Minimal Approach to Spinal Epidural Lipomatosis Using the Japanese Step Ladder Technique: A New Technique in 2 Cases

Karim Baayoud, Michel Triffaux, Triantafyllos Bouras, and Sarah Lonneville

ABSTRACT

Introduction: Spinal epidural lipomatosis (SEL) refers to a pathological condition characterized by an abnormal accumulation of fat within the epidural space. It’s a rare but well-known condition, that is mostly associated with long-term corticosteroid therapy or elevation of endogenous steroids.

Objectives: We aim to present a comprehensive account of our experience with SEL, while also introducing a novel technique and its outcomes. Our approach is informed by a rigorous review of the relevant literature.

Results: We report 2 cases of lipomatosis seen in the neurosurgery department. The first was a case of SEL due to obesity, manifested by intermittent claudication. The second case followed a lumbar arthrodesis, with clinical signs associated with radiculopathy. Both patients were treated conservatively, with no improvement, after which we opted for surgical treatment. The postoperative evolution of the 2 cases was marked by regression of symptoms in both patients.

Keywords: Glucosteroid treatment, interlaminar approach, obesity, spinal epidural lipomatosis.

1. INTRODUCTION

Spinal epidural lipomatosis (SEL) is a rare condition, due to an excessive and abnormal accumulation of fat within epidural space. The underlying cause can be attributed to the extended utilization of exogenous steroids or the heightened levels of endogenous steroids. SEL is usually asymptomatic but can be symptomatic in cases of spinal cord or radicular compression. The lumbar region is most frequently affected. If conservative management fails, decompressive laminectomy is recommended to improve neurological symptoms.

Even though this pathology is fairly well described, there are currently no guidelines to advise on how to approach this condition.

2. OBJECTIVES

To describe a new technique and document our experience with LES in the Department of Neurosurgery in the light of a systematic review of the literature.

3. PATIENT SAMPLE

The study was carried out retrospectively over 4 years, in the neurosurgery department of the Hospital Center of Wallonie-Picarde (CHWAPI), in Doornik, Belgium. At the end of this study, we report 2 cases of lipomatosis, who underwent surgery.

4. CASE PRESENTATIONS

4.1. First Case

A male patient, aged 58, with no prior medical history and no identified history of long-term corticosteroid therapy or endocrine disorders.

The patient had chronic low-back pain of a mechanical nature, of low intensity (VAS = 3/10). His clinical symptoms worsened over the last month with the appearance of neurogenic intermittent claudication, with a walking perimeter of 150. He had no muscle weakness or sphincter disorders.
The clinical examination showed morbid obesity with a BMI of 40.2. On palpation, his extremities exhibited normal vascular pulses. His neurological exam was normal.

Lumbar MRI revealed an excess of fat tissue from T12 to L5 (Fig. 1a), related to SEL, and responsible for canal stenosis (Fig. 1b).

We initially attempted conservative treatment for 3 months with a weight-loss diet and physiotherapy but saw no improvement.

The indication for decompression was retained given the failure of conservative treatment. The patient underwent a staged hemilaminectomy from L3 to L5.

### 4.2. Second Case

A 72-year-old man with a history of lumbar arthrodesis L3-L4: for multistage disc disease, causing low back pain with bilateral radiculalgia, with good evolution. As per the patient’s medical records, there is no documented history of corticosteroid therapy or endocrine pathology.

For several months, the patient presented with mal-systematized, intermittent, left sciatica, of low intensity (VAS = 4/10), with no limb weakness or sphincter disorders. The symptomatology was marked by an increase in the radiculalgia intensity (VAS = 7/10).

On general observation, the patient had a normal BMI estimated at 24.2. His neurological exam was normal.

We performed a lumbar MRI, which revealed an accumulation of fat between L1 and L3 responsible for canal stenosis (Fig. 2).

The patient underwent hemilaminectomy with alternating sides from L1 to L3. The procedure was performed without any intraoperative incidents.

### 5. Technique Presentation

We marked the incision extent after the fluoroscopic guidance (Fig. 3). After Intraoperative confirmation of the appropriate level with fluoroscopy, the decompressive procedure was performed. We used a minimal unilateral laminectomy, extended with the Bone Scalpel (Misonix) in a staggered fashion over the height of the lipomatosis, like a Japanese step ladder (Fig. 4).

After the realization of the hemi laminectomy, we proceeded to an ultrasound control to appreciate the extent of the lipomatosis, but also, its impact on the dural sac and the rootlets (Fig. 5).

