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Tinea Pedis

Alexander K C Leung[1*] and Benjamin Barankin[2]

[1]Clinical Professor of Pediatrics, University of Calgary, Pediatric Consultant, Alberta Children's Hospital, Canada

[2]Dermatologist, Medical Director and Founder, Toronto Dermatology Centre, Canada

Abstract

Tinea pedis, also known as "athlete foot", refers to a superficial fungal infection of the feet caused predominately by dermatophytes. Tinea pedis is most often caused by *Trichphyton rubrum* and *T. interdigitale*. It is estimated that 10 to 15% of the world population have tinea pedis. The prevalence is higher in adults than in children. The peak age incidence is between 16 and 45 years of age. Tinea pedis is more common among males than females. Human may become infected through close contact with infected persons, animals, fomites, or soil. The transmission of tinea pedis is facilitated by warm, moist environments and wearing of occlusive shoes. Three clinical forms of tinea pedis are recognized, namely, interdigital, moccasin, and vesicobullous. The diagnosis is often clinical, especially if the lesions are typical. If necessary, the diagnosis can be confirmed by potassium hydroxide wet-mount examination of skin scrapings of the active border of the lesion. Superficial or localized tinea pedis usually responds to topical antifungal therapy twice daily for 2 to 4 weeks. Systemic treatment is indicated if the lesions are extensive, chronic, recurrent or resistant to topical antifungal treatment or if the patient is immunocompromised, or there is evidence of concomitant nail involvement.

Keywords: Trichphyton rubrum; Trichphyton interdigitale; Interdigital; Moccasin; Vesiculobullous

***Corresponding Author:** Alexander K C Leung, Clinical Professor of Pediatrics, University of Calgary, Pediatric Consultant, Alberta Children's Hospital, #200, 233 – 16th Avenue NW, Calgary, Alberta, Canada T2M 0H5; Fax: (403) 230-3322; E-mail: aleung@ucalgary.ca

Introduction

Tinea pedis, also known as "athlete foot", refers to a superficial fungal infection of the feet caused predominately by dermatophytes. It is the most common superficial fungal infection of the skin [8].

Etiology

Tinea pedis is most often caused by *Trichophyton rubrum* and *T. interdigitale* (previously known as *T. mentagrophytes*), followed by *Epidermophyton floccosum* [1, 6]. Other causative dermatophytes include *T. tonsurans* and *Microsporum* spp [7]. Nondermatophyte molds such as *Scytalidium hyalinum*, *S. dimidiatum*, and *Scopulariopsis brevicaulis* as well as *Candida* species may also cause tinea pedis [6, 7].

Epidemiology

It is estimated that 10 to15% of the world population have tinea pedis [2, 6]. The prevalence is higher in adults (17%) than in children (4%) [2]. The condition is more common in adolescents than in prepubertal children [1]. The peak age incidence is between 16 and 45 years, when working and leisure activities are at a maximum [9]. Tinea pedis is more common

among males than females [6]. Humans may become infected through close contact with infected persons, animals (in particular, house pets), contaminated fomites, or soil [2]. Transmission among family members is the most common route; children often become infected by spores or infected skin fragments shed by a household contact [2]. Autoinfection by dermatophytes elsewhere in the body may also occur [2]. The transmission of tinea pedis is facilitated by a warm, moist environment and wearing of occlusive shoes [1]. The condition is more prevalent among athletes and manual laborers [4, 12]. Immunodeficiency, diabetes mellitus, atopic dermatitis, hyperhidrosis, insufficient foot care, and obesity are other predisposing factors [6, 12].

Pathogenesis

The causative organism can produce enzymes such as proteases that digest keratin and keratinase that penetrates keratinized tissue [11]. The hyphae then invade the stratum corneum and keratin and spread centrifugally outward. Infection is usually cutaneous and restricted to the non-living cornified layers because the fungus is not able to penetrate the deeper tissue of a healthy immunocompetent host [11]. Scaling results from increased epidermal replacement following inflammation.

Clinical Manifestations

Three clinical forms of tinea pedis are recognized, namely, interdigital, moccasin, and vesiculobullous [4, 6, 12]. Interdigital tinea pedis, the most common form, presents with erythematous plaques and white macerated areas between the toes, particularly the web space between the fourth and fifth toes [6]. Often there is peripheral scaling and fissuring. The condition is often pruritic. The disease can spread to the soles, sides, and dorsum of the involved foot (Figure 1). Secondary bacterial infection in the interdigital areas can result in erosions, foul odor, and crusting.

