MRI DIAGNOSIS OF SPINAL EPIDURAL LIPOMATOSIS
AND ITS CORRELATION WITH CLINICAL PRESENTATION

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TABLE OF CONTENTS

1. SUMMARY
2. ACKNOWLEDGEMENTS
3. CONFLICT OF INTEREST
4. ETHICS COMMITTEE CLEARANCE
5. ABREVIATIONS LIST
6. TERMS
7. INTRODUCTION
8. AIM AND OBJECTIVES
9. LITERATURE REVIEW
10. RESEARCH METHODOLOGY AND METHODS
11. RESULTS AND THEIR DISCUSSION
12. CONCLUSIONS
13. REFERENCES
SUMMARY

Aim: To acquire a better understanding about abundance of SEL in population as a cause of lower back pain and other neurological deficits.

Objectives: To calculate the prevalence of spinal epidural lipomatosis detected on lumbar magnetic resonance imaging scans. To compare spinal epidural lipomatosis frequency in male and female patients. To quantify normal and pathologic amounts of lumbar EF in relation to the thecal sac in order to calculate SEL grades (0 - III) and determine their frequency. Analyze and discuss a correlation between the grade of SEL and patients' clinical symptoms.

Methodology: From the total number of 5183 lumbar MRI examinations, 90 patients meeting criteria for SEL were chosen. By taking measurements of dural sac, spinal canal, extradural fat thickness and comparing them, the severity of SEL was determined by 4 grades. The correlation between SEL grades and clinical presentation was analyzed.

Results: MRI scans of 45 males and 45 females were examined (mean age 56.61 years). DuS/EF and EF/Spi C indexes were calculated. SEL was graded according to Borre et al. classification. The MRI grading showed the following distribution: grade 1 (n=9) 10%, grade 2 (n=78) 80%, grade III (n=9) 10%. SEL Grade II was the most common (n=78/90, 80%).

All patients were symptomatic and had lower back pain. Grade I SEL patients complained only of lower back pain. In grade II SEL group, 27.7% of patients had additionally complained of Right/Left/Bilateral sciatica, leg weakness, neurogenic claudication and sensory changes, while the rest had LBP as a single complain. In grade III SEL group, all patients had been complaining of LBP with either unilateral or bilateral sciatica, leg numbness and weakness, neurogenic claudication and even cauda equina syndrome. Additionally, in group of grade III SEL patients, MRI showed severe compression of dural sac and its morphological distortion: 55.5% of patients showed a "Y" sign of dural sac on lumbar MRI, 33.3% had a linear morphological appearance of dural sac, and 11.1% had severely compressed circular morphology.

Conclusions:
Spinal epidural lipomatosis is a rare medical condition, with a prevalence determined in this study of 1.73%. Men and women of middle age were equally affected by SEL. Grade II SEL was the most
frequent form. Clinical presentation of the patient is directly correlated with MRI grade of SEL. All patients with SEL grade I-III complained of lower back pain.
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The author, Nikolay Daykhovskiy
CONFLICTS OF INTEREST

The author reports no conflicts of interest
ETHICS COMMITTEE CLEARANCE

Ethics committee clearance was not required for this study.
ABBREVIATIONS LIST

A-Pd DuS - Antero-posterior diameter of the dural sac
A-Pd EF - Antero-posterior diameter of the epidural fat
A-Pd Spi C - Antero-posterior diameter of the spinal canal
EF - Extradural Fat
IC - Intermittent claudication
LEL - Lumbar Epidural Lipomatisos
MRI - Magnetic Resonance Imaging
SEL - Spinal Epidural Lipomatisos
TERMS

Computed Tomography, CT
Cauda equina syndrome
Corticosteroids
Dural sac
Epidural Fat
Epidural lipomatosis, Classification
Extradural fat
Glucocorticoids
Idiopathic
Incidental SEL
Low back pain
Lumbar spine
Lumbar canal stenosis
Magnetic resonance imaging, MRI
Myelopathy
Neurogenic claudication
Neurological deficits
Obesity
Paraparesis
Paresthesias
Prevalence
Radiculopathy
Retrospective analysis
Spinal canal
Spinal cord, compression
Spinal epidural lipomatosis, SEL
Spinal epidural lipomatosis grade
Stenosis
Y-sign
Spinal cord
INTRODUCTION

Spinal epidural lipomatosis (SEL) is a rare medical condition, which might be defined in different ways, all of them coincidental in their essence: “abnormal quantity of epidural fat”, “excess of epidural fat”, “increased amounts of normal epidural fat”, “excessive fat deposition within the spinal canal”, “pathological overgrowth of epidural fat”, and “simple hypertrophy of epidural fat”. [1] At the very first time it was described by Lee et al. in 1975 in a 15-year-old patient receiving steroids after renal transplantation. Badami and Hinck in 1982 described the same syndrome in an obese woman who did not receive steroids. [2]

This uncommon disorder characterized by an accumulation of excessive unencapsulated epidural adipose tissue, which might cause compression of the neural elements within the spinal canal. In an early stage of the disease, the spinal cord is slightly compressed, causing no or mild clinical symptoms. In an advanced stage, it is severely compressed, thereby causing clinical symptoms, such as lower back pain, lower extremities weakness, numbness, paresthesias, neurogenic claudication, cauda equina syndrome, and in some rare cases, urinary bladder and bowel dysfunction. [3]

