Chapter

Cauda Equina Syndrome

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Abstract

Cauda equina syndrome is a relatively rare clinical syndrome caused by compression of cauda equina and can result in significant morbidity if not treated. In this chapter, we describe briefly the anatomical background of the lumbar spine and the nerve supply of the urinary bladder, as the urinary symptoms play a crucial role in diagnosis of this syndrome. Then, we move on to discuss the etiology, symptoms, and signs of cauda equina syndrome. We also describe the different modalities to make the diagnosis including the CT scan, MRI, nerve conduction studies, and electromyogram. Finally, the management of this syndrome including the surgical procedures, complications, and prognosis. We enclosed five real-life cases of different causes of CES from our practice briefly describing the clinical background of the patients as well as CT and/ or MRI images of each case.

Keywords: CES: Cauda Equina Syndrome, CESI: Incomplete cauda equina syndrome, CESR: cauda equina syndrome retention, CNS: central nervous system, NCS: Nerve conduction studies, EMG: Electromyogram, MIS: Minimal Invasive Surgery, CSF: Cerebral Spinal Fluid

1. Introduction

Cauda equina syndrome (CES) is a relatively rare clinical condition caused by compression of the nerve roots forming the cauda equina (the tail end of the spinal cord). CES produces a characteristic set of clinical features and is a surgical emergency requiring urgent intervention to prevent permanent neurological deficits. In this chapter, we will discuss the anatomical background of the lumbar spine as well as clinical manifestations, causes, differential diagnosis, and management of this condition.

2. Anatomical background

Lumbar and sacral spine: Human body has five lumbar and five sacral vertebrae. Each vertebra consists of body and neural element which in turn composed of pedicles, laminae, spinous process, and two articular facets, namely superior and inferior.

An intervertebral disc is found between two successive vertebrae. The disc is formed by central nucleus pulposus and peripheral anulus fibrosis. The neural exit foramen is the space between two adjacent pedicles in the sagittal plane, and this space is normally filled with fat (**Figures 1–6**).

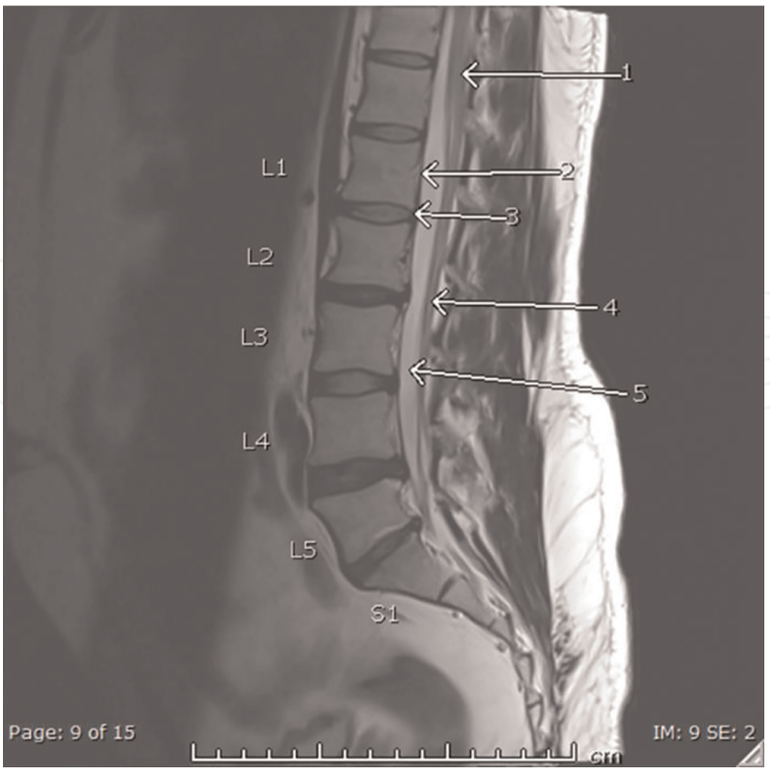


Figure 1.
1. Conus medullaris, 2. L1 vertebral body, 3. L1-L2 Intervertebral disc, 4. Cauda equina, 5. CSF within thecal sac.

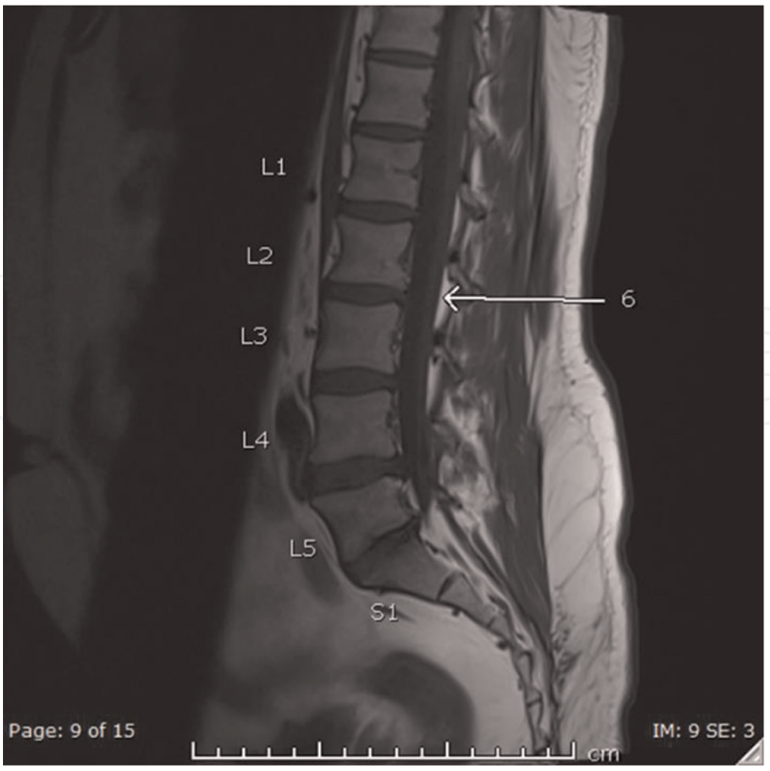


Figure 2.
6. Dorsal epidural fat.

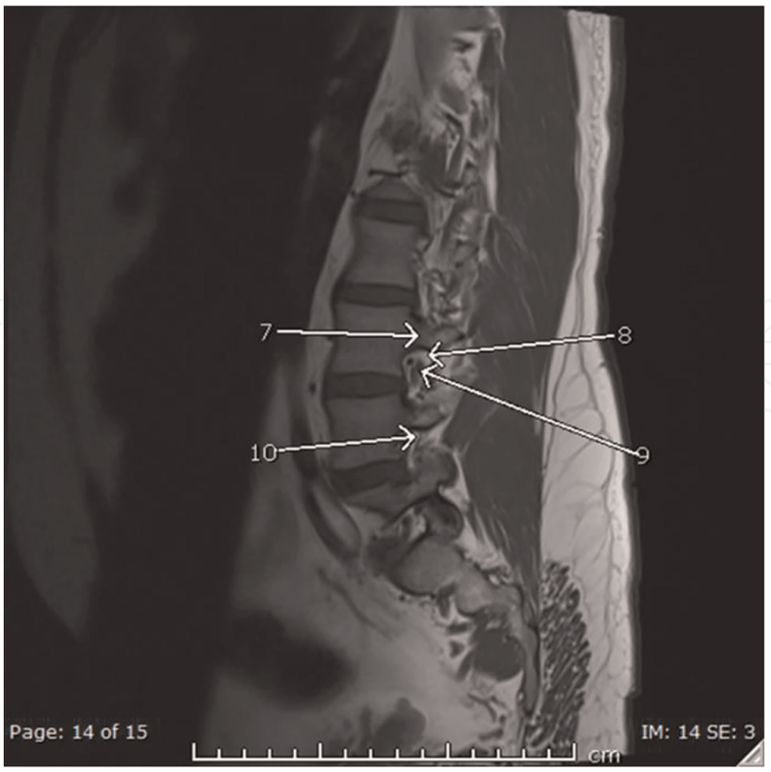


Figure 3.
7. Pedicle, 8. Fat within neural exit foramen, 9. Exiting nerve root, 10. Neural exit foramen.

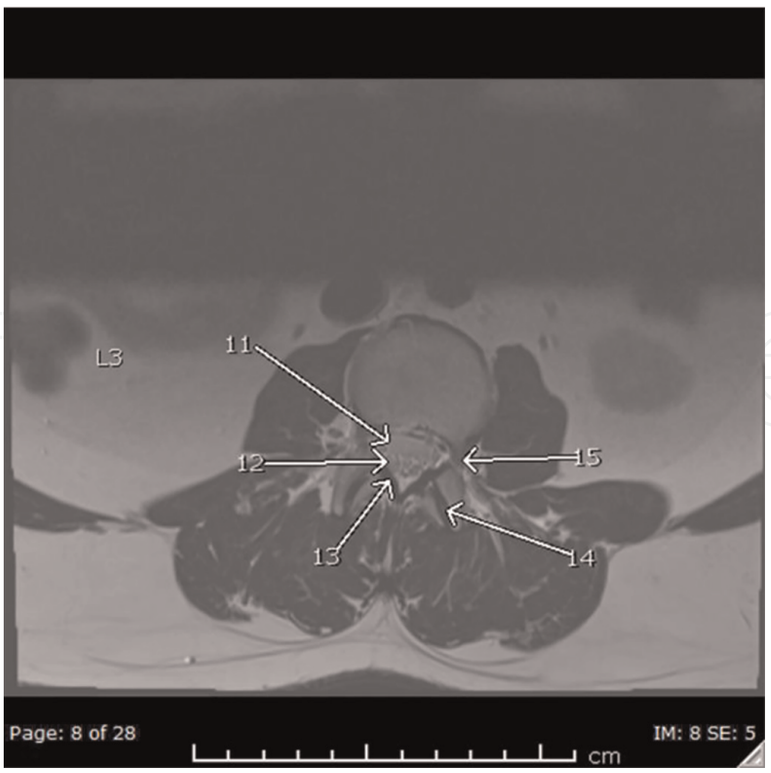


Figure 4.
11. CSF within thecal sac, 12. Cauda equina, 13. Ligamentum flavum, 14. Facet joint, 15. Pedicle.

