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Case Report

Acute Aortic Occlusion with Acute Onset of Paraplegia and Severe Hyposthenia

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ABSTRACT

A 75-years-old man presented at our ED with acute onset of paraplegia and severe bilateral lower limb hyposthenia. The patient 's neurological assessment was negative except for lower limbs positive Mingazzini test. CT angiography detected a complete lack of opacification of the abdominal aorta immediately below the emergency of the inferior mesenteric artery, caused by a coarse thrombus in the left ventricle. We present a case of acute aortic and lumbar arteries thrombosis with paraplegia and no clear symptoms of acute limb ischemia, in which the motor deficit in the lower extremities was explained by anterior spinal cord syndrome secondary to acute occlusion of lumbar arteries.

Background

Acute aortic occlusion (AAO) is a rare and infrequent medical condition with high mortality rate. Clinical symptoms depend on the level of the aortic occlusion and can be mistaken for stroke or similar neurological disease. It may appear under extremely various forms, from absolutely asymptomatic up to the most serious clinical presentations with severe pain and ischemia of the lower limbs, sometimes accompanied by paresthesia or paraplegia. Intravenous contrast enhanced Multidetector Computed Tomography (MDCT) is the imaging modality of choice for investigating aortic occlusion; MDCT also identifies possible comorbidities that can influence clinical management and therapeutic choices. We report a case of AOO with acute onset of paraplegia and severe hyposthenia.

Case Presentation

A 75-years-old man presented to our Emergency Department with acute onset of paraplegia and severe lower limbs hyposthenia. Acute symptoms arised,while he was seated, in a time lapse of one hour. The patient had a medical history of ischemic heart disease treated with PCI and stenting and controlled mild hypertension under drug medication, he had no history of previous trauma. Vital signs were normal except for PA of 140/80 mmHg. The cardiopulmonary examination was unremarkable, peripheral pulses were present but weak. The abdomen was slightly painful on palpation, skin was normal. Neurological assessment was negative except for positive Mingazzini test in the lower limbs.

Investigation

Laboratory tests showed a high white blood cell count ($11x10^3/mm^3$), moderate high levels of C-reactive protein (2mg/dl) and urea (55 mg/dl). Coagulation tests and lactate were unremarkable. EKG was normal. Patient underwent MDCT angiography in emergency settings. Chest and abdomen CT angiography were performed (supine position, peripheral venous access of 18G in right antecubital vein with 120 ml of iodinated contrast material at a rate of 4 mL/sec). At MDCT examination, a defect

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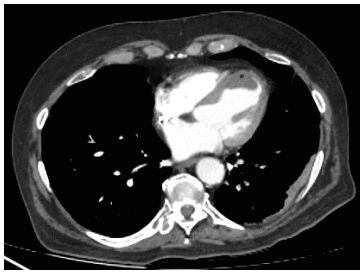
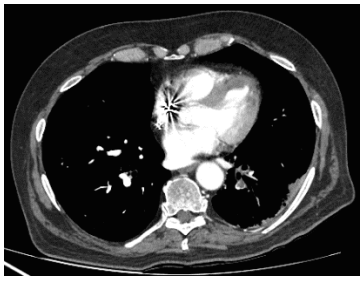
of opacification adherent to the left ventricle wall was visualized, caused by an intracardiac thrombus (42x23 mm) and pulmonary embolism in the lower left lobe was detected (Figures 1A & 1B). Complete lack of intravenous opacification of the abdominal aorta was detected, which extended immediately below the emergency of inferior mesenteric artery (Figures 2A & 2B) to the iliac bifurcation with associated lack of opacification of lumbar arteries below the vertebral body of the first lumbar vertebra (Figure 3). Final diagnosis of acute abdominal aortic occlusion caused by cardiac embolus was achieved by CT, and the patient underwent embolectomy.

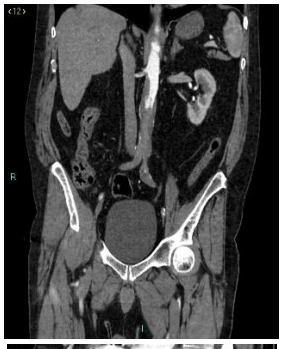


1A

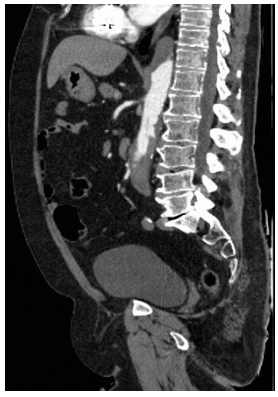


1B

Figures 1: Axial CT images with iv contrast. **A)** thrombus (42x23 mm) displayed as a defect of opacification (*) adherent to left ventricle's wall. **B)** pulmonary embolism in the lower left lobe.



