Worsening Epidural Lipomatosis Leading to Foot Drop Following an Epidural Steroid Injection: A Case Report

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ABSTRACT

Introduction: Epidural lipomatosis is a relatively rare condition resulting in the accumulation of unencapsulated fatty tissue within the epidural space. Steroids, either exogenous or endogenous, have been reported as a cause for this accumulation. The diagnosis is confirmed by computed tomography or magnetic resonance imaging. Symptomatic epidural lipomatosis has been reported to present with radiculopathy, myelopathy, claudication, cauda equina syndrome, or paraplegia. It is usually managed conservatively, including weight loss and avoidance of steroids.

Case Presentation: We report the case of a patient with sarcoidosis on oral prednisone who was referred for low back and leg pain of multifactorial origin. After addressing his low back pain, a fluoroscopically guided lumbar epidural steroid injection was performed for his neurogenic claudication. This provided 3 months of complete pain relief. But the patient also developed unilateral foot drop, possibly secondary to worsening epidural lipomatosis.

Conclusions: Epidural lipomatosis may result in complications that include neurological deficits. Although various disease states may cause it, prudence is advised in the use of exogenous steroids.

INTRODUCTION

Epidural lipomatosis (EL) is the excess accumulation of unencapsulated adipose tissue in the epidural space.[1] The first reported documentation of EL was in 1975 by Lee et al in a patient on steroids following renal transplant.[2]

Although it may be an incidental finding with nonspecific symptoms, EL also has been associated either with or resulting in myelopathy, radiculopathy, sensory deficits, spinal stenosis and, rarely, cauda equina syndrome.[3]

We report the case of a patient on oral steroids for sarcoidosis who developed a foot drop with worsening EL incidentally following an epidural steroid injection for spinal stenosis.

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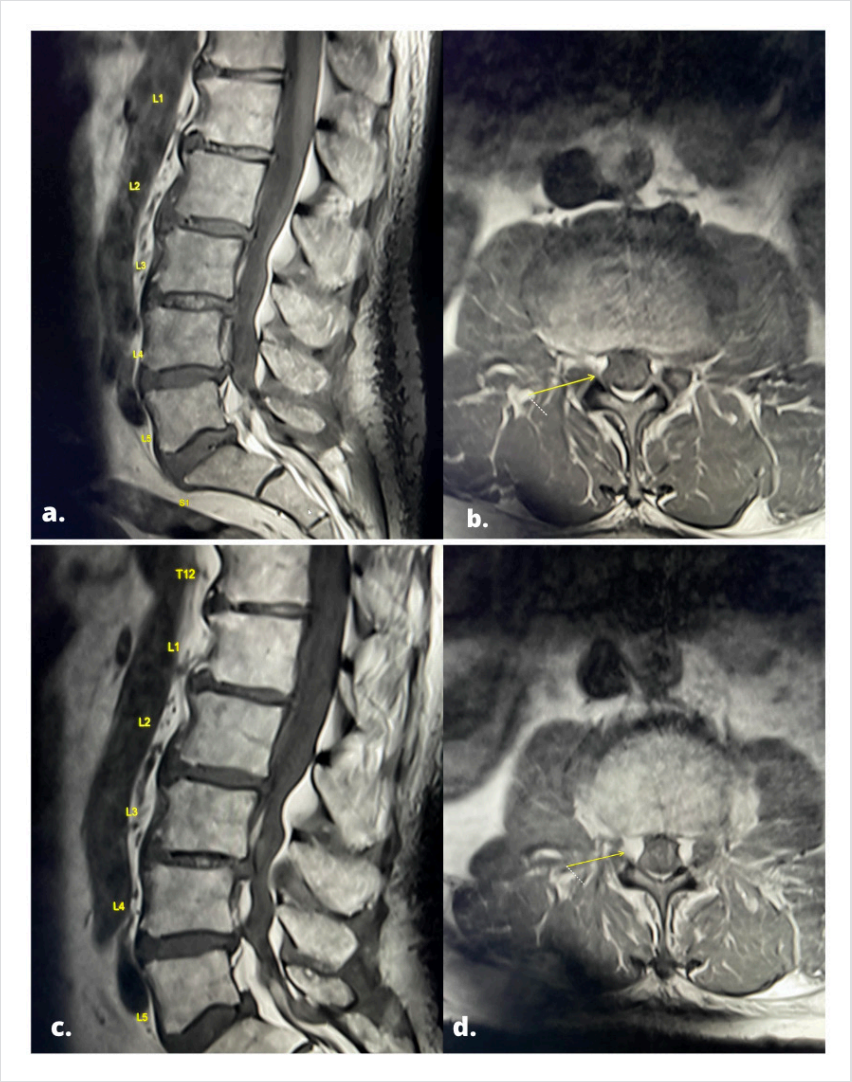
CASE PRESENTATION

