



## A REVIEW ON: ATHLETES FOOT INFECTION

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### ABSTRACT

This composition is aimed at furnishing a brief overview of the condition tinea pedis, generally appertained to as athlete's bottom. Tinea pedis is a veritably common fungal infection that affects a significantly large number of people encyclopedically. The donation of tinea pedis can vary grounded on the different clinical forms of the condition. The symptoms of tinea pedis may range from asymptomatic, to mild-to-severe forms of pain, itchiness, difficulty walking and other enervating symptoms. There's a range of preventative measures available to help infection, and both oral and topical medicines can be used for treating tinea pedis. This composition compactly highlights what athlete's bottom is, the different causes and how they present, the frequency of the condition, the variety of individual styles available, and the pharmacological and non-pharmacological operation of the condition. The body typically hosts a variety of saprotrophic micro-organisms that may beget infection. Athlete's bottom causes scaling, flaking and itching of the affected skin. Pocks and cracked skin may also do, leading to exposed raw towel, pain, swelling and inflammation. Secondary bacterial infection can accompany the fungal infection. This work examined the anti-fungal exertion of *Ardisia crispera* (AC) against common fungi that beget Athlete's bottom and several other pathogenic fungi. The antimicrobial exertion of water, ethanol and chloroform extracts of AC was tested against fungal strains using the slice proximity system. This antimicrobial exertion was compared to standard antifungal medicines (griseofulvin, fluconazole and itraconazole). Results revealed that chloroform extract of AC had potentiate antifungal exertion against *Trichophyton rubrum* ATCC 40051 and *Trichophyton mentagrophytes* ATCC 40004 which are the two most generally cause of Athlete's bottom.

### 1. INTRODUCTION

Athlete's foot, sometimes its also called as Tinea Pedia, Tenia pedum, moccasin foot and ringworm of the foot. Athlete foot is a skin disease caused by a fungus, usually occurring between the upper layer of skin in toes, but it can also affect the other areas of the feet. The fungus most commonly attacks the feet because shoes create a warm, humid dark, irritating and moist environment that encourages fungus growth. Not all fungus conditions are athlete's foot. Other conditions, such as eczema and psoriasis, may mimic athlete's foot. Tinea pedis is a habitual fungal infection of the bases, veritably frequently observed in cases who are immuno-suppressed or have diabetes mellitus. The rehearsing allergist may be called upon to treat this complaint for colorful reasons. occasionally tinea infection may be incorrect for atopic dermatitis or antipathetic eczema. In other cases, tinea pedis may complicate and asthma and may contribute to refractory atopic complaint. Cases with intermittent cellulitis may be appertained to the allergist/ immunologist for a vulnerable evaluation and discovered to have tinea pedis as a prepping factor. From a molecular viewpoint, superficial fungal infections may induce a type 2 T coadjutor cell response (Th2) that can aggravate atopy. Th2 cytokines may induce eosinophil reclamation and immunoglobulin E (IgE) class switching by B cells, thereby leading to exacerbation of atopic conditions. Three groups of fungal pathogens, appertained to as dermatophytes, have been shown to beget tinea pedis *Trichophyton* sp, *Epidermophyton* sp, and *Microsporum* sp. The complaint manifests as a pruritic, erythematous, scaled eruption on the bottom and depending on its position, three variants have been described interdigital type, moccasin type, and vesiculobullous type. Tinea pedis may be associated with intermittent cellulitis, as the fungal pathogens give a gate for bacterial irruption of subcutaneous. In some cases of refractory asthma, treatment of the associated tinea pedis infection may induce absolution in airway complaint. veritably frequently, prolonged topical and/or oral antifungal agents are needed to treat this frequently frustrating and morbid complaint. An evaluation for underpinning immuno-repression or diabetes may be indicated in cases with refractory complaint.

