



Derma Team 436

Common Skin Infections

Objectives:

Not Given

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Template by group B

Before you start.. CHECK THE EDITING FILE

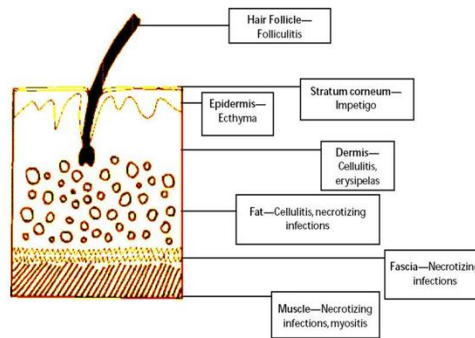
Sources: doctor's slides and notes + 436 group B team

[Color index: Slides | Slides | Important | doctor notes | Extra]

Why does skin get infected?

- There are multiple types of organisms which are normally present on the skin as normal flora such as: Staphylococcus epidermidis and yeasts.
- The presence of bacteria does not automatically lead to a skin infection.
- What is the difference between colonisation and infections?
Colonisation: Bacteria are present but causing no harm (no signs or symptoms).
Infection: Bacteria are present and causing harm.
- A break in the epidermal integrity can allow organisms to enter and become pathogenic. This can occur as a result of trauma, ulceration, skin disease such as eczema.

Figure 1. Skin And Soft Tissue Anatomy And Infection Types.



Bacterial skin infections

They are classified based on the site infected: Impetigo, Folliculitis, Furunculosis & Carbuncles, Ecthyma & Ecthyma gangrenosum, Erysipelas, Cellulitis, Erythrasma, SSSS.

Most of them are caused by staph & strept.

Impetigo (commonest skin infection)

- Acute superficial cutaneous infection (infection of stratum corneum)
- 2 forms (bullous 30%, **non-bullous 70% commonest**).
- The causative organism is usually Staphylococcus Aureus (>90% cases), but less often can be strept. Pyogenes (gp A beta-hemolytic streptococci).

The most important complication caused by this bacteria is post-streptococcal glomerulonephritis

- Very contagious, auto-inoculation is common.
- Can cause systemic symptoms (fever, LAD)
- Children, Adult.



IMPETIGO (non-bullous)

- Begins as tiny erythematous papule/pustule.
- Develops thin roofed vesicle/bulla with rim of erythema.
- Vesicle ruptures, releases thin yellow fluid which cause golden-yellow (**honey**) crust.
- Crust is a secondary lesion preceded by a primary lesion (a **ruptured flaccid bulla or pustule**).
- Predisposing factors:
 - Warm, humid climate, poor hygiene, trauma, insect bites, immunosuppression and **any break in skin**.



Bullous Impetigo

- Due to staph aureus. Phage group 2 toxins (exotoxins) which cleave desmoglein-1 leading to the formation of bullae (desmoglein keeps the keratinocytes attached to each other in the upper part of the epidermis).
- Newborn and in renal patients.
- Face, hands, diaper area or any part.
- Bullae (**flaccid**) on grossly normal skin.



Impetigo (Non-Bullous)



Non-bullous impetigo is a superficial skin infection that manifests as clusters of vesicles or pustules that rupture and develop a honey-colored crust.

Impetigo (Bullous)



Bullous impetigo is a superficial skin infection that manifests as clusters of vesicles or pustules that enlarge rapidly to form bullae. The bullae burst and expose larger bases, which become covered with honey-colored varnish or crust.

Prognosis: Scarring is unusual; why? Because it is very superficial involving only the stratum corneum, but postinflammatory hyperpigmentation or hypopigmentation.

Complications: Post-strept. GN (if caused by streptococcus pyogenes).

- Nephrogenic syndrome associated strains 49,55,57, 59.
- Rare.

Investigation: Swab > Gram stain and culture show gram positive cocci. and start anti-strept.

