CASE REPORT

From Scar to Infection: Cutaneous Chromomycosis under the Microscope

Jéssica Sandoval,* Fernanda Ron,* Cindy Tipán,* Estefany Proaño,* Wilma Vaca**

* *Dermatology Postgraduate Physicians – Universidad Tecnológica Equinoccial*
** *Dermatology Attending Physician – Calderón Teaching General Hospital*

Correspondence:
Quito, Ecuador
jesyquitao6@hotmail.com
0987444149

Key words: Chromomycosis, chromoblastomycosis, deep mycosis

How to cite this article:
Sandoval J., Ron F., Tipán C., Proaño E., Vaca W. De Cicatriz a Infección: La Cromomicosis Cutánea bajo el Microscopio – Reporte de un caso clínico. Rev Dermatol Cent Úraga. 2025;7(2).

Date of receipt: 26/01/2024
Date of acceptance: 28/02/2025

ABSTRACT

Chromoblastomycosis is a chronic, granulomatous, and refractory fungal infection caused by melanin-producing fungi found in soil, wood, and vegetation in tropical and subtropical regions. It primarily affects male agricultural workers following trauma to the extremities. Its clinical presentation is polymorphic (nodular, verrucous, plaque-like, tumoral, and scar-like), making differential diagnosis difficult with other infectious and non-infectious conditions such as verrucous tuberculosis, leprosy, or squamous cell carcinoma.

Diagnosis is based on the identification of muriform bodies through direct examination or fungal culture; molecular techniques help differentiate species, although they are often inaccessible in endemic areas. Treatment includes antifungal agents such as itraconazole and terbinafine, with complementary approaches like surgery or cryotherapy for small lesions. However, outcomes are limited and treatment tends to be prolonged.

We present the case of a 77-year-old male patient with a lesion initially classified as a scar, which was later confirmed to be Cladosporium spp., highlighting the importance of early diagnosis to prevent disease progression and complications.

INTRODUCTION

Chromoblastomycosis, or chromomycosis, is a chronic, granulomatous, polymorphic, and suppurative fungal infection of the skin and subcutaneous tissue, caused by several species of melanin-producing or dematiaceous fungi. Their parasitic forms are known as fumagoid or muriform cells (sclerotic bodies), which are round/oval, brown-colored cells with thick walls measuring 4–12 microns in diameter. These cells reproduce by septation in two distinct planes and are also referred to as Medlar bodies, representing the invasive form,[1-3] they are found in soil, plants, and decaying Wood, and are prevalent in tropical and subtropical regions of the world.[2]

Eight fungal species, grouped into four genera, are currently implicated: Cladosporium carrionii, Fonsecaea pedrosoi, Fonsecaea compacta, Phialophora verrucosa, Wangiella (ex-Exophiala) dermatitidis, Exophiala spinifera, Rhinocladiella cerophila y Rhinocladiella aquaspersa.[4-6]

This infection primarily affects the lower limbs (in 85% of cases), as well as the upper limbs (predominantly the back of the hand), and other anatomical areas such as the arms, trunk, neck, forearms, face, and buttocks.[7,8]

Most patients have a history of trauma caused by wood or vegetation, and over 80% are agricultural workers who often walk barefoot. Predominantly males aged 20 to 60 years represent 90% of the cases.[2]

The immune response is mediated by neutrophils and macrophages, leading to granulomatous processes. Macrophages are unable to destroy the phagocytosed fungal cells, which then develop inside them. Depending on the dominant immune response, five clinical forms can be distinguished: nodular, tumoral lesions, verrucous, plaque-like, and cicatricial; these may range from mild forms such as small nodules to extensive, debilitating disease.[9]

Clinically, it presents as oligosymptomatic or asymptomatic lesions, which may explain why patients tend to seek medical attention only after months or even years of living with the disease. The initial lesion usually appears in exposed areas at the site of infection, as a papule with centrifugal growth that evolves into any of the clinical forms described by Carrión in 1950 (Table 1).[2]

Nodular	Fibrotic, erythematous-violaceous nodules with a smooth or hyperkeratotic surface.
Verrucous	Hyperkeratotic, dry lesions with a cauliflower-like shape and black dots.
Plaque (infiltrative or erythematous)	Erythematous or violaceous, infiltrated, well-defined plaques with irregular, sharp, and raised borders, often with black dots.
Tumoral	Isolated or coalescent lobulated lesions with a smooth or vegetative surface.
Cicatricial o atrophic	Annular, serpiginous, or irregular lesions with centrifugal growth and central atrophic areas.

