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Retrospective Analysis and Literature Review of a Case of Spinal Epidural Lipomatosis

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Abstract: Spinal epidural lipomatosis (SEL) refers to the pathological overgrowth and accumulation of fat tissue within the spinal canal, forming an intraspinal space-occupying lesion. It is often associated with other spinal surgical conditions. This study retrospectively analyzes the clinical data of a patient diagnosed with SEL at Wuhan Hospital of Traditional Chinese Medicine on April 18, 2024, and reviews relevant literature. The study focuses on the etiology, clinical manifestations, diagnosis, and treatment of SEL to provide insights for clinical practice.

Keywords: Spinal epidural lipomatosis; Steroid hormones; Spinal stenosis; Spinal decompression surgery

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1. Introduction

Epidural fat (EF) refers to the adipose tissue filling the epidural space within the spinal canal. The dura mater, nerve roots, blood vessels, lymphatic vessels, and filum terminale within the spinal canal are all surrounded by EF, allowing for mutual sliding between these structures, thereby providing lubrication and stress-buffering protection [1]. EF contains mesenchymal stem cells (MSC), and extracellular vesicles derived from EF-MSC can improve neurological recovery after spinal cord injury by reducing the expression of inflammatory cytokines. Spinal epidural lipomatosis (SEL) is characterized by pathological hyperplasia and excessive accumulation of fat tissue within the spinal canal, leading to compression of the dural sac, spinal cord, cauda equina, and nerve roots, resulting in corresponding clinical symptoms. SEL is relatively rare in clinical practice and is often misdiagnosed or overlooked due to its similarity to common intraspinal space-occupying conditions such as lumbar disc herniation or ligamentum flavum hypertrophy. The authors retrospectively analyzed the clinical data of a patient with SEL treated at Wuhan Hospital of Traditional Chinese Medicine to provide references for clinical diagnosis and treatment.

2. Case presentation

The patient is a 56-year-old male who was admitted to the hospital due to "low back pain accompanied by soreness, numbness, and weakness in both lower extremities for 5 years, with symptoms worsening over the past month." The patient developed soreness and pain in the lower back without an obvious cause 5 years ago, accompanied by soreness, numbness, pain, and weakness in both lower limbs, as well as restricted mobility. He was diagnosed at an external hospital with "cervical spondylotic myelopathy" and "lumbar disc herniation." He underwent conservative treatments, cervical open-door surgery, and lumbar minimally invasive surgery, which initially relieved his symptoms. However, he continued to experience intermittent episodes, with symptoms worsening after exertion and slightly alleviated with rest. One month ago, his symptoms progressively worsened without an apparent cause, leading to difficulty walking and intermittent claudication, with no significant relief after rest. Seeking systematic treatment, he visited Wuhan Traditional Chinese Medicine Hospital on April 18, 2024, and was admitted to the Orthopedics and Traumatology Department, Hankou Third Ward, with the outpatient diagnosis of "lumbar disc herniation."

The patient has a history of hypertension for over 10 years, managed with long-term oral felodipine tablets, but blood pressure control was poor at admission. The patient also has a history of diabetes mellitus, managed with oral metformin tablets and subcutaneous insulin injections, but blood sugar control was also poor upon admission. The patient denied history of other underlying diseases. The patient underwent cervical single-door laminoplasty in 2019 and minimally invasive lumbar surgery in 2023 at an external hospital, but relevant surgical data was lost. There was no history of trauma, blood transfusion, or drug allergies. Physical examination revealed that the patient was in good general condition, with a height of 175 cm, weight of 100 kg, and BMI of 32.6 kg/m^2. The lumbar physiological curvature was flattened, with mild scoliosis. There was mild tenderness adjacent to L4/5 and L5/S1, no percussion pain in the lumbar region, positive deep tenderness in the right buttock, no significant tenderness in the bilateral sacroiliac joints, tenderness along the sciatic nerve trunk in both lower extremities (right: +, left: +), straight leg raising test (right: 50°, left: 60°), positive Bragard's test (right: +, left: +), negative bilateral "4" test, negative bilateral piriformis muscle stretch test, Grade 5-muscle strength in both lower extremities, Grade 5 muscle strength in both upper extremities, normal and symmetrical muscle tone in all extremities, normal and symmetrical knee and Achilles tendon reflexes, negative Babinski sign, normal skin temperature in both lower extremities, palpable dorsalis pedis arteries, and adequate peripheral circulation.

