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A Case of Chest Jolts: Pulmonary Vein Thrombus Extending into the Left Atrium

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Abstract

Pulmonary vein thrombosis (PVT) is a rare but life-threatening clinical condition, often found incidentally on imaging. In this report, we present an interesting case of PVT of the left inferior pulmonary vein with extension into the left atrium in a 78-year-old woman presenting with “jolts” in the chest. Initial imaging with plain chest film radiograph showed findings consistent with COPD and no acute intrathoracic process. A CT angiogram of the chest revealed a filling defect consistent with thrombus within the left inferior pulmonary vein extending into the left atrium. A transthoracic echocardiogram was remarkable for a severely enlarged right ventricular cavity with moderately reduced right ventricular systolic function and normal left ventricular size with preserved systolic function. She was not a candidate for any surgical interventions, and she was managed with systemic anticoagulation. Management of PVT mostly depends on the underlying cause as there are no well-defined treatment guidelines. The consensus recommends systemic anticoagulation until thrombus resolution. When anticoagulation is contraindicated, thrombectomy is indicated to restore blood flow. In patients with similar presentation and clinical history it is important to consider PVT, and to focus on prompt diagnosis and early initiation of appropriate treatment.

Keywords: Pulmonary vein thrombosis, Pulmonary vein thrombus, Thrombosis, Intracardiac thrombus, Left atrial thrombus, Pulmonary vein, Left atrium

1. Introduction

Pulmonary vein thrombosis (PVT) is a rare but life-threatening clinical condition, often found incidentally on imaging. The incidence of PVT is not well known, as prior documentation of the condition exists largely in the form of case reports. These reported cases occurred in the setting of complications from malignancy, major lung surgeries, and radio-frequency catheter ablation for atrial fibrillation. Less commonly, PVT may occur in the setting of atrial myxoma, mitral stenosis, congenital pulmonary vein narrowing, or idiopathic causes. This life-threatening disease predisposes to the development of pulmonary hypertension, reduced preload, increased right ventricular volume, and systemic embolization.¹ We present an interesting case of

PVT of the left inferior pulmonary vein with extension into the left atrium in a 78-year-old woman presenting with “jolts” in the chest.

2. Case presentation

A 78-year-old woman presented to the emergency room with a chief complaint of left-sided chest pain, described as “jolts,” and progressive shortness of breath of two days duration. Her medical history was remarkable for right breast cancer (status post radiation) in remission, nicotine dependence (approximately twenty pack years), chronic obstructive pulmonary disease (COPD) and deep vein thrombosis (DVT). She was wheelchair-bound at baseline, taking apixaban for DVT, and supplemental oxygen via nasal cannula (NC) at home (2 L/min nightly). A family member noted that the patient recently had

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lower oxygen readings on her pulse oximeter at home for three days prior to presentation. The patient described the left-sided chest pain as stabbing, non-radiating, and worse with inspiration. She denied antecedent fevers, chills, cough, or gastrointestinal symptoms. She endorsed frequently missing doses of her home medications.

On presentation, she was afebrile, tachycardic (115 beats per minute), mildly tachypneic (18 breaths per minute), hypertensive (148/66 mm Hg), and saturating 90% on room air. Physical exam was remarkable for coarse bilateral breath sounds (worse on the left), no chest wall deformity or crepitus, tachycardia with regular rhythm, and no appreciable murmurs, rubs or gallops. Laboratory diagnostics on presentation revealed leukocytosis (19.6 k/uL; reference range: 4.0–10.8 k/uL), neutrophilia (82.9 k/uL; reference range: 43.0–75.0 k/uL), lymphopenia (9.6 k/uL; reference range: 15.0–45.0 k/uL), and a mildly elevated d-dimer (0.82 mcg/mL FEU; reference: ≤ 0.78 mcg/mL FEU). Unfortunately, arterial blood gas (ABG) was not obtained on admission.

A 12-lead EKG revealed sinus tachycardia with a ventricular rate of 122 beats per minute, occasional premature atrial complexes, leftward axis, criteria for left atrial enlargement, and a right heart strain pattern of S1Q3T3 (Fig. 1). A plain film radiograph of the chest revealed findings consistent with COPD, but no acute intrathoracic process. A follow-up CT angiogram of the chest revealed a filling defect consistent with thrombus within the left inferior pulmonary vein extending into the left atrium (Fig. 2, Video 1 (https://scholarlycommons.gbmc.org/cgi/viewcontent.cgi?filename=2&article=1019&context=jchimp&type=additional&preview_mode=1), Video 2 (https://scholarlycommons.gbmc.org/cgi/viewcontent.cgi?filename=3&article=1019&context=jchimp&type=additional&preview_mode=1)), and bilateral airspace disease characterized by multiple small, round areas of patchy consolidation, with some having cavitation. The patient was admitted to the intermediate care unit for further management. She was placed on 5 L NC for oxygen supplementation, with adequate saturations at 97%. She was also initiated on intravenous antibiotics and a continuous infusion of heparin. Given the clinical complexity, specialists from infectious diseases, pulmonology, and cardiothoracic surgery were consulted.

