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Case Studies

Spinal epidural lipomatosis: case reports, literature review and meta-analysis

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Abstract

BACKGROUND CONTEXT: Symptomatic spinal epidural lipomatosis (SEL), a rare cause of spinal cord compression, has most often been associated with exogenous steroid use.
PURPOSE: Identify four associations with SEL, correlate the associated groups with level of disease and compare treatment with outcome data in these groups.
STUDY DESIGN/SETTING: Case reports of three patients and analysis of 104 cases from the literature.
PATIENT SAMPLE: Three patients from the senior author's practice.
OUTCOME MEASURES: Not applicable.
METHODS: The authors report three new cases of SEL not associated with steroid use. They review all available English literature and present a table of all 104 reported cases.
RESULTS: The clinical course of three new patients is reported.
CONCLUSIONS: Associated conditions are exogenous steroid use, obesity, endogenous steroid excess, and some remain idiopathic. Although SEL is a rare condition, our review of the literature reveals many more reported cases than previously thought. With increased awareness of this condition and improved imaging techniques, further studies of this disease should be undertaken.

Keywords: Dura mater/pathology; Lipoma/complications/pathology/radiography/surgery; Spinal cord compression/etiology

Introduction

Spinal epidural lipomatosis (SEL) is a disease consisting of an excessive deposition of normal adipose tissue in the spinal canal, compressing the spinal cord. Symptomatic SEL is exceedingly rare and often associated with exogenous steroid use. Although less common, obesity and Cushing syndrome/disease (hypercortisolism) have also played a role in SEL. Epidural lipomatosis becomes symptomatic in rare occasions by causing compression of the spinal cord or nerve roots. Because SEL can mimic other common spine conditions, such as spinal stenosis and degenerative joint disease, it was often underdiagnosed. Symptomatic epidural lipomatosis was first described in 1975 [1]. Since that time, a number of other cases have been diagnosed with the use of imaging in combination with clinical symptoms, history, surgical findings and the absence of other identifiable causes. Patients may present with progressive and longstanding complaints of pain, weakness, numbness, incontinence, ataxia, abnormal reflexes and even rarely paralysis. We report three new cases of SEL.

Case report 1

A 47-year-old man presented with low back pain of 2 years' duration that began after a work-related injury. The pain was situated in his lower back with radiation to the buttocks and thighs bilaterally. It was associated with decreased sensation and weakness in both lower extremities. This pain was not associated with any bowel or bladder incontinence. The patient also had a history of neck pain with associated

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numbness, weakness and loss of sensation in the right upper extremity. His past medical history was significant for renal cancer that was treated by nephrectomy 6 years prior. The patient had no history of steroid use.

On physical examination, the patient was 5 feet 6 inches tall and weighed 140 pounds, with a body mass index (BMI) of 23.4. He had no structural spinal deformity. His range of motion was limited on both flexion and extension. Some tenderness was noted over his lower back bilaterally. Neurological examination revealed that the patient was intact with no nerve root tension signs. A computed tomography (CT) myelogram revealed prominent epidural fat from L2 to S1 with thecal sac compression. Complete cutoff of the intrathecal dye occurred at the L4–L5 level. CT scan cuts through the L4–L5 and L5–S1 levels demonstrated thecal sac compression by an extrinsic circumferential mass consistent with epidural lipomatosis (Figs. 1 and 2).

A laminectomy and decompression was performed from L2 to L5. Operative findings included marked adipose tissue in the spinal canal and stenosis. The patient lacked any neurological abnormalities postoperatively, although he did still complain of lower back pain.

Case report 2

A 48-year-old man developed an acute onset of low back pain that he attributed to a work-related fall. The patient continued to work, and his pain eventually resolved. Subsequently, the patient sustained a lifting injury at work, causing him severe low back pain. He denied leg pain after this injury. The patient participated in physical therapy and took leave of work. Two days later, he was unable to continue the physical therapy because of pain.

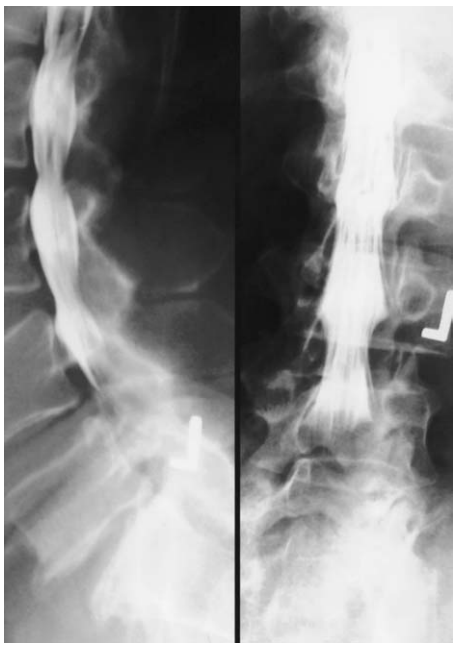

Fig. 1. First patient: anteroposterior and lateral views of the lumbar myelogram demonstrate some extrinsic compression of the thecal contents at L3–L4 with complete blockage of the intrathecal dye above the L4–L5 disc space. Hypertrophic spondylosis is seen radiographically only at L5–S1.

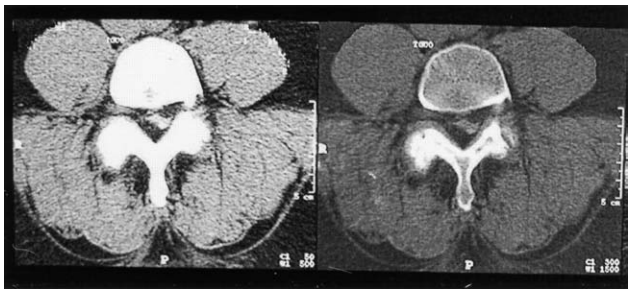

Fig. 2. First patient: axial computed tomography myelogram views of L4–L5 shown in Fig. 1 demonstrate the extrinsic compression of the thecal sac just above the L4–L5 disc space. The compression is symmetrical and is close to water density, certainly less dense than ligamentum flavum, capsule or disc. There is an abnormal dural sac shape changed by the extrinsic pressures of the epidural lipoma.

