JAYPEE

Comprehensive Approach to Infections in Dermatology

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Foreword
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Superficial Fungal Infections

Chander Grover, Suruchi Vohra

ABSTRACT

Infections caused by pathogenic fungi which are limited to the stratum corneum are known as superficial fungal infections. These infections are usually contagious and are prevalent worldwide.

Superficial fungal infections are a major cause of morbidity around the world. Their recognition and diagnosis is important in day-to-day practice because of high incidence, worldwide distribution and ease of transmission. Superficial fungal infections can be further divided into:

- Infections inducing a host inflammatory response (e.g., dermatophytosis) and
- The ones which are completely asymptomatic (e.g., piedra).

The most common types of superficial mycoses are: (1) dermatophytoses caused by various dermatophytes, which are the largest group of septate fungi present in our environment which have the potential to infect skin, hair, and nails; (2) pityriasis versicolor caused by *Malassezia furfur* (*Pityrosporum orbiculare*) which infects the skin, usually the trunk (pityriasis versicolor). Both organisms are otherwise normal human commensals; (3) candidiasis caused by yeasts. *Candida* species cause infections of the mucous membranes, skin and fingernails (candidiasis or thrush). In this chapter, in addition to the dermatophytosis, candidiasis, pityriasis versicolor, we will be covering tinea nigra and piedra as well.

INTRODUCTION

Cutaneous fungal infections (cutaneous mycoses) can be classified as *superficial, subcutaneous or systemic mycoses.* Of these, superficial fungal infections are the ones most commonly encountered. Herein, the infection remains confined to stratum corneum of skin, hair, or nails. Subcutaneous mycoses are caused by direct inoculation of the causative fungus into the skin or subcutaneous tissue generally by a foreign object, whereas systemic mycoses are characterized by disseminated infection. This chapter focuses on the various superficial fungal infections.

Irrespective of the infecting fungal species, there are a few general diagnostic principles which are useful in diagnosing the majority of these infections. These principles are summarized in the section below. Appropriate modifications to the techniques or any special requirements when a particular organism is suspected will be outlined in the respective sections.

LABORATORY DIAGNOSIS OF SUPERFICIAL FUNGAL INFECTIONS

Many cutaneous disorders can masquerade the superficial mycoses. So it becomes important to confirm the

diagnosis by various laboratory methods; before starting the patients on antifungal therapy. The laboratory diagnosis of superficial fungal infections mainly involves direct examination under microscope and species identification by culture.

Sampling Technique

The result of the laboratory tests depends on the sampling technique; hence, it is important to take samples properly to avoid false negative results. The sampling technique for various sites is given below in Table 1.

The specimen thus collected can be examined directly in KOH preparation or transported in a dry folded piece of paper after properly labeling it. It is important to avoid moisture to avoid rapid multiplication of bacteria.

Wood's Lamp Examination

Wood's lamp is a simple handy tool which is useful in diagnosing some of the superficial fungal infections including tinea capitis and pityriasis versicolor (Fig. 1). Wood's lamp emits UVA light and is fitted with a filter composed of barium silicate and 9% nickel oxide. Fluorescence of tissues occurs when Wood's (UV) light is absorbed and radiation of a longer wavelength, usually visible light, is emitted. Small-spored ectothrix infection caused by *M. canis* and *M. audouinii* produces brilliant green florescence under Wood's lamp. The causative agent of favus, *T. schonleinii* can also produce pale green florescence. Similarly, pityriasis versicolor shows pale yellow to white florescence.

TABLE 1: Sampling techniques when a superficial fungal infection is suspected

Skin	The blunt end of the clean scalpel blade can be used to scrape the scales from the border of the lesion
Hair	In case of black or white piedra, hair can be cut at the skin surface. While in tinea capitis, the hair should be plucked with the forceps. Skin scrapings can also be taken from scalp in case of gray patch
Nail (finger and toe nails should be sampled if both are affected)	The hyperkeratotic nail bed can be scraped off with the blunt end of disposable scalpel blade. The whole thickness of the nail plate can also be taken with the help of nail clipper

