

## Lung Abscess due to *Clostridium baratii* Infection in a Patient with Invasive Pulmonary Aspergillosis<sup>∇</sup>

Clostridial pleuropulmonary infections are rare; most of these infections are attributed to *Clostridium perfringens* (1), and none involving *C. baratii* have been reported. We describe here a fatal case of *C. baratii* lung abscess superimposed on invasive pulmonary aspergillosis.

A 47-year-old man was diagnosed with myelodysplastic syndrome with refractory anemia and excess blasts in December 2005. The disease course was complicated by disseminated *Mycobacterium avium* complex infection and invasive pulmonary aspergillosis. The patient underwent allogeneic hematopoietic stem cell transplantation in May 2006. One year later, he was admitted for leukemia relapse associated with febrile neutropenia. Chest radiography and computed tomography (CT) displayed multiple small opacities on the right upper and lower and left upper lung fields. Compared with chest CT results seen 1 year before, only the lesion on the left upper lung had increased in size (Fig. 1A). Low-grade fever persisted despite treatment with imipenem and voriconazole. Serum *Aspergillus* antigen results (Platelia *Aspergillus* enzyme immunoassay; Bio-Rad Laboratories) were negative. The lesion on the left lung progressed rapidly within 1 month (Fig. 1B).

Follow-up chest CT results showed an abscess with a thick wall (Fig. 1C and D). In August 2007, the patient was admitted to an intensive care unit (ICU) because of acute respiratory failure. A sonography-guided transthoracic lung biopsy was performed on the second ICU day. Pathology examination of the lung biopsy sample disclosed narrow, dichotomous, and septated hyphae compatible with *Aspergillus* spp. Cultures of the lung biopsy sample and aspirates of the lung abscess as well as blood cultures all yielded *C. baratii* that was susceptible to penicillin, chloramphenicol, metronidazole, and cefmetazole but was resistant to clindamycin by a disk diffusion test. The patient's hemodynamics deteriorated, and the patient died of multiple organ failure on the twelfth ICU day.

The genus *Clostridium* has more than 150 species, but fewer than 20 are associated with human diseases (5, 7). Members of the genus are mainly natural inhabitants of the human intestinal tract, and their disease spectra in humans are broad (7). *C. baratii* has attracted limited attention because of its lack of overt pathogenesis and failure to produce lethal toxin. Its most striking clinical feature is its neurotoxigenesis, type F botulism in infants (4) and in adults (3, 4). However, *C. baratii* pulmo-

