Cauda equina syndrome and severe lumbar-sacral radiculopathy in general practice: Finding the needle in the haystack

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Background
Cauda equina syndrome is a symptom complex of significant clinical and medico-legal concern to both primary care physicians and neurosurgeons. The clinical spectrum of presentation is a cause for significant confusion in the primary care and emergency department setting, which, coupled with the high frequency of litigation relating to these cases, results in physician discomfort in the assessment, investigation and triage of patients with lumbar spinal pathology.

Objective
This paper aims to discuss the clinical spectrum of 'cauda equina syndrome' and relate the signs and symptoms to their underlying anatomical basis, to provide a framework from which primary care physicians and neurosurgeons can triage the urgency of investigation and/or intervention.

Discussion
We recommend that 'cauda equina syndrome' should be better understood as a spectrum of severe radiculopathy relating to acute compression of lumbar and/or sacral nerve roots, which comprises multiple separate indications for investigation and intervention with variable urgency.

AS A GROUP, presentations with low back pain and radiculopathy are one of the most frequent attendances to general practitioners[1] and one of the leading causes of lost quality-adjusted life years worldwide. During any such attendances, practitioners inevitably enquire regarding 'red flags' of the rare cauda equina syndrome (CES; bowel, bladder or sexual dysfunction) and perform a neurological examination. Although such consultations are often straightforward, the presence of a positive response or abnormal examination finding is relatively uncommon and requires greater sophistication to interpret and appropriately triage. Indeed, approximately only 0.08% of back/radicular pain presentations to primary care physicians relate to CES.[2,3] Because of the disproportionate medico-legal burden relating to such cases, there is an appreciably low threshold for referral onwards; however, in large series, <10% of referrals to neurosurgical services for 'cauda equina syndrome' result in intervention, and very few of those represent true 'cauda equina syndrome', with the majority of interventions performed for what has been variably termed 'threatened', 'impending', or 'incomplete' cauda equina syndrome.[4–6] Likewise, diagnostic test accuracy studies for various clinical signs are hindered by the lack of a universally accepted 'true positive', whether that is a 'positive finding on magnetic resonance imaging (MRI)' or urgent/semi-urgent surgical intervention. Indeed, even subspeciality experts have poor consensus on what defines 'cauda equina syndrome'.[6] This results in confusing feedback to the primary care physician.

To avoid such confusing terminology and facilitate triage of these cases, it is simpler to consider a basic anatomical framework regarding the indications for urgent intervention and consider the common mimics of true lower sacral nerve root dysfunction.

Aim

This paper aims to briefly review the anatomical basis of 'cauda equina syndrome' and its spectrum of presentations, with a focus on differentiating the signs and symptoms of those rare cases that require urgent surgical intervention from the more common benign confounders.

Anatomical principles

The key concerns during the evaluation and treatment of lumbosacral spinal pathology both clinically and medico-legally relate to the possibility of motor deficits (weakness) arising from injury to (typically)

L2–S1 nerve roots (Table 1) or autonomic dysfunction relating to the lower sacral nerve roots resulting in incontinence or sexual dysfunction. Unfortunately, although motor function is readily assessed, assessment of lower sacral nerve root function is often more challenging and prone to confounding by other biopsychosocial factors including comorbities such a lower urinary tract obstruction, opiate use, pain-related behaviours such as limited mobility and psychological distress (Table 2). The classical history and examination findings are each reported to have poor sensitivity and specificity for detecting a lesion causing true 'cauda equina' syndrome. For example, in the population with back and/or radicular pain, urinary retention is very common in the setting of acute pain – even without true sacral nerve root injury (sensitivity 25%, specificity 72%).[4,7–11] Likewise, constipation is most commonly related to opiate use in that population, whereas faecal incontinence is likewise a poorly discriminating sign (in one series: sensitivity 19%, specificity 90%). Sexual dysfunction is often multifactorial and not unexpected in the setting of acute pain.[7–10,12]

In distinguishing true lower sacral nerve root dysfunction from these confounders, several anatomical principles are of value (Figure 1):[13,14]

1. nerve roots marginate (move to the lateral border of the spinal canal) before they exit at each level (ie the lower sacral nerve roots are centrally placed within the cauda equina)
2. lower sacral nerve roots are present bilaterally and there are multiple on each side with some clinical redundancy
3. acute neural compression is extremely rare below the level of the L5/S1 disc space (eg acute disc protrusion or pathological fracture generally occur above this level).

