



Medular Infarction in Patient with Descending Aorta Aneurysm with Intramural Hematoma

Infarto medular en paciente con aneurisma de aorta descendente con hematoma intramural

Juan Guillermo Rifaldo¹
Catalina Cuervo²
Diego Abreo³
Andrés Lozano¹
Juan Pablo Muñoz¹



Key words (MeSH)

Aneurysm, dissecting
Ischemia
Infarction
Myelitis

Palabras clave (DeCS)

Aneurisma disecante
Isquemia
Infarto
Mielitis

Summary

Spinal cord ischemia is a medical emergency due to hypoperfusion or embolization of the spinal artery. The onset of symptoms is abrupt and the clinical presentation depends on the medullary territory involved. The most frequent causes are atherosclerotic disease, aortic surgery and less commonly, aortic dissection, which is associated with serious complications and greatest risk of mortality. This article describes the case of a patient with an acute spinal infarction as the first manifestation of an intramural hematoma in a thoracic aortic aneurysm.

Resumen

La isquemia medular (IM) es una emergencia médica causada por hipoperfusión o embolismo de la arteria espinal. El inicio de los síntomas es abrupto, y su presentación clínica y gravedad es amplia, dependiendo del segmento medular comprometido. Entre las causas más frecuentes están la enfermedad aterosclerótica, las cirugías vasculares aórticas y, menos comúnmente, la disección aórtica (DA). La IM conlleva secuelas graves y alta mortalidad. Este artículo describe el caso de un paciente con infarto medular agudo como primera manifestación de un hematoma intramural (HIM) en un aneurisma de aorta torácica.

Introduction

Medullar infarction (MI) is a rare pathology with a devastating course and a wide clinical spectrum ranging from incomplete spinal syndrome to complete myelopathy. Its main causes are atherosclerotic disease and complications associated with aortic surgery. It can be associated with other aortic pathologies, such as aortic aneurysms, dissection, traumatic rupture, thrombosis or aortic coarctation.

Within the spectrum of acute aortic syndrome (AAS), aortic dissection (AD) represents the most common clinical presentation and the one most frequently associated with medullary ischemic complications. However, in clinical practice it is not common to find cases of acute spinal cord ischemia as the initial form of presentation of AAS.

Case description

A 71-year-old African-American man with a history of hypertensive and ischemic heart disease, percutaneously revascularized, diabetic, with chronic renal disease and extabachism. He consulted the emergency department for intermittent lumbosacral pain not trigge-

red by trauma, for a month, and 4 hours of increasing pain and irradiation to the dorsal region, accompanied by sudden loss of strength and hypoesthesia in the lower limbs.

On physical examination she was found with paralysis in lower limbs (0/5), patellar and achilles hyporeflexia, anesthesia to pain with sensory level T7, without bulbocarvenous reflex and with loss of tone in the anal sphincter.

Due to suspicion of spinal cord compression, magnetic resonance imaging (MRI) of the dorsolumbar spine was performed, with the finding of MI in the territory of the anterior spinal artery extending from T5 to T9 (Figures 1, 2 and 3); a descending aortic aneurysm was identified (Figures 4 and 5). An aortic aneurysm with intramural hematoma (IMH) was confirmed by angiography. The patient survived, but paraplegia persisted as a sequela.

Discussion

MI is a rare disorder, with an incidence of 2% of all neurological vascular disorders and 5-8% of acute non-traumatic myelopathies. Symptoms depend on the

¹General Physician, Pablo Tobón Uribe Hospital. Medellín, Colombia.

²Radiology Specialist, Hospital Pablo Tobón Uribe. Medellín, Colombia.

³Specialist in Emergency Medicine, Hospital Pablo Tobón Uribe. Medellín, Colombia.

involved spinal vascular area. Onset is sudden and occasionally develops within hours. Pain in the neck or dorsolumbar region frequently accompanies spinal ischemic symptoms and occurs in up to 70% of patients (1, 2).

