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Predictors of outcome in cauda equina syndrome

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Abstract This retrospective review examined the cause, level of pathology, onset of symptoms, time taken to treatment, and outcome of 19 patients with cauda equina syndrome (CES). The minimum time to follow up was 22 months. Logistical regression analysis was used to determine how these factors influenced the eventual outcome. Out of 19 patients, 14 had satisfactory recovery at 2 years post-decompression; 5 patients were left with some residual dysfunction. The mean time to decompression in the group with a satisfactory outcome was 14 h (range 6–24 h) whilst that of the group with the poor outcome was 30 h (range 20–72 h). There was a clear correlation between delayed decompression and a poor outcome ($P = 0.023$). Saddle hypoaesthesia was evident in all patients. In addition complete perineal anaesthesia was evident in 7/19 patients, 5 of whom developed a poor outcome. Bladder dysfunction was observed in 19/19 patients, with 12/19 regarded as having significant impairment. Of the five patients

identified as having a poor overall outcome, all five presented with a significant sphincter disturbance and 4/5 were left with residual sphincter dysfunction. There was a clear correlation between the presence of complete perineal anaesthesia and significant sphincter dysfunction as both univariate and multivariate predictors of a poor overall outcome. The association between a slower onset of CES and a more favourable outcome did not reach statistical significance ($P = 0.052$). No correlation could be found between initial motor function loss, bilateral sciatica, level or cause of injury as predictors of a poor outcome ($P > 0.05$). CES can be diagnosed early by judicious physical examination, with particular attention to perineal sensation and a history of urinary dysfunction. The most important factors identified in this series as predictors of a favourable outcome in CES were early diagnosis and early decompression.

Key words Cauda equina · Outcome

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Introduction

The cauda equina refers to that part of the nervous system consisting of peripheral nerves, both motor and sensory, below the level of the conus medullaris and within the spinal canal. The conus medullaris contains the myelomeres of the five sacral nerve roots. Damage to any part of this structure may result in the cauda equina syndrome.

This is typically characterised by low back pain, sciatica, saddle and perineal hypoaesthesia or anaesthesia, a decrease in anal tone, absent ankle, knee or bulbocavernosus reflexes, as well as bowel and bladder dysfunction.

Several causes of cauda equina syndrome have been reported in the literature, including traumatic injury [3, 5, 12, 17, 22], central disc protrusion [16, 27, 30], chiropractic manipulation [28] and metastatic invasion [11, 18].

Early diagnosis and decompression, regardless of the cause, has been recommended to minimise residual neurological deficit [28, 29, 30]. Many authors, however, have found that early decompression is not an indicator of a more favourable outcome [9, 14, 16, 21].

Presenting symptoms have also been put forward as indicators of eventual outcome in the cauda equina patient. Lower limb pain, both unilateral and bilateral, has been implicated as a harbinger of a poor outcome. O'Laoire et al. [21] in a review of 29 cases, determined that the outcome for patients with unilateral sciatica was more favourable than that for those who presented with bilateral involvement. This correlation between bilateral sciatica and a poor prognosis has been well documented [25, 28, 30]; however, other investigators have shown that severe residual dysfunction can exist with the absence of any lower extremity pain [24, 25]. Other presenting symptoms, including saddle hypoaesthesia, sphincter loss, and absent reflexes, have also been associated with variable outcomes in the literature [26].

It is clear, therefore, that the prognostic indicators for a good outcome following cauda equina syndrome are both confusing and vague. In this study, we examine the cause, presenting symptoms, duration of onset, level of injury and time to decompression of 19 patients who presented to the National Spinal Injuries Unit over a 7-year period. A correlation was sought between these parameters and the eventual outcome of each patient, to identify prognostic indicators and to establish practice guidelines for the future.

