CASE REPORT

Neurogenic claudication without spinal stenosis arising as a result of lumbar epidural varices

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Neurogenic claudication is most frequently observed in patients with degenerative lumbar spinal stenosis. We describe a patient with lumbar epidural varices secondary to obstruction of the inferior vena cava by pathological lymph nodes presenting with this syndrome. Following a diagnosis of follicular lymphoma, successful chemotherapy led to the resolution of the varices and the symptoms of neurogenic claudication.

The lumbar epidural venous plexus may have an important role in the pathogenesis of spinal stenosis. Although rare, epidural venous engorgement can induce neurogenic claudication without spinal stenosis. Further investigations should be directed at identifying an underlying cause.

Lumbar epidural varices can result from a number of conditions, including vascular malformations, agenesis, and thrombosis of the inferior vena cava (IVC), Budd-Chiari syndrome, portal venous hypertension and abdominal malignancy. Dilated epidural venous plexuses have been reported to mimic the presentation of lumbar disc prolapse, and acute cauda equina syndrome as a result of epidural venous engorgement has also been described. Although commonly found in the lumbar spine, symptomatic epidural varices have been reported in the cervicothoracic region. In this case report we describe a patient with lumbar epidural varices secondary to IVC obstruction, presenting with symptoms imitating lumbar spinal canal stenosis.

Case report
A 55-year-old man presented with a three-month history of low back and bilateral lower limb pain associated with numbness and tingling. The symptoms were exacerbated by standing and relieved by sitting, and his walking distance was approximately one mile (1.6 km). There was no significant past medical history.

Examination of the spine was normal and there were no abnormal neurological findings in the lower limbs; there was some bilateral pitting oedema of both feet, which was not due to cardiac causes.

MRI of the lumbar spine demonstrated lumbar epidural venous engorgement, which was most pronounced at L3/L4 and extended up to L2, but there were no degenerative features to suggest spinal canal stenosis (Fig. 1). A lytic spondylolysis at L5/S1 was noted but would not have explained the symptoms of spinal claudication. A CT scan of the abdomen showed extensive coeliac and para-aortic lymphadenopathy leading to occlusion of the IVC between the levels of the L1 and L3 vertebrae (Fig. 2).

A lymph node biopsy revealed positive Bcl-2 staining of reactive germinal centre B cells and the presence of neoplastic follicles, consistent with a diagnosis of follicular lymphoma. He had chemotherapy with six cycles of R-CVP (rituximab, cyclophosphamide, vincristine and prednisolone). A follow-up CT scan showed resolution of the coeliac and para-aortic lymphadenopathy. An MRI scan of the lumbar spine did not reveal any evidence of epidural venous congestion (Fig. 3), and there was complete resolution of the low back and leg pain by two months after completion of chemotherapy.

Discussion
A large valveless vertebral venous network arranged in a longitudinal pattern, with three distinct interconnecting divisions, was originally described by Breschet. Batson redefined the venous system into caval, pulmonary, portal and vertebral divisions and highlighted the role of this system in the spread of infection, tumour and emboli.

The valveless nature of this system allows free communication between the internal and external venous networks. At each level foraminal veins provide a drainage route for both networks. In the lumbar spine the L2 vertebra is a landmark for differential venous drainage. Either at or above this level drainage is either
directly into the IVC via transversely joining lumbar veins, or superiorly through the azygous and hemi-azygous systems. Below this level the iliolumbar veins drain the foraminal veins into the common iliac veins.¹³

Compression or occlusion of the IVC leads to an increase in venous pressure that is transmitted to the epidural venous plexuses via the foraminal veins. Similarly, increasing flow in the azygous and hemi-azygous veins, secondary to collateral pathways bypassing the obstruction, can contribute to epidural venous engorgement.

The presenting symptoms in our patient were suggestive of a diagnosis of spinal stenosis, which is often secondary to degenerative disc disease and hypertrophy of the facet joints and ligamentum flavum.¹⁴ The symptoms are usually of neurogenic claudication, and the pain radiates beyond the back into the buttocks and into the groin, thighs and legs. The symptoms typically increase with activities involving lumbar extension and axial loading, such as standing and walking.¹⁵

Narrowing of the spinal canal at a particular level may not on its own cause neurogenic claudication, and Porter’s two-level stenosis concept is widely accepted as the
explanation for the pathogenesis of neurogenic claudication. The concept is based on the model of venous pooling within the cauda equina between two levels of low-pressure occlusion of the central canal. Olmarker and Rydevik have identified critical mechanical pressures that can cause changes in the blood supply and nutrition of the nerve roots of the cauda equina, and have shown that pressures of 10 mmHg are sufficient to reduce total blood flow by 64%, leading to a 20% to 30% reduction in nutrition.

Ikawa, Atsuta and Tsunekawa demonstrated abnormal ectopic firing in neurons in the dorsal root ganglia secondary to artificially created venous stasis in animal models with lumbar stenosis. They observed a latent period between ectopic firing and the induction of venous stasis, as well as the resolution of ectopic activity within a short period after relief of stasis. These characteristics are recognised as similar to those of neurogenic claudication, which supports the concept of venous pooling in the pathophysiology of lumbar stenosis.

In patients with lumbar stenosis, epidural pressures of 15 mmHg to 18 mmHg have been recorded in flexion and between 80 mmHg to 100 mmHg in extension. An increase in epidural pressures has also been reported in patients with lumbar stenosis during walking. An increase in lower-limb venous return during exercise can lead to epidural venous engorgement and further increase in stenotic pressures.

Our patient had epidural varices resulting from occlusion of the IVC by coeliac and para-aortic lymphadenopathy, and the presenting symptoms were of neurogenic claudication. MRI did not identify stenosis. It is probable that lumbar epidural varices can create the two levels of low-pressure occlusion of the central canal, as proposed by Porter, leading to venous pooling within the cauda equina. This would result in a rise in the resting venous pressures in the occluded segment, but they would still remain below the threshold required for the onset of symptoms. However, any further increases in venous pressure, brought about by standing and walking, would lead to the onset of symptoms.

The finding of lumbar epidural varices or venous engorgement on MRI, although rare, should prompt clinicians to consider potential causes of IVC obstruction or occlusion that, if successfully treated, can lead to the resolution of symptoms.

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References