Magnetic resonance imaging (MRI) sagittal spin-echo T1-weighted images demonstrated widening of fat tissue in the epidural spinal canal rounding and compressing the thecal sac and nerve roots. Hypertrophic spondylosis of L2–S1 in the lumbar spine was noted. Axial T1-weighted MRI of the lumbar spine just cephalic to L5–S1 shows circumferential hyperintense and homogenous tissue elevating the dural sac and the nerve root. A sagittal T2-weighted image showed a high contrast between adipose tissue, and the dural sac on T2-weighted image permitted an accurate evaluation of the extent of pathologic overgrowth of epidural fat in the spinal canal. Hypertrophy of facet joints and end plates with discal bulging resulted in mild canal stenosis at multiple levels and increased epidural fat posterior to the dural sac that appeared compressed and indented below L2 (Figs. 3 and 4).

The patient received an epidural steroid shot. He reported that the pain became worse and began to radiate to both buttocks after the steroid injection. He was unable to walk after the steroid injection. He reported that his symptoms were occasionally accompanied by left leg numbness that extended to the bottom of the foot.

His past medical history was negative. His past surgical history was significant for a gunshot wound to the stomach sustained during the Gulf War. He denies taking steroids. His only medication was tramadol.

On physical examination, the patient was 6 feet tall and 270 pounds, with a BMI of 36.6. The patient had no spinal deformities. He stood with his lumbar spine flexed to 20 degrees, because of pain. He was unable to lie flat. Paravertebral muscle spasm was noted. He had no tenderness to palpation. His neurological examination showed that the patient was intact. A laminectomy and decompression was performed from L3-S1 with bilateral foraminotomy. Direct removal of the epidural lipomatosis was not reported.

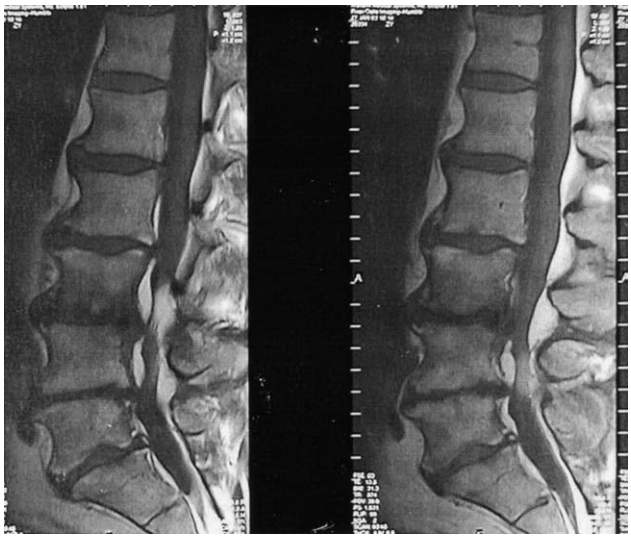

Fig. 3. Second patient: magnetic resonance imaging (MRI) sagittal spin-echo T1-weighted images demonstrate widening of fat tissue in the spinal canal rounding and compressing the thecal sac and nerve roots. This abnormal fat-density compression is thicker between the disc levels and thinner at the areas of degenerative discal bulging. MRI sagittal image also shows hypertrophic degenerative changes at L2–S1.

Postoperatively, the patient improved, although he still complained of low back pain and the inability to stand completely erect. The patient started a supervised physical therapy program with some improvement of his back pain.

Case report 3

A 43-year-old man developed an acute onset of back pain after sustaining a lifting injury. The pain was situated in his mid-thoracic spine and radiated into his neck. Several days later, the pain became much worse and he was unable to walk or stand. He was immediately brought to the emergency room and admitted. His past medical history was negative. The patient had had three prior operations on his back for work-related injuries. The patient had no history of steroid use.

On physical examination the patient weighed 270 pounds and measured 6 feet 4 inches, with a BMI in excess of 30. On palpation, the patient complained of tenderness throughout his spine. His range of motion was severely limited by pain. His motor examination revealed flexor paralysis of both lower extremities. There was also weakness in handgrip bilaterally. Deep tendon reflexes were absent in the lower extremities and for the triceps tendon bilaterally. Plantar stimulation elicited no response. Sensory examination revealed decreased sensation to pinprick from and including the T1 level and down.

MRI of the thoracic spine with axial and sagittal T1-weighted images showed a marked high contrast between adipose tissue and the dural sac on T1-weighted images. The contrast permits an accurate evaluation of the extent of pathologic overgrowth of epidural fat in the spinal canal. On the sagittal T1-weighted image, the posterior epidural stripe of hyperintense lipomatosis is 8 to 10 mm in width throughout the thoracic spine. Axial views of T5–T6 showed extradural compression from epidural fat. Axial cuts from T10–T11 showed restoration of the symmetry of the dural contents and cerebrospinal fluid without the extradural compression (Figs. 5 and 6).

The patient was diagnosed with thoracic SEL and cervical spondylosis. A laminectomy and decompression was performed from C5 to C7 and T1 to T7. Although the patient made some improvement, at time of discharge he was unable to stand or walk and lacked bowel and bladder continence.

Discussion

We conducted a complete review of the available English literature, charting all reported cases of SEL, noting the most common associations of SEL, correlating these associations to the location of the disease process and examining the treatment to outcome data. We found 104 cases of SEL reported in the literature, including our three cases (Table 1).

Pathogenesis

The underlying pathological mechanism of SEL is unknown. A review of the literature and the data reported in the chart below reveal four categorical associations with SEL: exogenous steroid use, obesity, endogenous steroid excess or Cushing syndrome and an idiopathic group. The most common association is exogenous steroid use. SEL has been

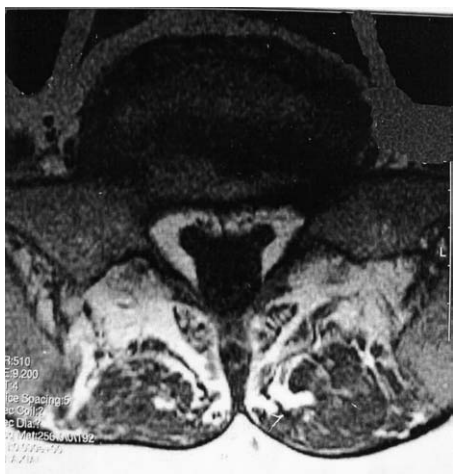

Fig. 4. Axial T1-weighted magnetic resonance imaging of the lumbar spine just cephalic to L5–S1 shows circumferential hyperintense and homogenous tissue elevating the dural sac and the nerve roots toward the posterior lamina, distorting and compressing the dural contents. Calculations of the dural sac to epidural fat anterior and posterior to the dural sac revealed a ratio of 1.05, and the epidural fat to spinal canal anteroposterior distance was 67%. This would be a moderate compression Grade II according to the classification of Borre el al [18].

