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Pulmonary Venous Thrombosis with Concomitant Arterial Thrombosis: A Case Report

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ABSTRACT

Background:
Pulmonary venous thrombosis (PVT) is a rare yet clinically important condition with potential for serious cardiopulmonary and systemic complications associated with lung malignancy, thoracic surgery, and post–radiofrequency ablation procedures, although idiopathic cases have also been reported. Its coexistence with arterial thrombosis is exceedingly uncommon and poses significant diagnostic and therapeutic challenges.

Case Presentation:
We report the case of a 55-year-old female presenting with dyspnoea, chest pain and abdominal pain for 8 days. Chest radiography demonstrated reticular opacities in the bilateral lower lung zones with an increased cardiothoracic ratio. Cardiac troponin levels were within normal limits. A 12-lead electrocardiogram revealed sinus tachycardia with right bundle branch block, right axis deviation, and left posterior fascicular block. Contrast-enhanced imaging showed pulmonary venous thrombosis involving the left lower lobe pulmonary vein extending into the left atrium, along with pulmonary arterial thrombosis involving both right and left pulmonary arteries extending into bilateral lower lobe segmental branches. The patient was treated with low molecular weight heparin, resulting in clinical improvement.

Conclusion:
This case underscores the importance of early recognition and prompt management of this rare combination of pulmonary venous and arterial thrombosis to prevent potential complications.

Keywords:
Pulmonary venous thrombosis; Pulmonary arterial thrombosis; Vascular pathology; Case report.

INTRODUCTION

Pulmonary venous thrombosis (PVT) is a rare and often underrecognized clinical entity, with limited data available due to its infrequent occurrence and nonspecific clinical presentation. It has been most commonly described in association with lung malignancy, post-lobectomy states, lung transplantation, and atrial fibrillation, particularly following catheter ablation procedures [1,2]. Despite its rarity, PVT is clinically significant because of its potential to cause serious complications, including pulmonary infarction, pulmonary oedema, and systemic embolization.

Unlike pulmonary arterial thromboembolism, thrombi originating in the pulmonary veins have direct access to the systemic circulation via the left atrium. This unique anatomical pathway

predisposes patients to arterial embolic events such as ischemic stroke, acute limb ischemia, and end-organ infarction [1,3]. The diagnosis of PVT is challenging due to nonspecific symptoms such as dyspnea, chest pain, cough, or hemoptysis, which often overlap with other cardiopulmonary conditions [2]. This case is rare condition because of the coexistence of pulmonary artery and venous thromboembolism.

CASE REPORT

A 55-year-old female presented to the medicine outpatient department with complaints of right-sided chest pain for one month. The pain was insidious in onset, non-exertional, and aggravated by inspiration. She also reported dyspnoea on exertion for eight days. She had a known history of hypertension for the past ten years and was non-compliant with her medications. Based on her presenting symptoms, she was admitted with a provisional diagnosis of acute coronary syndrome for further evaluation.

On clinical examination, the patient was tachycardic with a pulse rate of 150 beats per minute. Her blood pressure was 138/88 mmHg, respiratory rate was 24 breaths per minute, and oxygen saturation was 90% on room air. Respiratory system examination revealed normal bilateral breath sounds. Laboratory investigations showed significantly elevated D-dimer levels (8.03 µg FEU/mL) and elevated NT-proBNP levels (5025 pg/mL), while cardiac troponin levels were within normal limits. A comprehensive thrombophilia workup, including antiphospholipid syndrome, Factor V Leiden mutation, paroxysmal nocturnal haemoglobinuria, and JAK2 mutation, was negative, suggesting an idiopathic etiology.

Contrast-enhanced computed tomography pulmonary angiography revealed pulmonary arterial thrombosis involving both the right and left pulmonary arteries, extending into the bilateral lower lobe segmental branches (Fig 1and 2). In addition, pulmonary venous thrombosis was identified involving the left lower lobe pulmonary vein with extension into the left atrium (Fig. 3 and 4). Transthoracic echocardiography demonstrated mild pulmonary hypertension, mild tricuspid regurgitation, and mild concentric left ventricular hypertrophy, with no evidence of intracardiac thrombus (Fig. 5). Electrocardiography showed sinus tachycardia with right bundle branch block, right axis deviation, and left posterior fascicular block (Fig. 6). Chest radiography revealed reticular opacities in the bilateral lower lung zones, an increased cardiothoracic ratio, and blunting of the right costophrenic angle (Fig. 7).