#### Pathogens:

Three species of fungi, *Trichophyton rubrum*, *Trichophyton mentagrophytes*, and *Epidermophyton floccosum* are together responsible for the vast maturity of cases of tinea pedis throughout the world. Of these keratinophilic organisms, *Trichophyton rubrum* is the most common pathogen associated with habitual tinea pedis, while other fungal pathogens have also been associated with the complaint and are listed in Table 1. The factors affecting the transmission of these dermatophytic pathogens are dependent on the source of infection, which is generally either mortal (anthropophilic), beast (zoophilic) or soil. The most common anthropophilic dermatophyte infection seen is *T. rubrum*. A recent study showed that *T. rubrum* reckoned for over 76 of all dermatophyte infection, including tinea pedis (1) and may regard for over 2/3 of all tinea pedis infections. The spread of infections with this pathogen have been attributed to large population movements during World War II. Outbreaks of infection of rough skin have been associated with contact with infected, desquamated skin scales. This may do in military camps and in manufactories.

Rubrum appears in two forms. The first is generally white and ethereal in appearance with several upstanding hyphae and is called the "velvetlike form". The grainy form of *T. Rubrum*, still, is flat and has no upstanding hyphae (6). It's fluently confused with *T. Mentagrophytes* which is analogous in appearance and causes a more seditious form of tinea pedis. *T. rubrum* has several club shaped microconidia that form along the length of the hyphae (7). Conidia are asexual spores that form at the tip of conidiophores; in the same species, these are moreover large (macroconidia) or small (microconidia). *T. rubrum* isn't just common in Tinea pedis but in other tinea infections as well. *Trichophyton mentagrophytes* is morphologically and characteristically analogous to *T. rubrum*. Both have a downy or grainy appearance and are occasionally indistinguishable under the microscope. *T. mentagrophytes* species can be pale unheroic on the underpart while *T. Rubrum* is substantially, but not always, wine colored on the bottom (6).

*Mentagrophytes* is zoophilic and affects numerous beast species including rodents, pussycats, tykes, and nags. *Microsporum*, still, is presumably the most current of the zoophilic dermatophytes.

*Trichophyton tonsurans* is another anthropophilic fungus that causes tinea pedis. *T. tonsurans* isn't a common cause of tinea pedis but its frequency is adding in North America (6). Societies of this fungus have short, septate hyphae with several microconidia that vary from tear drop to club shape. Numerous chlamydoconidia, asexual conidia produced from the hyphae, may be seen but macroconidia are generally rare. *T. tonsurans* colonies can range in color from white to brown with an underpart ranging from unheroic to red (7). Another pathogen known to beget tinea pedis and is responsible for 5 of tinea pedis infections is *Epidermophyton floccosum* (6). *E. floccosum* is an anthropophilic fungus set up worldwide and has been indicted in several types of tinea infections. Colonies of this fungus are flat and coarse and range in color from unheroic to brown. *E. floccosum* has septate hyphae with club shaped macroconidia. No microconidia are observed in this species, but chlamydoconidia can occasionally be seen in aged colonies (7).

*Microsporum canis* is a zoophilic fungus contracted from tykes that's a rarer cause of tinea pedis infections (6). Colonies of *M. Canis* are white with a unheroic underpart and ethereal appearance while some remain tintless

(7). Their hyphae are septate, their microconidia are club shaped, they have numerous macroconidia, and their cell wall is generally thick (7). Lesions caused by this fungus are generally more severe and are frequently characterized by erythema (6).

#### **Pathogens other causes of athletes' foot: 1.**

Pathogens other causes of athletes' foot 1.