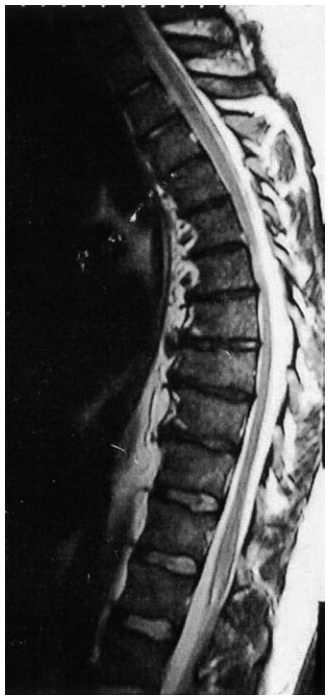

Fig. 5. Third patient: the sagittal T1-weighted image of the thoracic spine reveals that the posterior epidural stripe of hyperintense lipomatosis is 8 to 10 mm in width throughout the thoracic spine.

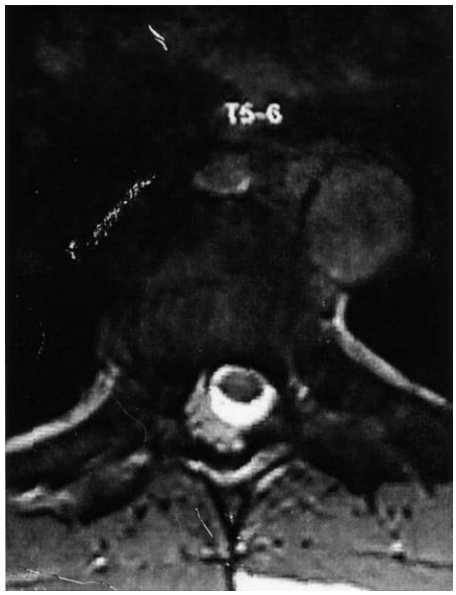

Fig. 6. Third patient: axial views of T5–T6 show displacement of the dural sac cerebrospinal fluid without extradural compression from epidural fat.

documented in association with steroid use for many conditions, including transplantation, systemic lupus erythematosus, rheumatoid arthritis, Graves disease, chronic hepatitis, dermatomyositis, nephritic syndrome, glomerulonephritis, sarcoidosis, Crohn's disease, multiple sclerosis, chronic obstructive pulmonary disease, atopic dermatitis, diabetes mellitus, prostatic cancer, lichen ruber planus, pineoblastoma, cerebral lymphoma, polyarthritis, asthma and polyarteritis nodosum. It is well established that hypercortisolism leads to an accumulation of adipose tissue in a typical distribution, on the face, neck, trunk and mediastinum [2]. Hypertrophy of adipose tissue already present in the spinal canal is theorized to be the cause of SEL in certain cases of exogenous steroid use [3]. Based on our review, 55.3% of all reported cases in the English literature were associated with exogenous steroid use. Although the majority of these cases were associated with long-term steroid use, three of the cases arose from multiple epidural steroid injections: cases 13, 43 and 47. Patient 13 received a total of 103 injections over 12 years, eventually resulting in an abrupt onset of neurologic deficits at the end of this period. Patient 43 received a series of injections over a period of 3 years totaling 1,200 mg of methylprednisolone. Her physical examination revealed sequelae of Cushing syndrome, including moon facies and buffalo hump. Patient 47 received a total of five injections; the last three injections reportedly exacerbated his symptoms. In fact, one of these patients developed Cushing syndrome sequelae from these local injections [3–5].

Obesity is the second most common associated category of SEL. Koch et al. [2] hypothesized that obesity in this patient class may be caused by a pseudo-Cushing state exhibiting elevated urinary free cortisol levels. Unfortunately, no studies of obese patients with SEL have reported cortisol levels to date. Furthermore, some investigators question whether obesity plays a causal role in SEL or is merely a predisposing factor [6]. Borre et al reported 53 severe cases of SEL with 39 obese patients. Additionally, 4 were on steroids, 2 were hypothyroid and 1 was obese, had hypothyroidism and was taking steroids [18]. Our case review revealed 24.5% of reported cases attributable to obesity alone (It is important to note that this percentage includes only those patients who did not take steroids and were classified as obese by either the case reporter or BMI.)

Cushing syndrome/disease from endogenous sources is the third associated category in SEL. To date, only three cases of Cushing syndrome associated with SEL have been reported in the English literature accounting for 3.2% of SEL cases [7–9].

We found no identifiable association with SEL in 17% of the cases. This patient group includes those patients who did not take exogenous steroids, were not obese and did not have an underlying Cushing syndrome/disease to account for the SEL. Unfortunately, some of these patients also lacked certain of the data criteria. Of the 16 cases, 2 of these lacked data on the patient's BMI. However, after consideration, we opted to include these cases in our analysis to be complete in analysis of all known cases in the English literature.

Hypothyroidism in previous papers has been associated with SEL [4,6,10,11]. Upon review, we disagree with this conclusion. There has been only one reported case of hypothyroidism associated with SEL [11]. This patient was obese,

Table 1
One hundred and four cases of spinal epidural lipomatosis from English literature review with diagnosis, presence of steroid usage or obesity, type and outcome of treatment

