Case Report

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Internal Medicine Section

Pulmonary Cavitation Following Acute Pulmonary Infarction: A Rare Case of Complication of Pulmonary Embolism

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ABSTRACT

Pulmonary infarction is a rare complication of Pulmonary Embolism (PE) due to dual blood supply of the lung. Pulmonary cavity formation following PE is a very rare but a recognised complication, typically resulting from ischaemic infarction of lung parenchyma. The majority of reported cases involve patients managed conservatively with anticoagulation or thrombolytic therapy, without surgical or invasive intervention. Cavity formation occurs due to occlusion of distal pulmonary arteries, leading to haemorrhage, necrosis and subsequent parenchymal breakdown. The authors report a rare case of a 35-year-old male patient with no known co-morbidities presented with acute PE with pulmonary infarction leading to cavitation with possible secondary infection following a thrombolysis and anticoagulation therapy. Chest radiography showed prominent descending right pulmonary artery-(Palla sign). As patient was haemodynamically unstable, he was immediately thrombolysed with intravenous Streptokinase (STK) followed by systemic anticoagulation with unfractionated heparin. Patient discharged on tab dabigatran 150 mg twice daily in a stable condition. This case highlights the importance of vigilance for post-embolism complications such as cavitation and infection, even after appropriate anticoagulant management and need for prompt management to prevent associated morbidity and mortality thereby improving the quality of life of the patient.

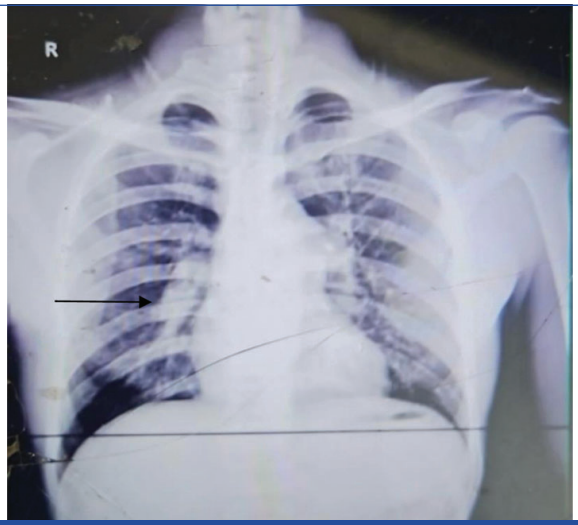
Keywords: Anticoagulation, Secondary Infection, Thrombolysis

CASE REPORT

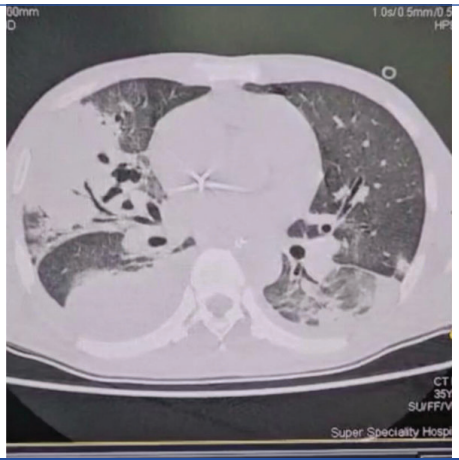
A 35-year-old male presented to the Emergency Department with breathlessness modified Medical Research Council (mMRC) grade 4, right-sided chest pain which was sharp and increased on coughing and blood-tinged sputum for 15 days. The patient had no known comorbidities, no history of immobility or prior thromboembolic events.

On initial examination, the patient was alert. His vital signs revealed tachycardia (pulse rate 130 beats per minute), tachypnoea (respiratory rate 30 breaths per minute), hypotension (blood pressure 90/60 mmHg) and oxygen saturation of 97% on room air. Auscultation of the chest revealed fine inspiratory crepitations bilaterally.