Fig. 1: Wood's lamp commonly used in dermatology clinics

Direct Microscopic Examination

Direct examination under microscope is the simplest and quickest method to diagnose superficial fungal infections. A thin layer of sample collected is mounted on a glass slide with potassium hydroxide (KOH 10–30%). The KOH causes destruction of keratin squames without affecting the fungus. Gentle heating of the slide accelerates the process, which might be needed in nail specimens. The skin and hair samples can be seen under the microscope immediately, while nail samples usually take few hours for preparation. The nail samples can be kept overnight before reporting it as negative. The alternative is addition of 35% dimethylsulfoxide (DMSO), dimethyl acetamide or dimethyl formamide to KOH to hasten the process of softening. Apart from this, some stains like Congo red, methylene blue, cotton blue and Parker's blue ink can be used to increase the contrast between the hyphae and the debris. Florescent microscopes if available can be helpful by staining the samples with calcofluor white (CFW) or acridine orange. The sensitivity of this test is high for cutaneous infections but varies from approximately 50–85% for cases with onychomycosis.

Fungal Culture

Fungal culture is essential in cases where long term systemic antifungal therapy is required for example onychomycosis and tinea capitis. A positive test takes around 7–21 days depending upon the causative fungus. The most common primary culture medium used is Sabouraud's dextrose agar (SDA) which is

