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UNDERSTANDING RINGWORM: A REVIEW ON CLINICAL, THERAPEUTIC AND RISK FACTORS OF TINEA INFECTIONS

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ABSTRACT

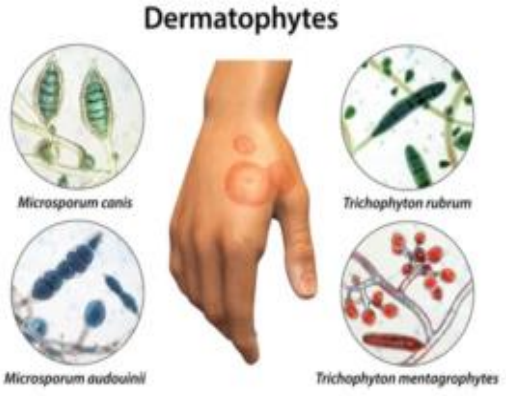
Dermatophytosis also known as Ringworm continues to be one of the most widespread superficial fungal infections globally, involving skin, hair and nails and being caused primarily by dermatophytes of the genera Trichophyton, Microsporum and Epidermophyton. While once held to be readily curable, recent years have seen significant shifts in its epidemiology, clinical presentation and therapeutic responsiveness. This review integrates existing evidence on four main areas – Clinical presentations, Treatment patterns- reporting topical and systemic antifungal treatment strategies , Risk factors and Emerging antifungal resistance pointing to increasing evidence of reduced susceptibility and documented molecular mechanisms facilitating resistance. The review highlights the imperative need for increased diagnostic vigilance, current treatment guidelines, prevention-focused education on hygiene and monitoring of resistant strains of dermatophytes. By synthesizing the most current understanding, this article is intended to assist clinicians and public-health partners in combating the changing problem of dermatophytosis in the 21st century.

KEYWORDS: Dermatophytes, Tinea Infections, Clinical Pattern, Treatment Pattern and Antifungal Resistance.

INTRODUCTION

Dermatophytosis, also called Tinea or Ringworm, is a cutaneous fungal infection of keratinized tissues such as skin, hair, and nails. Dermatophytes, their closely related fungi, infect them by being parasitic to keratin, the protein content in these tissues.

In 1934, Chester Emmons divided dermatophytes into three genera according to spore morphology and accessory organs: Trichophyton, Microsporum and Epidermophyton.



These genera may also be divided into three groups according to their main habitat

1. **Anthropophilic organisms:** They live on man and are spread directly from one individual to another. Examples – T. Rubrum, E. Floccosum.
2. **Geophilic organisms:** These fungi live in soil and can be transmitted to humans.
   Examples – M.Nanum, T. Terrestre.
3. **Zoophilic organisms:** These fungi are passed on to humans from the hosts of animals.

Examples – M.Canis, T.Simii.

EPIDEMIOLOGY

Tinea infections are prevalent worldwide and occur in about 20-25% of population. Dermatophytosis may be initiated by a number of factors including age, sex, climate, environmental conditions, socioeconomic status, and hygiene habits. Dermatophytes are favored by hot and humid climates, thereby causing an increase in these infections in tropical and subtropical regions. India alone is confronted with a serious problem as a result of the extraordinary increase in recurrent and chronic dermatophyte infections. Nevertheless, by the end of the 20th century, T. rubrum was the most frequent etiologic agent globally, especially in Central and North European nations, at 40 to 70 percent, followed by T. mentagrophytes. M.canis and Trichophyton verrucosum were the most commonly isolated zoophilic dermatophytes in Southern Europe. In Western Asia, Epidermophyton floccosum was the most frequently isolated organism, whereas Trichophyton violaceum was the predominant cause in Africa. In the 21st century, the occurrence of anthropophilic dermatophytes like E. floccosum, M. audouinii, and T. schoenleinii reduced in European countries, being substituted by other species of Trichophyton. T. rubrum emerged as the most common dermatophyte worldwide, infecting areas like South America, Asia, Africa, and Europe. Since the 20th century, T. rubrum infections have become very common and mostly occur in patients between the ages of 20 and 60, but also in older patients.

SYMPTOMS

- ✓ Itching
- ✓ Redness
- ✓ Rashes
- ✓ Scaling
- ✓ Blisters
- ✓ Burning Sensation
- ✓ Discomfort
- ✓ Irritation
- ✓ Pain
- ✓ Broken or Brittle Hair, Hair Loss
- ✓ Cracking of Skin
- ✓ Thickening of Nail or Nail Discolouration
- ✓ Onycholysis

MODE OF TRANSMISSION

1. **Direct Contact:** Contacting an infected individual. Contact with infected animals (usually dogs and cats).

