

Imaging diagnosis of cervical spinal cord infarction. Case Series

Diagnóstico por imágenes del infarto de la médula espinal cervical. Serie de casos

Diego Armando Guerrero Gómez[1]
Alfonso Javier Lozano Castillo[2]
https://doi.org/10.53903/01212095.187

Key words (MeSH)
Spinal cord ischemia
Magnetic resonance imaging
Diffusion magnetic resonance imaging

Palabras clave (DeCS)
Isquemia de la médula espinal
Imagen por resonancia magnética
Imagen de difusión por resonancia magnética

Summary

Spinal cord infarction (SCI) is one of the possible causes of acute myelopathy, which in the adult population has been associated with aortic disease, embolic event and vascular dissection, among others. The clinical manifestations of SCI will depend on the territory involved and the extent of the lesion with potential motor, sensory and/or autonomic involvement. We present two cases of young patients with extensive SCI secondary to infrequent causes, such as hypercoagulability due to autoimmunity and vertebral artery dissection, in which the diagnostic usefulness of magnetic resonance imaging and especially diffusion sequences (DWI) was proven. The cases included here represent atypical etiologies of an infrequent entity such as SCI.

Resumen

El infarto de la médula espinal (IME) es una de las posibles causas de mielopatía aguda, que en población adulta se ha asociado con enfermedad aórtica, evento embólico y disección vascular, entre otras. Las manifestaciones clínicas del IME dependerán del territorio comprometido y la extensión de la lesión con potencial afectación motora, sensitiva y/o autonómica. Se presentan dos casos de pacientes jóvenes con IME extenso secundario a causas infrecuentes, como hipercoagulabilidad por autoinmunidad y disección de arteria vertebral, en las que se comprobó la utilidad diagnóstica de las imágenes por resonancia magnética y especialmente de las secuencias de difusión (DWI). Los casos aquí incluidos representan etiologías atípicas de una entidad infrecuente como lo es el IME.

Introduction

The differential diagnosis of acute myelopathy syndrome includes a wide range of possibilities, among which the following etiologies stand out: infectious, post-immunization, associated with systemic autoimmune disease, demyelinating disease, paraneoplastic syndrome, vascular and idiopathic disease (1-3).

Spinal cord infarction (SCI), on the other hand, is the main vascular cause of acute myelopathy, considered an infrequent entity with poor prognosis. It represents about 6% of the cases of acute myelopathy and 1-2% of the neurological vascular pathologies (2, 4).

Multiple causes have been attributed to SCI; in the pediatric population it is frequently associated with cardiac malformations or trauma, and in adults, with atheromatosis, aortic pathology, aortic surgery, embolic disease, dissection, systemic hypotension, arteriovenous spinal malformations, coagulopathies, among others (1, 2, 5).

To understand the clinical spectrum and imaging findings of SCI it is important to remember the irrigation of the SCI, which depends on the anterior spinal artery and posterolateral arteries.

The anterior spinal artery originates, in most cases, from the vertebral arteries, supplies the anterior two thirds of the cord and is located in the pia mater along the median fissure with a vertical descent (2, 3, 6).

On the other hand, there are two posterolateral arteries arising from the posteroinferior cerebellar artery on each side, supplying the posterior third of the medulla (posterior column, posterior roots and dorsal bodies) (6).

The irrigation of the lower two thirds of the spinal cord and the conus medullaris also depends on the anastomosis between the anterior spinal artery and the radicular artery magna (Adamkiewicz artery) that arises on the left side of the aorta, between T8 and T11 (2, 6).

The clinical manifestations of SCI depend on the vascular territory involved, including anterior spinal syndrome - paralysis, pain, loss of thermal sensitivity and urinary or bladder incontinence - and posterior spinal syndrome - vibrational and proprioceptive hypoesthesia (Table 1) (1, 2, 4, 5).

The gold standard for the imaging diagnosis of SCI is MRI, in which high spinal cord signal in T2-weighted sequences with a central pattern described as "owl eyes", with involvement in most cases of the distal half

[1]Surgeon, specialist in Epidemiology. Resident of Radiology and Diagnostic Imaging, National University of Colombia, National University Hospital of Colombia. Bogotá, Colombia.

[2]Full Professor, Department of Diagnostic Imaging, Universidad Nacional de Colombia. Professor of Neuroimaging. Director of the Department of Diagnostic Imaging, Universidad Nacional de Colombia. Bogotá, Colombia.

of the thoracic spinal cord and with involvement of a vertebral body irrigated by the same artery, has classically been considered as the main suggestive criterion of SCI (2).

Recently, diffusion sequences (DWI) have gained field of action when ischemic etiology is suspected in patients with acute myelopathy.

