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Unilateral Posterior Spinal Cord Ischemia due to a Floating Thrombus: a case Report

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Abstract

Introduction: Spinal cord ischemia (SCI) accounts for less than 1% of all strokes, mostly affecting the anterior cord. The ascending aorta (AA) is the rarest site of localization for aortic thrombi (5%). We report a singular case of posterior SCI due to a floating thrombus in the AA.

Case presentation: A 75-year-old male presented with acute left hemiparesis and left tactile and proprioceptive sensory loss below the seventh thoracic vertebral level (NIHSS 3). Spinal cord MRI showed a C4-C6 ischemic lesion, involving the left lateral-posterior hemi-cord. CT angiography showed a 6-millimetre floating thrombus in the AA. According to cardiovascular surgeons, dual antiplatelet therapy and high-dose statin were started. After seven days, the patient was discharged with a mild left distal hemiparesis and an unchanged sensory deficit.

Conclusion: Posterior SCI is rarer than anterior ischemia and potentially unilateral. Its clinical presentation is mainly sensory with possible, but not systematic, weakness of the homolateral limbs. SCI is often caused by aortic pathologies in the elderly, but the incidence rate of non-aneurysmal aortic mural thrombus is about 0.45% and the AA represents a very rare location. In similar cases, conservative medical treatment is preferred despite the high-risk rate of embolic recurrences.

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Introduction

Compared to cerebral ischemic stroke, spinal cord ischemia (SCI) is quite an uncommon event, representing less than 1% of all strokes, mostly affecting the anterior cord[1]. Hypercoagulability and vessel wall alterations represent the major risks for thrombotic formation in the aorta[2]. Aortic thrombus is frequently detected at the thoracic descending tract (28%) or the distal arch (16%), while the ascending aorta (AA) is the rarest site of localization (5%)[3]. In the case described herein, we demonstrated a floating aortic thrombus in the ascending aorta likely responsible for a cervical posterior spinal cord infarction.

Case Presentation

A patient in their mid-70s was admitted to our hospital presenting with acute cervical pain, weakness in left upper and lower limbs, and left tactile and proprioceptive sensory deficit below the seventh thoracic vertebral level (National Institutes of Health Stroke Scale, NIHSS 3). Medical history was unremarkable, except for a former strong smoking habit (30 cigarettes per day, quitting 10 years before) and previous surgical excision of a lumbosacral herniated disk. The patient underwent brain computed tomography (CT) and brain magnetic resonance imaging (MRI), which excluded a cerebral involvement, while spinal cord MRI showed a C4-C6 ischemic lesion, involving the left posterior horn of the grey matter and the left lateral corticospinal tract (Figure 1). Diagnostic workup included blood laboratory tests (showing high triglyceridemia), clinical cardiac assessment (unremarkable findings) and a transthoracic echocardiogram (highlighting aortic ectasia). Finally, a contrast-enhanced CT angiography (CTA) was performed: the supra-aortic trunks study demonstrated severe atherosclerosis of the right vertebral artery (VA), with a lack of opacification from the V0 to the V3 segment and very filiform flow from V3 to basilar trunk (Figure 2), and a concomitant tortuous course of the left VA. The thoracic study also revealed a 6-millimeter floating thrombus in the proximal ascending aorta (Figure 3). A multidisciplinary committee discussed this clinical case; finally, dual antiplatelet therapy and atorvastatin 80 mg were started. The hospitalization was uneventful, and the patient was discharged on the seventh day, with an improved motor deficit: he was able to walk with unilateral support and the left hemiparesis was mild and predominantly distal (NIHSS 2). At the 3-month follow-up, no complications were reported, the neurological examination was unchanged, and the CTA scan confirmed the stability of the thrombus.

