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Curvularia-Induced Allergic Bronchopulmonary Mycosis: A Rare Case of Non-Aspergillus Allergic Bronchopulmonary Disease

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Abstract: Allergic bronchopulmonary mycosis (ABPM) is a hypersensitivity-mediated pulmonary syndrome most commonly associated with Aspergillus fumigatus but rarely caused by dematiaceous fungi such as Curvularia species. We present a case of an 80-year-old male with myelodysplastic syndrome on azacitidine who developed a 19-month history of non-productive cough, bronchiectasis, mucus plugging, and marked peripheral eosinophilia (37%) with a total IgE of 1949 IU/mL. Sputum cultures and lung biopsy demonstrated Curvularia without parenchymal invasion, and pathology revealed allergic mucin with abundant eosinophils and fungal hyphal elements consistent with the Curvularia equivalent of ABPM. Treatment with systemic corticosteroids and itraconazole resulted in clinical improvement. This case highlights the importance of considering non-Aspergillus fungi in the differential diagnosis of ABPM and the diagnostic and therapeutic challenges posed by this uncommon entity.

Keywords: Curvularia; allergic bronchopulmonary mycosis; dematiaceous fungi; eosinophilia; myelodysplastic syndrome

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Highlights

- *Curvularia species* are a rare but recognized cause of allergic bronchopulmonary mycosis (ABPM), functioning as the dematiaceous fungal equivalent of classic allergic bronchopulmonary aspergillosis (ABPA).

- Diagnosis requires synthesis of clinical, microbiologic, and pathologic data; no validated serologic or antigen tests exist for Curvularia.
- Key diagnostic features include markedly elevated total IgE (>1000 IU/mL), peripheral eosinophilia, bronchiectasis with mucus plugging on CT, and allergic mucin with non-invasive hyphal elements on biopsy.
- Treatment with systemic corticosteroids and itraconazole in combination generally yields clinical improvement initially, but treatment failure is common. Serial total IgE levels can be followed as a surrogate marker of disease activity and treatment response.
- No standardized diagnostic criteria or treatment guidelines exist for non-*Aspergillus* ABPM; management requires extrapolation from the ABPA literature.

1. Introduction

Allergic bronchopulmonary mycosis (ABPM) is a hypersensitivity-mediated disease of the lower airways caused by colonization of the bronchial tree by filamentous fungi, triggering combined type I and type III immune responses. The prototypical and most extensively described form is allergic bronchopulmonary aspergillosis (ABPA), first reported by Hinson and colleagues in 1952 in the United Kingdom [1].

Non-*Aspergillus* ABPM is considerably less common and less well-characterized. A comprehensive global review by Chowdhary and colleagues identified more than 100 species of fungi implicated in non-*Aspergillus* ABPM, with *Bipolaris* species, *Candida albicans*, and *Schizophyllum commune* accounting for the majority of reported cases [2]. *Curvularia* species, dematiaceous (melanin-containing) fungi ubiquitous in soil, are well-recognized causes of allergic fungal sinusitis but only rarely implicated in ABPM [3,4]. Diagnosis is further complicated by the absence of validated serologic tests and the lack of standardized diagnostic criteria for non-*Aspergillus* ABPM [1].

We present an unusual case of *Curvularia*-induced ABPM in an elderly male with myelodysplastic syndrome (MDS), emphasizing the diagnostic complexity of this condition and describing the clinical, microbiologic, and pathologic features that guided management.

2. Case Presentation

An 80-year-old male was referred to an Infectious Diseases (ID) outpatient clinic for evaluation of a non-productive cough with a 19-month duration. His past medical history was notable for myelodysplastic syndrome (MDS) managed with azacitidine. He had a remote history of tobacco use and denied recent travel, alcohol, or illicit drug use.

Review of systems was positive for cough, five-pound unintentional weight loss, anorexia, nausea (attributed to chemotherapy), constipation, and fatigue.

Thirteen months prior to the current evaluation, the patient had undergone a CT scan of the chest followed by bronchoscopy with bronchoalveolar lavage (BAL) without biopsy; sputum culture at that time grew *Curvularia* species. One year prior to the ID evaluation, he was hospitalized for *C. difficile* infection (considered unrelated to the pulmonary findings). During that same period, lung biopsy was performed to evaluate a pulmonary nodule and persistent cough. Pathologic examination demonstrated dilated bronchioles with allergic mucin, abundant eosinophils, and fungal hyphal elements, without evidence of invasion into the lung parenchyma. No antifungal therapy was initiated at that time.

At the time of the ID evaluation, the patient's cough had worsened. A repeat CT scan of the chest demonstrated bronchial dilation, mucus plugging, bronchiectasis, and varying degrees of airway inflammation. Repeat bronchoscopy confirmed persistent isolation of *Curvularia* species on sputum culture.

