Subcutaneous Chromoblastomycosis Caused by *Rhinocladiella* Species in Rhode Island

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ABSTRACT

Chromoblastomycosis (CBM) is a subcutaneous fungal infection caused by one of several dematiaceae molds with melanotic pigmentation in the cell walls. CBM is characterized by various clinical and dermatological features, leading to common misdiagnosis as several other infectious and noninfectious diseases. Histopathologically, in addition to the pathognomonic muriform cells or "copper pennies," the causative agents of subcutaneous and systemic mycosis lead to a granulomatous reaction due to the influx of mononuclear phagocytic cells and a suppurative infiltrate of neutrophils. Treatment of CBM can be challenging as no standard treatment has been established.

This case highlights a rare presentation of subcutaneous chromoblastomycosis caused by *Rhinocladiella* species in the United States with satisfactory management. It emphasizes the role of occupational and exposure history, keeping a high index of suspicion in cases of verrucous nodules or plaques with new satellite lesions on extremities, and recognizing this entity's distinct dermatopathology characteristics.

KEYWORDS: Chromoblastomycosis; Cutaneous Mycoses; Dematiaceous Fungi; Verrucous Dermatitis; *Rhinocladiella*

INTRODUCTION

Chromoblastomycosis (CBM), or chromomycosis, is a subcutaneous fungal infection caused by one of several dematiaceae molds with melanotic pigmentation in the cell walls. CBM is characterized by various clinical and dermatological features, leading to common misdiagnosis as several other infectious and noninfectious diseases.[1]

The main tissue form of the fungus in CBM is thick-walled, brown sclerotic bodies with internal septae.[2] Commonly denominated "Medlar/sclerotic bodies," "copper pennies," and "muriform cells," these entities represent an intermediate vegetative state between yeasts and hyphae. CBM is most frequently found in tropical and subtropical climates and is most commonly associated with the genera *Fonsecaea* and *Cladophialophora*, with fewer cases reported due to *Rhinocladiella, Phialophora*, and *Exophiala*.[1]

CASE REPORT

The patient is a 74-year-old male Veteran with a history of chronic obstructive pulmonary disease, hyperlipidemia, gastroesophageal reflux disease, and hypertension, who presented to the Veteran Affairs dermatology clinic in Providence, Rhode Island, with a reported year-long history of three, approximately 15 mm by 15 mm, verrucous nodules in a linear distribution across the left knee [**Figure 1A**]. The lesions were tender to palpation. They were treated by an outside dermatologist with topical steroids but continued to enlarge. Due to suspicion of cutaneous malignancy, broad shave biopsies of each nodule were completed for surgical pathology diagnosis. However, within a few weeks of the initial biopsies, the patient noted a similar lesion inferior to the original lesions [**Figure 1B**]. This prompted a repeat visit to the clinic, where a punch biopsy and tissue culture were taken of the newer lesion.

The patient was born and raised in Rhode Island. He served in the Navy from 1968–1972, stationed in the Mediterranean, Cuba, and Tunisia, predominantly working on ships, with notable exposure to asbestos, then worked as a bricklayer, and is now retired. His hobbies include gardening and camping. His only recent travel history was visiting Florida for a week at a time, with the last visit several years prior to presentation.

A review of systems was notable for occasional chills and chronic cough, but he denied fevers, chest pain, worsening dyspnea, abdominal pain, genitourinary symptoms, joint pains, and rashes. Laboratory studies revealed normal renal and liver function tests, along with negative hepatitis B, hepatitis C, and HIV serologies.

Histopathology

The surgical pathology diagnosis of all three lesions on the left knee revealed chromoblastomycosis, with associated florid pseudoepitheliomatous hyperplasia and prominent dermal suppurative granulomatous inflammation that includes several bronze-colored spores [**Figure 2A**]. GMS and PAS-D stains performed on all parts stained the fungal organisms, consistent with the diagnosis [**Figures 2B,C**].

Mycology

The fungal culture taken of the newer inferior lesion on the left knee isolated *Rhinocladiella* species.

Figure 1A. Presentation

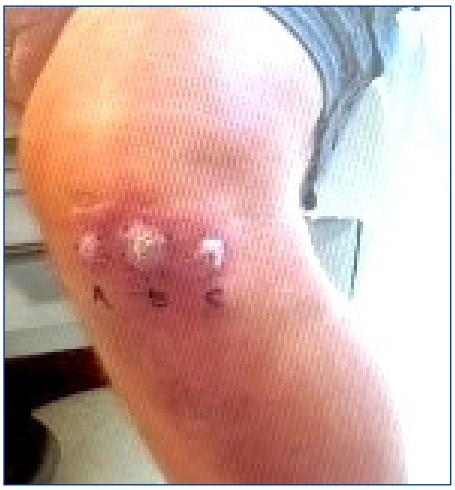


Figure 1B. Follow-up visit (one month later)

