Dermatophyte Infections – An Update

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Introduction

Dermatophyte infections are the most common superficial fungal infections involving the skin, nails or hair and they present with a wide range of clinical manifestations. The word ‘dermatophyte’ is derived from the Greek words for ‘skin’ and ‘plant’. Though simple to treat, the definitive diagnosis of this condition may be challenging, as there are many dermatological conditions which mimic a superficial dermatophyte infection. It is also mismanaged often with topical and oral steroids.

Dermatophyte infections cause lesions popularly known as ‘ringworm’. The word ‘Tinea’ is used to describe these infections, derived from a Latin word meaning “gnawing worm/moth”. Depending on the site of involvement, these infections have been termed tinea capitis, tinea barbae, tinea faciei, tinea corporis, tinea manuum, tinea pedis, tinea cruris, and tinea unguium.

It is caused by three genera of fungi: trichophyton, microsporum and epidermophyton. The unique characteristic of these fungi is their restriction to keratinized tissue-stratum corneum (the most superficial layer of the skin), nail and hair.

Epidemiology

Dermatophytes are prevalent worldwide and can affect usually healthy subjects. Tinea rubrum is considered to be the most common dermatophyte infection globally. In India, the incidence of dermatophyte infections is about 6.09% with a male predisposition and the most common type was tinea corporis (33.3% of the cases- Hanumanthappa et al.). Another study done in 2014 by Surendran et al., found tinea corporis with tinea cruris was the most common presentation (73.91%)7.

Predisposing conditions

Dermatophyte infections are more common if there are predisposing conditions like:
• Endocrine illnesses: diabetes mellitus, Cushing’s syndrome
• HIV infection: more severe and recalcitrant
• Immunosuppressed states: primary immunodeficiency syndromes, cancer chemotherapy, post-transplant patients, exogenous corticosteroid use
• Poor socioeconomic status with overcrowding and sharing of combs, towels and other fomites
• Young age: tinea capitis is common in children
• Environmental factors: contact with infected animals, tropical climate
• Defective cutaneous barrier caused by widespread disease eg.- ichthyosis

![Figure 1. Tinea corporis - Hypopigmented plaque with scaly dull erythematicus macules](image)

![Figure 2. Tinea imbricata. Note the concentric rings peripheral to the plaque (black arrow). An annular plaque with peripheral scaling (green arrow).](image)
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Pathophysiology of dermatophyte infections

Dermatophytes grow poorly at 37°C. As a result they do not penetrate beyond the superficial layers of the skin. The presence of serum inhibitory factor also prevents them from invading living tissue. The clinical manifestations depend on the infecting species, site affected, and immune status of the individual. These fungi also have the unique ability to produce keratinases which aids in their invasion of the stratum corneum. The mannans in the cell wall of these fungi have an immune inhibitory function which along with decreased epidermal turnover contributes to the chronicity of these infections.

Clinical Manifestations

**Tinea corporis:** is the infection of glabrous skin, i.e., skin of the trunk and extremities excluding skin of the palms, soles, groins. Person to person contact is the most common form of transmission.

The lesion is commonly seen in areas that are normally exposed, and is associated with itching. The lesion is characteristically a well demarcated, dry, scaly plaque with central clearing and peripheral activity with a slightly raised margin. This is due to the centrifugal spread of the fungus. This feature is absent in tinea of the palms and soles.

Other forms of presentation are:
- plaques with peripherally studded pustules
- concentric rings: this presentation is caused by tinea concentricum and is called as **tinea imbricata** (Fig. 2)
- papulopustules over the buttocks/shins: due to extensive inflammation of the hair follicles on the skin and

is known as **tinea profunda**
- perifollicular pustules or granulomatous nodules – known as **Majocchi’s granuloma**.

**Differential Diagnosis (T. corporis)**
- Annular psoriasis
- Pityriasis rosea
- Discoid eczema
- Subacute lupus erythematosus
- Granuloma annulare

**Tinea faceii:** is the dermatophyte infection of the face. Though it is an infection of the glabrous skin, it is considered as a separate entity (from tinea corporis) because of certain characteristics of the lesion - it has ill defined borders, less scaling, less prominent peripherally advancing edge and it may be associated with additional symptoms like photosensitivity.

