

# Double Compression of Left Atrium and Pulmonary Artery by a Huge Descending Aortic Aneurysm with Intramural Hematoma Mimicking Pulmonary Embolism: A Case Report

Djibril Marie BA<sup>1</sup>, Aminata Diack<sup>2</sup>, Alain Affangla<sup>1</sup>, Khadidiatou Dia<sup>3</sup>,  
Mouhamed Cherif Mboup<sup>3</sup>, Mouhamed Leye<sup>1</sup>, Abdoul Kane<sup>4</sup>

<sup>1</sup>Unit of Training and Research in Medical Sciences, University of Thies, Thies, Senegal

<sup>2</sup>Department of Radiology, Principal Hospital of Dakar, Dakar, Senegal

<sup>3</sup>Department of Cardiology, Principal Hospital of Dakar, Dakar, Senegal

<sup>4</sup>Department of Cardiology, Cheikh Anta Diop University, Dakar, Senegal

Email: gaby.11ba@yahoo.fr

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## Abstract

Hemodynamic instability secondary to left atrial (LA) compression by an aortic aneurysm is a rare entity. We report the case of a 43-year old woman with no previous diagnosis of congestive heart failure who was admitted for an initial diagnosis of pulmonary embolism (PE) based on shortness of breath, hypotension and D-Dimers elevation. The electrocardiogram and blood counts were within normal limits. The chest X-ray revealed widening of the mediastinum. Transthoracic echocardiography demonstrated LA compression by a large descending thoracic aortic aneurysm. Left and right ventricle systolic functions were preserved. Chest angiography showed LA and left pulmonary artery (LPA) compression by a descending aortic aneurysm and an intramural hematoma with no evidence of PE evidence. Emergency surgery could not be done because of her financial status. She was treated medically and was discharged 1 week later with significant improvement. However she remained hypotensive.

## Keywords

Aortic Aneurysm, Left Atrium Compression, Pulmonary Artery Compression, Intramural Hematoma, Echocardiography

## 1. Introduction

Thoracic Aortic aneurysm (TAA) may present catastrophically with dissection

or rupture or chronically with symptoms from compression of the surrounding structures. Extrinsic compression of the left atrium and the pulmonary vessels is an uncommon cause of hemodynamic compromise and may be secondary to the involvement of mediastinal structures, including TAA. We report a rare case of huge descending aortic aneurysm that caused dyspnea, palpitations and hypotension from compression of left pulmonary artery and left atrium that led to an initial diagnosis of pulmonary embolism.

## 2. Case Report

A 43-year-old woman was referred to our institution for initial diagnosis of pulmonary embolism based on shortness of breath, hypotension and D-Dimers elevation. The patient's electrocardiography (ECG) and blood counts were within normal limits.

Her medical history revealed a shortness of breath and palpitations that worsened within the past days. She had no previous history of congestive heart failure or cardiovascular risk factors.

The degree of dyspnea on admission corresponded to New York Heart Association grade II. Her blood pressure was 90/60 mmHg and she had regular pulses of 110 beats/min. No precordial murmurs or bruits were audible. There were inspiratory crackles in the basal segments of left lung.

The electrocardiogram showed a sinus regular rhythm. The chest X-ray (**Figure 1**) revealed widening of the mediastinum. Transthoracic echocardiography (TTE) in the apical four chamber view demonstrated left atrial compression by a large descending TAA (**Figure 2(a)**). Left atrium was of 7.2 cm<sup>2</sup> and right atrium of 11.8 cm<sup>2</sup> (**Figure 2(b)**). Left and right ventricle systolic functions were preserved with an estimated systolic pulmonary artery pressure of 25 mmHg.

Chest angiography was performed and revealed a giant 9.3 × 9.8 × 22.9-cm TAA with intramural hematoma (**Figure 3(a)** and **Figure 3(b)**), which compressed the left pulmonary artery and left atrium without any evidence of pulmonary embolism (**Figure 4(a)** and **Figure 4(b)**). No intimal flap was seen in any part of the thoracic aorta.

Surgery was planned, but the patient could not support surgery because of financial status.

