Ministry of Higher Education

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University of Diyala

College of Medicine

A Review Article :

Tinea

Submitted to the council of the College of Midicine, Diyala University, In Partial Fulfillment of Requirements for the Bachelor Degree in Medicine

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Dedication

I dedicate this study with much Gratitude and Love to;

His words of inspiration and encouragement in pursuit of excellence.

My Affectionate Mother;

Whose prayers and love took me to zenith of glory and transform my dreams into reality

My Brothers & Sister;

Which always encouraged and supported me.

Finally, to My Friends.

The content

Introduction

Dermatophytes (family Arthrodermataceae) are a group of fungi in the order Onygenales which have evolved relatively recently, almost exclusively with mammal hosts[1] .

Dermatophytes are a unique group of fungi that are capable ot intecting nonviable keratinize cutaneous tissues including stratum corneum, nails, and hair. Dermatophytic genera include Trichophyion, Microsporum, and Epidermophyton. The term dermatophytosis thus denotes a condition caused by dermatophytes. It can be further specified according to the issue mainly involved: epidermomycosis, trichomycosis, or onychomycosis. he term linea should be reserved for dermatophytoses and is modified according to the anatomic site of intection, e.g., tinea pedis.

Epidemiology

Only" Microsporum gypseum'* has been recovered to any extent from human infections. Several other dermatophytes have been isolated from soil, and may occasionally be isolated in the laboratory, particularly when material from animals is being cultivated. Not all of these have been shown to be pathogenic. An interesting outbreak due to M. gyp. seum was described by Whittle (6) in carnation greenhouses in the Cambridge area, and a similar one by Alsop & Prior[2] .

Pathogenesis of dermatophytoses

The secretion of proteolytic enzymes by dermatophytes is a key factor in their invasion and subsequent dissemination through the stratum corneum of the host. During the first stages of infection, dermatophytes respond to the skin by de-repressing a number of genes coding for proteins and enzymes such as adhesins, lipases, phosphatases, DNAses, non-specific proteases, and keratinases. These proteins have their optimal activity at acidic pH values, which matches the acidic pH of human skin, allowing the pathogen to adhere and penetrate the host tissue, scavenge nutrients and overcome host defence mechanisms. The conserved PacC/Rim101p signal transduction pathway mediates diverse metabolic events involved in ambient pH sensing and in the virulence of pathogenic microorganisms. The seven dermatophyte genomes analysed here revealed the presence of the PacC/Rim101p pH-responsive signal transduction pathway, which consists of the six pal genes (palA, B, C, F, H and I) and the transcription factor PacC. The PacC binding site was present in the promoter regions of pacC, palB, palI and palH genes of all dermatophytes, suggesting

functional equivalency with the signalling cascade of other fungi. Moreover, the promoter region of pacC gene of the seven dermatophytes had multiple PacC DNA-binding sites, suggesting that these genes, like their homologues in model fungi, are auto-regulated[3].

Types of Tinea[4]:

1- Tinea Corporis

2- Tinea Capitis

3- Tinea Barbae

4- Tinea Facialis

5- Tinea Pedis

6- Tinea Manum

7- Tinea Cruris

8- Tinea Unguium

Tinea corporis and cruris

Definetion :

Tinea corporis and tinea cruris are superficial dermatophyte infections, commonly known as ‘‘ringworm.’ Tinea corporis includes all superficial dermatophyte infections of the glabrous skin, excluding the scalp, beard, face, hands, feet, and groin. Tinea cruris includes infections of the genitalia, pubic area, perineal skin, and perianal skin.

Etiology and epidemiolog

Tinea corporis and tinea cruris may be caused by any of the dermatophytes making up the genera Trichophyton, Microsporum, and Epidermophyton[5]. Both conditions are common throughout the world, with men being affected by tinea cruris more frequently than women. The causative organism can invade both the stratum corneum and the terminal hair of the affected areas[6]. Once infected, scales may be transmitted through direct contact between individuals, or indirectly through contact with objects that carry the infected scales[7] .This

transfer of infection is thought to occur through arthroconidia that are shed by the infected host in skin scales[8] . Autoinfection by other dermatophytes elsewhere in the body, especially the foot to the groin, may also be a method of contracting a tinea infection[9] . Children are frequently infected with M canis, another causative organism of tineacorporis ,especially those exposed to infected animals, such as cats, dogs, horses, or cattle. Infection may also be transmitted by transfer of spores from the skin or hair of a child to another host[10] . The most common predisposing factor for most dermatophyte infections in adults is excessive perspiration. In addition, occlusive clothing may provide an environment where the dermatophyte organisms can thrive. Individuals involved in contact sports, such as wrestling, football, or rugby, may also be at risk of acquiring a tinea infection[11] .