The clinical corollary of these principles is that, in order to have clinical evidence of complete lower sacral nerve root dysfunction, a very large and centrally placed compressive pathology (eg intervertebral disc protrusion) would be required, and that pathology is at or above the level of the L5/S1 disc space. Such a pathology would very frequently, therefore, cause compression of nerve roots bilaterally resulting in bilateral radicular symptoms that extend below the knee. Bilateral symptoms have been found in multiple case series to have the greatest discriminating power out of individual symptoms for detecting significant pathology. For example, in a multivariate analysis of one large series, the only symptoms that remained independently predictive of neurosurgical intervention were bilateral leg pain (odds ratio [OR] 2.2), bilateral weakness (OR 2.1), bilaterally absent ankle jerks (OR 2.9) and dermatomal sensory loss (OR 1.8).[7–10,12]

Second, clinical assessment of lower sacral nerve roots will often be normal unless a large number of the roots are involved. Although assessment of the reflex function of those roots directly can be challenging, the immediately adjacent S1

Table 1. Nerve roots and associated myotome and reflexes

Nerve root	Distribution of weakness (myotome)	Reflex
L2	Hip flexion	
L3	Knee extension	
L4	Ankle dorsiflexion	Knee jerk
L5	Great toe dorsiflexion, inversion/eversion	Medial hamstring jerk
S1	Ankle plantarflexion	Ankle jerk
S2–5 'lower sacral nerve roots'	Continence	Bulbocavernosus anal wink

Table 2. Lower sacral nerve root symptoms and biopsychosocial mimics

Symptom	Mimics/confounders	Notes
Urinary retention	Pain	Painless urinary retention is highly concerning
Urinary hesitancy	Lower urinary tract obstruction	
Constipation	Opiate use, limited mobilisation, intolerance of sitting position	
Erectile dysfunction	Pain, comorbid psychological, vascular disorders	
Saddle paraesthesia	Elsberg syndrome (herpes simplex sacral radiculitis)[13]	Consider examination and investigation for herpes reactivation in 'MRI negative cauda equina'
Incontinence	Overflow faecal incontinence, urological disorder	Consider chronicity

MRI, magnetic resonance imaging.

nerve root and the corresponding ankle jerk reflex are readily assessed. In one series, bilaterally absent ankle jerk reflexes was highly specific for cauda equina syndrome (specificity of 95%).[8] Urinary retention is a cardinal sign of significant lower sacral nerve root dysfunction, especially in the absence of an alternative explanation such as severe pain. Even in the setting of significant pain, however, this is generally considered an indication for emergent investigation. In contrast, the assessment of anal tone is notoriously unreliable, with sensitivity and specificity each approximately 50%.[15] In the experience of the authors, assessment of anal tone is non-contributory to the triaging and management of these patients. In view of the invasive nature of such examination, the authors of this paper agree with international consensus recommending against its performance in the primary care setting.[16] As an alternative, conventional assessment of pinprick and light touch sensory modalities in the 'saddle region' (best described of as the parts of the genital and perineal region in contact with a bicycle seat/saddle). There is some limited evidence that this might have discriminatory value, is less invasive and allows assessment of the 'anal wink' reflex (the reflexive contraction of the anus in response to painful stimulation of the adjacent skin).[7,15,17–19] For example, in a large series of >1000 patients assessed for CES, altered perianal sensation was associated with a positive finding on MRI on univariate analysis (OR 2.1); however, this association did not quite maintain statistical significance on multivariate analysis (P=0.057). In the same series, the clinical assessment of anal tone was not significantly associated with a positive finding on MRI.[15]

Imaging

Because of the high degree of clinical and medico-legal aversity to type two error (missed diagnosis), MRI is generally required for definitive exclusion of significant cauda equina compression. Although one recent study with high-quality contemporary computed tomography (CT) scan confirmed a 98% sensitivity and 86% specificity for excluding significant central canal compression,[20] guidelines and expert consensus recommend that MRI is preferred in the setting of suspected cauda equina compression.[21,22] Indeed, acquisition of a CT scan in the community prior to referral is not required and might even delay the care of patients with concern for CES.

