# Fibrocartilaginous Embolization - a Rare Cause of Spinal Cord Infarction: Case Report

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ABSTRACT

A case of fibrocartilaginous embolization in 24-year-old female as a rare cause of spinal cord infarction is presented. It manifests as infarction syndrome with rapid progression of clinical signs - acute onset of quadriparesis and respiratory insufficiency. Among imaging studies MRI is the most accurate as it readily capable of detection of myelopathy and acute intervertebral disc lesion. Other laboratory tests and imaging modalities are usually normal. The final diagnosis is made by exclusion.





## CASE REPORT

### CASE REPORT

A 24-year-old female suddenly felt sharp pain in the thoracic region between scapulae while exercising sit-ups. She was admitted to hospital with acute onset of quadriparesis and respiratory insufficiency. During the diagnostic process, complete pentaplegia developed with the need for tracheal intubation and mechanical ventilation. Laboratory tests on admission were negative. Urgent head and cervical spine CT including CT angiography of the head were performed with a negative results. Because of suspected lesion of the spinal cord, urgent brain and cervical spine MRI examination was performed. Brain MRI scans were negative, but there were moderate signal changes found in anterior horns of the cervical spine reaching from the cranio-cervical transition to C6/7 (Fig. 1). Early follow-up MRI showed a progression of spinal cord abnormalities with typical radiological signs of an ischemic spinal cord lesion extending from the medulla oblongata down to Th2/3 (Fig. 2). Genetic and antibody screening tests did not confirm vasculitis nor congenital thrombophilic states; hemocoagulation tests were completely normal. Transesophageal ultrasound examination did not find the source of emboli and a spinal liquor tap revealed no abnormalities regarding inflammation. Percutaneous dilation tracheostomy was performed because of the necessity of prolonged mechanical ventilation. Patient was treated symptomatically with corticosteroids, anticoagulants, antiphlogistics and antibiotics. After 4 weeks, first signs of motor activity appeared, later on followed by spontaneous ventilation. Follow-up MRI scan revealed indistinctive regression of expansile lesions in the cervical spine and medulla oblongata (Fig. 3). During following one-year period, the patient's condition improved, with the onset of spontaneous breathing followed by motor function repair. Late follow-up MRI examination showed persistent pathological intramedullary signal consistent with MRI signs of myelopathy.

## DISCUSSION

*Etiology & Demographics:*

The first case report of fibrocartilaginous embolization was published in 1961 and it referred to a 15-year-old boy who became quadriplegic with breathing difficulties after minor fall on his coccyx [1,2,3]. Since then, several other cases were described in human medicine; on a contrary much more of them are presented in veterinary medicine literature. In human population, cases of patients with ages ranging from the first to the sixth decade with two peaks - in adolescent and in middle age - were described [4,5]. Some authors showed a higher incidence of the condition in young women and lesions occurring predominantly in the cervical spine [6], while others failed to prove gender differences and showed predominant occurrence of the lesions in the thoracic spine [7]. Since most of the cases published in literature were fatal, it can be assumed that there is a higher probability of cervical embolization since thoracic embolization is mostly non-fatal in the acute stage [5].

The cause of spinal cord ischemia is thought to be a migration of fibrocartilaginous nucleus pulposus material through the nearby vasculature to embolize into one of the spinal cord vessels, but the exact mechanism of embolization is still debated in many theories [8]. Young people have a conjoint blood supply for the nucleus pulposus and the spinal cord [1,4], that may be responsible for the higher incidence of fibrocartilaginous embolization in this age group. During adolescence, the disc becomes avascular. Neovascularization occurring with disc degeneration may stimulate similar conditions for fibrocartilaginous embolization in older age groups [4]. Another alternative may be high axial overload to the spine and intervertebral discs, that may lead to embolization of the nucleus pulposus into the vertebral bone marrow and from there to the connecting sinusoids and venules [4,7], or to retrograde embolus flow into the segmental arteries and then an antegrade flow into the spinal cord. [5]. Histopathological and imaging studies have not shown bleeding around the site of embolization, which would be expected if the cause for embolism was of traumatic origin [5].

*Prognosis:*

Prognosis of the disease is very unfavorable since, the definitive diagnosis can be established only by autopsy. It is possible that some cases of spinal ischemia clinically diagnosed as fibrocartilaginous embolization may have been caused by other unknown conditions [8, 9].