On examination, the patient was afebrile and normotensive with a BMI of 18.94 kg/m^2. He was alert and well-appearing, thin, and breathing comfortably on room air without acute distress. Cardiovascular examination revealed a regular rate and rhythm without murmur or peripheral edema. Pulmonary examination demonstrated good air entry with scattered rhonchi bilaterally. There was no hepatosplenomegaly, lymphadenopathy, oral thrush, or skin rash.

Laboratory data were remarkable for a white blood cell count of 5.3 $\times$ 10^9/L with a profound peripheral eosinophilia of 37% (absolute eosinophil count of 1961 cells/μL). Total serum IgE measured 1949 IU/mL (reference range: <100 IU/mL).

A four-month course of prednisone combined with itraconazole was initiated. The patient's cough improved. Serial total IgE measurements were tracked throughout the clinical course and demonstrated a striking decline corresponding directly to symptomatic improvement. At treatment initiation, total IgE was 1949 IU/mL. Four months later, at completion of the initial treatment course, IgE had fallen to 387 IU/mL and continued to decline to 257 IU/mL one year after initiation.

Approximately 18 months after completing initial therapy, the patient developed a recurrent cough with peripheral eosinophilia (15%; absolute count of 400 cells/μL) and a rising IgE, peaking at 1712 IU/mL, at which point therapy was resumed. Eosinophils had risen to 1000 cells/μL. With reinstitution of treatment, symptoms improved and IgE declined sequentially, with a nadir of 115 IU/mL 8 months later.

3. Discussion

3.1. Curvularia and Phaeohyphomycosis

Curvularia belongs to a group of organisms collectively termed dematiaceous (phaeoid) fungi, defined by the presence of melanin in their cell walls, which imparts dark pigmentation to their conidia and hyphae. More than 100 species have been associated with human disease [4]. These fungi are worldwide in distribution, most commonly isolated from soil, and frequently encountered as laboratory contaminants, which can complicate clinical interpretation [4,5].

In tissue, dematiaceous fungi appear as irregularly septate hyphae and yeast-like forms; hyphae are generally more fragmented and irregular than those of *Aspergillus*. Tissue staining with Fontana–Masson stain (melanin-specific) is a critical adjunct to identification. There are no validated serologic or antigen-based diagnostic tests for *Curvularia* or other phaeoid fungi; therefore, diagnosis depends on culture combined with histopathological examination [4,5].

Melanin serves as a key virulence factor for dematiaceous fungi. It scavenges the free radicals and hypochlorite generated by phagocytic cells during the oxidative burst, thereby attenuating the host innate immune response. Additionally, melanin binds to hydrolytic enzymes at the plasma membrane, further impairing fungicidal activity [4,5].

3.2. Pathophysiology and Diagnosis of Allergic Bronchopulmonary Mycosis

ABPM is driven by type I and III hypersensitivity responses to fungal antigens colonizing the bronchial mucosa. The cardinal histologic feature is eosinophilic (allergic) mucin harboring fungal hyphae within the bronchi, in which extracellular trap cell death (ETosis) of eosinophils induced by viable fungi is believed to play a central role [1]. Clinically, ABPM is characterized by peripheral blood eosinophilia, elevated total serum IgE, and characteristic CT findings including central bronchiectasis and mucus plugging [1,6].

Among non-*Aspergillus* causes, *Bipolaris* and *Curvularia* species are most frequently reported as triggers of allergic airway disease. The relatively large spore size of these organisms (20–30 μm $\times$ 8–12 μm for *Curvularia*, compared to 2–3 μm for *Aspergillus*) may affect aerodynamic deposition in

the airways and contribute to their allergenic potential, although the precise immunologic mechanisms remain incompletely understood [2,3].

The diagnostic criteria for ABPM remain a subject of ongoing refinement. Established criteria for ABPA include asthma, positive fungal skin tests, elevated total serum IgE, *Aspergillus*-specific IgE, and proximal bronchiectasis [6]. These criteria were designed for *Aspergillus* and do not translate directly to non-*Aspergillus* ABPM. The International Society for Human and Animal Mycology (ISHAM) working group has proposed revised criteria requiring elevated total IgE (>1000 IU/mL) as a mandatory criterion, with at least two of three additional components: cutaneous hypersensitivity or elevated specific IgE to the implicated fungus, relevant radiographic findings, and peripheral blood eosinophilia [1,7]. In our patient, the total IgE of 1949 IU/mL together with marked eosinophilia, bronchiectasis with mucus plugging on CT, pathologic demonstration of allergic mucin with eosinophils and hyphal elements, and repeated *Curvularia* isolation collectively supported the diagnosis of *Curvularia*-induced ABPM.

