



Atypical Chromoblastomycosis: A Case Report from Eastern India

Authors

Ankur Ghosh, Firdous Jahan

Department of Dermatology, Venereology & Leprosy, RIMS, Ranchi

Abstract

*Chromoblastomycosis is chronic fungal infection of the skin and subcutaneous tissue caused by dematiaceous fungi. We present here a case of atypical chromoblastomycosis in a 16-year old student from a city with facial involvement along with limbs. The diagnosis was confirmed by histopathological examination of skin biopsies and isolation of *Fonsecaea pedrosoi* on culture. Atypical Chromoblastomycotic lesions may be confused with other cutaneous diseases.*

Keywords: *Chromoblastomycosis, dematiaceous fungi, Fonsecaea pedrosoi.*

Introduction

Chromoblastomycosis first described by Max Rudolph in 1914. It is an uncommon chronic localized fungal infection of the skin and subcutaneous tissue presenting with slow progressive verrucous lesion.^[1,2] It is caused by different species of dematiaceous fungi such as *Fonsecaea pedrosoi*, *Phialophora verrucosa*, *Fonsecaea compacta*, *Cladophialophora carrionii* etc. The prevalence is higher in rural populations and in countries with a tropical or subtropical climate. The fungi is usually found in soil, wood, and rotting vegetables and infection often results from trauma such as puncture from a splinter of wood.^[3] The lower limbs and dorsum of the hand are commonly affected. The lesion presents as a slow-growing, verrucous nodule. The present report deals with an atypical case of cutaneous chromoblastomycosis from Eastern India.

Case Report

A 16-year old apparently healthy male from Bariatu, Ranchi presented in Dermatology OPD of a tertiary care centre in Jharkhand with moderately itchy, crusted, slowly progressive lesion over right cheek and nose for last 6 months. He also had similar lesions on the left lower limb. He gave a past history of thorn prick in his left foot. Initially he developed a painless papule on lateral malleolus of left leg after one year of thorn prick injury. Similar lesions continued to occur in the vicinity which coalesced to form a large lesion with a rough surface. 6 months later a thick plaque type lesion appeared on Rt. Ala of nose which progressed to involve the whole of rt. cheek & Rt. dorsum of nose.

There was no history of any grain-like discharge from the lesions. He had been treated for cutaneous tuberculosis for 2 months around 4 months back at a local hospital. However, no improvement was noticed.

On Examination, a dry looking nodular, verrucous lesion, oval in shape of size 5cm x 2cm with hyper pigmented border was found on lateral malleolus of left leg (Fig. 1). A large irregular flatter hyper pigmented plaque with central atrophy was found on the right side of dorsum and ala of nose, cheek (Fig. 2) and arm (Fig. 3). Multiple papulonodular satellite lesions were also present around the facial lesion. There was no regional lymphadenopathy. The general health of the patient was unaffected.



Figure 1: A nodular, verrucous lesion on lateral malleolus of left leg



Figure 2: A Flatter plaque with central atrophy on the right side of nose and cheek



Figure 3: A Flatter plaque with central atrophy on arm

Clinically, D/D of mycetoma, cutaneous TB, chromoblastomycosis, and nocardiosis were considered. Routine hematological and biochemical investigations were normal. Histopathology from a representative lesion clinched the diagnosis for us.

On histopathology, tuberculoid and suppurative granuloma were found in dermis consisting of lymphocytes, plasma cells, histiocytes, epithelioid cells with langhans giant cells.

Epidermis showed spongiotic psoriasiform change. (Fig. 4) Within giant cells & stratum corneum, clusters of thick walled brown coloured refractile yeast cells were seen (Clusters of pennies appearance) (Fig, 5). Fungal culture on Sabouraud's dextrose agar showed slowly growing, colonies of *Fonsecaea pedrosoi*.