Treatment:

- Wound care
- **Localized:**- Topical Abx (Mupirocin); it covers staph & strept.
 - Warm compresses to loosen crusts.
 - **Intranasal mupirocin for periodic decolonization in carriers** (Fusidic acid (fucidine) is used over the lesion or intra-nasally in patients having recurrent impetigo because of colonization of nostrils by staph).
- **Severe or widespread:** use systemic antibiotics (must cover both MRSA/GABHS (group A beta-hemolytic streptococci) such as:
 - Penicillinase-resistant PCN, 1st/2nd generation cephalosporin, clindamycin, or erythromycin (esp, if penicillin allergic)

Folliculitis

- Inflammation of the hair follicle.
- Presents as itchy or tender papules and pustules at the follicular openings.
- Complications include abscess formation and cavernous sinus thrombosis, carbuncles or furuncles if upper lip, nose or eye affected.
- Superficial infection of follicle ostium.
- Most common cause is Staph Aureus.
- Other organisms to consider include:
 - Gram negative bacteria – usually in patients with acne who are on oral broad-spectrum antibiotics for long time; in such cases, don't give antibiotics, but give isotretinoin.
 - Pseudomonas ("Hot tub folliculitis").
Present at bathing suit distribution that is very itchy usually in patients used to swim in hot tubs.



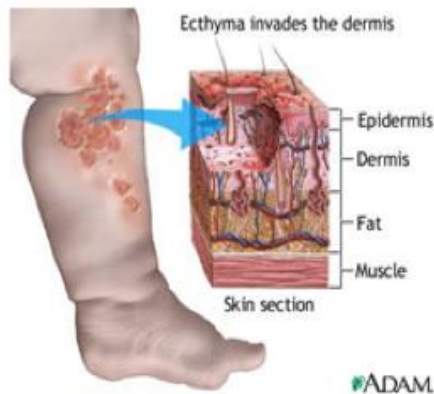
- Yeasts (candida and pityrosporum).
Involve areas rich in yeasts such as upper chest, upper back or seborrheic areas.
It is itchy & mono-morphous.
 - Demodex (usually seen in association with rosacea).
- Topical antiseptics such as Chlorhexidine.
 - Topical antibiotics, such as Fusidic acid, Mupirocin or clindamycin.
 - More resistant cases may need oral antibiotics (similar to impetigo).
 - Hot tub folliculitis (P. aeruginosa)– usually self-limited (ciprofloxacin in severe cases).
 - Gram negative – trimethoprim, isotretinoin.

Furunculosis (boils) and carbuncles

- Deeper staphylococcal abscess of the hair follicle
- Coalescence of boils leads to the formation of a carbuncle
- Presents as red tender nodule.
- Treatment is with systemic antibiotics and may need incision and drainage.
- Consider looking for underlying causes, such as diabetes.



Ecthyma and Ecthyma gangrenosum



Ecthyma is a skin infection similar to impetigo, but more deeply invasive. Usually caused by a streptococcus infection, ecthyma goes through the outer layer (epidermis) to the deeper layer (dermis) of skin, possibly causing scars.

Ecthyma presents as a superficial ulceration involving only the epidermis usually over the legs. Mostly caused by streptococcus & may heal with scarring.



Ecthyma gangrenosum is a bacterial skin infection (caused by *Pseudomonas aeruginosa*) that usually occurs in immunocompromised individuals

Typical scenario: ICU-setting, sick patients with pseudomonas sepsis & as a consequence they develop these ulcerations usually with black eschar (black necrosis in the center).
Ask them to do blood culture to rule out pseudomonas

Erysipelas

- Superficial infection with marked lymphatics involvement.
- **Sharply demarcated** (distinguishing feature) unilateral, hot red, tender & edematous. They might have oozing or crust.
- Infants, young children, & elderly patients (**most**)

Cellulitis

- Deeper involvement of the SC.
- Acute, raised, hot, tender, erythematous with poorly-defined border (leg)
- Strept. Pyogenes, staph.aureus
- Cutaneous abrasion or ulcer.
- There should be a port of entry for the organism either

commonly..)

- Face, leg
- **Beta hemolytic gp A Strept or staph.**
- **Risk factor;** Minor abrasion/lymphatic dysfunction
 - sup. Lymph vessels
- Might be associated with Leukocytosis & fever

Management:

- The diagnosis is clinical.
- **Smear for gram stain and culture (fluid, blood).**
- Cold compressor.
- Oral antibiotics or I.V. for severe infection.
- Oral penicillin or Erythromycin.



by insect bite, eczema or trauma.

