CLINICAL MANAGEMENT extra

Diagnosis and Management of Cutaneous Tinea Infections

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GENERAL PURPOSE:

To provide information about the epidemiology, clinical features, and management of cutaneous tinea infections.

TARGET AUDIENCE:

This continuing education activity is intended for physicians, physician assistants, nurse practitioners, and nurses with an interest in skin and wound care.

LEARNING OBJECTIVES/OUTCOMES:

After completing this continuing education activity, you should be better able to:

1. Summarize the epidemiology related to cutaneous tinea infections.
2. Describe the clinical features of cutaneous tinea infections.
3. Identify features related to the diagnosis and management of cutaneous tinea infections.

ABSTRACT

Dermatophyte or tinea infection refers to a group of superficial fungal infections of the hair, skin, and nails. Tinea infections are most commonly caused by fungi of the genus *Trichophyton*, *Microsporum*, or *Epidermophyton*. Cutaneous manifestations of tinea infections are seen worldwide and classified based on the affected body site. The diagnosis of these conditions is complicated by morphologic variations in presentation and overlap with other common infectious and noninfectious entities. As a result, diagnosis and appropriate management of these conditions are essential to avoid patient morbidity. This case-based review summarizes the epidemiology, relevant clinical features, microbiology, and management considerations for commonly encountered tinea infections.

KEYWORDS: case review, cutaneous tinea infection, dermatophyte, fungal infection, fungi, tinea

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INTRODUCTION

Tinea infections (dermatophytoses) are superficial infections of the keratinized epithelial layer. They are prevalent worldwide, and it is estimated that up to 25% of the global population has skin mycoses, including tinea infections.[1]

Dermatophytosis refers specifically to cutaneous infections caused by fungi from the *Trichophyton*, *Microsporum*, or *Epidermophyton* genera. Fungal organisms from each genus are preferentially associated with specific tinea manifestations. For example, *Trichophyton rubrum* is most commonly associated with tinea cruris ("jock itch").[1] Incidentally, epidemiologic studies show that dermatophyte infections vary by geography as well. Toenail, foot, and body involvement were most common in an Italian study, with *T rubrum* implicated in 64% of all dermatophyte infections.[2] In contrast, *Trichophyton tonsurans* was found to be the most isolated dermatophyte in a study based in the US.[3]

Dermatophyte infections are classified by the site of body involvement, which varies with age. Involvement of the scalp is common in children, whereas involvement of the torso, face, neck, inguinal area, foot, and nail is more common in persons of older ages. Indeed, most cases of dermatophytosis are seen in postpubertal individuals and may be attributable to pubertal changes in sebaceous gland physiology.

Tinea infections are often diagnosed based on clinical findings; they may provide a diagnostic dilemma for those not familiar with the varied presentations and the overlap in presentation with other dermatologic entities. In most cases, diagnoses of fungal conditions require laboratory confirmation because of the extent of overlap between these conditions, with few exceptions. Microscopy, fungal culture, or the use of Wood lamp examination may help to refine a differential diagnosis, but Wood light examination requires specialized technical expertise and may not be available.[4] Therefore, the diagnosis should always be confirmed by the appropriate culture technique prior to initiating treatment (Table).

Herein, the authors present and discuss the most common forms of dermatophytosis: tinea capitis, tinea corporis, tinea versicolor (pityriasis versicolor), tinea pedis, tinea unguium, and tinea incognito. Further discussion includes the clinical presentation of the varying conditions, relevant epidemiologic factors, and treatment considerations for each entity using a case-based approach.

TINEA CAPITIS

A 6-year-old boy adopted from East Africa with Fitzpatrick skin type 4 was brought into the clinic for evaluation of a 4-month history of patchy hair loss on the scalp (Figure 1). The areas of hair loss were occasionally itchy. On examination, nonscarred areas of alopecia with scale and erythema were visible. More recently, a related sibling had begun to develop a small, similar area of scalp alopecia. Skin scraping revealed fungal hyphae seen on direct examination, which was confirmed 4 weeks later by a positive fungal culture that grew T tonsurans.

