

Massive Haemothorax: A Rare Mask for Aortic Rupture

Hémothorax massif : une présentation rare de la rupture aortique

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SUMMARY

Thoracic aortic aneurysm is a potentially fatal condition that can lead to life-threatening complications such as aortic dissection or rupture. We report the case of a 65-year-old man admitted to our cardiology department complaining of rapidly progressive non-radiating chest pain with dyspnea. An initial examination showed a blood pressure of 140/90 mmHg bilaterally, heart rate of 90 beats/min. Cardiac enzymes were slightly elevated, and the electrocardiogram showed sinus tachycardia with inverted T waves in the anterior leads. A chest radiograph on admission demonstrated mediastinal widening and a left-sided pleural effusion. Given the suspicion of acute aortic syndrome, a chest computed tomography was performed, revealing a ruptured aneurysm involving the ascending aorta, the arch, and the descending aorta, resulting in an abundant left hemothorax. The patient was emergently referred for cardiovascular surgery. Death happened during the operation.

KEYWORDS

RÉSUMÉ

L'anévrisme de l'aorte thoracique est une affection potentiellement fatale pouvant entraîner des complications engageant le pronostic vital, telles que la dissection ou la rupture aortique. Nous rapportons le cas d'un homme de 65 ans admis dans notre service de cardiologie pour une douleur thoracique non irradiante à progression rapide associée à une dyspnée. L'examen initial a révélé un état hémodynamique stable. Les enzymes cardiaques étaient discrètement élevées, et l'électrocardiogramme mettait en évidence une tachycardie sinusale avec des ondes T inversées dans les dérivation antérieures. Une radiographie thoracique réalisée à l'admission a montré un élargissement du médiastin ainsi qu'un épanchement pleural gauche. Devant la suspicion d'un syndrome aortique aigu, une tomographie thoracique a été réalisée, révélant un anévrisme rompu touchant l'aorte ascendante, la crosse aortique et l'aorte descendante, responsable d'un hémothorax gauche abondant. Le patient a été orienté en urgence vers le service de chirurgie cardiovasculaire. Le décès est survenu en per-opératoire.

MOTS-CLÉS

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INTRODUCTION

A thoracic aortic aneurysm (TAA) is a progressive disease that may result in various of life-threatening complications. If it is not identified and managed promptly, the outcome is often fatal.[1] The most dreaded complication of aneurysm of descending thoracic aorta is free rupture into the left pleural space, an event that almost invariably leads to death due to uncontrolled hemorrhage. [2] We describe a patient with rupture of a TAA into the left pleural cavity presenting with left pleural hemorrhagic effusion.

CASE REPORT

A 65-year-old male with a history of heavy smoking, severe hypertension, and Alzheimer's disease was admitted to our cardiology department. He presented with rapidly progressive, non-radiating chest pain and dyspnea. Physical examination showed stable hemodynamics with a blood pressure of 140/90 mmHg and a heart rate of 98 beats/min. Laboratory results showed slightly positive cardiac enzymes, and an electrocardiogram (ECG) revealed sinus tachycardia with inverted T waves in the anterior leads.

Admission chest radiography showed an enlargement of the mediastinum and a left pleural effusion. (figure1)



Figure 1. A chest radiograph obtained with a mediastinum enlargement and a left pleural effusion.

Transthoracic echocardiography (TTE) revealed slight dilatation of the proximal aorta without insufficiency, left ventricular hypertrophy, a preserved ejection fraction (LVEF=50%), and an abundant left pleural effusion. Suspicion of an acute aortic syndrome prompted a chest computed tomography (CT) scan. The scan revealed an aneurysm involving the ascending aorta, the arch, and the descending aorta measuring 36.3 mm* 51mm * 58mm. (figure2)

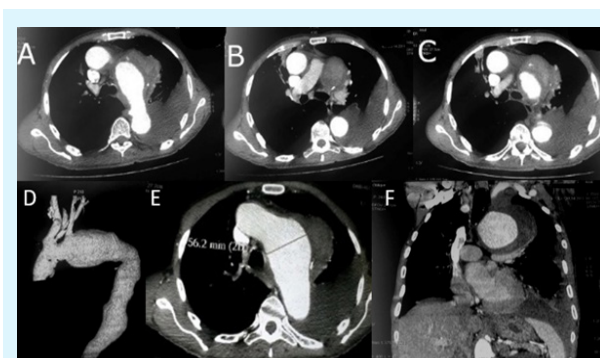


Figure 2.

