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Case Report

Cutaneous chromoblastomycosis-unusual presentation: a case report

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ABSTRACT

Chromoblastomycosis is a chronic, progressive, cutaneous and subcutaneous fungal infection following the traumatic implantation of certain dematiceous fungi. These are naturally pigmented fungi with two clinical forms viz. cutaneous and cerebral. We present a case of cutaneous chromoblastomycosis presenting as a swelling over right forearm in a 47-year-old lady farmer. Clinically cutaneous tuberculosis was suspected, however on histopathology, diagnostic 'sclerotic cells or medlar bodies' amidst the granulomatous inflammation and microabscesses were seen.

Keywords: Chromoblastomycosis, Dermatomycosis, Sclerotic bodies

INTRODUCTION

Chromoblastomycosis is a chronic, non-contagious, localised fungal infection of cutaneous and subcutaneous tissue. This mycosis is distributed worldwide, but most cases have been reported from tropical and subtropical regions.[1] Lesions of chromoblastomycosis are polymorphic and must be differentiated from those associated with many conditions. Dematiaceous (naturally pigmented or melanised) fungi are aetiological agents. These agents gain entrance through transcutaneous puncture wounds, usually by parts of plants, wood, soil or rotting vegetables.[2,3] The cutaneous lesions generally present as slowly progressive scaly-warty plaques in the lower extremities. That is why chromoblastomycosis is often confused clinically with verrucous carcinoma, squamous cell carcinoma or some other infectious process such as cutaneous tuberculosis. Routine histopathological examination settles this enigma.[4] Direct light microscopic demonstration of pathognomonic, brown sclerotic bodies also called as Medlar bodies, muriform cells and copper pennies in biopsy specimens and positive fungal culture confirm the diagnosis.[5,6]

Chromoblastomycosis is a therapeutic challenge for clinicians due to the recalcitrant nature of the disease. Treatment modalities include, physical treatment, chemotherapy and combination therapy.

CASE REPORT

A 47-year-old lady farmer presented with complaint of swelling over right forearm since few months. Clinically cutaneous tuberculosis was suspected, and surgical excision of the lesion was planned. We received specimen of excision of lesion over right forearm measuring 5.8x2.6x1cm. The covering skin measured 5.8x2.6cm and showed a grey white plaque like lesion measuring 0.5cm in diameter. On serial sectioning, the cut surface showed grey white to grey yellow ovoid area measuring 2.5x1.2x0.6cm beneath the plaque like lesion (Figure 1).

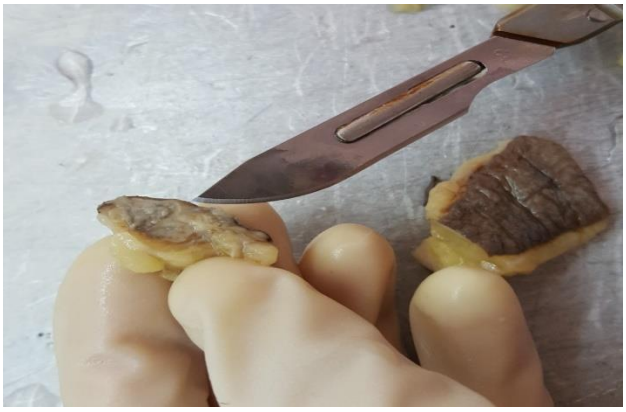

Figure 1: Cut section of specimen showing grey white to yellow lesion.

Microscopy of the lesion revealed hyperkeratosis and acanthosis of epidermis. In the dermis there were many epithelioid granulomas, numerous multinucleated giant cells, focal dense acute inflammatory infiltrate forming microabscesses and small foci of necrosis. Amidst this infiltrate, multiple thick walled dark brown, ovoid to spherical 'sclerotic cells' in single and in small clusters (Figure 2).

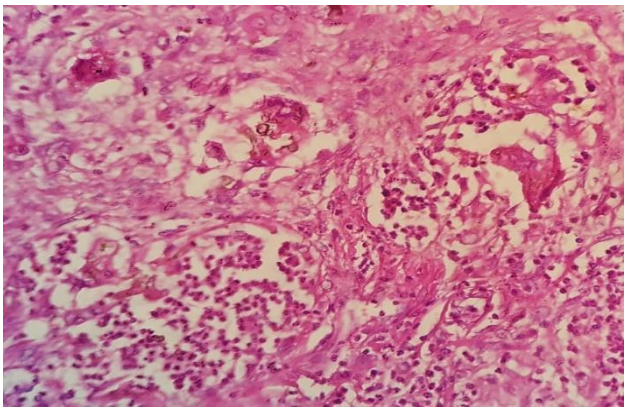

Figure 2: Neutrophilic microabscesses and few giant cells containing brown colored 'Muriform bodies' (H and E-40X).

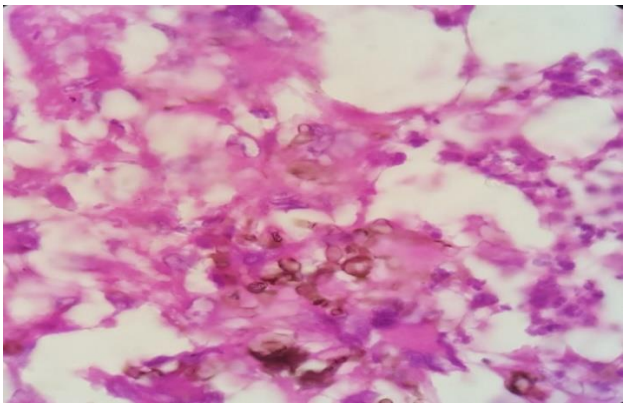

Figure 3: Many brown colored 'Sclerotic cells' or 'Medlar bodies' (H and E- 40X).

