging is the study of choice to confirm the diagnosis. Surgical decompression is the only effective treatment for CES. The prognosis depends on initial signs and symptoms, progression of neurologic deterioration, and timeliness of surgical decompression. Recovery may occur immediately after surgery or months or years postoperatively.

Introduction

Low back pain is a common complaint heard frequently by all physicians who provide primary care to adult patients. Because this common type of pain is generally not associated with clinically significant pathology, clinicians may overlook a rare but potentially disabling neurologic affliction such as cauda equina syndrome (CES). Most cases of CES result from lumbar disk herniation with excessive compression on the cauda equina. Clinical features may include low back pain, sciatica, saddle-area anesthesia, motor weakness, sensory deficit, and urinary or fecal incontinence. The condition may progress to permanent incontinence, paraplegia, or both.[1] Therefore, to diagnose and promptly treat CES, clinicians must be able to recognize the signs and symptoms of this neurologic syndrome.

Case Report

A 28-year-old man presented to the emergency department for low back pain and numbness in both lower extremities. Two days earlier, he had sharp, shooting pains in the back and buttocks after moving boxes. The pain was relieved with hydrocodone with acetaminophen. However, on the morning of presentation, the patient awoke with numbness in both lower extremities and had left leg weakness so severe that the patient was unable to stand or walk without support. The patient described the pain as mild while he was supine and worse when he sat or stood. The patient reported some urinary hesitancy, dribbling of urine, and constipation. He did have morning erections. The patient reported that he had had an industrial injury five years before that resulted in a herniated lumbar disk and subsequent laminectomy; he had been doing well since then until the time of presentation.

On physical examination, the patient was alert and oriented and had stable vital signs. The back was not tender when palpated. The straight-leg-raise test to 30° did not elicit additional pain in either leg. Motor strength examination showed some lack of effort on the right side but good motor function in all muscle groups of the right lower extremity. Motor strength of the left lower extremity was decreased to 3 out of 5 in the hamstrings, iliopsoas, and quadriceps muscles; 1 to 2 out of 5 in the ankle and toe plantar flexor muscles; and 0 out of 5 in the ankle dorsiflexor muscles and extensor hallicus longus muscle. Tests of the deep tendon reflexes showed normal right patellar reflex, absent left patellar reflex, and absent Achilles tendon reflexes bilaterally. Sensory examination demonstrated hyperalgesia of the left calf and hypesthesia of the scrotum, perianal area, and left foot. Anal sphincter tone was reduced.

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Lumbar spine radiographs revealed mild narrowing of the intervertebral disk spaces between L3-4 and L4-5. Emergent magnetic resonance images (MRI) of the lumbar spine showed herniated disk material located along the left lateral aspect of the vertebral canal. Disk material extending from the body of L3 to the body of L4 resulted in moderate central canal stenosis and compression of the cauda equina.

The radiograph and MRI both showed evidence of previous L4 laminectomy.

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A neurosurgery consultation was obtained urgently. The patient received an initial dose of dexamethasone, 10 mg, intravenously, followed by 4-mg intravenous dose every six hours. The patient was taken to the operating room the next morning. An L3 laminectomy was done, and herniated disk material at the level of L3-4 was removed in multiple fragments. Postoperatively, sensory and motor deficits persisted; the patient had decreased sensation on the left side of his penis and perineum, left foot drop, hyperalgesia of the left calf, and decreased anal sphincter tone. He was able to void without use of a catheter but had some difficulty with initiating urination. The patient was transferred to the rehabilitation unit for acute therapy, and the neurologic deficits gradually improved.

One year after surgery, the patient was able to walk, although the gait was broad and slow; he was not able to run. He had regained sensation in the left leg and perineum, although sensation was still mildly decreased. Sexual function was intact; the patient was able to have erections and had penile sensation. The patient was able to urinate, but initiating urination still required effort.

Discussion

Epidemiology of Low Back Pain, Sciatica, and CES

Seventy to 85% of adults in the United States report experiencing low back pain by the age of 50 years;[2] national annual incidence of low back pain is 5%.[3] One quarter of patients with back pain have sciatica,[4] a syndrome characterized by pain radiating from the buttocks down the posterior or lateral aspect of the lower limb below the knee.[5,6] Sciatica may be associated with motor, reflex, or sensory deficits. The most common cause of sciatica is herniation of the lower lumbar intervertebral disks, most often involving the disk between L4-5 and less often the disk between L5-S1 or L3-4; herniation causes compression or irritation of the lumbar nerve roots.[7] Symptomatic disk herniation most commonly occurs in patients who are 30 to 50 years old, although such herniation can occur at any age.[8] In contrast to sciatica, cases of CES after disk herniation are relatively rare; according to Chang et al, the incidence of CES due to lumbar disk herniation has been reported to range from 1% to 10% of operated disk cases.[9]