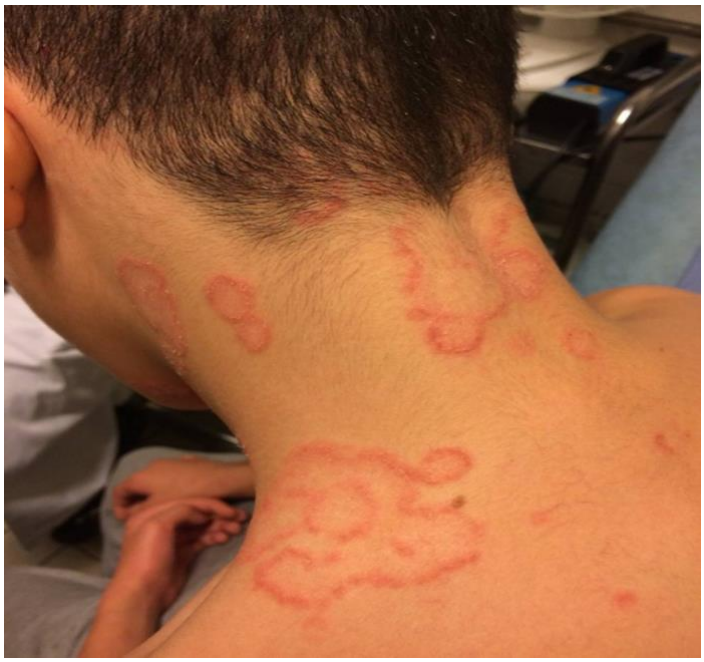
Clinical manifestation

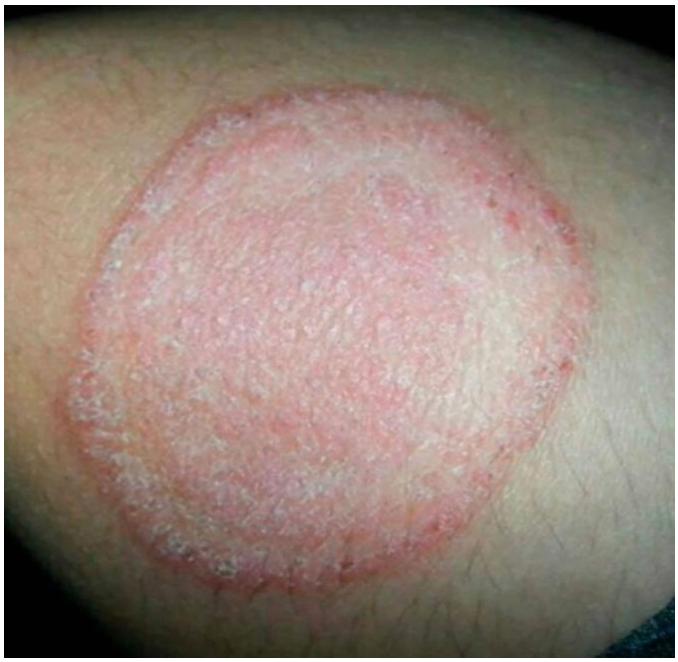
Tinea corporis and tinea cruris infections may present as an annular erythematous plaque with a raised leading edge and scaling. Clearance occurs in the center of the lesion; however, resolution is often incomplete, because nodules may be left scattered throughout the infected area[6] . The clearance in the center of the lesion may be the manifestation of an immune response of the host to the infecting organism[6] . Pruritus is a common symptom, and pain may be present if the involved area is macerated or secondarily infected[12] . The lesion of tinea cruris extends from the groin down the thighs and backward on the perineum or about the anus; the scrotum and labia majora are generally excluded[13] . Tinea corporis can also present in a non-ringworm fashion, where it may manifest as an erythematous papule or a series of vesicles[8] . When a zoophilic dermatophyte, such as Trichophyton verrucosum, is the responsible organism, an intense inflammatory reaction can result in large pustular lesions or a kerion[12] . In addition, occasionally frank bullae may appear as an expression of the inflammation, causing tinea corporis bullosa[14] . When viable hyphae invade and track down the hair shaft and into the dermis, perhaps because of trauma caused by shaving, inflammatory papules and pustules may develop. In addition, erythema and perifolliculitis may also be part of the clinical picture of Majocchi's granuloma[13] .

Differential diagnosis

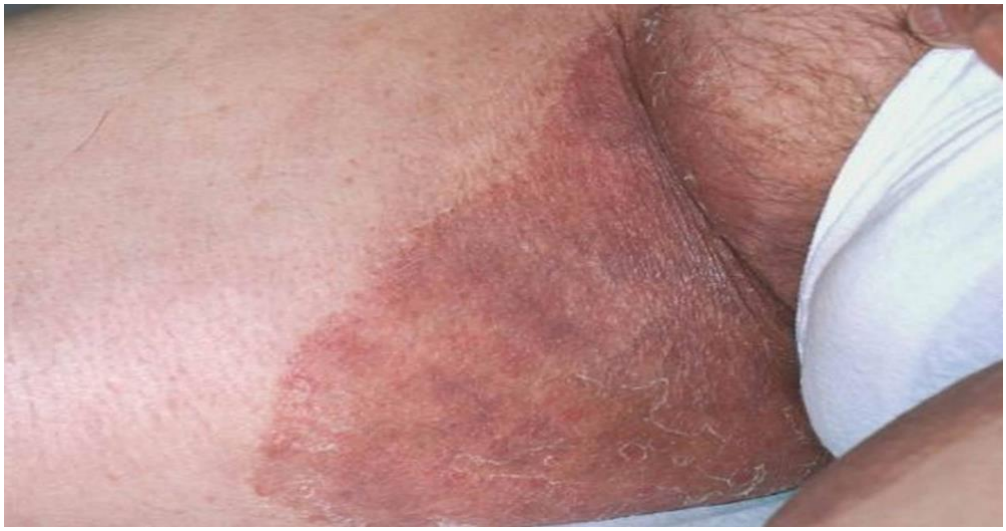
Other diseases closely resembling tinea corporis are impetigo, nummular dermatitis, and secondary and tertiary syphilis[15] . A tinea corporis eruption that is more papulosquamous in presentation may be mistaken for psoriasis, lichen planus, seborrheic dermatitis, pityriasis rosea, or pityriasis rubra pilaris[12] . The crural region may be infected by other dermatoses that present comparable clinical features as tinea cruris. Psoriasis, seborrheic dermatitis, candidiasis, erythrasma, lichen simplex chronicus, Darier's disease, and

pemphigus vegetans may be mistaken for tinea cruris. Cutaneous candidiasis, which oftenaffects women, may be distinguished from tineacruris of males. Satellite lesions and white pustules of Candida may affect the scrotum, whereas dermatophytes do not. Erythrasma produces a coral fluorescence under Wood's light, which is not seen in tinea cruris[16] .





Tinea corporis



Tinea cruris

Diagnosis and laboratory findings

Because of the broad range of differential diagnosisof dermatophyte infections, it is important to perform a mycologic examination, consisting of a 10% to 15% KOH preparation, from skin scrapings, and a fungal culture on Sabouraud's agar media. When tinea corporis or tinea cruris infection is suspected, examination of the infected scales from the leading edge of the lesion may reveal septate hyphae coursing through the squamas[12] . Cultures incubated at room temperature should grow the causative organism within 2 weeks.

Treatment

As in many cases of cutaneous fungal infections, topical therapy is sufficient, but systemic treatment is necessary when large areas of the body are involved, the incidence is chronic or recurrent, or when the infection is in immunocompromised patients[17] . Tinea corporis and tinea cruris respond satisfactorily to topical therapies, such as the azoles (sulconazole oxiconazole, miconazole, clotrimazole, econazole, and ketoconazole); the allylamines (naftifine and terbinafine); benzylamine derivative (butenafine); and hydroxypyridones (ciclopirox olamine). However, repeated application to large areas of the skin may not always be feasible or convenient for the patient. Thus, oral treatments may be preferred by the patient (Table 1)[18-23] . The use of oral ketoconazole has been limited by its rare association with hepatotoxicity[24] . Griseofulvin has a rapid disappearance from the stratum corneum after administration because it is not very keratophilic with poor binding with keratin. Patients may be at a higher risk of relapse[25] . The dosage of terbinafine is 250 mg/d given for 2 to 4 weeks. The triazoles, fluconazole and itraconazole, are also safe and effective treatments for tinea corporis and tinea cruris[22,24] . Fluconazole has shown high clinical and mycologic cure rates with once weekly therapy[26] . Itraconazole is effective when given a regimen of 200 mg daily for 7 days[27] . Topical corticosteroid application is a mistreatment and may lead to suppression of physical signs[6] and to the development of tinea incognito.

Tinea infection	Griseofulvin	Terbinafine	Itraconazole	Fluconazole	Ketoconazole
Tinea corporis And cruis	250 mg twice daily until cure is reached[18,19]	250 mg/d fore 2 to 4 weeks[20]	200mg/d for 1to wk[21]	150- 300 mg/wk for 2to4 wk[22]	200mg/d for 4 to 8 weeks[23]

Table (1) Systemic antifungal treatments for tinea corporis and tinea cruis

Prevention and control

Tinea corporis and cruris are dermatophyte infections particularly common in areas of excessive heat and moisture. A dry, cool environment may play a role in reducing infection[13] . In addition, avoiding contact with farm animals and other individuals infected with tinea corporis and cruris may help in preventing infection. In individuals with onychomycosis, it has been observed that there is a higher prevalence of tinea cruris; this may be the result of autoinfection acquired when the individual brushes fungal organisms onto the underwear following contact with the infected feet and toenails. In such an instance it may be prudent to cover the infected toenails by first putting on socks, followed by the undergarment.

