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Case Report

Spinal cord infarction complicating acute aortic syndrome: about 2 cases ☆,☆☆,★

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ABSTRACT

Spinal cord infarction is an uncommon but devastating disorder caused by various conditions. It remains however a rare neurological complication in acute aortic injuries. In this context, aortic dissection is the most frequent etiological factor. Acute aortic intra mural hematoma and atheromatous penetrating ulcer remain exceptional. We encountered two cases of spinal cord infarction associated with acute aortic intra mural hematoma in one case and atheromatous penetrating ulcer in the other case that presented without typical severe pain. Thus, acute aortic injuries should be considered a cause of spinal cord infarction even if there is little or no pain.

Introduction

Spinal cord infarction is a rare condition, usually marked by an acute onset. It clinically associates an acute and intense back pain, with motor, sensory, and sphincterians dysfunction. The etiologies are various and include aortic surgery, vascular malformations, aneurysm, and aortic dissection. We encountered 2 cases of spinal cord infarction with atypical etiologies including acute aortic intramural hematoma in one case and atheromatous penetrating ulcer in the other case manifesting as a transverse medullary infarction pattern but without the classic symptom of chest or back pain.

Case 1

A 70-year-old man presented to the emergency department with sudden onset of paraplegia and urine retention. He was

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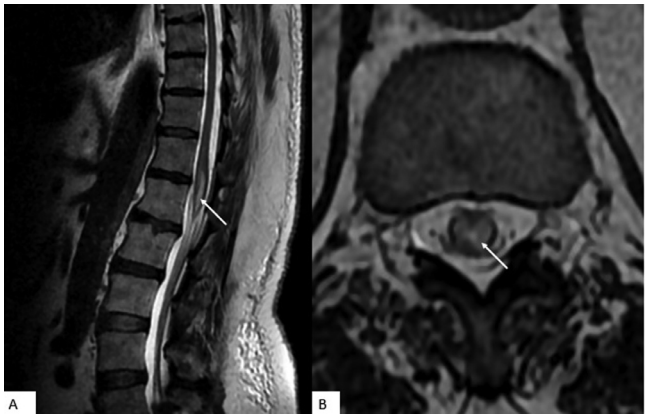

Fig. 1 – (Case 1): sagittal (A) and axial (B) T2WI images showing a large pencil like hyperintensity involving the conus medullaris. The axial section shows that the abnormality is essentially involving the grey matter (B)

transferred to our hospital 3 days after onset. His medical history was remarkable by an unwell managed hypertension and cholecystectomy 8 years ago. His blood pressure was 170/90 mmHg, and his heart rate was regular at 75 bpm. He was alert and oriented but had difficulty standing up. Physical examination revealed a complete impairment of muscle function in both lower extremities. Osteotendinous reflexes were absent on both sides. However, both sensory perception of pain, vibration and touch was preserved. Based on these findings, we graded his condition as Frankel grade A. We performed a magnetic resonance imaging (MRI) of spine and brain witch revealed no compression but a linear high signal intensity area on T2-weighted images in the conus medullaris (Fig. 1 image A). Axially, the abnormality involved the grey matter of the spinal cord (Fig. 1 image B). We also performed a full body enhanced computed tomography that revealed a mural hematoma in all parts of the aorta (Stanford type A) (Fig. 2). Spinal fluid was examined, but no abnormality was found. Therefore, we made a diagnosis of spinal cord infarction complicating an acute aortic intramural hematoma. Antihypertensive therapy was started associated to clean intermittent catheterization for his bladder dysfunction. After rehabilitation, his paralysis partially improved to the point that he was able to walk with the aid of a cane.

