



DIAGNOSIS AND TREATMENT OF DERMATOPHYTES INFECTIONS

Dr. Simant Ankit^{1*} and Prof. Dr. Tongxiang Zeng¹

¹*Department of Dermatology & Venereology, Jingzhou Central Hospital, Yangtze University, Jingzhou, Hubei, China*

ABSTRACT

Dermatophytes are fungi that require keratin for growth. These fungi can cause superficial infections of the skin, hair, and nails. Treatment of dermatophyte infection involves primarily oral and/or topical formulations of azoles or allylamines, particularly itraconazole and terbinafine. Topical medications applied once or twice daily are the primary treatment indicated for tinea corporis/cruris, and tinea pedis/manuum. Use of oral antifungals may be practical where the tinea involvement is extensive or chronic, or where application of a topical is not feasible. For tinea unguium (onychomycosis) and tinea capitis, oral therapies are the primary treatments provided. Recently, topical amorolfine and ciclopirox formulations have been approved for use in milder onychomycosis cases, and their role in the treatment of the different clinical forms of onychomycosis is currently being defined. Relapse of infection remains a problem, particularly with tinea pedis/unguium. This article reviews will update readers on the diagnosis and treatment of common dermatophyte infections.

Keywords: Dermatophytosis, superficial fungal infections, tinea corporis, tinea cruris, tinea pedis, tinea barbae, tinea mamuum.

INTRODUCTION

Dermatophytes are referred to as “tinea” infections. Dermatophytes require keratin for growth, they are restricted to hair, nails, and superficial skin. Thus, these fungi do not infect mucosal surfaces[1]. The dryness of the skin’s outer layer discourages colonization by microorganisms, and the shedding of epidermal cells keeps many microbes from establishing residence[2]. However, the skin’s mechanisms of protection may fail because of trauma, irritation, or maceration. Furthermore, occlusion of the skin with nonporous materials can interfere with the skin’s barrier function by increasing local temperature and hydration[3] With inhibition or failure of the skin’s protective mechanisms, cutaneous infection may occur. Some dermatophytes are spread directly from one person to another (anthropophilic organisms). Others live in and are transmitted to humans from soil (geophilic organisms), and still others spread to humans from animal hosts (zoophilic organisms). Transmission of dermatophytes also can occur indirectly from fomites (e.g., upholstery, hairbrushes, hats). Anthropophilic organisms are responsible for most fungal skin infections[4]. Transmission can occur by direct contact or from exposure to desquamated cells. Direct inoculation through breaks in the skin occurs more often in persons with depressed cell-mediated immunity. Once fungi enter the skin, they germinate and invade the superficial skin layers. In patients with dermatophytoses, physical examination may reveal a characteristic pattern of inflammation, termed an “active” border. The inflammatory response is usually characterized by a greater degree of redness and scaling at the edge of the lesion or, occasionally, blister formation. Central clearing of the lesion may be present and distinguishes dermatophytoses from other papulosquamous eruptions such as psoriasis or lichen planus, in which the inflammatory response tends to be uniform over the lesion. The location of the lesions also can help identify the pathogen. A dermatophytosis can most likely be ruled out if a patient has mucosal involvement with an adjacent red, scaly skin rash. In this situation, the more probable diagnosis is a candidal infection such as perleche (if single or multiple fissures are present in the corners of the mouth) or vulvo vaginitis or balanitis (if lesions are present in the genital mucosa). They are also named for the body site involved. *Microsporum*, *Trichophyton*, and *Epidermophyton* species are the most common pathogens in skin infections. Less frequently, superficial skin infections are caused by nondermatophyte fungi (e.g., *Malassezia furfur* in tinea [pityriasis] versicolor) and *Candida* species. Differential diagnosis of tinea infections is given in the table 2.

<p>1.Potassium hydroxide (KOH) microscopy</p> <p>Value: aids in visualizing hyphae and confirming the diagnosis of dermatophyte infection.</p>
<p>2.Wood’s lamp examination (ultraviolet light)</p> <p>Value: generally, of limited usefulness, because most dermatophytes currently seen in the United States do not fluoresce; may have value in the following situations: For diagnosing a brown, scaly rash in the scrotum or axilla: erythrasma, caused by the bacterium <i>Corynebacterium minutissimum</i>, fluoresces a brilliant coral red, whereas tinea cruris or cutaneous candidal infections do not fluoresce.</p> <p>For diagnosing tinea (pityriasis) versicolor, which fluoresces pale yellow to white</p> <p>For diagnosing tinea capitis caused by two zoophilic <i>Microsporum</i> species that fluoresce blue-green.</p>
<p>3.Fungal culture</p> <p>Value: slow and expensive, but useful to confirm the diagnosis of onychomycosis when long-term oral therapy is being considered.</p>
<p>4.Skin or nail biopsy</p> <p>Value: may guide treatment decisions when the diagnosis is difficult to establish, a dermatophyte infection has not responded to previous treatment, or KOH microscopy is negative in a patient with dystrophic nails.</p>