Materials and methods

We carried out a retrospective review of 397 consecutive patients with lumbar spine pathology treated at the National Spinal Injuries Unit between 1987 and 1994. We defined cauda equina compression as a complex of low back pain, sciatica (pain extending down the lower limb in a dermatomal pattern), saddle hypoaesthesia, and motor weakness in the lower extremities in association with either bowel or bladder dysfunction. Not all criteria were required for the diagnosis to be established. Patients were admitted to the study on the basis that they had symptoms of urinary dysfunction and sad-

dle hypoaesthesia, with varying degrees of motor and sensory loss in either of the extremities. Diagnosis of cauda equina syndrome was made on the basis of history and physical signs supplemented with either radiographs, CT, MRI and myelography or a combination of same. Any patients who did not satisfy these criteria were excluded from the study. Similarly all patients who had CES secondary to trauma or from long-standing malignant infiltration were excluded, to maintain a homogeneous cohort. We identified 32 patients who satisfied our criteria for cauda equina syndrome. In 19 cases all charts and radiographs were available for review and were standardised at a 2-year end point for evaluation (range 22–32 months).

Patients were divided into three groups with regard to onset of symptoms in accordance with Tandon and Sankaren [29]. Group I, in which the symptoms arose suddenly without previous history of backache, group II in which there was an acute onset of bladder dysfunction following a long history of low back pain and group III, in which CES arose gradually from a background of chronic low back pain and sciatica.

We defined early decompression as treatment within 24 h of the development of the full syndrome and late decompression any time following this. All patients underwent a laminectomy followed by bilateral decompression. Patients were commenced on a physical therapy and occupational therapy programme post operatively, and this was continued in a spinal rehabilitation facility until the patient was regarded as independent.

The term 'sphincter control' refers to both motor and sensory supply of bowel and bladder and not only to the actual sphincter. This follows the convention set down by Shepard [28]. Significant sphincter impairment was established as the inability to control micturition and or defecation, whilst partial impairment was attributed to patients with some functional control.

We defined a good outcome as an absence by the time of the 2-year follow-up of any residual deficit that was regarded as a physical or psychological impairment.

Logistical regression analysis was used to determine whether presenting symptoms, time to decompression or level of injury were significant predictors of poor outcome. The likelihood ratio (LR) chi-square test was used to evaluate the significance of each variable as univariate and multivariate prognostic indicators. Two-tailed values $P < 0.05$ were considered significant. Data analysis was conducted using the SPSS software package, version 6.1 (SPSS, Chicago).

Results

We evaluated the full medical records, operative records, and radiographs of the 19 available patients who satisfied

Table 1 Profile of patients with a poor outcome following decompression for cauda equina syndrome (EHL extensor hallucis longus)

Age	Sex	Aetiology	Level	Bilat. sciatica	Bowel & Bladder dysfunc.	Perineal anaesthesia	Motor loss	Time (h) to op.	Residual loss
34	F	Degen. disc	L4-L5	Y	Y	Y	Nil	24–36	Labial, bladder & rectum
31	F	Degen. disc	L4-L5	Y	Y	Y	Decreased eversion	36–72	Bladder alone
41	F	Degen. disc	L5-S1	Y	Y	Y	Nil	36–72	Bladder alone
70	M	Degen. disc & stenosis	L5-S1	N	Y	Y	Nil	36–72	Bladder & rectum
57	M	Degen. disc	L5-S1	Y	Y	Y	Decreased EHL	36–72	Scrotal & sexual dysfunc.

Table 2 Aetiology of patients presenting with cauda equina syndrome and level of injury

Aetiology	Level of injury	No. of patients
Degenerative disc	L3-L4	1
Disease (<i>n</i> = 17)	L4-L5	8
	L5-S1	8
Spondylosis (<i>n</i> = 2)	L4-L5	2

Table 3 The influence of early decompression on outcome in cauda equina syndrome

Outcome	Time to decompression	
	0–24 h	24–72 h
Satisfactory outcome (<i>n</i> = 14)	11 pts	3 pts
Poor outcome (<i>n</i> = 5)	0 pts	5 pts

the criteria for a diagnosis of cauda equina syndrome. Twelve of these were male and seven female. The mean age for this cohort was 55 years (range 31–76 years). All patients were treated with surgical decompression.