2A



2B

Figures 2: CT images with iv contrast. **A)** coronal multiplanar reconstruction(MPR). **B)** sagittal MPR: lack of intravenous opacification of the abdominal aorta immediately below the emergency of inferior mesenteric artery, extended to the iliac bifurcation.

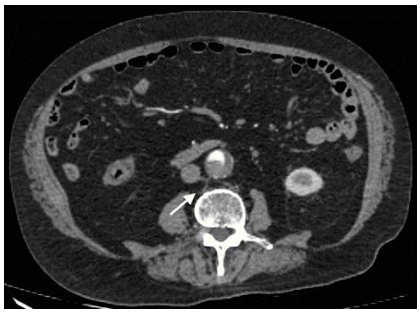


Figure 3: CT axial image with iv contrast. Lack of opacification of lumbar arteries below the vertebral body of the first lumbar vertebra (white arrow).

Discussion

AAO is a rare condition with high mortality rate, Grip *et al.* during a study of 20 years reported an incidence of 3.8 per 1 million person-years with mean age of 69.7 years [1]. AAO may be primary or secondary, primary aortic thrombosis occurs in an apparently normal aorta and the etiology of thrombus formation in a macroscopically normal aorta is not well understood, some causes are related to the patient's medical history: cancer chemotherapy, cocaine intake, essential thrombocythemia and some hypercoagulable states [2-4].

In case of secondary AAO, the most frequent etiologies are thrombosis in situ of an atherosclerotic aorta, an embolus in the aortic bifurcation, or thrombosis of an abdominal aortic aneurism; aortic thrombosis after a blunt trauma has also been described in a child [5-7]. In all cases, except for embolism, the pathogenesis is related to hypercoagulation disorder, hypotension or other low flow state or any other mechanism that affects platelet function, also high levels of stress-related hormones are considered a predisposing factor [8, 9]. Most patient present with bilateral acute limb ischemia with pain, coolness, paresthesia, absent pulses and mottling of the skin [10, 11]. Additional symptoms depend on level of thrombosis: acute renal failure in renal artery thrombosis, mesenteric ischemia in superior mesenteric thrombosis, gastric e\o hepatic failure in case of celiac trunk thrombosis. Johnson's *et al.* described 10 out of 17 patients with AOO presented with acute onset of lower limbs paraplegia [11].

The motor deficit in the lower extremities can be explained partially by acute occlusion of the lumbar arteries with secondary anterior spinal cord syndrome and to lower limb muscles ischemia [12]. In case of AAO rapid diagnosis is essential, because ischemic skeletal muscles injuries often occur within 2 hours of ischemia.; in patients presenting with paralysis definitive limb loss can occur within 6 to 8 hours [13].In our patient the atypical clinical presentation with acute paraplegia and severe hyposthenia, was determined by the fact that external iliac arteries still showed minimal opacification and by the rapid onset of symptoms just one hour before the patient access to ED. According to previous published studies CT represents the imaging of choice in the diagnosis of AAO [14, 15]. Aortic occlusion was clearly demonstrated, and the contemporary thrombosis of lumbar arteries was clearly detected. Acute neurological symptoms require a rapid differential diagnosis with acute

neurological syndromes in particular with spinal cord compression, therefore a careful neurological examination is important.

Although the CT diagnosis of acute aortic occlusion is not difficult, it is important to differentiate a thrombosed aorta from a saddle embolus: the therapeutic intervention in the former case is embolectomy, whereas a thrombosed aorta requires revascularization. Although in our case abdominal the aorta was apparently normal with no evidence of atherosclerotic disease, the diagnosis of primary AAO was excluded because of the evidence of a coarse thrombus in the left ventricle.

In conclusion acute aortic occlusion is a rare but life-threatening condition; lower limb ischemia is the most common clinical presentation, but a different presentation is possible depending on level of thrombosis. between these there is sudden paraplegia. MDCT angiography should be performed without delay for diagnosis, to distinguish primary from secondary AAO and to identify which branches of the aorta are affected.

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