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REVIEW

Tinea pedis: an updated review

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Abstract

Background: Tinea pedis is one of the most common superficial fungal infections of the skin, with various clinical manifestations. This review aims to familiarize physicians with the clinical features, diagnosis and management of tinea pedis.

Methods: A search was conducted in April 2023 in PubMed Clinical Queries using the key terms 'tinea pedis' OR 'athlete's foot'. The search strategy included all clinical trials, observational studies and reviews published in English within the past 10 years.

Results: Tinea pedis is most often caused by *Trichophyton rubrum* and *Trichophyton interdigitale*. It is estimated that approximately 3% of the world population have tinea pedis. The prevalence is higher in adolescents and adults than in children. The peak age incidence is between 16 and 45 years of age. Tinea pedis is more common amongst males than females. Transmission amongst family members is the most common route, and transmission can also occur through indirect contact with contaminated belongings of the affected patient. Three main clinical forms of tinea pedis are recognized: interdigital, hyperkeratotic (moccasin-type) and vesiculobullous (inflammatory). The accuracy of clinical diagnosis of tinea pedis is low. A KOH wet-mount examination of skin scrapings of the active border of the lesion is recommended as a point-of-care testing. The diagnosis can be confirmed, if necessary, by fungal culture or culture-independent molecular tools of skin scrapings. Superficial or localized tinea pedis usually responds to topical antifungal therapy. Oral antifungal therapy should be reserved for severe disease, failed topical antifungal therapy, concomitant presence of onychomycosis or in immunocompromised patients.

Conclusion: Topical antifungal therapy (once to twice daily for 1–6 weeks) is the mainstay of treatment for superficial or localized tinea pedis. Examples of topical antifungal agents include allylamines (e.g. terbinafine), azoles (e.g. ketoconazole), benzylamine, ciclopirox, tolnaftate and amorolfine. Oral antifungal agents used for the treatment of tinea pedis include terbinafine, itraconazole and fluconazole. Combined therapy with topical and oral antifungals may increase the cure rate. The prognosis is good with appropriate antifungal treatment. Untreated, the lesions may persist and progress.

Keywords: athlete's foot, dermatophytosis, interdigital, moccasin, *Trichophyton interdigitale*, *Trichophyton rubrum*, vesiculobullous.

Citation

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Introduction

Tinea pedis (commonly known as 'athlete's foot') refers to a superficial fungal infection of the skin on the foot caused predominantly by dermatophytes. Worldwide, tinea pedis is one of the most common, if not the most common, superficial fungal infections of the skin after puberty and its prevalence is increasing.[1–9] The condition was first described by Pellizzari in 1888.[10] Tinea pedis is a public health concern because it is contagious and recurrent and serves as an important reservoir for dermatophytosis elsewhere in the body.

Methods

A search was conducted in April 2023 in PubMed Clinical Queries using the key terms "tinea pedis" OR "athlete's foot". The search strategy included all clinical trials (including open trials, non-randomized controlled trials and randomized controlled trials), observational studies (including case reports and case series) and reviews (including narrative reviews, clinical guidelines and meta-analyses) published within the past 10 years. Google, UpToDate and Wikipedia were also searched to enrich the review. Only papers published in English were included in this review. The information retrieved from the search was used in the compilation of the present article.

Review

Aetiology

The most common etiological agents are *Trichophyton rubrum* and *Trichophyton interdigitale*.[11–18] The predominating dermatophytes may vary with geographic locations (related to climate characteristics and social factors) and change over time.[19] Other less common causative dermatophytes include *Epidermophyton floccosum*, *Trichophyton tonsurans*, *Trichophyton soudanense*, *Trichophyton violaceum* and *Microsporum audouinii*.[20,21] Non-dermatophyte moulds, such as *Neoscytalidium hyalinum*, *Neoscytalidium dimidiatum*, *Scopulariopsis brevicaulis* and *Fusarium* species, are uncommon causes of tinea pedis.[14,20,22] Rarely, *Cylindrocarpon lichenicola* and yeasts primarily *Candida* species may also cause tinea pedis.[14,20,22]