*Clinical Findings:*

From the neurological point of view, fibrocartilaginous embolization is a stroke syndrome of the spine [2]. It usually presents as a sudden onset of sharp back and neck pain with rapid progression of signs indicative of spinal ischemia [5]. Clinical signs vary depending on the extent of spinal cord injury from paraplegia (when thoracic cord is injured) to pentaplegia with respiratory insufficiency or even death (in case of injury to the cervical spine and medulla oblongata).

Even though there are no clear diagnostic criteria for fibrocartilaginous embolization, several authors have tried to specify clinical signs that make this diagnosis very probable [4,5,7,10,11,12]. Sudden onset of neurological impairment, minimal or no trauma to the back, accentuated stress to the back, Valsalva maneuver and radiologic attributes of spinal cord ischemia are all among these characteristics. Other signs that make the diagnosis highly probable are the absence of vascular risk factors, negative spinal tap and the absence of other signs explaining spinal ischemia (e.g. fistula or arteriovenous malformation of the spinal cord). Currently the diagnosis of fibrocartilaginous embolization is made by exclusion relying on patient history and clinical presentation [8].

*Imaging Findings:*

MRI imaging plays a significant role in the diagnostic process because of its high sensitivity in detection of possible pathologies of the spinal cord; on the other hand it has a relatively low specificity [5]. MRI can directly confirm spinal ischemia and rule out other causes of the disease. Other neurodiagnostic methods are in most of the cases only complementary.

The most important in establishing the diagnosis are T2 weighted images; in the acute phase, spinal cord edema with expansile effect and an abnormal intramedullary hyperintensity mainly in anterior columns are visible. In the early course of the injury, diffusion weighted images are helpful in identifying typical “ischemic” cytotoxic edema, with hyperintense signal in high B values and hypointense signal in ADC map. Another common sign is damage to intervertebral disc at the level of myelopathy and bone marrow edema neighboring the zone of disc herniation [5,12]. The intramedullary lesions typically do not enhance with gadolinium [8], but minimal postcontrast enhancement of injured adjacent structures may be present.

In the late-stage, spinal atrophy with heterogeneous signal is present [5].

*Treatment:*

To date there are no available specific treatment options for fibrocartilaginous embolization [8]. The treatment involves prevention of thromboembolic complications, therapy of spinal edema, plasmapheresis, that at the same time represents a preventive measure in case of spinal cord ischemia from other reasons. It also implies treatment of acute complications calling for intensive care therapy. Ultimately, there is possibility of rehabilitation, psychological and psychiatric care.

*Differential Diagnosis:*

The most common cause of spinal infarction is atherosclerosis (cholesterol or atheromatous emboli) [8,13]. If spinal ischemia is suspected we have to rule out a traumatic cause (prolapse or intervertebral disc herniation with spinal cord compression), cardiovascular diseases (prolonged arterial hypotension, thrombo-embolic disease, vasculitis, systemic

lupus erythematosus, antiphospholipid syndrome, dissecting aortic aneurysm, hypertensive microvasculopathy (small vessel disease), infection, and spinal involvement in disseminating carcinoma (carcinomatous meningitis). Despite an extensive array of possible causes, as much as 74% of spinal ischemias have no identifiable cause [4,9].

For differentiation of various etiologies of spinal infarction, clinical signs in correlation with diverse diagnostic tests are primarily used.

As the first step imaging modality, computed tomography or magnetic resonance is used to rule out traumatic compressive myelopathy caused either by extruded disc or collapsed vertebrae. Angiography of the aorta and spinal canal can rule out aortic dissection, occlusion of major cranial vessels, arteriovenous malformation or dural fistula. MR contrast enhanced scans can show a pathologic meningeal enhancement caused by infectious or carcinomatous processes. Presence of cardiac or aortic emboli can be detected by echocardiography. Main imaging characteristics of other potential pathological conditions are summarized in Table 2. Various laboratory tests are used to search for infectious or inflammatory processes, different types of vasculitis, hypercoagulation states, metabolic or demyelination diseases [7].

## TEACHING POINT

Fibrocartilaginous embolization represents a rare cause of spinal cord infarction and the diagnosis is made by exclusion. It is based on non-specific clinical information, negative laboratory tests, absence of vascular risk factors, negative spinal tap and positive MRI signs of spinal cord ischemia and in many instances acute intervertebral disc injury at the level of myelopathy.