Then we proceed with unilateral flavectomy, preserving the supraspinal, and the interspinous ligaments. The contralateral lamina is preserved with her ligament flavum.

We therefore proceeded to the ablation of the lipomatosis by suction on both sides through the hemilaminectomy (Fig. 6).

After the removal of the SEL, an ultrasound check was performed to ensure the emptiness of the epidural space (Fig. 7).

The evolution was marked by a regression of the symptomatology in terms of both pain and improvement in walking perimeter for the first patient and left leg pain for the second.
A radiological control was performed for the 2 patients. No recurrence was reported.

6. Discussion

6.1. Epidemiology

According to our findings, the prevalence of symptomatic SEL is 6.3%, with an incidence rate of 2.5% per year [1]. In their retrospective study, Malone et al. analyzed the charts of 831 patients and determined that the mean age of diagnosis was 62 years old [1]. The youngest patient ever described was 6 years old [2].

Multiple studies show that SEL is more common in males than females (3:1) [3–7].

SEL is more commonly found in the lower lumbar and lower thoracic spine [8], with values varying from one series to another [1], [9], [10]. However, no case has ever been described of cervical SEL.

Some authors suggest that certain causes of SEL may have a preference for particular locations in the spine. Fogel et al. showed in their study that 55.8% of SEL due to exogenous steroid administration were located in the thoracic region, 32.7% involved lumbosacral level, and 11.5% affected both. As for the endogenous steroid-related SEL, he found that it affected both the thoracic and lumbosacral spine equally [11].

Recent studies have indicated that the development of epidural lipomatosis (SEL) varies among different races. In particular, the Yoo et al. study conducted on the Korean population found that a significant proportion (68.8%) of SEL cases were idiopathic. In contrast, the Fogel et al. study carried out in Western countries reported a lower rate (17%) of idiopathic SEL cases. These findings suggest that there may be racial differences in the pathogenesis of SEL [3], [11], [12].

6.2. Etiology

It has been reported in the literature that SEL development is commonly associated with five major etiologies:
exogenous steroid use, obesity, surgery-induced, idiopathic, and endogenous steroid hormonal disease [11].

6.2.1. Exogenous Steroid Use

Recognized as the predominant etiology leading to SEL. Furthermore, it is considered the most significant risk factor for developing this condition [3], [11]. It is most usually seen in conditions that require long-term steroid administration with moderate to high dosages for years [4]. We found in the literature cases of SEL described for an organ transplant, Crohn’s disease, ulcerative colitis, and nephritic syndrome. The mean dosages of prednisone required for the manifestation of symptoms typically fall within the range of 30 to 100 mg per day. The duration of steroid use necessary for the manifestation of clinical symptoms has varied between 5 and 11 years [10]. However, cases of lipomatosis have been described following a shorter duration of steroid therapy at low doses [10], [13].

6.2.2. Endogenous Steroid Hormonal Disease

Several pathologies causing endogenous steroid hormone excess have been associated with SEL in the literature. The most frequently reported are hypothyroidism, Cushing’s syndrome, pituitary prolactin adenoma, and carcinoid tumors [4], [14], [15].

6.2.3. Obesity

In some studies, it’s considered the most frequent etiology of SEL. It has been postulated that obesity can potentially lead to the development of chronic inflammation, which in turn may result in hypertrophy of the adipose tissue within the epidural space [16]–[18]. A high level of inflammatory cytokines was observed, in obese patients, which is likely responsible for the growth of adipose cells [16]. Obesity is accepted as a risk factor for developing SEL. Numerous studies have been conducted to investigate the potential relationship between BMI and SEL. However, the results of these studies consistently demonstrate a lack of significant correlation between these two variables [19], [20].

6.2.4. Surgery-Induced SEL

Choi et al. described two cases of lipomatosis after surgery. The first case had an anterior lumbar interbody fusion for symptomatic spondylolisthesis. During the surgery, they did not find any hypertrophy of the epidural fat tissue. The evolution was marked by regression of the symptoms. The patient developed months later neurological symptoms, the MRI showed an SEL. The symptoms are the result of compression of neural structures by excess fat tissue within the spinal canal. The severity of the compression and the location of the adipose tissue determine the clinical manifestation.

6.2.5. Idiopathic SEL

Several meta-analyses estimate the percentage of idiopathic at 17% [11], [20], [22].

6.3. Pathophysiology

SEL is an accumulation in the epidural space of unencapsulated adipose tissue that will compress the spinal cord or the rootlets. Its pathophysiology is still not well described in the literature [23].