Epidemiology

It is estimated that the global prevalence of tinea pedis is approximately 3%.[2,23] The lifetime risk is up to 70%.[2,23] The prevalence is higher in adolescents and adults than in prepubertal children.[24,25] In the past decades, there has been an increase in prevalence due, at least in part, to an increase in recreational activities and growing urbanization.[26] The peak age of incidence is between 16 and 45 years, when working and leisure activities are at a maximum.[27] The condition is more common in industrial countries.[28,29] The male to female ratio is approximately 3:1.[30,31] Tinea pedis affects individuals of all races. In the United States, the condition is more common amongst Blacks and Hispanics.[32] Humans may become infected through close contact with infected persons, animals (in particular, house pets), contaminated fomites or soil.[25] Transmission amongst family members is the most common route; children often become infected by direct contact with spores of the causative organism or infected skin fragments shed by household contact.[25] On the other hand, transmission can also occur through indirect contact with contaminated belongings (e.g. shoes, socks, bedding) of an affected individual.[33,34] Autoinfection by dermatophytes elsewhere in the body may also occur.[25] The transmission of tinea pedis is facilitated by prolonged wearing of occlusive footwear (promoting a moist, warm local environment and a high dew point of footwear).[11,13,30,31] The condition is more prevalent amongst athletes (especially common amongst elite athletes who walk barefoot in swimming pool facilities or locker rooms), military personnel, manual laborers, individuals in long-term care facilities and homeless individuals.[35–45] In one study of the 169 employees at 21 swimming pools in the Netanya area, Israel, 78 (46%) of the employees had concurrent onychomycosis and tinea pedis and 50 (30%) had tinea pedis only; the diagnosis was based on KOH microscopy and fungal culture.[46] Past history of tinea pedis, concurrent tinea pedis amongst family members, hot humid climates, hyperhidrosis (especially plantar hyperhidrosis), prolonged exposure of the feet to water, communal bathing/sharing washing facilities, use of public swimming pools, insufficient foot care, poor personal hygiene, maceration or breaks in the pedal skin, diabetes mellitus, peripheral vascular disease, atopic dermatitis, psoriasis, obesity, immunodeficiency, depression, schizophrenia, and genetic predisposition or susceptibility are other predisposing factors.[47–58]

Pathophysiology

The causative fungus produces and releases enzymes, such as proteases, that digest keratin and keratinase that penetrates keratinized tissue.[59] The hyphae then invade the stratum corneum and keratin and spread centrifugally outward. Infection is usually cutaneous and limited to the non-living cornified layers because the fungus is not able to penetrate the deeper tissue of a healthy immunocompetent host.[59] Scaling results from increased epidermal replacement following inflammation. The wall of the fungus also contains mannans, which suppress the body's immune system, decrease lymphoproliferative response and reduce the proliferation of keratinocytes. The latter results in a decreased rate of sloughing of the affected skin and prolongs the infection. Sweating and warmth promote growth of the fungus.

Histopathology

Histological findings include hyperkeratosis, acanthosis and presence of neutrophils in the dermis, hyphae between the lower layer of the parakeratotic stratum corneum and the upper normal basket-weave stratum corneum ('sandwich sign').[2] Hyphae may be more readily seen on haematoxylin and eosin staining.[60]

Clinical manifestations

Three main clinical forms of tinea pedis are recognized: interdigital tinea pedis, hyperkeratotic (moccasin-type) tinea pedis and vesiculobullous (inflammatory) tinea pedis.[6,35,43] Interdigital tinea pedis, the most common form (especially in children), presents with erythema, silvery white scaling, peeling and maceration in the web spaces, typically in the web space between the fourth and fifth toes (most common) (Figures 1 and 2).[61–63] The web space may become white and soggy.[64] Presumably, the narrow interdigital spaces in children's feet may predispose them to this form of tinea pedis.[65] Pruritus is the most common symptom.[64,66] Often, there is peripheral fissuring, which may cause pain and burning sensation.[2,19] Adjacent areas such as the sole may also be affected (Figure 2). Secondary bacterial infections with gram-positive bacteria (*Staphylococcus aureus*, streptococci) and gram-negative bacteria (*Escherichia coli*, *Klebsiella* species, *Proteus* species, *Pseudomonas aeruginosa*) in the interdigital areas can result in foul odour, maceration, erosions and crusting – a condition referred to as dermatophytosis complex.[67]

Hyperkeratotic (moccasin-type) tinea pedis, the second most common form of tinea pedis, is characterized by chronic, hyperkeratotic, scaling plaques with varying degrees of underlying erythema on the heels, soles, lateral and medial sides and back of the foot, and distal dorsum of the foot (corresponding to the skin covered by this type of footwear) (Figure 3).[6,19,68] The dorsum of the foot, except the distal portion, is typically spared.[4,66] Erythema is more prominent on the distal dorsum of one or both feet.[19] A collarette of scale can be seen along the border of the feet in a moccasin-type distribution.[26] The leading edge of the eruption may be arcuate, annular and slightly elevated.[69] Occasionally, fissures may develop. Hyperkeratotic tinea pedis can be slightly pruritic but most often is asymptomatic.[35,43] The condition is usually chronic and quite resistant to treatment.[35,43]

