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Case Report

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Spinal Epidural Lipomatosis Due to the Corticosteroids; A Case Report

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Abstract

Spinal epidural lipomatosis (SEL), an abnormal localized or tumor-like accumulation of fat in the epidural space, is an infrequent complication of chronic steroid usage and an uncommon cause of spinal cord compression. A patient with a history of rheumatologic condition on chronic corticosteroids presented with a clinical picture of thecal sac compression and was diagnosed with SEL.

1. Background

Spinal epidural lipomatosis (SEL) is a condition in which adipose tissue accumulates excessively inside the spinal canal, causing the neural element to become compressed. The incidence of symptomatic SEL is extremely rare and often associated with the use of exogenous steroids. SEL has also been associated with obesity and Cushing syndrome/disease (hypercortisolism), although they are less common. On rare occasions, epidural lipomatosis causes nerve root compression or spinal cord compression, leading to symptoms. There is a high rate of misdiagnosis of SEL because it can mimic the symptoms of spinal stenosis or degenerative joint disease [1]. The symptoms of spinal cord compression are primarily determined by the level of compression in the spinal cord. Men are more likely to experience SEL than women, with a ratio of 3:1, and the average age at presentation is 43 [2]. The diagnosis of SEL can be best established with magnetic resonance imaging (MRI). The diagnostic criterion for SEL is an epidural adipose tissue thickness exceeding 7 mm [3,4]. We represent a case of spinal epidural lipomatosis due to the treatment with corticosteroids.

2. Case

A 48-year-old man was admitted to Al-Zahra University Hospital in Isfahan, Iran with complaints of progressive weakness and numbness of the right lower limb. The weakness progressed gradually from one year before admission in an ascending fashion to involve the lower limbs. His symptoms had worsened rapidly over a period of two weeks preceding his admission to our hospital, to the extent that he had difficulty moving his right lower limb. His past medical history was a rheumatologic condition, with the history of five mg of prednisolone daily in the last eight years. Physical examination on admission revealed a well-nourished male patient who weighed 95 kg and measured 180 cm tall (body mass index, 29.3). Neurological examination of his cognitive function and cranial nerves were within normal ranges. Motor examination of the lower limbs revealed a significant weakness. Sensory examination showed hypoesthesia and impaired proprioception in right lower limb, with decreased sensation. The motor and sensory examination of the upper limbs was normal. On investigation, routine laboratory blood tests including complete blood count, liver function, and renal function analysis were within normal ranges. An MRI study of the lumbar and sacral spine was done and the radiologist reported fatty degeneration of paraspinal muscles and spinal lipomatosis in L4-L5 and L5-S1. They also reported extradural lipomatosis with compression effect on thecal sac in the same area (Figure 1&2).

The patient underwent posterior laminectomy from L4 to S1 vertebral levels. A dorsally located and relatively vascular firm epidural fatty tissue was encountered, which was not infiltrating the bone or the dura; it was dissected hardly and incompletely removed due to its stickiness. Histological examination of the operative specimen demonstrated vascularized mature fibro-fatty tissue consistent with the diagnosis of lipomatosis. The patient had an uneventful postoperative period. The patient was able to walk with assistance two days after surgery and within a week, he was experiencing gradual improvement in his paraparesis and his motor function. He was discharged on the 7th postoperative day. He continued to show progressive neurological improvement with extensive outpatient physiotherapy program and finally was able to walk independently after 10 days. At 3-month follow-up, his examination revealed a complete neurological function.

