

Acute cervical cord infarction in anterior spinal artery territory with acute swelling mimicking myelitis

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ABSTRACT

الاحتشاء الحاد في الحبل الشوكي العنقي غير شائع. قد يشتكي بعض المرضى من أعراض وعلامات تحاكي التهاب النخاع المستعرض الحاد. قد تظهر كلتا الحالتين بشكل متشابه على تصوير الرنين المغناطيسي. ولكن الظهور المفاجئ للأعراض أساسي في التوصل إلى تشخيص احتشاء الحبل الشوكي الحاد في العنق. نبلغ هنا عن حالة احتشاء الحبل الشوكي العنقي الحاد في رجل يبلغ من العمر 40 عاماً والذي كان التشخيص الأولي له التهاب النخاع المستعرض الحاد، وكان يعالج بجرعة عالية من الستيرويدات القشرية تلتها 5 دورات تبادل البلازما. وكشف التصوير بالرنين المغناطيسي للنخاع الرقبي إشارة عالية غير طبيعية تمتد من الفقرة العنقية الثانية حتى السابعة في ثلثي الحبل الشوكي الأمامية والمنطقة الوسطى. وكانت هذه النتائج متسقة مع اعتلال الشريان الفقري الأمامي مسبباً احتشاء الحبل الشوكي العنقي الحاد.

Acute infarction of the cervical segment of the spinal cord is extremely uncommon. Patients may present with signs and symptoms mimicking that of acute myelitis. On imaging, both conditions may present as a hyperintense area on T-2 weighted MRI. History of sudden onset is essential in establishing the diagnosis. We report a case of cervical spinal cord infarction in a 40-year-old man who was diagnosed with acute transverse myelitis, and was treated with high dose intravenous corticosteroids followed by 5 sessions of plasma exchange. An MRI of the spine revealed abnormal high T2 signal intensity extending from the C2 to C7 level involving the anterior two-thirds of the cord with more central involvement. The findings were consistent with anterior spinal artery territory cervical cord infarction.

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Infarction of the spinal cord is rare.^{1,2} Presentation in the cervical region is unusual, especially at a young age. It mostly occurs in the territory of the anterior spinal artery.¹ Infarction of the cervical cord may present with pain, paralysis, dissociated sensory loss, and autonomic deficits.^{1,2} The posterior columns are relatively spared.^{3,4} Acute myelitis, on the other hand, may lead to damage of the functional areas at the affected segment of the cord.^{5,6} Like infarction, patients may present with pain, weakness or paralysis, sensory, and autonomic deficits.^{3,4,6} The disease may be idiopathic or attributed to other CNS, or systemic etiologies.⁷ Magnetic resonance imaging is essential for the diagnosis of both entities, and may reveal an area of hyperintensity on T-2 weighted imaging, suggesting infarction, or inflammation.^{1,8,9} Therefore, both conditions may have similar appearance, and accurate diagnosis can be challenging. We report a case of cervical spinal cord infarction (SCI) in a 40-year-old man who presented with a sudden onset of neck and back pain and paralysis. In addition, a literature review on SCI is presented. Our objective in presenting this particular case is to highlight the rare occurrence of acute medullary swelling in the first few hours following cord infarction simulating acute myelitis.

Case Report. A 40-year-old right-handed man with a known history of hypertension suddenly developed severe neck and back pain while driving. He was fully paralyzed, unable to walk, and developed numbness in all of his limbs. He was taken to the emergency room of a local hospital, where he was noted to have paralysis of all 4 limbs (quadriplegia), pain and temperature sensory loss below the neck, and urinary and bowel incontinence. Cranial nerves examination was unremarkable. There was no history of recent infections or vaccination.

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An MRI at presentation to the local hospital revealed intense swelling and high T2 signal in the cervical segment of the cord (Figure 1). He was transferred to our emergency department 7 days later. He was diagnosed with acute transverse myelitis, and was treated with

high dose intravenous corticosteroids, followed by 5 sessions of plasma exchange with daily physiotherapy. Laboratory investigations including complete blood count, international normalized ratio, rapid plasma regain, cytomegalovirus, Epstein-Barr virus, human immunodeficiency virus, enterovirus, coxsackie, arbovirus, mycoplasma, parvovirus, lyme, lupus, anticardiolipin, and homocysteine were unremarkable. Screening studies for vasculitis were negative. He had a neurogenic bladder, and a Foley catheter was placed. The CSF analysis revealed: glucose 4.34 mg/dL (normal range [NR]: 50-80 mg/dL), protein 291 mg/dL (NR: 15-60 mg/dL), 27 RBCs/mm³ (NR: 0 RBCs/mm³), <1 WBCs/mm³ (NR: 0-5 WBCs/mm³). A brain MRI was normal. After corticosteroids and plasma exchange, MRI of the cervical, thoracic, and lumbar spine revealed cervical spinal cord abnormal high T2 signal intensity extending from C2 to C7 level, and predominantly involving the anterior two-thirds of the cord and tapering distally with more central involvement. The cord swelling had receded, and minimal patchy enhancement could be seen after contrast administration (Figures 2 & 3). Mild degenerative disc disease with no spinal cord compression was seen at multiple levels. The thoracic and lumbar spine was unremarkable. The findings were consistent with anterior spinal artery territory cervical cord infarction, and the diagnosis of infarction in the anterior spinal artery was established. He showed improvement in movement as indicated in Table 1. Reflexes were +1. Babinski response was