The patient was initiated on therapeutic anticoagulation with low molecular weight heparin. Supportive management, including oxygen therapy and cardiovascular monitoring, was provided. The patient showed clinical improvement with treatment. Follow-up computed tomographic angiography demonstrated significant reduction in pulmonary arterial thrombus burden (Fig. 8) and resolution of pulmonary venous thrombosis in the left lower pulmonary vein (Fig. 9). Long-term anticoagulation was advised to reduce the risk of recurrence.

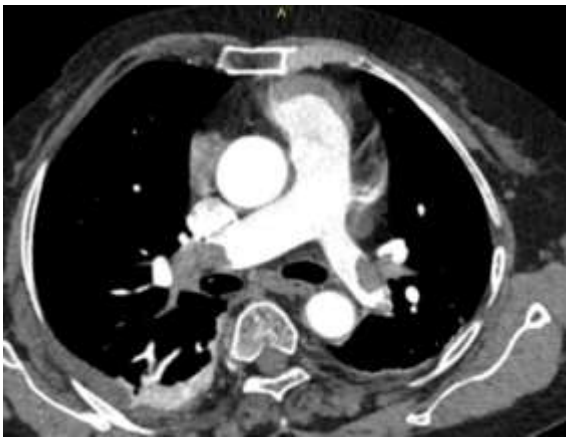

Fig 1. Contrast-enhanced computed tomography pulmonary angiography (CTPA) demonstrates hypodense intraluminal filling defects within the bilateral pulmonary arteries, consistent with pulmonary arterial thrombosis.

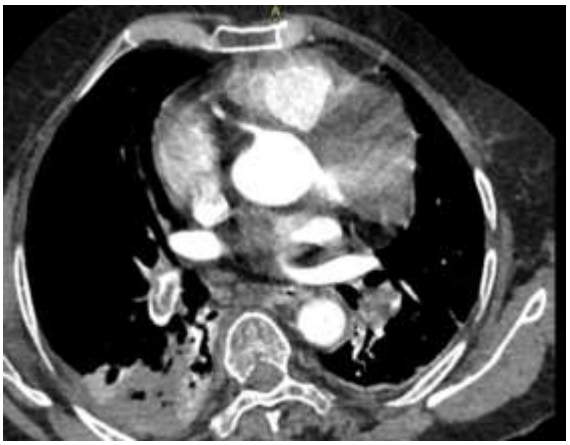

Fig 2. Contrast-enhanced computed tomography pulmonary angiography demonstrates filling defects extending into the lower lobe segmental branches of the pulmonary arteries.

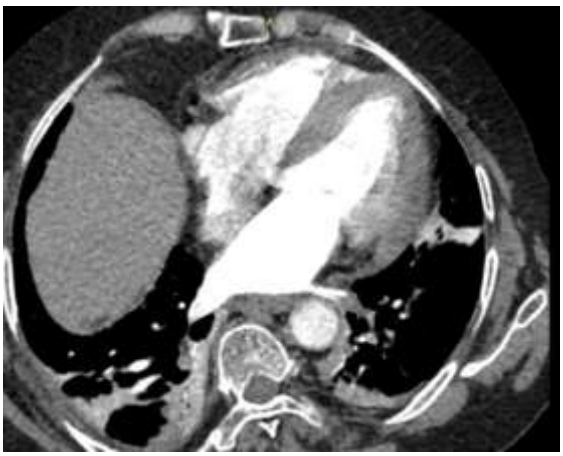

Fig 3. Contrast-enhanced CT image demonstrates an intraluminal hypodense filling defect within the left lower pulmonary vein, suggestive of pulmonary venous thrombosis.

