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In-situ Pulmonary Artery Thrombosis: Clinical and Echocardiographic Insights from a Four-patient Case Series

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Context: Thromboembolic disease is the third leading cause of cardiovascular death. In recent years, several studies have suggested the possibility of de novo thrombus formation in the pulmonary arteries without deep vein thrombus (DVT) in the lower extremities. Pulmonary artery thrombosis is a very rare diagnostic entity, typically due to embolic migration from another venous site, but it can also arise from an *In situ* thrombus. The aim of this study is to evaluate the clinical and echocardiographic (TTE) data of in situ pulmonary artery thrombosis (PATIS). This mini-series involves four cases observed in the intensive cardiac care unit (ICCU) at the cardiology department of Ibn Rochd University Hospital in Casablanca between January 1, 2024, and April 1, 2024.

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Case Presentation: We present a mini-series of four patients, consisting of three women and one man, admitted to the ICCU for high-risk pulmonary artery thrombosis. The mean age of the patients was 64 years. Two women had neoplasms, while the man was sedentary due to bilateral lymphedema in the lower limbs. The last patient was a woman on oestro-progestative pills. Altogether, these factors appear to be the main contributors to their thromboembolic events. Clinically, all patients experienced dyspnea and angina. Bedside echocardiography revealed evidence of acute cor pulmonale associated with right ventricular dysfunction and in situ pulmonary artery thrombosis (PATIS). We administered non-fractionated heparin (NFH) immediately after diagnosing PATIS, without waiting for the results of thoracic CT angiography, and none of the patients initially received thrombolysis. The prognosis was poor, with three out of four patients dying from cardiogenic shock during hospitalization. In our opinion, the main contributing factors were comorbidities such as age, neoplastic profile, and uncontrolled diabetes.
Conclusions: While there is much theory regarding the pathophysiology of in situ pulmonary artery thrombus, the primary pathogenic factors are thought to be local pulmonary factors, including dysfunction of pulmonary vascular endothelial cells, hypoxia, and inflammation.

Keywords: In situ pulmonary artery thrombosis (PATIS); thromboembolic disease; cardiovascular mortality; deep vein thrombosis (DVT); case series.

ABBREVIATIONS

DVT : Deep Vein Tthrombus
TTE : Echocardiography
PATIS : Pulmonary Artery Thrombosis in Situ
ICCU : The Intensive Cardiac Care Unit
NFH : Non-Fractional Heparin
CTPA : Computed Tomography Pulmonary Angiography
DOACs : Direct Oral Anticoagulants
PA : Pulmonary Artery
Ao : Aorta
RPA : Right Pulmonary Artery
LPA : Left Pulmonary Artery

1. INTRODUCTION

Thromboembolic disease represents a critical public health issue, ranking as the third leading cause of cardiovascular mortality worldwide. Recent research has increasingly highlighted the potential for de novo thrombus formation within the pulmonary arteries, even in the absence of deep vein thrombosis (DVT) in the lower extremities [1]. This phenomenon challenges traditional understandings of pulmonary artery thrombosis, which is typically associated with emboli originating from distant venous sites.

In this study, we aim to evaluate the clinical and echocardiographic (TTE) characteristics of in situ pulmonary artery thrombosis (PATIS). We present a mini-series involving four cases observed in the intensive cardiac care unit (ICCU) at the cardiology department of Ibn Rochd University Hospital in Casablanca, during the period from January 1, 2024, to April 1, 2024. By analyzing these cases, we hope to provide insight into the clinical presentation, underlying risk factors, and echocardiographic findings associated with PATIS. This study will contribute to a better understanding of this rare condition and its management, ultimately aiming to improve patient outcomes in similar clinical scenarios.

2. CASE PRESENTRATION

We present a mini-series of four patients—three women and one man—who were admitted to the intensive cardiac care unit (ICCU) for high-risk pulmonary artery thrombosis. The average age of our patients was 64 years, with one patient being notably younger at 45 years. Upon evaluating cardiovascular risk factors, we found that two patients were hypertensive, and three had type II diabetes, all of whom were undergoing insulin therapy. Notably, one of these diabetic patients was severely uncontrolled at the time of admission, presenting with diabetic ketoacidosis, which significantly elevated her risk of mortality. Additionally, all the female patients were menopausal.