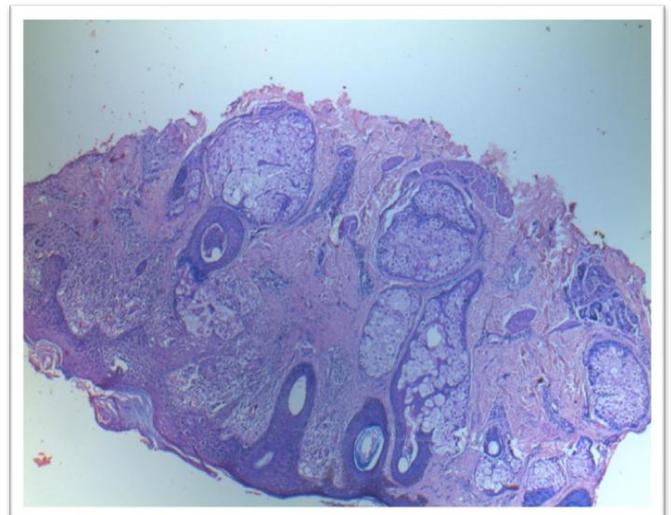


Figure 4: Histopathology from respective lesion

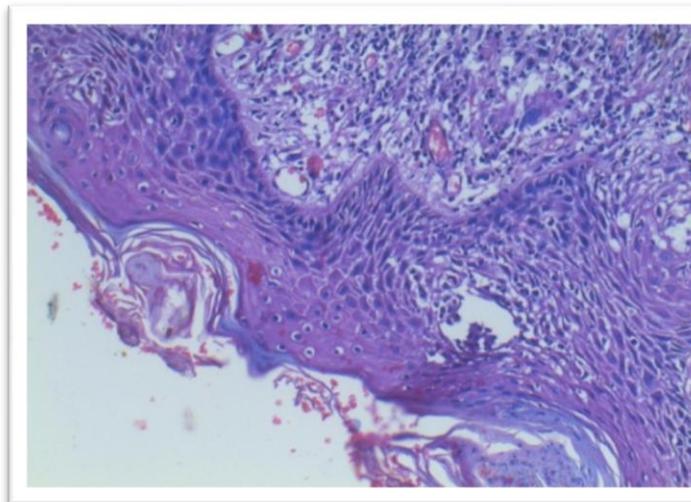


Figure 5: Clusters of pennies appearance

Discussion

Chromoblastomycosis is a chronic localized infection of the skin and subcutaneous tissue caused by a group of dematiaceous or black Fungi like *Fonsecaea pedrosoi*, *Fonsecaea compacta*, *Phialophora verrucosa*, and *Cladophialophora carrionii*, and occasionally by *Exophiala spinifera* and *Wangiella dermatitidis*.^[4,5,6,7] We have reported a case of cutaneous chromoblastomycosis caused due to a mold, *Fonsecaea pedrosoi*.

Cutaneous chromoblastomycosis clinically appear in the form of nodules, plaque like, verrucous, cicatricial or tumorous growth. Usually, the disease process remains localized to one lower extremity more common in middle-aged males (30-50 years) residing in rural areas.^[8,9] However our case was a 16 years boy from a city having a large irregular flatter hyper pigmented plaque with central atrophy on the right side of dorsum and ala of nose, cheek and arm along with a dry looking nodular, verrucous lesion, oval in shape of size 5cm x 2cm with hyper pigmented border on lateral malleolus of left leg. Multiple papulonodular satellite lesions were also present around the facial lesion. Ours was a case of cutaneous chromoblastomycosis of about 1 year duration, however skin involvement mimicked more long-standing forms as shown in the long duration cases presented by few authors previously.^[8,9]

Infection usually follows traumatic implantation of the etiological agent via minor wounds or penetration of foreign bodies such as wood splinters and is common among agricultural workers.^[7-9] However in our case, though history of thorn prick injury was present, the patient was a student studying in school.