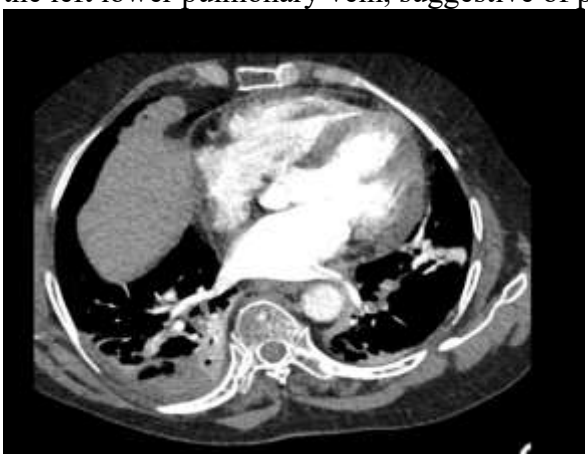

Fig 4. Contrast-enhanced CT image demonstrates intraluminal filling defect within the left lower pulmonary vein with extension into the left atrium.

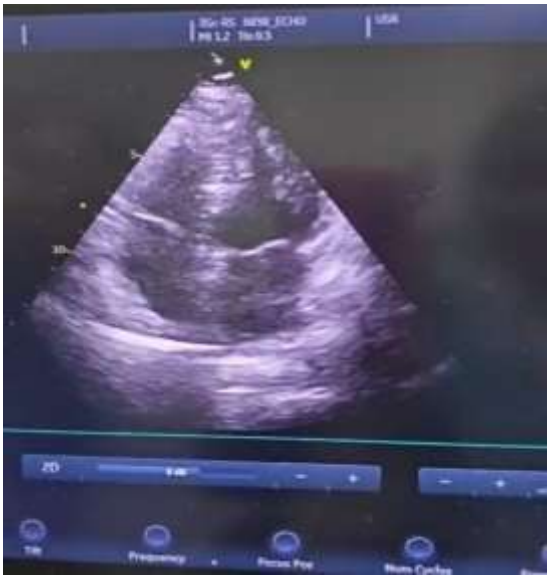

Fig 5. Transthoracic echocardiography demonstrating mild concentric left ventricular hypertrophy

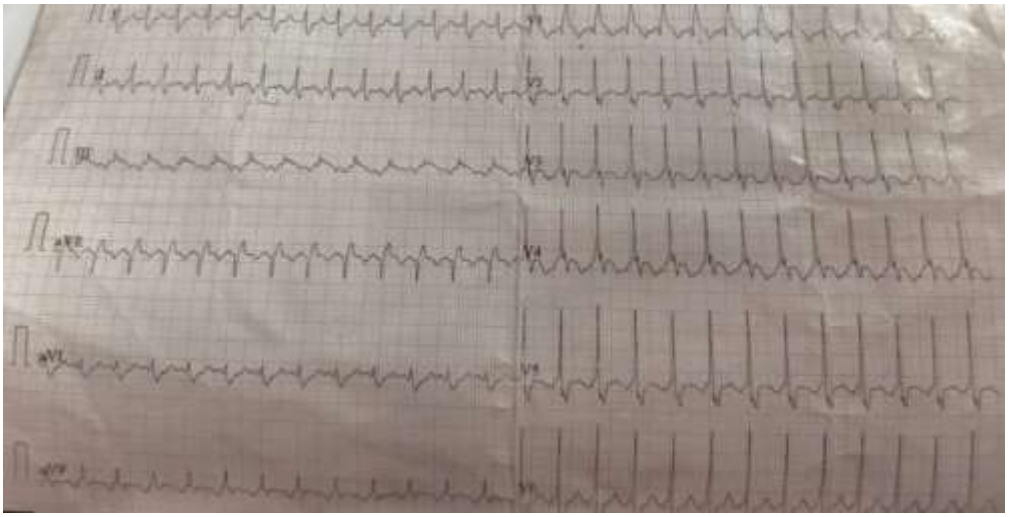

Fig 6. Electrocardiography showed sinus tachycardia with right bundle branch block, right axis deviation, and left posterior fascicular block.

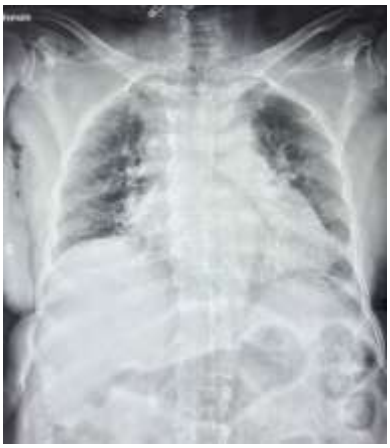

Fig 7. Chest radiograph shows reticular opacities in the bilateral lower lung zones, increased cardiothoracic ratio, and blunting of the right costophrenic angle.

