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CASE REPORT

Fibrocartilaginous embolism: a cause of acute ischemic myelopathy

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Study design: A case report of fibrocartilaginous embolism (FCE) presenting as acute myelopathy.
Objectives: To illustrate the clinical presentation and magnetic resonance imaging features of FCE.
Setting: Johns Hopkins Transverse Myelitis Center.
Case report: A 16-year-old boy was diagnosed with ischemic myelopathy secondary to FCE 2 years after symptom onset. Diagnosis was delayed because the clinical and radiological characteristics were not recognized initially. After rehabilitation, the patient made a modest recovery.
Conclusions: Diagnosis of FCE can be made by recognition of the characteristic clinical and radiological features and a high index of suspicion.
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Keywords: fibrocartilaginous embolism; ischemic myelopathy; intervertebral disc; herniation

Introduction

Spinal cord infarctions occur infrequently, resulting in long-term sequelae. They may be difficult to distinguish from other myelopathies and are sometimes mistaken for acute inflammatory demyelinating polyneuropathy, as these patients may present with flaccid paraparesis in acute spinal shock. Imaging of spinal cord ischemia is often difficult because of its small transverse area, cerebrospinal fluid artifact and inadequate resolution of magnetic resonance imaging (MRI).[1] We report a case of ischemic myelopathy secondary to presumptive fibrocartilaginous embolism (FCE) on the basis of the typical clinical and radiological features.

Case report

A 16-year-old boy awoke with bilateral lower-extremity weakness in March 2006. Within 1 h, he developed severe lower back pain, complete paraplegia, sensory loss below T10 dermatome and urinary and fecal incontinence. Two days earlier, he had been elbowed in the face during gym class, and was wrestling with his brothers the night before. Initial cerebrospinal fluid analysis was acellular with mildly elevated proteins. Brain computer tomography and spinal cord MRI scans were initially reported as normal. He was diagnosed with Guillain–Barre syndrome, was treated with intravenous immunoglobulins and made a gradual modest recovery.

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Nineteen months later, he was reevaluated for persistent fecal incontinence. Repeat spinal cord MRI scans showed a non-enhancing T2-weighted hyperintense lesion at T11 to L2, L1/2 disc collapse and Schmorl's nodes (vertical protrusions of intervertebral disc cartilage into the adjacent vertebral body), with adjacent vertebral end-plate signal changes (Figure 1), which in retrospect were present on the initial MRI scan (Figure 2). MRI brain, cervical spinal cord and remainder of the vertebral column were normal. Visual and brainstem auditory-evoked potentials, antinuclear antibody, neuromyelitis optica-IgG, Lyme titers and thyroid function test were normal.

We saw the patient 26 months after symptom onset. His history of sudden-onset paraplegia preceded by minor trauma, reaching nadir within an hour, non-inflammatory cerebrospinal fluid and MRI features were suggestive of FCE. Physical examination revealed residual asymmetric spastic paraparesis (motor strength: right leg 3/5, left leg 4/5) and reduced lower extremity sensation of all modalities. The sensory level and urinary and fecal incontinence had resolved by this time.

Discussion

Fibrocartilaginous embolism of the spinal cord is a rare cause of acute ischemic myelopathy.[1,2] The abruptness of symptom onset in this case suggests a vascular etiology. Ischemic spinal cord infarction can be caused by decreased blood flow due to hypotension, vascular injury, compression, other impairment or thromboembolism to spinal arteries.[1] Acute

