

Cauda equina syndrome with normal MR imaging

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Abstract The aim of this study was to compare the clinical characteristics of patients with and without abnormal MR imaging admitted to a neurosurgical unit with suspected cauda equina syndrome using a retrospective study of consecutive admissions to a regional neurosurgical unit over a 10-month period. Clinical details were obtained from the case notes. A lumbar spine MR scan to investigate possible cauda equina syndrome was performed in 66 patients. There were no significant differences between those with abnormal imaging ($n = 34$, 52%) and those with a normal scan ($n = 32$, 48%) in respect of sex, clinical history or features recorded on examination. Those with normal imaging had a high frequency of weakness ($n = 18$, 59%), saddle numbness ($n = 17$, 57%), leg numbness ($n = 24$, 80%), urinary incontinence ($n = 13$, 54%) and urinary retention ($n = 9$, 53%). A large number of patients present to neurosurgical units with symptoms suggestive of cauda equina syndrome without any radiological evidence of structural pathology. While some may have had an alternative organic cause, we

propose that these symptoms may have a “functional” origin in many patients.

Keywords Cauda equina syndrome · Magnetic resonance imaging · Functional symptoms · Conversion disorder

Introduction

Cauda equina syndrome (CES) includes paraparesis, sciatica, low back pain, saddle numbness and urinary retention among its clinical features [10]. A common cause is structural compression of the thecal sac [11]. Although clinical suspicion of CES in the community often triggers the urgent transfer of a patient to a neurosurgical unit for MR scanning, the small available literature suggests that a high proportion of patients have no obvious structural abnormality to explain the clinical findings [2]. We investigated whether any simple clinical characteristics would distinguish these patients from those with structural pathology.

Methods

We retrospectively identified potential subjects referred from primary or secondary care from (1) the neurosurgical Registrars’ daily-updated list of all referrals made to our unit over a 10-month period (1 March–31 December 2004), and (2) ward discharge records for the same period (diagnosis of CES or of ‘Suspected CES – scan normal’). Patients were eligible if they were admitted with symptoms suggestive enough of CES to warrant a lumbosacral MR scan. Scans were reported by a consultant neuroradiologist. Readmissions of the same patient were excluded.

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The case records of possible patients were retrieved. Data from the most senior clinical assessment on admission were extracted using a proforma with the following categories: age, sex, referral source, timing of scan, length of stay, clinical features in the history (low back pain, sciatica, saddle numbness, leg numbness, faecal/urinary incontinence, urinary retention, sphincter sensation) and clinical features on examination (leg power, reflexes, saddle numbness to pinprick, leg numbness). Fisher's exact test (two-sided) was used to compare each of these variables except for age and length of stay (unpaired *t* test).

Results

Recruitment

About 121 patients were newly referred with suspected CES in the 10-month study period. Records for 32 patients were incomplete and could not be analysed [mean age of this group: 45 years; 44% male ($n = 14$)]. Of the remaining 89 patients, 23 were excluded (already had MR scan in referring hospital: $n = 14$; on clinical re-evaluation CES felt unlikely and so not scanned: $n = 9$). This left 66 patients eligible for analysis.

Frequency of scan abnormality and associated clinical characteristics

Only 34 (52%) of the 66 eligible patients had a relevant abnormality on MR. Of these patients, 16 were operated on for a structural abnormality likely to explain their symptoms, 5 further patients were operated on for a nerve root lesion and 13 were managed conservatively. In 32 patients no structural abnormality was seen and no alternative organic cause found during the course of their neurosurgical admission.

Tables 1 and 2 show that there were no statistical differences in sex, timing of scan and clinical features in the history or examination between patients with and those without structural abnormality. The only differences between the two groups were referral source (hospital-referred patients were more likely to have a relevant abnormality), the average length of stay (patients without abnormality had a significantly shorter inpatient stay) and the average age (those with no radiological abnormality were marginally younger).

Discussion

Our study suggests that patients with symptoms suggestive of cauda equina syndrome, but without proven structural

abnormality or other obvious disease (what might be termed 'pseudo-cauda equina syndrome'), present as frequently to a regional neurosurgical unit as those with CES related clearly to a structural cause. Furthermore, in this retrospective study simple clinical features could not be used to distinguish between these two groups. The high proportion of 'out-of-hours' scans in each group suggests that the clinical opinion of the assessing doctor was also a poor discriminator.

