



## Tinea pedis: diagnosis and management

Guest reviewer: Ivan Bristow – Chair, UK Podiatric Dermatology Association

**T**inea pedis is one of the most commonly encountered foot infections. The fact that in most cases it is easily treatable yet still prevalent in the general population suggests that it is often undiagnosed or remains untreated by health care professionals and patients. The most common agent is *Trichophyton rubrum*, which is often mistaken for dry skin in its presentation. Treatment should include curative as well as preventative measures where possible.

### EPIDEMIOLOGY

Tinea pedis, fungal infection affecting the skin of the foot, is probably the most common infection encountered by podiatrists and chiropodists. Studies on selected populations have suggested a wide variation in prevalence ranging from 3 to 51% [1] while estimates in the general population have been around 16% [2]. Recently the Achilles study [3] highlighted that previous surveys were likely to be an underestimate of the true prevalence. In a sample of over 13 000 patients who were mycologically examined, just under 22% had fungal infection of the skin. This work also confirmed the reports of others – that the disease generally affects males three times more frequently and shows a rise in prevalence with increasing age – 60% of elderly patients having evidence of infection [4]. With an ageing population one can anticipate a rise in the prevalence of this disorder.

#### Key Points

*Tinea pedis* affects nearly a quarter of the population  
The prevalence increases to 60% in elderly patients  
Males are three times more frequently affected

### AETIOLOGY

The vast majority of fungal species responsible for tinea pedis belong to a group known as the dermatophytes. These are responsible for around 90% of all fungal foot infections [5] with yeast and non-dermatophyte moulds in the minority, only accounting for around 10% of foot infections. In Northern Europe, the causative dermatophytes are almost exclusively *Trichophyton* species; particularly *T. rubrum*, *T. mentagrophytes* (var. *interdigitale*) and to a lesser extent *Epidermophyton floccosum* [6].



Chronic plantar infection with *T. rubrum*

#### Key Point

Nearly all cases of tinea pedis are due to the dermatophyte *T. rubrum*

### SUSCEPTIBILITY AND SPREAD OF INFECTION

Most cases of dermatophyte foot infections are due to spread from human to human (anthrophilic species) although occasionally infections are acquired from animal sources (zoophilic species) such as horses, cats and dogs. Maruyama



et al [7] demonstrated that spread occurs most commonly in places where infected skin squames can be shed and transferred to the feet of healthy individuals. Fungal elements may remain viable in shed squames for up to two years.

Once attached to the epidermis, dermatophytes generally invade with hyphae growing between adjacent epidermal cells and destroy keratinocytes by the secretion of keratinases.

Although, potentially, any individual may be exposed to viable fungus in this manner not all will develop tinea pedis. Many practitioners may be aware of this fact having been exposed to fungal elements daily and not having acquired any infection. Precipitating factors for fungal foot disease are outlined in Table 1.

Table 1. Risk factors for susceptibility to tinea pedis

Adhesion of fungal elements	Proper drying of the foot with a towel has been demonstrated to remove infected particles [8]
Host skin condition	Fungi thrive in a humid, carbon dioxide rich environment. Skin pH can also make a difference as to whether overt infection occurs or not. Sebaceous secretions contain fatty acids and these can be fungicidal
Host immune response	Immune response can vary from individual to individual. People with diabetes are at a slightly higher risk of fungal infections due to the increased amounts of sugar in various tissues
Genetics	Studies have shown that particular families have a genetic predisposition to infection with <i>T. rubrum</i> . This is thought to be an autosomal dominant trait affecting cell mediated immunity [9]
Smoking	Two studies have reported a higher incidence of tinea in smokers [3, 10]
Systemic disease	Patients with endocrine disorders, vascular disease and obesity have all demonstrated a higher risk of developing tinea pedis [3, 10]

#### **PATHOLOGY AND CHRONIC INFECTION**

In normal subjects infection with dermatophytes causes a delayed T-cell mediated response that causes an acute inflammatory eruption. Growth factors are released from monocytes and lymphocytes that increase epidermal cell turnover and shed the invading fungus [11]. However, patients with chronic infections have been shown to demonstrate a defective cellular immunity that renders them incapable of clearing the dermatophyte infection. Linked to the high prevalence rate of fungal dermatoses, it has been suggested that about 10–20% of the population may possess this trait [12].