2. Indirect Contact: Contact with infested objects (clothes, towels, bedding). Fungal dissemination from other infected body locations.

3. Environmental Factors: Warm, moist environments, Sharing towels or clothes, Wearing occlusive clothing that holds moisture.

RISK FACTORS

1. **Hygiene and Lifestyle**
   - Poor hygiene.
   - Exchange of personal belongings (clothing, towels, combs).
   - Excessive sweating.

2. **Health Conditions**
   - Skin diseases such as eczema or psoriasis.
   - Immunosuppression.
   - Diabetes.
   - Cutaneous trauma.

3. **Genetics & Environment**
   - Hereditary.
   - Living in overcrowding.
   - Hot and humid environment.

4. **Medications & Occupation**
   - Use of corticosteroids (may suppress the immune system).
   - Occupations with high exposure to fungi or water (e.g.. athletes, gardeners, animal handlers).

5. **Foot & Skin Care**
   - Poor foot care, particularly with extended moist conditions.

CLINICAL PATTERNS OF TINEA INFECTIONS

Dermatophyte infections are classified based on the regions of the body they infect.

TINEA CORPORIS (Ringworm of the Body)

Involved Sites: Anywhere on the body except hands, feet, scalp, bearded regions, face, groin, and nails. Affects most commonly smooth, glabrous skin.

Etiologic Agents

Microsporum canis
Trichophyton rubrum
Trichophyton tonsurans

Clinical Presentation

- ➢ Well-demarcated, oval or circular scaly plaques or patches.
- ➢ Elevated, active border with central clearing .
- ➢ Border can be irregular, sometimes pustular, vesicular, or papular.
- ➢ Mild pruritus (itching) is usual.
- ➢ Central area can be hypopigmented or brown.

TINEA CRURIS (Jock Itch)
Infected Areas: Groin, perineal, pubic, vaginal, and perianal areas.

Causing Agents
Trichophyton mentagrophytes
Trichophyton rubrum

Clinical Features
- Circular or ring-shaped lesions with clear center.
- Red, crusted, or scaly patches or plaques.
- There may be itchy blisters or bumps.
- Prone to affect skin folds where wetness is likely.

TINEA PEDIS (Athlete's Foot)
A dermatophyte infection of the foot skin. First described by Pellizzari in 1888.

Causative Agents
Trichophyton interdigitale
Trichophyton rubrum.

Clinical Forms
1. Interdigital Tinea Pedis
Location: Web spaces (frequently between 4^{th} and 5^{th} toes).
Features: Erythema, white scaling, peeling, maceration

2. Hyperkeratotic
Location: Soles, heels, sides, back of foot, distal dorsum.
Features: Persistent scaling plaques, varying erythema.

3. Vesiculobullous
Highly puritic vesicles or bullae.

TINEA CAPITIS (Ringworm of the Scalp)
Fungal infection of the scalp, eyelashes, and eyebrows. First signs: itchy, scaly areas of hair loss.

Clinical Characteristics
1. Non-inflammatory Tinea Capitis: Fine, white, adherent dandruff-like scaling,Mild hair loss.One or more discrete alopecia plaques with broken hairs "black dots" appearance.

2. Inflammatory Forms
Kerion (Kerion celsi): Boggy, inflamed, pus-containinglesions.
Favus (Tinea favus): Scaly crusts (scutula) on scalp, chronic.

TINEA MANUUM
A superficial fungal infection of one or both palms, the dorsum of the hands, or interdigital folds.

Clinical Characteristics
Dry, scaly lesions or macules, sometimes more apparent in the palmar flexural creases. Lesions may be localized to a region or diffuse, occurring in one or both hands. Inflammatory types can take the form of erythematous, pustular, or vesicular lesions. Two feet, one hand syndrome can happen when tinea manuum is associated with onychomycosis and tinea pedis.

TINEA BARBAE (Barber's Itch)
Fungal infection of the skin, hair, and hair follicles in the beard and mustache region.

Clinical Characteristics
1. Superficial, moderate pruritic type
Erythemo-squamous plaques with active borders.
Involves hair and hair follicles

2. Deep type
Erythematous plaques with pustular folliculitis. May occasionally develop nodules and abscesses.

TINEA UNGUIUM (Onychomycosis)
Fungal infection of the nail unit (nail plate, nail bed, or matrix).