Vesiculobullous (inflammatory) tinea pedis (often acquired from animals) typically presents with intensely pruritic (sometimes painful) vesicles and/or bullae on a background of erythema (Figures 4 and 5). Vesicles typically range from 1 to 3 mm in diameter. Coalescence of vesicles leads to the formation of bulla.[2] Sites of predilection include the instep or medial plantar surface of the foot.[68,70] The lesions develop far more rapidly than the other forms of tinea pedis.[26] Rupture of the vesicles and/or bullae releases serous or purulent fluid with exposure of a red, raw and oozing surface.[2,71]

Other clinical variants include occult tinea pedis, ulcerative tinea pedis and tinea incognito. Occult tinea pedis

Figure 1. Interdigital tinea pedis presenting with maceration, desquamation and fissuring between the toes.

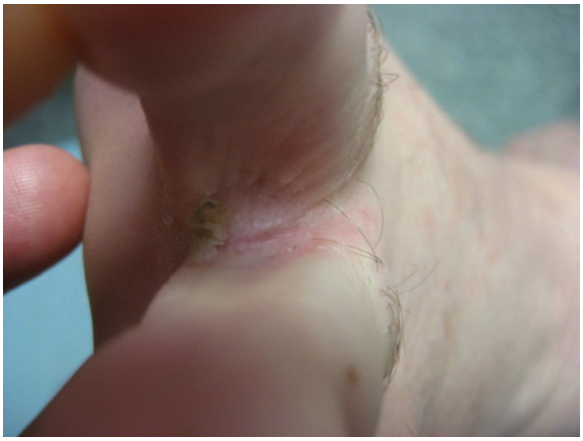
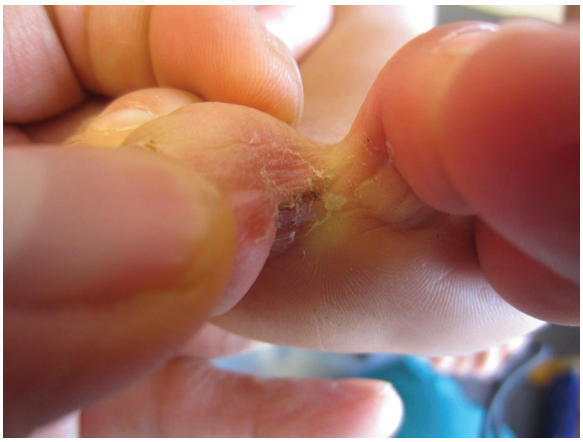


Figure 2. Interdigital tinea pedis presenting with erythema and scaling between the toes and the adjacent skin.



is common, particularly amongst elite athletes.[72,73] In one study of 150 regular swimmers, 22 (15%) swimmers had positive cultures for dermatophytes from toe-web samples;[72] 8 (36%) of these patients had no lesions.[72] In another study of 405 marathon runners, scrapings were taken from the fourth toe-web space of both feet and sent for fungal culture.[73] Cultures for dermatophytes were positive in 89 (22%) marathon runners, 43 (10.6%) of whom had no lesions. Individuals with occult tinea pedis are asymptomatic and they are often unaware of tinea pedis.[2] The diagnosis of occult tinea pedis is easily missed unless it is specifically sought. The majority of occult tinea pedis cases are caused by *Trichophyton interdigitale*.[2,72]

Ulcerative tinea pedis is rare and typically presents with rapidly spreading interdigital erosions and ulcers that

are more severe than those seen in vesiculobullous tinea pedis.[26,66] The condition is more commonly seen in patients with peripheral vascular disease, diabetes mellitus or immunodeficiency.[74] Ulcerative tinea pedis is often complicated by secondary bacterial infection, which can be severe and debilitating.[26]

Tinea incognito (also referred to as tinea atypica) (Figure 6) may result if tinea pedis is mistakenly treated with immunosuppressive agents, notably calcineurin inhibitors or corticosteroids, leading to loss of typical appearance of the lesion. Pruritus may be minimal or absent.[75,76] The active margin of the lesion may be lost, erythema and scale may be attenuated, and papules and pustules may be present.[4]

Very rarely, tinea pedis presents as scattered, asymptomatic, purpuric papules on the plantar surface of the foot in the absence of pruritus, erythema, maceration, desquamation or vesicles/bullae.[77] Physicians should be aware of the rare variant so that appropriate treatment can be initiated. Untreated tinea pedis poses a public health hazard as the condition is contagious.

