Penicillium marneffei infection (PM) is an important disease among HIV-infected persons in Southeast Asia. Discovered in 1956 from the bamboo rat, Rhizomys sinensis, in Vietnam (1), PM was first identified in HIV-infected persons in 1988 (2). The disease has now been reported among HIV-infected persons from the United States, the United Kingdom, The Netherlands, Italy, France, Germany, Switzerland, Sweden, Australia, and Japan after they visited the PM-endemic region (3).

PM occurs late in the course of HIV infection. Our study found that the CD4+ cell count at the time of the diagnosis of PM was consistently less than 50 cells/ml. Clinical presentation included fever (in 99% of the patients), anemia (78%), pronounced weight loss (76%), generalized lymphadenopathy (58%), and hepatomegaly (51%). However, these conditions were not specific for PM and could be caused by HIV or other HIV-related opportunistic infections. A more specific finding was skin lesions, most commonly papules with central necrotic umbilication (4), which were seen in 71% of the patients.

In 63% of the patients with PM, a presumptive diagnosis could be made several days before the results of fungal culture were available. This was done by microscopic examination of a Wright-stained sample of bone marrow aspirate, touch smears of a skin biopsy specimen, or a lymph node biopsy specimen. It was easy to culture P. marneffei from various clinical specimens. Bone marrow culture was the most sensitive (100%), followed by culture of the specimen obtained from skin biopsy (90%) and blood culture (76%)(4).

The fungus was sensitive to amphotericin B, itraconazole, and ketoconazole (5). The current recommended treatment regimen is to give amphotericin B, 0.6 mg/kg/day for 2 weeks, followed by itraconazole, 400 mg/day orally in two divided doses for the next 10 weeks (6). After initial treatment, the patient should be given itraconazole, 200 mg/day, as secondary prophylaxis for life (7).

P. marneffei has been isolated from several species of bamboo rats in the disease-endemic area, but epidemiologic studies have thus far failed to define an environmental exposure associated with the disease (8-10).

References