Right Buttock Rash for Thirty Years in a Patient from China
(See page 432 for the Photo Quiz)

Diagnosis: Chromoblastomycosis.

Sections of skin revealed pseudoepitheliomatous hyperplasia overlying a mixed inflammatory infiltrate with foreign body giant cells. Scattered pigmented spherical “sclerotic bodies,” the vegetative forms of the typical dematiaceous fungi that causes chromoblastomycosis, were visualized on hematoxylin-eosin stain (figure 1B and 1C). The skin sample sent for culture was plated onto Sabourad dextrose agar and grew brown-black velvety colonies over 2 weeks. On lactophenol aniline blue stain, septate dark hyphae with semi-erect conidiophores and barrel-shaped conidia were visualized. This is characteristic of Fonsecaea species, an etiologic agent of chromoblastomycosis [1].

Chromoblastomycosis is a heterogeneous group of skin and soft-tissue mycoses that was first described in 1911 by Alexandrino Pedroso [1–3]. The disease is endemic to parts of the Americas, Asia, and Africa [1–3]. The etiologic fungi are found in humid tropical and subtropical climates and live in decomposing plant matter and soil. There are multiple fungi of the Dematiaceae family (conidial fungi named for their darkly pigmented colonies and cell walls) that are known to cause chro-
moblastomycosis. These include Fonsecaea pedrosoi, Cladoiphala coronii, Rhinocladiella aquaspersa, Fonsecaea compacta, F. monophora, Phialophora verrucosa, Chaetomium funicola, Exophiala jeanselmei, and Exophiala spinifera [1, 4–5].

Transcutaneous penetrating trauma leading to inoculation of hyphal and conidial forms remains the most typical mode of infection. It is most commonly seen in men aged 30–50 years [6]. The lesions of chromoblastomycosis typically affect the lower extremities and, less commonly, the hands, arms, buttocks, and face. Lesions can begin as erythematous papules that develop into psoriasiform, verrucous or hyperkeratotic plaques, and nodules, ulcers, or exophytic tumors [1, 3, 6]. Superinfection may lead to lymphedema and elephantiasis [7]. Malignant transformation is a rare complication, and squamous cell carcinoma and malignant melanoma have been reported [7, 8].

Chromoblastomycosis is notoriously difficult to treat, and success rates are modest. Physical methods such as surgical excision, laser surgery, thermotherapy, and cryotherapy are most efficacious during the early phase of disease with localized lesions [9]. Long courses of antifungal monotherapy are required. Currently, itraconazole is favored, with a reported success rate of 89% for treatment of mild disease [10, 11]. Terbinaf ine has been shown to have a 74.2% cure rate, although the cost of therapy is prohibitive in many resource-poor settings [12]. Because of high rates of treatment failure with moderate to severe disease, combination therapy has been used, and in a small case series, itraconazole combined with terbinafine demonstrated an improved success rate in patients who experienced a failure of monotherapy [13]. Treatment with itraconazole in combination with cryo-therapy resulted in >70% of patients being disease free at a 3-year follow-up. Voriconazole has also been shown to have in vitro activity against dematiaceous fungi [14].

The difficulty in disease eradication and the high rates of recurrence have led to increased interest in newer antifungals for the treatment of chromoblastomycosis. In a phase III study, posaconazole demonstrated an 82% cure rate in refractory disease [15, 16]. Micafungin and caspofungin have also been found to have in vitro efficacy against dematiaceous fungi [17, 18].

In our patient, treatment with itraconazole in combination with monthly cryotherapy was initiated. He has experienced gradual improvement in his disease over 5 months of therapy.

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