Clinical characteristics
Discoloration of the nail: yellow-brown, white, green, or black. Thickening of the nail (onychochauxis), Detachment of the nail from the nail bed (onycholysis), Subungual hyperkeratosis.

TINEA FACIEI
Fungal infection involving the facial skin.

Clinical Characteristics
Scaly, flat plaques on the face,Macules with raised, active borders can develop papules,crusts,vesicles. Central zone is hypopigmented or brown.

TINEA INCOGNITO (Tinea Atypical)
Tinea incognito is a dermatophyte infection with an uncharacteristic appearance. This abnormal appearance is created by topical steroids or other immunosuppressive medications, which dampen the typical inflammatory reaction, concealing the hallmarks of fungal infections.

Causative Factors
Topical steroid use– Usually mistaken as eczema or dermatitis, which results in steroid application, further aggravating the fungal infections.

Clinical characteristics
Eczematous or dermatitis-like plaques and patches.
Scaling: Steroid-induced suppression of inflammation means that scaling is often minimal or absent.

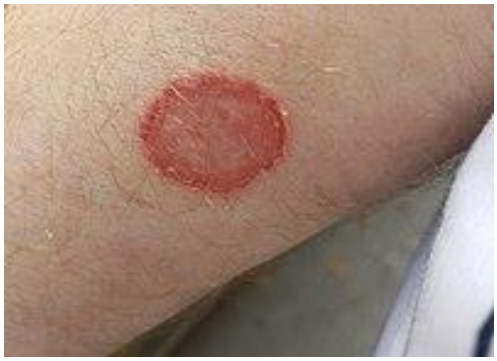
Symptoms: Mild pruritus may be present but can be less severe than in classical tinea.

PATHOPHYSIOLOGY
1. Transmission: Direct contact with infected people or animals. Indirect contact using contaminated objects (fomites).

2. Adherence and Colonization: Dermatophytes infect keratinized tissues—skin, hair, nails. They attach to the stratum corneum, the epidermis outermost layer. Dermatophytes grow on keratin, a structural protein of these tissues, as a source of primary nutrition.

3. Enzymatic Degradation: Dermatophytes produce several enzymes, among them: Keratinases – degrade keratin, Metalloproteases – break down proteins and extracellular matrix constituents, Cysteine dioxygenase – helps to use sulfur-containing amino acids. These enzymes break down keratin, proteins, lipids, and DNA, enabling fungi to colonize and disseminate. Enzyme activity disrupts epidermal barrier function and can modify epidermal differentiation

4. Host Immune Response: Keratinocytes (epidermal cells) recognize pathogens through Toll-like receptors (TLRs).TLR activation initiates the innate immune response releasing pro-inflammatory cytokine including:Interferon-gamma (IFN-γ), Tumor necrosis factor-alpha (TNF-α) , Interleukin-6 (IL-6), Interleukin-8 (IL-8), Cytokine release leads to inflammation, manifesting redness, swelling, and the classic lesions of **TINEA**

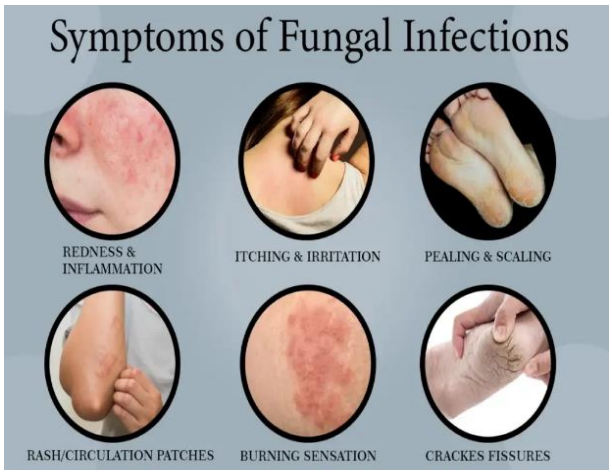


DIAGNOSIS

1. Clinical Features - Signs & Symptoms

Physical examination

Note the redness, scaling, cracking, or characteristic ring patterns on the lesion.



2. Diagnostic Methods

A. Direct Microscopic Examination

Procedure

1. Scrape the surface of the lesion.
2. Position specimen on glass slide with KOH solution.
3. KOH breaks down keratin, lipids, and proteins.
4. Fungal structures withstand digestion and become apparent.