SEL most commonly affects the thoracic (58- 61%) or lumbar (39-42%) spine, however cases affecting the cervical spine have not been described in the literature to date, possibly due to the small amount of epidural fat in this part of the spine. [2]

Most patients with SEL are associated with the administration of exogenous steroids or several endocrinopathies, including Cushing syndrome and hypothyroidism. [3] Idiopathic SEL, defined as case without evidence of definite predisposing factors and it seems to occur in obese patients, however the pathophysiology remains unclear. [4]

Magnetic resonance imaging (MRI) is the modality of choice in demonstrating the ongoing process of EF accumulation. Adipose tissue is characterized by hyperintense signal on T1W and intermediate signal intensity on T2W MR images. The typical presentation is circumferential compression of the dura by excessive deposition of adipose tissue. Advanced SEL can appear as a small oval, linear or Y-shaped on the axial slice of the MRI scan, commonly referred to as the stellate sign, or the “Y” sign. EF thickness greater than 7 mm is diagnostic feature of spinal epidural lipomatosis. [3, 5]
The goal of this study was to quantify the frequency of SEL detected on lumbar MRI scans, to quantify normal and pathologic amounts of lumbar EF in relation to the thecal sac to establish MRI grading of LEL, and to provide a correlation between severity of SEL and patients’ clinical symptoms.
AIM AND OBJECTIVES

The aim of this study is to acquire a better understanding about abundance of SEL in population as a cause of lower back pain and other neurological deficits.

Objectives:
1) To quantify the prevalence of spinal epidural lipomatosis detected on lumbar magnetic resonance imaging scans;
2) To compare spinal epidural lipomatosis frequency in male and female patients;
3) To quantify normal and pathologic amounts of lumbar EF in relation to the thecal sac in order to calculate SEL grades (0 - III) and determine their frequency;
4) To analyze and discuss a correlation between the grade of SEL and patients' clinical presentation.
LITERATURE REVIEW

Spinal epidural lipomatosis is a rare nonneoplastic condition with a strong male predominance, characterized by an abnormal accumulation of histologically normal unencapsulated fat in the extradural space. This leads to compression of the nerve roots and the spinal cord, which most often manifests clinically as lower back pain. As the disease progresses, the following symptoms are frequently encountered: lower extremities weakness, numbness, paresthesias, ataxia, intermittent claudication, paraparesis, cauda equina syndrome and in rare cases impaired urinary bladder and bowel functions. [6, 7]

SEL may be idiopathic, but is most often seen secondary to the endocrinopathies, including Cushing's syndrome and hypothyroidism and in the conditions requiring a long-term administration of exogenous corticosteroids. Obesity is the main accompanying risk factor in both the idiopathic and the secondary SEL [8, 9]. The secondary form affects the thoracic spine more frequently than it affects the lumbar region, whereas idiopathic form most often occurs in the lumbar region. [10]

Magnetic resonance imaging (MRI) is the imaging modality of choice in demonstrating the ongoing process of extradural fat accumulation. In an early stage of the disease, the dural sac is slightly reduced. In an advanced stage, it is severely compressed. [1]

9.1 Epidural fat as a normal content of spinal canal

9.1.1 Histology of epidural fat

Fat cells, or adipocytes, are unilocular with color varying from white to yellow, which depends on the amount of carotenes consumption in the diet. These large cells (up to 120 µm in diameter) are of a spherical shape and become polyhedral when large amounts of cells aggregate to become fat tissue. Every adipose cell has a single large lipid vacuole in the center and a peripheral oval nucleus. Fat tissue is being held together in packs within the epidural space by pedicles containing vessels. Epidural fat does not have a semifluid nature and contributes to the shape of the epidural space in the spinal canal. [34]

9.1.2 Distribution of EF

It is now certainly known that the epidural space contains abundant epidural fat tissue that distributes along the spinal canal in a predictable pattern [5, 34]. Although EF is distributed differently in the cervical, thoracic, and lumbar regions, however within each region the distribution remains consistent. [11]
In the cervical region, fat is absent, or almost nonexistent, in the anterior and lateral aspects of the epidural space, however a small deposit is sometimes might be seen in the posterior aspect. [12]

At thoracic levels, the epidural fat forms a broad posterior band with “indentations”. It is thicker near the intervertebral discs and much less prominent around the middle section of the vertebral bodies and close to the base of the spinous processes. Dorsal fat is present at all levels in the thoracic region, set between the vessels of the internal vertebral venous plexus. Lateral fat also exists at all levels between successive roots; that from T1 to T6 is small in quantity, becoming progressively larger in a caudal direction. Larger lateral masses are present from T7. Dorsal and lateral deposits appear to be continuous with each other at many places, except as prevented by the disposition of vessels. In the upper to middle thoracic levels (T1-7), epidural fat is continuous and the indentations are more evident. In the lower thoracic region, (T8-12) the distribution of epidural fat becomes patchy. [12, 13]

In the lumbar region, the fat in the anterior, and posterior, aspects of the epidural space forms two isolated structures. Epidural fat is also continuous with adipose tissue in the root canals. Both dorsal and lateral deposits appear in the lumbar region, however the two are frequently separated from each other. Lateral fat was found not only between successive roots but also ventral to them, forming a pad. Lateral aggregations became even more conspicuous at L5 and dorsal fat less so. The posterior epidural fat acquires its greatest volume in the caudal lumbar levels. [12, 13]

