

Chromoblastomycosis: A Case Report

Shanthala G.B., Rudresh S.M., Nagarathnamma T.

ABSTRACT

Chromoblastomycosis is a chronic fungal infection which is caused by the phaeoid fungi which are commonly seen in the tropical and subtropical climates. This infection is thought to be secondary to trauma or autoinoculation. The aetiological agent is a small group of dermatiaceous (pigmented) fungi. At least five species of fungi have been recognized to cause chromoblastomycosis: *Cladosporium carrionii*, *Fonsecaea compacta*, *Fonsecaea pedrosoi*, *Phialophora verrucosa*, and

Rhinocladiella aquaspersa. Here, we report a case of chromoblastomycosis from Karnataka, India. A 72 years old male patient presented with a history of a vegetating ulcer with crusting. The diagnosis of chromoblastomycosis was made by a demonstration of sclerotic bodies on KOH and the isolation of *Cladophialophora carrionii* on culture. The unique feature in this case is that it responded to the medical line of treatment with fluconazole.

Key Words: Chromoblastomycosis, Sclerotic body, *Cladophialophora carrionii*, Karnataka

INTRODUCTION

Chromoblastomycosis is a slowly progressing localized fungal infection of the skin and the subcutaneous tissues which is caused by several pigmented fungi. This disease is commonly seen in the rural workers working in the tropical and the sub-tropical climates [1]. Medlar reported the first case of Chromoblastomycosis in 1915 from Boston, in an Italian immigrant. He described the characteristic sclerotic bodies, which were thereafter named as the Medlar bodies, the other synonyms being “copper penny” bodies or “muriform” cells. In India, such cases have been reported from the sub-Himalayan belt and the coastal areas due to the hot and humid climates in those areas [1-3].

CASE REPORT

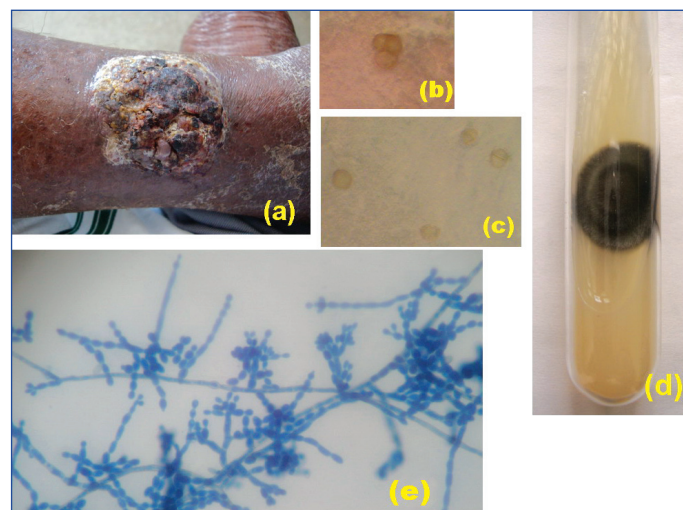
An 80 years male, an agriculturist by occupation, from the Ramanagara district of Karnataka, presented with history of verrucous ulcer, with crusting over the medial aspect of the left leg since one year. He gave a history of an injury which was caused by a coconut shell one year back. He developed a wound at the site of the injury which initially healed but it soon developed into an ulcer. He took medication from local practitioners, but the lesion did not heal. There is history of recurrent secondary infections which used to subside after a course of antibiotics but lesion remained same.

The examination revealed a solitary 5 x 5cm, well defined, painless ulcer with excoriation of the surrounding skin, the surface being covered with granulation tissue, with black crusts [Table/Fig-1a]. The regional lymph nodes were not palpable. The systemic examination was normal. The blood investigations and the chest x-ray were within normal limits. A differential diagnosis of tuberculosis verrucosa cutis or subcutaneous mycosis was made.