She was treated medically by beta blockers and low dose of diuretics and she was discharged after one week. Outcome after 6 months follow-up was favorable with regression of dyspnea but she remained hypotensive.

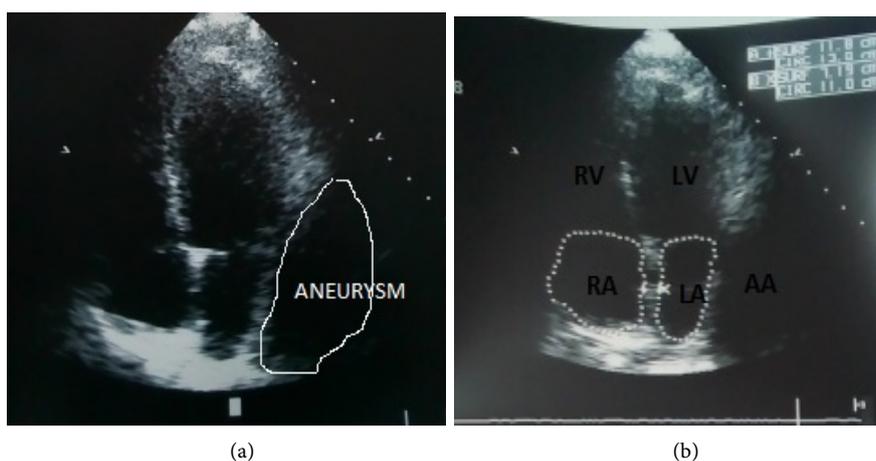
## 3. Discussion

Aortic intramural hematoma was first described by Krukenberg in 1920 as an aortic dissection without intimal tear [1].

The natural course of descending aortic intramural hematoma is thought to be less malignant than that of aortic dissections [2], which may be in part due to the lower frequency of its association with malperfusion syndromes, and there is



**Figure 1.** A chest radiograph showing widening of the mediastinum.

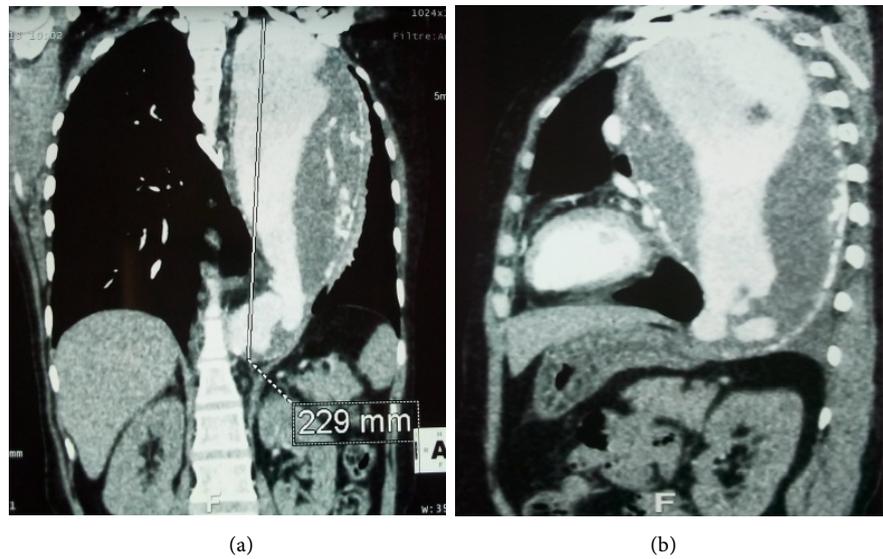


**Figure 2.** Transthoracic echocardiogram in the apical four chamber view demonstrating aneurysmal dilatation of the descending aorta compressing left atrium. LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle; AA: aneurysmal aorta.

