REVIEW ARTICLE

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Disorders of the Cauda Equina

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ABSTRACT

PURPOSE OF REVIEW: **Cauda equina dysfunction (often referred to as *cauda equina syndrome*) is caused by a diverse group of disorders that affect the lumbosacral nerve roots. It is important to recognize dysfunction of the cauda equina quickly to minimize diagnostic delay and lasting neurologic symptoms. This article describes cauda equina anatomy and the clinical features, differential diagnosis, and management of cauda equina disorders.**

RECENT FINDINGS: **The diagnosis of disorders of the cauda equina continues to be a challenge. If a compressive etiology is seen, urgent neurosurgical intervention is recommended. However, many people with clinical features of cauda equina dysfunction will have negative diagnostic studies. If the MRI is negative, it is important to understand the diagnostic evaluation and differential diagnosis so that less common etiologies are not missed.**

SUMMARY: **Cauda equina dysfunction most often occurs due to lumbosacral disk herniation. Nondiskogenic causes include vascular, infectious, inflammatory, traumatic, and neoplastic etiologies. Urgent evaluation and surgical intervention are recommended in most cases of compressive cauda equina syndrome. Other types of treatment may also be indicated depending on the etiology.**

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INTRODUCTION

Dysfunction of the lumbosacral nerve roots within the cauda equina can lead to symptoms of urinary retention and incontinence, constipation, bowel incontinence, sexual dysfunction, sensory changes (particularly saddle anesthesia), back pain, and lower extremity weakness; this is often referred to as *cauda equina syndrome*. The first description of cauda equina syndrome in the literature is attributed to Mixter and Barr,[1] who described a case due to disk herniation in 1934. The exact incidence of cauda equina syndrome is not known, but it is thought to be somewhere between 1 per 33,000 and 1 per 100,000,[2] which is consistent with an estimate from the United Kingdom of 1.9 per 100,000.[3] However, a study from Slovenia estimated an annual incidence of 3.4 per 1 million people.[4] The variability in the epidemiologic data may be partly because of poor consensus regarding the precise definition of cauda equina syndrome. As many as 17 different definitions were found in a literature review on the topic.[5] Still, it

is accepted that the most common cause is lumbar disk herniation, representing about 45% of cases.[5] Other etiologies include, but are not limited to, nondiskogenic structural changes, neoplasm, trauma, infection, inflammatory disorders, vascular disorders, and iatrogenic causes. Timely diagnosis is important since delays may result in worse neurologic outcomes. This article discusses the anatomy, clinical presentation, diagnosis, and management of disorders of the cauda equina.

ANATOMY

The last segment of the spinal cord, termed the *conus medullaris*, ends around vertebral level T12-L2. The nerve roots that exit via foramina below the conus medullaris (L2 through L5, S1 through S5, and the coccygeal nerve roots) create the cauda equina (FIGURE 9-1[6]), which was named for its resemblance to a horse's tail by French anatomists Andre du Laurens and Andreas Lazarius in the 1600s.[7] Damage to these nerve roots can cause symptoms of weakness, numbness, sexual dysfunction, and changes in urinary and bowel function since they control lower extremity movement and sensation (L2 through S3), external genitalia and perineal sensation (S2 through S4), sensation overlying the coccyx (S4-S5 and coccygeal nerve roots), bladder function (S2 through S4), and the external anal sphincter (S2 through S4).[8]

The cauda equina is located in the thecal sac inside the spinal canal. Similar to the spinal cord itself, it is surrounded by the vertebral bodies, intervertebral disks, and posterior longitudinal ligament anteriorly and the ligamentum flavum and spinous processes posteriorly.[9] The blood supply is not as well defined as that of the spinal cord.[10] One study found that each nerve root obtains blood supply from proximal and distal radicular arteries, with the ventral and dorsal proximal radicular arteries receiving blood supply from the anterior and posterior spinal arteries, respectively. This group also found the cauda equina to be an area of relative hypovascularity, which may make it more likely to be damaged by ischemia.[10,11]

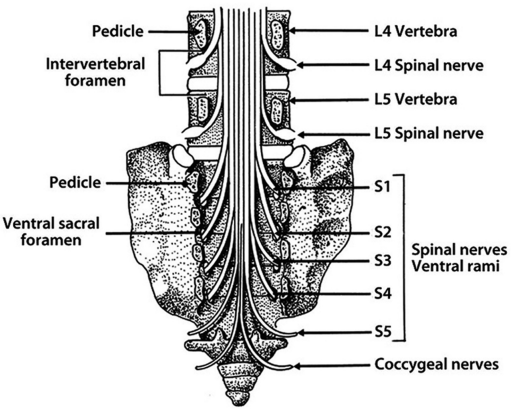

FIGURE 9-1
Cauda equina anatomy. Lumbar, sacral, and coccygeal nerve roots of the cauda equina.
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CLINICAL PRESENTATION

Despite a lack of consensus regarding the precise definition of cauda equina syndrome, the clinical features of bladder or bowel disturbance, sexual dysfunction, saddle anesthesia, weakness or numbness in the legs, and pain in the lower back or legs are well recognized. An individual patient with cauda equina dysfunction may have any of these symptoms to varying degrees, so high clinical suspicion is required. Symptoms may come on suddenly or develop over days to weeks.[12,13] Some patients even describe a more chronic course in which

back pain and sciatica are present with gradual progression of other symptoms over months.[13] The variability in time course may be because of differences in pathophysiology, with some patients having a sudden large disk herniation and others with more slowly developing canal stenosis or neoplastic disease, for example.