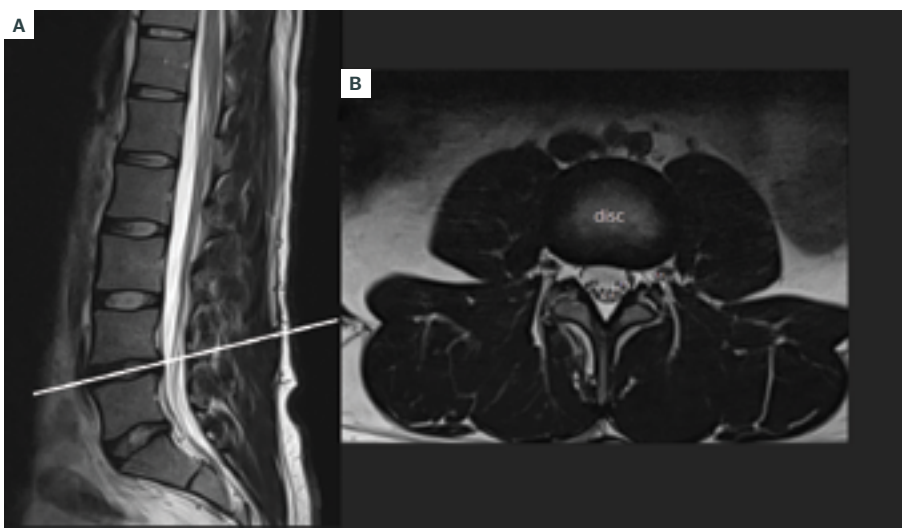

Figure 1. T2 weighted magnetic resonance imaging scan of the lumbar-sacral spine. (**A**) Mid-saggital section. (**B**) Axial section at level of the L4/5 disc space with intrathecal location of nerve roots labelled.

Correlating imaging and clinical findings

There are several well-recognised patterns of lumbar compressive pathology – the most common being lateral recess stenosis, foraminal stenosis, central stenosis and extraforaminal stenosis (Figure 2). Each present with a distinct clinical syndrome, and the goal of the practitioner is to correlate the imaging and clinical findings.

Lateral recess stenosis is the most common pattern because of it being the most frequent location for a disc protrusion, as directed by an intact posterior longitudinal ligament to the 'subarticular recess', which transmits the marginating nerve root numbered for the vertebrae below. Patients will frequently present with unilateral radiculopathy relating to that marginating nerve root.[23] Although such patients might experience significant weakness if the compression of that nerve root is severe, the presence of any sacral nerve root dysfunction is likely to be mild or incomplete, which has led to various terminology in the literature (eg threatened or incomplete cauda equina) relating to concerns for possible progression of compression and symptoms. The authors propose that it is simplest to avoid such terms and label such cases as severe sacral radiculopathy and manage with similar urgency to 'complete cauda equina'.

Foraminal stenosis and extraforaminal stenosis relate to compression of the exiting nerve root in the exit foramina or irritation of that root just beyond the foramen. These regions of compression would cause symptomatic involvement of the nerve root numbered for the vertebrae above. As these are quite distant from the more midline lower sacral nerve roots, symptoms such as urinary retention are likely to have an alternative explanation (eg severe pain).[24] Abnormal movement or positioning of one vertebrae upon the other (spondylolisthesis) might narrow the exit foramina bilaterally and provide an explanation for bilateral symptoms; however, this is generally not an acute pathology.[25]

Central canal stenosis can give rise to two separate syndromes depending on the speed of onset of the compression. In patients with acute compression (eg a large acute disc protrusion/extrusion), symptoms might be severe including significant involvement

