Acta Biomed 2021; Vol. 92, N. Supplement 1: e2021080 DOI: 10.23750/abm.v92iS1.8395

Case report

Spinal cord infarction. A case report and narrative review

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Summary. Spinal cord infarction is a rare but severe disorder, consistently less frequent than ischemic brain injury. It constitutes only 1-2% of all neurological vascular emergencies. Here we describe a case of spinal cord infarction in a 68-year-old Caucasian man without any neurological problem in his clinical history. The patient presented to the Emergency Department complaining for sudden onset of lower limbs weakness, as well as pain and mild loss of sensitivity in both legs. These symptoms suddenly arose after a 10 minutes bicycle race. He underwent a CT angiography, which confirmed the presence of a fusiform aneurysm of infra-renal abdominal aorta with thrombotic apposition on right lateral side and some ulcerated thrombi. As such, the patient underwent a spinal cord Magnetic Resonance Imaging, that showed images compatible with an acute ischemic injury (infarction) from D11 to medullary cone. He was then treated with low molecular weight heparin, aspirin, and methylprednisolone, and was then admitted to the Stroke Unit. He gradually regained lower limbs sensitivity totally, but the strength was only partially restored. Although a rare entity, Emergency Physicians should always keep in mind this possibility when evaluating patients with sudden loss of sensitivity and/or strength in their lower limbs. (www.actabiomedica.it)

Key words: spinal cord infarction, spinal cord ischemia, paraparesis, magnetic resonance imaging, MRI, emergency department

Introduction

Spinal cord infarction is a rare but severe disorder, caused by a kaleidoscope of pathologic states, consistently less frequent than ischemic brain injury. It constitutes only 1-2% of all neurological vascular emergencies (1), and shares some clinical features with other acute myelopathic syndromes. The onset of spinal cord infarction is typically abrupt, and only in a few cases symptoms progress over minutes or a few hours. The neurologic presentation depends on the vascular territory involved and its severity can vary from paraplegia to minor weakness. The involved cord level can vary widely, partially depending on the underlying etiology (2). Back or neck pain often (i.e., as many as 70% of cases) accompanies spinal cord infarction, typically occurring at the level of the lesion (3, 4).

Here we describe a case of spinal cord infarction arisen after a 10 minutes bicycle race in a patient without any neurological problem in his clinical history.

Case report

A 68-year-old Caucasian man presented to the Emergency Department of University Hospital of Parma on February 3rd, 2019, complaining for sudden onset of lower limbs weakness. This symptom suddenly arose after a 10 minutes bicycle race. The patient also complained for pain and mild loss of sensitivity

in both legs. He was a non-smoker, and his medical history was characterized by arterial hypertension and ischemic heart disease. He was previously treated with aspirin, nebivolol, atorvastatin, valsartan/hydrochlorothyazide. No allergies were reported. On arrival, his vital signs were as follows: blood pressure 120/70 mmHg, heart rate 75 beats/min, respiratory rate 14 breaths per minute, body temperature 36.8°C. The physical examination revealed severe lower limbs paresis (right>left), normal peripheral pulses, mild loss of sensibility. The medullary sensitive level seemed to be at L2-L3, without sphincter disorders. The electrocardiogram (ECG) showed sinus rhythm, with no significant alterations of the QRS complexes or ST-T waves. Standard blood tests were in the normal range. Given the clinical suspect of spinal cord disorder, the Emergency Physician (EP) ordered a thoracic and abdominal CT angiography, which confirmed the presence of a fusiform aneurysm of infra-renal abdominal aorta (diameter 37 x 37 mm) with thrombotic apposition on right lateral side and some ulcerated thrombi (Figure 1). Based on these results, after a few hours the patient underwent a spinal cord Magnetic Resonance Imaging (MRI), that displayed images compatible with an acute ischemic injury (infarction), extended from D11 level to the medullary cone (Figure 2). A vascular surgeon was consulted, but he did not consider an urgent surgical procedure, due to the fear that the clamping of the aorta could worsen the spinal cord ischemia. He suggested that the placement of an aortic prosthesis could be planned later. The patient was then treated with low molecular weight heparin (LMWH) in association with aspirin and methylprednisolone (the latter with the aim to reduce the spinal cord edema). The patient was admitted to the Stroke Unit. During the Stroke Unit staying, on day 2 the patient displayed a bladder dysfunction needing a catheter placement. He gradually regained lower limbs sensitivity totally, but the strength was only partially restored. Following the