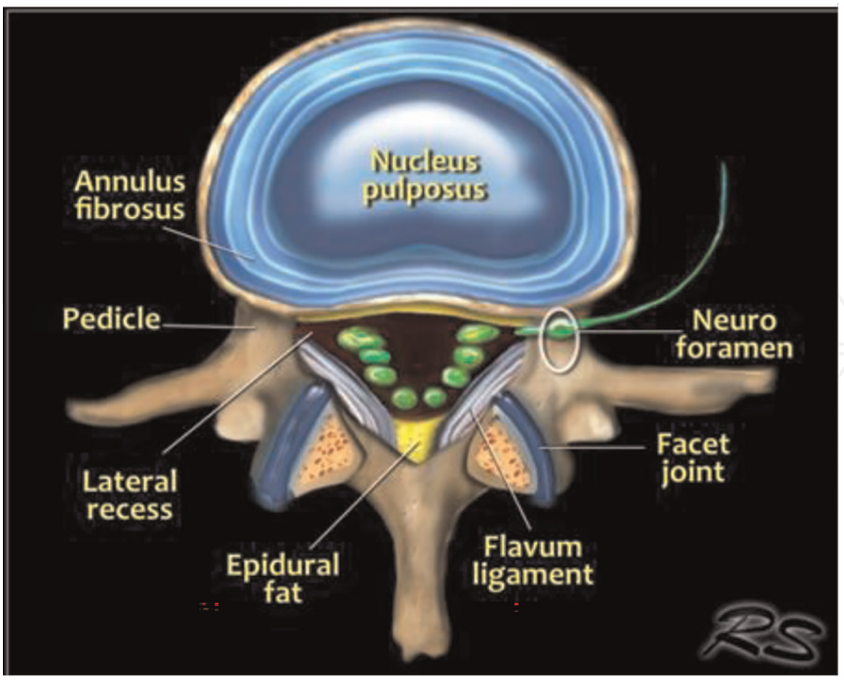


Figure 5.
Normal anatomy of lumbar vertebra with the neural elements and related structures.

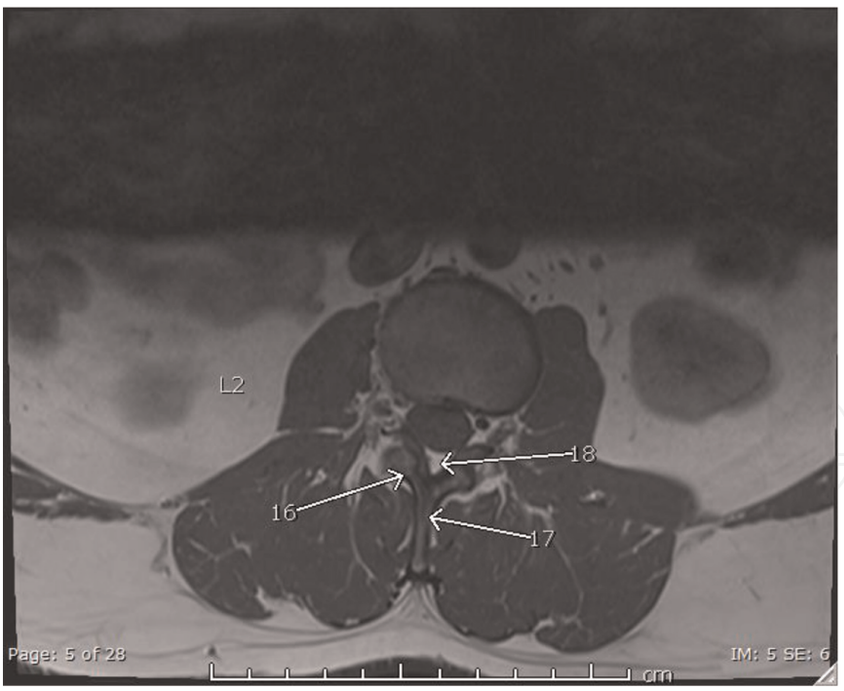


Figure 6.
16. Lamina, 17. Spinous process, 18. Dorsal epidural lipoma.

Spinal cord with the brain forms the central nervous system. The spinal cord starts at the cervicomedullary junction at the foramen magnum to the conus medullaris which is the terminal part of the cord, and this usually is seen at L1 level. Thirty-one

pairs of nerve roots are seen arising from spinal cord as follows: 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and one coccygeal. In axial section, the spinal cord is formed of central gray and peripheral white matter.

Cauda equina: The name of cauda equina came from Latin language and means horse tail. Cauda equina is the collection of nerve roots within the thecal sac extending from conus medullaris to the sacral canal through the lumbar spine (**Figure 7**). As we go down two nerve roots, one on each side, leave thecal sac through the exit foramen which is located between the two successive pedicles in the sagittal plane, we call these nerves exiting nerves. At the same time, another nerve root (transiting nerve) on each side starts to go peripherally preparing itself to leave the thecal sac in the next level. Like a train, in each station, two passengers are leaving from doors on each side of the train, and at the same time, two passengers carry their baggage and start to approach the doors to prepare themselves to leave the train in the next station.

The exiting nerve roots of the lower lumbar (L4 and L5) and sacral (S1, S2, S3, and S4) spine interconnect together to form the lumbosacral plexus which serves motor and sensory functions to the pelvis and lower limbs. Pudendal nerve is a branch of the sacral plexus formed by S2–S4 sacral nerve roots. It plays an important role in urinary, anal, and sexual functions. It has three branches, namely:

- Perineal nerve: motor to muscles of the urogenital triangle and sensory to the perineal region.

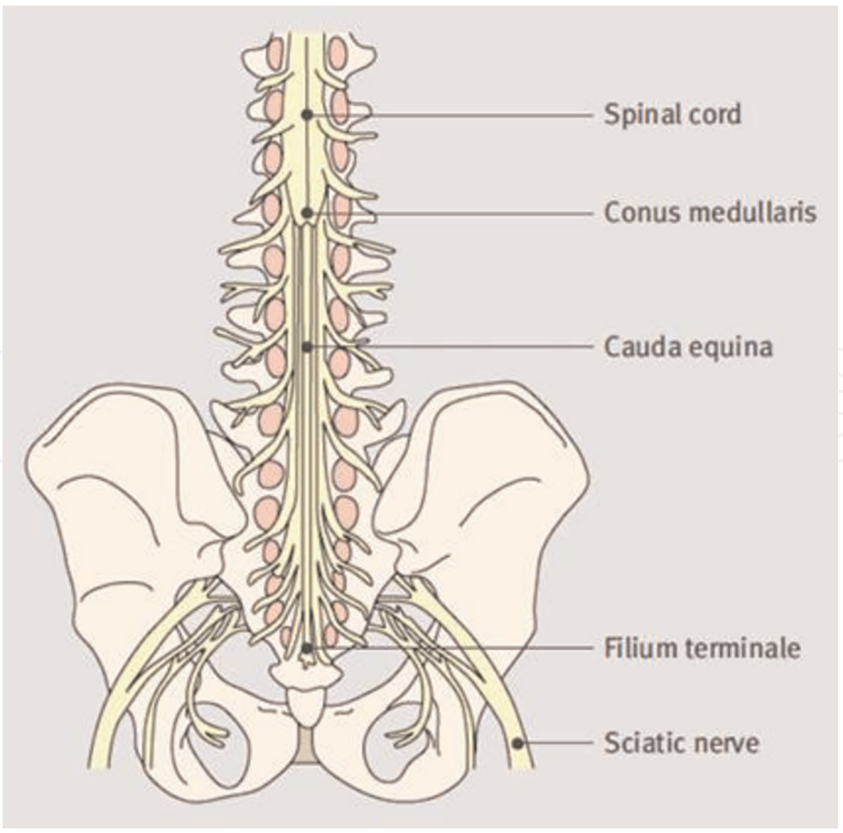


Figure 7.
Anatomy of the lumbosacral spine and the related cauda equina.

- Dorsal nerve of the penis (or clitoris): sensory.
- Inferior rectal nerve: motor to the external anal sphincter and sensory to perineal region.

The urinary symptoms are crucial in diagnosis and prognosis of the cauda equina syndrome as discussed later, and the nerve supply of the urinary bladder should be explained particularly.

The urinary bladder has normally autonomic and sensory blood supply as follows;

- Autonomic nerve supply:

  Sympathetic supply from L1 and L2. The effect of this type inhibits the contraction of the detrusor muscle which forms the wall of the urinary bladder and compress the internal urethral sphincter to prevent micturition.

  Parasympathetic supply from S1, S2, S3, and S4. The parasympathetic supply antagonizes the sympathetic effect leading to contraction of the muscle and relaxation of the sphincter.

- Sensory nerve fibers from the urinary bladder to spinal cord [1].

3. Micturition reflex

The stretch receptors in the wall of the urinary bladder are stimulated when the volume of urine in the bladder reaches around 300 ml. The generated impulses reach CNS, and the body starts to feel the desire to pass urine. The coming impulses reach the second, third, and fourth sacral segments of the spinal cord via pelvic splanchnic nerves. The sympathetic nerves send impulses to the first and second lumbar segments of the spinal cord through hypogastric plexuses. From the second, third, and fourth sacral segments of the spinal cord, the parasympathetic impulses pass through pelvic splanchnic nerves and the inferior hypogastric plexus to urinary bladder wall. As a result, there is contraction of the detrusor muscle and relaxation of the sphincter, which also receives some signals from pudendal nerve for relaxation. When the urine reaches urethra, additional afferent signals from the urethra are sent to the spinal cord to reinforce the reflex. The contraction of the abdominal muscles can also help in micturition by increasing the intraabdominal and pelvic pressures and therefore compress bladder to evacuate the contents [1].

4. Cauda equina syndrome

4.1 Definition

Although there is no consensus definition for cauda equina syndrome, this lower motor neuron condition occurs when there is dysfunction of cauda equina, precisely S2 and below nerve roots [2]. This usually occurs due to mechanical compression.