A 64-year-old man, with a body mass index of 31, presented with acute-on-chronic low back pain with occasional pain shooting to his knee. He described the left > right pain as constant, dull, and achy. The pain decreased marginally when he leaned forward. He described having worsening pain on standing for a prolonged time, as well as getting up from a seated position. He also was limited in his ability to walk more than a block because of his leg pain. He was managing this pain with exercises and ibuprofen as needed. His past medical history was significant for sarcoidosis, on nightly home oxygen and oral prednisone 20 mg daily, and diabetes on metformin 1000 mg twice a day.

On examination, the patient had tenderness over bilateral sacroiliac joint and over the low lumbar paraspinal area in the possible location of lower lumbar facet joints. His straight leg raise was negative and FABER (flexion, abduction, and external rotation) was positive bilaterally. The rest of his examination was within normal limits. Lumbar spine magnetic resonance imaging (MRI) revealed severe central canal stenosis from L4-S1, multilevel bilateral facet hypertrophy, ligamentum flavum thickening, severe multilevel bilateral foraminal stenosis from L2 to S1, and EL at L5-S1. Given the physical examination findings, along with worse low back pain and based on his preference, we performed therapeutic facet joint intraarticular steroid injection at L5-S1 levels bilaterally and decided to schedule him for diagnostic medial branch blocks at a later date.

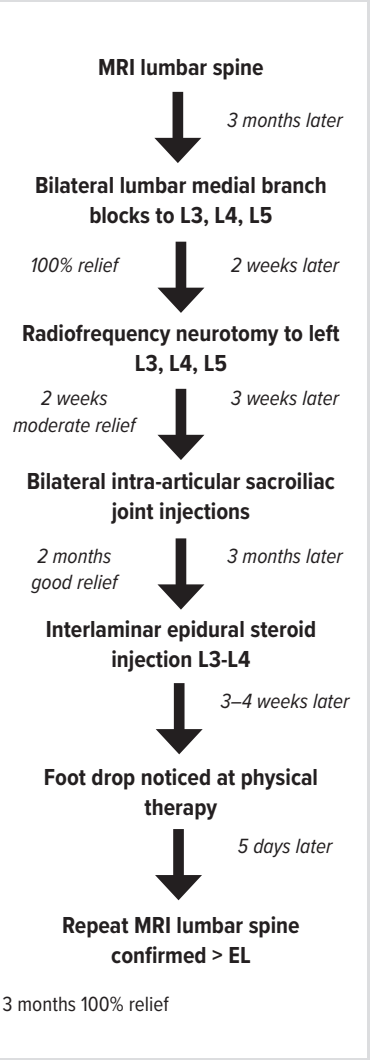
After 2 months, we performed bilateral lumbar medial branch

Figure 1. T1 weighted Sagittal and Axial Images via Magnetic Resonance Imaging Prior to Steroid Injection (A and B) and One Month After Epidural Steroid Injection (C and D)



Arrow pointing to epidural fat deposits

Figure 2. Timeline



Abbreviation: EL, epidural lipomatosis; MRI, magnetic resonance imaging.

blocks at levels 3-5 and, noticing appropriate short-term pain relief, went on to perform a thermal radio frequency ablation of his left lumbar medial branches 3-5 after 2 weeks, as his left side was causing him more pain. On his subsequent visit 3 weeks later, his pain was mostly on rising from sitting to standing with tenderness over the sacroiliac joints bilaterally and no pain over the lumbar area. We performed bilateral ultrasound-guided sacroiliac joint steroid injections with 10 mg of triamcinolone into each joint.