In the sacral region much fat is present surrounding the nervous elements on all sides. Remarkably little fat is visible from the ventral side. No mid-ventral fat is presenting rostral to L5, but from that point caudally a continuous pad appears. [12, 13]

9.1.3 Importance of EF

Thermal and mechanical protection is offered by the epidural fat. It protects the spinal cord within the dural sac and spinal nerves by buffering the pulsatile movements of the dural sac, facilitates dural sac movement over periosteum during flexion and extension, and creates reservoir for lipophilic substances. It fills the dural sac and protects its contents against the effects of lashing, deceleration, and rotational forces inflicted on the vertebral column. [12, 13]

9.2 Etiology and pathogenesis of SEL

The epidural space is lined with a thin layer of vascularized adipose tissue, which has a varying thickness of 3-6 mm showing in sagittal MRI. Since this space can't expand due to the tight
discoligamentary and rigid osseous limitations, an abnormal hypertrophy of the epidural fat tissue leads to a spinal cord compression in the spinal canal. The exact causes of this are still not known. [10]

Hypertrophy of epidural adipose tissue is most frequently observed in conditions, requiring chronic exogenous administration of glucocorticoid hormones (cortisone intake in rheumatic diseases, chronic inflammatory diseases or post-transplant patients) or in the conditions with an increased endogenous corticosteroid synthesis and secretion (ectopic hormone production). Obesity is an important factor contributing to an abnormal extradural fat hypertrophy. [14]

The predisposing factors for SEL development include the following:
1. Exogenous corticosteroids intake for conditions such as organ transplantation, pineoblastoma, cerebral lymphoma, prostate cancer, ulcerative colitis, Crohn's disease, chronic obstructive pulmonary disease, asthma, systemic lupus erythematosus, rheumatoid arthritis, polyarthritis, Graves disease, chronic hepatitis, dermatomyositis, nephritic syndrome, glomerulonephritis, sarcoidosis, multiple sclerosis, atopic dermatitis, diabetes mellitus, lichen ruber planus, polyarteritis nodosum (exogenous corticosteroid therapy accounts for 55% of the cases, being the most common risk factor for SEL); [3, 15]
2. Obesity (25% of the cases); [3, 15]
3. Idiopathic (17% of the SEL cases); [3, 15]
4. Cushing syndrome (3% of the cases); [3, 15]
5. Hypothyroidism;
6. Pituitary tumors;
7. Ectopic ACTH-producing tumors;
8. Epidural steroid injections;
9. Administration of Protease Inhibitors for HIV treatment (combined together with steroids); [1, 7, 10, 14]

The predominant numbers of SEL cases are associated with an exogenous use of corticosteroids. In most cases epidural lipomatosis precedes a long-term intake of corticosteroids of 5 to 11 years (30-100 mg prednisone equivalent/day), but there also known SEL cases detected after several months of glucocorticoid therapy [10] and even a single-shot epidural glucocorticoid injection (Lopez Gonzales et al. first presented a case of 45 years old diabetic man who received a single epidural steroid injection which had subsequently led to neurogenic claudication as a result of rapid epidural fat accumulation at the injection site, confirmed by MRI and surgery) [16]. Conditions related to hypercortisolism are characterized by a typical fat redistribution on face, trunk and mediastinum, contributing to the distinctive cushingoid appearance. It has been proposed that excess of steroids may
also lead to hypertrophy of the adipose tissue that is already present in the extradural space of the thoracic and lumbosacral spine. [7, 10]

9.2.1 Idiopathic SEL and obesity

In the idiopathic form neither a cause nor a genetic predisposition are known, however previous studies reports a strong association of idiopathic SEL with obesity [10, 17]. In general, the size of adipocytes contributes to the degree of obesity, due to the increase in triglyceride deposition within these cells. Interestingly, hypertrophic adipocytes constitutively express the pro-inflammatory cytokine tumor necrosis factor-α (TNF-α), and the serum TNF-α levels correlate with the degree of obesity in humans [18, 19]. Moreover, hypertrophic adipocytes also secrete high levels of adipokines (e.g., adiponectin and leptin) that are involved in the regulation of energy metabolism and inflammation [20]. These observations strongly indicate that chronic inflammation is causally related to adipocytes hypertrophy in humans. Since the majority of patients with idiopathic SEL are obese [8], there is a hypothesis that chronic inflammation and adipocyte hypertrophy are also involved in the pathogenesis of idiopathic SEL.

In the recent study, Fuhita et al. morphometrically analyzed the EF tissues collected from patients with idiopathic SEL and control subjects and found that the size of the adipocytes was significantly larger in patients with idiopathic SEL. Furthermore, an analysis of gene expression revealed higher transcript levels in two major cytokines, TNF-α and interleukin (IL)-1β, in patients with idiopathic SEL compared with the control subjects. Taken all those facts together, their study suggests that chronic inflammation and adipocytes hypertrophy are involved in the development of idiopathic SEL. [21]

Interestingly, Akhaddar et al. described a case of idiopathic SEL, where a 24-year-old man without obesity and any other risk factors presented with a 2-year history of mid-thoracic back pain and progressive neurogenic claudication with two episodes of unusual remitting and relapsing course. MRI demonstrated increased posterior spinal epidural fat (thickness more than 7 mm) causing variable anterior compression of the cord. After a bilateral T4-T9 laminectomy patient became completely free of pain. [22]
Such untypical cases are a rarity and do not present daily in clinical practice, however they definitely worth attention from medical personnel.
9.3 Epidemiology of SEL

In general, for SEL, men of the middle age are more affected than females and the most amounts of patients are obese. [23]

According to the reviewed literature, some sources state that SEL has a strong male predominance, with men (70-75%) are likely to be affected as much as twice or even more, compared to women (25-30%) [1, 14, 24], while other sources describe only a slight tendency for men to be affected by SEL more than females.