Staining : Optional stains such as Parker Quink enhance visualization.

Microscopic appearance

Dermatophytes - transparent, unpigmented, septate hyphae or arthrospores.

B. Fungal Culture

Purpose: Confirms presence and dermatophyte species identification.

Media: Sabouraud's dextrose agar (contains dextrose, peptone, agar; neutral pH).

Growth characteristics: Visible after 5–10 days, but complete identification can take up to 4 weeks.

Identification by: size, shape, color, texture, growth rate, spore pattern, and hyphal morphology.

C. Molecular Diagnosis

Purpose: Species differentiation on a genetic basis.

Techniques:

Conventional PCR - cheaper, slower

Real-time PCR - quicker detection

Post-PCR analysis - potentially time-consuming, but highly specific

D. Dermoscopy

Procedure: Apply oil/gel to lesion Observe fungal morphology and view lesion morphology

E. Skin Biopsy

Indication: For definitive diagnosis or ruling out other dermatoses (eczema, psoriasis)

Procedure: Local anesthetic punch biopsy

Fungal elements observed under microscopy

F. Wood's Lamp Examination

Procedure: A filtered UV light (>365 nm wavelength)

Findings: Some dermatophytes fluoresce under UV, particularly infected hairs.

PHARMACOLOGICAL TREATMENT
TOPICAL AGENTS

TYPE OF TINEA INFECTION	DRUG	FREQUENCY
Tinea unguium	Ciclopirox	Once daily
Tinea Faciei	Econazole cream Miconazole cream Ketoconazole cream	Once daily
Tinea Capitis	Ketoconazole cream Ketoconazole shampoo	Once daily
Tinea Cruris	Econazole cream Miconazole cream Ketoconazole cream Terbinafine cream Ciclopirox cream	Twice daily
Tinea Pedis	Econazole nitrate cream Ketoconazole Terbinafine Hydrochloride cream Ciclopirox cream	1% cream Twice daily 2 % cream Twice daily 1% cream Twice daily 0.77% Cream Twice daily
Tinea Corporis	Miconazole cream Clotrimazole cream Ketoconazole cream Econazole cream Terbinafine cream	2% cream Twice daily 1% cream Twice daily 2% cream Twice daily 1% cream Twice daily 1%cream Twice daily

ORAL AGENTS

TYPE OF TINEA INFECTION	DRUGS	DOSE	FREQUENCY
Tinea Corporis	Griseofulvin Itraconazole Fluconazole Terbinafine	500mg 200 - 400 mg 150 -300 mg 250 mg	Once daily
Tinea Pedis	Griseofulvin Itraconazole Fluconazole Terbinafine	500 mg 400mg 150 - 300 mg 250 mg	Twice daily Once daily 1 dose/week Once daily
Tinea Cruris	Griseofulvin Itraconazole Fluconazole Terbinafine	500 mg 200 - 300 mg 150 - 300 mg 250 mg	Once daily Once daily 1 dose/week
Tinea Capitis	Griseofulvin Ketoconazole Itraconazole Terbinafine Fluconazole	20 - 25 mg/kg 200 mg 5 mg/kg/day 3 - 6 mg/ kg/day 3 – 6 mg/kg/day	Once daily
Tinea Barbae	Griseofulvin Itraconazole	0.5 mg- 1 g/day 200 mg	Once daily
Tinea Unguium	Griseofulvin Terbinafine Itraconazole Fluconazole	500 mg 25 0mg 400 mg 150 – 300 mg	Once daily 1 dose/week

ANTI FUNGAL DRUGS
Drugs used to treat both superficial and deep fungal infections.

CLASSIFICATION
- **ANTIBIOTICS**
  - **Polyenes** – amphotericin B, nystatin, hamycin
  - **Echinocandins** – caspofungin, micafungin, anidulafungin
  - **Heterocyclicbenzofuran** – griseofulvin
- **ANTIMETABOLLITES** – flucytosine
- **AZOLES**

- **Imidazoles**
  - **Topical** – clotrimazole, econazole, miconazole, oxiconazole
  - **Systemic** – ketoconazole

- **Triazoles**- fluconazole, itraconazole, voriconazole, posaconazole
- **ALLYLAMINE** – terbinafine
- **OTHER TOPICAL AGENTS** – tolnaftate,amorolfine, undecylenic acid, benzoic acid, quiniodochlor, ciclopirox olamine, butenafine, sodium thiosulfate.

