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Case Report

Spinal Cord Infarction Due to Fibrocartilaginous Embolism: A Case Report and Review of Diagnostic Clues

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Abstract: Spinal cord infarction is a rare entity, accounting for 1-2% of acute ischemic disorders of the central nervous system. Fibrocartilaginous embolism (FCE), characterized by migration of nucleus pulposus material into the spinal cord vasculature, constitutes an uncommon and underrecognized etiology. We describe the case of a 49-year-old woman, previously independent, who developed acute myelopathy with flaccid tetraparesis, C5 sensory level, and dysautonomia. Magnetic resonance imaging (MRI) revealed an extensive intramedullary lesion from C4 to T1 with diffusion restriction and disc extrusion at C6-C7 with annular fissure. After exclusion of other etiologies, the diagnosis of spinal cord infarction secondary to FCE was established. The patient started antiplatelet therapy, statin, and intensive rehabilitation, with neurological stability but without significant early functional recovery. This case illustrates the importance of considering this entity in adults with low vascular risk and disc degeneration, highlighting the crucial role of diffusion-weighted MRI in diagnosis, thereby ensuring appropriate management with impact on functional outcomes.

Keywords: Spinal Cord Infarction; Fibrocartilaginous Embolism; Acute Myelopathy; Magnetic Resonance Imaging.

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1. Introduction

Spinal cord infarction is a rare neurological condition, representing approximately 1-2% of all acute ischemic disorders of the central nervous system [1,2,3]. Despite its low frequency, it is associated with significant morbidity, including severe neurological deficits and a substantial impact on patients' quality of life [3]. Unlike cerebral stroke, which is frequently related to atherosclerosis and cardioembolism, spinal cord infarction has a more heterogeneous etiology, particularly in younger populations, including iatrogenic causes (notably thoracic aortic surgery), aortic disease, atherosclerotic embolism, systemic hypoperfusion, and, more rarely, fibrocartilaginous material embolization [4-6].

This latter entity, known as fibrocartilaginous embolism, was first described in 1961 and is characterized by embolization of nucleus pulposus material from the intervertebral disc into the spinal vasculature. Fibrocartilaginous material may gain vascular access through several mechanisms, including disc revascularization associated with aging or degenerative disc disease, Schmorl's node formation, or persistence of disc vascularization into adulthood. The triggering event is often linked to increased intradiscal or intravertebral pressure, driven by axial loading forces on the spine during trauma, Valsalva maneuvers, or strenuous physical activity. Once in circulation, the embolus can reach the spinal cord via arterial or venous routes, causing ischemia and subsequent tissue necrosis [7].

Clinically, FCE is characterized by sudden onset cervical or thoracic pain, followed by rapid development of severe neurological deficits, including paraplegia or tetraplegia, sensory loss, and autonomic dysfunction. A time to nadir of less than 12 hours serves as a

strong clinical predictor of diagnosis [6,10]. This presentation often mimics other causes of acute myelopathy, such as transverse myelitis, spinal cord compression, or other forms of spinal cord ischemia, making diagnosis a significant clinical challenge [7]. MRI plays a central role in diagnosis, typically demonstrating T2 - hyperintense spinal cord lesions that, unlike inflammatory processes, generally lack gadolinium enhancement. Additionally, these lesions exhibit diffusion restriction on DWI sequences, corroborating the ischemic nature, and are frequently associated with degenerative disc changes at the corresponding level [5-8].

Therapeutic options are limited, with no specific treatment proven effective. Management is essentially supportive, focusing on early motor and functional rehabilitation, alongside the prevention of complications [7,9]. The prognosis is generally guarded, heavily influenced by the extent of the spinal cord lesion and the initial severity of motor dysfunction. Consequently, early intensive rehabilitation is paramount to optimize functional outcomes [2,7,9].

This case report aims to illustrate the clinical presentation and course, as well as the radiological findings of a spinal cord infarction due to FCE, emphasizing the importance of high diagnostic suspicion and judicious use of MRI in approaching this rare entity.

2. Case Report

A 49-year-old Caucasian woman, previously independent (modified Rankin Scale 0), with a history of arterial hypertension controlled with enalapril 20 mg and lercanidipine 10 mg, and hypothyroidism managed with levothyroxine 75 μg, was admitted to the emergency department with a 12-hour history of acute myelopathy. She denied recent history of trauma, Valsalva maneuvers, strenuous physical activity, cervical hyperextension or manipulation, or acute thoracic or abdominal pain.

On initial neurological examination, she presented flaccid tetraparesis, demonstrating plegia of the lower limbs (grade 0/5 on the Medical Research Council scale) and severe upper limbs' paresis (grade 2/5 proximally and grade 1/5 distally). A sensory level at C5 was identified, with loss of light touch and pain sensation below this dermatome. Deep tendon reflexes were absent in all four limbs, and plantar responses were bilaterally extensor. She also exhibited signs of dysautonomia, specifically arterial hypotension (blood pressure 90/60 mmHg) and acute urinary retention requiring catheterization.