Among the women, two had a history of neoplasia: the first patient had been treated for breast cancer, which involved a surgical mastectomy followed by five sessions of radiotherapy. The second had gallbladder cancer and was under a surveillance protocol. The male patient was sedentary due to bilateral lymphedema in the lower limbs. The last female patient was taking oestro-progestative pills.

Collectively, these factors are likely contributors to their thromboembolic events.

Clinically, all patients presented with dyspnea and angina, with one also experiencing hemoptysis. Despite normal blood pressure readings, they exhibited tachycardia, with an average heart rate of 119 bpm. Electrocardiographic findings showed sinus rhythm in all cases, with two patients displaying the S1Q3T3 pattern characterized by negative T waves from V1 to V4. Each patient underwent urgent bedside echocardiography, which revealed evidence of acute cor pulmonale associated with right ventricular dysfunction and in situ pulmonary artery thrombosis (PATIS), as illustrated in Figs. 1 to 6.

Biochemically, all patients had elevated troponin and BNP levels. The Pulmonary Embolism Severity Index (PESI) was positive for all patients, with an average score of 4 points. Three patients developed pulmonary infarction, which was confirmed by CT scan and treated with a combination of amoxicillin and clavulanic acid. Notably, none of the patients exhibited signs of lower extremity thrombosis.

Therapeutically, we initiated treatment with non-fractionated heparin (NFH) immediately after diagnosing PATIS, without awaiting the results of thoracic CT angiography. None of the patients received thrombolysis initially. The average length of hospitalization was 10 days, with only one patient being discharged from the intensive care unit. Unfortunately, the prognosis was grim; three out of the four patients—two women and one man—succumbed to cardiogenic shock during their hospital stay. In our analysis, we attribute the poor outcomes primarily to comorbidities, including advanced age, neoplastic conditions, and poorly controlled diabetes.

3. DISCUSSION

Pulmonary artery thrombosis is a complex condition characterized by diverse clinical presentations, which complicates its diagnosis and management [1]. This mini-series emphasizes the importance of integrating clinical assessments with echocardiography, particularly transthoracic echocardiography (TTE), to achieve an accurate diagnosis and guide therapeutic interventions.

Clinical evaluation is essential in the initial assessment of patients suspected of having pulmonary artery thrombosis. A comprehensive medical history, including risk factors such as obesity, immobilization, and recent surgery, provides valuable insights [2]. Presenting symptoms such as dyspnea, pleuritic chest pain, and hemoptysis are common but nonspecific, as they may also occur in other cardiopulmonary conditions. Consequently, a high index of suspicion is crucial to include pulmonary artery thrombosis in the differential diagnosis. Physical examination findings, including tachypnea, elevated jugular venous pressure, and a prominent pulmonic component of the second heart sound, further support the suspicion of a pulmonary embolic event [1,2].

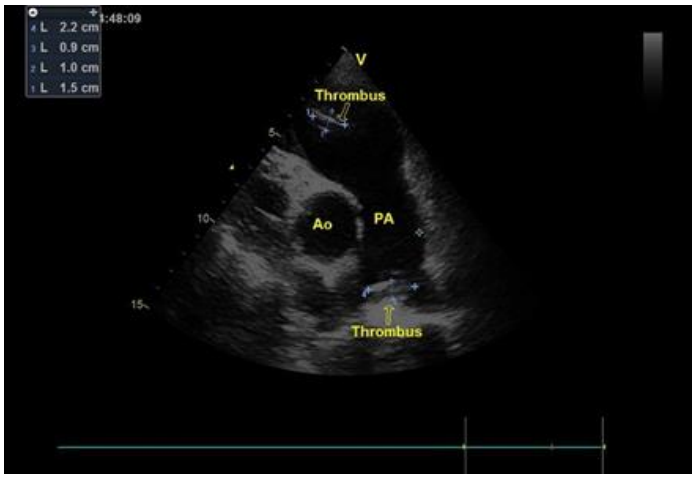

Fig. 1. First patient, PSAX view on echocardiography showing the presence of a thrombus within the right ventricle measuring 15x10mm, another thrombus located at the bifurcation of the pulmonary artery measuring 22x9mm