A concomitant secondary eruption, usually in the form of symmetric pruritic vesicles or papules, may occur at distant sites, such as the palmar aspect of the hands and sides of the fingers (referred to as a dermatophytid reaction), presumably due to an immunological reaction to the fungus.[78,79] It is estimated 7–17% of patients with tinea pedis develop a dermatophytid reaction;[2,79] the eruption does not contain fungal elements and eradication of tinea pedis results in a cure of the eruption.[66]

Diagnosis

Many physicians make the diagnosis of tinea pedis clinically. However, the accuracy of clinical diagnosis of tinea pedis is low. In one study, the specificities and sensitivities of clinical diagnosis were 0.94 and 0.47 for tinea pedis in the plantar area, respectively, and 0.95 and 0.37, respectively, for tinea pedis in the interdigital area.[80]

Reflectance confocal microscopy, a non-invasive, real-time imaging of the superficial layers of the skin, can be used as a tool for the diagnosis of tinea pedis, though this is seldom performed at present.[81] With tinea pedis, reflectance confocal microscopy typically shows branching hyphae and inflammatory infiltrate in the stratum corneum.[81]

To avoid misdiagnosis, especially in the setting of tinea incognito, a KOH wet-mount examination of skin scrapings of the active border of the lesion or the roof of a vesicle taken with a sterile instrument can be performed.[82,83] A drop of 10–20% solution of KOH, with or without dimethyl sulfoxide, is added to the scrapings on a microscopic slide. The specimen is then gently heated to accelerate

Figure 3. Diffuse scale and erythema on the sole and medial aspect of the foot in a patient with hyperkeratotic (moccasin-type) tinea pedis.

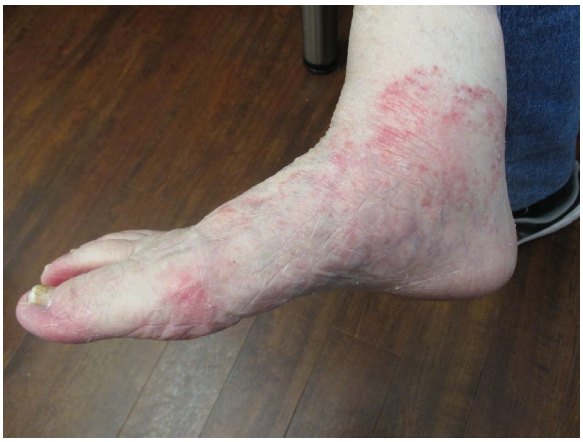


Figure 4. Vesiculobullous (inflammatory) tinea pedis presenting with pruritic vesicles and bullae over a background of erythema on the dorsal surface of the right foot.

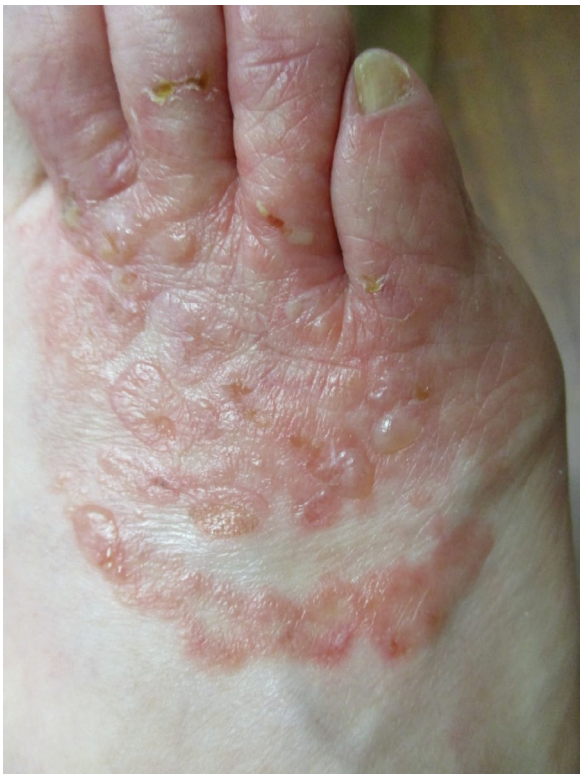


Figure 5. Vesiculobullous (inflammatory) tinea pedis presenting with pruritic vesicles and bullae over a background of erythema on the medial plantar surface of the left foot.