A A, B, C: CT shows a partially thrombosed aneurysm of the descending aorta, atelectasis band with bronchiectasis of the middle lobe and left pleural effusion.

D: CT showing a fusiform aneurysm of the proximal portion of the descending aorta (segment III), which appears to reach the left subclavian artery

E: CT showing a fusiform aneurysm of the descending thoracic aorta measuring 56.2 mm in diameter, partially thrombosed.

F: CT showing a left pleural effusion with a partially thrombosed aneurysm of the descending thoracic aorta.

Furthermore, wide ulceration on the anterior side of the arch was noted, likely responsible for the rupture into the pleural cavity and the resulting hemothorax. The patient was urgently transferred to the cardiovascular surgery department but died intraoperatively.

DISCUSSION

In this case, the patient presented with a left hemorrhagic pleural effusion as the primary manifestation of TAA rupture. Despite the absence of congestive heart failure or severe chronic obstructive pulmonary disease (COPD), dyspnea was the main complaint. While TTE raised suspicion of the rupture, a CT scan provided the definitive diagnosis.

Determining the true incidence of TAAs is difficult, as 95% of cases remain asymptomatic until a complication occurs. Most are detected incidentally during imaging for unrelated issues. [3]

The pathophysiology involves cystic medial necrosis—a focal degeneration of elastic and muscle tissue within the tunica media. This weakens the aortic wall, leading to dilatation under high intraluminal pressure. Acquired and hereditary conditions can exacerbate the process of medial necrosis.[4]

Indeed, TAAs can be a part of complex genetic syndromes, including Marfan syndrome (most common), Ehlers-Danlos syndrome, Loeys-Dietz syndrome, and Turner syndrome. These clinical syndromes, however, represent only a fraction of the total disease burden. As an increasing number of genetic loci associated with non-syndromic TAAs are identified, clinicians must prioritize the systematic screening of first-degree relatives of affected patients.[3]

Despite being a relatively rare event with an estimated incidence of 5.0 per 100,000 people per year, the risk of rupture can be as high as 50% in patients with a large descending TAA.[1]

Rupture of the thoracic aorta into the pleural cavity, principally due to aortic dissection, has been regularly described in the literature. The ascending thoracic aorta bleeds mainly into the pericardium. For obvious anatomical reasons, the ruptured descending thoracic aorta bleeds preferentially into the left pleural cavity.[5]

Although Most TAA tends to rupture into the left hemithorax, 5 to 10% of these aneurysms may do so on the right side.[5]

A ruptured TAA is a cardiovascular emergency with significantly high mortality rates of 97% to 100% if it is not recognized and managed immediately.[6]

It is only natural that the probability of rupture dictates the management approach to TAAs. One of the crucial factors significantly increasing the risk of rupture is the size of an existing aneurysm.[6] The degree of maximal dilatation, the rate of expansion of the aneurysm, a history of tobacco use, presence of COPD, advanced age, and a history of hypertension are independent risk factors for TAA rupture. Other factors include genetic disorders, advanced age, and female sex.[7]

When the diameter of an aneurysm reaches 5 cm, the likelihood of rupture increases, and this risk goes up considerably as the aneurysm expands. Indeed, the risk of rupture doubles for each additional centimeter of thoracoabdominal aneurysmal growth.[4]

The radiologic assessment of patients with a suspected aortic rupture or dissection is a cornerstone of the diagnostic process. A chest radiograph reveals pathologic findings such as abnormal aortic contour, widened mediastinum, "calcium signs," or pleural effusion in 80 to 85 percent of cases. Currently, a contrast-enhanced CT scan is the method of choice for the diagnosis of patients with suspected dissection or rupture.[8]

With recent advances in molecular imaging, there has been an increased focus on other imaging modalities

for TAA, including Positron emission tomography (PET) and Single photon emission computed tomography (SPECT). Akalihan et al. have shown that the PET scans "light up" in vulnerable aneurysms; this may prove to be a reliable predictor of an impending rupture.[9]

Once aneurysms become symptomatic or painful, however, surgical intervention is usually indicated regardless of aneurysm size.[3]

