Symptomatic Idiopathic Spinal Epidural Lipomatosis in 9 Patients: Clinical, Radiologic, and Pathogenetic Features

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■ BACKGROUND: **Symptomatic spinal epidural lipomatosis (SSEL) is characterized by hypertrophy of adipose tissue within the spinal canal and consequent neural compromise. The exact pathogenesis remains enigmatic. The authors describe a retrospective case series, define the full clinical spectrum, and discuss possible pathogenetic mechanisms.**

■ METHODS: **The medical notes and imaging of 9 patients with SSEL undergoing surgery from 2008–2018 were analyzed. Seven patients presented secondary to lumbosacral spinal epidural lipomatosis (SEL); 3 patients with chronic incomplete cauda equina syndrome (CES), 3 patients with acute CES (including a 25-week gravid patient and a 40-year-old patient with intravenous leiomyomatosis, both of whom had mild SEL) and 1 patient with chronic lumbar radiculopathy. In addition, 2 patients presented with progressive myelopathy secondary to thoracic SEL.**

■ RESULTS: **Patients presenting with acute CES had a mean age of 37 years (range 23–49 years) and mean extradural fat (EF)-to–spinal canal (SC) ratio of 47% (range 41%–58%), in comparison with patients with chronic CES; mean age 61 years (range 58–65 years) and EF:SC ratio 72% (range 65%–80%). Patients underwent laminectomy and resection of EF at compressive levels. All patients with CES experienced complete resolution of symptoms at follow-up (range 1–48 months).**

■ CONCLUSIONS: **The clinician should be astute to the radiologic features of SEL, particularly in patients presenting with CES in the absence of acute disk herniation. The outcome of patients with CES and SEL after surgery is excellent regardless of symptom duration. Venous impedance related to increased body mass index and EF deposition may play the predominant role in addition to mechanical compression in the pathogenesis of SSEL.**

INTRODUCTION

Historically, symptomatic spinal epidural lipomatosis (SSEL) has been reported to be a rare condition characterized by hypertrophy of unencapsulated adipose tissue within the spinal canal with consequent neural compromise. Current literature suggests that spinal epidural lipomatosis (SEL) has a higher prevalence than initially thought with idiopathic cases contributing a significant proportion of these cases.[1] The exact pathogenesis resulting in neurologic sequelae remains enigmatic. The authors describe a retrospective case series of 9 patients with SSEL presenting to 2 neurosurgical centers in London and define the full clinical spectrum of SSEL. In particular, a 23-year-old, 25-week gravid patient and 40-year-old patient with recurrent intravenous leiomyomatosis presented with acute cauda equina syndrome (CES). Both patients had venous abnormalities on imaging and may offer pathogenetic insight into SSEL.

MATERIALS AND METHODS

Materials

The medical notes of all patients undergoing surgery secondary to SEL from 2008–2018 were analyzed. Patient details were accessed following a search of respective operative databases. Patient

Key words
- Cauda equina
- Epidural lipomatosis
- Venous insufficiency

Abbreviations and Acronyms
BMI: Body mass index
CES: Cauda equina syndrome
IAP: Intraabdominal pressure
SC: Spinal canal
SEL: Spinal epidural lipomatosis
SSEL: Symptomatic spinal epidural lipomatosis

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demographics, presenting symptoms, co-morbidities (including risk factors for SEL), surgical details (including complications), radiographic findings, postoperative course, and follow-up assessment are presented. Preoperative magnetic resonance imaging is described, and the radiologic severity of lumbar SEL is graded.

Patient Population and Presenting Features

Six males and 3 females aged 23–67 (mean 52 years) presented with SSEL from 2010–2018. Six patients were documented as having an increased body mass index (BMI, including 1 patient with a BMI of 40 kg/m^2), and 3 patients were known to have noninsulin-dependent diabetes mellitus. None of the patients had a history of exogenous steroid therapy or had undergone previous spinal epidural injections.

Seven patients presented with neurologic symptoms secondary to lumbosacral SEL: 3 patients with chronic incomplete CES, 3 patients with acute CES (including 1 patient in the second trimester of pregnancy and 1 patient with recurrent intravenous leiomyomatosis), and 1 patient with a chronic lumbar radiculopathy. The average duration of symptoms in patients presenting with incomplete CES was 14 months (range 6–24 months), and all patients with acute CES presented within 24 hours of the onset of urinary symptoms. In addition, 2 patients presented with progressive myelopathy secondary to thoracic SEL and both patients presented with falls and paraparesis over several months (**Table 1**).