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FIGURES

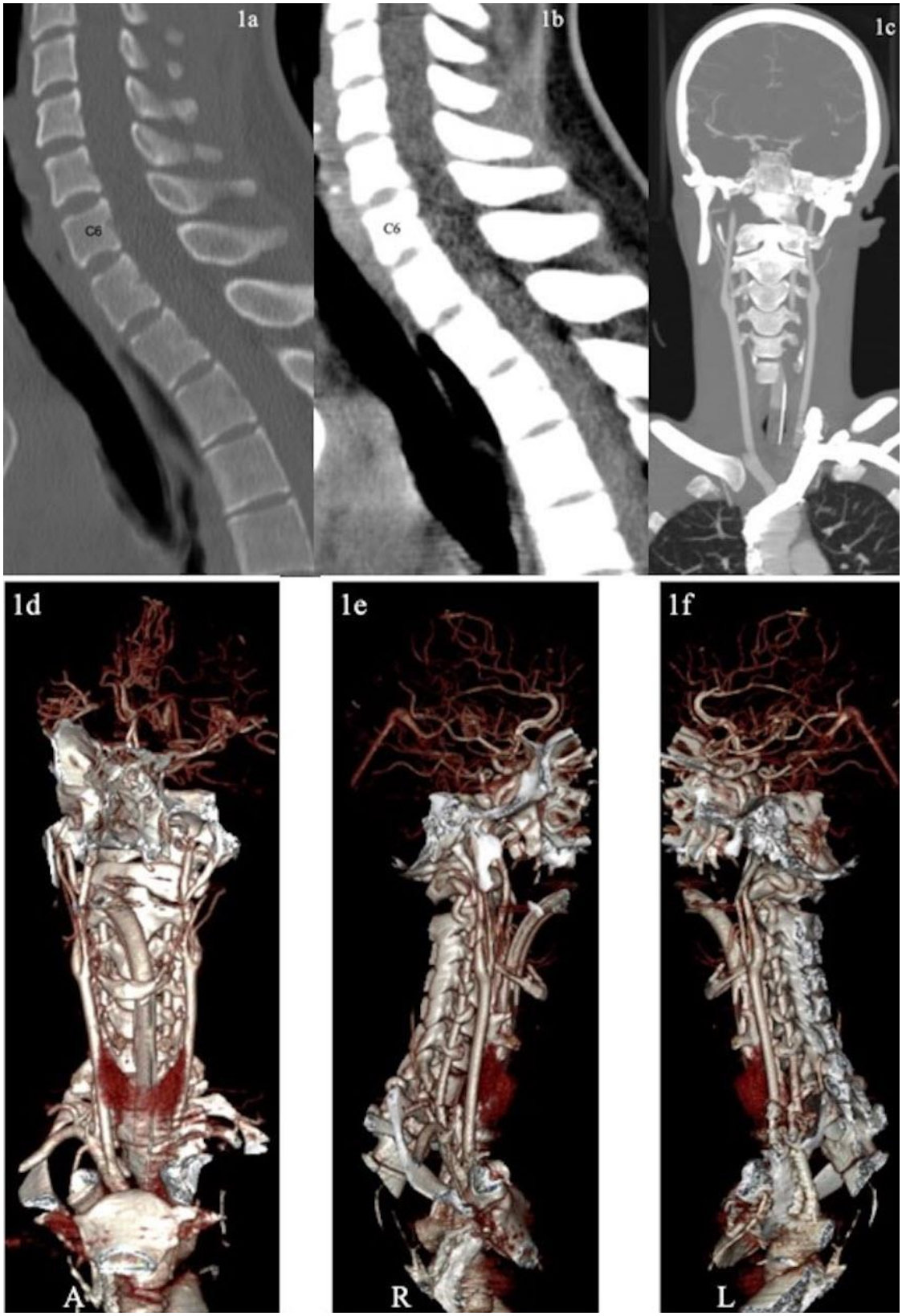


**Figure 1:** 24-year-old female with fibrocartilaginous embolization to the cervical spinal cord.
FINDINGS:
(a, b) CT of the cervical spine is negative. It shows normal width and no visible pathology in the spinal canal, no traumatic vertebral injury or discs protrusion.
(c, d, e, f) CT angiography of the extra- and intracranial arteries is normal; no significant stenosis or occlusion is visible.
TECHNIQUE: CT Siemens Sensation 16, 310 mAs, 120 kV. (a) Non-contrast CT, sagittal reformation, 3mm slice thickness, B70s very sharp and (b) B31s medium smooth. (c) coronal MIP images and (d, e, f) VRT, CT angiography, 80ml ioversolum (Optiray® 350).