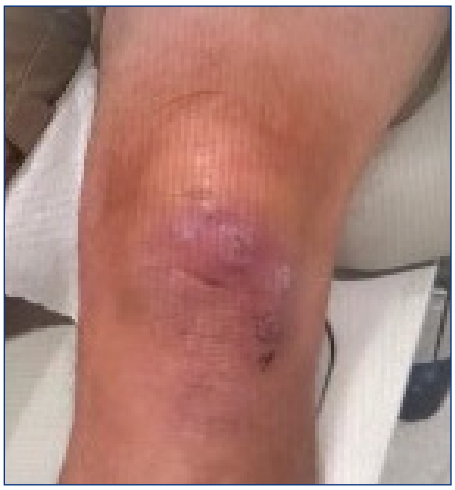


Figure 1C. Receiving terbinafine therapy (five months later)

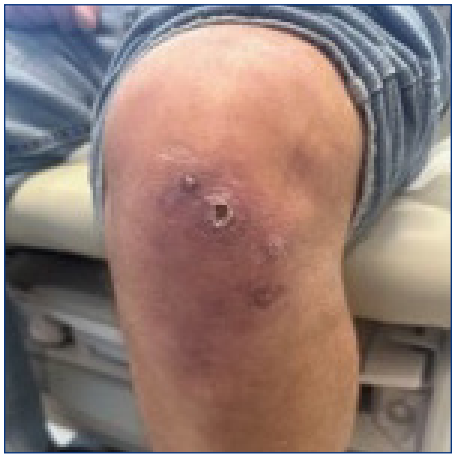


Figure 2A. High-power view shows giant cells with intracellular round pigmented sclerotic bodies ("copper pennies"), 200x

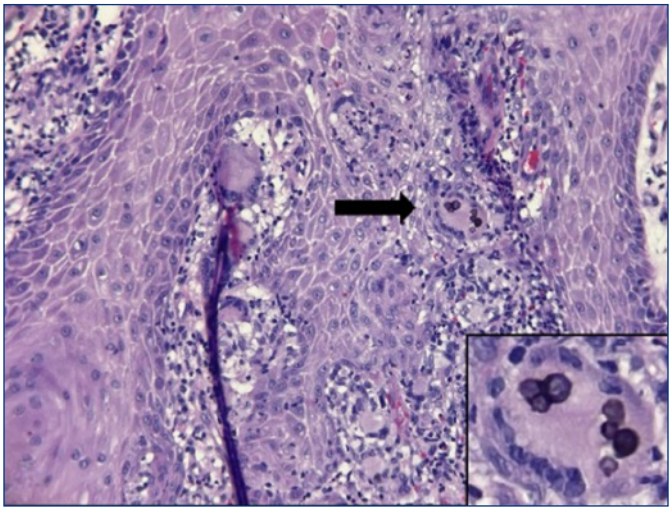


Figure 2C. Periodic acid-Schiff with diastase (PAS-D) stains the sclerotic bodies a magenta hue.

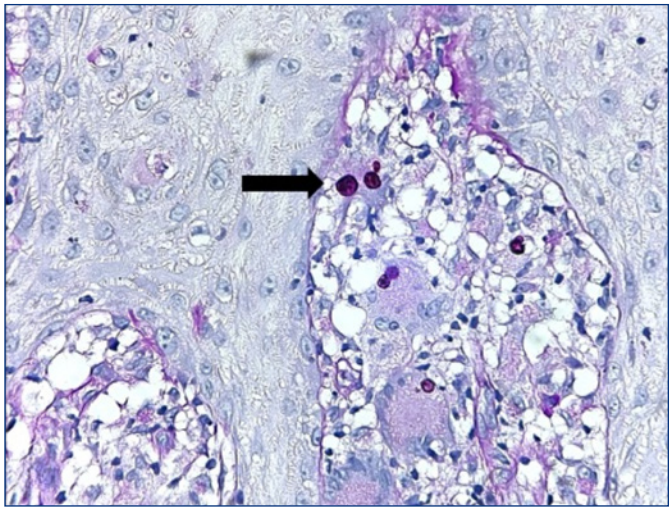
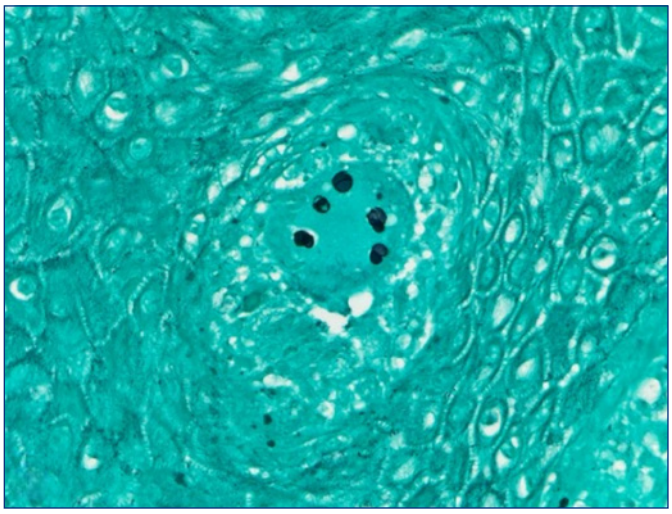


Figure 2B. Grocott's methenamine silver (GMS) stain highlights the sclerotic fungal wall black.



