

CASE REPORT

Progressive Epidural Lipomatosis with Steroid use in Severe Refractory Asthma

BENJAMIN WAKERLEY*, LOUISA KENT, AND MATTHEW JACKSON

Department of Neurology, John Radcliffe Hospital, Oxford, UK.

Background. Long-term immunosuppression with oral corticosteroids is frequently used to treat inflammatory diseases of the lung and is advocated in the management of some patients with asthma. **Methods.** The authors describe the case of a 35-year-old man with severe refractory asthma who developed a slowly progressive thoracic spinal cord syndrome. **Results.** Spinal imaging demonstrated the presence of spinal epidural lipomatosis, a rare complication of prolonged corticosteroid therapy, which is characterized by overgrowth of fat in the epidural space and neuronal compression. **Conclusions.** Spinal epidural lipomatosis should be considered in patients receiving long-term corticosteroid therapy who develop symptoms and signs suggestive of spinal cord compression.

Keywords asthma, corticosteroids, epidural lipomatosis

INTRODUCTION

The use of daily oral corticosteroids remains an important treatment option in some patients with chronic asthma and the recognition and the prevention of tablet-induced side effects, including hypertension, diabetes, osteoporosis, and cataracts, are well established (1). We describe an asthmatic patient who developed a slowly progressive spinal cord syndrome secondary to steroid-induced spinal epidural lipomatosis (SEL), a rare condition characterized by excessive overgrowth of normal fat in the epidural space that sometimes leads to symptomatic spinal cord or nerve root compression. In the majority of cases, this relates to an uncommon complication of prolonged corticosteroid therapy or endogenous overproduction by functional adenomas, although idiopathic forms exist (2).

CASE STUDY

A 35-year-old man with severe refractory asthma presented to the general neurology clinic with an 18-month history of slowly progressive weakness and sensory disturbance in his lower limbs. His symptoms started with difficulty picking up his legs and intermittent urinary incontinence. Over the previous 6 months he complained of numbness in both feet, which gradually ascended to the level of the breast bone and more recently erectile dysfunction.

On examination he was Cushingoid with a body mass index of 47. Tone was increased in the lower limbs with evidence of proximal muscle weakness, bilateral ankle clonus, and hyperreflexia. There was reduced sensation

to light touch and pin prick to the mid-thoracic level and reduced vibration and joint position sensation to the ankles.

He had allergic asthma since early childhood with poor control over the past 5 years and was atopic to perennial aeroallergen and dust mites. Lung function tests 1 month previously showed forced vital capacity = 2.35 L, forced expiratory volume in 1 second = 1.6 L, peak expiratory flow = 400–520 L/min. In addition to oral corticosteroids (prednisolone 30–60 mg), which he had been receiving continuously for the past 2 years, he was also using an inhaled corticosteroid (fluticasone), inhaled beta-2 agonists (salbutamol and salmeterol), and theophylline. Despite receiving fortnightly injectable anti-immunoglobulin E (IgE) (omalizumab) since 2008, which was of some benefit, he continued to have recurrent severe exacerbations with multiple hospital admissions and was corticosteroid dependent. During this time there was significant weight gain (75 kg in 2007, 140 kg in 2010). He had been requiring opiate analgesics for back pain for the past year but had no other past medical or family history of note.

An MRI scan of his spine revealed significant mid-thoracic cord compression secondary to epidural deposition of fat compounded by disc bulge (Figure 1). Before his steroids could be reduced, he died from an asthma attack complicated by pneumonia.

DISCUSSION

Neurological complications associated with long-term corticosteroid therapy are not uncommon, and steroid myopathy and psychosis are well described. Vertebral body fractures secondary to osteoporosis or avascular necrosis also occur and sometimes result in acute spinal cord syndromes. The development of SEL as a complication of prolonged corticosteroid therapy is rare and the

*Corresponding author: Dr. Benjamin Wakerley, Department of Neurology, John Radcliffe Hospital, Oxford, UK; Tel: +44(0)1865 741166; Fax: +44(0)1865 231885; E-mail: benwakerley@fastmail.fm