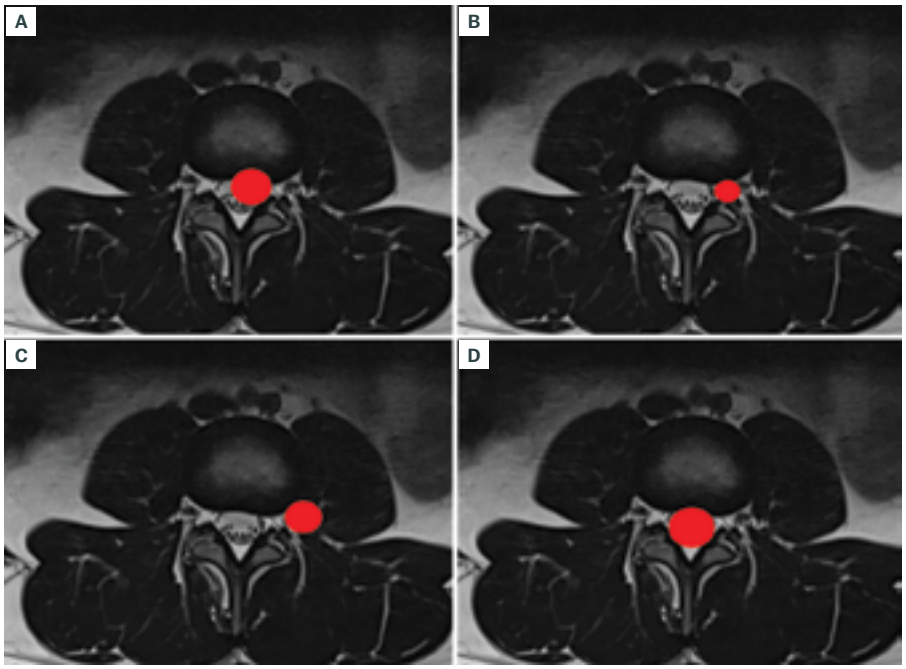

Figure 2. Axial sections of the lumbar spine with red circles indicating location of pathology (eg disc protrusion) causing: (**A**) lateral recess stenosis; (**B**) foraminal stenosis; (**C**) extraforaminal nerve root compression; and (**D**) central canal stenosis.

Box 1. Clinical indications for referral and implications for triage

Recommended indications for emergency referral

- Bilateral pain or altered sensation extending below the knee (including saddle and genital region)
- Bilateral weakness
- Bilaterally absent ankle jerks in the setting of radicular pain
- Unilateral weakness with power 3/5 or worse
- Any concern for urinary retention
- Any other 'red flags' of non-cauda equina significant spinal pathology (eg upper motor neuron signs, unexplained weight loss, night sweats, fevers, intravenous drug use, history of metastatic malignancy)

Recommended indications for urgent referral (within 24 hours/rapid assessment clinic)

- Bilateral pain or altered sensation extending below the knee, but without saddle/genital involvement or other signs as detailed above
- Single nerve root territory weakness with power 4/5 or better
- Severe radicular pain refractory to oral analgesia

Findings that do not require urgent assessment when isolated findings

- Axial lower back pain without radicular pain or altered sensation
- Lateral thigh pain alone without extension below the knees
- Constipation in the setting of opiate use
- Isolated finding of 'reduced anal tone'

of lower sacral nerve roots and likely the marginating nerve root or roots also. This is the classical lesion associated with CES and represents a neurosurgical emergency.[21,23]

Central canal stenosis might progress slowly, typically in the older population as a result of degenerative changes such as facet joint and ligamentum flavum hypertrophy. In such patients, despite radiologically severe compression, symptoms might be relatively modest, and lower sacral nerve root dysfunction is typically absent. The classical presentation of these patients is with neurogenic claudication (ie leg pain worsening with walking, typically relieved by rest or supported forwards posture – like using a shopping trolley) and they have the lowest clinical urgency.[26]

Making a positive anatomical diagnosis can help to stratify the patient's clinical urgency, and distills into the recommendations listed in Box 1.

Conclusion

There is significant confusion regarding the assessment of patients for CES during primary care consultations. Instead of considering CES as a separate entity with a checklist of symptoms, the authors recommend conceptualising a spectrum of severe radiculopathy patterns involving the lumbar and/or sacral nerve roots. The primary goal is to identify any signs of injury to those nerve roots, which have resulted in loss of motor, continence or sexual function, and those with bilateral symptoms at risk for progression – any of which is sufficient to warrant urgent investigation with MRI. Applying simple anatomical principles can guide the focused assessment of these patients and determine the urgency of further investigation without the requirement for invasive examinations.

Key points

- Cauda equina syndrome is best understood as a spectrum of severe sacral radiculopathy.
- An anatomical consideration of the lower sacral nerve roots should guide the clinical assessment.
- Bilateral radiculopathy and absent ankle jerks are highly concerning features.

- Clinical assessment of anal tone is not recommended in the primary care setting.
- Urinary retention in the setting of significant pain is most often due to the pain itself.
- Document examination findings of reflexes, pin prick and anal light touch over the saddle area if considering cauda equina syndrome.

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