- Palpable, tender LN.
- Fever, leukocytosis.

Risk factors:

- DM, HTN, obesity, immunocompromised patients, vascular insufficiency.
- **Complicated by lymphedema if recurrent**

Management:

- The diagnosis is clinical.
- Swab + blood culture.
- Semisynthetic Penicillin or Erythromycin if allergic.
- If severe or in immunocompromised, may require admission for IV antibiotics.
- After the acute attack has settled, especially in recurrent episodes – consider the underlying cause

Raised hot tender nodule



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CELLULITIS vs ERYSIPELAS



- Dermal and SC
- Ill-defined
- Indolent
- Less systemic symptoms



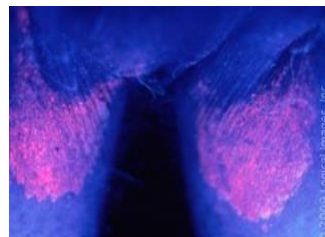
- Dermal lymphatics
- Well-demarcated
- Acute onset
- More systemic symptoms

Erythrasma

- Colonisation of axilla or groin (**flexures**) with *Corynebacterium Minutissimum*.
- Gram positive bacilli
- Present with itchy, dry, red (**erythematous**) to brownish **scaly patch**.
- asymptomatic, flexorures

Management:

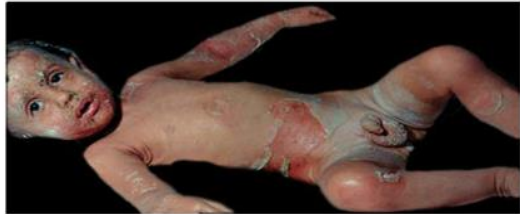
- Swab (for gram staining & culture).
- **wood's lamp: coral-red fluorescence (characteristic).**
- Topical: erythromycin.
 - fusidic acid.
 - clindamycin.
- Oral erythromycin X 7 d (**reserved for resistant to topical cases**).



Staphylococcal Scalded Skin Syndrome (SSSS) emergency

- A superficial blistering condition caused by **exfoliative toxins of certain strains of Staph Aureus (which break desmoglein-1)**.
- Usually in children less than 5 years old.
- Characterized by blistering and desquamation of the skin and **Nikolsky's sign** (shearing of the epidermis with gentle pressure **over normal skin**), even in areas that are not obviously affected.
- Begins with a **prodrome of pyrexia and malaise**, often **with signs and symptoms of an upper respiratory tract infection**.
- Discrete erythematous areas then develop and rapidly enlarge and coalesce, leading to generalized erythema - often worse in the flexures with sparing of the mucous membranes
- Large, fragile bullae form in the erythematous areas and then rupture
- Complications include hypothermia, dehydration and secondary infection.

Treatment: ABC, admit for IV antibiotics and fluids, may need referral to burn center or ICU.



Viral Skin Infections

Warts, Molluscum contagiosum, Herpes simplex, Varicella, Herpes zoster

Warts

- Caused by Human papillomavirus HPV (DNA virus).
- More than 100 subtypes of HPV have been identified with different epithelial preferences (skin vs mucosa) and different clinical patterns.
- **Clinical variants:**
 - Common warts (verruca vulgaris).
 - Plantar warts (verruca plantaris).
 - plane (flat) warts (verruca plana).
 - Genital warts (condyloma acuminata and bowenoid papulosis).
 - Mucosal warts.

Common warts (verruca vulgaris)

- Caused by HPV type 1,2,3,4,7,54.
- Affects the hands, could be periungual.
- Common in Children.
- Presents as hyperkeratotic (verrucous) papules.
- Koebner phenomenon d.t autoinoculation (self-infection from one site to another site) or contact.

Plane warts (Verruca plana)

- Affects Face, back of hands.
- Caused by HPV type 3,10, 28.
- Flat skin colored papules.

