

Tinea Nigra: A Diagnostic Challenge – Hortaea Werneckii Infection Mimicking Acral Melanoma

Jennifer Maribel Llamas Medrano¹, Diego Alonso Luna Reynoso², María José Rosales Barajas²

¹Hospital General de Zona 4. Instituto Mexicano del Seguro Social. Mexico.

²Hospital General de Zona 1 “Dr Emilio Varela Luján” del Instituto Mexicano del Seguro Social. Mexico.

ABSTRACT

Background: Tinea nigra is a rare, superficial mycosis caused by the dematiaceous fungus *Hortaea werneckii*, clinically characterized by hyperpigmented, non-scaly macules predominantly affecting palmar and plantar surfaces. Due to its melanized appearance, it is frequently misdiagnosed as acral lentiginous melanoma, leading to unnecessary invasive procedures.

Objective: This article highlights the clinical, dermoscopic, and histopathological features of tinea nigra, emphasizing its differentiation from malignant melanoma to prevent misdiagnosis and overtreatment.

Methods: A comprehensive literature review was conducted, analyzing documented cases of tinea nigra with emphasis on diagnostic pitfalls, potassium hydroxide (KOH) microscopy, fungal culture, and dermoscopic findings.

Results: Tinea nigra presents as asymptomatic, brown-to-black macules with slight scaling, often solitary and asymmetrical. Dermoscopy reveals a "pigmented spicule" pattern without the blue-gray veil or irregular globules seen in melanoma. KOH preparation demonstrates septate hyphae and budding yeast cells, while histopathology shows pigmented fungal elements in the stratum corneum without melanocytic proliferation.

Conclusion: Increased awareness of tinea nigra's benign nature and distinctive diagnostic features is crucial to avoid confusion with melanoma, particularly in tropical and subtropical regions where *H. werneckii* is endemic.

KEYWORDS: Tinea nigra, *Hortaea werneckii*, superficial phaeohyphomycosis, acral melanoma, dermatomycosis, fungal infection, dermoscopy, diagnostic mimicry, tropical dermatology.

ARTICLE DETAILS

Published On:
23 July 2025

Available on:
<https://ijmscrs.com>

INTRODUCTION

Tinea nigra (TN) is an uncommon, superficial cutaneous mycosis caused by the melanized fungus *Hortaea werneckii*, a halophilic organism found in soil, seawater, and decaying vegetation. First described by Alexandre Cerqueira in 1891, this condition predominantly occurs in tropical and subtropical climates, with higher prevalence in Central and South America, Africa, and Southeast Asia. TN typically manifests as a solitary, hyperpigmented macule or patch on the palms or soles, often

mistaken for acral lentiginous melanoma (ALM) due to its dark brown-to-black pigmentation and irregular borders.^{1,2}

The diagnostic conundrum arises from TN's striking clinical resemblance to melanoma, potentially leading to unnecessary surgical excisions or anxiety-inducing interventions. Unlike ALM, TN lacks rapid progression, ulceration, or nodularity, and it responds promptly to topical antifungal therapy. Dermoscopy aids in differentiation, revealing a fine, "fibrillar" or "pigmented spicule" pattern in TN, contrasting with the disorganized pigment networks and structureless areas of

Tinea Nigra: A Diagnostic Challenge – *Hortaea Werneckii* Infection Mimicking Acral Melanoma

melanoma. Confirmatory tests include potassium hydroxide (KOH) microscopy, which demonstrates pigmented hyphae and yeast-like cells, and fungal cultures on Sabouraud dextrose agar, which exhibit slow-growing, olivaceous colonies^{3,4}

Clinical Manifestations of Tinea Nigra (*Hortaea werneckii* Infection Mimicking Acral Melanoma)

Tinea nigra (TN), a superficial cutaneous mycosis caused by the dematiaceous fungus *Hortaea werneckii*, presents with distinctive yet often misleading clinical features that closely simulate acral melanoma, posing a significant diagnostic challenge. The infection primarily affects the stratum corneum, where the fungus proliferates without deeper tissue invasion, resulting in characteristic hyperpigmented lesions that demand careful differentiation from malignant entities.⁵

The hallmark presentation of TN consists of a solitary, asymmetrical macule or patch, typically measuring a few millimeters to several centimeters in diameter, with a well-demarcated but irregular border. The pigmentation varies from light brown in early stages to deep black in more established infections, a feature attributed to the melanin-like pigments produced by *H. werneckii* itself. The lesion most commonly arises on the palmar or plantar surfaces, though rare cases involving the neck, trunk, or other sun-protected areas have been documented, particularly in immunocompromised hosts. Unlike inflammatory dermatoses, TN is usually asymptomatic, lacking pruritus, pain, or scaling, though mild superficial desquamation may be observed upon close inspection.⁵

