Vaginal Ulceration and Local Lymphadenopathy in an African Immigrant

(See pages 441–2 for the Photo Quiz)

Figure 1. Immunohistochemical stain for lysozyme (arrow) revealing a giant cell with multiple ovoid cysts in the cytoplasm (original magnification, ×240).

Figure 2. Periodic acid Schiff stain showing ovoid yeasts (diameter, ∼10 μm) with thick cell wall, vacuolated cytoplasm, and narrow-based bud (arrow) (original magnification, ×420).

Diagnosis: Vaginal African histoplasmosis caused by *H. capsulatum var. duboisii* in an African patient with HIV infection.

The diagnosis was established by characteristic histopathological findings (figures 1–3) and IgG Western blot, immunodiffusion, and PCR results.

*H. capsulatum var. duboisii*, the causative agent of African histoplasmosis, is reported exclusively in Central and West Africa and in Madagascar [1, 2]. The natural habitat of *H. capsulatum var. duboisii* is in bird droppings and bat excrement. The seroprevalence of African histoplasmosis ranges up to 35% among cave guides, traders, and farmers living near bat caves [1]. It is thought that *H. capsulatum var. duboisii* enters the body through the lungs, although primary pulmonary infection has not been demonstrated. Infection occurs rarely via direct inoculation into the skin [3, 4]. The incubation period can vary from months up to several years after departure from regions in which the disease is endemic [1].

African histoplasmosis presents with granulomatous and suppurative lesions in cutaneous, subcutaneous, and osseous tissues, with local lymphadenopathy. The cutaneous manifestations include papular, nodular, ulcerative, ulceropolypous, eczematos, and psoriasiform lesions. Progressive systemic dissemination with fever, rigor, and miliary lesions in the liver, the spleen, and (rarely) in the lungs have been reported [1, 4].

The histopathological characteristics of African histoplasmosis are characteristic and distinct from those of American histoplasmosis. The typical lesions show clusters of multinucleated giant cells containing numerous oval, doubly contoured yeasts (8–15 μm in diameter), which have thicker walls and a greater diameter than *H. capsulatum var. capsulatum* (which are 2–4 μm in diameter).

Diagnosis relies on characteristic histopathological findings in biopsy specimens. Confirmation by culture is possible, but it is difficult because of the long generation time (6–30 h) [4]. Antigen detection in serum and urine samples is a sensitive test.
for \( H. \) capsulatum var. capsulatum. Serological tests often have negative results in patients with AIDS, but they are potentially useful for the diagnosis of African histoplasmosis, because \( H. \) capsulatum var. duboisii antigens cross-react with those of \( H. \) capsulatum var. capsulatum [5].

Despite the AIDS pandemic, there are no official reports on an increased incidence of acute disseminated African histoplasmosis [2]. In contrast with the regions of endemicity, where African histoplasmosis is common, Europe experiences few cases, with 23 documented cases since 1980, 6 of which involved patients with AIDS [4, 6–11].

Almost all AIDS-associated cases of \( H. \) capsulatum var. duboisii infection are disseminated rather than localized, suggesting the opportunistic character of this infection [1, 4, 5, 9].

Amphotericin B is the drug of choice in treating severe and disseminated cases; however, itraconazole and ketoconazole have shown good results in a large number of cases [4, 5, 7, 9]. Treatment for at least 6 months is mandatory, because the risk of relapse is greater with shorter durations of treatment [12]. Data on relapses and secondary prophylaxis in African histoplasmosis are rare; a maintenance therapy with itraconazole (200 mg daily) is proposed in cases of disseminated infection [4, 5, 7, 9, 12]. Recent data suggest that the risk of relapse among patients with HIV infection is small after 12 months of treatment with a sustained immunologic improvement (CD4+ cell count, >150 cells/μL) [5].

In our patient, treatment was started with itraconazole at a dosage of 400 mg/day administered orally for 9 months, and the vaginal ulceration resolved after 14 weeks of treatment. Prophylaxis against pneumocystis pneumonia and toxoplasmosis was initiated with sulfamethoxazole-trimethoprim at a dosage of 960 mg administered orally 3 times per week. HAART was started with tenofovir-emtricitabine administered once per day and nevirapine at a dosage of 200 mg administered twice per day. CD4+ cell counts increased to 260 cells/μL, and the HIV load decreased to <50 copies/mL. Because our patient had not been in Africa for 8 years, the vaginal ulceration seems to have been a late reactivation of an African histoplasmosis attributable to the immunosuppressive course of HIV infection.

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**Figure 3.** Grocott methenamine silver stain of a biopsy specimen, showing multinucleated giant cells, polymorphonuclear granulocytes, and multiple ovoid yeasts (arrow) (original magnification, ×124).