Case Report

Hypersensitivity Pneumonitis from *Pezizia domiciliana*
A Case of *El Niño* Lung

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A previously healthy woman developed severe dyspnea and was found to have restrictive lung disease and evidence of alveolitis. Open lung biopsy revealed extrinsic allergic alveolitis (hypersensitivity pneumonitis). The etiology was not initially apparent, but a home inspection showed an unusual mushroom growing in the patient's basement. Air sampling and serum precipitins against the fungal antigens confirmed that *Pezizia domiciliana* was the cause of the patient's hypersensitivity pneumonitis. This is the first described case of hypersensitivity pneumonitis cause by *P. domiciliana.* We speculate that unprecedented rainfall and flooding of the patient's basement as a result of *El Niño* rains produced ideal factors for the growth of this fungus. Wright RS, Dyer Z, Liebhaber MI, Kell DL, Harber P. Hypersensitivity pneumonitis from ***Pezizia domiciliana***: a case of ***El Niño*** lung.

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The winter of 1997–1998 has received considerable worldwide attention because of the weather phenomenon known as *El Niño*. This unusual weather pattern resulted in significant flooding in Western states of the United States and caused great property damage and personal loss. In the Santa Ynez Mountain Range of Southern California, total precipitation reached an unprecedented level (1). Not surprisingly, there has been some speculation on the health effects of this weather pattern (2, 3). *El Niño* has led to an unusual illness for the area of Santa Barbara and Southern California. We report the case of a patient in whom hypersensitivity pneumonitis was probably secondary to the excessive precipitation and flood damage to her home from *El Niño,* with consequent growth of an unusual fungus, *Pezizia domiciliana.*

CASE REPORT

A 54-yr-old woman presented with a 3-mo history of progressive dyspnea. She denied having fevers, chills, or weight loss. A chest radiograph showed discoid atelectasis, and a ventilation–perfusion lung scan was normal. Albuterol and triamcinolone acetonide given by metered-dose inhaler did not relieve the patient's dyspnea. At the time of her pulmonary evaluation, she was dyspneic after walking 50 ft. She had smoked cigarettes 30 yr earlier. She worked as a schoolteacher, and she denied having any unusual avocations or pet exposures. Examination showed a well nourished woman with a blood pressure of 120/70 mm Hg, pulse of 72 beats/min, and respiratory rate of 24/min, and bibasilar dry crackles.

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Measurement of oxygen saturation (Sa_{O_2}) by pulse oximetry gave a normal result at rest, but Sa_{O_2} dropped rapidly below 85% while the patient was ambulating. Pulmonary function tests showed a mildly reduced TLC (78% predicted by plethysmography) and a markedly reduced diffusing capacity for carbon monoxide ($D_{L_{CO}}$) (14% predicted after correction for alveolar volume). An echocardiogram was normal. A repeat chest-radiographic examination showed a mild increase in lung density in the middle and lower lung fields. High-resolution chest computed tomography (CT) showed ground-glass haziness and small centrilobular nodules (Figure 1).

An open lung biopsy showed patchy interstitial pneumonitis with a moderate lymphoplasmacytic infiltrate and peribronchiolar and interstitial nonnecrotizing granulomas (Figure 2). These findings were consistent with extrinsic allergic alveolitis. Because of the severity of the patient's disease, prednisone therapy (60 mg/d) was initiated.

Subsequent discussions with the patient revealed that the basement of her home had been flooded from *El Niño* rains and that mushrooms had been growing in the crevices of the basement floor. A home inspection by her physicians revealed considerable water damage to the basement area. The cement slab floor in the basement had been cracked by pressure from the roots of an adjacent tree, allowing flooding of up to 2 in. on several occasions during the winter and spring. Densely grouped pale yellow, tan, and brown cuplike fruiting bodies, from 1.5 to 3 cm in diameter, were observed growing out of the mortar of the lower course of bricks on the fireplace and between the junction of the cement slab floor and concrete blocks (Figure 3). The fungus was identified as a member of the class Ascomycetes, subclass Discomycetes (cup fungi), of the genus *Pezizia* and species *domiciliana* (4, 5). A daughter and grandaughter of the patient were living in this part of the house but were asymptomatic.