*Trichophyton*

*T. rubrum*

*T. mentagrophytes*

*c.T. tonsurans*

2. *Epidermophyton*

*E. floccosum*

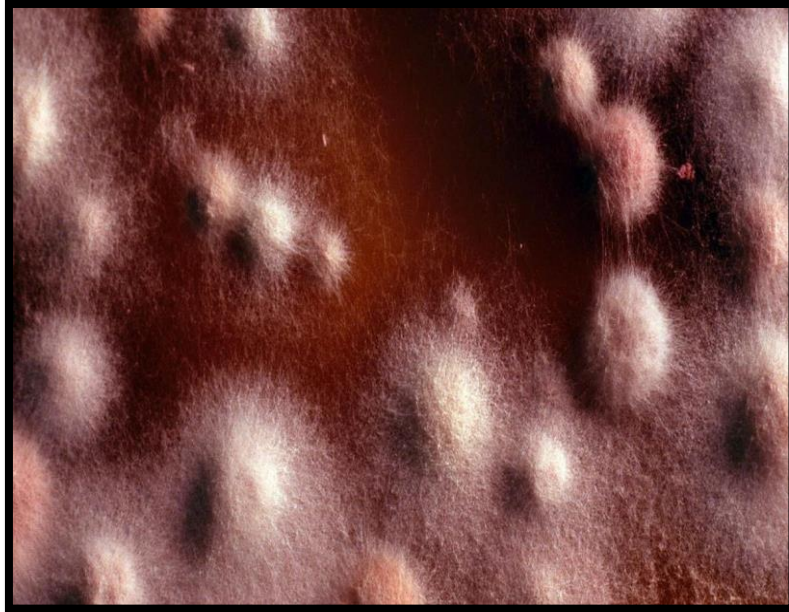
3. *Microsporum*

*M. canis*

The fungus that causes athlete's bottom is called *Trichophyton*. *Trichophyton rubrum* or *T. Mentagrophytes* is a fungus that's the most common cause of athlete's bottom. Your chances for getting athlete's bottom increases if

- Wear unrestricted shoes, especially if they're plastic-lined
- Keep your bases wet for long ages
- Develop a minor skin or nail injury
- Sweat a lot *Trichophyton Rubrum*.

The warmth and moistness of areas around swimming pools, showers, and locker apartments are also breeding grounds for fungi. Because the infection was common among athletes who used these installations constantly, the term "athlete's bottom" came popular.



**Fig. No. (1). T.rubrum**

#### **Symptoms**

- Symptoms
- The signs of athlete's bottom, independently or combined, include the following
- Itching and burning between the toes, which may increase as the infection spreads to other corridor of the bases
- Spanning or shelling skin
- Inflammation or swelling
- Pocks, which frequently lead to cracking or shelling skin to the bases and toes. When pocks break, small raw areas of towel are exposed, this may beget swelling.
- Athlete's bottom may spread to the soles of the bases and to the toenails. It can be spread to other corridor of the body by those who scratch the infection and also touch themselves away. The organisms causing athlete's bottom may persist for long ages.



Fig.No (2) Symptoms of Athletes foot.

**Risk factors for athlete's foot:**

**A. Host Factors**

1. Immunosuppression
  - a.) Chemotherapy
  - b.) Immunosuppressive Drugs
  - c.) Steroids
  - d.) Organ Transplant
  - e.) Acquired Immunodeficiency Syndrome (AIDS)
2. Poorly controlled diabetes mellitus
3. Obesity
4. Age

**B. Local Factors**

1. Trauma
2. Occlusive Clothing
3. Public Showering

#### 4. Moist Conditions

##### Epidemiology:

- Athlete's foot is maybe the foremost common dermatophyte infection within the World with up to seventieth of the population having had this infection.
- Athlete's foot is commonest among adolescents and in people World Health Organization wear occlusive shoes
- Men area unit infected 2–4 times a lot of usually than ladies.
- The risk of obtaining tinea will increase with age. Most cases occur when time of life. Infection is commonest between the ages of twenty and fifty years.
- It is a lot of common within the summer months.

##### Transmission of athlete s foot infection:

Athlete's foot may be very contagious and may be unfold via direct and oblique contact:

**Direct contact** – This entails pores and skin-to-pores and skin contact. For example, a person may also come to be inflamed in the event that they contact the affected place of your pores and skin and do now no longer wash their fingers afterwards

**Indirect contact** – This is in which the fungi may be surpassed on via infected gadgets including towels, mattress sheets and clothing. Communal showers, swimming swimming pools and converting rooms are not unusualplace locations in which athlete's foot is unfold. Like your feet, those locations are normally heat and humid, which inspires micro organism and fungi to multiply. Athlete's foot prospers in thick, tight footwear that squeeze the ft collectively and create heat, wet regions among them. Damp socks and footwear and heat, humid situations additionally choose the organisms' increase. Plastic footwear, in particular, offer a welcoming surroundings for fungal increase and contamination □ If you contact some thing that has fungi on it, you could unfold athlete's foot to different people-even in case you do not get the contamination yourself. The Typical Length for Athletes Foot An common time for an athletes foot to move away is kind of 1 to two weeks however after a quick time period or an extended time period, there are nonetheless many possibilities for the fungus to develop back.