The mean age at the time of diagnosis is 43 years, although cases are known in younger patients and even in children [7]. Literature reports as young as 5 years old girl with systemic lupus erythematosus that developed secondary SEL after 1, 4 years of steroid treatment. [25]

According to the literature, the prevalence of overall SEL among the population was 2.5%, of which:
- Incidental finding of SEL on lumbar MIR scans, 0.6% ;
- Patients with SEL, presenting with non-specific symptoms(lower back pain), 1.8% ;
- Patients with SEL, presenting with specific symptoms (lower extremities numbness, IC, paresthesias) 0.1%. [26]
The authors of the recent researches found, that about 1 in 40 patients undergoing a spine MRI procedure had signs of SEL; 23% of whom didn't have any symptoms, 72% of whom were complaining about spine-related non-specific symptoms (such as lower back pain), and 5% had neurological deficits related to SEL. [26]

The SEL is mostly thoracic and lumbar. Thoracic infiltration is slightly more common with the 58-61% than the lumbar (39-42%) and 10% of the patients may present with the involvement of both. An isolated sacral lipomatosis is a rarity. Cervical involvement has not been described in the literature to date [7, 26]. The thoracic SEL is most commonly found at T6-T8 levels of the spine and the lumbar SEL at L4-L5 levels of the lumbosacral region. Patients with spinal epidural lipomatosis at the thoracic level generally present at an earlier age than the lumbosacral region group (average age 38.1 years for thoracic versus 51.4 years for lumbar). One of the possible reasons for this might be explained by anatomico-physiological peculiarities of the spine: the narrower space of the thoracic spinal canal, limited vascularity, and the fact that a larger proportion of epidural fat offers very little compliance to compressive effects. [4, 14]
9.4 Clinical presentation of SEL

In spinal epidural lipomatosis, abnormal overgrowth of adipose tissue in the epidural space causes compression of the nerves roots and the spinal cord. In an early stage of the disease, the spinal cord is slightly compressed, causing no or mild non-specific clinical symptoms. In an advanced stage, increased amount of extradural fat causes severe mechanical compression of the dural sac and vascular compromise, contributing to the neurological deficits. [6]

Obviously, the clinical presentation strongly depends on the level of spinal canal compromise (spinal cord, conus medullaris or cauda equina). For thoracic SEL, observation will show signs of spinal irritation, mostly ataxia as well as clinical signs and symptoms of myelopathy. In SEL at lumbar levels, there may be symptoms and signs of radicular impairment, neurogenic claudication and even cauda equina syndrome. [14, 27, 28]

In general, the onset of symptoms is gradual and the condition progresses slowly. Epidural lipomatosis commonly presents with localized chronic pain, which has often lasted months to several years, followed by progressive or sudden neurologic deficits [14, 28]. Back pain is being the most frequently reported symptom associated with SEL and often presents long before the other neurologic symptoms [6]. Lower-extremity weakness (which appears to be slowly progressive in most cases) and sensory changes with numbness, paresthesias, or radicular symptoms are the most common finding during physical examination, though decreased pinprick sensation and altered deep tendon reflexes are also frequently encountered. Bowel and bladder incontinence are reported, but appear to be rare complaints. [6, 14]

9.4.1 Clinico-radiological correlation of SEL

At year 2003, Daniel G. Borre and his associates published an article [1], where they proposed an MRI classification of lumbosacral spinal epidural lipomatosis based on the gradual process of extradural fat hypertrophy. By calculating the anterior posterior diameters of the dural sac, the spinal canal and the EF and comparing them, Borre and company have established four grades of the disease progression:
- Grade 0, which contributes to the absence of extradural fat hypertrophy.
- Grade I (Mild), the early stage, representing an incipient hypertrophy of epidural adipocytes.
- Grade II (Moderate), an intermediate stage, represents a moderate hypertrophy of epidural fat.
- Grade III (Severe), the end stage, represents the highest degree of epidural adipose tissue accumulation. [1]
In the group of LEL grade I all the examined patients were asymptomatic. This is attributed to the only a mild overgrowth of the epidural fat. On MRI scans the EF/Spi C index were slightly elevated (41-50%), however the dural sac is still not compressed, thereby not causing any neurologic symptoms nor signs. [1]

In the group of LEL grade II, 14, 5% of the patients were symptomatic, complaining about non-specific neurologic symptoms (low back pain, weakness, dysesthesias), while the rest of them were asymptomatic. This stage represents a moderate overgrowth of EF, with an EF/Spi C index varying from 51-74%. While the epidural adipose tissue hypertrophy is objectively present, the dural sac, though still rounded in shape, has been only a partially compromised. [1]