Air sampling with a Burkard Personal Sampler (Burkard Manufacturing, Ltd., Rickmansworth, UK) revealed significant

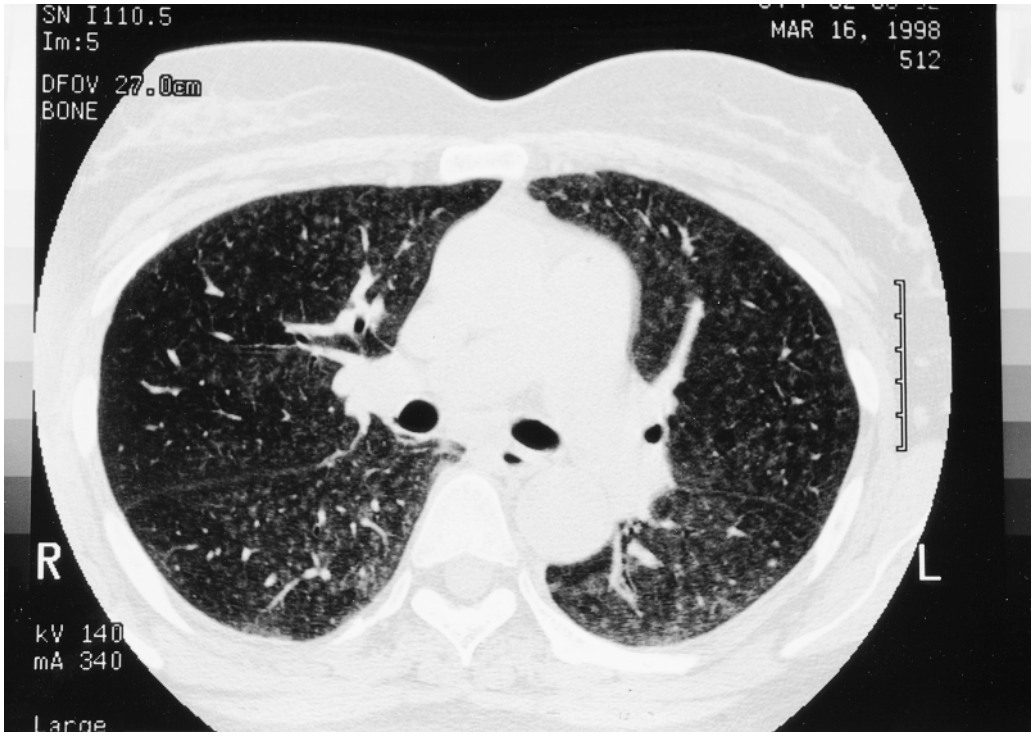

Figure 1. High-resolution chest CT scan of patient made before treatment, showing centrilobular nodules consistent with hypersensitivity pneumonitis (9).

contamination of ambient air with spores from *P. domiciliana* as well as other common spores (Table 1). The patient's serum was tested for precipitating antibodies to *P. domiciliana* and other common antigens through the micro-Ouchterlony gel double-diffusion technique (6) (IBT Reference Laboratories, Lenexa, KS). Her serum tested positive for precipitins to *P. domiciliana*, but was negative for preciptins to *Thermoactinomycetes vulgaris*, *Micropolyspora faeni*, *Aspergillus fumigatus* #1 and *A. fumigatus* #6, and *Aureobasidium pullulans*. Control sera were reacted with the *P. domiciliana* extract. Negative controls included six asymptomatic adults, seven patients positive for reaction to each of the commercial antigens of the other previously named fungi, and four patients with high-titer precipitating antibodies to *A. fumigatus* #1. The latter were used as specificity controls. The results were verified by repeating the test for *P. domiciliana* on two different occasions. The micro-Ouchterloney plates were checked for precipitins at 24 and 48 h after rinsing and staining. A strong precipitin band was formed between the patient's serum and the *P. domiciliana* antigen. A repeat study demonstrated identical line formation. All other test wells were negative for precipitins. The control sera did not contain precipitins to *P. domiciliana*. These results indicate that the patient had high levels of precipitating antibodies that were specific for *P. domiciliana*.