Table 1. Clinical syndromes in spinal ischemic lesions

Injury	Manifestation
Anterior spinal artery complete	Most common. Loss of bilateral motor function, thermal sensation and pain. May present with autonomic dysfunction (hypotension, sexual, bowel or bladder dysfunction; respiratory dysfunction with cervical involvement).
Incomplete anterior spinal artery	Ischemia in the anterior horns. Acute paraplegia (peudopolyomyelic) without sensory alterations or sphincter involvement.
Posterior spinal artery	Proprioceptive and vibratory hypoesthesia inferior to the lesion site and total anesthesia at the level of the lesion.
Sulco-commissural syndrome	Partial Brown-Sequard. Respect of postural sensitivity. Hemiparesis with contralateral sensory spinothalamic deficit.
Infarction in conus medullaris	Similar to cauda equina syndrome.
Central spinal infarction	Post cardiac arrest or prolonged hypotension. Bilateral sensory spinothalamic deficit with respect to the posterior columns.
Transverse spinal cord infarction	Plegia or sudden paresis. It is usually of embolic origin.
Spinal transient ischemic attack	Transient motor and/or sensory compromise of minutes-hours duration.

Source: Own elaboration.

Case series

Case 1

This is a 19-year-old female patient with a history of systemic lupus erythematosus, lupus nephritis and antiphospholipid syndrome, who was admitted for progressive weakness and flaccid quadriparesis of hours of evolution, with initial diagnostic impression of Guillain-Barré syndrome.

As a diagnostic complement, MRI of the cervical and thoracic spine identified marked edema of the medulla with increased volume and high signal in sequences with T2 information with extension from the bulbar-medullary junction to T6. Due to diagnostic suspicion of SCI, diffusion gradient imaging (DWI) sequences and ADC map were performed, in which marked diffusion restriction was found, compatible with acute SCI (Figure 1).

During the intensive care unit stay, the patient suffered hypoxemic respiratory failure and hemodynamic compromise, secondary to brain stem involvement, which led to death.

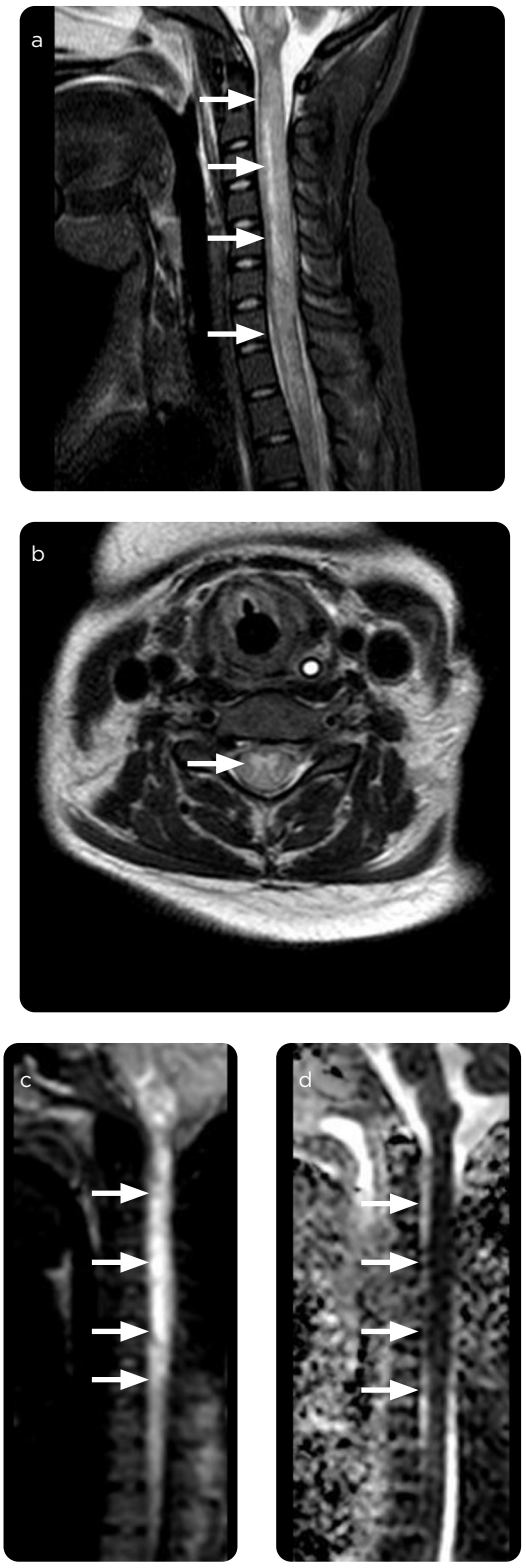


Figure 1. MRI of the patient in case 1. a) Sequences with sagittal and b) axial T2 information; c) sagittal DWI b-400 and d) ADC map. Significant marrow edema with volume increase, with predominance in the anterior aspect, high signal in sequence with T2 information with extension from the medulla oblongata to the upper thoracic segment (arrows in a), with "owl eyes" aspect in axial image (arrow in b), which corresponds with high signal in DWI (arrows in c) and low signal in ADC map (arrows in d), findings that configure diffusion restriction, with greater high cervical involvement, related to acute MSI.