In the group of LEL grade III, all of the cases had clinical symptoms at presentation, such as low back pain, radiculopathy, numbness, paresthesias, neurogenic claudication and even in some cases, cauda equina syndrome. This end stage of SEL represents the highest degree of EF accumulation (EF/Spi C index ≥75% with extreme compression of the thecal sac, leading to the neurologic dysfunctions. [1]

9.5 Diagnosis of SEL

The clinical diagnosis of SEL is very challenging. Symptoms and signs of SEL imitate those observed in other conditions such as vertebral and disc disease (herniated disc) [29], epidural abscess or hemorrhage, transverse myelitis, paraspinal lesions, multiple sclerosis and so on. Patients with underlying malignancy and metastatic disease of the spine may mimic clinical presentation of SEL as well. Thus, definitive diagnosis of SEL requires a complex approach including a clinical suspicion, imaging studies, surgical evaluation, and in some cases even a biopsy. [7]

Firstly, a complete physical examination is required with thorough evaluation of the individual’s medical history. A careful anamnesis should account for all of the causes and associated risk factors (see chapter 9.2), especially for the symptomatic patients undergoing a long-term treatment (5-11 years) with exogenous steroid therapy. [17]

Laboratory tests recommended to be performed in order to rule out an endocrinopathy or other pathological conditions. [28]

However, plain radiographs of the lumbar spine are usually normal and cannot diagnose SEL, X-ray imaging of the affected portion of the spine might be helpful to exclude other possible causes of low back pain, like degenerative disease and tumors. [2, 27]
Myelography, a type of radiological exam using a contrast to detect cord compression. It reveals an obstruction to the contrast material at the level of the spinal canal compression, and the filling pattern is useful for determining whether the obstruction is intradural or extradural. An hourglass appearance of contrast obstruction is usually associated with an epidural mass (such as SEL), however it's not specific. [14]

Interestingly, Schulz et al. indirectly discovered an epidural lipomatosis case due to high impedance values observed during a failed spinal cord stimulator trial; this case was an old age morbidly obese female with a history of chronic lower back pain and bilateral neuropathic leg pain encompassing the L3-S1 dermatomes. The authors of this article suggest that this finding may have a clinical application for patients with obesity or a risk of endocrinopathy, when high impedance is identified during a spinal cord stimulation trial with no obvious explanation nor reason. [30]

CT scan of the affected region can be used in the cases of strongly obese patients or for whom MRI is contraindicated [10]. Axial CT scans are used to help differentiate the cause of compression based on the density of the tissue in the epidural space. Adipose tissue has a density ranging between 80 and 120 HU (Hounsfield Units) on CT scans, which helps to differentiate it from most other tissues in the area. [14]

Before the MRI era, most case reports relied heavily on CT imaging and myelography, however nowadays MRI is being the modality of choice for diagnosis of SEL. [28]

Nerve conduction studies are used, if the nervous system is affected and neurological symptoms are present. Additional tests might be carried out with respect to the underlying condition or disorder. A biopsy of EF might be performed and sent to a laboratory for a pathological examination. After putting together clinical findings, special studies on tissues (if needed) and with microscope findings, the pathologist arrives at a definitive diagnosis.

9.5.1 The role of MRI in diagnosis of SEL

In recent times, MRI is recognized as the most sensitive and specific modality of choice in demonstrating the ongoing process of extradural fat accumulation. [5]

Spinal canal compression and obliteration of cerebrospinal fluid spaces can be appreciated on axial and sagittal MR images. On conventional spine echo MRI, adipose tissue demonstrates a high signal intensity on non-contrast T1-weighted images and an intermediate signal intensity on T2-weighted images [5]. The high contrast between adipose tissue and the thecal sac permits an accurate evaluation of the extent of pathologic epidural adipose tissue overgrowth in the spinal canal. This
allows us to differentiate epidural fat from dural content with a high degree of specificity as well as to measure the adipose thickness. [1, 7]

Quent et al. conducted a study in order to calculate the normal range of the spinal canal adipose tissue, where an MRI scanning was done for a group of 28 healthy patients. On sagittal MR images, the mean thickness of their epidural fat posterior to the spinal cord at the thoracic level was measured 4.6 mm (range, 3-6 mm) [31]. Epidural adipose tissue that has a thickness greater than 7 mm has been reported to be the diagnostic criterion for SEL. [14]

On axial MRI of the lumbar spine, the thecal sac is intact and has a round, circular appearance. As the accumulation of fatty tissue progresses, the dural sac becomes compressed which leads to a different morphology on MRI scan. In the mild-moderate stage, thecal sac may appear slightly flattened or oval. However, in severe lipomatosis it acquires a stellate appearance with three rays radiating from the central core. This configuration was described by Kuhn et al. as the "Y-sign" of the thecal sac, a finding which is pathognomonic for LEL [32]. Borre et al. in their study found that 46% of patients with III grade SEL had a "Y-sign" configuration of thecal sac on their axial MRI scans. [1]

9.5.2 MRI grading of LEL

As discussed before (Chapter 9.4.1), Daniel G. Borre et al. at the year 2003 [1] conducted a study, that was reviewed by Pinkhardt et al. at year 2007 [33]. They observed axial MRI scans of 2528 patients. For each patient they obtained four linear measurements at the level of the superior end plate of the S1 vertebral body:

