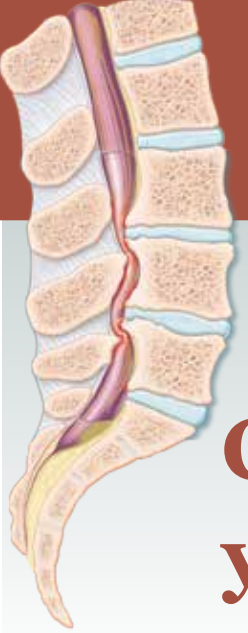

Cauda Equina Syndrome: a review of all you need to know

ABSTRACT

Cauda equina syndrome (CES) is the collection of signs and symptoms produced by severe compression of the lumbar spinal nerves that form the cauda equina. The compression can be caused by lumbar degenerative changes, intraspinal tumors, epidural hematoma, and infections. Rapid diagnosis and treatment are paramount as CES requires emergent surgical decompression. With delay, the patient could develop permanent neurological deficits including loss of lower limb sensorimotor function, bladder, bowel, and/or sexual dysfunction. Unfortunately, even with expeditious surgery, neurological improvements remain unpredictable. Failure to fully explain the possible prognoses can involve all the healthcare providers in medicolegal consequences.

KEYWORDS: Cauda Equina Syndrome, Spine Emergency, Urinary retention, MRI scanning, Saddle Anesthesia

CME Pre-test Quiz

Introduction and Background

Cauda Equina Syndrome (CES) is a devastating spine surgical emergency with potentially significant neurologic morbidity, including bladder, bowel, and sexual dysfunction, leg numbness, weakness, and radicular pain.[1-4] CES is caused by a large space-occupying lesion within the central canal of the lumbosacral spine. It is the most common non-traumatic spinal surgical emergency with high morbidity that can lead to irreversible neurological sequelae when misdiagnosed and/or treatment is delayed beyond 48 hours of its clinical establishment.[4]

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What is the Cauda Equina?

Definition of Cauda Equina Syndrome

The term Cauda Equina was coined by the French anatomist Andreas Lazarius (1558-1609) while describing "rope-like" fibers at the end of the spinal cord and its resemblance to a horse's tail (from Latin: Cauda—tail, and Equinus—horse)[5] (Figure 1). Hence, "Cauda Equina" only refers to the collection of the lower lumbar, sacral, and coccygeal nerves distal to the conus medullaris. "Cauda Equina Syndrome" is the acute clinical syndrome of lower back pain, sensory loss and motor weakness in the lower extremities, saddle anesthesia, and bowel / bladder dysfunction (the last two are required to establish the diagnosis of CES).[4,6]

Epidemiology

CES presents in 2%-3% of all lumbar disc herniations mostly at the L4 - 5 and L5 - S1 levels.[2,7,8] It is very uncommon with an incidence of only 5-10 cases per million back patients per year and a prevalence of 1:33,000 to 1:100,000.[1,6,8] CES impacts both males and females between the second and 4th decade of life (30s),[7-9] with a slightly higher preponderance among men possibly due to higher rates of thoracolumbar trauma in this group.[7,10]

Neuroanatomy, Pathogenesis and Etiology of Cauda Equina Syndrome

Neuroanatomy

The spinal cord originates at the pyramidal decussation of the medulla oblongata in the foramen magnum, and (in adults) it ends at the L2 vertebral body level or L1 - L2 disc space as the conus medullaris.[2,3] The dorsal and ventral rootlets from L2 to L5, S1 to S5, and Co1 descend vertically from the conus medullaris as the cauda equina[3-5] (see Figure 1).

Each of these nerves has specific sensory, motor, sympathetic, and parasympathetic functions in the lower extremities.[2] Notably, there is a somatotopic organization of the nerve roots such that the lumbar nerve roots are more peripheral while the sacral nerve roots are more centrally located.