**Differential Diagnosis (T. faceii)**
- Polymorphous light eruption
- Patch of a borderline tuberculoid Hansen’s disease
- Contact dermatitis
- Rosacea

**Tinea incognito:** is the term used to describe tinea corporis which has been treated with topical steroids thereby modifying its clinical features. The signs of inflammation are absent – erythema, scaling and the well defined peripheral raised margin. This gives the patient the false impression of being successfully treated as even symptoms like itching will improve with topical steroids. However the fungus thrives in this immunomodulated environment leading to a flare-up of the lesion on stopping the steroids. There may be peripherally studded pustules in some cases.

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Figure 3: Tinea cruris with involvement of the groin and the pubic area with characteristic scaling.

Figure 4: Tinea cruris: involving the groins, medial aspect of thighs and the pubis.
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**Tinea barbae**: is the infection of the beard area. Therefore it is an exclusive diagnosis for an adult male. This is usually caused by zoophilic species like T.mentagrophytes and T. verrucosum. It is thought to be spread by contaminated razors in barber shops. Inflammation is more pronounced due to the zoophilic spread, more terminal hairs in the site and may present as pustule-nodules, abscesses and sinus tracts. This is also associated with alopecia. Lack of pain is a striking feature unlike the other causes for folliculitis.

**Tinea cruris**: Dermatophyte infection of the groins, medial aspect of thighs and the lower abdomen (Fig3-4). T.rubrum is the usual etiological agent. This entity is more common in the tropics. Men are predisposed due to the warm and moist environment of the scrotal skin. Obesity and sweating further predispose this condition. It usually co-exists with tinea pedis and onychomycosis. The differential diagnoses can be ruled out by the potassium hydroxide test (Box 1: Laboratory test).

**Tinea capitis**: Dermatophyte infection of the scalp hair follicles and of the surrounding skin.

Dermatophyte infections are mostly seen in post-pubertal individuals, except T.capitis which is more common in children. It can be caused by all dermatophytes except Epidermophyton floccosum. The fungus can cause either an endothrix (invasion can be within the hair shaft with an intact cuticle) or an ectothrix infection (from outside the hair shaft). The spores of the fungus can be disseminated even in the air close to the patient, so a direct contact is not required for the spread of this condition. As the hair invasion occurs at a deeper plane than the stratum corneum of the skin, topical treatment is not effective. The species involved may be zoophilic (more intense inflammation or anthropophilic with less inflammation). In most cases, they present as one or more patches of alopecia with/without scaling.

**Common clinical types of tinea capitis**

T. capitis may be of the non-inflammatory or inflammatory type.

Non-inflammatory type can be of three types:

- **Grey patch type** – where there are circumscribed scaly patches of non-scarring alopecia with broken hair shafts.
- **Black dot type** - usually caused by endothrix infection. In this type, there will be broken off stumps of hair often in rounded patches with crusts or pustules. The broken off hairs are loose and when examined, are found to be surrounded by or contain fungi. A hair pull test is positive.
- **Diffuse scaly type** - is similar to seborrheic dermatitis and is seen with some fungi like Trichophyton tonsurans.

**Differential Diagnosis (T.barbae)**

- Herpetic folliculitis
- Bacterial folliculitis
- Grade 3 acne vulgaris
- Cervicofacial actinomycosis

**Differential Diagnosis (T. cruris)**

- Erythrasma
- Intertrigo — streptococcal, candida

**Differential Diagnosis (T. capitis)**

- Non inflammatory T. capitis
- Alopecia areata
- Seborrheic dermatitis
- Scalp psoriasis
- Trichotillomania

**Figure 5.** Tinea capitis presenting as non-scarring alopecia with dull erythema and broken hair shafts.

**Figure 6.** Tinea capitis as a scaly plaque with adherent crusts and broken hairs.
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Figure 7. Tinea pedis. Note the superficial scaling of the sole of the foot. The three patterns are:

**Inflammatory type**

i. Kerion: is an inflammatory fungal infection that may mimic a bacterial folliculitis or an abscess. It produces tender boggy plaques exuding pus. The scalp is tender to the touch and the patient usually has posterior cervical lymphadenopathy. This may be followed by scarring and permanent alopecia.

ii. Favus (Latin —“honeycomb”): is a chronic dermatophyte infection of the scalp caused by Trichophyton schoenleinii. It presents with scarring alopecia and cup-shaped, thick yellow concave, crusts called scutula. Air spaces are seen within the air shaft.