These findings support the recent conclusions of a similar but smaller study of 23 consecutive community-based referrals to our neurosurgical unit, with all patients reporting back pain and a recent onset of urinary symptoms in addition to varied neurological abnormalities. In this study, which drew on a different sample of patients from ours, only 57% of MR scans revealed a compressive structural pathology [2]. No clinical feature was found to predict radiological findings although small numbers precluded meaningful statistical analysis.

Apart from this study we could find little data on this topic. In the pre-MR era, normal myelography was reported in a group of 19 patients with symptoms suggestive of compressive CES [16]. Although two were later found to have compression of the thoracic spinal cord, the majority recovered with conservative treatment. In patients with low back pain and sciatica, imaging often fails to show nerve root impingement with one study reporting a 'non-explanatory scan' frequency of 45% [7].

To date, patients with pseudo-CES have not been specifically highlighted as a clinical group even though they have dramatic symptoms that cause anxiety in the community, leading to continued admission to hospital and the use of precious out-of-hours MR resources.

An organic differential diagnosis of pseudo-CES

What could be causing patients to present with leg weakness, numbness and bladder disturbance in the absence of obvious structural pathology? There are several potential 'organic' explanations for an acute or sub-acute cauda equina or conus medullaris lesion in which neuroimaging, even with contrast, may still be normal.

These include transverse myelitis [12], procedure-related damage (e.g. injection of anaesthetic during lumbar puncture), a spinal dural arteriovenous fistula, spinal ischaemic stroke, inflammatory lumbosacral polyradiculopathy (autoimmune or infective), vasculitis, lymphoma, epidural abscess and meningeal malignancy.

However, none of these explanations emerged in any of the patients with normal imaging in our study (at least during their admission to the neurosurgical ward). Could 'pseudo-CES' sometimes be explained by 'functional' disorder of the lower limbs and sphincters, possibly related to acute back pain?

Table 1 Demographic and historical features of patients with ‘pseudo-cauda equina syndrome’ compared to those with relevant structural abnormalities on magnetic resonance imaging

	No relevant abnormality on scan (n = 32) n (%) ^a	Relevant abnormality on scan (n = 34) n (%)	P value (Fisher’s exact test)
Mean age (whole years)	40	46	0.049 ^b
Range	13–59	22–72	
Sex			0.79
Female	24 (75)	24 (71)	
Referral source			0.02
Primary	26 (81)	18 (53)	
Tertiary	6 (19)	16 (47)	
Scan time			0.21
In-hours (0900–1700 hours)	15 (48)	22 (67)	
Out-of-hours	16 (52)	11 (33)	
Unrecorded (n)	1	1	
Average stay (days)	3.6	7.2	0.001 ^b
Range	1–11	1–22	
Low back pain			0.61
Yes	29 (91)	29 (97)	
No	3 (9)	1 (3)	
Unrecorded (n)	0	4	
Numb saddle			0.66
Yes	12 (71)	10 (83)	
No	5 (29)	2 (17)	
Unrecorded (n)	15	22	
Sciatica			0.19
Unilateral	14 (50)	19 (70)	
Bilateral	9 (32)	7 (26)	
No	5 (19)	1 (4)	
Unrecorded (n)	4	7	
Numb leg			0.53
Unilateral	10 (53)	11 (52)	
Bilateral	7 (37)	5 (24)	
No	2 (11)	5 (24)	
Unrecorded (n)	13	13	
Incontinence			0.37
Urine	13 (54)	9 (38)	
Faeces	1 (4)	0 (0)	
Both	2 (8)	1 (4)	
No	8 (33)	14 (58)	
Unrecorded (n)	8	10	
Retention			0.46
Urine	9 (53)	7 (30)	
Faeces	1 (6)	3 (13)	

Table 1 continued

	No relevant abnormality on scan (n = 32) n (%) ^a	Relevant abnormality on scan (n = 34) n (%)	P value (Fisher’s exact test)
Both	0 (0)	1 (4)	
No	7 (41)	12 (52)	
Unrecorded (n)	15	11	
Insensate			0.75
Urine	9 (45)	7 (32)	
Faeces	0 (0)	0 (0)	
Both	1 (5)	1 (5)	
No	10 (50)	14 (64)	
Unrecorded (n)	12	12	

^a ‘Unrecorded (n)’ subtracted from the denominator before calculation

^b Two-sided *t* test

Functional symptoms

Functional symptoms such as paralysis and numbness are common in neurology. Also known as ‘non-organic’ or ‘conversion’ symptoms, or ‘symptoms unexplained by disease’, they are present in up to one-third of new neurology outpatients depending on the definition. Functional paralysis has an incidence of around 3–5/100,000 [3] (similar to multiple sclerosis) and has previously been reported to mimic CES [9]. A systematic review of the literature on motor and sensory conversion symptoms found that physical injury, and thus pain, was common just prior to onset (37%), particularly so in patients with paraparesis (64%) [14]. The theory that weakness and sensory symptoms are functional in some of these patients is therefore plausible.