Table 1: Clinical classification of chromoblastomycosis types according to Carrión (1950)

The chronicity of chromomycosis and the difficulties in treatment ultimately lead to complications and sequelae, including chronic lymphedema, secondary bacterial and parasitic infections, neoplastic transformation, ectropion, ulceration, and limb ankylosis.[10]

Due to the high polymorphism of the lesions, the differential diagnosis includes conditions such as verrucous tuberculosis, leishmaniasis, leprosy, sporotrichosis, lobomycosis, tinea corporis, secondary and tertiary syphilis, mycetoma, coccidioidomycosis, and elephantiasis. It should also be differentiated from non-infectious entities such as squamous cell carcinoma, psoriasis, or sarcoidosis.[2,7,11]

Diagnosis includes direct examination using 10–20% potassium hydroxide (KOH) of crusts or skin fragments, particularly from so-called "black dots," which represent transdermal fungal elimination and reveal muriform cells. Cultures are also performed on Sabouraud dextrose agar, with and without antibiotics, showing velvety colonies in dark brown, olive green, or black. Differentiation among fungal species is difficult and relies on reproductive structures and molecular identification.[1,2,12] Histopathology usually reveals hyperkeratosis, pseudoepitheliomatous hyperplasia, neutrophilic microabscesses containing muriform cells, and tuberculoid-type granulomas.[10,13]

It is a difficult-to-treat, refractory disease; however, various therapeutic approaches have been proposed, ranging from the use of antifungals—primarily itraconazole, voriconazole, amphotericin B, and terbinafine.[1,7,14] In the case of oral itraconazole, the recommended dose is 200 mg/day for 6 to 12 months; terbinafine is administered at 250–500 mg/day for 12 months 9. Other treatment options described include surgery, cryotherapy, electrosurgery, radiotherapy, CO2 laser, and photodynamic therapy, particularly when the lesion is small.[2,10,15]

CLINICAL CASE

A 77-year-old male patient, dedicated to agriculture, with a medical history of type II diabetes mellitus, arterial hypertension, heart failure, and renal

insufficiency, presented with a lesion on the right forearm (Figure 1) of several months' evolution. He described it as a scar that had been progressively increasing in size and causing pruritus, but without pain or any applied treatment. Physical examination revealed localized dermatosis on the right forearm characterized by an atrophic, scar-like plaque (Figure 2) with an erythematous base, infiltration, and peripheral scaling areas (Figure 3), approximately 7 cm in diameter. A skin biopsy and fungal culture were performed (Figure 4), which identified Cladosporium spp. (Figure 5). Treatment with itraconazole was initiated; however, the patient passed away due to complications from his other underlying conditions.

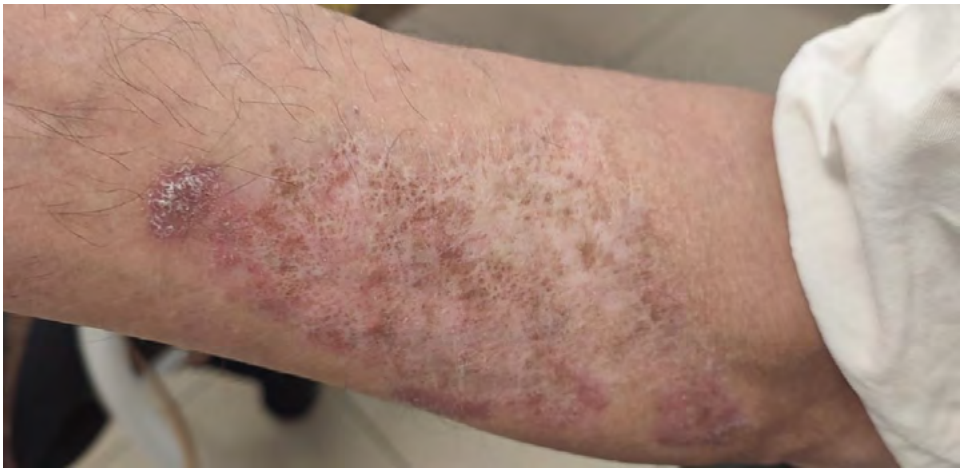

Figure 1: Lesion on right forearm.

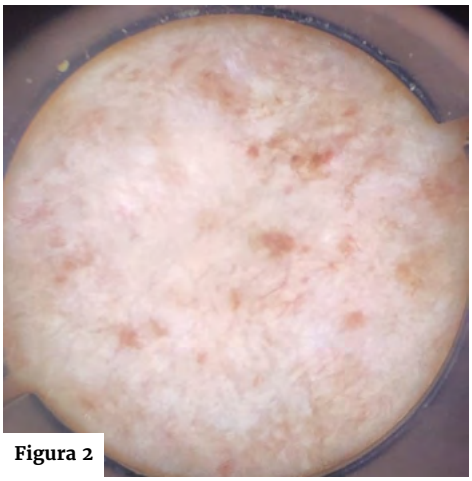

Figura 2

Figure 2: Scar-like lesion.