Bladder and bowel

In the bowel, the primary nerve pathway is mostly the enteric nervous system (ENS), a complex neuronal network within the walls of the gastrointestinal tract that controls peristalsis. Bladder control is maintained through complex interactions between autonomic (sympathetic and parasympathetic nerves) and somatic efferent pathways (mediated by pudendal nerves) in a coordinated, controlled fashion.[11]

The parasympathetic control of the bladder is through efferent

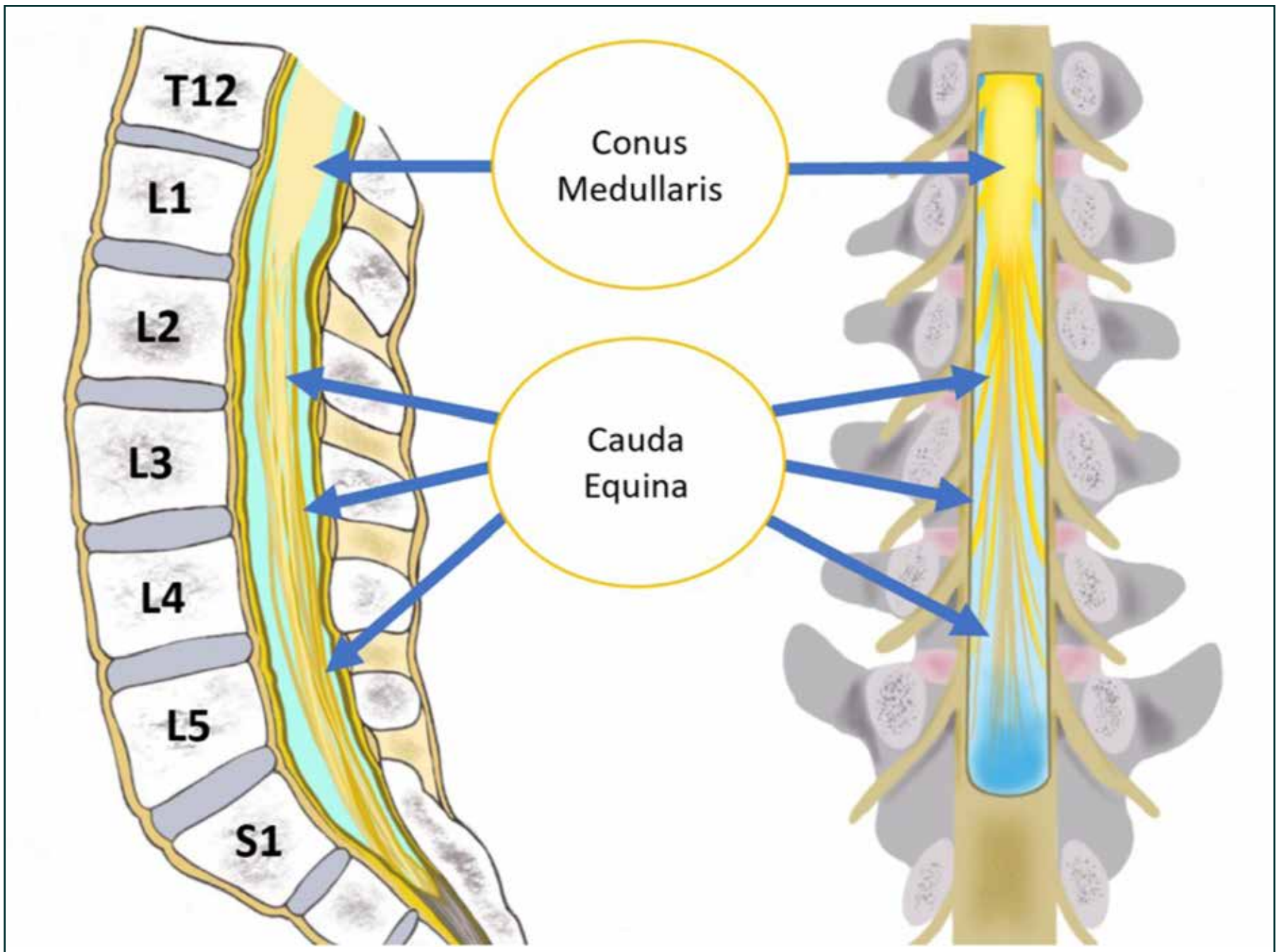

Figure 1. Artistic representation showing the termination of the spinal cord as it ends in the conus medullaris (usually below L1) with nerve roots continuing caudally to become the cauda equina.