These 'copper pennies or muriform bodies' were also present extracellularly, within the macrophages and the multinucleated giant cells (Figure 3). With these features diagnosis of cutaneous chromoblastomycosis was given and microbiological studies were advised. Patient is responding well to treatment and is on follow up.

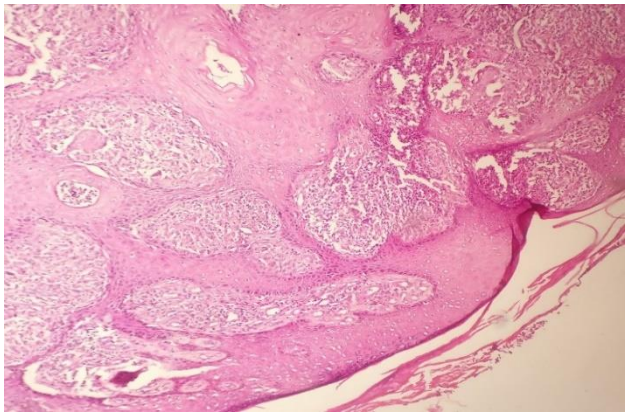

Figure 4: Hyperkeratosis and acanthosis of epidermis (H and E-40X).

DISCUSSION

Chromoblastomycosis is also known as chromomycosis, cladosporiosis, fonseca's disease, pedroso's disease, phaeosporotrichosis and verrucous dermatitis. It was first reported in Brazil in 1914 by Max Rudolph, a German physician.[7] in 1915, Lane and Medlar described the sclerotic cells which were subsequently called Medlar bodies.[5,6]

The international society for Human and Animal Mycology in 1992, recommended the name Chromoblastomycosis to define the disease, which Terra et al coined in 1922.[8,9] in 1920, Pedroso and Gomes, Brazilain Physicians reported four cases that had been under observation for many years.[10]

The causative agent of chromoblastomycosis is dematiaceous fungi like *Fonsecaea pedrosoi*, *Fonsecaea compactum*, *Phialophora verrucosa* and *Cladophialophora carrionii*. Rarely *Erophiala spinifera* and *Wangiella dermatitidis* have also been implicated as causative agents.[11] These agents are saprophytes found in soil, decaying vegetation or, rotten wood in subtropical and tropical countries.

The most common mode of infection in cuataneous chromoblastomycosis is traumatic caytaneous injury that is often not remembered or realised by the patient. Men and women in the age group of 30-40 years are most commonly affected as they are involved in agricultural work and are prone to injury. Hematogenous dissemination is another rare mode which may cause extensive cautaneous lesions and this mode carries grave prognosis.

The lesions develop slowly. Initially a warty nodule limited to the skin and the subcutaneous tissue is produced at the site of implantation. As the disease spreads, it forms plaques. In long standing cases, lesions may appear tumorous and even cauliflower like. While some of the lesions heal with scarring, new lesions may appear in the vicinity resulting from the spread of fungus along superficial lymphatics. Common complications are ulceration, lymphedema and secondary infection, however mortality is rare. Very rarely, epidermoid carcinoma can occur in long standing cases. Legs, arms and buttocks are common sites of involvement. Unusual sites include pleural cavity, ileocecal region, tonsil and laryngo-tracheal region.[12] Our case presented with the lesion on forearm. Chromoblastomycosis is more concentrated in tropical and subtropical countries. The African country Madagascar has highest incidence of chromoblastomycosis in world.[13] In India, it was first reported by Thomas et al in 1957, from Assam.[14] Cutaneous chromoblastomycosis has many mimickers like cutaneous tuberculosis, mycetoma, leprosy, leishmaniasis, syphilis and other fungal infections. The advanced or long standing cases can also be reported as suspicious of a malignant lesion. In our case the clinical diagnosis was cutaneous tuberculosis.

A combined histopathological and mycological diagnosis is a highly sensitive approach. Histologically, owing to its association with pseudoepitheliomatous hyperplasia, cutaneous chromoblastomycosis can mimic a number of epithelial malignancies. Roy D et al, encountered similar difficulties in distinguishing chromoblastomycosis from squamous cell carcinoma.[15] Microscopic demonstration of characteristic dark brown, thick walled, ovoid to spherical fungal cells also known as sclerotic or muriform bodies is diagnostic of chromoblastomycosis. Recognition of exact fungal agent requires culture studies.

Cutaneous chromoblastomycosis has a high recurrence rate and is a therapeutic challenge. Antimycotic medication with itraconazole and/or terbinafine, surgery, local cryotherapy and thermotherapy are the popular eradicative measures. However, choice of treatment varies with case.[4,16] In refractory cases, photodynamic therapy using 5-aminolevulinic acid and irradication in combination with antifungal therapy has been successfully used. To prevent local dissemination, a margin of uninfected tissue should be removed. Trauma et. al preferred to reduce the lesion size with oral terbinafine before surgical excision.[16]

CONCLUSION

A cutaneous chromoblastomycosis should be kept as a differential in patients presenting with chronic skin lesions. Histopathological examination is diagnostic and fungal culture is essential for identification of causative agent. As cutaneous chromoblastomycosis is refractory to treatment and complications are known to occur, early diagnosis of this condition is important.

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