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Case Report

Unruptured aneurysm with intramural thrombus is an unusual cause of spinal cord infarction: a case report ^{☆,☆☆}

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ARTICLE INFO

Article history:

Received 19 November 2021

Revised 23 November 2021

Accepted 28 November 2021

Keywords:

Anterior spinal cord infarction

Abdominal aortic aneurysm

ABSTRACT

Infarction of the spinal cord is a rather rare occurrence. Paraparesis or quadriparesis with vibration and proprioceptive senses sparing are symptoms of anterior cord syndrome. Ischemic anterior cord syndrome can result from an obstruction of the anterior spinal artery or the Adamkiewicz Artery. Spinal infarction due to abdominal aortic aneurysm with intramural thrombosis is an extremely rare condition, because of its rarity, it presents a diagnostic difficulty to clinicians, which may result in an inaccurate or delayed diagnosis. We present a case of spontaneous spinal cord infarction due to a previously asymptomatic aortic aneurysm with intraluminal thrombus, with a review of the literature.

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Introduction

Acute spinal infarctions are uncommon occurrences marked by abrupt paralysis or sensory impairments below the level of damage. Spinal cord trauma, vascular injury, arterial dissection, thromboembolic disease, chronic inflammatory diseases, or a mass effect on the spinal cord are all possible causes.

Spinal infarction due to acute thrombosis of abdominal aortic aneurysm (AAA) is an extremely rare condition.

We encountered a case of spontaneous spinal cord infarction due to a previously asymptomatic aortic aneurysm with

intraluminal thrombus, and we present the case with a review of the literature.

Case report

We report the case of this 77-year-old patient, with a history of hypertension under therapy, and an episode of AVCI 6 months ago, who presented to the emergency for the sudden onset of paraplegia preceded by paresthesia with sphincteric disorders.

[☆] Garant of submission: The corresponding author is the garant of submission.

^{☆☆} Competing interests: The authors declare that they have no conflicts of interest.

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<https://doi.org/10.1016/j.radcr.2021.11.068>

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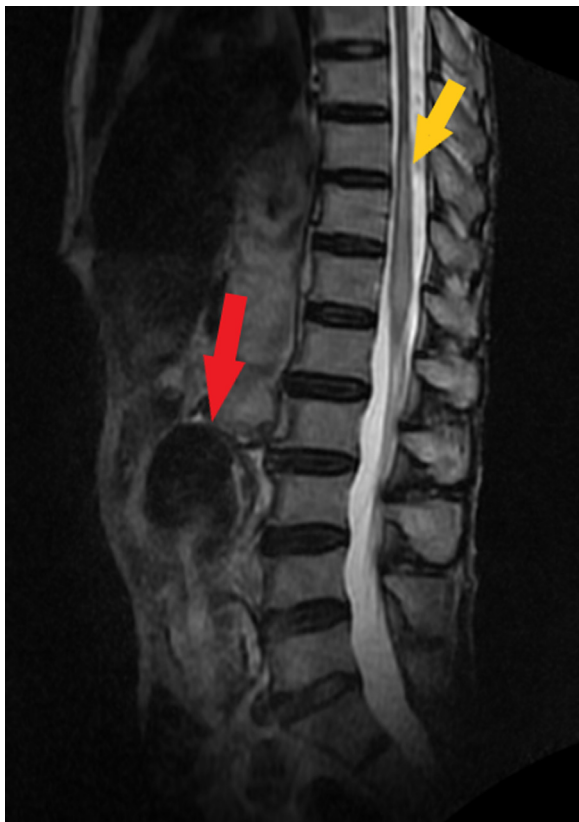


Fig. 1 – Representative magnetic resonance image on sagittal section T2, from the present case of a 77-year-old man showing presenting with paraplegia showing a long T2 hypersignal in the spinal cord extend from D10 to L1(yellow arrow), we also identify the aneurysmal dilatation of the abdominal aorta(red arrow).

The clinical examination indicated a paresis on the right lower limb (1/5) and a paresis on the left lower limb (2/5). However, the vibration and proprioception were both intact.

Furthermore, no deficits in the upper limbs were discovered, and everything is progressing in the framework of apyrexia.

A palpable enlarged bladder with a relaxed anal sphincter tone was discovered during the abdominal examination. The cardiac and respiratory systems were also examined and found to be in good working order. The condition of the mind and cognition were unaltered.

In our case, there have been no data that suggested a systemic infection or infection, such as fever, at the time of symptom onset, and no viral infections occurred prior to symptom onset. Furthermore, due to the COVID-19 pandemic, we undertake a systematic PCR-SARS COV 19 on our patients before their admission, which has proven to be negative. In addition, there was no evidence of COVID-19 infection in the patient's medical history.

Compressive myelopathy was suspected and we performed a magnetic resonance imaging (MRI) without gadolinium of the spinal cord revealed a long T2 signal extend of D10 to L1 (Fig. 1), we also visualized the dilatation of the abdominal aortal extend with intramural thrombosis (Figs. 1 and 2). So



Fig. 2 – Representative magnetic resonance on sagittal section T2 showing the aortic aneurysmal dilatation with a mural thrombus(red arrow).



Fig. 3 – Angio CT on axial section, which revealed marked atherosclerotic plaques in the abdominal aorta with a mural thrombus(green arrow).

we completed an abdominal CT angiography which revealed marked atherosclerotic plaques in the abdominal aorta with a mural thrombus (Figs. 3 and 4). The aneurysmal dilatation extended to 14 cm in height, from L1 to L4 (Fig. 4) with bilateral intramural thrombosis but more marked on the right side (Fig. 4).

We presumed that spinal cord infarction produced by an AAA with intraluminal thrombus was the cause of abrupt paralysis of both lower limbs.

