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Dermatologic Fungal Neglected Tropical Diseases—Part I. Epidemiology and Clinical Features

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Abstract

In this part 1 of a 2-part continuing medical education series, the epidemiology, clinical features, and diagnostic methods for fungal skin neglected tropical diseases (NTDs), which include eumycetoma, chromoblastomycosis, paracoccidioidomycosis, sporotrichosis, emergomycosis, talaromycosis, and lobomycosis, are reviewed. These infections, several of which are officially designated as NTDs by the World Health Organization (WHO), cause substantial morbidity and stigma worldwide and are receiving increased attention due to the potential for climate change-related geographic expansion. Domestic incidence may be increasing in the setting of global travel and immunosuppression. United States dermatologists may play a central role in early detection and initiation of appropriate treatment, leading to decreased morbidity and mortality.

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Attachments: CME proposal form, CME clinical vignette quiz section

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Keywords

neglected tropical diseases; fungal infections; endemic mycoses; implantation mycoses; systemic mycoses; eumycetoma; chromoblastomycosis; paracoccidioidomycosis; sporotrichosis; emergomycosis; talaromycosis; lobomycosis; epidemiology; diagnostics

Introduction

Key points

- Fungal skin neglected tropical diseases (NTDs) primarily affect socioeconomically disadvantaged populations globally.
- Familiarity with fungal skin NTDs is important given associations with severe morbidity.
- We provide an overview of fungal skin NTDs, focusing on epidemiology, clinical features, and diagnosis.

Fungal skin NTDs are a diverse group of environmental fungal infections (Table 1) which cause substantial disability and receive little public health attention. Consequently, the World Health Organization (WHO) designated mycetoma, chromoblastomycosis, and “other deep mycoses” as NTDs.¹ NTDs disproportionately affect low-income countries, but due to travel to endemic regions and immunosuppression, NTDs are also reported in high income countries. NTD geographic expansion may occur with climate change, globalization, and increased host susceptibility with use of immunosuppressive therapies.^{2–5} The majority of fungal skin NTDs are not reportable diseases,^{6–12} resulting in a large unknown disease burden. Diagnosis is challenging, as clinical presentation frequently resembles other infectious and non-infectious conditions (Table 2). In the United States (US), dermatologists have limited fungal skin NTD patient encounters, precluding clinical pattern recognition and affecting long term outcomes.¹³ This review addresses this practice gap by outlining epidemiology, clinical features, and diagnosis of fungal skin NTDs to improve patient care by dermatologists.

In addition to NTDs recognized by the WHO, this review also includes mycoses on the WHO NTD 2021–2030 roadmap, mycoses recognized as NTDs by the *Public Library of Science Neglected Tropical Diseases*, and mycoses for which there are global calls for NTD recognition.

Eumycetoma

Key points

- Eumycetoma occurs most commonly in the tropical and subtropical mycetoma belt, but cases have been reported worldwide.
- Immunocompetent men in their 30s–40s in the mycetoma belt without sufficient foot coverage are most often affected.

- Physical examination is commonly notable for subcutaneous swelling, multiple discharging sinuses, and presence of macroscopic granules.

Overview. Mycetoma is caused by fungi (eumycetoma), bacteria (actinomycetoma), or combinations. Eumycetoma is most frequently caused by *Madurella mycetomatis*,¹⁴ which is present in soil and gains cutaneous entry after minor trauma, which has led to the term “Madura foot.” However, metagenomic studies demonstrate diversity of implicated pathogens.¹⁵

Epidemiology. Populations of tropical/subtropical regions in the mycetoma belt (latitudes 15° S -30° N) are primarily affected (Figure 1).¹⁶ Sudan has the largest reported mycetoma burden;¹⁷ other endemic regions include Senegal,¹⁸ India,¹⁹ Mexico,²⁰ Brazil, and Argentina.²¹ Men in their 30s-40s are most frequently affected, likely related to agriculture occupations.²² Risk factors include walking barefoot or wearing footwear without sufficient coverage.

Epidemiologic trends. In an epidemiologic study (2020),²³ eumycetoma and actinomycetoma were reported beyond tropical endemic areas of the mycetoma belt, to the US (57 cases), Italy (8), China (9), and Australia (5). There are eumycetoma cases in Mexican patients who migrate to the US.^{24–26} In a US epidemiologic study¹³ of implantation mycoses (2017–2021), there were 238 eumycetoma cases (5.2 per 1,000,000), with highest prevalence in the Midwest and more commonly among immunosuppressed patients. There was a female predominance for eumycetoma in the US in contrast to international settings, possibly due to different care-seeking behaviors.²¹

Clinical features. The classic triad of disease includes slow, progressive subcutaneous swelling, multiple discharging sinuses, and macroscopic granules.²⁷ Lower extremities are most frequently affected, with painless plaques demonstrating woody swelling and communicating sinuses, with the latter draining purulent material and granular grains (black-fungal, red-bacterial, white/yellow-fungal or bacterial) (Figure 2).²⁸ Clinical variants include nodules without sinuses, cysts,²⁹ and verrucous plaques.^{30,31} If left untreated, infection may spread via fascial planes to involve muscle or bone.^{28,32}

Diagnosis and evaluation. Ultrasound may distinguish eumycetoma from other subcutaneous masses, and between eumycetoma (black grains produce sharp hyperechoic foci) and actinomycetoma (grains less distinct and seen at bottom of lesions).^{33,34} Sensitivity and specificity for ultrasound examination of eumycetoma were 94.3% and 50%, respectively, in a cross-sectional study³⁵ of 222 patients suspected of fungal mycetoma caused by *Madurella mycetomatis*. Sinus material may be obtained via ultrasound-guided fine needle aspiration for histopathologic examination, which is required for definitive diagnosis (Figure 3).³⁶ Tissue fungal culture identifies the causative organism. Magnetic resonance imaging (MRI) may assess for extent of soft tissue involvement.^{37,38} The “dot-in-circle” sign on MRI refers to small, round-shaped hyperintense lesions with small central focus correlating to granulomata-containing grains (Figure 4).³⁹ X-ray and computed tomography (CT) may assess for bone involvement.

Chromoblastomycosis

Key points

- Chromoblastomycosis most commonly occurs in tropical and subtropical regions.
- Immunocompetent male agricultural workers are most frequently affected, but anyone may be affected.
- Physical examination is notable for diverse clinical findings, including macules, papules, plaques, nodules, and verruciform or scar-like lesions. Cayenne pepper black dots may be present.

Overview. Chromoblastomycosis is a chronic granulomatous infection most frequently caused by *Fonsecaea* and *Cladophialophora* spp. and acquired through traumatic inoculation.

Epidemiology. Chromoblastomycosis most commonly occurs in tropical and subtropical regions, and most frequently affects agricultural workers, though not limited to this population (Figure 5). In a review⁷ of 7,740 chromoblastomycosis cases (1914–2020), cases were most common in South America, Africa, Central America and Mexico, and Asia. Men (81.7%) were frequently affected with mean age 57.1 years.

Epidemiologic trends. In a US epidemiologic study¹³ of implantation mycoses (2017–2021), there were 667 chromoblastomycosis and phaeohyphomycotic abscess cases (14.7 per 1,000,000), with highest prevalence in the Northeast and more commonly among immunosuppressed patients.

Clinical features. Chromoblastomycosis most frequently affects lower extremities, followed by upper extremities, and head/neck. In early disease stages, chromoblastomycosis may present as a small verrucous papule following trauma (Figure 6). Lesions enlarge over time, forming hypertrophic and verrucous plaques, sometimes associated with pruritus and pain. Black dots with “cayenne pepper” appearance may be present on the surface (Figure 7).⁴⁰ In the aforementioned study of 7,740 chromoblastomycosis patients, the most common morphologies were verruciform, tumorous, and plaque-like.⁷ Only one lesion type was present in 63.2% of patients, while combinations were observed in 36.8%. In the US, chromoblastomycosis typically presents as small to moderate sized plaques with heavy scale-crust, unlike the tumoral masses and large verrucous plaques more typical of long-neglected lesions in endemic regions (Figure 8).⁴¹ Chromoblastomycosis lesions may clinically and histopathologically mimic squamous cell carcinoma.⁴²

Diagnosis and evaluation. Diagnosis is confirmed through identification of sclerotic bodies (thick-walled cells [4–12µm in diameter] resembling copper pennies), also known as muriform cells or medlar bodies, seen on potassium hydroxide (KOH) preparation with microscopy or skin biopsy with histopathology (Figures 9, 10).⁴³ Skin biopsy is preferentially performed in an affected area with visible black dots.⁴⁴ Skin scraping is typically sufficient due to the robust nature of medlar bodies.⁴⁵ Skin scrapings may be used for early and long-standing lesions.⁴⁵ Tissue fungal culture identifies the causative

organism.⁴⁴ In a retrospective review of chromoblastomycosis cases (1989–2018) in Brazil, average time to diagnosis was nine years.⁴⁶

Paracoccidioidomycosis

Key points

- Paracoccidioidomycosis is endemic in tropical and subtropical regions of Latin America.
- Male agricultural workers in their 30s–50s are primarily affected.
- Dermatological examination is notable for mucosal and cutaneous ulcers with hemorrhagic spots, often accompanied by severe local adenopathy.

