Net Quiz

Multiple asymptomatic verrucous plaques over the legs

A 60-year-old male farmer presented to us with verrucous lesions over the right leg of five-year duration. Patient does not remember any trauma to this site prior to the onset of the lesion. On clinical examination, he had mild pallor. Cutaneous examination revealed a well demarcated, indurated, non-tender, verrucous plaque measuring 10 cm × 12 cm, situated over the lateral aspect of the lower half of the right leg, extending on to the pre-tibial area [Figure 1]. Surface was covered with blackish crusts at some places. A few lesions of similar morphology but of smaller sizes were distributed around the bigger lesion. There was a similar plaque measuring 3 cm in diameter situated over the medial aspect of the middle one-third of the left leg. There was no significant, regional lymphadenopathy. Systemic examination was unremarkable.

On investigation, the patient had hemoglobin of 10 gm/dl. Other routine investigations and radiological examinations were normal. KOH mount of the crust revealed brownish muriform bodies [Figure 2]. The lesion over the right leg was biopsied and subjected to the histopathological examination which revealed sub-epithelial dense aggregates of lymphocytes, neutrophils and epithelioid cells forming granulomas and brownish sclerotic bodies. The crusts present over the lesion were collected in a sterile bottle and sent for culture. After two weeks of incubation in Sabouraud's 2% glucose agar, velvety olive-black colonies were grown [Figure 3]. Lactophenol cotton blue mount of the smear prepared from the culture revealed long chains of elliptical conidia borne from erect, tall, branching conidiophores. Hyphae were septate and brown in color. Conidiophores were long and branched, and gave rise to chains of darkly pigmented budding conidia.

WHAT IS YOUR DIAGNOSIS?

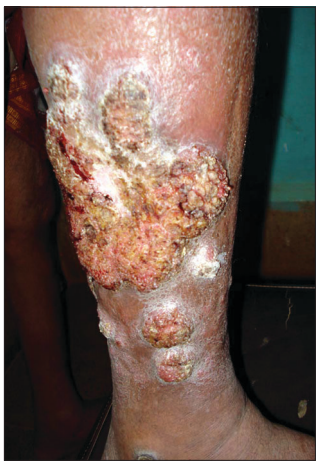

Figure 1: Verrucous growth over the right leg with crusting over the surface-close-up view

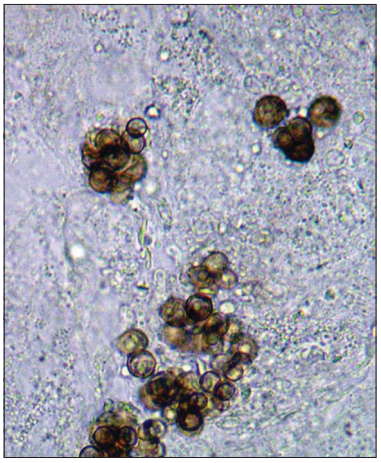

Figure 2: KOH mount of the crust showing muriform bodies

Figure 3: Fungal culture at the end of two weeks showing blackish colonies

How to cite this article: Bhagwat PV, Tophakhane RS, Kudligi C, Noronha T. Multiple asymptomatic verrucous plaques over the legs. Indian J Dermatol Venereol Leprol 2010;76:86.

Received: May, 2009. **Accepted:** October, 2009. **Source of Support:** Nil. **Conflict of Interest:** None declared.

Diagnosis

Cutaneous Chromoblastomycosis caused by *Cladosporium carrionii*

DISCUSSION

Chromoblastomycosis is a localized, chronic subcutaneous mycosis caused by any of the several species of dematiaceous fungi with melanin-type pigment in the wall, commonly seen in tropical and subtropical climates. It usually occurs in the lower extremities following traumatic implantation of the organism. The condition is characterized by verrucoid, ulcerated and crusted lesions, either flat or raised above the skin surface. Although dematiaceous fungi have a world-wide distribution, it has a higher prevalence in areas of tropical and sub-tropical climate. Chromoblastomycosis was first described in Brazil in 1914 by Max Rudolph, a German physician. He published a report of six cases and isolated a dark, grey to black-colored fungus. Medlar, in 1915, described the characteristic histologic appearance of sclerotic bodies, which thereafter named as Medlar bodies. Other synonyms include "copper-penny" bodies or "muriform" cells. They are globose-shaped, cigar-colored, thick-walled structures that are 4-12 μm in diameter. These structures multiply by septation, and they induce a purulent and granulomatous inflammatory reaction in tissue. Six species or genera are accepted to cause chromoblastomycosis-*Fonsacea pedrosoi*, *Phialophora verrucosa*, *Cladophialophora carrionii* and *Fonsacea compacta* are common causes. Rarely caused by Rhinocladiella aquaspersa and Exphiala species,[1] the disease is characterized by the presence, in infected tissue, of sclerotic bodies or Medlar bodies.