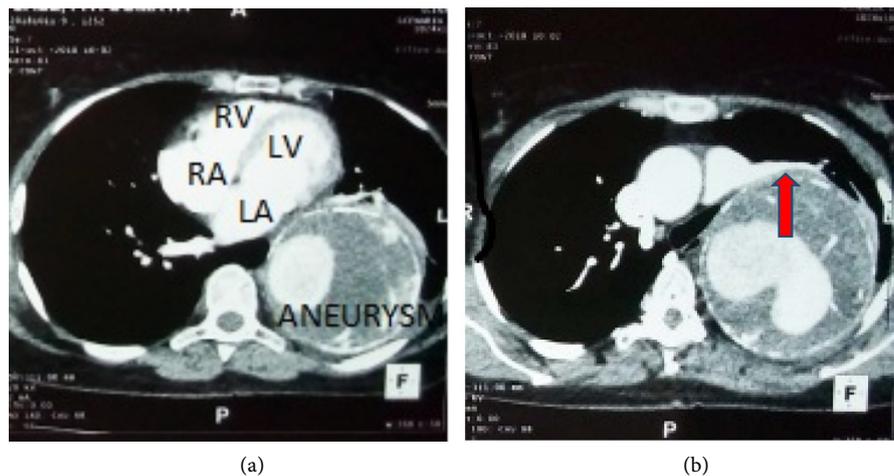
consensus on medical treatment with follow-up for its evolution to aortic dissection or rupture [2] [3]. Although late progression to aneurysm or frank dissection may occur, complete resolution of hematoma has been observed in 50% - 80% of cases [2] [3].

Since the descending aorta have parts located very close to the left atrium, occurrence of both dissecting and non dissecting aneurysms compressing left atrium is a well-defined, but uncommon entity with few reports in the literature [4] [5].

Left atrium compression is classified into three categories based on the severity of anatomical deformation and its hemodynamic consequences: proximity (a contiguous or adjacent structure without chamber deformation), encroachment (distortion of normal cardiovascular architecture without hemodynamic effect), and compression [6]. Proximity and encroachment are defined as conditions



**Figure 3.** Chest angiography, sagittal view. A large descending aortic aneurysm with in-tamural hematoma.



**Figure 4.** (a) Chest angiography, Horizontal view. A large descending aortic aneurysm is compressing the left atrium (LA). RA (right atrium) RV (right ventricle) LV (left ventricle); (b) Chest angiography, Horizontal view. A large descending aortic aneurysm is compressing the left pulmonary artery (red arrow).

that may not lead to symptoms, whereas compression causes severe inflow obstruction resulting in hemodynamic instability and symptoms such as hypotension, hypoxia, tachypnea, and tachycardia, as in our case [6].

The possible mechanism of congestive heart failure in our case may be reduced left atrium volume due to compression leading to low cardiac output. In addition, as left atrium pressure rises with subsequently elevated pulmonary venous pressure, this may have eventually led to pulmonary edema.

Case of aneurysm compressing pulmonary artery are published but these relatively rare cases were reported in the literature more frequently in the period when syphilis was much more common [7].

Hemodynamic compromise encountered in patients with pulmonary artery compression is related to right ventricular hypertension and right heart failure.

Traditionally, the degree of mechanical obstruction of the pulmonary vascular bed was considered to be the only determinant of the Pulmonary Vascular Resistance increase. Over the years, however, a number of observations have challenged this concept. Firstly, several studies have shown that the correlation between the degree of mechanical obstruction and the hemodynamic manifestations of pulmonary embolism is either absent, or poor at best [8] [9]. Furthermore, bringing about a strictly mechanical obstruction by cross-clamping the left or right pulmonary artery during a surgical procedure, or by unilateral balloon occlusion, causes only a modest rise in pulmonary artery pressure, and almost never results in right-sided heart failure [10] [11] [12], whereas pulmonary embolism with obstruction of 25% of the pulmonary vascular tree can cause marked pulmonary hypertension [13].

In our patient, since there was no right ventricular failure or pulmonary hypertension we think that symptoms were more linked to left atrial compression than pulmonary artery compression.

#### 4. Conclusion

Hemodynamic instability secondary to left atrial compression by an aortic aneurysm is a rare entity. Chest angiography provides an accurate diagnosis. Transthoracic echocardiography is a noninvasive, cheap, and easily applicable method and, thus, should be the first choice in investigating the cause of dyspnea particularly in sub-Saharan countries where most facilities are understaffed.

#### Conflicts of Interest

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

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