Overview. Paracoccidioidomycosis is caused by dimorphic fungi of the genus *Paracoccidioides*, which are soil saprophytes.⁴⁷ Infection occurs via inhalation of conidia, with hematogenous spread to other organs, including the skin.⁴⁸ Direct cutaneous or mucosal inoculation is rare.^{49,50} Paracoccidioidomycosis is classified into acute-subacute (juvenile) and chronic (adult) forms. Most cases (74–96%) are chronic,⁵¹ and are classified into unifocal form (single organ involvement—typically lungs), and multiple form, (multiple organ involvement—typically skin, mucosa, and lungs).⁴⁹

Epidemiology. Paracoccidioidomycosis is endemic to tropical and subtropical regions of South and Central America (Figure 5). Infection is associated with exposure to rural environments and agricultural activity, with higher risk in people working in coffee, sugar cane, corn, and tobacco plantations.^{52,53} Men in their 30s–50s are most frequently affected in the chronic form,⁵¹ and 90% are smokers, with disease risk 14 times higher among smokers vs. non-smokers.^{54,55} Adolescents of both sexes are most frequently affected in the acute-subacute form.⁵¹

Epidemiologic trends. Brazil has the highest proportion of cases (80%),⁵⁶ with incidence rising in the northern region (Amazon) due to new settlements and concurrent deforestation.^{51,57–59} Urban cases of paracoccidioidomycosis may be increasing.⁶⁰

Clinical features of chronic form. The lungs are most frequently involved (90%), followed by upper respiratory tract mucosa and skin.⁵¹ Mucosal lesions are painful, erythematous ulcers with ragged borders and hemorrhagic spots.⁶¹ In a retrospective study of 161 paracoccidioidomycosis patients, 60.2% presented with oral lesions, most commonly mulberry-like ulcers and gingival hemorrhagic spots (Figure 11),⁶² and 27.8% presented with skin involvement. Cutaneous lesions usually present as erythematous papules or ulcers/vegetations with fine granulations and hemorrhagic spots, most commonly involving the face, extremities, and trunk (Figure 12).⁶³ There is a rare sarcoidosis-like form (Figure 13).⁶⁴

Clinical features of acute-subacute form. Most symptoms involve the phagocytic-mononuclear system and frequently include generalized lymphadenopathy and hepatosplenomegaly.⁵¹ Cutaneous lesions are present in 16.3% of cases.⁶⁵

Diagnosis and evaluation. Direct microscopy with KOH of tissue samples or skin scrapings demonstrates large yeasts (10–30µm) with a thick, birefringent cell wall with multiple buds resembling a “mariner’s wheel” or “Mickey Mouse Ears.”^{8,66,67} Histopathologic examination with Gomori’s methenamine silver (GMS) stain aids in organism identification (Figure 14). Tissue fungal culture identifies the causative organism. Serologic testing is used to monitor response to therapy.⁶⁶ Chest CT is recommended to evaluate for pulmonary involvement.⁴⁷

Sporotrichosis

Key points

- Sporotrichosis is thought to be the most prevalent implantation mycosis worldwide.
- Patients in contact with vegetation, soil, and cats are most frequently affected.
- Physical examination classically shows ascending nodular lymphangitis.

Overview. Sporotrichosis is caused by the dimorphic fungal genus *Sporothrix* spp. The most common species vary by region, including *Sporothrix schenckii* (North America, Australia, South Africa),⁶⁸ *Sporothrix globosa* (Asia),^{68,69} and *Sporothrix brasiliensis* (Brazil).^{70,71} Transmission varies by species, with the classical route defined as traumatic inoculation of soil or plant material for *Sporothrix schenckii*, and the emerging route defined as inoculation through a bite, scratch or contact with respiratory droplets (sneezing, licking) from infected cat for *Sporothrix brasiliensis*.⁷² In feline sporotrichosis, transmission to humans may occur via contact with infected animal secretions without trauma.

Epidemiology. Sporotrichosis has a global distribution, with endemic areas being Latin America (especially Brazil, Mexico, Colombia), Asia (especially China, India, Japan), and to a lesser extent, US and Australia (Figure 5).⁷³ It is often considered the most prevalent and widespread implantation mycosis worldwide.⁷⁴ Veterinarians and those who work with animals may have increased infection risk.^{72,75}

Epidemiologic trends. An ongoing sporotrichosis epidemic in Rio de Janeiro, Brazil, primarily from feline transmission of *Sporothrix brasiliensis*, caused an increase in human cases from 759 in 1998–2004 to >4,000 in 2014.⁷⁴ In a case series of 122 sporotrichosis patients infected with *Sporothrix brasiliensis*, 70.5% were female, which may be related to the high proportion of women working as housewives or cleaners in Brazil with close contact to domesticated cats.⁷⁶

Clinical features. The cutaneous form is most common, including lymphocutaneous (55%) and fixed cutaneous (25%) involvement (Figures 15, 16).⁷⁷ In the lymphocutaneous form, a papulonodular lesion develops days to weeks after inoculation and subsequently ulcerates, often resembling the initial lesion of syphilis and therefore referred to as chancreiform (Figure 17). Similar lesions thereafter occur along regional lymphatic channels proximal to the original lesion (nodular lymphangitis). In the cutaneous form, polymorphic skin lesions, including papules, nodules, ulcers, verrucous lesions, and plaques develop

following trauma.⁷⁴ Extracutaneous involvement is associated with chronic obstructive pulmonary disease, alcoholism, steroids, AIDS, solid organ transplantation, and TNF-alpha inhibitors, and may include pulmonary, osteoarticular, ophthalmic, and central nervous system infection.^{74,78}

Diagnosis and evaluation. Tissue fungal culture is the primary diagnostic test.^{79–83} Histopathology has low sensitivity, showing only an inflammatory or granulomatous reaction, and rarely oval or spherical yeasts (2–6µm in diameter) sometimes elongated in a cigar shape (Figure 18).⁷⁹ Because organisms may be sparse, immunohistochemistry may enhance sensitivity. Polymerase chain reaction (PCR) is a fast, accurate, and highly sensitive diagnostic method.⁸⁴ However, immunohistochemistry and PCR are not readily available for sporotrichosis diagnosis in the US.

Emergomycosis

Key points

- Emergomycosis is a recently recognized dimorphic fungal infection with known distribution in North America, Africa, Asia, and Europe.
- Immunosuppressed patients are most frequently affected.
- Physical examination shows widespread polymorphic cutaneous lesions.

Overview. Emergomycosis is caused by a group of previously unknown dimorphic fungal species that have recently been reclassified to the genus *Emergomyces*. *Emergomyces* spp. are found in soil and are likely spread by inhalation of conidia.⁸⁵ It is unknown whether *Emergomyces* spp. have truly emerged or whether they are now recognized due to increased immunosuppressed, susceptible hosts, and the adoption of molecular identification techniques.⁸⁶

Epidemiology. Emergomycosis cases have been reported in North America, Africa, Asia, and Europe.⁸⁷ The most cosmopolitan and widespread species is *Es. pasteurianus*,⁸⁸ diagnosed in the Netherlands,⁸⁹ Italy,⁹⁰ Spain,⁹¹ France,⁹² India,⁹³ China,^{94,95} Uganda,⁹⁶ and South Africa.⁹⁷ Other species include *Es. canadensis* (North America), *Es. orientalis* (Asia), *Es. europaeus* (Europe) and *Es. africanus* (Southern Africa).^{85,87,98,99} Emergomycosis most commonly affects immunosuppressed people, including those with advanced HIV, solid organ transplantation, diabetes mellitus, hematologic malignancy, and those taking immunosuppressants.^{88,100,101,102} The highest burden of cases is among HIV-infected patients in South Africa.¹⁰³

Epidemiologic trends. Emergomycosis is now recognized as the most commonly diagnosed dimorphic fungal infection in South Africa.⁸⁶ Recent cases of emergomycosis have been reported in North America, including four cases in immunocompromised patients in the US (Colorado, New Mexico) and Canada (Saskatchewan).¹⁰⁴

Clinical Features. Emergomycosis may involve the skin, mucous membranes, lungs, gastrointestinal tract, liver, spleen, bone marrow, lymph nodes, and cervix.^{85,86,88,105–107} In

a retrospective study of 55 emergomycosis patients in South Africa, 96% had skin lesions at presentation, and 88% had pulmonary involvement.¹⁰⁸ Cutaneous lesions are typically widespread and polymorphic,¹⁰⁵ and may include umbilicated papules, hyperkeratotic plaques, nodules, verrucous lesions, and ulcerations (Figures 19, Figure 20).^{85,105,106,108,109}

Diagnosis and evaluation. Diagnosis is via tissue fungal culture in conjunction with histologic evidence of tissue invasion.¹⁰⁰ Punch or incisional skin biopsies may be used for tissue culture and histopathology.¹⁰⁰ Histopathologic sections demonstrate 2–5µm intracellular (in histiocytes) and extracellular oval yeasts with narrow-based budding and inflammatory change, best visualized with hematoxylin-eosin, periodic acid Schiff (PAS), GMS, and Wright-Giemsa stains (Figure 21).^{85,88,101} Histopathology alone is not sufficient for diagnosis, as yeast cells of *Es. africanus* and *Es. orientalis* resemble those of *Histoplasma capsulatum* and *Blastomyces dermatitidis*, respectively.^{85,88,100}

Talaromycosis

Key points

- Talaromycosis is endemic to tropical and subtropical regions of Asia.
- Patients living with HIV are most frequently affected.
- Physical examination findings are papules with central umbilication involving the face, neck, and extremities.

