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# In Shortly about Cauda Equina Syndrome

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**Abstract**

*Cauda equina syndrome affects a group of nerve roots called Cauda equina (in latin "horse tail"). These nerves are located at the lower end of the spine in the lumbosacral part. They send and receive messages for the legs, feet and pelvis. Trauma, overexertion, violent injuries, and some diseases and conditions result in compression of the roots of these nerves. There is severe lower back pain, loss or change of sensation in the legs, buttocks, difficulty in urinating. Surgical treatment must be performed quickly to prevent permanent damage, such as leg paralysis, bladder and bowel dysfunction, sexual function, or other problems.*

**Keywords:** *Cauda equina, Spinal cord, Pathology, Treatment*

## Introduction

Cauda equina syndrome (CES). The signs and symptoms of lower extremity weakness and pain developing acutely after heavy lifting should raise suspicion for a herniated intervertebral disc, which is the commonest cause of CES [1]. Depending on the location and degree of the herniation, a combination of nerve root and cord could be impinged. The spinal cord typically terminates at the L1/2 level in an adult. Caudal to this, the cord continues as the cauda equina. If the spinal cord itself is compressed by a herniated disc, myelopathic signs such as hyperreflexia, Hoffmann's sign, Babinski's sign and clonus would be expected.

When the cauda equina is compressed by a very large central disc, patients can present with a combination of urinary retention, bowel incontinence, loss of anal tone and saddle anaesthesia. These findings are related to the compression of the somatic and autonomic fibres from nerve roots S2–4, which control the detrussor muscle of the bladder, external and internal anal sphincters and perineal sensation. The caliber of the spinal canal can also be reduced by osteophytes and soft tissue such as ligamentum flavum hypertrophy. In these cases, a smaller degree of herniated nucleus pulposus will produce marked signs and symptoms.

CES is a neurosurgical emergency. The goal is to prevent irreversible loss of bowel and bladder function and motor function of the lower extremities. While CES is primarily a clinical diagnosis, further imaging will be required to aid decision-making and operative planning. Ultimately, an MRI of the lumbar spine will help guide the neurosurgeon.

A multitude of alternative diagnoses may masquerade as CES – stroke, vascular claudication, deep venous thrombosis, muscle cramps and peripheral neuropathy. A good history and examination will help differentiate these from true CES. If there is a concern for bony fracture from trauma, a non-contrast CT computer tomography) should be performed. If an unstable fracture is found on initial CT, spinal precautions should be maintained until definitive management. The scan time is in the order of seconds to minutes and hence will be available for inspection much quicker than an MRI (magnetic resonance imaging), which can take 30–60 minutes. The lumbar spine is often scanned in isolation, but consideration should be given to extending the imaging to the thoracic and cervical spine as well as the brain in order to rule out a higher level lesion in cases where clinical correlation is less than expected.

## Spinal Cord

As a fetus, the spinal cord spans the entire length of the spine [2]. As the infant ages, the spine lengthens at a faster rate compared to the growth of the spinal cord. As a result, the spinal cord ends at the L3 level in early childhood, and in adulthood the spinal cord extends from the foramen magnum to the L1-2 level.

As the spinal cord approaches L1 it tapers off into the conus medularis and eventually becomes the cauda equina, or "horse's tail," a bundle of nerve roots that float relatively freely in the cerebrospinal fluid (CSF) of the spinal canal. A spinal anesthetic performed below L1 would be in the territory of the cauda equina and decrease the potential for spinal cord trauma as the needle theoretically displaces the nerve roots to the side.

There are 31 pairs of spinal nerves (8 cervical, 12 thoracic, 5 lumbar, and 5 sacral). The nerve roots exit the spinal column through bilateral intervertebral foramen. In the cervical spine the nerve roots exit above their corresponding vertebral body. At the C7 body, the C7 root exits above and the C8 root exits below (between C7 and T1). Starting at T1, the nerve roots then exit below their respective vertebral bodies.

The dural sac, as well as the subarachnoid space, extends below the cauda equina to S2 in adults and S3 in children. The dura often continues to sheath the nerve roots as they exit the spinal canal into the intervertebral foramen. As such there is a risk of subdural or subarachnoid injection even when approaching from a caudal or transforaminal approach. Although the dura mater extends to S2, the pia mater continues as a thin strand of tissue to form the filum terminale, which connects the conus to the coccyx.