Tinea nigra

Definition

Tinea nigra is an asymptomatic mycotic skin infection affecting the stratum corneum[12] . It occurs mainly on the palms, but may also involve the soles. Tinea nigra infection has also been reported on the neck and trunk.

Etiology and epidemiology

The organism responsible for tinea nigra, Hortaea werneckii (formally known as Phaeoannellomyces werneckii, Exophiala werneckii, and Cladosporium werneckii), is a dematiaceous fungus commonly found in nature. It has been isolated from superficial dermal lesions in humans, such as inflammatory scalp lesions; macerated interdigital lesions; and environmental sources (such as salted dried fish, soil samples, and house dust)[28,29] . Infection with H werneckii is thought to occur by inoculation through trauma[30] . Incubation times may range from a few weeks to 20 years, and is thought to produce clinical disease when a change in the balance between the fungus and the host occurs, causing the fungus to proliferate more rapidly[31] . The fungus adheres to the skin in a hydrophobic manner and can survive for prolonged periods in the environmental conditions prevailing on the skin because it is able to endure high salinity and low pH[32] . Tinea nigra typically occurs in children and young adults with female predominance[29] . It is commonly observed in patients living in warm countries or in those who have lived in or visited the tropics or subtropics and brought the infection back to North America[31] . Infection with

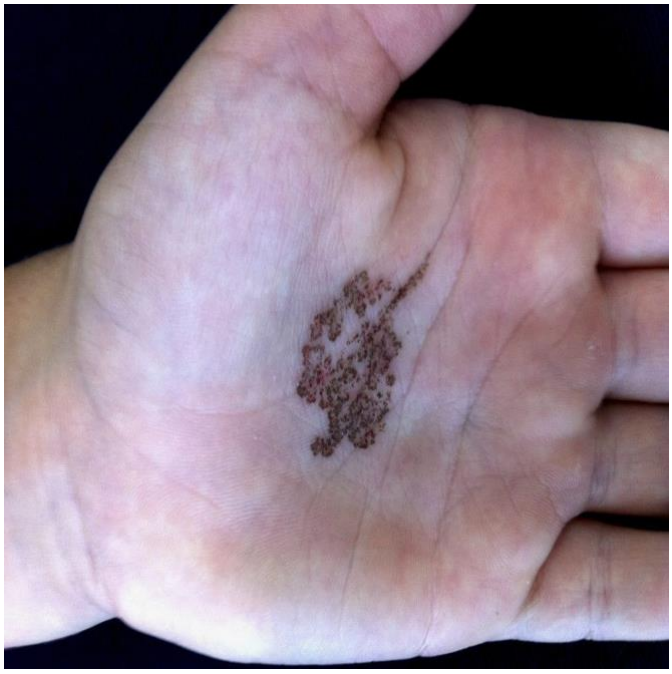
tinea nigra has been reported from South Africa, Brazil, Panama, Cuba, and Puerto Rico and many cases have been reported from the coastal areas of southeastern United States[33] .

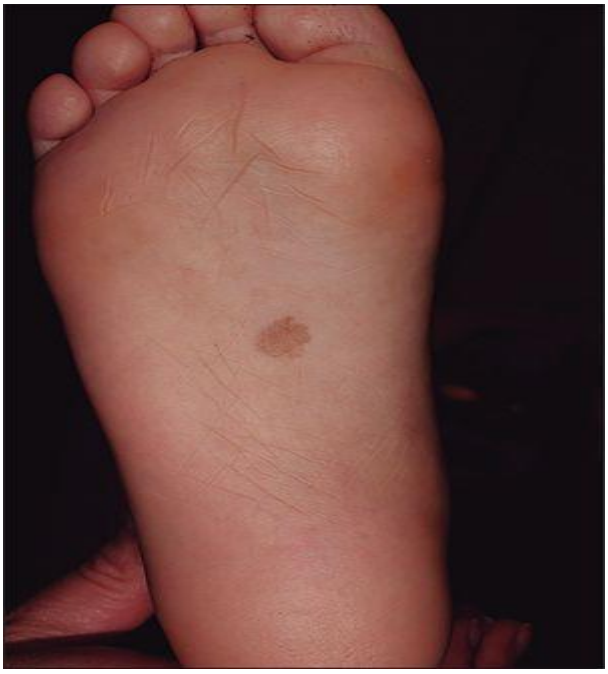
Clinical manifestation

Hortaea werneckii presents as a brownish black, velvety macular lesion that is neither elevated nor scaly, and occasionally pruritic. The lesion may darken, especially at the borders, while it gradually spreads at an uneven rate, producing an irregular outline[6,12] .

Differential diagnosis

Because of the similarity in color and growth of the lesion, tinea nigra is most frequently misdiagnosed with pigmented junctional nevus or malignant melanoma[34] . An accurate diagnosis of tinea nigra is important to prevent the diagnostic and excisional surgery[35] and concomitant scarring associated with treatment of nevomelanocytic lesions. Tinea nigra can also be mistaken for a lentigo, pityriasis (tinea) versicolor, drug eruption, chromhidrosis, contact dermatitis, syphilis, pinta, or staining from a variety of chemical or dyes[12,35] .





Diagnosis and laboratory findings

Culture of tinea nigra grows readily at room temperature, but sometimes slowly on primary isolation averaging 2 to 4 weeks before identification is possible[35] . Microscopic examination of scrapings of the stratum corneum reveals numerous dark-colored branching

septate hyphae and round to oval spores with some budding. The colonies are initimoist, shiny, black, and yeast-like[34] .

Treatment

Tinea nigra responds to treatments with keratolytics (Whitfield's ointment) and simple abrasion; however, topical imidazoles, such as 2% miconazole cream and 2% ketoconazole cream, are more popular[36] . Topical thiabendazole and ciclopirox olamine may also be effective[37,38] . Topical tolnaftate and oral griseofulvin are usually ineffective, and topical undecylenic acid gives variable results[39] .

Piedra

Definition

Piedra, meaning stone in Spanish, is limited to the hair shaft without involvement of the adjacent skin[40] . Two varieties of piedra may be seen: black piedra and white piedra.

Etiology and epidemiology

The causative organism of black piedra, P hortae, and Trichosporon ovoides, T inkin and T asahii, of white piedra have a worldwide distribution. Black piedra occurs frequently in humid, wet tropical areas and is common in certain tropical areas of central South America and Southeast Asia, whereas white piedra occurs in semitropical and temperate countries[41] . P hortae has been found on the hairs of animals, including primates, and stagnant water, soil, and vegetables[42] . It has been suggested that for some native populations, black piedra may have cosmetic importance[43] . The natural habitats of Trichosporon species are soil, lake water, and plants, and such fungi are occasionally seen as normal flora of the human skin and mouth[44] . White piedra has been found on animal hairs, including monkeys, horses, and lower mammals[45] . Infection with piedra does not seem related to personal hygiene or exposure to an infected person, nor does white piedra of the pubic hair seem to spread by sexual contact[46] .