Overview. Talaromycosis is caused by the fungus *Talaromyces marneffe*, which is spread via inhalation of spores.¹¹⁰

Epidemiology. Talaromycosis is endemic to tropical and subtropical regions of Asia and primarily affects HIV patients.¹¹¹ Patients with other immunosuppressive conditions, including cancer and organ transplantation, may also be affected.¹¹² A total of 24,622 talaromycosis cases were reported in 33 countries by late 2018, with an estimated 288,000 talaromycosis cases globally 1964–2018, and approximately 17,300 cases diagnosed annually.¹¹³

Epidemiologic trends. It is estimated that talaromycosis incidence will increase by 35% 2020–2025,¹¹³ due to rising incidence of advanced HIV among newly diagnosed HIV patients in Indonesia, Thailand, Vietnam, and the Philippines.¹¹¹

Clinical features. The main clinical manifestations are painless papules involving the face, neck, and extremities.^{114–117} In a retrospective study of 80 HIV patients with disseminated talaromycosis, 71% had cutaneous findings, and 87% of papules had central umbilication (Figure 22). Non-HIV patients less often had skin lesions (44% vs. 71%),^{118,119} and had longer time to diagnosis (180 vs. 45 days)¹²⁰ and higher mortality (29% vs. 21%)¹²¹ compared to HIV-coinfected patients. Papules less often have central umbilication in non-HIV-coinfected vs. HIV-coinfected patients.¹²²

Diagnosis and evaluation. Tissue fungal culture is the primary diagnostic test.¹²³ Presumptive diagnosis may be made via microscopy of Wright-stained touch smears from a skin biopsy, showing clear basophilic, oval yeasts with central septation.^{124–126} Histopathology with GMS or PAS staining demonstrate round or oval yeast cells within macrophages (Figure 23),¹²⁶ but may resemble that of other fungal diseases associated with parasitized histiocytes, including emergomycosis and histoplasmosis. Antigen detection is an accurate and inexpensive diagnostic option for advanced HIV patients with high fungal burden.¹⁰⁰ An inexpensive, point-of-care immunochromatographic strip test with high sensitivity/specificity using clinical urine specimens was recently developed, which is not yet readily available in the US.¹²⁷

Lobomycosis

Key points

- Lobomycosis primarily occurs in tropical regions of Latin America.
- Individuals with trauma in the setting of environmental exposure are most frequently affected.
- Physical examination is notable for keloid-like nodules involving the lower extremities or ears.

Overview. Lobomycosis, also known as lacaziosis or Jorge Lobo's disease, is caused by the fungus *Lacazia loboi*, which presumably gains cutaneous entry after minor trauma.¹²⁸ This pathogen is non-cultivable, and taxonomy is therefore debated.^{129,130} Dolphins are the only other known host. Recent increase in dolphin varieties infected with *Lacazia loboi*¹³¹ raises concern for increased geographical distribution of disease in humans.

Epidemiology. Lobomycosis primarily occurs in Central and South America, and most commonly affects forest workers, farmers, hunters, manual laborers, and fishermen. There were 907 reported cases globally as of 2022, which is likely an underestimate.¹³⁰

Epidemiologic trends. Incidence has increased in Acre, Brazil.^{132,133} Cases have been reported in the US and Canada, likely caused by preceding exposure in the Amazon Basin.^{134,135}

Clinical features. Lobomycosis is characterized by long incubation period (sometimes years) followed by slow, progressive development of painless keloid-like nodules, which may ulcerate or become verrucous (Figure 24).¹⁰ In the localized form, lower extremities are most frequently affected, followed by ears, upper extremities, and head (Figure 25).^{136,137} In a retrospective study of 249 lobomycosis cases in Brazil, localized, multifocal, and disseminated cutaneous lesions were observed in 61%, 22%, and 16% of patients, respectively.¹³⁸

Diagnosis and evaluation. Diagnosis is made via skin biopsy with histopathology,¹³⁹ showing isolated round yeast-like cells with a thick wall containing melanin, or in 2–10 cell

chains connected via tubular projections (Figure 26).¹³⁹ In scaly lesions, fungal visualization using direct microscopy on lesional scrapings is useful.^{10,140}

Conclusion. Fungal skin NTDs have been and continue to be neglected, despite association with substantial morbidity and stigma. Public health efforts are needed to improve surveillance and to increase access to diagnostic tools in endemic regions, since diagnosis is challenging. A high index of suspicion for fungal NTDs based on demographics and clinical presentation is necessary, with adequate tissue biopsy for histological examination and fungal culture. Recognition of cutaneous manifestations and prompt diagnosis is imperative for improved outcomes.

Abbreviations

AIDS	Acquired immunodeficiency syndrome
CT	Computed tomography
HIV	Human immunodeficiency virus
GMS	Gomori's methenamine silver
KOH	Potassium hydroxide
MRI	Magnetic resonance imaging
NTD	Neglected tropical disease
PAS	Periodic acid Schiff
PCR	Polymerase chain reaction
TNF	Tumor necrosis factor
US	United States
WHO	World Health Organization

