Diagnosis of Cauda Equina Syndrome in the Emergency Department

David Metcalfe *clinical lecturer in emergency medicine*[1,2], Ingrid Hoeritzauer *consultant neurologist*[3,4], Michelle Angus *consultant physiotherapist*[5], Alex Novak *consultant in emergency medicine*[2], Michael Hutton *consultant spinal surgeon*[6], Julie Woodfield *honorary clinical lecturer*[3].

1. Oxford Trauma and Emergency Care (OxTEC), Nuffield Department of Orthopaedics, Rheumatology and Musculoskeletal Sciences, University of Oxford, Oxford, U.K.
2. Emergency Medicine Research in Oxford (EMROx), John Radcliffe Hospital, Oxford, U.K.
3. Centre for Clinical Brain Sciences, University of Edinburgh, Edinburgh, U.K.
4. Salford Royal NHS Foundation Trust, Salford, U.K.
5. Royal Devon and Exeter NHS Foundation Trust, Exeter, U.K.

Correspondence to Dr D. Metcalfe, Kadoorie Centre for Critical Care Research, Level 3, John Radcliffe Hospital, Headley Way, Oxford, OX3 9DU. david.metcalfe@ndorms.ox.ac.uk

Key points:

- The care pathway for CES should aim to diagnose or exclude this rare but potentially devastating condition as soon as possible.
- No single examination finding, or combination of findings, is sufficient to exclude CES in a patient with symptoms suspicious for the diagnosis.
- Any patient with back and/or radicular lower limb pain that has recently developed urinary symptoms, saddle sensory changes, bowel dysfunction, sexual dysfunction, or severe or progressive bilateral lower limb neurological deficits should undergo emergency MRI scanning to exclude the diagnosis of CES.

ABSTRACT

Cauda equina syndrome (CES) is a spinal emergency that can be challenging to identify from amongst the many patients presenting to Emergency Departments with low back and/or radicular leg pain. This article presents a practical guide to the assessment and early management of patients with suspected CES as well as an up-to-date review of the most important studies in this area that should inform clinical practice in the ED.

Low back and radicular leg pain are common reasons for attending an Emergency Department (ED). Although these symptoms are typically self-limiting, around 1 in 300 patients with low back pain in the ED have cauda equina syndrome (CES)[1]. Identifying CES can be challenging but delayed diagnosis may lead to potentially avoidable long-term disability, including permanent lower limb paralysis/paresis, pain or sensory disturbance, impaired bladder and/or bowel control, and sexual dysfunction in a predominantly working age population[2].

This review presents a practical guide to the assessment and early management of patients with suspected CES. It is informed by a broad literature search (using the term "cauda equina syndrome") and synthesises the best available research to help inform clinical practice in the ED.

Anatomy and aetiology

The "cauda equina" describes the bundle of nerve roots that extends caudally from the spinal cord, which usually terminates around the L1/L2 vertebral level in adults (Figure 1). It was described in 1605 by the French anatomist Andre du Laurens who wrote that the lower part of the spinal cord "*dispersio caudae equinae similis*" ("scatters like a horse's tail")[3]. The nerve roots that make up the cauda equina provide somatic, visceral, and autonomic innervation to the perineum, bladder, bowel and sexual function and lower extremities (Box 1).

Cauda equina syndrome (CES) describes the symptoms that occur when multiple lumbosacral nerve roots are compromised within the vertebral canal. The most common cause is compression due to a large central disc herniation, usually at L4/L5 or L5/S1. Other causes of cauda equina compromise include malignancy (primary or metastatic), epidural abscess or haematoma, fracture, vascular malformations, nerve root infiltration, chemical irritation, and degenerative changes. Compression of the cauda equina can lead to permanent nerve root injury as a result of direct compression, venous congestion, and/or ischaemia[4]. The cauda equina is particularly vulnerable to ischaemia because it is supplied by end arteries (without anastomoses) and there is a region of relative hypovascularity just below the conus medullaris[5]. Cauda equina compression may also be caused by lumbar spinal stenosis,

which is a degenerative condition that may affect up to 60% of adults aged ≥65[6]. Patients with features of cauda equina compromise developing slowly as a result of degenerative lumbar spinal stenosis are less likely to present to an ED and/or to require emergent management. So-called "grumbling" CES is therefore beyond the scope of this article but interested readers may wish to access a recent review of this condition published elsewhere[6].

Why is early diagnosis important?

Cauda equina syndrome is an emergency and surgical decompression aims to reduce the duration of neural compromise and halt progression of paralysis, impaired bladder and/or bowel control, and sexual dysfunction[7]. Unfortunately, misdiagnosis and diagnostic delay in the ED still occur and lead to potentially avoidable harm for this group of patients[8].

A number of factors may contribute to diagnostic delay. First, although low back pain, sciatica, and bladder problems are very common, CES is comparatively rare[9]. Based on an annual incidence of 2.7 cases per 100,000 population[10], there are fewer than 2000 patients with confirmed CES across the UK each year. Second, many "red flags" for CES occur commonly in the low back pain population. For example, pain on movement may masquerade as lower limb motor deficit, pain inhibition may cause urinary symptoms, and altered bowel habit is prevalent amongst patients with reduced mobility and those using opioids/gabapentinoids[11]. Third, clinical judgement performs poorly in terms of identifying patients with CES[9, 12] and the only clinical prediction rule that has been proposed is insufficiently sensitive to exclude CES[13]. Finally, the definitive diagnostic modality (i.e. MRI) is not readily available to many acute hospitals out of hours[14]. These factors contribute to patient harm, and – perhaps unsurprisingly – CES is a prominent cause of healthcare litigation[8, 15]. One survey in the UK found that 69% of CES litigation claims alleged diagnostic error or delayed management, and most of these claims (88%) were successful with mean costs in the region of £400,000[8].