Case 2

A 81-year-old woman presented to the emergency department with sudden onset of lower limb weakness, legs numbing and urine retention. She reported having Sars Cov-2 infection 20 days before the onset, that was well managed. She was transferred to our hospital 5 days after onset. Her medical history was remarkable by pulmonary tuberculosis since 4 years and unwell managed blood hypertension. At the admission, her blood pressure was 100/70 mmHg, and her heart rate was regular at 80 bpm. She was alert and oriented but had difficulty standing up. Physical examination revealed sensory loss below T10 in which sensory perception of pain, vibration and touch were absent. Muscle function was completely impaired in both lower extremities. Osteotendinous reflexes were absent on both sides. Based on these findings, we graded her condition as Frankel grade A. She underwent a magnetic resonance imaging (MRI) including brain and spine. MRI revealed a linear high signal intensity area on T2-weighted images extending from T3 to T9 (Fig. 3 image-A). Axially, the abnormal signal the gray matter throughout the affected area of the spinal cord (Fig. 3 image- B). There was no brain lesion. Spinal fluid was examined and was normal. The patient underwent a hole body CT. Unfortunately, we found out a penetrating atheromatous ulcer of the aortic isthmus (Fig. 4). Therefore, we made a diagnosis of spinal cord infarction complicating a penetrating atheromatous ulcer due to the embolism of Adamkiewicz artery. Antihypertensive therapy was started associated to clean intermittent catheterization for his bladder dysfunction. After rehabilitation, his paralysis did not improve and patient had severe physical handicap requiring a wheelchair and bladder catheterization.

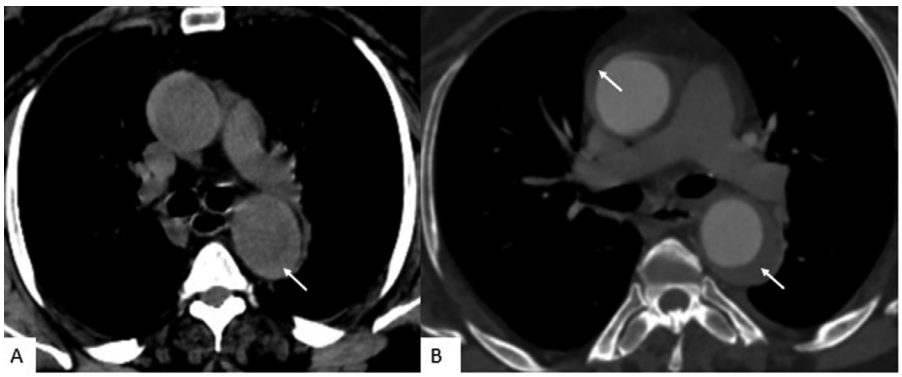

Fig. 2 – (Case 1): axial unenhanced (A) and enhanced (B) CT scan images showing a crescent like mural hyperdensity of aorta, unenhanced after iodine injection relative to a mural hematoma of aorta

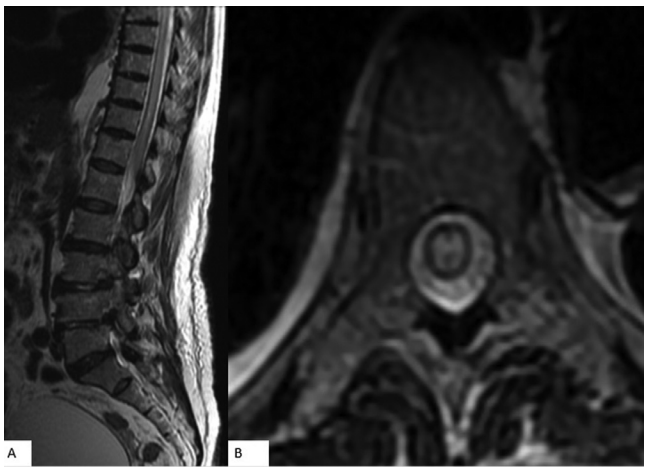

Fig. 3 – (Case 2): sagittal (image A) and axial (image B) T2WI images showing a large hyperintensity (greater than one vertebral body height) involving the thoraco lumbar spinal cord. The axial section shows that the abnormality is essentially involving the grey matter (B)

Discussion

Spinal cord vascularization is complex. In one hand, it is insured by spinal arteries. There is one anterior spinal artery and two posterior spinal arteries emanating from intradural vertebral arteries. These arteries run vertically from the cervical medullary segment to the conus medullaris. In the other hand, this vascular system receives supplies from anterior and posterior radiculomedullary arteries arising from intercostal and lumbar arteries, The most important are "Adamkiewciz artery" located at a variable height, generally next to T9 and the artery of "Von Haller" in the upper thoracic region. These branches form with the spinal arteries a perimedullary anastomotic circle called "corona medulla" which essentially supplies the white matter of the spine. The gray matter it is vascularized by sulcal arteries emanating directly from spinal arteries. Despite this vasculature richness, they are some watershed zones, especially thoraco lumbar junction.