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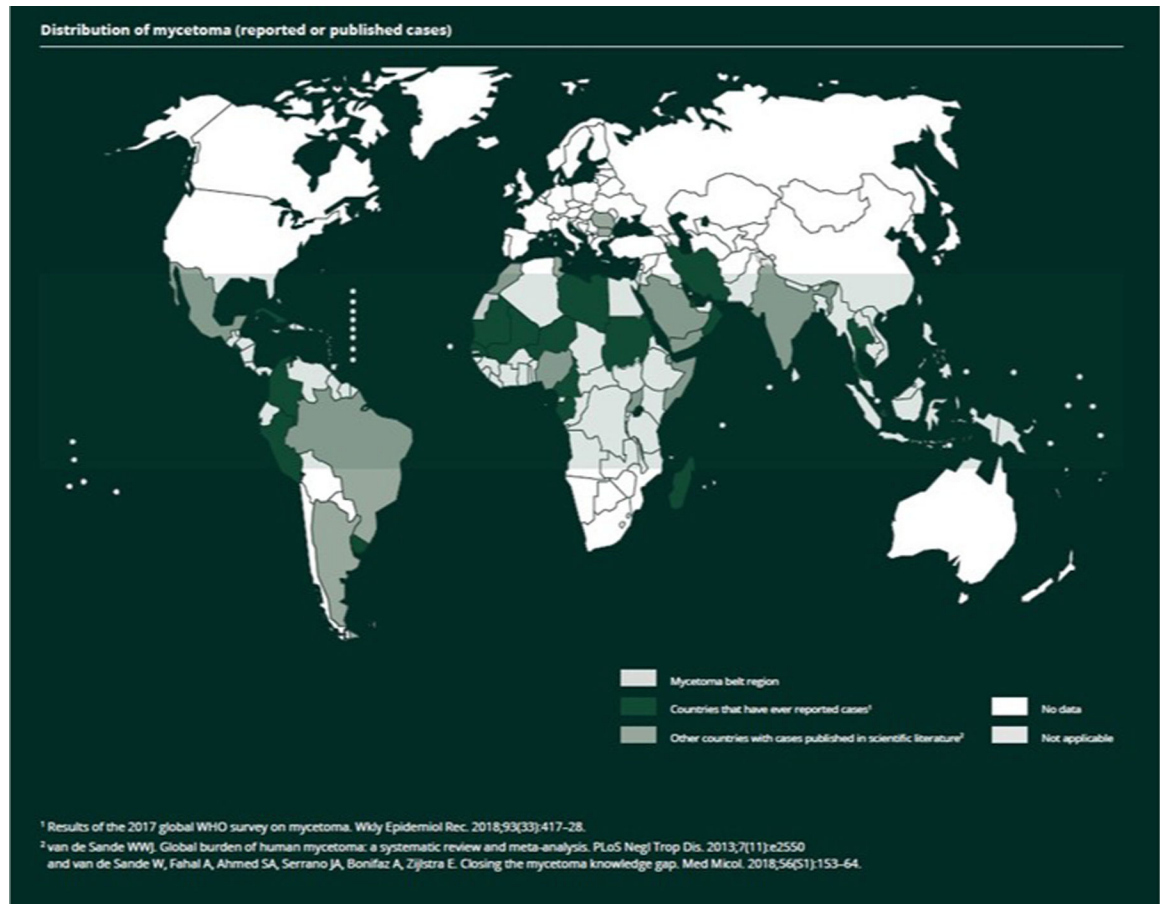
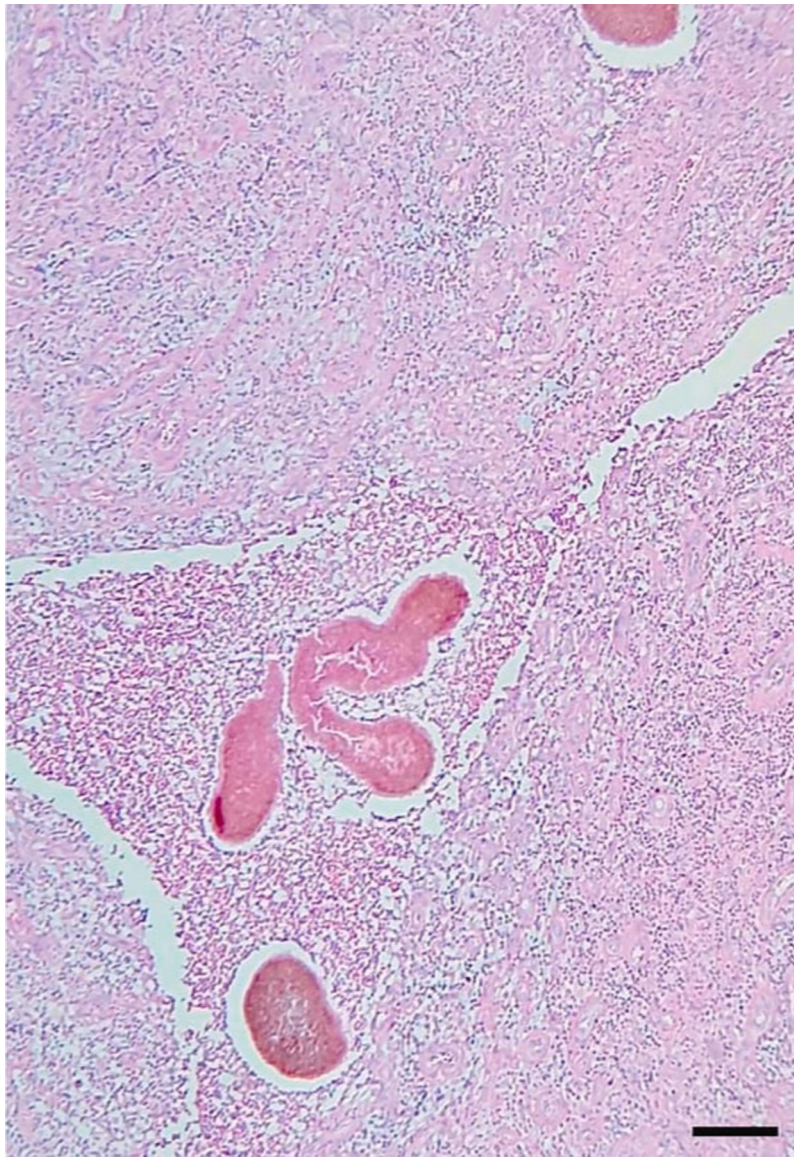
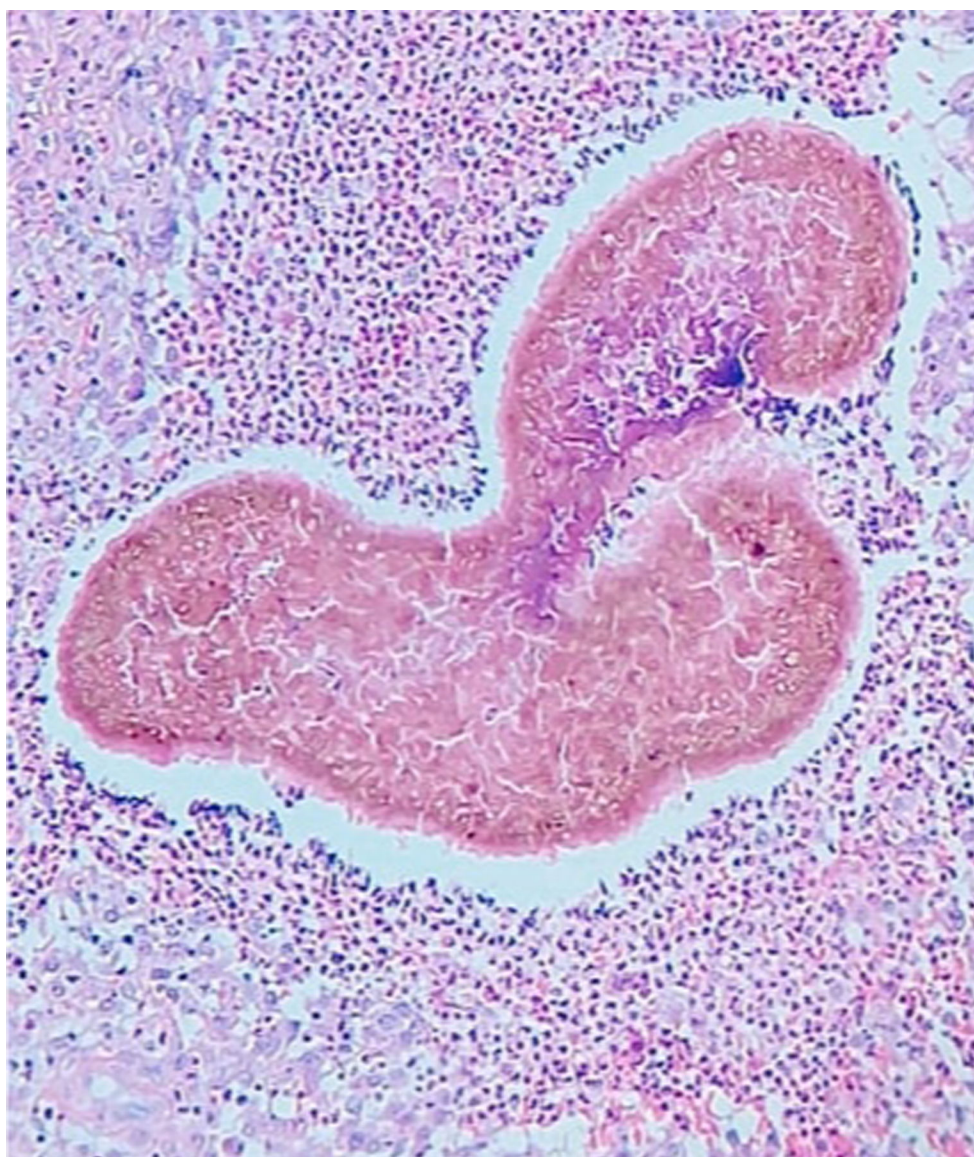


Figure 1.
Distribution of mycetoma (reported or published cases). Reproduced from the World Health Organization (published under Creative Commons CC BY-NC-SA 4.0 DEED license).



Figure 2.
A massive eumycetoma lesion of the plantar foot with multiple discharging sinuses and black grains, Mexico.





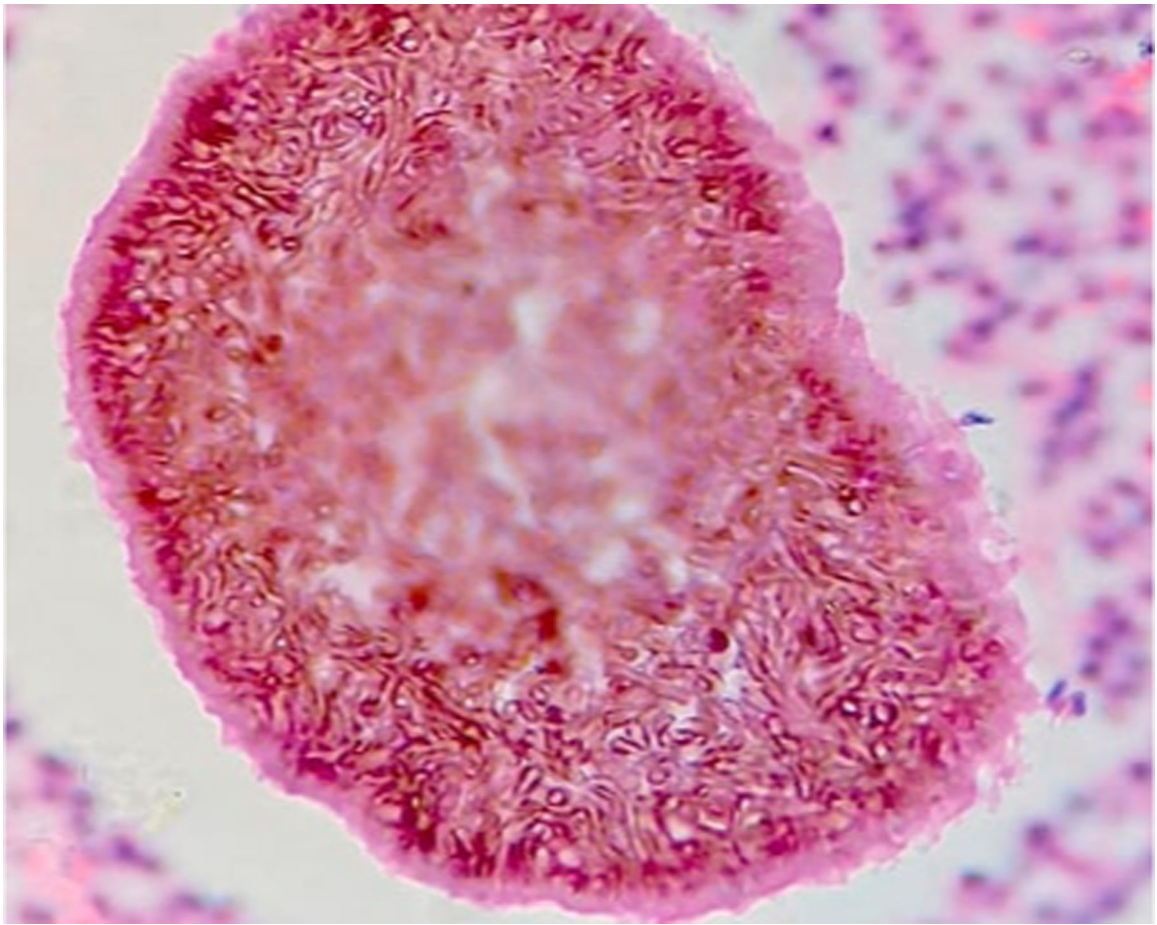


Figure 3.

Eumycetoma histopathologic examination (skin biopsy) demonstrating A. suppurative granulomatous inflammatory infiltrates with neutrophils, lymphocytes, and histiocytes; B. large multilobed grains with Splendore-Hoeppli phenomenon; C. clusters of radially branched brown hyphae (Hematoxylin and eosin stain, 100x). Reproduced from Arteaga et al.¹⁴¹ (published under Creative Commons CC-BY-NC-ND license).