Fig. No. (3) Transmission of Infection

##### Stages of athletes foot infection:

There are three stages of athletes foot infection causes:

Stage 1. Normally foot skin is red and crack also formed

Stage 2. The skin of foot is yellow colors and peel off

Stage 3. sites of the skin from surface of feet is separate and under them wounds are visible

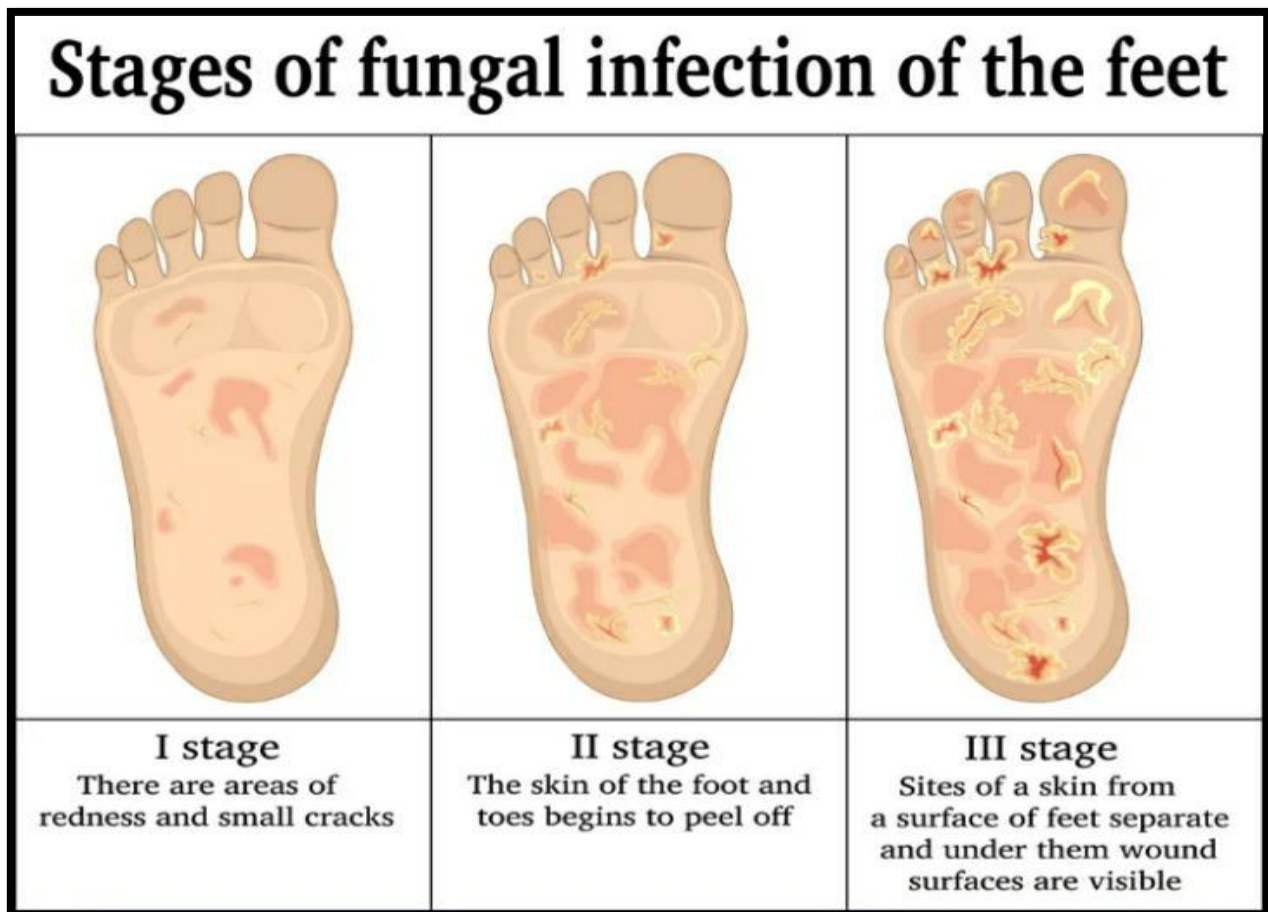


Fig. No. (4) Stages of fungal infection of the feet

#### Types of Athlete Foot:

- Toe web infection
- A moccasin-type foot infection
- Vascular-type infections

### 1] Toe Web foot Infection



**Fig. No. (5) Interdigital tinea pedis affecting the space between the third and fourth digits.**

Tinea infections between the toes are common due to high moisture content and occlusion and often present with itching, burning, and/or malodor. This figure shows a man with dry-type tinea pedis in the third interspace. (Photograph kindly provided by Dr. Stuart Leicht,

Division of Dermatology, East Tennessee State University

Toe web infection is the most common and easiest type of athlete's foot infection to treat. Most infections occur between the fourth and little toe, but it can occur between any of your toes. The skin in these areas may be dry, scaly, and peel or crack. These irritated areas of skin are more prone to allowing other bacteria to enter your body Dry & Scaly skin.

### 2] Moccasin Type foot Infection •



**Fig. No. (6) Moccasin Type foot Infection**

A moccasin-type infection is often indicated by soreness on the soles or heels of your feet. The skin in these areas can crack and thicken, and in severe cases the infection often spreads into the toenails. This can cause thick or crumbly nails and even complete nail loss. If your toenails are affected they will require separate care and treatment methods. Soreness on the soles Thick and Crumbled Toe Nails

### **3] Vascular Type foot Infection •**



**Fig. No. (7) Vascular type foot**



Fig .Vesiculobullous type feet Figure 3tinea pedis on the plantar surface of the Vesiculobullous type tinea pedis on the plantar surface of the feet. This type of Tinea pedis usually causes pustules or vesicles on the instep and plantar surfaces of the feet. Bacterial infection should be ruled out by microscopy or culture .