Imaging and Surgical Management

All patients had preoperative magnetic resonance imaging and were graded according to the study by Borre et al[2] on the basis of the degree of epidural fat occupying the lumbosacral canal (extradural fat [EF]-to–spinal canal [SC] ratio); grade 1, 41%–50% of the canal; grade 2, 51%–74% of the canal; and grade 3, >75% of the canal. In all lumbosacral cases abnormal fat deposition was observed below the level of L2 dorsal to the theca. Caudal to L5 the distribution of epidural fat encircles the theca, as the theca naturally tapers to form the coccygeal ligament, with varying degrees of compression. In both thoracic cases there was a continuous pattern of epidural fat almost the entire length of the thoracic spine dorsal to the spinal cord. In Patient 5 the classic "Y-sign" associated with SEL is seen secondary to significant compression of the thecal sac at the lumbosacral junction (**Figure 1**).

RESULTS

The average follow-up was 27 months (range 1–48 months). Patients presenting with acute CES had a mean age of 37 years (range 23–49 years) and mean EF-to-SC ratio of 47% (range 41%–58%), in comparison with patients presenting with chronic CES. The mean age was 61 years (range 58–65 years), and the mean EF-to-SC ratio was 72% (range 65%–80%). All patients underwent laminectomy and excision of epidural fat at the levels most severely compressed according to preoperative magnetic resonance studies. Inadvertent durotomy occurred in 4 patients with lumbar SEL. All 4 cases underwent primary closure without further sequelae. Patient 1 underwent further surgery for ongoing sciatica secondary to coexisting degenerative lateral recess stenosis at 6 months and re-presented at 8 months with wound infection requiring wound washout.

All patients presenting with CES experienced an immediate improvement in autonomic symptoms postoperatively and complete resolution at last follow-up. Patient 1 experienced initial improvement in radicular leg pain despite recurrence of pain requiring reoperation as described earlier. Patient 8 had been bedbound for 4 weeks before admission with Medical Research Council scale 3/5 paraparesis. There was immediate improvement in lower limb strength postoperatively, and at discharge he was able to take several steps with a walking aid. Patient 9 has shown no improvement and remains wheelchair bound. The clinical picture is complicated by concomitant severe chronic demyelinating polyneuropathy (see **Table 1** and **Figure 2**). Immediate postoperative imaging was not performed routinely given the majority of patients reported immediate clinical improvement.

Illustrative Case 1

A 23-year-old, 25-week gravid patient (Patient 7) of moderately increased BMI and without significant past medical history presented to neurosurgical services with a 2-week history of bilateral sciatica, 1-day history of unilateral saddle sensory disturbance, and urinary incontinence. There was a preceding 2-year history of lumbar back pain and left sciatica. Imaging revealed focal (caudal to the L5/S1 disk space) grade 1 (41% EF/SC ratio) lumbosacral epidural lipomatosis in the absence of significant lumbar degenerative disease or acute disk herniation (**Figures 3** and **4**).

After discussing conservative versus surgical management and obstetric consult, the patient elected for surgical decompression. The patient was positioned prone on a Montreal mattress with additional support ensuring the abdomen was elevated and hanging freely. L5-S1 laminectomy and excision of a dense adipose plaque dorsal to the theca were performed without maternal or fetal complication. Postoperatively the patient reported immediate resolution of sciatica, saddle sensory disturbance, and urinary symptoms. The patient was discharged at day 3, and there has been no recurrence of symptoms at follow-up.

Illustrative Case 2

A 40-year-old female patient with recurrent intravenous leiomyomatosis presented to neurosurgical services with a 2-week history of lumbar back pain, right-sided sciatica, and 1-day history of urinary incontinence and saddle sensory disturbance. She had previously undergone hysterectomy, cardiopulmonary bypass, cavotomy, and atriotomy for extensive intravascular leiomyomatosis. Follow-up imaging under gynecology had revealed a recurrent 74 × 57 × 57 mm pelvic mass with intravenous involvement of the inferior vena cava (superiorly extending to the left renal vein) and both common iliac veins (**Figure 5**). She was on the waiting list for repeat surgery at the time of presentation.

Magnetic resonance imaging of the lumbar spine excluded an acute disk herniation; however, it did reveal prominent EF caudal to the lumbosacral junction, predominantly ventral to the thecal sac. Axial imaging in addition to epidural lipomatosis