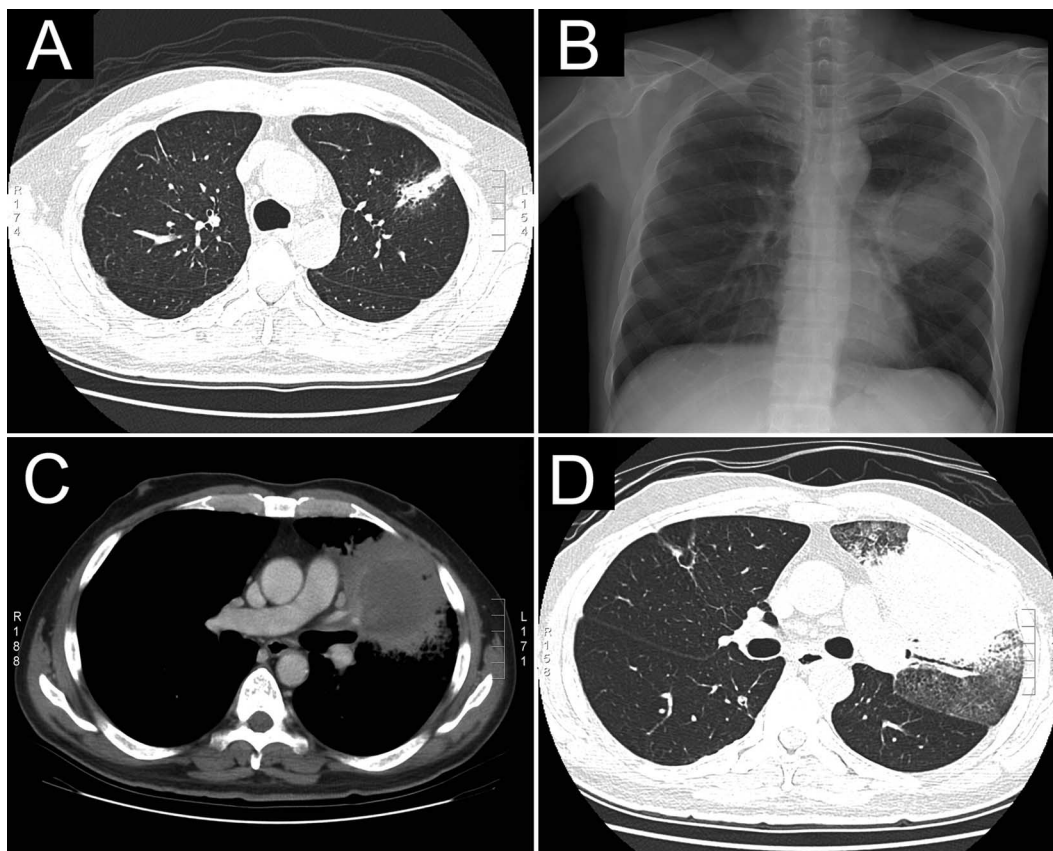


FIG. 1. (A) CT scan of the chest on admission, showing a small faint patch at the left upper lung field (LULF) and old fibrotic changes at the right upper lung field. (B to D) Follow-up chest radiography 1 month later disclosed a mass lesion at the LULF (B), and a CT scan at the same period (C and D) revealed a mass with a fluid-retaining central cavity, thick irregular wall, and a focal area of consolidation over the LULF.

nary infection in adults has never been previously reported in the literature. We speculate that the development of a lung abscess caused by *C. baratii* in this case may be closely related to the patient's leukemia relapse with persistent neutropenia and invasive pulmonary aspergillosis. The hyphae of *Aspergillus* species tend to invade blood vessels, which in turn leads to local thrombosis and tissue infarction (2, 6). Simultaneously, clostridial bacteremia originating from the disruption of the gastrointestinal mucosa results in seeding into the lung, with subsequent formation of lung abscess. The infection is refractory to antimicrobials in the face of tissue infarction despite the use of antimicrobial agents with in vitro activity against *C. baratii*. In conclusion, among high-risk hosts, the rapid progression of fungal pneumonia into lung abscesses may suggest the presence of a superimposed infection due to an anaerobic bacterial species such as *C. baratii*.

## REFERENCES

1. Bayer, A. S., S. C. Nelson, J. E. Galpin, A. W. Chow, and L. B. Guze. 1975. Necrotizing pneumonia and empyema due to *Clostridium perfringens*. Report of a case and review of the literature. *Am. J. Med.* **59**:851–856.
2. Fraser, R. S. 1993. Pulmonary aspergillosis: pathologic and pathogenetic features. *Pathol. Annu.* **28**(Pt. 1):231–277.
3. Gupta, A., C. J. Sumner, M. Castor, S. Maslanka, and J. Sobel. 2005. Adult botulism type F in the United States, 1981–2002. *Neurology* **65**:1694–1700.
4. Harvey, S. M., J. Sturgeon, and D. E. Dassey. 2002. Botulism due to *Clostridium baratii* type F toxin. *J. Clin. Microbiol.* **40**:2260–2262.
5. Hatheway, C. L. 1990. Toxigenic clostridia. *Clin. Microbiol. Rev.* **3**:66–98.
6. Kamai, Y., L. Y. Chiang, L. M. Lopes Bezerra, T. Doedt, A. S. Lossinsky, D. C. Sheppard, and S. G. Filler. 2006. Interactions of *Aspergillus fumigatus* with vascular endothelial cells. *Med. Mycol.* **44**(Suppl. 1):S115–S117.
7. Lorber, B. 2005. Gas gangrene and other clostridium-associated diseases, p. 2828–2838. In B. Mandell, J. E. Bennett, and R. Dolin (ed.), *Principles and practice of infectious diseases*, 6th ed. Churchill Livingstone, New York, NY.

**Chin-Chung Shu**  
**Ming Yao**  
**Chien-Ching Hung**  
**Shih-Chi Ku\***  
**Chong-Jen Yu**  
*Department of Internal Medicine*

**Yih-Leong Chang**  
*Department of Pathology*  
*National Taiwan University Hospital*  
*Taipei, Taiwan*

\*Phone: 886-2-23562905  
 Fax: 886-2-23582867  
 E-mail: scku1015@ntu.edu.tw

<sup>v</sup> Published ahead of print on 3 January 2008.