Chromoblastomycosis must be differentiated from other conditions such as blastomycosis, leprosy, mycetoma, cutaneous tuberculosis, tertiary syphilis, malignancy, dermal leishmaniasis, sporotrichosis, paracoccidiomycosis, lobomycosis, protothecosis, phaeohyphomycosis, varrucosactis, and varrucosa vulgaris.^[10] Since there are no serological tests available for diagnosis of chromoblastomycosis mycological; histopathological investigations are essential to confirm the diagnosis of the disease. Histologically, the tissue response in chromoblastomycosis is nonspecific. Usually histopathological examination shows pseudoepitheliomatous type of hyperplasia, hyperkeratosis, intraepidermal abscess containing inflammatory cells, and medlar bodies.^[11] Microscopy and culture provides highly sensitive means of diagnosis that are both simple and inexpensive. Culture is found to be positive in 72% of the cases studied, while sclerotic bodies have been observed in 84% of the cases reported by various authors from different regions.^[9] In our case, both the investigations were found to be positive.

Our case is unique because so far very few reports on chromoblastomycosis due to *Fonsecaea pedrosoi* have been documented from Eastern India. Secondly, our patient was a 16 year old student from a city having history of trauma or wood stick injury prior to onset of infection. Thirdly, our patient presented with a large irregular flatter hyper pigmented plaque with central atrophy on the right side of dorsum and ala of nose, cheek and arm along with a dry looking nodular, verrucous lesion, oval in shape of size 5cm x 2cm with hyper pigmented border on lateral malleolus of left leg.

The treatment for chromoblastomycosis is cryosurgery in smaller lesions; triazole derivatives and terbinafine for larger ones; and in some cases, a combination of both.^[12] Here our patient responded well with prolonged oralitraconazole therapy.

In conclusion, instead of initiating treatment with antitubercular drugs, physician should consider chromoblastomycosis in the differential diagnosis of long standing skin lesions in patients from tropical and subtropical areas. Our report emphasizes the need for awareness about this disease

References

1. López Martínez R, Méndez Tovar LJ. Chromoblastomycosis. *Clin Dermatol* 2007;25:188-94.
2. Rubin HA, Bruce S, Rosen T, McBride ME. Evidence for percutaneous inoculation as the mode of transmission for chromoblastomycosis. *J Am Acad Dermatol* 1991;25:951-4.
3. Santos AL, Palmeira VF, Rozental S, Kneipp LF, Nimrichter L, Alviano DS, *et al.* Biology and pathogenesis of *Fonsecaea pedrosoi*, the major etiologic agent of chromoblastomycosis. *FEMS Microbiol Rev* 2007; 31:570-91.
4. Fatemi MJ, Bateni H. Oral chromoblastomycosis: A case report. *Iran J Microbiol* 2012;4:40-3.
5. Khan I, Khan AR, Khan MS. Clinicopathological study of cutaneous chromoblastomycosis in Pakistan. *J Pak Assoc Dermatol* 2012;22:122-5.
6. Chander J. In: *Textbook of Medical Mycology*. 2nd ed. Ch. 13. Chromoblastomycosis. New Delhi: Mehta publishers; 2002. p. 144-6.
7. Pradeepkumar NS, Joseph NM. Chromoblastomycosis caused by *Cladophialophora carrionii* in a child from India. *J Infect Dev Ctries* 2011;5:556-60.
8. Ramraje SN, Gokhale J, Gupta S. Cutaneous chromoblastomycosis. *JCase Rep* 2013.
9. Shah H, Verma K, Dhand PL, Soni R, Shakya P. Chromoblastomycosis. *Natl J Integret Res Med* 2011;2:57-9.
10. Bajpai T, Nandedkar S, Bhatambare G, Gagrani N. Cutaneous chromoblastomycosis: A case report from central India. *Int Med J SifaUniv* 2015;2:59-61.
11. Suh MK, Sung YO, Yoon KS, Ha GY, Kim JR. A case of chromoblastomycosis caused by *Fonsecaea pedrosoi*. *Korean J Dermatol* 1996;34:832-6.
12. Ranawaka RR, Amarasinghe N, Hewage D. Chromoblastomycosis combined treatment with pulsed itraconazole therapy and liquidnitrogen cryotherapy. *Int J Dermatol* 2009;48:397-400.