What should happen at initial assessment?

Patients with suspected CES should be escalated at triage and prioritised for clinical assessment. This is particularly important when early assessment might permit easier access to MRI within normal working hours. As patients are often in significant pain and require assessment on an examination trolley, they should ideally be nursed in a bed-space rather than in the waiting room.

Prompt and generous analgesia should be administered to relieve pain, facilitate effective clinical examination and MRI scanning, and to minimise ED length of stay in patients later deemed suitable for discharge. The need to achieve rapid and effective pain control mean that slow up-titration of analgesics (as recommended by the World Health Organization pain ladder) is unlikely to be appropriate.

There is trial evidence to support the use of a range of drugs (including paracetamol, non-steroidal anti-inflammatory drugs, and opioids) for patients with acute back pain[16]. There is little evidence to support the use of benzodiazepines for back pain alone[17] but they are sometimes tried in patients with evidence of significant lumbar muscle spasm. A combination of agents is usually required to treat acute back and leg pain, and drug choice should be tailored to individual patients depending on their preferences and risk of side effects. Intravenous, intramuscular, and/or rectal administration is likely to achieve analgesia more rapidly than enteral administration. Trial evidence to support the use of specific pharmacological agents for radicular pain is limited[18].

How should patients be evaluated?

The features of CES are highly variable depending on the degree, pattern, and duration of nerve root compression. Patients with CES usually have radicular lower limb and/or back pain but this is not ubiquitous and may not be a dominant symptom[19-21]. Any patient presenting to an ED with low back and/or leg pain, altered bladder or bowel disturbance, new sexual dysfunction, and/or abnormal lower limb neurology should therefore undergo an assessment of their spine and lower limbs for evidence of CES. Although the focus should be on the prompt identification of patients with CES, emergency physicians should be alert to alternative causes, particularly as this may influence the choice of radiological imaging.

Important pitfalls include failure to identify serious pathology above the lumbosacral spine (e.g. cervicothoracic compression, discitis or malignancy) or to consider non-spinal causes (e.g. acute aortic syndrome or primary urological causes of urinary retention or incontinence)[22].

History

A thorough history should elicit symptoms of cauda equina compression as well as considering potential aetiologies (Box 2) and alternative causes for the presentation. The symptoms that have been associated with CES include saddle sensory change[9, 19-21, 23] (sometimes reported as altered sensation on wiping, urinating, or defecation), urinary disturbance (frequency[20, 24], incontinence[9, 19-21, 24, 25], or retention[9, 19-21, 24, 25]), bowel incontinence[9, 19, 20, 24], bilateral lower limb weakness[9], and bilateral radicular leg pain[9, 19, 20, 25]. However, a recent systematic review of diagnostic test accuracy studies reported low sensitivity and specificity across all individual symptoms[12]. As it is important not to miss *any* patients with CES, these features should generally be elicited to "rule in" rather than to exclude the diagnosis.

As CES is an urgent surgical condition, relevant comorbidities and potential anaesthetic and surgical risk factors should also be considered in the history. For example, the time last ate, anti-coagulant use, breast feeding, potential or confirmed pregnancy, or significant cardiorespiratory comorbidities may impact on investigation, management, and surgical timing. Features of non-discogenic causes of CES may be elicited at this stage to guide adjunctive investigations, e.g. blood cultures in an intravenous drug user with rigors and weight loss.

Physical examination

As with the history, no single physical finding can be used to exclude a diagnosis of cauda equina compression[12]. This fact has been misinterpreted as suggesting that physical examination has minimal role in the assessment of suspected cauda equina compression[26]. However, careful physical examination is important for a number of reasons:

1. Careful neurological examination will help identify alternative causes for symptoms, such as spinal pathology outside the lumbosacral spine or primary urological problems.

2. The priority should be to avoid missing *any* patients with CES. The patients who may gain most from decompression are those presenting early whose neurological signs may be subtle or absent[26].
3. Baseline neurological status is necessary to measure deteriorating neurological function, which may inform the urgency of decompression, and to counsel patients regarding likely recovery.

Patients with suspected CES should undergo a comprehensive physical examination of the spine and lower limbs. This includes testing sensation in all lumbar and sacral dermatomes, power in the lower limb myotomes, and lower limb reflexes. Deficits that may be observed in CES include lower limb weakness, peri-anal sensory change (S2-S4) and diminished reflexes (knee L4 and ankle jerk S1). Brisk reflexes, increased tone, and clonus suggest an upper motor neurone cause for symptoms, i.e. above the level of the cauda equina. A stocking distribution of numbness suggests peripheral neuropathy or upper motor neurone causes and cannot be readily explained by compression of lumbosacral nerve roots.

Digital rectal examination (DRE) can be used to assess anal tone (internal anal sphincter), anal squeeze (external anal sphincter), anal canal sensation, and the bulbocavernosus reflex. However, DRE performs poorly compared with objective measures of anal tone in both simulation[27] and clinical studies[28]. Reduced anal squeeze has also been proposed as a late sign[29], which would limit its value for early detection of CES. Overall, there is mixed evidence about the usefulness of DRE but a recent meta-analysis reported low sensitivity for reduced anal tone (23-53%), anal squeeze (29%), and anal canal sensation (40%)[30]. Although traditionally taught as a mandatory part of assessing a patient with suspected CES, the evidence does not support a need for DRE during the initial assessment or its use in determining whether a patient should undergo MRI scanning. However, DRE may subsequently have a role to establish a functional baseline in patients with confirmed CES before they undergo decompression. Although DRE is not required, peri-anal sensation should still be assessed as part of testing the S2-S4 dermatomes.

