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# Spinal Cord Infarction Owing to Likely Fibrocartilaginous Embolism

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**Abstract** **Case Report**

Fibrocartilaginous embolism (FCE) is one of the rare causes of acute spinal cord infarction. We report the case of a previously healthy 16-year-old adolescent with a presentation suggestive of this condition. A few hours after lifting heavy objects, he developed sudden paraplegia. On examination, he had flaccid areflexic paraplegia, and thermoalgic anesthesia below the level of the D4 dermatome. Magnetic resonance imaging (MRI) of the spine confirmed the diagnosis of spinal infarction, FCE was retained as a likely cause after eliminating other differential diagnoses. Although FCE is rare, it should be kept as one of the differential diagnoses of an acute neurological deficit of the spinal cord.

**Keywords**: spinal cord infarction, adolescent, fibrocartilaginous embolism, lifting heavy objects.



## INTRODUCTION

Fibrocartilaginous embolism (FCE) of the spinal cord is a seemingly rare ischemic myelopathy that probably tends to be overlooked in clinical practice because of the diagnostic difficulty [1]. This ischemic myelopathy, is assumed to be caused by the migration of fibrocartilaginous nucleus pulposus components through retrograde embolization to the spinal artery. Symptoms include rapidly progressive plegia, paresthesia, and bladder or bowel dysfunction typically following back or neck pain [2]. We report a case of spinal cord infarction secondary to presumptive fibrocartilaginous embolism on the basis on the history, clinical and radiological features.

## CASE REPORT

A 16-year-old adolescent with no significant medical history, presented with the sudden development of severe pain in the interscapular region while lifting heavy objects. He subsequently developed rapidly progressive weakness of lower limbs, with numbness and vesico-sphincter disorders. The clinical examination revealed flaccid areflexic paraplegia, and thermo-algic anesthesia below the level of the D4 dermatome, with respect for other sensory modalities. MRI made it possible to establish the diagnosis of spinal infarction, with a characteristic snake-eyes appearance without Schmorl nodes within the vertebral body (figure 1). Aortic and supra-aortic trunk imaging was normal (CT angiogram and TEE), as were lumbar puncture and usual biological assessment, as well as cardiac, autoimmune and infectious examinations. Given the absence of cardiovascular risk factors, the age and the context of lifting heavy objects, the possibility of fibrocartilaginous embolism was raised. So he was given aspirin and a daily motor rehabilitation was started. Despite this our patient did not recover after 6 months of rehabilitation.

## DISCUSSION

FCE is a very rare cause of acute ischemic myelopathy and is not often suspected on presentation. It was identified only 25 cases of FCE of the spinal cord in pediatric patients younger than 18 years in the available English literature from 1961 [2]. FCE shows a bimodal distribution, with peaks in adolescence and late middle age [3]. Sex differences differ depending on the reports (male predominance [4], and female predominace [5]. The pathophysiology of FCE remains unclear. The most accepted hypothesis is that forceful herniation of the

intervertebral disk nucleus pulposus material into the intradiscal or vertebral body vessels secondary to minor trauma or some axial loading forces induces the prolapse of cartilaginous material into the spinal artery [3]. The nucleus pulposus may also extend into the vertebral body by Schmorl node [6], or probably abnormal vertebral body or disk changes [7]. Schmorl nodes, focal masses of fibrocartilage within the vertebral body, are commonly found in adults but are rare among children [8]. Although a definitive diagnosis of FCE can only be made after an autopsy, diagnosis by clinical and radiological features is considered possible [9]. Patients with FCE typically present with weakness that progresses to paralysis over a period of minutes to hours, often with sudden back pain, sensory problems, or bladder dysfunction after a suggestive history of a minor traumatic event [10].

The MRI findings of expansion of the spinal cord with increased signal on T2WI involving the spinal cord without early contrast enhancement in association with a narrowed disk or Schmorl nodes strongly suggest the diagnosis of FCE [3]. The diagnosis of spinal cord FCE is often difficult possibly because the responsible intervertebral disc cannot be determined definitively. Follow-up MRI performed with a relatively short interval may contribute to the diagnosis of FCE when chronological changes in the disc are observed [1]. Diffusion-weighted imaging sequence on MRI is also reported as a good modality because abnormalities can be found even within a few hours in patients with spinal cord ischemia. However, MRI sometimes reveals normal results in the early stage of FCE. Therefore, repeat MRI with a T2WI sequence or an enhanced MRI may be needed later [11]. Upon the diagnosis of FCE, it's recommended to start a long-term rehabilitation with home physiotherapy which led to significant improvement, albeit with noticeable deficits.

## CONCLUSION

In our patient, the clinical history in combination with cerebrospinal fluid and MRI features were suggestive of FCE. This entity should be kept as one of the differential diagnoses of an acute neurological deficit of the spinal cord.

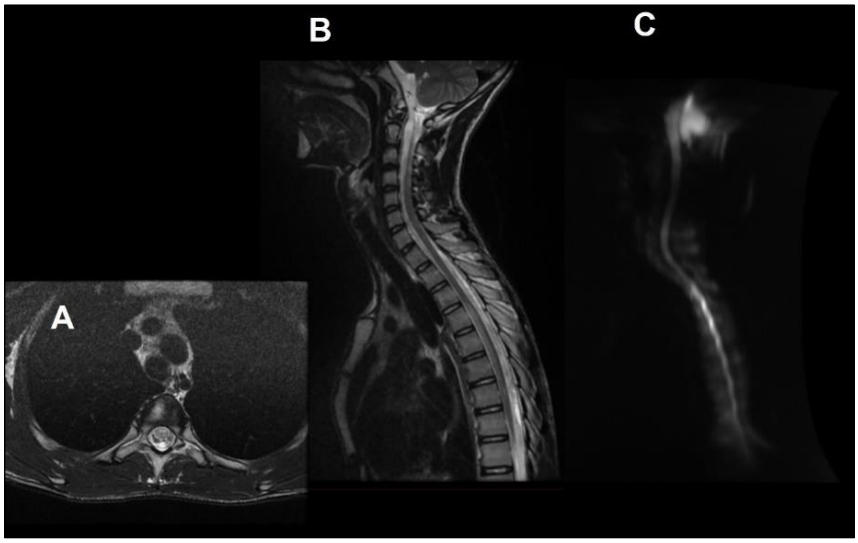


**Figure 1:**

A) Spinal MRI in axial T2-weighted section: central hyper signal from the anterior horns of the spinal cord in snake eyes.
B) Spinal MRI in sagittal section T2 sequence: hyper signal of the cord next to D4-D7
C) Diffusion sequence in the sagittal plane confirming the presence of spinal hypersignal.

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