Clinical manifestation

Black piedra is a condition that presents as astone-hard black nodule on the scalp, beard, moustache, and pubic hair shaft[47] . Brown-black hard nodules along the hair shaft characterize black piedra, with the fungal activity limited to the cuticle and with no penetration of the hair shaft. Black piedra is more frequent and less sporadic than white piedra. White piedra is characterized by white-to-tan nodules along the shafts of hair in the scalp, beard, eyebrows, eyelashes, and groin, genital and perigenital area[41] . Numerous

discrete, soft nodules that are barely visible to the naked eye are attached to the hair shaft, and produce a gritty sensation when palpated[48] . The nodules may be detached easily, and the affected hairs may be split or broken[40] . T asahii and T inkin can behave as opportunistic pathogens, particularly in immunosuppressed patients, where they can cause serious and life-threatening symptoms[49] .

Differential diagnosis

Clinically, many hair disorders can be confused with piedra[40] . White and black piedra should be distinguished from each other and nits, hair casts, developmental defects of the hair shaft, and trichomycosis axillaris[12] . Infections can co-exist with dermatophyte or *Candida* infections, and erythrasma[13] . White piedra should be differentiated from pediculosis[50] .

Diagnosis and laboratory findings

Infection with P hortae (black piedra) reveals tightly packed, darkly pigmented hyphae, asci, and ascospores attached to the hair shaft, whereas infection with Trichosporon species (white piedra) shows loosely arranged hyphae, blastoconidia, and arthroconidia attached to the hair shaft[41] . Fungal cultures are performed on Sabouraud's dextrose agar. Some Trichosporon species involved in white piedra (eg, T ovoides) are inhibited by cycloheximide, which is found in dermatophyte test medium, Mycosel, and Mycobiotic[41] .

Treatment

Shaving or clipping the infected hair is the treatment of choice for both types of piedra; however, this method may not be esthetically pleasing to all patents, especially women. Antifungal therapy may be initiated in conjunction with shaving[12] . Black piedra may be treated with oral terbinafine[47] . Effective therapies against white piedra include imidazoles, ciclopirox olamine, 2% selenium sulfide, 6% precipitated sulfur in petrolatum, chlorhexidine solution, and zinc pyrithione[41] . In the older literature other reported treatments are Castellani's paint, amphotericin B lotion, and 2% to 10% glutaraldehyde[41] .

Prevention and control

Black piedra rarely occurs after treatment; however; white piedra is prone to sporadic recurrence and familial spread may also occur[41] . The cause of spreading is not known. There is suggestion of person-to-person transmission and transmission through animal contacts; however, both are rare[49] . Travel abroad is not the source of infection of piedra[12] . If untreated, black piedra may last for several years. It is suggested that individuals with

either black or white piedra avoid spreading the infection by not sharing combs, hairbrushes, and other hair accessories[47] .

Tinea capitis

Definition

Tinea capitis is an infection caused by dermatophyte fungi (usually species in the genera Microsporum and Trichophyton) of scalp hair follicles and the surrounding skin.

Epidemiology

Tinea capitis is predominantly a disease of preadolescent children, adult cases being rare[51]. Although world-wide in distribution, its prevalence in the U.K. has been relatively low in the past. An increase in prevalence has recently been reported in urban areas, particularly in children of Afro-Caribbean extraction.[51-53] The main pathogens are anthropophilic organisms with Trichophyton tonsurans now accounting for .90% of cases in the U.K. and North America.([52,54,55] These infections frequently spread among family members and classmates.[55,56] Certain hairdressing practices such as shaving of the scalp, plaiting or the use of hair oils may promote disease transmission, but their precise role remains the subject of study. In nonurban communities, sporadic infections acquired from puppies and kittens are due to M. canis, which, however, accounts for less than 10% of cases in the U.K. Occasional infection from other animal hosts (e.g. T. verrucosum from cattle) occur in rural areas.

Pathogenesis

There are three recognized patterns: endothrix, ectothrix and favus. The latter, a pattern of hair loss caused by T. schoenleinii, is rarely seen in the U.K. being largely confined to eastern Europe and Asia and is not considered further here. Endothrix infections are characterized by arthroconidia (spores) within the hair shaft. The cuticle is not destroyed. Ectothrix infections are characterized by hyphal fragments and arthroconidia outside the hair shaft, which leads eventually to cuticle destruction.

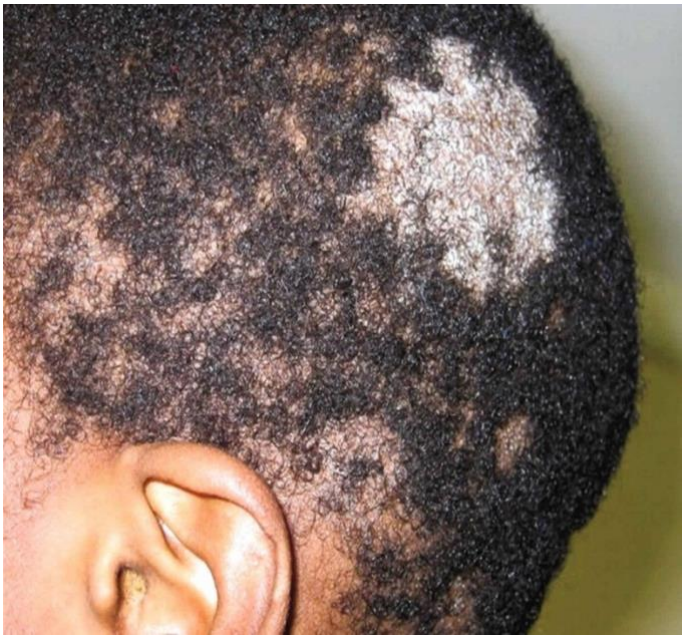
Clinical diagnosis of scalp ringworm

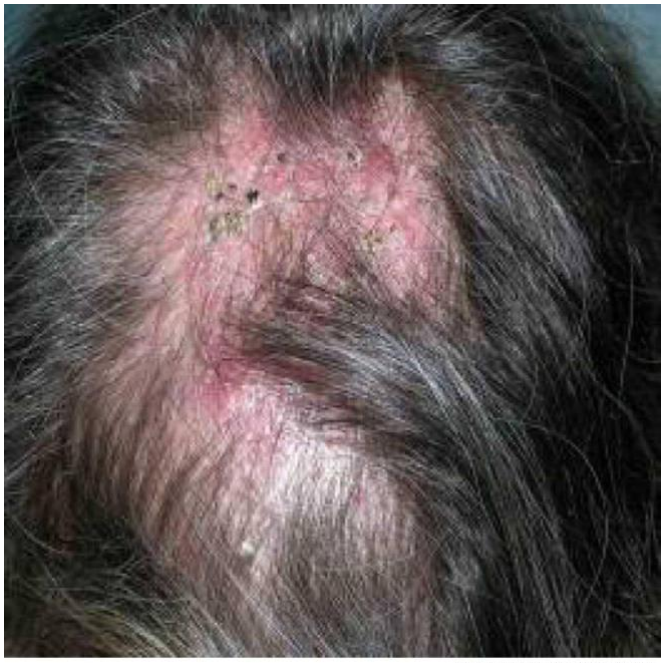
A variety of clinical presentations are recognized as being either inflammatory or non-inflammatory and are usually associated with patchy alopecia (Table2). However, the infection is so widespread, and the clinical appearances can be so subtle, that in urban areas,

tinea capitis should be considered in the diagnosis of any child over the age of 3 months with a scaly scalp, until dismissed by negative mycology. Infection may also be associated with painful regional lymphadenopathy, particularly in the inflammatory variants. A generalized eruption of itchy papules particularly around the outer helix of the ear may occur as a reactive phenomenon (an `id' response). This may start with the introduction of systemic therapy and so be mistaken for a drug reaction.

