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CASE REPORT

Pulmonary thromboembolism presenting with multiple cavitary infarcts: A diagnostic challenge with overlap to granulomatous vasculitis

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Abstract

Pulmonary embolism (PE) is a life-threatening condition that can present with a wide range of symptoms, often complicating its diagnosis. Pulmonary infarction (PI), a rare consequence of PE, may mimic other conditions such as pneumonia, malignancy, or vasculitis. A 65-year-old woman presented with progressive dyspnoea, chest pain, and haemoptysis. She was initially diagnosed with pneumonia; however, imaging revealed cavitating pulmonary infarcts. Further investigations excluded tuberculosis, fungal and viral infections, as well as granulomatosis with polyangiitis presenting with multiple cavitary lesions. Contrast-enhanced CT pulmonary angiography confirmed the diagnosis of PE with multiple cavitary infarcts. The patient was treated with anticoagulation and broad-spectrum antibiotics, resulting in complete clinical and radiological resolution within eight months. This case highlights the importance of considering infected cavitary pulmonary infarction in the differential diagnosis of rapidly cavitating pulmonary lesions, particularly in the presence of clinical signs of infection, to avoid misdiagnosis and ensure appropriate management.

Keywords: pulmonary embolism, pulmonary infarction, cavitation, sepsis, differential diagnosis

Introduction

Pulmonary embolism (PE) is a potentially life-threatening condition caused by sudden obstruction in the pulmonary circulation, leading to impaired perfusion and possible parenchymal ischemia. It carries considerable morbidity and mortality, with fatality rates reported between 10% and 28% when diagnosis or treatment is delayed [1,2], highlighting the need for early recognition.

Common symptoms include dyspnea, pleuritic chest pain, and hypoxemia, though presentations can be nonspecific. Pulmonary infarction (PI), a consequence of distal arterial occlusion in PE, occurs in 10% to 30% of cases [1,3], despite dual pulmonary and bronchial arterial supply. Diagnosis may vary based on clinical, imaging, or pathological findings.

Infarcts typically appear as non-cavitating opacities; however, cavitation may develop, particularly in infected or immunocompromised cases. Postmortem data suggest cavitation occurs in 4–5% of pulmonary infarcts [4], potentially from sterile necrosis or secondary infection [5]. Cavitating infarctions can mimic infections, malignancies, or vasculitides like granulomatosis with polyangiitis (GPA), making diagnosis challenging [6,7]. A multidisciplinary approach is essential. We present a rare case of histologically confirmed septic cavitary pulmonary infarction initially resembling GPA.

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CASE HISTORY

A 65-year-old woman presented with a two-month history of progressive dyspnea, productive cough, chest discomfort, and intermittent hemoptysis. She reported yellowish sputum with recent blood-tinging and sharp chest pain. Her history included type 2 diabetes, hypertension, and prior coronary stenting. There was no smoking, tuberculosis (TB) exposure, or occupational risk.

On exam, she had bilateral (2+) pretibial edema and reduced breath sounds. SpO_2 was 92% on 3 L/min oxygen, and temperature was 38.2°C. Labs showed leukocytosis (white blood cell count [WBC] 17,100/mm^3; neutrophils 16,000/mm^3), C-reactive protein (CRP) 272 mg/L, hemoglobin A1c (HbA1c) 13.5%, and postprandial glucose 355 mg/dL. Urine studies showed nephrotic-range proteinuria (1297 mg/day) and low spot urine sodium (11 mmol/L).

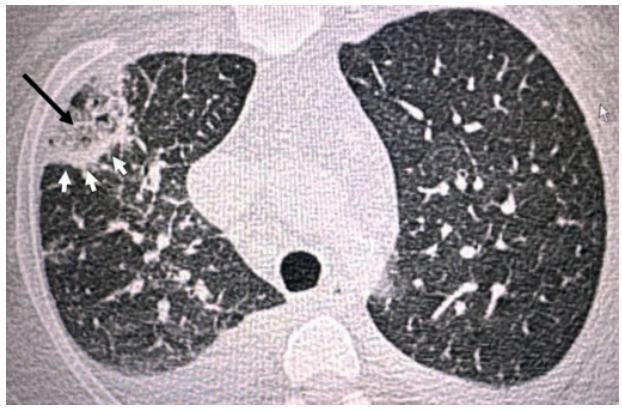

Figure 1. On non-contrast axial chest CT, a pleural-based, triangular consolidation (white arrows) is noted in the anterior segment of the right upper lobe, demonstrating smooth margins and a central ground-glass component (black arrow), raising suspicion for pneumonic infiltration.