The bulbocavernosus reflex may be elicited by feeling for anal sphincter contraction In response to pressure on the clitoris or glans penis[31], which suggests intact reflex arcs of the

S2-S4 spinal segments. Impairment of the bulbocavernosus reflex was found to be associated with CES by electrophysiological studies in the 1970s. Although one small clinical study (n=142 with 10 cases) reported that the absence of this reflex was 100% sensitive for CES[31], this manoeuvre is unlikely to be practical or appropriate in contemporary medical practice.

Bladder scanning can be helpful as an adjunct to the physical examination and a post-void residual ≥200ml has been associated with CES[32, 33]. However, normal bladder emptying is insufficiently sensitive to exclude CES[9] and over-reliance on bladder scanning may provide false reassurance and contribute to diagnostic delay. Although bladder scanning showed promise as an adjunct in earlier diagnostic studies[32, 33], three subsequent reports have highlighted that up to 80% of patients with confirmed CES have a post-void residual <200ml[9, 34, 35]. Bladder scanning may therefore have a role in ruling *in* CES but should not be used to exclude the diagnosis[34]. Bladder scanning may also be helpful in identifying painless urinary retention requiring early catheterisation[35].

Investigations

Any patient with an acute onset or progressive constellation of symptoms compatible with CES (Box 3) should undergo emergency MRI[36]. Patients with low back pain and/or unilateral radicular leg pain without features of CES should not routinely undergo MRI in the ED.

A normal physical examination does not obviate the need for MRI if the history is suspicious for CES. However, the diagnosis of CES requires both clinical features *and* explanatory findings on spinal imaging. A large central disc prolapse can be diagnosed on MRI but this alone is not sufficient for a diagnosis of CES. Although clinicians should have a low threshold for considering the diagnosis, there is no role for indiscriminate use of MRI to screen for cauda equina compression in the absence of concerning clinical features.

The gold-standard diagnostic test is MRI of the lumbosacral spine, which may show compression of the cauda equina by a large central disc prolapse (Figure 2) or other causes. MRI provides the best possible soft tissue resolution for the evaluation of lumbar spine

pathology. As MRI is sensitive to motion and can be uncomfortable for patients, adequate analgesia is required prior to scanning.

Patients should be counselled that MRI will only identify a structural cause for symptoms in a minority of cases. They should understand that some features (e.g. minor disc bulges) are normal and do not mean that anything is "wrong" with the spine. Providing patients with this information early, ideally before the MRI scan, can influence whether someone engages wholeheartedly with physiotherapy after discharge or feels they have a "degenerative" spine and so becomes fearful of movement. This information can be reinforced by standardised patient-information leaflets[37].

Limited sequence MRI (e.g. T2 sagittal +/- axial sequences[13]) has been associated with reduced scan time and improved patient experience. Two case series report having identified 15 cases amongst 210 patients with suspected CES without missing any cases using limited sequence MRI (sensitivity 100% [95% CI 78-100%]; specificity 100% [98-100%])[14, 38]. However, limited sequence scans are less sensitive for other abnormalities (e.g. nerve root compression)[39] and some patients with "normal" limited sequence scans may subsequently require re-imaging with full protocol MRI. The two studies evaluating limited sequence protocols did not directly compare these scans to full protocol MRI or follow patients up for other missed diagnoses[14, 38]. Although limited sequence MRI can reduce scanning time from 19 to 10 minutes[13], other steps (e.g. completion of a safety questionnaire and transfer of someone in pain on and off the scanner table) will not be affected by the choice of MRI protocol. Similarly, if the MRI does not show cauda equina compression a T2 sagittal of cervical and thoracic spine can demonstrate other structural pathologies acutely with minimal additional scanning time. However, concurrent imaging of the thoracic spine may require entering the scanner head first, which may increase the feeling of claustrophobia for some patients.

Alternatives to MRI have been explored for those unable to undergo MRI and when MRI is unavailable. CT has been used to risk stratify patients with suspected CES in one study that found thecal sac effacement <50% (measured by CT with or without contrast) excluded cauda equina compression in their population[40]. However, CT cannot match MRI for soft tissue

resolution, exposes patients to ionising radiation, and risks delay between CT reporting and subsequent MRI scanning. It is therefore not an acceptable choice of imaging modality unless MRI is contraindicated in which case both plain CT (windowed specifically for the canal) and CT myelography have been used. CT myelography is an invasive procedure and requires the placement of a needle and dye directly into the spinal canal as well as expertise that may not be readily available, especially out of hours[41].

Plain radiographs have no role in the assessment of patients with suspected CES.

How long do I have to make the diagnosis?

The aim of diagnosing CES promptly is to expedite decompressive surgery. However, the timing of decompressive surgery remains controversial. Nerve root viability may be threatened as early as 6 hours[42] and some retrospective studies suggest that decompression within 48 hours is associated with better outcomes than delayed intervention, although this finding is not universal[43]. The thresholds (e.g. <24 or <48 hours) reported by these retrospective studies have sometimes been misinterpreted as suggesting that there is a period within which it is safe to delay decompression[43, 44]. However, it is more likely that on-going damage to the cauda equina is a continuous process and that there are no true "safe" time thresholds for intervention. It is for this reason that the Society of British Neurological Surgery and British Association of Spinal Surgeons warn that "nothing is to be gained by delaying surgery [which should be] undertaken at the earliest opportunity… considering the duration and clinical course of symptoms and signs, and the potential for increased morbidity while operating in the night"[45].