Table1: Diagnostic Methods for Dermatophyte Infections:[5, 6]

Infection	Common causative species	Differential diagnosis
1.Tinea capitis	-Trichophyton tonsurans -Microsporum audouinii* -Zoophilic Microsporum canis*	Alopecia areata , Impetigo , Pediculosis , Psoriasis Seborrheic dermatitis , Traction alopecia Trichotillomania
2.Tinea corporis	-Trichophyton rubrum -Epidermophyton occosum	Cutaneous lupus erythematosus, Drug eruption, Eczema , Erythema multiforme, Granuloma annulare , Nummular eczematous dermatitis, Pityriasis rosea, Psoriasis , Secondary syphilis
3.Tinea cruris	-T. rubrum, -E. occosum -Trichophyton mentagrophytes	Candida lintertrigo, Contact dermatitis, Erythrasma* , Psoriasis, Seborrhea
4.Tinea manuum Tinea pedis	-T. rubrum , - T. mentagrophytes -E. occosum	Same as with tinea pedis Bacterial or candidal infection, Contact or atopic dermatitis Dyshidrosis, Eczema, Pitted keratolysis, Psoriasis.
5.Tinea unguium	-T. rubrum -T. mentagrophytes	Contact dermatitis Lichen planus Onychodystrophy Psoriasis

Table 2: Differential Diagnosis of Tinea Infections: [7, 8]

*Fluoresces under a Wood’s lamp: M. audouinii and M. canis uoresce blue-green; Malassezia furfur, the fungus that causes tinea (pityriasis) versicolor, uoresces pale yellow to white, and Corynebacterium minutissimum, the bacterium that causes ery- thrasma, uoresces bright coral red.

Tinea Capitis:

Tinea capitis, the most common dermatophytosis in children, is an infection of the scalp and hair shafts[9]. Transmission is fostered by poor hygiene and overcrowding, and can occur through contaminated hats, brushes, pillowcases, and other inanimate objects. After being shed, affected hairs can harbor viable organisms for more than one year. Tinea capitis is characterized by irregular or well-demarcated alopecia and scaling. When swollen hairs fracture a few millimeters from the scalp, “black dot” alopecia is produced[10]. Tinea scalp infection also may result in a cell-mediated immune response termed a “kerion,” which is a boggy, sterile, inflammatory scalp mass[11]. Cervical and occipital lymphadenopathy may be prominent. Today, about 90 to 95 percent of tinea scalp infections in adults and children are caused by *Trichophyton tonsurans*, which does not fluoresce. Therefore, Wood’s lamp examination has become a less useful diagnostic test for tinea capitis[12]. Tinea capitis is generally identified by the presence of branching hyphae and spores on KOH microscopy. If hyphae and spores are not visualized, Wood’s lamp examination can be performed. If KOH microscopy and Wood’s lamp examinations are negative, fungal culture may be considered when tinea capitis is strongly suspected. Alternatively, clinical features can point to the diagnosis. In one study[13], tinea capitis was confirmed by culture in 92 percent of children who had at least three of the following clinical features: scalp scaling, scalp pruritus, occipital adenopathy, and diffuse, patchy, or discrete alopecia. When scaling and inflammation are prominent, other diagnoses to consider include seborrheic dermatitis (no hair loss), atopic dermatitis (lesions in flexural folds of the neck, arms, or legs), and psoriasis (nail changes and silvery scales on the knees or elbows). When alopecia is prominent, diagnoses to rule out include alopecia areata (complete, rather than patchy, hair loss), traction alopecia (history of tight hair braiding), and trichotillomania (hairs of differing lengths and a history of obsessive hair manipulation).