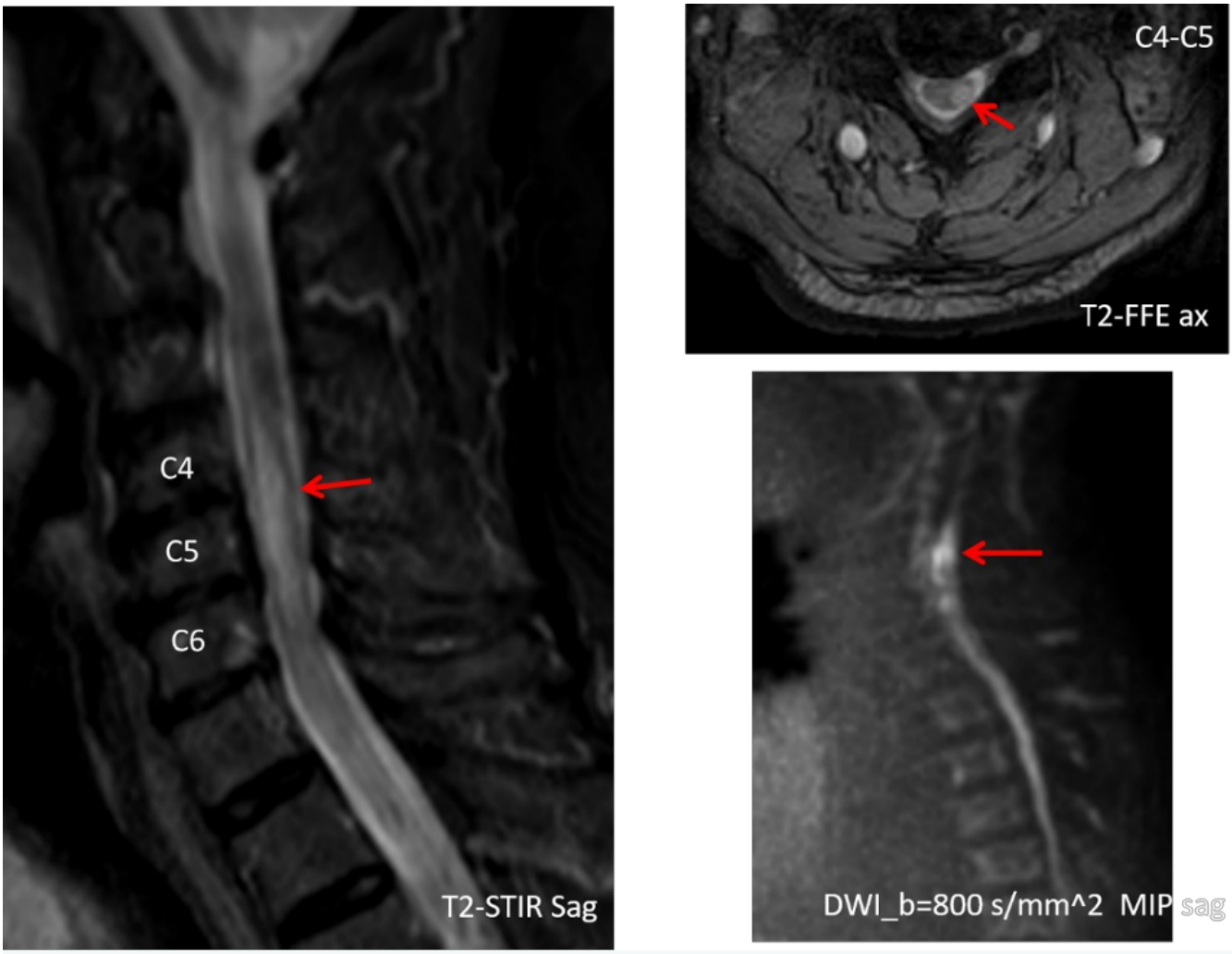

Figure 1. *Magnetic resonance imaging (MRI) of the cervical spinal cord. On the left panel, MRI showing a C4-C6 ischemic lesion on T2-STIR (short TI inversion recovery) sequences (sagittal projection, blue arrow). Right panel: on the upper row, T2-FFE (fast field echo) sequences (axial view) showing the ischemic lesion of the left posterior horn of the grey matter (blue arrow); on the lower row, DWI (diffusion-weighted imaging) sequences (sagittal projection) showing the involvement of left lateral corticospinal tract (blue arrow).*