Clinical patterns	Clinical description	Differential diagnosis
Diffuse scale	Generalized diffuse scaling of the scalp	Seborrhoeic and atopic dermatitis, psoriasis
Grey patch	Patches , scaly alopecia	Seborrhoeic and atopic dermatitis, psoriasis
Black dot	Patches of alopecia studded with broken-off hair stubs	Alopecia areata , trichotillomania
Diffuse pustular	Scattered pustules associated with alopecia scaling ± associated lymphadenopathy	Bacterial folliculitis , dissecting folliculitis
Kerion	Boggy tumour studded with pustules± associated lymphadenopathy	Abscess , neoplasia

Table (2) summarizing the clinical patterns of tinea capitis





Actas Dermosifiliogr. 2012;103:144–8

Laboratory diagnosis of scalp ringworm

If tinea capitis is suspected, specimens should be taken to confirm the diagnosis as systemic therapy will be required.

Taking specimens

Affected areas should be scraped with a blunt scalpel to harvest affected hairs, broken-off hair stubs and scalp scale. This is preferable to plucking, which may remove uninvolved hairs. Scrapings should be transported in a folded square of paper preferably fastened with a paper clip, but commercial packs are also available (e.g. Dermapak, Dermaco, Toddington, Bedfordshire, U.K. and Mycotrans, Biggar, Lanarkshire, U.K.). It is easier to see affected hairs on white paper rather than black. Alternatively, the area can be rubbed with a moistened gauze swab[57,58] or brushed gently 10 times with a plastic, sterile, single use toothbrush [BR8 (unpasted), Brushaway Products, Chislehurst Railway Station, Station Approach, Chislehurst, Kent BR7 5NN, U.K.]. The brush can then be sent in the container provided to the laboratory for culture[59]. (Strength of recommendation . A; quality of evidence III.)

Microscopy and culture

Samples should be sent to laboratories with a particular interest and expertise in mycology. Microscopy provides the most rapid means of diagnosis, but is not always positive. Scalp scales and broken off hair stumps containing the root section (rather than intact hairs) are mounted in a 10±30% potassium hydroxide solution and viewed under the light microscope. Positive microscopy (when the hairs or scales are seen to be invaded by spores or hyphae) confirms the diagnosis and allows treatment to commence at once. Scales, hairs or samples obtained with a sterile single use brush are often not suitable for microscopy, but are inoculated on to a suitable culture medium, e.g. Sabouraud's. Culture allows accurate identification of the organism involved, and this may alter the treatment schedule. Culture is more sensitive than microscopy; results may be positive even when microscopy is negative, but may take up to 4 weeks to become available. Conventional sampling of a kerion can be difficult. In these cases negative results are not uncommon and the diagnosis and decision to treat may need to be made clinically. A moistened standard bacteriological swab taken from the pustular areas and inoculated on to the culture plate may yield a positive result.[58]

Wood's light examination

This is useful for certain ectothrix infections, e.g. those caused by M. canis, M. rivalieri and M. audouinii, which cause the hair to fluoresce bright green. However, as most of the current infections in the U.K. are endothrix and so negative underWood's light, it is of limited value for screening and monitoring these infections.

Therapy of tinea capitis

The aim of treatment is to achieve a clinical and mycological cure as quickly as possible. Oral antifungal therapy is generally needed[60]. (Strength of recommendation . A; quality of evidence IIi.)

Drug	Current standard dose	Duration
Griseofulvin	10-25 mg/kg daily taken with food divided dose	8-10 weeks
Terbinafine	< 20 kg 62.5 mg do: > 20 < 40 kg 125 mg od:> 40 kg 250 mg do	4 weeks
Itraconazole	5mg / kg per day	1-4 weeks

Table (3) Dosing reginmen for tinea capitis

Topical

Topical treatment alone is not recommended for the management of tinea capitis[60]. (Strength of recommendation . A; quality of evidence . III.) It may however, reduce the risk of transmission to others in the early stages of systemic treatment. (Strength of recommendation . B; quality of evidence . IIii.) Selenium sulphide[61] and povidone iodine[62] shampoos, used twice weekly, reduce the carriage of viable spores and are assumed to reduce infectivity[4].

Oral

Griseofulvin. This is fungistatic, and inhibits nucleic acid synthesis, arresting cell division at metaphase and impairing fungal cell wall synthesis. It is also antiinflammatory. It remains the only licensed treatment for scalp ringworm in U.K. It is available in tablet or suspension form. The recommended dose, for those older than 1 month is 10 mg/kg per day. Taking the drug with fatty food increases absorption and aids bioavailability[63,64]. Dosage recommendations vary according to the formulation used, with higher doses being recommended by some authors for micronized griseofulvin as opposed to ultramicronized griseofulvin, but up to 25 mg/kg may be required. The duration of therapy depends on the organism (e.g. T. tonsurans infections may require prolonged treatment schedules) but

varies between 8 and 10 weeks. (Strength of recommendation . A; quality of evidence . IIii.) Shorter courses may lead to higher relapse rates. Side-effects include nausea and rashes in 8±15%. The drug is contra-indicated in pregnancy and the manufacturers caution against men fathering a child for 6 months after therapy. (Strength of recommendation . B; quality of evidence . III.)

Advantages. Licensed; inexpensive; syrup formulation is more palatable; suspension allows accurate dosage adjustments in children; and extensive experience. Disadvantages: Prolonged treatment required.

Contra-indicated in lupus erythematosus, porphyria and severe liver disease. Drug interactions. Include warfarin, cyclosporin and the oral contraceptive pill.

Terbinafine. This acts on fungal cell membranes and is fungicidal. It is effective against all dermatophytes. It is not yet licensed for tinea capitis, although a licence for children of .2 years is being considered. It is at least as effective as griseofulvin and is safe for the management of scalp ringworm due to Trichophyton sp. In children[65,67] .Its role in management of Microsporum sp. is debatable[68]. Early evidence suggests that higher doses or longer therapy (. 4 weeks) may be required in microsporum infections[69.70]. Dosage depends on the weight of the patient, but lie between 3 and 6 mg/kg per day (see Table 2). Side-effects include; gastrointestinal disturbances and rashes in 5% and 3% of cases, respectively[71]. (Strength of recommendation . A/B; quality of evidence . I/IIi.) Advantages.Fungicidal so shorter therapy required (cf. griseofulvin) so increased compliance more likely. Disadvantages. No suspension formulation and no U.K. licence for tinea capitis. Drug interactions. Plasma concentrations are reduced by rifampicin and increased by cimetidine.