4.2 Epidemiology

It is considered a rare entity with a prevalence estimated by approximately 1 in 65,000 in one study [3]. It is seen in around 3% of lumbar spine disc herniation.

4.3 Etiology

Cauda equina syndrome occurs when there is significant spinal canal stenosis and mechanical compression on cauda equina enough to produce symptoms. The commonest cause is disc herniation. Other less common causes are neoplasms, trauma, hematoma, abscess, and inflammation (**Table 1**) [3].

4.4 Disc disease

This is the commonest cause for spinal canal stenosis and cauda equina syndrome [2–5]. Disc herniation is displacement of disc material like nucleus pulposus, parts of the annulus fibrosus, and cartilage, beyond the limits of the intervertebral disc space (**Figure 8**). This can be either diffuse (disc bulge) or focal (herniation). Focal disc herniation can be further subdivided into protrusion (broad base at parent disc), extrusion (narrow or no base at parent disc), and extrusion with sequestration (extruded disc without contiguity to parent disc) [6], (see Case 1).

MRI shows disc extrusion at L4-L5 compressing cauda equina.

Had surgical laminectomy and microdiscectomy, 2 months after surgery patient still complains of weakness in the lower limbs with urinary incontinence and constipation (**Figures 9** and **10**).

The central spinal canal stenosis and cauda equina compression by degenerative disc disease are commonly associated by other risk factors like hypertrophic facet joint arthropathy and ligamentum flavum thickening as well as spondylolisthesis [6] and epidural lipomatosis [7, 8].

Disc herniation
Trauma
Spinal stenosis
Tumors: primary and secondary
Infection
Arteriovenous malformation
Hemorrhage (subarachnoid, subdural, epidural)
Ankylosing spondylitis
Iatrogenic causes
Continuous spinal anesthesia
Postsurgery
Postintradiscal therapy
Postchiropractic manipulation

Table 1.
Causes of Cauda Equina syndrome.

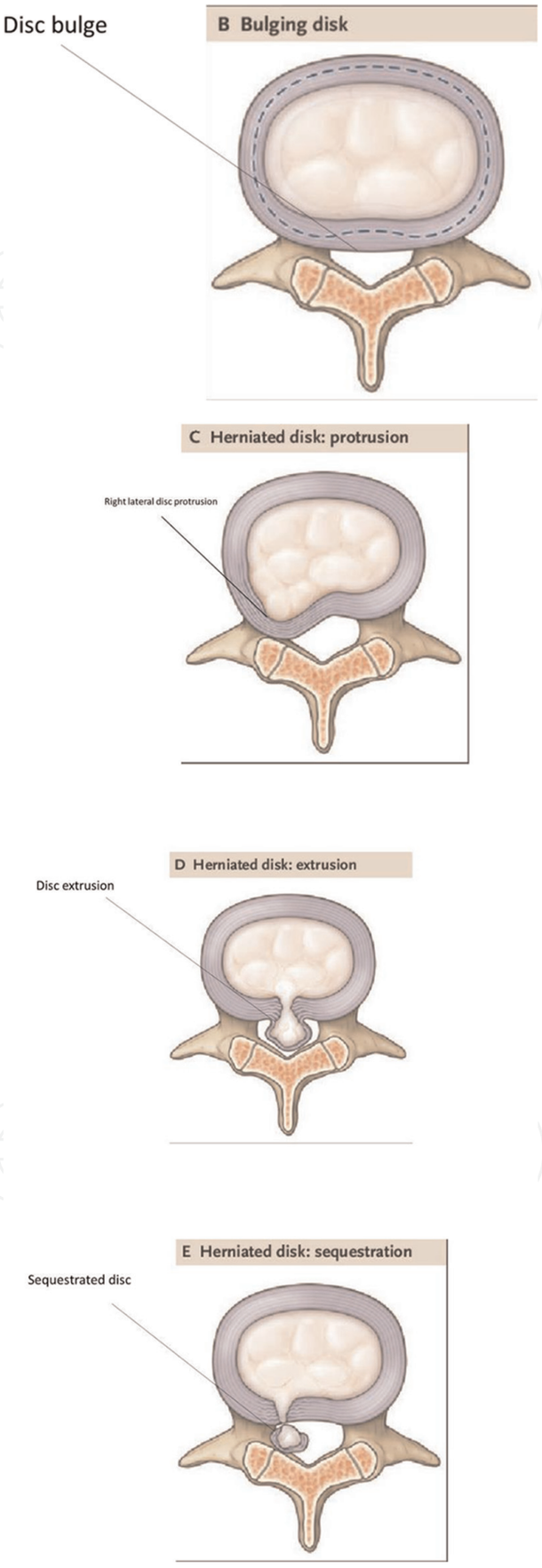


Figure 8.
Classification of degenerative disc disease.

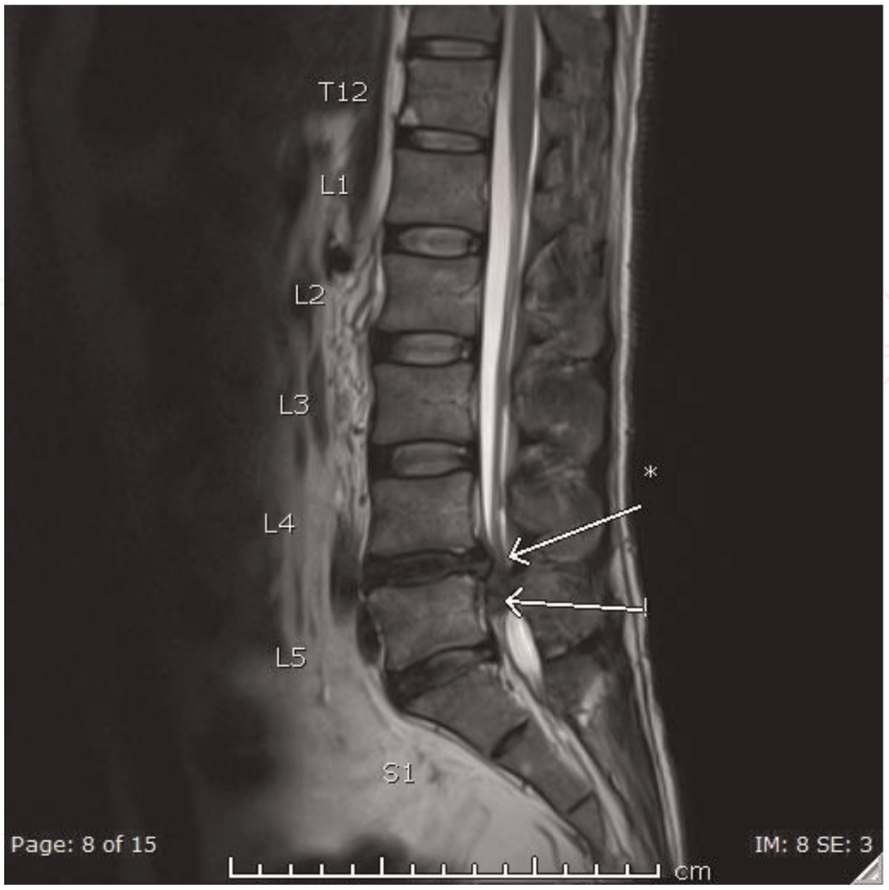
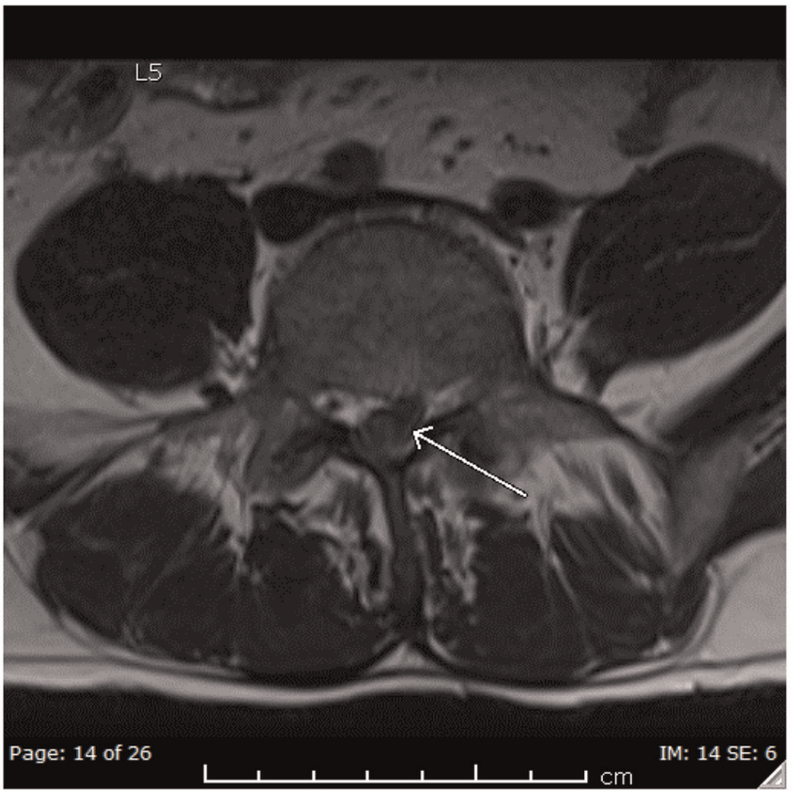
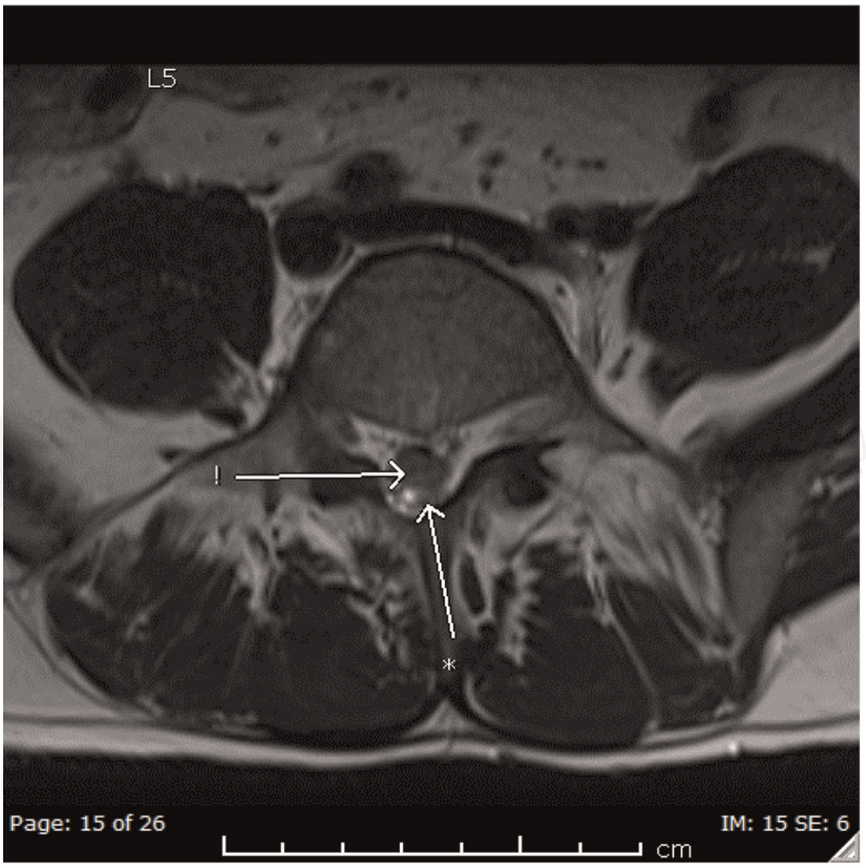


