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Case report

Pediatric fibrocartilaginous spine embolism induced by trauma ^{☆,☆☆}

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ABSTRACT

Fibrocartilaginous embolic infarction of the spinal cord is a rare cause of acute back pain and motor weakness. Most symptoms start after minor trauma that is often considered harmless and forgotten, however these minor injuries can result in lethal consequences. It is quite rare to diagnose fibrocartilaginous embolism in a timely manner and start treatment to prevent poor outcomes. We present the case of a previously healthy eight-year-old female with sudden onset neck pain and progressive bilateral upper extremity weakness following an injury while playing with her younger sister. Magnetic resonance imaging of the cervical spinal cord without contrast revealed a posterior disc protrusion suggestive of post-traumatic spinal cord infarction due to fibrocartilaginous embolism. In young, otherwise healthy, patients with acute motor deficits, radiographic imaging can help identify rare presentations like fibrocartilaginous embolism in order to rapidly diagnose and efficiently treat such patients.

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Introduction

Fibrocartilaginous embolism is a rare cause of spinal cord infarction, gaining prominence in the literature when the first reported case in 1961 described the death of a fifteen-year-old male after a “harmless fall” during a basketball game [2]. The source of a fibrocartilaginous embolism is the internal nucleus pulposus of the intervertebral disc, and

the embolism can navigate its way into the thin-walled blood vessels lining the intervertebral discs [1]. These blood vessels begin to regress shortly after birth with complete disappearance by the second decade of life [1]. The incidence of fibrocartilaginous emboli is bimodal, with peaks observed around adolescence, due to the persistence of blood supply between spinal cord and nucleus pulposus, and older age, from neovascularization due to degenerative disc disease [1].

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Fig. 1 – Sagittal T2-weighted MRI sequence showing posterior disc protrusion at the C5-C6 level.



Fig. 2 – Sagittal STIR MRI sequence demonstrating increased signal consistent with edema within the anterior cord from C4 through C7 with minimal spinal cord expansion.

Case report

A previously healthy eight-year-old female presented with sudden onset back and neck pain with bilateral upper extremity weakness following repetitive kicks to her back between the shoulder blades while playing with her sibling. The patient did not lose consciousness, but reported feeling weak and being unable to move her hands later that night. The following morning, the mother noticed that the weakness in her daughter's hands had worsened significantly, making it difficult for the patient to pick up things.

Upon arrival at the hospital, the patient presented with stable vitals, back and neck pain, weakened grip in bilateral hands, and no sensory deficits in the upper extremities. The patient had intact motor and sensory function in the lower extremities and was alert and interactive with medical staff. Neurological exam revealed absence of motor function in her triceps, wrist flexors, wrist extensors, and intrinsic muscles of the hands bilaterally (0/5 on muscle strength grading scale). The patient was only able to activate her biceps and abduct her shoulders demonstrating 5 out of 5 strength bilaterally. There was no difficulty with urination, gait, or bowel function, and perianal sensation and rectal tone were intact. The patient underwent cervical spine magnetic resonance imaging (MRI) without contrast to evaluate for fracture or spinal cord injury as well as x-ray of the left humerus and right shoulder. The T1 weighted imaging demonstrated a posterior disc protrusion at the C5-C6 level (Fig. 1 & 3) with increased signal on the STIR consistent with edema within the anterior cord from C4 through C7 as well as minimal spinal cord expansion (Fig. 2). The axial T2 weighted images localized the edema to the anterior 2/3 of the cord (Fig. 4). Additionally, diffusion weighted images demonstrated increased signal in the anterior cord spanning C4-C6 with corresponding signal drop out on ADC



Fig. 3 – Sagittal T1-weighted MRI with arrow pointing to disc protrusion at C5-C6.

consistent with diffusion restriction (Fig. 5). Five days later the post gadolinium MR cervical spine was performed which showed linear long enhancement (Fig. 6) in the anterior third of the cord at the same level where restriction diffusion was seen in the prior scan. Pseudo-normalization of the restricted diffusion was noted (Fig. 7), confirming the diagnosis of subacute infarct. Given the above findings within the spinal cord in the presence of disc protrusion and history of trauma, fibrocartilaginous embolic infarction was strongly suspected. However, given the long segment hyperintense signal with overlap

