



## Review

## Severity of presentation is associated with time to recovery in spinal epidural lipomatosis



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## ARTICLE INFO

## Article history:

Received 6 March 2015

Accepted 14 March 2015

## Keywords:

Epidural lipomatosis

Glucocorticoids

Prednisone

## ABSTRACT

We present a patient with prednisone-induced spinal epidural lipomatosis (SEL) and relatively acute neurologic deterioration followed by rapid recovery after surgical decompression. SEL is a rare disease characterized by hypertrophy of epidural fat, most commonly associated with exogenous steroid use. To our knowledge, an analysis of the dynamics of steroid dose related to time to onset has never been performed, or of patient presentation features with respect to patient outcome. We retrospectively reviewed the literature for English language series and case reports of SEL associated with prednisone use from 1975–2013. Data were compiled for 41 patients regarding the prescribed dose of prednisone and length of treatment, as well as the severity of symptoms on the Ranawat scale, time to onset, time to recovery, and degree of recovery of neurological symptoms. Fisher's exact test and analysis of variance were used for comparing proportions, and  $p$  values  $<0.05$  were considered statistically significant. We found that the mean cumulative dose of prednisone trended towards an association with a lack of recovery ( $p = 0.06$ ) and may be related to rate of recovery. Prescribed prednisone dose varied inversely with the time before onset of neurological symptoms, but failed to reach statistical significance. Higher severity of presenting symptoms on the Ranawat scale were found to be associated with a higher likelihood of delayed recovery ( $p = 0.035$ ). Patients with symptoms lower on the Ranawat scale more frequently experienced complete neurologic recovery, though this did not reach significance. The acuity of neurological deterioration was not related to the time to recovery or ultimate degree of recovery. Severity of presentation on the Ranawat scale is associated with rate of recovery and may be related to degree of recovery in SEL patients. Cumulative dose of prednisone may be related to degree and rate of recovery. Prescribed dose of prednisone may be related to time to onset of neurological symptoms. Acuity of neurological deterioration is not related to rate or degree of recovery.

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### 1. Introduction

Spinal epidural lipomatosis (SEL) is a rare condition first described by Lee et al. in a postoperative renal transplant patient in 1975 [1]. SEL is characterized by increased deposition of adipose tissue in the epidural space and is most commonly associated with exogenous administration of corticosteroids [2]. Less commonly, SEL may be due to obesity or elevated endogenous steroids, as seen in Cushing's syndrome. Patients generally present with insidious development of weakness, paresthesia, numbness, abnormal reflexes, incontinence and/or paralysis [3]. We present a patient with acute onset cauda equina syndrome due to SEL after treatment with oral glucocorticoids for temporal arteritis and showed rapid neurological recovery postoperatively. We conducted an

analysis of the dynamics of SEL to evaluate the potential relationships between relevant parameters including dose of steroids, length of onset of symptom development and recovery after treatment.

### 2. Case report

A 58-year-old woman with a past medical history of type 2 diabetes mellitus, chronic obstructive pulmonary disease and steroid-responsive orbital pain with vision changes presented with 1 month of symptoms consistent with progressive cauda equina syndrome. In the week prior to presentation, her lower extremity weakness and numbness became more severe and she developed new urinary and bowel incontinence. Due to complaints of orbital pain and vision loss, which was diagnosed clinically as temporal arteritis, she had been taking 55 mg prednisone once daily for 1 year and was responsive to the steroids.

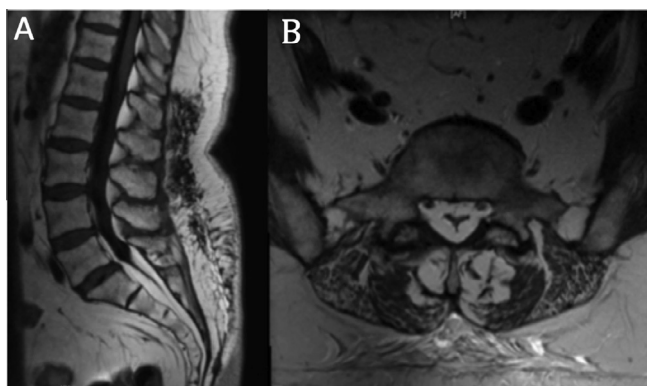
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On physical examination, she was overweight with a body mass index of 29 and was cushingoid in appearance. She had striking moon facies, buffalo hump and central fat deposition. Her upper extremities were 5/5 in strength with normal sensation and reflexes. However, she had 4-plus strength bilaterally in dorsiflexion and plantarflexion. She also had diminished sensation to light touch, pain and temperature in her ankles and feet bilaterally. Lower extremity reflexes were absent. MRI demonstrated a prominent epidural fat signal on T1-weighted images in the spinal canal with circumferential compression of the thecal sac extending from L1 to the sacrum. The Y sign was present in the axial plane indicating circumferential dural sac compression by epidural fat [4] (Fig. 1). Measurements of the spinal canal demonstrated that the woman had Grade III lumbar epidural lipomatosis by the Borre MRI classification [5].