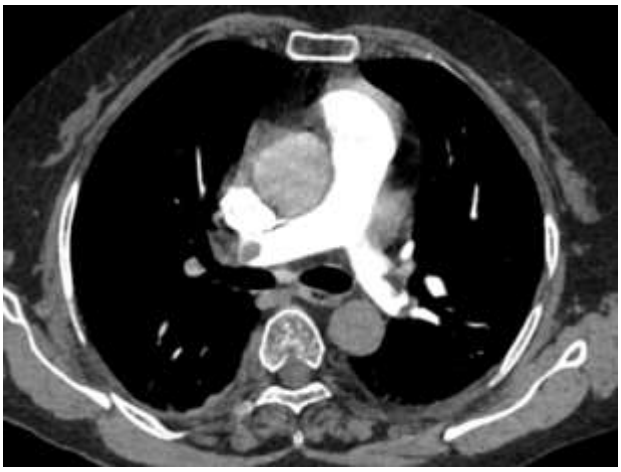

Fig 8. Follow-up computed tomography shows significant reduction in pulmonary arterial thrombus burden

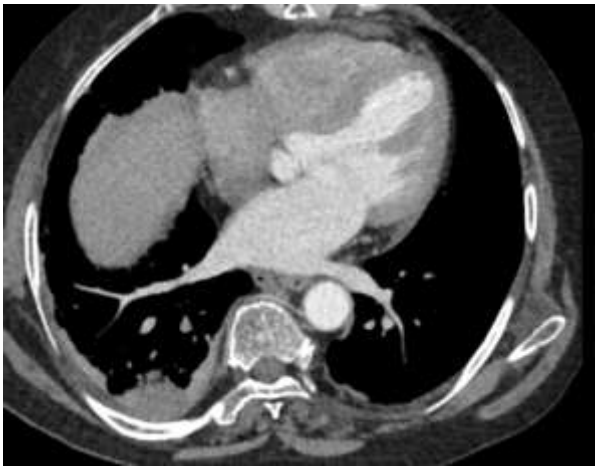

Fig 9. Follow-up computed tomography shows resolution of left lower pulmonary vein thrombus.

DISCUSSION

Pathophysiology

Pulmonary venous thrombosis (PVT) is an uncommon and frequently underrecognized clinical condition, with limited evidence available in the literature. The present case is notable for the coexistence of **pulmonary venous thrombosis with concomitant arterial thrombosis**, along with **elevated D-dimer and brain natriuretic peptide (BNP) levels, electrocardiographic (ECG) abnormalities, and a background history of hypertension**. This constellation of findings suggests a multifactorial and systemic thrombotic process involving both vascular and cardiac components.

PVT is most commonly associated with predisposing conditions such as lung malignancy, thoracic surgery, or atrial fibrillation; however, it may also occur in the absence of these risk factors, making diagnosis challenging [1,2]. A key feature of pulmonary venous thrombosis is its direct anatomical communication with the left atrium, which allows thrombi to enter the systemic circulation [1,3].

The presence of **elevated D-dimer** in this patient is consistent with active thrombogenesis and fibrinolysis. D-dimer is a sensitive marker of thrombus formation and degradation and is frequently elevated in both venous and arterial thrombotic conditions. In this case, markedly elevated D-dimer levels support the diagnosis of an ongoing systemic thrombotic process rather than an isolated vascular event [2,6].

In addition, the observed **elevation of BNP** provides important insight into the hemodynamic impact of the disease. In the context of pulmonary vascular pathology, elevated BNP may reflect **right ventricular strain or increased pulmonary venous pressure**, even in the absence of overt pulmonary arterial embolism [8].

The presence of **ECG changes** further supports cardiac involvement. Although specific ECG findings in PVT are not well established, nonspecific abnormalities such as sinus tachycardia, ST-segment changes, or T-wave inversions may occur as a result of myocardial strain, hypoxia, or demand ischemia. In patients with underlying cardiovascular risk factors, these changes may be more pronounced and may mimic acute coronary syndromes, thereby complicating the diagnostic process.

