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Case Report

Acute paraplegia as a presentation of acute aortic occlusion

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ABSTRACT

Acute aortic occlusion is a rare life-threatening event. We present a case of a heavy smoking, 54-year-old man who was admitted in the emergency room with sudden paraplegia, associated to severe lower back and lower limbs pain. A neurologic examination showed paralysis of the lower limbs and cold lower extremities. The pedal and femoral pulses were absent. A computed tomography revealed occlusion of the mesenteric superior artery, abdominal aorta, and both iliac arteries. Despite medical treatment, the patient died before evaluation of vascular surgery. Paraplegia is a rare presentation of acute aortic occlusion and clinicians should be alert to make an early intervention.

Introduction

Acute aortic occlusion (AAO) is an uncommon vascular emergency with a high degree of morbidity and mortality. Some studies report a 30-day mortality between 20% and 75% [1].

The etiology of this condition is variable and could be embolism from proximal aorta or the heart, in situ thrombosis as well progression of atherosclerotic lesions [1–3].

The most frequent clinical presentation is acute limb and low back pain [1,2]. The prompt recognition is important to prevent morbidity and mortality. The treatment could be with open aortic surgery, thrombolysis, or with endovascular techniques [1,4].

Case description

A 54-year-old man, without regular medical surveillance, presented to the emergency room (ER) with acute onset of paraplegia and severe lower back and lower limbs pain, in a time lapse of 1 hour. He had no history of previous trauma. In the last 6 months, he was admitted several times in the ER with intermittent claudication, lower back and lower extremities pain. It was assumed that he had sciatica due to lumbar herniated disc.

The patient had a medical history of smoking (60 cigarette packs/year) and chronic alcohol consumption. He had no regular medication.

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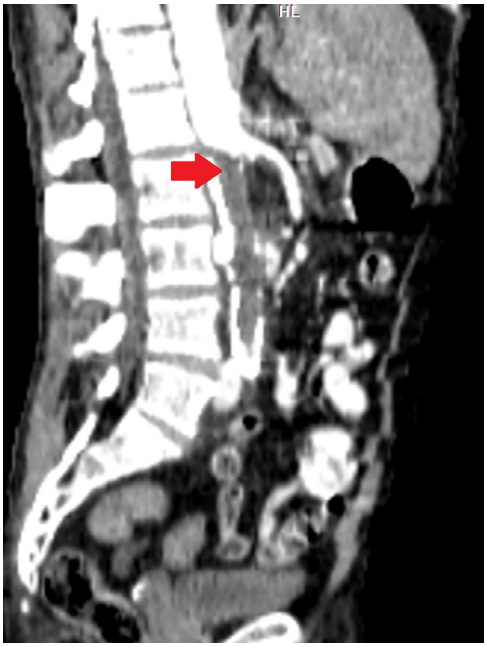

Fig. 1 – Computed tomography angiography, sagittal plane, showing acute abdominal aortic occlusion (red arrow). (Color version of figure is available online.)

On examination he had a blood pressure of 174/120 mm Hg in the right arm and 154/100 mm Hg in the left arm. He had tachycardia and the others vital signs were normal. The cardiopulmonary examination was unremarkable. The abdomen was painless on palpation. A neurologic examination showed normal motor function of the upper extremities and paralysis of the lower limbs, with bilateral hypoesthesia until L2 dermatome, mottled skin, and cold feet. The pedal pulses and the femoral pulses were absent.

The laboratory test results showed: macrocytic anemia (Hb = 11.8 g/dL, Normal range (NR) 13.5-18 g/dL), normal platelets (265 000/μL, NR 150-450/μL), normal leukocytes (11,000/μL, NR 4-10.5/μL), high international normalized ratio (1.2, NR 0.9-1), high d-dimers (1 352 ng/mL, NR <243 ng/mL), normal blood urea nitrogen (8.8 mg/dL, NR 6-20 mg/dL), normal creatinine (1.1 mg/dL, NR 0.7-1.2 mg/dL), elevated erythrocyte sedimentation rate (53 mm/hr, NR <20 mm/hr), and C-reactive protein (76.46 mg/L, NR <5 mg/L). The arterial blood gas test revealed a metabolic acidemia: pH 7.216 (NR 7.35-7.45), pCO2 10.4 mm Hg (NR 35-45 mm Hg), pO2 84 mm Hg (NR 83-108 mm Hg), $HCO3^-$ 4.1 mmol/L (NR 24-26 mmol/L), SO2 91.6%, Na^+ 131 mmol/L (NR 135-145 mmol/L), K^+ 5.2 mmol/L (NR 3.5-5.1 mmol/L), lactate 15 mmol/L (NR 0.5-1.6 mmol/L).

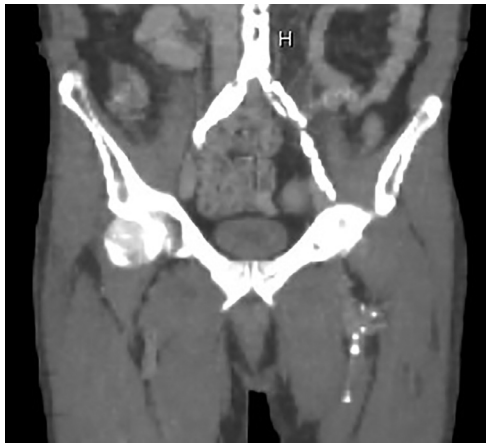

Fig. 3 – Computed tomography angiography, coronal plane, revealing diffuse atherosclerotic calcifications.

