Case Report

ISSN 2639-8486

Cardiology & Vascular Research

Resistant HTA Revealing Aortic Dissection: A Case Report from the Cardiology Department of the Kara University Hospital

Mario Abalo BAKAI[1]*, Tchaa TCHEROU[1], Eyram Makafui Yoan Yawo Amekoudi[2], Lihanimpo DJALOGUE[3], Sylvain MBAIDEDJIM[4], Nafi OUEDRAOGO[5] and Machihude PIO[1]

[1]*Cardiology Department, CHU Kara, Togo.*

[2]*Néphrology Department, CHU Kara, Togo.*

[3]*Internal Medecine Department, CHU Kara, Togo.*

[4]*Cardiologie Department, CHU Renaissance Ndjamena, Tchad.*

[5]*Physiology Laboratory CHU INSSA, Burkina Faso.*

***Correspondence:**
BAKAI Abalo Mario, Cardiology Department, CHU Kara, Togo.

Received: 15 Mar 2025; **Accepted:** 25 Apr2025; **Published:** 06 May 2025

Citation: Mario Abalo BAKAI, Tchaa TCHEROU, Eyram Makafui Yoan Yawo Amekoudi, et al. Resistant HTA Revealing Aortic Dissection: A Case Report from the Cardiology Department of the Kara University Hospital. Cardiol Vasc Res. 2025; 9(2): 1-3.

ABSTRACT

Introduction: *Arterial hypertension of the subject is a particular entity, as it requires a meticulous search for the etiology.*

Objective: To report the case of an aortic dissection discovered fortuitously during the etiological search for arterial hypertension in a 36-year-old patient.

Clinical Case: *Mrs. A., 36 years old, non-smoker, non-alcoholic, followed in nephrology for hypertension and CKD for two years on perindopril 10mg, amlodipine 10mg, indapamide 2.5mg, aldomet 2g, nebivolol 5mg daily treatment regularly taken for more than one (1) year. She had no other cardiovascular history. She had been hospitalized for elevated blood pressure. On examination, blood pressure was 234/145 mmHg, pulse 90bpm, and room air saturation 99%. General condition was good. Cardiac and pulmonary auscultation was normal. ECG showed left ventricular hypertrophy. Creatinemia was 60 mg/l, and CBC showed severe microcytic hypochromic anemia. Transthoracic Doppler echocardiography revealed severe pulmonary hypertension, no visualized intracavitary thrombus, and good biventricular systolic function. DE BAKEY type IIIb aortic dissection. Doppler ultrasonography of the renal arteries had revealed an abdominal aortic aneurysm (3.35cm) post-dissection. The left renal artery (occluded after the ostium), with extension of the dissection to the iliac arteries. Renal ultrasonography revealed small, dedifferentiated stage III kidneys suggestive of chronic renal failure. Thoracic-abdominal-pelvic angioscanner showed CT evidence of a STANDFORD type A aortic dissection extending from the aortic arch to the subrenal abdominal aorta, with a pelvic fluid layer.*

Conclusion: *Etiological research remains an essential phase in the management of hypertension in young subjects.*

Keywords

Hypertension, Young subjects, Aortic dissection.

Introduction

Arterial hypertension (AH) is the most common cardiovascular risk factor. Its prevalence in sub-Saharan Africa ranges from 16 to 40% [1]; in Togo, its prevalence was estimated at 30.5% in 2019 [2]. Depending on the mode of evolution, several nosological entities exist, including resistant hypertension, defined by the French hypertension society as uncontrolled hypertension in consultation despite a therapeutic strategy comprising adapted hygienic-dietary rules and anti-hypertensive triple therapy, for at least 4 weeks, at optimal dose, including a thiazide diuretic [3]. Depending on the etiology, a distinction is made between secondary hypertension,

which accounts for 10% of all cases [4]. The particularity of the latter lies in its complete recovery once the cause has been identified and treated. Among secondary hypertensives, we distinguish renovascular hypertensives, whether due to renal parenchymal or renal artery damage. The evolution of hypertension is fraught with complications, one of the most dreaded being aortic dissection, which is an absolute hypertensive medical emergency.

This dissection may reach the renal arteries and also cause secondary hypertension [5]. We report here a case of resistant hypertension revealing an aortic dissection.

Patient and Observation

Mrs. A. B., aged 36, followed in nephrology for chronic renal failure and arterial hypertension for two years on maximum-dose quadritherapy consisting of : Perindopril 10mg, Amlodipine 10mg, Indapamide 2.5mg, Aldomet 2g, Nebivolol 5mg daily, and with no other pathological cardiovascular history, was referred for better management of her blood pressure.