MRI of the lumbar spine indicated degenerative changes, with L2, L3, and L5 vertebral bodies showing posterior spondylolisthesis (Grade I). There was degeneration of the L2/3–L5/S1 intervertebral discs, bulging and herniation of the T12/L1 disc (central type), bulging and mild herniation of the L2/3 disc (central type), bulging of the L3/4 disc, and bulging and herniation of the L4/5 and L5/S1 discs (left lateral type), resulting in narrowing of the left lateral recess. Stenosis of the spinal canal was observed at the L4/5 level. Synovial cysts were considered adjacent to the right facet joints of L4/5 and the left facet joints of L5/S1. There was edema in the subcutaneous soft tissue of the waist, and inflammation of the interspinous ligaments from L2/3 to L5/S1. After departmental discussion, the patient's condition was communicated with the patient and family. Surgical treatment was recommended, and both the patient and family requested surgery. The patient's general condition was stable in the perioperative period, with well-controlled blood pressure and blood glucose. Preoperative examinations showed no significant contraindications to surgery. Thus, the following procedure was performed: posterior lumbar discectomy for herniated disc + spinal canal decompression + interbody fusion with cage

implantation + pedicle screw fixation.

The surgical procedure is as follows: After successful general anesthesia, the patient was placed in the prone position, and routine disinfection and draping were performed. A midline longitudinal incision of approximately 12 cm was made with the lower edge of the L3 spinous process as the center. A micro radiofrequency tungsten needle electrode was used to incise the skin, superficial fascia, and lumbar dorsal fascia. The bilateral erector spinae muscles were dissected and stripped laterally along the L2–L5 spinous processes to expose the bilateral laminae, facet joints, and parts of the transverse processes of L2–L5.

Using C-arm X-ray fluoroscopy for confirmation, two guide pins were inserted bilaterally at L4 with an approximately 15-degree angle to the sagittal plane and parallel to the transverse plane. Similarly, two guide pins were inserted bilaterally at L5 with the same angulation. The positioning was confirmed to be satisfactory via C-arm fluoroscopy. A total of four 6.5 × 50 mm universal pedicle screws were inserted bilaterally at L4 and L5.

After confirming positioning via C-arm fluoroscopy, an ultrasonic bone scalpel was used to remove part of the left L4 lamina, the inferior articular process, and parts of the superior articular process and lamina of L5, exposing the ligamentum flavum and dura mater. A large amount of adipose tissue was observed in the spinal canal. The L4–L5 segment dura mater was visibly compressed, appearing white with reduced pulsation. The left L5 nerve root was found to be congested and edematous.

The left nerve root canal was expanded, and the nerve root was carefully mobilized medially. Bipolar coagulation was used for hemostasis, and the herniated nucleus pulposus was removed entirely, ensuring clearance within the intervertebral space. The nerve root canal was checked and found to be unobstructed and decompressed. Bone grafting was performed in the L4/5 intervertebral space, and a suitable cage (2.2 × 1.3 cm) was implanted. C-arm fluoroscopy confirmed good cage positioning.

An ultrasonic bone scalpel was then used to remove the middle and lower parts of the right L2 lamina and part of the right L3 lamina while preserving the L2 and L3 isthmus. After removing the lamina, hypertrophic ligamentum flavum, adipose tissue, and significant compression on the dura mater were observed. The hypertrophic ligamentum flavum and adipose tissue were meticulously removed, revealing a pulsating and expanded dura mater.

Two connecting rods were installed on the pedicle screws, and four locking nuts were sequentially tightened. C-arm X-ray fluoroscopy confirmed satisfactory internal fixation positioning. A final instrument and gauze count was performed. The surgical field was thoroughly irrigated with a large amount of normal saline, and hemostasis was achieved using absorbable hemostatic fleece. A drainage tube was placed in the incision, and the wound was closed layer by layer.