Plantar warts (verruca plantaris)

- Affects the soles.
- Caused by HPV type 1,2,4, 60,63.
- To be differentiated from plantar corn (مسماة القدم).
- Corn is only thickening of the skin at the sites of pressure.



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4 or few hyper-keratotic skin colored papules over the thumb.
Papules is well-defined by definition



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Pin-point hemorrhage indicates presence of wart.



Management:

- Involute spontaneously within 2 years.
We don't wait until it involutes spontaneously because patient might infect others or himself by autoinoculation.
- Cryotherapy.
- Topical keratolytics: Salicylic acid, TCA.
- Electrocautery, curettage.
- Laser.
- Topical retinoids in flat warts especially on the face.
- Others: bleomycin, cantharidin.
- PPD, Candida antigen to stimulate the immune response.

Genital wart

- Most common STD.
What is the next step after encountering a patient with genital wart?
Screen for other STDs.
Screen the partner.
Pap smear.
- Condylomata acuminata.
- Cauliflower like.
- Penile, vulvar skin, mm, perianal area.
- Sexual partner.
- Child---? sexual abuse.
- Caused by HPV type 6,11, 16, 18.
- Oncogenic strains: 16, 18.
- Vaccination
 - Vaccine (**GARDASIL-9**) provides immunity against 9 HPV type: 6,11,16,18,31,33,45,52,58.

Table 79.2 Management of anogenital warts with grading of recommendations. Grading of recommendation: (1), based on randomized, controlled trials of good quality and consistency; (2), well-conducted clinical studies but no randomized clinical trials⁶⁷.

MANAGEMENT OF ANOGENITAL WARTS WITH GRADING OF RECOMMENDATIONS
Cytotoxic agent
• Podophyllotoxin 0.5% solution, 0.15% cream (1)
Physical destruction
• Cryotherapy (liquid nitrogen, cryoprobe) (1) • Trichloroacetic acid (TCA) 80–90% solution (1) • Electrosurgery (1) • Scissors excision (1) • Laser vaporization (2)
Immunomodulatory
• Imiquimod 5% cream (1)

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Table 79.1 Clinical manifestations and associated HPV types.

CLINICAL MANIFESTATIONS AND ASSOCIATED HPV TYPES		
	Frequently detected	Less frequently detected
Skin lesions		
• Common, palmar, plantar, myrmecial and mosaic warts	1, 2, 4	26, 27, 29, 41, 57, 60, 63, 65
• Flat warts	3, 10	28, 29
• Butcher's warts	7, 2	1, 3, 4, 10, 28
• Digital squamous cell carcinoma and Bowen's disease	16	34, 35
• Epidermodysplasia verruciformis (EV)	3, 5, 8	9, 12, 14, 15, 17, 19-25, 36-38, 46, 47, 49, 50, etc.
• EV - squamous cell carcinoma	5	8, 14, 17, 20, 47
Mucosal lesions		
• Condylomata acuminata	6, 11	42-44, 54, 55, 70
• High-grade intraepithelial neoplasias (including cervical condylomata plana, bowenoid papulosis, erythroplasia of Queyrat)	16	18, 31, 33-35, 39, 40, 51-59, 61, 62
• Buschke-Löwenstein tumor	6, 11	
• Recurrent respiratory papillomatosis, conjunctival papillomas	6, 11	
• Heck's disease (focal epithelial hyperplasia)	13, 32	

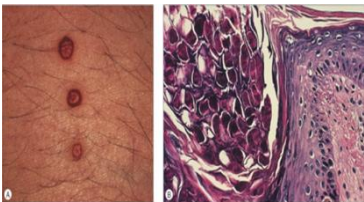
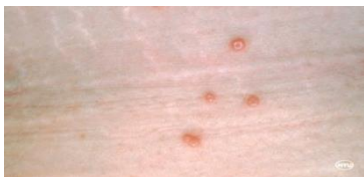
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Molluscum contagiosum

- Caused by Poxvirus (DNA virus).
- Common in children by contact.
- In adults: **immunosuppression, STD.**
- Face, neck or genitalia.
- Skin colored papules with central punctum (umbilication).
- **Koebner phenomenon d.t autoinoculation.**
- H/P: Hunderson-patterson bodies.