Tinea capitis is a dermatophyte infection of the scalp area. It is common in children and infrequent in adults. Cases of tinea capitis occur more frequently in children of African descent.[5] Of the usual genera causing dermatophytosis, only *Trichophyton* and *Microsporum* are causative of tinea capitis, but geographic variations in the epidemiology of tinea capitis exist.[1,5–7] In the US, *T tonsurans* is the most prevalent pathogen, accounting for more than 90% of cases.[8] In contrast, *Microsporum canis* causes the majority of tinea capitis cases in Europe and rural areas in the United Kingdom,[7,9] and *Trichophyton violaceum* is frequently identified in east Africa and Tehran, Iran.[6,10]

Clinical presentations depend on the causative pathogen involved, although several presentations have been previously described.[11] Infection by *T tonsurans* often results in "black dot" tinea capitis because of hair breakage at the level of the scalp within marked areas of alopecia. This is referred to as an endothrix infection. Conversely, "gray patch" tinea capitis presents as a dry, annular patch of alopecia with marked scaling and is associated with an ectothrix infection caused by *Microsporum audouinii*. In severe cases, it may present as a kerion, characterized by a pustular reaction with accompanying alopecia. Kerion results from an exaggerated host immune response to the causative pathogen and may present as a localized boggy and swollen area.

The most common sign of tinea capitis is the presence of alopecia with or without scale. Therefore, differentiation of this condition from alopecia areata (an autoimmune condition causing hair follicle destruction) is important. Accompanying lymphadenopathy with involvement of the posterior cervical and/or posterior auricular lymph nodes is suggestive of tinea capitis. Lymphadenopathy is also often seen with kerion.

Table.
METHODS FOR DIAGNOSING DERMATOPHYTE INFECTIONS[4]

Test	Value	Suitability
Skin scraping: culture	Criterion standard for diagnosis Positive culture takes 7-14 d but specimens are held for 21 d before a negative result is reported Noninvasive	Tinea capitis Pityriasis versicolor Tinea corporis Tinea pedis Tinea unguis Tinea incognito
Skin scraping: direct examination	May be done in office or sent to the microbiology laboratory Positive skin scraping is considered diagnostic Noninvasive	Tinea capitis Pityriasis versicolor Tinea corporis Tinea pedis Tinea unguis Tinea incognito
Wood light examination	Requires • a Wood lamp (UV-A source) • a darkened room • trained technician	Pityriasis versicolor fluoresces pale yellow to white Tinea corporis and capitis caused by *Microsporum* Infections may fluoresce a blue-green color
Skin or nail biopsy	Limited value as diagnostic procedure (uncommon) Considered in cases where dermatophytosis is unexpectedly refractory to treatment Invasive	Under certain circumstances for diagnosis of: • Tinea capitis • Pityriasis versicolor • Tinea corporis • Tinea pedis • Tinea unguis • Tinea incognito

Although hair loss because of tinea capitis is often temporary, chronic tinea capitis may increase the risk of scarring alopecia. Considering and ruling out tinea capitis is important because effective therapeutic options exist.[12] Topical agents are not effective in tinea capitis because of impaired penetration of the hair follicles. Oral antifungal therapy typically involves the use of terbinafine 250 mg (or 5 mg/kg) daily for 4 weeks or griseofulvin 500 mg (or 20 mg/kg) daily for 6 weeks. Local epidemiology can influence treatment choice; oral terbafine is typically more effective in treating *T tonsurans*-associated tinea capitis.[12] Additional therapies include the use of 1% or 2.5% selenium sulfide shampoo or 2% ketoconazole shampoo; these are thought to reduce the risk of transmission between persons by decreasing aerosolized spores. Treatment and examination of asymptomatic individuals close to the infected patient should be considered because of the likelihood of fungal shedding.