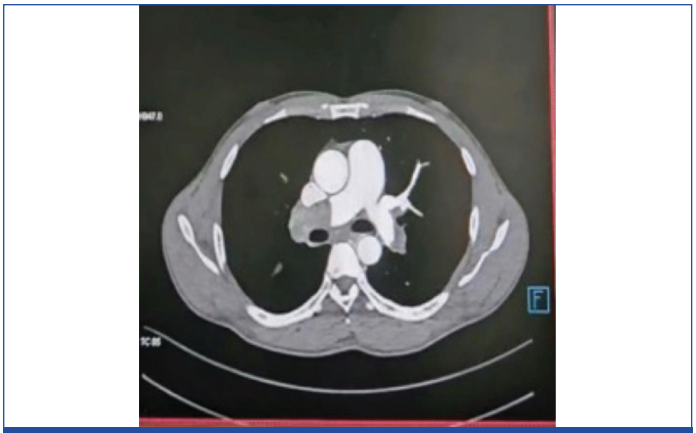
Laboratory investigations, including a complete haemogram, were unremarkable except for an elevated D-dimer level of >10,000 ng/mL. Electrocardiogram (ECG) demonstrated an S1Q3T3 pattern along with T-wave inversions in precordial leads V2-V4, suggestive of right heart strain. Troponin-I and Creatine Phosphokinase-Myocardial Band (CPK-MB) were within normal limits. His modified Wells score [1] was 5.5, indicating a high likelihood of PE. Chest radiography showed prominent descending right pulmonary artery-(Palla sign) [Table/Fig-1]. Considering his clinical and laboratory findings, Computed Tomography Pulmonary Angiography (CTPA) was immediately planned. CT pulmonary angiogram axial CT lung window showed dense consolidation in right upper lobe middle lobe with cavitation within and surrounding ground glass opacities [Table/Fig-2] and contrast window showed a thrombosis of right pulmonary artery extending into right upper lobe, middle lobe and lower lobe and their segmental and subsegmental branches. Similar thrombosis noted in left pulmonary artery extending into segmental and subsegmental branches [Table/Fig-3]. Echocardiography further demonstrated right atrial and right ventricular dilatation, severe tricuspid regurgitation, severe pulmonary arterial hypertension and a congested inferior vena cava with <50% collapsibility,



[Table/Fig-1]: Chest X-ray on admission showing right descending pulmonary artery prominence (Palla sign).



[Table/Fig-2]: Axial CT lung window showing dense consolidation in right upper lobe middle lobe with cavitation within and surrounding ground glass opacities.

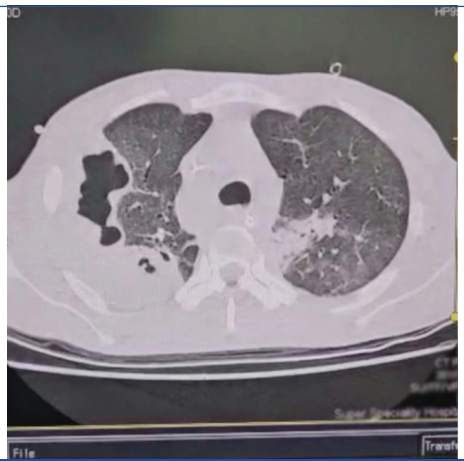


[Table/Fig-3]: Axial CTPA showing hypodense filling defect in right pulmonary artery and left pulmonary artery.

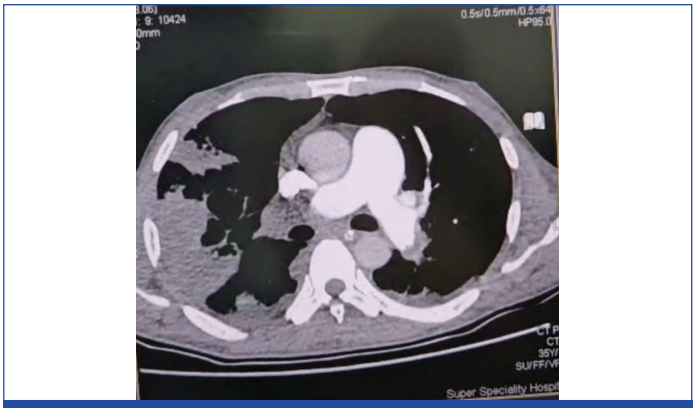
indicating significant right heart strain. Bilateral lower limb Doppler did not show any thrombosis.