A transthoracic echocardiogram was remarkable for a severely enlarged right ventricular cavity, moderately reduced right ventricular systolic function, moderate tricuspid regurgitation, and a normal left ventricular size with preserved systolic function (60–65%) (Video 3 (https://scholarlycommons.gbmc.org/cgi/viewcontent.cgi?filename=4&article=1019&context=jchimp&type=additional&preview_mode=1)). Antibiotics were discontinued given low concern for an infectious etiology. Over the next few days, the patient experienced dysphonia and was often unable to make any audible sound due to increased pleuritic chest pain, limiting inspiratory effort. This respiratory splinting led to worsening hypoxia superimposed on her COPD. The patient was placed on heated high-flow nasal cannula (HHFNC) during the day, bilevel positive airway pressure at night, and ipratropium-albuterol nebulization treatments. Cardiothoracic surgery was consulted for her care, and after determining that she was not a candidate for any surgical interventions, continued anticoagulation was recommended for a minimum of six months.

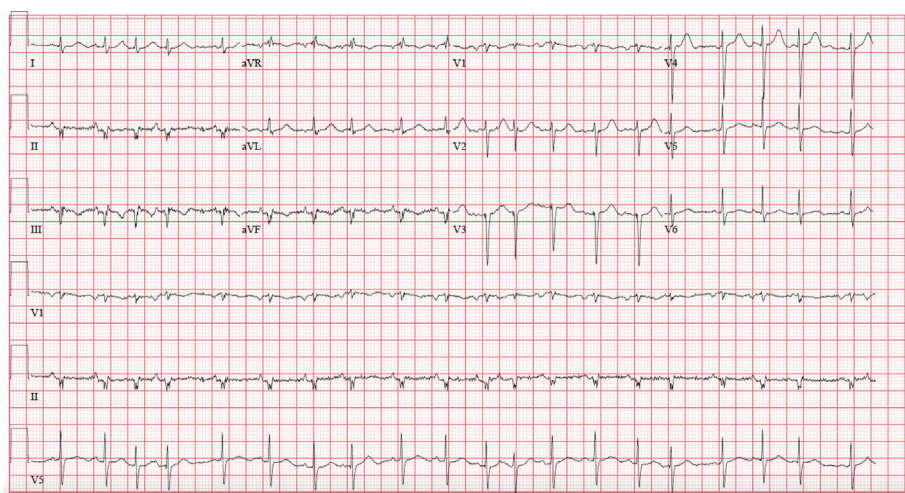


Fig. 1. Twelve-lead electrocardiogram showing sinus tachycardia, occasional premature atrial complexes, leftward axis, criteria for left atrial enlargement, and right heart strain pattern of S1Q3T3.

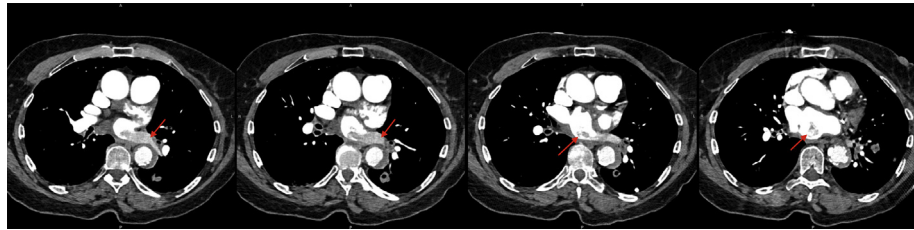


Fig. 2. CT angiogram of the chest (axial slices) revealing a filling defect consistent with thrombus within the left inferior pulmonary vein extending into the left atrium.

The patient's condition progressively worsened, with complete collapse of the left lung, likely due to mucus plugging. She was continued on HHFNC, with slight improvement respiratory status, and interval imaging showed a decrease in the left hemithoracic volume as well as interval development of moderate left-sided and mild right-sided pleural effusions. Her hospital course was further complicated by an episode of atrial fibrillation with rapid ventricular response, controlled after initiation of a continuous infusion of diltiazem.