Clinical

Blood pressure in the right arm was 234/145 mmHg and in the left arm 230/140 mmHg, pulse 90 beats/min, no pulse asymmetry, ambient air saturation 99%, cardiac and pulmonary auscultation normal. General condition was unchanged, with palpebral pallor predominating.

Laboratory Investigations

- **Electrocardiogram**

Regular sinus rhythm at 85 beats/min, normal QRS axis, left ventricular hypertrophy.

- **Transthoracic Doppler echocardiography**

- dilatation of the heart chambers
- grade II diastolic dysfunction
- severe pulmonary hypertension
- absence of visualized intracavitary thrombus
- good biventricular systolic function
- DE BAKEY type IIIb aortic dissection

Doppler ultrasound of the renal arteries

A post-dissection abdominal aortic aneurysm (3.35cm) involving the celiac trunk, superior mesenteric artery and left renal artery (occluded after the ostium), with extension of the dissection to the iliac arteries (Figure 1 and 2). Renal ultrasound revealed small, dedifferentiated stage III kidneys suggestive of chronic renal failure.

Thoracic-abdominal-pelvic angioscanner

STANDFORD type A aortic dissection extending from the aortic arch to the subrenal abdominal aorta, with a pelvic fluid layer elsewhere (Figure 3).

- **Biology:** creatinemia was 60 mg/l with a calculated clearance of 11.62 m/min. A week later, creatinine was checked and found to be 75 mg/l with a clearance of 8.98 ml/min; the CBC showed severe microcytic hypochromic anemia associated with isolated lymphopenia.

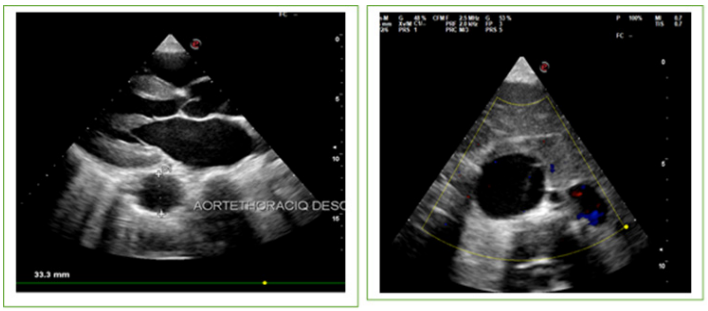

Figure 1: Aneurysm of the abdominal aorta (a) Cardiac ultrasound, parasternal long-axis section; b) Abdominal aorta, subcostal section).

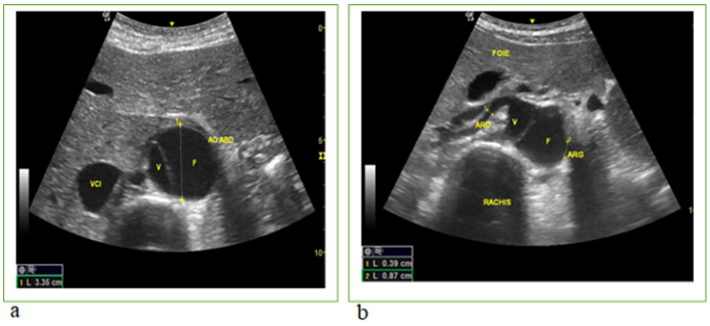

Figure 2: Echodoppler of the renal arteries. (a) abdominal aortic dissection with distinction between false and true channels. (b) with false channel taking up the left renal artery and true channel taking up the right renal artery with static stenosis identified by Doppler).

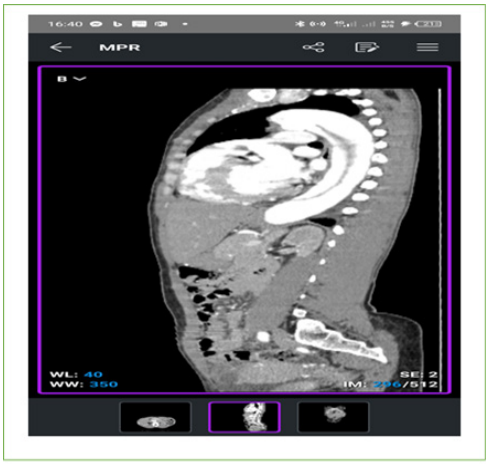

Figure 3: Thoracoabdomino-pelvic angioscan showing the extent of aortic dissection .