Prednisone therapy and mitigation measures to rid the patient's home of the fungus led to marked improvement of her symptoms and pulmonary $D_{L_{CO}}$, and of her chest radiographic and high-resolution chest computed CT results. Four months later, the patient's high-resolution chest CT results and $D_{L_{CO}}$ were normal.

DISCUSSION

This case is significant for several reasons. First, it is the first well documented case of hypersensitivity pneumonitis caused by *P. domiciliana*. Second, it demonstrates that respiratory disease in a particular locale may vary significantly with weather conditions. Third, it demonstrates the essential role of a home visit in unusual and otherwise unexplained cases of pulmonary disease.

The diagnosis of hypersensitivity pneumonitis caused by *P. domiciliana* was established in this case for the following reasons: (*1*) The patient's lung biopsy findings were classical for hypersensitivity pneumonitis (7). (*2*) The onset of illness following flooding and mushroom growth, and the rapid improvement after mitigation of the exposures, are strongly supportive of *P. domiciliana* as the cause of the patient's illness. (*3*) The patient had precipitins in her serum to *P. domiciliana*. Because *P. domiciliana* is otherwise uncommon in California, this finding is unlikely to be purely coincidental. (*4*) The patient did not have serum precipitins to other common causes of hypersensitivity pneumonitis such as *M. faeni* and *A. fumigatus*. (*5*) *P. domiciliana* was found in significant concentrations in the patient's home, whereas elevated levels of other common causes of hypersensitivity pneumonitis were not identified.

The diagnosis of hypersensitivity pneumonitis as a result of exposure to the fungus *P. domiciliana* meets the following major criteria as outlined by Schuyler and Cormier (8): (*1*) symptoms compatible with hypersensitivity pneumonitis; (*2*) evidence of exposure, based on documented spore concentrations in ambient air and the detection of precipitating serum antibody to *P. domiciliana*; (*3*) compatible roentenographic changes (9); and (*4*) compatible pulmonary histopathology (7). Additional minor criteria that were met in the patient's case included: (*1*) bibasilar crackles on lung auscultation; (*2*) decreased $D_{L_{CO}}$; and (*3*) hypoxemia with exertion. An inhalation challenge study was not done because the patient responded well to medical and environmental mitigation therapy and because inhalation challenge testing entails risk.

P. domiciliana is not often found in California. In the United States, the fungus is most prevalent in the Northeast. It is favored