TINEA CORPORIS

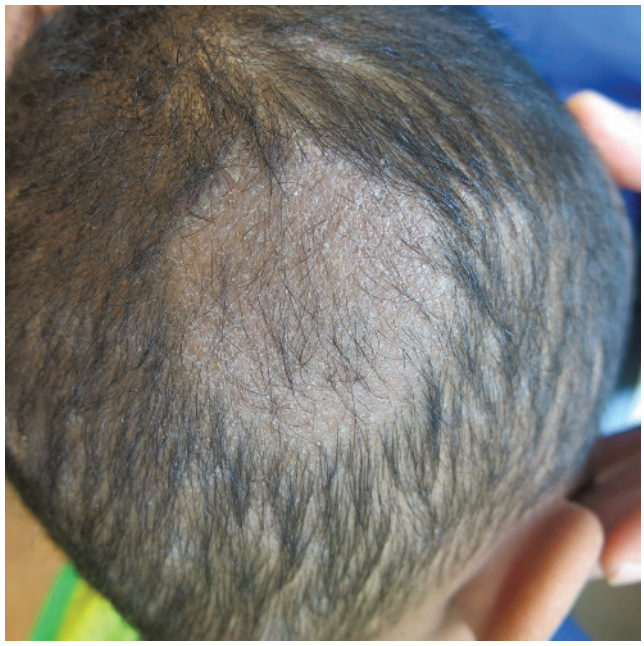
A 16-year-old amateur wrestler presents with a slowly expanding red, itchy, and scaly patch on his left forearm (Figure 2). He was prescribed a topical steroid cream 4 weeks prior to this assessment by another care provider. Although this has helped with pruritus, it has not cleared the patch. His providers decide to do a skin scraping for fungal examination and culture. The skin scraping shows no hyphae, but the culture comes back with a positive result for Trichophyton *species. Treatment with a topical antifungal cream clears the rash in 3 weeks and there is no recurrence after treatment is discontinued.*

Tinea corporis is a superficial dermatophyte infection involving the chest, face, arms, and/or legs. It is estimated that up to 20% of persons will be affected over their lifetime.[13] It is seen more commonly in children and younger adults.[14] Although person-to-person transmission of tinea corporis is possible, the risk is comparatively low, with rates of transmission between family members or individuals in school reported around 10%.[15] Given the limited rates of transmission, individuals with tinea corporis do not need to be isolated if proper precautions are taken.

The diagnosis of tinea corporis is based on clinical appearance. Tinea corporis manifests as erythematous patches or plaques with a scaly, advancing raised edge and a central clearing. Once established, the rash typically spreads centrifugally. The rash may be annular in shape and is frequently pruritic.

Treatment of tinea corporis requires the use of topical antifungals. First-line therapy of tinea corporis is topical terbinafine 1% cream once daily for up to 6 weeks or at least 1 week following the clearance of the rash.[16,17] Alternative choices involve naftifine

Figure 1.
TINEA CAPITIS



1%, butenafine 1%, or topical azoles (ie, ketoconazole, miconazole, clotrimazole creams).[16] Newer therapies for tinea corporis include luliconazole cream applied once daily.[18] Oral therapies are not used to treat tinea corporis except in extensive or refractory cases because of the potential for adverse effects.

TINEA VERSICOLOR (PITYRIASIS VERSICOLOR)

A 15-year-old African American male adolescent presents with an 8-month history of a slowly spreading eruption on the torso (Figure 3). The eruption is mildly pruritic and consists of sharply demarcated macules with a fine scale. In some areas, the macules are coalescing to form larger patches.

Tinea versicolor (now called pityriasis versicolor) is not caused by dermatophytes but rather by a yeast of the genus *Malassezia*. It merits inclusion in this article because it is still referred to as tinea and often mistakenly attributed to dermatophyte causes. The fungus is found normally as part of the skin flora but may cause infection in response to altered extrinsic environmental and intrinsic host factors. Indeed, while pityriasis versicolor has a worldwide distribution, it is more commonly seen in tropical environments likely attributed to elevated temperatures and humidity that enhance proliferation of the fungus.

It is most commonly seen in younger to middle-aged adults, but may affect individuals of all ages. Pityriasis versicolor follows sebaceous gland density and commonly presents on the upper torso, back, and neck. Involvement of the face is uncommon and is typically associated with children.[19]

Pityriasis versicolor presents as multiple round or oval macules or patches and draws the name versicolor from its variable appearance. These lesions can present as areas of hypopigmentation (whitish/tan) or hyperpigmentation (brown). The altered pigmentation is the result of inflammation and the organism's effect on melanin-producing structures. Fine scale is often present and may be accentuated by stretching or scraping of the skin. This clinical feature is known as the "evoked-scale sign" and may aid in clinical diagnosis. Pruritus occurs infrequently, and transmission of pityriasis versicolor between persons does not occur.