Figure 9.
Sagittal T2 showing disc extrusion (!), compressed cauda equina (*).



(a)



(b)

Figure 10.
Axial T2 showing extruded disc (!), compressed cauda equina (*).

4.5 Neoplasms

Primary:

- Myxopapillary ependymoma: intradural extramedullary tumor arises from ependymal glia of the filum terminale. It is usually seen below conus medullaris [9].
- Schwannoma: benign intradural extramedullary tumor. It is the commonest nerve sheath tumor of the spine (**Figure 11**).
- Meningioma: intradural extramedullary spinal canal tumor arises from spinal meninges. It is more common in female [3].

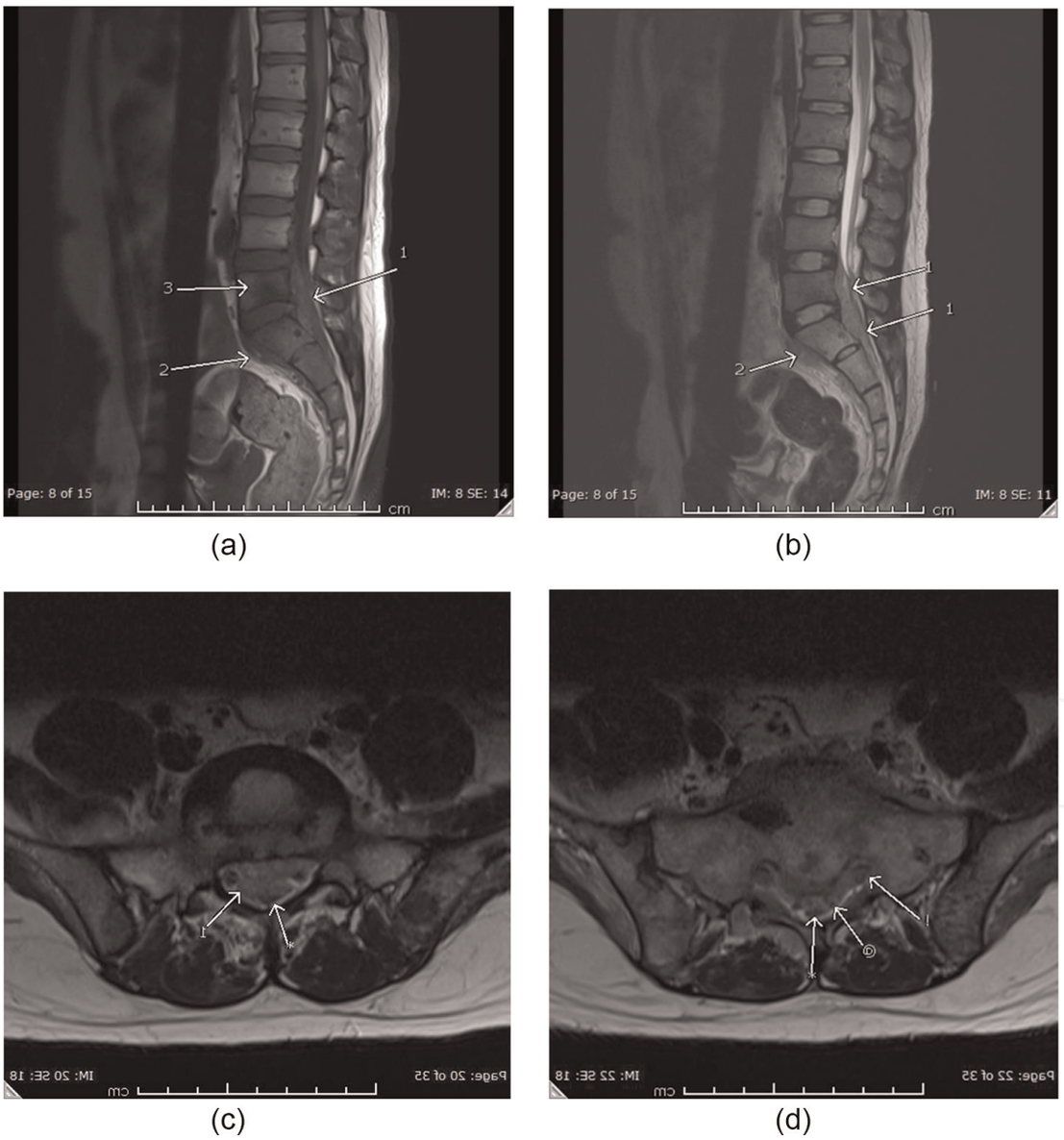


Figure 11.
*a: Sagittal T1; (1: epidural metastatic lesion, 2: prevertebral metastatic lesion, 3: bone metastasis), b: sagittal T2; (1: epidural metastatic lesion, 2: prevertebral metastatic lesion), c and b: axial T2; (1: epidural metastatic lesion, *: cauda equina, !: S1 exiting nerve root, @: S2 transiting nerve root).*

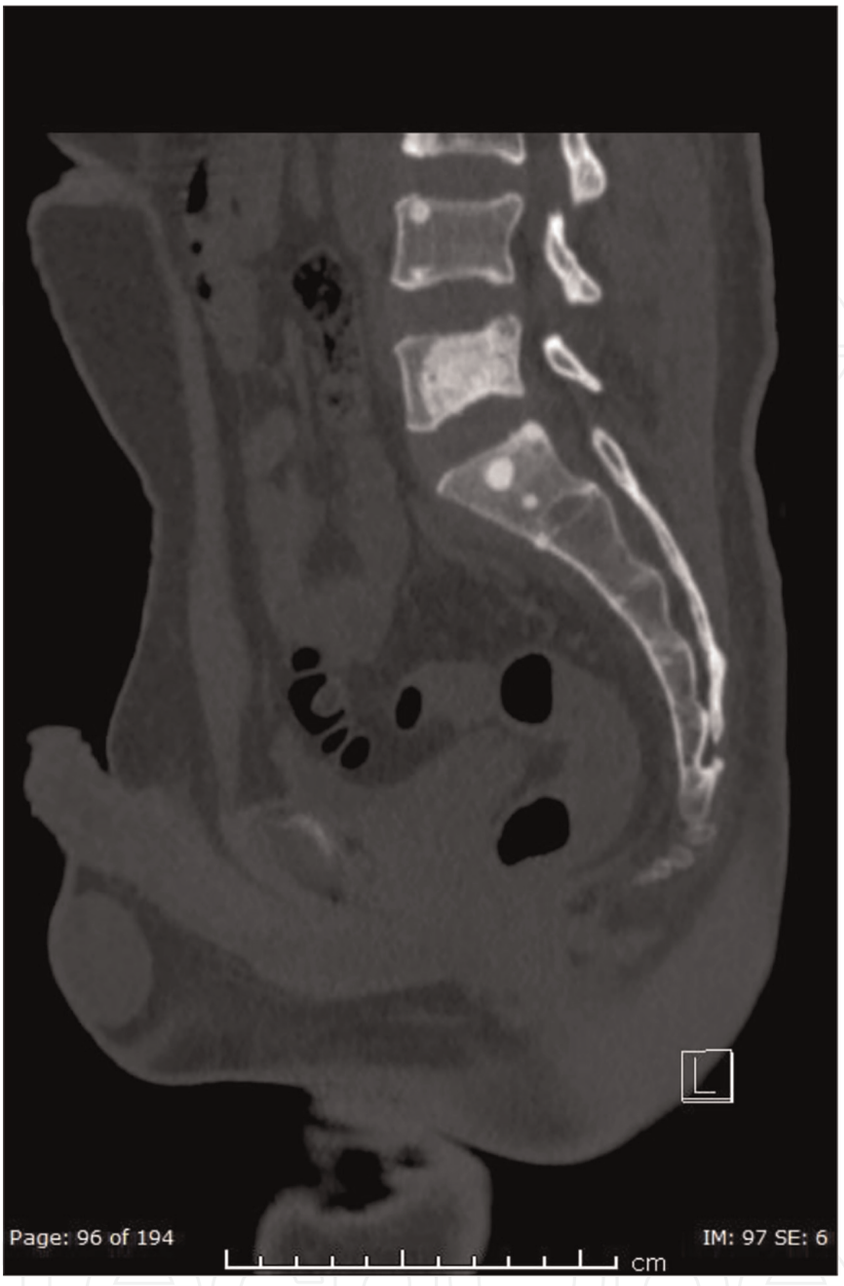


Figure 12.
CT scan showing multiple sclerotic bone metastasis.

