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Case Report

Thrombotic Events and Pulmonary Thromboembolism as Primary Manifestation of Aspergillus Infection with Presence of Aspergilloma: A Case Report

Daniela Flores Hernandez[1*], Pérez-Millán Karla Janeth[2], Monroy-Meneses Carlos Eduardo[3], Ruiz-Gonzalez Samantha Lizeth[3], Carmona-Tapia Daniela Alejandra[3]

[1]Department of Internal Medicine, Toluca General Hospital, Institute of Security and Social Services of State Workers, Mexico
[2]Department of Diagnostic and Therapeutic Imaging, Centro Médico Lic, Arturo Montiel Rojas, Instituto de Seguridad Social del Estado de México y Municipios, México
[3]Department of Internal Medicine, General Hospital Toluca, Instituto de Seguridad y Servicios Sociales de los Trabajadores del Estado, México

***Corresponding Author:** Daniela Flores Hernandez
Department of Internal Medicine, Toluca General Hospital, Institute of Security and Social Services of State Workers, Mexico

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Abstract: Pulmonary aspergillosis is an opportunistic infection caused by species of the genus Aspergillus, encompassing a broad clinical spectrum, from saprophytic colonization to potentially fatal invasive disease. Among its chronic forms, aspergilloma represents an intracavitary fungal mass, usually associated with pre-existing pulmonary cavities due to tuberculosis, emphysema, or other structural lung diseases. Although classically considered non-invasive, it may cause severe complications in specific clinical settings. Pulmonary Aspergillus infection can present atypically, especially in immunocompromised patients. Thrombotic events, including pulmonary thromboembolism (PTE), have been described as rare but possible complications in these patients.

Keywords: Aspergilloma, Pulmonary Thromboembolism, Aspergillus Fumigatus, Invasive Fungal Infection, Voriconazole, Galactomannan, Aspergillus Infection, Thrombotic Events.

INTRODUCTION

Fungal lung infections caused by Aspergillus species are responsible for a spectrum of clinical entities, classified according to the host's immune response, the presence of structural lung diseases, and the degree of tissue invasion [1].

Pulmonary aspergillosis comprises a wide range of manifestations from colonization to severe invasive forms, depending on the patient's immunological status and the presence of pre-existing pulmonary cavities [1].

In immunocompromised patients or those with prior lung damage, a chronic form known as aspergilloma may develop, characterized by the formation of a fungal ball within pre-existing pulmonary cavities.

Aspergilloma typically forms within pre-existing cavities, most commonly secondary to tuberculosis, neoplasms, or surgical interventions, and is generally considered non-invasive. However, recent literature has reported atypical presentations such as local thrombosis or vascular damage, particularly in the context of angioinvasion by Aspergillus [2].

Aspergilloma is usually asymptomatic or may present with hemoptysis. In rare cases, it may be associated with systemic manifestations such as fever, dyspnea, or even thrombotic events. Recent studies suggest that fungal infections may induce a prothrombotic state through complex mechanisms

including endothelial activation, release of inflammatory cytokines, formation of neutrophil extracellular traps (NETs), and platelet dysfunction [3].

Pulmonary thromboembolism (PTE) is a potentially life-threatening complication, and its association with Aspergillus infections is uncommon but has been documented, particularly in immunocompromised patients. Serum antigen detection, such as galactomannan, may aid in the timely diagnosis of these infections [4].

Although the association between aspergilloma and thrombotic events is rare, recent case series have hypothesized that inflammatory and endothelial activation mechanisms may induce hypercoagulability, particularly in patients with risk factors such as diabetes, cancer, or cytotoxic treatments [3]. Therefore, the presence of an aspergilloma in a patient with PTE warrants a high index of suspicion for early diagnosis and comprehensive therapeutic management.

This case highlights an unusual presentation of Aspergillus infection with thromboembolic events as the primary manifestation, which should be considered in the differential diagnosis of patients with oncological history or immunosuppression.