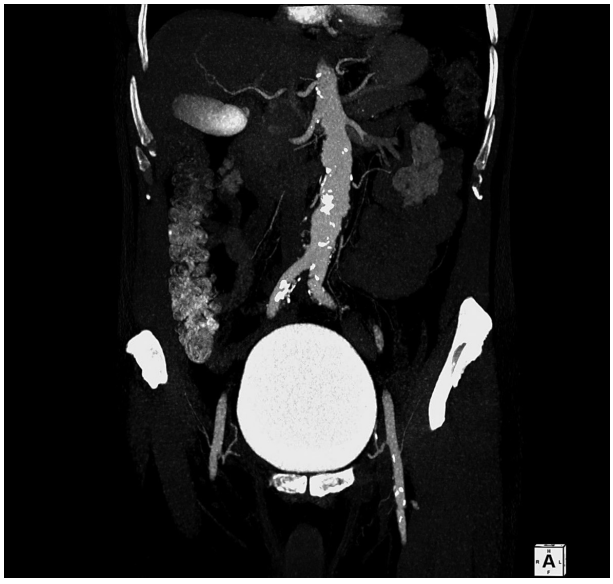

Figure 1. CT angiography displaying infra-renal aortic aneurysm, with thrombotic apposition

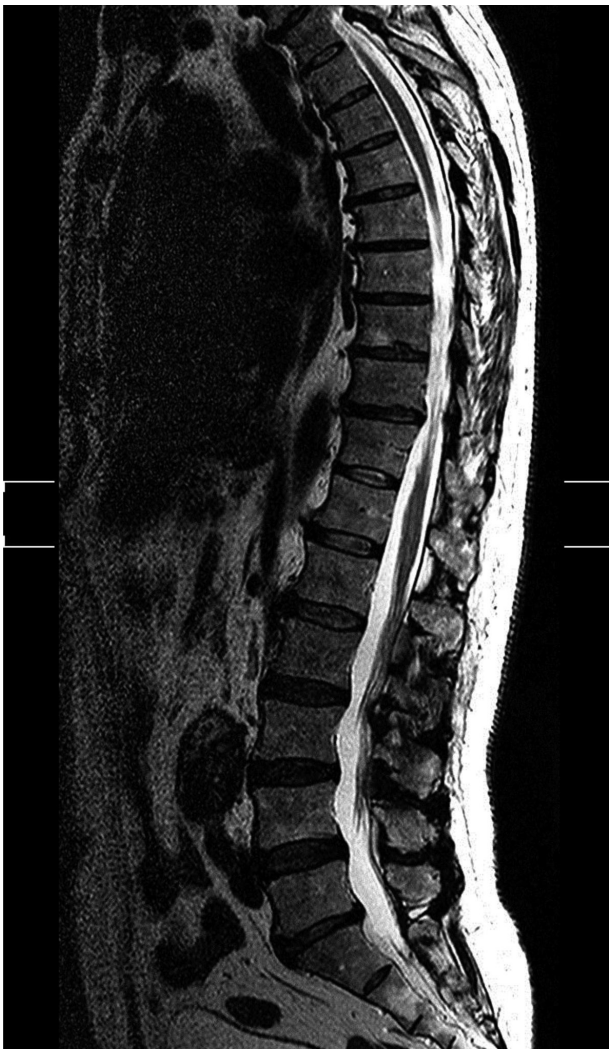

Figure 2. MRI imaging displaying a large spinal cord infarction extended from D11 level to the medullary cone

patient's clinical improvement, a double anti-platelet therapy (clopidogrel plus aspirin) was prescribed, thus stopping the anticoagulant therapy, that was replaced with LMWH at prophylactic doses. On day 3, a thoracic and abdominal CT angiography was repeated, not showing any new finding. On day 8, the patient was transferred to a rehabilitation clinic. After 8 weeks of rehabilitation, the patient was able to walk with the support of a medical walker, also displaying an almost complete recovery of the peripheral sensitivity.