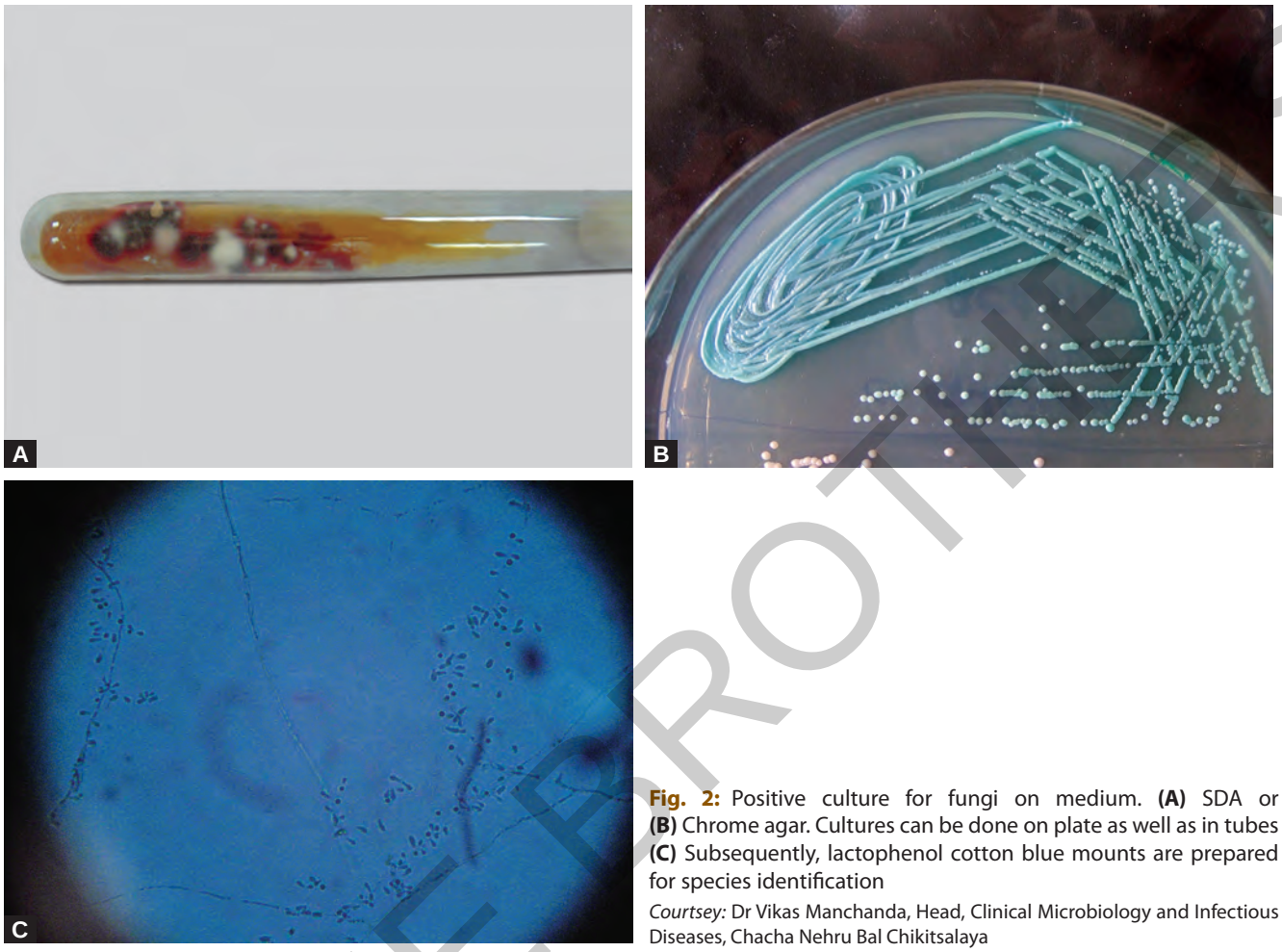

Fig. 2: Positive culture for fungi on medium. **(A)** SDA or **(B)** Chrome agar. Cultures can be done on plate as well as in tubes **(C)** Subsequently, lactophenol cotton blue mounts are prepared for species identification

Courtsey: Dr Vikas Manchanda, Head, Clinical Microbiology and Infectious Diseases, Chacha Nehru Bal Chikitsalaya

composed of 4% sugar, 1% peptone and has an acidic pH. Antibacterial agent like chloramphenicol (0.005%) or gentamicin (0.0025%) is usually added to avoid bacterial contamination of the growth. Additionally, to ensure isolation of dermatophytes, cycloheximide (0.04%) is also added to inhibit nondermatophytic mold (NDM) growth (Fig. 2).

DERMATOPHYTOSIS

Introduction

Dermatophytoses are superficial fungal infections caused by dermatophytes. These are a group of molds which are predominantly environmental pathogens belonging mostly to various asexual genera. Some of the species may be zoophilic or anthropophilic as well.

Etiology

Dermatophytes are a group of related fungi capable of breaking down and digesting the keratin in skin, hair and nails. These are mostly environmental pathogens, but based on their preferred habitat or ecological niche,[1] they can be classified as geophilic, anthropophilic and zoophilic organisms (Table 2).

Mycologically, most of these dermatophyte species are named according to their sexual or asexual forms. The asexual forms, which reside in vertebrate host, are classified into three main genera: *Microsporum, Trichophyton and Epidermophyton.* The sexual forms have been classified into a single genus known as *Arthroderma.* The three asexual forms can be distinguished on the basis of morphology of the multicellular macroconidia produced by them (Table 3). Expectedly, this differentiation is not

TABLE 2: Classification of medically significant dermatophyte species on the basis of their ecological niche

Dermatophyte species	Habitat	Mode of transmission	Immune response	Worldwide cases
Geophilic (originating in soil) *Microsporum gypseum* *Microsporum praecox*	Soil	Contact with soil	Inflammatory	Sporadic
Anthropophilic (largely restricted to humans) *Epidermophyton floccosum* *Microsporum audouinii* var. *rivalieri* *Microsporum audouinii* var. *langeronii* *Microsporum ferrugineum* *Trichophyton concentricum* *Trichophyton gourvilii* *Trichophyton mentagrophytes* var. *interdigitale* *Trichophyton megninii* *Trichophyton rubrum* *Trichophyton schoenleinii* *Trichophyton soudanense* *Trichophyton tonsurans* *Trichophyton violaceum* *Trichophyton yaoundei*	Humans	Direct contact or fomites	Varies, non-inflammatory to inflammatory	Epidemic
Zoophilic (commonly infecting animals) *Microsporum canis* var. *canis* *Microsporum canis* var. *distortum* *Microsporum equinum* *Microsporum gallinae* *Microsporum nanum* *Microsporum persicolor* *Trichophyton equinum* *Trichophyton mentagrophytes* var. *mentagrophytes* var. *erinacei* var. *quinckeanum* *Trichophyton simii* *Trichophyton verrucosum*	Animals (cats, dogs)	Direct contact with animal or indirectly through infected hair in the clothing	Inflammatory (spontaneous cure may occur)	Sporadic

TABLE 3: Differentiation of dermatophyte genera on the basis of characteristics of macroconidia produced

Genus	Shape of macroconidia	Character of macroconidia	Number of septae
Microsporon	Fusiform or obovate	Thick-walled, rough	1–12 septae
Trichophyton	Cylindrical, fusiform or clavate	Thin-walled, smooth	Up to 12 septae
Epidermophyton	Clavate, broadened	Thin-walled, smooth	Up to 5 septae

possible under light microscopy (in KOH mount), as microcondia or macroconidia are not produced in the parasitic phase of the fungal growth, which is encountered *in vivo*.