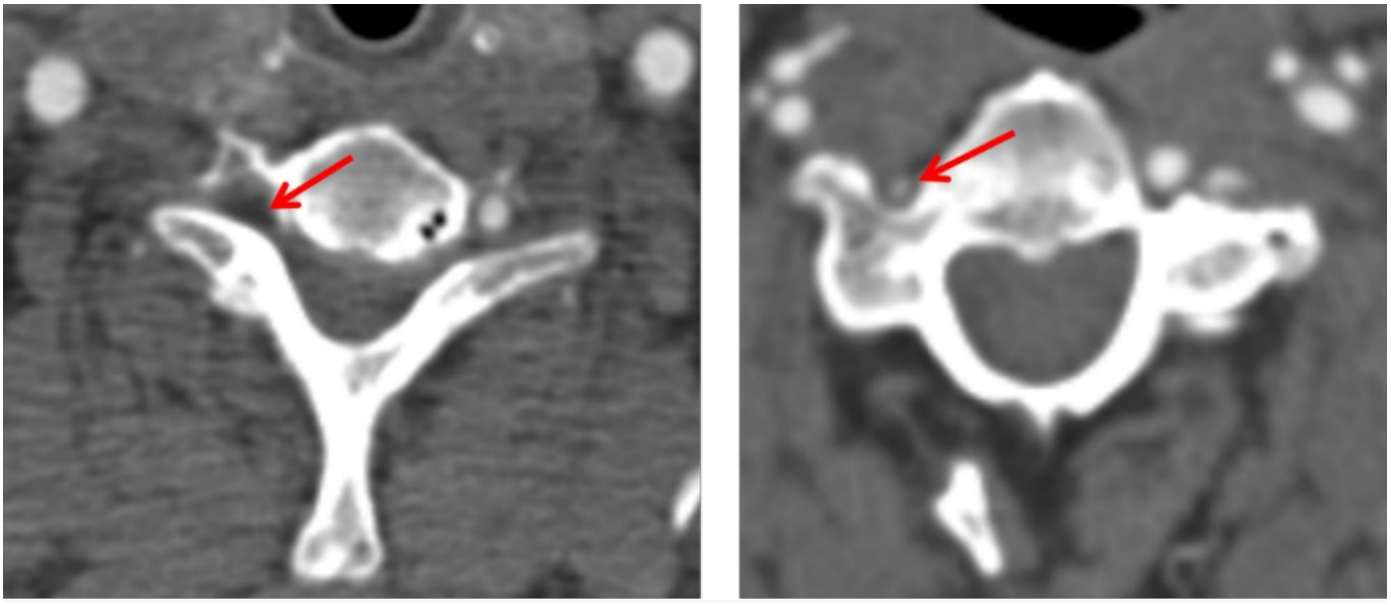

Figure 2. *CTA on axial view at the level of C2 (left panel) and C1 (right panel) vertebra. The supra-aortic trunks study demonstrating severe atherosclerosis of the right vertebral artery (blue arrows), with a lack of opacification from the V0 to the V3 segment (left panel) and filiform flow from V3 to the basilar trunk (right panel).*

