‘Scan-negative’ cauda equina syndrome: what to do when there is no neurosurgical cause

Ingrid Hoeritzauer, Biba Stanton, Alan Carson, Jon Stone

ABSTRACT
Suspected cauda equina syndrome is a common presentation in emergency departments, but most patients (≥70%) have no cauda equina compression on imaging. As neurologists become more involved with ‘front door’ neurology, referral rates of patients with these symptoms are increasing. A small proportion of patients without structural pathology have other neurological causes: we discuss the differential diagnosis and how to recognise these. New data on the clinical features of patients with ‘scan-negative’ cauda equina syndrome suggest that the symptoms are usually triggered by acute pain (with or without root impingement) causing changes in brain–bladder feedback in vulnerable individuals, exacerbated by medication and anxiety, and commonly presenting with features of functional neurological disorder.

INTRODUCTION
When a neurosurgeon asks a neurologist to see a patient with cauda equina symptoms but a negative scan, how often is there another neurological disease? And if there is no other recognised pathophysiological disease, can we still understand their symptoms and support them to recover?

A small number of patients without clear structural abnormalities have other neurological pathology causing cauda equina syndrome and we discuss this differential below. However, most patients have no recognised pathophysiological cause.

In the last few years, new research in this area has informed our knowledge of these ‘scan-negative’ cauda equina syndrome patients with a focus on what may be wrong, rather than just the lack of structural abnormalities or neurological rarities. The evidence suggests that in most scan-negative patients, the problem relates to the acute experience of pain, exacerbated by medication such as opiates, and this commonly overlaps with a functional disorder—the disorder is much more than just of the cauda equina.

This article aims to equip neurologists with a differential diagnosis of cauda equina syndrome, some understanding of newer mechanistic hypothesis of ‘scan-negative’ cauda equina syndrome, and our thoughts on explanation and treatment.

NEUROLOGICAL DISEASE MASQUERADING AS A SURGICAL PROBLEM?
Cauda equina syndrome is a rare but potentially devastating condition caused by compression of the cauda equina nerve roots, most often by a prolapsed intervertebral disc. The estimated incidence in working age adults is 7 per 100 000.1 There are no internationally agreed clinical or radiological definitions of cauda equina syndrome, so we use the definitions from a systematic review of its descriptions. Suspected cauda equina syndrome is defined as having one or more of the following:

1. Bladder and/or bowel dysfunction,
2. reduced sensation in the saddle area or
3. sexual dysfunction, with possible neurologic deficit in the lower limb (motor/sensory loss, reflex change).2 The presentation is primarily one of sphincter disturbance, but it is often accompanied by back pain, sciatica and sensorimotor symptoms in the legs. However, about two-thirds of patients presenting with suspected clinical cauda equina syndrome have no identifiable structural cause for their symptoms.3 4

From recent research in two different centres, only a few (2%-7%) patients end up having an alternative neurological problem causing their symptoms,3 4 and these are typically identified during the initial admission. The numbers of ‘late’ presentations of alternative neurological causes is much smaller. Figure 1

Correspondence to
Dr Ingrid Hoeritzauer,
Department of Clinical Neurosciences, Royal Infirmary of Edinburgh, Edinburgh, UK;
Ingrid.hoeritzauer@ed.ac.uk
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Acute "Cauda Equina" presentations.
Identifiable surgical and medical causes (blue) only account for one third of presentations.

Table 1 Neurological differential diagnosis of cauda equina syndrome

| Category          | Conditions                                                                 |
|-------------------|-----------------------------------------------------------------------------|
| Inflammatory      | Transverse myelitis, for example, due to multiple sclerosis                 |
|                   | Inflammatory conus lesions, for example, due to neuromyelitis optica spectrum disorder, particularly associated with MOG antibody |
|                   | Acute and chronic inflammatory demyelinating polyradiculoneuropathy        |
|                   | Sarcoidosis                                                                |
|                   | Vasculitis                                                                 |
|                   | Meningitis retention syndrome                                              |
| Vascular          | Spinal dural arteriovenous fistula                                         |
|                   | Spinal stroke/ischaemia                                                    |
|                   | Epi/intradural haematoma                                                   |
| Infective         | Elsberg’s syndrome (herpes simplex virus type 2 lumbosacral radiculitis/myelitis) |
|                   | Varicella zoster                                                           |
|                   | Cytomegalovirus                                                           |
|                   | HIV                                                                        |
|                   | Tuberculosis                                                               |
|                   | Epidural abscess                                                          |
| Neoplastic        | Neoplastic or radiation induced                                            |
| Neurodegenerative | Multiple system atrophy                                                   |
|                   | Parkinson’s disease                                                       |
| Medications       | Opiates                                                                    |
|                   | Benzodiazepines                                                           |
|                   | SSRIs                                                                      |
|                   | Anticholinergics (eg, tricyclics)                                         |
|                   | Pregabalin                                                                 |
| Structural        | Pudendal neuropathy (eg, after prolonged cycling, pelvic injury, childbirth) |