innervation from the pelvic organ stimulating center (POSC) and the Onuf's nucleus in the sacral cord at S2 to S4.[12] This stimulates the bladder via the pelvic nerves leading to detrusor contraction and urethral relaxation. The sympathetic neurons maintain bladder relaxation and run through the hypogastric nerve plexus that originates from the T10 to T12 level of the spinal cord.[12,13] This input causes detrusor relaxation and closes the bladder neck and urethra by constricting the internal urethral sphincter. The voluntary/somatic control for both urinary and fecal continence is achieved by the pudendal nerves allowing the voluntary contraction of the external urethral sphincter and the anal sphincter muscles.[14] Pudendal nerves also carry sensory signals from the external genitals, perineum, and the skin around the anus.

Pathogenesis

The pathophysiology of nerve injury begins with direct mechanical compression. Because the nerve roots lack Schwann cell coverage, direct mechanical compression causes venous congestion, neural ischemia, and decreased nutrient nerve diffusion from the CSF.[2,15] With persistent compression, intraneural edema within the nerve roots progresses to nerve ischemia due to impaired arterial perfusion and cytotoxic nerve damage in a compartment-like syndrome.[4,9,16]

Etiology

CES can result from a variety of de novo or exacerbated spine conditions reducing the available space within the lumbosacral canal. By far, the most common etiology is central disc herniations (45%). These most commonly occur at the L4 – L5 and L5 – S1 levels.[7,8] Patients with preexisting degenerative lumbar stenosis may be prone to develop CES with a lesser degree of mechanical compression.[2,17,18] Surgery can produce the syndrome in several ways such as postoperative hematomas in the first 24 hours following spine surgery.[19,20] Avoiding early anticoagulation after surgery may mitigate this risk. CES can also occur secondary to spinal or epidural anesthesia[21] and after aggressive surgical retraction of the thecal sac during surgery. Other causes of CES are described in Table 1.

Table 1: Summarized Causes of Cauda Equina Syndrome. All of them behave as a space-occupying lesion.

Herniated Disc	Acute central disc herniation > L4 – L5 or L5 – S1
Degenerative	Spondylolisthesis, spine stenosis
Neoplastic	Primary: meningioma, ependymoma (myxopapillary), neurofibroma, Schwannoma, lymphoma Secondary: metastasis
Traumatic	Retropulsion of bone fragment (burst fracture) into the canal, vertebral dislocation, or vertebral body collapse
Infectious	Spinal epidural abscess
Iatrogenic	After neuraxial anesthesia procedures (epidural), secondary to surgery, after spinal manipulation
Vascular	Arteriovenous malformations, spinal subdural hematoma, spinal epidural hematoma, inferior vena cava thrombosis
Miscellaneous	Ankylosing spondylitis, arthritis, osteoporotic collapse, Paget's disease

Clinical Presentation, Neurological Findings, and Patient Evaluation

Clinical Presentation

About 70% of patients have a previous history of low back pain and/or sciatica.[4] Cauda equina syndrome can appear suddenly or in a delayed fashion and the presentation can vary considerably between patients. Several authors have subdivided CES into three categories (Table 2).[2,3,6,22,23]

Bowel or bladder incontinence without motor or sensory symptoms is unlikely to be cauda equina syndrome. Although the sacral nerve roots are somatotopically organized in the center of the cauda equina nerve fibers, it is likely that the more peripherally located lumbar nerve roots would also be affected by a large compressive lesion. Patients with isolated bowel or bladder incontinence without motor or sensory symptoms should be evaluated for other potential causes of incontinence[12,14] bearing in mind that a lesion in the lowest portion of the sacral canal, below the exit points of the lumbar roots, could cause bowel or bladder incontinence without pain, weakness, or sensory symptoms.