#### *Key Point*

*A section of the general population may have increased susceptibility to superficial skin infections*



**Interdigital tinea pedis**

#### **CLINICAL RECOGNITION**

Tinea pedis presents in a number of forms:

1. Interdigital infection
2. Tinea Incognito
3. Vesicular eruption
4. Plantar moccasin spread.

Classic interdigital infection typically arises in the lateral web spaces. The presence of a dry fissure with pruritis is the typical sign of resident fungi. The macerated web space may indicate the presence of Gram-negative bacteria alongside fungi. Swabbing of moist web spaces is generally less likely to recover fungal elements as they are often inhibited by the release of bacterial agents [13]. Clinically, to eradicate a moist interdigital infection an agent must be selected that has both antibacterial and antifungal properties.

#### **Key Point**

*Moist interdigital spaces often indicate a mixed fungal and bacterial skin infection*



**Tinea Incognito**

# Continuing Professional Development



**Tinea pedis affecting the heel**

Tinea Incognito is a term coined by dermatologists to describe the clinical appearance of a fungal infection that has been treated, inappropriately, by the application of topical steroids. The effect is that the infection spreads widely, typically from the interdigital spaces onto the dorsum and may appear less florid in its eruption as the immune response is suppressed.

Vesicular eruption is a less common type characterised by the formation of vesicles on a background of erythema and intense itching. These typically arise from the interdigital spaces but also occur as asymmetrical lesions on the plantar surface. Zoophilic (animal acquired) species are more likely to be the causative agents in this variety. Secondary bacterial infection may often complicate the clinical picture [14]. Recurrent bouts of inflammation are common with this type.

Plantar moccasin spread, due to *T. rubrum* is probably the most common form of tinea pedis. Clinically, it appears as a dry, powdery eruption on the soles (more evident in skin creases) with a mildly erythemic background. Owing to the thickness of the plantar epidermis, itching is often absent in this form of the disease. Despite its innocuous appearance it is often the most difficult to detect and eradicate. For many sufferers the condition is entirely unnoticed as there are no outward symptoms and the condition is often diagnosed as dry skin.

### Key point

*Many cases of dry skin are in fact tinea pedis due to T. rubrum*

Whether many cases of tinea pedis go unnoticed or are treated depends on their presentation. Maruyama and colleagues [15] demonstrated in a population of adults that treatment was more likely to be sought if itching and erythema were present. This may help to explain the high prevalence of *T. rubrum* infections owing to the lack of symptoms in many patients. Furthermore, many sufferers were unable to recognise the disease.

### Key point

*Patients are more likely to seek treatment if itching is present*

Literature in the last few years has focused on the extent to which *T. rubrum* has become almost the ultimate parasite in this form. Zaias and Rebell [9] described this condition as a syndrome as many patients with this presentation have

evidence of hand, groin and nail involvement with the plantar surface infection acting as the source. Chronic infection may represent a genetic trait in individuals who possess a defective immune response to this particular agent [16].

### Key Point

*Patients with tinea pedis may have evidence of infection elsewhere*

## WHY TREAT PATIENTS WITH TINEA PEDIS?

For many patients, tinea pedis is nothing more than an inconvenience and it could be argued that if so many of the population have it why bother? Fungal skin infection leads to itching, erythema and fissuring. However, for a small percentage of patients a breach in skin integrity may leave them vulnerable to secondary bacterial infection from staphylococci and streptococci. The extent of the problem is not known but recent research has demonstrated that many patients presenting with cellulitis also have demonstrable, concurrent fungal foot infection [17]. Furthermore, the presence of web space maceration is a known risk factor for the development of cellulitis. The more web spaces affected – the higher the risk [18].

