Chromomycosis
Chromomycosis is a slowly progressive cutaneous mycosis caused by various dematiaceous fungi prevalent in tropical and humid countries. Major endemic areas include Madagascar [1], some islands of the Indian Ocean, South Africa, Asia, the Caribbean and Amazonian areas. Chromomycosis is attributed to several different dematiaceous fungi, including *phialophora, cladosporium*, *Wangiella*, and *Fonsecaea*, as well as a few other genera anecdotally found in the literature [2]. These agents are found worldwide in soil and in decaying plant materials, including wood.

In the Maghreb countries, chromomycosis is rare. Among 30 observations of deep mycoses in
Morocco, only 5 patients presented chromomycosis [3]. In Algeria, chromomycosis was first described by Perrin et al. in 1989 [4]. Subsequently, seven cases have been reported [5]. In Tunisia, this disease is rarely encountered. There is only one previous report, published in 2002, involving a woman living in a rural area [6].

The primary lesion is thought to develop as a result of percutaneous traumatic inoculation. From the site of inoculation, the lesion usually restricts itself to cutaneous and subcutaneous tissue, especially in parts of the body not protected by clothing.

The disease affects predominantly men. Typical lesions grow slowly over many years and tend to be found on the lower limbs. According to Minotto R et al., 27 percent of the cases involve other areas, including the medial canthus of the eye, the ear, neck, shoulder, chest, wrist and buttocks [7]. The morphology of the lesion may be tumor (as in our patient), nodular, verrucous, plaque-like, psoriasiform, or scar [2, 8]. Our patient worked as a butcher and trauma was frequent. However, his lesion occurred curiously in a covered area.

In our country chromomycosis lesions may be confused with leishmaniasis, verrucous tuberculosis, and tertiary syphilis. Scraping from a verrucous lesion in potassium hydroxide preparation reveals mycelia arising from sclerotic bodies. Mycologic tests are used to confirm the diagnosis. Culture in Sabouraud glucose agar media or Micosel agar allows isolation and identification of causal organisms within about 15 days. The principal causal agent is Fonsecaea a pedrosoi, followed by four species in order of frequency, Phialophora verrucosa, Cladosporium carrionii, Fonsecaea compacta, and Rhinocladiella aquaspera [8].

Histologic features of a skin biopsy stained with H&E show a dermal granulomatous infiltrate with a predominance of epithelioid cells surrounding fumagoid bodies.

If not diagnosed and treated early, chromomycosis has a chronic evolutional course. The most
frequent complication was secondary bacterial infection. Chronic chromomycosis also has potential association with epidermoid carcinoma (14 cases are reported in the literature) [9]. Central nervous system invasion is possible and may be fatal [10].

Our patient presented with a 1½ years duration tumor. Surgical removal followed by oral terbinafine therapy was associated with a favorable outcome—a complete remission within a 3-year followup period.

Treatment in localized lesions can be surgical as in our patient. Widespread lesions can be treated with itraconazole (100-200 mg/day) monotherapy [11] or its combination with oral 5-fluorocytosine (100-200 mg/day) [12]. In long-standing cases, removal of prominent cutaneous lesions by shave excision plus cryotherapy is also very helpful. Terbinafine (250-500 mg/day) for up to 12 months achieved good results in 42 Malagasy patients suffering from chromoblastomycosis [13]. Amphotericin B (1mg/kg/day) intravenously has been used in severe forms.

Chromomycosis is quite rare in our country; diagnosis was made by the histopathologic identification of fumagoid cells and then cultures revealing *Fonsecaea pedrosoi*. Even in Maghreb, dermatologists should be aware of this diagnosis. For limited involvement, treatment of choice appears to be excision followed by oral terbinafine.