Lung Abscess due to *Clostridium barati* in a Patient with Invasive Pulmonary Aspergillosis

Clostridial pleuro-pulmonary infections are rare, most of which are attributed to *Clostridium perfringens* [1] and none have been reported involving *C. barati*. We describe here a fatal case of *C. barati* 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 one year before, only the lesion on the left upper lung increased in size (Figure 1A). Low grade fever persisted despite treatment with imipenem and voriconazole. Serum *Aspergillus* antigen [Platelia *Aspergillus* enzyme immunoassay; Bio-Rad Laboratories] was negative. The lesion on the right lung progressed rapidly within
one month (Figure 1B). Follow-up chest CT showed an abscess with a thick wall (Figures 1 C and D). In August 2007, the patient was admitted to the intensive care unit (ICU) because of acute respiratory failure. Sonography-guided trans-thoracic lung biopsy was performed on the 2nd ICU day. Pathology examination of the lung biopsy disclosed narrow, dichotomous and septated hyphae compatible with *Aspergillus* spp. Cultures of the lung biopsy and aspirates of the lung abscess as well as blood cultures all yielded *C. barati* that was susceptible to penicillin, chloramphenicol, metronidazole, and cefmetazole but was resistant to clindamycin by disk diffusion test. The patient’s hemodynamics deteriorated and died of multi-organ failure on the 12th ICU day.

The genus *Clostridium* has more than 150 species but fewer than 20 are associated with human diseases [2, 3]. Members of the genus are mainly natural inhabitants of the human intestinal tract and their disease spectra in humans are broad [3]. *C. barati* has attracted limited attention because its lack of overt pathogenesis and failure to produce lethal toxin. Its most striking clinical feature is its neuro-toxogenesis, the type F botulism in infants [4] and in adults [4, 5]. However, *C. barati* pulmonary infection in adults has never been reported in literature. We speculate that the development of *C. barati* lung abscess in this
case may be closely related to the patient’s leukemia relapse with persistent
neutropenia and invasive pulmonary aspergillosis. The hyphae of *Aspergillus*
tends to invade blood vessels which in turn leads to local thrombosis and tissue
infarction [6, 7]. Simultaneously, clostridial bacteremia originating from the
disruption of the gastro-intestinal mucosa becomes 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. barati*. In conclusion, among high-risk hosts, the rapid
progression of fungal pneumonia into lung abscess may suggest a superimposed
infection due to anaerobic bacteria such as *C. barati*.

Chin-Chung Shu¹, Ming Yao¹, Chien-Ching Hung¹,
Yih-Leong Chang², Shih-Chi Ku¹, and Chong-Jen Yu¹

Departments of Internal Medicine¹ and Pathology², National Taiwan University
Hospital
REFERENCES


FIGURE LEGENDS

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