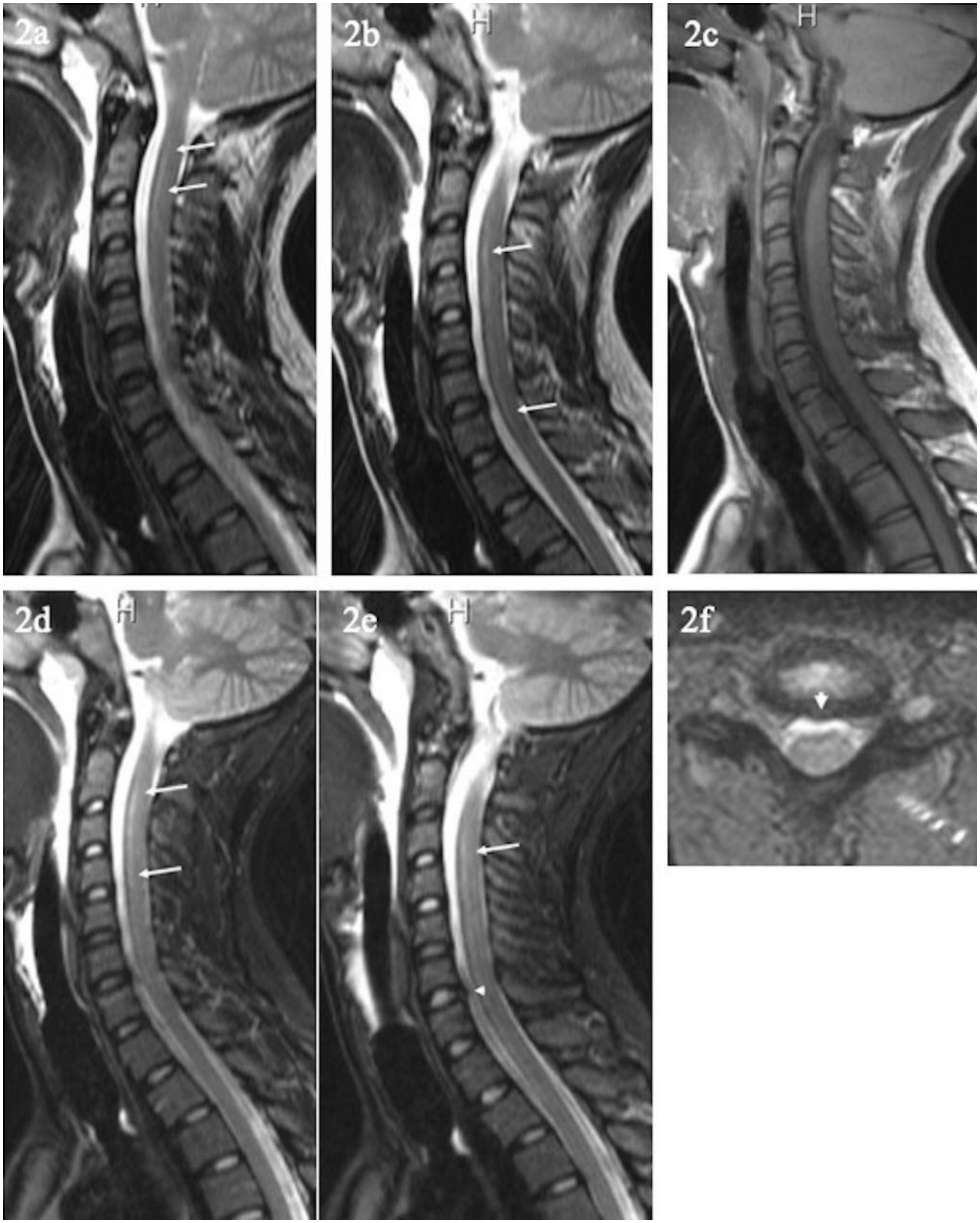


**Figure 2:** 24-year-old female with fibrocartilaginous embolization to the cervical spinal cord.
FINDINGS: (a, b) MRI of the cervical spine shows moderate T2W hyperintensity in anterior horns of cervical spinal cord from the cranio-cervical transition down to C6/7 (arrows). (c) T1W images doesn't show any abnormality. (d, e) On sagittal TIRM sequence, the intramedullary hyperintensity is even more visible. (e, f) Arrowheads show a mild protrusion of C6/7 intervertebral disc to the spinal canal visible on sagittal TIRM and axial T2W image.
TECHNIQUE: 1,5T Siemens MAGNETOM Avanto MRI scanner. (a, b) Sagittal T2W, TR 3400, TE 102, 3mm slice thickness. (c) Sagittal T1W, TR 570, TE 10, 3mm slice thickness. (d, e) Sagittal TIRM W, TR 4700, TE 73, TI 150, 3mm slice thickness. f) Axial T2*, TR 1670, TE 27, 3mm slice thickness.