Figure 2. Digital subtraction angiography (DSA) of the patient in case 2. DSA in AP projection: absence of opacification with contrast medium of the left vertebral artery (short arrow), compatible with dissection, associated with poor filling of the upper cervical portion of the anterior spinal artery by probable dependence of the same in its origin from the left vertebral artery, as an anatomical variant. Adequate opacification of the right vertebral artery (long arrow).

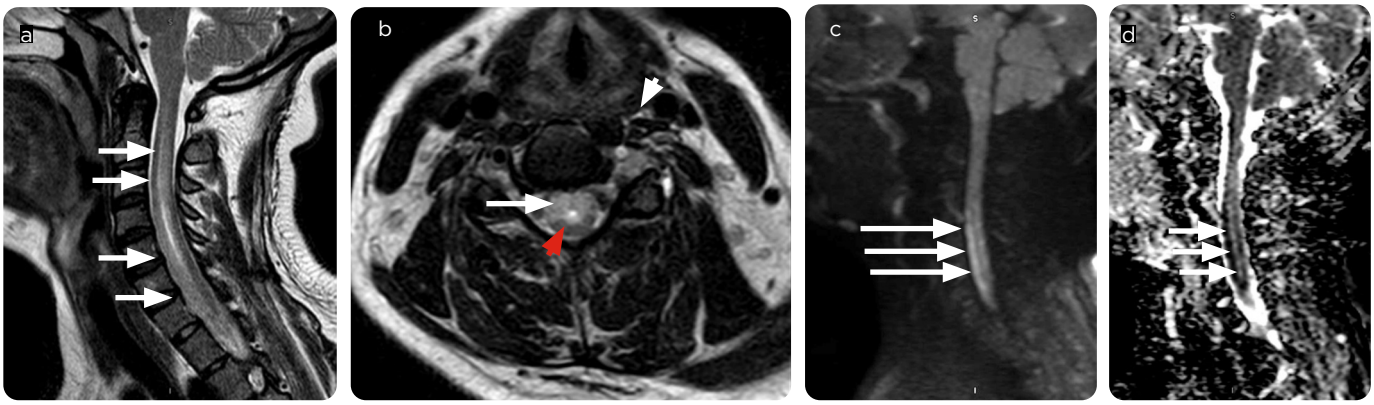


Figure 3. MRI cervical spine of the patient of case 2. a) Sagittal and b) axial T2 sequences; c) sagittal DWI b-600 sequence and d) ADC map: Spinal cord edema with increased volume associated with high signal in sequences with T2 information (long arrows in a and b) corresponding with high signal in DWI sequence (long arrows in c) and low signal in ADC map (long arrows in d) which configure diffusion restriction, predominantly in the anterior aspect of the spinal cord, with extension from C4 to C7. Additionally, mild dilatation of the central ependymal canal (white arrowhead in b) and loss of signal void by flow in left vertebral artery due to known dissection (red arrowhead in b). Findings related to acute SCI.

Case 2

29-year-old female patient with overweight as the only relevant medical history, who consults for flaccid quadriparesis of sudden onset and cervicalgia of hours of evolution.

Brain MRI identified findings suggestive of left vertebral dissection, so a cerebral panangiography was performed which confirmed this finding; it also showed poor filling of the upper cervical segment of the anterior spinal artery due to its probable dependence on the left vertebral artery at its origin, as an anatomical variant (Figure 2).

As a diagnostic complement, MRI of the cervical spine was performed, which showed marked edema with high signal in sequences with T2 information of predominance in the anterior aspect of the spinal cord, as well as high signal in DWI sequence and low signal in the ADC map, which confirmed the diffusion restriction, compatible with acute SCI in the anterior spinal artery (figure 3).

Discussion and conclusions

Acute myelopathy due to SCI is a medical emergency with great implications in terms of morbidity and mortality (1, 2, 4).

Among the potential causes of SCI are, in addition to aortic pathology, hypercoagulable state (typical of entities such as antiphospholipid syndrome) and vascular dissections, entities that should be considered in the presence of non-traumatic myelopathy in young patients (1, 2, 5).

MR images are the gold standard in the approach to DWI, thanks to the possibility of detecting spinal cord edema with high signal in T2-weighted sequences depending on the vascular territory and extension involved (3, 7), as illustrated in the cases presented, in which the "owl eyes" sign is also observed.