Topical treatment is not effective for tinea capitis. Systemic antifungal therapy is required to penetrate the hair follicles. Griseofulvin is the only agent that the U.S. Food and Drug Administration (FDA) has labeled for the treatment of tinea capitis[14]. Although griseofulvin remains the gold standard, it is a less than ideal agent for several reasons[15], resistant organisms require dosage increases to affect a cure; treatment must be continued for six to 12 weeks; relapse rates are high because of rapid clearance of the drug from the skin with the cessation of therapy; and the liquid form for young children is a bitter-tasting solution. Compared with griseofulvin, ketoconazole is no more effective and has the potential for adverse hepatic effects and drug interactions[16]. In one study involving a small number of children, treatment with itraconazole, in a dosage of 3 to 5 mg per kg per day for four weeks, resulted in clinical and mycologic cure rates of 90 to 100 percent[17] [Evidence level B, nonrandomized clinical trial]. Fluconazole and terbinafine are promising agents; randomized, comparative studies with griseofulvin should clarify their role in the treatment of tinea capitis[18]. One randomized trial[19] in patients with tinea capitis caused by *Trichophyton* species showed that treatment with terbinafine, fluconazole, or itraconazole for two weeks was as effective as

six weeks of griseofulvin therapy.

Adjunctive topical therapy with selenium sulfide (e.g., Exsel), ketoconazole, or povidone iodine (Betadine) lotion or shampoo (applied for five minutes twice weekly) is useful to decrease shedding of viable fungi and spores[20]; over-the-counter 1 percent selenium sulfide shampoo works as well as the prescription 2.5 percent strength[21].

Tinea Corporis:

Tinea corporis or ringworm[22], typically appears as single or multiple, annular, scaly lesions with central clearing, a slightly elevated, reddened edge, and sharp margination on the trunk, extremities or face. The border of the lesion may contain pustules or follicular papules. Itching is variable. The diagnosis of tinea corporis is based on clinical appearance and KOH examination of skin [23]scrapings from the active edge. The differential diagnosis [24]includes nummular eczema, pityriasis rosea, Lyme disease, tinea versicolor, contact dermatitis, granuloma annulare, and psoriasis. Previous topical corticosteroid use can alter the appearance of the lesions, so that raised edges with central clearing are not present. Corticosteroid use may also be a factor in the development of Majocchi's granuloma, a deep follicular tinea infection that usually involves the legs and is more common in women[22]. Treatment of tinea corporis usually consists of measures to decrease excessive skin moisture and the use of topical antifungal creams[25]as shown in table 3. Rarely, widespread infections may require systemic therapy.

Tinea Barba:

Tinea barbae involves the skin and coarse hairs of the beard and mustache area[26]. This dermatophyte infection occurs in adult men and hirsute women[26]. Because the usual cause is a zoophilic organism, farm workers are most often affected. Tinea barbae may cause scaling, follicular pustules, and erythema. The differential diagnosis[27] includes bacterial folliculitis, perioral dermatitis, pseudo folliculitis barbae, contact dermatitis, and herpes simplex. One clue to the diagnosis is that hair removal is painless in tinea barbae but painful in bacterial infections[28]. Like tinea capitis, tinea barbae is treated with oral antifungal therapy as shown in table 3. Treatment is continued for two to three weeks after resolution of the skin lesions.

Tinea Faciei:

Tinea faciei tends to occur in the non- bearded area of the face. The patient may complain of itching and burning, which become worse after sunlight exposure[29]. Some round or annular red patches are present. Often, however, red areas may be indistinct, especially on darkly pigmented skin, and lesions may have little or no scaling or raised edges. Because of the subtle appearance, this dermatophytosis is sometimes

known as “tinea incognito”[30]. The differential diagnosis includes seborrheic dermatitis, rosacea, discoid lupus erythematosus, and contact dermatitis[31]. A high index of suspicion, along with a KOH microscopy of scrapings from the leading edge of the skin change, may help in establishing the diagnosis[32]. Treatment is similar to that for tinea corporis as shown in table 3.

Tinea Manuum:

Tinea manuum is a fungal infection of one or, occasionally, both hands. It often occurs in patients with tinea pedis[33]. The palmar surface is diffusely dry and hyperkeratotic. When the fingernails are involved, vesicles and scant scaling may be present, and the condition resembles dyshidrotic eczema. The differential diagnosis[34] includes contact dermatitis, psoriasis, and callus formation. Topical antifungal therapy and the application of emollients containing lactic acid are effective[35]. Relapses may be frequent if onychomycosis or tinea pedis is not resolved[35].