Secondary (metastasis): can be either from CNS through drop metastasis (where the primary tumors usually high grade glioma, germinoma, medulloblastoma, and choroid plexus tumors), or hematogenous metastasis from outside the CNS commonly lung and breast primaries [4, 10]. Metastasis can be from outside CNS commonly lung and breast malignancies (see Case 2) (**Figures 12** and **13**).

4.6 Trauma

Burst fracture of lumbar and/or sacral vertebral bodies with retropulsion of fractured fragments or vertebral subluxation may encroach on the spinal canal and compress cauda equina causing this syndrome (see Case 3) (**Figures 14** and **15**).

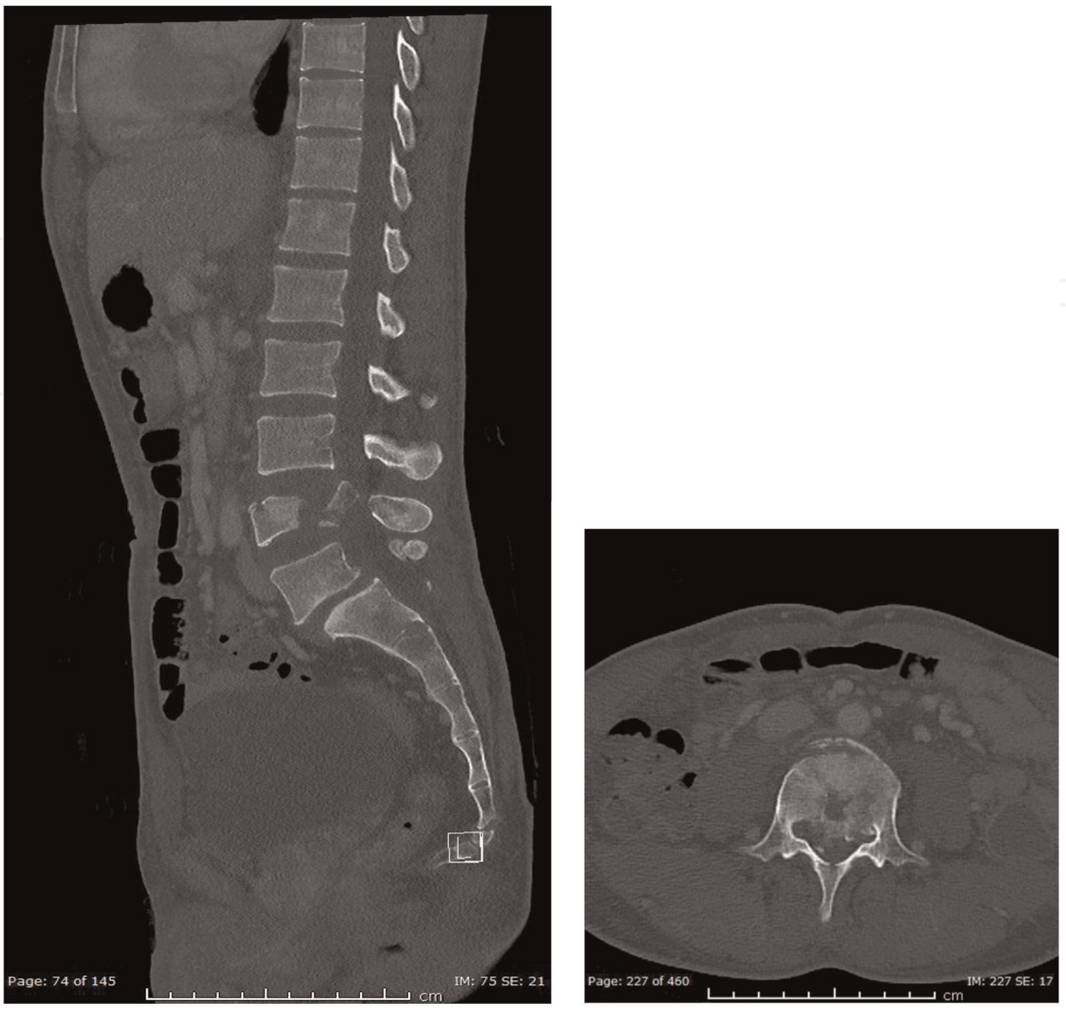
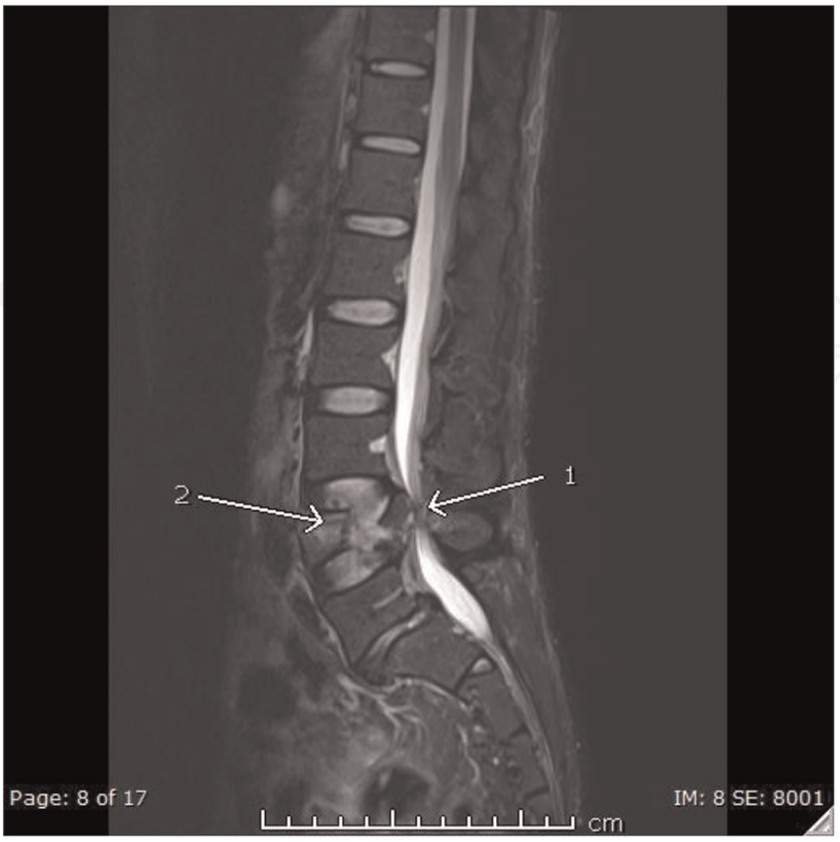
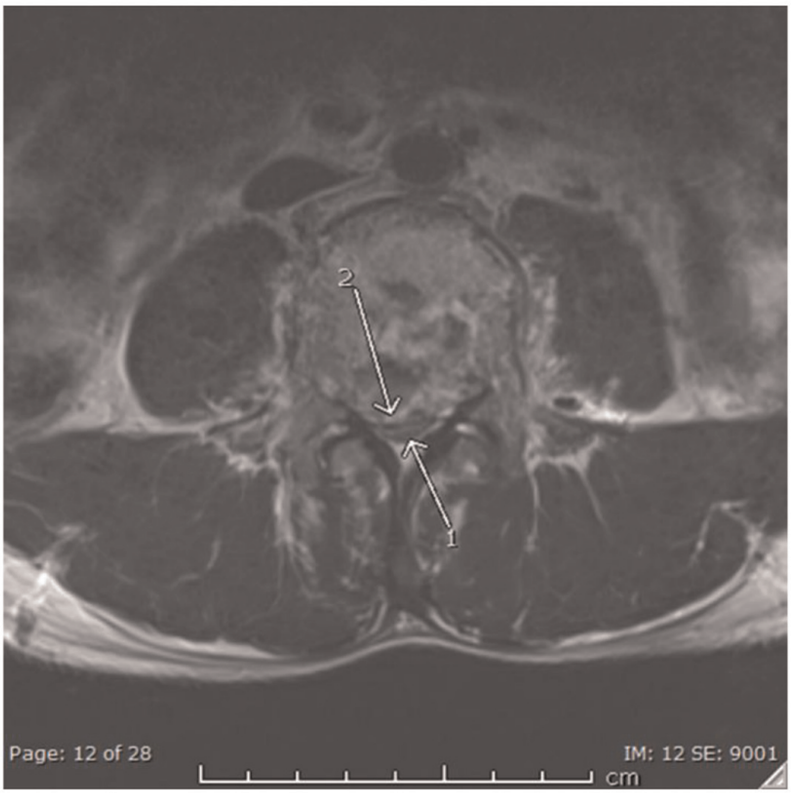


Figure 13.
Burst fracture of L4 vertebral body with retropulsion encroaching on the spinal canal.



(a)



(b)

Figure 14.
(a) Sagittal STIR showing 1: compressed cauda equina, 2: fractured vertebral body (b) Axial T2 showing compressed cauda equina 1 and the fractured vertebral body.

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This can be also sometimes associated with significant hematoma adding to cauda equina compression and spinal canal stenosis.

4.7 Infection

- Tuberculous spondylodiscitis: Inflamed soft tissue (phlegmon) with abscess can compress the cauda equina and cause significant spinal canal stenosis.
- Pyogenic spondylitis: Sizable pyogenic abscess can compress cauda equina and produce symptoms (see Case 4).

MRI Exam showed anterior epidural collection along the lumbar spine compressing cauda equina. It also showed enhancement of L5 and S1 vertebral body suggesting spondylitis. Surgical drainage of spinal abscess was done. There was significant improvement with almost complete recovery after surgical drainage and full course of bacterial antibiotics (**Figure 15**).

