Underlying diseases associated with pulmonary pseudallescheriasis include diabetes mellitus, leukemia, lymphoma, aplastic anemia, Cushing's disease, collagen-vascular diseases, and alveolar proteinosis. *P. boydii* may cause pulmonary infiltration (with or without cavitation) to occur and fungus balls to develop. However, to our knowledge, we report the first case of intrabronchial pseudallescheriasis. Moreover, we also report the first case of pseudallescheriasis in a healthy person who had no immunologic defects. Since *Pseudallescheria* species and *Aspergillus* species both produce septate hyphae and share some morphologic features, *Pseudallescheria* may be histologically misdiagnosed as *Aspergillus* in the absence of identification by culture [9, 10]. Although in our case the endobronchial biopsy findings were initially thought to be consistent with aspergillosis, the fungus was identified as *S. apiospermum* by culture.

Itraconazole therapy was administered after the fungus was identified since the MIC of this drug was lower than that of other drugs. However, the intrabronchial lesion persisted after 12 weeks of itraconazole therapy.

Shuichi Yano, Shinji Shishido, Takeaki Toritani, Katsuhiko Yoshida, and Hiroko Nakano

The Department of Pulmonary Medicine, National Sanatorium Matsue Hospital, Agenogi, Matsue City, Shimane, Japan

References

1. Bell WE, Myers MG. Allescheria boydii. Brain abscess in a child with leukemia. Arch Neurol 1978;35:386–8.
2. Winston DJ, Jordan MC, Rhodes J. Allescheria boydii infections in the immunosuppressed host. Am J Med 1977;63:830–5.
3. Kisch S, Taylor J, Bergfeld W, Hall G. Petriellidium boydii mycetoma in an immunosuppressed host. Cleve Clin Q 1983;50:209–11.
4. Anderson RL, Carroll TF, Harvey RT, Myers MG. Petriellidium (Allescheria) boydii orbital and brain abscess treated with miconazole. Am J Ophthalmol 1984;97:771–5.
5. Alture WE, Edberg SC, Singer JM. Pulmonary infection with Allescheria boydii. Am J Clin Pathol 1976;66:1019–24.
6. Forno LS, Billingham ME, Allescheria boydii infection of the brain. J Pathol 1972;106:195–8.
7. Nomdedeu J, Brunet S, Martino R, Altes A, Ausino V, Domingo AA. Successful treatment of pneumonia due to *Scedosporium apiospermum* with itraconazole: case report. Clin Infect Dis 1993;16:731–3.
8. Green WO, Adams TE. Mycetoma in the United States. Am J Clin Pathol 1964;42:75–91.
9. Lutwick LI, Galgiani JN, Johnson RH, Stevense DA. Visceral fungal infections due to Petriellidium boydii (Allescheria boydii). In vitro drug sensitivity studies. Am J Med 1976;61:632–9.
10. Shih LY, Lee N. Disseminated petriellidiosis (allescheriasis) in a patient with refractory acute lymphoblastic leukaemia. J Clin Pathol 1984;37:78–82.

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/Mie/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 suspected 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 dissemina-

Reprints or correspondence: Dr. Yoshinobu Kimura, Department of Microbiology, Fukui Medical School, Fukui, 910-11, Japan.

Clinical Infectious Diseases 1997;24:736–7

1058–4838/97/2404–0028$02.00

Table 1. Use of PCR for detection of viremia in children with virologically confirmed influenza.