The management of a ruptured ascending aortic aneurysm, primarily handled within the framework of acute Type A aortic syndromes, represents an emergency surgical priority where immediate intervention is mandatory. Current guidelines emphasize that emergency surgical consultation and evaluation should occur without delay, as surgical repair is the definitive treatment to improve survival rates. [10]

Whenever possible, and if it does not cause significant surgical delay, patients should be transferred to high-volume aortic centres where multidisciplinary teams can manage the complexities of the procedure. Surgical strategies often include an open distal anastomosis to improve long-term outcomes and promote false lumen thrombosis. [10] Furthermore, while aortic valve resuspension is recommended when the leaflets are not pathological, more extensive aortic root replacement with a valved conduit is indicated in cases involving genetic disorders or severe root destruction. For complex scenarios where the dissection extends into the descending aorta, techniques such as the frozen elephant trunk may be considered to reduce late-stage distal complications. Throughout the peri-operative period, strict medical therapy involving blood pressure control and pain relief remains a critical component of stabilization. [10]

According to the guidelines, open repair remains the method of choice in ruptured ascending and aortic arch TAA management, whereas thoracic endovascular aortic repair has become the gold standard for ruptured descending TAA.[1]

CONCLUSION

This case highlights the diagnostic challenges inherent in TAA rupture, emphasizing that hemodynamic stability does not exclude life-threatening pathology. Atypical symptoms, such as dyspnea as a primary complaint, may mislead clinicians and lead to diagnostic delays for what is often considered a «silent killer». Early use of advanced imaging is crucial, particularly when clinical and radiological findings

diverge. This case underscores the need for a high index of suspicion for aortic pathology in patients with unexplained pleural effusion and supports the earlier use of contrast-enhanced CT in atypical presentations.

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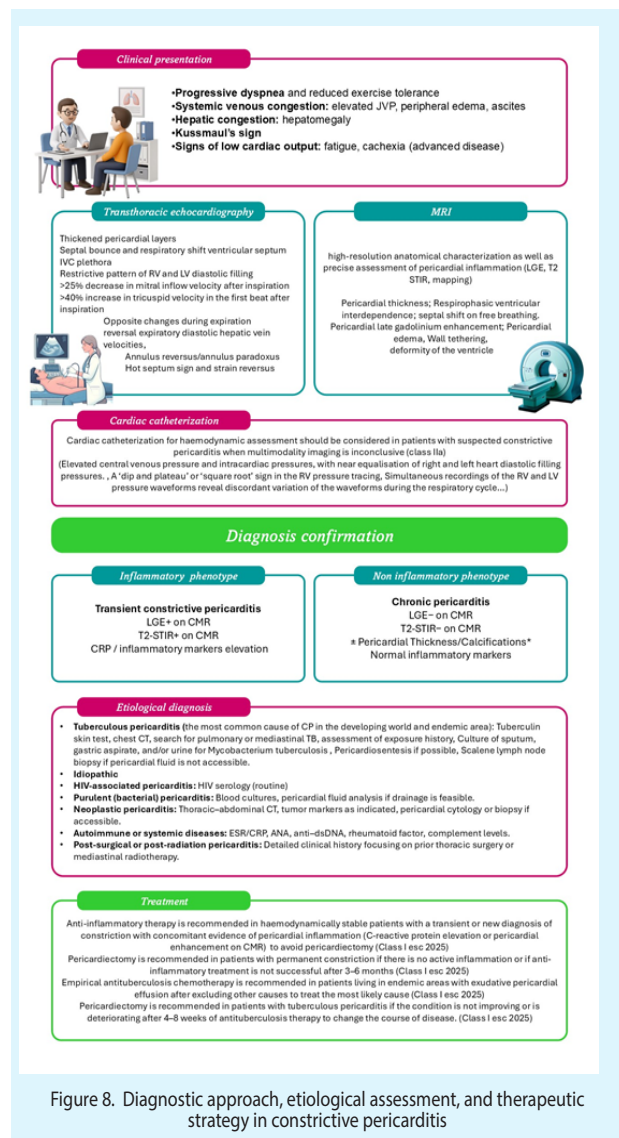


Figure 8. Diagnostic approach, etiological assessment, and therapeutic strategy in constrictive pericarditis