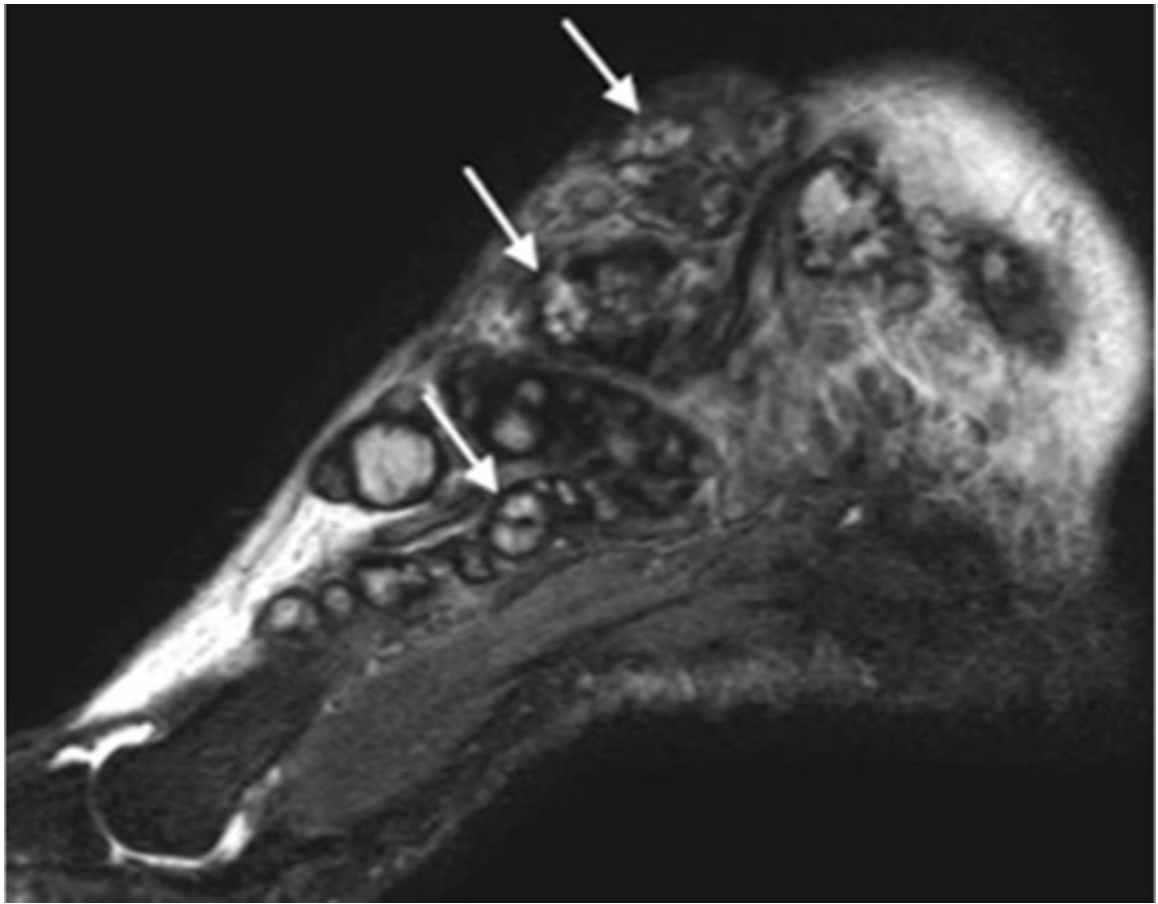


Figure 4. Sagittal MRI show multiple, small, round-to-spherical hyperintense mycetoma lesions separated by peripheral hypointense tissue. Some of the lesions contain a central hypointense dot, resulting in the 'dot-in-circle' sign (arrows). Reproduced from Laohawiriyakamol et al.³⁹ (published under Creative Commons CC-BY license).

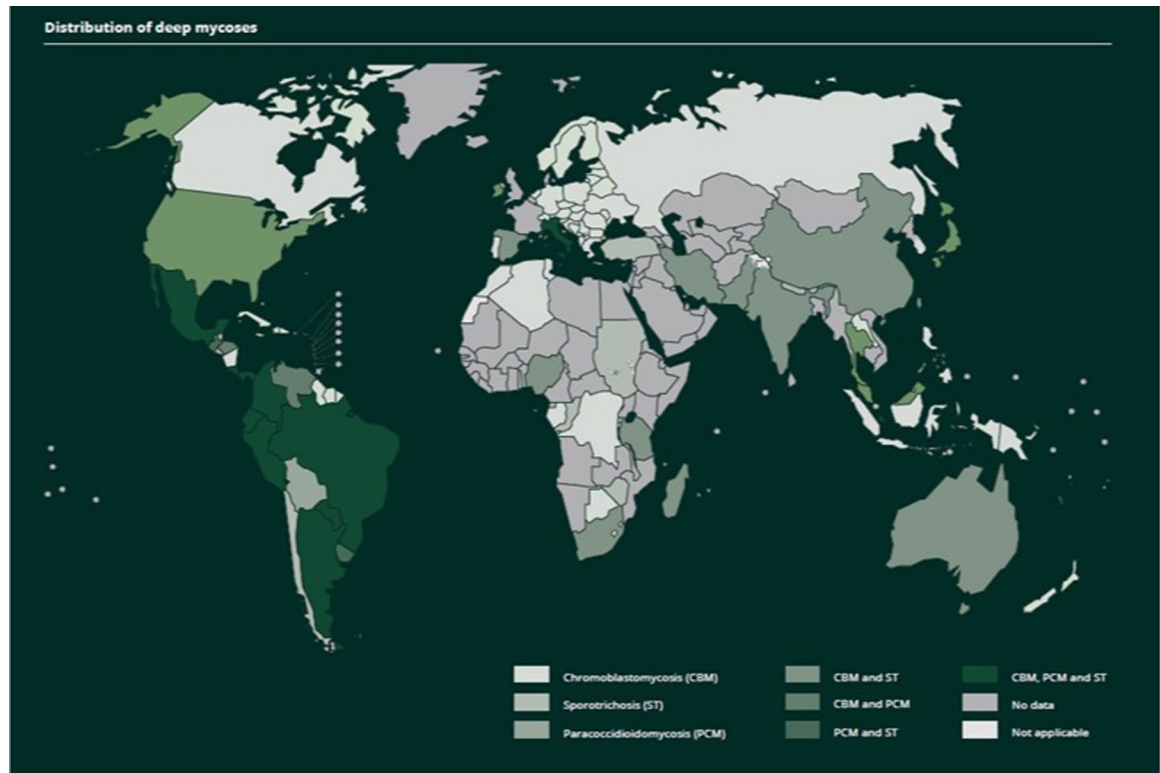


Figure 5. Distribution of deep mycoses. Reproduced from the World Health Organization (published under Creative Commons CC BY-NC-SA 4.0 DEED license).



Figure 6.
Small, scale-crust covered lesion on the forearm found to be chromoblastomycosis;
submitted for biopsy as Bowen's disease. Patient from Houston, Texas.



Figure 7. Chromoblastomycosis, hyperkeratotic plaque, with warty dry lesions, with moderate severity disease localized to the distal third of the leg. Lesions with small black dots (cayenne pepper appearance).



Figure 8. Tumoral mass clinically diagnosed as verrucous squamous cell carcinoma and diagnosed with biopsy as chromoblastomycosis. Patient from Panama.



Figure 9. Chromoblastomycosis, clustered muriform cells depicted as round to polyhedral (chestnut-like) cells, visualized on direct microscopy potassium hydroxide exam.

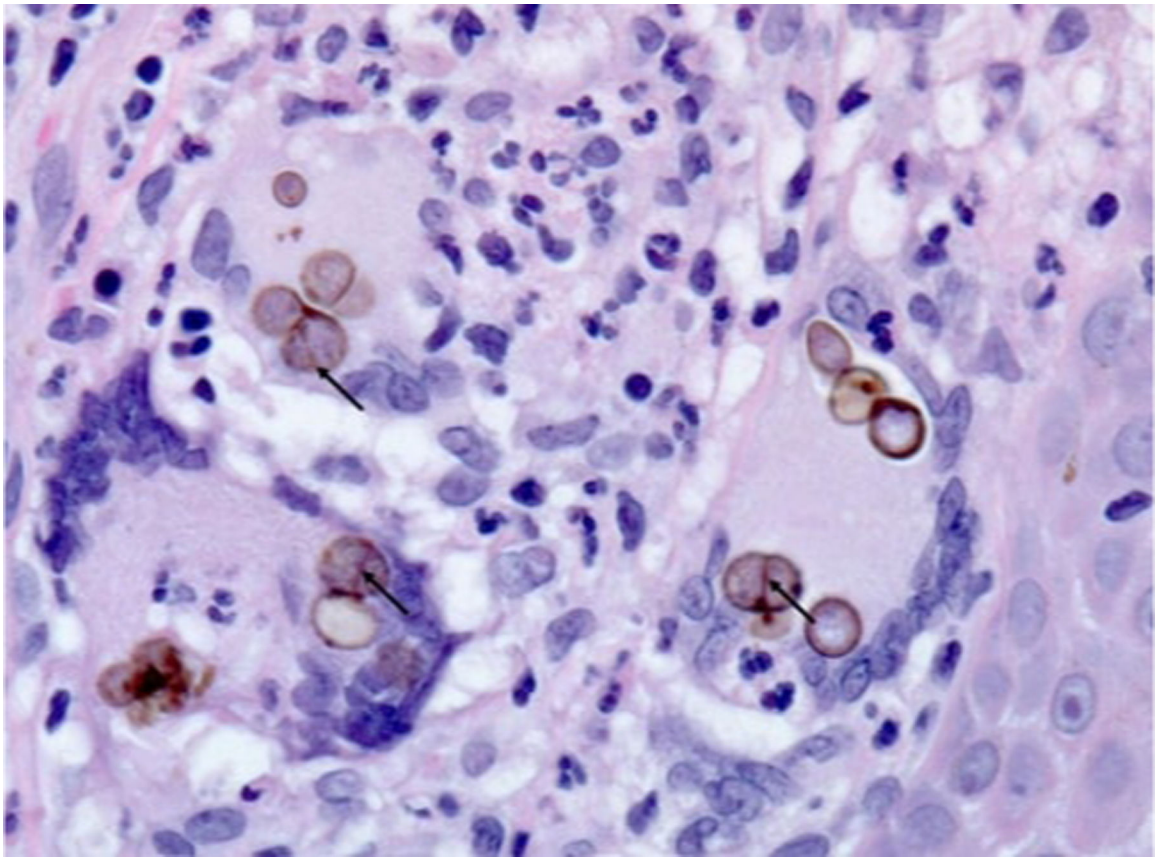


Figure 10.