While patients are most commonly asymptomatic, pityriasis versicolor is often treated because of its unsightly appearance. Topical therapies are the preferred agents, and systemic treatments are generally reserved for extensive skin disease in which topical therapy is impractical for the patient. Treatment choices include topical antifungal creams such as azoles, terbinafine, and ciclopirox creams and shampoos including 2% ketoconazole, zinc pyrithione shampoo, selenium sulfide shampoo, and sulfur plus salicylic acid shampoo.[20] Systemic therapies include oral azole antifungals such as itraconazole and fluconazole. Recent evidence suggests that 5 days of itraconazole 200 mg/d is as effective in curing the condition as the traditionally used 7-day course.[21,22] Although the full 7-day course can be used in more severe cases, a shorter course of antifungals can limit hepatotoxicity, particularly in

Figure 2.
TINEA CORPORIS

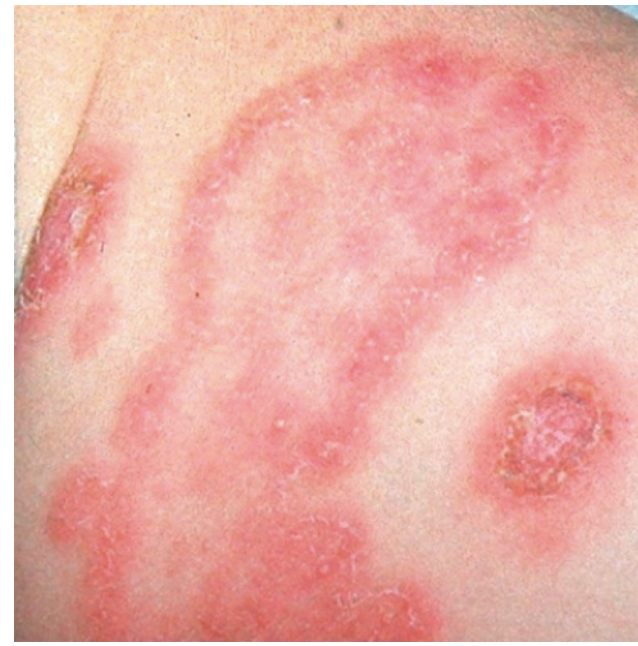
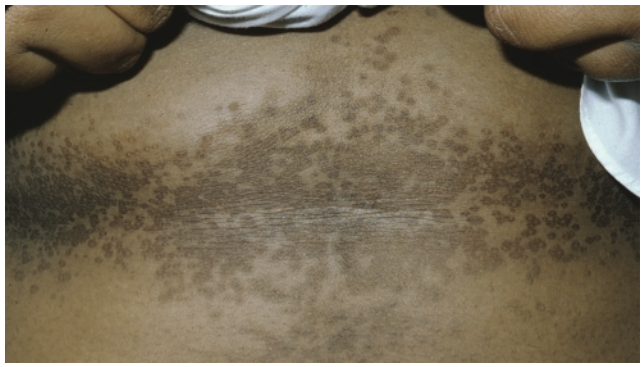


Figure 3.
TINEA VERSICOLOR



younger patients.[22] Oral terbinafine is not effective in treating pityriasis versicolor because it does not reach high-enough levels in the skin; however, topical terbinafine can be effective.

Because pityriasis versicolor has been reported to reoccur in up to 60% of patients following cessation of treatment in the first year,[19] prophylactic treatment is generally recommended. The use of 2.5% selenium sulfide shampoo on the first and third days of each month for 6 months following a treatment course has been used to prevent recurrence.[19] However, 400 mg of oral ketoconazole monthly or 200 mg itraconazole twice daily on the first day of each month for 6 months has been shown to significantly reduce recurrence.[23] That said, in 2016, the FDA issued a black box warning for oral ketoconazole because of the risk of fulminant hepatitis, and this drug should no longer be prescribed.