Table 1. Patient Demographics

Patient	Age	Sex	Presentation/Duration of Symptoms Before Surgery	Comorbidities	Operation	Complications	Postoperative Course	Follow-Up, Months	Symptoms at Follow-Up	Borre Grade (EF:SC Ratio)
1	54	M	3/52 LBP and bilateral sciatica, leg weakness, and numbness	CKD, multiple DVTs	L2-5 Laminectomy	Dural tear—primary repair	Uncomplicated	30	Complete resolution of CE sx	Grade 2 (67%)
2	61	M	6/12 Longstanding claudicant sx, mild urinary symptoms and numbness	Increased BMI	L4-S1 Laminectomy	Nil	Uncomplicated	48	Initial improvement, recurrent sx at 6/12 and infection at 8/12	Grade 2 (72%)
3	65	M	1-year claudication progressive numbness and urinary sx	HTN, Hypercholesterolemia, increased BMI	L2-S1 Laminectomy	Dural tear—primary repair	Uncomplicated	6	Back pain. No numbness, CE sx improved	Grade 3 (80%)
4	58	M	2 years' claudication urinary sx and numbness	HTN, NIDDM, increased BMI	L2-S2 Laminectomy	Dural tear—primary repair	Uncomplicated	6	Complete resolution of sx	Grade 2 (65%)
5	49	F	2/52 hx of CES, 1/7 hx urinary retention. Longstanding claudicant sx	Increased BMI, HTN, IHD, hypothyroid	L4 -S2 Laminectomy	Dural tear—primary repair	Uncomplicated	1	Complete resolution of CE sx	Grade 2 (58%)
6	40	F	2/52 history of lower back pain, right sciatica and 1/7 urinary incontinence	Increased BMI, Recurrent Leiomyomatosis	L5- S1 Laminectomy	Nil	Uncomplicated	2	Complete resolution of CES sx	Grade 1 (43%)
7	23	F	2/52 Longstanding back pain and b/l sciatica. 1/7 urinary sx and perianal numbness. 25 weeks pregnant	Nil, increased BMI	L5- S1 Laminectomy	Nil	Uncomplicated	2	Complete resolution of CE sx	Grade 1 (41%)
8	67	M	6 month hx of falls, paraperesis and T7 sensory level	IHD, DM2, AF, Peripheral neuropathy, DISH,	T2 - 9 Laminectomy	Nil	Uncomplicated	2	Improvement in sensation and leg power, now walking	N/a
9	53	M	12 month hx of progressive thoracic myelopathy and falls, paraparesis MRC 2/5, thoracic sensory level and sphincter dysfunction.	NIDDM, hyperparathyroidism, hypercholesterolemia	T1-T12 Laminoplasty	Nil	Uncomplicated	3	Paraplegic, urinary and faecal incontinence	N/a

LBP, lower back pain; CKD, chronic kidney disease; DVT, deep vein thrombosis; BMI, body mass index; HTN, hypertension; NIDDM, non–insulin-dependent diabetes mellitus; IHD, ischemic heart disease; DM2, non–insulin-dependent diabetes; AF, atrial fibrillation; DISH, diffuse idiopathic skeletal hyperostosis.

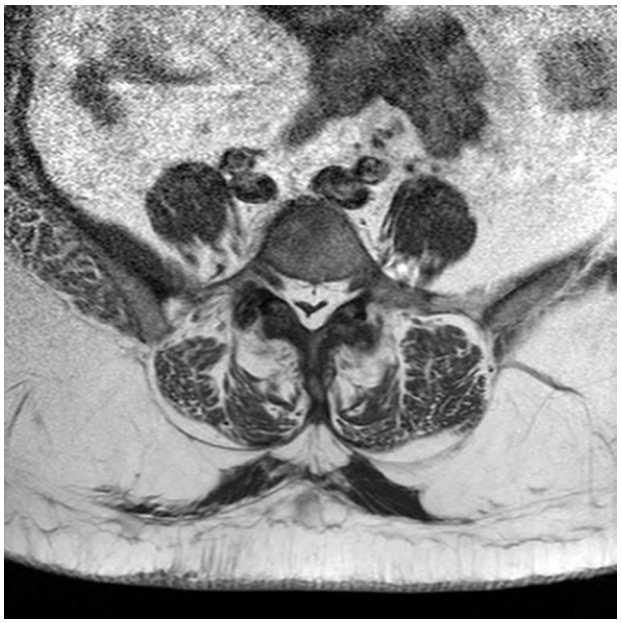

Figure 1. T1 axial Patient 5: classic "Y-sign" thecal compression secondary to epidural lipomatosis.