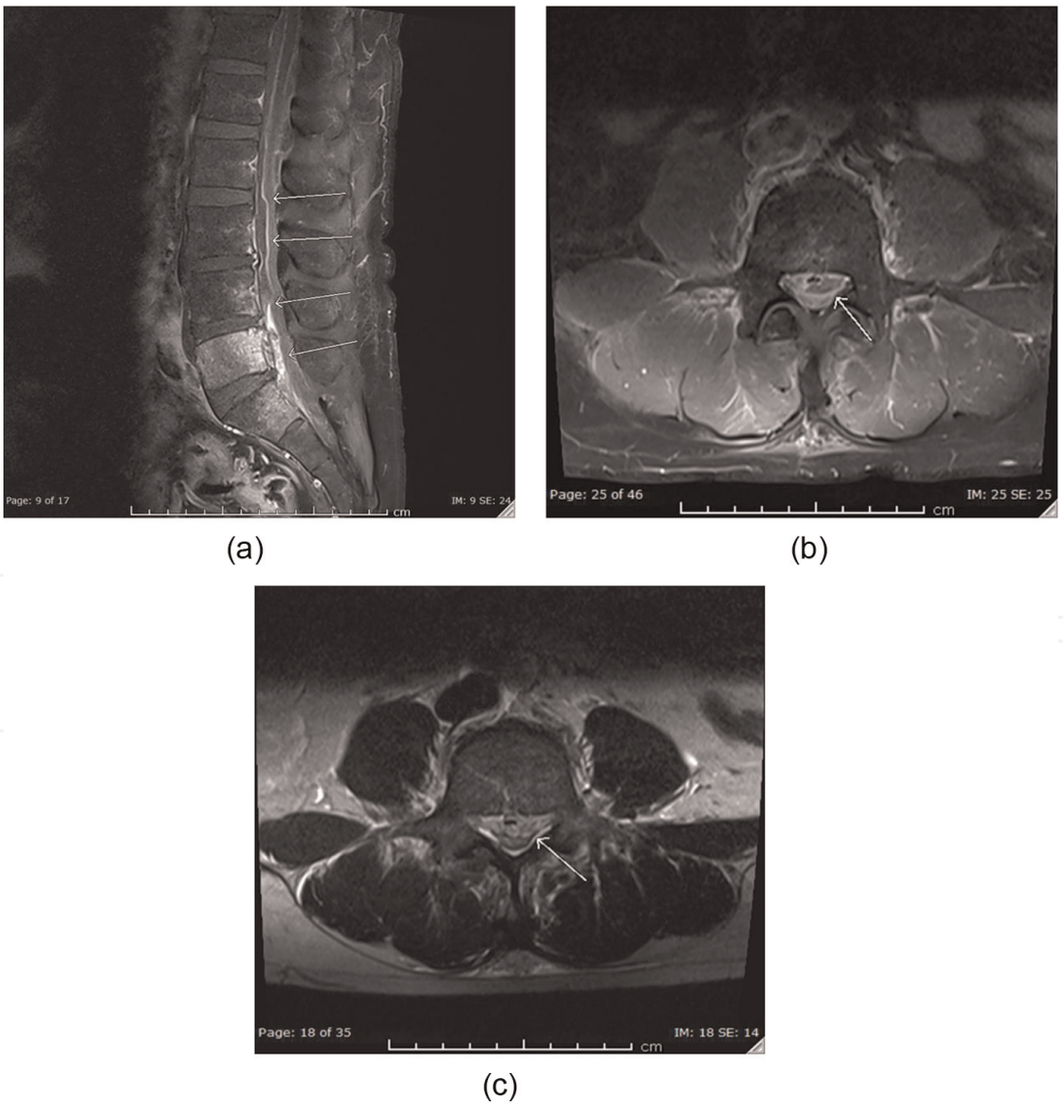

Figure 15.
a: Sagittal T1 post contrast and b: axial T1 post contrast: shows epidural collection with marginal enhancement. c: axial T2: shows bright collection compressing cauda equina.

4.8 Inflammatory

A rare cause of CES is ankylosing spondylitis [11, 12]: This is a chronic progressive inflammatory disease of the joints which can cause ligamentous calcification and osteophyte formation, and this may result in significant spinal canal stenosis and cauda equina compression.

4.9 Degenerative

Synovial cysts, spondylolisthesis, facet joint arthropathy, and hypertrophy of ligamentum flavum can cause canal stenosis; however, these more commonly add to spinal canal stenosis in the presence of other factors like disc herniation (**Figure 16**) [3, 13, 14].

Hematoma: can be due to trauma, bleeding tendency, or iatrogenic this can be a cause.

Epidural lipomatosis: This can rarely alone cause CES [15]. This is usually related to prolonged use of corticosterioids, obesity, or idiopathic [16].

4.10 Symptoms and signs

Symptoms depend grossly on the degree of compression and the nerve roots being compressed.

The following are the symptoms seen in cauda equina syndrome:

1. Lower back pain.

2. Unilateral or bilateral leg pain (sciatica). *Bilateral sciatica should be always taken seriously.*

3. Unilateral or bilateral lower limb weakness.

4. Saddle anesthesia: This includes perineal and perianal areas as well as inner thighs, and the severity of the sensory disturbance correlates with the number of the involved nerve roots.

5. Bladder, bowl or sexual dysfunction; the later two are only rarely seen.

Although all these symptoms can be seen in cauda equina syndrome, the latter two [4 and 5] are the hallmarks for definite diagnosis [2, 3].

4.11 Presentation

The onset of symptoms can be either acute (rare), acute on top of chronic (commonest presentation) or chronic [2].

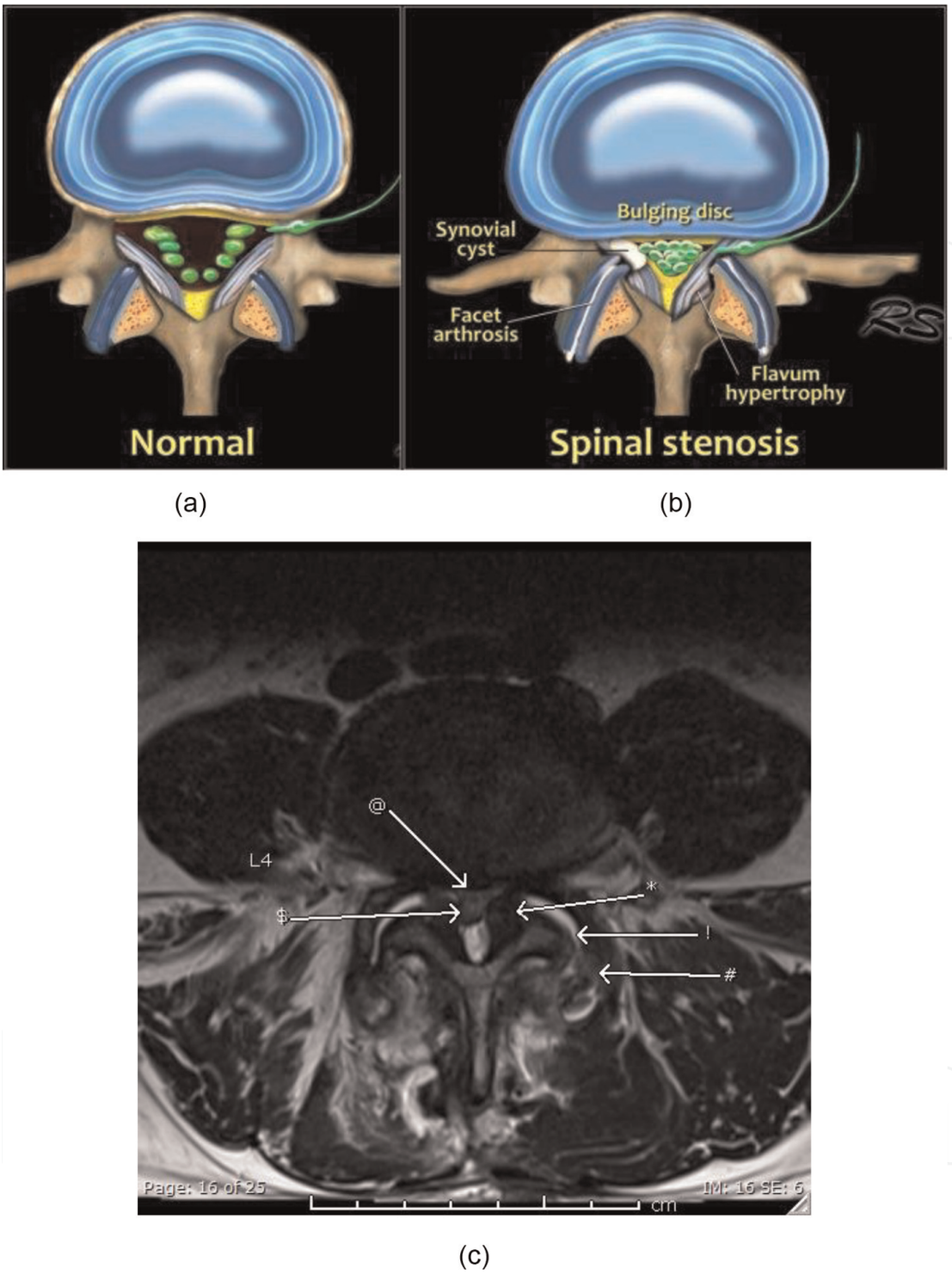

Figure 16.
*@: Protruding disc, $: Compressed cauda equina within thecal sac, *: Thickened ligamentum flavum, !: Joint effusion, #: Hypertrophied facet joint.*

4.12 Classification of CES

This widely used classification to assess the severity of the damage and predict the outcome of CES was proposed by **Gleave and Macfarlane**, and accordingly CES is classified to incomplete CES (CESI) and CES with painless bladder retention/ CES (CESR).

In CESI, the patient has urinary difficulties of neurogenic origin such as altered urinary sensation, loss of desire to void, poor stream or the need to strain, but there is still executive control of bladder function and voiding is possible even if difficult.

Meanwhile CESR occurs when the bladder is no longer under executive control and there is painless retention of urine with overflow [2, 5].

Another less commonly used classification was proposed by J Shi and et al. based on the clinical presentation and physiological disability, where CES is classified into four stages, namely preclinical, early, middle, and late. However, this classification has not been widely adopted by the neurosurgeons community [17].