Management:

- Involute spontaneously within 2 years but we have to treat.
- **Curettage** (preferred modality of treatment), cryotherapy.
- Other: Salicylic acid.



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Cup-shaped invagination of the epidermis which represent the umbilication filled with eosinophilic & basophilic bodies. The bodies represent the viral particles & are called Hunderson-patterson bodies.

Herpes simplex

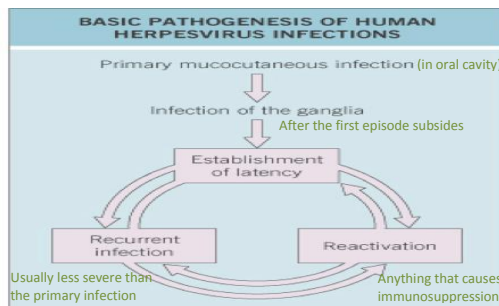
- Group of small blisters on background of erythema.
- HSV-1 (H. labialis).
- HSV-2 (genital herpes).
- Herpetic whitlow mostly caused by HSV-1.
- Eczema herpeticum (mostly caused by HSV-1): Infection with HSV in patients with previous skin disease (eg: atopic dermatitis, pemphigus, Darrier disease).

Diagnosis:

- Tzanck Smear showing multi-nucleated giant cells.
- Direct fluorescent antibody (DFA).
- Viral culture- **most definitive.**

Treatment:

Oral /IV acyclovir for Genital, Recurrent, immune suppressed, neonatal, Eczema Herpeticum (best initiated in the first 24h of blister/vesicle appearance).



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The first episode is usually severe



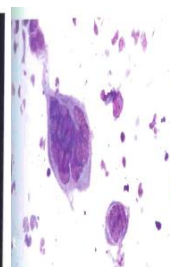
Painful group of blisters on background of erythema.



Eczema herpeticum



Herpetic whitlow



Varicella (chicken pox)

- Initial infection with varicella zoster virus (VZV).
- Incubation period: 2 weeks.
- Prodrome of respiratory (with or without fever) coryza followed by disseminated red macules with central vesicles or pustules. The most characteristic feature in varicella is the presence of multiple morphologies (hallmark) in the same patient; for example, they have vesicles, pustules, macules & papules all in the same patient.
- The whole illness: 3 weeks
- The patient contagious 5 days before and 5 days after skin eruption.
- Children.
- Adults as a primary disease: immunosuppression, individuals never infected during childhood, pneumonia (complication).

Diagnosis: tzanck smear (doesn't differentiate between varicella & HSV), DFA, Viral culture.

Treatment: symptomatic for itching, Systemic antiviral in immunocompromised patients.

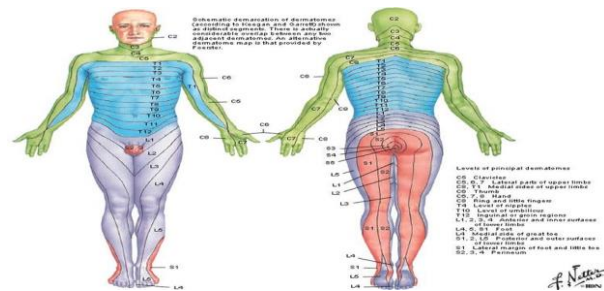
Vaccination for non-infected individuals.

- Varicella in pregnancy:
 - 1st and 2nd trimester: risk of varicella embryopathy syndrome, abortion.
 - 3rd trimester: congenital varicella.
 - Pregnant patients with varicella should receive VZ immunoglobulin within 96 h from exposure and antiviral therapy.
- Scenario:** a pregnant mother & one of her kids developed varicella, what should you do? give VZ immunoglobulin.



Herpes zoster

- Following initial varicella infection VZV remain latent in sensory ganglia when reactivated it will affect skin dermatome supplied by this ganglia.
- Adult, immunocompromised.
- Prodromal pain—dermatomal (grouped blisters and vesicles on background of erythema)—post-herpetic neuralgia.
- **Diagnosis:**
 - Clinical diagnosis.
 - Tzanck Smear.
 - Direct fluorescent antibody (DFA)
 - Viral culture
- **Treatment:**
 - Analgesia.
 - **ANTIVIRAL (within 72 hours of skin eruption).** Oral for immune-competent. Admission & IV acyclovir for immune-compromised & patients who develop ophthalmic zoster or Ramsay hunt syndrome.