Computed tomography (CT) of the brain showed no acute changes, while CT of the cervical and thoracic spine revealed diffuse degenerative disc disease, with posterior disc herniations at C5-C6 and C6-C7 causing dural sac compression, without evidence of fractures or acute bony injury. Laboratory testing demonstrated negative inflammatory markers. Cerebrospinal fluid (CSF) analysis revealed cellularity <2 cells/μL, normal protein levels and normal glucose, excluding inflammatory or infectious central nervous system processes. Spinal cord MRI, performed 24 hours after symptom onset, revealed an intramedullary lesion with contiguous extension from C4 to T1, demonstrating T2 hyperintensity (Figure 1) and diffusion restriction on DWI sequences with a corresponding hypointense signal on the apparent diffusion coefficient (ADC) map (Figure 2), without gadolinium enhancement. These findings were highly suggestive of acute ischemic injury. Additionally, posterior disc extrusion at C6-C7 was observed, causing spinal cord deformation and signs of an annular fissure at the corresponding level (Figure 3).

Following multidisciplinary discussion with Neurosurgery, surgical decompressive intervention was not indicated, given the absence of significant acute spinal cord compression and the established ischemic nature of the lesion. Single antiplatelet therapy with acetylsalicylic acid (100mg/day) and high-intensity statin therapy with atorvastatin (40mg/day) were initiated, combined with a structured motor and functional rehabilitation program. The complementary etiological workup revealed no evidence of a cardioembolic source on transthoracic echocardiogram and Holter monitoring.

Figure 1. Cervical spine MRI (sagittal T2 sequence) showing extensive intramedullary hyperintensity, with contiguous involvement from C4 to T1.

Figure 2. Cervical spine MRI (sagittal diffusion sequences) demonstrating DWI hyperintensity with corresponding hypointense signal on ADC map, compatible with diffusion restriction.

Doppler ultrasonography of the neck vessels showed no evidence of arterial dissection or hemodynamically significant stenosis. A thrombophilia screen (lupus anticoagulant, anticardiolipin antibodies, anti-beta-2-glycoprotein I, protein C, protein S, antithrombin III, factor V Leiden, prothrombin mutation) was unremarkable. Additionally, an extended autoimmune panel (including anti-aquaporin-4 and anti-MOG antibodies) and infectious serologies (HIV, syphilis, Borrelia, herpes viruses) were negative. Given the absence of an alternative etiology and imaging findings of a disc fissure at C6-C7 adjacent to the ischemic spinal cord lesion, the diagnosis of spinal cord infarction secondary to fibrocartilaginous embolism was established.

Figure 3. Cervical spine MRI (sagittal view) showing posterior disc extrusion at C6-C7, with spinal cord deformation and signs of annular fissure at the corresponding level.

During hospitalization, the patient remained clinically stable, without progression of neurological deficits or the need for ventilatory support. After three weeks, she was transferred to a specialized Physical Medicine and Rehabilitation center to continue an intensive rehabilitation program. At the time of transfer, she presented with persistent severe tetraparesis (modified Rankin Scale 4), showing a slight improvement in proximal upper limb muscle strength (grade 3/5), but without recovery of remaining deficits. During the three- month post-discharge monotoring, mild functional improvement was observed; she was able to perform some activities of daily living with assistance, although she remained wheelchair-dependent on mobility.

3. Discussion and Conclusion

This case illustrates the typical clinical and radiological characteristics of fibrocartilaginous embolism as a cause of spinal cord infarction. The patient presented several highly suggestive features of this etiology: relatively young age, the absence of significant vascular risk factors, sudden onset of severe neurological deficits, and imaging evidence of a disc fissure adjacent to the ischemic spinal cord lesion. The absence of a clear precipitating event does not exclude the diagnosis, as apparently innocuous activities can trigger this entity [9,10].

The pathophysiology of FCE in this case aligns with the established literature. Fibrocartilaginous material from the nucleus pulposus likely entered the vascular network via

disc revascularization secondary to degenerative disc disease, as evidenced by the multilevel degenerative changes and disc herniations at C5-C6 and C6-C7. The annular fissure at C6-C7 suggests an acute disc extrusion event that may have increased intradiscal pressure, thereby facilitating the embolization of fibrocartilaginous material [7]. Once in the circulation, the emboli reached the spinal cord vasculature, inducing ischemia across the C4-T1 segments, which correlates with the clinical presentation of tetraparesis and a C5 sensory level. The comprehensive workup performed in this case effectively excluded alternative causes, further substantiating the diagnosis of FCE.

Therapeutic options for FCE remain limited; in this case, antiplatelet therapy and a statin were initiated, following the principles of secondary prevention of ischemic events, although their specific efficacy in FCE is not established. While some studies suggest that early corticosteroid therapy may exert protective effects on cellular function and reduce oxidative stress following ischemic injury, evidence remains limited [9]. Consequently, intensive rehabilitation remains the cornerstone of treatment.

The patient's clinical evolution, characterized by persistence severe tetraparesis at 3 months, aligns with the prognosis described in the literature for spinal cord infarction. Studies report that only 18-41% of survivors achieve independent ambulation at hospital discharge, while 20-57% remain wheelchair dependent. The initial severity of motor dysfunction constitutes the main predictor of functional outcomes [9]. In this case, the severity of the baseline deficit portended a guarded prognosis, as observed. This case highlights the importance of considering FCE in the differential diagnosis of acute myelopathy, particularly in young or middle-aged individuals with low vascular risk and evidence of disc degeneration. The combination of a typical clinical presentation, suggestive MRI findings, and the rigorous exclusion of other etiologies allows the clinical diagnosis to be established with high probability.

In conclusion, FCE represents a rare and likelly underdiagnosed cause of spinal cord infarction. Greater awareness of this entity among clinicians may improve its recognition and the understanding of its epidemiology and natural history. Furthermore, the identification of more precise prognostic markers and the development of potential neuroprotective therapies constitute crucial areas for future research aimed at optimizing functional outcomes in these patients.

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