Vesicular-type infections are identified by fluid-filled blisters that appear on the feet. They typically appear on the soles of the feet but can also appear on the top and heels. If the blisters rupture they can cause fluid to spread under the skin or create open sores where other bacteria can cause infections. Fluid filled Blisters

#### **When to Visit a Podiatrist:**

If you believe that you have a fungus infection that does not respond to proper foot hygiene and there is no improvement within two weeks, consult a podiatrist.

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## **2. DIAGNOSIS**

Usually diagnosed by visual inspection of the skin

**KOH test-** Direct microscopy of a potassium hydroxide preparation of a skin scraping

**Using a wood's lamp (black light)** - Although useful in diagnosing fungal infections of the scalp (Tinea capitis), is not usually helpful in diagnosing athlete's foot,

Since the common dermatophytes that cause this disease do not fluoresce under ultraviolet light Your podiatrist will determine if a fungus is the cause of the problem. If it is, a specific treatment plan, including the prescription of antifungal medication, applied topically or taken by mouth, may usually be suggested. Such a treatment appears to provide better resolution of the problem when the patient follows the course of treatment prescribed by the podiatrist; if it's shortened, failure of the treatment is common. Topical or oral antifungal drugs are often prescribed. If the infection is caused by bacteria and not fungus, antibiotics that are effective against a broad spectrum of bacteria, such as penicillin, may be prescribed. It is important to keep the feet dry by using foot powder in shoes and socks. The feet should be bathed frequently and all areas around the toes dried thoroughly. If someone in your family develops athlete's foot, disinfect home showers and tubs after each use to discourage transmission of infection.