3.3. Differential Diagnosis

The clinical presentation prompted a broad differential diagnosis. Leading considerations were ABPM with *Curvularia* as the causative organism (the functional equivalent of ABPA) and classic ABPA itself. Beyond allergic fungal disease, the differential encompassed *Curvularia* colonization without true infection, bronchiectasis with recurrent bacterial superinfection, recurrent aspiration, pulmonary neoplasm, pulmonary tuberculosis, eosinophilic pneumonia, and asthma with fungal sensitization. Given the patients underlying MDS and five-pound weight loss, pulmonary neoplasm was also considered, as was pulmonary tuberculosis, eosinophilic pneumonia, and cystic fibrosis (less likely given age). Rheumatologic or autoimmune disease and medication-associated pulmonary toxicity from azacitidine rounded out the differential. The latter was given particular attention, as azacitidine has been associated with eosinophilic lung disease [8]; however, the chronology of symptoms preceding azacitidine initiation and the pathologic identification of allergic mucin with fungal hyphal elements on biopsy were felt to be more consistent with ABPM than drug toxicity.

3.4. Treatment Considerations

The management of ABPM rests on two principal strategies: suppression of the eosinophilic inflammatory response with systemic corticosteroids, and reduction in fungal burden with antifungal therapy [9,10]. Systemic corticosteroids are the cornerstone of treatment and have been shown to reduce total serum IgE by 35–50% over 6–8 months. However, corticosteroid therapy alone may not prevent exacerbations or long-term decline in lung function.

For ABPA, two standard corticosteroid protocols have been described: high-dose prednisolone (0.75 mg/kg/day for 6 weeks, then taper) or medium-dose prednisolone (0.5 mg/kg/day for 2 weeks, then taper) [1]. These regimens achieve remission and reduce IgE levels but require careful monitoring, particularly in immunocompromised patients.

Among antifungal agents with activity against *Curvularia*, itraconazole and voriconazole demonstrate the most consistent in vitro activity [4,5]. Fluconazole is not reliably active against this organism, and amphotericin B retains activity for invasive disease. Echinocandins have higher minimum inhibitory concentrations against dematiaceous fungi compared to *Aspergillus* but may retain clinical utility [4,5]. No standard of care has been established, and treatment decisions must be individualized.

The likelihood of treatment success in patients treated for Aspergillus-related ABPM is sobering. Patients treated with steroids alone face an 81% likelihood of failure to control symptoms [11]. Antifungals may improve the odds of symptom control by just over 2-fold but leave patients with an expected treatment success of 46%. The largest ABPA clinical trial defined disease control as a decrease in steroid use by at least 50%, a 25% decrease in general serum IgE, and at least one

of the following criteria: resolution of pulmonary infiltrate or improvement of exercise tolerance or pulmonary function tests by at least 25% [11]. Clinicians might apply these parameters while treating patients with ABPM. Admittedly, data for the treatment of Curvularia-associated ABPM is limited, as it is less well-described in the literature [2]. Extrapolating and applying lessons learned from the ABPA literature should prepare treating physicians for a protracted course of treatment and monitoring.

With this being said, hope exists in emerging treatments. Patients initially treated with corticosteroids and antifungal agents but with incomplete response have responded well to anti-IgE therapy with omalizumab, followed by the anti-IL5R agent benralizumab [12]. Additionally, dupilumab has shown significant improvement in symptoms and imaging in patients with failure of itraconazole and benralizumab [13].

In our patient, a four-month course of prednisone combined with itraconazole resulted in symptomatic improvement with resolution of cough. The observed decline in total IgE over the treatment course corresponded directly with clinical improvement, reinforcing the value of serial IgE monitoring as a surrogate marker of disease activity and response to therapy [1,6]. After completion of treatment, the ABPM recurred, necessitating additional treatment.

3.5. The Role of Underlying Immunosuppression

This case is complicated by the patient's underlying MDS and ongoing treatment with azacitidine, a hypomethylating agent. Azacitidine has been associated with pulmonary adverse effects including eosinophilic pneumonitis [8]. While the timeline and pathologic findings argue strongly for ABPM rather than drug toxicity in this case, the immunologic milieu created by MDS and cytotoxic therapy may have contributed to impaired fungal clearance or altered the inflammatory response. MDS itself is associated with immune dysregulation, potentially permitting prolonged *Curvularia* airway colonization and facilitating hypersensitivity.

3.6. Conclusion

Curvularia-induced ABPM represents a diagnostically challenging entity for several reasons. First, *Curvularia* is a common environmental contaminant, and its isolation from respiratory specimens must be interpreted in a clinical context. Second, there are no validated serologic tests to confirm sensitization, and skin testing with fungal antigens is not routinely available in most clinical settings. Third, the diagnostic criteria used for ABPA have not been formally validated for non-*Aspergillus* pathogens, creating diagnostic uncertainty [1,7]. Lastly, the lack of a standard treatment protocol requires clinicians to extrapolate from the ABPA literature and individualize care.

Heightened clinical awareness of ABPM caused by non-*Aspergillus* dematiaceous fungi is essential to avoid diagnostic delay. In any patient presenting with chronic cough, bronchiectasis, mucus plugging, peripheral eosinophilia, and a markedly elevated total IgE, ABPM should be considered even when sputum cultures yield organisms not classically associated with this syndrome.

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