The surgery was completed successfully, with approximately 650 ml of intraoperative blood loss. 400 ml of autologous blood was reinfused intraoperatively. The patient remained hemodynamically stable and was safely transferred back to the ward. Vital signs were monitored postoperatively. The patient received anti-inflammatory, analgesic, infection prevention, gastric protection, neurotrophic support, and fluid replacement therapy. The preoperative MRI images (**Figure 1**) and postoperative lumbar X-rays **(Figure 2**) are as follows:

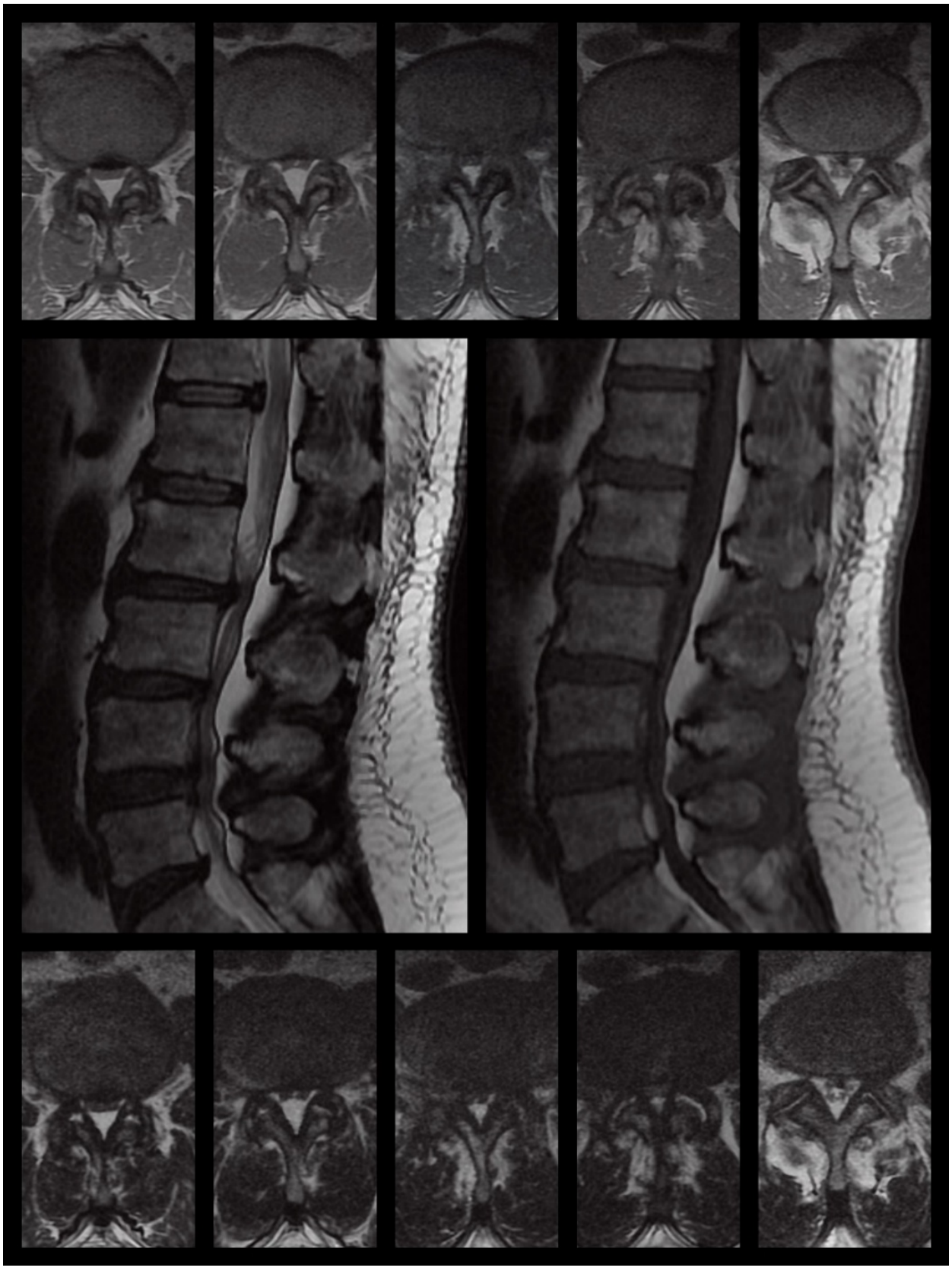

Figure 1. Preoperative MRI images

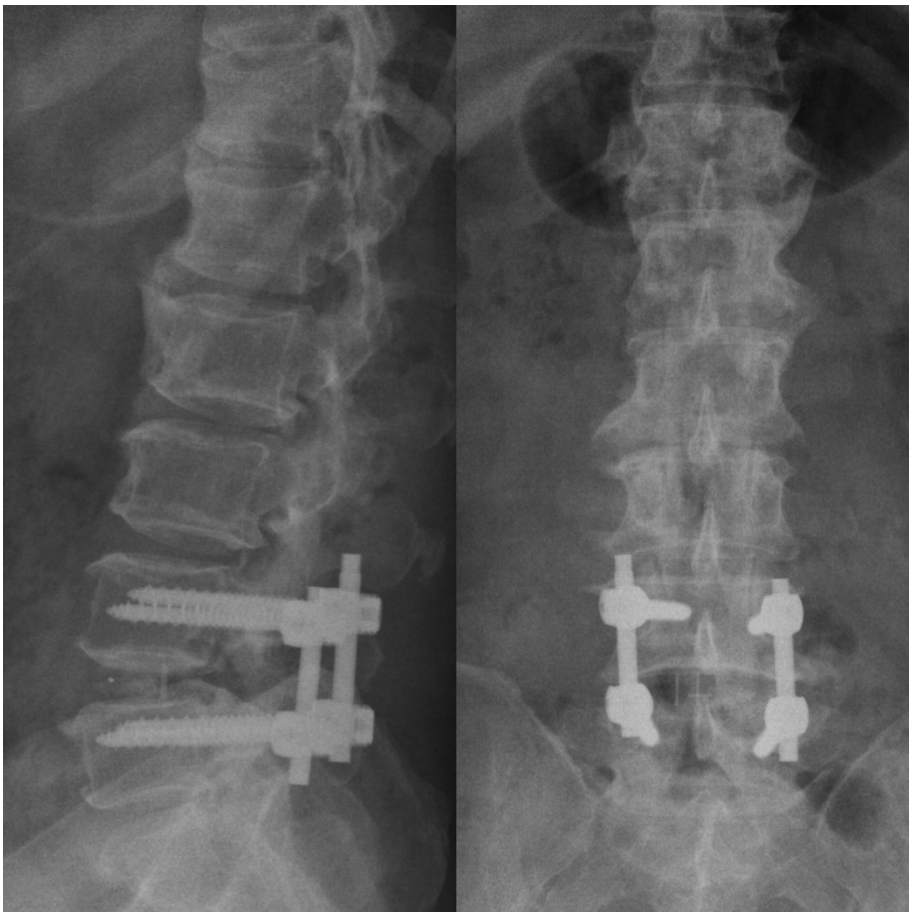

Figure 2. Postoperative lumbar X-rays

3. Literature review

3.1. Etiology of SEL

Spinal epidural lipomatosis (SEL) refers to a space-occupying lesion within the spinal canal caused by the pathological proliferation and excessive accumulation of EF. It is characterized by the compression of spinal canal contents such as the dural sac, spinal cord, cauda equina, and nerve roots, leading to varying degrees of lower back pain, unilateral or bilateral limb soreness, numbness, swelling pain, cauda equina syndrome, intermittent claudication, and other neurological compression symptoms.

The exact pathogenesis of SEL remains unclear. The prevailing view is that excessive corticosteroid hormones, whether due to exogenous corticosteroid use or increased endogenous cortisol, are the primary causes of SEL [2]. Increased endogenous cortisol is usually secondary to conditions such as Cushing's syndrome, hypothyroidism, hyperprolactinemia, and diabetes. Non-steroid-related SEL is considered idiopathic epidural lipomatosis and is often associated with obesity, metabolic syndrome, and chronic inflammation. Ishihara *et al.* [3] conducted a statistical evaluation of the correlation between SEL and metabolic syndrome or metabolic-related diseases, finding that the degree of EF accumulation was significantly associated with BMI, abdominal circumference, and visceral fat area, suggesting a relationship between EF deposition and metabolic diseases. Greenish *et al.* [4] reported the first case of acute SEL following spinal surgery and suggested that SEL should be considered a postoperative complication of spinal surgery. Currently, various hypotheses exist regarding the

etiology and pathogenesis of SEL, and further research is needed to supplement and clarify these aspects.

