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Atypical presentation of pulmonary aspergilloma in an immunocompetent individual: a case study and clinical conundrum

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Abstract

Pulmonary aspergilloma typically develops in pre-existing lung cavities, while hemoptysis is the most recognized complication, spontaneous pneumothorax in immunocompetent patients is exceptionally rare. We report a case of a 58-year-old immunocompetent male who presented with acute respiratory distress due to spontaneous pneumothorax. Chest imaging revealed an aspergilloma with cavity rupture into the pleural space. Despite chest tube drainage, persistent pneumothorax necessitated surgical intervention. The patient underwent conservative thoracotomy and received six months of voriconazole therapy, achieving complete recovery. This case highlights an unusual presentation of pulmonary aspergilloma, emphasizing the importance of considering fungal etiology in post-tuberculosis patients presenting with pneumothorax.

Key words: Chronic Cavitary Pulmonary Aspergillosis (CCPA), tuberculosis sequelae, spontaneous pneumothorax, immunocompetent patient.

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Introduction

The most dangerous complication of pulmonary aspergillosis is massive hemoptysis, which can be life-threatening and often requires urgent surgical intervention due to the risk of severe bleeding caused by the fungal ball's erosion of blood vessels in the cavity wall.[1]

The occurrence of spontaneous pneumothorax in a former chronic smoker with a tuberculosis history led to a discussion of diagnoses such as: relapse of tuberculosis, rupture of an emphysematous bulla, rupture of a cyst, pneumonia, pulmonary neoplasia, whereas in our case, the etiology was a rupture of a chronic aspergillary cavitary complex in the pleural cavity.[2]

According to global epidemiological studies analyzing the relationship between Pulmonary Tuberculosis (PTB) and Chronic Pulmonary Aspergillosis (CPA), the development of pulmonary cavities after PTB varies by geographical region, ranging from 21% in the United States to 35% in Taiwan/China. Of these patients with post-tuberculosis cavities, approximately 22% go on to develop CPA. The five-year period prevalence of CPA shows significant geographical variation, with Western European countries and the United States reporting less than 1 case per 100,000 population, while African nations like the Democratic Republic of the Congo and Nigeria demonstrate much higher rates at 42.9 per 100,000. Asian countries show intermediate prevalence rates, with China at 16.2 and India at 23.1 cases per 100,000 population over a five-year period.[3]

Interestingly, while various complications of pulmonary aspergillosis have been well-documented in the medical literature, pneumothorax is not typically recognized as a common sequela of ruptured mycetoma in non-immuno-compromised individuals.[4]

This observation raises intriguing questions about the physiopathological process of the Aspergillus infection in a non-immunocompromised individual and highlights the diagnostic and therapeutic difficulties encountered by the clinician.

Case Report

A 58-year-old ex-smoker, with a 40-pack-year smoking history, which stopped in 2019, a history of type 2 diabetes, and previously treated pulmonary tuberculosis (2019), with no recent TB exposure, presented with acute respiratory distress. The patient developed sudden-onset right pleuritic chest pain and severe dyspnea (mMRC grade 4) 24 hours before admission. The patient also reported a chronic cough, absence of fever, and a context of weight loss. This clinical presentation suggested several possible diagnoses, including pneumothorax, pulmonary embolism, and tuberculosis reactivation. While mentally alert with normal consciousness, physical examination revealed significant respiratory compromise with hypoxemia (90% oxygen saturation), tachypnea (22 breaths/minute), absent breath sounds, and vocal fremitus over the

right hemithorax. Hemodynamic parameters indicated physiological stress, with blood pressure elevated to 150/80 mmHg and tachycardia present at 107 beats/minute. Cardiac auscultation was normal, and examination of the lower extremities revealed no edema or tenderness, effectively ruling out certain differential diagnoses such as deep vein thrombosis.

A chest radiograph (Figure 1) was promptly performed, revealing a right-sided pneumothorax (black arrows) characterized by a hyperlucent area devoid of pulmonary vascular markings, with visible retraction of the visceral pleural line (red arrows) from the chest wall, immediate intervention with chest tube thoracostomy was initiated, however, despite a continuous and effective chest tube drainage for eight days, follow-up imaging demonstrated persistent non-expansion of the right lung.

This unexpected complication of prolonged pneumothorax, despite appropriate management, raised suspicion for underlying pathology impeding lung re-expansion, such as a bronchopleural fistula or endobronchial obstruction. The persistence of the pneumothorax warranted further investigation with advanced imaging modalities to elucidate the aetiology of the refractory pneumothorax and guide subsequent management strategies.