Figure 1: T1 and T2-weighted magnetic resonance images of individual included in this study. The images showed complete CSF block in L4-L5 and L5-S1 levels

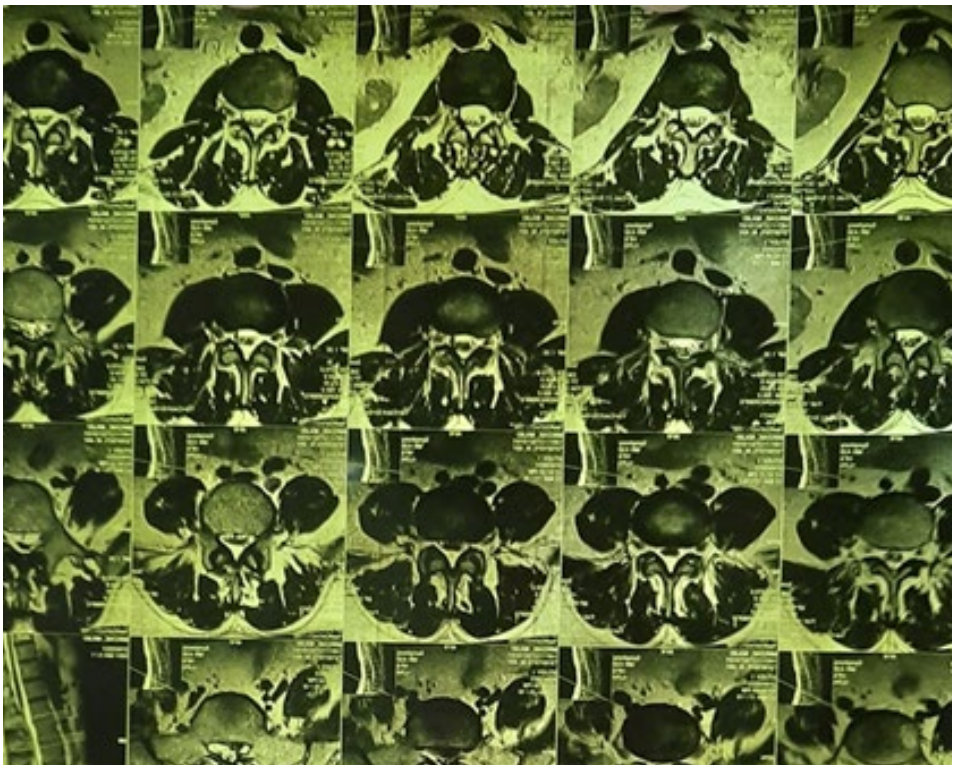

Figure 2: T2-weighted magnetic resonance images of individual included in this study. The images sac compression in L4-L5 and L5-S1 levels

3. Discussion

Lipomatosis is an abnormal localized or tumor-like accumulation of fat in tissue. This abnormal collection of fat in the epidural space, known as epidural lipomatosis, is an uncommon complication of chronic steroid usage, Cushing's disease, and obesity [5]. Normal epidural fat responds to steroid use with progressive hypertrophy, but there is no clear rationale why it is more evident with exogenous steroid than in Cushing's disease. It is believed that corticosteroids cause epidural lipomatosis by inducing hypertrophy of adipose tissue normally present in the epidural space of the spinal cord [6]. SEL most commonly occurs in the thoracic region due to narrow cord width, limited vascularity, and a large portion of epidural fat normally in that area. It is less common in the lumbar region and has not yet been reported in the cervical region. Clinical presentations differ with the anatomical location of the epidural lipomatosis. However, back pain and paraparesis are the most common symptoms, with decreased sensation and altered reflexes occurring infrequently. Signs and symptoms have an insidious onset but may occur acutely [7].

Treatment options include surgical or conservative methods depending on the patient's neurological assessment. A slower resolution of symptoms is possible with conservative management, including weight loss and a reduction or discontinuation of the corticosteroids. A permanent neurological deficit is possible, so close monitoring of the patient's neurologic symptoms is important. For progressive neurologic symptoms, a spinal decompression with excision of the excess fatty tissue is required [8].

4. Conclusion

SEL is a rare disease-causing spinal cord compression due to accumulation of adipose tissue, which is commonly reported with endogenous or exogenous steroid excess or obesity. Asymptomatic individuals are treated conservatively, but those who present with progressive neurological deficiencies should be treated urgently with neural elements decompression and removal of the fatty tissue, preferably through laminectomy for children and adolescents.

5. Declarations

Consent statement: Written informed consent was obtained from the patient to publish this report in accordance with the journal's patient consent policy

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