4.13 Differential diagnosis

The main differential diagnosis is conus medullaris syndrome (see Case 5), the main difference between the two syndromes is that CES presents only with lower motor neuron deficit, meanwhile conus medullaris syndrome will present with signs of both upper and lower motor neuron lesion.

MRI with IV contrast was done. It showed extramedullary lesion at T12-L1 level compressing conus medullaris (**Figure 17**). This was surgically resected. In histopathological examination the lesion was proved to be schwannoma. Patient showed significant improvement after surgical resection.

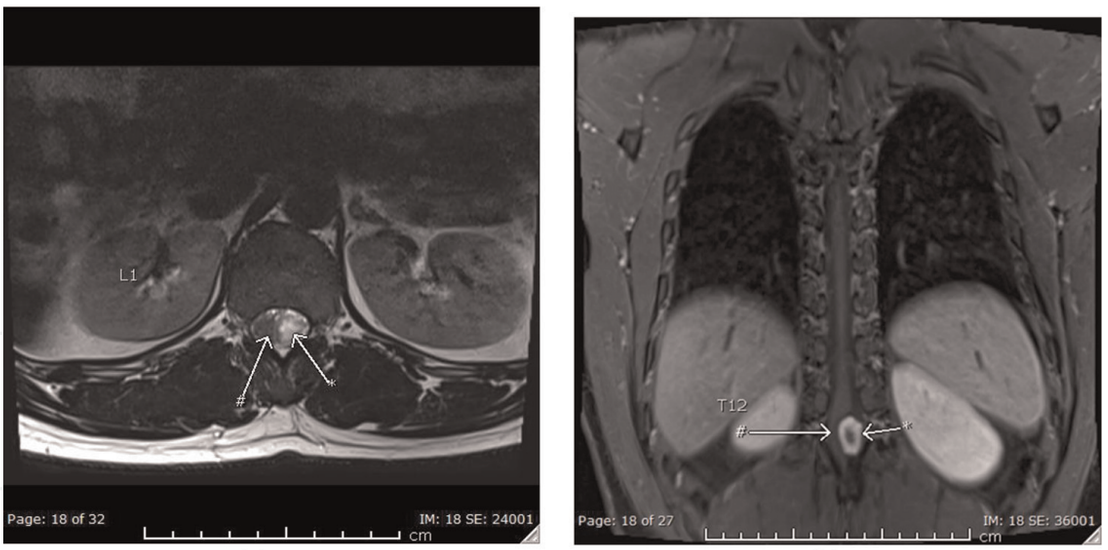

Figure 17.
**: Lesion, #: Conus medullaris.*

4.14 Investigation

Cauda equina syndrome is a clinical condition. Imaging plays an important role in detecting the cause of this condition. MRI is the best modality and is enough in most patients. In case of CES, MRI will reveal the cause in most of the patients, which can be large disc herniation, tumor, or collection (which can be either hematoma or abscess) compressing cauda equina nerve roots. There will be usually significant spinal canal

stenosis. From the radiological point of view, central spinal canal stenosis of the lumbar spine can be classified based on the cauda equina nerve root aggregation. Grade 1 (mild stenosis) is when the anterior CSF space is mildly obliterated, but all the nerves in the cauda equina can be clearly separated from each other. Grade 2 or moderate stenosis indicates cauda equina aggregation, while grade 3 signifies severe stenosis with the entire cauda equina appearing as a one bundle (**Figures 18** and **19**) [6].

CT is a good alternative when the MRI is contra indicated, and it is very good in assessing bony tissues and bony spinal canal stenosis. CT scan can highly exclude cauda equina impingement if the thecal sac is effaced by less than 50%, as one study concluded [18]. The possibility of CT to identify soft tissue lesions can be sometimes limited by artifacts. Myelogram is another modality; however, it is considered relatively invasive as it requires intrathecal injection of contrast.

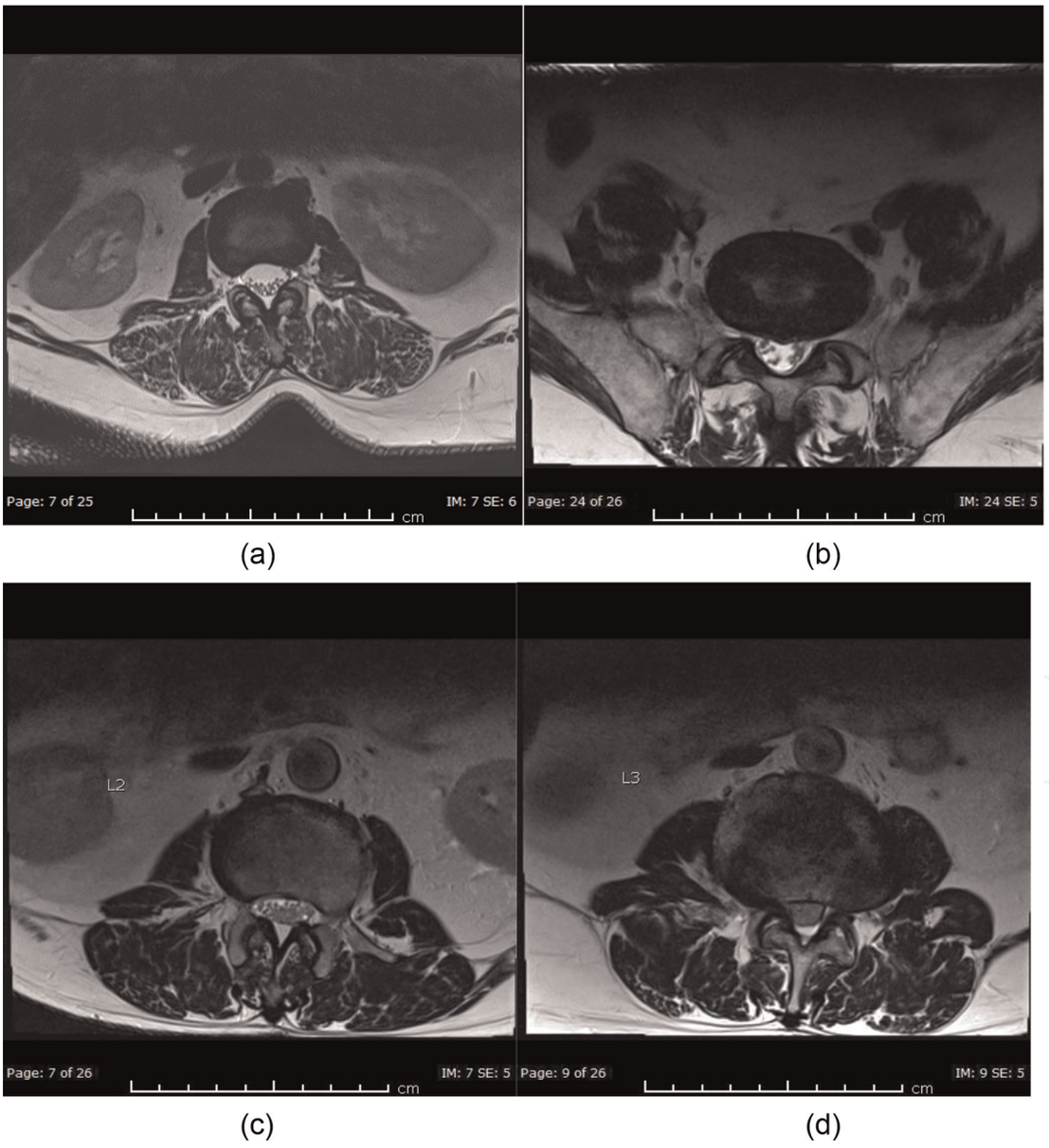

Figure 18.
Degrees of central spinal canal stenosis: (a) no stenosis (b) mild canal stenosis (c) mild canal stenosis (d) severe canal stenosis.