Age	Date of onset of influenza	Clinical sign; diagnosis	Temperature		Serum HAI antibody titer to influenza virus			Sample (d)*	Virus isolation	Results of PCR				
					Conv. Acute	Conv. Acute	Conv. Acute							
			°C	No. of days	H1N1	H3N2	B		Throat	Throat	PBMC	RBC	Plasma	CSF
6 mo	12/22/94	FC; bronchiolitis	40.0	6	<4 <4	<4 256	<4 <4	2	ND	ND	ND	ND	ND	–
8 y†	1/8/95	Convulsion; bronchitis	40.4	5	128 128	<4 1,024	32 32	0	–	–‡	–	–	–	ND
2 y	1/11/95	Bronchitis	40.2	7	<4 <4	<4 128	32 64	1	–	A§	–	–	–	ND
3 y	1/11/95	Bronchitis	41.5	5	<4 <4	<4 128	32 32	1	ND	ND	–	–	–	ND
3 y	1/12/95	FC; URI	39.5	7	<4 <4	<4 128	16 16	1	–	A§	–	–	–	–
2 y	1/12/95	Drowsiness; URI	39.8	5	8 8	<4 64	8 8	0	H3N2	A§	–	–	–	–
1 y, 5 mo	1/25/95	FC; URI	40.3	6	<4 <4	<4 128	<4 <4	0	H3N2	A§	–	–	–	–
9 mo	2/1/95	URI	40.1	11	<4 <4	<4 256	8 8	1	H3N2	A§	–	–	–	–
1 y	2/5/95	FC; bronchitis	39.0	3	<4 <4	<4 128	32 128	1	ND	ND	–	–	–	–
5 y†	2/5/95	Convulsion; bronchitis	39.2	5	<4 <4	<4 128	<4 128	1	–	A§	–	–	–	ND
1 y, 4 mo	2/9/95	Bronchiolitis	39.7	7	<4 <4	<4 128	<4 <4	5	H3N2	A§	–	–	–	ND
1 y, 4 mo	2/9/95	Bronchiolitis	38.5	8	<4 <4	<4 512	<4 <4	5	–	A§	–	–	–	ND
4 y	2/13/95	URI	40.0	5	<4 <4	256 256	16 2,048	4	–	B‖	–	–	–	ND
5 y	3/5/95	URI	40.0	2	<4 <4	256 256	32 128	1	–	B‖	–	–	–	ND
1 y, 7 mo	3/26/95	Bronchiolitis	39.6	7	<4 <4	<4 <4	<4 32	2	–	–	–	–	–	ND

NOTE. Conv = convalescent; FC = febrile convulsion; HAI = hemagglutination-inhibiting; ND = not done; PBMC = peripheral blood mononuclear cells; URI = upper respiratory infection.

* No. of days after the onset of illness.

† This patient had a history of intractable epilepsy and mental retardation.

‡ Negative for both influenza A and B viruses.

§ Positive for influenza A virus–specific sequences.

‖ Positive for influenza B virus–specific sequences.

tion if it is pneumotropic enough to cause pneumonia. Host factors should also be considered when investigating virus spread in immunocompromised individuals because one might expect them to have more serious illnesses [5].

Isamu Mori, Hiroshi Nagafuji, Kazuo Matsumoto, and Yoshinobu Kimura

Department of Microbiology, Fukui Medical School, Fukui; Department of Pediatrics, The Tazuke Kofukai Medical Research Institute, Kitano Hospital, Osaka; and the Fukui Prefectural Institute of Public Health, Fukui, Japan

References

1. Murphy BR, Webster RG. Orthomyxoviruses. In: Fields BN, Knipe DM, Howley PM, eds. Virology. 3rd ed. Philadelphia: Lippincott-Raven, 1996; 1397–445.
2. Rantala H, Uhari M, Tuokko H. Viral infections and recurrences of febrile convulsions. J Pediatr 1990; 116:195–9.
3. Mori I, Komatsu T, Takeuchi K, et al. Viremia induced by influenza virus. Microb Pathog 1995; 19:237–44.
4. Zhang W, Evans DH. Detection and identification of human influenza viruses by the polymerase chain reaction. J Virol Methods 1991; 33:165–89.
5. Apalsch AM, Green M, Ledesma-Medina J, Nour B, Wald ER. Parainfluenza and influenza virus infections in pediatric organ transplant recipients. Clin Infect Dis 1995; 20:394–9.