**Differential Diagnosis** (Inflammatory T.capitis)
- Bacterial folliculitis
- Abscesses

**Tinea pedis**: is the infection of the sole of the foot. It is predisposed in people who wear occlusive footwear. The sweat and the warmth acts as favorable factors for the growth of the fungi. Clinically this can present in 3 distinct patterns which in turn depends on the causative organism.

1. **Vesicular type** - is usually an acute presentation, where there will be vesicles along the lateral aspect of the soles or the instep. The fungus can be isolated from the roof of the vesicle. This gets controlled when the primary site of infection is treated.

2. **Intertriginous** type – the toe web spaces are involved, most commonly the forth and the fifth. These present as dry, scaly and fissured or moist, macerated and soggy. Sometimes there can be a co-infection with the bacterial flora like the *Staphylococcus aureus*, *Corynebacterium minutissimum* and *Micrococcus sedentarius*.

3. **Squamous hyperkeratotic/Moccasin type** - is a chronic form of tinea pedis, usually caused by T. rubrum. It presents as superficial scaling of the soles, heels and sides of feet and is also known as the moccasin type. Two feet—one hand syndrome may be seen when there is involvement of both the feet and one hand. There may be associated infection of the nails.

**Tinea manuum**: refers to infection of the palmar skin. It usually presents as hyperkeratosis of the palmar skin with accentuation over the flexural creases which is a characteristic feature or as circumscribed vesicular

Figure 8. Tinea pedis with tinea unguium. Note the unilateral involvement of the right foot with scaling.

Figure 9. Tinea pedis with tinea unguium. Yellow discolouration of the distal nail plate and superficial scaling of the periungual skin.
patches. The closest differential will be hyperkeratotic eczema and palmar psoriasis; these are often bilateral while dermatophyte infection is usually unilateral.

**Tinea unguium**: is the dermatophyte infection of the nail apparatus. (Other fungi like non dermatophyte moulds and candida can also infect the nail). It is seen in approximately 40% of patients with fungal infections in other locations.

**Types of tinea unguium**

There are 4 major types -

1. **Distal and lateral onychomycosis**- (most common type) presents as streaks or patch of discoloration, white or yellow at the free edge of the nail plate (**Fig 9**) or near the lateral end with secondary involvement of the underside of the nail plate of the finger or toe nails.

2. **Proximal subungual onychomycosis**: involves the nail plate mainly from the proximal nail fold. It may be an indication of HIV infection.

3. **White superficial onychomycosis**: this is an invasion of the nail plate on the surface of the nail.

4. **Total dystrophic onychomycosis**: is the involvement of the entire nail with its destruction and it may have started as any of the above mentioned types.

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**Box 1: Laboratory test for dermatophyte infection**

In any of the suspected dermatophyte infections a potassium hydroxide mount helps in a quick confirmation by the demonstration of fungal hyphae. Depending on the site, the specimen is obtained by scraping the lesion onto a slide (using blade No.10 for skin and scalp lesions and with 15 for the nail specimens). Scraping is usually taken from the active peripheral margin.

Potassium hydroxide (10% or 20% solution) is applied with a dropper to the edge of the cover slip and allowed to run under via capillary action. The preparation is gently heated under a low flame and then pressed to facilitate separation of the epithelial cells and fungal hyphae. The advantage of potassium hydroxide is that it dissolves material that fuses cells but does not distort the epithelial cells or fungi.

**Microscopy**: The preparation is studied under the cover slip at low power. The presence of hyphae should be confirmed by examination with the 40 X objective. Dermatophytes appear as translucent, branching, rod-shaped filaments (hyphae) of uniform width, with lines of separation (septa). The regular septae with characteristic bending and branching distinguish hyphae from other structures like hair or debri.
## Table 1. Some of the commonly used topical antifungal agents