The patient was referred to the vascular surgery department for potential endovascular Exclusion of AAAs with Stent-Grafts but unfortunately, he died of a cardiac attack before receiving endovascular treatment.



Fig. 4 – Coronal section of an angio CT showing the extent of the abdominal aneurysm from L1 to L4 with individualization of a bilateral intramural thrombus more marked on the right side (red star) than on the left side (black star).

Discussion

Spinal cord syndrome is divided into five main categories: anterior cord syndrome, posterior cord syndrome, central cord syndrome, Brown-Sequard syndrome, and Cauda equina syndrome [1].

The anterior spinal cord region has a higher rate of spinal cord infarction; this may be because the radiculomedullary arteries in the anterior territory are less numerous than those in the posterior territory [2].

The Adam Kiewitz (AKA) artery is the most dominant anterior radiculomedullary artery and is responsible for the arterial blood supply to the spinal cord from T8 to the conus medullaris [3]. This artery supplies the lower one-third of the spinal cord [4]. It has a wide range of origins, ranging from the mid-thoracic to lumbar levels, and includes the bilateral T3-T12 intercostal arteries and the L1-L4 lumbar arteries. It is most commonly seen in the T8-L1 neural foramina. The T8-L1 neural foramina and the intercostal or lumbar arteries are the most common sources [3].

The descending aorta's intercostal or lumbar arteries split into an anterior and a posterior branch. The radiculomedullary artery, the muscular branch, and the dorsal somatic branch are all part of the posterior branch. The radiculomedullary artery further subdivides into the anterior

(AKA artery) and posterior radiculomedullary arteries [4]. Side 76.6% of AKAs came from the left side in 76.6% while 23.4% came from the right side, according to the findings. According to a review of 43 research, 89% of arteries start between T8 and L1 [3].

The terminal section of the spinal cord was affected in our case by a spinal infarction; and the aneurysmal dilatation with intramural thrombus in our instance extended from L1 to L4, which anatomically corresponds to the origin of the lumbar artery from which the AKA artery arises, we can assume that the spinal infarction is caused by thrombosis of this one.

The nature and degree of the neurological injury are determined by the infarction's height, its transverse extension, and individual anatomical variances in spinal cord vascularization. The issues may appear acutely, subacutely, or chronically, and the neurological deficiency may appear immediately (as a result of spinal cord hypoperfusion) or later (as a result of protracted hypoxia's repercussions) [2]. When a spinal cord lesion is suspected, the first test that should be done is a spinal MRI. Despite this, a normal MRI in the first few hours does not eliminate the diagnosis [2,5]. The MRI is performed using gadolinium injections in the axial and sagittal planes in T1- and T2-weighted sequences. In a properly formed infarction, a hyper signal in T2 and an isosignal or hyposignal in T1 are translated. Vasogenic edema can cause the spinal cord to enlarge in size [2]. Diffusion sequences may improve the MRI's sensitivity during the acute phase. A distinct hyper signal with a decreased coefficient of diffusion translates the infarction [2].

CT angiography is very useful in emergencies. Because of its ease of use and ability to determine aorta diameter perpendicular to the axis of blood flow at various levels, CT is the examination of choice. It can also help detect penetrating aortic ulcers and intramural hematoma [6].

Aortic pathologies that cause spinal cord infarction include cardiac or aortic surgery, aortic aneurysm rupture, aortic dissection, aortic intramural hematoma, and acute aortic occlusion [5].

Our patient exhibited quadriparesis, proprioception, and vibration are spared, indicating anterior cord involvement; a potential vascular etiology is suggested by the acute history. Furthermore, the pathognomonic aspect of elevated T2-signal intensity, as well as the absence of vertebral disease, point to anterior spinal cord infarction on MRI. However, neither aortic dissection nor rupture of an aortic aneurysm was found in this patient's instance, and no surgery or procedure had been conducted previously. We objective only the presence of aneurysmal dilatation of the abdominal aorta with a thrombus.

In our case, the diagnosis was suspected on MRI and confirmed with a CT angiography.

Weisman and Adams reported the first cases of spinal cord ischemia caused by occlusion of the intercostal and lumbar arteries in Aortic dissection patients in 1944 and postulated that paraplegia in Aortic dissection is caused by vascular obstruction. Ischemia is caused by obstruction of the segmental medullary arteries, which supply the anterior spinal artery, which then supplies the spinal cord [7]. Hand et al reported, a 72-year-old male smoker with acute paraplegia and sensory level at T12/L1 was found to have an infrarenal abdominal

unruptured aortic aneurysm with significant mural thrombus. The presence of liquid thrombus during surgery led to the diagnosis of a substantial cholesterol embolism in the distal vasculature [8]. A thrombosis occluding a segmental artery vital for the vascularization of the spinal cord or an occlusive thrombosis of the aorta might cause medullary infarcts [9]. The probable explanation is detachment of a piece of the thrombus of the aortic aneurysm resulting in embolism of the artery of AKA or mural thrombus blocking the origin of the segmental artery that branches out into the artery of AKA [10]. The cause of anterior cord syndrome and the risk-benefit analysis of implementing a specific therapy determine the course of treatment [1]. When an emergency physician first recognizes a patient with atraumatic acute cord syndrome caused by a vascular blockage, the patient should be started on anticoagulation and a vascular surgeon should be notified to discuss revascularization options [1].

Conclusion

Spinal cord infarction is a rare entity. Aortic dissection/surgical procedures involving the aorta, intrinsic arterial occlusion of the blood vessels perfusing the spinal cord (the anterior spinal artery or radicular branches), hypotension, and venous infarctions are the etiopathogenic mechanisms. Only a few cases of unruptured AAA generating anterior cord syndromes due to mural thrombi had been documented previously.

Patient consent statement

Written informed consent for publication was obtained from patient.

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