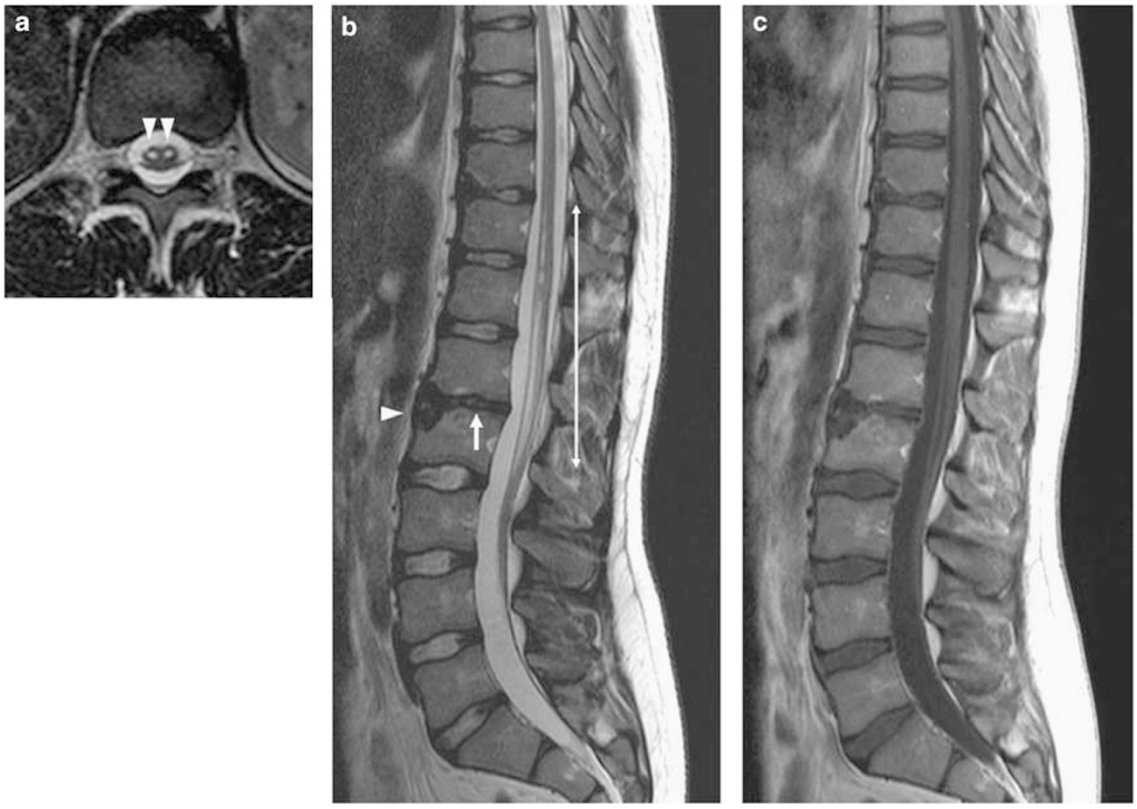

Figure 1 Axial (**a**) and sagittal (**b**) T2-weighted and sagittal (**c**) contrast-enhanced thoracolumbar spine MRI scans 19 months after acute myelopathy, showing increased signal intensity of bilateral anterior horn cells (arrowheads in **a**) at T11–L2 (line segment in **b**), L1/2 disc collapse (arrow in **b**), Schmorl's nodes (arrowhead in **b**) and adjacent vertebral end-plates signal changes, without contrast enhancement.

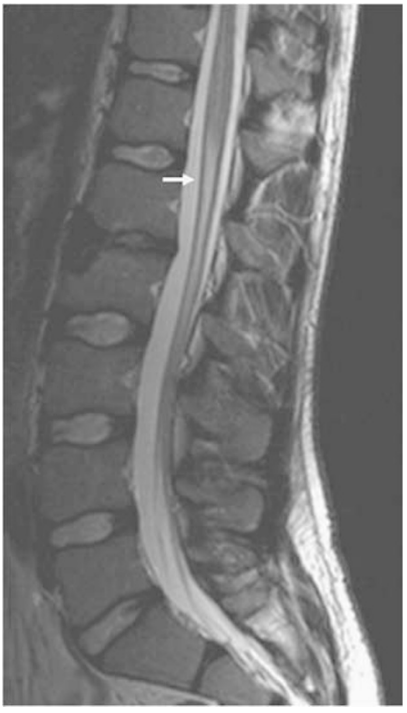

Figure 2 Sagittal T2-weighted MRI scan of the thoracolumbar spine on initial presentation, showing the lesion (arrow).

transverse myelitis is unlikely, as the diagnostic criteria were not fulfilled.[3] Occasionally, acute myelopathy may be misdiagnosed as Guillain–Barre syndrome, as acute spinal shock may present with flaccid paraparesis.