Unfortunately, the available retrospective studies are confounded because the timing of surgery is influenced by factors that are independently associated with outcome, such as disc size and degree of neurological deficit on examination. A recent prospective study of patients with CES found that time to decompression was not associated with outcome at either one month or one year[35]. The only factor associated with outcome was symptom severity pre-operatively (urinary retention yes/no). However, those patients with urinary retention pre-operatively were operated on more quickly than those without which could lead to

confounding. It therefore remains unclear whether long-term outcome is determined by the duration of nerve root compression and/or pressure severity at the time of disc prolapse.

Although the question of timing remains controversial, it is plausible that continued impingement of the nerve roots leads to on-going harm[43] and the courts have certainly expressed their expectation that the cauda equina be decompressed expeditiously[46]. Emergency physicians should therefore prioritise the earliest possible diagnosis and onward referral to a spinal surgeon[29, 45].

Even after diagnosis, surgical decompression may be delayed by logistical challenges, such as referral to a spinal surgeon, transfer between hospitals, and access to an appropriate operating theatre. However, these processes cannot begin without a diagnosis, which should therefore be sought at the earliest possible opportunity.

What if the MRI does not show cauda equina compression?

Between 5-11% undergoing MRI for suspected CES are found to have cauda equina compression and require immediate surgical referral. If the MRI does not show cauda equina compression, it may reveal an alternative structural cause, such as a prolapsed disc causing isolated nerve root compression[14]. However, once CES has been excluded by MRI, the patient journey does not end. The differential diagnosis for CES symptoms is broad (Box 2) and alternative causes should be considered[11, 47]. Although MRI is highly sensitive, structural abnormalities may still be overlooked by limited sequence MRI scans undertaken for the sole purpose of excluding cauda equina compression[14, 38, 39] and even full-sequence MRI may not detect abnormalities such as small inflammatory cord lesions.

One prospective cohort study found that 7% patients with suspected CES had important alternative causes for their symptoms: inflammatory cord lesions, polyneuropathy, vertebral fracture, abscess, discitis, cervical myelopathy, cord infarct, tumour, and plexopathy[47]. A further 2% patients were discharged after normal lumbosacral imaging but subsequently found to have an alternative cause: cord inflammation, cervical epidural haematoma, and sacral chordoma.

Hoeritzauer et al have proposed a list of “red flags” that may alert emergency physicians to the possibility of an important alternative cause (Box 4)[11]. Although these are not emergency diagnoses, they highlight the continued importance of clinical assessment and the role for follow-up and/or referral in some cases despite a normal MRI scan.

Up to 85% of patients will not have an identifiable structural cause for their symptoms[1]. There is a small but growing literature that recognises “scan-negative CES”[11, 48], which may be caused by central and/or peripheral inhibition of bladder, bowel and lower limb function as well as acute pain, medications, panic, and functional leg weakness. For example, severe pain (such as that caused by single nerve root compression) may lead to pelvic floor contraction causing urinary retention or inability to contract the pelvic floor causing urinary incontinence even though the neurological injury could not directly cause structural bladder, bowel or sexual visceral damage[50]. Severe pain from nerve root compression or mechanical back pain can also cause acute panic and dissociation.

It is helpful to counsel patients about the likely trajectory of their symptoms. Patients presenting to an ED with non-specific low back pain typically experience rapid reductions in pain intensity over the first week but may still experience mild symptoms 6 months later[49]. Patients being discharged without a diagnosis of CES require a good explanation, adequate and regular short-term analgesia (3-5 days regularly before reducing), advice to ensure bowels are moving early (with the help of laxatives if required) and early gentle mobilisation. The UK National Low Back and Radicular Pain Pathway recommends that physiotherapy referral should be considered if the pain remains after 2 weeks. Patients with pre-existing stress incontinence should be encouraged to contact their GP for onward pelvic health physiotherapy which leads to resolution in over half of cases[50]. Those with ongoing leg weakness should be considered for referral to a spinal surgeon or a neurologist depending on whether a structural cause was identified.

System considerations

The management of suspected CES is challenging in some settings given the limited value of clinical assessment and relative unavailability of definitive imaging. However, the consequences of delayed CES diagnosis can be devastating and so health *systems* should prioritise not missing the diagnosis. Clear guidelines and pathways are necessary to:

1. Ensure that patients receive consistent assessment, investigation, and treatment regardless of where and when they present.
2. Reduce unwarranted variation in decision making and over-reliance on clinical judgement, which is insufficiently sensitive to exclude the diagnosis.
3. Reduce delays caused by negotiations between individual clinicians about access to imaging and transfer between hospitals.

Referral pathways are particularly important in the case of hospitals without access to all-hours MRI scanning. Transfer of patients between hospitals for the purposes of diagnostic imaging is expensive[51] and uncomfortable in cases of severe back pain but may be necessary in some cases. Systems should accept a substantial proportion of "negative" scans and not chase higher yields because it is critical not to miss cases of CES[34]. Protocols that facilitate direct ED access to emergency MRI can expedite imaging for suspected CES without overburdening radiology services[52, 53]. The optimal configuration of diagnostic and spinal surgical services is unknown and will likely vary between health settings. In the meantime, local, regional, and national agreements should be sought to streamline the assessment of patients with suspected and confirmed CES. For example, this has recently been achieved in the UK by the multidisciplinary adoption of a National Suspected CES Pathway[36], which was developed by representatives from all stakeholder groups and endorsed by almost all of the relevant professional organisations[36]. Such a pathway ensures that all components of the health system have aligned expectations and are working towards a common goal.