Epidemiology

All dermatophytic infections are more common in regions with hot and humid climate. In such tropical climates, dermatophytoses constitute the major source of skin infections. In India, tinea corporis is the most common type of dermatophytic infection reported, followed by tinea cruris.[2,3] Tinea capitis is more commonly seen in children.

Pathogenesis

The source of infection is the arthroconidia or spores deposited on the host surface. Dermatophytes colonize the stratum corneum and use keratin as the source of nutrition. As under normal circumstances, they are incapable of penetration into the viable layers of skin; their *in vivo* activity is restricted to specific zones like the newly differentiated keratin of skin; the nail plate and the Adamson's fringe within the hair shaft.[4]

Under favorable circumstances, the inoculated arthroconidia undergo well-characterized phases of growth to produce clinically manifest infection.[4]

- *Adherence:* The arthroconidia adhere to the keratinocyte through, as yet unknown mediators. This adherence is immediately followed by their germination into hyphal element. *In vitro* models have revealed the presence of fibril-like structures on the arthroconidia which help them in attaching to the keratinocyte surface[5]
- *Penetration:* The dermatophyte hyphal elements secrete an arsenal of proteases which are capable of breaking down the keratin.[6] It is postulated that mechanical factors may also play a role in this invasion. The site preference of certain dermatophyte species (e.g., *Epidermophyton floccosum* rarely infect the hair follicle and occasionally the nail) may be explained on this basis
- *Host response*: The fungal penetration invokes both innate as well as acquired immunity in the host. The antimicrobial peptides like cathelicidins and human beta-defensins, which have antifungal activity, are proposed to be the main factors involved. Apart from this, certain serum factors like transferrins also have inhibitory effect on fungal growth. These molds have chemotactic properties and can also activate the alternate pathway of the complement system.

The cell mediated immune response is the protective immune response and is primarily a delayed type of hypersensitivity. The dermatophyte antigens are taken up by the Langerhan's cell present in the epidermis and are carried to the local lymph nodes, where they are presented to CD4+ T lymphocytes. The sensitized T lymphocytes proliferate and migrate to the dermis to produce the inflammatory response. Patients with widespread infection may also have detectable levels of antibodies to the dermatophyte antigen, though they do not appear to be protective.

Many other factors like host's age, sex, race, rate of sebum production (sebum has inhibitory action), break in the skin barrier, immune status, glucocorticoid usage, etc. may affect the clinical response and pattern. In diabetics, the prevalence of dermatophytoses is not reported to be increased unlike superficial bacterial infections. An association between atopy (characterized by increased IgE levels) and chronic dermatophytic infection has also been reported.[7]

Clinical Features

Traditionally, the dermatophytoses are classified on the basis of body site involved: tinea corporis, tinea barbae, tinea faciei, tinea capitis, tinea pedis, tinea manuum, tinea cruris and tinea unguium. Owing to unique characteristics afforded by the different sites involved, we shall discuss these manifestations accordingly.

Tinea Corporis

Introduction

It is the dermatophytic infection of the glaborous skin except for palms, soles and groins.

Epidemiology

Tinea corporis is usually seen in hot, humid climates. Although, it can occur in all age groups, but it is more common in adults.

Etiopathogenesis

All dermatophytes species are reported to cause tinea corporis, but the commonest ones involved are *T. rubrum, T. mentagrophyte, M. canis* and *M. tonsurans.*

Incubation period

Post inoculation, the reported incubation period is 1–3 weeks.

Clinical features

The clinical presentations may vary depending on the species involved and host immunity. In less inflammatory cases, there are characteristic annular plaques with raised erythematous borders (Fig. 3). The central clearing seen is due to the inflammatory response against the pathogenic fungi causing the fungi to migrate centrifugally in the horny layer of the epidermis; however, central clearing may not always be present. The presence of scales at the erythematous borders is generally appreciated. In severe inflammatory cases, there may be vesicles and pustules at the margin, instead of scales. Rarely, bulla formation can also occur especially with *T. rubrum*.