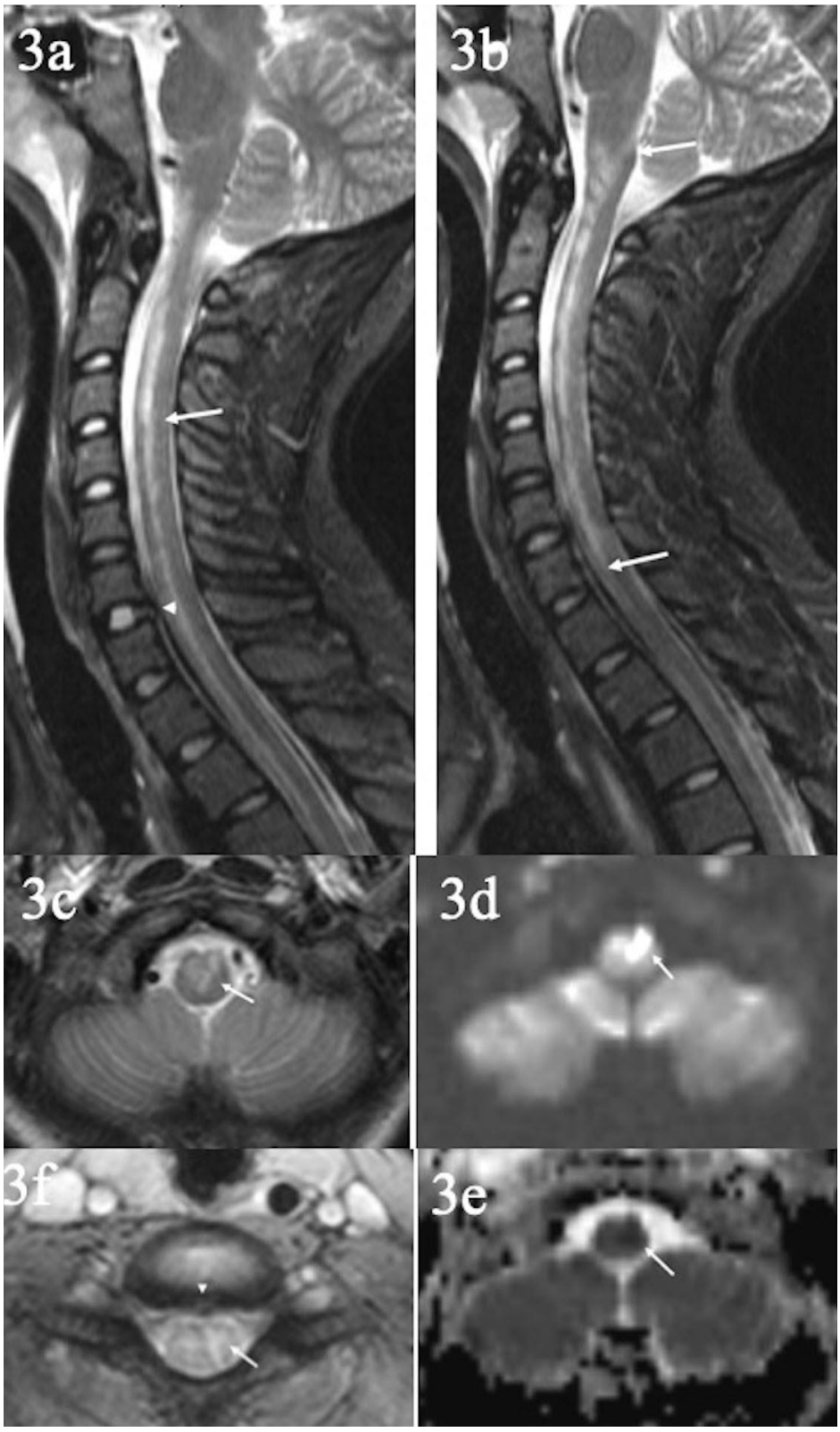


**Figure 3:** 24-year-old female with fibrocartilaginous embolization to the cervical spinal cord.
FINDINGS: A follow up MRI two days later shows progression of spinal cord hyperintensity on TIRM sequence (a, b) extending from the medulla oblongata to Th2/3 level. Arrowhead indicates intervertebral disc protrusion and nucleus pulposus rupture (central hyperintensity of nucleus pulposus extending to the hypointense anulus fibrosus). (c) Axial T2W image at the level of medulla oblongata shows hyperintense signal with restricted diffusion on DWI b=1000 (d) with hypointense signals on ADC map (e) - a finding corresponding to cytotoxic edema. (f) Axial T2*W image at the level of C6/7 shows both - disc protrusion (arrowhead) and hyperintensity affecting anterior horns of the cervical spinal cor.
TECHNIQUE: 1,5T Siemens MAGNETOM Avanto MRI scanner. (a, b) Sagittal TIRM W, TR 5000, TE 73, TI 150, 3mm slice thickness. (c) Axial T2W, TR 4000, TE 99, 3mm slice thickness. (d, e) Axial DWI/ ADC, TR 3200, TE 94, B = 0/1000, 5mm slice thickness. (f) Axial T2*W, TR 1160, TE 27, 3mm slice thickness.

