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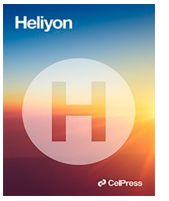

Case report

Curvularia lung infection mimics malignancy

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ABSTRACT

Curvularia rarely causes human infections despite its ubiquity in the environment. It is most associated with allergic diseases such as chronic sinusitis and allergic bronchopulmonary mycosis; however, causing a lung mass is rarely reported in the literature. We describe an interesting case of a 57-year-old man with a history of asthma and localized prostate cancer diagnosed with a *Curvularia*-caused lung mass that responded quickly to itraconazole.

1. Introduction

Curvularia is a dematiaceous fungus widely distributed in soil but rarely infects humans [1,2]. Due to the presence of melanin in their cell wall, dematiaceous fungi have dark-pigmented conidia and hyphae. More than 100 species of dematiaceous fungi have been linked to human disease [1]. These fungi can infect both immunocompetent and immunocompromised individuals. Clinical syndromes range from cutaneous infection to potentially fatal disseminated disease [1]. The diagnosis is made by culture and histopathological examination; there are no serological or antigen tests to detect these fungi [1]. They appear in tissues as yeast-like forms that can be solitary or in short chains, and/or irregularly septate hyphae [1–3]. *Curvularia* is frequently associated with eosinophilia and allergic diseases; however, causing a solitary lung mass without disseminated infection is uncommon [2]. We present a case of a solitary lung mass that mimicked malignancy and was found to be caused by *Curvularia* and responded quickly to itraconazole.

2. Case presentation

A 57-year-old man was evaluated in the surgery clinic for supraumbilical left paramedian incisional hernia and possible hernia repair following robotic prostatectomy. Computed Tomography (CT) of the abdomen and pelvis revealed bilateral small pulmonary nodules in the lung bases, so he was referred to the pulmonary clinic to evaluate these pulmonary nodules and optimize respiratory status before hernia repair.

The patient had a history of hypertension, asthma since childhood, a former smoker who quit three years ago (30 pack-year), chronic obstructive pulmonary disease (COPD), and localized prostate cancer (p T2N0 with positive margins), which was treated by robot-assisted laparoscopic prostatectomy (RALP) three years ago.

The patient reported shortness of breath and dry cough when exposed to allergens like dust, cutting grass and trees, and hot weather. He had been using short-acting bronchodilators (Levalbuterol) 1–2 times per day on average. However, the patient denied any persistent productive cough, fever, hemoptysis, and night sweats. He had no recent travel history, and no use of intravenous or

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inhalational drugs.

His vital signs were stable, and his physical examination was unremarkable except for supraumbilical left paramedian incisional hernia.

CT chest without contrast revealed a lobulated solid partially calcified mass in the right upper lobe, compromising the upper lobe bronchial system with distal tree-in-bud abnormality, bilateral small lung nodules, and no hilar or mediastinal lymphadenopathy (Fig. 1).

Laboratory work-up revealed normal total white blood cells of 6 K/μL with 7.9% eosinophils, normal *C*- reactive protein (<0.29 mg/dl), and IgE level of 799 IU/ml, a negative aspergillus antibody by complement fixation (<1:8), normal prostate-specific antigen (PSA) (<0.01 ng/ml), a negative purified protein derivative (PPD) test, and a negative HIV test.

Positron emission tomography (PET) scan showed irregular lobulated right upper lung mass with faint or mild irregular fluorodeoxyglucose (FDG) uptake, scattered bilateral tiny lung nodules with no avid FDG uptake, no other abnormal foci of increased FDG in the remaining region of the body.

Bronchoscopy showed abnormal endobronchial tissue in the right upper lobe posterior segment with some distal narrowing. Bronchoalveolar lavage (BAL) and endobronchial biopsies were performed. Fine needle aspiration (FNA) showed mixed inflammatory cells, including numerous eosinophils and Crystalline debris with no malignant cells. Pathology showed benign respiratory mucosa with acute and chronic inflammation with lymphocytes, plasma cells, neutrophils, and eosinophils. No fungal elements were seen, and PAS and GMS were both negative. Two BAL fungal cultures grew Curvularia species after nine days of incubation which was identified by growth pattern, colony morphology, and lactophenol cotton blue stain. Gram stain and routine BAL cultures did not show any microorganisms. Three acid-fast bacilli (AFB) smears and cultures were negative.

Since no microorganisms were found apart from *Curvularia* and the PET scan revealed no avid intake, we believe *Curvularia* was the most likely cause of this lung mass. The patient was given itraconazole, and a CT chest scan four weeks later revealed that the lung mass had disappeared completely (Fig. 2). There was no mass recurrence on the CT chest at the one- and two-year follow-up intervals (Figs. 3 and 4, respectively).

3. Discussion

We initially assumed that the lung mass, found in our case, was caused by a cancer due to the patient's history of COPD and Prostate Cancer, but the PET scan revealed that no avid intake, and the histopathology showed no definitive evidence of malignancy, thus cancer was excluded. Furthermore, a mucus plug as another causing potential was also excluded as it would resolve quickly after a bronchoscopy, but the lung mass was repeatedly visible in several radiological imaging over a course of five months as well as in the chest X-ray taken immediately after the bronchoscopy. Another possibility was that a *Curvularia* was only colonization based on the fact that the histopathology did not show any evidence of fungal elements. However, the absence of other microorganisms in the microbiological cultures, combined with the rapid response to Itraconazole, led us to assume that Curvularia was the true pathogen.

Lung infections caused by *Curvularia* presenting as a consolidation or mass are rarely reported in the literature and have been reported in a few cases and most commonly as a part of disseminated disease [2].