Neurological Findings

Patients can present with a wide constellation of neurological signs

Table 2: Subgroups or Categories of Cauda Equina Syndrome

Signs and Symptoms	CESS[1]	CESI[2]	CESR[3]
Pain	Back pain with bilateral radiculopathy	Back pain +/– bilateral radiculopathy	Back pain +/– bilateral radiculopathy
Motor and sensory disturbances	Subjective sensory or motor deficits	Objective motor and/or sensory deficits	Objective motor and/or sensory deficits
Bladder integrity/function	Preserved	Neurogenic bladder dysfunction *Diminished desire to void, difficulty starting and stopping the urine stream	Painless retention with overflow incontinence
Bowel integrity/function	Preserved	Anal sphincter tone diminished	Fecal incontinence
Saddle anesthesia	Abesnt	Partial	Complete perianal sensory loss

[1]CESS = Cauda Equina Syndrome Suspected, [2]CESI = Cauda Equina Syndrome Incomplete, [3]CESR = Cauda Equina Syndrome with Retention

and symptoms including combined motor and sensory deficits, decreased reflexes, and radicular pain in the lower extremities.[7,18] However, there is a consensus that (1) bladder and/or bowel dysfunction, (2) reduced sensation in the saddle area, or (3) sexual dysfunction MUST be present to establish the diagnosis of CES.[4,7,17,18,24]

Motor

Cauda equina syndrome typically produces radicular pain and lower motor neuron symptoms with weakness in both lower extremities. Motor examination of the lower extremities can be performed by testing myotomes as described in Table 3.

Evaluate the lower sacral nerve roots by assessing the rectal tone and anal sphincter contraction with digital rectal examination; decreased rectal tone can be an early physical finding.[16] A distended bladder or a postvoid residual greater than 400 ml bladder volume are indicative of urinary retention.[4,25,26] Patients develop overflow incontinence following an extended period of urinary retention.[26]

Sensory

Testing sensory impairment in the lower extremities should include

Table 3: Lumbar Spine Exam and Test Maneuvers

Nerve	Sensory – Dermatome	Muscle – Myotome	Associated test
L1	Groins, upper hips	–	–
L2	Front of thighs	Iliopsoas (**L2**, L3)	Hip flexion
L3	Medial knee, inner legs	Quadriceps femoris (**L3**, L4)	Knee extension
L4	Medial malleolus	Tibialis anterior (**L4**, L5)	Ankle dorsiflexion
	Patellar tendon		
L5	Top/dorsum of the feet, front of the lower legs	Extensor hallucis longus Gluteus Medius	Great toe extension Hip abduction or Trendelenburg test
S1	Plantar foot, outer toes	Gastrocnemius (**S1**, S2)	Ankle plantar flexion
	Achilles tendon		
S2	Back of the thigh area	Biceps femoris (S1, **S2**)	Knee flexion
S3, S4, S5	Perimeum, genitals perianal area	Bladder and anal sphincter	Rectal Exam
	Bulbocavernosus reflex		

both pinprick and assessment of light touch and pressure although light touch and pressure may be preserved. The dermatomes are shown in Figure 2. Patients should be tested for saddle anesthesia. To evaluate the S2, S3, and S4 sacral dermatomes, pinprick testing should be performed in the posterior thighs and perineal region.[16,2] Patients with CES will develop painless urinary retention and deep anal anesthesia.[27]