NON-PHARMACOLOGICAL TREATMENT

1. Hygiene Measures

Shower or bathe the affected skin area regularly.
Shower daily with mild soap and water; dry thoroughly particularly in skin folds, between toes, and groin area.
Do not share personal items (towels, clothing, combs, shoes, bedding).

2. Clothing and Shoes

Wear loose, well-ventilated clothing (e.g., cotton materials). Change clothing, socks, and undergarments every day. For tinea pedis (athlete's foot), use open shoes or sandals to promote air circulation. Do not use tight or synthetic shoes and clothing that trap moisture.

3. Environmental Control

Wash or disinfect soiled items (towels, hats, combs, bedding) in hot water and dry them under the sun.
Clean shower floors and showers regularly, particularly in communal areas (locker rooms, gyms, dorms).
Utilize antifungal powders or sprays in shoes if needed.

4. Behavioral and Lifestyle Measures

Refrain from walking barefoot in public.
Refrain from close skin contact with infected people or animals.Treat household contacts or pets if they develop evidence of infection to avoid re-infection.

REOCCURENCE OF TINEA INFECTION

Recurring dermatophyte infections like ringworm, athlete's foot, or tinea cruris are frequent and may be annoying. Recurrence typically occurs because the primary cause or the source of infection is not completely cleared. Below are the chief causes of recurrence

1. Inadequate treatment- Premature discontinuation of antifungal therapy, Inadequate dosing or application of low-strength antifungal creams. Inadequate treatment of all affected areas, particularly in spreading or mixed infections.
2. Reinfection from untreated contacts or fomites
3. Inadequate hygiene or moisture control
4. Misdiagnosis or mixed infection
5. Antifungal resistance
6. Host factors

Diabetes mellitus or obesity (promoting moist skin folds).
Immunosuppression - HIV, corticosteroid treatment, organ transplant.
Poor nutrition or chronic diseases compromise skin defenses.

ANTIFUNGAL RESISTANCE

Alteration of Drug Target
Mechanism: Mutation in the gene for the antifungal target protein decreases the binding affinity of the drug.
Examples: Azole resistance in Candida albicans mutations in ERG11 (lanosterol 14α-demethylase) decrease azole binding.

Overexpression of Drug Target

Mechanism: The fungus overproduces the target enzyme, and therefore higher concentrations of the drug are required to inhibit it.
Example: Overexpression of ERG11 results in decreased susceptibility to azoles.

Efflux Pump Overactivity

Mechanism: Overexpression of membrane transport proteins that actively pump the drug out of the fungal cell. Transporter families involved - ATP-binding cassette (ABC) transporters (e.g., CDR1, CDR2 in Candida albicans)
Result: Reduced intracellular drug concentration.

Bypass Pathways or Compensatory Mechanisms

Mechanism: The fungus employs an alternative biochemical pathway to circumvent the drug's target.
Example: Certain fungi upregulate secondary ergosterol biosynthesis pathways as a response to azole inhibition.

Reduced Drug Uptake

Mechanism: Changes in cell wall or membrane permeability decrease entry of antifungal drug.
Example: Decreased flucytosine (5-FC) uptake resulting from mutations in FCY2 (cytosine permease) or FUR1 (uracil phosphoribosyltransferase).

Biofilm Formation

Mechanism: Fungal biofilms form an extracellular matrix that repels the antifungal and changes metabolic processes.Biofilm cells are 1,000× more resistant to antifungals (particularly azoles and amphotericin B).

Stress Response and Adaptation

Mechanism: Induction of stress response pathways (e.g., Hsp90, calcineurin) enhances survival during drug stress.
Effect: Increases tolerance and provides time for stable resistance mutations.

CONCLUSION

Tinea infections remain a major global dermatologic burden because of their multiple clinical presentations, changing therapeutic challenges, and rising antifungal resistance. A definite comprehension of the multifarious

clinical patterns and presentations facilitates correct diagnosis and proper management. Identification of predisposing risk factors and the history of recurrence are critical to avoid chronicity and reinfection. The increasing trend of antifungal resistance, especially to agents in regular use, highlights the importance of wise use of drugs, compliance with treatment guidelines, and continued monitoring of resistance patterns. Future approaches should target early diagnosis, patient education, enhanced hygiene protocols, and the discovery of new antifungal drugs to improve the effectiveness of treatment and prevent recurrence.

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