The moccasin type is characterized by fine, silvery scaling plaques with varying degrees of underlying erythema on the heels, soles, and lateral aspects of the feet [4]. This form is frequently asymptomatic and is quite resistant to treatment [4, 12].

Vesiculobullous tinea pedis, the least common form, presents with vesicles and/or bullae, typically on the instep of the foot [4]. The condition is intensely pruritic [12].

Concomitant secondary eruptions may occur at distant sites presumably due to an immunologic reaction to the fungus [5]. This is referred to as a dermatophytid reaction [5].

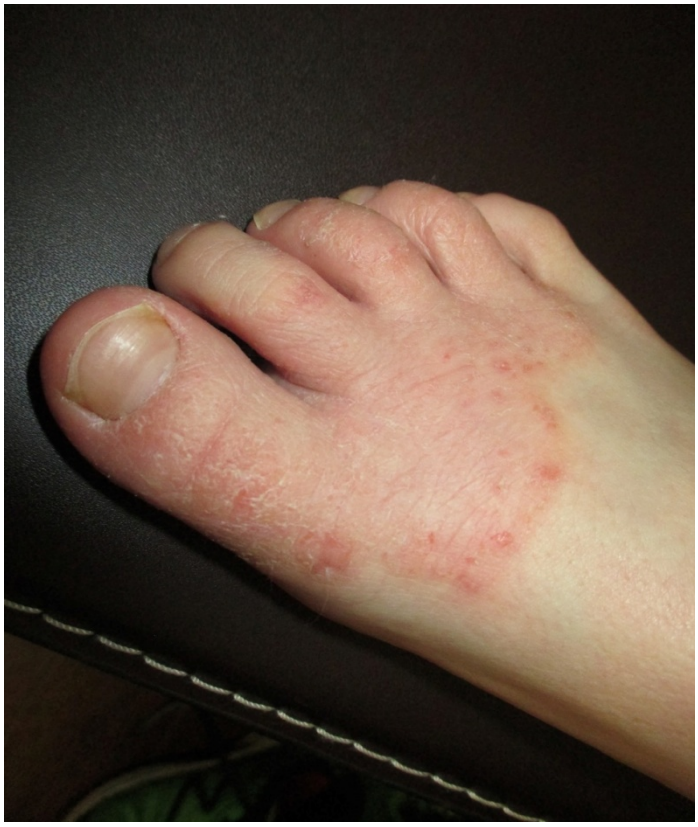

Figure 1: Tinea pedis in an 8-year-old boy with involvement of the dorsum of the right foot.

Diagnosis

The diagnosis can often be made clinically, especially if the lesion is typical. However, the diagnosis can be difficult with prior use of medications such as corticosteroids or calcineurin inhibitors. Tinea incognito refers to a dermatophytosis that has lost its typical morphological features because of the use of corticosteroids or calcineurin inhibitors. If the diagnosis is in doubt, a potassium hydroxide wet-mount examination of skin scrapings of the active border of the lesion

or the roof of a vesicle should be performed [6]. A drop of 10 to 20% potassium hydroxide, with or without dimethyl sulfoxide, is added to the scrapings. The specimen is then gently heated to accelerate the destruction of the squamous cells if no dimethyl sulfoxide is added. The potassium hydroxide dissolves the epithelial tissue, leaving behind easily visualized septate hyphae. A negative result, however, does not necessarily rule out the possibility of dermatophyte infection especially in inflammatory cases [12].

Although fungal culture is the gold standard to diagnose dermatophytosis, culture is rarely needed, unless the diagnosis is in doubt, the infection is severe, widespread, or resistant to treatment. Culture is expensive and it takes 7 to 14 days for results. The most common culture medium is Sabouraud's peptone-glucose agar. Super-infection with Gram-negative bacteria may result in decreased sensitivity of cultures [4]. Wood lamp examination is not useful as the lesions of tinea pedis usually do not fluoresce with a Wood lamp. A skin biopsy for histopathology can be useful as well when the diagnosis is in doubt.