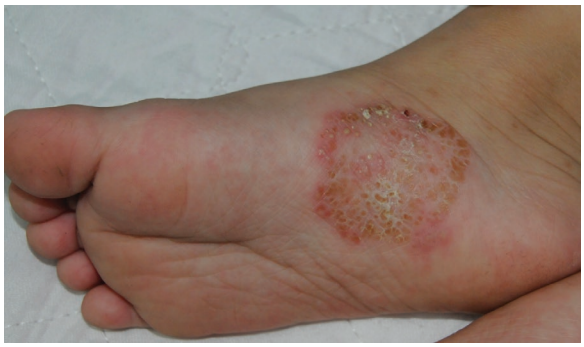
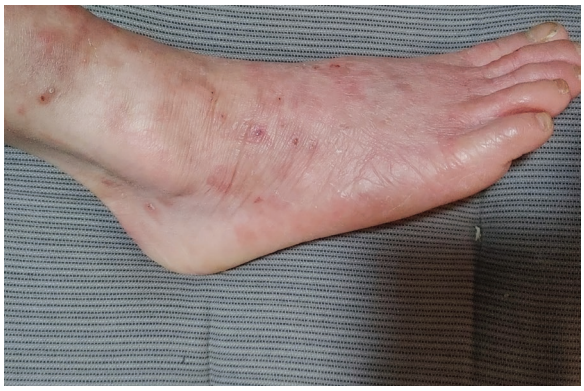


Figure 6. Tinea incognito in a patient with tinea pedis treated with topical corticosteroid resulting in loss of typical appearance of the lesion.



the destruction of the squamous cells. KOH dissolves the epithelial tissue, leaving behind easily visualized dichotomously branched, septate hyphae and spores. The addition of 20–40% of dimethyl sulfoxide to the KOH preparation allows rapid examination of the scrapings even without heating. Direct microscopic examination of a KOH wet-mount preparation of skin scrapings of the active border of the lesion or the roof of a vesicle, however, cannot distinguish pathogenic species of dermatophytes and whether the dermatophyte is alive. On the other hand, the procedure is easily performed, the cost is low and the result is readily available (usually less than 2 hours). A pooled analysis of data from 460 patients showed that the specificity and sensitivity of the test was 42.5% (95% CI 36.6–48.6) and 73.3% (95% CI 66.3–79.5), respectively.[84] Some authors suggest repetition of the test if the initial result is negative, and the clinical picture is suggestive of tinea pedis.[85]

Although fungal culture is the gold standard to diagnosing dermatophytosis and the pathogenic species, culture is rarely needed, unless the diagnosis is still in doubt after KOH wet-mount examination of skin scrapings of the active border of the lesion or roof of a vesicle, or the infection is severe, widespread or resistant to antifungal treatment. Culture is expensive and it takes 7–14 days for results to be available. The most common culture medium is Sabouraud dextrose agar, which contains 10 g/L peptone, 40 g/L dextrose and 15–20 g/L agar, at pH 5.6. As contamination or coinfection with Gram-negative bacteria may result in decreased sensitivity of cultures, gentamycin or chloramphenicol (0.005%) is added to reduce bacterial contamination.[35,60] Cycloheximide should not be added to the culture medium because it inhibits growth of non-dermatophyte moulds, which may be a cause of tinea pedis. A skin biopsy for histopathology is rarely necessary but, when performed, may show fungal elements and neutrophils within the stratum corneum.

More recently, a DNA-chip assay of the clinical specimen has been developed that allows rapid diagnosis of tinea pedis.[86] In the DNA-chip assay, fungal DNA is amplified by using multiplex polymerase chain reaction with subsequent determination by hybridization on a microarray chip.[86] The DNA-chip assay has a high sensitivity (90.7%).[86] The sensitivity can be further increased to 96.3% by factoring results from microscopic examination of KOH wet-mount preparation or direct immunofluorescence microscopy.[86] However, the DNA-chip assay is expensive and unavailable in most physicians' offices or laboratories.