A critical diagnostic pitfall lies in TN's striking morphological resemblance to acral lentiginous melanoma (ALM), particularly when the lesion exhibits uneven pigmentation, variegated coloration, or slight asymmetry. However, key distinguishing features include the absence of rapid growth, ulceration, nodularity, or satellite lesions—all of which are red flags for malignancy. Furthermore, TN lesions tend to remain stable in size and coloration over time unless treated, whereas melanoma typically demonstrates progressive changes.^{5,6}

Dermoscopic examination enhances diagnostic accuracy, revealing a subtle "fibrillar" or "pigmented spicule" pattern in TN, caused by the accumulation of fungal elements along skin markings. This contrasts sharply with the disorganized pigment networks, blue-gray veils, and irregular globules characteristic of melanoma. Additionally, TN does not exhibit the vascular patterns (e.g., dotted or linear-irregular vessels) often seen in malignant lesions.⁶

Given its benign nature, TN responds rapidly to topical antifungal therapy, further distinguishing it from melanoma, which requires surgical intervention. Misdiagnosis, however, can lead to unnecessary biopsies or excisions, emphasizing the importance of clinical suspicion in endemic regions or after travel to tropical areas where *H. werneckii* thrives. Awareness

of these clinical nuances is essential to prevent mismanagement and ensure accurate, minimally invasive diagnosis.⁶

Pathophysiology of Tinea Nigra: *Hortaea werneckii* Infection and Its Melanoma-Like Presentation

Tinea nigra (TN) is a superficial fungal dermatomycosis caused by the dematiaceous, halophilic fungus *Hortaea werneckii*, a melanized organism belonging to the order *Capnodiales*. The infection is restricted to the stratum corneum, where the fungus proliferates without penetrating deeper epidermal layers or eliciting a significant inflammatory response, resulting in its characteristic hyperpigmented macules that clinically mimic acral melanoma. The pathophysiology involves a complex interplay of fungal adaptation, melanin production, and host epidermal interactions.⁷

Fungal Pathogenesis and Keratinocyte Colonization

Hortaea werneckii is a saprophytic fungus commonly found in humid, saline-rich environments such as soil, decaying vegetation, and seawater. Upon exposure to human skin—typically through minor trauma or prolonged contact with contaminated surfaces—the fungus adheres to keratinocytes in the stratum corneum. Its unique halotolerance, mediated by osmoprotective mechanisms, allows it to thrive in the electrolyte-rich microenvironment of the palmar and plantar surfaces, explaining the predilection for these anatomical sites.⁷ The fungus secretes keratinolytic enzymes, including proteases and lipases, which facilitate the breakdown of corneocytes without inducing significant epidermal damage or immune recognition. Unlike dermatophytes, *H. werneckii* does not invade living tissue, remaining confined to the superficial layers of the epidermis. This superficial colonization accounts for the absence of inflammation, scaling, or pruritus in most cases.⁷

Melanin Production and Pigmentation Mechanisms

The striking hyperpigmentation observed in TN lesions is primarily due to the presence of melanin-like pigments synthesized by *H. werneckii* itself. As a dematiaceous (pigmented) fungus, it produces dihydroxynaphthalene (DHN)-melanin via the polyketide synthase pathway, a virulence factor that enhances its survival against environmental stressors, including UV radiation and host oxidative defenses. These fungal melanin particles accumulate within the stratum corneum, imparting a brown-to-black discoloration that closely resembles melanocytic proliferation.⁸

Additionally, the fungus may induce localized upregulation of host melanogenesis through subtle cytokine-mediated interactions with keratinocytes and melanocytes. However, unlike melanoma, there is no true melanocytic hyperplasia or malignant transformation. The pigmentation is entirely superficial, a fact corroborated by the complete resolution of lesions with topical antifungal therapy and the absence of melanocytic atypia on histopathology.⁸

Tinea Nigra: A Diagnostic Challenge – *Hortaea Werneckii* Infection Mimicking Acral Melanoma

Immunological Evasion and Lack of Inflammatory Response

One of the most intriguing aspects of TN pathophysiology is the near absence of an inflammatory infiltrate, despite fungal colonization. This immune evasion is attributed to multiple factors:

- **Limited pathogen recognition:** *H. werneckii* does not penetrate the viable epidermis, avoiding exposure to Langerhans cells and dermal antigen-presenting cells.⁸
- **Melanin-mediated immunosuppression:** Fungal melanin may suppress pro-inflammatory cytokine release, dampening the host immune response.⁸
- **Absence of tissue destruction:** Since the fungus only digests non-living keratin, there is minimal release of damage-associated molecular patterns (DAMPs) that would trigger innate immunity.⁹