MR images in DWI and apparent diffusion coefficient (ADC) map are especially useful in the suspicion of ischemic etiology, since they allow confirmation of cytotoxic edema and, therefore, of SCI (8-11).

The literature has described the low overall diagnostic yield of SCI with conventional MR images (45%), which increases to 100% when

DWI sequences and ADC mapping are added, which is why their inclusion in MRI protocols for acute myelopathy should be considered (10).

It is therefore essential to perform multi-sequence analysis, starting from the series of images with anatomical information (enhanced in T1 and T2) and complementing the findings with the determination of diffusion restriction in DWI images and ADC map (11).

In the cases presented, the MRI protocol included sagittal cervical spine diffusion sequences: DWI b-400 and b-600, as well as sagittal ADC map with good resolution and diagnostic quality. The usefulness of this sequence is outstanding.

It is important to highlight that, as in ischemic stroke, the imaging findings of MSI depend on its evolutionary stage, and may go unnoticed in the hyperacute phase, which is why dynamic analysis with evolutionary control MRI may help to increase the diagnostic yield compared to single MRI in the acute stage (8).

The MR evolution of SCI has in diffusion restriction the first radiological sign, which can potentially be identified from the onset of symptoms and up to 7-10 days (hyperacute and acute phases), with subsequent pseudo-normalization of the ADC, similar to that seen in cerebral infarction. In turn, in chronological order, the second radiological sign to appear is the increase in signal intensity in T2/FLAIR-weighted sequences, which can be observed from 12-24 hours after the onset of symptoms, followed by the appearance of peripheral enhancement after gadolinium administration, which will be observed in the subacute stage (after 1 week of evolution of the SCI). Finally, in the late stages, sequelae findings such as myelomalacia and atrophy of the spinal cord are identified (8-11).

In this regard, the authors of this article schematically propose the following MRI characteristics according to the evolutionary stage of SCI (Table 2).

Table 2. MRI findings according to the evolutionary stage of the SCI

Evolutionary stage of SCI	Time (after symptom onset)	MRI findings
Hyperacute	Min-24 h	• High signal in DWI and low signal in ADC: diffusion restriction.
Acute	24 h-1 week	• Diffusion restriction. • High T2/FLAIR signal.
Subacute	1-4 weeks	• Pseudonormalization of ADC. • High signal in T2/FLAIR sequences. • Peripheral enhancement after gadolinium.
Chronic: old SCI	More than 4 weeks	• Myelomalacia. • Spinal cord atrophy.

Source: Own elaboration.

References

1. Frohman E, Wingerchuk D. Transverse myelitis. New Eng J Med. 2010;363(6):564-72.
2. Vargas M, Gariani J, Sztajzel R, Barnaure-Nachbar I, Delattre B, Lovblad K, et al. Spinal cord ischemia: Practical imaging tips, pearls, and pitfalls. Am J Neuroradiol. 2015;36(5):825-30.
3. Masson C. Spinal cord infarction: clinical and magnetic resonance imaging findings and short term outcome. J Neurol, Neurosurg Psychiat. 2004;75(10):1431-5.
4. Nedeltchev K, Loher T, Stepper F, Arnold M, Schroth G, Mattle H, et al. Long-term outcome of acute spinal cord ischemia syndrome. Stroke. 2004;35(2):560-5.
5. Novy J, Carruzzo A, Maeder P, Bogousslavsky J. Spinal cord ischemia. Arch Neurol. 2006;63(8):1113.
6. Ross J, Moore K. Diagnostic imaging: Spine. Philadelphia, PA: Elsevier; 2015.
7. Weidauer S, Nichtweiss M, Lanfermann H, Zanella F. Spinal cord infarction: MR imaging and clinical features in 16 cases. Neuroradiology. 2002;44(10):851-7.
8. Alblas C, Bouvy W, Lycklama à Nijeholt G, Boiten J. Acute spinal-cord ischemia: Evolution of MRI findings. J Clin Neurol. 2012;8(3):218.
9. Thurnher M, Bammer R. Diffusion-weighted magnetic resonance imaging of the spine and spinal cord. Sem Roentgenol. 2006;41(4):294-311.
10. Thurnher M, Bammer R. Diffusion-weighted MR imaging (DWI) in spinal cord ischemia. Neuroradiol. 2006;48(11):795-801.
11. Fujikawa A, Tsuchiya K, Takeuchi S, Hachiya J. Diffusion-weighted MR imaging in acute spinal cord ischemia. Eur Radiol. 2004;14(11):2076-8.

Correspondence

Diego Armando Guerrero Gómez
Carrera 30 # 45
Bogotá, Colombia
daguerrerog@unal.edu.co

Received for evaluation: April 10, 2022
Accepted for publication: July 10, 2022