The ulcer was cleaned with sterile saline and the crusts were collected. Gram staining and ZN staining were done with the swabs which were taken from the ulcer and they did not reveal any pathology. A direct 10% KOH mount of the black crusts over the

ulcer showed 10–15 µm, golden-brown coloured sclerotic bodies with a pseudoseptate arrangement (a pathognomonic feature of chromoblastomycosis) [Table/Fig-1b,c]. The specimen was cultured onto Sabouraud's dextrose agar (SDA) with and without actidione. After three weeks of incubation at 25°C, both the SDA tubes showed velvety black flat colonies with a raised center and a black reverse [Table/Fig-1d]. Microscopy showed septate hyphae with a cladosporium type of conidiation [Table/Fig-1e]. The conidia were oval with smooth walls and variable sizes and shield cells were also seen. With this background, the case was diagnosed as Chromoblastomycosis which was caused by *Cladophialophora carrionii*.

The patient was put on 150mg of oral fluconazole (once a week) and Terbinafine local ointment was also prescribed for 12 weeks. The patient showed improvement with a decrease in the size of the lesions after 2 months.



[Table/Fig-1]: Showing verrucous ulcer with crusting (a); sclerotic bodies seen in 10% KOH mount (x400) (b, c); growth on SDA showing velvety black flat colony with raised center (d); cladosporium type of sporulation with long chains of oval smooth walled conidia on slide culture (x400) (e).

DISCUSSION

Chromoblastomycosis is a chronic disease of the skin and subcutaneous tissues which is caused by the phaeoid fungi of the genera *Fonsecaea*, *Phialophora*, *Cladophialophora* and *Rhinocladiella*, the commonest causative agent being *Fonsecaea pedrosoi* [2]. Rarely may the *Wangiella*, *Exophiala* and the *Chaetomium* species cause the disease.

This disease affects the exposed parts of the body, generally the lower extremities and it is more common in males. It is most commonly seen in agricultural workers, following a traumatic implantation of the aetiologic agent beneath the epidermis [2-3]. The disease spreads to the adjacent skin, thus causing satellite lesions and it rarely metastasizes to other organs [4]. The lesions are polymorphic or verrucoid. Secondary bacterial infections are common and repeated infections may lead to lymphatic fibrosis and elephantiasis of the legs [1]. Recurrences are common and this disease has a potential to predispose for the development of squamous cell carcinoma.

Chromoblastomycosis must be differentiated from tuberculosis verrucosa cutis, leprosy, leishmaniasis, mycetoma and tertiary syphilis. The diagnosis is based on a demonstration of sclerotic bodies in the tissue sections/KOH mounts and on the isolation and identification of the causative agent.

There are various reports of chromoblastomycosis from different parts of India. Sharma A et al have reported 2 cases of chromoblastomycosis which were caused by *Cladosporium carionii*, from Assam, India.[6] To the best of our knowledge, this is the first report of Chromoblastomycosis from Karnataka, south India.

Various treatment modalities like cryotherapy, thermotherapy, laser therapy and surgical excision are available [1, 5]. Generally, the disease responds poorly to the medical line of treatment [3, 4]. Newer azoles like itraconazole and fluconazole have been tried, with success [1].

Physical treatment modalities like local thermotherapy, cryotherapy, electrosurgery and radiation have the advantage of reducing the duration of the therapy and these are also non-

expensive. Their efficacies are largely anecdotal. But the evidence of clinical studies on these treatments is lacking.

Triazole derivatives and terbinafine are more effective in treating *Fonsecaea pedrosoi* and *Cladosporium carionii*. Rui Yao et al have reported a case of chromoblastomycosis which was successfully treated with fluconazole [7]. Sayal et al have reported a case of chromoblastomycosis which was treated with itraconazole, with significant improvement [4]. The efficacy of these newer anti-fungal agents is based on case reports and open labeled trials. The optimal dose and the duration of the treatment are still to be defined.

The therapeutic success is related to the causative agent (*C. carionii* is more sensitive than *Fonsecaea pedrosoi*), as well as to the clinical form and the severity of the chromoblastomycosis [8]. In the present case, the size of the lesion was small and it was superficial and less severe. Hence, with early intervention, we were able to achieve a favourable therapeutic response. An early diagnosis of the cases and appropriate treatment helps in preventing the morbidity which is caused by this disease.

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DECLARATION ON COMPETING INTERESTS:

No competing Interests.

Date of Submission: Oct 25, 2010

Date of Peer Review: Dec 06, 2011

Date of Acceptance: Jan 01, 2011

Date of Publishing: Aug 08, 2011