The electrocardiogram had a supraventricular tachycardia without ST alterations.

A lumbar computed tomography (CT) scan excluded medullary compression. A CT angiography of the abdomen and pelvis showed occlusion of the mesenteric superior artery, occlusion of the abdominal aorta in the beginning of the renal arteries with extension to the iliac arteries and division branches (Figs. 1 and 2) and signs of bilateral renal hypoperfusion without signs of intestinal distress. The exam also revealed diffuse atherosclerotic calcifications (Fig. 3) and hepatomegaly.

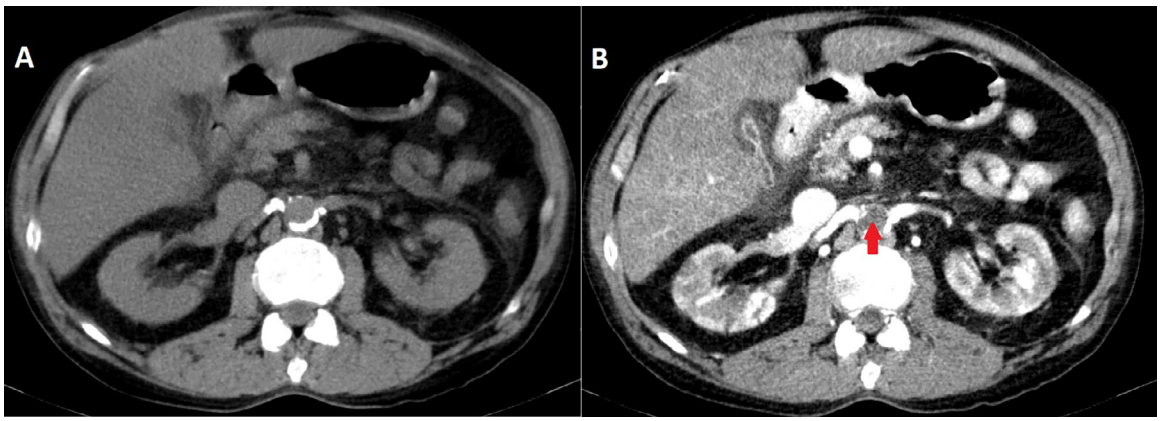

Fig. 2 – Computed tomography (CT) scan revealing an atherosclerotic aorta (A) and CT angiography showing acute abdominal aorta occlusion (red arrow) in the beginning of the renal arteries (B). (Color version of figure is available online.)

After 45 minutes after admission, while the patient was being transported from the radiology department to the ER, for posterior transport to vascular surgery of other hospital, he suffered a cardiorespiratory arrest, with a nonshockable rhythm (asystole). Cardiopulmonary resuscitation was started, the patient was intubated and it was assumed the diagnosis of paraplegia secondary to AAO. He was treated with low-molecular weight heparin, calcium gluconate, and sodium bicarbonate. The patient kept on a nonshockable rhythm and after 16 cycles of advanced life support, with 8 mg of adrenalin it was decided to stop the advanced life support and the patient was declared dead.

Discussion

As previous mentioned AAO is a rare condition with a wide variety of causes. In this case, the most probable etiology was superimposed thrombosis of an atherosclerotic abdominal aorta, secondary to heavy smoking.

The clinical presentation may vary from abdominal symptoms, acute limb ischemia, low back pain, and neurological symptoms of the lower extremities [1–3]. Low back pain is a common presenting complaint, and only a small percentage has concomitant neurologic symptoms. Acute painful paresis or paraplegia is a rare form of AAO presentation and it is a consequence of spinal cord ischemia [3]. A nontraumatic paraplegia represents a diagnostic challenge, with a broad spectrum of etiologies including spinal cord external compression, infection, and ischemia. A structured approach to early intervention and management is imperative to preserve the neurological function. A clinical examination of peripheral pulses in these patients is mandatory [1–3]. In the reported case, a heavy smoker with a 6-month history of intermittent claudication should raise the suspicion of atherosclerotic aortic disease. Beside that the absent/weak pulses with cold extremities were other clue to vascular disease.

The CT angiography is the gold standard modality of the AAO diagnosis [2]. After the diagnosis is made the anticoagulation should be immediately initiated and urgent revascularization procedures (thromboembolectomy, aortic reconstruction, bypass, and thrombolysis) should be attempted [3,4]. In this case, the patient had a cardiorespiratory arrest before revascularization was attempted, probably due to the acute renal injury and metabolic acidemia. This patient had a normal intestinal perfusion, because, probably the collateral vasculature was able to maintain adequate basal perfusion of the intestines, revealing a chronic superior mesenteric artery occlusion. However, he had normal size kidneys, with signs of low perfusion, a fact that support an AAO, without collateral circulation stabilized. Despite the medical efforts the patient did not survive, revealing the high mortality rate associated to this condition.

In conclusion, the AAO should be considered in the differential diagnosis of sudden-onset paraplegia, especially in the presence of pain and weak or absent peripheral pulses. Prompt diagnosis and intervention may prevent morbidity and death.

Patient consent

The patient passed away so we do not have a writen consent. To protect the patient identity all the personal information has been removed.

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