CASE PRESENTATION

A 68-year-old male presented to the emergency department. He was a lawyer with a 5-year history of prostate cancer, previously managed with radical prostatectomy and partial bladder resection, and had undergone 19 cycles of chemotherapy. He was currently in remission. His past medical history also included a cerebrovascular accident nine months prior, with a modified Rankin score of 1, secondary to deep vein thrombosis, for which he was receiving apixaban 5 mg every 24 hours. He had also been diagnosed with type 2 diabetes mellitus three months earlier but had not started treatment.

He presented with swelling of the left lower limb, accompanied by severe pain rated 10/10, decreased strength in the left lower limb (Daniels 4/5), sudden-onset dyspnea, and tachycardia. On admission, his vital signs were: blood pressure 100/60 mmHg, heart rate 138 bpm, respiratory rate 21 bpm, temperature 36°C, and SpO2 86%. Physical examination revealed signs of respiratory distress, decreased chest wall motion, coarse bilateral basal crackles on auscultation, and decreased ventilation at the right apex. He required supplemental oxygen via nasal cannula at 2 L/min. The left lower limb exhibited marked edema (+++), increased warmth, and discoloration. A 12-lead ECG revealed sinus tachycardia. A posteroanterior chest radiograph showed a right apical cavitary lesion (Image 1).

Laboratory tests revealed thrombocytosis (platelet count 484,000), lymphocytosis (26,840) due to neutrophilia (22,000), D-dimer of 11,814, arterial blood gas with pH 7.50, PCO2 24, PO2 59, on FiO2 21%, PaFi ratio 280. Doppler ultrasound of the left lower limb showed chronic complete deep vein thrombosis and subacute superficial vein thrombosis. Internal medicine consultation led to hospital admission. Given suspicion of pulmonary thromboembolism and possible tuberculosis, a contrast-enhanced chest CT was performed, revealing pulmonary embolism in the posterior parahilar segment of the right lung and a lesion consistent with aspergilloma (Images 2 and 3).

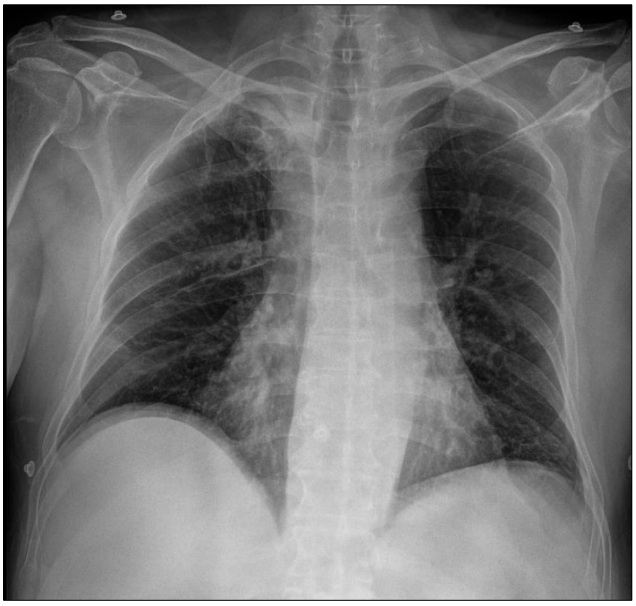

Image 1: Posteroanterior chest X-ray: A right apical ovoid lesion with irregular, partially defined radiopaque borders and a radiolucent center