Itraconazole. Itraconazole exhibits both fungistatic and fungicidal activity depending on the concentration of drug in the tissues, but like other azoles, the primary mode of action is fungistatic, through depletion of cell membrane ergosterol, which interferes with membrane permeability. 100 mg/day for 4 weeks or 5 mg/kg per day in children is as effective as griseofulvin[72] and terbinafine[73]. (Strength of recommendation . B; quality of evidence . I.) Small studies suggest that shorter or pulsed regimens may also be effective[74]. (Strength of recommendation . B; quality of evidence . I.) Itraconazole is currently unlicensed in the U.K. for use in tinea capitis and for use in children and there are no plans to change this. Advantage. Pulsed shorter treatment regimens are possible.

Disadvantage. Lack of U.K. licence to treat tinea capitis and possible side-effects. Potential drug interactions. More studies needed to confirm paediatric requirements. Drug interactions. Enhanced toxicity of anticoagulants (warfarin), antihistamines (terfenadine and

astemizole), antipsychotics (sertindole), anxiolytics (midazolam), digoxin, cisapride, cyclosporin and simvastatin (increased risk of myopathy). Reduced efficacy of itraconazole with concomitant use of H2-blockers, phenytoin and rifampacin. Fluconazole. Has occasionally been assessed for tinea capitis but its use has mainly been limited by sideeffects. Doses of 3-5 mg/kg per day for 4 weeks are effective in children with tinea capitis[75]. There are no large published series of its use and it is not licensed for the treatment of tinea capitis in the U.K. (Strength of recommendation . C; quality of evidence . I.)

Ketoconazole. Has occasionally been assessed for tinea capitis but its use has mainly been limited by sideeffects. Doses of between 3´3 and 6´6 mg/kg per day. Resolution is comparable with griseofulvin but the response may be slower. However, side-effects are arguably sufficiently significant to lead to the recommendation by some authors that it should not to be used in children[76].Studies have not shown it to be consistently superior to griseofulvin and its use in children is limited by hepatotoxicity. Not licensed for the treatment of tinea capitis in the U.K. (Strength of recommendation . D; quality of evidence . I.)[4].

Additional measures

Exclusion from school. Although there is a risk of the transmission of infection from patients to unaffected class-mates, for practical reasons children should be allowed to return to school once they have been commenced on appropriate systemic and adjuvant topical therapy[53,62]. (Strength of recommendation . B; quality of evidence IIiii.)

Familial screening. Index cases due to the anthropophilic T. tonsurans are highly infectious. Family members [77] as well as other close contacts should be screened (both for tinea capitis and corporis) and appropriate mycological samples taken preferably using the brush technique, even in the absence of clinical signs. (Strength of recommendation . B; quality of evidence . III.)

Cleansing of fomites. Viable spores have been isolated from hairbrushes and combs. For all anthropophilic species these should be cleansed with disinfectant[78]. (Strength of recommendation . B; quality of evidence . IV.) Proprietary phenolic disinfectants are no longer available, but simple bleach or Milton should be suitable alternatives. (Strength of recommendation . B; quality of evidence . III.)

Soaking off crust from kerions or pustules. This is recommend by some authors. Although this is not necessary, it is often soothing[79]. (Strength of recommendation . C; quality of evidence III.)

Steroids. The use of corticosteroids (both oral and topical) for inflammatory varieties, e.g. kerions and severe id reactions is controversial, but may help to reduce itching and general discomfort[80]. Although in the past steroids have been thought to minimize the risk of permanent alopecia secondary to scarring, current evidence does not suggest any reduction in clearance time compared with griseofulvin alone[79,80]. (Strength of recommendation . C; quality of evidence . I.)

Treatment failures

Some individuals are not clear at follow-up. The reasons for this include:

1- Lack of compliance with the long courses of treatment.

2- Suboptimal absorption of the drug.

3- Relative insensitivity of the organism.

4- Reinfection.

T. tonsurans and Microsporum sp. are typical culprits in persistently positive cases. If fungi can still be isolated at the end of treatment, but the clinical signs have improved, the authors recommend continuing the original treatment for a further month. If there has been no clinical response and signs persist at the end of the treatment period, then the options include:

1- Increase the dose or duration of the original drug: both griseofulvin (in doses up to 25 mg/kg for 8-10 weeks) and terbinafine have been used successfully and safely at higher doses or for longer courses to clear resistant infections. (Strength of recommendation . C; quality of evidence . IV.)

2- Change to an alternative antifungal, e.g. switch from griseofulvin to terbinafine or itraconazole. (Strength of recommendation . C; quality of evidence . IV.)

Carriers

The optimal management of symptom-free carriers (i.e. individuals without overt clinical infection, but who are culture positive) is unclear. In those with a heavy growth/high spore count on brush culture, systemic antifungal therapy may be justified as these individuals are especially likely to develop an overt clinical infection, are a significant reservoir of infection[53] and are unlikely to respond to topical therapy alone[55,81]. Alternatively, they may represent a missed overt clinical infection. For those with light growth/low spore

counts on brush culture, twice weekly selenium sulphide or povidone iodine shampoo is probably adequate[61,62]. (Strength of recommendation .B; quality of evidence . IV.)

Follow-up

The definitive end-point for adequate treatment is not clinical response but mycological cure; therefore, follow-up with repeat mycology sampling is recommended at the end of the standard treatment period and then monthly until mycological clearance is documented. (Strength of recommendation . A.) Treatment should, therefore, be tailored for each individual patient according to response.

Tinea manuum

Tina manuum is a chronic dermatophytosis of the hand(s), often unilateral, most cortonly on the dominant hand, and usually associated with tinea pedis[4].

Epidemiology and Etiology

Etiology Most often T. rubrum, T. mentagro-phytes, and E. floccosum, the same fungi that cause tinea pedis and tinea cruris.

Pathogenesis

Usually associated with tinea pedis and, often, tinea cruris.

History

Duration Months to years Skin Symptoms Frequently symptomatic. Pruritus. Pain if secondarily infected or fissured. Dyshidrotic type: episodic symptoms of pruritus.

