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Hypersensitivity pneumonitis induced by *Penicillium expansum* in a home environment

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Summary

An episode of fever, cough, shortness of breath and leucocytosis developed in a 31-year-old atopic housewife from mould exposure in her home environment is evaluated. A chest radiograph revealed diffuse tiny nodular infiltrations in both whole lung fields. Spirometry revealed a severe restrictive type of ventilation impairment. Bronchoalveolar lavage (BAL) showed an increased lymphocyte count with reversed CD4+/CD8+ ratio and transbronchial lung biopsy showed markedly increased lymphocytic infiltration in alveolar septa. Fungal cultures in the air of her home were positive for *Penicillium expansum* and other fungi. Double immunodiffusion test with the patient's serum showed two precipitin bands to *P. expansum* antigens. Her symptoms, abnormal findings of radiograph, and spirometric abnormalities disappeared after 2 months' avoidance. The serum precipitin disappeared after 1 month's avoidance. This study indicates that the patient had hypersensitivity pneumonitis (HP) on exposure to *P. expansum* in her home environment.

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Surveys of airborne fungi in home environments have revealed a correlation between the presence of moulds and respiratory diseases [1–4]. There have been some reports of hypersensitivity pneumonitis (HP) associated with an occupational exposure to mould-contaminated wood dusts containing *Cryptostroma corticale* [6,7], *Alternaria* [8], *Rhizopus*, *Paecilomyces* and *Aspergillus* [9–11], *Penicillium* spp. [11–13]. In this paper, we report a case of HP from mould exposure (*P. expansum*) in the home environment of a 31-year-old woman.

Case report

The patient was a 31-year-old, non-smoking housewife. She had been in excellent health except for seasonal rhinitis symptoms during spring of the previous 2 years. However, several months ago, she experienced fever, myalgia and arthralgia after she removed and mended a wooden floor in the living room of her home. These symptoms disappeared spontaneously on the following day. One month ago, she started to cough and experience shortness of breath and these symptoms were progressively aggravated. When she was examined on admission in November 1992, she was febrile (38·8°C) for 1 day but there were no other remarkable findings. Her leucocyte count was 12400/mm^3 without eosinophilia. Diffuse tiny nodular densities with increased interstitial markings were noted. Spirometry results: FVC-1190 ml (38% predicted), FEV_1-1000 ml (33% predicted), FEV_1/FVC-84% and $MEFR_{25-75}$-3·14 l/s (48% predicted). The patient was observed without medication. Rheumatoid (RA) factor was positive and C_3 and C_4 levels were normal. Serum IgM level was increased (4·83 g/l) but IgG and IgA levels were normal.

After 3 days of hospitalization, she became asymptomatic. One week later, her leucocyte count decreased to 5700/mm^3 and the chest radiograph showed a slight decrease in tiny nodular densities. Follow-up spirometry results were FVC-1750 ml (70% predicted), FEV_1-1170 (55% predicted), FEV_1/FVC-67% and $MEFR_{25-75}$-2·53 l/s (44% predicted). A skin-prick test revealed positive responses to tree pollens (alder-4+, birch-4+, poplar-4+ and hazel-4+), dog hair (3+), grasses (rye-3+ and timothy-3+) and plantain pollen (3+), and negative results to moulds. Total IgE by PRIST was 161 IU/ml and RAST results were t_2 (class 2) and t_5 (class 2). Her methacholine PC20 was 25 mg/ml. BAL fluid analysis

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revealed that lymphocytes were increased up to 40% and CD4+/CD8+ ratio was reversed (0·8). The pathologic findings of transbronchial lung biopsy specimens demonstrated extensive lymphocytic infiltration within alveolar walls. When the subject was sent home, the symptoms recurred several hours after her arrival. She was re-admitted and her symptoms improved after 2 days' hospitalization. The patient was advised to move to another house. Her chest radiograph and spirometry results were normalized after 2 months' avoidance and she complained of no more episodes of the symptoms until July 1993.

Laboratory methods

Isolation and identification of fungi

Airborne fungi in the patient's home were isolated by the open plate method. Sabouraud dextrose agar plates containing 0·06 g/l of chloramphenicol were left open for 2 h in the patient's living room, bedrooms, and on the floor and then incubated at 25°C and at 37°C for 10 days. Colonies appearing on the plates were subcultured and identified by morphology [14].

Crude antigen preparation

Fungal isolates were cultured in sabouraud dextrose broth for 4 weeks on a gyrotory shaker at 25°C or 37°C depending on the requirements of the strains. The culture filtrate was separated and concentrated by vacuum dialysis against de-ionized water at 4°C.

Double-diffusion tests

Double immunodiffusion tests were performed according to the method previously described by Crowle [15] with the following modification: 1% Difco noble agar contained 1% sodium azide as the electrolyte [16]. Serial serum of the patient were reacted with antigens of *Aspergillus fumigatus*, *flavus*, *niger*, *versicolor*, *Candia albicans*, two *Penicillium* spp. (A & B), *Pseudallescheria boydii*, and *Rhizopus* sp., which were prepared with isolates from patient's home environment except *P. boydii* and *C. albicans*.

Results

Penicillium spp. were the predominant isolates (50–80 colonies per plate) from all sites of the air in the patient's home. A few colonies of *A. niger* and *versicolor*, *C. albicans*, *Fusarium* sp., *Scopulariopsis* sp., and *Rhizopus* spp. were also present. Double immunodiffusion tests showed two positive precipitin lines only with antigen strain A of *Penicillium* spp. and the second serum after 1 month's avoidance showed a negative reaction.

Discussion

This study describes a patient having clinical and immunological features of HP resulting from mould exposure (*P. expansum*) in her home environment. Although we did not perform an inhalation challenge, many elements of the history and laboratory findings support the diagnosis of HP. There are a history of continuous exposure to the suspected environment before onset of symptoms, recurrent episodes of fever, cough and dyspnoea that occurred several hours after exposure to the suspected environment, resolution of symptoms within several days away from her home without medication, leucocytosis which normalized after avoidance along with spirometry and chest radiograph, absence of symptoms after avoidance of the suspected antigen exposure, BAL and pathologic findings compatible with HP.

The presence of serum precipitin to the *P. expansum* antigen isolated from the patient's home provides an additional support for the diagnosis of HP and suggests that *P. expansum* was the causative agent. *Penicillium* spp. have been reported to be responsible for HP in association with a variety of exposures to mould-contaminated wood work [8,9,11,12]. It has also been reported to cause HP in a cheese worker [17], in an entomologist [18], in a library outbreak of the disease [19], and from a leak in a central heating system [20]. Presumably in this case, moulds growing under her wooden floor could have been dispersed into the air and subsequent inhalation by the patient resulted in an immunologic response.

This patient has had allergic rhinitis to tree and grass pollens and airway hyperresponsiveness to methacholine. Her symptoms were controllable by topical applications of sodium cromoglycate and steroids during the season. There are some studies showing different results on the surface phenotypes of BAL lymphocytes in HP [21–25]. Our result of increased lymphocytes with reversed CD4/CD8 ratio was consistent with HP.

This case report underlines the need to be conscious of the clinical presentation of HP from mould exposure in the home environment. This diagnosis should be suspected and investigated in those patients who manifest episodic cough, fever, and dyspnoea whether their environments have been previously reported to be associated with HP or not.

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