Examination findings that suggest cauda equina dysfunction include reduced or absent reflexes in the lower extremities, loss of perineal or lower extremity sensation, reduced rectal tone, and lower extremity flaccid weakness. Classically, this is a syndrome that affects only lumbosacral nerve roots, so no upper motor neuron signs and no signs or symptoms in the upper extremities should be present (eg, if the cauda equina dysfunction is caused by lumbosacral disk herniation). However, some disorders that affect the cauda equina may also affect the spinal cord, leading to upper motor neuron signs, or may involve the upper extremities if the specific pathologic process also causes dysfunction of cervical nerve roots.

Pain

Low back pain and leg pain are very common in cauda equina syndrome, with sciatica reported in up to 97% of patients in one retrospective review.[14] However, within the larger population of those with low back pain (about 90% of the population will experience low back pain at some point in their lives),[15] cauda equina syndrome is rare, with an estimated prevalence of 4 per 10,000.[16] Of those with cauda equina syndrome, unilateral sciatica may be more common than bilateral sciatica,[8,13,14] and the pain may worsen in the supine position.[8]

Sensory Changes

The sensory changes in cauda equina syndrome can be unilateral or bilateral, with the most common areas of involvement being the posterior thighs, buttocks, and perineum.[5] Saddle anesthesia was noted to be present in 93% of patients with cauda equina syndrome in one study.[14] It is important to specifically ask patients about perineal sensory deficits in a way that is understood by everyone, including asking about sensation during toileting activities such as wiping or defecation.[17] Sensation in the extremities should be assessed as part of the basic neurologic examination, but if cauda equina syndrome is suspected, perineal sensation, particularly to pinprick, should also be assessed.[5]

Urinary Symptoms

Symptoms of urinary dysfunction include decreased urethral sensation and urinary stream, urinary retention, and incontinence. Urinary incontinence in cauda equina syndrome is thought to be overflow incontinence that occurs secondary to the retention.[8,18] At least some of these symptoms occur in the majority of patients with cauda equina syndrome.[14] Careful history taking is important since patients may not fully recognize their symptoms.[19]

Bowel Dysfunction

Bowel dysfunction in cauda equina syndrome can range from constipation to incontinence, with the former usually preceding the latter.[17] Overall, bowel incontinence is not as commonly reported as urinary dysfunction.[14] The reason for this is not entirely clear but may be because of a reporting bias, or bowel

KEY POINTS

- **Cauda equina syndrome results from dysfunction of lumbosacral nerve roots leading to symptoms of urinary retention and incontinence, constipation, bowel incontinence, sexual dysfunction, sensory changes (particularly saddle anesthesia), back pain, and lower extremity weakness.**
- **Examination findings that suggest cauda equina dysfunction include reduced or absent reflexes in the lower extremities, loss of perineal or lower extremity sensation, reduced rectal tone, and lower extremity flaccid weakness.**
- **The sensory changes in cauda equina syndrome can be unilateral or bilateral, with the most common areas of involvement being the posterior thighs, buttocks, and perineum.**

problems may simply take longer to become apparent.[12] It is still recommended to test rectal tone,[5] although testing may have limited utility since some studies have noted poor physician accuracy in assessing rectal tone.[20] Testing for the presence of the bulbocavernosus reflex can also aid in the evaluation of anal function. This reflex, which may be absent in cauda equina syndrome, is elicited by squeezing the glans penis or clitoris and watching for contraction of the anal sphincter. Examination elements such as assessing rectal tone and the bulbocavernosus reflex should be elicited only when clinically indicated and in the presence of an appropriate chaperone. Furthermore, given the sensitive nature of these maneuvers, the clinician should explain the rationale and mechanics of these examinations and obtain verbal consent before conducting these assessments.

Sexual Dysfunction

Of patients with cauda equina syndrome, 12% to 96% will report sexual dysfunction if asked.[14,21] The symptoms of sexual dysfunction are variable and can include erectile or ejaculatory impairment, impotence, priapism, dyspareunia, and urination during intercourse.[14,19] Asking specific questions about these symptoms is important but often omitted or not documented by physicians.[5,22]

Weakness

Weakness in the lower extremities can occur in any distribution (proximal or distal, symmetric or asymmetric) in cauda equina syndrome.[5] However, it is not necessary for weakness to be present since a process affecting only the lower sacral and coccygeal roots will not cause weakness. On examination, motor strength should be assessed in detail and reflexes evaluated since lower extremity reflexes may be reduced.

DIAGNOSIS

The diagnosis of cauda equina syndrome can be challenging since many of the symptoms are common in the general population and could be secondary to other causes. For example, urinary retention and constipation are common side effects of medications. Delays in diagnosis are not infrequent, with one study citing a median of 11 days from the onset of symptoms to diagnosis and another citing an average of 9 days until diagnosis.[2,18] Furthermore, no single symptom or sign has been found to have consistently high sensitivity and specificity in diagnosing MRI-positive cauda equina syndrome.[23–25] In one study, bowel dysfunction, reduced perineal sensation, and abnormal rectal tone were the most specific findings but had low sensitivity.[17,26] That being said, measuring the postvoid residual volume may be a helpful early step (and can be done as an adjunct to the examination) in evaluating a patient with suspected cauda equina syndrome. A postvoid residual of 200 mL or more was found to have a sensitivity of 94% and specificity of 72% in predicting MRI-positive cauda equina syndrome in one study.[27]

MRI and Other Imaging

In patients with possible cauda equina dysfunction, MRI of the lumbosacral spine is the imaging modality of choice, with many studies using it as the standard for diagnosis. It allows for optimal evaluation of soft tissue pathology and can help evaluate for both diskogenic and nondiskogenic causes of cauda equina

dysfunction. If diskogenic disease is highly suspected, contrast administration can be withheld, but if the etiology is unclear, a contrast-enhanced scan can be helpful. Neoplastic, infectious, and inflammatory disorders will often show nerve root enhancement. If MRI cannot be performed, CT of the lumbar spine should be done. In a study evaluating the sensitivity and specificity of CT in cauda equina syndrome, it was found that CT was 98% sensitive and 86% specific for identifying significant spinal stenosis (using MRI as the standard).[28] If the CT does not reveal a clear etiology, CT myelography should be performed as soon as possible to help rule out a compressive etiology.