1. Antero-posterior diameter of the dural sac (A-Pd DuS)
2. Antero-posterior diameter of the epidural fat located ventrally to the dural sac (Segment A)
3. Antero-posterior diameter of the epidural fat located dorsally to the dural sac (Segment B)
4. Antero-posterior diameter of the spinal canal (A-Pd Spi C), which is equivalent to the addition of the A-Pd DuS plus segments A and B

These measurements allowed them to establish the relation between the dural sac and the epidural fat as well as between the EF and the spinal canal. They obtained these relations by means of the following equations:

\[
\text{DuS/EF index} = \frac{A-Pd \text{ DuS}}{EF (\text{segment } A+B)}
\]
EF/Spi C index = \frac{\text{minor diameter of DuS}}{A-Pd SpiC - \text{minor diameter of DuS}}

However, in some cases of advanced LEL the fat tissue hypertrophy produced a severe distortion of the dural sac (DuS), changing its characteristic oval shape into a "Y" form or a thin band (linear shape). Hence, in these cases, the A-Pd DuS was not reliable measurement to calculate the indexes. Therefore, they modified the previous equations to avoid paradoxical results:

Modified DuS/EF index = \frac{\text{minor diameter of DuS}}{A-Pd SpiC - \text{minor diameter of DuS}}

Modified EF/Spi C index = \frac{A-Pd SpiC - \text{minor diameter of DuS}}{A-Pd SpiC} \times 100

Subsequently, an MRI grading of LEL was developed attending to the spatial relation between DuS and EF and the following classification was proposed (Table 1):

Table 1. MRI classification of spinal epidural lipomatosis

<table>
<thead>
<tr>
<th>MRI grade</th>
<th>DuS/EF index</th>
<th>EF/Spi C index</th>
<th>Meaning</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal 0</td>
<td>≥1.5</td>
<td>≤40</td>
<td>Normal amount of EF</td>
</tr>
<tr>
<td>Grade I</td>
<td>1.49-1</td>
<td>41-50</td>
<td>Mild overgrowth of EF</td>
</tr>
<tr>
<td>Grade II</td>
<td>0.99-0.34</td>
<td>51-74</td>
<td>Moderate overgrowth of EF</td>
</tr>
<tr>
<td>Grade III</td>
<td>≤0.33</td>
<td>≥75</td>
<td>Severe overgrowth of EF</td>
</tr>
</tbody>
</table>
MATERIALS AND METHODS

10.1 MRI procedure
For our patients the MR examinations were performed on either "Siemens Magnetom Concerto" 0.2 T or "Siemens Magnetom Skyra" 3 T. Sequences included the following:
1. Spin-echo (SE) T1-weighted sagittal images (TR 690ms/TE 8.5 ms, with a 320x320 matrix, 4-mm slice thickness);
2. SE T2-weighted sagittal images (TR 3600ms/TE 107 ms, with a 320x320 matrix, 4-mm slice thickness);
3. Fast spin-echo (FSE) T2-weighted sagittal images (TR 4890/TE 97 ms, with a 321x321 matrix, 4-mm slice thickness);
4. FSE T2-weighted axial images (TR 4000ms/TE 90 ms, with a 221x221 matrix, 3-mm slice thickness).

10.2 Subjects selected for the retrospective analysis
Out of 5183 MR examinations of the lumbar spine performed in MRI department of Republic Klaipeda Hospital during a period from 2011.01.01 to 2017.04.06, 90 cases were selected (45 females and 45 males; age range 20-85 years; mean age 56.61 years).
The main criteria for all selected patients was the presence of epidural fat hypertrophy affecting the same spine level (S1) on sagittal and axial MRI scans.
The exclusion criteria for this retrospective study were: Spondylitis, discitis, disc herniation (when extended intradurally or severely medially extruded at L5-S1 level), epidural hematomas, extradural tumors, lumbosacral trauma or pathologic fractures (such as metastases) with anatomic distortion of spinal canal, previous lumbosacral surgery.

10.3 Patients classification
By evaluating mainly T2W axial scans, for each patient we obtained four linear measurements at the level of S1 vertebral body (Figure 1):
1. Antero-posterior diameter of the dural sac (A-Pd DuS)
2. Antero-posterior diameter of the epidural fat located ventrally to the dural sac (Segment A)
3. Antero-posterior diameter of the epidural fat located dorsally to the dural sac (Segment B)
4. Antero-posterior diameter of the spinal canal (A-Pd Spi C), which is equivalent to the addition of the A-Pd DuS plus segments A and B
Figure 1. Demonstration of the process of measuring the A-Pd DuS, A-Pd of EF and A-Pd Spi C. (MRI, T2W, axial plane, S1 vertebral level)

Subsequently, the two indexes were calculated in order to establish SEL grades:

\[ \text{DuS/EF index} = \frac{A-Pd \ DuS} {EF \ (\text{segment} \ A+B)} \]

\[ \text{EF/Spi C index} = \frac{\text{minor diameter of DuS}} {A-Pd \ SpiC - \text{minor diameter of DuS}} \]

In cases of severe distortion of morphological appearance of the dural sac ("Y" shape or linear shape), we used modified equations of previously mentioned indexes:

Modified DuS/EF index = \[ \frac{\text{minor diameter of DuS}} {A-Pd \ SpiC - \text{minor diameter of DuS}} \]

Modified EF/Spi C index = \[ \frac{A-Pd \ SpiC - \text{minor diameter of DuS}} {A-Pd \ SpiC} \times 100 \]

Based on those measurements, the severity of SEL was determined by four grades: Grade 0, Grade I, Grade II and Grade III.
RESULTS AND THEIR DISCUSSION

11.1 Gender and age

Out of 5183 MR examinations of the lumbar spine 90 patients (1.73%) were selected meeting the criteria for SEL (Table 3). Age range was 20-85 years; mean age 56.61 with a standard deviation of ±11.77 years. 45 patients (50%) were males and 45 females (50%), with a male-to-female ratio being 1:1.