Case	Reference	Pathology	Levels	Steroids	Obesity	Treatment	Outcome
		Glomerulonephritis					
1	[1]	Transplantation	C7–L4	Yes	NR	Laminectomy	Improved
2	[20]	Asthma	T2–T10	Yes	Yes	Laminectomy	Unchanged
3	[21]	Arteritis nodosum	T1–T9	Yes	NR	Laminectomy	Improved
4	[22]	Thyroiditis	L4–S1	Yes	Yes	Laminectomy	CR
		Chronic hepatitis					
5	[23]	Cushing syndrome	T7–T9	Yes	Yes	Laminectomy	CR
		Polycystic kidney					
6	[23]	Cushing syndrome	T2–T10	Yes	Yes	Laminectomy	Improved
		Polyarthritis					
7	[23]	Cushing syndrome	T2–T10	Yes	NR	Laminectomy	CR
		Glomerulonephritis					
8	[24]	Transplantation	S1–S3	Yes	Yes	Laminectomy	Improved
9	[25]	Asthma	L4–S1	Yes	NR	Laminectomy	CR
10	[26]	Urethral stenosis	T1–L5	Yes	NR	Steroid taper	CR
11	[27]	Asthma	T4–T8, L4–L5	Yes	NR	Laminectomy	Unchanged
12	[28]	Polyarthritis, asthma	L4–S1	Yes	No	Laminectomy	CR
		Multiple sclerosis					
13	[28]	Multiple epidural steroid injections	T10–L5	Yes	No	Laminectomy	Improved
14	[29]	Heart transplant	L3–L4	Yes	Yes	Laminectomy	CR
15	[30]	Asthma	T4–T8	Yes	Yes	Laminectomy	Unchanged
16	[30]	Asthma	T5	Yes	Yes	High-dose steriods	NR
17	[31]	Rheumatoid arthritis	T4–T8	Yes	NR	Laminectomy	Improved
18	[32]	Heart transplant	T4–T10	Yes	Yes	Laminectomy	LE paretic
19	[32]	Heart transplant	T2–T10	Yes	NR	Steroid taper	CR
20	[17]	Asthma	T4–T9	Yes	NR	Laminectomy	Improved
21	[17]	Sarcoidosis	T4–T9, L5–S1	Yes	NR	Laminectomy	Improved
22	[17]	Crohn disease	T2–T8	Yes	NR	Observation	Lost of FU
23	[17]	Prostatic cancer	T4–T8	Yes	NR	Medical	Improved
24	[17]	Pineoblastoma	T3–T9	Yes	NR	Laminectomy	Worse
25	[13]	Heart-lung transplant	T6–T8	Yes	No	Laminectomy	CR
26	[33]	Heart transplant	L3–S1	Yes	NR	Laminectomy	CR
		Asthma				Weight reduction	
27	[33]	Treated with steroid inhaler	L3–S1	Yes	Yes	and steroid taper	Improved
28	[33]	Chronic obstructive pulmonary disease	T5–T12	Yes	NR	Laminectomy	Unchanged
29	[33]	Lichen ruber planus	L3–S1	Yes	NR	Steroid taper	CR
30	[33]	Renal transplantation	T4–T7	Yes	NR	Laminectomy	Improved
31	[34]	Renal transplantation	L5–S2	Yes	Yes	Laminectomy	CR
32	[34]	Cerebral lymphoma	L4–L5	Yes	NR	None	NR
		Systemic lupus					
33	[35]	erythematosis	L1–S1	Yes	Yes	Laminectomy	CR
		Systemic lupus					
34	[36]	erythematosis	T3–T9	Yes	Yes	Laminectomy	Improved
		Radiation pneumonitis					
35	[37]	For adenocarcinoma	T8–T10	Yes	NR	Laminectomy	Improved
36	[38]	Nephrotic syndrome	T5–T10	Yes	NR	Steroid taper	Improved
		Atopic dermatitis					
37	[39]	Cushing syndrome	T6–T9	Yes	NR	Laminectomy	Paraplegic
38	[40]	Renal transplantation	T3–T9	Yes	NR	Laminectomy	CR
39	[41]	Renal transplantation	T1–T12	Yes	Yes	Weight reduction and steroid taper	CR
40	[42]	Dermatomyositis	T4–T7	Yes	NR	Laminectomy and steroid taper	CR
41	[43]	T-cell lymphoma	T10–T11	Yes	NR	Laminectomy	Unchanged
42	[44]	Polymyalgia rheumatica	L4–L5	Yes	Yes	Laminectomy	CR
		Cushingoid sequelae from multiple epidural steroid					
43	[4]	injections	L3–S1	Yes	NR	Steroid taper	CR
44	[45]	Juvenile rheumatoid arthritis	T4	Yes	Yes	Laminectomy and steroid taper	CR

(Continued)

Table 1
Continued

Case	Reference	Pathology	Levels	Steroids	Obesity	Treatment	Outcome
45	[46]	Ulcerative colitis	T1–T11	Yes	Yes	Laminectomy	Improved
46	[47]	Temporal arteritis	T1–T10	Yes	NR	Laminectomy	Improved
47	[5]	None Multiple epidural steroid injections	L2–L5	Yes	No	Laminectomy	CR
48	[48]	Systemic lupus erythematosis Cushing syndrome	Thoracolumbar	Yes	Yes	Weight reduction and steroid taper	Improved
49	[49]	Juvenile rheumatoid arthritis Cushing syndrome	T6–T7	Yes	NR	Laminectomy	CR
50	[49]	Juvenile rheumatoid arthritis Cushing syndrome	L5–S1	Yes	NR	Laminectomy	Improved
51	[50]	None; previous anabolic steroid use for body building	Lumbar	Yes	No	Bed rest, analgesics	Improved
52	[51]	Thrombocytopenia purpura Human immunodeficiency virus treated with ritonavir	L5–S1	Yes	No	Laminectomy	Improved
53	[35]	None	T1–T10	No	Yes	Weight reduction	CR
54	[35]	None	T6–T8	No	Yes	Laminectomy	CR
55	[35]	None	L5–S1	No	Yes	Laminectomy	Improved
56	[15]	None	T5–T8	No	Yes	Laminectomy	Improved
57	[52]	None	L4–L5	No	Yes	Laminectomy	NR
58	[53]	None	L3–L5	No	Yes	Weight reduction	CR
59	[16]	None	L5–S1	No	Yes	Weight management	CR
60	[6]	Diabetes mellitus	T3–T9	No	Yes	Laminectomy	Unchanged
61	[11]	Hypothyroidism	T1–T12	No	Yes	Laminectomy	Unchanged
62	[54]	None	L4–S1	No	Yes	Laminectomy	Almost CR
63	[55]	None	L3–S1	No	Yes	Weight reduction	CR
64	[56]	None	L3–S1	No	Yes	Weight reduction	Improved
65	[57]	Prostatic cancer treated with cyproterone acetate	L2–L4	No	Yes	Weight reduction and cyproterone cessation	CR
66	[58]	None	L5–S1	No	Yes	Weight reduction	CR
67	[59]	None	L4–L5	No	Yes	Laminectomy	CR
68	[59]	None	L4–S1	No	Yes	Weight reduction	CR
69	[60]	None	L5–S1	No	Yes	Weight reduction	Unchanged
70	[60]	None	L4–S1	No	Yes	Weight reduction	Improved
71	Our case	None	L3–L5	No	Yes	Laminectomy	Improved
72	[61]	Spermatocytic seminoma	L4–S1	No	Yes	Laminectomy	CR
73	[19]	None	T1–T10	No	Yes	Laminectomy	Improved
74	[62]	None	L4–S1	No	Yes	Physiotherapy	NR
75	Our case	Cervical spondylosis	T1–T7	No	Yes	Laminectomy	Paraplegic
76	[63]	None	T3–T7	No	No	Laminectomy	CR
77	Our case	None	L2–L5	No	No	Laminectomy	CR
78	[64]	None	T1–T10	No	No	Laminectomy	Improved
79	[12]	None	L4–S1	No	No	Laminectomy	CR
80	[65]	None	L3–S2	No	No	Laminectomy	Improved
81	[66]	None Cerebral palsy Intrathecal baclofen	L4–S1	No	No	Laminectomy and endoscopic decompression	Improved
82	[67]	Pump placement	Thoracolumbar	No	No	Laminectomy	Unknown
83	[17]	None	T4–T8	No	NR	Laminectomy	Improved
84	[68]	None	L3–L4	No	No	Laminectomy	Improved
85	[68]	None	L3–L5	No	No	Laminectomy	CR
86	[68]	None	L3–S1	No	No	Laminectomy	Improved
87	[10]	None	T3–T10	No	No	Laminectomy	CR
88	[69]	None	C6–L3	No	No	Laminectomy	Improved
89	[70]	None	T4–T9	No	No	Laminectomy	CR
90	[71]	None	L5–S1	No	NR	Laminectomy	Improved
91	[19]	None	T6–T8	No	No	Laminectomy	Improved