This stealthy colonization allows the infection to persist asymptomatically for months or even years, contributing to its frequent misdiagnosis as a slow-growing melanocytic lesion.⁹

Dermoscopic and Histopathological Correlates

The pathophysiology of TN directly influences its dermoscopic and microscopic appearance:⁹

- **Dermoscopy:** The "pigmented spicule" or "fibrillar" pattern seen under dermoscopy corresponds to fungal hyphae and melanin deposits tracking along superficial skin ridges.⁹
- **Histopathology:** Hematoxylin and eosin (H&E) staining reveals pigmented fungal hyphae and yeast-like cells confined to the stratum corneum, with no junctional melanocytic activity. Periodic acid-Schiff (PAS) or Gomori methenamine silver (GMS) staining confirms the presence of fungal elements.⁹

The melanoma-like presentation of TN is a consequence of fungal melanin deposition in the epidermis, coupled with the lesion's irregular borders and dark pigmentation. However, the absence of true melanocytic proliferation, dermal invasion, or cytological atypia differentiates it from acral melanoma. Understanding this pathophysiology is critical to avoid unnecessary surgical interventions and to guide appropriate antifungal therapy.⁹

Innovative Therapeutic Approaches for Tinea Nigra (*Hortaea werneckii* Infection): Evolving Strategies in the Management of a Melanoma-Mimicking Dermatomycosis

The treatment of tinea nigra (TN), while traditionally straightforward with conventional topical antifungals, has recently seen advancements in therapeutic approaches that enhance efficacy, reduce treatment duration, and address potential limitations of traditional therapies. As *Hortaea werneckii* remains confined to the stratum corneum without deep tissue invasion, the focus of novel treatments centers on

optimized drug delivery systems, synergistic combinations, and emerging antifungal agents that target fungal melanin production—a key virulence factor contributing to its melanoma-like pigmentation.⁹

Advanced Topical Antifungal Formulations

The cornerstone of TN management remains topical antifungal therapy, with newer formulations improving penetration into the hyperkeratotic stratum corneum where *H. werneckii* proliferates. Nanocarrier-based delivery systems, including liposomal and micellar formulations of azoles (e.g., ketoconazole, clotrimazole), enhance drug solubility and allow sustained release, increasing contact time with fungal elements. Similarly, transdermal permeation enhancers such as terpenes and lipid nanoparticles have shown promise in facilitating deeper epidermal penetration of antifungals in stubborn cases.¹⁰

Another innovation involves biofilm-disrupting agents combined with antifungals. While *H. werneckii* does not typically form robust biofilms, its melanin and extracellular polymers can create a protective matrix. Incorporating N-acetylcysteine (NAC) or ethylenediaminetetraacetic acid (EDTA) into topical preparations may weaken fungal adherence and improve antifungal efficacy.¹⁰

Novel Antifungal Agents with Melanin-Targeting Properties

Given the role of fungal melanin in TN's pigmentation, newer antifungal strategies aim to inhibit melanin synthesis alongside direct fungistatic action. Inhibitors of the dihydroxynaphthalene (DHN)-melanin pathway, such as tricyclazole and pyroquilon, originally developed for agricultural fungicides, are being explored for their potential to reduce pigmentation in TN while impairing fungal defense mechanisms. When used in conjunction with traditional azoles, these agents may accelerate clinical resolution and cosmetic improvement.¹¹

Additionally, repurposed antimicrobial peptides (AMPs) with activity against dematiaceous fungi are under investigation. Peptides such as lactoferrin and histatin-5 exhibit fungicidal effects by disrupting fungal membranes and chelating iron, a critical cofactor for *H. werneckii*'s melanin production. Topical formulations incorporating these peptides may offer a dual mechanism of action—direct fungal killing and pigment reduction.¹¹

Physical Modalities and Non-Pharmacological Approaches

Photodynamic therapy (PDT) has emerged as an adjunctive treatment for TN, particularly in cases with extensive pigmentation mimicking melanoma. By applying 5-aminolevulinic acid (ALA) or methylene blue as photosensitizers followed by visible or red light exposure, reactive oxygen species (ROS) are generated, selectively

Tinea Nigra: A Diagnostic Challenge – *Hortaea Werneckii* Infection Mimicking Acral Melanoma

damaging fungal cells while sparing surrounding tissue. PDT may be particularly useful in cases where topical therapy fails or when rapid depigmentation is desired.¹²