According to the literature, SEL has a strong male predominance with men (70-75%) are likely to be affected as much as twice or even more, compared to women (25-30%). [1, 14, 24] However in this study we found, that men (n=45, 50%) and women (n=45, 50%) were equally affected by SEL.

11.2 Frequency and prevalence of SEL

In this study, prevalence of SEL was estimated to be 1.73% (90 cases out of 5183), meaning that approximately each 57th patients would have epidural lipomatosis signs on MRI. Grade 0 patients (n = 5093) accounted for 98.26%, grade I patients (n = 9) accounted for 0.17%, grade II patients (n=72) were 1.38% and grade III patients (n=9) were 0.17% (Graph 1).

Graph 1. Prevalence of spinal epidural lipomatosis

![Prevalence of SEL](null)

Out of those 90 cases (100%), 9 patients (10%) had I grade (mild) SEL, 72 patients (80%) were diagnosed with grade II (moderate) SEL and 9 patients (10%) had grade III (severe) SEL (Graph 2).
Previous articles have shown, that the prevalence of overall SEL among the population was 2.5% [26], where in our study SEL prevalence was only of 1.73%, which is less if compared to the published data.

Borre et al. in their study calculated overall prevalence for SEL and its grades (Table 2). [1]

### Table 2. Comparison of SEL prevalence found in our study with reviewed literature

<table>
<thead>
<tr>
<th></th>
<th>SEL grade 0</th>
<th>SEL grade I</th>
<th>SEL grade II</th>
<th>SEL grade III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Our study</td>
<td>98.26%</td>
<td>0.17%</td>
<td>1.38%</td>
<td>0.17%</td>
</tr>
<tr>
<td>Borre</td>
<td>79.2%</td>
<td>12.2%</td>
<td>6.5%</td>
<td>2.1%</td>
</tr>
</tbody>
</table>

However, our values are not 100% reliable in terms of discussing the overall prevalence of SEL due to the selection criteria for our data base (all SEL cases had to be at the same level (S1), cases with other pathologies on MRI scan had to be excluded). Rather, in our study we discussed a prevalence of SEL among population at S1 vertebral body level without any other pathological findings on lumbar MR images.

#### 11.3 Clinico-radiological correlation

All 90 cases (100%) were symptomatic (Table 3). Each of those 90 patients reported low back pain as a recent or ongoing complain. However, the other symptoms were varying:

- In the group of grade I SEL (n=9), all patients complained only of lower back pain;
- In group of grade II SEL (n=72), among lower back pain, 27.7% (n = 20) of patients had additionally complained of LBP irradiating to either one or both legs, leg weakness, neurogenic claudication,
sensory changes such as numbness and abnormal Achilles tendon reflexes (Table 1);
- In group of grade III SEL (n=9), 100% of patients had additional complains such as LBP with either unilateral or bilateral sciatica, leg numbness and weakness, neurogenic claudication and even cauda equina syndrome.

According to this analyzed data, the strong correlation can be seen between the severity of SEL progression (grade of SEL) and clinical presentation of the affected individuals. Patients in early stage of disease complain of nonspecific symptoms (such as lower back pain), while as the condition worsens, the more neurological deficits are noted (sciatica either bilateral or unilateral, leg weakness, neurogenic claudication).

According to the patients' clinical presentation, we divided our patients into 4 groups (Graph 3):
- Group 1 patients had low back pain as the single complain at the time of diagnosis;
- Group 2 patients had low back pain radiating to the left leg;
- Group 3 patients had low back pain radiating to the right leg;
- Group 4 patients had low back pain radiating to the both legs.

**Graph 3. Clinical presentation of patients with SEL**
Our results differ from that of reported in the literature. Borre et al. at year 2003 provided a correlation between MRI grade of LEL and clinical presentation of the patients.

In the group of LEL grade I all the examined patients were asymptomatic [1], whereas all our grade I SEL patients complained of low back pain only.

In the group of LEL grade II, 14.5% of the patients were symptomatic, complaining about non-specific neurologic symptoms (low back pain, weakness, dysesthesias), while the rest of them were asymptomatic [1]. In our group of SEL grade II, 27.7% of patients complained of additional neurological deficits besides LBP, while the rest had LBP only as a complain.

In the group of LEL grade III, all of the cases had clinical symptoms at presentation, such as low back pain, radiculopathy, numbness, paresthesias, neurogenic claudication and even in some cases, cauda equina syndrome [1], which is the common point among our study and the literature, because all our SEL grade III patients (100%) were symptomatic (LBP with other neurological deficits, such as unilateral or bilateral sciatica, leg numbness and weakness, neurogenic claudication and even cauda equina syndrome).

