

## Lower Back Pain and Leg Weakness

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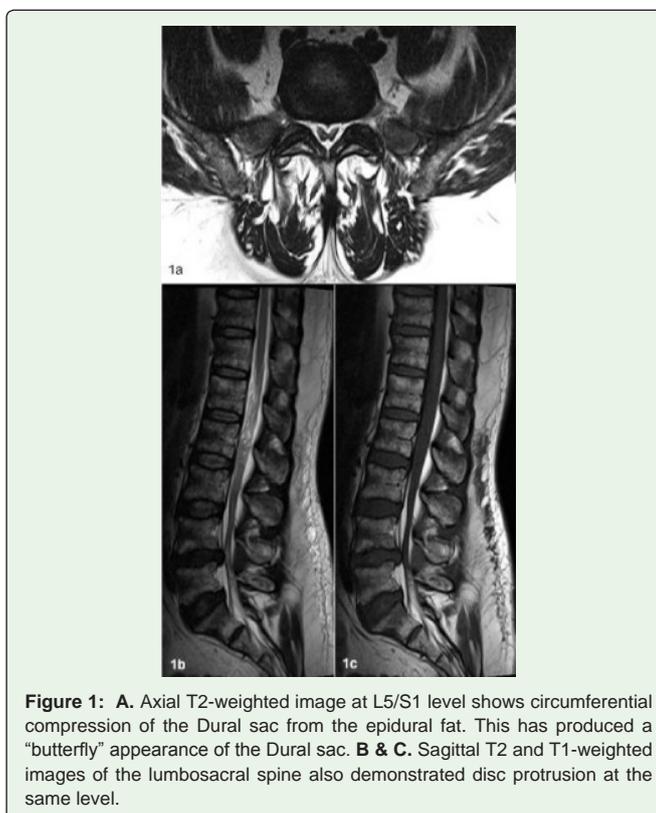
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## Clinical image

An 59 year-old patient complaining of progressive worsening of lower back pain with new bilateral leg weakness limiting mobility. Physical examination showed paraesthesia localising to L5/S1 dermatomes bilaterally. Patient has no previous injury or co-morbidities.

Magnetic Resonance Imaging (MRI) is the imaging investigation of choice. In our patient, on the axial T2-weighted image at the L5/S1 level, there is accumulation of adipose tissue in the spinal canal causing circumferential compression of the dural sac and producing a “butterfly” appearance of the dural sac (Figure 1). The appearance is consistent with Spinal Epidural Lipomatosis (SEL). The accumulation of adipose tissue in the spinal canal can be clearly depicted on conventional spin-echo MRI with non-contrast T1 and T2-weighted sequences – homogeneous high T1 signal intensity and intermediate T2 signal intensity. The normal epidural fat thickness is between 3-6 mm. A thickness of more than 7 mm in a symptomatic patient is suspicious for SEL [1]. On the axial images, the classic appearance of lumbar SEL is described as the Y-shaped dural sac [2]. It is important to be aware that the fat deposition can be asymmetrical and lead to displacement of the spinal cord [3]. Anterior displacement is more commonly seen in the thoracic region, where epidural fat tend to accumulate posteriorly [4].

SEL is an uncommon condition due to the excessive growth of adipose tissue within the epidural space leading to neural compression and vascular compromise. It most commonly affects the thoracic spinal canal followed by the lumbar region. The build up of epidural fat in the spinal canal causes direct compression of the dural sac producing the classic symptoms of back pain with motor and sensory deficits. Urinary and faecal incontinence are rare presentations of the condition [5]. The condition was first reported by Lee et al. in 1975 in a renal transplant recipient receiving steroid therapy [6]. Exogenous steroids including oral, inhaled, and parenteral formulations have all been reported to be associated with SEL. It is also linked to obesity and endocrine diseases that



**Figure 1:** A. Axial T2-weighted image at L5/S1 level shows circumferential compression of the Dural sac from the epidural fat. This has produced a “butterfly” appearance of the Dural sac. B & C. Sagittal T2 and T1-weighted images of the lumbosacral spine also demonstrated disc protrusion at the same level.

promote deposition of unencapsulated adipose tissue within the epidural space, such as Cushing disease and Cushing syndrome, hypothyroidism, and pituitary prolactinoma [5,7]. Therefore, patients not taking corticosteroids should be referred for endocrinological workup before given the diagnosis of idiopathic SEL.

When MRI is contraindicated, alternative imaging options include Computed Tomography (CT) and myelography. CT is able to demonstrate fatty attenuation of the tissue in the epidural space, which can help provide the diagnosis. On myelography, SEL produces the hourglass pattern of contrast obstruction at the level of compression, although this is not pathognomonic for the condition [8].

Management of SEL can be divided into conservative and surgical. Conservative strategies include weight loss and weaning patients off corticosteroids [1,9,10]. An underlying endogenous corticosteroid overproduction should be considered before referral for spinal decompression. Laminectomy with debulking of epidural fat is an effective treatment option for spinal cord compression and when conservative management is ineffective [1,4,7].

In cases of chronic compressive myelopathy without history of trauma, SEL should be part of the differential diagnosis for epidural mass particularly when the patient is overweight or taking long-term corticosteroids.

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