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## **3. PREVENTION**

It is easy to prevent athlete's foot where bare feet come in contact with the fungus by practicing good foot hygiene. You can prevent fungal infection by practicing the following: Wash feet daily with soap and water; dry carefully, especially between the toes Avoid walking barefoot; use shower shoes in public showers. Reduce perspiration by using talcum powder Wear light and airy shoes Change shoes and socks regularly to decrease moisture Wear synthetic blend socks that wick away moisture, and change them frequently if you perspire heavily

To Prevent Athletes foot, you would need to follow some steps you could do these steps daily

- At home, first take your shoes off and put them in the air to make them not moist
- Change your socks and underwear everyday when you sweat or when it is hot outside
- Dry your feet carefully and especially between your toes after using a shower or a locker room
- Avoid walking in bare foot in especially on public areas
- Throw away any type of worn out shoes or exercise of shoes and never borrow anybody's shoes`

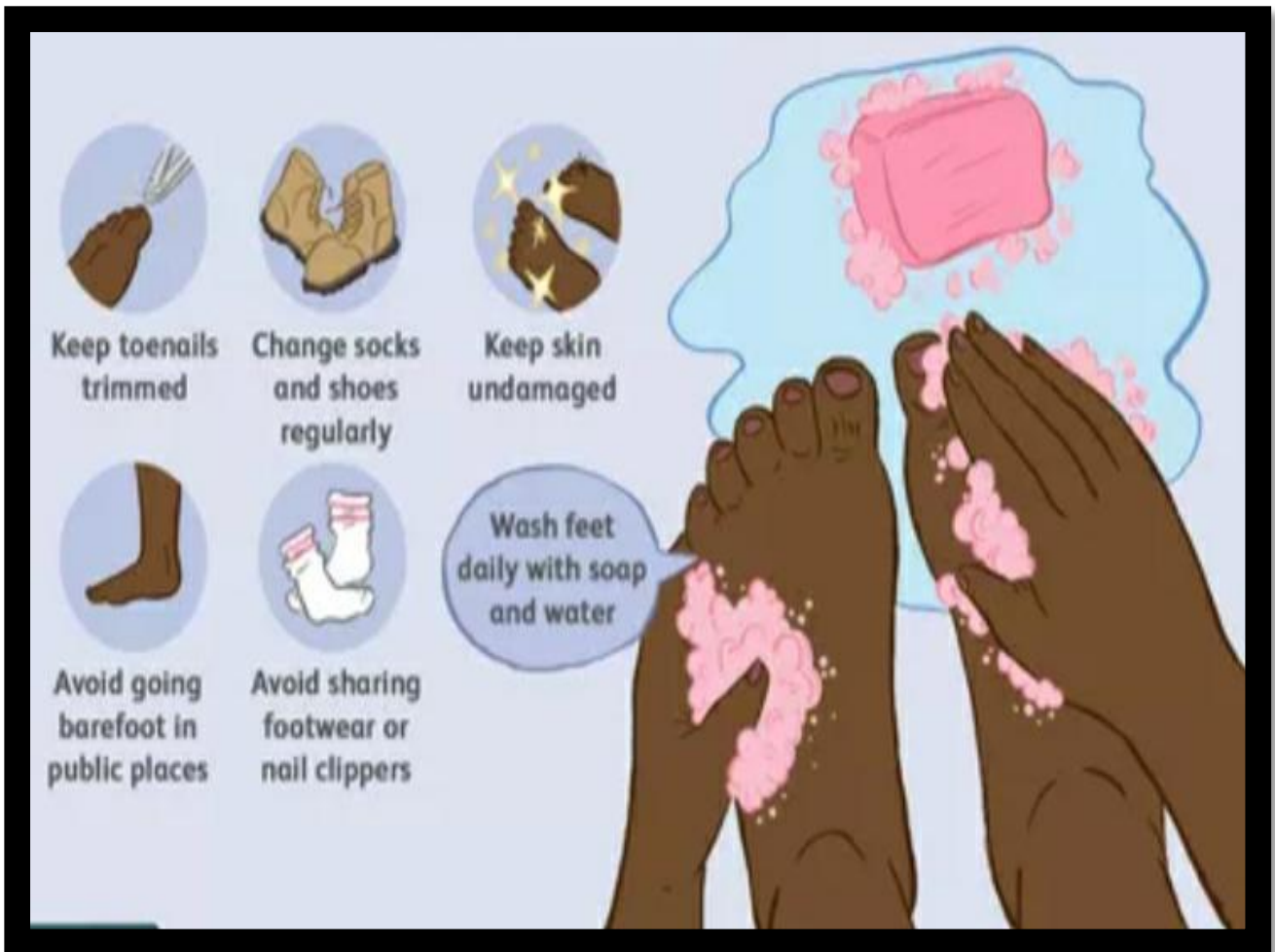


Fig. No. (8) Prevention of Athletes foot Infection

#### Treatment:

(Self care) In addition the following steps can help treat athlete's foot:

- Wash your feet regularly and thoroughly using soap and water.
- After washing, dry your feet, paying particular attention to the areas between your toes.
- Wear clean cotton socks.
- Change your shoes and socks regularly to help keep your feet dry.
- Do not share towels, and wash your towels regularly.

#### Medicines used in Athlete's Foot:

Use anti-fungal creams or sprays

- Topical- Clotrimazole, Miconazole, Tolnaftate, Naftifine, Butenafine.
- Hydrocortisone treatment- Hydrocortisone reduces inflammation and eases irritation and itching But cannot be used for longer than seven days. → Take oral tablets
- Oral- Terbinafine, Fluconazole, Itraconazole, Griseofulvin.
- Oral drugs are comparatively toxic and expensive.

**The azole antifungal agents:**

The azoles are a class of five-membered, heterocyclic compounds containing a nitrogen atom and at least one other non-carbon atom (i.e. nitrogen, sulfur or oxygen) as part of the ring structure. Azoles that are available for clinical use are classified as either Imidazoles or triazoles, according to the number of nitrogen atoms they contain in their chemical structure<sup>3,17</sup>:

- Imidazoles (ketoconazole, miconazole, econazole and Clotrimazole)
- Triazoles (fluconazole, itraconazole and voriconazole).

Although these medications share a similar mechanism of action and spectrum of activity, their pharmacokinetics and therapeutic uses vary significantly. Imidazoles, in general, are fungistatic.

**Mechanism of action:** They inhibit C-14 alpha demethylase, thereby blocking the demethylation of lanosterol to ergosterol. Ergosterol is the primary sterol of fungal membranes. Inhibition of ergosterol synthesis disrupts fungal membrane structure and function, which in turn, prevents fungal growth.<sup>16</sup> The selective toxicity of the Imidazoles results from their greater affinity for fungal, rather than for human cytochrome P450-enzymes. Resistance is seen with prolonged therapy in advanced HIV-infection, or following bone marrow transplant procedures. Mutations in the C-14 alpha demethylase gene that leads to decreased azole binding can also occur. Some strains of fungi have developed efflux pumps that actively pump out azole molecules from their cell contents. Spectrum of activity: The azoles are broad-spectrum antifungal agents, with their spectrum of activity including the following:

- Many species of *Candida* and *Cryptococcus neoformans*
- Dermatophyte-mycoses.

Drug interactions: All azoles inhibit the hepatic CYP450 isoenzymes (especially CYP450 3A4) to varying degrees; decreasing the metabolism of other drugs, and leading to numerous drug interactions. Potent CYP450-inducers, e.g. rifampicin, can lead to decreased effect of azoles. On the other hand, potent CYP450-inhibitors, e.g. ritonavir, can lead to increased adverse effects of the azoles.

**Terbinafine:**

Terbinafine<sup>17,19,23</sup> is a synthetic allyl amine (squalene epoxidase inhibitor) that is available in both oral and topical formulations.

**Mechanism of action:** Allyl amines act by inhibiting the squalene epoxidase enzyme thereby blocking the biosynthesis of ergosterol, an essential component of fungal cell membranes. Accumulation of toxic amounts of squalene results in increased membrane permeability and death of fungal cells.<sup>19</sup> Terbinafine is the drug of choice for treating onychomycosis. Applicable pharmacokinetics: More than 70% of the drug is absorbed and it is highly bound to plasma proteins. However, due to the first-pass effect only 40% of the ingested drug is available to the systemic circulation. It is metabolised in the liver by several CYP 450 isoenzymes and mainly excreted in the urine.<sup>17</sup> It accumulates in breast milk and should not be given to nursing mothers.