Chromoblastomycosis, skin, H&E stain: Clusters of round, thick-walled, brown-pigmented fungal spores (“sclerotic bodies,” “Medlar bodies” or “copper bodies”) within multinucleated giant cells in a focus of subepidermal granulomatous inflammation. Sclerotic bodies are fungal spores that are usually 5–12 microns and divide by internal septation (arrows). Original magnification: x630.



Figure 11.

Oral lesions of paracoccidioidomycosis in the gingiva and mulberry-like ulcers with hemorrhagic dots. Photograph from a patient treated at University Hospital Cassiano Antonio Moraes, Federal University of Espirito Santo. Reproduced from Dutra et al.¹⁴² (published under Creative Commons CC-BY-NC-ND license).



Figure 12. Paracoccidioidomycosis and liver transplantation. Acneiform lesions and erythematous papules, some with an ulcerated center, disseminated on the face. Reproduced from Valentim et al.¹⁴³ (published under Creative Commons CC-BY license).



Figure 13. Paracoccidioidomycosis, infiltrated, sarcoid-like cutaneous lesions in the right malar region. Reproduced from Cárcano et al.⁶⁴ (published under Creative Commons CC-BY-NC-ND license).

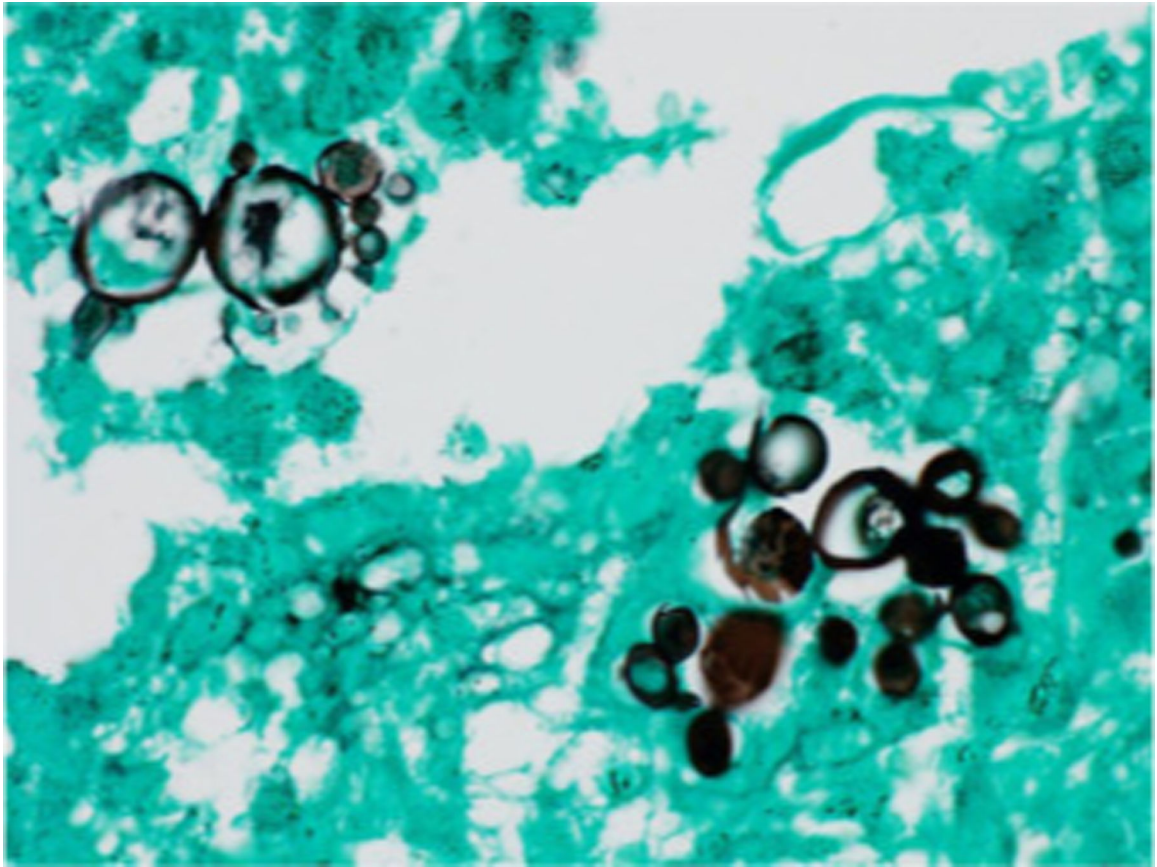


Figure 14. Paracoccidioidomycosis and liver transplantation, histopathologic examination (skin biopsy) demonstrating multi-budding fungal cells, “ship’s wheel” finding pathognomonic for disease (Grocott-Gomori, Immersion). Reproduced from Valentim et al.¹⁴³ (published under Creative Commons CC-BY license).



Figure 15.
Sporotrichosis, feline dissemination. Erythematous lesions, with subcutaneous nodules, painful on palpation, with lymphatic distribution involving the right arm.



Figure 16.
Sporotrichosis fixed cutaneous form, verrucous lesion on the dorsum of the hand.
Reproduced from Orofino-Costa et al.¹⁴⁴ (published under Creative Commons CC-BY license).



Figure 17.
Primary chancriform lesion of sporotrichosis after trauma with rose thorn.

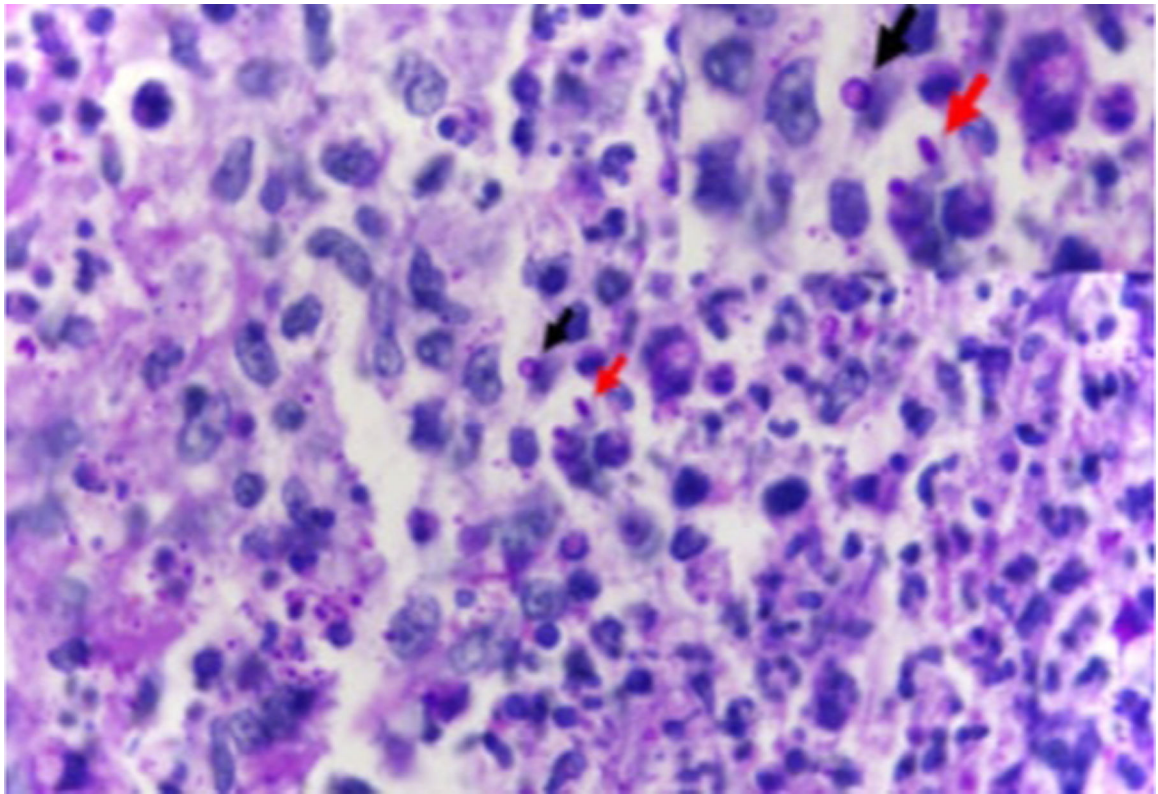


Figure 18.