Initial chest X-ray demonstrated blunting of the bilateral costophrenic angles and an ill-defined opacity in the mid-lung zone. Because pneumonia was suspected, a non-contrast chest CT was obtained, revealing bilateral pleural effusions and a wedge-shaped consolidation in the anterior right upper lobe with central ground-glass changes adjacent to the pleura (Figure 1). Empiric Sulbactam/Cefoperazone together with intravenous furosemide was initiated. Despite appropriate treatment, the patient showed no clinical improvement by the third day, prompting a switch to Piperacillin/Tazobactam. On the seventh day, she developed a new fever (37.9°C), and *E. coli* grew in blood and sputum cultures; therefore, Meropenem was started. During this period, a markedly elevated D-dimer level (2620 ng/mL) was noted. Given the persistent lack of clinical response to broad-spectrum antibiotics together with the unexpectedly high D-dimer, pulmonary embolism became a significant diagnostic consideration. On the twelfth day, a computed tomography pulmonary angiogram (CTPA) was performed and demonstrated cavitation within the right upper-lobe consolidation as well as newly developed cavitary lesions in the left lower and right lungs (Figure 2). Segmental and subsegmental pulmonary arteries in both upper lobes and in the distal left lower lobe showed intraluminal filling defects consistent with thromboembolic occlusion (Figure 3). The patient had multiple recognized factors for pulmonary thromboembolism, including advanced age, poorly controlled diabetes mellitus, acute systemic infection with bacteremia, and proteinuria attributed to diabetic nephropathy, all of which may have contributed to a prothrombotic state.

A detailed diagnostic evaluation excluded tuberculosis, fungal and viral infections (negative serology and bronchoalveolar lavage (BAL) cultures), and systemic autoimmune or vasculitic diseases (negative antinuclear antibody (ANA) and antineutrophil cytoplasmic antibody (ANCA) panel). The coexistence of multiple cavitary pulmonary lesions and bacteremia prompted evaluation for infective endocarditis; however, transthoracic echocardiography was unremarkable, with no evidence of valvular vegetations or structural abnormalities. Proteinuria and hyponatremia were attributed to diabetic nephropathy, and upper respiratory tract examination, including sinus CT, showed no abnormalities. Despite the radiological diagnosis of pulmonary embolism, the rapid progression of multiple cavitary lesions, their atypical upper-lobe predominance, and the rarity of cavitation in pulmonary infarction raised persistent concern for alternative diagnoses, particularly vasculitis. Therefore, tissue sampling was pursued to exclude granulomatosis with polyangiitis and other inflammatory or malignant processes.A CT-guided transthoracic core-needle biopsy (CT-TTNB) showed acute inflammation and

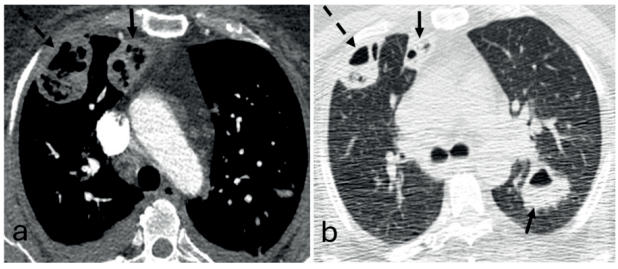

Figure 2. Cavitation has developed in the previously observed consolidation in the right upper lobe (dashed black arrow). New cavitary lesions are seen adjacent to the first lesion and in the superior segment of the left lower lobe (black arrows). These findings are demonstrated in both the mediastinal (a) and parenchymal (b) windows.