Side-effects: These include headaches, gastrointestinal disturbances and skin rashes. Terbinafine should be used with caution in the presence of renal and hepatic impairment.<sup>23</sup>

**Griseofulvin:**

Griseofulvin<sup>19</sup> was first discovered in 1939 from *Penicillium griseofulvum*, and was the first available oral agent for the treatment of dermatophytosis. Now it has been largely replaced by oral Terbinafine for the treatment of onychomycosis, although it may still be used for the treatment of dermatophytoses.

**Mechanism of action:** Following oral administration, griseofulvin is deposited in the keratin precursor cells and has a greater affinity for diseased tissue. It tightly binds to newly synthesised keratin, forming a keratin-griseofulvin complex, which becomes highly resistant to fungal invasion. Once the complexes reach the site of action in the skin, they bind to fungal microtubules (tubulin), thus altering fungal mitosis. Applicable pharmacokinetics: This agent is available in oral tablet form only; it is ineffective topically. In terms of its distribution, it tends to become concentrated in the skin, hair, nails and adipose tissue. Griseofulvin is metabolised by the liver to 6-desmethyl-griseofulvin and its glucuronide conjugate, and is ultimately excreted in urine, faeces and perspiration.

Side-effects: These include skin rashes, headache, urticaria, gastrointestinal disturbances and oral thrush. This agent is contraindicated in patients with porphyria.

**Topical agents:**

Several antifungal agents may be employed to effectively manage dermatophytoses (Tinea infections) of the skin. These include undecenoic acid (and zinc undecenoate) and Tolnaftate. The azoles (refer to the afore-mentioned text) and Terbinafine (an allylamine; as is naftifine) can also be used, and are effective against candidiasis as well. Topical nystatin may be used for mucocutaneous candidiasis, such as oropharyngeal and vulvovaginal thrush, but is not effective against tinea infections (including tinea pedis). Fungal infections of the nails (onychomycoses) may be treated topically with

amorolfine. Ciclopirox is another topical treatment option in the management of dermatophytosis, and is also available as a shampoo, as well as a nail lacquer solution for the treatment of mild onychomycosis.<sup>17,18,24</sup>

#### Home remedies to Cure Athlete's Foot:

- Try a bleach soak.
- Soak your feet in betadine.
- Make a vinegar soak.
- Use a bit of black tea.
- Improve your feet with cinnamon.
- Tea tree oil is an antifungal and antibacterial agent derived from the Australian *Melaleuca alternifolia* tree. Although it reduces fungi and resulting symptoms, tea tree oil may not completely kill the infection.
- Ajoene is an antifungal compound found in garlic. It is sometimes used to treat athlete's foot.
- Treatment During Pregnancy
- Dilute vinegar soaks or sprays (roughly one part white household vinegar to four parts water) and Clotrimazole cream twice a day for two to three weeks to the soles. Antifungal pills are generally not recommended during pregnancy because of the potential side effects and possible fetal harm.

#### Complications In athletes foot treatment:

If untreated, skin blisters and cracks caused by athlete's foot can lead to severe bacterial infections. In some types of athlete's foot, the toenails may be infected. All types of athlete's foot can be treated, but symptoms often return after treatment. Athlete's foot is most likely to return if: . You don't take preventive measures and again exposed to fungi that cause athlete's foot. 2. You don't use antifungal medicine for the prescribed length of time and the fungi are not completely killed. The fungi are not completely killed even after the full course of medicine. 4. Severe infections that appear suddenly, and keep returning, can lead to long-lasting infection. Effected Toe Nails.

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## 4. CONCLUSION

Fungal bottom infections, particularly athlete's bottom, are veritably common. They're more current with adding age and utmost frequently do in men. Athlete's bottom is a veritably contagious condition and spreads relatively fluently; the infection can frequently spread from one Point on the body to other spots. The spores live in the wettish warm areas of the body, or the terrain, and the threat of infection increases when in contact with such an terrain. Tinea pedis is generally diagnosed via clinical observation, but there are variety of other styles used to diagnose it. Prevention can effectively be done non-pharmacologically. There's also a variety of pharmaceutical products available for the operation and treatment of this condition..We Are Successful in making you Aware About Athlete's Foot

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