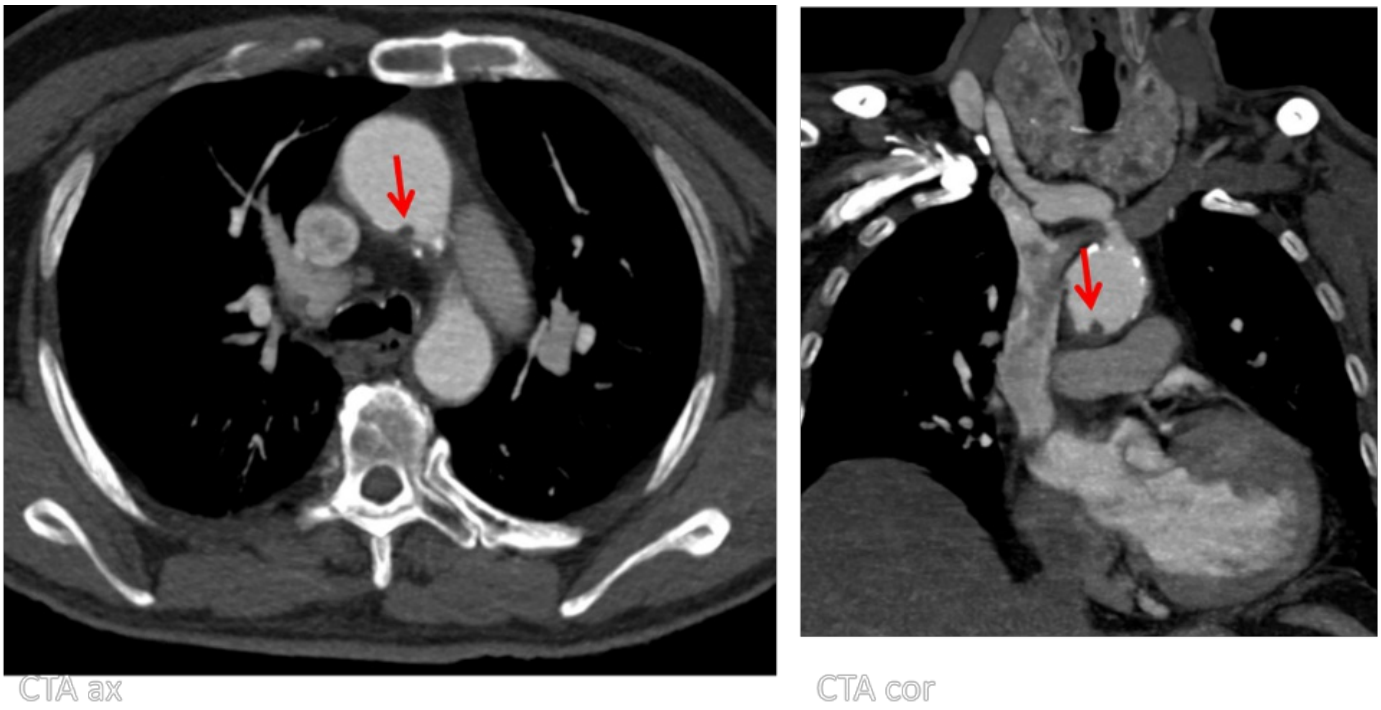

Figure 3. Contrast-enhanced CT angiography (CTA) in axial (left panel) and coronal (right panel) view. The thoracic study revealed a 6-millimetre floating thrombus (blue arrows) in the proximal ascending aorta.

Discussion

Posterior SCI is rarer than anterior ischemia. It is potentially unilateral, and its clinical presentation is mainly sensory with possible, but not systematic, weakness of the homolateral limbs. Conversely, anterior ischemia is more frequently bilateral[4]. In this case, the sensory deficit was ipsilateral to the motor deficit, differently from the classical spinal cord syndromes, which usually present with bilateral sensory/motor abnormalities. However, the presence of a sensory level suggested the possible injury of the spinal cord. Once spinal cord MRI was performed, a clear ischemic lesion was shown in T2-weighted short tau inversion recovery (STIR) and diffusion-weighted imaging (DWI) scans. DWI has the highest sensitivity showing hyperintense signal changes and abnormalities that could be detected within 3 hours. A transient lesion expansion on MRI after SCI has been previously reported as pencil-shaped necrosis[5], with a length usually limited to several vertebral segments[6]. Actually, vasogenic edema in the spinal cord may occur from the acute phase, when a cervical cord compression due to cervical spondylosis causes secondary venous congestion[7]. Venous congestion is a known cause of longitudinally extensive spinal cord swelling, and may initially mimic a peripheral nerve disorder, but T2-hyperintense lesion is most often seen in the center of the spinal cord with peripheral sparing[7][8].