Physical Examination

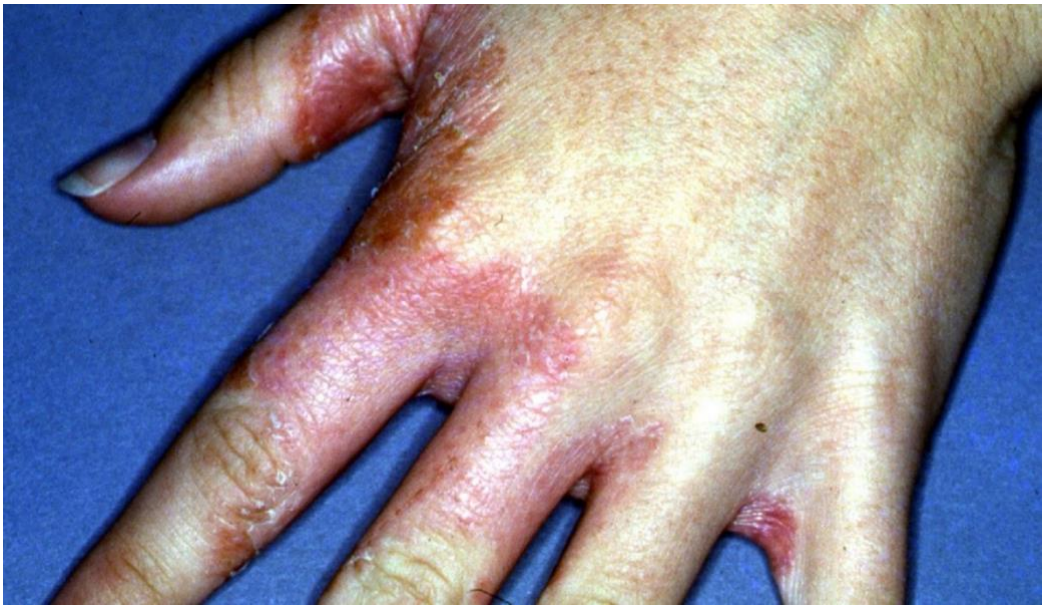
Skin Lesions

TYPE Dyshidrotic Type Papules, vesicles, bulla (uncommon on the margin of lesion) Hyperkeratotic Type Well-demarcated scaling patches, hyperkeratosis and scaling confined to palmar creases, fissures on palmar hand. Borders well demarcated; central clearing. Often extends onto dorsum of hand with follicular papules, nodules, pustules with der- matophytic folliculitis. Secondary Changes Lichen simplex chroncus, prurigo nodules, impetiginization COLOR Erythematous SHAPE Annular, polycyclic, especially on the dorsum .

DISTRIBUTION Diffuse hyperkeratosis of the palms with pronounced involvement of palmar creases or patchy scaling on the dorsa and sides of fingers; 50 % of patients have unilateral involvement .Usually associated with tinea pedis, ‡tinea cruris. If chronic, often associated with tinea unguium of fingernails.

Differential Diagnosis

Erythema/Scaling Hands Atopic dermatitis. lichen simplex chronicus, allergic contact dermatitis. irritant contact dermatitis, psoriasis vulgaris, pityriasis rubra pilaris, in situ squamous cell carcinoma, tinea nigra.



Laboratory and Special Examinations

Direct Microscopy . Hyphae in scales. Fungal Culture Dermatophytes. Rule out Scytalidium hyalinum, Hendersonula toruloidea, Candida albicans. Diagnosis Clinical findings confirmed by direct mieroscopy or isolation of dermatophyte on culture course Chronic, does not resolve spontaneously. After treatment, recurs unless dermatophytosis of fingernails, feet, and toenails is eradicated. Fis- sues and erosions provide portal of entry for bacterial infections.

Management

trevention Must eradicate tinea unguium of agemails as well as toenails, tinea pedis-Hina cruris as well; otherwise, tinea manuum ill recur. hail ungal Agents MCAL AGENTS See Dermatophytoses, Failure common.TANIC NiENTS Because of thickness of PRimer stralum corneum, and especially if associated with tine unguium of fingernails, tinea manuum is impossible to cure by top- ical agents. Oral agents eradicate dermato- phytoses

of hands, feet, and nails. Terbinafine 250 mg/day for 14 days. Itraconazole 200mg q.d. for 7 days. Griseofulvin 500 mg micronized per day for 21 days. NOTE: Eradication of dermatophytosis of nails requires longer use .

Tinea facialis

Tina facialis is dermatophytosis of the glabrous facial skin, characterized by a well-eire/ascribed erythematous patch, and is more commonly misdiagnosed than any other dermatophytos. Synonyms: Tina faciei, ringworm of the face[(4)].

Epidemiology and Etiology

Age More common in children Etiology T. mentagrophytes; T. rubrum most commonly; also M. audouinii, M. canis Predisposing Factors Animal exposure, chronie topical application.

History

Skin Symptoms Most commonly asymptomatic. At times, pruritus and photosensitivity.

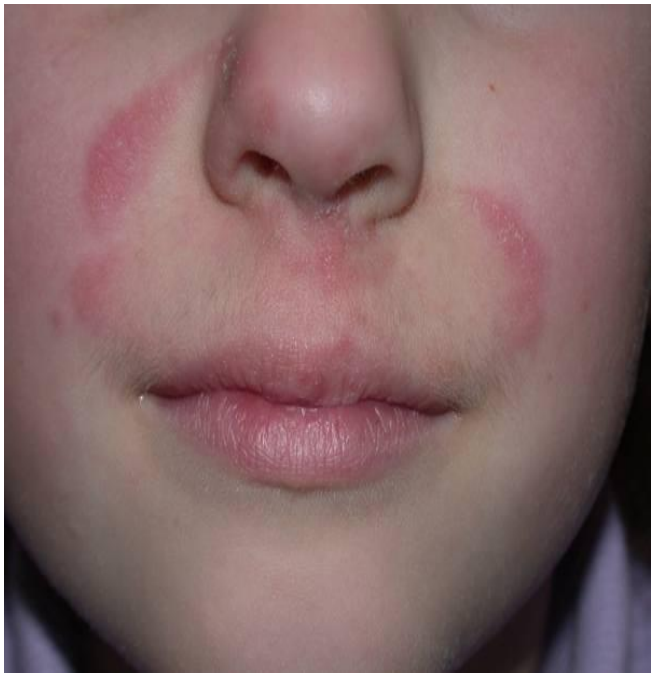
Physical Examination

Skin Lesions

TYPE Well-circumscribed macule to plaque of variable size: elevated border and central re-gression.Scaling often is minimal but can be pronounced. COLOR Pink to red. In black patients, hyper- pigmentation. DISTRIBUTION Any area of face but usually not symmetric.

Differential Diagnosis

Scaling Facial Patches Seborrheic dermatitis, contact dermatitis, erythema migrans, lupus erythematosus, polymorphic light eruption. phototoxic drug eruption, lymphocytic infiltrate.



Laboratory and Special Examinations

Direct microscopic examination of scraping shows hyphae. Scrapings from patients who have used topical corticosteroid show massive numbers of hyphae.

Management

Topical antifungal preparations Eradicate dermatophyte infection at other sites such as feet and hands.

Tinea barbae

Tina barbae is a dermatophytic trichomycosis involving the beard and moustache areas, closely resembling tinea capitis, with invasion of the hair shaft. Synonym: Ringworm of the beard[(4)].

Epidemiology and Etiology

Age Adult Sex Males only Etiology T. verrucosum, T. mentagrophytes most commonly. May be acquired through an- imal exposure. Predisposing Factors More common in farmers.