Apart from this prototype of tinea corporis, various *special types* have also been described.

- *Tinea faciei* (dermatophytic infection of the skin of the face) is most commonly caused by *T. rubrum* and *T. mentagrophytes*. Apart from itching, patient may complain of burning sensation after exposure to the sun. These lesions are frequently modified by application of topical corticosteroids (Fig. 4)
- *Tinea imbricata* is a special subtype, caused by *T. concentricum* (anthropophilic), especially seen in South Asia, Mexico and Brazil. It presents as multiple concentric polycyclic erythematous lesions with scales at the border. This type is usually chronic and may involve the whole body. This dramatic clinical picture has been described due to *T. concentricum*, which is confined to endemic areas and is associated with negative delayed-type hypersensitivity to *T. concentricum*[8]
- *Tinea indecisiva* (commonly caused by *T. mentagrophytes* and *T. tonsurans*) clinically manifest as erythematous annular plaques with multiple concentric rings with in the plaque resembling tinea imbricata (Fig. 5) caused by *T. concentricum.*[8] Long-term cyclical therapy with topical antifungals and corticosteroids may produce this manifestation due to similar underlying mechanisms of immunosuppression with topical corticosteroids and reinfection due to early discontinuation of topical antifungals[8,9]

Fig. 3: Annular lesions of tinea corporis

Fig. 4: Tinea faciei in a young male

Fig. 5: Concentric rings in a case of tinea indecisiva, extensive involvement over the trunk can be seen

- *Majocchi's granuloma* (most commonly caused by *T. rubrum*) is a type of deep folliculitis, most commonly seen in the women who regularly shave their hair. It presents as perifollicular pustules and nodules with surrounding erythema (Fig. 6)
- *Tinea incognito* or tinea modified by topical or systemic corticosteroids is generally a result of suppression of inflammation and is difficult to recognize due to absence of raised margins and scales (Fig. 7)
- Other rare forms described in literature are *subcutaneous mycetoma like, agminate folliculitis* (plaque studded with pustules at the periphery) and *subcutaneous abscesses*.

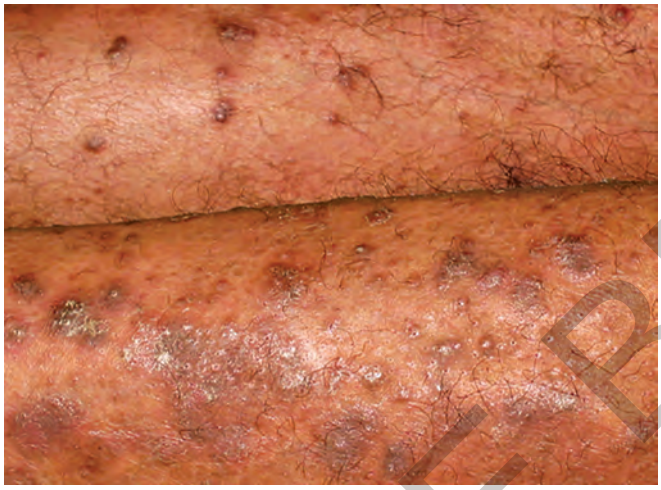

Fig. 6: Deep-seated inflammation on the legs in a case with extensive tinea corporis

Fig. 7: Steroid modified tinea or tinea incognito. Note the minimal to absent scaling

Box 1: Differential diagnosis of tinea corporis

- Seborrheic dermatitis
- Psoriasis
- Annular erythema
- Pityriasis versicolor
- Pityriasis rosea
- Secondary syphilis
- Subacute lupus erythematosus

Differential diagnosis

The differential diagnoses are summarized in Box 1.

Tinea Cruris or Dhobi Itch

Introduction

It is an infection of the groins, perianal and perineal areas caused by dermatophytes.

Epidemiology

It is most commonly reported in young men, living in hot climates. The condition is more common in summer months and is associated with increased sweating and heavy manual work.