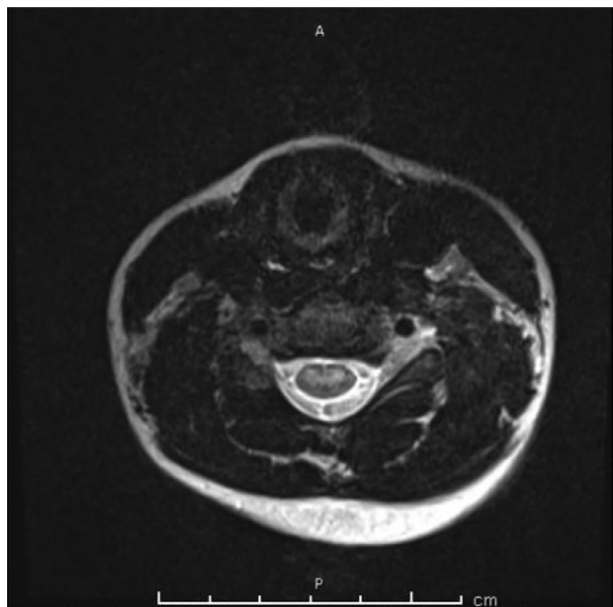


Fig. 4 – Axial T2 weighted MRI highlighting edema localized to the anterior 2/3 of the cervical spinal cord.

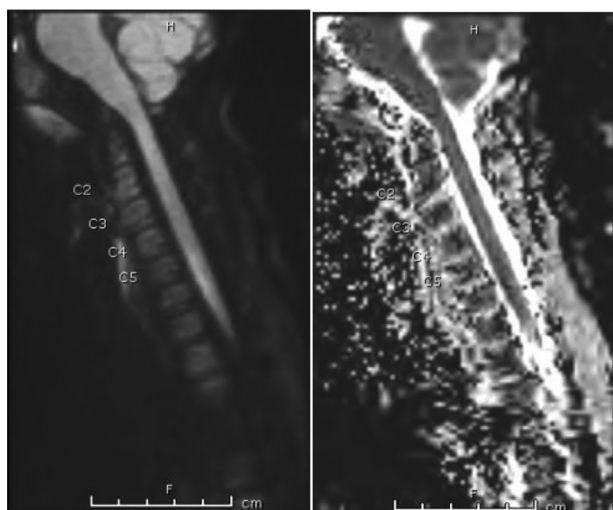


Fig. 5 – DWI (left) and ADC (right) with increased signal on DWI and signal drop out on ADC in the anterior Cord from C4-5 through C5-6 consistent with diffusion restriction.

of findings with a potential inflammatory myelitis, a follow up lumbar puncture and cerebrospinal fluids (CSF) analysis were ordered to help rule out inflammatory myelitis such as NMO. Results showed < 3 nucleated cells per μL , < 2 red blood cells per μL , total protein 28 mg/dl and glucose of 56 mg/dl. Tests for viral and bacterial sources of infection were all negative. Zero oligoclonal bands were observed in the CSF and no myelin oligodendrocyte glycoproteins were detected in the sample, making an inflammatory source unlikely.

After ruling out inflammatory causes for the patient's symptoms, the use of steroids was not recommended as treatment for our patient. Instead, she was treated with an intensive inpatient rehabilitation program requiring 3 hours of

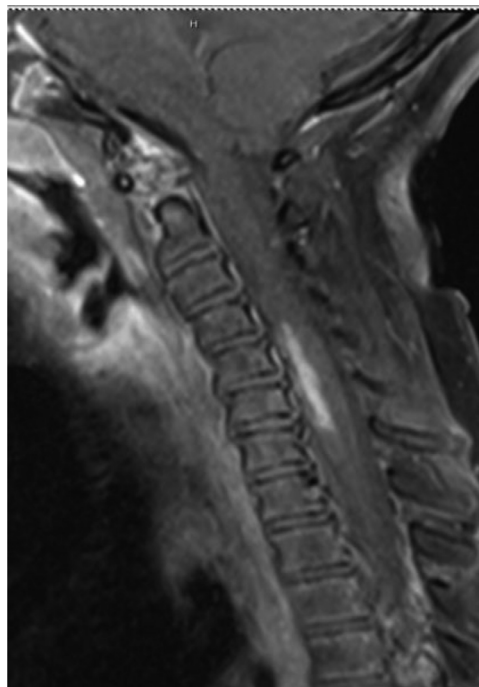


Fig. 6 – Post gadolinium MR cervical spine with linear long enhancement.

therapy for at least 5 days a week. While working with physical medicine and rehabilitation, the patient completed and reached her rehabilitation goals such as completing self-care tasks, activities of daily living (ADLs), and ambulating independently within 10 days of inpatient therapy and was subsequently discharged. Upon discharge, the patient was scheduled to follow up with outpatient pediatric neurology within 2 months and recommended to obtain an EMG. Unfortunately, the patient was lost to follow up and had not attended her outpatient neurology appointments or EMG.