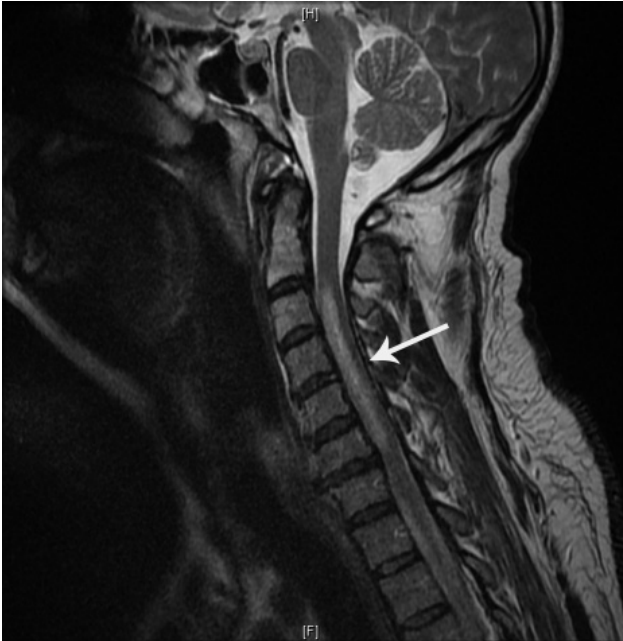


Figure 1 - Sagittal T2 MRI demonstrating intense swelling and high T2 signal in the cervical segment of the cord.



Figure 2 - Sagittal T2 MRI demonstrating: A) cervical spinal cord abnormal high T2 signal intensity extending from C2 to C7 level and predominantly involving the anterior two-thirds of the cord and tapering distally with more central involvement (arrow); and B) sagittal T1 MRI with contrast depicting minimal patchy enhancement.

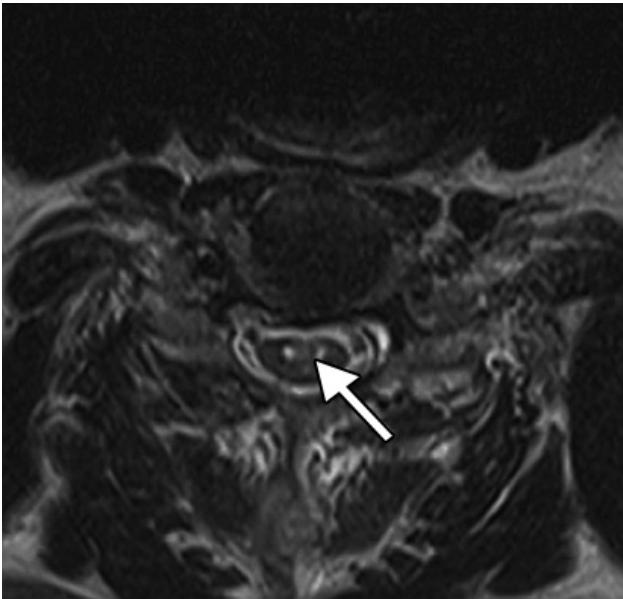


Figure 3 - Axial T2 MRI demonstrating abnormal high T2 signal intensity of the anterior cervical cord horns "owl's eyes" sign (arrow).

Table 1 - Motor examination after treatment of the patient with acute cervical cord infarction.

location	Left	Right
Shoulder	5/5	5/5
Elbow	1/5	1/5
Wrist	3/5	4/5
Hand flexors	3/5	4/5
Hip	5/5	5/5
Knee	4/5	5/5
Ankle	4+/5	5/5

negative. His sensations were as follows: vibration was absent in the upper limbs and reduced in the lower limbs; proprioception was intact in all limbs; pinprick was absent in all limbs; pinprick from the face to T2 level was intact; and pinprick and vibration below T2 level were absent. A follow up MRI 2 weeks later revealed interval improvement of high signal intensity, as well as improvement of enhancement seen in T2-weighted imaging of the spinal cord. He was advised to continue his rehabilitation and physiotherapy at an advanced center.