MRI-negative Cauda Equina Dysfunction

Of patients with suspected cauda equina dysfunction, 14% to 48% will have the diagnosis confirmed on MRI of the lumbosacral spine.[23] The recommended evaluation for the other patients, those who are "scan negative," is not well delineated. In these patients, less common causes of cauda equina dysfunction should be considered. For example, patients with abdominal aortic disease may have ischemia leading to cauda equina dysfunction, in which case imaging of the aorta may be helpful. In other cases, magnetic resonance angiography (MRA) of the spine may be needed to visualize vascular lesions causing cauda equina dysfunction. In addition, further neurologic workup with CSF analysis and nerve conduction studies and EMG is recommended unless a clear explanation is already evident. CSF analysis is helpful in the evaluation of infectious, inflammatory, or neoplastic disorders. Nerve conduction studies and EMG can help to confirm a pathologic preganglionic process or help to diagnose disorders such as Guillain-Barré syndrome (GBS) or chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) that can rarely present resembling cauda equina syndrome. However, it is necessary to take the timing of the presentation into consideration when performing nerve conduction studies and EMG, as some findings may not be evident in the first 2 to 3 weeks.

In patients with symptoms of cauda equina dysfunction whose scan is negative, consideration of other diagnoses that can have similar symptoms is also important. If any question exists regarding the localization based on history and examination, then imaging of the entire neuraxis (brain and spinal cord) should be considered. This can help rule out other etiologies, such as spinal cord compression, infarction, or myelitis, that may be missed by imaging only the lumbosacral spine. Other potential mimickers of cauda equina dysfunction include pudendal neuralgia (discussed in more detail below), side effects of medications such as opiates and anticholinergics, neurodegenerative disorders such as multiple system atrophy, and urologic disorders such as Fowler syndrome (urinary retention in young women associated with polycystic ovaries due to poor relaxation of the external urethral sphincter).[22,29]

In a 2018 series that reviewed 276 patients presenting with symptoms of cauda equina dysfunction, 28% had positive scans, with 86% of those due to a herniated disk; other causes included fracture, primary tumor, metastatic disease, and a cyst.[22] The study authors did not describe the full evaluation done on the patients with negative MRIs but noted that these patients were more likely to have comorbid psychiatric and functional disorders as well as chronic pain at follow-up.[22] In addition, only 1 out of 191 patients with negative MRIs was found to have an explanatory neurologic disorder (transverse myelitis in that case) at follow-up (mean follow-up time was 13 to 16 months).[22]

KEY POINTS

- **No single symptom or sign has been found to have consistently high sensitivity and specificity in diagnosing MRI-positive cauda equina syndrome.**
- **If any question exists regarding the localization to the cauda equina based on history and examination, then imaging of the entire neuraxis (brain and spinal cord) should be considered.**

NEUROSURGICAL INTERVENTION AND PROGNOSIS

Neurosurgery should be consulted immediately in a case of suspected cauda equina dysfunction due to a compressive lesion. Early surgical intervention is thought to be important in improving patient outcomes. In the literature, surgery within 48 hours of symptom onset and after 48 hours is often compared. A large meta-analysis found that surgery within 48 hours of symptom onset was associated with improved outcomes, but no difference in outcomes was seen in those who had surgery in less than 24 compared to within 48 hours.[30] However, others have questioned this conclusion,[31] and some studies have even found that timing of surgery does not significantly alter outcomes.[32,33] Because of such mixed conclusions, surgical decompression is generally recommended at the earliest time that it can be performed safely by an experienced surgeon.[33,34]

The degree of neurologic dysfunction before surgery is the most consistent prognostic factor in cauda equina syndrome.[34] Most studies divide patients into two categories based on presurgical symptoms: complete cauda equina syndrome or incomplete cauda equina syndrome. Complete cauda equina syndrome is defined as cauda equina syndrome with painless urinary retention and overflow incontinence, whereas incomplete cauda equina syndrome is defined as cauda equina syndrome with altered urinary sensation, poor stream, or a need to strain during urination. Patients with incomplete cauda equina syndrome are often cited as having a more favorable prognosis than those with complete cauda equina syndrome.[13,35] One large retrospective study (n = 136) found that all patients had some recovery of bowel and bladder function postoperatively independent of cauda equina syndrome type[36]; however, a smaller study (n = 22) found that only pain improved after surgery in those with complete cauda equina syndrome after a median follow-up of 75 months.[37] Clinical improvement can occur soon after surgery, but often improvement continues to occur slowly over months to years.[12,33] This is supported by the findings of Korse and colleagues,[38] who noted that more dysfunction was reported in studies with a shorter follow-up time. Although patients often have some improvement, persistent deficits are common. Many patients (one study cited about 50%[38]) will still have bowel, bladder, or sexual dysfunction at long-term follow-up.[14,38,39]

ETIOLOGIES OF CAUDA EQUINA DYSFUNCTION

The etiologies of cauda equina dysfunction are varied. The most common cause is lumbar disk herniation. Other causes include nondiskogenic lumbar spinal disease, vascular disorders, infectious or neoplastic disease, trauma, and inflammatory or iatrogenic disorders (**TABLE 9-1**).