Sporotrichosis histopathologic examination (skin) demonstrating suppurative granuloma and parasitic fungus. Epithelioid cells on the left, neutrophils and myocytes on the right, round yeast-like (black arrow) and elongated or navicular fungal cells (red arrow) (Periodic acid-Schiff stain, 1000x). Reproduced from Orofino-Costa et al.¹⁴⁴ (published under Creative Commons CC-BY license).



Figure 19.

Mucocutaneous manifestations of HIV-associated disseminated cryptococcosis in a South African patient who presented with a 1-year history of nodular lesions on the face, limbs, and trunk. Papules and plaques with central necrosis. Reproduced from Reddy et al.¹⁴⁵ with permission. Figure courtesy of Dr. Matilda Mphahlele (Division of Dermatology, Chris Hani Baragwanath Academic Hospital, Faculty of Health Sciences, University of the Witwatersrand, Johannesburg, South Africa).



Figure 20. Clinical image of a South African patient with HIV-associated disseminated emergomycosis, showing hyperpigmented and violaceous plaques with surface scaling. Reproduced from Reddy et al.¹⁴⁵ with permission.

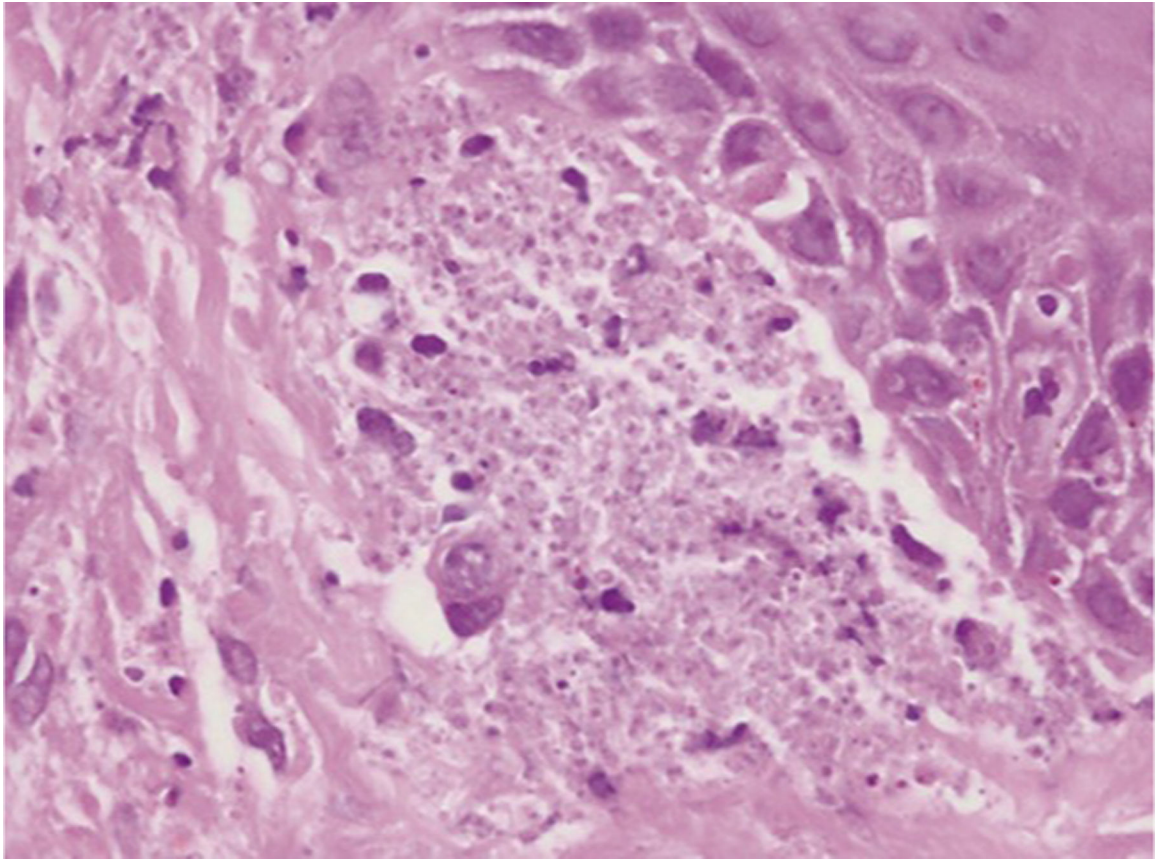


Figure 21.

Histopathology of skin biopsies from South African patients with HIV-associated disseminated emeryomyces caused by *Es. Africanus*. *Es. africanus* organisms occupying the papillary dermis, as seen under oil emersion (haematoxylin and eosin, original magnification x 1,000). Reproduced from Govender et al.¹⁰⁶ with permission.



Figure 22.

Talaromycosis physical examination. Multiple cuticolor follicular papules with or without central umbilication, nodules, and acne-like lesions were developed densely on his face, neck, and upper anterior chest. Some of them were covered with reddish brown crusts. Reproduced from Xian et al.¹⁴⁶ (published under Creative Commons CC-BY license).

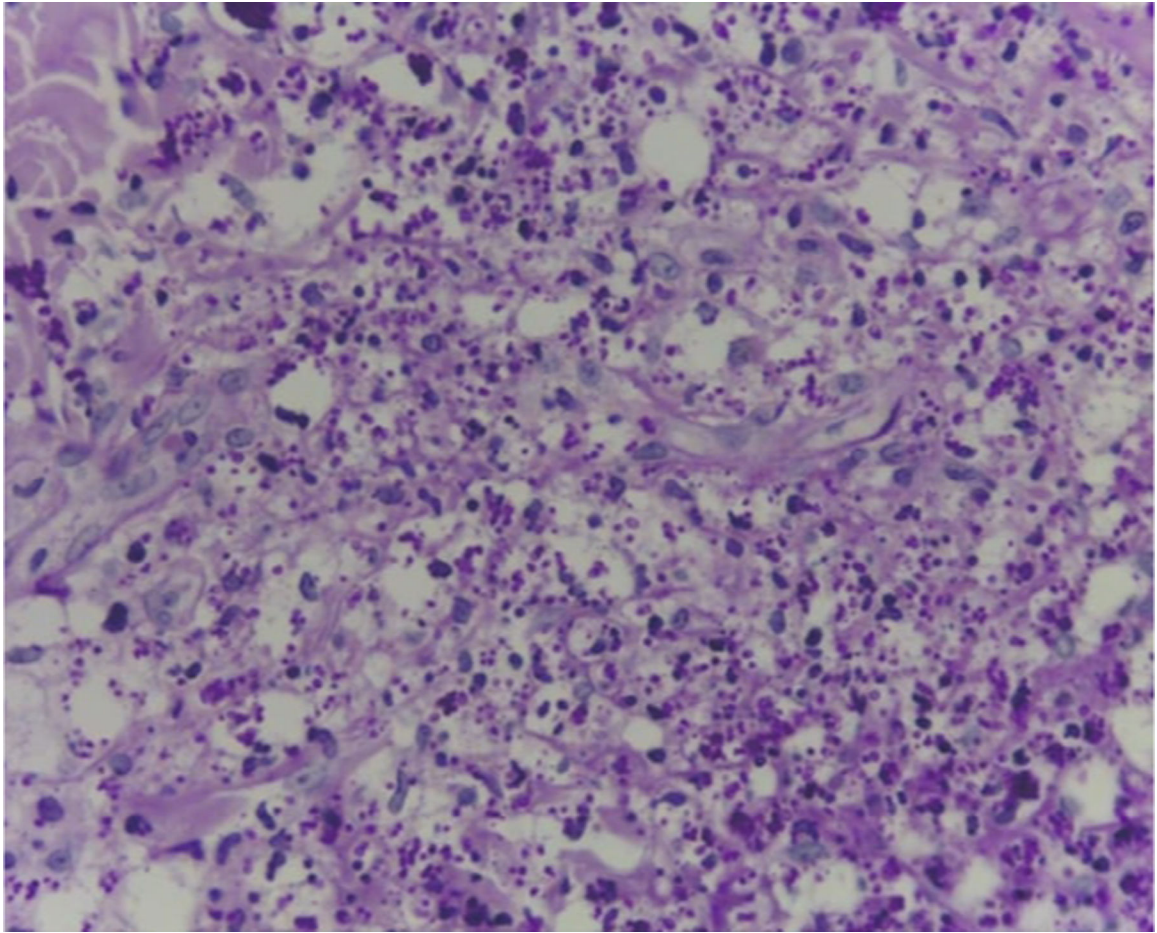


Figure 23.

Talaromycosis histopathologic examination (skin biopsy) demonstrating abundant yeast-like organisms in the cytoplasm of histocytes. The organisms were spherical to oval, about 3-µm in diameter and occasional contained septum (Periodic acid-Schiff stain, 40x). Reproduced from Xian et al.¹⁴⁶ (published under Creative Commons CC-BY license).



Figure 24. Lobomycosis, classic infiltrative nodules on the right outer ear. Reproduced from Talhari et al.¹³⁹ Permission granted for figure reproduction.



Figure 25.
Large aggregate of keloid-like nodules admixed with sclerotic areas diagnosed as lobomycosis on biopsy. Patient from Panama.