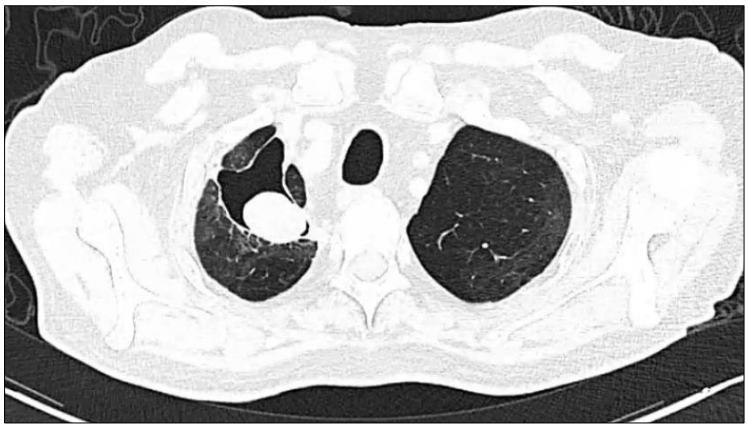

Image 2: Axial CT scan (lung window): A rounded, solid-appearing mass partially occupying a pulmonary cavity in the right apical segment (S1), separated from the cavity wall by an air crescent (air crescent sign), with focal areas of diffuse ground-glass opacities

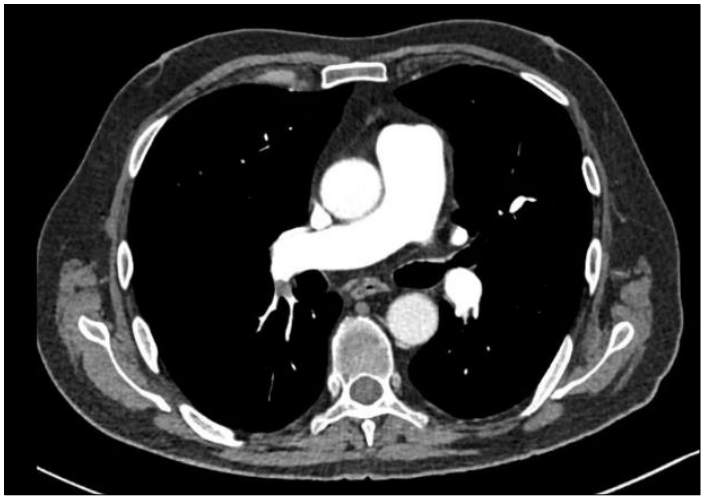

Image 3: Axial pulmonary CT angiography (mediastinal window): A partial filling defect is seen in the right middle lobar artery, with a hypodense image and obtuse margins following contrast administration; no other vascular abnormalities noted

Based on imaging findings, treatment with voriconazole 200 mg every 12 hours and enoxaparin was initiated. Galactomannan test was positive. After six days of treatment, symptoms improved and oxygen requirement decreased. At seven days, the patient no longer required supplemental oxygen. A follow-up chest CT showed a stable or reduced lesion (Image 4).

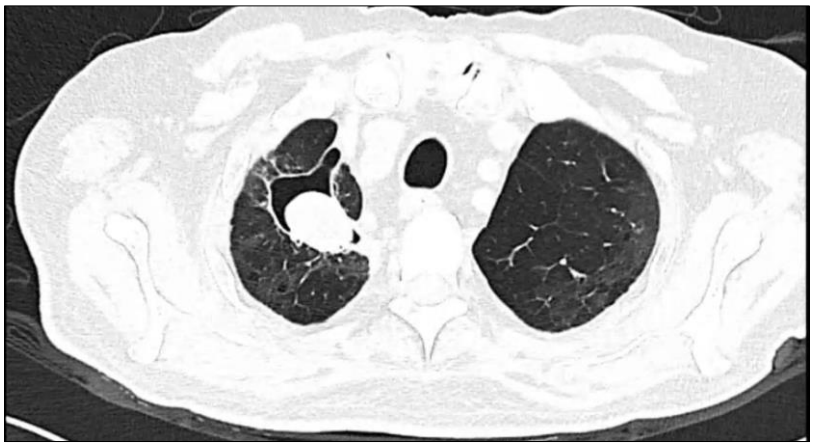

Image 4: Axial CT scan (lung window): Rounded, solid-appearing mass partially occupying a pulmonary cavity in the right apical segment (S1), with reduced size compared to prior imaging

Surgical intervention was deemed unnecessary. Outpatient treatment with voriconazole 200 mg every 12 hours and apixaban 5 mg every 12 hours was continued. The patient failed to attend follow-up visits and was lost to follow-up.