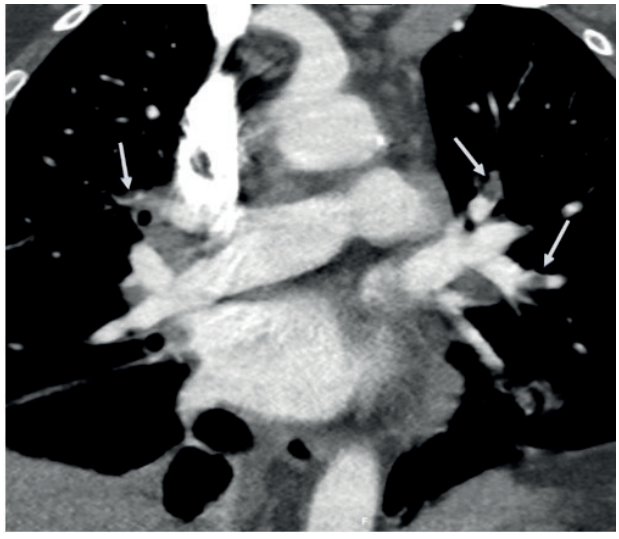

Figure 3. Contrast-enhanced pulmonary CT angiography shows filling defects compatible with thromboembolism in the segmental branches of both pulmonary arteries in the upper lobes and in the left lower lobe branch (arrows).

focal fibrosis, but no vasculitis. The final diagnosis was pulmonary embolism with multiple pulmonary infarcts. Persistent inflammation, E. coli growth in blood/sputum cultures, and rapid cavitation supported septic pulmonary infarction. Enoxaparin was started, later replaced by oral rivaroxaban. The CT performed at 8 months showed complete resolution of the cavitary lesions with minimal remaining fibrosis.

DISCUSSION

Pulmonary embolism is a critical cardiovascular emergency with variable presentations, complicating diagnosis. Pulmonary infarction, although uncommon, may present with hemoptysis or pleuritic pain. Incidental PE is more often detected without infarction (35%) than with infarction (11%) [1]. PI is frequently reported in delayed PE because of its resemblance to pneumonia or vasculitis [6,7]. In this case, initial imaging suggested pneumonia; however, lack of response to treatment prompted CTPA on day 12, revealing multiple cavitary consolidations. Final diagnosis of PI was confirmed by histopathology.

On CT, the classic radiographic finding of pulmonary infarction (PI)—Hampton's hump—has limited value. Instead, peripheral consolidations with central low attenuation and hypoenhancement on contrast-enhanced scans are more specific, reported in approximately 98% and 95% of cases, respectively [8,9]. Cavitation remains rare[4,10]. In this case, atypical imaging findings prompted further evaluation. Differential diagnoses for multiple cavitary lung lesions include infectious processes such as septic pulmonary embolism and vasculitis [3,7].

GPA was initially suspected due to overlapping imaging features with pulmonary infarction, including wedge-shaped morphology and angiocentric distribution [11]. However, GPA lesions typically show peribronchovascular distribution, irregular margins, and heterogeneous enhancement [12], unlike the subpleural, smooth-bordered, non-enhancing, and rapidly cavitating lesions seen in this case. Together with negative vasculitis markers, these findings favored septic pulmonary infarction secondary to untreated PE. CTPA confirmed PE by revealing central and segmental arterial filling defects with multiple infarcts. Comprehensive clinical, laboratory, imaging, and histopathological evaluation showed no evidence of systemic vasculitis, effectively excluding GPA.

Septic pulmonary embolism (SPE) is an important differential diagnosis in patients with multiple cavitary pulmonary lesions and is most commonly associated with infective endocarditis. On imaging, SPE typically presents with peripherally distributed cavitary nodules, predominantly involving the lower lobes, often accompanied by peripheral wedge-shaped opacities;

a feeding vessel sign may also be observed [13,14]. In this case, however, the lesions were predominantly located in the upper lobes and were characterized mainly by wedge-shaped infarcted areas rather than a nodular pattern, with no feeding vessel sign identified. In addition, none of the common predisposing factors for SPE—such as intravenous drug use, indwelling vascular catheters, cardiac implantable devices, or infective endocarditis—were present. The absence of valvular vegetations or structural cardiac abnormalities on transthoracic echocardiography further reduced the likelihood of SPE. Taken together, the clinical, radiological, and echocardiographic findings support a diagnosis of septic cavitating pulmonary infarction rather than SPE.