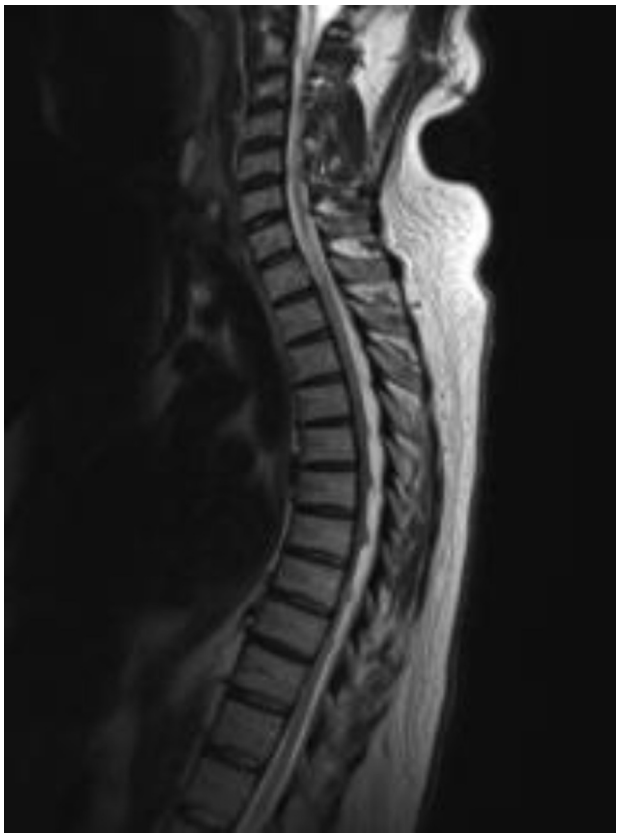

Figure 2. T2 sagittal Patient 9: thoracic epidural lipomatosis and cord compression.

demonstrated intravascular leiomyomatosis involving the common iliac vessels and segmental lumbar veins (Figure 6).

After discussing the imaging findings, as well as conservative versus surgical management, the patient opted for surgery. An uncomplicated L5 and S1 laminectomy was performed. Minimal dorsal lipomatosis and a prominent bulging thecal sac were noted intraoperatively (presumably secondary to ventral lipomatosis). Postoperatively, the patient experienced complete resolution of CES and radicular leg pain. She was discharged at day 2 without complication. There has been no recurrence of symptoms at last follow-up.

DISCUSSION AND LITERATURE REVIEW

EF cushions the pulsatile movements of the thecal sac, protects nerve structures, and facilitates the movement of the dura over the periosteum of the spinal canal during flexion and extension. Spinal epidural lipomatosis results from hypertrophy of normal unencapsulated adipose tissue within the spinal canal and can compress adjacent neural structures, resulting in neurologic symptoms.

SEL has a male preponderance and occurs most commonly in middle-aged and elderly adults. It affects the lumbosacral spine most commonly followed by the thoracic spine, occurring in isolation or coexisting with lumbar SEL, and cervical involvement is rare.[3] There is possible genetic susceptibility with SEL reported more commonly in Afro-Caribbean and Korean populations.[4] The vast majority of SEL cases are asymptomatic. The radiologic study by Borre et al[2] of 2528 patients with lumbosacral SEL concluded that all cases of SSEL occur as a result of grade 3 SEL (EF-to-SC ratio >75%) and this group accounted for only 2.1% of all cases.

The association of SEL and long-term systemic steroids, epidural injections, Cushing disease, and obesity has been extensively reported. The first reported case of SSEL was described in association with long-term steroid immunosuppression in a renal transplant patient.[5] The association with renal disease is related to long-term systemic steroid use. More recent literature recognizes the association with metabolic syndrome and highly active retroviral therapy–related lipodystrophy.[6,7] Idiopathic SEL denotes patients without a history of systemic steroid therapy or endocrinopathy; however, the vast majority of idiopathic cases have an increased BMI.

The prevalence of SSEL is greater than initially thought with the current literature suggesting a rate between 6.2% and 8.6%.[8] This may represent a combination of increased imaging, rising obesity, and an aging population. The House of Commons obesity statistics briefing 2018 reported 25% of the UK population is affected by obesity, an increase from 15% in 1995 with obesity levels highest among those aged 45–74. Valcarenghi et al[9] also reported the case of a 48-year-old obese man with a history of