The adult spinal cord begins at the level of the cervicomedullary junction as the spinal cord passes through the foramen magnum of the skull to join the medulla of the brainstem [3]. The spinal cord is surrounded by the three layers of the meninges which are contiguous with the cranial compartment. The dura mater is the outermost, tough layer, followed by the arachnoid that loosely invests the spinal cord and contains space for the cerebrospinal fluid (CSF). The pia mater is tightly adherent to the spinal cord itself. The spinal cord tapers to an end at the conus medullaris posterior to the L1–L2 vertebral body.

The spinal cord contains white matter tracts consisting of axons originating from upper motor neurons (UMNs) within the brain. These axons synapse with lower motor neurons (LMNs) within the gray matter of the spinal cord that send axons out in the form of nerve roots and the cauda equina. Lesions of the UMNs or their axons result in spasticity, while lesions of the LMNs or their axons result in flaccid paralysis. Generally speaking, UMN lesions carry a worse prognosis than LMN lesions, as nerve roots have better capacity for repair than the spinal cord. Further, because LMN have overlapping motor and sensory distributions, minor injuries may have limited sequela. After exiting the spinal canal, the nerve roots in the cervical and lumbar regions fuse as the cervical and lumbar plexuses before separating again as specific nerves.

All spinal nerves are classified as mixed nerves because they contain the axons of both sensory and motor neurons [4]. However, the motor and sensory fibers are separate inside the vertebral canal, where each spinal nerve divides into two branches. The posterior branch, called the dorsal root, contains the axons of sensory neurons that bring information to the spinal cord. The anterior branch, called the ventral root, contains the axons of motor neurons that carry commands to muscles or glands. Each dorsal root of a spinal nerve is also associated with a dorsal root ganglion. A ganglion is any collection of neuron cell bodies in the PNS. In a dorsal root ganglion, the cell bodies are those of sensory neurons. The cauda equina consists of spinal nerve roots that extend inferior to the tip of the spinal cord. The spinal nerves they form leave the vertebral canal between vertebrae in the lower lumbar and sacral regions.

Spinal shock, which must always be a diagnosis of exclusion (to the extent that the physician must prove that hemorrhage does not explain the patient's hypotension), may be noted immediately after injury to the spinal cord [5]. Most patients present with systolic blood pressures in the range of 70 to 90 mm Hg and have warm extremities and a normal or only slightly elevated pulse, neither of which is an expected finding in patients with hemorrhagic shock. Additionally helpful findings include neck pain; flaccid areflexia, including the rectal sphincter; diaphragmatic breathing or apnea; priapism; a sensory level; and facial gesturing in response to painful stimuli above the level of the clavicles but not below them. The

institution of pressors, if required, may be helpful and should be instituted in patients with spinal shock after blood loss is excluded or corrected.

## Structure

The major clinically relevant tracts of the cord are the dorsal columns (ascending), which convey tactile discrimination, vibration, and joint position sense; the spinothalamic tracts (ascending), which convey pain, temperature, and crude touch; and the corticospinal tracts (descending), which convey fibers used for motor control [6]. The spinal cord ends between the first and second lumbar vertebrae in adults. This distal area is called the conus medullaris, and its continuation as the filum terminale is composed of connective tissue that attaches to the coccyx. The cauda equina is a collection of nerve roots that begins at the end of the spinal cord and exits from the third lumbar vertebra to the fifth sacral vertebra. The spinal cord is protected from the bony canal by a layering of fatty connective tissue and by the meninges. The three meninges from inner to outer are pia, arachnoid, and dura. The subarachnoid space contains cerebrospinal fluid and separates the pia from the arachnoid.

Other structures that protect the spinal cord can be divided based on their location relative to the cord. The intervertebral foramen is the opening between the pedicles of adjacent vertebrae for the spinal nerve to pass through. Spinal nerves are composed of dorsal and ventral roots. The first seven pairs of cervical spinal nerves exit above the same-numbered vertebral bodies, whereas all the subsequent nerves exit below the same numbered vertebral bodies because of the presence of eight cervical spinal cord nerves but only seven cervical vertebrae. Intervertebral disks separate the vertebral bodies and act as shock absorbers. The avascular disk consists of an eccentrically located nucleus pulposus and the surrounding annulus fibrosus. The nucleus pulposus is a semi gelatinous mass composed of 70–80% water. The water content declines with advancing age, and by the sixth or seventh decade of life, the nucleus has been transformed to fibrocartilage.

