Fibrocartilaginous embolism: a rare aetiology of spinal cord infarction

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DESCRIPTION

A male in late adolescence experienced sudden onset of neck pain followed by quadriplegia during shoulder press exercises. Cranial nerve examination was normal. He had flaccid quadriplegia, areflexia, flexor plantar responses and urinary retention without other autonomic dysfunction. Sensory examination showed a spinothalamic sensory level at T2, while dorsal column sensation remained intact. MRI with contrast of the brain and whole spine done 12 hours after the onset of paralysis was normal. Cerebrospinal fluid analysis showed no abnormality. A repeat MRI performed on day 5 revealed a long segment anterior spinal cord infarct (figure 1). CT angiogram was normal. Vasculitis screen, Anti-Myelin Oligodendrocyte Glycoprotein (MOG) and aquaporin four antibodies were negative.

Figure 1  (A) Sagittal T2 of the cervical and thoracic spine showing central T2 hyperintense signal of the cord extending from C2 level until the upper thoracic cord without evidence of cord compression. (B) Axial T2 at C4 level showing central hyperintense signal involving the grey matter and sparing the peripheral cord (typical ‘owl’s eyes’ appearance is not seen as the infarction in this case was extensive). (C) Axial T2 at T3 level showing hyperintense signal involving the anterior cord aspect and extends to the left side. (D) Axial Diffusion-weighted Imaging (DWI) along the upper thoracic cord showing hyperintense signal with corresponding signal drop in ADC denoting restricted diffusion. Features are suggestive of an extensive long segment cord infarction. Given the extent and location of the infarction along the anterior spinal artery territory.

A diagnosis of spinal cord infarction was made. Cord infarction typically manifests acutely, the most common being anterior spinal artery syndrome (ASAS). The characteristic clinical features of ASAS include back or neck pain, initial flaccid weakness, autonomic dysfunction and loss of spinothalamic sensation, with relative sparing of dorsal column sensation.

Anterior spinal cord infarction occurring during exercise and weightlifting may involve the thoracic or cervical spinal cord. Fibrocartilaginous embolism (FCE) has been proposed as a likely aetiology. The proposed mechanism of FCE involves lateral rupture of the intervertebral disc, leading to entry of disc fragments into the intradiscal artery under pressure then travel retrogradely via radicular branch to anterior spinal artery causing the cord infarction. (NL created this figure.)

MRI has limited sensitivity in detecting anterior cord infarction in the early hours following symptom onset. In cases with a high clinical suspicion, follow-up imaging should be obtained as we did in this case. MRI findings associated with spinal
cord ischaemia include hyperintensities on T2-weighted and short-tau inversion recovery images. A more specific finding is the presence of an ‘owl’s eyes’ or ‘snake eyes’ sign, as well as vertebral body infarction adjacent to a cord signal abnormality. Detection of intervertebral disc disease at the level of infarction may suggest a possible FCE. Vascular imaging, such as CT angiography or magnetic resonance angiography, should be performed to rule out aortic or vertebral artery dissection.

In this case, we highlight that healthcare providers should be aware of this rare and potentially devastating FCE when a patient presents with quadriplegia during weightlifting or exercising, along with appropriate imaging findings. Compressive myelopathy and inflammatory transverse myelitis are important differential diagnoses. Acute spinal pain, followed by quadriplegia/paraplegia, characteristic MRI findings, exclusion of other vascular causes by angiography support the diagnosis.

Management is symptomatic. Antiplatelet or anticoagulation is not indicated. Despite the initial devastating presentation, prognosis is generally good but early neurorehabilitation is important. Initial intravenous methylprednisolone before diagnosis of FCE made no improvement. He began to show spontaneous improvement in 3 weeks and at 6 weeks, he was able to self-care with activities of daily living.

**Contributors** The following authors were responsible for drafting of the text, sourcing and editing of clinical images, investigation results, drawing original diagrams and algorithms, and critical revision for important intellectual content: TMH, MS, LN, NL. The following authors gave final approval of the manuscript: TMH, MS, LN, NL.

**Funding** The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

**Competing interests** None declared.

**Patient consent for publication** Consent obtained from parent(s)/guardian(s).

**Provenance and peer review** Not commissioned; externally peer reviewed.

Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

**Learning points**

- Anterior spinal cord infarction should be suspected when a patient presents with flaccid weakness, autonomic dysfunction and loss of spinothalamic sensation, with relative sparing of dorsal column sensation during weightlifting or exercising.
- Early MRI may be negative and does not rule out fibrocartilaginous embolism (FCE), so a follow-up MRI after a few days is important in suspected FCE.
- The MRI of FCE-related spinal cord infarctions may show a long lesion that resembles an inflammatory myelopathy, and the characteristic signs of ischaemic myelopathy, such as ‘snake’s eyes’ or ‘owl’s eyes’, may not be present. The disc herniation that causes FCE may be subtle and hard to detect.

**REFERENCES**