Chromoblastomycosis: An Unusual Diagnosis in Europe

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Sir,

Chromoblastomycosis is a chronic cutaneous and subcutaneous infection due to a group of dematiaceous fungi including Fonsecaea pedrosoi, Cladophialophora carrionii, Phialophora verrucosa, F. compacta and Rhinocladiella aquaspersa (1). This infection occurs in tropical and subtropical regions and results from traumatic implantation of material contaminated by the fungus (1, 2). We report here a case of chromoblastomycosis imported from French Polynesia which was diagnosed in metropolitan France and responded to itraconazole.

CASE REPORT

An 86-year-old French woman sought medical advice for a cutaneous lesion of the right calf lasting for 6 years. She had no past medical history and had no treatment for several weeks. The patient had been living in Tahiti, French Polynesia, for almost 30 years and had not travelled abroad in this period of time except for metropolitan France. The lesion developed soon after a prick with bougainvillea thorn, increasing progressively in size, despite multiple topical and oral antibiotics, antifungal creams and recently topical corticosteroids. Physical examination disclosed a slightly painful plaque, 10 × 7 cm in diameter, verrucous in the centre surrounded by vegetating, ulcerated tissue covered with crusts and small abscesses (Fig. 1). Inguinal lymph nodes were palpable. Blood cell count disclosed eosinophilia at 1452/mm3. Renal and liver function tests were normal. A search for intestinal helminthiasis, filariasis and visceral larva migrans was negative. Histopathological examination of a cutaneous biopsy specimen disclosed a granulomatous inflammation in the dermis without necrosis, and thick-walled brown cells among the epithelioid and giant cells (Fig. 2). Direct microscopic examination of skin scraping in 10% sodium sulfur showed multiple sclerotic (muriform) bodies, presenting as dark brown sebaceous cells occurring in small clusters. A fungal culture grew F. pedrosoi within 3 days, confirming the diagnosis of chromoblastomycosis. The fungus was highly sensitive to ketoconazole, miconazole, itraconazole, fluconazole and amphotericin B, but resistant to 5-fluorocytosine and nystatin. Treatment with itraconazole was given at a daily dose of 300 mg. Within 3 months, dramatic clinical improvement was observed and the eosinophil cell count progressively returned to normal values. Treatment was stopped after 8 months. Five years later no relapse had occurred.

DISCUSSION

Chromoblastomycosis is caused by inoculation of fungus living as a saprophyte in soil and in decaying wood and vegetation, but the injury may be minor or may have been so long ago that it is not recorded (1, 2). F. pedrosoi is the most common agent for chromoblastomycosis and is frequent in tropical and subtropical zones. It has been reported mainly from Madagascar, Africa, North, Central and South America, Australia, Caribbean Islands, India, Japan and in Europe as an imported infection (1–5). However, cases have been confirmed as autochthonous in Northern Europe (6). The clinical presentation of chromoblastomycosis

Fig. 1. Verrucous plaque on the calf.

Fig. 2. Sclerotic bodies surrounded by inflammatory cells in dermis.
consists of nodules, verrucous or ulcerated plaques on the limbs (1). Secondary infection is possible and lymphoedema and elephantiasis may occur, as well as squamous cell carcinoma at affected sites. Lymphatic and haematogenic dissemination is unusual. Brain lesions have been described (5). Histological examination shows granulomatous reaction associated with an extensive fibrosis in dermis and subcutaneous tissue. Diagnosis is established by the presence in exudate and tissue of typical sclerotic bodies and by isolation and identification of the fungus. Our observation is typical of chromoblastomycosis as regards clinical and mycological features. However, the diagnosis was not established in European Polynesia but suggested on the basis of clinical features by one of us and then confirmed in the laboratory. Indeed the diagnosis can be difficult, but the subsequent application of topical corticosteroids may have made it easier. Eosinophilia in our patient is an unusual pattern and was noted on several blood cell counts (the search for helminthiasis carried out in French Polynesia was negative). It returned to normal after treatment with itraconazole. Eosinophilia has been reported in only one case of chromoblastomycosis but a search for parasitic disease was not exhaustive (7). Of note, eosinophilia has been observed in patients with other fungal infections, especially coccidioidomycosis and aspergillosis (8).

Chromoblastomycosis is notoriously difficult to treat. Small lesions can be resected with curettage and destruction and Mohs surgery. Treatments with carbon dioxide laser, cryotherapy and topical heat therapy have been reported (1, 9–11). However, recurrences are common. Widespread lesions require systemic treatment. Amphotericin B, thiabendazole, 5-flucytosine and ketoconazole are variably effective in this condition (1, 9, 12, 13). Itraconazole is becoming the treatment of choice (12) because it accumulates in the skin and subcutaneous tissue due to its lipophilicity. Terbinafine has also been used successfully for the treatment of chromoblastomycosis (13–15). However, total cure is difficult to assess and prolonged remission is acceptable. In our patient itraconazole was given for 8 months and no relapse had developed 5 years later.

As a result of international travel, tropical diseases may be observed in Europe. Physicians should keep in mind unusual infections such as chromoblastomycosis and laboratory diagnosis requires a high index of suspicion.

REFERENCES