Intrabronchial Pseudallescheriasis in an Immunocompetent Woman

Scedosporium apiospermum, the asexual stage of the imperfect form of Pseudallescheria boydii, has been reported as a cause of local or disseminated infection in immunocompromised patients. To our knowledge, we report the first case of intrabronchial pseudallescheriasis as well as the first case of pseudallescheriasis in a healthy woman who had no immunologic defects.

Fiberoptic bronchoscopy revealed a whitish polypoid lesion that obstructed the right lower bronchus (figure 1). After the whitish lesion was removed with biopsy forceps, the right basal bronchus was found to be completely obstructed by a dark gray necrotizing lesion. The endobronchial biopsy specimens contained hyphal fungal elements and necrotic tissue.

Culture of endobronchial biopsy material on Sabouraud medium yielded white-grayish cotton-like colonies. The fungal isolates were stained with lactophenol cotton blue stain, and microscopic examination revealed branching septate hyphae with brownish ovoid conidia at the ends or short sides of conidiophores, findings characteristic of the asexual form of *S. apiospermum*.

The patient's refractory cough decreased markedly after part of the lesion was removed. *S. apiospermum* was isolated from the lesion, and the MIC of itraconazole for this organism was 1 mg/L. The MIC of itraconazole for *S. apiospermum* was lower than that of fluconazole, amphotericin B, or miconazole. Therefore, treatment with oral itraconazole was begun on 30 November 1995 at a dosage of 100 mg/d. Twelve weeks later, the lesion had healed slightly but remained detectable on bronchoscopy. Therapy with itraconazole was continued at a dosage of 200 mg/d.

Pseudallescheria boydii, the sexual stage of the imperfect fungus *S. apiospermum*, is known to be a causative agent of Madura foot in the United States [1]. The fungus lives saprophytically in soil and sewage. *S. apiospermum* is the etiologic agent of infection in many compromised hosts, including patients with lymphoma [2], patients with leukemia [3, 4], patients with systemic lupus erythematosus who are receiving corticosteroid therapy [2, 4], renal transplant recipients [5], and trauma patients [2, 6]. The most common extracutaneous site of *P. boydii* infection is the lungs [2, 7–9].

Figure 1. Endobronchial findings for an immunocompetent woman with intrabronchial pseudallescheriasis. Left: A whitish polypoid lesion obstructs the right lower bronchus. Right: The right basal bronchus is completely obstructed by a dark gray necrotizing lesion after the whitish lesion is removed.
Use of the Polymerase Chain Reaction for Demonstration of Influenza Virus Dissemination in Children

Most investigators believe that influenza virus does not usually induce viremia [1]. Although CNS, cardiac, and skeletal muscle complications have been described in relation to influenza, virus was successfully isolated from the blood and extrapulmonary organs in only a limited number of cases [1, 2]. We recently demonstrated with use of PCR that influenza A/PR/8 virus produces viremia in a mouse model during the acute phase of disease [3].

We searched for influenza virus in the blood and CSF of children with virologically confirmed influenza from 22 December 1994 to 26 March 1995 (table 1). Patients ranged in age from 6 months to 8 years; bronchiolitis was clinically diagnosed in four cases, bronchitis in five cases, and upper respiratory infection in six cases. No abnormal shadows were found in the lung fields on any of the children's chest roentgenograms. None of the children had a history of recurrent serious infectious diseases.

Serum hemagglutination inhibition titer of antibody to A/Kitakyushu/159/93 (H3N2) virus significantly increased (at least a fourfold increase from acute titer to convalescent titer) in 12 cases, it significantly increased to B/Mic/1/93 virus in five cases, and it significantly increased to both strains in two cases. Culture of throat swab specimens in MDCK cell suspension yielded H3N2 virus for 4 of 12 children. PCR and successive Southern hybridization were performed with primer sets for influenza A and B virus matrix gene as previously described [3, 4]. Influenza A and B viruses were detected by PCR in eight and two cases, respectively. However, blood fractions of virus could not be detected by PCR in any of the 14 cases (table 1).

Six children, including two epileptic patients with mental retardation, had convulsions during the course of our study. One child showed signs of somnolence. Because CNS infection was suspect in these cases, CSF was examined for a greater than normal number of cells and an increased protein concentration; however, pleocytosis was not detected, and the protein concentration was within normal limits. PCR was performed with these CSF samples, but they were negative for influenza A and B virus. Influenza virus was not isolated from blood samples or CSF.

This study has verified that viremia and transmission of the virus to the CNS cannot be easily detected among children infected with recent strains of influenza virus. We have previously shown that the PR8 strain of influenza A virus becomes viremic in immunocompetent mice [3]. Furthermore, we tentatively concluded that the virus enters the bloodstream through the infected alveolar septum. This hypothesis is supported by the finding that viremia does not occur when alveolitis is prevented by previous intraperitoneal administration of the antiserum to the virus. The fact that it was difficult to detect viremia among the children in our study might support this hypothesis since none of our patients had obvious pneumonia on the basis of chest roentgenogram findings.

In addition, we could not find any direct evidence that influenza virus invades the CNS of these infected children. Rantala et al. described the successful isolation of influenza B virus from the CSF of a child with febrile convulsions [2]. It might be possible that a certain strain of influenza virus induces systemic dissem-