Cauda equina syndrome is diagnosed when acute neurological impairment occurs in those structures supplied by the sacral nerve roots, notably causing bowel or bladder dysfunction or perineal (“saddle”) anesthesia [7]. This is a surgical emergency that requires urgent diagnosis and treatment. The most common causes are large paracentral disk herniations and tumors. Even with prompt decompression, some patients do not recover completely.

## Compression

The clinical presentation of epidural spinal cord compression is well known and depends on the level of spinal involvement [8]. Axial pain is the most common presenting symptom (prodromal phase), occurring in 95% of adults and 80% of children with epidural spinal cord compression. Therefore, spinal cord compression should be considered in any patient with cancer and axial pain. The local pain corresponds to the site of the lesions and is described as dull and aching. Tenderness over the affected spinal element is usually readily elicited. Approximately 15% of patients will develop paraplegia despite a long duration of painful symptoms (compressive phase), because spinal cord compression was not anticipated. Pain may persist for several weeks or months before symptoms of radiculopathy are manifested. Cervical or lumbar disease usually but not always presents as unilateral radiculopathy, whereas thoracic disease produces bilateral symptoms resulting in a band like distribution of pain. Radicular pain may be accompanied by sensory or motor loss, as determined by the involved nerve root, and may be easily confused with disk herniation. Pain is usually worse at night and is aggravated by movement, coughing, or the Valsalva maneuver. Because midthoracic back pain is less likely to be due to benign causes,any patient localizing pain and tenderness to this area regardless of a history of malignancy should be evaluated carefully.

Neurologic deficits seen in spinal cord compression usually begin with motor impairment. These are seen more commonly in the distal part of the body or the lower extremities owing to the greater frequency of thoracic and lumbar spine involvement. Anterior spinal cord compression is more common than posterior involvement. Accordingly, patients usually have more motor than sensory disability, at least in the early stages. Sensory impairment follows, parallels the development of motor deficit, and is present in half of patients at the time of diagnosis of spinal cord compression. Autonomic dysfunction occurs later and is present in half of cases.

The neurologic deficit is caused either by mechanical compression by the tumor on the spinal cord or cauda equina or by destruction of a vertebral body sufficient

to make it collapse and compress the spinal cord. Once spinal cord compression occurs, progression may be very rapid.

## Pathology

The conus medullaris is the most distal part of the spinal cord, adjacent to the vertebral body of L1 in most adults, and the cauda equina ("the horse's tail") is the sheaf of nerve roots which lies below it, the roots running distally to the intervertebral foramina of the lower lumbar, sacral, and coccygeal vertebrae [9]. Local pathology may involve both structures or, less likely, affect them individually. Although this anatomical distinction may be attempted on clinical grounds, it is often difficult and of little practical value.

Recognized causes of cauda equina and conus medullaris syndromes include

- Central disc herniation. If acute, this is a neuro surgical emergency.
- Tumor: primary (e.g., ependymoma, meningioma, schwannoma); metastasis.
- Hematoma.
- Apscess.
- Lumbosacral fractures.
- Inflammation (e.g., cauda equina syndrome in neurosarcoidosis; rare).
- Ankylosing spondylitis (cauda equina syndrome in association with dorsal arachnoid diverticula: rare).

Cauda equina syndrome is an acute polyradiculo neuropathy of the conus medullaris and lumbosacral nerve roots [10]. Cauda equina syndrome manifests as lower extremity neuropathic pain, sensory disturbance, bowel and bladder dysfunction, and asymmetric BLE weakness. There are many causes, including neoplastic invasion, cytomegalovirus infection, and acute compression, such as from an epidural hematoma. Evaluation includes emergent imaging by MRI, often followed by lumbar puncture to evaluate the CSF.

Cauda equina syndrome (CES) may result from any lesion that compresses CE nerve roots [11]. The most common causes of CES are lumbar stenosis, spinal trauma (including vertebral fractures), disc herniation, severe spondylolisthesis, tumors, spinal infection, complications of epidural injections, mass effect from a spinal hemorrhage, and tethered cord. Tumors of the spinal cord are a rare cause of CES or conus medullaris syndrome (CMS). The incidence of spinal cord tumors is 0.5–2.5 cases per 100,000.

