Symptomatic concurrent spinal epidural lipomatosis and spinal pathology

Spinal epidural lipomatosis (SEL) is a rare condition defined by an excess deposition of extradural adipose tissue. SEL is often an incidental finding and the majority of patients remain asymptomatic. SEL is typically associated with excess steroid, either endogenous or exogenous, as well as a number of endocrinopathies. Few reports have described the disorder in conjunction with other spinal pathologies.

A 47-year-old man presented to our hospital following a 14-day history of progressive paraparesis and gait disturbance. He had no significant past medical history and clinical examination demonstrated bilateral decreased power and pyramidal signs of his lower limbs. Magnetic resonance imaging (MRI) of his whole spine demonstrated dorsal thoracic epidural lipomatosis and a focal central disc extrusion at T6/7 with canal stenosis and increased cord signal (Fig. 1).

Because of the ventral location of the disc and the presence of dual pathology (ventral compression by the disc and multilevel dorsal compression by the lipomatosis) he underwent a combined anterior and posterior approach, decompression and instrumented fusion (Fig. 3a). Histopathology of excised fat confirmed SEL. There were no perioperative complications, and at 4 weeks follow-up, there was near complete resolution of his lower limb weakness and gait disturbance.

A 70-year-old man was referred with a 4-week history of mid-thoracic back pain and progressive lower limb numbness. Relevant past medical history included Wegener’s granulomatosis treated with 5–10 mg of prednisolone daily for 5 years and had no recent history of trauma. Clinical examination demonstrated reduced sensation with a sensory level of T6 and mild motor deficit in the lower limbs. Osteopenia and a T6 burst fracture with mild retropulsion was noted on his CT, while MRI suggested extensive SEL (T4 to L2) with spinal cord compression (Fig. 2).

The patient developed worsening lower limb weakness and new urinary incontinence during a 5-day period of conservative management. Subsequently, he underwent an extensive T3–10 posterior decompressive laminectomy and instrumented fusion (Fig. 3b). Histopathology again confirmed SEL. At 12-month follow-up, the patient was continent and mobilizing independently with a frame.

We present two novel cases of neurological deficit resulting from acute spinal pathology in pre-existing SEL. Symptomatic SEL in the

Fig. 1. Case 1: (a) preoperative sagittal T1-weighted images and (b) preoperative sagittal T2-weighted images.

Fig. 2. Case 2: (a) preoperative sagittal T1-weighted images and (b) preoperative sagittal T2-weighted images.

Fig. 3. (a) Case 1 post-operative anteroposterior radiograph. (b) Case 2 post-operative anteroposterior radiograph.
setting of concurrent spinal pathology has rarely been reported in the literature. To our knowledge, this is the first report of idiopathic SEL with thoracic disc herniation.

The majority of patients with SEL remain asymptomatic, even with partial compression of the dural sac. However, the presence of SEL can greatly reduce the spinal canal diameter, rendering the spinal cord and cauda equina susceptible to compression in the presence of additional pathology. SEL may cause neurological injury by direct compression or vascular compromise. In the current report, symptoms originated from ventral compression (by thoracic disc or bone fragment) in established canal stenosis leading to cord compression and neurological deficit.

Few reports have documented pathology compounding SEL. Celek et al.² and Andress et al.³ described similar cases of SEL complicating an acute thoracic compression fracture. Both cases were treated with a posterior extended laminectomy, resection of epidural fat and instrumented fusion that resulted in improvement of neurological deficits. SEL occurring with concurrent epidural abscess,⁴ syrinx⁵ and spinal fluorosis⁶ have also been described.

SEL is commonly asymptomatic; however, it can contribute to cord compression in the setting of concurrent acute spinal pathology. The accommodating ability of the spinal cord is limited in SEL and otherwise clinically insignificant lesions, as described in the reported cases, can become symptomatic.

References


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