Bladder symptoms might seem to be an unusual concomitant of functional symptoms and indeed have only rarely been reported in the literature of the last 100 years. Urinary retention as part of hysteria (hysterical ischuria) was however noted by Charcot and others [4]. Our own clinical experience is that urinary and bowel sphincter symptoms can occur in patients with functional paraparesis in the absence of disease, especially at moments of acute back pain. However, urinary retention secondary to impaired relaxation of striated muscle of the urethral sphincter has been described by Fowler et al. [6], and may account for some bladder symptoms previously characterised as psychogenic.

In addition, the reflex changes seen in the patients with normal imaging needs to be explained (reflex loss was seen in 42% of patients). These examination findings may indicate an alternative organic diagnosis but also could have been inaccurate, a consequence of previous problems

Table 2 Examination features of patients with ‘pseudo-cauda equina syndrome’ compared to those with structural abnormalities on magnetic resonance imaging

	No relevant abnormality on scan (<i>n</i> = 32) <i>n</i> (%)	Relevant abnormality on scan (<i>n</i> = 34) <i>n</i> (%)	<i>P</i> value (Fisher’s exact test)
Power			>0.9
Normal	13 (42)	13 (39)	
Unilateral loss	10 (33)	11 (33)	
Bilateral loss	8 (26)	9 (27)	
Unrecorded (<i>n</i>)	1	1	
Reflexes			0.81
Normal	14 (45)	15 (45)	
Unilateral loss	6 (19)	8 (24)	
Bilateral loss	7 (23)	5 (15)	
Unilateral increase	1 (3)	0 (0)	
Bilateral increase	3 (10)	5 (15)	
Unrecorded (<i>n</i>)	1	1	
Saddle numbness			0.80
Yes	17 (57)	17 (54)	
No	13 (43)	15 (47)	
Unrecorded (<i>n</i>)	2	2	
Leg numbness			>0.9
Yes	24 (80)	25 (81)	
No	6 (20)	6 (19)	
Unrecorded (<i>n</i>)	2	3	

or may even be consistent with a ‘functional’ problem. The literature on reflex changes in functional paralysis [1] and in volunteers simulating [13] suggests that asymmetry of the deep tendon reflexes can occur in these situations.

Physical and psychological aspects of back pain

Many of us may have experienced feeling transiently weak at the knees during acute back pain. Could an abnormally persistent physiological response to acute back pain lead to weakness and numbness of the legs in the absence of a structural cause, and independent of emotional or conscious processing? Evidence of mechanisms at both spinal and cerebral level that supports this possibility is emerging [8]. The CNS could have evolved deliberately to be inhibited at times of acute pain [5]. Therefore, it could be inaccurate to label patients presenting with ‘pseudo-CES’ as ‘psychogenic’, if they present acutely in relation to pain.

Limitations

This was a retrospective case note survey prone to observer bias; it reports clinical data that were not recorded

systematically. Over the course of a 10-month period the notes for 32 patients referred with suspected CES were incomplete. The use of discharge summary data may have misrepresented (increased) the relative numbers of patients with CES compared to pseudo-CES, who may have been discharged with a simple diagnosis of ‘back pain’. Although most of the recorded assessments were by a senior neurosurgeon, some were based on a more junior opinion making the data more generalisable but perhaps less reliable. Potential organic causes could have been more actively excluded, especially with CSF analysis.

Implications for future research

Prospective research is needed to further define this group of patients, their prognosis and their treatment. Our hypothesis that many of these patients have functional symptoms related to back pain needs testing. The search for discriminating clinical features should also not be abandoned given the small numbers studied to date. A prospective study including detailed history of the timing and severity of weakness, pain and sphincter disturbance, the presence of dissociative or panic symptoms, previous symptoms unexplained by disease and the presence of positive signs of functional symptoms [15] could yet find clinically relevant features. Such research is warranted on both symptom severity and economic grounds.

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