Of the 19 patients, 14 were determined to have a good outcome. The remaining five were deemed to have a poor outcome 2 years after surgery. A profile of these patients and their residual deficits are presented in Table 1.

The aetiology of cauda equina syndrome in this cohort was weighted heavily towards prolapse of a lumbar intervertebral disc, accounting for 17 of the 19 patients. Of these, 14 were central disc prolapses and 3 posterolateral. The two remaining patients were diagnosed as suffering a lumbar spondylosis.

The level of injury varied; however, L4-L5 and L5-S1 were the most commonly affected levels (Table 2). When level of injury was examined in the subset of patients with a poor outcome, two patients had pathology at the L4-L5 level and three at the L5-S1 level. These findings were indicative of the prevalence of this level of pathology in the group overall. On statistical analysis no correlation could be found between a poor outcome and the level of pathology.

All patients were operated on within 72 h of presentation, the average time to decompression being 30 h (range 6–70 h). In those who had a complete recovery, 11 patients were decompressed within 12 h and three operated on within 36 h. Of the five patients regarded as having an unsatisfactory outcome, one was decompressed between 24 and 36 h and four between 36 and 72 h (Table 3). This represents a clear association between delayed decompression and a poor outcome. On statistical analysis, delayed decompression of greater than 24 h was shown to be a significant predictor of a poor outcome ($P = 0.0350$).

All patients presented with a pre-existing history of backache. The duration of backache ranged from 3 days to 1 year. Eighteen patients presented with symptoms of

sciatica. In 14 of these, the sciatica was bilateral and in the remaining 4 it was unilateral at initial presentation. Of the 14 patients who presented with bilateral sciatica, 11 gave a history of initial unilateral sciatica, which progressed to a bilateral condition. The one patient who had back pain but no sciatica developed impaired sphincter disturbance both in the bowel and bladder immediately following lifting heavy machinery at work secondary to a central prolapsed disc. The presence of bilateral sciatica or the subsequent development of bilateral symptoms from unilateral sciatica was not a predictor of a poorer outcome overall. No correlation could be determined between residual dysfunction and bilaterality.

The onset of symptoms prior to the development of the full syndrome varied across the 19 patients in this review. One patient had a rapid onset of the syndrome without prodromal symptoms – a Tandon and Sankaren type I. Eight patients with a prolonged history of backache who developed acute sphincter dysfunction were regarded as type II onset CES, and ten patients who had a gradual onset of the syndrome were classified as type III. A correlation was established between a more rapid onset of symptoms and a poorer outcome, but this did not reach statistical significance ($P = 0.052$).

All patients had bladder dysfunction prior to decompression. Bladder dysfunction symptoms ranged from post-micturition residual volume to urinary retention and painful bladder distension. Of the 19 patients, 12 required urinary catheterisation due to complete loss of bladder control. Eight of these 12 were type II CES, three type III CES and one type I. These were regarded as having significant sphincter dysfunction.

The remaining seven patients were determined to have partial sphincter loss, as they were able to control micturition by manual compression or breath holding and did not have urinary retention at presentation, necessitating immediate catheterisation. These seven patients were all type III Tandon and Sankaren CES.

Of those patients with urinary retention, the average time to voiding without a catheter following decompression was 6 days (range 72 h – 3 months). Of the five patients regarded as having a poor overall outcome, all had significant sphincter dysfunction at presentation and four complained of residual bladder compromise following decompression. One patient with painful bladder distension still required intermittent self-catheterisation 2 years after onset of symptoms. Three other patients had hypotonic bladders identified by cystometrogram at 2 years post decompression.

Of the 19 patients identified, 15 were determined to have decreased anal tone and an absent perianal wink at presentation. Normal anal tone recovered on average within 10 days of decompression in the group with a satisfactory outcome (range 24 h – 2 months). Of the five patients with a poor overall outcome, four presented with significant anal sphincter dysfunction, two recovered full

function and two patients complained of poor faecal continence at 2 years, but could minimise this by regular bowel evacuations either with laxatives or manually. The one residual deficit that they were unable to control was flatulence. There was a clear association between the severity of preoperative bladder and/or anal sphincter disturbance and overall residual dysfunction. Bladder and anal sphincter dysfunction were both univariate and multivariate predictors of a poor outcome (Table 4).