Legends: PA: Pulmonary Artery, Ao: Aorta

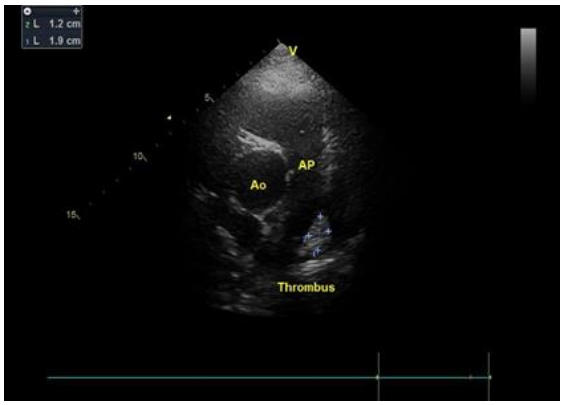

Fig. 2. Second patient, PSAX view on echocardiography showing the presence of a thrombus located at the bifurcation of the pulmonary artery measuring 19x12 mm

Legends PA: Pulmonary Artery, Ao: Aorta

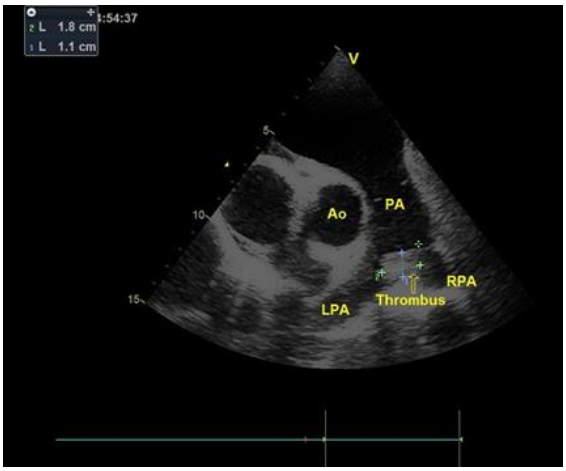

Fig. 3. Second patient, after a few days of anticoagulation, we performed an echocardiography control showing the regression of the pulmonary artery thrombus measuring 18x11mm

Legends: PA: Pulmonary Artery, Ao: Aorta, RPA: Right Pulmonary Artery, LPA: Left Pulmonary Artery

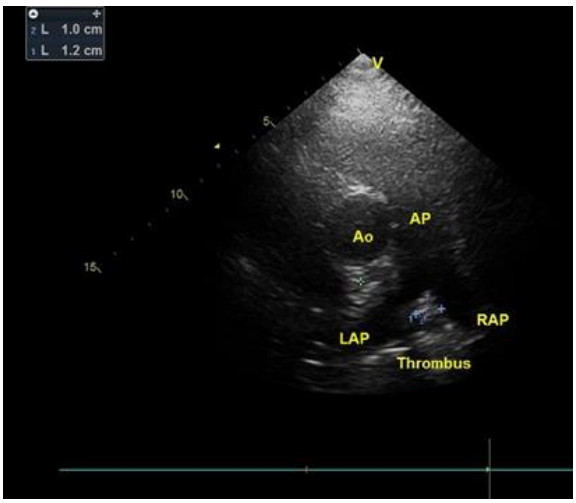

Fig. 4. Second patient, two weeks of anticoagulation, showing significant regression of the pulmonary artery thrombus measuring 10x12mm

Legends: PA: Pulmonary Artery, Ao: Aorta, RPA: Right Pulmonary Artery, LPA: Left Pulmonary Artery

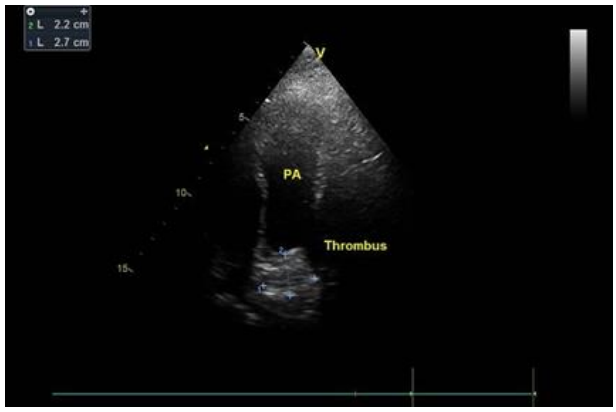

Fig. 5. Fourth patient, PSAX echocardiographic view showing a thrombus at the bifurcation of the pulmonary artery with an occlusive appearance measuring 22x27mm
Legends PA: Pulmonary Artery