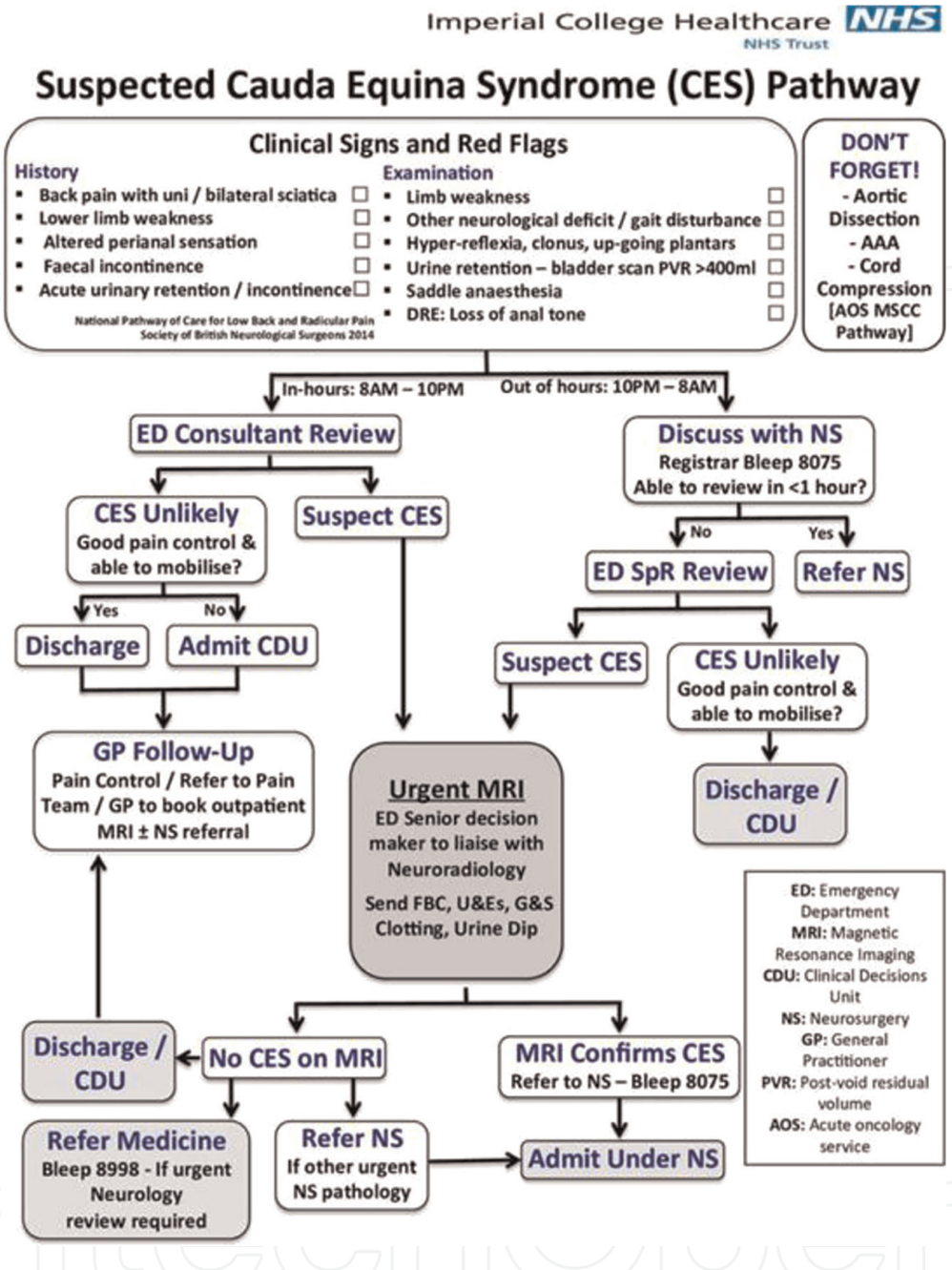

Figure 19.
Summary of the management pathway for suspected cauda equina syndrome cases in the Charing Cross Hospital (CXH) ED. NHS, National Health Service UK.

Nerve conduction studies (NCS) and electromyogram (EMG): These are two electrodiagnostic tests frequently done together to assess the functional integrity of the targeted nerve and therefore the degree and extent of nerve damage (which cannot be achieved by imaging). When done together, they have high specificity and sensitivity regarding the involved nerve root. Electrodiagnostic tests are usually complementary to MRI, especially when there is mismatch between the MRI and clinical findings. These exams can be also used to assess prognosis [19].

They are more helpful in patients with chronic rather than acute onset. This is because abnormality in EMG needs at least two weeks from the clinical onset to be detected (**Figure 19**) [10].

4.15 Treatment

Indications of surgery:
Absolute indications to the surgical decompression include.

- Compressive etiologies.

- Acute to subacute progression of neurologic deficits including lower limb weakness and bladder disturbances.

The surgical intervention is the only option for preventing further deterioration and for recovery, either open microdiscectomy or MIS.

Surgical issue: Some advise a bilateral laminectomy (but this is not mandatory).

Time of surgery: Most evidence supports within 24 hours is desirable if possible, some evidence supports that surgery within 48 is acceptable, and after that the chance of recovery is less and less. The Standard surgery is open microscopic lumbar laminotomy/ laminectomy and discectomy [20] microdiscectomy similar to standard procedure, but a smaller incision is utilized.

Advantages may be cosmetic, shortened hospital stay, and lower blood loss. Overall efficacy is similar to standard discectomy.

4.16 Steps of the procedure

Great care should be taken to position the patient correctly on the operating table to avoid pressure sores and neural peripheral nerve compression. As known, excessive abdominal compression during the prone position can result in excessive epidural bleed due to venous congestion. Landmarks for skin incision are the spinous processes, posterior superior iliac spine, and iliac wings.

A line that is drawn between the posterior superior iliac spines usually projects to the disk level of L4–15. However, this is unreliable, and image intensifier control is necessary in every case.

For CES secondary to lumbar stenosis, the principal surgical option is decompression +/− fusion.

The aim of decompression is to create more room for nerves and thecal sac and release the compression from soft tissue structures as the following disk herniation, hypertrophic ligamentum flavum, hypertrophic facet joints, osteophytes, and narrowed bony canal [21].

Performing complete laminectomies in the past was the standard method for decompression in central spinal stenosis. But from other hand found that complete laminectomies may increase chances of instability at those levels. Recently inter-laminar approaches involve leaving intact portions of the lamina and the connection between the facet joints, laminae, and pedicles, and pars interarticularis approach significantly reduces the chance of instability.

A midline approach exposes the inter-laminar windows at L3–L4 and L4–L5 as well as the facet joints to decompress a spinal stenosis at these levels.

The decompression inter-lamina is opened with a Kerrison rongeur half of laminae, ligamentum flavum, and hypertrophy facet. It is important to release that the narrowest part of the stenosis is typically under the lamina. The remaining part needs to be undercut from the superior and inferior sides, respectively. In some cases, decompression and fusion are necessary where the spine is deemed unstable. In the

case of tumor or inflammation, decompressive laminectomies alone are rarely indicated. The goals of surgical intervention are better accomplished by combining decompression of neural structures, debulking of tumor mass, realignment of spinal deformity, and spinal reconstruction with instrumentation and bone grafting.

4.17 Post-operative actions

Several therapeutic options are available for patients with CES post operation, according to its underlying cause.

- Anti-inflammatory agents including corticosteroids (especially methylprednisolone) can be effective in patients with inflammatory processes, including ankylosing spondylitis.

- Patients with CES caused by an infection should receive appropriate antibiotic therapy.

- Patients affected by CE neoplasms confirmed by tissue [22].

Sampling should be evaluated for chemotherapy and radiation therapy.

- In most cases, treatment with medications alone is not indicated because of a need for emergency release of nerve compression.

- If CES is due to tumors, traumas, metabolic diseases (i.e., lipomatosis), or chronic inflammation, a staged surgery with initial decompression and subsequent operation to correct the underlying cause may be the best approach. This can provide the greatest chance of resolution of CES, without compromising the treatment of the underlying pathology [23].

4.18 Contraindications

- Patients not suitable for surgery due to significant comorbidities or advanced age.

4.19 Complications

Complications can be classified into two groups:

- Procedure-specific complications (i.e., problems related to surgical approach or spinal implants)

- General postsurgical complications (may involve the neuro-logic, pulmonary, cardiovascular, and gastrointestinal systems).

Potential causes of neurologic deficits diagnosed after spine procedures include

- Direct intraoperative trauma to neural structures

- Acute vascular etiologies (including intraoperative hypotension, disruption of crucial segmental vessels supplying the spinal cord during anterior surgical approaches)

- Patient malpositioning during surgery (including brachial plexus injuries, compressive neuropathy involving the peroneal nerve)
- Post-operative bleeding with resultant epidural hematoma and neuronal compression
- Persistent pain
- Bone graft migration with resultant neurologic compromise
- Deep venous thrombosis (DVT)
- Instrumentation failure and/or persistent instability (e.g., due to nonunion or pseudoarthrosis).

4.20 Outcomes and prognosis

The prognosis for CES has traditionally been determined by multiple factors including

- Etiology
- Speed of onset and progression of symptoms: It seems that a more rapid onset corresponds to a poorer outcome
- Duration of compression: Immediate surgical decompression is often recommended to minimize the chances of permanent nerve injury
- Degree of neurologic deficit [20]
- Symptoms and signs: Bladder and/or anal sphincter disturbances or perianal anesthesia seems to be correlated with poor prognosis
- Levels of spinal involvement

5. Rehabilitation program

CES is one of the most common pathology post-surgeries need extensive rehabilitation programs. It is an emergency and needs to be managed by a multidisciplinary team. Rehabilitation programs are the most important part of multidisciplinary team after surgery of CES [24].

A proper rehabilitation results in making the patient functionally able in performing activities of daily living with ease.

Following 4–6 weeks of rehabilitation given significant improvement in movement, muscle strength, pain reduction, and functional sphincters.

6. Conclusions of CES

- CES is a complex of symptoms, and signs need urgent intervention.

- The surgery interventions are a mandatory option for CES to save lower limbs and sphincter function.
- Early intervention within 48 hours from onset will be good results.
- Delay intervention, the lower-extremity motor weakness that occasionally progresses to paraplegia or not improvement after surgery.
- Patients with CES should have undergone rapid radiologic evaluation and diagnosis.
- Lumbosacral trauma: It is important to maintain a high index of suspicion for the diagnosis of CES and its implications both diagnostically and prognostically.
- If the clinician has suspicious, this is a cauda equina syndrome,should obtain a post-void residual urine volume. In addition, trauma patients often receive an indwelling catheter at the time of admission to the emergency department.
- A rectal examination and evaluation of perianal sensation is mandatory when assessing any patient with acute onset of severe back pain or significant lumbosacral trauma.
- If an MRI is inadequate or contraindications, a CT-myelography) is alternative.
- For good results the rehabilitation after surgery is the key point for recovery or improvement.

7. Case reports of Cauda equina syndrome

- **Case 1:** 39 year old male patient complaining of more than one year history of back pain, presented to ED complaining of tingling sensation in the pelvis and thighs, with acute urinary retention since one day.
- **Case 2:** 34 year old patient with lung cancer with 2 weeks history of back pain, presented to ED complaining of new onset of saddle anesthesia ,urinary and stool incontinence. Physical exam shows lower limb weakness (**Figures 12** and **13**).
- **Case 3:** 35 year old male patient sustained multiple fractures after full from height complaining of severe back pain and urinary retention. CT Showed burst fracture of L4 vertebral body encroaching on the spinal canal. MRI revealed compression of cauda equina by the displaced bone fragment (**Figures 14** and **15**).
- **Case 4:** 50-year-old diabetic and hypertensive male patient presented to ED complaining of acute back pain and lower limb weakness with urine retention and perianal paresthesia. Patient was febrile. Blood test showed significant leukocytosis.
- **Case 5:** 38 year old male patient presented with progressive right sciatica, decreased sensation over the right lower limb as well as urine and stool incontinence of the last one month. Deep tendon reflexes were increased in physical examination. Diagnosed as conus medullaris syndrome.

Conflict of interest

No conflict of interest.

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