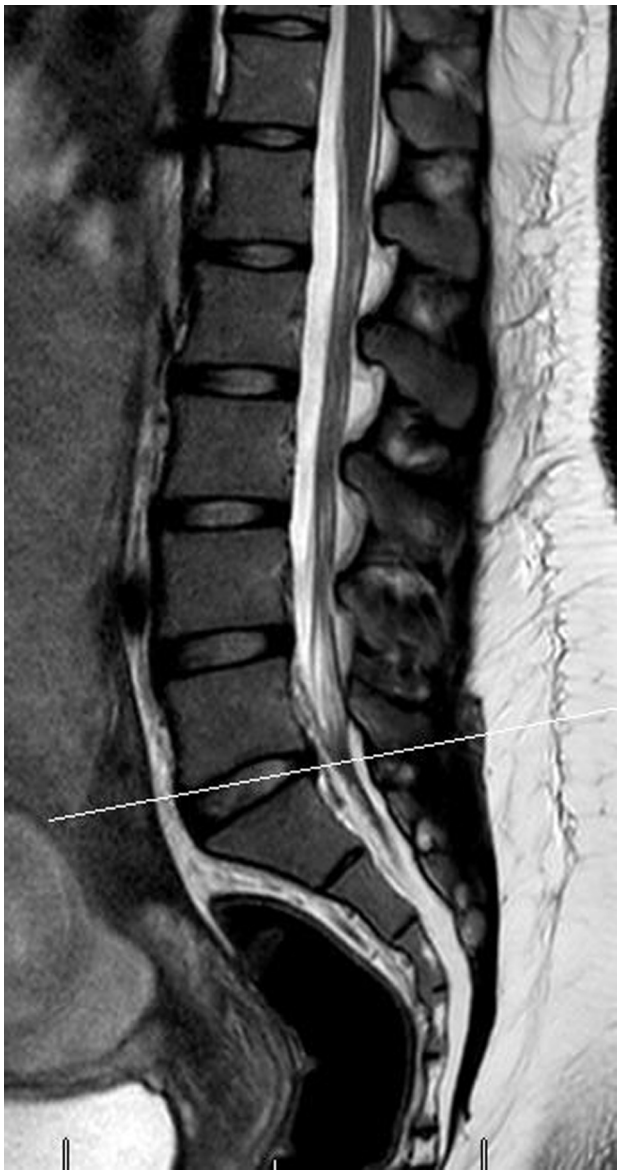

Figure 3. T2 sagittal and axial Patient 7: focal epidural lipomatosis caudal to L5 inferior end plate and thecal compression.

chronic back pain and sciatica secondary to SEL, which subsequently resolved completely after sleeve gastroplasty over a 6-month follow-up period.

The full clinical spectrum of SSEL has not been clearly described. The most common presentation is one of chronic neurogenic claudication or lumbar radiculopathy associated with lumbar back pain. A literature search excluding coexisting spinal pathology identifies a number of authors reporting SEL associated with chronic incomplete CES.[10-12] With reference to this literature, the mean age range at presentation was 72 years (range 60–84 years) and the duration of symptoms ranged from 3–24 months.

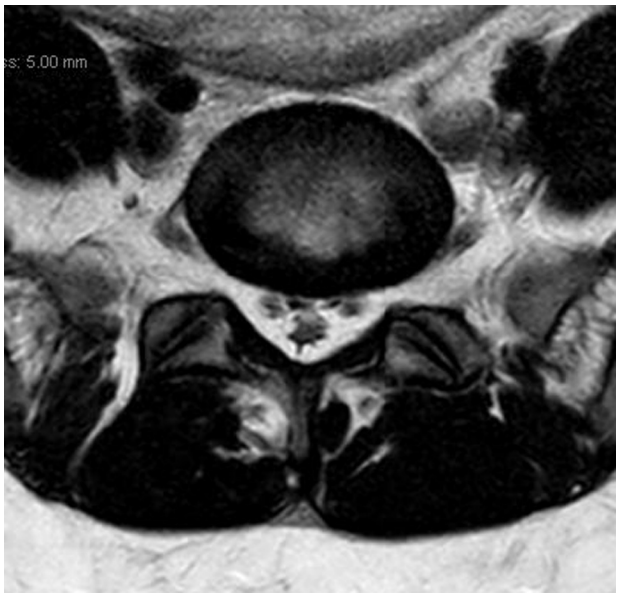

Figure 4. T2 axial imaging of Patient 7 showing moderate thecal compression and prominent anterior internal venous plexus.

There is only 1 reported case of rapidly progressive CES over a 2-week period in a 55-year-old male.[8]

Patients 5, 6, and 7 presented with acute CES. The mean age was 37 years (range 23–49 years), and all 3 cases presented with a

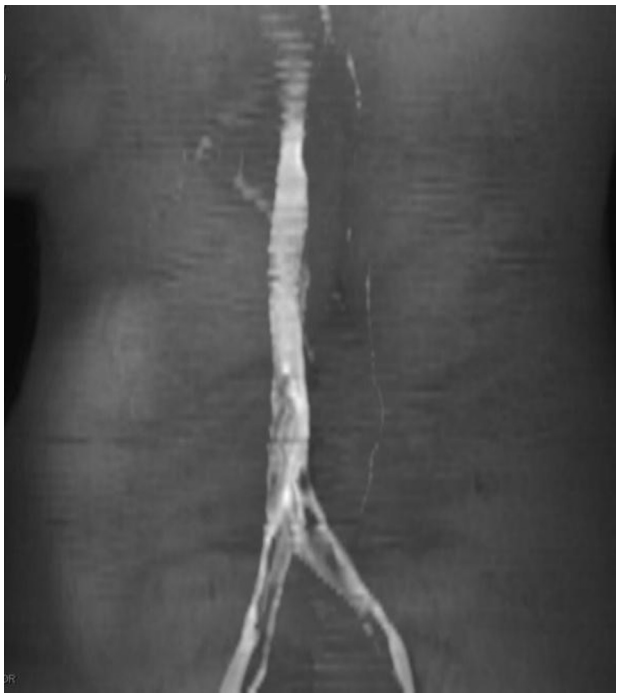

Figure 5. Magnetic resonance time-of-flight cavogram Patient 6: filling defects seen within the inferior vena cava and both common iliac veins.