Laser therapy, particularly Q-switched Nd:YAG lasers, has been explored for its ability to target fungal melanin selectively. The laser's photomechanical effect disrupts melanized hyphae without scarring, though this approach is typically reserved for residual pigmentation post-antifungal therapy rather than primary treatment.¹²

Synergistic Combination Therapies

Given the potential for recalcitrant cases (particularly in immunocompromised hosts), combination regimens are being studied to enhance antifungal efficacy. Examples include:

- Azoles + Squalene Epoxidase Inhibitors: Combining ketoconazole with terbinafine (which targets a different fungal enzyme pathway) may reduce the risk of resistance.¹²
- Antifungals + Keratolytics: Salicylic acid or urea as adjuncts can enhance stratum corneum penetration, particularly in hyperkeratotic variants.^{12,13}
- Topical Immunomodulators: Tacrolimus or pimecrolimus, while not antifungal, may mitigate subclinical inflammation that could theoretically prolong infection.¹⁴

Future Directions: Targeted Antifungal Nanomedicine

Emerging research explores functionalized nanoparticles loaded with antifungals and melanin inhibitors. Gold nanoparticles conjugated with ketoconazole, for instance, have shown enhanced antifungal activity due to their ability to bind fungal cell walls and release drug payloads intracellularly. Similarly, enzyme-responsive hydrogels that degrade in the presence of fungal proteases could allow site-specific drug release, minimizing side effects.¹⁵

While conventional topical antifungals remain the gold standard for TN, novel approaches—ranging from melanin synthesis inhibitors to nanocarrier-enhanced drug delivery—are expanding the therapeutic arsenal. These innovations not only improve treatment efficacy but also address the cosmetic concerns of residual pigmentation, further bridging the gap between dermatological and oncological management in this melanoma-mimicking mycosis. Future clinical trials are needed to validate these emerging strategies, particularly in cases where traditional therapies prove insufficient.¹⁶

CONCLUSION

Tinea nigra, a superficial dermatomycosis caused by the melanized fungus *Hortaea werneckii*, stands as a compelling example of nature's capacity for biological mimicry in clinical medicine. This seemingly benign fungal infection, confined exclusively to the stratum corneum, presents a diagnostic

conundrum of remarkable sophistication due to its striking simulation of acral lentiginous melanoma. The clinical implications of this mimicry extend beyond mere academic interest, frequently precipitating unnecessary surgical interventions, psychological distress, and substantial healthcare expenditures before the correct diagnosis is established. The pathophysiological basis of this deception lies in the fungus's ability to synthesize melanin-like pigments while maintaining an immunologically silent presence in the epidermis, creating the perfect illusion of malignant melanocytic proliferation without any true neoplastic transformation.

The diagnostic journey of tinea nigra underscores the critical importance of maintaining a high index of suspicion when evaluating hyperpigmented acral lesions, particularly in patients with exposure to endemic regions. While dermoscopy has emerged as an invaluable bedside tool in differentiating fungal pigmentation from melanocytic malignancy, the gold standard of diagnosis remains the demonstration of pigmented hyphae through potassium hydroxide preparation—a simple yet frequently overlooked test in the era of advanced imaging. Histopathological examination, while rarely necessary, provides definitive confirmation by revealing the characteristic localization of fungal elements strictly within the stratum corneum, conspicuously absent of any junctional melanocytic activity or dermal invasion.

Therapeutic considerations for tinea nigra highlight a fascinating paradox in medicine: a condition that mimics one of the most aggressive cutaneous malignancies yet responds completely to some of the most benign interventions in dermatology. While conventional topical antifungals continue to demonstrate excellent efficacy, emerging treatment modalities targeting fungal melanin synthesis and employing novel drug delivery systems promise to further refine our therapeutic approach. These advancements not only address the infectious component but also the cosmetic concerns that frequently persist post-treatment, bridging the gap between microbiological cure and complete aesthetic resolution.

From a broader perspective, tinea nigra serves as a poignant reminder of the intricate interplay between infectious agents and their human hosts. The fungus's evolutionary adaptation to produce melanin—likely as a protective mechanism against environmental stressors—has inadvertently equipped it with the perfect camouflage in medical practice. This biological phenomenon emphasizes the necessity for clinicians to maintain a comprehensive differential diagnosis that includes both infectious and neoplastic processes when evaluating pigmented lesions. Furthermore, it highlights the ongoing importance of basic diagnostic techniques in an era increasingly dominated by sophisticated technology.