Tinea Cruris:

Tinea cruris, frequently called “jock itch,” is a dermatophyte infection of the groin[36]. This dermatophytosis is more common in men than in women and is frequently associated with tinea pedis[23]. Tinea cruris occurs when ambient temperature and humidity are high. Occlusion from wet or tight-fitting clothing provides an optimal environment for infection. Tinea cruris affects the proximal medial thighs and may extend to the buttocks and abdomen. The scrotum tends to be spared. Patients with this dermatophytosis frequently complain of burning and pruritus[27]. Pustules and vesicles at the active edge of the infected area, along with maceration, are present on a background of red, scaling lesions with raised borders. The feet should be evaluated as a source of the infection. Adjunctive treatment can include a low-dose corticosteroid (e.g., 2.5 percent hydrocortisone ointment for the first few days. Rarely, systemic antifungal therapy is needed for refractory tinea cruris. Patient education on avoiding prolonged exposure to moisture and keeping the affected area dry is important.

Tinea Pedis:

Tinea pedis, or athlete’s foot, has three common presentations[37]. The interdigital form of tinea pedis is most common. It is characterized by fissuring, maceration, and scaling in the interdigital spaces of the fourth and fifth toes[38]. Patients with this infection complain of itching or burning. A second form, usually caused by *Trichophyton rubrum*[39], has a moccasin-like distribution pattern in which the plantar skin becomes chronically scaly and thickened, with hyperkeratosis and erythema of the soles, heels, and sides of the feet. The vesiculobullous form of tinea pedis is characterized by the development of vesicles, pustules, and sometimes bullae in an inflammatory pattern, usually on the soles[38]. The differential diagnosis includes contact dermatitis, eczema, and pustular psoriasis[27]. Streptococcal cellulitis is a potential complication of

all three forms of tinea pedis. Streptococcal infection of normal skin is unlikely. However, the presence of fungal maceration and fissuring permits streptococci to colonize the web spaces between the toes in patients with tinea pedis. The clinical features of symptomatic athlete's foot are a result of the interaction of fungi and bacteria. Treatment of tinea pedis involves application of an antifungal cream to the web spaces and other infected areas[40]. Infrequently, systemic therapy is used for refractory infections. In several studies, twice-daily application of the allylamine terbinafine resulted in a higher cure rate than twice-daily application of the imidazole clotrimazole (Lotrimin; 97 percent versus 84 percent), and at a quicker rate (one week for terbinafine versus four weeks for clotrimazole)[41]. A pharmacoeconomic analysis of tinea treatments found topical terbinafine to be more cost-effective than imidazole or ciclopirox cream. When marked inflammation and vesicle formation occur and signs of early cellulitis are present, the addition of a systemic or topical antibiotic with streptococcal coverage is warranted. Reinfection is common, especially if onychomycosis is present[6]. Nail infections should be treated. In addition, footwear should be disinfected, and patients with tinea pedis should avoid walking barefoot in public areas such as locker rooms. Other measures to reduce recurrence include controlling hyperhidrosis with powders and wearing absorbent socks and nonocclusive shoes[42].

Tinea Unguium:

Tinea unguium, a dermatophyte infection of the nail, is a subset of onychomycosis, which also may be caused by yeast and non-dermatophyte molds[43]. Risk factors for this infection include aging, diabetes, poorly fitting shoes, and the presence of tinea pedis. Onychomycosis accounts for about 40 to 50 percent of nail dystrophies[44]. The differential diagnosis includes trauma, lichen planus, psoriasis, nail-bed tumor, peripheral vascular disease, atopic dermatitis, contact dermatitis, and yellow nail syndrome. Because onychomycosis requires expensive, prolonged therapy (three to four months for fingernail infections and four to six months for toenail infections), the diagnosis should be confirmed before treatment is initiated[7]. Periodic acid-Schiff staining with histologic examination of the clipped, distal free edge of the nail and attached subungual debris is the most sensitive diagnostic method and is painless for patients[45]. Tinea unguium, especially of the toenails, is difficult to eradicate. Topical agents have low efficacy. Mycologic cure rates for ciclopirox nail lacquer, applied daily for up to 48 weeks, have ranged from 29 to 47 percent[45]. Oral treatment with griseofulvin must be continued for 12 to 24 months, and ketoconazole carries a risk of hepatotoxicity. Fluconazole has not been studied extensively in the treatment of onychomycosis and is not labeled by the FDA for this indication. Mycologic and clinical cure rates are similar for 12 weeks of treatment with itraconazole in a dosage of 200 mg per day and terbinafine in a dosage of 250 mg per day as shown in table 3[40]. Itraconazole costs more for the same regimen. Continuous terbinafine therapy has a better mycologic cure rate than intermittent or "pulse" terbinafine therapy, in which 500 mg of terbinafine is given once daily for seven days of each of four months (94 percent versus 80 percent); however, continuous treatment is more expensive. Intermittent itraconazole therapy, in a dosage of 400 mg per day for seven days

of each of four months, and intermittent terbinafine therapy are similarly effective.

