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Case Reports & Case Series

Spinal epidural lipomatosis due to obesity presenting with back and leg pain and weakness successfully treated with weight loss

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ABSTRACT

Spinal Epidural Lipomatosis (SEL) is an uncommon but previously characterized ailment typified by excessive overgrowth epidural adipose tissue. This overgrowth may lead to compression of local tissue including the underlying thecal space and nervous tissues. Common causes are medication induced (particularly glucocorticoid administration) and obesity. Prompt recognition of the diagnosis and initiation of treatment, be it medication stoppage or surgical decompression is imperative. In this report we describe the case of a 51-year-old female who presented with back and leg pain. She was diagnosed with SEL due to obesity with noted compression of the underlying nervous tissue noted on MRI. The patient was successfully treated with a course of weight loss with remarkable clinical and noted resolution of thecal sac compression on follow up MRI. Early consideration of this entity can increase the likelihood of timely diagnosis and successful treatment.

1. Introduction

Spinal Epidural Lipomatosis (SEL) is an uncommon but known condition characterized by excessive overgrowth of normal adipose in the epidural space [1,2]. This overgrowth can compressive the underlying thecal space and nervous tissues [3,4]. Common causes are medication induced (particularly glucocorticoid administration) and obesity [1,2,5]. Prompt recognition of the diagnosis and initiation of treatment is imperative [1,3,6]. In this report we describe the case of a 51-year-old female who presented with back and leg pain diagnosed with SEL and successfully treated with a course of weight loss. The patient presented herein has provided written informed consent for the use of her medical narrative.

1.1. Case presentation

A 51-year-old obese woman presented to the emergency department reporting a 6-month history of progressively worsening back and leg pain along with subjective bilateral lower extremity weakness. She did not have bowel or bladder symptoms. She reported a history of hypertension and hypercholesterolemia, as well as recent coronary stent placement 2 months earlier requiring dual anti-platelet therapy with aspirin and clopidogrel. She was not taking any steroid medications. Current weight was 100.2 kg with the BMI of 36.8 kg/m^2. On examination she was awake, alert, and oriented. Her cranial nerves were intact. There was full strength in the bilateral upper extremities. There was 4+/5 strength in her lower extremities. Sensation was decreased below the knees symmetrically. Deep tendon reflexes were all symmetric and physiologic, except a decrease in patellar reflex bilaterally. There was no Babinski sign, Hoffman's sign, or ankle clonus noted. Vital signs were normal and she was afebrile. Serum electrolytes, glucose, complete blood cell count, and urinalysis were all within normal limits. A T1 and T2-weighted magnetic resonance imaging (MRI) revealed lumbosacral spinal epidural lipomatosis with anterior displacement and significant compression of the thecal sac (Fig. 1A, B, and D). A diagnosis was given of symptomatic spinal epidural lipomatosis secondary to obesity.

Decompressive surgery was considered, but conservative management was pursued due to the longstanding mild nature of the symptoms as well as the dual anti-platelet therapy required for the recent coronary stent placement. At 4-month follow-up, she had lost over 10 kg with a new weight of 86.6 kg and BMI of 31.8 kg/m^2. Her pain had completely abated. She continued to have 4+/5 strength in her lower extremities. Repeat imaging revealed reduction of the epidural adipose tissue and resolution of thecal sac compression (Fig. 1C and E).