(Continued)

Table 1
Continued

Case	Reference	Pathology	Levels	Steroids	Obesity	Treatment	Outcome
		Cushing disease				Surgical removal	
92	[9]	Pituitary microadenoma	C7–L1	No	NR	of pituitary tumor	Improved
		Adrenal tumor					
93	[8]	Cushing syndrome	C5–T2	No	No	Tumor removal	Improved
		Bronchial carcinoid				Medical: metyrapone,	
94	[3]	Cushing syndrome	T5–T10	No	NR	ketaconazole	Improved
95	[72]	Diabetic, paraplegic	T1–T9	No	Yes	Laminectomy	Improved
		Klippel-Trenaunay-Weber					
96	[73]	syndrome	Thoracic	No	NR	Laminectomy	Improved
		Obese posttraumatic cauda					
97	[74]	equina syndrome	Lumbar	No	Yes	Laminectomy	Improved
98	[75]	Cushing syndrome	Thoracic	No	NR	Medical management	Improved
		Cushing syndrome with					
		pituitary tumor				Surgical removal of	
99	[76]	(macroprolactinoma)	Thoracic	No	NR	pituitary tumor	Improved
100	[77]	Becker nevus associated	T4–T7	No	NR	Laminectomy	Improved
101	[78]	None	Lumbar	No			
102	[78]	None	Lumbar	No	Yes	Laminectomy	Improved
103	[78]	None	Lumbar	No	Yes	Laminectomy	Improved
104	[78]	None	Lumbar	No	Yes	Laminectomy	Improved

CR=complete recovery; FU=follow-up; LE=lower extremity; NR=not reported.

the second leading association with SEL. There is the persuasive argument that hypothyroidism is associated with generalized fat deposition resulting from decreased lipolysis, However, the data are insufficient to link hypothyroidism and SEL [3,4,10,11]. This linkage would require cases of nonobese patients with hypothyroidism and SEL to be considered an independent risk factor. The case of an obese patient with hypothyroidism and SEL can only be considered a case of obesity associated with SEL.

Imaging

Most case reports relied heavily on CT imaging and myelography [12,13]. However, MRI is now recognized as the most sensitive and specific modality for evaluating fatty tissue [14–16]. T1-weighted images differentiate epidural fat from dural content with a high degree of specificity and allow for measurement of adipose thickness. Quint et al. [17] conducted a study in which 28 normal patients were imaged and their epidural adipose measured. The mean sagittal thickness of their epidural fat was 4.6 mm with a normal range of 3 to 6 mm. In contrast, imaging in 6 patients with SEL revealed a mean thickness of 8 mm [17]. Borre et al. [18], on the lumbar MRIs of 2,258 patients, measured the anterior posterior diameters of the dural sac and spinal canal and the thickness of the epidural fat. Borre et al. developed an MRI classification based on these ratios. Grade 0 or normal was defined as epidural fat less than 40% of canal width, and dural 150% width of epidural fat. Grade I was defined as epidural fat less than 50% of the canal width and less than 50% of the dural width. Grade I was not symptomatic. Grade II was defined as the epidural fat 50% to 75% of the canal and 100% to 150% the width of the dural sac. Grade II SEL was symptomatic in 14% of cases. Grade III was defined as the epidural fat more than 75% of canal width and the dural 30% the width of the fat. All Grade III SEL cases were symptomatic. In Grade III cases, there was a 42% rate of associated substantial pathology, such as disc herniation or stenosis. Also in the Grade III SEL, the epidural fat produces centripetal pressures on the thecal sac changing the morphology or shape. Most commonly a trifid or Y shape may be seen. Borre et al. [18] report six other patterns seen in axial magnetic imaging.

Level of disease

Examining all available case reports, we were able to determine incidences of spinal level involvement. From the reported cases, 45.8% had thoracic involvement only, 43.6% had lumbosacral involvement only and 10.6% had involvement in both thoracic and the lumbosacral area. We correlated location of disease to the associated categories to ascertain whether any trends existed. Of the 52 patients with a history of steroid use, 55.8% were found to have thoracic involvement only, 32.7% with lumbosacral involvement only and 11.5% with both thoracic and lumbosacral involvement. Data from the patients in the obese category reveal a stronger trend: 69.6% had lumbosacral involvement only, and 30.4% had thoracic involvement only. The idiopathic group consisted of 16 patients. Of the 16 patients, 37.5% had thoracic spine involvement only, 50% had lumbosacral involvement only and 12.5% had involvement of both the lumbosacral and thoracic spine. Of the three patients with Cushing syndrome/disease from endogenous sources, 66.6%, had both thoracic and lumbosacral involvement and 33.3% had thoracic involvement only. Although four of the

cases reported cervical involvement, these cases had SEL throughout the spine and were included in the percentages of thoracic and lumbosacral involvement. We have found no cases of SEL isolated to the cervical region.