Conclusion

Cauda equina syndrome is a spinal emergency that can be challenging to diagnose but quickly results in severe disability. Clinicians should assess patients with suspected CES carefully and adopt a low threshold for organising MRI. However, the rapid diagnosis and decompression

of patients with CES requires careful pathway design and a whole system approach with engagement from EDs, radiology, and specialist spinal services.

LEGENDS

Figure 1: Anatomy of the cauda equina showing its origin arising caudally from the lower end of the conus medullaris at the L1/L2 vertebral level.

Figure 2: Axial (top left) and sagittal (top right) T2-weighted views of lumbosacral spine MRI showing normal anatomy. (a) Intervertebral disc, (b) intervertebral foramen, (c) cerebrospinal fluid within the thecal sac (high T2 signal), (d) individual nerve roots of the cauda equina (appearing as darker grey within the CSF), (e) spinous process, (f) paraspinal muscles. Lumbosacral spine MRI (bottom left and right) showing a large central L4/L5 disc herniation compressing the cauda equina and completely effacing the CSF (arrows).

CONFLICTS

None declared.

CONTRIBUTORSHIP

DM conceived the need for this article and wrote the first draft. DM, IH, MA, AN, MH, and JW helped develop the idea, and made important critical revisions to the manuscript. All authors approved the final version.

FUNDING

DM is supported by an NIHR Advanced Fellowship and the NIHR Oxford Biomedical Research Centre.

References

1. Hoeritzauer I, Wood M, Copley PC, Demetriades AK, Woodfield J: **What is the incidence of cauda equina syndrome? A systematic review**. *J Neurosurg Spine* 2020:1-10.
2. Korse NS, Veldman AB, Peul WC, Vleggeert-Lankamp CLA: **The long term outcome of micturition, defecation and sexual function after spinal surgery for cauda equina syndrome**. *PLoS One* 2017, **12**(4):e0175987.
3. Olry R, Haines DE: **Between Andre Du Laurens' horse tail and William Cadogan's pony tail**. *J Hist Neurosci* 2012, **21**(3):327-331.
4. Mauffrey C, Randhawa K, Lewis C, Brewster M, Dabke H: **Cauda equina syndrome: an anatomically driven review**. *Br J Hosp Med (Lond)* 2008, **69**(6):344-347.
5. Grasso G, Munakomi S, Salli M: **Red Flag for Cauda Equina Syndrome in Symptomatic Lumbar Disc Herniation**. *World Neurosurg* 2020, **143**:232-234.
6. Comer C, Finucane L, Mercer C, Greenhalgh S: **SHADES of grey - The challenge of 'grumbling' cauda equina symptoms in older adults with lumbar spinal stenosis**. *Musculoskelet Sci Pract* 2020, **45**:102049.
7. Hogan WB, Kuris EO, Durand WM, Eltorai AEM, Daniels AH: **Timing of Surgical Decompression for Cauda Equina Syndrome**. *World Neurosurg* 2019, **132**:e732-e738.
8. Hamdan A, Strachan RD, Nath F, Coulter IC: **Counting the cost of negligence in neurosurgery: Lessons to be learned from 10 years of claims in the NHS**. *Br J Neurosurg* 2015, **29**(2):169-177.
9. Angus M, Curtis-Lopez CM, Carrasco R, Currie V, Siddique I, Horner DE: **Determination of potential risk characteristics for cauda equina compression in emergency department patients presenting with atraumatic back pain: a 4-year retrospective cohort analysis within a tertiary referral neurosciences centre**. *Emerg Med J* 2021.
10. Woodfield J, Lammy S, Jamjoom AA, Fadelalla MA, Copley PC, Arora M, Glasmacher SA, Abdelsadg M, Scicluna G, Poon MT *et al*: **Demographics of Cauda Equina Syndrome: A Population Based Incidence Study**. *Neuroepidemiology* 2022.
11. Hoeritzauer I, Stanton B, Carson A, Stone J: **'Scan-negative' cauda equina syndrome: what to do when there is no neurosurgical cause**. *Pract Neurol* 2022, **22**(1):6-13.
12. Dionne N, Adefolarin A, Kunzelman D, Trehan N, Finucane L, Levesque L, Walton DM, Sadi J: **What is the diagnostic accuracy of red flags related to cauda equina syndrome (CES), when compared to Magnetic Resonance Imaging (MRI)? A systematic review**. *Musculoskelet Sci Pract* 2019, **42**:125-133.
13. Angus M, Berg A, Carrasco R, Horner D, Leach J, Siddique I: **The Cauda Scale - Validation for Clinical Practice**. *Br J Neurosurg* 2020, **34**(4):453-456.
14. Gnanasekaran R, Beresford-Cleary N, Aboelmagd T, Aboelmagd K, Rolton D, Hughes R, Seel E, Blagg S: **Limited sequence MRI to improve standards of care for suspected cauda equina syndrome**. *Bone Joint J* 2020, **102-B**(4):501-505.
15. Machin JT, Hardman J, Harrison W, Briggs TWR, Hutton M: **Can spinal surgery in England be saved from litigation: a review of 978 clinical negligence claims against the NHS**. *Eur Spine J* 2018, **27**(11):2693-2699.
16. Oliveira CB, Amorim HE, Coombs DM, Richards B, Reedyk M, Maher CG, Machado GC: **Emergency department interventions for adult patients with low back pain: a systematic review of randomised controlled trials**. *Emerg Med J* 2021, **38**(1):59-68.

