Case Report

Spinal Cord Infarction due to Fibrocartilaginous Embolism: A Report of 3 Cases

Jeffrey N. Quinn, MD, Hannah Breit, MD, and Rima M. Dafer, MD, MPH

Fibrocartilaginous embolism (FCE) is an uncommon cause of spinal cord infarction often misdiagnosed as transverse myelitis. The mechanism of ischemia is suspected to be due to retrograde embolization of nucleus pulposus material originating from Schmorl's nodes to the spinal vessels following acute disk herniation. We describe the clinical and imaging findings of FCE in 3 healthy young women with history of trivial spinal cord trauma, and recommend that FCE should be considered in the differential diagnosis of acute myelopathy.

Key Words: Stroke—infarction—fibrocartilaginous embolism—spinal cord

Introduction

Spinal cord infarction (SCI) is a rare clinical entity representing 1%-2% of all strokes.[1] Causes and etiologies of SCI vary, with a well-established association with vascular surgical interventions. Due to the anatomy of the vascular supply to the spinal cord and surrounding structures, a unique etiology of infarction exists in the form of fibrocartilaginous embolism (FCE). First described in 1961 in a 15-year-old boy with acute quadriplegia with fatal outcome following a minor fall during a basketball game,[2] there have been 68 cases to-date in the literature, with two thirds diagnosed histopathologically.

We present 3 additional cases seen within 1-year at a tertiary care center with clinical diagnosis of FCE.

Case Reports

Case 1: An 18-year-old healthy woman presented with acute chest and back pain followed by bilateral upper extremity paralysis and sensory loss after bending to pick up a shirt from the ground. She had flaccid paraplegia of the upper extremities with areflexia, minor weakness in the hip flexors bilaterally, and sensory level at T1. Magnetic resonance imaging (MRI) of cervical and thoracic spine (C/T spine) revealed anterior T2 hyperintensity with corresponding restricted diffusion from C4-T1 without cord enhancement (Fig 1, A-C). Six months later, she continued to have significant distal hand weakness.

Case 2: A 20-year-old healthy woman presented with sharp midline back pain, followed by acute bilateral upper extremities numbness and weakness, flank and abdomen numbness, and urinary incontinence. Symptoms occurred after bending to pick up laundry basket. She had bilateral upper extremity weakness, sensory level at T4-T12, and diffused hyperreflexia. MRI C/T spine showed anterior T2 hyperintensity with restricted diffusion C6-C8 without cord enhancement (Fig 1, D-F). At 6 months, she showed significant improvement in overall motor strength, with residual hand weakness bilaterally.

Case 3: A 32-year-old woman presented with sudden back pain and acute bilateral lower extremity weakness and paresthesia with numbness up to the mid-abdomen and loss of sphincteric control after bending down to pick up laundry. She is diabetic and overweight. She had severe bilateral weakness in the lower extremities predominantly proximally, absent deep tendon reflexes, extensor plantar responses, and sensory level to T6. MRI C/T spine showed central cord T2 hyperintensity T4-T11, with prominent owl sign on transverse views (Fig 1, G,H). She was discharged to inpatient rehabilitation, but was lost for further follow-up.

From the Department of Neurological Sciences, Rush University Medical Center, Chicago, Illinois.

Received October 16, 2018; accepted March 3, 2019.

Address correspondence to Jeffrey N. Quinn, MD, Hannah Breit, MD, and Rima M. Dafer, MD, MPH, Department of Neurological Sciences, Rush University Medical Center, 1725 W. Harrison St, Suite 1118, Chicago, IL 60612. E-mails: Jeffrey_n_quinn@rush.edu, Hannah_Breit@rush.edu, rima_dafer@rush.edu.

1052-3057/$ - see front matter

https://doi.org/10.1016/j.jstrokecerebrovasdis.2019.03.012

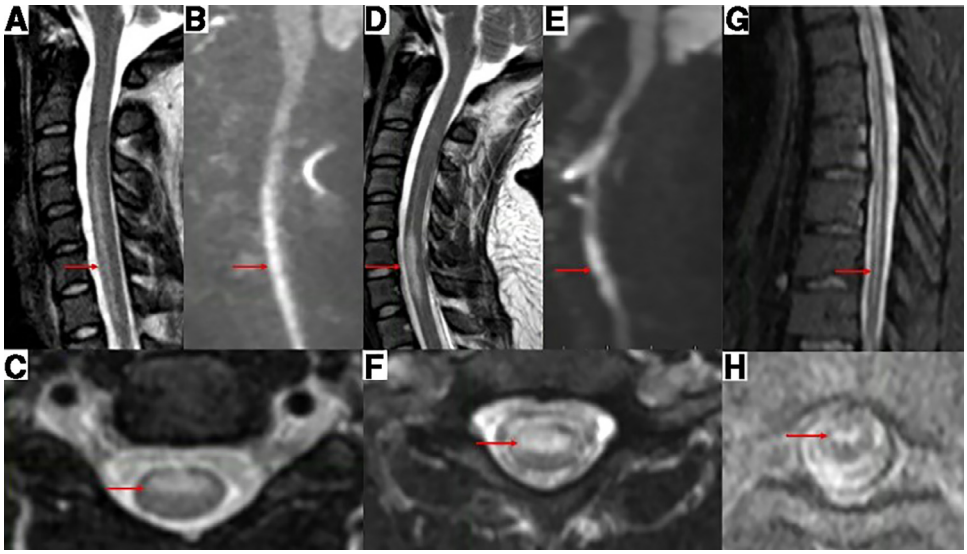

Figure 1. *Magnetic resonance imaging in 3 patients demonstrating a T2 hyperintensity on sagittal views from case 1 (A), case 2 (D), case 3 (G), and corresponding restricted diffusion seen in case 1 (B) and case 2 (E). Transverse T2-weighted images at the corresponding levels for case 1 (C), case 2 (F), and case 3 (H) with increased signal intensity and characteristic "owl sign."*

In all 3 patients, extensive work up including cerebrospinal fluid analysis, cardiac sonography, and laboratory panels was unremarkable. Direct spinal angiography was pursued in 2 patients without vascular abnormality. Based on the clinical presentations, work up, and imaging findings, the diagnosis of acute SCI due to FCE was made in all 3 patients.

Discussion

FCE is a rare cause of SCI wherein transient increased intravertebral body pressure on intervertebral disks—often triggered by minimal stresses leading to retrograde embolization of debris from the nucleus pulposus into the vascular supply of the spinal cord, typically the anterior spinal artery, leading to infarction. Unlike the classical etiologies of cerebral or SCIs, FCE has a higher incidence in young adults. The clinical presentation in all 3 of our patients was triggered by bending to lift objects, suggesting an acute increase in intraosseous pressure with acute disk herniation. FCE appears to be more common than previously thought and should be in the differential diagnosis of acute myelopathy in the young.

References

1. Rigney L, Cappelin-Smith C, Sebire D, et al. Nontraumatic spinal cord ischaemic syndrome. JClin Neurosci 2015;22:1544-1549.
2. Naiman JL, Donohue WL, Richard JS. Fatal nucleus pulposus embolism of spinal cord after trauma. Neurology 1961;11:83. 83.