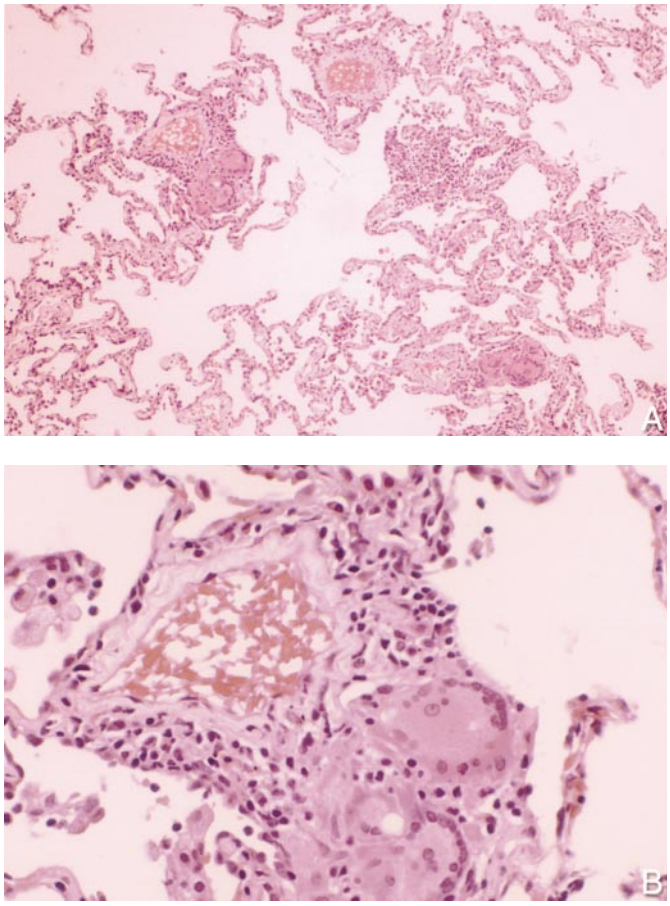

Figure 2. Histopathology of patient's open lung biopsy specimen, demonstrating an interstitial lymphoplasmacytic infiltrate (*A*) and interstitial nonnecrotizing granulomas (*B*).

by an alkaline environment and damp conditions, and grows indoors in cellars, on plaster, mortar, sand, and coal dust (5). In this case, the alkaline environment was provided by the brick and mortar of the patient's basement, and the excessive flooding in the patient's home created the damp conditions. More than 42 in. of rain fell in the winter of 1997–1998, setting a record for the Santa Ynez Mountain Range (1). It was also notable that spore concentrations for *P. domiciliana* were similar in the upper levels of the patient's house (where the fungus was not growing) and in the basement. This was probably attributable to the spores being transported by internal convection currents and turbulence from a forced air heater. Thus, although the patient rarely spent time in the basement area, significant exposures occurred in the kitchen and second floor areas, where she spent most of her time.

Hypersensitivity pneumonitis has many causes (10–12) *Thermophilic actinomycetes* from moldy hay causes farmer's lung in up to 8% of the farming population (13). Similarly, *T. actinomycetes* is believed to be the causative agent of bagassosis and also of mushroom worker's lung (14–16). Typically, occupational or avocational activities lead to repetitive exposures to an organic antigen. The immune response develops as a result of the exposure and subsequently leads to an inflammatory response of the alveoli and pulmonary parenchyma. However, not every person with serum precipitins to these organic antigens develops hypersensitivity pneumonits.

In addition to resulting from occupational exposures, such as to moldy hay, commericall grown mushrooms, and bagasse (sugar cane), hypersensitivity pneumonitis results from home exposures. Bird fanciers develop hypersensitivity pneumonitis relatively often because of avian antigens (17). Hypersensitivity pneumonitis has been associated with home ultrasonic humidifiers (18, 19). Contamination of home dwellings with *Trichosporon cutaneum* has been described with Japanese summer-type hypersensitivity pneumonitis (20). Heated swimming pools and composting waste at home have also been associated with hypersensitivity pneumonitis (21, 22).

Figure 3. Photograph of mushrooms growing along lower basement wall of patient's home.

TABLE 1

SPORE CONCENTRATION IN AMBIENT AIR OF PATIENT'S HOME*

Fungi and Fungal Components	Kitchen	Basement
Pezizia domiciliana	2,750	2,250
Aspergillus/Penicillium sp.	2,750	22,000
Cladosporium	10,000	5,000
Basidiospores	2,250	500
Ascospores	750	0
Coprinus	750	0
Rusts	250	250
Alternaria	0	250
Smuts	0	250

* Fungal spores per cubic meter.

Hypersensitivity pneumonitis is a serious disease that can lead to permanent lung dysfunction (23, 24). Although early stages are reversible, chronic hypersensitivity pneumonitis leads to irreversible pulmonary fibrosis (25, 26). The home visit in this patient's case led to mitigation of exposures and prevention of disease progression.

In summary, this was a case of extrinsic allergic alveolitis occurring in the home setting and caused by the fungus *P. domiciliana*. Excessive rainfall as a result of the *El Niño* weather pattern, and a structural defect in the patient's home basement, led to flooding of the basement. These conditions, along with the cement, brick, and mortar construction of the basement, created a microenvironment within the patient's home that provided the ideal growth conditions for *P. domiciliana*. The association between the fungus and the patient's illness was not immediately recognized, but was subsequently documented. This case highlights the usefulness of a home visit to assess potential causes of illness.

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