Etiopathogenesis

Although *T. rubrum* is most commonly implicated, *T. mentagrophyte var. interdigitale* and *E. floccosum* may also be involved in some cases. The infection is a result of the moist conditions in the folds along with chronic friction.

Clinical features

Although, the morphology is similar to tinea corporis, maceration is a more prominent feature. Superadded bacterial infection may also develop. The lesions are highly pruritic (Fig. 8). Infection with *T. rubrum* may spread from groins to thighs, lower abdomen, back and buttocks. *Epidermophyton floccosum* is generally associated with fungal infection of the foot, while *T. mentagrophyte var. interdigitale* presents with more inflammation, vesicles and pustule formation at the margins.

Differential diagnosis

The differential diagnoses are summarized in Box 2.

Candidal intertrigo should be differentiated from cases of tinea cruris. The clinical clues toward tinea cruris are well-defined borders, less maceration, more scales and rare presence of satellite pustules. Some cases of

Fig. 8: Tinea cruris with involvement of groins. Extensive areas of involvement can be seen

Fig. 9: Tinea barbae with marked inflammation. Deep-seated lesions with scarring can be appreciated in the beard area

Box 2: Differential diagnosis of *tinea cruris*

- Candidal intertrigo
- Erythrasma
- Pityriasis versicolor
- Flexural psoriasis
- Seborrheic dermatitis

Box 3: Differential diagnosis of *tinea barbae*

- Syscosis barbae
- Acne vulgaris
- Rosea
- Perioral dermatitis

erythrasma and pityriasis versicolor may be confused with tinea cruris, though the two conditions do not show central clearing. Flexural psoriasis and seborrheic dermatitis may mimic tinea cruris.

Tinea Barbae

Introduction

Tinea infection of the beard and moustache area is known as tinea barbae; thus, it is seen only in males.

Epidemiology

This infection is more commonly seen in adult males in rural areas due to contact with livestock. It can also be transmitted through barbers who use shared razors.

Etiopathogenesis

It is most commonly caused by *T. mentagrophyte* and *T. verrucosum* (zoophilic species) and occasionally by other species like *M. canis*, *T. violaceum* and *T. schoenlenii*.

Clinical presentation

It typically presents as multiple folliculocentric papules and pustules with surrounding erythema (Fig. 9). There is associated exudation and crusting, quite resembling a kerion. The hairs in the area are easily pluckable. Chronic cases present with abscess and sinus formation, ultimately leading to scarring. Occasionally, less of inflammatory changes may be seen in infections with anthropophilic species which present with circular, scaly plaques.

Differential diagnosis

The differential diagnoses are summarized in Box 3.

The inflammatory variant of tinea barbae needs to be distinguished from sycosis barbae (deep folliculitis caused by *S. aureus*). Sycosis barbae is relatively more painful and the hair in the center of the pustule is only occasionally loosened up. Acne vulgaris, papulo-pustular stage of rosacea or cases of perioral dermatitis may mimic the less inflammatory variants of tinea barbae.

Tinea Capitis or *Tinea Tonsurans*

Introduction

Dermatophytic infection of the scalp hair follicles is known as tinea capitis.

Epidemiology

Majority of the cases occur in preadolescent age group, though rarely adults can also develop this infection. There is no gender predilection. It is common in India, Africa and certain urban areas of North, Central and South America. It is common in school-going children and in households with overcrowding.

Etiology

All dermatophytic molds are known to invade the hair shaft except for three species *T. concentricum*, *T. mentagrophyte var. interdigitale*, and *E. floccosum*.

Epidemiology

Tinea capitis is a worldwide problem. The predominant species causing tinea capitis varies from region to region and time to time. The most common species worldwide is *M. canis*, though recently there is an increase in *T. tonsurans* prevalence especially in US and western Europe.[10] The incidence of tinea capitis in India varies from 0.5 to 10%.[11-13] In India, *T. violaceum* is now the most common species isolated[14,15] while in United States, the predominant species causing this infection is *T. tonsurans.*[16,17]