Differential diagnosis

Tinea pedis is very common and mimics many other plantar dermatoses. The differential diagnosis includes intertrigo from *Candida* or bacterial infection, atopic dermatitis, xerosis, irritant contact dermatitis, allergic contact dermatitis, interdigital erythrasma, impetigo, friction blisters, pemphigus, cellulitis, herpes simplex, acute palmoplantar (dyshidrotic) eczema, scabies, pitted keratolysis, juvenile plantar dermatosis, palmoplantar psoriasis, palmoplantar pustulosis, palmoplantar keratoderma, keratolysis exfoliative (lamellar dyshidrosis sicca), mycosis fungoides and pityriasis rubra pilaris.[87–95]

Complications

Bacterial superinfection such as cellulitis (most common), pyoderma and lymphangitis may complicate tinea pedis as this may provide a portal of entry for bacteria, especially in patients with vesiculobullous tinea pedis and ulcerative tinea pedis.[96–99] The fungus can spread to other parts

of the body such as the nails (onychomycosis), groin (tinea cruris), hands (tinea manuum), bearded areas (tinea barbae) and face (tinea faciei).[100] The fungus can invade the hair follicle, leading to the formation of perifollicular granulomatous inflammation (Majocchi granuloma).[12] Invasion of subcutaneous tissue results in subcutaneous dermatophytosis.[101]

It is estimated that approximately 84% of patients with tinea manuum have concomitant tinea pedis.[102] Tinea manuum is often unilateral, whereas coexisting tinea pedis is bilateral (referred to as two feet-one hand syndrome).[102–105] Two feet-one hand syndrome likely results from contact (usually scratching) of the feet with the hand.[102–105] In a study of 80 patients (72 men and 8 women) with mycologically confirmed two feet-one hand syndrome, tinea was found to begin in the feet.[106] Occasionally, tinea manuum and concurrent tinea pedis can be bilateral (referred to as two feet-two hands syndrome).[107]

Concurrent tinea pedis with onychomycosis is a frequent phenomenon.[108–113] Untreated or inadequately treated tinea pedis is a predisposing factor for the development of onychomycosis. In a 2022 systematic review of 44 articles including 2382 children with onychomycosis, 527 (22%) children had concurrent tinea pedis.[114] Whilst onychomycosis can serve as a reservoir for the dermatophyte causing tinea pedis, the dermatophyte causing tinea pedis can also spread to the nail, resulting in onychomycosis.[115]

Treatment

General measures

Because fungi thrive best in moist warm environments, patients should be advised to wear non-occlusive, clean, natural fibre or cotton socks and shoes (ideally, sandals) and dry their feet thoroughly after bathing.[14] Prolonged use of occlusive footwear should be avoided. The importance of proper washing of the feet, good personal hygiene, avoidance of footwear sharing and proper disinfection of footwear of affected patients cannot be over-emphasized.[66] In one study, *Trichophyton* species was detected from the footwear of 47% of patients with tinea pedis.[116] If necessary, footwear of affected individuals can be sterilized by ultraviolet-C (UVC) sanitizing devices and ozone gas.[117,118] Some authors suggest disinfection of socks by soaking in quaternary ammonium compounds.[119] Hot washing of socks or running shoes can also kill fungus. Application of desiccating, antiseptic powders to the feet (particularly between toes) as well as the footwear may help.[14,64] Hyperhidrosis of the feet should be treated appropriately.

Topical antifungal therapy

Topical antifungal therapy is the mainstay of treatment for superficial or localized tinea pedis. Topical antifungal agents are generally applied once to twice daily for 1–6 weeks (usually 2–4 weeks) depending on the severity of the lesion, the type of medication used and the response of the lesions to treatment.[2,21] A variety of topical antifungal agents are available for the treatment of tinea pedis, including azoles (e.g. econazole, ketoconazole, sertaconazole, efinaconazole, miconazole, clotrimazole, luliconazole, isoconazole, oxiconazole, sulconazole, tioconazole), allylamines (e.g. naftifine, terbinafine), benzylamine (e.g. butenafine), ciclopirox, tolnaftate and amorolfine.[120–140] It has been shown that patients with interdigital tinea pedis usually respond to 1 week of treatment with topical terbinafine whereas those with hyperkeratotic tinea pedis may require treatment for 4 weeks.[2] In a mixed-treatment comparison (head-to-head trials and trials with a common comparator) meta-analysis involving 14 topical antifungal treatments, there was no significant difference in the efficacy amongst the antifungal agents.[141] A 2022 systematic review of seven randomized controlled trials (n=680) showed that the likelihood of successful treatment of tinea pedis with topical terbinafine and topical butenafine was 3.9 (95% CI 2.0–7.8) and 5.3 (95% CI 1.4–19.6) times that of patients treated with a placebo, respectively.[142] Topical terbinafine and topical butenafine had similar efficacy.[142] Topical nystatin is not effective for the treatment of tinea pedis and is therefore not recommended.[68] As topical ciclopirox has antidermatophytic, anticandidal and antibacterial activities, the medication is particularly effective for the treatment of dermatophytosis complex.[2,143] Other topical antifungal agents that have antibacterial activity include miconazole, sulconazole and naftifine.[144] Topical antifungal agents are well tolerated.[140] Side-effects are uncommon and consist mainly of pruritus, stinging and burning sensation at the site of applications.[140,145] Most of the relapses are a result of poor compliance, which is more commonly seen in patients/parents who are working long hours or individuals taking multiple medications; these individuals tend to discontinue the medication prematurely if there is no response (not enough time for the medication to work) or with partial cure.[146] In this regard, topical antifungals, such as sertaconazole, terbinafine and econazole, which can be used once daily, may help to improve compliance.[35,43] Terbinafine 1% cream might be the best strategy for maintaining remission, especially for the treatment of interdigital tinea pedis.[56,141]