<table>
<thead>
<tr>
<th>Agent</th>
<th>Indication</th>
<th>Dosage</th>
<th>Mechanism of action</th>
<th>Other characteristics</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Terbinafine cream</td>
<td>Tinea corporis, T.cruris, T.pedis</td>
<td>Local application twice daily for 2-3 weeks</td>
<td>Inhibits squalene epoxidase in the fungal cell membrane</td>
<td>Fungicidal unlike other antifungals which are fungistatic.</td>
<td>Rarely may have irritant / allergic contact dermatitis</td>
</tr>
<tr>
<td>5% Amorolfin nail lacquer</td>
<td>Tinea unguium</td>
<td>Applied once or twice a week for 6-12 months</td>
<td>Inhibits enzymes in the ergosterol pathway</td>
<td>Prior to application the diseased nail has to be gently filed</td>
<td>Usually rare Erythema, pruritus and burning</td>
</tr>
<tr>
<td>8% ciclopirox nail lacquer</td>
<td>Tinea unguium</td>
<td>Applied daily for 48 weeks</td>
<td>Inhibits metal dependent enzymatic processes</td>
<td>Cure rates are lower</td>
<td>Erythema of the periungual area</td>
</tr>
</tbody>
</table>

## Table 2. List of some of the common oral antifungal agents (adapted from Fitzpatrick’s Dermatology in General Medicine)

<table>
<thead>
<tr>
<th>Agent</th>
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</tr>
</thead>
<tbody>
<tr>
<td>Allylamines - Terbinafine: is the first line agent</td>
<td>Tinea corporis, T.capecit T.unguium</td>
<td>Tinea corporis- 250 mg once daily for 2 weeks</td>
<td>inhibition of squalene epoxidase, essential for the ergosterol pathway required for fungal cell wall synthesis.</td>
<td>Terbinafine also has a few drug interactions with rifampicin, cimetidine, terfenadine and cyclosporine. A complete blood count should be done prior to the initiation of oral terbinafine.</td>
<td>Gastrointestinal disturbances (nausea, diarrhea and taste disturbance). Skin eruptions including rarely Steven Johnson syndrome. Hepatobiliary dysfunction (rare) Therefore LFT should be monitored in patients treated for longer than 6 weeks and it is not recommended in patients with chronic or acute liver disease.</td>
</tr>
<tr>
<td>Itraconazole</td>
<td>Tinea corporis, T.capecit T.unguium</td>
<td>Tinea corporis/ cruris- 200 mg twice a day for 7 days</td>
<td>Acts by inhibiting cytochrome P 450 required for fungal cell wall synthesis.</td>
<td>It has a lower in vitro susceptibility than terbinafine. Most common - headache and gastrointestinal disturbances, elevated liver enzymes(occur in 5-10% of patients). Hepatotoxicity (with prolonged therapy). Therefore, liver function tests should be monitored if the treatment extends beyond 6 weeks, for pre-existing liver disease or if being used with other hepatotoxic drugs.</td>
<td></td>
</tr>
<tr>
<td>Griseofulvin</td>
<td>Tinea capitis: 1st line in children</td>
<td>Paediatric (&gt;2 yrs): microsize preparation 15-20 mg/kg for 6-12 weeks (or) ulramicrosize preparation 5-10mg/kg/day for 6-12 weeks Max dose 1000mg/day Max dose 750mg/day</td>
<td>Acts by inhibiting microtubule formation in cell division.</td>
<td>It is the only agent licensed for treatment in children. Not recommended in adults, due to lower cure rates, better alternative agents (like terbinafine), longer duration of treatment.</td>
<td>It is contraindicated in pregnancy. Should be avoided in hepatic impairment. Can cause photosensitivity, lupus erythematosus. It is to be taken with food as bile increases its absorption.</td>
</tr>
</tbody>
</table>

Note: 1. Topical steroids alone or in combination are not to be used as it only decreases the inflammatory symptoms and has no effect on the fungus. It also encourages the patients to use it over- the-counter as it gives a false impression of temporary cure. Long term use of topical steroids lead to irreversible changes like cutaneous atrophy, depigmentation (Fig 12), easy bruisability and telangiectasias. 2. Nystatin is not effective against dermatophytes.....

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Treatment of dermatophyte infections

The principles of treatment are:
1. Eradication of the dermatophyte infection by the use of antifungal agents.
2. Alleviation of the associated symptoms like pruritus.
3. Use of other adjuvants like keratolytics for hyperkeratotic tinea pedis (eg: Whitfield’s ointment)

The treatment is dependant upon certain factors like the age of the patient, site of involvement, the extent of involvement and the underlying co morbidities of the patient. Topical and systemic treatments are available.

