Bronchial Mucoid Impaction Due to the Monokaryotic Mycelium of Schizophyllum commune

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We report, to our knowledge, the first case of mucoid impaction of the bronchi due to a hypersensitivity reaction to the monokaryotic mycelium of *Schizophyllum commune*. The patient was hospitalized because of mild asthma attacks, persistent cough, peripheral eosinophilia, and “gloved finger” shadows on a chest roentgenogram. Bronchoscopic examination disclosed mucoid impactions that consisted of accumulations of eosinophils, Charcot-Leyden crystals, and nondichotomously branched hyphae in B³, B⁹, and B¹⁰ of the left lung. Cultures of the mucous plugs and sputum samples yielded white, felt-like mycelial colonies that were later identified as the monokaryotic mycelium of *S. commune* by use of mating tests with established monokaryotic and dikaryotic strains of *S. commune*. The results of tests for serum antibody to *S. commune* cytosol antigen were positive. Repeated bronchoscopies for performing bronchial toilet were effective in removing the mucous plugs and relieving the patient’s symptoms. We suggest that the monokaryotic mycelium of *S. commune* should be considered as one of the fungi that can cause hypersensitivity-related lung diseases.

The term *mucoid impaction of the bronchi* (MIB) was first coined by Shaw [1] in 1951 to designate the distinct clinical entity of localized accumulation of inspissated mucus in the bronchi of patients with asthma or chronic obstructive bronchitis. Although there have been several reports of MIB that occurred distally to obstructing endobronchial lesions such as tumors and atresia [2], it is believed that MIB occurs most commonly as a manifestation of a hypersensitivity state in patients with bronchial asthma or in association with allergic bronchopulmonary aspergillosis (ABPA), and there is considerable clinical overlap between MIB and ABPA [3–5]. Since Hinson et al. [6] proposed *Aspergillus fumigatus* as the cause of ABPA, there have been several reports of allergic bronchopulmonary mycoses (ABPM) caused by other fungi including *Aspergillus oryzae*, *Aspergillus ochraceus*, *Candida albicans*, *Torulopsis glabrata*, and *Penicillium* species [7]. In 1994, Kamei et al. [7] reported a case of ABPM caused by the homobasidiozymotic fungus *Schizophyllum commune* in dikaryotic mycelial form [7]. We report, to our knowledge, the first case of MIB caused by a hypersensitivity reaction to the monokaryotic mycelium of *S. commune*, and we discuss identification of this fungus.

Case Report

A 67-year-old female was admitted to The Hospital of the Chest Disease Research Institute, Kyoto University (Kyoto, Japan) in August 1991 because of persistent cough, recurrent mild wheezing, and abnormal shadows on a chest roentgenogram. She had had pulmonary tuberculosis and tuberculous pleurisy at the age of 20, and she had had a similar episode of bronchial asthma in April 1990. She had dealt with antique dolls for the past 40 years. She had never smoked.

On admission, chest roentgenograms and CT scans demonstrated Y-shaped densities resembling gloved fingers in the B³, B⁹, and B¹⁰ of the left lung (corresponding to the dilated bronchi containing inspissated mucus) and a consolidation in the left hilar zone. Physical examination revealed slight expiratory rhonchi in both lungs. Laboratory studies demonstrated peripheral eosinophilia (WBC count, 5,300/mm³ with 12% eosinophils). Serum chemistry and immunologic values, including IgG, IgA, IgM, and IgE levels, were within normal limits. A radioallergosorbent test for specific IgE antibodies and an Ouchterlony test for precipitating antibodies to common fungi including *A. fumigatus*, *C. albicans*, and *Cryptococcus neoformans* were negative. Results of urinalyses were normal. The first bronchoscopic examination, performed on the third hospital day, disclosed thick white-yellowish mucous plugs in B³, B⁹, and B¹⁰ of the left lung (figures 1A and 1B). Bronchoscopy was repeated five times during a 2-month period to remove all the mucous plugs by means of forceps and suctioning. The largest mucous plug taken from the B¹⁰ bronchus was elastic and hard, measuring 0.8 cm in diameter and 5 cm in length.

Cultures of the mucous plugs, bronchial aspirates, and sputum samples yielded the same mycelial colonies, which were white and had a felt-like, fluffy appearance on Sabouraud dextrose agar (Nissui Pharmaceutical, Tokyo) plates; however, no other pathogenic microorganisms were isolated. The mycelial fungus was not immediately identified because spores were absent, but it was later identified as the monokaryotic mycelium...
Figure 1. Results of the first bronchoscopic examination of a patient with mucoid impaction of the bronchi caused by *Schizophyllum commune*. A, mucous plug in B3 of the left lung; B, mucous plugs in B9 and B10 of the left lung. The mucous plugs were stuck tightly in the bronchi.

The patient’s symptoms gradually abated, and the abnormal shadows on her chest roentgenograms gradually disappeared. From October 1991 until October 1994, her clinical symptoms did not recur, although she was not receiving any medications.

