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Sudden Paraplegia Revealed Acute Aortic Dissection Type A with Adamkiewicz Artery's Involvement: A Congolese Case Report

Thibaut Naïbe Gankama[1,2*], Jospin Karel Makani Bassakouahou[2], Eric Gibrel Kimbally Kaky[1,2], Rog Paterne Bakekolo[1,2], Solange Flore Mongo Ngamami[1,2], Christian Michel Kouala Landa[1,2], Kivié Ngolo Letomo[2], Bijou Moualengue[2], Franck Bienvenu Ekoba Othendet[2], Ruddy Junior Taty[2], Rud Ampat Effonouan[2], Méo Stéphane Ikama[1,2]

[1]Cardiology Department, Faculty of Health Sciences, Marien Ngouabi University, Brazzaville, Republic of Congo
[2]Cardiology Department, Teaching Hospital of Brazzaville, Brazzaville, Republic of Congo
Email: *thibautgankama@gmail.com

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Abstract

Aortic dissection with neurological symptoms is a usual presentation and rare condition. It poses a major diagnostic challenge due to the risk of misdiagnosis and worse prognosis (functional or vital). We present the clinical case of a 53-year-old female patient admitted for chest pain and sudden onset paraplegia in cardiology department. Her medical history included hypertension and diabetes mellitus. Physical examination showed paraplegia associated to anesthesia of legs and painless back. BP: 120/60 mmHG. Cariopulmonary auscultation was normal. Pulses presents. Chest X-ray revealed mediastinal widening. Cardiac ultrasound revealed an intimal tear of aortic dissection and severe aortic insufficiency. Chest CT angiography showed a Stanford type A aortic dissection with left renal infarction. Spinal cord MRI was not available. In the absence of a cardiac surgery center, the patient died one month later from trophic and infectious complications. Paraplegia leads to aortic dissection when there is no pain in the back and occurs suddenly. Hypertension is the common cause in subafrica. Early diagnosis and management prevent death.

Keywords

Aortic Dissection, Paraplegia, Spinal Cord, Malperfusion, Ischeamia

1. Background

Aortic dissection is a longitudinal split in the wall of the aorta between the inner third and the two outer thirds of the media, separating it into two channels or

lumens by the intimal flap: the true lumen and the false lumen [1]. It can extend to a visceral artery and cause ischaemia: this is the malperfusion syndrome. Aortic dissection is a cardiovascular medical and surgical emergency requiring immediate treatment. It is a rare but serious condition with a high mortality rate. Its severity is underpinned by the risk of tamponade, severe acute aortic insufficiency and malperfusion syndrome. In Western countries, its incidence is 3 to 5 per 100,000 inhabitants per year (based on the international registry of acute dissection aortic) [1] [2]. Distal malperfusion occurs in 25% - 31% cases of acute type A aortic dissection and paraplegia into 2% to 5% [3].

In Africa, data is sparse, with low numbers ranging from 4 to 19 cases over a decade [4]-[6]. Some cases of aortic dissection with paraplegia are also described [7] [8]. The causes are dominated by hypertension [2].

In Congo, a few cases have been described [9]-[11]. However, there are no studies available on malperfusion syndrome complicating aortic dissection in Congo. Hence the interest of this work. We report a case of aortic dissection with involvement of the Adamkiewicz artery or intercostal artery (the artery responsible for spinal cord vascularisation in the lower dorsal, lumbar and sacral regions).

The aim of this study was to highlight its extreme severity, the clinical polymorphism that can be seen in a clinical picture of visceral ischaemia during aortic dissection, and to emphasise the limitations of surgical management in Congo.

2. Patient and Observation

2.1. Reason

A 53-year-old female patient admitted to the cardiology department of Brazzaville Teaching Hospital on July, 2017 for an aortic dissection, Stanford type A, complicated by spinal, renal and coronary ischaemia.

2.2. Medical History

This is a 53-year-old diabetic patient who was hospitalised in the metabolic diseases department of Brazzaville Teaching Hospital for decompensation of her diabetes mellitus(ketoacidosi). Three days after admission, she presented with sudden, intense, prolonged retrosternal chest pain radiating to the back, accompanied by syncope and functional impairment of the lower limbs. Due to an abnormal ECG, the patient was transferred to the cardiology department.

She has had hypertension since 2012 and is treated with amlodipine. She has type 2 diabetes and is treated with insulin. She does not consume alcohol or tobacco.

2.3. Clinical Examination

Patient conscious, dyspnoeic

T = 37˚C, slight pallor of the mucous membranes, weight = 93 kg, height = 1.67 m, BMI = 33.34 kg/m^2 (moderate obesity). Hand span: 1.60 m. There was no Marfan-type (slender) body type.

Karnofsky index of 60 (dependent patient).

