

Case Report

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Pulmonary Infarction Mimicking Organised Pneumonia in a Patient with Pulmonary Embolism: A Case Report

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ABSTRACT

Diagnosing a Pulmonary Embolism (PE) is often challenging, as the symptoms, signs and results of basic diagnostic tests are similar to those of other common conditions. It is essential to investigate risk factors for PE because, if associated with a major risk factor, it is considered provoked and the duration of anticoagulant treatment will be for months; whereas in an unprovoked episode, treatment will, in principle, be indefinite, although always tailored to the individual case. The association between unprovoked PE and a radiological lesion under investigation necessitates ruling out the presence of an occult tumor. We present the case of a man with unprovoked PE and a pulmonary lesion suggestive of Organised Pneumonia (OP).

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Introduction

The clinical manifestations of acute pulmonary embolism (PE) are wide-ranging, extending from a complete absence of symptoms to shock or sudden death. It is essential to maintain a high level of clinical suspicion so as not to miss clinically significant cases. Several conditions form part of the differential diagnosis of PE and may sometimes co-exist; therefore, one diagnosis does not completely rule out another. There are several scales for stratifying the probability of PE; different algorithms are recommended depending on the score, and management varies according to clinical severity [1]. Radiological findings of pulmonary infiltrates with an air-bronchogram suggest a differential diagnosis of slow-resolving pneumonias, which include OP, certain tumours and Pulmonary Infarcts (PI), amongst others; therefore, clinical presentation, radiological findings and clinical course are key to distinguishing between them [2].

Clinical Case

A 58-year-old male, a former smoker with a pack year of 13, works as a bus panel fitter. He suffers from hypertension, dyslipidaemia, BMI 32 and severe bronchial asthma with a TH2 inflammatory pattern and 300 eosinophils/uL, FENO 20 ppb; his spirometry shows a moderate non-obstructive pattern and a positive bronchodilation test, with poor clinical control characterized by limited physical activity, dyspnoea on strenuous exertion and a dry cough. An outpatient high-resolution chest CT scan was requested to complete the investigation, which was carried out six months later, on 25 January 2024, and which revealed findings suggestive of OP in segment 10 of the left lower lobe (LLL), Figure 1.

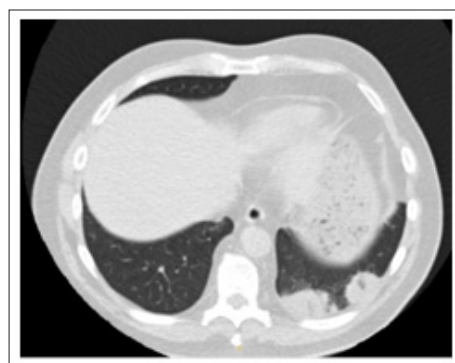


Figure 1: In the Posterior Basal Segment of Left Lower Lobe, Ground-Glass Opacities are Observed, Surrounded by Areas of Airspace Consolidation Forming a Complete Ring, Consistent with the 'Atoll Sign', Which is Highly Suggestive of Organised Pneumonia

The patient had attended Emergency Department (A&E) in late 2024 with a cough, mucous sputum and pleuritic pain in the left chest, presenting with a radiological infiltrate in the posterior segment of LLL; he had received empirical antibiotics for 14 days and prednisone 1 mg/kg/day for 10 days. During the follow-up consultation in mid-February 2025, he continued to experience a cough with purulent sputum and, on occasion, mild haemoptysis, chest pain and dyspnoea; a bronchoscopy, respiratory function tests and a follow-up CT scan were requested, and prednisone 1 mg/kg/day was initiated for 3 months. On 25 February, he attended again A&E due to worsening dyspnoea and oedema of the right lower limb; he was haemodynamically stable, conscious and afebrile, with a baseline SpO₂ of 96%. Laboratory results showed

renal function, electrolytes and haemoglobin within normal limits, elevated white blood cell count with other acute-phase reactants within normal limits, and DD 2755 ngr/ml, respiratory alkalosis on arterial blood gas analysis; and a contrast-enhanced urgent chest CT scan revealed findings consistent with acute central pulmonary embolism involving both main pulmonary arteries, extending to all lobar and segmental arteries—primarily those of both basal pyramids—with no evidence of right ventricular overload; the lesions in LLL, identified in a previous study and consistent with possible OP, remain unchanged, although other aetiologies could not be ruled out, Figure 2.

