in the previous 6 months as preemptive therapy or prophylaxis while receiving chemotherapy. Perea et al. [1] suggested that empirical or prophylactic use of azole agents could cause the selection of pathogenic non-albicans species of Candida, including C. krusei and C. glabrata, that exhibit decreased susceptibility to these agents in immunosuppressed patients. Thus, in our patients, it is likely that azole administration was effective against azole-susceptible C. albicans, allowing potentially azole-resistant C. albicans to emerge during neutropenia. The finding that all patients who developed candidemia had mucositis and previous colonization of C. albicans in the digestive tract supports the premise that azole-resistant C. albicans strains could have been acquired through the orogastrointestinal tract rather than through catheter insertion sites. Unfortunately, Candida isolates of surveillance cultures obtained from our patients were not available for in vitro susceptibility testing.

In conclusion, our findings emphasize that fungemia in neutropenic patients who are receiving azole prophylaxis and have previously received azole compounds should raise suspicion that organisms resistant to azole compounds are present, even if laboratory testing suggests that the isolate is C. albicans. Since this type of infection is associated with a high mortality rate, it should be intensively treated with alternative compounds, such as amphotericin B, caspofungin, and micafungin, until neutrophil levels recover. We are currently studying the prevalence of azole-resistant C. albicans fungemia in neutropenic patients with hematologic malignancies in our hospital to define the etiology of and the mechanisms involved in the development of azole resistance.

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References

**Campylobacter fetus**

Bacteremia in a Patient with Adult T Cell Leukemia

Sir—We describe a 69-year-old Japanese woman with adult T cell leukemia (ATL) who developed *Campylobacter fetus* bacteremia. The patient received a diagnosis of chronic-type ATL after hematologic examination in 1990. In March 1998, her disease transformed into acute-type ATL. Clinical examination revealed skin eruption, lymph node swelling, and splenomegaly. Laboratory tests revealed an elevated level of lactic dehydrogenase (1609 IU/L; normal range, 270–523 IU/L). Human T cell leukemia virus type 1 (HTLV-I) antibodies were present, and monoclonal integration of HTLV-I proviral DNA in PBMCs was demonstrated by Southern blot analysis. Chemotherapy consisting of adriamycin, cyclophosphamide, vincristine, and prednisolone resulted in a partial remission of symptoms.

However, 1 month later, the disease relapsed, and the leukemia cells became gradually resistant to the anticanancer agents. Subsequently, the patient had a high fever for 6 days. At the time of fever onset, her WBC count was 3.09 × 10^10 cells/L, with 94% ATL cells, and her neutrophil count was 5.86 × 10^9 neutrophils/L. Blood culture yielded spiral-shaped gram-negative bacilli (figure 1). Results of tests for atalase and nitrate reductase were positive. Results of tests for hippurate hydrolysis, H2S production, and urease were negative. On the basis of these findings, the bacillus was identified as *C. fetus*. After the start of therapy with sulbactam/cefoperazone, the patient’s septicemia improved immediately. Although she was discharged from the hospital 8 days after this episode, she died of tumor progression 1 month after discharge.

This is the first case report of *C. fetus* bacteremia complicating ATL. This disease is a highly aggressive, mature T cell neoplasm associated with HTLV-I infection, which is endemic in southwest Japan [1]. Patients with ATL frequently develop opportunistic infections during their disease course [1]. *Pneumocystis carinii, Strongyloides stercoralis, cytomegalovirus*, and a variety of fungi have been reported to cause infection in patients with ATL. The extent of immunodeficiency is similar to that in patients with AIDS. Although the frequency of opportunistic infections is higher in patients with ATL than in those with other hematologic malignancies [2], the mechanisms underlying this phenomenon are not known. In HTLV-I–infected individuals, the number of normal CD4+ cells is decreased, as in AIDS-infected individuals [3], and the production of naive T lymphocytes is impaired [3], as is the T cell response to Epstein-Barr virus [4].

*C. fetus* is commonly found in the gastrointestinal tract of wild or domestic cattle, sheep, goats, swine, dogs, cats, and fowls [5]. In contrast, *C. fetus* is an infrequent human pathogen but is sometimes identified in debilitated and immunocompromised patients, such as those with AIDS, liver disease, diabetes mellitus, chronic alcoholism, or malignancy [6–8]. *C. fetus* has been considered as a model system for bacterial pathogenesis on mucosal surfaces: intestinal colonization leads to bacteremia in the portal circulation. In a host with an impaired immune response, this bacteremia may be sustained and may lead to sepsis [9]. *C. fetus* strains are highly serum resistant because of the presence of

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**Table 1**

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**Figure 1**

*Campylobacter fetus* bacteremia in a patient with adult T cell leukemia. The patient received a diagnosis of chronic-type ATL after hematologic examination in 1990. In March 1998, her disease transformed into acute-type ATL. Clinical examination revealed skin eruption, lymph node swelling, and splenomegaly. Laboratory tests revealed an elevated level of lactic dehydrogenase (1609 IU/L; normal range, 270–523 IU/L). Human T cell leukemia virus type 1 (HTLV-I) antibodies were present, and monoclonal integration of HTLV-I proviral DNA in PBMCs was demonstrated by Southern blot analysis. Chemotherapy consisting of adriamycin, cyclophosphamide, vincristine, and prednisolone resulted in a partial remission of symptoms.

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Figure 1. Micrograph of the spiral-shaped gram-negative bacilli isolated from the patient. Micrography was performed after 4 days of primary culture on blood agar under microaerophilic conditions. The length of individual cells is longer than that of other Campylobacter species, and chaining is present, which further confirms its identity as Campylobacter fetus (original magnification, ×100).

a surface layer (a capsule-like protein structure [10]) that inhibits C3 binding and thus inhibits clearance by phagocytic cells [11]. In patients with AIDS, in particular, C. fetus has been recognized as a cause of relapsing bacteremia [6]. In consideration of the similarities of impaired T cell function between patients with ATL and patients with AIDS, C. fetus should be considered as a cause of bacteremia in patients with ATL.

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References
2. White JD, Zaknoen SL, Kasten-Sportes C, et al. Infectious complications and immunode-

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Cholelithiasis Possibly Induced by Protease Inhibitors in 3 Patients

Sir—We read with interest the article by Verdon et al. [1], who reported a human immunodeficiency virus (HIV)–infected patient with indinavir-induced cholelithiasis. Parallel to the major declines in HIV-associated morbidity and mortality, the toxicity of highly active antiretroviral therapy (HAART) has been increasingly recognized in daily clinical practice. It is interesting that the patient described by