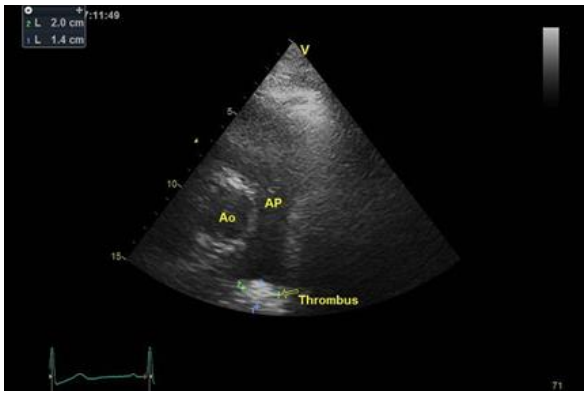

Fig. 6. Fourth patient, PSAX echocardiographic view showing a thrombus at the bifurcation of the pulmonary artery with an occlusive appearance measuring 22x9mm
Legends: PA: Pulmonary Artery, Ao: Aorta

Laboratory investigations play a significant role in the diagnostic process. D-dimer levels, which indicate the breakdown of fibrin clots, can be elevated in various conditions, including pulmonary artery thrombosis [3]. However, elevated D-dimer levels lack specificity and may be influenced by factors such as age, inflammation, and malignancy. Additionally, arterial blood gas analysis and coagulation profiles provide further insights into the patient's respiratory status and coagulation parameters [4].

Echocardiography, especially TTE, is a valuable imaging modality in evaluating pulmonary artery thrombosis. It offers real-time information about cardiac structure and function and allows for direct visualization of thrombi within the pulmonary vasculature [5]. TTE can also identify right ventricular dilation, paradoxical septal wall motion, and the presence of thrombi within the main pulmonary artery and its branches. These findings are typically indicative of acute right ventricular strain due to pulmonary artery thrombosis [5,6].

However, diagnosing pulmonary artery thrombosis can be challenging, particularly in cases involving distal or small vessels. Thrombi in the pulmonary artery may not always be easily visualized with TTE alone [6].

In such cases, additional imaging modalities, such as computed tomography pulmonary angiography (CTPA) or ventilation-perfusion scanning, may be required to complement TTE findings and achieve a definitive diagnosis [7,8].

Management of pulmonary artery thrombosis focuses on prompt therapeutic interventions to prevent further thrombus propagation, alleviate right ventricular strain, and reduce the risk of complications. Anticoagulation therapy, typically initiated with heparin, is followed by a transition

to oral anticoagulants such as warfarin or direct oral anticoagulants (DOACs) [9]. Close monitoring of the patient's clinical status, laboratory values, and imaging studies, including follow-up TTE, is essential for assessing treatment response and guiding further management decisions.

In the management of in-situ pulmonary artery thrombosis, cardiovascular interventional treatments have demonstrated significant efficacy. Several clinical cases have shown favorable outcomes following timely intervention, particularly with catheter-directed thrombolysis and surgical embolectomy [8]. Beyond internal medicine and chronic diseases, pulmonary embolism also presents challenges in obstetrics and gynecology, where pregnancy and the postpartum period are well-documented risk factors [9]. Understanding the pathogenesis of pulmonary embolism is crucial; it is a complex process involving endothelial injury, stasis of blood flow, and a hypercoagulable state [10]. Furthermore, the interplay between diabetes and thrombosis is increasingly recognized. Diabetes contributes to a prothrombotic state through mechanisms such as endothelial dysfunction, platelet hyperreactivity, and increased coagulation factors, further elevating the risk of thrombotic events [11,12].

4. CONCLUSION

Pulmonary artery thrombosis is a rare diagnostic entity, typically resulting from embolic migration from another venous site, but it can also occur due to the formation of an *In situ* thrombus. While there are various theories regarding the pathophysiology of in situ pulmonary artery thrombus, the primary pathogenic factors are thought to include local pulmonary influences such as dysfunction of pulmonary vascular endothelial cells, hypoxia, and inflammation. Notably, radiation therapy is recognized as a significant iatrogenic factor contributing to the development of pulmonary artery thrombus.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

CONSENT

In accordance with international and university standards, written consent from the patient(s) has been obtained and is retained by the author(s).

ETHICAL APPROVAL

In line with international and university standards, written ethical approval has been obtained and is preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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