TINEA PEDIS

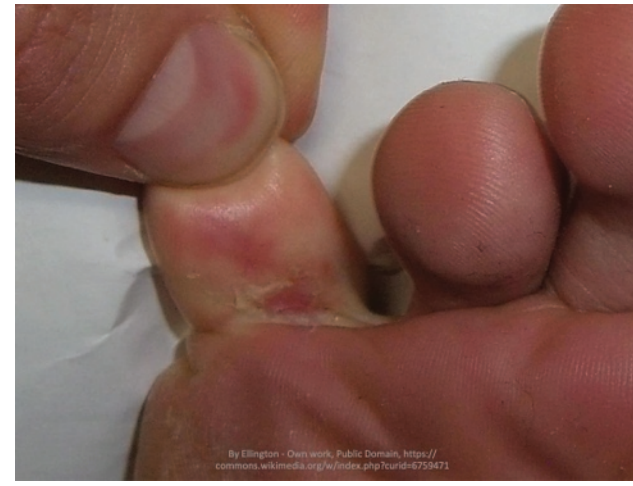
A 45-year-old man presents with a 6-week history of episodic pruritus involving the fourth web space of the right foot (Figure 4). He has tried over-the-counter treatment for athlete's foot with minimal improvement. This has been a recurrent problem and seems to worsen with wearing warm footwear and under humid conditions. On closer examination, there is erythema and scaling involving the lateral foot that extends onto the heel.

Tinea pedis is the most common dermatophyte infection in the US and involves the soles and interdigital spaces of the foot. It is commonly known as athlete's foot and is seen in athletes, military soldiers, and homeless individuals.[24,25] Tinea pedis occurs more frequently in adults, with a peak prevalence noted in men between 31 and 60 years of age.[26] It is associated with humid environments and occlusive footwear that contribute to impaired sebaceous gland function.[4,26,27] As such, tropical and subtropical environments have widespread incidence of dermatophyte infections. However, dermatophytes may still be acquired from public facilities, including gyms, swimming pools, and locker rooms. *T rubrum*, *Trichophyton interdigitale*, *Trichophyton mentagrophytes*, and *Epidermophyton floccosum* are among the dermatophytes commonly associated with tinea pedis in adults.[11]

Tinea pedis is classified into four clinical types: interdigital, moccasin, inflammatory, and ulcerative. These definitions are classified based on the characteristic pattern of foot involvement. Most commonly, tinea pedis presents as the interdigital variety, which is characterized by interdigital erythema, scaling, maceration, and fissuring between the toes. Initial involvement of the fourth and fifth toes is seen frequently. Involvement of the dorsal surfaces of the toe is not common, although it can occur in persons with immune compromise. The moccasin distribution, also known as chronic hyperkeratotic type, is characterized by demarcated fine scaling with accompanying erythema and/or fissuring. Inflammatory tinea pedis manifests as rigid vesiculation, bullae formation, or pustule formation on the midanterior plantar surface. The ulcerative clinical type is characterized by rapid progression of vesiculopustular lesions and ulceration of the skin. The ulcerative phenotype is seen more commonly in individuals with diabetes or those who are immunocompromised. Symptoms associated with all presentations include pruritus and a pain/burning sensation.

Treatment of tinea pedis most commonly involves the use of topical antifungal creams until clearance is achieved. A wide range of effective topical antifungals exist for the treatment of tinea pedis.[28,29] These include terbinafine 1% cream, butenafine 1% cream, naftifine 1%, or azoles, including econazole 1% or ketoconazole 2% creams. While topical options are effective for most cases of tinea pedis, oral therapy should be considered in those with severe or chronic disease, concomitant onychomycosis, and those recalcitrant to topical therapy. Oral terbinafine is prescribed

Figure 4.
TINEA PEDIS



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250 mg once daily in adults or 125 mg twice daily therapy and used most frequently because of its known safety profile. It is also a fungicidal agent.

Alternative therapies include griseofulvin 750 mg/d and itraconazole 100 mg/d. These are both fungistatic. A systematic review of the evidence suggests that while all oral therapies are effective as compared with placebo, terbinafine achieves clearance better than griseofulvin.[30]