WHAT SUGGESTS A NEUROLOGICAL CAUSE OF CAUDA EQUINA SYNDROME?

No back pain
If severe back pain is not a predominant symptom at the onset, then proceed with caution. Neuromyelitis optica spectrum disorder can present with ‘cauda equina’ symptoms and myelin oligodendrocyte glycoprotein inflammatory myelopathy is particularly associated with bladder symptoms at onset.6 However, these conditions are usually have other neurological signs (eg, optic nerve involvement, pyramidal signs) and a longitudinally extensive spinal cord signal abnormality that would be picked up on the initial T2 sagittal MR imaging of the cervical and thoracic spine.

Sensory level
Is there a spinal sensory level? This would also push us to look very carefully for an underlying structural cause for their symptoms. For instance, an inflammatory cord lesion in multiple sclerosis might present with prominent bladder symptoms (although rarely with bladder symptoms alone). Here, finding a sensory level could provide evidence of localisation to the spinal cord (even if a small lesion were not picked up on imaging).

Box 1 ‘Red flags’ suggesting an identifiable neurological cause in a scan-negative cauda equina presentation

- No history of back pain.
- A sensory level.
- Urinary retention despite adequate analgesia and resolution of constipation for more than 72 hours.
- Progressive weakness, particularly with loss of reflexes.
- Recent genital ulceration suggesting herpes simplex virus-2 infection.
- Progressive perineal pain.
Urinary retention
Does the patient have urinary retention of >100 mL despite adequate analgesia and resolution of constipation for more than 72 hours? If yes, then we suggest considering neurological causes of cauda equina syndrome.

Progressive weakness with reflex loss
Has there been gradually progressive weakness with loss of reflexes without prominent pain? Patients with a spinal arteriovenous fistula can take up to 3 years to receive a diagnosis as they often present with lower motor neuron symptoms before gradually developing upper motor neuron signs. Spinal dural arteriovenous fistula most commonly affects men at a mean age of 55–60 years and most occur in the thoracolumbar region, often much higher than the anatomical localisation of the problem.

Recent genital ulceration
Were there flu-like symptoms before onset? Look for a history of genital ulcers and prodromal symptoms such as myalgia or headache in the presence of persistent urinary retention. Elsberg syndrome refers to acute or subacute lumbosacral radiculitis/myelitis associated with herpes simplex virus-2 infection (genital herpes) that may result in urinary retention.

Progressive perineal pain
Is the pain more progressive and does it localise better to the perineum than to the back or legs? If so, especially if it is worse on sitting, consider pudendal neuropathy. This can occur in women after prolonged labour or in keen cyclists (as many neurologists are) who initially note perineal neuralgia that can be followed by numbness and even bladder, bowel or sexual dysfunction if the compression is bilateral.

In any patient with red flags for a neurological cause, we suggest a lumbar puncture looking for evidence of infective or inflammatory lumbosacral plexopathy, malignancy or spinal inflammation. CSF analysis may include varicella zoster virus, cytomegalovirus and herpes simplex virus, cytology and oligoclonal bands.

WHO GETS SCAN-NEGATIVE CAUDA EQUINA SYNDROME?
For the rest of this article, we focus on those individuals who have neither a surgical nor a clear alternative neurological cause. We use the term ‘scan-negative’ cauda equina syndrome to indicate that this an area of ongoing investigation based on four studies of 469 patients. We also acknowledge that the mechanisms of these symptoms occur in a distribution wider than the cauda equina alone.