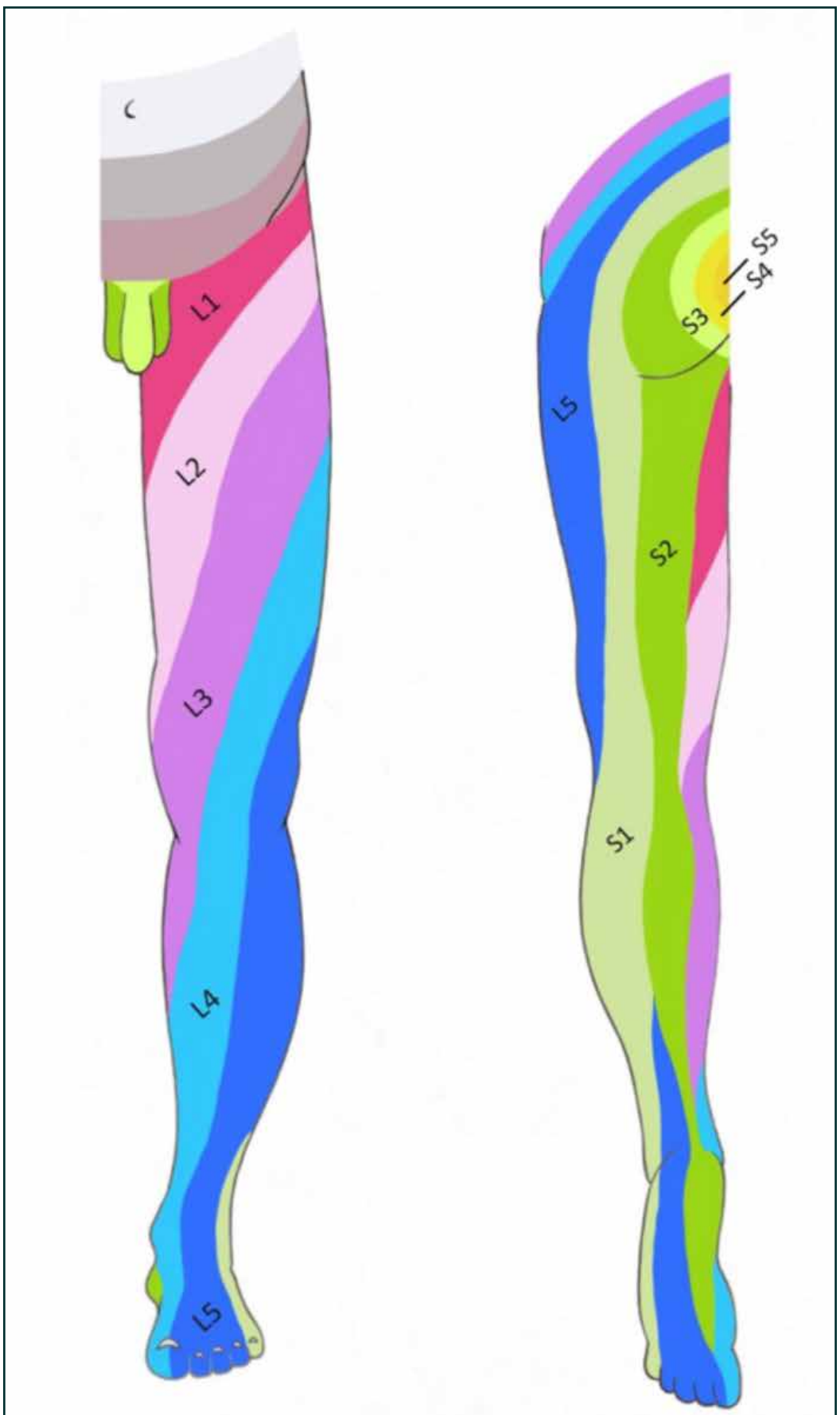

Figure 2. Lumbar (L) and sacral (S) dermatomes of the lower extremities.

Patellar and Achilles tendon reflexes

Testing lower extremity reflexes includes the patellar tendon (L4) and Achilles tendon (S1) reflex arcs. The deep tendon reflexes may be decreased with more than half of patients with cauda equina syndrome having an absent Achilles reflex.[4]

The bulbocavernosus reflex, which assesses the S1-S3 sacral nerve roots, should be evalusated.[2] Pressure on the glans penis or clitoris can be applied by gently pulling on a Foley catheter while performing a rectal exam. In the presence of an intact reflex, the anal sphincter will contract.[16,25,26]

Cauda Equina Syndrome vs Conus Medullaris Syndrome

The symptoms and signs of CES are consistent with only lower motor neuron findings, while those of conus medullaris syndrome (CMS) are a combination of both lower and upper motor neuron findings, since both the spinal cord and the exiting lumbar nerve roots may be affected. Lesions above the L1–L2 disc space cause CMS with upper motor neuron findings, such as

increased reflexes, pyramidal and long tract signs. Lesions below L2 produce only lower motor neuron symptoms such as diminished or absent reflexes, and weakness in a myotome pattern. Lesions below L2 may also cause asymmetric neurological deficits[15] (Table 4). In both conus medullaris syndrome and cauda equina syndrome, patients will lose bowel or bladder control. Similarly, in CMS as in CES, the neurological recovery is variable and unpredictable. A delay in either diagnosis may lead to permanent neurological impairment.[28,10,28,29]