Resistance to terbinafine therapy may be due to infection with non-dermatophyte moulds or infection with a dermatophyte that has acquired resistance to terbinafine.[19] Acquired resistance to terbinafine may be due to a missense mutation in the squalene epoxidase-encoding gene.[19,147] Squalene epoxidase is an enzyme involved in ergosterol synthesis, which is a target for terbinafine.[19,148]

Use of a special carrier system where topical antifungal agents are attached to carriers (e.g. noisomes, liposomes,

ethosomes, nanostructured lipid) to enhance their ability to penetrate the stratum corneum and to increase their bioavailability opens the door for future research in this area.[149,150] New formulations of topical antifungal agents that may enhance the efficacy and, potentially, compliance include BB2603 (a nano-formulation of terbinafine with the polymer polyhexamethylene biguanide to enhance solubility and drug delivery to the skin)[123] and a miconazole–urea combination (claimed to potentiate the spectrum of antifungal activity whilst reducing the chance of drug resistance).[151]

Systemic antifungal therapy

Systemic treatment should be considered if the condition is extensive, recurrent, chronic or resistant to topical antifungal treatment, if the patient is immunocompromised, or if there is evidence of concomitant onychomycosis.[61,83] Oral antifungal agents used for the treatment of tinea pedis include terbinafine (62.5 mg/day for body weight 10–20 kg, 125 mg/day for body weight >20 kg and <40 kg, and 250 mg/day for body weight ≥40 kg for 2 weeks), itraconazole (3–5 mg/kg/day divided into two doses for 1 week; maximum 200 mg twice daily), and fluconazole (6 mg/kg once weekly; maximum 150 mg once weekly for 2–6 weeks).[25,68] These oral antifungal agents and their adverse events are listed in Table 1.[25,68,103,144,152–155] Oral ketoconazole has fallen out of favour and is rarely used now because of adverse events, notably hepatotoxicity.[144,156] Other adverse events include neurotoxicity, renal toxicity, pancytopenia, suppression of testosterone synthesis, gynecomastia, drug–drug interactions, gastrointestinal upset, urticaria, rhabdomyolysis, headache, asthenia, fatigue and suicidal ideation.[156] The use of oral ketoconazole for the treatment of tinea pedis has been replaced by other oral antifungal agents that have greater efficacy and less adverse events.

In a meta-analysis of 15 randomized controlled trials (n=1438) comparing the efficacy of various oral antifungals, no significant difference in efficacy was detected between terbinafine and itraconazole, fluconazole and itraconazole, and fluconazole and ketoconazole in the treatment of tinea pedis.[25] A 2022 systematic review showed that oral terbinafine was more efficacious than oral itraconazole in the treatment of tinea pedis with a risk ratio of 1.3 (95% CI 1.1–1.5).[142] Oral terbinafine was found to be more effective than oral griseofulvin with a risk ratio of 2.26 (95% CI 1.49–3.44).[25] Additionally, oral griseofulvin has more adverse events (photosensitivity, fixed drug eruption, gastrointestinal upset, hepatic dysfunction) than other oral antifungal agents as well as its poor and highly variable bioavailability and contraindication for use in pregnancy; therefore, the medication is rarely used nowadays.[157–160] Combined therapy with topical and oral antifungals likely increases the cure rate.

Table 1. Oral antifungal agents for the treatment of tinea pedis and side-effects.