Spinal cord infarction is an uncommon condition. It incidence is 1% that of cerebral infarction [1].

Clinical presentations are various and are defined by the vascular territory involved with the severity of the impairment varying from paraplegia to minor weakness. Back pain is often associated to spinal cord infarction and has been reported in as many as 70% of patients typically at the level of the lesion [2].

Classic clinical forms include anterior spinal artery pattern and posterior spinal artery pattern.

Typically, anterior spinal artery (ASA) infarct causes involvement of anterior two-thirds of cord involving gray and white matter, causing bilateral weakness, dissociative sensory loss with or without autonomic symptoms [3].

Posterior spinal artery (PSA) infarct is usually unilateral and less severe, because of presence of two posterior spinal arteries. This syndrome leads to ipsilateral loss of light touch, vibration, and proprioception while mostly sparing the motor function. However, there can be segmental deep tendon areflexia due to posterior horn involvement and paresis below the level at which the posterior portion of the cord is affected [4].

Some clinical forms are incomplete including:

Sulcocommissural syndrome causing as a partial Brown-Sequard syndrome which consists of hemiparesis with a contralateral spinothalamic sensory deficit.

Central spinal infarct occurring after cardiac arrest or prolonged hypotension; its clinical presentation includes bilateral spinothalamic sensory deficit with sparing of the posterior columns. Motor deficit and sphincter dysfunction are usually absent

Finally, a rare presentation is transverse medullary infarction (full transverse lesions) presenting with sudden inability to walk due to paraplegia or tetraplegia. There may be com-

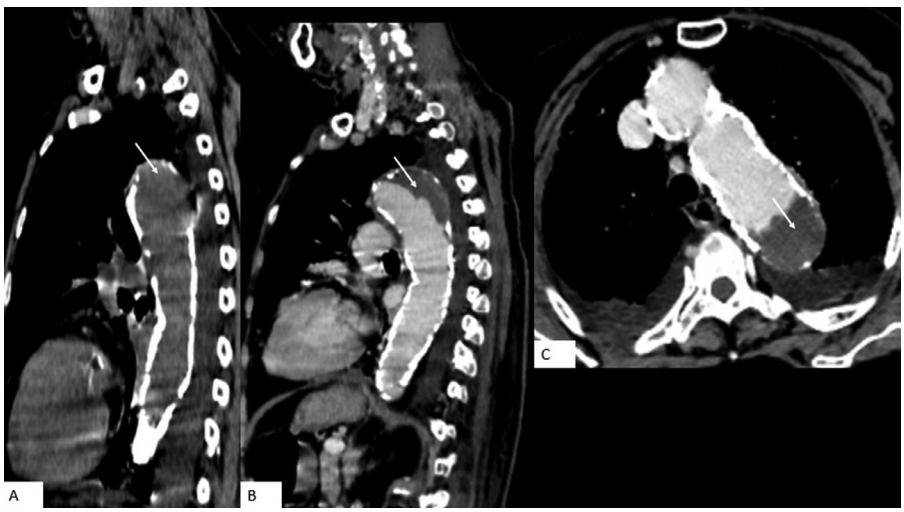

Fig. 4 – (Case 2): Sagittal unenhanced (A) CT, sagittal (B) and axial (C) enhanced CT scan images showing a penetrating atheromatous ulcer of the aorta isthmus

plete sensory loss involving all modalities and pain reported as circumferential tightness. Autonomic dysfunction is noted. Usually it is of embolic origin as in our cases [4].

The etiologies of spinal cord infarction are various. They are usually a consequence of systemic arteriosclerosis (19%), or attributable to aortic dissection or aortic aneurysm (8%) [5]. Iatrogenic causes include vascular surgery (25%) with aortic cross clamping, aortic stent-grafting, open hiatal hernia repair, lumbar sympathectomy, adrenalectomy and anesthetic procedures including epidural anesthesia, intercostal nerve block and celiac plexus. Rare causes include other causes of acute aortic injuries including intramural hematoma and atheromatous penetrating ulcer, cardiac embolism, decompression sickness, coagulopathy, spinal arteriovenous malformation, fibrous cartilaginous embolism, sickle cell disease, vasculitis, and medication [3]. Embolism of the artery of Adamkiewicz is also a possible etiology. It often causes thoracolumbar medullary infarction. It remains however idiopathic in 35% [3].