Discussion. Myelitis is an uncommon inflammatory disorder of the spinal cord.⁹ The inflammatory reaction may damage areas of the spinal cord, leading to either temporary or permanent motor, sensory, or autonomic loss of function of variable extent.⁶ It may affect any segment of the spinal cord.⁵ Patients commonly present

with subacute back pain along with motor, sensory, and autonomic deficits.^{3,5,6} Laboratory tests and CSF analysis may aid in establishing diagnosis and identification of the etiologic agent.⁵ Like infarction, myelitis may present as cord swelling at the level of the lesion on MRI T2-weighted images with hyperintensity.⁹ There is no definitive cure for acute transverse myelitis. Corticosteroids are usually administered to decrease inflammation and swelling, and to improve symptoms.⁵ Plasma exchange has also been suggested in moderate to severe cases. Rehabilitation is of a great importance in such patients.⁵

The cervical segment of the spinal cord receives its radicular vascularization from the V4 segment of the vertebral artery at the C3 level, and from the ascending cervical arteries at the levels of C6-C7. These small arteries are of great importance to the blood supply of the functional areas in the cord, especially through their connections with the anterior spinal artery.^{10,11} The spinal cord anterior horns, ventral spinothalamic tracts, the anterior segment of the lateral spinothalamic tracts, and lateral corticospinal tracts bilaterally obtain their vascular supply from the central branches of the anterior spinal artery.^{1,11}

Unlike the cerebral arteries, the spinal arteries are positioned over a mobile structure, predisposing them to increased risk of injury. The radicular arteries are usually involved in movement injuries. Acute infarcts in the anterior spinal artery territory can be attributed to various causes. These include, but are not limited to, radicular artery occlusion, cervical disc herniation compressing the vasculature, trauma, vasculitis, iatrogenic injury during spinal surgery, spinal cord compression, epidural anesthesia, chronic arachnoiditis, vertebral artery dissection, vertebral artery stenosis due to atherosclerosis, stroke, prothrombin mutation, embolism, global hypotension, and cocaine misuse.¹ Half of the reported SCI cases lack detectable etiology.² Similarly, no identifiable etiology could be found in our case.

Infarction of the spinal cord is extremely uncommon.^{1,2} It occurs mostly in the anterior spinal artery territory. Unlike cerebral infarction, SCI lacks conclusive epidemiological data.¹ Infarction of the cervical segment of the spinal cord is unusual. The number of reported cases in the medical literature remains small. This can be attributed to the abundance of collateral vessels supplying the spinal cord, making it relatively resistant to infarction.^{1,10}

Infarction of the spinal cord presents with a sudden onset. The initiating event can be in the form of mechanical tension or sudden movement. When partial anterior spinal artery territory in the cervical segment is

involved, patients may present with Horner syndrome on the same side of the infarction. The preservation of light touch, proprioception, and deep sensations is attributed to the dorsal column sparing along with the spinocerebellar tracts. The cervical and thoracolumbar segments of the spinal cord are very sensitive to ischemia. This can be attributed to the high density of motor neurons at those levels.¹² Infarction of the anterior horns appears as “owl’s eyes” on MRI scan as seen in our patient (Figure 3).

The MRI remains the modality of choice for diagnosis of spinal infarction.⁸ It is important to rule out other causes, such as, tumors or cord compression. A hyperintense region on T2-weighted imaging demonstrating infarction is the typical finding on MRI for medullary infarction. Its appearance has been described as “pencil-like” in longitudinal sections by different authors. Such findings can only be demonstrated several hours after the onset of symptoms.⁸ In the cervical region, the hyperintensity tends to appear in the medial and central parts of the cord. The T1-weighted images may demonstrate medullary enlargement at the affected segment.¹ Although only few studies described the role of diffusion-weighted imaging (DWI) in detecting cord infarction, it has been shown that DWI may increase the sensitivity in confirming SCI by depicting diffusion restriction in the acute settings.^{1,8}

It has been reported that MRI features of SCI have a predictable course. In the acute stage (<24 hours), the MRI findings are usually normal, with no cord enlargement, gadolinium enhancement, or increased signal intensity on T2-weighted imaging. However, these features emerge and appear clearly on MRI after 1-2 days. On the contrary, nearly all patients with acute myelitis have increased signal intensity on T2-weighted imaging in the acute phase with 62-84% of them demonstrating gadolinium cord enhancement.^{13,14} This distinction in the acute phase may aid in differentiating between the 2 conditions. Therefore, it is essential to obtain repeated imaging in such patients in order to document the temporal profile of changes and facilitate accurate diagnosis.

Diagnosing SCI from the anterior spinal artery territory is based on acute onset of cord symptoms, neurological examination indicating dissociated sensations, MRI changes, and a high index of suspicion. The management of SCI remains controversial. Corticosteroids and/or anticoagulation for secondary prevention can be used. However, studies have failed to document that one of these treatments is superior to the other.¹⁵ The role of rehabilitation in these patients is essential. Patients should be encouraged to participate in rehabilitation programs for improvement of their symptoms.

In conclusion, acute medullary swelling is not typical in the first few hours following cord infarction. This case illustrates an unusual presentation of cervical cord infarction with acute swelling mimicking acute myelitis. Therefore, acute medullary infarction should be included in the differential diagnosis of acute cord swelling. A history of sudden onset along with detailed neurological examination, and radiological images are essential in establishing the diagnosis.

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