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PRESENTATION AND MEDICAL MANAGEMENT OF FIBROCARTILAGINOUS EMBOLISM IN THE EMERGENCY DEPARTMENT

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☐ **Abstract—Background: Fibrocartilaginous embolism is an exceedingly rare condition that was formerly a clinical diagnosis based on mechanism of injury, physical examination findings, and older magnetic resonance imaging (MRI) technologies without a specific histologic diagnosis. Spinal cord MRI diffusion-weighted imaging allows for a more specific diagnosis. Case Report: A 14-year-old male felt a sudden pop in his back while running sprints in his gym class. He slowly developed bilateral lower extremity weakness and urinary incontinence, prompting an emergency department evaluation. A MRI scan of his lumbar spine revealed degeneration, desiccation, and bulging of the T12–L1 disc with an accompanying subacute Schmorl's nodule. There was adjacent cord swelling and central cord T2 hyperintensity, with accompanying restricted diffusion consistent with spinal cord infarction. These findings, in conjunction with paraplegia and mechanism of injury, were highly suggestive of fibrocartilaginous embolism. Why Should an Emergency Physician Be Aware of This?: An emergent MRI scan with the proper sequencing and immediate consultation with a spine surgery specialist are important to exclude a compressive myelopathy that would necessitate acute surgical decompression. There is significant uncertainty in the initial management and stabilization of this rare condition that has not been addressed in the emergency medicine literature.** © 2016 Elsevier Inc. All rights reserved.

☐ **Keywords—fibrocartilaginous embolism; paraplegia; pediatrics**

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INTRODUCTION

Fibrocartilaginous embolism (FCE) is a rare cause of ischemic myelopathy that arises when embolic debris from the intervertebral discs lead to spinal cord infarction through retrograde embolization to the central artery (1,2). FCE has been primarily a clinical diagnosis based on mechanism of injury, physical examination findings, and older magnetic resonance imaging (MRI) technologies without a specific histologic diagnosis (3). However, the development of spinal cord MRI diffusion-weighted imaging, as used in this more recent case, permits a more specific diagnosis (4). A review of reported cases showed that FCE presents in a wide range of ages (13–77 years of age), in a bimodal age distribution with peaks at ages 22 (median age for men 20.5; median age for women 28) and 60 (median age for women 61, median age for men 45). When analyzing those below and above 40 years of age, respectively, the majority of cases have been in European and North American whites (84%), with an increased frequency in females (69%) (5).

It has been hypothesized that clinical risk factors include young age (because the nucleus pulposus volume is larger with vascularization that persists into adolescence); osteoporosis and cartilage degeneration, both of which preferentially affect postmenopausal white women, also partially account for the epidemiologic trends documented in patients with FCE (5–7). Other potential predisposing factors might include a history of low back pain, previous lumbar disc surgery, vertebral

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fractures, motor vehicle accidents, and prolonged corticosteroid treatment (5). One author cites studies that document that FCE is associated with minor trauma to the vertebrae, with described mechanisms including lifting weights, stooping, forced head movements, falling, intense physical activity, and prolonged coughing often accompanied by the Valsalva maneuver (2). The typical clinical presentation of FCE is that of sudden back pain or neck pain that is followed by a progression of signs and symptoms that indicate an evolving spinal stroke (3). For cervical lesions, symptoms may include neck, chest, or abdominal pain with evolving quadriplegia, respiratory involvement, lower cranial nerve involvement, or Horner's syndrome. For lumbar involvement, lower back pain with evolving paraplegia is not uncommon. In all cases, spinal shock with a loss of reflexes and sphincter function is common (5).

Although there is no clear consensus on the pathophysiology of FCE, predisposing factors include the presence of Schmorl's nodes and spinal arteriovenous malformation accompanied by axial pressures required to produce retrograde flow through existing arteriovenous shunts (1,5). Pathophysiologic models have been proposed and include anatomic contact between the nucleus pulposus and venous sinusoids, migration of FCE from vertebral body sinusoids into spinal veins that are devoid of valves, and communication of spinal veins with spinal artery via arteriovenous communication that propagate the embolus to cause infarction of the spinal cord (5). It is important for emergency physicians to recognize the salient clinical features that distinguish FCE from other causes of spinal cord infarction in order to effectively initiate proper management of these patients.

CASE REPORT

A healthy 14-year-old male presented to the emergency department (ED) with lower back pain, bilateral lower extremity weakness for 1 day, and urinary incontinence. The day before, the patient was at gym class running sprints and making quick turns during which he felt a sudden pop and developed an abrupt onset of left-sided back pain. He completed the remainder of the class. That evening he had progressive numbness and unsteadiness of his legs to the point that he crawled into bed. When he woke the next morning he was unable to get out of bed, and his mother, thinking it was severe pain from exercise, allowed him to stay home from school. When his mom returned from work at 2:30 PM he was unable to move his legs and described unintentionally urinating on himself. She tickled his toes and noted absence of movement in either leg, prompting a trip to the ED.

In the ED, he was hemodynamically stable and afebrile. The physical examination revealed reproducible tenderness over the T12–L1 disc. He was alert and oriented on his neurologic examination, cranial nerves 2 to 12 were grossly intact, and he had 5/5 strength in his upper extremities. He had 0/5 strength in his lower extremities (i.e., hip flexion, knee extension, knee flexion, ankle dorsiflexion, plantar flexion, and extensor hallicus longus) with the exception of 2/5 strength of his left extensor hallicus longus and 1/5 strength with dorsiflexion of his left ankle. Sensation to the level of the inguinal crease was preserved; however, he had 1/2 sensation from L2 to S1 distributions bilaterally. His patellar and Achilles reflexes were 0/4 with absent Babinski reflexes bilaterally. There was no volitional contraction of his sphincter but preserved resting rectal tone.

Upon evaluation in the ED, the differential diagnosis included acute causes of spinal compression syndrome, including cauda equina, conus medullaris syndrome, and spinal cord compression. An infectious etiology was felt to be unlikely in the setting of no fever or other infectious symptoms. A post-void residual ultrasound revealed 1 liter of retained urine. A spine surgery specialist was emergently consulted and radiographs and emergent MRI scans were ordered while the operating room was notified of possible surgical intervention. Lumbar spine radiographs showed no evidence of acute fracture and mild narrowing of the T12–L1 interspace. An MRI scan of the lumbar spine showed a T12–L1 mildly degenerated and desiccated disk with an associated bulge that mildly deformed the ventral cord with the central canal widely patent with a small posterior Schmorl's node at this level (Figure 1). The adjacent cord was mildly swollen and demonstrated significant cord T2 hyperintensity predominately over its anterior two-thirds. This was accompanied by restricted diffusion on diffusion-weighted imaging, indicative of cord infarct in the anterior spinal artery distribution. There was no evidence of prominent or serpiginous vessels to suggest an arteriovenous fistula. The spine surgery specialist determined that the patient's condition was nonoperative. The patient was admitted to the pediatric intensive care unit to maintain a mean arterial pressure (MAP) of ≥90 mm Hg to maximize neurologic recovery. There was a discussion about heparinization to prevent clot progression; given that the working diagnosis was FCE to the anterior spinal artery, heparin would be ineffective. However, in an effort to prevent any propagation of the clot within the artery and resulting ascending paralysis, aspirin therapy was initiated. Dexamethasone 10 mg intravenously was given to decrease swelling.

Neurosurgeons elected not to place a lumbar drain given the time since the infarct, because any effect of reducing cerebrospinal fluid pressure would be minimal.

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