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Phanerochaete chrysosporium and granulomatous lung disease in a mulch gardener

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Abstract

A 50-year-old woman who gardens regularly with rotting bark mulch presented with exertional dyspnea, diffusion impairment, and radiographic abnormalities (centrilobular nodules, tree-in-bud and ground glass opacities, calcified mediastinal and hilar lymph nodes) on a computed tomogram. Moderate lymphocytosis was noted on bronchoalveolar lavage. Surgical biopsy of her lung revealed granulomatous changes, and biopsies grew *Phanerochaete chrysosporium*, a fungus that causes white rot in tree bark. She was treated with voriconazole and instructed to avoid gardening, which led to radiographic and symptomatic improvement. She had recurrence of symptoms when she started doing yard work again. *P. chrysosporium* has been demonstrated to cause hypersensitivity pneumonitis in animal models. This case is the first documented report of *P. chrysosporium* associated with granulomatous lung disease in a human.

Introduction

We report recovery of a fungus in a patient with granulomatous lung disease.

Case Report

In June 2010, a 50-year-old woman with abdominal complaints received a computed tomogram (CT) of her abdomen with incidentally noted tree-in-bud opacities in the basal segments of both lungs. These abnormalities prompted a dedicated CT of the chest (Fig. 1), which demonstrated numerous, bilateral, ill-defined centrilobular nodules with basilar predominance, tree-in-bud and ground glass opacities, and multiple calcified mediastinal and hilar lymph nodes. Her past medical history was significant for ulcerative colitis, treated by proctocolectomy 15 years ago, with no further symptoms of inflammatory disease or need for immunosuppressive therapy. She complained of mild dyspnea when ascending a flight of stairs, but no dyspnea exercising on level ground, and no other respiratory complaints, such as cough or wheeze. Her only medications were antidepressants. She had never smoked, drank alcohol, or used recreational drugs. Her travel history included South America and extensive travel in the United States. She had no pets. She is an avid gardener, and gardens daily. Over the past 2 years, she had been digging in rotting bark mulch.

Initial examination was unremarkable, except for a room air SpO2 of 88% (at 1400 m altitude). Spirometry was normal (forced expiratory volume at 1 s [FEV1] = 2.91 L, 97% predicted; forced vital capacity [FVC] = 3.64 L, 96% predicted, FEV1/FVC = 80%), and diffusing capacity of lung for carbon monoxide (DLCO) was mildly reduced (22.27 mL/mmHg/min, 78% predicted). Serologic testing for occult collagen vascular disease was negative. Bronchoscopic evaluation revealed normal airways. Bronchoalveolar lavage revealed moderate lymphocytosis (68% macrophages, 24% lymphocytes, 8% bronchial lining cells), with no microscopic evidence for malignancy, pneumocystis, fungal or viral infection. Lavage culture grew a *Penicillium* species.

The patient underwent video-assisted thoracoscopic surgery of the right lung, with biopsies of the upper, middle, and lower lobes. The pathologic examination revealed numerous noncaseating granulomata and mild to moderate emphysematous changes (Fig. 2). While no fungal elements were seen on histopathologic examination, the biopsy cultures rapidly grew *Phanerochaete chrysosporium* from the upper and lower lobes (Fig. 3).

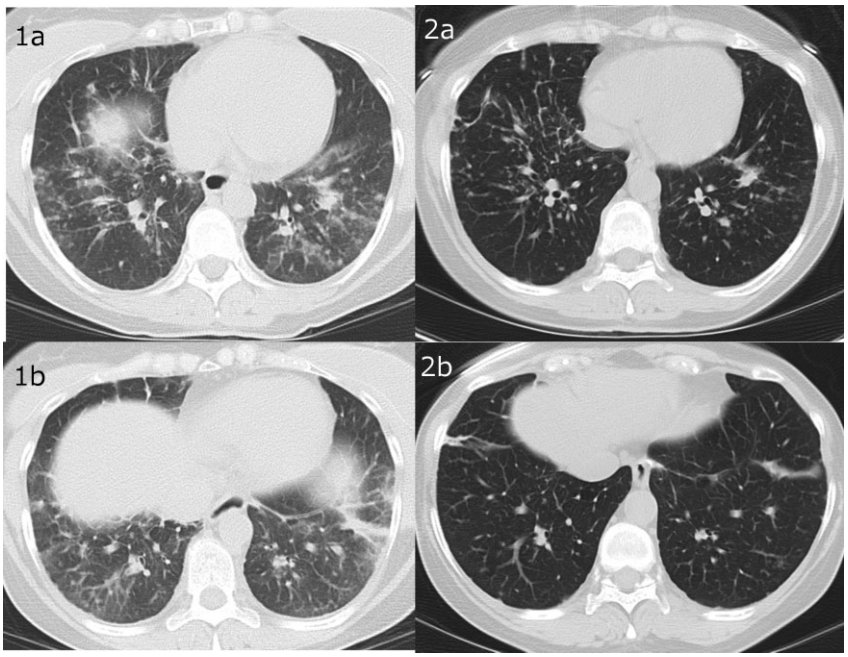

Figure 1. Computed tomograms at presentation (1a and 1b) and after initiation of antifungal therapy and avoidance of gardening (2a and 2b).