‘Scan-negative’ cauda equina syndrome is relatively common: a recent study involving 28 of the 30 UK neurosurgery centres found 4441 referrals for possible cauda equina syndrome over a 6-month period. A systematic review of cauda equina syndrome incidence and three scan-negative cauda equina syndrome studies found 69%–84% of patients referred as possible cauda equina syndrome had negative or non-explanatory scans, with no other neurological cause found. Extrapolating across the UK, this suggests that around 6000–7000 people present to hospitals with these symptoms every year. Current data suggest that it is not benign. Representation is more common in patients with scan-negative than scan-positive cauda equina syndrome (11% vs 4%) and only 10% of patients describe resolution of their symptoms at 3-year follow-up.

Patients with ‘scan-negative’ cauda equina syndrome can be divided roughly evenly into those with nerve root compression or impingement, and those with normal (or ‘normal for age’) scans. These two groups share several clinical features that are more frequent in the patients with normal (or ‘normal for age’) scans. They are usually young (average age 40s) and predominantly female (70%). Most present with severe back pain (70%–97%) and have chronic back pain (41%–45%). Patients may have panic symptoms, or a panic attack, when their back pain occurs or in the emergency department (57%–70%). Patients may have preceding bladder symptoms: stress incontinence is an embarrassing but common problem, affecting about one in five women over 40, and is more likely in people with chronic back pain. Patients often take many medications (approximately one-third taking opioids and/or gabapentinoids) and are more likely to have prior chronic pain, functional and psychiatric disorders. Those patients with nerve root compression or impingement usually have more prominent leg than back pain.

Most strikingly, this scan-negative group commonly have positive evidence of functional neurological symptoms, including weakness, gait and sensory symptoms (34%–82%), which we discuss further below.

MAKING SENSE OF ACUTE SYMPTOMS
While taking a step-by-step history, it’s useful to ask the patient to elaborate on what they were thinking and feeling at each point. Their description can help to assemble a clinical narrative to explain their symptoms. Box 2 gives a typical patient story, illustrating how terrifying it is for people experiencing these symptoms. Table 2 lists specific features to look out for in the history.

Asking the patient to describe a typical day in detail can give a feel for disability and psychiatric comorbidity. Are they still finding things to do that they enjoy? Can they get out of bed or do they spend a lot of time worried about pain recurring? Can they go out alone or do they worry too much about falling or something bad happening? If they have chronic pain, are they skipping meals regularly?
It is essential to evaluate a patient’s illness perceptions and expectations. What do they think has been causing their back pain, and this acute presentation? Do friends or family take a different view? What has been getting in the way of improving previously?

It is worth investing the time in doing this: a positive explanation of the mechanism of their symptoms, tailored to their own history, aims to give patients greater confidence in the diagnosis.

**Box 2 A patient’s account of their experience**

When the pain started it was so severe that I was really scared. I thought ‘what if I can never walk or hold my children again?’ My heart started racing and I felt as though my legs didn’t really belong to me. I was on my own in the bathroom, so I crawled into the bedroom. I honestly thought I might die. When my partner found me he was terrified too. Then I was incontinent because I couldn’t manage to get up to go to the toilet. The ambulance came really quickly. They were really good, but it was hard to get me down the stairs. Once we got to hospital we were rushed straight through to resus. When the doctor examined me I realised I couldn’t feel anything normally around my bottom and I couldn’t move my legs at all. The nurses said I had 200 mL of urine in my bladder, so I had a catheter put in. They said I needed a scan straight away. I was in so much pain during the scan, and it seemed to take a long time. Then after the scan they just told me everything was normal and there was nothing to worry about. But I was still lying there in pain and I couldn’t move my legs. So I knew there must be something wrong.

**EXAMINATION**

Our recent data suggest that some clinical signs may have predictive value in discriminating between scan-positive and scan-negative cauda equina patients, although none mean that an urgent MRI should be avoided (table 3). In patients with leg weakness, it is useful to look for signs of functional limb weakness like Hoover’s sign or the hip abductor sign. In our study, there was a positive Hoover’s sign (weakness of hip extension that normalises with contralateral hip flexion) in 16% of scan positive patients, 42% of those with only nerve root compromise, and 82% of patients with completely scan-negative cauda equina syndrome. For patients with bilateral leg weakness, neurologists can use a modified version of Hoover’s sign. Ask the patient to sit forward and then push back with their trunk against resistance: this is another way to normalise weakness of voluntary hip extension. Functional sensory loss, such as sharply demarcated loss of sensation in the whole leg with cut-off at the top of leg, often with a patient reporting that the leg feels like it’s ‘not there’, showed a similar ‘dose–response’ relationship in these three groups (3% scan positive group, 25% nerve root compression group, 49% in patients with normal scans) (table 3).