After eight days of continuous chest tube drainage without lung re-expansion, a CT scan was performed on day 9 of hospitalization (Figure 2), which revealed a well-defined cavitary lesion in the right lung, likely a sequela of the patient's previously documented pulmonary tuberculosis, within this cavity, a characteristic rounded opacity was observed, consistent with an aspergilloma (white arrow), the Computed Tomography (CT) scan also elucidated a communication between the cavitary lesion and the pleural space, evidenced by a defect in the visceral pleura (red arrow). This radiological finding provided a plausible explanation for the refractory pneumothorax observed in this case.

The initial laboratory results demonstrated leukocytosis with an elevated white blood cell count of 11.5×10^9/L (reference range: 4-10×10^9/L) and an elevated absolute neutrophil count of 9.1. Hemoglobin was within the normal range at 13.5 g/dL, and the platelet count was 385×10 µ/L. Electrolytes, liver function tests, and kidney function tests were all within normal limits. The patient's glycemic control was adequate, with a Glycated Hemoglobin (HbA1c) of 5.9 mmol/L.

Given the pulmonary cavity finding, the differential diagnosis included autoimmune disease, pulmonary embolism, septic emboli, infections, and congenital malformation. A comprehensive diagnostic workup was performed, including bronchoscopy with Bronchoalveolar Lavage (BAL), which showed chronic inflammation but was negative for acid-fast bacteria. Additional testing revealed negative Human Immunodeficiency Virus (HIV) and hep-

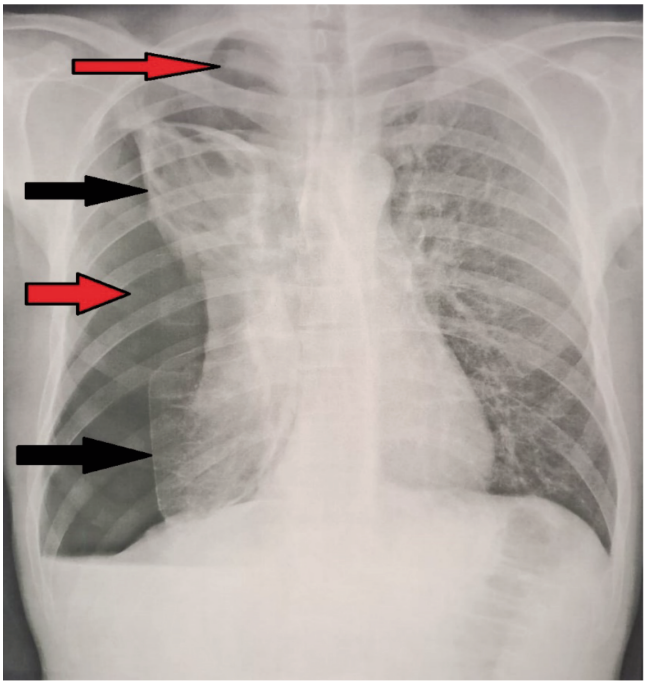

Figure 1. Chest X-ray showing a right-sided pneumothorax (red arrow) and the retracted pleural line (black arrow).

