


25. Nucci M, Anaissie E. Should vascular catheters be removed from all patients with candide-

**Fatal Fungemia Due to Phaeoacremonium inflatipes in a Child with Severe Aplastic Anemia**

SIR—Infection remains a major cause of death in patients with aplastic anemia [1–3]. Bacterial pathogens, particularly Escherichia coli, Pseudomonas aeruginosa, and gram-positive cocci, constitute the main causes of infectious episodes [1–3]. Invasive infection due to fungal pathogens, especially Candida and Aspergillus species, has emerged recently and is associated with mortality in this patient population [2]. We report an invasive infection caused by *Phaeoacremonium inflatipes* in a boy with severe aplastic anemia for whom amphotericin B treatment failed. To our knowledge, this is the first reported fatal case of fungemia due to *P. inflatipes* [4, 5].

**Case report.** A boy began to experience easy bruising at the age of 14 months. Fever developed 2 months later. He was found to have pancytopenia (WBC count, 3600 cells/μL; hemoglobin level, 5.2 mg/dL; platelet count, 2000 platelets/μL) and was admitted to a hospital in June 2003, when he was 18 months of age. Diagnostic workup revealed hypocellular bone marrow. Evidence of Epstein-Barr virus (EBV) infection was noted on the basis of a test result positive for IgM antibody against EBV capsid antigen. Other laboratory investigations, including microbiological cultures, echocardiography, and abdominal CT, did not yield significant findings. Immunosuppressive therapy with antithymoglobulin and prednisolone was given twice during June 2003. The patient’s activity and appetite remained fair during these 2 months, but the intermittent fever persisted despite the use of antibiotics. At the family’s request, he was transferred to our hospital (National Taiwan University Hospital, Taipei).

On admission to our hospital on 27 June 2003, pancytopenia (WBC count, 140 cells/μL; hemoglobin level, 8.1 mg/dL; platelet count, 8000 platelets/μL), hemophagocytosis (the liver was palpable 3 cm below the right costal margin) and good general condition were observed. Bone marrow aspiration performed on 1 July revealed infection-associated hemophagocytosis. Furthermore, a species of mold grew from the bone marrow culture. CT performed on 15 July showed spleen infarction; inflammatory changes in the mesentery; and multiple, less-enhanced, small focal areas at the left lateral, left medial, and right inferior liver. Chronic sinusitis involving the maxillary, ethmoid, and sphenoid sinuses was demonstrated by CT on 16 July. Culture of a nasal discharge specimen grew *Stenotrophomonas maltophilia* and *Candida parapsilosis*. A species of mold was repeatedly cultured from peripheral blood samples obtained on 1 July, 3 July, 5 July, and 7 July. The MIC of amphotericin B against this mold species was 0.75 μg/mL. However, high, spiking fever and pancytopenia persisted during therapy with amphotericin B (1 mg/kg/day) and broad-spectrum antibiotics (vancomycin and meroxepen).

Treatment failure first manifested as ulceration on the patient’s lower lip. The lesions evolved to swelling and gangrene of the lips, swelling over the left periorbital area, and, finally, swelling of the neck. A CT scan performed on 28 July revealed multiple small areas of adenopathy on both posterior cervical spaces and diffuse edema bilaterally on the posterior neck and on the left eyelid with inferior extension to the upper mediastium. The family refused any surgical intervention. The patient died of septic shock on 7 August 2003.

**Discussion.** The isolates were recognized as *P. inflatipes* by conventional morphological identification methods. The isolates were further identified by partial
sequencing of rRNA genes [6]. The high percentage of identity in the rRNA loci 5.8S–28S (94%), 18S–28S (93%), and 28S (95%) confirmed the identification of *P. inflatipes*. For all isolates, MICs of amphotericin B determined by the E-test (AB Biodisk) were 0.064 μg/mL.

Of the 6 species belonging to the new genus *Phaeoacremonium* proposed by Crous et al. [7], 3 are associated with infection in humans: *P. inflatipes*, *Phaeoacremonium parasticum*, and *Phaeoacremonium rubrigenum*. These 3 species were previously recognized as *Phialophora parasticida*. Previously reported human infections caused by *P. parasticus* or *Phaeoacremonium* species included subcutaneous abscess, arthritis, infected cysts, and disseminated infections, mostly in immunocompromised patients [7–9]. Our patient had severe aplastic anemia and EBV infection–associated hemophagocytosis syndrome, and he developed invasive *P. inflatipes* infection (proven by positive results of cultures of blood and bone marrow) during the neutropenic stage. The patient’s condition failed to respond to amphotericin B therapy, which was active against the isolate in vitro [10]. Previous studies have indicated that patients with severe aplastic anemia are at high risk of infection, probably because of profound neutropenia. Torres et al. [1] further demonstrated the high mortality (83%) associated with invasive mold infection (all due to *Aspergillus* species) in patients with aplastic anemia; the fatal cases were in patients with prolonged and severe neutropenia. Without bone marrow recovery, the prognosis for invasive fungal infections was grave.

In summary, we report a fatal case of *P. inflatipes* fungemia in a child with severe aplastic anemia. *P. inflatipes* should be included as an etiologic agent of invasive fungal infection in neutropenic patients with severe aplastic anemia.

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References


Rhinovirus and Lower Respiratory Tract Infection in Adults

SIR—In their prospective study about the role of viruses in community-acquired pneumonia, Tsoila et al. [1] used real-time PCR to identify an unexpectedly high prevalence (34 [45%] of 75 samples) of rhinovirus RNA in nasopharyngeal wash samples obtained from hospitalized school-aged children. Of 35 rhinovirus-positive patients, 20 (57%) had mixed infection, mostly due to rhinovirus and *Mycoplasma pneumoniae*. These findings are consistent with recent evidence, obtained by sensitive methods for detecting rhinovirus [1, 2], showing that the prevalence of rhinovirus is much higher than was previously thought, and suggest that rhinovirus can infect the lower respiratory tract.

However, almost all of the evidence about rhinovirus infection in the lower respiratory tract has been accumulated in studies involving pediatric populations, and few such data are available for adults. We recently conducted a laboratory-based prospective study to assess the prevalence of respiratory viruses among and their potential role on outcomes in hospitalized adults.

In brief, consecutive sputum, bronchoalveolar lavage, and endotracheal aspirate samples were collected and sent to the microbiology laboratories of 2 general hospitals. The sputum smears were accepted as suitable for the study if there were >25 polymorphonuclear leukocytes per high-power field (magnification, 40×). In addition to analysis with routine microbiological tests (i.e., direct sputum analysis and sputum culture), clinical specimens were analyzed by PCR or RT-PCR for the presence of 11 different viruses (influenza virus serotypes A and B; metapneumovirus; adenovirus; parainfluenza viruses 1, 2, and 3; respiratory syncytial virus; rhinovirus; and coronaviruses OC43 and 229E), as well as *Chlamydia* species, *M. pneumoniae*, and *Legionella pneumophila*. Nested PCR was used for detection of rhinovirus. Positive and negative control