Antifungal agent	Dosage	Duration of treatment	Adverse events
Terbinafine	62.5 mg/day for body weight 10–20 kg; 125 mg/day for body weight >20 kg but <40 kg; 250 mg/day for body weight ≥40 kg	2 weeks	Headache, difficulty to concentrate, dysgeusia, ageusia, anorexia, nausea, vomiting, abdominal cramps, diarrhoea, pruritus, drug eruption, visual disturbance, drug–drug interactions (uncommon), mild transaminitis, fulminant hepatic failure (rare), neutropenia (rare), depression (rare), Steven–Johnson syndrome (rare)
Itraconazole	3–5 mg/kg/day divided into two doses; maximum: 200 mg bid	1 week	Drug–drug interactions (common), pruritus, pyrexia, peripheral oedema, hypertension, hypokalaemia, dermatitis, hepatic dysfunction, headache, dizziness, nausea, vomiting, abdominal pain, diarrhoea, dyspnoea, cardiac failure (rare), thrombocytopenia (rare)
Fluconazole	6 mg/kg once weekly; maximum 150 mg once weekly	2–6 weeks	Drug–drug interactions (common), pyrexia, nausea, vomiting, diarrhoea, headache, pruritus, transaminitis, drug eruption, serum sickness-like reaction (uncommon), Steven–Johnson syndrome (rare), prolongation of QT-interval (rare), Torsades de pointes (rare)

Alternative and complementary therapies

In some cultures, complementary and alternative therapies are popular for the treatment of tinea pedis. A wide variety of products from medicinal plants have been shown to have antifungal activity and some therapeutic effects on tinea pedis. These include red onion (*Allium cepa L.*) extract, Griseococcin extracted from *Bovistella radicata* (a species of puffball mushroom), extract from the root of *Impatiens tinctoria A.* (an herbal plant), extract from the leaves of *Cestrum schlechtendalii* (a plant in the Solanaceae family), extract from the leaves and twigs of *Isodan flavidus* (a Miao medicinal plant), extract from the leaves of *Eucalyptus globulus labill* (a species of flowering plant in the family Myrtaceae), extract from *Coptis* (a flowering plant in the family of Ranunculaceae), soleshine (a polyherbal preparation containing extracts of resin of Sal tree, leaves of neem, henna, sesame oil and castor oil), and essential oils extracted from *Lavandula luisieri* (a Portuguese lavender) and *Cymbopogon citratus* (lemon grass).[161–169] These products have not been subjected to rigorous studies or randomized clinical trials. Well-designed, large-scale, multicentre, randomized, placebo-controlled trials are needed before the use of these products can be routinely recommended.

Antibiotic use should be considered for secondary bacterial infection. Interdigital tinea pedis may benefit from placing a separator between the affected toes to ensure better aeration and dryness, in turn increasing the efficacy of antifungal therapy.[170] Hyperkeratotic tinea pedis may benefit from combining antifungal treatment with a topical keratolytic such as salicylic acid, lactic acid and urea.[83,151,171–173] Topical keratolytics reduce the thickness of the horny layer, thereby facilitating absorption of topical antifungal agents.[173]

Prognosis

The prognosis is good with appropriate antifungal treatment. Untreated, the lesions of tinea pedis may persist and progress.[68] Underlying conditions such as immunodeficiency, hyperhidrosis and diabetes mellitus may have an adverse effect on the prognosis.

Conclusion

Tinea pedis is one of the most common superficial fungal infections of the skin worldwide. The most common aetiological agents are *T. rubrum* and *T. interdigitale*. The accuracy of clinical diagnosis of tinea pedis is low. KOH wet-mount examination of skin scrapings of the active border of the lesion is recommended as a point-of-care testing to identify fungal hyphae and spores before antifungal treatment is initiated. The diagnosis can be confirmed, if necessary, by fungal culture or culture-independent molecular tools of skin scrapings. Accurate diagnosis and proper antifungal treatment are important to alleviate pruritus, prevent the fungus from spreading to other parts of the body and to other individuals, reduce the risk of complications that may arise, and improve quality of life. Topical antifungal therapy is the mainstay of treatment for superficial or localized tinea pedis. Oral antifungal therapy should be reserved for severe disease, failed topical antifungal therapy, with concomitant onychomycosis, or for immunocompromised patients. Patient compliance is often an issue, as patients tend to cease treatment too early when clinical symptoms have improved, which may lead to recurrence of the disease.

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