Pathogenesis

The arthroconidia in the air get trapped into the scalp hair and invade the scalp skin at the level of stratum corneum. The incubation period is approximately 3 weeks. Three main patterns of hair shaft invasion by the dermatophytic molds have been described. These include *ectothrix pattern* where the fungus penetrates at the midfollicular level of a mid-to-late anagen phase of hair follicle, and grows downward toward the hair bulb until the upper border of keratinized zone. At this zone, also known as the Adamson's fringe, the fungus multiplies in equilibrium with the rate of keratinization, thus invading the newly keratinized cells. There are two types of ectothrix spores. The small-spored ectothrix is seen with *M. canis, M. audouinii, M. equinum* or *M. ferrugineum* species mainly. Secondary extrapilary hyphae grow out in tortuous manner and then segments into a mass of small arthroconidias of size 2–3 μm. This type shows green fluorescence under Wood's lamp. The large-spored ectothrix pattern is a result of infection with *T. mentagrophyte var. mentagrophyte*, *T. verrucosum*, *M. gypseum,* and *M. fulvum.* Here, the primary straight extrapilary hyphae break into chain of large arthroconidias of size 5–8 μm on the surface of hair follicle and there is no fluorescence on Wood's lamp examination.

In the *endothrix pattern*, the penetration is in a manner similar to ectothrix but the intrapilary hyphae of *T. tonsurans* and *T. violaceum* species break into multiple large arthroconidias (8–10 μm) inside the hair shaft making it weak and fragile. As a result, the hair follicle breaks at the level of skin surface leaving behind black dots while the cortex remains intact.

The third pattern *favus* is caused by *T. schoenleinii.* The intrapilary hyphae, rather than breaking up into arthroconidia, remain intact inside the hair shaft. As a result, the hair grows and attains normal length unlike endothrix, forming tunnels inside the hair follicle, which appear as air spaces under KOH mount.

Clinical features

The clinical presentations of tinea capitis vary greatly, depending on the causative species, type of hair invasion and level of host resistance. The appearance may vary from noninflammatory types like gray patch to severe inflammatory variants like kerion. Inflammatory variants may have associated cervical or occipital lymphadenopathy. However, a great degree of overlap and coexistence of different patterns exists.[15] The common clinical presentation in all forms is partial hair loss.

- *Gray patch or noninflammatory tinea capitis* is generally associated with *M. audouinii or M. ferrugenium* which produces a small-spored ectothrix type of penetration. It is typically seen as well-circumscribed round, patches of noncicatricial alopecia, with multiple broken stumps of gray, lusterless hair and mild scaling (Fig. 10). The arthroconidia coating the

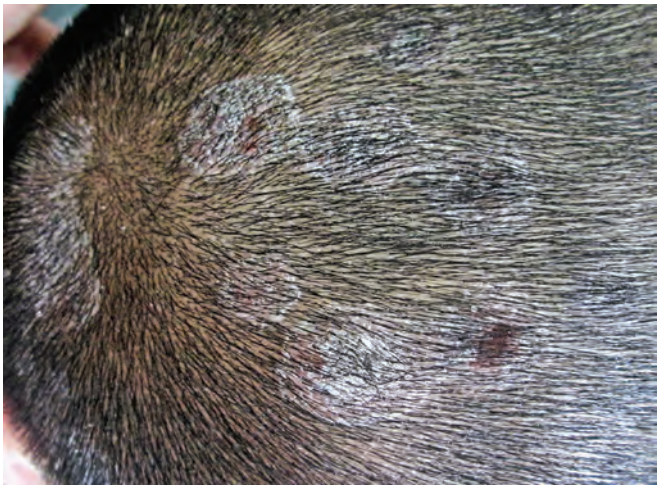

Fig. 10: Gray patch tinea capitis

hair give rise to the gray appearance. *Microsporum canis canis* may produce a similar picture albeit with a little more inflammation

- *Black dot variant* is caused by species like *T. tonsurans, T. violaceum and T. soudanense* which produce an endothrix pattern of invasion. It is characterized by grouped black dots (swollen hair shafts) with diffuse scaling (Fig. 11). The inflammation may vary from mild scaling to a frank pustule. The lesions are usually multiple and have angulated borders unlike gray patch
- *Inflammatory types* are usually seen with zoophilic species like *T. mentagrophyte var. mentagrophyte* and *T. verrucosum* and geophilic species like *M. gypseum*. This is characterized by a hypersensitivity response to the invading fungi. The clinical presentation may vary from follicular pustule (Fig. 12) to frank *kerion* (an inflammatory, painful boggy swelling studded with follicular pustules) with associated lymphadenopathy (Fig. 13). Self-resolution may occur in some cases, but scarring is usual. The affected patients may also develop an id eruption
- *Favus* is caused by *T. schoenleinii,* occurring sporadically, mainly in South African countries, Middle East and Pakistan. It is long-standing infection which may extend many years and is characterized by yellow-colored scutulum around the hair follicles, which may become confluent to form yellow crusts. The infection is usually chronic with little tendency for self-resolution and with risk of development of cicatricial alopecia.