11.4 Grade III SEL dural sac morphology

Borre et al. in their study have found eight different morphologic patterns of the dural sac caused by the severe hypertrophy of epidural adipose tissue in group of SEL grade III patients:

The "Y" shaped morphology was seen in 46% of SEL grade III cases; rounded shape was seen in 36.5% of cases; other six patterns were unusual. [1]

In our group of grade III SEL patients, MRI showed severe compression of dural sac and its morphological distortion. Out of 9 patients with Grade III SEL, 55.5% of patients (n =5) showed a "Y-sign" of dural sac on lumbar MRI (Figure 2), 33.3% (n=3) had a linear morphological appearance of dural sac, and 11.1% (n=1) had severely compressed circular morphology.

Therefore, the "Y" shaped morphological pattern of dural sac is the most common finding in advanced SEL, followed by round and compressed linear shape morphology on axial MRI scans.
**Figure 2-A.** MRI, T1W, sagittal view. Advanced overgrowth of epidural EF is seen.

**Figure 2-B.** MRI, T2W, axial view. Distorted morphological appearance of dural sac, "Y" - sign, caused by severe EF hypertrophy.
Table 3. Presentation of the results.

<table>
<thead>
<tr>
<th>Patient's number</th>
<th>Gender</th>
<th>Age</th>
<th>DuS/EF index</th>
<th>EF / Spi C index</th>
<th>SEL grade</th>
<th>Clinical presentation</th>
</tr>
</thead>
<tbody>
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<tr>
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<td>0.43</td>
<td>II</td>
<td>LBP; R sciatica; R leg weakness.</td>
<td></td>
</tr>
<tr>
<td>73.</td>
<td>female</td>
<td>64</td>
<td>0.41</td>
<td>II</td>
<td>LBP; bilateral sciatica; Neurogenic claudication.</td>
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</tr>
<tr>
<td>74.</td>
<td>male</td>
<td>69</td>
<td>0.67</td>
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<td>LBP</td>
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</tr>
<tr>
<td>75.</td>
<td>female</td>
<td>69</td>
<td>0.49</td>
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<td>LBP</td>
<td></td>
</tr>
<tr>
<td>76.</td>
<td>female</td>
<td>58</td>
<td>0.36</td>
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<td>LBP; L sciatica; Impaired Achilles tendon reflex.</td>
<td></td>
</tr>
<tr>
<td>77.</td>
<td>female</td>
<td>73</td>
<td>0.26</td>
<td>II</td>
<td>LBP; R sciatica; R leg weakness; Neurogenic claudication.</td>
<td></td>
</tr>
<tr>
<td>No.</td>
<td>Gender</td>
<td>Age</td>
<td>BMI</td>
<td>Status</td>
<td>Diagnosis</td>
<td></td>
</tr>
<tr>
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<tr>
<td>78.</td>
<td>female</td>
<td>64</td>
<td>0,45</td>
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<td>LBP; R sciatica.</td>
<td></td>
</tr>
<tr>
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<td>II</td>
<td>LBP</td>
<td></td>
</tr>
<tr>
<td>80.</td>
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<td>56</td>
<td>0,85</td>
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<tr>
<td>81.</td>
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<td>72</td>
<td>0,55</td>
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<td>LBP</td>
<td></td>
</tr>
<tr>
<td>82.</td>
<td>male</td>
<td>46</td>
<td>0,73</td>
<td>II</td>
<td>LBP</td>
<td></td>
</tr>
<tr>
<td>83.</td>
<td>male</td>
<td>65</td>
<td>0,39</td>
<td>II</td>
<td>LBP; R sciatica; R leg weakness, numbness. Impaired Achilles tendon reflex; Neurogenic claudication.</td>
<td></td>
</tr>
<tr>
<td>84.</td>
<td>female</td>
<td>52</td>
<td>0,62</td>
<td>II</td>
<td>LBP</td>
<td></td>
</tr>
<tr>
<td>85.</td>
<td>male</td>
<td>53</td>
<td>0,47</td>
<td>II</td>
<td>LBP</td>
<td></td>
</tr>
<tr>
<td>86.</td>
<td>female</td>
<td>26</td>
<td>0,47</td>
<td>II</td>
<td>LBP; R sciatica.</td>
<td></td>
</tr>
<tr>
<td>87.</td>
<td>female</td>
<td>56</td>
<td>0,44</td>
<td>II</td>
<td>LBP; L sciatica; L leg weakness; L feet numbness.</td>
<td></td>
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<tr>
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<td>1,17</td>
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<td>LBP</td>
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<td>0,5</td>
<td>II</td>
<td>LBP; R sciatica; R leg weakness.</td>
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</tr>
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<td>51</td>
<td>1,1</td>
<td>I</td>
<td>LBP</td>
<td></td>
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</tbody>
</table>

DuS - dural sac; EF - extradural fat; Spi C - Spinal canal; SEL - spinal epidural lipomatosis; LBP - low back pain; R - right; L - left.
CONCLUSIONS

1. Spinal Epidural lipomatosis is a rare medical condition, with a prevalence of 1.73%.

2. Men and women of middle age are equally affected by SEL.

3. Grade II SEL was the most frequent form.

4. Clinical presentation of the patient is directly correlated with MRI grade of SEL. All patients complained of lower back pain.
REFERENCES