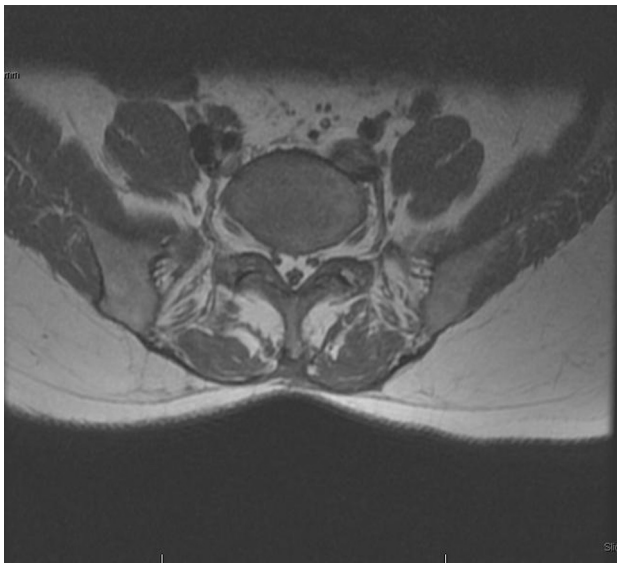

Figure 6. T1 axial Patient 6: mild epidural lipomatosis, intravascular leiomyomatosis involving the common iliac vessels, and prominent lumbar segmental veins.

24-hour history of incomplete CES. They represent a younger subpopulation presenting with acute incomplete CES distinct from the current literature. Also, the mean EF-to-SC ratio in this group was 47% (range 41%–58%), in comparison with patients presenting with chronic CES. Mean age at presentation was 61 years (range 58–65 years), and the mean EF:SC ratio was 72% (range 65%–80%).

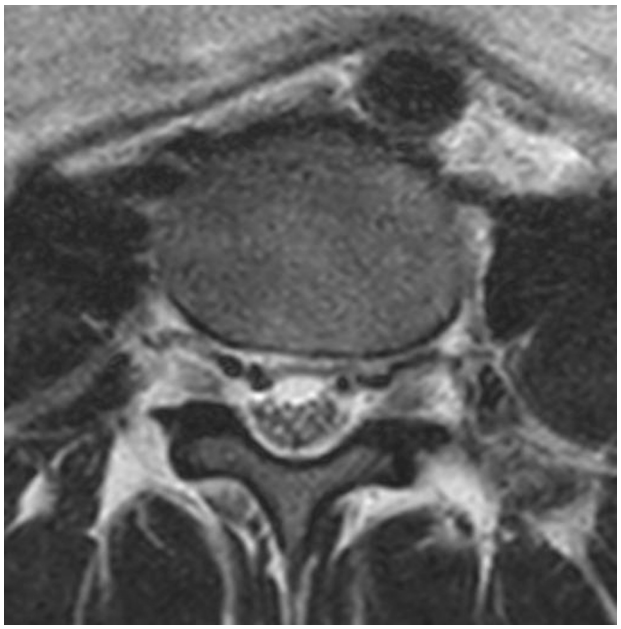

Figure 7. T2 axial patient 7: distended anterior longitudinal venous plexi and intervertebral veins.

Most interestingly, Patient 7 was 25 weeks pregnant and had the lowest EF-to-SC ratio within the series. This is the first reported case of a gravid patient presenting with acute CES secondary to SEL. Closer inspection of the imaging of Patient 7 revealed engorged epidural venous tributaries (**Figure 7**). Also of interest, Patient 6 had extensive central venous disease and prominent segmental veins in addition to a low EF-to-SC ratio (see **Figure 6**).

Previous authors have hypothesized that neurologic symptoms result from a combination of mechanical compression from hypertrophied EF and venous insufficiency.[13,14] Idiopathic SEL is strongly associated with obesity. Ishihara et al[15] demonstrated a positive correlation among EF accumulation, BMI, abdominal circumference, and visceral fat. Several authors have also reported the positive correlation between BMI and intraabdominal pressure (IAP).[16-18] In addition, pathologic BMI has been found to be associated with a significant positive pressure gradient between the thoracic and abdominal vena cava.[19]

Fuchs et al[20] measured mean IAP in pregnancy via intravesicular manometry before and after caesarean delivery; preoperative IAP was significantly greater than postoperative values. Also, obese term patients were found to have significantly raised IAP values in comparison with nonobese term patients. The authors hypothesize that in the case of Patient 7, the combination of a gravid uterus and increased adipose deposition (associated with pregnancy) resulted in raised IAP, venous insufficiency, and acute decompensation in the context of SSEL.