Along with multidisciplinary management with Infectious Disease specialists, terbinafine was initiated [**Figure 1C**] with a plan for discontinuation once the patient met the following criteria for three consecutive months: clinical resolution, defined as lesion clearing except for scarring; resolution of local pruritus and pain; and negative monthly skin biopsies, potassium hydroxide preparations, and tissue cultures. Terbinafine was favored over itraconazole due to significantly fewer drug-drug interactions with his home medications, given the anticipated extended course of treatment.

DISCUSSION

CMB is characterized as one of the Neglected Tropical Diseases caused by inoculation of pathogenic dematiaceous

fungi.[3] Based on published case series and epidemiological surveys, incidence in the United States is estimated at one per 8,625,000.[4] However, actual incidence and prevalence are unknown, as reporting of this disease is not mandatory. Most cases are caused by the *F. pedrosoi* species complex, with higher prevalence in Africa and Latin America than in Asia and Australia; rarely are CBM cases observed in northern Europe and the United States of America.[1] This was a case of a patient who developed subcutaneous chromoblastomycosis caused by *Rhinocladiella* species in the United States. There have been reports of the genus Rhinocladiella relating to CBM, including *R. aquaspersa*, a classic chromoblastomycosis agent; R. tropicalis, which was isolated from four Brazilian patients; and *R. phaeophora*, whose only report derives from a case in Thailand. There have been two cases in the worldwide literature of *R. similis* causing CBM.[5]

The clinical diagnosis of CBM is challenging due to the polymorphic dermatologic characteristics of lesions, with the differential diagnoses including cutaneous malignancies (as was initially suspected in our case), Bowen's disease, squamous cell carcinoma, and keratoacanthoma; and cutaneous infections such as cutaneous mycobacteria, cutaneous leishmaniasis, lobomycosis, paracoccidioidomycosis, phaeohyphomycosis, and sporotrichosis, necessitating confirmation of the etiological agent in the patient's tissues. Maintaining a high index of suspicion for a rare cutaneous fungal infection is crucial, especially when new lesions occur, such as in this patient. As in this case, most patients do not remember a traumatic inoculation event prior to the disease because it may go unnoticed, or a long time may have elapsed from the injury to the onset of the disease. Studies have reported the duration of disease until diagnosis from one month to four decades with a mean duration of 3.1 to 8.3 years.[4] Despite the patient's limited travel history and thus low epidemiological suspicion for CBM, his occupation as a bricklayer may have exposed him to contaminated soil. CBM has been more frequently reported on the extremities in men and rural workers, with researchers postulating that this distribution is related to the notion that men have been more historically involved in activities that expose them to environments where etiological agents are present. Although infrequent, extracutaneous dissemination with muriform cells and confirmatory isolate in tissues has been described mainly in immunocompetent individuals, warranting a comprehensive review of systems in affected patients.[4]

Histopathologically, in addition to the pathognomonic muriform cells or "copper pennies," the causative agents of subcutaneous and systemic mycosis lead to a granulomatous reaction due to the influx of mononuclear phagocytic cells and a suppurative infiltrate of neutrophils. This results in what has been recently described as mixed organized mycotic granuloma (MOMG).[6]

Lastly, treatment of chromoblastomycosis can be challenging as no standard treatment has been established. Low cure rates and high relapse rates can complicate therapy, especially in chronic and extensive disease, with the most common causes of treatment failure being treatment abandonment, prolonged therapy, side effects, and antifungal resistance.[7] Reported treatments have ranged from physical therapeutic methods (surgery for smaller lesions, cryosurgery, electrosurgery, and local heat), antifungals such as itraconazole or terbinafine administered as monotherapy, and combined therapy with other antifungals or with physical agents.[4] Eradication is important as there are reports of progression to squamous cell carcinoma in long-standing, inadequately treated lesions.[4,8]

In conclusion, this case highlights a rare presentation of subcutaneous chromoblastomycosis caused by *Rhinocladiella* species in the United States. It emphasizes the role of occupational and exposure history, keeping a high index of suspicion in cases of verrucous nodules or plaques with new satellite lesions on extremities, and recognizing this entity's distinct dermatopathology characteristics.

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Disclosures

Our institution does not require ethical approval for reporting individual cases or case series.

Written informed consent for treatment and publication was obtained from the patient and is on file. The patient gave written permission for the publication of clinical details and images. Identifying information has been removed to protect anonymity.

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