Fungal Skin Infections

Superficial mycosis

Table 77.2 Superficial mycoses of the skin.

SUPERFICIAL MYCOSES OF THE SKIN		
	Cutaneous disorder	Pathogen(s)
Minimal, if any, inflammation	Pityriasis (tinea) versicolor	<i>Malassezia furfur</i> (<i>Pityrosporum ovale</i>)
	Tinea nigra Black piedra White piedra	<i>Exophiala werneckii</i> <i>Piedraia hortae</i> <i>Trichosporon beigelii</i>
Inflammatory response common	Tinea capitis, barbae, faciei, corporis, cruris, manuum, pedis Cutaneous candidiasis	<i>Trichophyton</i> , <i>Microsporum</i> , <i>Epidermophyton</i> spp. <i>Candida albicans</i>

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Candidiasis

- Candidiasis is a normal flora in the skin especially in the moist & flexural areas.
- Physiological (old age, neonate and pregnancy)
- Pathological (DM, HIV and organ transplant, on immunosuppression).
- Iatrogenic (long course of Antibiotics).
- *Candida albicans* (normal commensal of GIT)
 - Napkin candidiasis & Intertrigo (**satellite lesions**).
 - Paronychia.
 - MM--oral, urogenital and oesophagus.
 - Vulvovaginitis---irritation, discharge.
 - *Candida* folliculitis.
 - Generalized Systemic infection.
 - Chronic mucocutaneous candidiasis.

Management:

- **Clinical diagnosis.**
- Swab and KOH.
- Alter moist warm environment.
- Nystatin-containing cream.
- Imidazole (Daktarin, canastein).
- Oral antifungal (itraconazole): immune suppressed, persistent infection.



Pityriasis versicolor

- It is called versicolor because it may present with hypopigmentation, hyperpigmentation or erythematous thin plaques.
- Caused by *Malassezia furfur* (hyphae) & *Pityrosporum orbiculare* (yeast).
- Upper Trunk (upper chest, upper back & neck).
- Asymptomatic or mild itching.
- Yellowish-brown or hypopigmented thin scaly patches. It can be admixed with hypopigmentation or hyperpigmentation.
- Once the rash has gone, it leaves hypopigmented macules which takes time to tan.

Investigation:

- Wood's lamp (coppery-orange fluorescence).
- Scraping for KOH and fungal c/s.
- Skin biopsy for PAS stain.

Treatment:

- Topical imidazole (nizoral) creams or shampoo. From the neck to the waist for 10 mins then washed away.
- Oral Antifungal (azoles).
- Recurrence.



A 22-year-old lady returns from a holiday in Spain after She has a tan. She noticed hypopigmented lesions on her chest and back?

DDX: vitiligo, post inflammatory hypopigmentation, pityriasis colour.

Dermatophyte infections

The dermatophytes like the keratin, which is present in the hair, nail & skin.

3 main genera:

- Trichophyton
- Microsporum
- Epidermophyton
- Invade the keratin of the stratum corneum, hair or nail.

Can be:

- Anthrophilic – contracted from humans.
- Zoophilic – contracted from animals.
- Geographic/geophilic – contracted from soil.
- Clinical appearance depends on the organism involved, the site and the host reaction.
- Skin
- Hair
- Nails

Notes:

- Tinea pedis: Infection of foot.
- Tinea Unguium: Infection of nails.
- Tinea manum: Infection of Hand.
- Tinea corporis: Infection of trunk.
- Tinea cruris: Infection of groin.
- Tinea capitis: Infection of scalp and hair.

Tinea pedis

1.Erosive interdigitalis (web space), athletes because the moisture is a good environment for the dermatophytes to

Tinea unguum (onychomycosis)

Different presentations including:
- White superficial Onychomycosis.

grow.