The presentation varies from mild weakness to quadriplegia, associated with loss of bladder and bowel function, spinal pain and sensory involvement with “sensory level”.

There are 31 spinal cord segments, each with a pair of ventral and dorsal roots that allow sensory and motor function, respectively; these roots join on each side, to form the spinal nerve that exits through the foramen of junction. The spinal cord is divided into cervical, thoracic, lumbar and sacral segments; however, the cord only extends to the height of the first lumbar vertebra and from this point the roots of the lumbar, sacral and coccygeal nerves form the cauda equina (3).

Vascular disorders affecting the spinal cord are a medical emergency. There are two types of MI: medullary infarction in radicular arterial territory -unilateral or bilateral infarcts in territories irrigated by the anterior or posterior spinal artery- and central and transverse infarction -due to extensive medullary hypoperfusion- (4, 5). They present with different clinical manifestations, of which anterior spinal artery syndrome is the most common, as in this case. In the patient under study, the sagittal sequence with T2 information of the MRI shows it: high signal in the ventral region of the spinal cord from T5 to T9, suggesting involvement of the vascular territory corresponding to the anterior spinal artery. The usual presentation is bilateral loss of motor function with relative alteration of proprioception and vibratory sensation below the level of the lesion; it is accompanied in the acute phase by flaccidity and hyporeflexia, with onset of spasticity and hyperreflexia after days or weeks. It is associated with compromised respiration, if the lesion is located in the rostral region of the cervical spinal cord.

Other presentations correspond to incomplete spinal artery syndrome, in which the ischemia is located in the lateral horns, and causes acute paraplegia, without sphincter or sensory dysfunction, painful bilateral brachial dysplegia in case of cervical involvement, and progressive distal amyotrophy due to chronic lesions of the anterior horns, which can be confused with amyotrophic lateral sclerosis.

Posterior spinal artery syndrome manifests with loss of proprioception inferior to the level of the lesion and with total anesthesia occurring at the same level of the lesion; it may be accompanied by usually mild and transient weakness. Unilateral involvement is the most common. Less typical presentations include sulcocommissural syndrome, which manifests as a partial Brown-Sequard syndrome, hemiparesis with contralateral spinothalamic sensory deficit-; conus medullaris infarction-, which may be confused with cauda equina syndrome; central spinal infarction, which occurs after a state of shock following cardiac arrest or prolonged hypotension due to another cause, whose main finding is bilateral spinothalamic sensory deficit, and transverse spinal cord infarction with complete motor and sensory deficit, depending on the level of the lesion, which simulates a myelopathy of another origin -mainly embolic etiology- (6).

These clinical syndromes associated with MI are explained by the vascularization of the spinal cord, which is mainly given by the anterior spinal artery, the posterolateral spinal artery and the spinal radiculomedullary artery, also known as magna or Adamkiewicz artery. The anterior spinal artery supplies the anterior two thirds of

the spinal cord; it is located in the pia mater, it is formed from two small branches originating from the fourth segment of the vertebral arteries with anastomoses in the foramen magnum. Adamkiewicz's artery has a particular hairpin twist, but it must be differentiated from the radiculomedullary vein, which has the same shape, but is longer and more tortuous. It originates from the left side of the aorta between segments T8 and L1 to anastomose with the anterior spinal artery. It perfuses the lower two thirds of the spinal cord and the conus medullaris. Finally, the two posterolateral spinal arteries originate from the posteroinferior cerebellar arteries and supply the posterior third of the spinal cord (posterior columns, posterior roots and dorsal horn) (7).

The etiologies of MI are multiple. Spinal cord infarcts are often secondary to acute interruptions in the blood flow of a main or radicular artery, usually secondary to an embolus or atherosclerotic plaque; however, in adults, systemic hypotension, cardiac and aortic surgeries, minimally invasive procedures, compression secondary to vertebral disc disease and cervical degenerative disc disease are also relevant causes of MI.