Perhaps just as relevant are the lessons we are learning about how unhelpful some potentially invasive parts of the examination are. Anal tone and saddle numbness appear, in real-world clinical settings, to have no diagnostic value in the assessment of cauda equina symptoms. There is now a valid question as to whether there should be routinely carried out (table 3).

**Table 2 Key points in the history of scan-negative cauda equina syndrome**

| Pain | ► Back or leg pain?  
|► Leg pain stopping at the knees or down to the ankles? Pain that stops at the knees suggests muscle spasm with referred pain and tightening rather than a radicular or lumbosacral problem  
|► Time course: was it acute on chronic, chronic with gradual worsening or acute? |
| Bladder and bowels | ► Any previous bladder or bowel difficulties? Many people experience stress incontinence, or difficulties in urinating or defecating in a toilet that is not their home toilet  
|► Usual frequency of bowel opening |
| Sensory/motor symptoms | ► Did the weakness occur in emergency department or at home?  
|► Did the patient notice loss of perianal sensation (usually described as loss of sensation on wiping) or was it only picked up in emergency department? (Sometimes being examined—especially in a stressful situation—can trigger symptoms through changes in attention) |
| Medication | ► Be open and non-judgemental. Patients with worsening pain often have ramped up their medication in recent days, which then worsens their bladder and bowel symptoms. But people are often afraid to admit they have taken more medication than they should. You might say ‘Many people end up taking more than is recommended. Has the pain been so bad that you needed more painkillers than normal?’ |
| Panic symptoms | ► If the pain or leg symptoms were acute it is useful to ask specifically about panic symptoms: palpitations, sweating, trembling or shaking, shortness of breath, feeling of choking, chest pain or discomfort, nausea, dizziness, depersonalisation or derealisation, fear of losing control or ‘going crazy’, fear of dying, paraesthesia in fingers or around their mouth, chills or hot flushes.  
|► A panic attack is a discrete period in which ≥ four of the symptoms above reach a peak within 10 min |
| Depersonalisation or derealisation | ► If a patient describes ‘dizziness’, ask whether this felt like spinning (vertigo), light-headedness (presyncope) or a feeling of being ‘there but not there’ (dissociation)  
|► If they have leg symptoms, do they have an odd feeling of their legs not belonging to them or a sense of not being there? |
| Specific thoughts about symptoms | ► Did they think they might be paralysed or incontinent forever?  
|► Have they had some other experience of this in themselves or others that made this even more scary? |
As stated earlier, we suggest introducing, as standard for people with cauda equina presentations, a T2 sagittal of the cervical and thoracic cord during the initial MR imaging, as 70%–80% of scans will not show cauda equina compression. The entire length of the sacral spine should also be included in all routine cauda equina imaging. This adds a minimal time to the scanning, and often avoids the need for a further scan. Brain imaging, contrast imaging, lumbar puncture, neurophysiology and other investigations may all be needed depending on the assessment, but none of these is mandatory.

Knowledge of what disc and other degenerative changes are normal for age are essential for neurological practice, but especially in this area (table 4). A systematic review of asymptomatic people has shown that more than 30% of patients aged over 20 have minor disc bulges on imaging. In asymptomatic patients in their 40s, almost 70% have degenerative changes in the spine, half have minor disc bulges and one fifth have evidence of nerve root irritation. As a general rule, the per cent likelihood of a disc bulge is the patient’s age plus 10. It may be helpful for patients to understand the role that scan changes may have played in triggering their symptoms, but also that often such changes are irrelevant, or that their symptoms can improve even if the scan changes remain.