Patients 5, 6, and 7 contrast to the findings of Borre's[2] radiologic study in which all patients with grade 1 and 2 SEL were asymptomatic. The authors therefore hypothesize that venous insufficiency is the predominant mechanism resulting in acute decompensation and CES in both Patients 6 and 7. In the case of Patient 5, a pathologically raised BMI and IAP in the context of grade 2 SEL could be a possible explanation for decompensation. The authors consider whether symptomatic idiopathic SEL is the spinal equivalent of idiopathic intracranial hypotension. Are Patients 6 and 7 extreme examples of a pathologic model generalizable to patients with idiopathic SSEL who have significantly raised BMI and IAP resulting in venous insufficiency?

In addition to venous insufficiency, as is the case with degenerative lumbar canal stenosis, concentric compression of the cauda equina by dense hypertrophic epidural fat may result in decreased CSF supply to neural tissues, impaired nutritional supply, and accumulation of noxious substances. This in turn results in microcirculatory changes and edema, setting in motion a self-perpetuating cycle exacerbated by venous insufficiency. The increased neural metabolic demand during exercise may explain why claudication is the most common presenting syndrome associated with SEL.[21]

Thoracic SEL is extremely rare; generally, patients who present at a younger age are more frequently symptomatic. It is less often associated with obesity when compared with lumbosacral SEL. Al-Khawaja et al[4] report that 75% of cases of thoracic SEL are secondary to systemic steroid therapy or Cushing disease. The most common presenting symptom is thoracic back pain followed by a chronic but progressive paraparesis. Occasionally patients may present with acute paraplegia presumably related to vascular insult.[4] Both Patients 7 and 8 had mild and

moderately compressed cords, respectively, but presented with profound and progressive myelopathy. This may be related to the relatively high cord-to-canal ratio within the thoracic spine and its relatively tenuous blood supply.

Management of Symptomatic Spinal Epidural Lipomatosis

The management of SSEL includes conservative and surgical management. Conservative options should be exhausted in patients with secondary SEL and elevated BMI (tapering of steroid use and weight loss). Endocrinopathy-related cases (predominantly Cushing disease and hypothyroidism) should be referred to an endocrinologist before surgical consideration. Despite the long duration of autonomic dysfunction in the majority of patients presenting with incomplete CES, autonomic function normalizes after decompression. It is unclear whether conservative management would offer similar outcomes in this subset of patients.

Surgical decompression through laminectomy or laminoplasty (in pediatric cases) with excision of the hypertrophic adipose plaque is considered the mainstay of treatment in patients presenting with progressive neurologic deficit. Removal of the dorsal portion of the fatty plaque is sufficient to ensure adequate decompression (even in the lumbosacral spine where the theca becomes encircled by epidural fat). Care should be taken to develop a plane between the dorsal theca and fatty plaque as the theca is elevated and flattened by ventral epidural fat increasing the risk of inadvertent durotomy and cerebrospinal fluid leak. Engorged veins may also be encountered more laterally in the intervertebral foramen and ventral to the thecal sac. If epidural bleeding is encountered, this is easily managed with gentle pressure and bipolar and hemostatic agents.

Patients 5, 6, and 7 presented with an acute incomplete CES. Given the rapidity of symptom progression, urgent surgical decompression was favored and performed safely. The outcomes after surgery in this case series including patients presenting with CES are excellent and align with the study by Ferlic et al[22] concerning patient-reported outcomes in SEL.

Limitations

SSEL is still a rare condition; accordingly, the numbers involved in this study are such that any conclusions drawn should be interpreted with some caution. It is acknowledged that epidural venous engorgement (a sign of venous hypertension) is not always observed in cases of SSEL, but does this exclude spinal venous hypertension at the level of the cauda equina? Could a high EF-to-SC ratio in grade 3 patients mask epidural venous congestion? The authors also acknowledge that cases 6 and 7 could represent 2 independent processes resulting in CES, and the finding of SEL was incidental; however, the intraoperative findings and response following decompression would suggest otherwise.

CONCLUSION

The prevalence of obesity is increasing, and therefore the clinician should be mindful of SEL when reviewing imaging that at first glance appears radiologically benign, particularly in patients with raised BMI and CES. Surgical decompression in SSEL offers excellent results including patients presenting with CES regardless of symptom duration. This case series includes evidence that suggests venous insufficiency is a key pathologic mechanism in SSEL in addition to mechanical compression.