Magnetic Resonance Imaging (MRI) and Computed Tomography (CT)

While cauda equina syndrome remains a clinical diagnosis, advanced imaging studies help to confirm and/or rule out the pathology. Lumbosacral magnetic resonance imaging (MRI) is the optimal imaging study as neural elements can be directly visualized.[22,30] MRI provides better resolution and

Table 4: Cauda Equina Syndrome vs Conus Medullaris Syndrome

	CES[a]	CMS[b]
Clinical Presentation	Gradual and unilateral (asymmetric)	Sudden and Bilateral (symmetric)
Reflexes	Both ankle and knee jerks are affected (absent)	Knee jerks preserved but ankle jerks affected (hyperactive)
Radicular pain	More severe	Less severe
Low Back Pain	Less frequent	More frequent
Sensory symptoms and signs	Localized to saddle area and sensory dissociation	Localized to perianal region, may have sensory dissociation
Motor strength	Areflexic paraplegia (LMN)[c]	Hyperreflexic distal paresis (UMN)[d]
Impotence	Less common	Frequent
*Sphincter dysfunction	Late with mostly urinary incontinence	Sudden with both urinary and fecal incontinence

[a]CES = Cauda Equina Syndrome, [b]CMS = Conus Medullaris Syndrome, [c] LMN = Lower Motor Neuron, [d]UMN = Upper Motor Neuron * In conus medullaris syndrome as well as cauda equina syndrome, the patients will loss bowel or bladder control

helps to facilitate surgical planning.[16,23] The MRI finding is typically a big central disc herniation that compresses the lumbosacral nerves and obliterates the lumbar cistern (Figure 3).[30,31] Disadvantages to the use of MRI include a lack of 24-hour availability to many clinicians working in primary care and contraindications such as pacemakers and poor patient tolerance due to claustrophobia.[4] MRI-compatible metallic implants from previous lumbar instrumentation generate significant artifacts.[17]

Plain computed tomography (CT) scanning or CT myelograms are alternatives under urgent circumstances or when patients have contraindications.[2,6] CT is superior to MRI at defining bony abnormalities but uses ionizing radiation to generate images and does not provide the neural soft