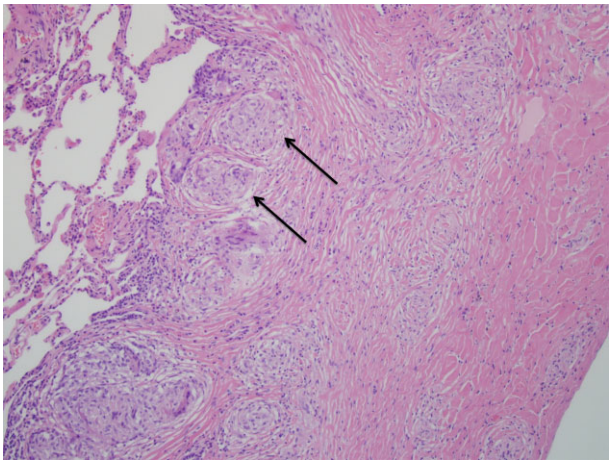

Figure 2. Surgical biopsy of right lung. Noncaseating granulomata are seen (arrows) near the airways.

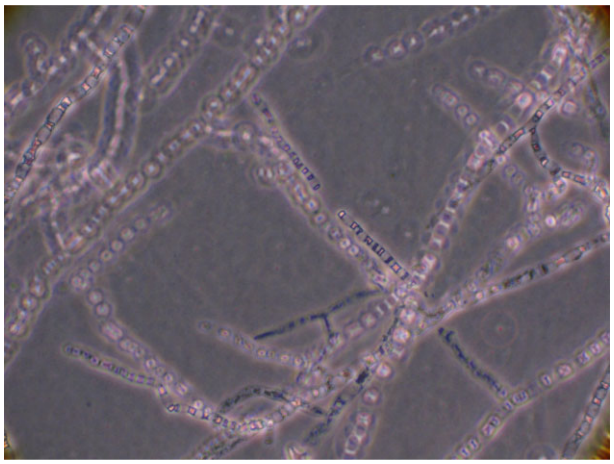

Figure 3. *Phanerochaete chrysosporium*, grown from the patient's lung biopsy.

Despite no evidence for invasive fungal infection, the patient was empirically treated with 6 months of oral voriconazole, 200 mg p.o. b.i.d., and was instructed to stop gardening. She reported mild improvement in symptoms during the first 2 months of antifungal therapy. A repeat CT of the chest at that time demonstrated significant improvement in ground glass opacities, with persistent nodules and calcified lymph nodes. After a total of 6 months of antifungal therapy, the patient had no significant change in spirometry or DLCO. The voriconazole was discontinued, and prednisone was initiated (20 mg daily). The prednisone was tapered off after 1 month with no additional improvement in spirometry, DLCO, or symptoms.

She returned to clinic in August 2011 with complaints of worsened dyspnea over the preceding 2 weeks, which was associated with her mowing and edging her lawn, although no significant change was noted in spirometry or DLCO. Since that time, she has not interacted significantly with bark mulch, and she is asymptomatic at the time of writing this manuscript.

Discussion

P. chrysosporium, a fungus found in decaying wood, has not been reported as a pathogen in humans before, although inhalation of its spores has been reported to cause hypersensitivity pneumonitis in animal models, with granuloma formation and alveolar wall thickening [1, 2]. This fungus grows in moderate to higher temperatures, and is found in forests of North America, Europe, Asia, and Africa [3]. This organism has received particular attention because of its ability to biodegrade phenolic resin plastics through extracellular enzymes, and has been suggested for industrial use to degrade plastics [4]. We believe this patient's lung disease may represent either fungal infection or subacute hypersensitivity pneumonitis as a result of recurrent exposure to this fungus while gardening with rotting wood mulch. She had compatible clinical, radiographic, and histopathologic findings, lymphocytosis on lavage, radiographic, and symptomatic improvement with administration of an antifungal and avoidance of gardening, and recurrence of symptoms when she resumed yard work.

Hypersensitivity pneumonitis is the result of a cell-mediated immune response of the lung to an inhaled antigen. Diagnosis requires obtaining a thorough exposure history. Radiographic findings include diffuse micronodules, ground glass attenuation, and focal air trapping. Histopathology often demonstrates poorly defined granuloma formation [5]. Once identified, avoidance of the offending agent is typically sufficient for management. Hypersensitivity pneumonitis is difficult to recognize when subacute or chronic in presentation, as it may have a more insidious presentation and an offending agent may be difficult to isolate. A high clinical suspicion is required for diagnosis. Because hypersensitivity pneumonitis is a complex syndrome with a variable clinical presentation, it can often be confused with other disease states, such as idiopathic pulmonary fibrosis, nonspecific interstitial pneumonitis, and sarcoidosis. Because the patient's granulomata were well defined, we favor a diagnosis of fungal infection over subacute hypersensitivity pneumonitis. We believe sarcoidosis is less likely due to the radiographic and symptomatic improvement that occurred with antifungal administration and avoidance of gardening, the absence of further improvement with administration of steroids, and the recurrence of symptoms with return to gardening. A low CD4/CD8 ratio on bronchoalveolar lavage may have aided in diagnosing hypersensitivity pneumonitis, but was not performed due to an initially low clinical suspicion for hypersensitivity pneumonitis at the time of bronchoscopy.

If, in the future, this organism is applied in a widespread fashion to degrade plastics, it would be important to recognize its potential as a human pathogen.

Disclosure Statements

No conflict of interest declared.

Appropriate written informed consent was obtained for publication of this case report and accompanying images.

Acknowledgment

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