DISCUSSION

This case is notable for several clinically relevant reasons. First, it highlights the relationship between Aspergillus infection and thromboembolic events, a rare but plausible association. Although aspergilloma is generally considered a non-invasive form of aspergillosis, its presence in immunocompromised patients may facilitate dissemination or induce systemic inflammatory responses that promote thrombogenicity [5].

Aspergillus species can invade the vascular wall (angioinvasion), leading to local thrombosis, tissue necrosis, and endothelial damage [2]. This vascular injury promotes in situ thrombus formation and, in severe cases, distal embolization.

Additionally, the inflammatory response mediated by neutrophil extracellular traps (NETs) and pro-inflammatory cytokines contributes to a systemic prothrombotic state in invasive fungal infections [3].

In a recent report, Yanoma and Ugajin (2024) described a case of chronic aspergillosis secondary to pulmonary infarction, initially misdiagnosed as cryptogenic organizing pneumonia. Later, pulmonary thromboembolism was identified by contrast-enhanced CT and positive anti-Aspergillus IgG antibodies. The patient improved with anticoagulation and antifungal treatment [6].

Kamath *et al*., (2023) reported a post-COVID case in which a patient developed pulmonary aspergillosis and pulmonary hypertension associated with PTE, despite no prior immunosuppression. This shows that even immunocompetent individuals can present with this complication [7].

Furthermore, pulmonary vein thrombosis secondary to invasive aspergillosis was documented in a patient with thrombocytopenia, where antifungal treatment alone was sufficient to reverse the thrombosis, even without anticoagulation [2].

In this patient, multiple risk factors for thrombotic events were present: history of cancer, previous deep vein thrombosis, recent diagnosis of diabetes mellitus, and the prothrombotic state induced by fungal inflammation. Despite being on anticoagulation with apixaban, the patient developed a new PTE, which may also reflect pharmacologic resistance or poor adherence to treatment.

The CT finding of a right apical cavitation, along with a positive galactomannan assay, allowed identification of the probable underlying infectious cause. Galactomannan is a polysaccharide component of the Aspergillus cell wall, and its detection in serum or bronchoalveolar lavage has high specificity in compatible clinical contexts. Although its sensitivity is lower in non-neutropenic patients, its diagnostic value increases when combined with radiological findings [4].

Voriconazole, a second-generation triazole with excellent pulmonary penetration and activity against A. fumigatus, is the first-line antifungal treatment for chronic pulmonary aspergillosis. Its concurrent use with anticoagulation presents clinical challenges due to potential drug interactions; however, in this case, the patient tolerated both regimens without major adverse effects [1].

Literature supports immediate antifungal therapy when angioinvasion or vascular thrombosis by Aspergillus is evident, along with simultaneous management of thrombotic risk, despite potential pharmacologic interactions [2,3]. Clinical resolution after six days of medical management without surgery is notable, unlike other cases requiring surgical intervention or cavity drainage.

The decision to avoid surgery was appropriate given the patient's clinical stability, absence of massive hemoptysis or pleural invasion signs, and good response to medical treatment. However, outpatient follow-up remains essential, as the patient was lost to follow-up, limiting full assessment of long-term outcomes.

CONCLUSIONS

This case highlights the need for a high index of suspicion for fungal infections such as Aspergillus in patients presenting with atypical thromboembolic events, particularly in those with pre-existing pulmonary cavities or oncologic history.

The finding of aspergilloma in the context of PTE should prompt a comprehensive diagnostic approach, including imaging and fungal biomarkers. Treatment with voriconazole and anticoagulation may be sufficient for medical management, provided clinical and radiologic follow-up is ensured.

Loss to follow-up limits the evaluation of long-term outcomes and underscores the importance of strengthening outpatient care transition systems, especially for patients with complex pathologies and immunosuppression.

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