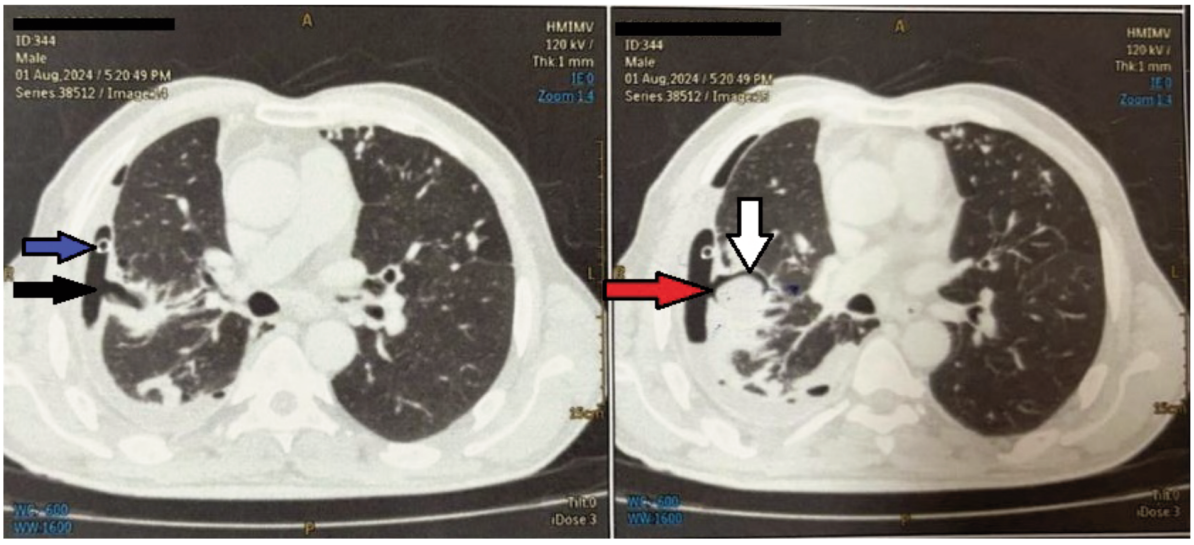

Figure 2. Chest Computed Tomography (CT) scan showing a right pulmonary cavity containing a rounded mass, consistent with an aspergilloma (white arrow), and a bronchopleural fistula identified by an interruption of the visceral pleura (red arrow), as well as the pneumothorax (black arrow) with a chest tube in place (blue arrow).

atitis panel results, normal immunoglobulin levels, and negative autoimmune markers (ANA, ANCA). The diagnosis of Chronic Cavitary Pulmonary Aspergillosis (CCPA) was ultimately confirmed through positive serum Immunoglobulin G (IgG) 21 g/L (reference: 7.0-16.0 g/L) and mycologic cultures positive for *Aspergillus fumigatus*.

Following the CT findings and the workup tests, a multidisciplinary team meeting was convened on day 10 of hospitalization, bringing together infectious disease specialists, pulmonologists, and thoracic surgeons. After careful consideration of the patient's clinical condition, imaging findings, and the persistent nature of the pneumothorax, the team reached a consensus that surgical intervention would be the most appropriate therapeutic approach.

On day 14 of hospitalization, the patient underwent surgery through a posterolateral right thoracotomy performed through the 5th intercostal space. This approach was chosen over Video-Assisted Thoracic Surgery (VATS) due to the complex pulmonary pathology, extensive pleural adhesions, and the need to thoroughly explore the bronchopleural fistula. Intraoperatively, dense pleural adhesions were encountered, particularly in the upper lobe region, requiring careful adhesiolysis. A wedge resection was performed, specifically targeting the right upper lobe region where the aspergilloma was located, with a 2 cm margin of healthy tissue around the cavitary lesion. The resection specimen measured approximately 6 × 4 cm, encompassing both the aspergilloma and the compromised visceral pleura. Total estimated blood loss was minimal (<200 mL). To minimize the risk of recurrence and future complications, a complete mechanical pleural abrasion was performed on the remaining visceral and parietal pleural surfaces. Two chest tubes (28F) were placed, one anterior and one posterior, to ensure adequate drainage and lung expansion.

The patient was treated with voriconazole (800 mg loading dose, followed by 400 mg daily maintenance dose for six months) for CCPA, with careful monitoring of potential side effects. This extended antifungal therapy was chosen to prevent pleural aspergillosis and ensure complete eradication of the infection.

During the 6-month follow-up period, the patient tolerated voriconazole therapy well without adverse effects. Regular clinical and radiological monitoring showed no evidence of disease recurrence, indicating successful treatment of the CCPA.

Discussion

Aspergillus infections and related complications, such as aspergilloma, primarily affect individuals with compromised immune systems or underlying lung diseases, rather than the general population who are regularly exposed to Aspergillus spores without developing serious health issues.[5]

Aspergillus species are capable of inducing chronic, non-invasive pulmonary infections characterized by a spectrum of overlapping clinical manifestations that vary widely depending on the host's immune status. The implications can vary from life-threatening, as is noted in invasive pulmonary aspergillosis and invasive rhinosinusitis seen in the severely immuno-compromised, to non-urgent in the case of small aspergillomas in the immuno-competent.[6,7]

The pathophysiological mechanisms leading to pneumothorax in pulmonary aspergilloma have been well-documented in the literature. First, the mechanical friction between the fungal ball and cavity wall leads to local tissue necrosis and erosion, second, Aspergillus species produce proteolytic enzymes that gradually weaken the cavity wall integrity, third, the local inflammatory response to the fungal infection causes tissue destruction and pleural compromise.