2.4. Physical Examination

There are no signs of heart failure. Heart sounds are regular with a heart rate of 96 bpm. Diastolic murmur, soft and aspiratory, parasternal 3/6e of aortic insufficiency. Lungs are clear. Peripheral pulses are present and symmetrical in all four limbs. BP = mmHg. There was no hyperpulsatility of the pulses. There were no signs of cuff, Musset's sign, pectus excavatum or prominent thumbs.

Muscle strength is rated 5/5 in the thoracic limbs and 1/5 in the pelvic limbs (motor deficience), with loss of osteotendinous reflexes and hypotonia in the pelvic limbs (flaccid paraplegia). There is also descending anaesthesia from L5 to the pelvic limbs (toes) associated with saddle anaesthesia with preserved pulses.

Front chest X-ray: Mild cardiomegaly, CTR at 56%, convex lower left arch with, mediastinal widening (wide and unfolded aorta), normal-appearing lungs, free cul-de-sacs (Figure 1).

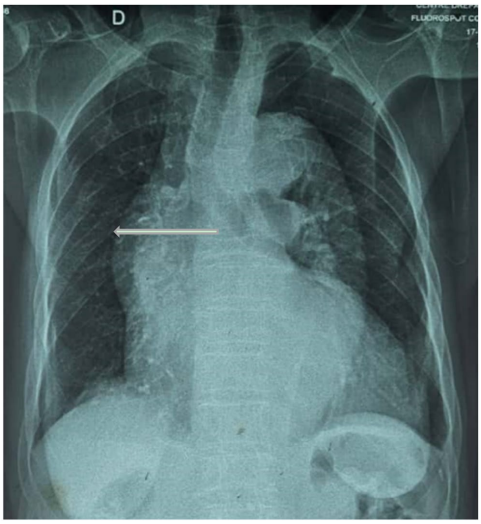

Figure 1. Chest X-Ray showing mediastinal widening.

ECG: Sinus rhythm, HR: 92 bpm, extensive anterior ST elevation (Figure 2).

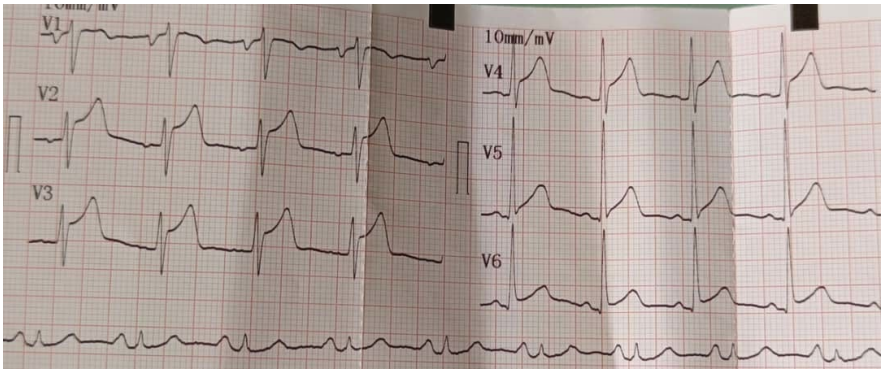

Figure 2. ECG showing anterior segment ST elevation.

Cardiac Ultrasound:

Severe acute aortic insufficiency with Stanford type A aortic dissection. LVEF: 74%; ascending aorta diameter:43 mm; heart chambers were not dilated. Normal wall thickness. Normal mitral profile, PAPS: 33 mmHG, IVC not dilated, minimal pericardial effusion.

Thoracoabdominal CT angiography: Stanford type A aortic dissection with involvement of the left renal artery causing left renal infarction (Figure 3).

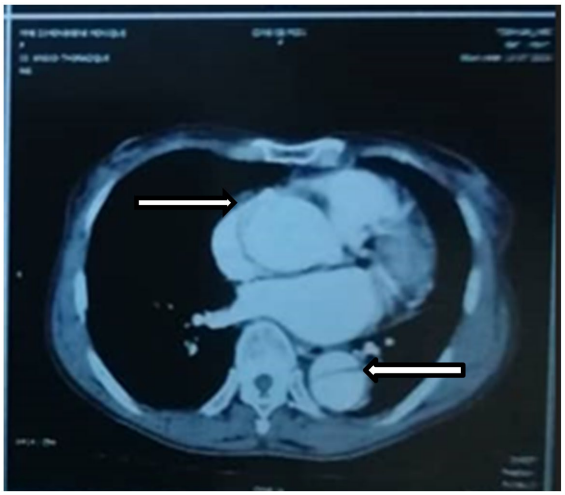

Figure 3. CT showing true and false lumens of aortic dissection.

Spinal cord MRI (Magnetic Resonance Imagery) was not available.

2.5. Biology

Complete blood count: WC at 8500 cells, haemoglobin at 9.6 g/dl, blood glucose at 2.6 g/l, creatinine at 53 mg/l. Normal troponins, total cholesterol 1.19 g/l, LDL 0.76 g/l, HDL 0.26 g/l, triglycerides 0.88 g/l.

Diagnosis: Aortic dissection, Stanford type A with severe aortic insufficiency complicated by medullary, renal ischeamia in an obese diabetic patient with hypertension.

