

PNEUMONIA FROM INTRABRONCHIAL PSEUDALLESHERIASIS IN A WOMAN WITH NO IMMUNOCOMPETENT DEFECTS

Shuichi YANO and Shinji SHISHIDO

Department of Pulmonary Medicine, National Sanatorium Matsue Hospital, Matsue 690-8556, Japan

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We describe the first report of intrabronchial *Pseudallesheriasis* in a 74-year-old Japanese woman with a one-year history of refractory cough and an intrabronchial lesion. After endoscopic removal of a part of the lesion, her symptoms were markedly improved. Oral itraconazole therapy was begun, but right lower lobe pneumonia developed due to *S. apiospermum*. Continuous infusion of amphotericin B in a total dose of 1096 mg was performed and the pneumonia resolved completely. Our experience suggests that intravenous amphotericin B may be the treatment of choice for *Pseudallesheriasis*, even if the lesion is limited in bronchus.

Key words: intrabronchial pseudallesheriasis

CASE REPORT

A 74-year-old Japanese woman was first examined at our hospital on 14 November 1995 with a one-year history of persistent dry cough. She had no immunological abnormalities. A CT scan of the thorax showed a round intrabronchial opacity with calcification in the right truncus intermedius bronchus (Fig. 1a). Fiberoptic bronchoscopy revealed a whitish polypoid lesion which obstructed the right lower bronchus (Fig. 2a). Culture of endobronchial biopsy material on Sabouraud medium yielded whitish-gray cotton-like colonies, and microscopic examination using lactophenol cotton blue staining of the original isolates revealed characteristic features of the asexual form of *Scedosporium apiospermum*.

Her refractory cough improved markedly after partial removal of the lesion. As the minimum inhibitory concentration (MIC) of itraconazole for the pathogen was lower ($1 \mu\text{g/ml}$) than that of fluconazole ($25 \mu\text{g/ml}$), amphotericin B ($3.13 \mu\text{g/ml}$), or miconazole ($12.5 \mu\text{g/ml}$), treatment with oral itraconazole was begun at a dose of 100mg/day on November 30. Twelve weeks later, the dose of itraconazole was increased to 200mg/day. The plasma concentration of itraconazole was 621ng/ml with a daily itraconazole dose of 200mg.

She was followed up at our outpatient clinic. The obstruction of right lower bronchus had not worsened according to fiberoptic bronchoscopic examination on 15 March 1996. On 29 May 1996, she presented with the sudden onset of high fever and malaise. A chest radiograph showed infiltration of the right lower lobe, and she was admitted (Fig. 1b). The temperature was 38.1°C. Analysis of arterial blood gases showed that the partial pressure of oxygen was 73mmHg, the partial pressure

of carbon dioxide was 46mmHg, and the pH was 7.45.

The white blood cell count was 4600/mm³, of which 72% neutrophils, 18% lymphocytes, 9% monocytes, and 1% eosinophils. The serum albumin level was low (2.8g/dl), and the CRP level was 9.2mg/dl. Fiberoptic bronchoscopy was performed. Biopsy specimens from right lower lung showed inflammatory granulation tissue with eosinophils, and culture on Sabouraud medium yielded whitish-gray cotton-like colonies. Therefore, we judged that infiltration of the right lower lobe was due to *S. apiospermum*. Since the MIC of amphotericin B was the second lowest after that of itraconazole, continuous infusion of amphotericin B was begun at a dose of 1mg/day, and the dose was gradually increased to 30mg/day. The maximum dose was determined to be 30mg/day, because of renal dysfunction. On 30 July 1996, the orifice of the right basal bronchus was open and a small reddish polypoid cystic lesion was seen, which decreased in size after biopsy (Fig. 2b). Administration of amphotericin B was stopped when the total dosage reached 1096mg. Chest radiographs showed marked improvement (Fig. 1c), and the polypoid lesion disappeared (Fig. 2c). No recurrence has occurred after nine months.

DISCUSSION

Pseudallesheria boydii, the sexual stage of the imperfect fungus *Scedosporium (S) apiospermum*, is a well known causative agent of Madura foot in the United States (1). The fungus lives saprophytically in the soil and sewage. *S. apiospermum* is the etiologic agent of infection in many compromised hosts (2,3,4), and the most common extracutaneous site of infection is the lungs. Pulmonary infiltration may occur with or without cavitation and fungus balls may develop. We reported the first case of intrabronchial *Pseudallesheriasis* in a woman without immunodeficiency (5). A CT scan of the thorax showed a round intrabronchial opacity with calcification in the right truncus intermedius bronchus. The calcification of intrabronchial lesion was probably formed in the fungal infection, because there were no other old opacities. Itraconazole was administered after identification of the fungus, since the MIC was lower than those of other drugs (amphotericin B, miconazole, and fluconazole). However, the intrabronchial lesion persisted after seven months of itraconazole therapy and right lower lobe pneumonia occurred. Accordingly, amphotericin B was administered by continuous infusion, since the MIC was lower than that of fluconazole or miconazole. There were three reasons for the initial choice of itraconazole. First, the patient had no significant symptoms after removal of part of the intrabronchial lesion, so oral treatment as an our outpatient was desired. Second, itraconazole is reported to

Correspondence: Shuichi Yano, Department of Pulmonary Medicine, National Sanatorium Matsue Hospital, 5-8-31, Agenogi, Matsue city, Shimane 690-8556, Japan

