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Case Report

# A case report of chromoblastomycosis in a female farmer

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## Abstract

Chromoblastomycosis is recognized as a "Neglected Tropical Disease" (NTD) by WHO and is included in NTD road map 2021-2030 to increase surveillance and visibility of disease. It is a slowly progressing localized fungal infection of the skin and the subcutaneous tissues which is caused by several pigmented fungi. It affects individuals in tropical and sub-tropical regions often with history of trauma involving plant material, and has a male preponderance. The characteristic sclerotic bodies are seen microscopically, the other synonyms being "copper penny" bodies and medlar bodies. Chromoblastomycosis presents with polymorphic lesions often mimicking other dermatological conditions leading to diagnostic delays. Identification of pathognomonic sclerotic bodies in histopathology clinches the diagnosis to initiate early treatment and better patient outcomes. The disease may easily be misdiagnosed by those who are not sensitized to its clinical presentation and recognition of sclerotic bodies in the tissue sections. Here we present a case of Chromoblastomycosis in a female farmer, presenting with itchy skin lesion since 20 years.

**Keywords:** Chromoblastomycosis, Tropical, Pigmented fungi, Sclerotic bodies

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## 1. Introduction

Chromoblastomycosis is a slowly progressive cutaneous mycosis caused by pigmented (dematiaceous) fungi that occur as round, nonbudding forms in tissue sections. It is most often caused by one of five closely related species: Phialophora verrucosa, Fonsecaea pedrosoi, Fonsecaea compactum, Exophiala (Fonsecaea, Wangiella) jeanselmei, Exophiala spinifera, Rhinocladiella aquaspersa, and Cladosporium carrionii may each produce chromoblastomycosis. These fungi are saprophytes and thus can be found growing in soil, decaying vegetation, or rotten wood in subtropical and tropical countries. The primary lesion is thought to develop as a result of traumatic implantation of the fungus into the skin. The cutaneous lesions generally arise on the lower extremities and are variably pruritic, papular, nodular, verrucous, or plaque like lesions. While some of the lesions heal with scarring, new ones may appear in the vicinity as a result of spreading of the fungus along superficial lymphatic vessels or autoinoculation. Lymphatic disruption with elephantiasis may occur.[1,2,3,4,5]

## 2. Case Report

A 67 year old female farmer presented with itchy skin lesion over left side of face since 20 years. Initially lesions were small, coin sized, gradually increased over the years to involve left side of cheek, angle of mouth. She complained of itching of moderate intensity. There was also history of photosensitivity.

On examination there was sparseness of hair noted. Cutaneous examination showed well-defined atrophic plaque measuring around 6x7cms present over left side of face. Clinically she was provisionally diagnosed to have Discoid lupus erythematosus (DLE).

A skin biopsy was done for confirmation of diagnosis.

On microscopy, there was pseudoepitheliomatous epidermal hyperplasia, extensive dermal infiltrate of

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epithelioid histiocytes, lymphocytes, plasma cells and multinucleated giant cells, pigmented(dark brown) round to ovoid structures, and cross walls in spores equatorial septation rather than budding- features suggestive of Chromoblastomycosis.

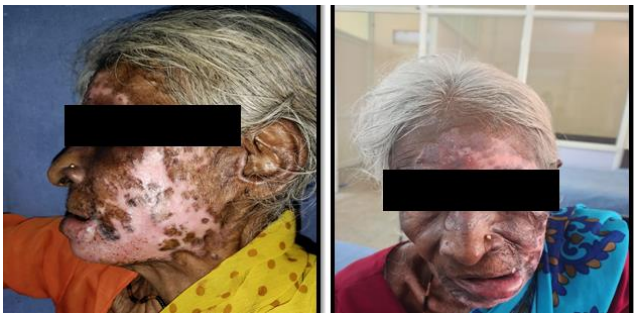

**Figure 1:** Well-defined atrophic plaque over left side of face (Hypopigmented areas)

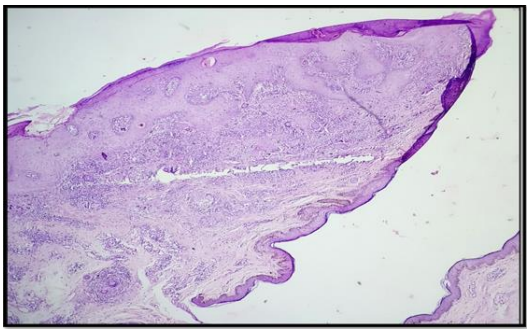

**Figure 2:** H&E, 5X – Pseudoepitheliomatous hyperplasia

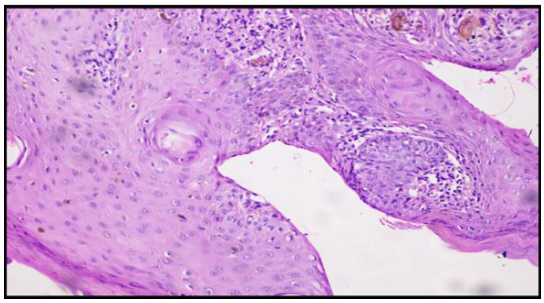

**Figure 3:** H&E, 40X – Pseudoepitheliomatous hyperplasia

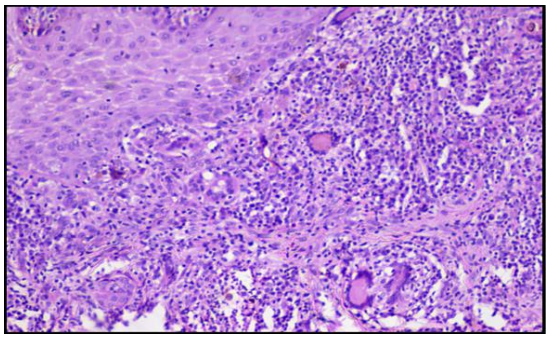

**Figure 4:** H&E, 40X, Externsive dermal infiltrate of epithelioid histiocytes, lymphocytes, plasma cells and multinucleated giant cells