In October 1994, she had a recurrence of MIB in B4 and B5 of the left lung. Cultures of the mucous plugs and sputum samples again yielded a monokaryotic mycelium of *S. commune* but no other significant microorganisms. Bronchial toilet, performed by means of repeated bronchoscopies, as well as inhalation of procaterol HCl (2 puffs every 8 hours daily) resulted in improvement in her condition.

**Mycological Study**

Homogenates of the mucous plugs from the patient’s left lung and of her sputa were initially cultured in September 1991 on several Sabouraud dextrose agar plates. During incubation at 25°C for 7 days, several white, felt-like, and fluffy mycelial colonies appeared. After a pure culture was established from these colonies, the fungus was labeled as IFM 46097 (IFM is the abbreviation for the strains maintained in the Research Center for Pathogenic Fungi and Microbial Toxicoses, Chiba University, Chiba, Japan). Despite several microscopic examinations, spores or hyphae with clamp connections could not be found. However, nondichotomously-branched septate hyphae with tubercles, which seem to be peculiar to *S. commune* [8], were observed. The second isolate from the patient, which was detected in November 1994 and labeled as IFM 46800, had macroscopic and microscopic features that were almost identical to those of IFM 46097.

As both isolates from the patient were assumed to be the monokaryotic mycelium of *S. commune* on the basis of these macroscopic and microscopic features, mating tests were performed as follows. Tester strains of *S. commune* used for the mating tests were a dikaryotic strain (IFM 46581) derived from a wild *S. commune* basidiocarp and four monokaryotic strains (IFM 46101, IFM 46102, IFM 46103, and IFM 46578). The clinical isolates IFM 46097 and IFM 46800 were inoculated onto potato dextrose agar (Difco, Detroit) plates opposite each of the tester strains. Duplicate sets of the crosses were made. The plates were incubated in transparent plastic boxes at 25°C for 7–9 days in darkness; the boxes were then transferred to a common laboratory table to permit exposure to daylight, and the strains were cultured at room temperature (~20°C) for 2–3 weeks.

The mating tests were carried out two times per isolate. The first clinical isolate, IFM 46097, was dikaryotized; it produced tuberculated hyphae with clamp connections when cocultured with the dikaryotic strain IFM 46581, although it did not produce any basidiocarps. When the isolate IFM 46097 was cocultured with each of the four monokaryotic tester strains, the testers were dikaryotized. In addition, various types of basidiocarps [9] were produced, although IFM 46097 itself did not...
show any reactions. The second clinical isolate, IFM 46800, was dikaryotized by the four monokaryotic tester strains, and it made the tester strains dikaryotic. In some crosses, typical basidiocarps of *S. commune* were produced after a 2- to 3-week incubation period (figure 2). The second isolate was also dikaryotized by the dikaryotic tester strain. On the basis of these mating behaviors, the clinical isolates IFM 46097 and IFM 46800 were identified as monokaryotic mycelia of *S. commune*.

**Discussion**

*S. commune* is a member of the homobasidiomycetous fungi, which include mushrooms, toadstools, and their relatives and can produce macroscopic fruiting bodies (basidiocarps). *S. commune* is ubiquitous in the environment and has been considered to be nonpathogenic in humans. Consequently, its detection in human specimens has been regarded as contamination in most cases. However, *S. commune* can cause infectious and hypersensitivity-related disease in humans. Since Kligman [10] reported the first case of human onychomycosis caused by *S. commune* in 1950, several cases of disease caused by this fungus have been reported [7]. Kamei et al. [7] reported the first case of ABPM caused by *S. commune* in dikaryotic mycelial form, although MIB was not demonstrated in this case. These authors cautioned that the characteristic basidiocarps of *S. commune* do not grow in the dark (a common environment of incubators in clinical microbiology laboratories), and this circumstance may result in nongrowth of the basidiocarps and, therefore, misdiagnosis in many cases of basidiomycosis. Moreover, it seems more difficult to detect the monokaryotic mycelium of *S. commune* because the monokaryotic mycelium of this fungus has neither clamp connections, which are specific to the dikaryotic mycelium of the basidiomycetes, nor the ability to produce basidiocarps by itself. In the case of the monokaryotic mycelium of *S. commune*, it is important to be aware of the white, felt-like, fluffy appearance of the mycelial colonies on the culture plates and, on microscopic examination, the formation of tubercles by the septate hyphae; mating tests should be performed with established monokaryotic and dikaryotic strains of *S. commune* to correctly identify the isolate.

For our patient, inhalation of a β2-agonist and oral therapy with a mucolytic agent were not sufficient to relieve the symptoms of MIB, but repeated bronchoscopies and use of forceps and suctioning were effective in removing the mucous plugs. We were able to avoid the use of steroid therapy because of the excellent efficacy of bronchoscopy for performing bronchial toilet. The mechanisms by which the bronchi of our patient were colonized with *S. commune* are not yet known and remain to be clarified in the future.

**References**