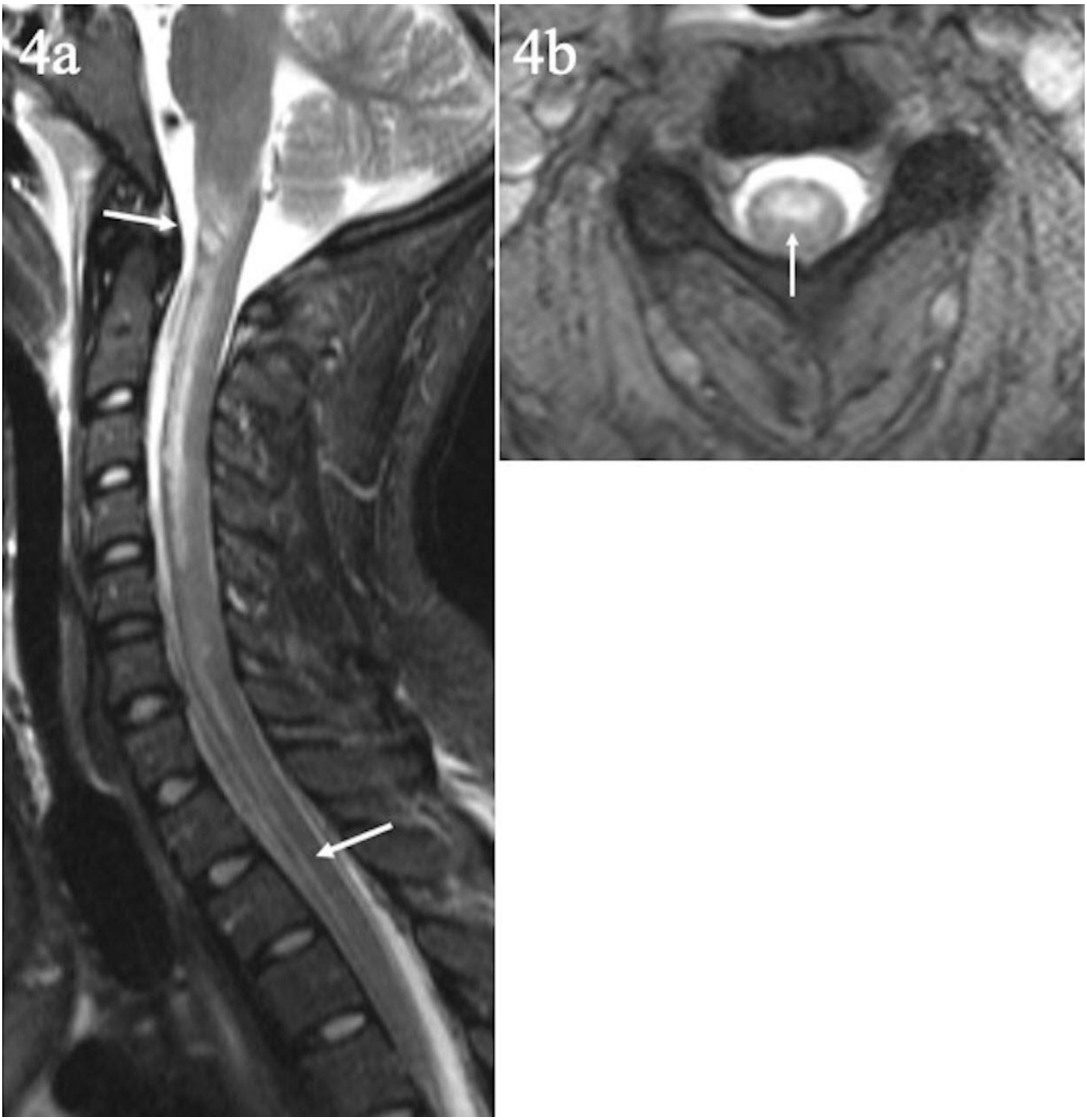


**Figure 4:** 24-year-old female with fibrocartilaginous embolization to the cervical spinal cord.
FINDINGS: A follow up MRI of cervical spine 14 days later revealed mild regression of expansile lesions in the cervical spine and medulla oblongata. The hyperintense signal on TIRM sequence (a) and T2* (b) is still visible but became less pronounced with sharper margins.
TECHNIQUE: 1,5T Siemens MAGNETOM Avanto MRI scanner. (a) Sagittal TIRM W, TR 5000, TE 73, TI 150, 3mm slice thickness. (b) Axial T2*W, TR 1160, TE 27, 3mm slice thickness.