Ultrasound guided thoracentesis of the left-sided pleural effusion was performed, with drainage of 170 cc of bloody fluid. Fluid analysis was remarkable for pleural fluid pH of 7.56 (reference range: pH 7.60–7.64), red blood cell count of 639,000/mm³ (reference range: 0–10000/mm³), estimated pleural hematocrit of 6.39%, white blood cell count of 161/mm³ (reference range: 0–1000/mm³), 55% neutrophils (reference range: 0–25%), LDH >200 u/L, and protein of 5.8 g/dL. Light's criteria for exudative effusion were met. Although the pleural fluid leukocyte count was elevated, given the elevated erythrocytes, and the expected ratio of one leukocyte per 500 erythrocytes, there was no significant pleural fluid leukocytosis. These results favored a pulmonary bleed related to anticoagulation over an inflammatory or neoplastic process. On hospital day 16, the patient unfortunately succumbed to persistent hypoxia and developed Cheyne-Stokes respirations. Given her clinical worsening and poor prognosis, the family ultimately requested comfort measures and the patient passed away from her worsening hypoxia.

3. Discussion and conclusions

PVT is a rare disease with limited information due to its unclear incidence. PVT predisposes to increased pulmonary venous pressure with compensatory pulmonary arteriolar constriction similar to pulmonary veno-occlusive disease. This process subsequently

leads to pulmonary arterial hypertension.² These triggers are most commonly seen in complications of malignancy, major lung surgeries, and radio-frequency catheter ablation for atrial fibrillation.

The clinical presentation of PVT is usually nonspecific, potentially resulting in diagnostic delays. Common symptoms include pleuritic chest pain, shortness of breath, cough and hemoptysis, which may lead to progressively pulmonary edema.³ Our patient presented with acute hypoxia and pleuritic chest pain.

A high index of suspicion and prompt imaging with CT chest angiography remains key to diagnosing PVT.¹ In particular, the pulmonary venous phase of contrast allows for better visualization of filling defects within the pulmonary vein and left atrium. Visualization of the pulmonary veins by transesophageal echocardiography in the mid-esophageal 4-chamber, 2-chamber and bicaval views may also aid in diagnosis.⁴ MRI is another modality that may allow for further characterization of the thrombus as bland or tumor.¹ Nevertheless, CT chest angiography is often the first line for diagnostic imaging due to the presentation being similar to pulmonary embolism.

Immediate complications from PVT include elevated pulmonary artery pressure, right ventricular diastolic end pressure, and right ventricular dilation.⁵ Additional clinical features can be secondary to further immediate complications such as peripheral embolization leading to transient ischemic attack, stroke, and secondary infection. PVT that remains undetected for a prolonged period can potentially result in pulmonary fibrosis or precipitate the development of pulmonary gangrene.^{2,3} The radiographic findings of pulmonary nodules, some cavitating, as seen in our patient, are most commonly seen with septic pulmonary emboli, but could also represent metastatic cancer with necrosis and cavitation.

Management of PVT mostly depends on the underlying cause as there are no well-defined treatment guidelines. The consensus recommends

systemic anticoagulation until thrombus resolution. When anticoagulation is contraindicated, thrombectomy is indicated to restore blood flow. Lobectomy or pneumonectomy are indicated with a worsening clinical course or unexpected cases of lung gangrene.⁵ Antibiotic therapy is often used due to the likelihood of superimposed infection. Our patient was managed with anticoagulation and follow up CT chest angiography to evaluate for resolution.

This case illustrates the multifaceted possible causes for the development of PVT in this patient - previous history of breast cancer with radiation treatment in presumed remission with noted pulmonary nodules, possibly reflecting metastatic recurrence, however no additional biopsy was performed. Her history of breast cancer with presence of pulmonary nodules may have contributed to hypercoagulability and endothelial injury. Her comorbidities and anticoagulation noncompliance may have also likely been the direct cause of her developing PVT. In patients with similar presentation and clinical history it is important to consider PVT, and to focus on prompt diagnosis and early initiation of appropriate treatment.

Conflict of interest

The authors report no conflict of interest.

Appendix

Video 1. CT angiogram of the chest (axial) demonstrating thrombus within the left inferior pulmonary vein extending into the left atrium.

Video 2. CT angiogram of the chest (coronal) demonstrating thrombus within the left inferior pulmonary vein extending into the left atrium.

Video 3. Transthoracic echocardiogram (apical 4-chamber view) showing moderately reduced right ventricular systolic function, normal left ventricular size with normal systolic function and ejection fraction of 60–65%.

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