This accumulation of fat occurs slowly and will be responsible over time for neurological symptoms [24], [25]. The symptoms can be due to the mass effect caused by the accumulation of fat or by the venous engorgement. The adipose tissue has glucocorticoid receptors [26]–[28]. Koch et al. suggest that glucocorticoids are responsible for the adipose tissue’s enlargement [15].

A mass in the thoracic canal, no matter how small, can produce symptoms as a result of the canal’s limited width and vascularity [3].

SEL is often localized posterior to the cord, however, it can be located in different compartments of the spinal canal depending on the etiology of the disease [13].

6.4. Clinical Presentation

It is commonly observed that back pain often manifests as the initial symptom, it usually has a progressive onset [4].

The symptoms of SEL are non-specific, they can be divided into myelopathy, radiculopathy or claudication, and in some rare cases cauda equina syndrome [29], [30]. These symptoms are the result of compression of neural structures by excess fat tissue within the spinal canal. The severity of the compression and the location of the adipose tissue determine the clinical manifestation.

6.5. Imaging Features

The MRI is the most reliable imaging modality for the diagnosis, but also for evaluating impingement upon the cauda equina [31].

In the CT, lipomatosis can easily be differentiated from other lesions due to its low density (80 to 120 Hounsfield units) [32].

In the MRI signal follows fat (high signal on T1WI, intermediate on T2WI). The diagnosis could be considered when the epidural adipose is superior to 7 mm thickness [3], [33]. It can also help distinguish excess fat from degenerative lesions, which leads to a narrowing of the spinal canal.

Borré et al. have proposed a scale for the classification of lipomatosis, which comprises four distinct grades ranging from 0 to 3. (Table 1) The grading system is based on the ratio of the dural sac, epidural fat, and anteroposterior diameter of the spinal canal, and is designed to provide an objective measure of the severity of the condition [31].

6.6. Treatment

SEL can be managed conservatively or with surgical decompression. The choice of treatment depends on the etiology and severity of the clinical presentation. Given the variety of etiology and pathogenesis of the SEL, every patient should have a unique approach [5].

6.6.1. Conservative Treatment

Conservative therapy includes weight loss and weaning of steroids. It has shown its efficacy [3], [10], [34]. Weight loss has shown its efficacy when obesity is the etiology of SEL. [3], [34], [35] by decreasing the amount of epidural fat within the spinal canal [36]. Robertson et al. have
recommended in their article, that at least 15 kg weight loss to control the SEL symptoms.

Many patients couldn’t withdraw steroid therapy, due to chronic diseases that required it.

There is a current controversy surrounding the use of epidural steroid injection as a treatment [5]. Others report successful pain management [6], [9].

6.6.2. Surgical Treatment

The main indications of surgical decompression are failure of the conservative treatment or the appearance of a neurological deficit [17], [37], [38]. Surgical decompression has shown its efficacy regardless of the technique used [16], [24], [39], [40]. It mostly consists of a laminectomy with resection of the excess epidural fat [6], [11], [41]. Kim et al. suggest that patients with obese patients should avoid surgery unless weight loss fails [17].

Minimally invasive procedures have shown their efficacy as an option in treating SEL [42], [43]. They have lower postoperative pain, shorter rehabilitation time, and less cosmetic damage compared to the open procedure [44]. Endoscopic removal of fat has been described as an option [45].

Given the infrequency of this pathology, there have been no studies comparing the results of open and minimally invasive management of SEL [46].

The technique we describe allows multi-stage decompression while preserving spinal stability, thus avoiding the need for the patient to undergo arthrodesis or its extension if this has already been performed.

6.7. Outcome

Surgery is usually associated with a significant improvement of the symptomatology [3]. The success rate of the surgical decompression for SEL secondary to exogenous steroid use is estimated at 77%. Preoperative steroid doses are also a factor that influences neurological recovery. Roy-Camille et al. report that patients receiving lower doses of steroids have a better outcome [32].

SEL tends to have a better outcome following surgical decompression in Lumbar locations compared to the thoracic location [32]. There is a correlation between the severity of the clinical presentation and the time to recovery.

Idiopathic SEL seems to have a better prognosis compared to the other etiologies [47]. Recurrences requiring repeat surgery due to reaccumulation of epidural fat are very rare [3].

7. Conclusion

SEL is a rare and complex entity due to the accumulation of fat in the spinal canal, causing symptoms secondary to neural structure compression. Surgical decompression may be reserved for patients with neurological symptoms refractory to the conservative treatment. Our technique enables a multi-stage approach to LES without compromising spinal stabilization.

Conflict of Interest

Authors declare that they do not have any conflict of interest.

References