Investigators initially reported a significantly higher incidence of thoracic SEL. This was theorized to be secondary to the fact that the thoracic region has the largest amount of epidural fat [10,17,19]. Contrary to this popular belief, our case review indicates that SEL is found in approximately the same number of cases in both the thoracic and the lumbosacral region. The previous causal relationship is no longer present with this new information. It is clear that further studies into the etiology of SEL are required.

Treatment

Treatment of SEL ranges from conservative management to surgical excision. The success rates were calculated assuming that none of the patients in the surgical group were first treated conservatively. In the event that the reviewed data revealed a failed conservative treatment that then proceeded to surgery, these patients were included in both groups or were specifically discussed. In the steroid patient group, three of the cases lacked outcome data and are therefore considered to have no improvement for purposes of this analysis. Thirty-nine patients received a laminectomy and debulking, accounting for 75% of those patients in the steroid group (39 of 52). The success rate of this modality was 77% (31 of 39 patients receiving a laminectomy had results ranging from improved symptoms to complete recovery). The remaining 13 patients (25%) received a combination of different medical treatments, including weight loss, steroid taper, analgesics, bed rest and observation. One of the 13 received high-dose steroids for symptoms. The success rate of medical management of these patients was 77%. Borre et al. [18] reported that 26 patients were treated medically and 18 surgically. In the surgical group 16 of 18 had associated spinal pathology treated, such as disc herniation and stenosis decompression. Two patients were decompressed surgically with such a high ratio of epidural fat to canal and dural width and presented with neurologic compromise and cauda equina syndrome [18]. Although both modalities appeared to treat SEL successfully in approximately 75% of the cases, these data do not reveal criteria for selecting a treatment modality.

The obese patient group was split between surgical correction and weight loss treatment modalities: 52.2% were treated by laminectomy and debulking, 47.8% by weight loss. The success rate of the surgically managed group was 66.7%. The patients managed conservatively by weight loss improved in 81.8% of the cases, with the one patient lacking outcome data considered having no improvement. Two patients not included in the conservative group previously failed conservative treatment before surgical treatment was administered. One of the patients was managed by physiotherapy and nonsteroidal anti-inflammatory medication, not weight loss. The other patient failed to lose weight. Because conservative care was not successful, they were not included in that group. Based on these data, weight loss as a treatment modality appears to be very successful and should be considered the first line of treatment in this patient group, with surgical correction reserved for those patients who fail to respond clinically to a weight loss plan or are unable to lose weight.

One of the patients in the idiopathic group lacked outcome data and was therefore considered to have a poor outcome. All of the patients in this group were treated by laminectomy, with a success rate of 93.75%. The only patient without improvement lacked outcome data. Surgical treatment of patients with no discernable cause for SEL appears to be the treatment of choice.The three patients who were diagnosed with SEL associated with endogenous steroid excess or Cushing disease/syndrome were treated differently. Because all three of these patients had an underlying disorder causing endogenous steroid excess, the underlying disorder was treated. Two of the patients had surgical removal of the tumor causing the steroid excess, and the remaining patient was treated with ketaconazole, an inhibitor of steroidogenesis. All three of the patients improved.

Conclusions

Symptomatic SEL is a rare condition consisting of excess adipose tissue in the spinal canal causing compression of the spinal cord and resulting neurologic symptoms. Four categories have been identified as associated with SEL: exogenous steroid use, obesity, endogenous steroid excess and idiopathic. Thoracic and lumbosacral levels are usually affected by SEL, with the incidence between the two roughly equal. No case of isolated cervical involvement was found. A new MRI grading scale may help define those patients who will improve expectantly (Grade I) from those who may require surgical decompression (Grade III). Surgical treatment in the Grade III SEL may also treat commonly associated degenerative stenosis, and facet pathology. Obese patients tend to develop SEL in the lumbosacral region three times more often than in the thoracic, whereas steroid use tends to cause SEL in the thoracic region slightly less than twice as often as in the lumbosacral region. Obese patients should be managed by diet alone initially, with surgery reserved for those without a significant clinical response. Although SEL is a rare condition, our review of the literature reveals many more reported cases than previously thought. With increased awareness of this condition and improved imaging techniques, further studies of this disease should be undertaken.

References

[1] Lee M, Lekias J, Gubbay SS, Hurst PE. Spinal cord compression by extradural fat after renal transplantation. Med J Aust 1975;1(7):201–3.