## DIAGNOSIS

Diagnosis of tinea pedis is often made on clinical grounds alone but for formal confirmation and identification of the pathogen a microbiological diagnosis is required. For accurate diagnosis, it is important to collect a skin sample from an area likely to contain viable fungus. Clinically, this represents the edge of a skin lesion – at the interface with healthy skin. In order to increase the likelihood of a result, sufficient material should be taken to allow the microbiology laboratory to undertake microscopy and culture. Specimens may be sent to the laboratory in coloured cardboard. This allows for easier collection and processing at the lab. Unlike bacterial swabs, fungal scrapings can remain viable for many months and so do not need immediate transport to the laboratory.

Microscopy, although it can be carried out in a clinical setting, is a skill that can be technically difficult [19]. A sample skin is placed on a slide and finely sliced with a scalpel. A solution of 10% potassium hydroxide is added to soften the sample. After the slide has been warmed gently and left to clear, a microscopic examination can be carried out. The presence of fungal hyphae under microscopy is sufficient to confirm the presence of a fungal infection and treatment can be commenced.

Culture of specimens is normally carried out by a microbiology laboratory. Culture is a means to identify the species as this may inform choice of treatment. Typically the specimen is cultured on a medium for 2–3 weeks at 27°C and examined regularly for the presence of fungal growth.

## TREATMENT OF TINEA PEDIS

Effective management of tinea pedis should focus on two aspects: treating the infection and preventing reoccurrence. For skin infection a number of topical agents are available in the UK over the counter (Table 2).

Antifungal agents can be described as being either fungicidal or fungistatic in activity, that is, being able to directly kill fungus or prevent fungal replication and growth, respectively. Fungicidal agents by their nature tend to be faster to cure.



Table 2. Common agents used in the treatment of tinea pedis

Agent	Class of Drug	Examples	Availability
Amorolfine	Morpholine	Loceryl® Cream (Galderma)	POM
Clotrimazole	Azole	Canesten AF® cream and spray (Bayer consumer care)	OTC
Miconazole	Azole	Daktarin® cream (Janssen-Cilag)	OTC
Sulconazole	Azole	Exelderm® Cream (Centrapharm)	OTC
Terbinafine	Allylamine	Lamisil AT® spray and cream (Novartis)	OTC
Undeconoate	Zinc Undeconoate	Mycota® (Thornton & Ross)	OTC

A systematic review comparing the efficacy of various agents was undertaken by Hart et al [20]. From this work it was concluded that azoles, allylamines and undecenoates were all effective when compared with placebo in controlled trials.

Data were also examined comparing azoles with allylamines. The findings confirm other research [21] that allylamines such as terbinafine are more effective than azoles in the treatment of tinea pedis, in both time to cure and spectrum of activity. Terbinafine is able to work in around half the time reflecting the fact that azoles are primarily fungistatic in their action at this concentration [22] while terbinafine is fungicidal. Duration of therapy is an important factor in determining patients' compliance. On this basis, the more rapid effects of terbinafine cream may reduce the likelihood of treatment failures due to non-compliance.

#### Key point

*Allylamines, such as terbinafine, have been found to cure faster than than azoles (such as clotrimazole and miconazole)*

One longer-term study followed up a group of patients who had received either clotrimazole or terbinafine cream for interdigital tinea pedis. Based on the available data it was reported that those using clotrimazole required re-treatment within 6 months while those on terbinafine remained without the need for further treatment for around 12 months [23]. Amorolfine cream has been tested independently and has been shown to be equally effective as terbinafine cream [24].

In cases of severe tinea pedis or where a rapid cure is required, short courses of oral agents may be indicated such as oral terbinafine, griseofulvin and itraconazole. The use of oral terbinafine (250mg/day) for one week has been found to be as effective as a four-week course of clotrimazole 1% cream [25].