Hypertension is a critical contributing factor in this case. Chronic hypertension is associated with **endothelial dysfunction, vascular remodelling, and increased arterial stiffness**, all of which promote a prothrombotic state. In addition, hypertension is a well-established risk factor for arterial thrombosis, providing a unifying link between the venous and arterial components observed in this patient.

The coexistence of arterial and venous thrombosis also raises the possibility of a **systemic hypercoagulable state**. Even in the absence of identifiable inherited or acquired thrombophilia, transient or subclinical prothrombotic conditions may contribute to widespread thrombosis. Increasing evidence suggests that arterial and venous thromboses share common pathophysiological pathways, including inflammation, endothelial activation, and platelet dysfunction [5].

Another important mechanism is **immunothrombosis**, which involves the interaction between the immune system and coagulation pathways. Activation of inflammatory mediators, neutrophil extracellular traps (NETs), and endothelial cells can amplify thrombus formation across multiple vascular beds. This mechanism may be particularly relevant in cases such as the present one, where extensive thrombosis occurs without clear precipitating factors.

Comparison with existing literature provides further context for this case. Porres et al. emphasized the role of pulmonary veins as a potential source of systemic emboli due to their direct communication with the left atrium [1]. Chaaya and Vishnubhotla highlighted the diagnostic challenges and varied etiologies associated with PVT, underscoring its underrecognized nature [2]. Similarly, Baig and Wickramasinghe reported a case of idiopathic concurrent pulmonary embolism and pulmonary venous thrombosis, suggesting a systemic thrombotic predisposition even in the absence of identifiable risk factors [6].

Additionally, reports of simultaneous thrombosis involving multiple vascular territories—including coronary, intracardiac, and pulmonary circulations—support the concept that **multi-territory thrombosis represents a distinct clinical entity driven by systemic mechanisms**

rather than coincidental events [7]. The findings in the present case align with this emerging perspective.

Diagnosis

The diagnosis of PVT requires a high index of suspicion and appropriate imaging. Contrast-enhanced computed tomography (CT), particularly with careful evaluation of pulmonary venous structures, remains the most reliable diagnostic modality [3]. However, PVT may be overlooked if attention is focused solely on pulmonary arterial pathology, emphasizing the need for comprehensive imaging assessment.

Management

Management strategies are not well defined due to the rarity of this condition. Anticoagulation remains the cornerstone of therapy, aiming to prevent thrombus propagation and systemic embolization [2]. In cases of concomitant arterial thrombosis, additional interventions such as surgical or endovascular thrombectomy may be necessary. Given the presence of elevated BNP and ECG abnormalities, careful cardiac monitoring and supportive management are also essential.

The optimal duration of anticoagulation therapy is uncertain; however, prolonged or indefinite treatment is often considered in cases of idiopathic or extensive thrombosis due to the risk of recurrence [6]. Long-term follow-up is crucial to monitor for recurrent events and to assess cardiovascular status.

Conclusion

In conclusion, this case illustrates a rare and clinically significant presentation of **pulmonary venous thrombosis with concomitant arterial thrombosis**, accompanied by elevated D-dimer, elevated BNP, ECG changes, and underlying hypertension. The findings suggest a complex interplay between systemic hypercoagulability, endothelial dysfunction, and cardiovascular stress. Recognition of this condition is essential for timely diagnosis and management, as early intervention can significantly reduce morbidity and prevent life-threatening complications.

Author Contributions

Concept: Saranya Ravi, Smit Shrivastava
Data collection: Saranya Ravi, Smit Shrivastava
Analysis: All authors
Manuscript drafting: Saranya Ravi, Smit Shrivastava
Review: All authors.

Patient Consent

Written informed consent was obtained from the patient.

Conflict of Interest

The authors declare that there are no conflicts of interest.

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No funding was received for this study.

Abbreviations

PVT – Pulmonary venous thrombosis; **CT** – Computed tomography; **CTPA** – Computed tomography pulmonary angiography; **ECG** – Electrocardiogram; **BNP** – Brain natriuretic

peptide; **NT-proBNP** – N-terminal pro–brain natriuretic peptide; **RBBB** – Right bundle branch block; **APS** – Anti phospholipid syndrome; **PNH** – Paroxysmal nocturnal haemoglobinuria; **JAK2** – Janus kinase 2 mutation; **LV** – Left ventricle / Left ventricular; **FEU** – Fibrinogen equivalent units

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