On the other hand, in the pediatric population, cartilaginous embolism secondary to minor trauma, cardiac surgeries, scoliosis correction, sickle cell anemia and umbilical catheterization in neonates are described as common causes (8). To a lesser extent, spinal cord infarction is explained by prolonged hypoperfusion, vasculitis and local or systemic infections (9). Regarding MI secondary to AAS, it occurs in 1-2% of AD and ruptured abdominal aortic aneurysms (10, 11).

In the case described here, the clinical presentation and etiology are rare in the medical literature, since the MI was secondary to an aortic intramural hematoma (hemorrhage of the vasa vasorum in the middle layer of the aortic wall, with the formation of a hematoma, without evidence of primary intimal tear). This is considered a precursor of AD in approximately two out of three cases (12-14), in which ischemic spinal cord involvement is estimated at 3% and only 1% debuts with ischemic spinal cord injury (15-17).

The imaging diagnosis of MI is based on the finding in MRI of an area with high signal in sequences with T2 and STIR information with confirmed diffusion restriction of the ischemic area; the application of contrast medium can help in the acute phase, since it allows ruling out another etiology (18). The vascular etiology of MI can be evaluated with CT angiography or MRI angiography.

The aortic intramural hematoma is observed as a lesion -with high density in CT and high signal in MR sequences with simple T1 information; it is of circular or crescent morphology located in the aortic wall with a thickening greater than 5 mm. It has no blood flow or filling with contrast medium, unlike aortic dissection, which has a false lumen and a true lumen, both with filling with contrast medium.

In this case medullary compression was initially suspected. The unexpected finding was an aneurysm of the descending aorta with intramural hematoma on spinal MR images. For this reason, aortic MRI angiography was performed. The spinal cord infarction was not markedly represented in the diffusion images, but the finding in MRI, high signal in sequences with T2 information in the ventral region of the spinal cord suggested spinal cord ischemia, a diagnosis supported by the clinical presentation of the patient. Subsequent images are not available due to the loss of the patient's follow-up.

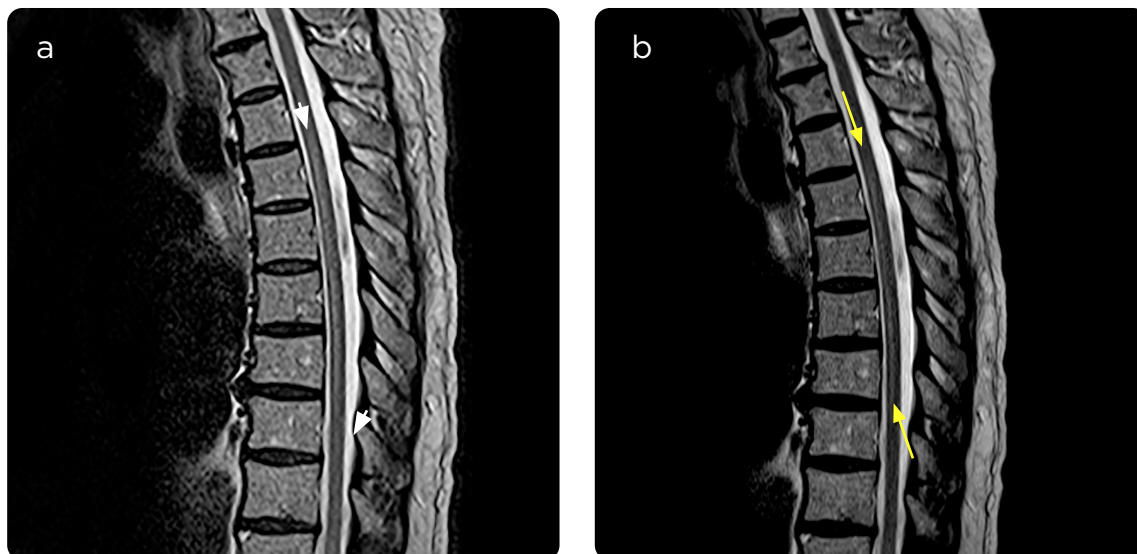


Figure 1. Dorsolumbar spine MRI, sagittal sequence with T2 information. It shows high signal in the spinal cord towards its ventral aspect extending from T5 to T9 (indicated between white arrowheads in a and between yellow arrows in b). The anterior location suggests ruling out infarction in the anterior spinal artery.