17. Friedman BW, Irizarry E, Solorzano C, Khankel N, Zapata J, Zias E, Gallagher EJ: **Diazepam Is No Better Than Placebo When Added to Naproxen for Acute Low Back Pain**. *Ann Emerg Med* 2017, **70**(2):169-176 e161.
18. National Institute for Health and Care Excellence. In: *Evidence review for pharmacological management of sciatica: Low back pain and sciatica in over 16s: assessment and management: Evidence review A.* edn. London; 2020.
19. Balasubramanian K, Kalsi P, Greenough CG, Kuskoor Seetharam MP: **Reliability of clinical assessment in diagnosing cauda equina syndrome**. *Br J Neurosurg* 2010, **24**(4):383-386.
20. Domen PM, Hofman PA, van Santbrink H, Weber WE: **Predictive value of clinical characteristics in patients with suspected cauda equina syndrome**. *Eur J Neurol* 2009, **16**(3):416-419.
21. Rooney A, Statham PF, Stone J: **Cauda equina syndrome with normal MR imaging**. *J Neurol* 2009, **256**(5):721-725.
22. Mayo E, Herdman G: **Acute Aortic Thrombus Presenting as Cauda Equina Syndrome**. *J Emerg Med* 2020, **58**(5):802-806.
23. Raison NT, Alwan W, Abbot A, Farook M, Khaleel A: **The reliability of red flags in spinal cord compression**. *Arch Trauma Res* 2014, **3**(1):e17850.
24. Gooding BW, Higgins MA, Calthorpe DA: **Does rectal examination have any value in the clinical diagnosis of cauda equina syndrome?** *Br J Neurosurg* 2013, **27**(2):156-159.
25. Bell DA, Collie D, Statham PF: **Cauda equina syndrome: what is the correlation between clinical assessment and MRI scanning?** *Br J Neurosurg* 2007, **21**(2):201-203.
26. Todd NV: **Clinical Examination and the Diagnosis of Cauda Equina Syndrome. More Examination, Not Less**. *Global Spine J* 2022, **12**(6):1301-1302.
27. Sherlock KE, Turner W, Elsayed S, Bagouri M, Baha L, Boszczyk BM, McNally D: **The Evaluation of Digital Rectal Examination for Assessment of Anal Tone in Suspected Cauda Equina Syndrome**. *Spine (Phila Pa 1976)* 2015, **40**(15):1213-1218.
28. Tantiphlachiva K, Rao P, Attaluri A, Rao SS: **Digital rectal examination is a useful tool for identifying patients with dyssynergia**. *Clin Gastroenterol Hepatol* 2010, **8**(11):955-960.
29. Todd NV: **Guidelines for cauda equina syndrome. Red flags and white flags. Systematic review and implications for triage**. *Br J Neurosurg* 2017, **31**(3):336-339.
30. Tabrah J, Wilson N, Phillips D, Bohning D: **Can digital rectal examination be used to detect cauda equina compression in people presenting with acute cauda equina syndrome? A systematic review and meta-analysis of diagnostic test accuracy studies**. *Musculoskelet Sci Pract* 2022, **58**:102523.
31. Zusman NL, Radoslovich SS, Smith SJ, Tanski M, Gundle KR, Yoo JU: **Physical Examination Is Predictive of Cauda Equina Syndrome: MRI to Rule Out Diagnosis Is Unnecessary**. *Global Spine J* 2022, **12**(2):209-214.
32. Alshahwani AA, Boktor J, Elbahi A, Banerjee P: **A Systematic Review of the Value of a Bladder Scan in Cauda Equina Syndrome Diagnosis**. *Cureus* 2021, **13**(4):e14441.
33. Katzouraki G, Zubairi AJ, Hershkovich O, Grevitt MP: **A prospective study of the role of bladder scanning and post-void residual volume measurement in improving diagnostic accuracy of cauda equina syndrome**. *Bone Joint J* 2020, **102-B**(6):677-682.

34. Todd N, Dangas K, Lavy C: **Post-void bladder ultrasound in suspected cauda equina syndrome-data from medicolegal cases and relevance to magnetic resonance imaging scanning**. *Int Orthop* 2022, **46**(6):1375-1380.
35. Woodfield J, Hoeritzauer I, Jamjoom AAB, Jung J, Lammy S, Pronin S, Hannan CJ, Watts A, Hughes L, Moon RDC *et al*: **Presentation, management, and outcomes of cauda equina syndrome up to one year after surgery, using clinician and participant reporting: A multi-centre prospective cohort study**. *Lancet Reg Health Eur* 2023, **24**:100545.
36. Hutton M: **Spinal Surgery: National Suspected Cauda Equina Syndrome (CES) Pathway**. In. London, U.K.; 2023.
37. **BestMSK Spinal MRI: Patient Advice** [https://www.england.nhs.uk/wp-content/uploads/2022/10/B1206-Best-MSK-Spinal-Patient-Advice-030822.pdf]
38. Beresford-Cleary N, Lane J, Fullerton M, Butler K, Hughes R, Blagg S: **Is there a role for limited sequence MRI in the evaluation of cauda equina syndrome?** *The Spine Journal* 2017, **17**(3):S20.
39. Chawalparit O, Churojana A, Chiewvit P, Thanapipatsir S, Vamvanij V, Charnchaowanish P: **The limited protocol MRI in diagnosis of lumbar disc herniation**. *J Med Assoc Thai* 2006, **89**(2):182-189.
40. Peacock JG, Timpone VM: **Doing More with Less: Diagnostic Accuracy of CT in Suspected Cauda Equina Syndrome**. *AJNR Am J Neuroradiol* 2017, **38**(2):391-397.
41. Ozdoba C, Gralla J, Rieke A, Binggeli R, Schroth G: **Myelography in the Age of MRI: Why We Do It, and How We Do It**. *Radiol Res Pract* 2011, **2011**:329017.
42. Delamarter RB, Sherman J, Carr JB: **Pathophysiology of spinal cord injury. Recovery after immediate and delayed decompression**. *J Bone Joint Surg Am* 1995, **77**(7):1042-1049.
43. Chau AM, Xu LL, Pelzer NR, Gragnaniello C: **Timing of surgical intervention in cauda equina syndrome: a systematic critical review**. *World Neurosurg* 2014, **81**(3-4):640-650.
44. Thakur JD, Storey C, Kalakoti P, Ahmed O, Dossani RH, Menger RP, Sharma K, Sun H, Nanda A: **Early intervention in cauda equina syndrome associated with better outcomes: a myth or reality? Insights from the Nationwide Inpatient Sample database (2005-2011)**. *Spine J* 2017, **17**(10):1435-1448.
45. Society of British Neurological Surgeons, British Association of Spine Surgeons: **Standards of care for investigation and management of cauda equina syndrome.** In.; 2018.
46. **Hewes v. West Hertfordshire Acute Hospitals NHS Trust & Ors [2020] EWCA Civ 1523**. In: *Hewes.* 2020.
47. Hoeritzauer I, Carson A, Statham P, Panicker JN, Granitsiotis V, Eugenicos M, Summers D, Demetriades AK, Stone J: **Scan-Negative Cauda Equina Syndrome: A Prospective Cohort Study**. *Neurology* 2021, **96**(3):e433-e447.
48. Gibson LL, Harborow L, Nicholson T, Bell D, David AS: **Is scan-negative cauda equina syndrome a functional neurological disorder? A pilot study**. *Eur J Neurol* 2020, **27**(7):1336-1342.
49. Coombs DM, Machado GC, Richards B, Oliveira CB, Herbert RD, Maher CG: **Clinical course of patients with low back pain following an emergency department presentation: a systematic review and meta-analysis**. *Emerg Med J* 2021, **38**(11):834-841.