As patient was haemodynamically unstable, he was immediately thrombolysed with intravenous STK followed by systemic anticoagulation with unfractionated heparin. Despite initial management, the patient's oxygen requirement progressively increased, culminating in respiratory distress necessitating endotracheal intubation and mechanical ventilation.

The patients repeated CTPA showed thrombosis involving right and left subsegmental arteries with wedge shaped peripheral areas of cavitatory consolidation in right upper and middle lobe and left upper lobe suggestive of infarction [Table/Fig-4,5]. Serial haemograms showed leukocytosis with neutrophilic predominance (89% neutrophils). Endotracheal aspirate culture isolated *Acinetobacter lwoffii*, which was intermediately sensitive to minocycline. Inflammatory markers were markedly elevated with procalcitonin at 1.79 ng/mL and C-Reactive Protein (CRP) levels peaking at 242 mg/L, serum homocysteine level 17.45 µmol/L. Since patient was thrombolysed and was on anticoagulants, his work-up for both congenital and acquired thrombophilia profile was not done. The diagnosis of pulmonary infarction with cavitary consolidation complicated by secondary bacterial infection was established. The patient was managed with broad-spectrum intravenous antibiotics, including meropenem 1 gram thrice daily, tigecycline 100 mg twice daily followed by oral minocycline 100 mg twice daily alongside therapeutic anticoagulation with heparin dose adjusted as per activated Partial Thromboplastin Time (aPTT). The patient's clinical condition gradually improved with resolution of leukocytosis and reduction in oxygen requirements and review echocardiography revealed no evidence of embolus in the visualised portion of main pulmonary artery, left pulmonary artery, right pulmonary artery with severe pulmonary arterial hypertension and

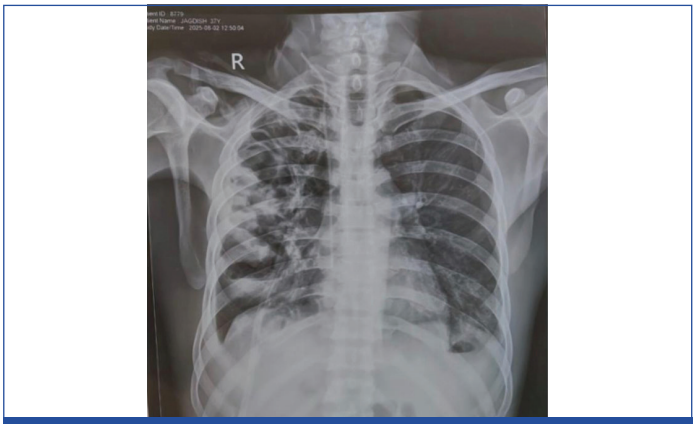


[Table/Fig-4]: Axial CT lung window showing dense consolidation with large cavities in right middle lobe with cavitation within and surrounding ground glass opacities.



[Table/Fig-5]: Axial contrast CT showing partial contrast opacification of bilateral pulmonary artery with pulmonary infarct and cavitatory consolidation.

no right ventricular dysfunction. He was subsequently weaned off from mechanical ventilation and transitioned to oral anticoagulation therapy with dabigatran 150 mg twice daily. His follow-up chest X-ray done after 10 days showed improvement [Table/Fig-6]. The patient got discharge on tab dabigatran 150 mg twice daily in a stable condition.



[Table/Fig-6]: Chest X-ray showing resolving consolidation with bilateral minimal pleural effusion with small air fluid level within cavities on right-side.

DISCUSSION

The PE is a common but potentially life-threatening condition with varied clinical presentations, ranging from asymptomatic incidental findings to severe cardiopulmonary collapse. Despite the occlusion of pulmonary arteries, pulmonary infarction occurs in only approximately 10% of cases [2], owing to the lungs' dual blood supply from both the pulmonary and bronchial arteries. Among those who do develop infarction, cavitation is a rare, reported in only 4-5% of cases [3]. One of the risk factor for the development of aseptic necrosis and subsequent cavity formation is the size of the infarct, with lesions larger than 4 cm posing a significantly higher risk [3]. PE and pulmonary infarction should be kept in differential diagnosis of patients presenting with cavitatory consolidation [4].