Newer Topical Antifungals:

Luliconazole, an azole antifungal has fungicidal action against Trichophyton species similar to or more than that of terbinafine. Available in 1% cream formulation, it is effective as once daily application for 1–2 weeks for dermatophytic infection. Approved by the US Food and Drug Administration for the treatment of interdigital tinea pedis, tinea cruris, and tinea corporis, it has a favorable safety profile[46]. Econazole nitrate foam preparation has also shown its efficacy over foam vehicle for tinea pedis[47]. However, these newer drugs are costlier which in turn may lead to issues of adherence to treatment in resource-poor settings, and may predispose to development of resistance.

Newer Oral Antifungal Agents:

There is lack of any recent literature regarding systemic antifungals in the treatment of tinea cruris and corporis. Although few newer systemic antifungals have been approved in last two decades but most of them are reserved for more severe life-threatening invasive systemic mycoses with paucity of evidence on efficacy in superficial mycoses. Recently, posaconazole was found to be effective in a patient with extensive dermatophytic skin and nail infection with underlying CARD9[48].

New and Potential Therapies:

Other than the antifungals already mentioned, few plant extract (Chinese herbals) are also found to be effective against common dermatophytic infection. One of them is macrocarpal C, an active ingredient obtained from the fresh leaves of Eucalyptus globulus Labill with antifungal action against T. mentagrophytes and T. rubrum[49]. Demicidin, an antimicrobial peptide has antifungal action at a concentration normally present in sweat providing an insight to newer therapeutic target for dermatophytic infection[50].

Drug	Doses
1.Tinea unguium -Terbinafine -Itraconazole	250 mg orally every day for 12 weeks† <i>or</i> 500 mg orally every day during the rst week of each month for four month 200 mg orally every day for 12 weeks 400 mg orally every day during the rst week of each month for four months
2.Tinea capitis, barbae -Griseofulvin, micronized (Grifulvin) -Terbinafine	20 mg per kg per day for eight weeks 62.5 mg per day for four weeks
3.Tinea corporis, pedis, cruris, faciei -Butenafine (Mentax) -Terbinafine -Miconazole (Micatin) -Clotrimazole (Lotrimin AF)	Applied to the lesion and a 2-cm area surrounding the lesion once daily for approximately 14 days Applied to the lesion and a 2-cm area surrounding the lesion twice daily for approximately 14 days Applied to the lesion and a 2-cm area surrounding the lesion twice daily for approximately 14 days Applied to the lesion and a 2-cm area surrounding the lesion twice daily for approximately 14 days

Table 3: Antifungal Agent for Treatment for Tinea Infestions [13, 23, 27]

CONCLUSION

Dermatophytes are fungi that invade and multiply within keratinized tissues (skin, hair, and nails) causing infection. Based upon their genera, dermatophytes can be classified into three groups: Trichophyton (which causes infections on skin, hair, and nails), epidermophyton (which causes

infections on skin and nails), and *Microsporum* (which causes infections on skin and hair). Based upon mode of transmission, these have been classified as anthropophilic, zoophilic, and geophilic. Finally, based upon the affected site, these have been classified clinically into tinea capitis (head), tinea faciei (face), tinea barbae (beard), tinea corporis (body), tinea manus (hand), tinea cruris (groin), tinea pedis (foot), and tinea unguium (nail). Dermatophyte infections can be readily diagnosed based on the history, physical examination, and potassium hydroxide (KOH) microscopy. Diagnosis occasionally requires Wood's lamp examination and fungal culture or histologic examination. Topical therapy is used for most dermatophyte infections. Cure rates are higher and treatment courses are shorter with topical fungicidal allylamines than with fungistatic azoles. Oral therapy is preferred for tinea capitis, tinea barbae, and onychomycosis. Orally administered griseofulvin remains the standard treatment for tinea capitis. Topical treatment of onychomycosis with ciclopirox nail lacquer has a low cure rate. For onychomycosis, "pulse" oral therapy with the newer imidazoles (itraconazole or fluconazole) or allylamines (terbinafine) is considerably less expensive. Although there is sufficient evidence to demonstrate the efficacy of topical antifungals in limited disease yet, there is scarce data on the frequency of relapse once topical monotherapy is discontinued and need more research in the future for more improvement. Appropriate follow-up duration and education of patients on proper foot hygiene are also important components in providing effective therapy.

Conflict Of Interest:

There are no conflicts of interest.

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