Diskogenic Cauda Equina Dysfunction

Disk herniations are the most common cause of cauda equina dysfunction, occurring the majority of the time at the L4-L5 or L5-S1 level (**CASE 9-1**).[17,18] However, only about 2% to 3% of all disk herniations requiring surgery result in cauda equina syndrome.[5,35] It is thought that a history of spinal disease, such as spinal stenosis, is a risk factor for diskogenic cauda equina dysfunction since a smaller disk herniation in this setting could lead to symptoms.[8,17] Obesity is another potential risk factor for cauda equina dysfunction due to disk herniation.[40,41] Cushnie and colleagues[40] suggested an increase in epidural fat (and thus reduced spinal canal diameter) in patients who are obese as a potential

mechanism. As discussed above, MRI should be obtained to confirm the diagnosis, and urgent neurosurgical intervention is recommended.

Lumbosacral Spinal Stenosis and Other Nondiskogenic Spine Disease

The onset of symptoms in cauda equina dysfunction from a herniated disk can be sudden, but in cauda equina dysfunction secondary to lumbosacral spinal stenosis, the symptoms usually have an insidious onset because of degenerative changes to the spinal canal over time. The degenerative changes result in loss of intervertebral disk height and hypertrophy of the ligamentum flavum, leading to central stenosis and eventually compression of the cauda equina.[42] Symptoms of degenerative spinal stenosis commonly occur in the sixth or seventh decade but can occur earlier in those with a congenitally narrow spinal canal.[42] Symptoms usually start with pain that radiates from the low back and is made better by flexion of the low back. This can eventually progress and cause bladder dysfunction (and cauda equina syndrome), but this is rare.[42,43] Similar to the way that degenerative lumbar spinal stenosis can lead to cauda equina dysfunction, cases of spinal epidural lipomatosis directly causing cauda equina dysfunction have also been reported.[44,45] Spinal epidural lipomatosis may be idiopathic or secondary to obesity, chronic corticosteroid use, or other endocrinopathies.[44]

Vascular Causes

People with aortic disease, especially abdominal aortic disease, can rarely present with symptoms of cauda equina dysfunction, which is thought to be secondary to nerve root ischemia from hypoperfusion or embolization.[46] From these case reports, it is not always entirely clear if the lumbosacral roots or the lumbosacral spinal cord were actually affected. Still, in cases in which patients present with symptoms of cauda equina dysfunction and MRI of the lumbosacral spine is unrevealing, imaging of the abdominal aorta with MRA or CT angiography should be considered. Symptoms similar to cauda equina dysfunction may also be seen after a severe hypotensive event such as cardiac arrest. One study evaluating pathology specimens from patients who died after a cardiac arrest or severe hypotension found that neurons in the lumbosacral spinal cord were actually much more susceptible to ischemic injury than those in other spinal cord levels (even midthoracic), but the nerve roots were not examined.[47]

Spinal hematomas, either intradural or epidural, can cause compression of the lumbosacral nerve roots and cauda equina dysfunction. In some cases, neurologic symptoms may be preceded by hours or days of back pain.[48] Spinal hematomas can occur spontaneously; may be secondary to coagulopathy, trauma, or vascular anomalies; or may occur after intervention from a surgery or procedure. Asymptomatic spinal epidural hematomas are common after lumbosacral spine surgery, occurring in 33% to 100% of people, whereas symptomatic postoperative spinal epidural hematomas are rare, occurring in only 0.1% to 0.2% of cases.[49] However, in one large retrospective study of 15,668 patients in whom the etiology was known, 6.3% of cauda equina syndrome cases were listed as secondary to postoperative hematoma.[50] For this reason, clinicians should have a high index of suspicion and patients should be rescanned after surgery if any concern exists.

One case report describes engorgement of the epidural venous plexus occurring postoperatively, leading to dural sac shift and cauda equina dysfunction.[51] In this particular case, the dural sac shift was treated with laminoplasty and had a good clinical outcome.[51] Epidural venous plexus

KEY POINTS

- Neurosurgery should be consulted immediately in a case of suspected cauda equina dysfunction due to a compressive lesion.
- The degree of neurologic dysfunction before surgery is the most consistent prognostic factor in cauda equina syndrome.
- Disk herniations are the most common cause of cauda equina dysfunction, occurring the majority of the time at the L4-L5 or L5-S1 levels.

enlargement (seen as signal flow voids on MRI of the lumbosacral spine) leading to cauda equina dysfunction can also occur secondary to occlusion of the inferior vena cava.[52] This most commonly occurs because of thrombosis in the inferior vena cava but can occur secondary to occlusion from a mass or from pregnancy. Occlusion of the inferior vena cava can be visualized on MRI of the lumbosacral spine but is usually confirmed with Doppler ultrasound. In one case series, all

TABLE 9-1

Causes of Cauda Equina Dysfunction

Diskogenic and nondiskogenic structural spinal disease

- Lumbosacral disk herniation
- Lumbosacral spinal stenosis
- Spinal epidural lipomatosis

Vascular

- Spinal hematoma
- Aortic disease (eg, dissection, thrombosed aneurysm)
- Dural arteriovenous fistula

Neoplastic

- Myxopapillary ependymoma
- Leptomeningeal metastasis
- Skeletal metastases (eg, lung, breast, prostate cancer)
- Schwannoma
- Paraganglioma
- Neurofibroma
- Meningioma
- Non-Hodgkin lymphoma
- Chordoma
- Chondrosarcoma
- Ewing sarcoma
- Hemangioblastoma
- Dermoid tumor
- Glioma
- Drop metastasis from primary brain tumors