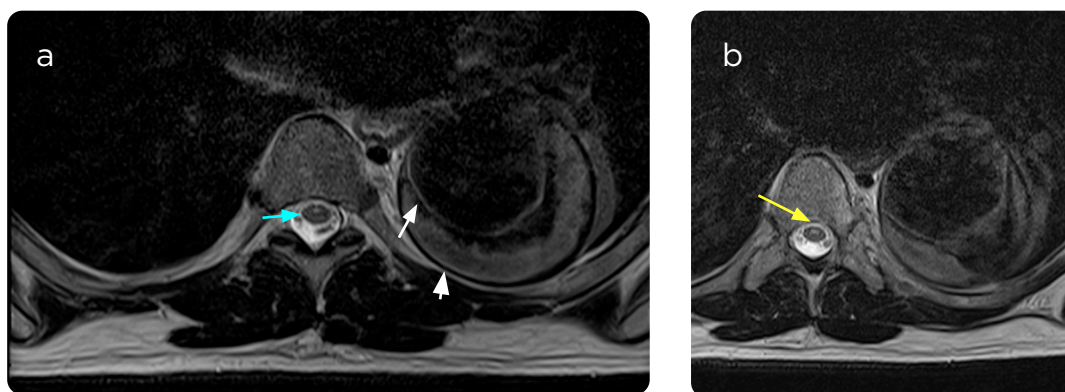


Figure 2. MR of dorsolumbar spine, axial sequence with T2 information. High ventral signal of the spinal cord (blue arrow in a, yellow arrow in b) due to spinal cord infarction; in addition, aneurysmal dilatation of the descending aorta (white arrow) with eccentric mural thrombus of high signal in T2 (white arrow head). No periaortic hematomas.



Figure 3. MR of dorsolumbar spine, sagittal T2 STIR sequence. It confirms alteration of the signal intensity of the anterior portion of the spinal cord, in sequences sensitive to fluid, from T5 to T9 (arrows).

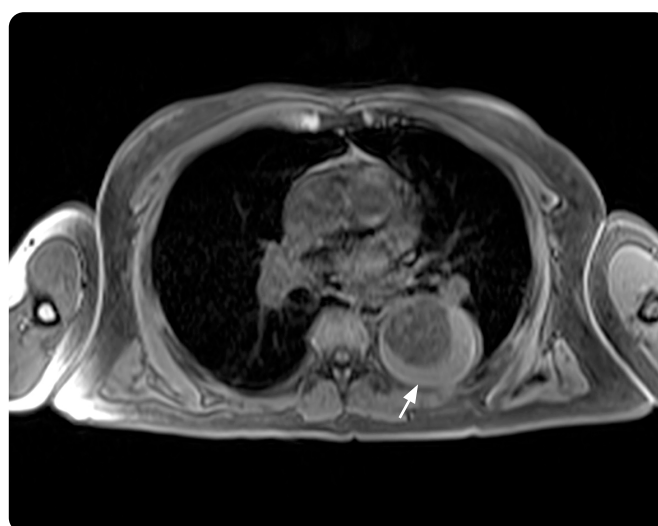


Figure 4. AngioMR of thoracoabdominal aorta, axial T1 sequence with fat saturation (VIBE). Confirms aneurysm of descending thoracic aorta, beginning 3 cm from the left subclavian artery, measuring 6x6x13 cm (APxTxL), without extension to the abdominal aorta, with acute intramural hematoma, high signal in T1 and T2 sequences (white arrow). Stanford type B classification.