REFERENCES

1. Malone JB, Bevan PJ, Lewis TJ, Nelson AD, Blaty DE, Kahan ME. Incidence of spinal epidural lipomatosis in patients with spinal stenosis. *J Orthop*. 2017;15:36-39.
2. Borré DG, Borré GE, Aude F, Palmieri GN. Lumbosacral epidural lipomatosis: MRI grading. *Eur Radiol*. 2003;13:1709-1721.
3. Papastefan ST, Bhimani AD, Denyer S, et al. Management of idiopathic spinal epidural lipomatosis: a case report and review of the literature. *Childs Nerv Syst*. 2018;34:757-763.
4. Al-khawaja D, Seex K, Eslick GD. Spinal epidural lipomatosis—a brief review. *J Clin Neurosci*. 2008;15:1323-1326.
5. Lee M, Lekias J, Gubbay SS, Hurst PE. Spinal cord compression by extradural fat after renal transplantation. *Med J Aust*. 1975;1:201-203.
6. Morishita S, Arai Y, Yoshii T, Sakai K, Hirai T, Okawa A. Lumbar epidural lipomatosis is associated with visceral fat and metabolic disorders. *Eur Spine J*. 2018;27:1653-1661.
7. Cersósimo MG, Lasala B, Folgar S, Micheli F. Epidural lipomatosis secondary to indinavir in an HIV-positive patient. *Clin Neuropharmacol*. 2002;25:51-54.
8. Wells AJ, McDonald MJ, Sandler SJI, Vrodos NJ. Lumbosacral epidural lipomatosis causing rapid onset cauda equina syndrome. *J Clin Neurosci*. 2014;21:1262-1263.
9. Valcarenghi J, Bath O, Boghal H, Ruelle M, Lambert J. Benefits of bariatric surgery on spinal epidural lipomatosis: case report and literature review. *Eur J Orthop Surg Traumatol*. 2018;28:1437-1440.
10. Mariotto S, Bianchi MR, Ferrari S, et al. Cauda equina syndrome caused by lumbosacral epidural lipomatosis. A case report. *Clin Neurol Neurosurg*. 2013;115:1549-1551.
11. Kim S, Lim D. Epidural lipomatosis with cauda equina syndrome in chronic alcoholic patient: a case report. *Int J Surg Case Rep*. 2017;33:12-15.
12. Bushkar JB, Menkinsmith LP, Krywko DM. Idiopathic spinal epidural lipomatosis causing cauda equina syndrome. *Clin Pract Cases Emerg Med*. 2017;1:305-308.
13. Fogel GR, Cunningham PY, Esses SI. Spinal epidural lipomatosis: case reports, literature review and meta-analysis. *Spine J*. 2005;5:202-211.
14. Yasuda T, Suzuki K, Kawaguchi Y, et al. Clinical and imaging characteristics in patients undergoing surgery for lumbar epidural lipomatosis. *BMC Musculoskelet Disord*. 2018;19:66.
15. Fujita N, Hosogane N, Hikata T, et al. Potential involvement of obesity-associated chronic inflammation in the pathogenesis of idiopathic spinal epidural lipomatosis. *Spine (Phila Pa 1976)*. 2016;41:E1402-E1407.
16. Sugerman H, Windsor A, Bessos M, Wolfe L. Intra-abdominal pressure, sagittal abdominal diameter and obesity comorbidity. *J Intern Med*. 1997;241:71-79.
17. Wilson A, Longhi J, Goldman C, McNatt S. Intra-abdominal pressure and the morbidly obese patients: the effect of body mass index. *J Trauma*. 2010;69:78-83.
18. Varela JE, Hinojosa M, Nguyen N. Correlations between intra-abdominal pressure and obesity-related co-morbidities. *Surg Obes Relat Dis*. 2009;5:524-528.
19. Linicus Y, Kindermann I, Cremers B, Maack C, Schirmer S, Böhm M. Vena cava compression syndrome in patients with obesity presenting with edema and thrombosis. *Obesity (Silver Spring)*. 2016;24:1648-1652.

20. Fuchs F, Bruyere M, Senat MV, Purenne E, Benhamou D, Fernandez H. Are standard intra-abdominal pressure values different during pregnancy? *PLoS One*. 2013;8:e77324.

21. Boos N, Aebi M. *Spinal Disorders: Fundamentals of Diagnosis and Treatment*. Berlin, Germany: Springer-Verlag; 2008.

22. Ferlic PW, Mannion AF, Jeszenszky D, et al. Patient-reported outcome of surgical treatment for lumbar spinal epidural lipomatosis. *Spine J*. 2016; 16:1333-1341.

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