**A PROPOSAL FOR THE MECHANISM OF SCAN-NEGATIVE CAUDA EQUINA SYNDROME**

These emerging data have led us to suggest a model in which scan-negative cauda equina syndrome arises as an end pathway of acute pain, sometimes with partly structural findings and/or vulnerability to functional disorders (figure 2). We hypothesise that severe pain and panic (especially in those with pre-existing bladder symptoms) cause changes in the higher brain centres monitoring safety to void and therefore inhibiting bladder emptying. This is often aggravated by medication (including opioids and gabapentinoids), which act centrally and peripherally to create or exacerbate bladder dysfunction. In people who are vulnerable to functional disorders, these factors, particularly dissociation as part of a panic attack, can also cause functional limb weakness and sensory disturbance.

**MANAGEMENT**

Figure 3 shows a suggested care pathway for patients with scan-negative cauda equina syndrome that could be adapted to different settings. There is no evidence to support a specific treatment approach,
so these suggestions are based on clinical experience and informed by the proposed model of symptom mechanism.

**ACUTE MANAGEMENT**

**Explanation**

Patients expect and deserve a clear explanation for what has happened to them. Just saying ‘the scan is normal so there’s nothing to worry about’ is not enough to provide the patient the confidence needed to engage in treatment. The best terminology to use may depend on the patient: you might just use the label ‘acute back pain’ where this is clearly the dominant feature and it looks as if symptoms are resolving rapidly, or you might talk about ‘functional leg weakness’ in patients with predominant motor symptoms and a positive Hoover’s sign. For some patients, it can help to use the term ‘scan-negative cauda equina syndrome’. We have created a factsheet using this heading, available on neurosymptoms.org. More important than the exact label is providing an individualised explanation of the mechanism of the symptoms necessitating the scan. Based on the model described above, neurologists can explain how acute pain (especially on a background of pre-existing bladder and bowel symptoms) can trigger changes in brain–bladder feedback and brain processing. Addressing the patient’s fears about walking, especially in relation to their understanding of changes on the scan, is often important. Consider providing printed written information (and not just a weblink) (figure 4). In an inpatient setting a printed document is more likely to be shared with family, friends and other health professionals to create a shared understanding of what is wrong.

**Pain control**

Pain control is the first priority for acute management. Using strong analgesia in the short term to promote early mobilisation has to be balanced against the risks...
of adverse side effects that could worsen some of the symptoms such as constipation and urinary voiding dysfunction. The best choice depends on the individual patient: paracetamol and non-steroidal anti-inflammatory drugs are the least likely to aggravate sphincter symptoms. Gabapentinoids may be useful for radicular pain but are best avoided if possible. Benzodiazepines are often considered for muscle spasm but are especially problematic in worsening symptoms of dissociation in patients with functional leg paralysis. They are often not reviewed carefully and may be harmfully continued after discharge. Physiotherapy input to support early mobilisation is invaluable. Walking aids should be avoided wherever possible.

Even if doctors avoid prescribing opiates acutely, patients may present with a long history of opiate use. It is surprising how many patients have partially realised that their opiates do not work, and have unpleasant side effects, but are lost in the need for pain control. Many patients respond differently to discussion in which they are facilitated to arrive at the question of how to tackle the problem themselves. Techniques of motivational interviewing suggest taking a step back to find out what is preventing them from changing. Often it is fear of further pain with psychological and physical withdrawal. It can help to ask questions such as ‘are the medications stopping the pain?’, ‘what are benefits and the downsides of continuing to take opiates?’, ‘how is your life now, compared with before you started opiates?’, ‘if you imagine taking opiates for the next 5 years, what would that be like?’; ‘are there any benefits to stopping the opiates?’.

Consider discussing opiate-induced hyperalgesia (worsening pain due to opiates) and that sometimes physiological dependence may be causing their pain to flare if they miss or delay opiate medication. We often tell people that their pain is likely to flare up but then settle to how it currently is if they come off opiates but they will not have any of the medication side effects. Although a modest claim it is often enough to spur patients at least to consider reducing or stopping their medications. Neurologists should reassure patients that any opiate reduction will be done slowly, and ideally at a pace they choose. It is worth seeking help from their general practitioner on discharge.

**Bowel and bladder management**

Bowel management is crucial, particularly because constipation will aggravate urinary retention. In addition to a laxative, patients may need to be prescribed suppositories (to be used if no bowel movement after 24 hours) and enemas (to be used if no bowel movement after 48 hours, or sooner if there is faecal impaction on rectal examination).