Inflammatory

- Sarcoidosis
- Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP)
- Chronic immune sensory polyradiculopathy
- Acute inflammatory demyelinating polyradiculoneuropathy (AIDP)
- Vasculitis

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patients had resolution of their neurologic symptoms after treatment of the vena cava occlusion.[51] Similarly, a spinal dural arteriovenous fistula can lead to or mimic cauda equina syndrome. This disorder can be difficult to diagnose; contrast-enhanced MRA of the spine can help localize the fistula, and digital subtraction angiography is used to confirm the diagnosis.[53] For more information regarding spinal dural arteriovenous fistulas and other vascular disorders of the

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- Ankylosing spondylitis
- Graft versus host disease

Infectious

- Spinal epidural abscess
- Lyme disease
- Elsberg syndrome (herpes simplex virus type 2)
- Cytomegalovirus
- Varicella-zoster virus
- Human immunodeficiency virus (HIV)
- Herpes simplex virus type 1
- Epstein-Barr virus
- Syphilis
- Tuberculosis
- Cysticercosis
- Schistosomiasis
- Hydatid cysts
- Cryptococcosis
- Nocardiosis
- Brucellosis
- Tick-borne encephalitis

Iatrogenic

- Radiation induced
- Arachnoiditis
- Epidural pneumorrhachis (entrapment of air/gas within spinal column)
- Intrathecal cytarabine or methotrexate
- Complication of spinal anesthesia

Other

- Trauma
- Extramedullary hematopoiesis
- Calcifying non-neoplastic pseudoneoplasm

spinal cord, refer to the article "Vascular Myelopathies" by Nicholas L. Zalewski, MD,[54] in this issue of *Continuum*.

Infectious Causes

Constitutional symptoms, such as fevers, night sweats, and weight loss, should lead to consideration of an infectious etiology of cauda equina dysfunction in the appropriate clinical setting. Spinal epidural abscesses are a potential infectious cause of cauda equina dysfunction. Back pain at the level of the abscess (present in 75% of patients) and fever (present in 50% of patients) are the most common symptoms.[55] The symptoms may be present for days or up to 2 months in some cases. Predisposing factors include, but are not limited to, diabetes, alcohol use disorder, human immunodeficiency virus (HIV), spine abnormality or recent intervention, sepsis or other current infection, and IV drug use.[55] Two-thirds of cases are caused by *Staphylococcus aureus*; other common pathogens include *Staphylococcus epidermidis*, *Escherichia coli*, and *Pseudomonas aeruginosa*.[55] Infection from other pathogens, such as tuberculosis, fungi, or parasites, is rare.[55–57] If infection is present, erythrocyte sedimentation rate and C-reactive protein will be elevated, and blood cultures should be obtained. Diagnosis is

CASE 9-1

A 35-year-old man with a history of chronic back pain presented to the emergency department with worsening back pain, lower extremity and saddle paresthesia, urinary retention, and a feeling of weakness in his feet. He said he had been playing basketball 4 days prior and "twisted" his back, causing worsening of his typical back pain. He thought the pain was improving, but shortly before coming to the emergency department, he stood up and felt a sudden worsening of low back pain and developed numbness and tingling in his legs and perineum. He felt the need to urinate but had been unable to do so; he denied bowel symptoms. He had no other significant past medical history except for morbid obesity.

Neurologic examination revealed normal strength, left greater than right perianal numbness and patchy distal lower extremity numbness to pinprick, a reduced left ankle reflex, and normal rectal tone. MRI of the lumbosacral spine (FIGURE 9-2) revealed a large disk herniation at L5-S1 compressing the cauda equina. Underlying central spinal stenosis existed secondary to significant ventral epidural fat.

Emergent L5-S1 microdiskectomy was performed within 24 hours of symptom onset. At a follow-up visit 8 months later, he reported a continued pins and needles sensation in his feet, saddle paresthesia, and a feeling of weakness in his feet that was still gradually improving. He denied any residual bladder dysfunction.

made with MRI with and without contrast or CT myelography if MRI cannot be performed, as both are more than 90% sensitive.[55] CSF evaluation is not recommended (unless myelography is needed) since the culture yield is low and a small risk exists of causing meningitis or a subdural infection from the lumbar puncture. In most cases, emergency decompressive surgery and antibiotic treatment are recommended.[55]

In a patient with HIV, usually with a CD4 count of less than 100 cells/mm^3, an infectious cause of cauda equina dysfunction must be considered. An acute polyradiculopathy due to involvement of lumbosacral nerve roots occurs in about 2% of patients with HIV.[58] In this population, this is most frequently caused by cytomegalovirus (CMV), herpes simplex virus type 1 (HSV-1) or type 2 (HSV-2), varicella-zoster virus (VZV), or tuberculosis.[59] HIV itself is also a rare cause of polyradiculopathy or polyradiculoneuropathy, especially at the time of seroconversion. This is more likely to present as GBS or pure motor polyradiculopathy than as cauda equina syndrome.[60]

CMV (usually seen in patients with HIV or other patients who are immunocompromised, as previously mentioned) can cause cauda equina dysfunction through invasion of the lumbosacral nerve roots, with clinical signs