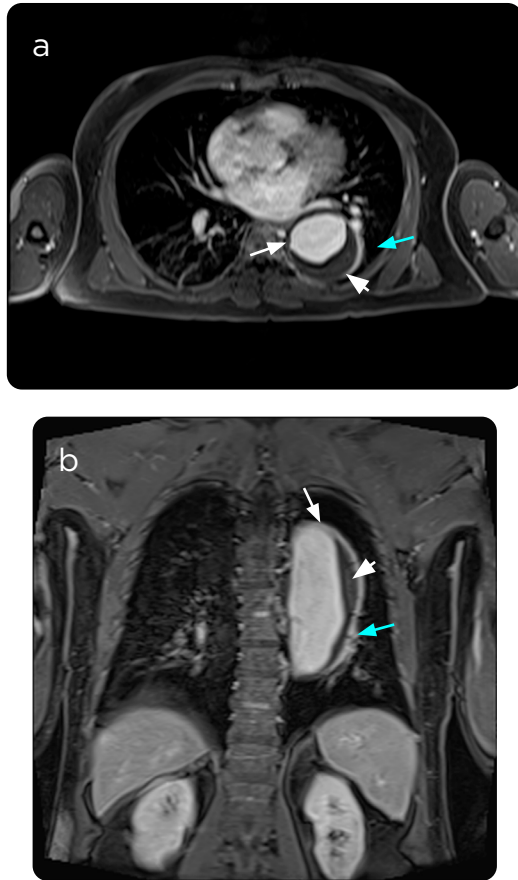


Figure 5. a) AngioMR of thoracoabdominal aorta, axial T1 VIBE sequence with contrast medium. Aortic aneurysm is observed (white arrow), with acute intramural hematoma, without enhancement or filling with contrast medium of the false lumen (white arrow head), without dissection flaps. In addition, left basal laminar pulmonary atelectasis in intimate contact with the aneurysm wall (blue arrow). b) Coronal T1 VIBE sequence with contrast medium. Aortic aneurysm is observed (white arrow), with acute intramural hematoma, without enhancement or filling of the false lumen (white arrowhead), without dissection flaps. Laminar pulmonary atelectasis in intimate contact with the aneurysm wall (blue arrow).

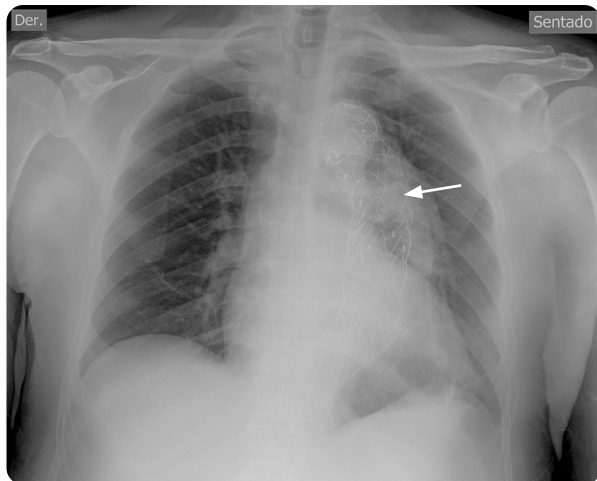


Figure 6. Chest X-ray. Mediastinal widening due to known thoracic aortic aneurysm. Endovascular management with endoprosthesis (white arrow) was performed, with adequate result, exclusion of the aneurysm, without immediate complications.

Conclusión

Medullary ischemia is an infrequent condition. Due to anatomical predisposition, the thoracic medullary segment has a higher risk of ischemia. Acute aortic syndrome and intramural hematoma, as in this case, should be considered in the etiology of acute ischemic spinal cord injury.