If a catheter has been inserted, we suggest changing to a ‘flip–flow’ device as soon as possible, and then try removing the catheter as soon as the patient has opened their bowels and mobilised. If there is a delay in being able to remove the catheter then intermittent self-catherisation should be considered as this permits an assessment of natural voiding as the patient recovers.

**Physiotherapy**

Most physiotherapists are used to treating patients with mechanical back pain. Recent major papers on back pain treatment have advocated physiotherapy and education to improve outcomes. There is also positive evidence emerging about the role of physiotherapy in improving symptoms in patients with functional neurological disorders. In a recent randomised trial the outcome of patients with functional motor symptoms at 6 months improved in 72% of patients with specialist physiotherapy input.

In our experience patients derive most benefit from seeing a physiotherapist after having had an explanation of their symptoms. Physiotherapists may find it helpful to review consensus physiotherapy recommendations for functional motor disorders. Kinesiophobia (fear of movement) is often particularly prominent in this patient group. Our experience is that it can be particularly helpful to equip physiotherapists with the basic principles of cognitive–behavioural therapy so that they can have some tools and a ‘language’ for targeting this while getting the patient up and moving.

**Psychiatric treatment**

Psychiatric disorders do seem to be more prominent in this group of patients than functional disorders in general. In our study, 90% of the scan-negative group had a current psychiatric disorder, usually anxiety, panic, post-traumatic stress disorder, obsessive symptoms or depression. These features are often pertinent to the acute presentation and need a combination of pharmacological and psychological management. In addition, a subgroup of patients has significant suicidal ideation and risk assessment is important. It is essential to have a close working relationship with neuropsychiatry or liaison psychiatry colleagues.

**LONGER-TERM MANAGEMENT**

The available follow-up studies of patients with scan-negative cauda equina syndrome are small but suggest high rates of persistent symptoms. At present, we have no treatment intervention data to know if those outcomes can be moderated.

Appropriate follow-up depends on the individual patient. Self-management advice alone may be appropriate for some, whereas others may benefit from community rehabilitation teams or ongoing advice.
from a continence team. In the approximately 40% of patients with chronic pain, referral to a multidisciplinary pain service can be helpful. Sometimes an understandable explanation of why their symptoms have occurred will encourage patients who have been unable to engage fully with the pain service to do so. Neurologists might discuss referral to psychological therapy or psychiatry for problems with mood disturbance that might impact on improvement (figure 5). At present neurologists follow-up only a few of these patients. They may be best served by the multidisciplinary approach and expertise in functional disorders present in a neurology clinic at least after discharge to assess whether they have understood the explanation.

Although analgesia may be essential in the acute setting, it is important to explain the limitations of pharmacological approaches to chronic pain. There is only very limited evidence for benefits from opioids in non-cancer pain after 12 weeks of treatment. At 2 years, opioid use is not associated with any improvement in functional outcome or quality of life. In chronic low back pain, opioids are associated with higher rates of harm and lower benefit than non-opioid analgesia.

CONCLUSION

‘Scan-negative’ cauda equina syndrome is common. Neurologists should exclude alternative neurological causes by targeted clinical assessment and investigations. We propose that in most patients, the symptoms are triggered by acute pain (with or without root impingement), causing changes in brain–bladder feedback in vulnerable individuals, exacerbated by medication and anxiety, and commonly presenting with features of functional neurological disorder. Detailed history taking can help to develop an individualised formulation of the mechanism of symptoms. Explaining why and how a patient’s symptoms have occurred can be a rewarding therapeutic experience and can lay the foundations for positive patient engagement in rehabilitation to improve their pain, weakness, bladder and bowel symptoms.

Key points

- In suspected cauda equina syndrome, only 20%–30% of patients have a surgical or medical identifiable cause.
- Around half of people with ‘scan-negative’ cauda equina syndrome have nerve root pathology on imaging, but half have normal-for-age scans.
- ‘Scan-negative’ cauda equina syndrome appears to be triggered by a mixture of pain, anxiety/panic, medication side effects and features of functional neurological disorder.
- Management is helped by formulating the patient’s experience with their physical and psychiatric comorbidities and providing early education and rehabilitation.

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