Treatment: Nebivolol 2.5 mg/day. Amlodipine 5 mg/day. Rapid-acting insulin 14 IU in the morning and at lunchtime, and long-acting insulin 16 IU in the evening.

2.6. Outcome

The patient's condition was marked by persistent pain, paraplegia and the onset of trophic disorders (buttock bedsores: a consequence of paraplegia) leaded skin infection. Atriaf fibrillation tachycardia occurred also. In beginning, her haemodynamic status was stable with preserved diuresis. The pulses are preserved. The treatment was supplemented by nursing care and functional motor rehabilitation sessions and antibiotics. Bedsores appeared later.

The patient was transferred one month later to Morocco for aortic dissection

surgery. She succumbed to septic shock originating in the skin (buttock bedsores), with surgery being postponed due to infection.

3. Discussion

This case perfectly illustrates the polymorphism of clinical presentation. Paraplegia and anaesthesia, often of neurological or rheumatological origin, were in this rare case of vascular origin (spinal cord ischaemia due to damage to the Adamkiewicz artery). Adamkiewicz artery is an intercostal artery. This ischaemia was caused either by the extension of the dissection tearing the wall of this artery, or detachment(separating) of artery's origin tfrom the true lumen or intimal flap prolapse into vessel origin or by the obstruction of the ostia of this vessel by the false lumen of a wide aortic dissection or by the obstruction caused by partial thrombosis of a false lumen. All of which mechanisms lead to low arterial flow [3]. In this case ischeamia was certainly due to separating of artery's origin from true lumen.

Unlike rheumatological damage, which is painful, paraplegia of vascular origin is painless (without lumbar) because the mechanism is ischaemia of the lower spinal cord, *i.e.* the nerves that innervate the pelvic limbs and the small pelvis.

Several authors in both the West Countries and sub-Saharan Africa have reported cases of aortic dissection complicated by paraplegia [3] [6]-[8] [12]-[14].

Renal failure secondary to left renal infarction was due to prolonged ischaemia in the left renal artery caused by low flow or renal hypoperfusion. Renal failure is one of the most common types of malperfusion during dissection due to its declive position on the aorta. Some authors have referred to this [10] [15].

The factors contributing to dissection may be constitutional (elastic tissue disease such as Marfan syndrome) or acquired, including hypertension, diabetes mellitus, smoking and aneurysms, which weaken the aortic wall [1] [2] [15]. In our case, the patient was hypertensive and diabetic.

She was a relatively young adult. In Africa or blacks, hypertension tends to appear early, is severe and frequently leads to complications. This contrasts with the West Countries, where the average age of onset is over 60 [2] [15]. Hypertension remains the main aetiological factor, a fact highlighted by most African studies [4]-[6] [9]-[11] [15].

Cardiac Doppler ultrasound in expert hands allows diagnosis to be made by revealing the intimal flap with the existence of a true, a false channel and aortic insufficiency. This diagnosis is confirmed by chest CT angiography. Spinal cord MRI show a spinal cord ischeamia for linking paraplegia to aortic dissection. In sub-Saharan Africa, these imaging techniques are becoming increasingly available. Their cost limits patient access to these techniques in countries without universal health insurance. In our case, the CT angiography was obtained on the third day of hospitalisation. There was not spinal MRI.

The severity and complexity of the treatment, unlike in classic cases of aortic dissection, lay in the fact that it was life-threatening due to the risk of imminent

aortic rupture, tamponade, worsening aortic insufficiency and the risk of secondary infection of pressure sores, but also the functional risk due to functional impotence following paralysis and, above all, spinal cord ischaemia, which exposes the patient to lower spinal cord infarction.

While awaiting surgery, medical treatment consists of reducing the shock wave generated by the blood flow on the affected aortic wall through the use of beta-blockers and 10 mg of amlodipine per day. The use of beta-blockers is widely described in the literature [1]-[5] [9]-[14].

The lack of surgical treatment after one month of admission reflects the difficulty of providing care in our environment (Congo), which lacks a functional cardiovascular surgery unit despite the presence of cardiovascular surgeons and a catheterisation or angiology room for endovascular treatment. Medical evacuation, the only emergency solution available after one month, illustrates the sluggishness of the Congolese healthcare system in managing emergencies due to the lack of universal health insurance. In Stanford type A dissection, in-hospital mortality remains high in the absence of emergency surgery. This is the bitter conclusion that emerges from all the African literature on aortic dissectioni [4]-[6] [10] [11].

4. Conclusion

The diagnosis of aortic dissection has become possible in sub-Saharan Africa thanks to the use of non-invasive medical imaging. The association with paraplegia reflects the clinical complexity that can mislead clinicians. It's malperfusion syndrome whom is an emergency needing a multidisplinary and intensive management. Type A dissection is often fatal without surgery. Endovascular repair is helpful to cure ischeamia. The fight against arterial hypertension must be carried out.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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