Local treatment

Indications:
1. Tinea cruris
2. Tinea corporis —with limited skin involvement without complications
3. Tinea unguium —[Table 1]
   - white superficial onychomycosis
   - post treatment prophylaxis
4. Tinea pedis
5. Tinea manuum
6. When systemic therapy is contraindicated

Local therapy

1. Topical antifungals (Table 1): Terbinafine is the most commonly used topical antifungal agent. Alternatives which may be used (when terbinafine is not available) are clotrimazole, miconazole, ciclopirox olamine and tolnaftate.

   Apply any one of the medications mentioned two times a day for 2-3 weeks. Extended use till 4 weeks may prevent relapse.

2. Other adjunctive therapies:
   - Selenium sulfide shampoo or ketoconazole shampoo may be used in instances of tinea capitis for elimination of the fungal spores. It can be used on the scalp for 5 minutes, three times a week for 6-8 weeks.
   - Keratolytics like urea, lactic acid, salicylic acid paste (Whitfield’s ointment: 3% salicylic acid, 6% benzoic acid) in case of hyperkeratotic tinea pedis can be applied at night till resolution.
   - Mild potent topical steroid (1% hydrocortisone) may be used in certain instances like tinea incognito.
   - Desiccating powders may be used to prevent recurrences of intertriginous type of tinea pedis.
   - Nail sanding to be done prior to application of nail lacquers for better penetration.
   - Avoid use of occlusive footwear and moist undergarments to prevent further recurrences of tinea pedis or tinea cruris respectively.

Systemic treatment

Indications:
1. Hairy sites (eg: T. capitis, T.barbae): as the drugs need to penetrate the hair follicle for efficient treatment
2. Tinea corporis — if extensive/complicated
3. Tinea unguium —
   - involvement of the nail matrix,
   - total nail dystrophy,
   - thickening of the nail plate > 2mm
4. Those who fail topical therapy
5. Those with acute inflammatory reactions

Common causes of recurrence are:
1. Other untreated family members
2. Infected pets
3. Poor compliance /discontinuation of medicines
4. Hidden sources of persistent infection like tinea cruris, tinea pedis and tinea unguium

Complications of Dermatophyte infections

If left untreated these infections may progress to develop any of the following.

1. Id reaction: is an allergic reaction to a distant focus of infection. It may vary in its morphology and may be papular, pustular, vesicular, erythema multiforme-like or urticarial. These reactions resolve when the primary focus of infection is treated. The id reaction which occurs secondary to dermatophyte infection is called dermatophytid.

2. Erythroderma: is a dermatological emergency when there is erythema and scaling of the skin with > 90% of body surface area involvement. It is very rare and occurs in patients who are immunosuppressed.

3. Majocchi’s granuloma: when there is invasion of dermis and subcutaneous tissue by the dermatophytes via penetration of hair follicles. It usually occurs in immunosuppressed individuals.
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**KEY POINTS**

1. All red scaly lesions are not “ringworms”. When the KOH mount is negative for fungal hyphae, the following conditions need to be ruled out by doing a histopathological examination.

<table>
<thead>
<tr>
<th>Type of disorder</th>
<th>Common examples</th>
</tr>
</thead>
</table>
| Papulo- squamous | Psoriasis  
                      Pityriasis rosea |
| Infections       | Borderline Tuberculoid Hansens disease in reaction  
                      -The lesion will be hypo/anaesthetic  
                      -Nerves may be enlarged  
                      Chromomycosis  
                      (A KOH mount will reveal pathognomonic copper penny bodies) |
| Eczemas          | Discoid eczema  
                      Allergic contact dermatitis  
                      (A patch test may reveal the allergen) |
| Miscellaneous    | Sarcoïdosis  
                      Plaque stage of cutaneous T cell lymphoma |

2. All sites must be examined in case of a dermatophyte infection as these hidden sites may continue to contribute for further recurrences – nails, scalp, soles, groins and axilla.

3. In cases of recurrences, other affected household members and pets are to be treated.

4. Oral and topical steroids are not to be used for the treatment of dermatophyte infection

5. Sometimes even after the treatment of tinea unguium the nails may remain dystrophic due to other causes like traumatic dystrophy, non fungal nail disease like psoriasis or lichen planus.

References:

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