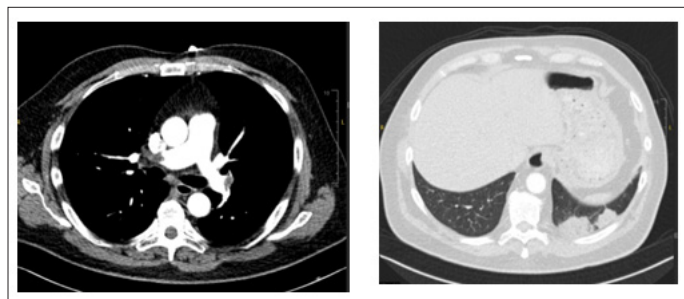


Figure 2: Central Filling Defect in Both Main Pulmonary Arteries (Left), Opacities in the Posterior Basal Segment of the Left Lower Lobe (Right)

He was admitted to the respiratory medicine department, where a Doppler ultrasound of the lower limbs was requested, confirming a thrombus in the right internal saphenous vein. A bronchoscopy was performed, revealing signs of chronic bronchitis, and a blind transbronchial biopsy was taken of the known lesion. A comprehensive investigation was completed with CT scans of the skull and abdomen-pelvis, both of which were negative. He was classified as having low-risk PE and right deep vein thrombosis (DVT); anticoagulation was initiated with heparin at 1 mg/kg every 12 hours, and on 1 March he was discharged whilst on heparin at 1.5 mg/kg every 24 hours, with the pathological results pending to determine the nature of the pulmonary lesion. Three weeks later, he returned to A&E with pleuritic-type left-sided chest pain, purulent secretions and increased shortness of breath; a chest X-ray revealed opacification of the left lung base. The results of the transbronchial biopsy were reviewed and found to be consistent with fragments of alveolar parenchyma, some of which show haemorrhagic necrosis compatibles with PI. Broad-spectrum empirical antibiotic therapy was initiated to cover superinfection of the PI, and a chest CT scan was performed on 2 April, revealing a large left-sided pleural effusion and vascular defects as signs of chronic PE. *Haemophilus parainfluenzae* was isolated from sputum, and the antibiotic regimen for the complicated nosocomial pneumonia was switched to levofloxacin in accordance with the antibiotic susceptibility test. However, the patient's condition remained sluggish, with no fever but reports of uncontrolled chest pain and an increase in the pleural effusion; so, it was decided to perform a chest ultrasound, which revealed an organized pleural cavity containing debris and septa. A diagnostic thoracentesis was performed, extracting 40 cc of sero-haematic fluid with a pH of 7.33; the procedure had to be halted due to symptomatic hypotension. Due to the complicated parapneumonic effusion, a pleural drain was inserted on 10 April; this was removed two days later and the patient was discharged to continue the antibiotic treatment for a further two weeks and to switch his anticoagulation

therapy to Dabigatran. An outpatient contrast-enhanced chest CT scan was performed on 4 July, showing reduction in the left-sided pleural effusion, a round atelectasis and other lamellar atelectasias adjacent to the effusion. Additional note from the radiologist: The chronology of radiological investigations was reviewed, leading to the conclusion of a pulmonary haematoma in LLL, probably in the context of a PI secondary to PE; there were no radiological findings suggestive of OP (Figure 3).



Figure 3: Airspace Consolidation with an air Bronchogram and Curvature of the Bronchovascular Bundles in Relation to a Round Atelectasis, Associated with Laminar Atelectasias in Both Lower Lobes

At the follow-up in August 2024, the patient was feeling better; he reported mMRC grade 1 baseline dyspnoea and mild left-sided pleuritic discomfort at the site where the pleural drain had been inserted. The respiratory function tests from July 2024 are as follows: VC 2.63 L (52%), FEV1: 2.33 L (62%), FEV1%VC: 88%. KCO: 99%, TLC: 72%, RV: 102%. A follow-up appointment was scheduled for one year later, at which the patient remained stable; a thrombophilia screening test was negative apart from elevated factor VIII; a chest X-ray showed thickened left basal parenchymal bands secondary to parenchymal scarring; spirometry in June 2025 showed improvement with a VC of 71.5 per cent; an echocardiogram was normal, but the lower limb Doppler scan revealed chronic post-phlebotic changes in the right internal saphenous vein territory at the middle and distal thirds, associated with a small endoluminal thrombus; the patient was therefore discharged whilst continuing indefinite anticoagulation due to the unprovoked pulmonary embolism and deep vein thrombosis.

Conclusion

This case concludes that the infiltrate initially interpreted as organised pneumonia was the parenchymal manifestation of a pulmonary infarction, which was further complicated by a secondary infection and an organised parapneumonic pleural effusion; and that, as this was an embolism not associated with any identifiable major transient factor, it warrants indefinite anticoagulation [3,4]. A key point is that the initial misdiagnosis of the pulmonary lesion—which presented with pleuritic symptoms and haemoptysis—and its subsequent reinterpretation as a pulmonary infarction, illustrates that an infarction can mimic pneumonia, including organised pneumonia and/or a tumour on imaging; therefore, the clinical and radiological timeline and histological confirmation are crucial to avoiding inappropriate treatments [2].

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