Differential Diagnosis

Differential diagnosis includes contact dermatitis, allergic dermatitis, atopic dermatitis, xerosis, dyshidrotic eczema, erythrasma, candidiasis, psoriasis, and pityriasis rubra pilaris [1, 6].

Complications

Complications include secondary bacterial infection and spread of the fungal infection to other parts of the body such as the nails (onychomycosis), groin (tinea cruris), face (tinea faciei), bearded areas (tinea barbae), and hands (tinea manuum).

Treatment

Superficial or localized tinea pedis usually responds to topical antifungal therapy twice daily for 2 to 4 weeks. Commonly used topical antifungal agents ciclopirox, econazole, clotrimazole, ketoconazole, butenafine, naftifine and terbinafine [12]. 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 among the antifungals [10]. Terbinafine might be the best strategy for maintaining cured status [10]. Nystatin is not effective for the treatment of tinea pedis [5]. Topical antifungal agents are well tolerated and side effects are uncommon, except for rare instances of contact dermatitis. Most of the relapses are a result of poor compliance. In this regard, topical antifungals such as terbinafine, sertaconazole, and econazole which can be used once daily help to improve compliance [4, 12]. Because fungi thrive best in moist warm environments, patients should be advised to wear nonocclusive, clean, natural fiber socks and shoes and dry the feet after bathing [6]. Antifungal powders may be placed in shoes daily [6]. Shoes can also be sterilized by ultraviolet-C (UVC) based devices.

Systemic treatment is indicated if the lesion is extensive, chronic, recurrent, resistant, or non-compliant to topical antifungal treatment, if the patient is immunocompromised, or there is evidence of concomitant nail involvement [3]. Oral antifungal agents used for the treatment of tinea pedis include itraconazole, fluconazole, ketoconazole, terbinafine, and butenafine [2]. In a meta-analysis of 15 randomized controlled trials (n = 1438) of oral antifungals, no significant difference was detected between terbinafine and itraconazole, fluconazole and itraconazole, and fluconazole and ketoconazole [2]. Terbinafine was found to be more effective than griseofulvin which is rarely used now [2]. Combined therapy with topical and oral antifungals may increase the cure rate.

Prognosis

The prognosis is good with appropriate treatment. Untreated, the lesions persist and progress [5].

References

1. Andrews MD, Burns M. Common tinea infections in children. Am Fam Physician 2008; 77:1415-1420.

2. Bell--Syer SE, Khan SM, Torgerson DJ. Oral treatments for fungal infections of the skin of the foot. Cochrane Database Syst Rev 2012;Oct 17;10:CD003584.
3. Ely JW, Rosenfeld S, Stone MS. Diagnosis and management of tinea infections. Am Fam Physician 2014;90: 702-710.
4. Field LA, Adams BB. Tinea pedis in athletes. Int J Dermatol 2008; 47:486-492.
5. Goldstein AO, Goldstein BG. Tinea pedis. In: Post TW, ed. UpToDate. Waltham, MA. (Accessed on February 18, 2015)
6. Gupta AK, Chow M, Daniel CR, et al. Treatments of tinea pedis. Dermatol Clin 2003; 21:431-462.
7. Mistik S, Ferahbas A, Koc A, et al. What defines the quality of patient care in tinea pedis? J Eur Acad Dermatol Venereol 2006;20:158-165.
8. Parish LC, Parish JL, Routh HB, et al. A randomized, double-blind, vehicle-controlled efficacy and safety study of naftifine 2% cream in the treatment of tinea pedis. J Drugs Dermatol. 2011; 10: 1282-1288.
9. Pau M, Atzori L, Aste N, et al. Epidemiology of tinea pedis in Cagliari, Italy. G Ital Dermatol Venereol 2010; 145:1-5.
10. Rotta I, Ziegelmann PK, Otuki MF, et al. Efficacy of topical antifungals in the treatment of dermatophytosis: a mixed-treatment comparison meta-analysis involving 14 treatments. JAMA Dermatol 2013; 149(3):341-349.
11. Surendran KA, Bhat RM, Boloor R, et al. A clinical and mycological study of dermatophytic infections. Indian J Dermatol 2014; 59(3):262-267.
12. Weinberg JM, Koestenblatt EK. Treatment of interdigital tinea pedis: once-daily therapy with sertaconazole nitrate. J Drugs Dermatol 2011; 10(10):1135-1140.