History

Skin Symptoms Pruritus, tenderness, pain

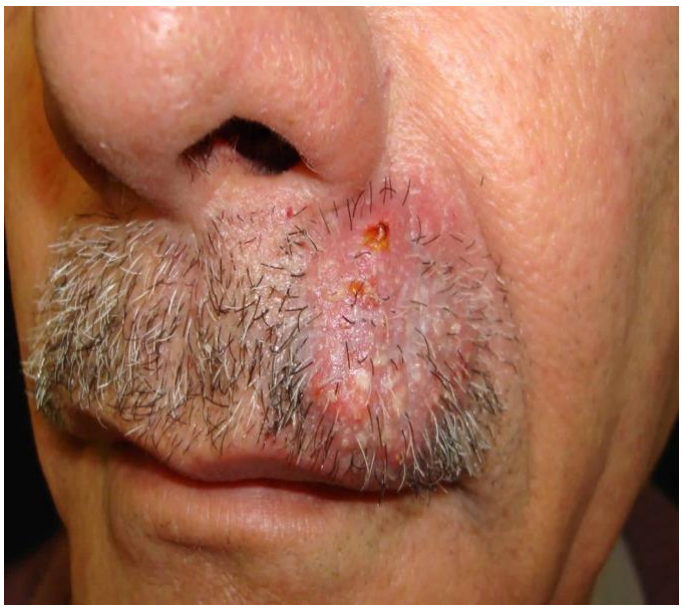
Physical Examination

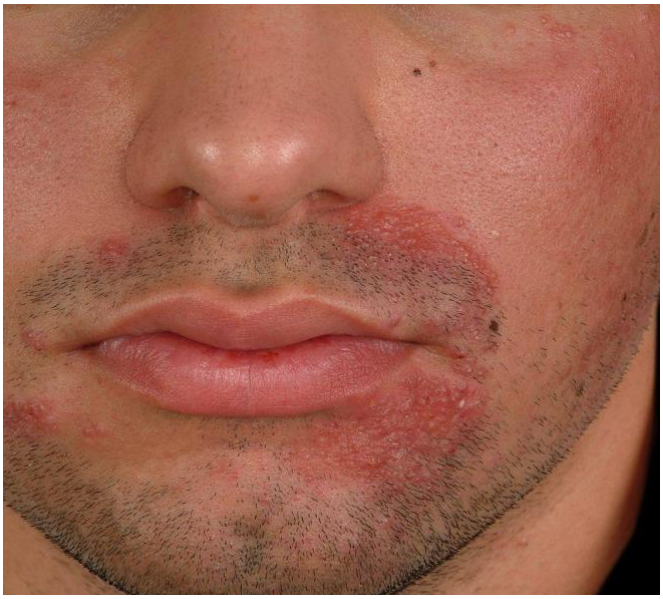
Skin Lesions

TYPPS Pustular folliculitis i.e,. hair follicles surrounded by red inflammatory papules or pustules, often with exudation and crusting. Involved hairs are loose and easily removed. With less follicular involvement, there are scaling, circular, reddish patches in which hair is broken off at the surface, Papules may coalesce to inflammatory plaques topped by pustules. Kerion: boggy purulent nodules and plaques aswith tinea capitis . COLOR Red

DISTRIBUTION Beard and moustache areas, rarely eyelashes, eyebrows Systemic Findings Regional lymphadenopathy, especially if of long duration and if superintected

Differential Diagnosis Beard Folliculitis Staphylococcus aureus folliculitis, furuncle, carbuncle, acne vulgaris. rosacea, pseudofolliculitis.





Laboratory and Special Examinations Same as tinea capitis.

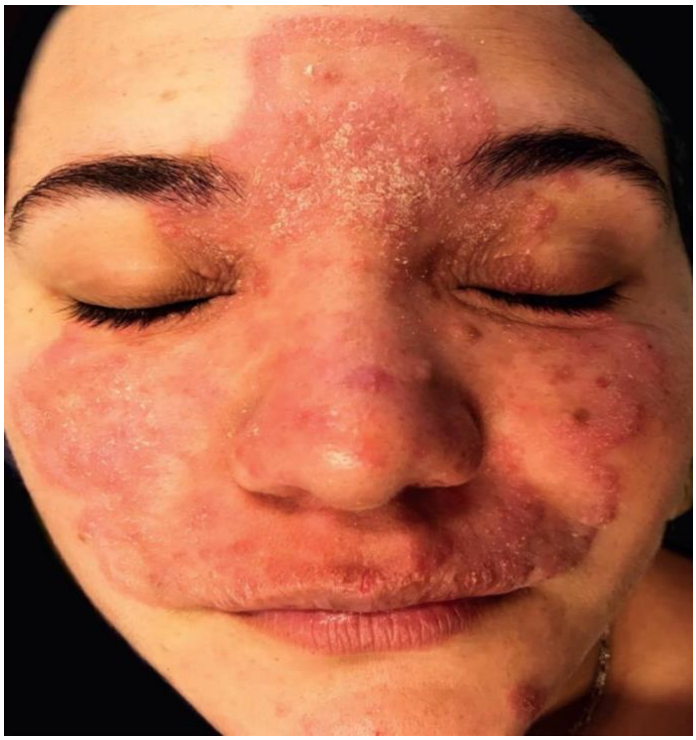
Management ,Topical Agents, Inettective Systemic same as Tinea Capitis.

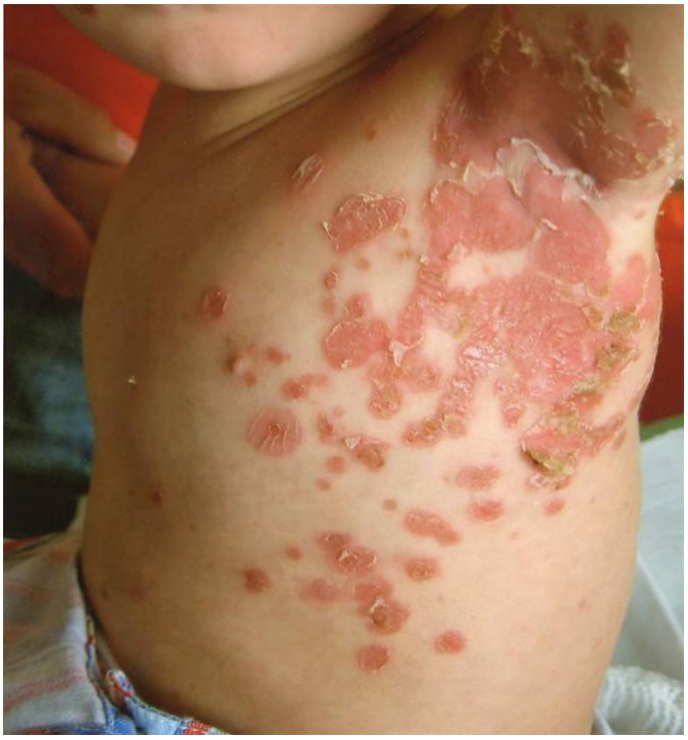
Tinea Incognito

A ringworm infection, modified by topical and/or systemic corticosteroids, given mistakenly or for co-existing pathology is known as tinea incognito[82]. Application of even as mild as 1 % hydrocortisone can cause this condition.It may mimic a number of other

dermatological conditions like lupus erythematosus, contact dermatitis, psoriasis and eczema.This similarity further delays proper diagnosis and treatment of this condition[83].

The pathogenesis of the condition is presumed to be a steroid modified response of the host to fungal infection rather than a direct pharmacological effect of the drug. Corticosteroids being immuno- suppressives decreases the resistance to infection, and suppress the inflammatory reaction. The infection therefore spreads unchecked and acquires a form quite different from clinical ring form[84].





Conclusion

Skin infections are a major dermatological issue because of the prevalence and the slow response to treatment. The patient compliance is important factor that should be researched and studied to decrease the rate of occurrence and finally the eradication of all known skin infections.

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