Although definitive FCE diagnosis can only be made at autopsy, several clinical and radiological features strongly suggest it. Clinical features vary on the basis of the embolization site but, in general, include severe back pain and rapidly progressive myelopathy, which may be preceded by Valsalva-like maneuvers, intensive physical effort or trauma. Although acute symptoms typically occur within minutes to hours, FCE (one pathologically confirmed) has been reported in patients with symptom-free intervals of days following the presumed precipitating event.[2,4] Similarly, FCE has occurred in patients who suffered seemingly minor or no trauma.[5,6]

Fibrocartilaginous embolism occurs in a bimodal distribution, with peaks in adolescence and late middle age (range 6–77 years).[2] Myelopathy predominate in cervical (69%) and lumbosacral (22%) segments.[6] MRI findings include spinal cord swelling associated with collapsed intervertebral disc at the appropriate level, Schmorl's nodes and the absence of early contrast enhancement. Longitudinally extensive spinal cord lesions are commonly seen even when intervertebral disc pathology occurs at only one spinal segment (L1/2 in our patient).[4–6] Compromise of the anterior spinal artery

(by presumed FCE) may explain why multiple spinal segments are involved. Spinal fluid examination is generally unrevealing except for mild protein elevation.[6]

The proposed pathophysiology of FCE is forceful herniation of intervertebral disc nucleus pulposus material into intradiscal or vertebral body vessels secondary to trauma, with retrograde flow into and compromise of spinal cord vasculature.[2,6] The minor trauma suffered by our patient may have precipitated vertical disc herniation. However, the mechanism for the symptom-free interval following the precipitating event remains unclear.

Fibrocartilaginous embolism-related ischemic myelopathy is often not suspected on presentation. With the bias toward case detection by histological diagnosis at autopsy, it was once thought to be rare and fatal.[2] In our patient, the clinical history in combination with cerebrospinal fluid and MRI features were highly suggestive of FCE. Awareness of this entity and recognition of the characteristic clinical and radiological features is the key in establishing an antemortem diagnosis. Prognosis is favorable (54% of patients with spinal cord infarction show improvement) as seen in our patient.[1]

Disclosure

The authors report no conflict of interest.

References

1 Nance JR, Golomb MR. Ischemic spinal cord infarction in children without vertebral fracture. *Pediatr Neurol* 2007; **36**: 209–216.
2 Han JJ, Massagli TL, Jaffe KM. Fibrocartilaginous embolism—an uncommon cause of spinal cord infarction: a case report and review of the literature. *Arch Phys Med Rehabil* 2004; **85**: 153–157.
3 Transverse Myelitis Consortium Working Group. Proposed diagnostic criteria and nosology of acute transverse myelitis. *Neurology* 2002; **59**: 499–505.
4 Duprez TP, Danvoye L, Hernalsteen D, Cosnard G, Sindic CJ, Godfraind C. Fibrocartilaginous embolization to the spinal cord: serial MR imaging monitoring and pathologic study. *AJNR Am J Neuroradiol* 2005; **26**: 496–501.
5 Tosi L, Rigoli G, Beltramello A. Fibrocartilaginous embolism of the spinal cord: a clinical and pathogenetic reconsideration. *J Neurol Neurosurg Psychiatry* 1996; **60**: 55–60.
6 Toro G, Roman GC, Navarro-Roman L, Cantillo J, Serrano B, Vergara I. Natural history of spinal cord infarction caused by nucleus pulposus embolism. *Spine* 1994; **19**: 360–366.