This patient presented with a classic embolic event, managed promptly with thrombolysis and anticoagulation. However, despite reperfusion strategies, he developed cavitary pulmonary lesions, a scenario that highlights the pathophysiological complexity beyond mere vascular occlusion. The occurrence of cavitation in this context reflects extensive parenchymal necrosis, a process that is influenced by factors such as the size of the infarct, vascular perfusion pressures and the patient's immune and inflammatory responses and secondary infection. Patient had secondary infection with *Acinetobacter*. The average time required for the formation of a cavity after a PE in a patient with infected embolism is 18 days while that in a non infected infarct is 28 days [5,6].

In a case reported by Koroscil MT and Hauser TR, patient developed cavitation three weeks following initial presentation while in this case cavity was seen within one week of presentation [7]. In another

similar case reported by Kumar S and Kumari J, elderly female initially treated as community acquired pneumonia, developed cavitation on fifth day, CT done showed cavitatory pulmonary infarct with lower limb deep venous thrombosis [8]. In a case reported by Elgohary A et al., elderly male presented with cavitatory lesion in right lung suspicious of malignancy was later found to have cavity secondary to PE [5]. In another case described by Venugopal V et al., patient developed cavitatory lesion secondary to pulmonary infarction due to factor V Leiden mutation [9]. This patient additionally had Chronic Thromboembolic Pulmonary Hypertension (CTEPH) which was treated with riociguat.

Several case reports, including that by Nilgiri KM et al., have emphasised the diagnostic challenge posed by cavitating pulmonary infarcts. Radiologically, these lesions can mimic infectious or neoplastic processes, leading to diagnostic ambiguity. In this case, the peripheral location of the cavities with surrounding consolidation raised suspicion for infectious causes such as bacterial pneumonia, lung abscess, or tuberculosis. However, extensive microbiological work-up yielded a pathogen, confirming the diagnosis towards septic infarct cavitation [10].

One noteworthy aspect of this case is the infarct size, which exceeded 4 cm, a threshold recognised in the published literature [3] as a strongest risk factor for septic necrosis and cavitation. This observation reinforces the concept that infarct burden, rather than the success of vascular reperfusion alone, dictates the risk of structural lung damage. Management strategies remain primarily supportive and anticoagulation-based. The development of cavitation, in the absence of infection, does not typically necessitate antibiotics, underscoring the importance of judicious antimicrobial stewardship. Nonetheless, close monitoring is warranted to detect secondary bacterial infections and complications such as massive haemoptysis and pleural effusion.

Advanced imaging modalities like CT Pulmonary Angiography (CTPA), are indispensable for diagnosing PE and evaluating infarct morphology, cavity formation and excluding alternative diagnoses [11]. Serial imaging may be necessary to track the resolution or progression of cavities, which helps in therapeutic decisions.

Patient was initially haemodynamically unstable with hospital acquired infection successfully managed with effective ventilatory strategies and culture guided antibiotics to treat hospital acquired infection. Patient gradually improved and was discharged with oral anticoagulant. This case stresses the need for high clinical awareness regarding rare complications of PE, such as cavitating pulmonary infarction. A systematic approach involving detailed imaging assessment, identification of infectious aetiologies and vigilant clinical monitoring can prevent misdiagnosis, inappropriate interventions and help in reducing mortality. Furthermore, it highlights a gap in current understanding regarding the precise mechanisms and predictors of cavitation post-PE, emphasising the need for further research in this domain.

CONCLUSION(S)

A systematic approach involving detailed imaging assessment, identification of infectious aetiologies and vigilant clinical monitoring can prevent misdiagnosis, inappropriate interventions and helps in reducing mortality. Early recognition through advanced imaging and identification of infections is crucial for guiding appropriate management, improving clinical outcomes, preventing long-term complications and mortality. Possibility of pulmonary infarction should be kept in mind in patients presenting with lung cavities. Secondary bacterial infection should be ruled out in patients with prolonged hospitalisation.

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