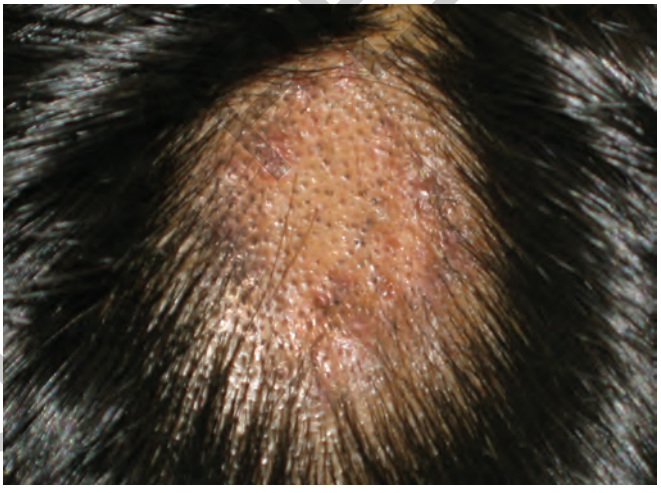

Fig. 11: Black dot tinea capitis

Fig. 12: Inflammatory tinea capitis

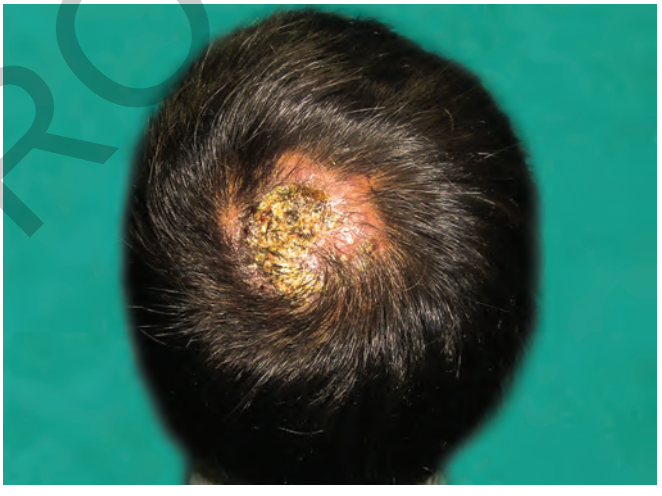

Fig. 13: Kerion

Differential diagnosis

The differential diagnoses are summarized in Box 4.

Box 4: Differential diagnosis of *tinea capitis*

- Seborrheic dermatitis
- Psoariasis
- Atopic dermatitis
- Alopecia areata
- Trichotillomania
- Bacterial folliculitis or impetigo
- Folliculitis decalvans

Comprehensive Approach to

Infections in Dermatology

Key Features

- The book focuses on infections encountered in dermatologic practice, wherein these are discussed in detail from a dermatological perspective
- All important and relevant skin infections–bacterial, fungal, viral, mycobacterial (including tuberculosis and leprosy), parasitic and protozoal, and sexually transmitted infections and infestations have been included with detailed accounts of their epidemiology, varied clinical details, differential diagnoses, and management approaches, including diagnostic guidelines
- Text has been summarized in to key points at frequent intervals for ease of retention
- Ample number of good quality, representative photographs have been included to aide easy recognition both by practitioners and students
- Additional information on the diagnostic procedures resorted to by the clinicians have been included. A step-by-step approach to bed side diagnostic procedures makes this book a valuable reference guide
- Liberal use of clinical photographs, tabulation of data, graphics, and inclusion of the latest therapeutic guidelines and references make this book a reader friendly and must have manual not only for dermatologists but for pediatricians, physicians, and general practitioners alike.

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