The most common manifestation of lung involvement by *Curvularia* is allergic bronchopulmonary mycosis (ABPM) which is commonly reported in patients with asthma or cystic fibrosis. Criteria for ABPM are not established, but high IgE level, positive skin test for fungal allergens, positive culture or serology for *Curvularia*, and response to systemic steroids may suggest that [1]. We considered ABPM in our patient due to a history of asthma and the presence of bronchiectasis in imaging, but the patient was not severely symptomatic; the mass was discovered incidentally, and the IgE level was less than 1000 IU/ml measured twice, initially 799 IU/ml, then 192 IU/ml, so we decided not to give him a systemic steroid and instead optimized his bronchodilator therapy by adding inhaled corticosteroid.

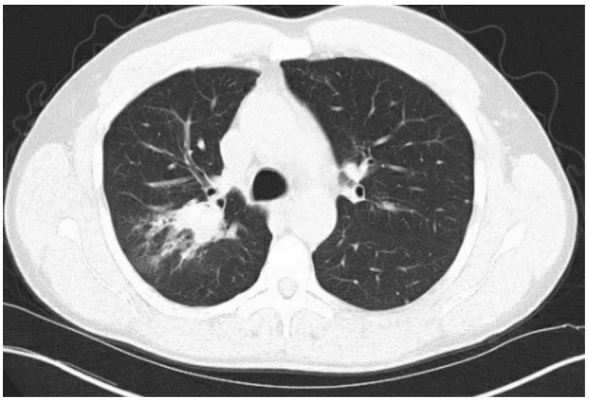

Fig. 1. An axial chest CT scan shows a 2.9 × 2.6 cm lobulated mass in the right upper lobe with focal calcifications compromising the upper lobe bronchial system with distal retained material and tree-in-bud abnormality.

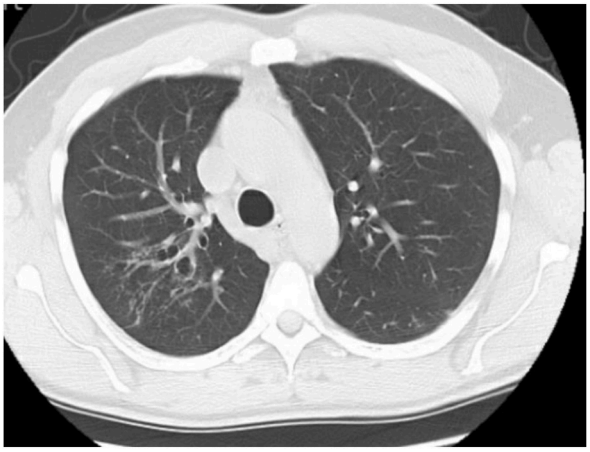

Fig. 2. After 4-week of treatment, an axial chest CT scan shows interval resolution of the right upper lobe mass with residual cluster bronchiectasis, tree-in-bud abnormality, and bandlike parenchymal scars.

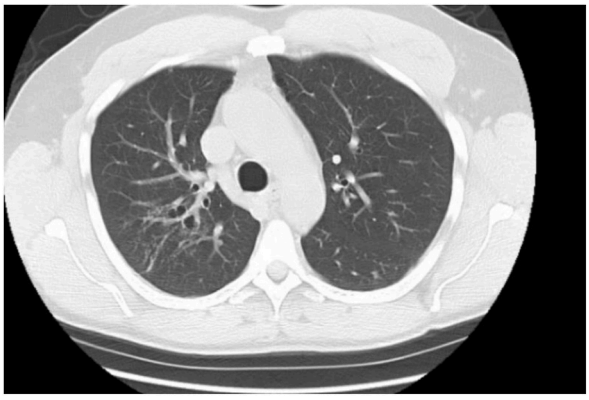

Fig. 3. At a one-year follow-up, an axial chest CT scan shows no focal mass in the right upper lobe, with residual focal bronchiectasis.

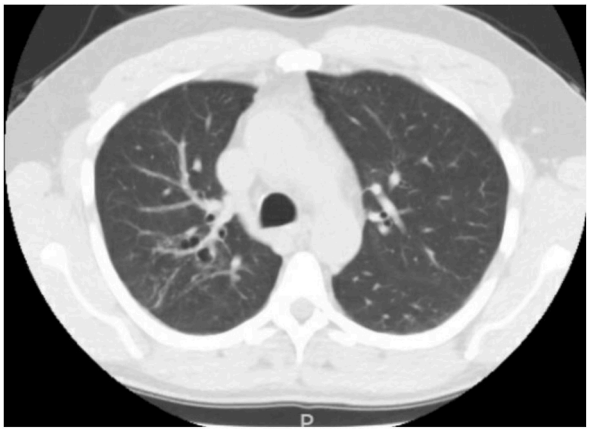

Fig. 4. At a two-year follow-up, an axial chest CT scan shows no focal mass in the right upper lobe, with residual focal bronchiectasis.

The optimal treatment of *Curvularia* infection is still undetermined. Most localized diseases like sinusitis did well with surgery alone even without antifungal therapy, and the disseminated disease did poorly even with systemic antifungal therapy [2,4]. Also, the best antifungal agent for these fungi is still not determined, but amphotericin and azoles have been used with variable responses [2–4]. The length of therapy is generally based on the clinical response and ranges from several weeks to several months or longer [1].

Author contributions

Nour Daoud: Conceptualization, Data curation, Writing - original draft, Writing - review & editing.
Neerja Gulati: Conceptualization, Supervision, Writing - review & editing.

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Consent

Written informed consent for publication was taken from the patient.

Author contribution statement

All authors listed have significantly contributed to the investigation, development and writing of this article.

Data availability statement

Data will be made available on request.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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