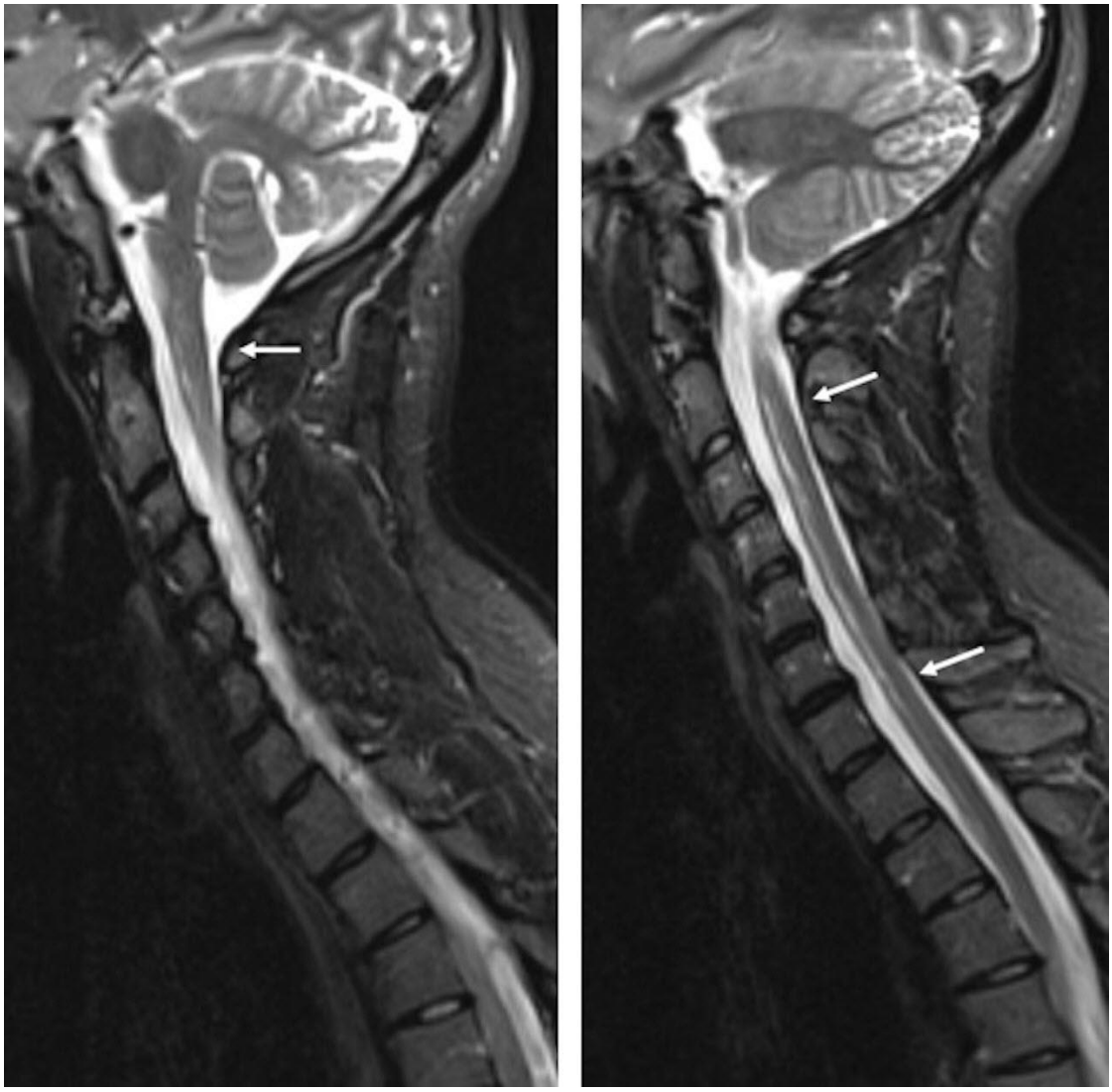

**Figure 5:** 24-year- old female with fibrocartilaginous embolization to the cervical spinal cord.
FINDINGS: MRI of cervical spine one year after injury shows persistent intramedullary hyperintensity on TIRM sequence, with sharp margins and without expansile characteristics - a finding consistent with myelopathy.
TECHNIQUE: 1,5T Siemens MAGNETOM Avanto MRI scanner. (a, b) Sagittal TIRM W, TR 6000, TE 86, 3mm slice thickness.





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|---|---|
| **Etiology** | Retrograde embolization of an intervertebral disc into the spinal artery |
| **Incidence** | Unknown, but rare |
| **Gender ratio** | Unknown |
| **Age predilection** | Two peaks – adolescents and late middle age |
| **Risk factors** | Mostly axial overload of spine |
| **Treatment** | Non-specific |
| **Prognosis** | Depends on level of spinal cord injury |
| **Imaging findings** | MRI - T2-hyperintense signal intramedullary |

**Table 1:** Summary table for fibrocartilaginous embolization.

| | CT/ CTA | MRI |
|---|---|---|
| **Fibrocartilaginous embolization** | No changes | • T1 - No changes /mild enlargement of the spinal cord<br>• T2 - Hyperintense signal<br>• CE T1 - None / faint enhancement<br>• DWI - Hyperintense and hypointense signal in ADC map in acute phase |
| **Cardiovascular diseases** (thromboembolic disease, vascular malformations, vasculitis etc) | No changes/ occlusion of the spinal artery / vascular malformation | • T1 - No changes/ hyperintense signal in subacute thrombus<br>• T2 - Lack of flow voids in occluded large vessels or prominent flow void in vascular malformation<br>• CE T1 - None / vascular malformation enhancement<br>• DWI - None / hyperintensity if acute ischemia is present |