Extradural spinal cord tumors comprise 55% of spinal cord tumors, intradural–extramedullary tumors account for 45%, and intramedullary tumors account for only 5%. Most of the extradural tumors come from tumor metastasis; most intradural–extramedullary tumors are schwannomas, neurofi bromas, or menigiomas. The intramedullary (within the cord) tumors are usually ependymomas (60%) or astrocytomas (33%).

## Treatment

The treatment of any trauma patient begins with basic life support measures [12]. Spinal immobilization must be maintained with a cervical collar and back board while en route to the ED, and strict spinal precautions must be observed once the patient arrives at the ED. If a patient with suspected cervical injuries requires intubation, it should be performed by personnel with experience performing in-line intubation using the Sellick maneuver, or by using fiberoptic guidance if it is readily available. The neurological examination should be performed on a nonintubated, nonsedated patient, if possible. For any patient with evidence of neurological deficit, methylprednisolone is given with an initial bolus dose of 30 mg/ kg over 1 hour, and then continued at 5.4 mg/kg/hr x 23 hours. Emergent surgical decompression and stabilization is considered for any patient with MRI findings of acute spinal cord compression by bone fragmentation, herniated or ruptured disk, spinal epidural hematoma, or cauda equina syndrome. Patients with fracture-dislocations will be placed in traction using Gardner-Wells tongs for attempted preoperative closed reduction. Once reduction is accomplished, the patient is usually operated on within a matter of hours. If closed reduction cannot be accomplished, then stabilization surgery is performed with hopes of intraoperative reduction at the time of surgery. Patients with stable neurological examination findings will usually be allowed to stabilize for a few days before surgical intervention is planned. Fractures that are nonsurgical are usually treated with bracing for 6–12 weeks, depending on the location and severity.

With any spinal cord injury, the success of the treatment depends on the timing from diagnosis to intervention, and the degree of injury sustained. Patients with incomplete injuries will usually have some improvement in their neurological function, where as patients with initial complete injuries are unlikely to regain any function below the level of injury. Most of the patients from both groups will require long-term rehabilitative treatment. The most common long-term complications in patients with spinal cord injury are pressures ulcers, occurring at a 1-year incidence of 15% and steadily increasing thereafter.

The suspicion for the diagnosis is raised by an awareness of the syndrome coupled with clinical suspicion for CES [13]. Confirmatory evidence is supplied by advanced radiographic imaging. Initially, a plain view radiograph is performed to rule out lumbar fracture and this film will likely be normal or nonspecifically abnormal in this presentation. Additional appropriate imaging includes magnetic resonance imaging (MRI) scanning. Ideally, all patients with suspected CES should undergo MRI of the spine for confirmation and localization of the lesion. If MRI is not available, then CT-myelography is an alternative imaging tool.

In the emergency department, treatment should include intravenous steroids and surgical consultation. Steroid therapy is recommended early in the course of evaluation; dexamethasone at a dose of 6–8mg is a reasonable choice. Surgical consultation, either from a neurosurgeon or orthopedic spine surgeon, should also be performed. The urgency with which the surgeon decides on operative management is clearly not a decision made by the emergency clinician. Controversy exists within the surgical community regarding the urgency of operation. Most authors agree that early surgical intervention is the best approach with "early" defined as occurring within the first 48 hours after diagnosis. Other surgeons feel that urgent surgery at the time of diagnosis is the most appropriate. It is widely believed that patients who have earlier operations have decreased neurologic disability. Unfortunately, many patients are left with permanent deficits. In general, the patient is likely to be discharged from the hospital with the same neurologic status present as was noted at the time of surgery.

## Conclusion

Cauda equina syndrome is primarily a stressful condition for the patient. Sudden pain, inability to urinate, and loss of sensation in the legs are a great fear for the patient. Every hospital stay, regardless of the severity of the symptoms, causes emotional distress and anxiety and depression. Therefore, the patient's health care must also be focused on identifying changes in the mental state, and in addition to medically, ongoing psychological support is needed. It is important to motivate and encourage the patient to participate in the planning and delivery of health care and independence. Good communication and a positive relationship reduces uncertainty and concern. Family support is also very important. Quality of life after treatment for caude equine syndrome in some people leads to significant improvement in symptoms and a normal lifestyle as before, while others may have permanent neurological impairment, chronic pain and/or mental health problems due to the impact of symptoms on social life and relationships.

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