Our cases had a lower thoracic spine and conus medullaris injuries, which possibly due to the involvement of Adamkiewicz artery. In the first case, we think that Adamkiewicz artery was accidentally occluded because it ostia emanated from the mural hematoma. In the second case, the hypothesis of Adamkiewicz artery embolism from the ulcer clot was more pleasant.

The sensitivity of initial MRI of the spinal cord is limited, nearly 17% to 45% of clinically suspected spinal cord infarction cases have a normal MRI. An abnormality may become apparent on T2-weighted images a week later [6]. It allows in the other hand to rule out other causes of acute spinal syndrome, such as compressive myelopathies, vascular malformations, infective myelitis, demyelinating disorders that have abnormal MRI findings at an early stage. Thus, the absence of abnormal findings on MRI in the initial few hours to days should raise suspicion for spinal cord infarction and MRI should be repeated within a week. abnormal findings on MRI in the initial few hours to days should raise suspicion for spinal cord infarction.

On a T2 sequence, hyper-intensity of the ischemic cord is present, in the central cord more than the peripheral cord. This T2 hyper intensity is generally seen in greater than one vertebral section. Hyper-intense marrow signal may also be present within the medullary bone of the vertebral bodies secondary to vertebral body infarction. If the infarction is predominately in the watershed zone of the grey matter of the anterior horns, then the classic “owl’s eyes” or “snake eyes” sign can develop on T2 weighted axial images and “pencil like” hyper intensities on T2 weighted sagittal images [7,8]. Diffusion restriction will also be present in the spinal cord, just as it is in the brain during infarction it has been reported to be more sensitive for detection of spinal cord infarction in the early stage. Patchy contrast enhancement can sometimes be seen during the sub-acute phase [9].

On a T1 sequence, slight cord expansion, decreased signal, or no obvious signal abnormality may be seen. Atrophy of the spinal cord may be seen in the late stage

Regardless of the etiology, acute aortic injuries such as aortic dissection, intramural aortic hematoma and atheromatous penetrating ulcer are life-threatening conditions and should be investigated carefully in the presence of spinal cord infarction. Patients with acute aortic syndrome such as in our cases often complain of back pain. However, there are some reports of aortic acute lesions presenting with little or no pain [10,11]. In addition to this, pain could arise from spinal cord infarction alone. Therefore, cases of spinal cord infarction caused by aortic acute injuries that present without severe pain are quite rare. Although misdiagnosis of these acute aortic injuries can have serious clinical consequences and enhanced computed tomography should be performed in a patient with acute myelopathy to rule out aortic disease.

Just as with infarctions of the brain, treatment of spinal cord infarctions is based on prevention of secondary injury or repeat infarctions. Maintaining systemic perfusion with supportive treatment of possible neurogenic shock make up the mainstay of treatment. Different medications can be used including antihypertensive drugs, antiplatelet or anticoagulant agent, a corticosteroid, or continuous spinal drainage, depending on the cause However, corticosteroid usage in spinal cord infarction is controversial. Some small series advocate the usage of lumbar drain for removing cerebrospinal fluid. The theory being that lower cerebrospinal fluid pressure decreases resistance to spinal cord arterial flow [12].

Although the prognosis of spinal cord infarction has been reported to be more benign than that of cerebral stroke in terms of cognitive function and mental state, it is however responsible of severe physical handicap. About half of patients with spinal cord infarction require a wheelchair or bladder catheterization [13,14]. According to the literature, authors are suggesting that patients with Frankle scores of A or B have worse outcomes than other scores [14].

In summary, we encountered two cases of spinal cord infarction associated with acute aortic injuries that presented without typical severe chest or back pain. These etiologies should be considered as cause of spinal cord infarction and an enhanced CT angiogram should be done even if there is no or little pain.

Patient consent

Consent was obtained from the patient. The study was conducted anonymously.

Availability of data and materials

The data sets are generated on the data system of the university hospital of Fès.

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