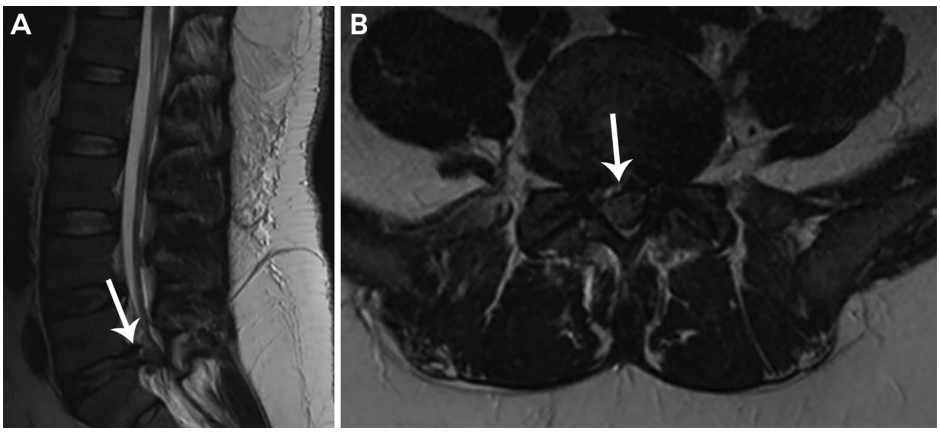

FIGURE 9-2

Imaging of the patient in CASE 9-1. Sagittal (*A*) and axial (*B*) T2-weighted MRIs of the lumbosacral spine show a large extruded disk fragment (*arrows*) compressing the thecal sac. The prominent epidural fat seen in this patient is not shown in these T2-weighted images.

COMMENT

This case illustrates a typical case of cauda equina dysfunction secondary to lumbosacral disk herniation at the L5-S1 level. This patient had a risk factor of obesity with central canal stenosis that was already present to some degree because of epidural lipomatosis. None of the hospital notes for this patient commented on sexual dysfunction, but this should be discussed with every patient in whom cauda equina dysfunction is suspected. Surgery was performed within 24 hours of symptom onset in this patient. At follow-up, the patient reported improvement but still had some neurologic symptoms, which is not uncommon.

and symptoms of areflexia, weakness, sensory disturbance, and urinary retention. In some patients, the infection can spread to affect the upper extremities and cranial nerves as well. Often MRI of the spinal cord will show contrast enhancement of the nerve roots and meninges and T2-hyperintense lesions in the cord itself.[61] CSF will often have a mixed pleocytosis, low glucose, and protein elevation. CMV DNA found in the CSF is specific, and patients should be treated with IV ganciclovir or foscarnet, or both.[61]

HSV-2 and VZV can cause a presentation similar to CMV. Elsberg syndrome is an acute or subacute presentation of lumbosacral radiculitis, often with myelitis, that is associated with HSV-2 infection. A retrospective review of patients from the Mayo Clinic with Elsberg syndrome found that Elsberg syndrome likely accounts for about 10% of patients with a clinical presentation of cauda equina syndrome and myelitis.[62] Out of 30 patients, only two had a preceding genital herpes infection. Other clues to the diagnosis include an acute presentation (potentially distinguishing it from neoplastic or inflammatory causes), smooth (rather than nodular) nerve root enhancement on MRI, and, if present, spinal cord lesions that are multiple and discontinuous. HSV can rapidly clear from the CSF, so a high clinical suspicion should exist even in the absence of positive CSF polymerase chain reaction (PCR). VZV may be more likely than HSV-2 when more lesions are present higher in the spinal cord, in the presence of a longitudinally extensive lesion, or when encephalitis is present. Both VZV and HSV-2 are treated with acyclovir, and treatment should be considered even when CSF studies are nondiagnostic.[62]

Polyradiculopathy can be seen in half of patients with peripheral nerve involvement due to Lyme disease. Pain and sensory loss are the most common symptoms and typically have an acute onset within the first couple of months of infection.[63] This disorder may be underrecognized since the symptoms can be self-limited and CSF may not be abnormal (although a lymphocytic pleocytosis and elevated protein are commonly seen). On EMG, abnormalities may be seen in paraspinal muscles; however, this should be true in most polyradiculopathies, so this finding is not specific. Previous rash, arthralgia, facial palsy, or a known environmental exposure can be helpful in making the diagnosis.[63]

Other potential infectious causes of cauda equina dysfunction include tuberculosis,[64] cryptococcosis,[65] neurosyphilis,[66] schistosomiasis,[67] cysticercosis,[68] Epstein-Barr virus,[69] nocardiosis,[70] brucellosis,[71] and tick-borne encephalitis.[72] For more information on infectious causes, refer to the article "Infectious Myelopathies" by Michel Toledano, MD,[73] in this issue of *Continuum*.

Neoplastic Causes

In one large retrospective study, cauda equina syndrome was secondary to tumor in 3.1% of patients (478 of 15,668 patients with a reported etiology).[50] Neoplastic disease can lead to cauda equina dysfunction by direct compression from primary or metastatic lesions or secondary to nerve root invasion from meningeal-based disease. It is more common for cauda equina dysfunction from tumors to present in a subacute or chronic fashion compared to cauda equina dysfunction resulting from other causes.