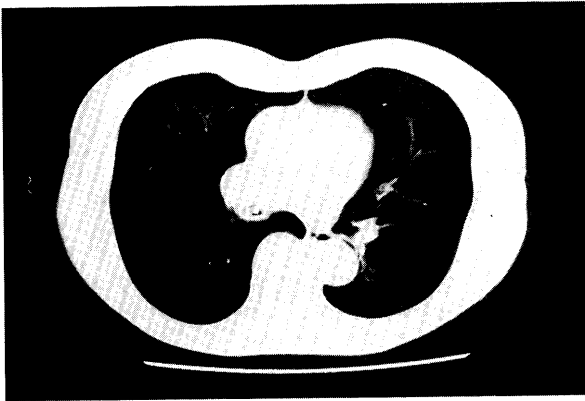


Fig. 1a; CT scan of the thorax shows an intrabronchial round opacity with calcification in the right intermedium bronchus.

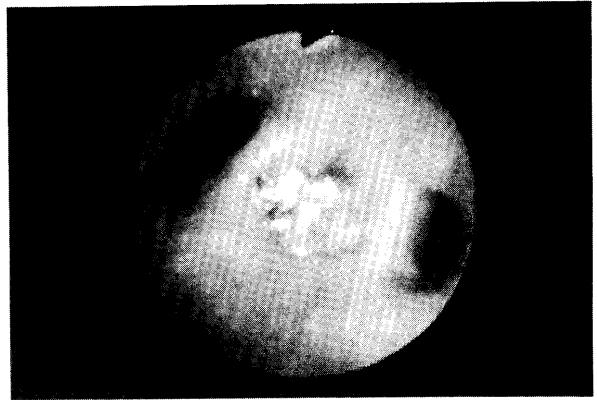


Fig. 2a; Obstruction of the right lower lobe bronchus is seen on fiberoptic bronchoscopy.

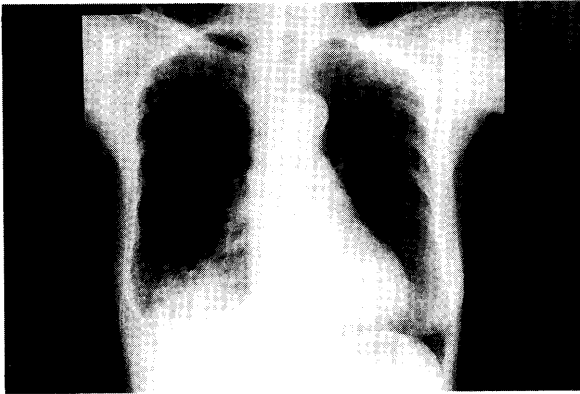


Fig. 1b; A chest radiograph shows infiltration of the right lower lobe.

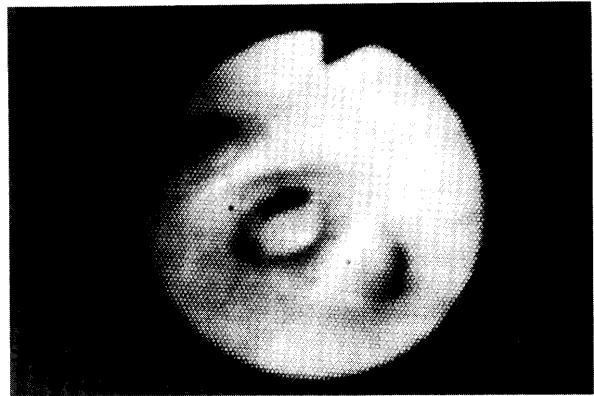


Fig. 2b; The orifice of right basal bronchus is open and there is a small reddish polypoid cystic lesion, which decreased in size after biopsy.



Fig. 1c; The chest radiograph shows marked improvement.

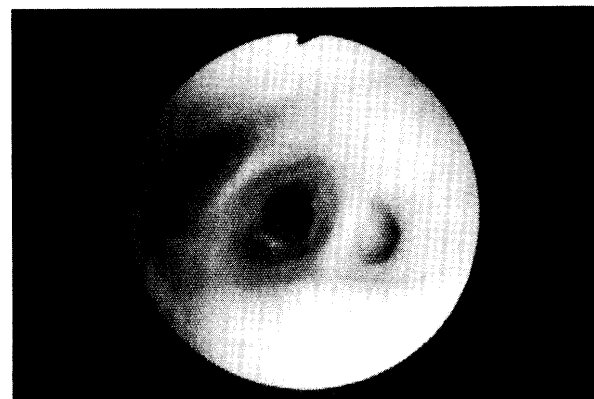


Fig. 2c; The polypoid lesion has disappeared after therapy.

be effective against filamentous fungi, the MIC of itraconazole was lower than those of other drugs, and the plasma concentration was adequate. Third, amphotericin B is reported to cause severe side effects and a continuous infusion is the preferred method of administration, so we could not treat her with amphotericin B as an outpatient. Data in the literature on the treatment of this fungus are controversial (6,7,8,9,10). In our case, the *in vitro* MIC may not have completely reflected the effect of the lesion. We did not find any immunological problems in this

patient. However, since she had an intrabronchial lesion which has not been reported in normal subjects, she might have some presently unknown immunological defects. Although we used itraconazole for the above reasons, even if the lesion is limited to the bronchus in patients with *Pseudallesheriasis*, it might be better to employ continuous intravenous infusion of amphotericin B. However, the permissible limit of oral itraconazole and the best therapy against intrabronchial fungal infection are unknown.

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