Discussion

A kaleidoscope of predisposing factors for spinal cord infarction include, in adults, aortic aneurysms, venous thromboembolism, coagulopathies and all surgical procedures performed on the aorta. Additional, albeit less specific, factors could be atherosclerosis, arterial hypertension, diabetes and other cardiac disease (1, 5).

In one of the largest series ever published (i.e., 55 patients in up to 20 years), etiologies of infarcts were arteriosclerosis of the aorta and vertebral arteries (23.6%), aortic surgery or interventional aneurysm repair (11%), and aortic and vertebral artery dissection (11%), whereas in 23.6 %, etiology remained unclear (5).

In children, trauma and vascular malformations are the most common causes of ischemia (6).

The spinal cord, due to its length, is perfused by a complex network of arterial vessels. Spinal arteries, including anterior spinal arteries (ASAs) and posterior spinal arteries (PSAs), are the primary source of blood supply. They run along the spinal cord and are small-diameter vessels (0.2-0.8 mm and 0.1-0.4 mm respectively). The small diameter favors the obstruction by emboli (7).

The anterior spinal artery (ASA) syndrome is the most common clinical presentation of spinal cord infarction. The affected patient presents a loss of motor function and pain/temperature sensation, with relative sparing of proprioception and vibratory sense below the level of the lesion. Initially, flaccidity and loss of deep tendon reflexes are predominant, but subsequently spasticity and hyperreflexia develop over ensuing days and weeks. Autonomic dysfunction, as hypotension (orthostatic or frank hypotension), sexual dysfunction, bowel/bladder dysfunction, can be present. Posterior spinal artery (PSA) syndrome produces loss of proprioception and vibratory sense below the level of the injury and total anesthesia at the level of the injury. Weakness is typically transient and mild. ASA syndrome is more often bilateral, whereas PSA syndrome tends to be unilateral (8).

In the aforementioned large series, infarcts occurred in 38.2% at the cervical and thoracic level, respectively, with 49% of patients suffering from centromedullar syndrome caused by ASA occlusion (5).

The clamping of aorta could worsen the spinal flow across these small vessels; few cases of medullary ischemia induced by surgery (both open surgery and endovascular methods) are reported in literature. Wachowski et al. reported a case of medullary ischemia after stent-graft implantation in a patient with abdominal aortic aneurysm. On day 2 the patient revealed bilateral flaccid paralysis as well as deep and superficial sensory disturbances of the lower limbs. The MRI showed both edematous and ischemic lesions (9).

The diagnosis of spinal cord infarction is mainly based on clinical findings, whilst neuroimaging is aimed to confirm the diagnosis and/or exclude other conditions. MRI is the most appropriate test for the diagnosis of spinal cord infarction, being its most important role to rule out the causes of compressive myelopathy. It can also confirm the presence of ischemic lesions and can provide information about the underlying etiology. The test should be performed as soon as possible, although it can be delayed if the patient is scheduled for emergent aortic surgery or other life-saving interventions. MRI shows ischemic lesions as hyperintensities on T2-weighted images (T2WI) (10). In a large series, MRI disclosed hyperintense lesion pattern on T2WI in 98.2%, cord swelling in 40%, enhancement on post-contrast T1WI in 42.9% and always hyperintense signal on diffusion-weighted imaging (DWI) (5).

It should be noted that hyperintensities on T2WI can also be seen in inflammatory diseases, but the sudden onset supports a diagnosis of spinal cord infarction (11). The sensitivity of standard MRI is limited, especially during first hours. If the clinical suspicion is

high and the initial MRI is normal, follow-up imaging should be repeated (10).

Unfortunately, with the exception of compressive or inflammatory causes of an acute spinal cord syndrome, there is no well-established effective therapy for spinal cord infarction, but life-threatening causes like aortic dissection could be found and managed in an emergency setting (5).

Our case is paradigmatic of this rare but potentially devastating condition. As such, EPs should always keep in mind this possibility when evaluating patients with sudden loss of sensitivity and/or strength in their lower limbs.

Conflict of interest: None to declare

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Received: 18 April 2019
Accepted: 18 April 2019
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