PI may rarely lead to cavitation—either aseptic due to sterile necrosis or more commonly septic due to secondary infection [3,7]. Septic cavitation typically presents with fever, leukocytosis, and positive sputum cultures [5], as seen in this case where *Escherichia coli* was isolated from both blood and sputum. Unlike the typically solitary, large cavitary infarcts reported in literature [15], this case exhibited multiple small lesions. This was likely due to emboli involving several segmental and subsegmental arteries, creating numerous small infarcts prone to infection. Cavitation in infected emboli may occur sooner than in aseptic cases—typically around two weeks [16]. In this case, cavitation appeared within 12 days, aligning with this timeframe and supporting a diagnosis of septic pulmonary infarction.

In PI, sputum typically contains minimal bacterial or inflammatory content despite hemoptysis. However, secondary infection can mimic bacterial pneumonia [7]. In this case, positive sputum cultures confirmed infection of the infarcted tissue. This distinction is crucial, as sterile infarcts are managed with anticoagulation alone, while septic infarcts require antibiotics. This case responded well to combined anticoagulant and broad-spectrum antibiotic treatment.

PI can lead to severe complications like sepsis, empyema, pneumothorax, and bronchopulmonary fistula [17]. In cases with cavitation, surgical intervention may be recommended, especially when complications like sepsis are present [10]. However, this case showed complete clinical and radiological resolution, with only minor fibrotic changes after 8 months, indicating that medical therapy alone was sufficient.

Cavitary lung lesions are associated with a broad spectrum of infectious and non-infectious diseases and often pose significant diagnostic challenges. This case, the presence of multiple cavitary lesions initially raised suspicion for GPA. However, careful evaluation of radiological findings, absence of systemic involvement, negative microbiological and serological test results, and histopathology supported the diagnosis of infected pulmonary infarction. Although cavitation in pulmonary infarction is a recognized phenomenon, this case is distinctive because of the rapid development of multiple cavitary lesions with upper-lobe predominance and septic features, closely mimicking vasculitis and septic pulmonary embolism.The patient responded well to broad-spectrum antibiotic therapy combined with anticoagulant treatment and achieved complete clinical and radiological recovery without the need for surgical intervention. This case highlights the necessity of considering infected pulmonary infarction in the differential diagnosis of rapidly cavitating pulmonary lesions, especially in the presence of clinical signs of infection.

Informed Consent: Informed consent was not obtained because the case report contains no identifiable patient data, and the study was conducted in compliance with ethical standards for retrospective analyses.

Author contribution

Conception and design: B.S.A.; Data acquisition: B.S.A.; Data analysis: B.S.A.; Data interpretation: B.S.A.; Drafting of the manuscript: B.S.A.; Critical revision of the manuscript: B.S.A. The author reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of this study.

Ethical approval

Written informed consent was obtained from the patient(s) or their legal guardians for the publication of this study and any accompanying images.

Data availability statement

The data supporting the findings of this study are not publicly available due to containing patient information

and are not publicly available due to privacy and ethical restrictions.

Conflict of interest

The author declares that this study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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Generative AI statement

The author declares that during the preparation of this study, the following AI-assisted technology was used: ChatGPT-5 (OpenAI) on 15/07/2025 – 08/08/2025. Extent of Use: ChatGPT was used solely to assist with language refinement and minor phrasing adjustments in the manuscript. The author confirms that he/she has critically reviewed and edited any AI-generated content and takes full responsibility for the integrity, accuracy, and originality of the publication. The author certifies that the original human contribution is maintained and that AI-assisted tools are not listed or cited as authors..

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