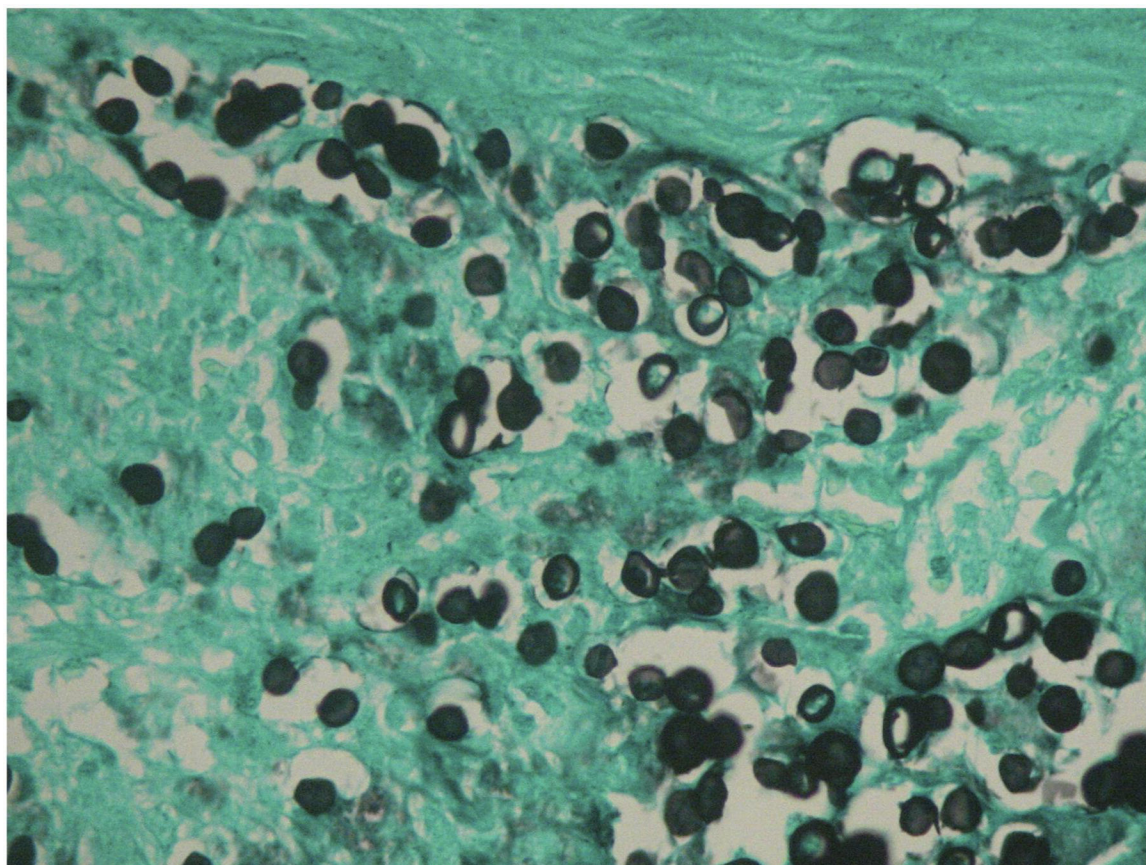


Figure 26. Lobomycosis histopathology examination demonstrating multiple isolated and chained parasites (Grocott stain, 40x). Reproduced from Talhari et al.¹³⁹ Permission granted for figure reproduction.

Table 1.

Summary of fungal skin NTDs—epidemiology and clinical features

Fungal skin NTD	Endemic region	Mode of transmission	Patient population	Dermatologic clinical features	Cutaneous areas of involvement	Other organ involvement
Eumycetoma	Tropical and subtropical regions between latitude 15°S and 30°N ('mycetoma belt')	Traumatic transcutaneous inoculation	Barefoot-walking immunocompetent populations, male (3:1 male/female), third to fourth decade	Painless, firm subcutaneous masses, formation of multiple sinuses within the mass, and a purulent or seropurulent discharge containing sand-like particles called "grains,"	Lower extremities are most common, but other anatomical sites can be involved	Muscle, bone
Chromoblastomycosis	Tropical and subtropical regions	Traumatic transcutaneous inoculation	Immunocompetent male agricultural workers	Verrucous papule (early stage), which progressively enlarges into variable morphologies (nodular, verrucous, tumorous, cicatricial, plaque). Black dots (sclerotic lesions) may be present on plaques	Lower extremities are most common, but other anatomical sites can be involved	Internal organ involvement very rare
Paracoccidioidomycosis	Tropical and subtropical regions of South and Central America	Inhalation of conidia, possible rare direct transcutaneous inoculation	Male agricultural workers between third and fifth decades, smokers	Ulcers or vegetations with fine granulations or hemorrhagic spots	Upper respiratory tract mucosa, face, extremities, trunk	Lungs, adrenal glands
Sporotrichosis	Global distribution	Traumatic transcutaneous inoculation or contact with exudate from infected cat	Patients in contact with vegetation, soil, or cats; veterinarians or those that work with animals	Lymphocutaneous (nodular lymphangitis) or cutaneous (polymorphic skin lesions including papules, nodules, ulcers, verrucous lesions, or plaques following trauma)	Extremities	Pulmonary, osteoarticular, ophthalmic, central nervous system
Emergomycosis	North America, Europe, Africa, Asia	Inhalation of conidia	HIV-infected patients or patients with other immunosuppressive conditions, such as diabetes mellitus, hematologic malignancy, or organ transplant.	Unbilicated papules, hyperkeratotic plaques, nodules, verrucous lesions, ulcerations. Lesions may be polymorphic in one patient or in one lesion over time	Widespread	Lungs, gastrointestinal tract, liver, spleen, bone marrow, lymph nodes, and cervix
Talaromycosis	Tropical and subtropical regions of Asia	Inhalation of spores	HIV-infected patients or patients with other immunosuppressive conditions, such as cancer, organ transplant, or auto-immune diseases.	Painless papules with central umbilication	Face, neck, extremities	Upper and lower respiratory tract, bone, gastrointestinal tract
Lobomycosis	Central and South America	Traumatic transcutaneous inoculation	Forest workers, farmers, hunters, and fishermen	Slowly developing (months, years, decades), keloid-like nodular lesions	Lower extremities, ears, upper extremities, head	No internal organ involvement; testicular involvement reported in one case

Table 2.

Common pathogens and differential diagnoses of fungal skin NTDs

Fungal skin NTD	Common pathogens	Infectious diseases differential diagnoses	Non-infectious diseases differential diagnoses
Eumycetoma	<i>Maduraella mycetomatis</i> <i>Maduraella pseudomycetomatis</i> <i>Maduraella fahalii</i> <i>Maduraella tropicana</i> <i>Trematosphaeria grisea</i> <i>Medicopsis romeroi</i> <i>Scedosporium boydii</i> <i>Falcatiformispora senegalensis</i>	Cutaneous tuberculosis Coccidioidomycosis Chromoblastomycosis Hyalohyphomycosis Sporotrichosis Blastomycosis Dermatophyte pseudomycetomas Botryomycosis	Fibroma Rheumatoid nodule Keloid Fibrolipoma Dermatofibroma Dermatofibroma protuberans Kaposi's sarcoma Malignant melanoma Verrucous carcinoma
Chromoblastomycosis	<i>Fonsecaea pedrosoi</i> <i>Fonsecaea monophora</i> <i>Fonsecaea nubica</i> <i>Cladophialophora carrionii</i> <i>Phialophora verrucosa</i> <i>Rhinocladia aquaspersa</i> <i>Exophiala</i> spp.	Lobomycosis Sporotrichosis Phaeohyphomycosis Protothecosis Tuberculosis verrucosa Leishmaniasis Hanseniasis Eumycetoma Botryomycosis	Keratoacanthoma Lupus erythematosus Cutaneous sarcoidosis Basal Cell Carcinoma Psoriasis
Paracoccidioidomycosis	<i>Paracoccidioides brasiliensis</i> <i>Paracoccidioides lutzii</i>	Tuberculosis Coccidioidomycosis Histoplasmosis Leishmaniasis Chromoblastomycosis Leprosy Syphilis	Sarcoidosis Pneumoconiosis Interstitial pneumonitis Neoplasm
Sporotrichosis	<i>Sporothrix schenckii</i> <i>Sporothrix globosa</i> <i>Sporothrix luriei</i> <i>Sporothrix brasiliensis</i>	Mycobacteriosis Actinomycosis American tegumentary leishmaniasis Blastomycosis Cryptococcosis Paracoccidioidomycosis	Pyoderma gangrenosum
Emergomycosis	<i>Emergomycetes pasteurianus</i> <i>Emergomycetes africanus</i> <i>Emergomycetes canadensis</i> <i>Emergomycetes orientalis</i> <i>Emergomycetes europaeus</i>	Histoplasmosis Cryptococcosis Sporotrichosis Blastomycosis Tuberculosis Non-tuberculous mycobacterial infection Bacillary angiomatosis Varicella Papular pruritic eruption of HIV Secondary syphilis	Drug reactions Kaposi's sarcoma Guttate psoriasis Pyoderma gangrenosum
Talaromycosis	<i>Talaromycetes marnettii</i>	Histoplasmosis Molloscum contagiosum Mycetoma Viral wart Cutaneous tuberculosis	Epidermoid cyst Lipoma

Fungal skin NTD	Common pathogens	Infectious diseases differential diagnoses	Non-infectious diseases differential diagnoses
Lobomycosis	<i>Lacazia loboi</i>	Sporotrichosis Chromoblastomycosis Meliodosis Lepromatous leprosy Diffuse cutaneous leishmaniasis Sporotrichosis Chromoblastomycosis Paracoccidioidomycosis	Keloids Dermatofibrosarcoma protuberans Kaposi's sarcoma Squamous cell carcinoma