TINEA UNGUIUM

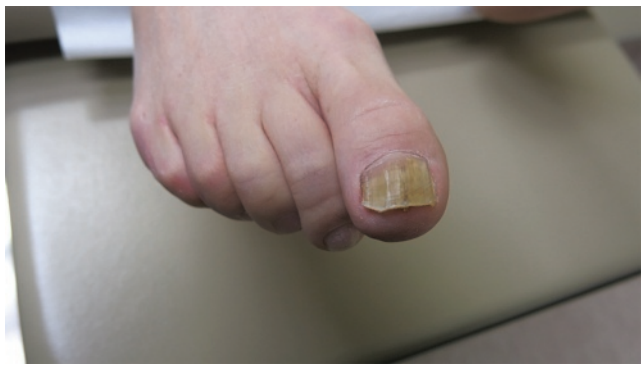
A 58-year-old man presents for thickening and discoloration of the right great toenail (Figure 5). He has noticed a slow progression of this change over the last 2 years, and he has had episodic tinea pedis since his early 20s. This change is asymptomatic, but he now has difficulty cutting the nail because of its increasing thickness.

Fungal infection of the nail is referred to as onychomycosis, which includes both dermatophyte and nondermatophyte infections. Tinea unguium refers specifically to nail infections caused by dermatophytes, which are the associated pathogen in up to 90% of onychomycosis cases.[31] Of the dermatophytes, *T rubrum* and *T interdigitale* are most common. Nondermatophyte molds (eg, *Candida albicans*) make up approximately 5% of cases.[31]

Interestingly, it is seen more frequently in men than women.[31] Tinea unguium is mainly seen in adults but can infrequently occur in children. When it occurs in children, *T rubrum* is the prevalent organism and has been reported in 67% of cases.[32] Less prevalent organisms in children include *T mentagrophytes* and species of *Candida*.

Tinea unguium shares similar risk factors with tinea pedis and is associated with occlusive footwear, hyperhidrosis, immunosuppressive agents, diabetes, HIV, increasing age, and trauma to the nail. Both fingernails and toenails may be affected, although toenail infections are more common. Patients who have tinea unguium may also have concomitant tinea pedis.[33]

Figure 5.
TINEA UNGUIUM



Tinea unguium may be diagnosed clinically but should be confirmed with supportive laboratory findings, including fungal culture (Table). Generally, it presents with nail thickening, discoloration, and potential friability, although many patterns exist. Distal and lateral subungual onychomycosis most commonly involves the nail bed via the hyponychium (underside of distal nail) and presents as thickening (hyperkeratosis) of the nail bed. Yellowing of the nail, thickening of the distal nail, and separation of the nail from the nail bed (onycholysis) are seen with progression of the condition. Eventual thickening of the entire nail may be seen.

Superficial white onychomycosis involves the dorsum of the nail surface, which presents as discrete white patches and is caused by *T mentagrophytes*. Atypical presentations may involve white transverse bands on the nail because of invasion of the nail fold and are typically associated with *T rubrum*. Proximal subungual onychomycosis involves invasion of the proximal nail bed and is associated with immunosuppression, including HIV. Multiple patterns of infection may occur simultaneously and form a mixed pattern.

While many patients consider tinea unguium a cosmetic disturbance, individuals may experience pain or discomfort while wearing occlusive footwear and activities including walking or running. In cases where tinea unguium contributes to morbidity, a conversation surrounding medical therapy is warranted. In addition, transmission to close contacts is possible and as such infections may benefit from treatment.

Tinea unguium represents a challenge to treat and should be confirmed prior to initiation of therapy because of treatment burden. When initiated, treatments are often carried out until absolute cure has been reached. Absolute cure is a combination of mycologic and clinical cure, that is, negative nail cultures and microscopy and a normal-appearing nail.[34] Topical treatments are not very efficacious because of impaired penetration into the nail and limited by an onerous 48-week duration of therapy. These are typically reserved for mild involvement of the nail and superficial white onychomycosis or for patients for whom oral antifungals are contraindicated. Topical treatments have variable results, with mycologic cure rates ranging from 31% to 50%.[28,35-37]

Therapeutic options include efinaconazole 10% solution, tavaborole 5% solution, ciclopirox 8% lacquer, and butenafine 2%.[38] Efinaconazole 10% solution used once daily for 48 weeks was relatively efficacious with mycologic cure rates of approximately 50% and absolute cure rates of 15%.[37] Oral therapies are more effective for treating tinea unguium but are burdened by potential adverse effects, the need for routine blood work monitoring, treatment duration, and the risk of recurrent infection. The most common adverse effects of oral terbinafine include headache, altered taste, and gastrointestinal upset. Terbinafine 250 mg taken once daily for 12 weeks is a first-line treatment of tinea unguium, with absolute cure rates ranging from 51% to 71%.[39,40] However, it is contraindicated in patients with

renal or hepatic disease and requires routine monitoring of liver enzymes.