Because of the unilateral posterior hemi-cord involvement, we supposed that the identified cervical lesion might be due to posterior spinal artery occlusion. In fact, the spinal cord is supplied by three spinal arteries (one anterior and two posteriors, arising from the VAs) and several radicular arteries (often arising from direct aortic branches).

SCI is mostly caused by VA pathologies (e.g., arteriosclerosis, dissection), hypotension, inflammatory vascular diseases, hematologic alterations, and drug abuse. In the elderly population, most reports identify aortic aneurysms and aortic/spinal surgical procedures as the main causes of spinal syndromes, but in clinical practice, the pathogenesis of spinal cord strokes remains frequently unclear. Aortic mural thrombus (AMT) is often found on atherosclerotic or aneurysmatic walls, and it might be able to determine significant arterial emboli[1]. In this case, the patient's medical history did not reveal any cardiac disease, previous vascular surgery, or aortic aneurysm, while former strong cigarette smoking only suggested an atherosclerotic predisposition. Searching for a source of embolism, we investigated the heart and the aorta with electrocardiogram, transthoracic echocardiogram, and thoracic contrast-enhanced CTA, finally finding a 6-millimetre floating thrombus in the ascending aorta. Most arterial emboli originate from cardiac disease or severe aortic pathologies (80-90%)[3]. CTA is the first-choice investigation when there is a suspicion of arterial thrombus, but a transesophageal echocardiogram could be preferred if the patient has potential iodine-anaphylaxis history. We supposed that the detected non-aneurysmal aortic thrombus could have been the possible source of arterial embolism causing cervical SCI. As opposed to aneurysmal AMT, the incidence rate of non-aneurysmal AMT seems to be only about 0.45%[1]. Considering how rarely a floating thrombus in the aorta can occlude a posterior spinal cord artery, an alternative mechanism that could explain this case could be a transient hypoperfusion, caused by the severe atherosclerosis of the right VA and the concomitant tortuous course of the left VA detected by CTA. Anyway, as shown, the left vertebral artery is widely patent without significant stenosis or atherosclerotic disease (unlike the right) and thus the left infarct is most likely embolic from the detected thrombus, as we supposed.

Possible treatment for SCI due to a floating thrombus is very heterogeneous, including antiplatelets, anticoagulants, thrombolytics, endovascular surgery and open surgery. The choice of treatment mostly depends on the patient's conditions, thrombus morphology, and physician experience and nowadays there is no consensus on the best management of this pathology[2]. Generally, when unstable aortic plaques or aortic floating thrombus are detected, conservative medication is preferred because of the elevated efficacy rate and the fewer adverse effects, even if it coexists with a high risk of embolic recurrences (about 73% of floating thrombus, compared to 12% of sessile thrombus)[3]. Following the ESC guidelines on diagnosing and treating aortic diseases, anticoagulation or antiplatelet therapy should be considered when aortic thrombus were detected after stroke or peripheral embolism[9]. Contraindication to anticoagulation or recurrent embolism can lead to surgical intervention[3].

In conclusion, SCI is a rare event, and the unilateral involvement of the posterior medullary territories is extremely peculiar; the identified primary source of the embolism was supposedly a floating thrombus located in the ascending aorta. Spinal cord MRI should be a routine investigation when ischemic events are suspected, and CTA best identifies any aortic wall alteration. Generally, unilateral infarcts are less severe, with a better prognosis. Because of the rarity of this disease, there is no standardized protocol or clinical guideline, and therapy relies on clinical judgment and physician experience.

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