Discussion

Fibrocartilaginous embolic infarction is a rare and often undiagnosed cause of spinal cord infarctions. It is a clinical diagnosis limited by definitive correlation via postmortem examination of the vascular supply to the spinal cord [3]. However, the diagnosis of fibrocartilaginous embolism should remain on the differential for acute paralysis or weakness from minor trauma or physical activity immediately preceding symptom onset [4]. The incidence of fibrocartilaginous embolism is higher in females and presents with bimodal peaks in late adolescence and late middle age [5]. Patients often presented with sudden back pain as the initial symptom, later followed by motor deficits ranging from weakness to paralysis within minutes to hours depending on the extent of injury from infarction [6]. In addition, patients may present with sensory deficits and loss of autonomic functions such as bladder or bowel dysfunction.

It is still not clearly understood how the fibrocartilaginous embolism enters the vascular system, but the commonly accepted theory is that a strong force from trauma or physi-



Fig. 7 – DWI scan with pseudo-normalization of the restricted diffusion.

cal exertion leads to expulsion of the fibrocartilaginous nucleus pulposus into the spinal cord vascular system [1]. Another common cause is the presence of Schmorl's nodes, small nodes of fibrocartilage located within the vertebrae that are located in close proximity to the blood vessels supplying the vertebral body, which results in infarction with herniation from increased axial forces applied along the spine [1,6]. In younger patients, the vascular supply to the vertebral column has not yet regressed, as it occurs during the second decade of life [1]. The blood supply of the nucleus pulposus regresses in adolescents as a result of Fas ligand expression, which leads to vascular endothelial cell apoptosis [7]. As the blood vessels undergo apoptosis, the intervertebral discs receive nutrients from the cartilaginous endplates by diffusion or by the blood vessels still present in the outer annulus fibrosis [7]. In older patients, the process of neovascularization from degenerative disc disease leads to increased occurrence of spinal cord infarctions [4]. In these patients, the increased intradisc pressure induces collagen disruption and loss of proteoglycans in

the nucleus pulposus [8]. The loss of proteoglycans in patients with degenerative disc disease stimulates an anabolic process that drives the growth of blood vessels and nerves, allowing emboli to traverse the new blood supply in older patients who suffered with spinal cord trauma [8].

Diagnosing fibrocartilaginous emboli based on MRI is difficult as early stages of the infarction will present normally. The presence of a narrowed disk or Schmorl nodes in addition to expanding spinal cord with increased signal on T2 weighted image (T2WI) without early contrast enhancement is highly suggestive of a fibrocartilaginous embolus [3]. Repeat MRI with a T2WI sequence or enhanced MRI may be important. In the initial stages of presentation, MRI studies presented with signs of infarction with cord swelling and increased T2 signal in the acute phase followed by a chronic phase characterized by atrophy and nonuniform signal intensity [6].

For our patient, we were considering an inflammatory condition as the cause of her symptoms given her family history of multiple sclerosis. However, on follow up lumbar puncture and CSF analysis, results were normal and had no indication for an inflammatory source to cause the patient's symptoms. The presence of a tiny central disc protrusion at the C5-C6 level on MRI supports post-traumatic spinal cord infarction due to fibrocartilaginous embolism.

There is no well-defined treatment for fibrocartilaginous emboli within the spinal cord. Previous patients have been treated with anticoagulation therapy, corticosteroids, or even emergent surgery, however, there was no significant improvement in patients clinically [3]. Patients have also been managed in the long term with blood pressure control, physical therapy, and supportive care [3]. Our patient has been following physical therapy and occupational therapy as well as recommendations of physical rehabilitation specialists to develop strength in her distal upper extremities.

Conclusion

The pathophysiology of fibrocartilaginous emboli causing spinal cord infarction remains rare in the literature and in clinical practice as many patients do not often survive such "harmless" traumatic events. Based on radiographic imaging and clinical presentation of our patient, we established the presence of fibrocartilaginous embolism. After examining similar fibrocartilaginous embolic cases, we hope to increase awareness of such similar cases in medical practice and the literature in order to help rapidly diagnose and efficiently treat such patients.

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