Ependymomas are the most common primary tumor of the spinal cord in adults and the most common primary tumor to affect the cauda equina. Specifically, myxopapillary ependymomas are more likely to occur below the conus medullaris than other types of ependymomas.[74] These tumors grow slowly, arising from the ependymal cells in the central canal. On MRI, they typically enhance with

contrast and appear to be intradural-extramedullary when affecting the cauda equina. Other tumors that can rarely lead to cauda equina dysfunction or involve the lumbosacral nerve roots include schwannomas, neurofibromas, meningiomas, hemangioblastomas, dermoid tumors, chordomas, and gliomas.[74-76] Cauda equina dysfunction can also occur secondary to compression from metastatic disease, most frequently from lung cancer, breast cancer, prostate cancer, or multiple myeloma.[74,75]

Leptomeningeal disease can also lead to cauda equina dysfunction. This is often due to lung or breast cancer but has been reported from a large range of cancers. Rarely, invasion of the lumbosacral nerve roots can occur with leukemia[77] or lymphoma, most commonly related to diffuse large B-cell lymphoma.[78] Drop metastases from brain tumors such as medulloblastomas, ependymomas, germinomas, choroid plexus carcinomas, and glioblastomas can also lead to leptomeningeal disease.[74] MRI of the spine with and without contrast may show nodular enhancement along the nerve roots or meninges. At least 10.5 mL of CSF should be sent for cytology, and the test should be repeated if negative to minimize false-negative results.[79] Flow cytometry should also be conducted when hematologic malignancy is suspected to increase the sensitivity.[80]

Inflammatory Causes

Sarcoidosis presenting with cauda equina dysfunction is rare, but it is still likely the most common inflammatory cause of cauda equina syndrome (CASE 9-2).[75,82] About 5% of patients with sarcoidosis will have nervous system involvement (neurosarcoidosis), with only about one-fifth of these cases affecting the peripheral nerves or spinal roots.[83] Of those with neuropathy, polyradiculoneuropathy was the most common pattern in one series, seen in 22 of 57 patients. The most commonly reported symptoms were pain and sensory symptoms, and only 8 of the 57 patients reported bowel or bladder symptoms.[83] Other symptoms commonly seen in this case series that may serve as diagnostic clues included fatigue, fever, unexplained weight loss, arthralgia, and skin and eye involvement. The disease course was most commonly acute or subacute, but some patients had an insidious onset.[83] MRI of the lumbosacral spine in neurosarcoidosis may show nodular thickening of the nerve roots and enhancement. CSF findings are nonspecific; protein elevation is the most common finding, whereas a pleocytosis or reduced glucose are less common. As in any case of suspected neurosarcoidosis, CT of the chest is important to look for pulmonary involvement. Positron emission tomography (PET)-CT may be more sensitive to detection of systemic sarcoid and can be considered if CT of the chest is negative. If the diagnosis is still unclear, biopsy is necessary (in cases of polyradiculoneuropathy, biopsy would likely be of the leptomeninges or nerve and muscle).

Peripheral nerve vasculitis presenting with cauda equina dysfunction has been described in association with systemic lupus erythematosus.[84] Cauda equina syndrome has also been reported in association with ankylosing spondylitis and graft versus host disease.[85,86] Other inflammatory disorders to consider include GBS,[87] CIDP,[88] and chronic immune sensory polyradiculopathy (CISP), since lumbosacral root involvement can predominate the clinical picture and bowel and bladder dysfunction can rarely occur.

Iatrogenic Causes

As discussed above, spinal hematomas occurring after surgery may cause cauda equina syndrome. Epidural injections of anesthetics and analgesics have also

KEY POINTS

- Constitutional symptoms, such as fevers, night sweats, and weight loss, should lead to consideration of an infectious etiology of cauda equina dysfunction in the appropriate clinical setting.
- Elsberg syndrome likely accounts for about 10% of patients with a clinical presentation of cauda equina syndrome and myelitis.
- Myxopapillary ependymomas are the most common primary tumor to affect the cauda equina.
- Sarcoidosis is likely the most common inflammatory disorder that can present with cauda equina dysfunction.

CASE 9-2

A 45-year-old man with no significant past medical history presented with several weeks of worsening low back pain, progressive lower extremity weakness and sensory changes, saddle anesthesia, and urinary retention. He had also noticed shortness of breath and arthralgia over the past 2 months.

Neurologic examination showed normal cranial nerves and upper extremity strength, 4/5 strength diffusely in the lower extremities, reduced lower extremity reflexes, and reduced sensation in the perineum and in a length-dependent pattern in the lower extremities. MRI of the lumbosacral spine showed nodular enhancement in the cauda equina and in the vertebrae, similar to the MRI example in FIGURE 9-3.[81] CSF analysis showed a protein of 101 mg/dL, white blood cell count of 50 cells/mm^3, low glucose, and a normal angiotensin-converting enzyme level. Cytology was negative for malignant cells, and all infectious studies were negative. Nerve conduction study/EMG was also performed and showed abnormal spontaneous activity in the lumbar paraspinal muscles and a length-dependent sensorimotor axonal neuropathy. CT of the chest showed hilar lymphadenopathy, which was ultimately biopsied and consistent with sarcoidosis. He was treated with IV methylprednisolone followed by oral prednisone and started on mycophenolate mofetil.

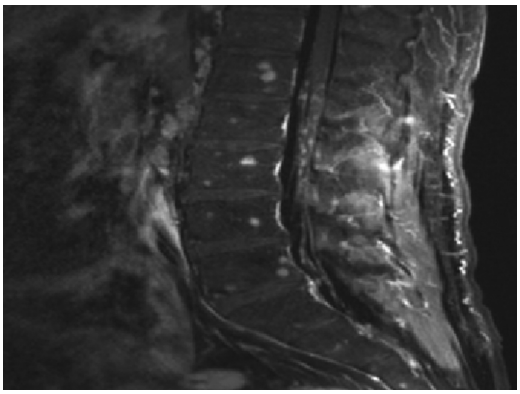

FIGURE 9-3
Nodular enhancement of the cauda equina and vertebrae.
Reprinted from Hoyle JC, et al, Neurohospitalist.[81] © 2014 SAGE Publications.