Other therapeutic choices include oral azoles such as itraconazole, fluconazole, and griseofulvin, but these may be less effective than terbinafine.[40] Untreated, tinea unguium may lead to cellulitis in immunocompromised individuals such as those with diabetes or on active immunosuppression. Preexisting tinea unguium in patients with diabetes increases the risk of developing diabetic foot conditions.[41]

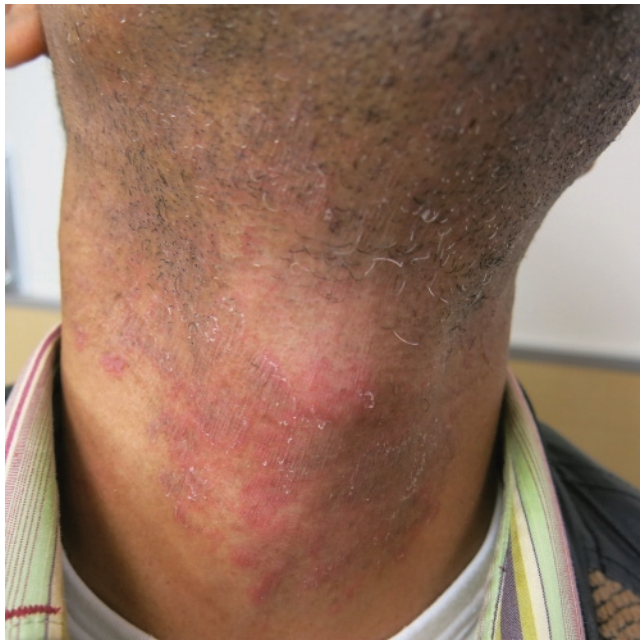
TINEA INCOGNITO

A 24-year-old woman presents with a pruritic red rash on her upper left arm (Figure 6). She has had some relief of the pruritus with a midpotency topical corticosteroid cream, but the eruption continues to spread. There is some evidence of central clearing of this eruption since the application of the steroid cream.

Cutaneous tinea infections may be misdiagnosed as eczematous reactions and treated with immunosuppressive therapies. Treatment of the offending cutaneous manifestation often results in changes from the initial presentation. The masking of the typical clinical dermatophyte infection morphology is known as tinea incognito.[41] Often, the margin of the rash will be flattened and pustular, with a loss of fine scale. The rash will also enlarge after inadequate treatment.[42]

Tinea incognito results from treatment with topical corticosteroids, topical calcineurin inhibitors, and even oral steroids for a presumed dermatologic condition.[43–45] Continued use of topical steroids despite persistence of the rash may give rise to cutaneous atrophy including thinning of the skin and the development of stretch marks. Treatment of tinea incognito centers around stopping immunosuppressive therapy, followed by a treatment course of antifungals. In many cases, topical treatment is adequate to treat tinea incognito and involves many options discussed within this review.[38] Most commonly, topical terbinafine is used for up to 6 weeks or until the rash has resolved. However, extensive or widespread tinea incognito may require oral medications including terbinafine or fluconazole.[46,47]

Figure 6.
TINEA INCOGNITO



CONCLUSIONS

Dermatophyte infections are common skin mycoses encountered in the healthcare setting. These infections can be a significant source of morbidity in affected patients because of their cosmetic appearance, discomfort, pruritus, and hair loss. Early medical intervention for these patients will help to prevent secondary complications arising from infection. Recognizing these conditions will help to choose proper therapeutic interventions at an early stage. In addition, recognizing and treating asymptomatic individuals will help to reduce the recurrence and transmission of these common cutaneous infections.

PRACTICE PEARLS

- Fungal infections of the hair, skin, and nails are very common and can lead to significant morbidity.
- Early recognition and diagnosis, including differentiating these from other conditions, are critical for appropriate treatment.
- Diagnosis is made based on clinical assessment but may require laboratory and microbiology tests in certain cases.
- Treatment is curative in most cases and will help to prevent secondary infections and reduce risk of transmission.

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