Aspergillus is notable for angioinvasion, with thrombosis of blood vessels causing tissue necrosis and allowing spread to distant sites, this explains the frequency and the gravity of hemoptysis as the first complication of aspergillosis.[8,9]

Besides hemoptysis, multiple complications of aspergillosis were described in the literature, like continued wheezing, lung fibrosis, respiratory failure, central nervous system infection, endocarditis, and death.[9]

Pneumothorax as a result of rupture of aspergilloma into the pleural space in non-immuno-compromised patients is extremely rare, it has been reported in granulocytopenic patients undergoing intensive cytotoxic therapy for hematologic malignancies.[9]

Adzic-Vukovic *et al*. reported a rare and unusual case of pneumothorax in an immunocompetent 40-year-old woman patient with chronic necrotizing pulmonary aspergillosis, she was immunocompetent according to the laboratory parameters, but clinically, she was immune-compromised with end-stage chronic obstructive pulmonary disease and cachexia, in our case, the patient was immunocompetent according to the clinical and laboratory parameters.[10]

Post-Tuberculosis (TB) sequelae can be categorized into parenchymal, airway disease, pleural/chest wall, vascular, and mediastinal. These residual changes can be minor however, some can be debilitating and even fatal.[10]

According to Denning *et al*., pulmonary cavities develop in 21-35% of pulmonary TB patients (with rates varying from 21% in the United States to 35% in Taiwan, China). Subsequently, approximately 22% of these patients progress to develop Chronic Cavitary Pulmonary Aspergillosis (CCPA).[3]

Many risk factors for CCPA probably exist, and they include some genetic defects, deficiency of surfactant A2, and toll-like receptor 4. In CCPA patients, cytokine production profiles typically show a Th2 cytokine profile, and gamma interferon production may be absent or poor.[11]

Denning and colleagues proposed the following criteria for diagnosing CCPA: one large cavity or two or more cavities on chest imaging with or without a fungal ball (aspergilloma) in one or more of the cavities (exclude patients with other chronic fungal cavitary lesions, e.g., pulmonary histoplasmosis, coccidioidomycosis, and para-coccidioidomycosis); and at least one of the following symptoms for at least 3 months: fever, weight loss, fatigue, cough, sputum production, hemoptysis, or shortness of breath; and a positive *Aspergillus* IgG with or without culture of *Aspergillus* spp from the lungs.[12,13]

The most sensitive microbiological detection method is the Aspergillus IgG antibody test, followed by sputum PCR, which shows higher sensitivity than culture. Definitive diagnosis can also be achieved through histopathological examination of tissue samples.[14]

In our case, the patient's anamnesis revealed weight loss progressing over 12 months, chronic productive cough with shortness of breath progressing over 4 months, but without fever and hemoptysis, the CT showed a typical aspect of fungus ball inside a cavity complicated with a bronchopleural fistula and a right pneumothorax and the diagnosis has been made on clinical, radiological and biological evidence (serum IgG for *Aspergillus fumigatus* and mycologic cultures were also positive for *A. fumigatus*)

When dealing with aspergilloma, surgeons weigh the risks of watching and waiting versus immediate treatment. While tricky, surgery is often the go-to solution. European experts recommend surgical removal of uncomplicated aspergillomas, preferably using

minimally invasive methods.[12] VATS is the top choice.[15] However, in our practice with the VATS approach, this case was too complex due to severe air leaks and extensive scarring, making an open surgery (conservative thoracotomy) necessary.

The occurrence of this complication in an immunocompetent individual highlights a significant diagnostic challenge in clinical practice. Aspergilloma-related pneumothorax may be underdiagnosed in immunocompetent patients as clinicians typically associate fungal infections with immune-compromised states. This bias can lead to delayed diagnosis and appropriate intervention, particularly in patients with structural lung disease such as post-tuberculosis cavities. We emphasize the importance of maintaining a high index of suspicion for fungal etiology in all patients presenting with spontaneous pneumothorax and underlying cavitary lung disease, regardless of immune status. Early recognition and appropriate intervention are crucial for optimal outcomes, as delayed diagnosis may lead to increased morbidity and potentially more complex surgical management.

Conclusions

This case highlights the importance of recognizing atypical presentations of pulmonary aspergilloma, even in low-risk patients. The successful individualized treatment and follow-up demonstrate the value of personalized care. Future research should focus on understanding fungal ball mechanics and documenting unusual manifestations to enhance our knowledge and improve patient outcomes.

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