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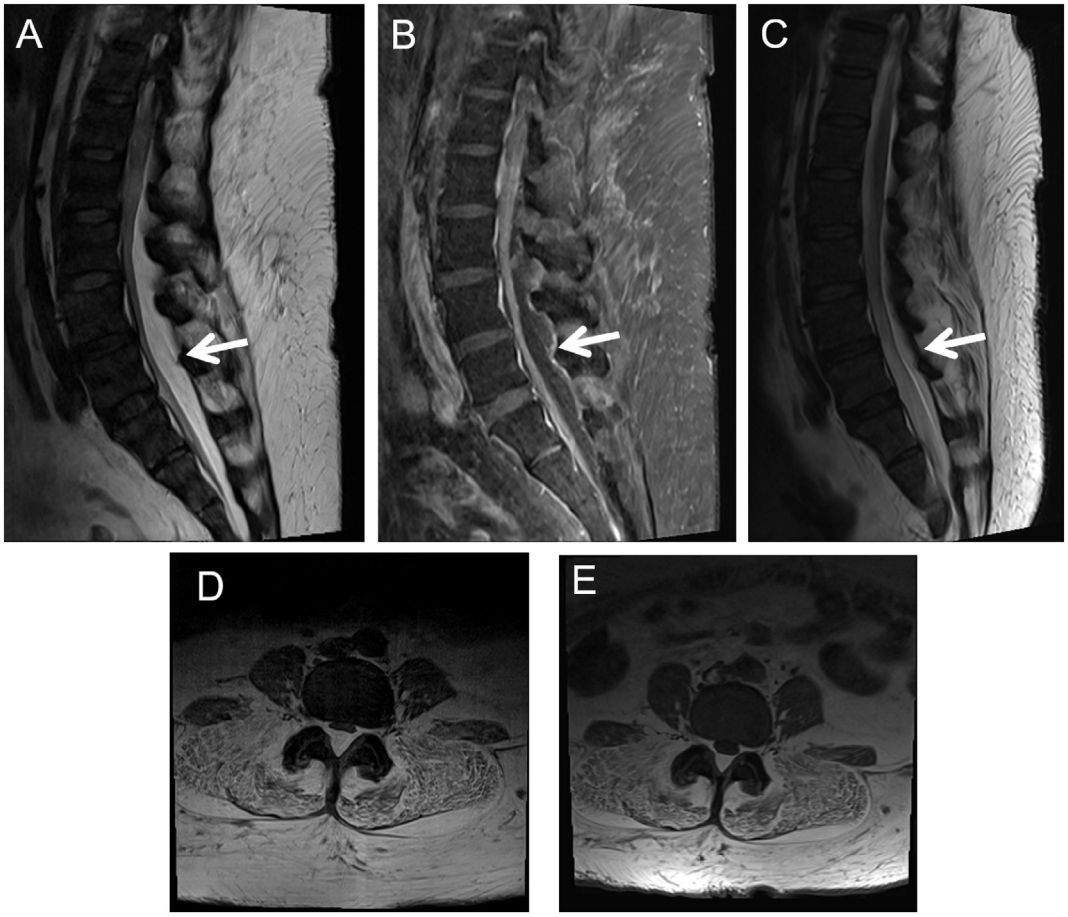

Fig. 1. T2 sagittal sequence shows significant compression and anterior displacement of the thecal sac by epidural adipose tissue (A, arrow and D), confirmed to be adipose tissue based on signal dropout on T1 sequence with fat suppression (B, arrow). T2 sequence obtained 4 month later shows significant reduction of epidural fat compression (C and E).

2. Discussion

The key clinical feature in this case is recognition of significant compression of the thecal sac (noted as areas of low signal intensity on MRI T1 sequence), which contains the spinal cord and nerve roots, by excessive amount of the spinal epidural fat (noted as areas of high signal intensity on MRI T1 sequence). Although surgical intervention is to be considered in patients with acute/severe neurological deficits [7], conservative management with weight reduction is the appropriate management in idiopathic cases with pain as the primary presenting symptom [8].

Symptomatic spinal epidural lipomatosis (SEL) is a rare presentation of abnormal overgrowth of epidural fat (normally present in small amount) within the spinal canal causing neurological symptoms first described in 1982 [2,3,7,9]. Fatty overgrowth is most commonly intradural, but may also be intramedullary or extradurally [7]. SEL symptoms, in descending order of commonality, include pain, weakness, numbness, and bowel or bladder dysfunction [6]. These symptoms may present in an abrupt or progressive manner [3].

SEL can be categorized as either idiopathic/primary (associated with obesity) or secondary (associated with an endocrinopathy or steroid use) [3]. Examples of secondary SEL include Cushing's syndrome, hyperprolactinemia, adrenal tumor, and exogenous steroid administration [3,10]. Increased fatty deposits within the epidural space are induced via this excessive cortisol signaling. Exogenous steroid use is by far the most common culprit for SEL, with one series noting it being responsible for just over half of its observed cases [3]. Ruling out exogenous steroid administration is an important step in the workup of SEL.

The second most common cause is obesity and metabolic disorders [9]. The idea of a direct relationship of obesity with fatty overgrowth within the spinal canal was first proposed in a 1991 manuscript [5]. In one large retrospective series of 104 cases of SEL, 24.5% instances were linked to obesity alone [3]. Given its link with obesity, SEL is expected to become more common with the current obesity epidemic.

Treatment of SEL is dictated based on patient presentation. With no acute neurological deficits, conservative management consisting of removal of the offending medication or a structured weight loss program should be initiated. In the setting of obesity, the effectiveness of weight loss and/or bariatric surgery has been previously shown [6,8]. If there is an acute development of neurological deficit or bowel/bladder symptoms, emergent surgical decompression may be required [1–3]. Decreasing the time to diagnosis and initiation of appropriate treatment will decrease the chance of patients requiring invasive interventions.

Declaration of funding and competing interests

All the above authors declare that they have no competing interests or funding conflicts of interest. This manuscript in part or whole is not currently published or under consideration for publication with any other journal or publication house.

Authorship statement

This final manuscript has been reviewed and approved by all of the above co-authors prior to submission.

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