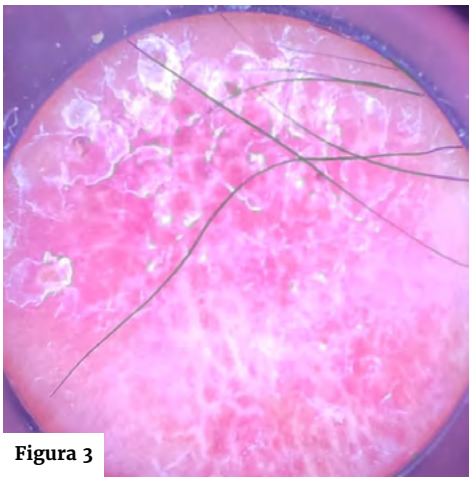

Figura 3

Figure 3: Scaling area with erythematous base.

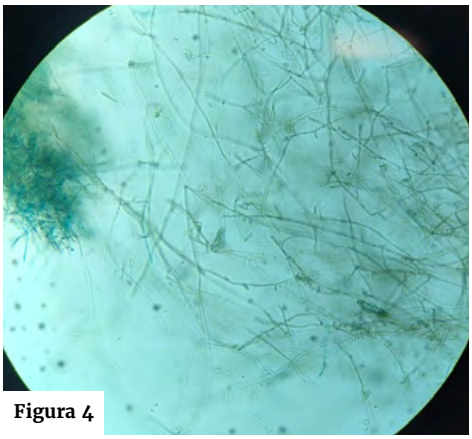

Figura 4

Figure 4: Cladosporium spp.

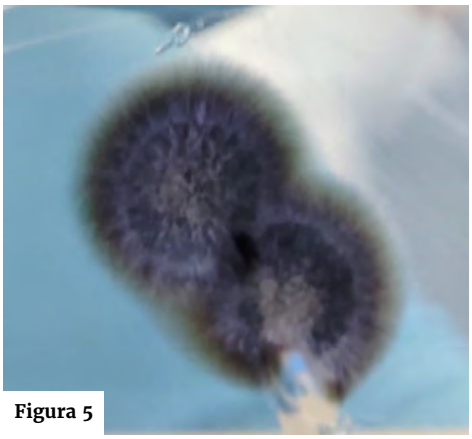

Figura 5

Figure 5: Velvety black colonies.

DISCUSSION

Chromoblastomycosis represents a medical challenge due to its chronicity, clinical variability, and resistance to treatment. It primarily affects rural and vulnerable populations, such as agricultural workers. The clinical presentation makes differential diagnosis difficult with other infectious and non-infectious diseases, such as verrucous tuberculosis, leishmaniasis, leprosy, and even squamous cell carcinoma—or, as in our case, it was mistaken for a scar.

Definitive diagnosis is based on the identification of muriform bodies through direct examination or fungal culture, although molecular techniques have proven to be valuable tools for differentiating species. However, these are not always available in endemic areas.

Treatment remains a challenge. Although antifungals such as itraconazole and terbinafine have shown efficacy, the prolonged duration and potential lack of adherence complicate clinical outcomes. Complementary methods such as surgery or cryotherapy can be helpful but are limited to small lesions and require access to specialized services.

It is crucial to promote preventive strategies and education in at-risk communities. Furthermore, research into more effective and accessible treatments is necessary to address this debilitating disease, thereby improving clinical outcomes and the quality of life of affected patients.

CONCLUSION

Chromoblastomycosis is a chronic and difficult-to-manage fungal infection, prevalent in tropical and subtropical areas, primarily affecting agricultural workers exposed to trauma from wood or vegetation. Its clinical diversity and slow progression can delay diagnosis, worsening its impact on quality of life. Although multiple therapeutic options exist, such as antifungal agents and surgical procedures, treatment is prolonged and often refractory. Prevention, through measures such as the use of footwear and adequate protective equipment, is crucial to reducing its incidence, especially in at-risk populations.

INFORMED CONSENT

The patient included in this study signed the informed consent form, approving the use of their images and clinical data exclusively for research and scientific publication purposes. It is guaranteed that no personal data was provided, nor were any photographs used that could allow their identification.