The woman underwent successful L5–S2 laminectomy with debulking of excess epidural fat to decompress the thecal sac. During the operation, large quantities of epidural fat appeared to be under pressure and herniated out immediately following removal of the lamina and ligamentum flavum (Fig. 2).

Postoperatively, the woman showed immediate improvement in strength and sensation and complete resolution of her incontinence. She remained in the hospital for 4 days postoperatively and was discharged to acute inpatient rehabilitation. Her symptomatic and functional improvements were maintained through her last follow-up visit, 1 year after surgery. She has been gradually weaned off her prednisone as tolerated with respect to recurrence of her temporal arteritis symptoms. She currently takes 10–15 mg prednisone once daily.



**Fig. 1.** Spinal MRI of a 58-year-old woman with lower extremity weakness, numbness and incontinence at presentation. (A) Sagittal T1-weighted and (B) axial T1-weighted images demonstrating expansion of fat tissue in the spinal canal engulfing and compressing the thecal sac and nerve roots. A Y sign is present in the axial plane.



**Fig. 2.** Intraoperative photograph of epidural fat herniating dorsally through the laminectomy defect following removal of lamina and ligamentum flavum. This figure is available in colour at [www.sciencedirect.com](http://www.sciencedirect.com).

### 3. Materials and methods

We present a woman who was being treated with prednisone with relatively acute neurologic deterioration from SEL and showed a remarkably rapid recovery after surgical decompression. We retrospectively reviewed the literature for English language case reports and series of SEL associated with prednisone use from 1975–2013. Data were compiled for 41 patients and analyzed regarding the prescribed daily dose of prednisone and total duration of steroid therapy, as well as the severity, time to onset, time to recovery, and degree of recovery of neurological symptoms. Prescribed dose was calculated as a weighted mean of all prescribed doses over the patient's clinical course, where available. Cumulative steroid dose was calculated as the product of the mean daily prescribed dose and the total days of steroid treatment. Fisher's exact test and analysis of variance were used for comparing proportions and *p* values <0.05 were designated as statistically significant.

### 4. Results

#### 4.1. Prescribed steroid dose

The weighted mean daily prescribed doses of prednisone and time to onset of neurological symptoms were available for 32/41 SEL patients found in the literature. Seven of these patients were managed conservatively with reduction of their steroid dose, 18 were managed with surgery alone and seven were managed with both surgery and steroid reduction. Time to onset of neurological symptoms was determined based on the patients' case description and compared with daily prescribed prednisone dose (Table 1) [1,6–29]. The relationship between these two variables is represented in a scatter plot and appears to be non-linear and inverse (Fig. 3). Notably, 11/19 patients who presented within 15 months

**Table 1**

Daily prednisone dose and the respective time to onset of symptoms in spinal epidural lipomatosis patients published in the English language literature

Author, year	Daily dose prednisone (mg)	Time to symptom onset (months)
Lee, 1975 [1]	48	15
Butcher, 1979 [8]	150	15
Godeau, 1979 [15]	80	6
Lipson, 1980 [20]	92	6
Chapman, 1981 [10]	24.5	11
Chapman, 1981 [10]	50	7
Chapman, 1981 [10]	15	48
Guegan, 1982 [16]	60	15
Archer, 1982 [6]	10	156
George, 1983 [14]	42	6
Randall, 1986 [26]	20	6
Jungreis, 1987 [19]	10	240
Haid, 1987 [17]	20	72
Zampella, 1987 [29]	47.6	21
Zampella, 1987 [29]	60.5	8
Quint, 1988 [25]	20	12
Fessler, 1992 [13]	16	6
Fessler, 1992 [13]	20	12
Fessler, 1992 [13]	10	84
Healy, 1987 [18]	51	44
Soloniuk, 1989 [27]	60	15
Maehara, 1988 [21]	50	44
Tobler, 1988 [28]	24	4
Buthiau, 1988 [9]	40	11
Perling, 1988 [23]	15	48
Pinsker, 1988 [24]	60	4
Crayton, 1992 [11]	30	120
Arroyo, 1988 [7]	39	13
Miwa, 2013 [22]	7	36
Emon, 2011 [12]	5	72

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