50. Dumoulin C, Cacciari LP, Hay-Smith EJC: **Pelvic floor muscle training versus no treatment, or inactive control treatments, for urinary incontinence in women**. *Cochrane Database Syst Rev* 2018, **10**(10):CD005654.
51. Hutton M: **Spinal Services GIRFT Programme National Specialty Report**. In. London, U.K.; 2019.
52. Buell KG, Sivasubramaniyam S, Sykes M, Zafar K, Bingham L, Mitra A: **Expediting the management of cauda equina syndrome in the emergency department through clinical pathway design**. *BMJ Open Qual* 2019, **8**(4):e000597.
53. Fraig H, Gibbs DMR, Lloyd-Jones G, Evans NR, Barham GS, Dabke HV: **Early experience of a local pathway on the waiting time for MRI in patients presenting to a UK district general hospital with suspected cauda equina syndrome**. *Br J Neurosurg* 2022:1-7.

Box 1: Function of the lumbosacral nerve roots that compose the cauda equina

Nerve roots	Function	Signs and symptoms
L2-S2	Lower limb motor function	Lower limb motor weakness, which may be unilateral or bilateral and involve one or multiple myotomes in a lower motor neurone pattern
L2-S3	Lower limb sensation	Sensory change (numbness, pain, or paraesthesia) in a dermatomal pattern involving one or multiple dermatomes
S2-S4	Bladder control	Defective bladder emptying with urinary retention Loss of awareness of bladder fullness until abdominal pressure occurs (with complete loss of sensation) Loss of or alteration of usual sensation on passing urine
S2-S4	Defecation reflex triggered by rectal distension External anal sphincter control	Constipation Faecal incontinence (inability to control passing stool with awareness or complete loss of awareness of passing stool) Loss of awareness of fullness of rectum / need to pass stool Loss of or alteration of usual sensation on passing stool Inability to pass stool
S4-S5	Peri-anal and internal buttock sensation	Peri-anal and buttock sensory change
	Sexual function	Loss of internal vaginal sensation Loss of ability to get or maintain an erection Loss of ability to ejaculate / orgasm

Box 2: Causes of cauda equina compression

Cause	Features
Prolapsed intervertebral disc	Sudden severe back pain while moving. Pre-existing disc disease
Spinal infections, e.g. discitis, epidural abscess, facet joint infection	Fever, raised inflammatory markers Source of infection – either local (e.g. spinal surgery/instrumentation) or distant (e.g. intravenous drug use)
Tumour – primary or metastatic	Known or previous cancer Systemic features, e.g. involuntary weight loss
Trauma	History of injury
Vascular – e.g. epidural haematoma, dural arteriovenous fistula	Recent surgery

Box 3: Symptoms compatible with CES that would usually indicate emergency MRI[36]

Low back and/or radicular leg pain* within 2 weeks of presentation and any one of:	Urinary symptoms: difficulty initiating micturition or impaired sensation of urinary flow.
	Saddle sensory change: altered peri-anal, perineal, and/or genital sensation (subjectively reported or objectively tested).
	Motor weakness: severe or progressive neurological deficit of both legs, such as major motor weakness of knee extension, ankle eversion, or foot dorsiflexion.
	Bowel dysfunction: loss of sensation of rectal fullness.
	Sexual dysfunction: inability to achieve erection and/or ejaculate or loss of vaginal sensation.

*Note that bilateral radicular leg pain in the absence of CES features may still be a warning symptom that CES is going to occur.