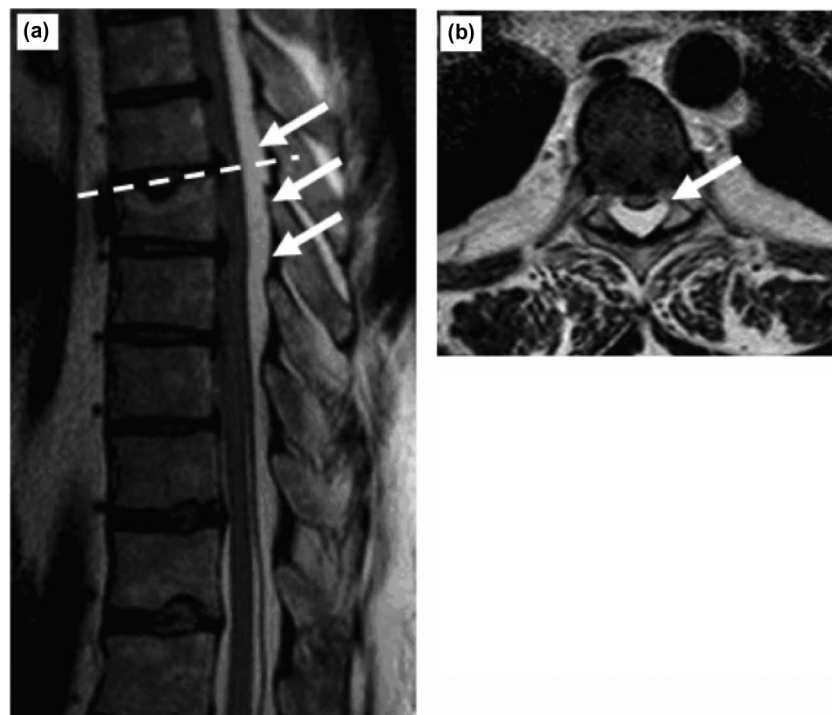


FIGURE 1.—T₂-weighted sagittal (a) and axial (broken line, b) MRI of thoracic spinal cord showing epidural overgrowth of fat (white arrows) leading to significant cord compression. There is also mild disc bulge at T5/6, T6/7, and T7/8 and a crush fracture at T7.

vast majority of spinal cord syndromes secondary to compressive lesions arising in the epidural space are a direct consequence of tumor, hematoma, or abscess formation rather than overgrowth of epidural fat.

In a significant proportion of SEL, there is a history of previous corticosteroid excess. This includes both exogenous long-term immunosuppressive therapy (3) and endogenous overproduction by functional adenomas of the pituitary or adrenal glands (4). The redistribution of body fat that occurs in Cushing's disease is well recognized but in addition to causing central obesity, corticosteroids also precipitate fat overgrowth in other organs including the liver and, as this case highlights, the epidural space.

SEL has been reported in other steroid-responsive respiratory conditions, including sarcoidosis and interstitial lung disease (5), but there are very few case reports in asthma (6). Other associations without corticosteroid excess include hyperprolactinemia and hypothyroidism (7, 8). Obesity alone is described as a predisposing factor in many cases (9), and a significant proportion is idiopathic (44%, 2).

Neurological symptoms usually develop insidiously and the clinical presentation depends on the site of nerve compression. Overgrowth of epidural fat in the thoracic spine results in myelopathy and paraplegia, whereas lumbar involvement frequently leads to a painful radiculopathy, cauda equina, or neurogenic claudication (10). In both cases bladder and erectile dysfunction is often a feature.

Whole spine MRI should be considered in such cases and epidural fat, which returns a high signal on T1- and

T2-weighted images, can easily be differentiated from CSF surrounding the cord. Overgrowth of fat in the epidural space is not uncommon on spinal imaging, especially in obese patients, but rarely causes neurological problems unless very significant. Subdural fat greater than 7-mm thickness in the anteroposterior dimension has been used to define SEL, but in symptomatic cases it is often more pronounced (11). Although SEL is sometimes the sole cause of neuronal compression, the presence of other structural abnormalities, including disc protrusions or a congenitally narrowed spinal canal, is often contributory.

Initial treatment in early SEL should be conservative, especially in obese patients, and weight loss and corticosteroid tapering have been shown to improve radicular pain and leg strength (5, 9). Neurosurgery is necessary if there are signs of acute cord compression although despite intervention only half of the patients make a full recovery and many are left with persistent neurological deficit (2).

Use of long-term corticosteroid therapy in respiratory diseases including asthma is commonplace and although rare, SEL should be considered in these patients who develop symptoms suggestive of early spinal cord or root compression as conservative measures may prevent the need for surgical intervention and irreversible disability.

DECLARATION OF INTEREST

The authors report no competing interests. The authors alone are responsible for the content and writing of this paper.

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