[2] Koch CA, Doppman JL, Patronas NJ, Nieman LK, Chrousos GP. Do glucocorticoids cause spinal epidural lipomatosis? When endocrinology and spinal surgery meet. Trends Endocrinol Metab 2000;11(3): 86–90.
[3] Koch CA, Doppman JL, Watson JC, Patronas NJ, Nieman LK. Spinal epidural lipomatosis in a patient with the ectopic corticotropin syndrome. N Engl J Med 1999;341(18):1399–400.
[4] McCullen GM, Spurling GR, Webster JS. Epidural lipomatosis complicating lumbar steroid injections. J Spinal Disord 1999;12(6):526–9.
[5] Sandberg DI, Lavyne MH. Symptomatic spinal epidural lipomatosis after local epidural corticosteroid injections: case report. Neurosurgery 1999;45(1):162–5.
[6] Badami JP, Hinck VC. Symptomatic deposition of epidural fat in a morbidly obese woman. Am J Neuroradiol 1982;3(6):664–5.
[7] Doppman JL. Epidural lipomatosis. Radiology 1989;171(2):581–2.
[8] Noel P, Pepersack T, Vanbinst A, Alle JL. Spinal epidural lipomatosis in Cushing's syndrome secondary to an adrenal tumor. Neurology 1992;42(6):1250–1.
[9] Sivakumar K, Sheinart K, Lidov M, Cohen B. Symptomatic spinal epidural lipomatosis in a patient with Cushing's disease. Neurology 1995;45(12):2281–3.
[10] Selmi F, Davies KG, Sharma RR, Redfern RM. Idiopathic spinal extradural lipomatosis in a non-obese otherwise healthy man. Br J Neurosurg 1994;8(3):355–8.
[11] Toshniwal PK, Glick RP. Spinal epidural lipomatosis: report of a case secondary to hypothyroidism and review of literature. J Neurol 1987;234(3):172–6.
[12] Bednar DA, Esses SI, Kucharczyk W. Symptomatic lumbar epidural lipomatosis in a normal male. A unique case report. Spine 1990;15 (1):52–3.
[13] Hierholzer J, Benndorf G, Lehmann T, et al. Epidural lipomatosis: case report and literature review. Neuroradiology 1996;38(4):343–8.
[14] Hierholzer J, Vogl T, Hosten N, Lanksch W, Felix R. Imaging in epidural lipomatosis. Crit Rev Neurosurg 1998;8(5):279–81.
[15] Kumar K, Nath RK, Nair CP, Tchang SP. Symptomatic epidural lipomatosis secondary to obesity. Case report. J Neurosurg 1996;85(2): 348–50.
[16] Qasho R, Ramundo OE, Maraglino C, Lunardi P, Ricci G. Epidural lipomatosis with lumbar radiculopathy in one obese patient. Case report and review of the literature. Neurosurg Rev 1997;20(3):206–9.
[17] Quint DJ, Boulos RS, Sanders WP, et al. Epidural lipomatosis. Radiology 1988;169(2):485–90.
[18] Borre DG, Borre GE, Aude F, Palmieri GN. Lumbosacral epidural lipomatosis: MRI grading. Eur Radiol 2003;13(7):1709–21.
[19] Stern JD, Quint DJ, Sweasey TA, Hoff JT. Spinal epidural lipomatosis: two new idiopathic cases and a review of the literature. J Spinal Disord 1994;7(4):343–9.
[20] Butcher DL, Sahn SA. Epidural lipomatosis: a complication of corticosteroid therapy. Ann Intern Med 1979;90(1):60.
[21] Godeau P, Brunet P, Wechsler B, Fohanno D. [Compressive extradural lipomatosis as an unusual complication of corticosteroid therapy: one case (author's translation)]. Nouv Presse Med 1979;8(47):3889–91.
[22] Lipson SJ, Naheedy MH, Kaplan MM, Bienfang DC. Spinal stenosis caused by epidural lipomatosis in cushing's syndrome. N Engl J Med 1980;302(1):36.
[23] Chapman PH, Martuza RL, Poletti CE, Karchmer AW. Symptomatic spinal epidural lipomatosis associated with Cushing's syndrome. Neurosurgery 1981;8(6):724–7.
[24] Guegan Y, Fardoun R, Launois B, Pecker J. Spinal cord compression by extradural fat after prolonged corticosteroid therapy. J Neurosurg 1982;56(2):267–9.
[25] Archer CR, Smith KR. Extradural lipomatosis simulating an acute herniated nucleus pulposus. Case report. J Neurosurg 1982;57(4): 559–62.
[26] George WE, Wilmot M, Greenhouse A, Hammeke M. Medical management of steroid-induced epidural lipomatosis. N Engl J Med 1983; 308(6):316–9.
[27] Russell NA, Belanger G, Benoit BG, et al. Spinal epidural lipomatosis: a complication of glucocorticoid therapy. Can J Neurol Sci 1984;11(3): 383–6.
[28] Roy-Camille R, Mazel C, Husson JL, Saillant G. Symptomatic spinal epidural lipomatosis induced by a long-term steroid treatment. Review of the literature and report of two additional cases. Spine 1991;16(12): 1365–71.
[29] Randall BC, Muraki AS, Osborn RE, Brown F. Epidural lipomatosis with lumbar radiculopathy: CT appearance. J Comput Assist Tomogr 1986;10(6):1039–41.
[30] Jungreis CA, Cohen WA. Spinal cord compression induced by steroid therapy: CT findings. J Comput Assist Tomogr 1987;11(2):245–7.
[31] Haid RW, Kaufman HH, Schochet SS, Jr, Marano GD. Epidural lipomatosis simulating an epidural abscess: case report and literature review. Neurosurgery 1987;21(5):744–7.
[32] Zampella EJ, Duvall ER, Sekar BC, et al. Symptomatic spinal epidural lipomatosis as a complication of steroid immunosuppression in cardiac transplant patients. Report of two cases. J Neurosurg 1987;67(5): 760–4.
[33] Fessler RG, Johnson DL, Brown FD, et al. Epidural lipomatosis in steroid-treated patients. Spine 1992;17(2):183–8.
[34] Peeters FL, Koster PA. Epidural lipomatosis simulating spinal canal stenosis or herniated nucleus pulposus. Rofo Fortschr Geb Rontgenstr Neuen Bildgeb Verfahr 1991;154(3):342–3.
[35] Haddad SF, Hitchon PW, Godersky JC. Idiopathic and glucocorticoid-induced spinal epidural lipomatosis. J Neurosurg 1991;74(1):38–42.
[36] Healy ME, Hesselink JR, Ostrup RC, Alksne JF. Demonstration by magnetic resonance of symptomatic spinal epidural lipomatosis. Neurosurgery 1987;21(3):414–5.
[37] Soloniuk DS, Pecoraro SR, Munschauer FE. Myelopathy secondary to spinal epidural lipomatosis. A case report. Spine 1989;14(1):119–22.
[38] Maehara T, Tanohata K, Noda M, Nakayama D. Medically treated steroid-induced epidural lipomatosis. Neuroradiology 1988;30(3):281.
[39] Kaplan JG, Barasch E, Hirschfeld A, et al. Spinal epidural lipomatosis: a serious complication of iatrogenic Cushing's syndrome. Neurology 1989;39(8):1031–4.
[40] Tobler WD, Weil S. Epidural lipomatosis and renal transplantation. Surg Neurol 1988;29(2):141–4.
[41] Vazquez L, Ellis A, Saint-Genez D, Patino J, Nogues M. Epidural lipomatosis after renal transplantation—complete recovery without surgery. Transplantation 1988;46(5):773–4.
[42] Buthiau D, Piette JC, Ducerveau MN, et al. Steroid-induced spinal epidural lipomatosis: CT survey. J Comput Assist Tomogr 1988;12(3): 501–3.
[43] Pennisi AK, Meisler WJ, Dina TS. Lymphomatous meningitis and steroid-induced epidural lipomatosis: CT evaluation. J Comput Assist Tomogr 1985;9(3):595–8.
[44] Taborn J. Epidural lipomatosis as a cause of spinal cord compression in polymyalgia rheumatica. J Rheumatol 1991;18(2):286–8.
[45] Perling LH, Laurent JP, Cheek WR. Epidural hibernoma as a complication of corticosteroid treatment. Case report. J Neurosurg 1988;69 (4):613–6.
[46] Andress HJ, Schurmann M, Heuck A, Schmand J, Lob G. A rare case of osteoporotic spine fracture associated with epidural lipomatosis causing paraplegia following long-term cortisone therapy. Arch Orthop Trauma Surg 2000;120(7-8):484–6.
[47] Pinsker MO, Kinzel D, Lumenta CB. Epidural thoracic lipomatosis induced by long-term steroid treatment case illustration. Acta Neurochir (Wien) 1998;140(9):991–2.
[48] Crayton HE, Partington CR, Bell CL. Spinal cord compression by epidural lipomatosis in a patient with systemic lupus erythematosus. Arthritis Rheum 1992;35(4):482–4.
[49] Arroyo IL, Barron KS, Brewer EJ. Spinal cord compression by epidural lipomatosis in juvenile rheumatoid arthritis. Arthritis Rheum 1988;31(3):447–51.
[50] Fiirgaard B, Madsen FH. Spinal epidural lipomatosis. Case report and review of the literature. Scand J Med Sci Sports 1997;7(6):354–7.