Etiology of CES

The adult spinal cord terminates at the level of vertebra L1 to L2 with the terminal bundle of lumbar and sacral nerve roots within the spinal canal forming the cauda equina below; the nerve roots then separate and exit at their specific foramina.[10] Compression of the cauda equina is most commonly caused by herniation of a large quantity of lumbar disk material, often in association with degenerative or congenital spinal stenosis, and can result in CES. According to Delamarter et al, extremely rare causes of CES include compression by tumor, fracture, penetrating trauma, chiropractic manipulation, chemonucleolysis, postoperative hematoma, free epidural fat graft, and ankylosing spondylitis.[11]

Risk factors for disk herniation include obesity,[12] male gender,[12] age more than 40 years,[12] heavier lifetime loading during occupational and leisure time activities,[13] and history of back disorders.[13] Factors associated with degeneration of the intervertebral disk include genetic factors and changes in disk hydration and collagen.[14] These factors reduce effectiveness of the nucleus pulposus (the inner disk layer) for absorbing shock, providing resistance to compression, and permitting flexibility of the vertebral column.[10] Instead, the nucleus transmits a greater portion of applied loads to the surrounding annulus asymmetrically, an imbalance that may lead to weakness of the annulus and herniation of the nucleus pulposus material into the spinal canal.[14]

Clinical Presentation and Physical Examination for CES

Three variations of CES have been described: 1) acute CES that occurs suddenly in patients without previous low back problems; 2) acute neurologic deficit in patients who have history of back pain and sciatica; and 3) gradual progression to CES in patients who have chronic back pain and sciatica.[15] However, in more than 85% of the cases, the signs and symptoms of CES develop in less than 24 hours.[7]

Signs of CES include severe bilateral sciatica; bilateral foot weakness; saddle-type hypesthesia or anesthesia in the areas innervated by nerve roots S2 to S5; and retention or incontinence of urine, stool, or both.[9] Thus, asking all patients with back pain about the presence of associated neurologic deficits is imperative and should include questions about lower extremity and saddle paresthesia, numbness, weakness, gait disturbance, bowel or bladder dysfunction, and impotence.[6] Positive responses to these symptoms warrant further investigation to rule out the diagnosis of CES. Coughing, sitting, or bearing down (Valsalva maneuver) may aggravate sciatic pain, and lying supine may alleviate pain.[6] The straight-leg-raise test, during which the examiner raises the supine patient's fully extended leg up to 70 degrees, is considered positive for disk herniation and nerve irritation when it produces a radicu-

lar pain radiating down the lower limb to below the knee in one or both limbs at between 30 and 60 degrees.[6,16] A positive straight-leg-raise test result for the limb on the affected side is 80% sensitive and 40% specific for disk herniation, a result which suggests involvement of the L5 to S1 nerve roots or the sciatic nerve. A positive straight-leg-raise test result for the limb on the contralateral side is 25% sensitive and 90% specific for disk herniation, a result which suggests involvement of the L2 to L4 nerve roots.[17]

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Neurologic examination should evaluate each of the spinal nerve roots. Lumbar disk herniation typically affects the nerve root inferior to the disk space. Thus, herniation of the L4-5 intervertebral disc would typically impinge on the L5 nerve root.[6] Sensory examination should be conducted using both light touch and pinprick;[6] cold temperature sensation can be easily tested using the cold metal end of a tuning fork. Sensory, motor, and reflex innervation by nerve roots L1 through S5 are summarized in Table 1. Because the L4 nerve root controls ankle dorsiflexion, the L4 nerve root can be tested by heel walking.[6,14] The L5 nerve root can be evaluated by using the Trendelenburg test.[6,14] The Trendelenburg test requires the patient to stand on one leg and the physician to stand behind the patient with hands on the patient's hips; a drop in the pelvis on the side opposite the raised leg implies presence of either L5 nerve root or hip joint pathology.[14] The S1 and S2 nerve roots together are responsible for plantarflexion of the ankle and can be tested by asking the patient to stand and to walk on the toes.[6]

CES or spinal cord compression should be considered until proven otherwise in all patients who have low back pain with bowel or bladder incontinence.[6] Bladder dysfunction usually is secondary to detrusor muscle weakness and an areflexic bladder; this dysfunction initially causes urinary retention followed by overflow incontinence in later stages.[18] Patients who have back pain with urinary incontinence but who have normal neurologic examination results should have a urinary postvoid residual volume measured.[6] A postvoid residual volume greater than 100 mL indicates overflow incontinence and mandates further evaluation;[6] a volume less than 100 mL rules out diagnosis of CES.[6] The anal wink reflex, elicited by gently stroking the skin lateral to the anus, normally causes reflexive contraction of the external anal sphincter.[6] Rectal examination should be done to assess anal sphincter tone and sensation if any of the characteristic signs or symptoms of CES are present.[6]