All patients in this series demonstrated saddle hypoaesthesia as part of the inclusion criteria to the study. Saddle hypoaesthesia recovered on average 6 days post decompression (range 12 h – 2 weeks). Complete perianal sensory impairment was recorded in seven patients overall. Of the five patients with a poor outcome, all had complete perianal anaesthesia in addition to saddle hypoaesthesia. Two men suffered residual scrotal sensory impairment and two women residual labial sensory impairment following decompression. There was a significant correlation between the presence of complete perineal anaesthesia and a poor outcome following decompression ($P = 0.023$). As saddle hypoaesthesia was a defining element of the syndrome it was not a predictor of the outcome.

Eleven patients were documented as having weak extensor hallucis longus grades of 4/5 or less. Twelve patients demonstrated reduced subtalar eversion, ten bilateral and two unilateral. Six patients had documented plantar flexion weakness, five bilateral and one unilateral. Full motor recovery was seen in all at 4 months post surgery.

Knee and ankle tendon reflexes were consistently recorded on all patients. The bulbocavernosus reflex was, however, performed only on ten patients, all male. Twelve out of 19 patients had absent or reduced ankle reflexes, while eight patients demonstrated absent or reduced knee reflexes. No significance could be attributed to the presence or absence of reflexes at presentation and eventual outcome following decompression ($P = 0.854, 0.9116$).

Discussion

Cauda equina syndrome is uncommon, accounting for a reported incidence of 1–5% of spinal pathology in the literature [10, 14, 20, 27]. Acute trauma as a cause of this syndrome is rare [12, 25], and this paper supports lumbar disc herniation as the most common aetiological factor [2, 7, 13]. The most consistent presenting features in this series were bladder symptoms in association with saddle hypoaesthesia and loss of motor function. Our recovery rate of greater than 70% at 2-year follow-up, to a satisfactory functional recovery following decompression, compares favourably with that of other studies [1, 15, 17, 27].

Early surgical decompression has been suggested to obtain a satisfactory recovery. Shepard [28] associated delayed surgical decompression with a poor outcome and advocates early intervention as a prerequisite to return of

full neurological function. Nielson et al. [19] supported this concept of early decompression, establishing by urodynamic parameters that early intervention correlates with improved bladder function. The literature is replete with reports of the advantages of early operative decompression [1, 8, 14, 21, 23, 30]. Despite this, however, several papers have questioned the role of early decompression. Bohlman [4], in a paper on post-traumatic cauda equina, demonstrated significant recovery 30 years following the initial injury with late decompression. Kostuick et al. [16] have also demonstrated excellent return of motor function with decompression up to 3 days following presentation. Delamater et al. [9] confirmed these findings with canine experimental cauda equina syndrome. He could find no differences in somatosensory evoked potentials between animals treated within the 1st h and those treated at 1 week.

In our study, early decompression was a significant factor in preventing a poor outcome. All five patients with residual impairment were operated on 24–72 h following presentation, whilst in the group with no residual impairment post decompression 78% were operated on within 24 h of presentation and the remainder within 36 h (Table 1).

We believe the decision for early intervention prevents the progression of partial sphincter disturbance to a complete lesion in many cases. This progression has been documented as occurring even within the first 24 h [21]. Clearly, early decompression of neural elements prevents the progression of the syndrome and promotes an early return of function.