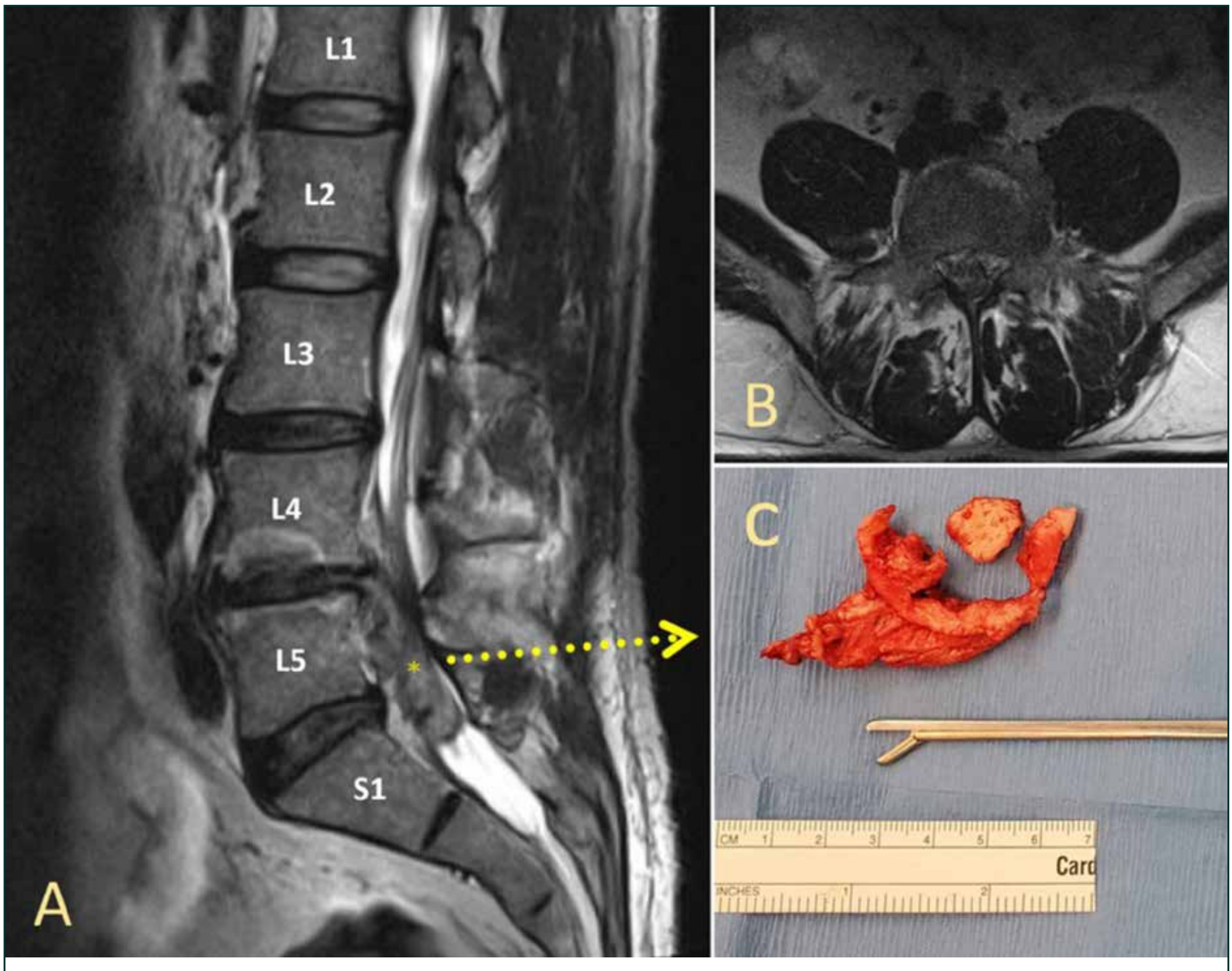

Figure 3. T2 mid-sagittal (A) magnetic resonance imaging of the lumbar spine showing a large herniated L4/5 disc with the corresponding axial (B) cut showing severe canal stenosis. Following herniated disc removal after surgery (C).

tissue information seen on MRI. CT myelography gives excellent soft tissue and bony definition sufficient to demonstrate a complete or near complete block but it is an invasive procedure that requires the injection of dye into the thecal sac.[17] It can be useful when a patient has instrumentation in the lumbar region and MRI may not be diagnostic.

Treatment

The diagnosis of cauda equina syndrome requires prompt neurosurgical or orthopedic consultation and urgent spine surgery referral. The operation can be a technically demanding procedure that should be performed as soon as possible by an adequately skilled surgeon.[6,32] The primary goal is to achieve a wide decompression and complete removal of the mechanical compression.[9,2,22] The type of surgical intervention may vary based on the nature of the compressive lesion. In the typical disc herniation, a laminectomy, and discectomy may be sufficient while in more uncommon cases of trauma, tumors, or infections, stabilization with internal fixation may be required to maintain the spinal column stability.[17,24,28,29] Recent technological advances include the use of percutaneous endoscopic lumbar discectomy though the North American experience with this technique for cauda equina syndrome remains limited.[33]

Prognosis

Prognosis is largely determined by the severity of neurological compromise and the timing of surgery. Maximizing good outcomes necessitates diagnosing and treating cauda equina syndrome as a surgical emergency. Early diagnosis gives the patient the best chance of a satisfactory outcome; time to decompression directly correlates to a good neurological outcome.[6,32]

SUMMARY OF KEY POINTS

1. Cauda Equina Syndrome results from pathologies that compress the nerves in the lumbosacral spinal canal, most commonly due to an acute lumbar disc herniation.
2. Early diagnosis is crucial and is made clinically by distinctive symptoms of saddle anesthesia, acute urinary incontinence combined with acute back and leg pain.
3. The most consistent early clinical sign of CES is urinary retention, and the prognosis is worse when present.
4. Urgent MRI is the study of choice and should be performed to confirm or rule out CES.
5. Surgery is highly recommended within 24 hours after CES is identified.