COMMENT

The progression of symptoms over several weeks in this patient was suspicious for an inflammatory process, and the symptoms of arthralgia and shortness of breath were clues to the diagnosis. The possibility of infection should be evaluated fully, especially since CSF findings in sarcoidosis can appear similar to an infection, with a high white blood cell count and low glucose. In addition, given the CSF findings and nodular enhancement on MRI, it was also necessary to ensure that no signs of malignancy were present in the CSF or elsewhere in this patient. The diagnosis of sarcoidosis is made from tissue biopsy. If no lung involvement had been found in this patient, then biopsy of the meninges would have been considered.

been associated with cauda equina dysfunction. In some cases, a subarachnoid (rather than epidural) injection was performed, inadvertently leading to lumbosacral polyradiculopathy.[89] In other cases in which an epidural injection was performed correctly, it is thought that patients who developed cauda equina dysfunction may have been at greater risk because of a history of spinal stenosis or adhesive arachnoiditis.[89,90] A possible reason for this is that edema caused by the injection may lead to compression of the lumbosacral nerve roots.[89] Adhesive arachnoiditis itself may also present with cauda equina dysfunction.[91] In adhesive arachnoiditis, the arachnoid is thickened and scarred and may lead to nerve ischemia.[75] It is most commonly caused by myelographic contrast agents or other intrathecal injections and spinal operations.[91] The treatment of adhesive arachnoiditis is usually conservative, with medications and physical therapy; surgical treatment is sometimes used, depending on the specific case.[92]

Radiation can also cause lumbosacral polyradiculopathy. This typically occurs about 6 years after exposure but can occur as soon as several months after or as late as 25 years after radiation.[75] This is mostly reported as a pure motor syndrome, with less than one-third of patients reporting sensory symptoms and many without pain. MRI may sometimes show nodular nerve root enhancement, making it difficult to distinguish from a neoplastic meningitis.[75,93,94]

Other Causes

Trauma, especially from motor vehicle accidents, falls, and gunshot wounds, is a potential cause of cauda equina dysfunction. Often this is because of low lumbar or transverse sacral fractures.[95,96] No prospective trials have evaluated the best treatment for such fractures. A 2018 meta-analysis of case reports and retrospective case series that included a total of 521 patients with transverse sacral fractures found that surgically treated and nonoperatively treated patients showed no significant difference in neurologic recovery. In patients who were surgically treated, fracture fixation in addition to decompression resulted in greater neurologic recovery.[97]

Several case reports have described extramedullary hematopoiesis as a cause of cauda equina dysfunction. Extramedullary hematopoiesis is hematopoiesis that occurs outside of the bone marrow, which is normal during embryonic and fetal development. However, it can occur as a pathologic process in people with thalassemia and, less commonly, myelofibrosis, polycythemia, and sickle cell disease. The spleen, liver, and lymph nodes are common sites of extramedullary hematopoiesis, but rarely extramedullary hematopoiesis can occur in the spinal canal, leading to neurologic symptoms.[98] On MRI of the spine, this may appear as multiple rounded masses or a single mass in the epidural space, typically contrast enhancing with variable signal characteristics.[98,99] No clear treatment of choice has been identified, but surgical decompression, transfusion, hydroxyurea, and radiation therapy have all been tried.[98]

Although not technically a disorder of the cauda equina, pudendal neuropathy can closely mimic cauda equina dysfunction since the pudendal nerve originates from the S2 through S4 nerve roots and innervates the perineum.[100] It most commonly presents as a neuralgia, with perineal pain that is worse with sitting because of pressure on the perineum.[75] Sensory symptoms of numbness can occur, as can bowel, bladder, and sexual dysfunction if the neuropathy is bilateral.[75,101] A key difference between pudendal neuropathy and true cauda

KEY POINTS

- **Trauma, especially from motor vehicle accidents, falls, and gunshot wounds, is a potential cause of cauda equina syndrome, often because of a low lumbar or transverse sacral fracture.**
- **Although not technically a disorder of the cauda equina, pudendal neuropathy can closely mimic cauda equina syndrome since it originates from the S2 through S4 nerve roots and innervates the perineum.**

equina syndrome is that the pain in cauda equina syndrome is more commonly in the lower back and legs and less often described as in the perineum. Pudendal neuralgia occurs secondary to compression or entrapment from pelvic fractures, bicycle seats, neoplasm, childbirth, or muscle or iatrogenic injury.[101-103] The diagnosis is challenging since objective findings can be difficult to demonstrate. Nerve conduction studies of the pudendal nerve are not often performed and are of unclear sensitivity and specificity for identifying a nerve injury.[75] Denervation of the external anal sphincter would be supportive of nerve injury; however, external anal sphincter EMGs are technically challenging, and if the EMG is normal, pudendal nerve dysfunction still cannot be ruled out (the same is true when using these techniques to assess for cauda equina dysfunction in general). MRI, especially MRI neurography, may be helpful in some cases.[104] Symptoms of pudendal neuropathy can be treated symptomatically with medications or sometimes with pudendal nerve block.[105] Surgery should generally not be performed unless a clear etiology is identified or if the patient is severely affected.[75,106]

CONCLUSION

Cauda equina syndrome is a neurologic emergency requiring timely diagnosis and treatment. Important clinical characteristics include radiating low back pain, perineal and lower extremity sensory disturbances, urinary and bowel dysfunction, and lower extremity weakness. Although diskogenic causes account for most cases, nondiskogenic etiologies must also be considered. An understanding of cauda equina anatomy; clinical presentation; and the value of diagnostic studies such as MRI, CSF evaluation, and nerve conduction studies and EMG is necessary to provide the best chance for neurologic recovery.

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