Tricbophyton rubrum tinea capitis in an HIV-positive patient with generalized dermatophytosis



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INTRODUCTION

Generalized dermatophytosis, or chronic widespread dermatophytosis, is an uncommon disease most often caused by *Trichophyton rubrum* in the immunocompromised or chronically ill host.^{1,2} Tinea capitis caused by *T rubrum* is rare in adult patients and has not, to our knowledge, been described in the HIV/AIDS population. We report the unusual case of a 62-year-old HIV-positive woman with generalized dermatophytosis and tinea capitis caused by *T rubrum*.

CASE REPORT

A 62-year-old Hispanic woman with a history of HIV/AIDS, noncompliance with highly active antiretroviral therapy, untreated hepatitis C, and severe chronic obstructive pulmonary disease presented with progressively worsening dyspnea on exertion. She was intubated for respiratory failure and treated with intravenous methylprednisolone, before transition to oral prednisone. The dermatology department was consulted for a diffuse, peeling rash that was reportedly present for years. Generalized tinea was found on biopsy more than 1 year prior, although treatment status was unknown.

Examination found generalized, monomorphous, purpuric 2- to 3-mm papules with an annular distribution on the abdomen, forearms, and legs (Fig 1). There was diffuse scale with localized areas of sparing. The patient's palms and soles were hyperkeratotic, and all nails were thickened and yellow, with significant subungual debris. Her scalp

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had thick, scaly plaques with easily extractable hairs (Fig 2).

Laboratory findings were remarkable for a CD4 count of 135 cells per cubic millimeter and negative blood cultures. A toenail clipping showed numerous periodic acid-Schiff-positive hyphae. Scalp hairs, scalp skin, trunk skin, and nail clippings were positive for hyphae on potassium hydroxide examination, and all speciated T rubrum on fungal culture. Hyphae were only seen on the outside of the hair shaft. Two punch biopsies were performed from purpuric papules on the forearm and abdomen. Hematoxylin-eosin staining found folliculitis with evidence of follicular rupture (Fig 3). Periodic acid-Schiff staining confirmed the presence of fungal organisms at the periphery of the hair shaft, without dermatophyte invasion of the dermis. The patient was started on fluconazole, 200 mg daily, for generalized dermatophytosis, and clinical improvement was noted after 3 weeks of treatment. She was discharged on a prolonged course of fluconazole.

DISCUSSION

Generalized dermatophytosis is a chronic, widespread dermatophyte infection across multiple body sites. This infection is distinct from disseminated dermatophytosis, as the dermatophyte does not penetrate into the dermis or disseminate to the subcutaneous tissue and lymph nodes. The source of widespread infection is thought to originate from fungal reservoirs secondary to longstanding onychomycosis, moccasin-type tinea pedis, or tinea cruris.¹

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Fig 1. Widespread dermatophytosis. Generalized, monomorphous, red-purpuric, 2- to 3-mm papules on the abdomen (**A**) and forearm (**B**).



Fig 2. Tinea capitis. Diffuse, thick plaques overlaid with white scale on the scalp.



Fig 3. Widespread dermatophytosis on histology. Hematoxylin-eosin staining shows fungal organisms at the periphery of the hair shaft, without dermal infiltration.

The most common pathogen of generalized dermatophytosis is *T rubrum*, an anthropophilic dermatophyte with multiple, geographically patterned phenotypes.¹

Although generalized dermatophytosis has been reported in immunocompetent patients, it more frequently occurs in those with immunocompromise or skin barrier dysfunction.³ Widespread dermatophytosis is uncommon in the HIV population; however, severe immunologic dysfunction is thought to render increased risk through an impaired cellmediated response.^{2,3} In a study of more than 400 HIV-1–infected patients, the 3 patients who had generalized dermatophytosis all had CD4 counts less than 200.²

Although chronic widespread dermatophytosis alone is rare, the concurrence of tinea capitis makes this case even more unusual. Tinea capitis is largely considered a disease of prepubertal children, with adult cases accounting for only approximately 3% of all infections.⁴ Sweat, sebum, the presence of Pityrosporum ovale, and increased hair thickness offer protection against tinea capitis after puberty.^{4,5} Adults with a history of diabetes, anemia, immunosuppression, steroid use, hormonal abnormalities, or pathogen exposure are thought to be predisposed.⁴ Although theoretically at increased risk of tinea capitis owing to immunocompromise, it has been proposed that heightened hair follicle colonization by Pityrosporum species is a protective factor against tinea capitis in patients infected with HIV.⁵

Review of the literature found only 7 cases of tinea capitis in the setting of HIV infection. A study of 117 patients with AIDS or AIDS-related complex recorded a single case of tinea capitis, and the organism was not stated.³ Three cases caused by *Microsporum canis* and individual cases caused by *Microsporum langeroni, Trichophyton violaceum*, and *Microsporum audouinii* are reported.⁵ To our knowledge, we report the first case of *T rubrum* as a cause of tinea capitis in a patient with HIV/AIDS.

T rubrum is a very uncommon cause of tinea capitis in the general population, comprising 0.78% of cases worldwide, with only 127 cases reported as of 1995 according to one survey of the literature.⁶ Despite its extreme rarity, *T rubrum* led to a robust case of tinea capitis in this patient, likely owing to autoinoculation from generalized *T rubrum*

dermatophytosis and onychomycosis. In contrast to other dermatophyte species, *Trubrum* can uniquely function as both an endothrix and an ectothrix infection in the setting of hair invasion,⁷ which may have allowed long-term colonization and development of tinea capitis in this immunocompromised patient. Microscopically, hyphae were noted exclusively on the outside of the hair shaft, suggesting ectothrix infection; this was further supported clinically by her absence of alopecia.

In widespread dermatophytosis, systemic antifungal therapy is the treatment of choice, particularly terbinafine, itraconazole, or fluconazole. In 3 HIVpositive patients with generalized chronic dermatophytosis, treatment was successful with 7 days of oral ketoconazole, 200 mg twice daily, followed by oral ketoconazole, 200 mg daily over 24 months.² However, ketoconazole is not considered first-line treatment, given the potential for severe side effects. Other reports describe clearing of widespread dermatophytosis with 12 days to 21 weeks of systemic terbinafine, with or without combination topical antimycotic therapy.^{8,9} Complete or near-complete recovery has also been observed within 1 to 2 months of systemic itraconazole therapy in patients with generalized dermatophytosis from T rubrum.¹⁰ In the patient described here, lesions resolved within 3 weeks of treatment with fluconazole, 200 mg daily.

To our knowledge, this is the first report of a unique presentation of generalized dermatophytosis and tinea capitis secondary to T rubrum in a patient with HIV/AIDS. This case emphasizes the

importance of recognizing widespread dermatophytosis and tinea capitis in the setting of HIV infection, with the potential for T rubrum as a causative organism.

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