REFERENCES

1. Carrasco-Zuber JE, Navarrete-Dechent C, Bonifaz A, Fich F, Vial-Letelier V, Berroeta-Mauriziano D. Cutaneous Involvement in the Deep Mycoses: A Literature Review. Part I-Subcutaneous Mycoses. Vol. 107, Actas dermo-sifiliograficas. 2016. p. 806–15.
2. de Brito AC, Bittencourt M de JS. Chromoblastomycosis: An etiological, epidemiological, clinical, diagnostic, and treatment update. An Bras Dermatol. 2018 Jul 1;93(4):495–506.
3. López Mayra Marisela S. Diagnóstico microbiológico de la Cromoblastomicosis en Latinoamérica. UNIVERSIDAD NACIONAL DE CHIMBORAZO FACULTAD CIENCIAS DE LA SALUD CARRERA LABORATORIO CLÍNICO E HISTOPATOLÓGICO. 2022. Disponible en: http://dspace.unach.edu.ec/handle/51000/9416
4. Morand JJ. Manifestaciones cutaneomucosas de las micosis profundas y/o tropicales (excluidas las candidiasis). EMC - Dermatología. 2012 Dec;46(4):1–12.
5. Fernández Ronquillo TE, Acosta Y, Almeida R. Cromoblastomicosis en el Ecuador. Medicina (B Aires). 2019 Feb 11;19(4):246–51.
6. Marina Romero Navarrete, Roberto Arenas, Víctor Fernando Muñoz Estrada, Carlos Enrique Atoche Diéguez, Jorge Mayorga, Alexandro Bonifaz, et al. Cromoblastomicosis en México: revisión de 603 casos en siete décadas. Dermatología CMQ. 2014;12(2):87–93.
7. Badilla Ramírez Y, Madrigal Zúñiga K, Lara Campos P. Cromoblastomicosis: características clínicas y microbiológicas. Gestión en Salud y Seguridad Social [Internet]. 2023 Dec 6;2(1):e197. Available from: https://www.binasss.sa.cr/ojssalud/index.php/gestion/article/view/197

8. Badilla Ramírez Y, Madrigal Zúñiga K, Lara Campos P. Cromoblastomicosis: características clínicas y microbiológicas. Gestión en Salud y Seguridad Social [Internet]. 2023 Dec 6;2(1):e197. Available from: https://www.binasss.sa.cr/ojssalud/index.php/gestion/article/view/197
9. Clínico C, Nacional U, Gallo PR, Lambayeque P, Ventura-Flores R, Failoc-Rojas V, et al. Cromoblastomicosis: características clínicas y microbiológicas de una enfermedad desatendida [Internet]. 2017. Available from: www.sochinf.cl
10. Toro Álvareza A, Lotero Cadavid Á, Morales Posada C, García Rincón C. Cladosporium spp. como causante de cromomicosis verrucosa. Piel. 2021 Jun 1;36(6):421–4.
11. Gaviria-Giraldo CM, Cardona-Castro N. Esporotricosis y cromoblastomicosis: revisión de la literatura. Ces Medicina [Internet]. 2017;31(1):77–91. Available from: http://revistas.ces.edu.co/index.php/medicina/article/view/77
12. Chávez N, Rosero C, Nuñez M, Vicuña Y, Guevara Á, Quito DE. Cromoblastomicosis en Ecuador: reporte de caso. 2022. Available from: https://www.rcderm.org/index.php/rcderm/article/view/425/435
13. Leiro, V., Fernández Pardal, P., Coringrato, M., Piñeiro, O., Maronna, E., & Santiso, G. (2023). Cromoblastomicosis: abordaje con terapia combinada. Actualizaciones En Sida E Infectología, 31(112). https://doi.org/10.52226/revista.v31i112.16
14. Muñoz Estrada VF, Valenzuela Paz GA, Rochín Tolosa M. Cromomicosis: Reporte de un caso con topografía atípica. Rev Iberoam Micol. 2011 Jan;28(1):50–2.
15. Bassas-Vila J, Fuente MJ, Guinovart R, Ferrándiz C. Cromomicosis. Respuesta al tratamiento combinado con crioterapia y terbinafina. Vol. 105, Actas Dermo-Sifiliograficas. Ediciones Doyma, S.L.; 2014. p. 196–8.

ORCID

Jéssica Sandoval https://orcid.org/0009-0000-1975-8039
Fernanda Ron https://orcid.org/0000-0002-4752-0244
Cindy Tipán https://orcid.org/0009-0003-3120-2473
Estefany Proaño https://orcid.org/0000-0001-7522-084X