Vascular anatomy of the spinal cord and cord infarction

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INTRODUCTION
Spinal cord infarction is an uncommon but important problem. Often it is a diagnosis of exclusion, having ruled out other causes of acute spinal symptoms such as multiple sclerosis, transverse myelitis, disc herniation, spinal canal stenosis, contusion, congestive myelopathy secondary to dural arteriovenous fistula and spinal tumours. In this article we discuss the important arterial anatomy of the spinal cord and use it to make sense of a case of spinal cord infarction.

CASE PRESENTATION
A 39-year-old woman was admitted with sudden onset of pain in the back and legs, accompanied by spasm and numbness, whilst in bed at night. On examination she had bilaterally spastic legs with profound ankle weakness, particularly on the right. Ankle reflexes were absent, while her plantar reflexes were mute. There was loss of pin-prick sensation in the S2/3 dermatomes, with loss of anal tone and reflexes. MRI examination on the day of admission showed an equivocal area of increased signal within the conus medullaris, and ruled out a compressive lesion. A repeat MRI 2 days later confirmed the presence of high signal centrally within the conus (Fig. 1). Other investigations were negative. A diagnosis of spontaneous spinal cord infarction was made. She gradually improved until she was able to walk on her own. Follow-up revealed some residual weakness, particularly around the right ankle.

Figure 1 (a) Sagittal and (b) axial T2-weighted MRI images showing high-signal change centrally within the conus medullaris (arrows).
Spinal cord infarction

There are various causes of spinal cord infarction: vertebral artery dissection, atherosclerosis of the vertebral arteries with severe stenosis, hypotension, dissecting aortic aneurysm and aortic surgery, epidural anesthesia, vasculitis and trauma. Usually, as in our case, the onset evolves over minutes and more than 80% of cases are associated with pain in the back. Neurological symptoms reflect ischaemia in the anterior spinal artery territory with involvement of the spinohalamic and corticospinal tracts, anterior and lateral, causing para- or tetra-paresis, bladder dysfunction and bilateral dissociated sensory deficit with loss of temperature and pain sense below the level of infarction. Sparing of the posterior columns is typical with preservation of proprioception, light touch and vibration sense. Treatment is mainly supportive.

Vascular anatomy

The traditional student textbook account of the spinal cord blood supply shows the anterior spinal artery as a branch of the vertebral arteries, descending the length of the spinal cord. Two or four posterior spinal arteries are also described. Spinal cord blood supply thus depends on two systems: medullary perforating arteries from the anterior spinal artery supplying the anterior and central portions and the grey matter core, and pial circumferential arteries from the posterior spinal arteries supplying the periphery. In reality, the anterior spinal artery represents the embryonic fusion of the paired longitudinal neural arterial axis, in effect a continuation of the basilar artery, itself resulting from the fusion of the posterior communicating arteries. This is a highly stable phylogenetic system conserved over more than 500 million years of evolution, from fish to humans. This anterior spinal artery, perhaps better thought of as an anterior spinal axis, is fed in turn by the vertebral arteries, deep and ascending cervical arteries, and by the thoracic (intercostal) and lumbar segmental arteries (Fig. 2).

Figure 2 Schematic illustration of the major sources of supply to the anterior spinal artery. Note the basket anastomosis with the posterior spinal arteries at the conus medullaris and the small artery of the filum terminale.
In the embryo, segmental arteries at each level reach the anterior spinal artery (31 pairs). Progressive regression of these vessels during development leaves four to eight ventral arteries contributing to the anterior spinal artery. A dominant feeding vessel in the cervical region is commonly present – the artery of the cervical enlargement. In the thoracic region, feeders are sparse and of small calibre, making the cord here vulnerable to ischaemia. A further dominant trunk is typically present in the lumbar enlargement, the so-called ‘artery of Adamkiewicz’. All these feeding arteries to the anterior spinal artery are termed radiculo-medullary arteries and have a characteristic hairpin appearance (Figs 2 and 3).

Inferiorly, the anterior spinal artery anastomoses around the conus medullaris with the posterior spinal arteries in the arterial ‘basket’. The circumferential, pial network of the spinal cord is mainly supplied by dorsal feeding arteries termed radiculo-pial arteries. Developmental regression is less pronounced dorsally, and 10–20 dorsal radiculo-pial vessels persist (Fig. 4).

Sulcal penetrating arteries from the anterior spinal artery have few anastomoses and are in effect end-arteries. The few feeding vessels to the

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**Figure 3** Spinal angiogram showing the ‘artery of Adamkiewicz’ at T9 level on the right. Note the characteristic hairpin appearance (white arrow) of the radiculo-medullary artery joining the anterior spinal artery (black arrows). The catheter tip lies in the intercostal artery (block arrow).

**Figure 4** Schematic illustration of the arterial supply to the spinal cord from behind. The posterior elements of the vertebrae have been removed and the dura reflected. Note that at T9, on the left side there are radiculo-medullary (ventral) and radiculo-pial (dorsal) arteries, while on the right there is only radiculo-pial supply. At T10, on the left, there is only radicular supply with branches to nerve root and dura, while on the right there is radiculo-pial supply.
anterior spinal artery make this system appear quite vulnerable and explain the typical anterior location of spinal cord infarcts (Fig. 5). However, the system is sufficiently robust to make cord infarcts much less common than brain infarcts. The pial network has many feeders and extensive anastomoses, which explains the rarity of posterior spinal infarcts.

Radicular arteries also supply the vertebra and occlusion may result in vertebral body bone infarcts adjacent to cord infarcts. This can be a useful sign on MRI in confirming the diagnosis.

CONCLUSION
A diagnosis of spinal cord infarction requires exclusion of surgically treatable lesions such as spinal haematomas, empyemas and disc protrusions. Knowledge of spinal vascular anatomy, which is both simpler and more consistent although less familiar than cerebral vascular anatomy, allows recognition of typical patterns of involvement (central and anterior) of the spinal cord.

FURTHER READING