[51] Ebright JR, Stellini MA, Tselis AC. Spinal epidural lipomatosis in a human immunodeficiency virus-positive patient receiving steroids and protease inhibitor therapy. Clin Infect Dis 2001;32(5):E90–1.
[52] Gero BT, Chynn KY. Symptomatic spinal epidural lipomatosis without exogenous steroid intake. Report of a case with magnetic resonance imaging. Neuroradiology 1989;31(2):190–2.
[53] Beges C, Rousselin B, Chevrot A, et al. Epidural lipomatosis. Interest of magnetic resonance imaging in a weight-reduction treated case. Spine 1994;19(2):251–4.
[54] Stambough JL, Cheeks ML, Keiper GL. Nonglucocorticoid-induced lumbar epidural lipomatosis: a case report and review of literature. J Spinal Disord 1989;2(3):201–7.
[55] Berenbaum F, Revel M, Deshays C, Rousselin B, Amor B. [Lumboradicular pain caused by epidural lipomatosis in an obese patient: recovery after hypocaloric diet]. Rev Rhum Mal Osteoartic 1992; 59(3):225–7.
[56] Pouchot J, Si-Hassen C, Damade R, et al. Cauda equina compression by epidural lipomatosis in obesity. Effectiveness of weight reduction. J Rheumatol 1995;22(9):1771–5.
[57] Grayling M, Jardine DL, McClintock AD, Spar J, Wilton GN. Symptomatic epidural lipomatosis following cyproterone acetate therapy. Aust N Z J Surg 2000;70(3):233–5.
[58] Boutsen Y, Donckier J. Epidural lipomatosis. Postgrad Med J 2000; 76(891):60–1.
[59] van Rooij WJ, Borstlap AC, Canta LR, Tijssen CC. Lumbar epidural lipomatosis causing neurogenic claudication in two obese patients. Clin Neurol Neurosurg 1994;96(2):181–4.
[60] Borstlap AC, van Rooij WJ, Sluzewski M, Leyten AC, Beute G. Reversibility of lumbar epidural lipomatosis in obese patients after weight-reduction diet. Neuroradiology 1995;37(8):670–3.
[61] Millwater CJ, Jacobson I, Howard GC. Idiopathic epidural lipomatosis as a cause of pain and neurological symptoms attributed initially to radiation damage. Clin Oncol (R Coll Radiol) 1992;4(5):333–4.
[62] Sundram SR. CT of epidural lipomatosis: a case report. Radiogr Today 1990;56(632):26–8.
[63] Iplikcioglu AC, Berkman MZ, Sengoz A. Idiopathic spinal epidural lipomatosis. Acta Neurochir (Wien) 1998;140(4):405–6.
[64] Citow JS, Kranzler L. Thoracic epidural lipomatosis with associated syrinx: case report. Surg Neurol 2000;53(6):589–91.
[65] Kurt E, Bakker-Niezen SH. Neurogenic claudication by epidural lipomatosis: a case report and review of literature. Clin Neurol Neurosurg 1995;97(4):354–7.
[66] Frank E. Endoscopic suction decompression of idiopathic epidural lipomatosis. Surg Neurol 1998;50(4):333–5.
[67] Huraibi HA, Phillips J, Rose RJ, et al. Intrathecal baclofen pump implantation complicated by epidural lipomatosis. Anesth Analg 2000;91(2):429–31.
[68] Lisai P, Doria C, Crissantu L, et al. Cauda equina syndrome secondary to idiopathic spinal epidural lipomatosis. Spine 2001;26(3):307–9.
[69] Hogg JP, Shank T, Gingold M, et al. Childhood presentation of idiopathic epidural lipomatosis: a case report with magnetic resonance imaging and pathologic confirmation. J Child Neurol 1996;11(3): 236–40.
[70] Hughes JP, Jones EW. Case of the month: inside a thin man—an engrossing problem. Br J Radiol 1995;68(806):213–4.
[71] Supik LF, Broom MJ. Epidural lipoma causing a myelographic block in a patient who had sciatica and lumbosacral spondylolisthesis. A case report. J Bone Joint Surg Am 1991;73(7):1104–7.
[72] Draganescu M, Daka M, Manon-Espaillat R. Epidural lipomatosis. Neurology 2002;58(9):1387.
[73] Ashkan K, Moore AJ. Spinal cord compression caused by an extradural lipoma in Klippel-Trenaunay-Weber syndrome. Case illustration. J Neurosurg 2002;97(2 suppl):269.
[74] Schizas C, Ballesteros C, Roy P. Cauda equina compression after trauma: an unusual presentation of spinal epidural lipoma. Spine 2003;28(8):E148–51.
[75] Dumont-Fischer D, Rat AC, Saidenberg-Kermanac'h N, et al. Spinal epidural lipomatosis revealing endogenous Cushing's syndrome. Joint Bone Spine 2002;69(2):222–5.
[76] Fujisawa H, Hasegawa M, Tachibana O, Yamashita J. Spinal epidural lipomatosis associated with pituitary macroprolactinoma. Acta Neurochir (Wien) 2002;144(2):213–4.
[77] Kawai M, Udaka F, Nishioka K, et al. A case of idiopathic spinal epidural lipomatosis presented with radicular pain caused by compression with enlarged veins surrounding nerve roots. Acta Neurol Scand 2002;105(4):322–5.
[78] Payer M, Van Schaeybroeck P, Reverdin A, May D. Idiopathic symptomatic epidural lipomatosis of the lumbar spine. Acta Neurochir (Wien) 2003;145(4):315–21.