In the presence of saddle anesthesia or bowel/bladder dysfunction, surgical decompression more than 48 hours after onset, is associated with poor outcomes.[18] A meta-analysis in 2005 demonstrated that patients undergoing surgery earlier than 24 hours after the onset of symptoms were more likely to recover bladder function than those treated after that point.[32] Patients with unilateral saddle dysesthesias have a better prognosis for return of bladder function than those with bilateral saddle anesthesia.[18,28]

Unfortunately, severe neurological compromise may not be reversable even with early surgery.[22] The severity of injury may be more predictive of outcomes than the timing of the operation since neurological recovery depends not only on removing the mechanical obstruction but also on the resolution of the ischemic nerve damage and intraneural edema.[16,29]

Comparing the three subtypes of cauda equina syndrome (Table 2), Cauda Equina Syndrome Suspected (CESS) is the group with the best prognosis. Cauda Equina Syndrome Incomplete (CESI) patients may have restoration of bladder and bowel function to the extent that catheterization is not required. Patients with Cauda Equina Syndrome with Retention (CESR) have a low chance of recovery even after surgery.[9] It is essential to establish the state of anal tone since there is a correlation between poor overall outcome and anal paresthesia and sphincter dysfunction.[26]

CLINICAL PEARLS

1. Cauda Equina Syndrome is caused by a large space-occupying lesion within the central canal of the lumbosacral spine, most commonly a large disc herniation. However, compression can also be caused by lumbar degenerative changes, intraspinal tumors, epidural hematoma, and infections.

2. Cauda equina syndrome generally presents with varying degrees of sensory loss and motor weakness in the lower extremities, saddle anesthesia, and bowel/bladder dysfunction (these last 2 are required to establish the diagnosis of CES).

3. The main clinical feature between differentiating Cauda Equina Syndrome vs Conus Medullaris Syndrome, is the absence of UPPER MOTOR NEURONS findings in CES).

4. About 70% of patients with cauda equina syndrome have a previous history of lower back pain and/or sciatica.

5. Although the prognosis is largely determined by the preoperative severity of neurological deficits, early surgery improves the chance of significant recovery so patients with CES require urgent surgical intervention.

CME

Post-test Quiz

Members of the College of Family Physicians of Canada may claim MAINPRO-M2 Credits for this unaccredited educational program.

Medico-Legal Consequences

The referring physician must communicate the urgency of the situation to the appropriate spine consultant. The most common medical "error" is the failure to diagnose, recognize the urgency and adequately treat an established cauda equina syndrome.[34,35] This is a pathology with potentially devastating consequences for the patient's and the patient's family's social function and quality of life.[36]

When the patient's deficits persist, especially when the likelihood of a poor outcome has not been fully explained and understood by the patient, litigation is common.[19,34] To avoid misunderstandings, the diagnosis demands a careful and extensive discussion of the long-term prognosis.[2,34] Permanent disability can require ongoing supportive multidisciplinary management.

Conclusion

Cauda equina syndrome results from compression of the sacral nerves within the lumbosacral canal. The presence and diagnostic value of the individual symptoms can vary, but the most consistent and clinically important initial sign is urinary retention followed by insensible overflow. Decreased rectal tone can be a relatively early physical examination finding. In patients with clinical features suggestive of CES, urgent referral to the local spinal unit and an emergency MRI scan are indicated. Early surgery offers the best chance of an improved prognosis, even though irreversible neurologic sequelae, related to ischemic–intraneural nerve damage, can occur despite emergent decompression. The severity of neurological symptoms significantly affects the prognosis. The possibility of a poor outcome must be discussed and clearly understood. Not surprisingly, litigation is more common in patients with residual symptoms.

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