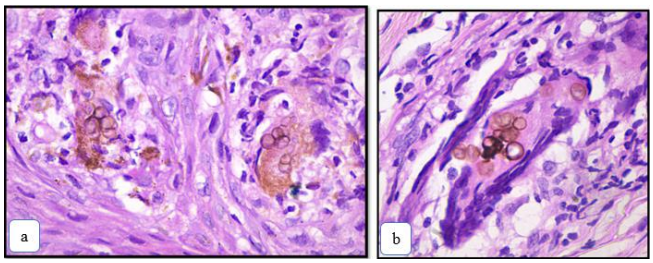


**Figure 5:** H&E, 40X, Pigmented (dark brown) round to avoid structure (a) Similar structure within giant cells (b)

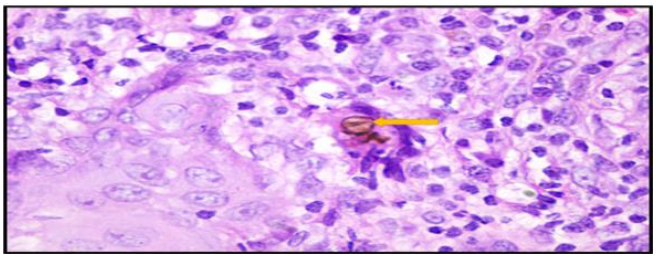

**Figure 6:** H&E, 40X, Cross walls in spores (arrow)

## 3. Discussion

Chromoblastomycosis has a higher prevalence in tropical and subtropical regions, which can be explained by the favourable environment for the fungus. The usual clinical presentation is a slowly enlarging exophytic warty plaque with superficial crusting and black dots. The disease is slowly progressive - the average time between the initial lesion and the clinical diagnosis is 15 years. Lesion starts as a small firm red / grey bump on the traumatized skin on the foot or hand. Grows slowly (2 mm/year) to form raised hyperkeratotic (crusted, warty-looking) plaque. Up to 70% of cases occur in males. Barefooted farmers account for almost 75% of patients with chromoblastomycosis.[6]

Although percutaneous inoculation of the fungus is widely accepted as the mode of infection, the fact that the cutaneous manifestations show no chancriform syndrome but rather a granulomatous infiltrate resembling that of North American blastomycosis suggests that the cutaneous lesions of chromoblastomycosis may arise by hematogenous dissemination from a silent primary pulmonary focus.[7]

Histopathologically in chromoblastomycosis, there is pseudoepitheliomatous epidermal hyperplasia and an extensive dermal infiltrate composed of many epithelioid histiocytes. Other components of the infiltrate include multinucleated giant cells; small abscesses and clusters of neutrophils; and variable numbers of lymphocytes, plasma cells, and eosinophils. Tuberculoid formations may be present, but caseation necrosis is absent. Fungi are found within giant cells as well as free in the tissue, especially in the abscesses. They appear as conspicuous, dark brown, thick-walled, ovoid or spherical spores varying in sizes from 6 to 12 µm that are likened to “copper pennies” and that may lie either singly or in chains or clusters.[8]

Because of their brown pigmentation, the spores can be seen easily without the use of special stains. Reproduction is by intracellular wall formation and septation, not by budding, and cross walls can be seen in some of the spores.

Transepidermal elimination of fungal spores may be observed, resulting in clinically visible black dots.

Scrapings for microscopic examination using 10% KOH should be taken from a site where black dots are seen on the surface of the lesion. It represents the transdermal elimination of fungal agents.[9]

Although chromoblastomycosis is not a fatal disease, it is chronic, and known for complications due to lymphatic damage and neoplastic transformation. Rarely, squamous cell carcinoma develops within longstanding chromoblastomycosis.

Most cases of chromoblastomycosis are chronic, indolent infections and eradication may be difficult even with prolonged therapy. A multiagent approach including surgical debridement, physical agents such as cryotherapy and systemic antifungal therapy may therefore be employed, although no one strategy is standard or universally effective.

## 4. Conclusion

The hallmark features of chromoblastomycosis are its diverse presentation and its refractoriness to treatment. The disease may easily be misdiagnosed by those who are not sensitized to its clinical presentation. Chromoblastomycosis, though is not frequent can be considered in the differential diagnosis of chronic skin lesions particularly in patients from tropical and subtropical regions for a early and appropriate therapy since it has a good prognosis if detected and treated at early stages. A multiagent approach including surgical debridement, physical agents such as cryotherapy and systemic antifungal therapy may therefore be employed, although no one strategy is standard or universally effective.[10,11,12]

## 5. Author Contributions

Sharon krupa contributed significantly to the investigation, conceptualization, data curation, formal analysis, visualization, original draft writing, and editing. Soumya B M and Vardendra Kulkarni played a key role in approving the final manuscript, providing critical revision assistance, and contributing to supervision, project administration, and validation. All authors confirm that they have no competing interests concerning this manuscript and have diligently reviewed and consented to its final version.

## 6. Data Availability Statement

All data that reported in this study is available from the corresponding author on reasonable request.

## 7. Source of Funding

None.

## 8. Conflicts of Interest

There is no conflict of interest.

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