Diagnosis, Treatment, and Prognosis of CES

Although plain radiographs are of limited value for diagnosing lumbar disk herniation, they can be used to rule out other pathology.[14] Plain lumbar spinal radiographs should be obtained if neurologic dysfunction is discovered on physical examination or if patient

Table 1. L1 to S4 nerve roots with associated motor, sensory, and reflex functions

Nerve root	Sensory	Motor	Reflex	Reference
L1	Anterior aspect of thigh	Hip flexion (iliopsoas muscle)		14
L2	Anterior aspect of thigh	Hip flexion Knee extension (quadriceps muscle)	Patellar	6,14
L3	Anterior aspect of thigh	Hip flexion Knee extension	Patellar	6,14
L4	Medial aspect of leg and foot Great toe	Knee extension Ankle dorsiflexion and foot inversion (tibialis anterior muscle)	Patellar	6,14
L5	Lateral aspect of calf First dorsal web space	Great toe dorsiflexion (extensor hallicus longus muscle) Hip abduction (gluteal muscles)		6,14
S1	Lateral aspect of foot Posterolateral aspect of calf	Foot eversion (peroneal muscles) Ankle plantarflexion (gastrocnemius, soleus muscles)	Achilles	6,14
S2	Perineum, perianal	Ankle plantarflexion (gastrocnemius, soleus muscles) Bladder and bowel control	Anal wink	6
S3	Perineum, perianal	Intrinsic foot muscles Bladder and bowel control	Anal wink	6
S4	Perineum, perianal	Intrinsic foot muscles Bladder and bowel control	Anal wink	6

history suggests the presence of tumor, infection, or fracture.[6] Although radiograph findings are often unremarkable, the presence of decreased disk height may be suggestive of disk herniation.[14]

Computed tomography (CT) or magnetic resonance imaging (MRI) may be considered for evaluation of a patient with signs of disk herniation.[19,20] MRI is the widely accepted standard for the rapid and complete evaluation of a patient with clinically significant spinal pathology and should be obtained emergently when the diagnosis of CES is suspected.[19] Abnormalities on MRI are commonly found in asymptomatic patients;[20] MRI should therefore be used as a means of confirming a diagnosis in the presence of neurologic signs rather than as a screening tool.[20] In the series of CES cases reported by Shapiro, 75% of CT or MR images of CES cases showed large quantities of disk material occupying more than one third of the spinal canal diameter.[7]

Treatment with high doses of steroids may provide rapid relief of pain as well as improve function …

Treatment with high doses of steroids may provide rapid relief of pain as well as improve function while appropriate diagnostic studies and consultations are being obtained.[6] Dexamethasone is commonly given intravenously at doses of 4 to 100 mg.[6]

CES is an absolute indication for emergent surgical decompression;[11] laminectomy followed by gentle retraction of the cauda equina (to avoid complications of increased neurologic compromise) and diskectomy is the technique of choice.[7] Timing of the decompression has not been unanimously agreed upon. Traditionally, patients with CES who have surgery within 24 hours of initial symptoms are believed to have clinically significantly better neurologic recovery.[7] However, some studies[1,7,21] found no statistically significant improvement in outcome between patients surgically treated within 24 hours compared with those surgically treated within 24 to 48 hours. Other studies[9,11] suggest that surgery performed on an expedient rather than emergent basis did not compromise neurologic recovery.

Outcome for patients with CES can be predicted primarily by their symptoms at presentation.[6] Patients who are ambulatory at initial evaluation generally remain ambulatory;[6] those who are paretic but can walk with assistance have a 50% chance of walking unassisted after recovery; those who are paralyzed when seen initially rarely will walk again.[6] About 79% of patients who require urinary catheterization at initial evaluation will continue to use a catheter after recovery.[6] Patients with a history of chronic low back pain have an increased risk of urinary and rectal dysfunction after surgery.[1] Postoperative recovery time can range from months to years. Most patients improve within the first two years after surgical decompression, although some continue to clinically improve for up to five years after surgery.[1]

Conclusion

Acute compression of the cauda equina is a neurologically compromising and potentially debilitating syndrome. Physicians who evaluate low back pain must be able to recognize the signs and symptoms of this relatively rare but critical spinal syndrome and must expedite emergent evaluation with appropriate history and physical examination, imaging studies, and consultations. Patients with neurologic deficits of the lower extremities, perianal region, scrotum, penis, bowel or bladder (or both) need further evaluation. Patients with bowel or bladder incontinence should be considered to have neurologic spinal compromise until proven otherwise and need emergent imaging studies, preferably MRI. If the diagnosis of CES is confirmed, surgical intervention should be done as soon as possible to prevent progression of neurologic symptoms and to allow maximum neurologic recovery. ❖