References

1. Bar C, Cheuret E, Bessou P, Pedespan JM. Childhood idiopathic spinal cord infarction: Description of 7 cases and review of the literature. *Brain Develop.* 2017;39:818-27.
2. Rigney L, Cappelen-Smith C, Sebire D, Beran RG, Cordato D. Nontraumatic spinal cord ischaemic syndrome. *J Clin Neurosci.* 2015; 22:1544-9.
3. Witham CL, Fisher KM, Edgley SA, Baker SN. Corticospinal inputs to primate motoneurons innervating the forelimb from two divisions of primary motor cortex and area 3a. *J Neurosci.* 2016;36:2605.
4. Nardone R, Pijika S, Mutzenbach JS, Seidl M, Leis S, Trinka E, et al. Current and emerging treatment options for spinal cord ischemia. *Drug Discov Today.* 2016;21(10):1632-41.
5. Ando T, Abe H, Nagata T, Makuuchi H. Successful staged operation for acute type A aortic dissection with paraplegia. *Asian Cardiovasc Thorac Ann.* 2013;21(3):335-7.
6. Novy J, Carruzzo A, Maeder P, et al. Spinal cord ischemia: clinical and imaging patterns, pathogenesis, and outcomes in 27 patients. *Arch Neurol.* 2006;63:1113-20.
7. Vargas MI, Gariani J, Sztajzel R, Barnaure-Nachbar I, Delattre BM, Lovblad KO, et al. Spinal cord ischemia: practical imaging tips, pearls, and pitfalls. *AJNR.* 2015;36(5):825-30.
8. Hernández Pinzón J, Mora C, Aguilar M. Signo de los ojos de lechuga. *Rev Argentina Radiol.* 2017;81(2):154-6.
9. Kumral E, Polat F, Gulluoglu H, Uzunkopru C, Tuncel R, Alpaydun S. Spinal ischaemic stroke: clinical and radiological findings and short-term outcome. *Eur J Neurol.* 2011;18:232-9.
10. Weidauer S, Nichtweiß M, Hattingen E, Berkefeld J. Spinal cord ischemia: aetiology, clinical syndromes and imaging features. *Neuroradiology.* 2015;57:241-57.
11. Peppelenbosch AG, Vermeulen Windsant IC, Jacobs MJ, Tordoir JHM, Schurink GWH. Open repair for ruptured abdominal aortic aneurysm and the risk of spinal cord ischemia: Review of the literatura and risk-factor analysis. *Eur J Vasc Endovasc Surg.* 2010;40:589-95.
12. Lee SJ, Kim JH, Na CY, Oh SS, Kim YH, Lee CK, et al. Eleven years of experience with the neurologic complications in Korean patients with acute aortic dissection: a retrospective study. *BMC Neurol.* 2013;13:46.
13. Strayer RJ. Thoracic aortic syndromes. *Emerg Med Clin N Am.* 2017;35:713-25.
14. Lech C, Swaminathan A. Abdominal aortic emergencies. *Emerg Med Clin N Am.* 2017;35:847-67.
15. Upadhye S, Schiff K. Acute aortic dissection in the Emergency Department: Diagnostic challenges and evidence-based management. *Emerg Med Clin N Am.* 2012;30:307-27.
16. Erbel R, Aboyans V, Boileau C, Bossone E, Di Bartolomeo R, Eggerbrecht H, et al. Guía ESC 2014 sobre diagnóstico y tratamiento de la patología de la aorta. *Rev Esp Cardiol.* 2015;68(3):242.e1-e69.
17. Robertson CE, Brown RD Jr, Wijdicks EF, Rabinstein AA. Recovery after spinal cord infarcts: long-term outcome in 115 patients. *Neurology.* 2012;78:114-21.
18. Heldner MR, Arnold M, Nedeltchev K, Gralla J, Beck J, Fischer U. Vascular diseases of the spinal cord: a review. *Curr Treat Options Neurol.* 2012;14:509-20.

Correspondence

Diego Abreo
Hospital Pablo Tobón Uribe
Medellín, Colombia
abreo87@gmail.com

Received for evaluation: October 14, 2020

Accepted for publication: December 6, 2020