3.2. Clinical manifestations and diagnosis of SEL

SEL has a higher incidence in males and most commonly occurs in the thoracolumbar region and at the S1 level, with the L5/S1 segment being particularly affected. It is rarely observed in the cervical region or below the S2 level. A small number of asymptomatic SEL cases have also been reported. Salna Iv *et al.* [5] described a case of a patient who experienced three days of right upper and lower limb weakness, initially evaluated for a stroke but ultimately diagnosed with SEL. This case was the first to suggest that SEL could present with stroke-like prodromal symptoms, highlighting the need for careful clinical diagnosis and differential diagnosis.

MRI can clearly visualize excessive epidural fat within the spinal canal. Fat tissue appears as a high signal intensity on both T1WI and T2WI sequences and as a low signal intensity on fat suppression sequence images. Combined sagittal and axial imaging can accurately display the distribution and morphology of excess fat within the spinal canal, as well as the degree of compression on the dural sac and spinal cord. Ge *et al.* [6] noted that the distribution of EF varies across spinal segments, and using a fixed MRI threshold to diagnose SEL at different spinal locations may be inaccurate.

3.3. Treatment of SEL

For patients with a lower grade of EF accumulation, mild and stable clinical symptoms, or asymptomatic cases, conservative treatment is recommended. This primarily involves treating the underlying condition, reducing or discontinuing exogenous corticosteroids, encouraging weight loss, optimizing pain management, and utilizing physical therapy. Surgery is considered only when conservative treatment proves ineffective. The primary surgical approach for SEL is decompression surgery, which includes laminectomy and epidural fat resection [7]. In pediatric SEL patients, laminoplasty is the preferred option to prevent future spinal deformities [8]. Minimally invasive procedures, such as unilateral biportal endoscopic decompression, have also been applied in SEL treatment, achieving decompression effects and improving clinical symptoms.

4. Discussion and conclusion

Reviewing this case, the patient was overweight, with a BMI of 32.6 kg/m^2, and had a history of “cervical spondylotic myelopathy” and “lumbar disc herniation.” The spine was in a state of chronic inflammation, and the patient also had a long history of diabetes, indicating an underlying metabolic disorder. Additionally, the patient had undergone spinal surgery in the past. Although the surgical records were lost, standard spinal surgery typically involves the use of a certain amount of corticosteroids for anti-inflammatory treatment. Combining these factors with the content discussed above, the etiology of SEL in this case closely aligns with what has been reported in related literature.

From an imaging perspective, the patient’s DuS/EF was ≤ 0.33, and EF/SpiC was $\geq 75\%$, meeting the criteria for SEL grade III. Axial images showed that the patient’s dural sac was compressed into an oval or star-shaped deformation. Since the radiologists at our hospital had not previously encountered such cases, the initial imaging report did not describe the increased fat content. However, after discussing this case with the radiology department, subsequent imaging reports for some patients included descriptions of epidural fat accumulation in the spinal canal. This case also contributed to the experience and knowledge base of our hospital’s radiology

department.

Due to the patient's concurrent lumbar disc herniation and progressively worsening symptoms, the department decided on surgical treatment. The procedure included posterior lumbar discectomy, spinal canal decompression, cage-assisted interbody fusion, and pedicle screw fixation. Intraoperatively, the hypertrophic adipose tissue was resected. However, this case had certain limitations. Since the patient sought treatment in a different city, postoperative MRI follow-up was not feasible. Follow-up was conducted via telephone, where the patient reported normal walking ability and expressed high satisfaction with the surgical outcome. In conclusion, SEL is a relatively rare clinical condition, and further research is needed to deepen our understanding. Clinicians should enhance their knowledge and awareness of this disease to avoid missed or incorrect diagnoses.

Disclosure statement

The authors declare no conflict of interest.

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