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References

1. Ahn UM, Ahn NU, Buchowski JM, Garrett ES, Sieber AN, Kostuik JP. Cauda equina syndrome secondary to lumbar disc herniation: a meta-analysis of surgical outcomes. Spine 2000 Jun 15;25(12):1515-22.
2. Research on low back pain and common spinal disorders. NIH Guide 1997 May 16;26(16). Available from: http://grants.nih.gov/grants/guide/pa-files/PA-97-058.html (accessed June 2, 2003).
3. Carey TS, Evans AT, Hadler NM, et al. Acute severe low back pain: a population-based study of prevalence and care-seeking. Spine 1996 Feb 1;21(3):339-44.
4. Borenstein DG, Wiesel SW, Boden SD. Low back pain: medical diagnosis and comprehensive management. 2nd ed. Philadelphia: Saunders; 1995.
5. Kahanovitz N. Sciatica: verifying the diagnosis, offering relief. J Musculoskeletal Med 1998 Jan;15(1):51-9.
6. Della-Giustina DA. Emergency department evaluation and treatment of back pain. Emerg Med Clin North Am 1999 Nov;17(4):877-93, vi-vii.
7. Shapiro S. Medical realities of cauda equina syndrome secondary to lumbar disc herniation. Spine 2000 Feb 1;25(3):348-51; discussion 352.
8. Murrey DB, Hanley EN Jr. Surgery for lumbar disc

herniation: what are the choices? J Musculoskeletal Med 1999 Jan;16(1):39-45.
9. Chang HS, Nakagawa H, Mizuno J. Lumbar herniated disc presenting with cauda equina syndrome. Long-term follow-up of four cases. Surg Neurol 2000 Feb;53(2):100-4;discussion 105.
10. Arce D, Sass P, Abul-Khoudoud H. Recognizing spinal cord emergencies [published erratum appears in Am Fam Physician 2002 May 1;65(9):1751]. Am Fam Physician 2001 Aug 15;64(4):631-8.
11. Delamarter RB, Sherman JE, Carr JB. 1991 Volvo Award in experimental studies. Cauda equina syndrome: neurologic recovery following immediate, early, or late decompression. Spine 1992 Sep;16(9):1022-9.
12. Kostova V, Koleva M. Back disorders (low back pain, cervicobrachial and lumbosacral radicular syndromes) and some related risk factors. J Neurol Sci 2001 Nov 15;192(1-2):17-25.
13. Borenstein DG. Epidemiology, etiology, diagnostic evaluation, and treatment of low back pain. Curr Opin Rheumatol 1997 Mar;9(2):144-50.
14. Humphreys SC, Eck JC. Clinical evaluation and treatment options for herniated lumbar disc. Am Fam Physician 1999 Feb 1;59(3):575-82, 587-8.
15. Tandon PN, Sankaran B. Cauda equina syndrome due to lumbar disc prolapse. Indian J Orthop 1967;1:112-9.
16. Bratton RL. Assessment and management of acute low back pain. Am Fam Physician 1999 Nov 15;60(8):2299-308.
17. Rose-Innes AP, Engstrom JW. Low back pain: an algorithmic approach to diagnosis and management. Geriatrics 1998 Oct;53(10):26-8, 33-6, 39-40 passim.
18. Comarr AE. Neurourology of spinal cord-injured patients. Semin Urol 1992 May;10(2):74-82.
19. Coscia M, Leipzig T, Cooper D. Acute cauda equina syndrome. Diagnostic advantage of MRI. Spine 1994 Feb 15;19(4):475-8.
20. Boden SD, Davis DO, Dina TS, Patronas NJ, Wiesel SW. Abnormal magnetic-resonance scans of the lumbar spine in asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am 1990 Mar;72(3):403-8.
21. Shapiro S. Cauda equina syndrome secondary to lumbar disc herniation. Neurosurgery 1993 May;32(5):743-6; discussion 746-7.

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Do you realize that you have one of the last of the Ivory Towers? You have no Senatorial responsibility. You don't have committees, you don't have to work on registration committees, admission groups and waste your time. You don't have to teach in the sense that we do in Academia. You really study.

Theodore van Brunt, former Director of the Department of Research quoting a man he described as a world-class epidemiologist from the University of California at Berkeley. As a co-investigator on a DOR project, the epidemiologist had been very impressed by the research conditions at Kaiser Permanente and at the Department of Research.