Box 4: “Red flags” for patients in whom CES has been excluded

Feature	Potential conditions
No history of back pain	Neuromyelitis optic spectrum disorder Myelin oligodendrocyte glycoprotein inflammatory myelopathy
Sensory level	Structural cause, e.g. outside the lumbosacral spine Multiple sclerosis
Urinary retention despite adequate analgesia and resolution of constipation for >72 hours	Various neurological causes
Progressive weakness, particularly with loss of reflexes	Spinal dural arteriovenous fistula
Recent genital ulceration	Eisberg syndrome; acute or subacute lumbosacral radiculitis/myelitis associated with herpes simplex virus-2 infection
Progressive perineal pain	Pudendal neuropathy, e.g. after prolonged labour or cycling

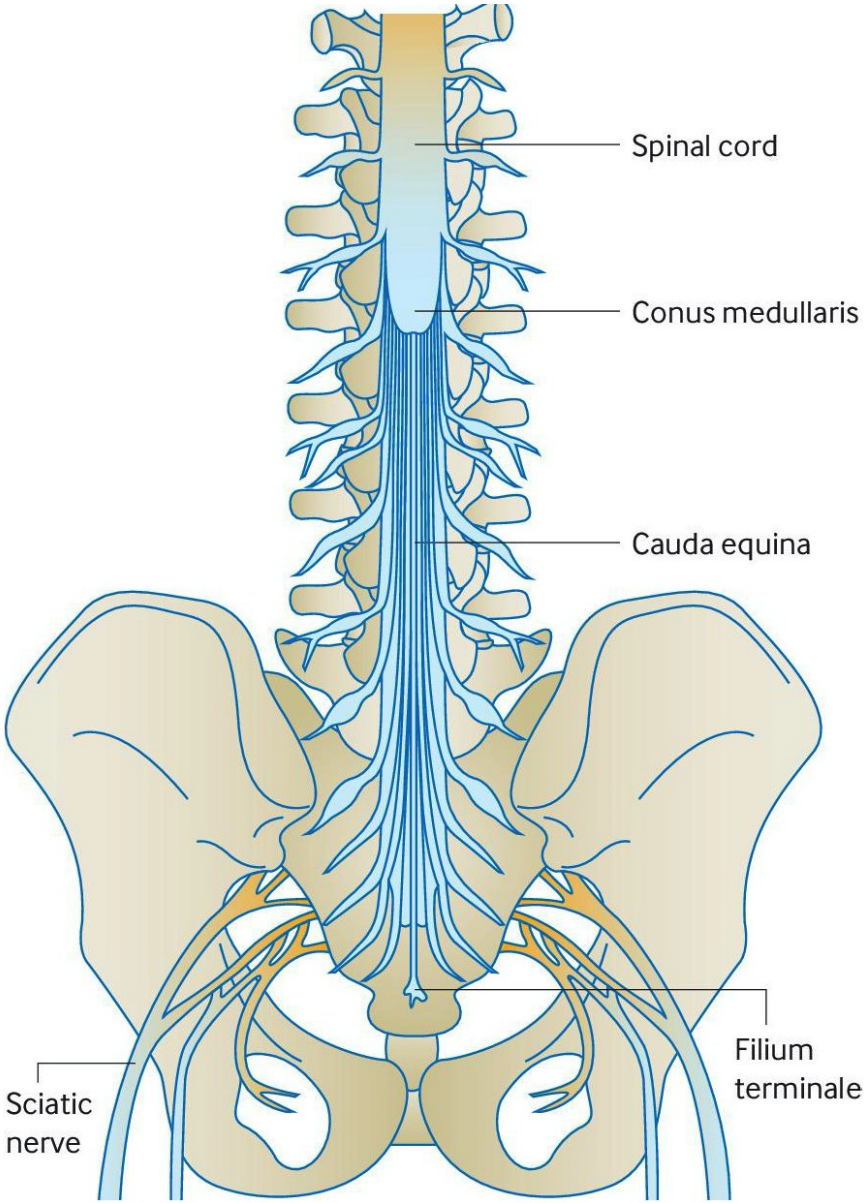
Spinal cord
Conus medullaris
Cauda equina
Filium terminale
Sciatic nerve

Figure 1

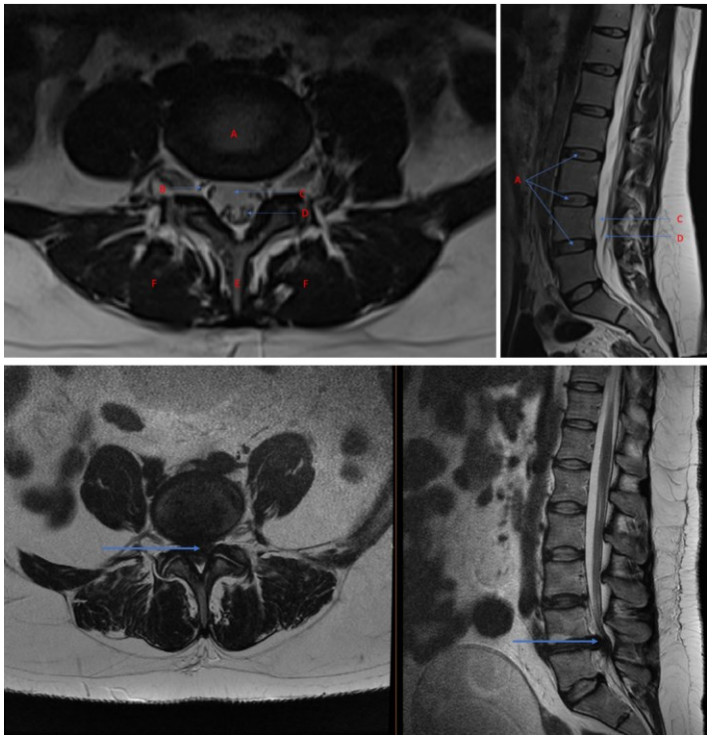

Figure 2