Discussion

The overriding question in this clinical picture was the occurrence of an aortic dissection that had gone unnoticed, the diagnosis having been established only retrospectively after the onset of AH resistance. This contrasts with the noisy chest pain accompanying aortic dissection. It has also been proven that in 70% of cases,

hypertension is the predisposing factor in aortic dissection, as it weakens the vascular endothelium in areas of turbulence, thus representing the mechanical and hemodynamic factor. It is also important to remember the classification of acute, sub-acute and chronic dissection [6]. In the case of acute patients, after the first few days, it can be assumed that the most serious patients have died, and that the only patients to survive are those with none of the serious complications of the accident. As a result, patients who survive are generally in good clinical condition, and surgery is no longer indicated in the most urgent cases. In the course of aortic dissection, especially of type B, the renal arteries may be involved.

Extension of aortic dissection to the renal arteries can lead to significant stenosis, resulting in renovascular hypertension and ischemic nephropathy [7]. The incidence of AD extending to the renal arteries varies from 12% to 60%, depending on the study [8]. Aortic dissection with extension to the renal arteries leads to renal malperfusion. The pathogenesis of malperfusion may be dynamic, due to systolic expansion of false channel movements, or static, due to permanent obstruction by the false channel, or both. In both cases, angioscanner coupled with vascular echodoppler can be used to assess the lesion and determine the pathophysiological mechanism. In our case, the two mechanisms are associated, leading to renal failure [9].

In terms of management, medical treatment of aortic dissection remains the preferred option in the acute phase. Initial surgical management has been virtually abandoned, given its high morbidity and mortality. Endoluminal treatment is increasingly being proposed, based on three techniques: for dynamic malperfusion, closure of the aortic dissection portal by covered endoprosthesis or fenestration (perforation of the intimal flap close to the malperfusion) to reduce pressure in the false channel; for static malperfusion, stenting of the ostium of the renal artery [10]. These treatments improve perfusion of the affected kidney, facilitate blood pressure control and reduce the risk of renal failure.

In our patient with a chronic type IIB aortic dissection, given the inadequate technical platform, medical treatment and ongoing discussion for management probably involved stenting of the ostium of the renal artery in a country with a substantial technical platform.

Conclusion

Despite being an absolute medical and surgical emergency in the acute phase, aortic dissection, with its dismal prognosis, can be discovered spontaneously in the chronic phase, revealed by the chronic complications it can engender. These complications include renal failure if the dissection reaches the renal arteries. Renal damage may also be responsible for resistant hypertension. In the case of resistant hypertension in the young, a secondary etiology should always be sought.

References

1. Houehanou C, Amidou S, Preux PM, et al. Hypertension artérielle (HTA) en Afrique subsaharienne. JMV-J Médecine Vasc Mars. 2018; 43: 87.
2. https://sante.gouv.tg/politique-nationale-de-sante-au-togo/.
3. https://www.larevuedupraticien.fr/article/hypertension-arterielle-resistante
4. Vesin C, Nana A, Manzo-Silberman S, et al. Hypertensions artérielles secondaires d'origine surrénalienne : syndromes de Conn, de Cushing et autres entités. EMC - Cardiol janv. 2009; 4: 1-12.
5. Sory BI, Yaya BEH, Abdoulaye C, et al. Dissection de l´aorte abdominale révélatrice de la maladie de Takayasu: à propos d´un cas en Guinée. Pan Afr Med J. 2020; 37: 34.
6. Bachet J. Dissections aiguës de l'aorte : physiopathologie et diagnostic. EMC - Chir. 1 août. 2004; 1: 301-323.
7. Vautrin E, Thony F, Chavanon O, et al. Dissection aortique étendue aux artères rénales: intérêt de la volumétrie rénale après angioplastie.Annales de cardiologie et d'angéiologie. 2012 ; 61 : 203-208.
8. Miller DC. The continuing dilemma concerning medical versus surgical management for patients with acute type B dissections. Semin Thorac Cardiovasc Surg. 1993; 5: 33-46.
9. Favre JP, Chavent B, Albertini JN, et al. Dissections artérielles rénales et conséquences rénales des dissections aortiques. JMV-Journal de Médecine Vasculaire. 2017; 42: 89-90.
10. Fabre O, Guesnier L, Renaut C, et al. Prise en charge actuelle des dissections aortiques de type A. Traitement chirurgical et traitement des syndromes de malperfusion. Ann Cardiol Angéiologie. 2005; 54: 332-338.