#### WHICH DRUGS ARE AVAILABLE FOR SUPPLY TO PATIENTS?

For skin infection, virtually all topical creams and sprays are available for supply by podiatrists or from pharmacies and other retailers. The only exception to this is amorolfine cream (Loceryl® cream, Galderma UK) which is currently a prescription only medicine.

#### COMBINED TOPICAL ANTIFUNGAL AND TOPICAL STEROID PREPARATIONS

A number of combination topical antifungal and steroid preparations are currently available on prescription, indicated for use in tinea pedis. Evidence for their success in clearing fungal foot infections compared with plain antifungal agents is lacking. One American review paper [26] has quoted workers who suggested that combination therapy of this type is more expensive, risks higher cutaneous reactions and is generally less effective. This type of preparation is more likely to be prescribed in primary practice, perhaps when the diagnosis is not so clear.

#### PREVENTION OF RELAPSE

Despite the availability of effective antifungal agents, many patients suffer with recurrent bouts of tinea pedis. Relapse may result from reoccurrence (a previous infection has failed to be fully eradicated) or from re-infection (an infection is completely cured and after a period of time a new infection develops).

Research investigating successful methods to prevent reoccurrence and re-infection are sparse and therefore suggestions can only be made. When an infection has apparently cleared, prophylactic use of topical antifungal agents on a weekly basis may help to reduce reoccurrence. Topical terbinafine has been shown in studies to remain in the tissues at an active concentration for over 7 days following cessation of treatment [27, 28]. Furthermore, fungal spores (or arthroconidia) may remain in footwear and hosiery from which relapse may develop. For this reason, it is advisable for patients to discard old hosiery and to disinfect footwear with a preparation such as terbinafine (Lamisil AT®) spray, which has been effective in inactivating arthroconidia from various species of dermatophytes [29]. Another consideration is that a percentage of patients who have tinea pedis may also have evidence of fungal infection in other parts of the body, typically the hands and groin. So, these areas also need to be treated effectively to prevent re-infection from other parts of the body.

Re-infection can occur in particular individuals who may be exposed to high levels of fungal elements (i.e. sports club members, swimmers, etc) or patients at a higher risk of infection due to underlying diseases such as peripheral vascular disease and diabetes, so preventative action should be taken. Basic hygiene measures have been shown to be effective. For example, drying of feet following deliberate inoculation by dermatophyte elements has been shown to be effective in removing them before an infection is established [8]. Table 3 summarises basic measures that may reduce the likelihood of relapse.

Table 3. Recommendations to prevent tinea pedis – adapted from [30]

Avoid going barefoot in public places
Dry feet properly after bathing
Encourage prophylactic application of antifungal agents
Check there are no other active sources of infection elsewhere, i.e. hands, groin
Discard old footwear and hosiery
Ensure footwear is properly fitted and not occlusive
Hosiery should be natural fibre (i.e. cotton, not nylon or rayon) and changed regularly
Ensure other family members are free of tinea pedis

## Post-reading activity

### Short Answer Questions

1. What is the most common age group affected by tinea pedis?

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2. How common is tinea pedis among the general population?

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3. What is the most common form of presentation of the disease?

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4. Where else may fungal infection co-exist?

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5. What factors make some people more susceptible to infection than others?

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6. Outline the main complications associated with fungal skin infection.

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7. How would you confirm a diagnosis of tinea pedis?

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8. What are the main groups of drugs available for treating tinea pedis?

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9. Which agents are considered to be superior in treating the disease?

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10. What advice should be given to patients with fungal foot infection?

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## Reflection

After reading this CPD article, take a few minutes to reflect on tinea pedis and how it is managed. Areas for reflection may include:

- How often do I often recognise the condition?  

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- Am I treating the disease effectively and giving the patient the best care possible?  

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- How do I assess my treatment outcomes?  

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- How does this article change my practice, if at all?  

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## Notes

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