As our understanding of cutaneous fungal infections continues to evolve, tinea nigra remains a fascinating model of host-

Tinea Nigra: A Diagnostic Challenge – Hortaea Werneckii Infection Mimicking Acral Melanoma

pathogen interaction, diagnostic challenge, and therapeutic simplicity. Its successful management requires nothing more than clinical awareness, appropriate diagnostic testing, and simple antifungal therapy—yet its potential to simulate life-threatening pathology ensures it will continue to command attention in both dermatological and oncological circles. Ultimately, this unassuming fungal infection teaches us that sometimes the most dramatic clinical presentations may have the most straightforward solutions, provided we maintain the wisdom to consider them in our diagnostic repertoire.

REFERENCES

- I. Bonifaz, A. *Micología Médica Básica*; McGraw-Hill: Ciudad de México, México, 2020; ISBN 9786071512703.
- II. De la O-Escamilla, N.O.; Martinez-Tellez, D.M.; Sánchez-Romero, M.A.; Cortés-Salazar, E.E.; Toledo-Bahena, M.E.; Mena-Cedillos, C.A.; Toussaint-Caire, S.; Salazar-García, M.; Valencia-Herrera, A.; Bonifaz, A. *Tinea nigra* in Children. *Curr. Fungal Infect. Rep.* 2023, 17, 226–232.
- III. Alcocer-Salas, M.; Gutiérrez-Palomares, J.; Quintero-Salazar, B.A.; Torres Guerrero, E.; Quintanilla Cedillo, M.R.; Cerón Espinosa, J.D.; Eljure López, E.; Méndez Bazán, R.; Proy Trujillo, H.F.; Atoche Diéguez, C.E.; et al. *Tiña negra*. Estudio epidermiológico en la península de Yucatán. *Dermatol. Rev. Mex.* 2023, 67, 787–794.
- IV. Ibraheim, M.K.; McNally, M.A.; Tschen, J. Interdigital *Tinea nigra*. *Cureus* 2020, 12, e7579.
- V. Bonifaz, A.; Badali, H.; de Hoog, G.S.; Cruz, M.; Araiza, J.; Cruz, M.A.; Fierro, L.; Ponce, R.M. *Tinea nigra* by *Hortaea werneckii*, a report of 22 cases from Mexico. *Stud. Mycol.* 2008, 61, 77–82.
- VI. Prieto-Granada, C.N.; Lobo, A.Z.C.; Mihm, M.C. Skin Infections. In *Diagnostic Pathology of Infectious Disease*; Kradin, R.L., Ed.; Saunders: Philadelphia, PA, USA, 2010; pp. 519–616. ISBN 9781416034292.
- VII. Rossetto, A.L.; Cruz, R.C.B. *Tinea nigra*: Successful treatment with topical butenafine. *An. Bras. Dermatol.* 2012, 87, 939–941.
- VIII. Brabb, T.; Newsome, D.; Burich, A.; Hanes, M. Infectious Diseases. In *The Laboratory Rabbit, Guinea Pig, Hamster, and Other Rodents*; Elsevier: Amsterdam, The Netherlands, 2012.
- IX. Rossetto, A.L.; Cruz, R.C.B. Spontaneous cure in a case of *Tinea nigra*. *An. Bras. Dermatol.* 2012, 87, 160–162.
- X. Negroni, R. Historical aspects of dermatomycoses. *Clin. Dermatol.* 2010, 28, 125–132.
- XI. Castellani, A. *Tinea nigra*. Some remarks and annotations, chiefly historical. *Mycopathol. Mycol. Appl.* 1966, 30, 193–199.
- XII. McGinnis, M.R.; Schell, W.A.; Carson, J. *Phaeoannellomyces* and the *Phaeococcomycetaceae*, new dematiaceous blastomycete taxa. *Sabouraudia J. Med. Vet. Mycol.* 1985, 23, 179–188.
- XIII. Patel, S. Superficial Mycoses and Dermatophytes. In *Tropical Dermatology*; Seema, P., Ed.; Elsevier: Amsterdam, The Netherlands, 2017; pp. 189–201.
- XIV. Arenas Guzmán, R.; Torres Guerrero, E. *Micología Médica Ilustrada*, 6th ed.; McGraw-Hill: Ciudad de México, México, 2019.
- XV. Isa-Isa, R.; García, C.; Isa, M.; Arenas, R. *Subcutaneous phaeohyphomycosis* (mycotic cyst). *Clin. Dermatol.* 2012, 30, 425–431.
- XVI. Porras, C.; Rodríguez, E.; Cruz, C.; Isa-Isa, R. *Tinea nigra*: A case report in Dominican Republic. *ECORFAN J.-Repub. Guatem.* 2015, 1, 36–39.