Clinically, plaque type of cutaneous chromo blastomycosis may resemble cutaneous tuberculosis, warts, cutaneous phaeohyphomycosis or cutaneous sarcoidosis. Histopathological differential diagnoses would be other subcutaneous mycoses and foreign body granuloma. But, KOH mount of the crust will clinch the diagnosis of chromoblastomycosis. Histopathological examination is highly characteristic.

The taxonomy of the organism that causes chromoblastomycosis is complex. Their identification is based on somewhat distinct microscopic morphologic features. Cladosporium is characterized by long chains of elliptical conidia (2 to 3 μm × 4 to 5 μm) borne from erect, tall, branching conidiophores. Hyphae are septate and brown in color. Conidiophores are long and branched, and give rise to chains of darkly pigmented budding conidia. Conidia are usually single-celled and exhibit prominent attachment scars (disjunctors) that may resemble "shield" cells. This organism may often fail to reveal chains of conidia on wet mounts because conidia are easily dislodged. *Phialophora verrucosa* produce tube-like or flask-shaped phialides each with a distinct colarette. Conidia are produced endogenously and occur in clusters at the tip of phialide. *Fonsacea pedrosoi* and *Fonsacea compacta* show conidial heads with sympodial arrangement of conidia, with primary conidia giving rise to secondary conidia.[2]

There are many case reports of chromoblastomycosis from India caused by agents other than Cladosporium carrionii.[3,4] Even though, it has been mentioned in the literature that *Cladosporium carrionii* is one of the common agents causing cutaneous chromoblastomycosis, we could not find many case reports in the literature and in India, it appears to be rare.

The main treatment of chromoblastomycosis involves the use of antifungal chemotherapy. Itraconazole with or without flucytosine is often successful, although responses to itraconazole alone are thought to be better if the causative organism is Cladosporium carrionii. Flucytosine alone or combined with amphotericin B may also be effective. Other options include terbinafine 250 mg/day, thiabendazole, cryotherapy or local application of heat. Surgery is indicated in very small lesions, and even in these should be combined with chemotherapy.[5]

Pradeep Vittal Bhagwat, Raghavendra S. Tophakhane, Chandramohan Kudligi, Tonita Noronha

Department of Skin and STD, Karnataka Institute of Medical Sciences, Hubli, Karnataka, India

Address for correspondence:
Dr. P.V. Bhagwat, Department of Skin and STD, Karnataka Institute of Medical Sciences, Hubli - 580 022, Karnataka, India.
E-mail: sharadapbagwat@yahoo.com

DOI: 10.4103/0378-6323.58707 - **PMID:** 20061751

REFERENCES

1. Momin YA, Raghuvanshi SR, Lanjewar DN. Cutaneous chromoblastomycosis. Bombay Hosp J 2008;50:299-301.
2. Forbes BA, Sahm DF, Weissfeld AS. Laboratory methods in

basic mycology, chapter 53. In: Bailey and Scott's diagnostic microbiology, 11th ed. Mosby, St. Louis: 2002. p. 774-7.
3. Rasul ES, Hazarika NK, Sarma A, Borua PC Sen SS. Chromoblastomycosis. J Assoc Physicians India 2007;55:149-51.
4. Muhammed K, Nandakumar G, Asokan KK, Vimi P. Lymphangitic chromoblastomycosis. Indian J Dermatol Venereol Leprol 2006;72:443-5.
5. Hay RJ, Moore MK. Mycology. In: Burns T, Breathnach S, Cox N, Griffiths C, editors. Rook's textbook of dermatology. 7th ed. London: Blackwell Science; 2004. p. 31.81-31.83.