| **Traumatic changes** (herniated intervertebral disc, vertebral fracture with compression of the spinal cord etc) | Disc herniation or fracture line in the vertebra with spinal canal stenosis | • T1 - Disc herniation or fracture line and spinal cord compression<br>• T2 - Intramedullary hyperintensity in compression myelopathy /disc herniation, bone bruise hyperintensity in vertebral body<br>• CE T1 - None / bone bruise enhancement<br>• DWI - None / hyperintense signal if acute ischemia is present |
| **Infections / inflammatory changes** | No changes visible | • T1 - No changes visible<br>• T2 - Intramedullary hyperintensity in myelitis<br>• CE T1 - None or faint meningeal enhancement<br>• DWI - None / hyperintense signal |
| **Carcinomatous meningitis** | Usually no changes or enhancement of the dural carcinomatous plaques | • T1 - Usually no changes<br>• T2 - Intramedullary hyperintensity or dural thickening or small foci on dura<br>• CE T1 - Meningeal enhancement<br>• DWI - None / hyperintense signal |

**Table 2:** Differential diagnoses table for fibrocartilaginous embolization.





## ABBREVIATIONS

CT - Computed Tomography
CTA - Computed Tomography Angiography
DWI - Diffusion Weighted Images
MIP - maximum intensity projection
MRI - Magnetic Resonance Imaging
T2W - T2 Weighted (images)
TE - Echo Time
TIRM - Turbo Inversion Recovery Magnitude
TR - Repetition Time
VRT - Volume Rendering Technique

## KEYWORDS

fibrocartilaginous embolization; material disc embolism; spinal cord ischemia; back pain; minor trauma; spinal cord imaging; MRI

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