The most common level of pathology in this group was at the L4-L5 level, and the L5-S1 level. This correlates well with other investigations into cauda equina [27, 28]. We could find no significant difference between the levels of pathology as predictors of the eventual outcome. There is evidence in the literature, however, to suggest that involvement of the lower sacral levels manifests clinical symptoms that are more subtle than those of lumbar root involvement [21]. Sacral root lesions may not cause motor reflex changes, and the diagnosis of cauda equina may be delayed or missed if careful attention is not directed towards the evaluation of sensation in the saddle and perineal area. This observation has been previously recognised by Scott [26], who associates permanent bladder paralysis with complete perineal anaesthesia at presentation. In our study, L5-S1 pathology was associated with negligible motor loss and a resultant delay in both diagnosis and surgical decompression. Although the level of pathology was not a predictor of outcome per se, the correlation between a loss of perineal sensation and a poor outcome in our study may be related to the time taken to diagnose the more subtle sacral level pathology.

In this review, all four patients with residual sphincter disturbance at 2-year follow-up were regarded as having had severe sphincter dysfunction prior to decompression. Severity of sphincter dysfunction was concluded to be an

independent predictor of a poor prognosis. It was also found to be a multivariate predictor of outcome in association with time taken to decompression and perineal anaesthesia, the clear implication being that a delay in diagnosis or treatment manifests itself in greater levels of nerve root impairment and consequent unsatisfactory outcome. Both Shapiro [27] and Robinson [23] have reported rates of 70–100% recovery in urological function in patients who were treated within 48 h. Jennett [14] and Nielson et al. [19], have similarly demonstrated that significant sphincter dysfunction can remain if left untreated for greater than 48 h. It is clear that sphincter disturbance is a prognostic indicator both in isolation and in conjunction with duration of symptoms.

In this series, sciatica was a presenting symptoms in all cases of cauda equina, with the exception of the one patient who developed sudden-onset CES. Bilateral sciatica was more common at initial presentation and, using logistical regression analysis, was found to be a predictor of a poor outcome. This is in agreement with O'Laoire et al. [21] and others [1, 5, 9], who identified bilateral sciatica as a prognostic indicator. Our results, however, cannot establish unilateral sciatica as a predictor of a more favourable outcome. Bilateral sciatica in this series would appear to be a progression of single-sided symptoms. These observations are similar to those of Robinson [23] and Kostuick et al. [16], who identified unilateral sciatica as a common mode of presentation in patients who subsequently developed bilateral symptoms and the cauda equina syndrome.

When motor function was evaluated, no correlation could be found between a poor outcome and an initial motor loss at presentation. All patients recovered full motor recovery both in the group with poor overall outcome and in the group with satisfactory overall outcome.

The onset of symptoms in this series ranged from sudden onset to a gradual progression over several weeks.

The time taken to decompression was measured from the time that the patient presented with the complete syndrome. Of those who had a poor outcome there were three type II onsets, one type I onset and one type III onset. Although a correlation between a more rapid onset of symptoms and a poor outcome was shown, this did not reach statistical significance. Kostuick et al. [16] also demonstrated a slightly poorer prognosis following decompression in those with a more acute onset of symptoms in their review of 31 cases. This may be due to those with more severe symptoms and consequently the more significant pathology presenting earlier than those with a slower onset. This is supported by the association of complete perineal anaesthesia and significant sphincter impairment with a poorer outcome.

Cauda equina syndrome is a relatively uncommon condition that may be seen in traumatic injury, spondylosis, metastatic disease and, most commonly, following lumbar disc herniation. We have identified a clear correlation between a delayed decompression of 24 h or more and a poor outcome. Furthermore, we have identified predictors of residual dysfunction as the presence of significant sphincter disturbance and complete perineal anaesthesia at presentation. A sudden onset of CES may be a harbinger of a poorer prognosis. We were unable to find any correlation between either the level of injury or the presence of initial motor dysfunction and a poor outcome.

We recommend detailed examination not only of the saddle area but also, more importantly in terms of prognosis, the perineal area. Bladder function may often be difficult to assess, but in those patients in whom CES is suspected, urodynamic studies should be performed at initial presentation. Operative intervention should, however, not be delayed for the completion of urologic diagnostic tests. We recommend that decompression be done within 24 h of presentation, particularly in the presence of complete perineal anaesthesia and significant sphincter disturbance.

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