At his 2-month follow-up, the patient's most bothersome pain was the leg pain with features predominantly of neurogenic claudication, including pain behind the thighs and calf worsened with prolonged walking and relieved with rest. Hence, we performed an intralaminar epidural steroid injection (ESI) above the area of severe spinal stenosis at L3-4 using 40 mg of methylprednisolone with 4 ml of preservative-free normal saline. This provided him significant relief from his leg and back pain and facilitated participation in physical therapy.

Several weeks after the ESI, the physical therapist noticed a foot drop. Repeat MRI was ordered and revealed diffuse worsening of his EL from L2-S1 resulting in worsening severe canal stenosis and likely the cause of his foot drop (Figure 1). He was then referred to physiatrists for suitable orthotics (Figure 2). Despite his requests to have the ESI repeated again, which provided him the maximum pain relief, we elected to adopt conservative measures including weight reduction, as he was unable to reduce his prednisone for sarcoidosis. He currently manages his pain with tramadol 3 times daily and has decreased his weight. He continues with his home exercise program but is currently limited by the development of atypical mycobacterial pneumonia.

DISCUSSION

We present a case of foot drop after an epidural steroid injection in a patient with prior documented EL. Besides his body habitus and obesity, the patient's potential for developing EL was that he was

on oral steroids daily for sarcoidosis. He had MRI-documented EL, increasing his chances of worsening of EL.

The prevalence of EL has been reported to be between 1.1% and 6.2%[4,5] and is more common in men. Its cause may be unknown/idiopathic in 17% of patients.[6] Besides obesity, its causes include both exogenous steroid therapy and endogenous overproduction of steroids. Exogenous steroid therapy-related EL (>50% of cases) occurs in patients with organ transplantation, Crohn's disease, ulcerative colitis, and nephritic syndrome; endogenous overproduction of steroids-related EL is seen in Cushing's syndrome, hypothyroidism, carcinoid syndrome, and pituitary prolactinoma.[2,6,7] Other reported possible scenarios include patients on highly active retroviral therapy and Scheuermann's disease.[8] It is possible that etiology may determine the location/level of the EL in the spine. Interestingly, obese patients—in whom EL is more common—have an increase in inflammatory markers such as interleukin-1β and TNF-α that are responsible for enhanced adipose tissue growth.[7]

Computed tomography or MRI imaging is the usual means of identifying EL. Besides volumetric analysis, epidural fat (EF)/antero-posterior diameter of the dural sac also has been proposed as a measure to gauge the severity of EL. Epidural fat in excess of 6 mm also has been proposed as an indicator of EL.[9] In late stages of EL, "Y" sign or polygonal deformations of the dura are noted.[10] EL has been reported to be more frequent in the thoracic than lumbosacral spine with exogenous steroid use, whereas EL related to endogenous steroid affects the thoracic and lumbosacral spine equally.[7]

Previous studies have noted that patients with EL have baseline neurological deficits, with a cohort reporting up to 14.0% of patients having a motor deficit.[1] The only studies thus far demonstrating neurologic weakness in association with EL have reported acute paraplegia following exogenous steroid administration, but there was also documentation of osteoporotic vertebral fractures making it difficult to establish a cause and effect relationship.[6,11] Although a positive correlation between the number of ESI and EL has been demonstrated, no threshold dose of cumulative steroids has been established thus far.[12,13] Interestingly, despite some confounding factors, a few reports have demonstrated resolution of EL-related radicular symptoms with an ESI or by a series of ESIs.[14,15] Symptom severity may be associated with delayed recovery.

Most often, EL is an incidental finding requiring no major interventions. Management of EL associated with neurological injuries is individually tailored with conservative management, including weight loss, decreasing steroid use when possible, and management of the primary condition such as supplementing with thyroid hormones in patients with hypothyroidism. When unsuccessful, some patients have opted for decompression surgery with good results.

CONCLUSIONS

This is a report of foot drop caused by worsening epidural lipomatosis secondary to steroids chronologically following an epidural steroid injection. This report highlights the need for prudence in the use of exogenous steroids, especially when evidence of epidural lipomatosis is present. Although cause and effect cannot be established, cautious use of steroids is recommended to avoid such complications.

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