2. Hyperkeratotic type (*T. rubrum*) affecting the sole.

3. Inflammatory type (*T. mentagrophyte*) forming blisters on the sole of the foot.

Table 77.9 The four major types of 'tinea pedis' (including dematiaceous and dermatomycoses). *Because of the thickness of stratum corneum on plantar surfaces and the inability of *T. rubrum* to elicit an immune response sufficient to eliminate the fungus¹⁶. †Often *Pseudomonas*, *Proteus* or *Staphylococcus aureus*. ‡Allergic reaction to fungal elements presenting as a dyshidrotic-like eruption on the fingers and palms (culture-negative for fungus). CMI, cell-mediated immunity.

THE FOUR MAJOR TYPES OF 'TINEA PEDIS' (INCLUDING DEMATIACEOUS AND DERMATOMYCOSIS)			
Type	Causative organism	Clinical features	Treatment considerations
Moccasin	<i>T. rubrum</i> <i>E. floccosum</i>	Diffuse hyperkeratosis, erythema, scaling, and fissures on one or both plantar surfaces; frequently chronic and difficult to cure*; may be associated with fungal CMI deficiency	Topical antifungal plus product with urea or lactic acid; may also require oral antifungal therapy
	<i>S. hyalinum</i> <i>S. dimidiatum</i>		
Interdigital	<i>T. mentagrophytes</i> (var. interdigitale) <i>T. rubrum</i> <i>E. floccosum</i>	Most common type; erythema, scaling, fissures, and maceration occur in the web spaces; the two lateral web spaces are most commonly affected; associated with the 'dermatophytosis complex' (fungal infection followed by bacterial invasion [†]); pruritus common; may extend to dorsum and sole of foot	Topical antifungal; may require topical or oral antibiotic if superimposed bacterial infection
	<i>S. hyalinum</i> <i>S. dimidiatum</i> <i>Candida</i> spp.		
Inflammatory (vesicular)	<i>T. mentagrophytes</i> (var. <i>mentagrophytes</i>)	Vesicles and bullae on the medial foot; associated with the dermatophytid reaction [‡]	Topical antifungal usually sufficient
Ulcerative	<i>T. rubrum</i> <i>T. mentagrophytes</i> <i>E. floccosum</i>	Typically an exacerbation of interdigital tinea pedis; ulcers and erosions in the web spaces; commonly secondarily infected with bacteria; seen in immunocompromised and diabetic patients	Topical antifungal; may require topical or oral antibiotics if secondary bacterial infection

■ Dermatophytes ■ Non-dermatophytes

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- Onycholysis (distal or proximal).
- Distal or proximal Subungual hyperkeratosis.
- Thickening of nail plate.
- Caused by *T. rubrum*, *T. mentagrophytes*.

How to differentiate from psoriasis?

Psoriasis is a systemic disease affecting all nails (finger & toenails) while fungal infection usually affects only few nails.

How to confirm the diagnosis:

Send nail clipping for KOH & fungal culture.

PAS stain.

Treatment: oral antifungal.



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Tinea manuum

- Diffuse dry scaling over the palm & itching.



Tinea corporis

2 Types:

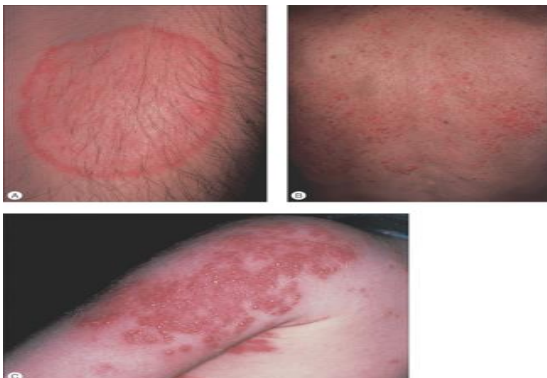
1. Hyperkeratotic type (*T. rubrum*) well-demarcated annular red hyperkeratotic plaque with central clearing (Ringworm).

If it is limited (few plaques), it can be treated topically.

If extensive it needs oral anti-fungal.

2. Inflammatory type (*T. mentagrophyte*) well-demarcated edematous red plaque with superimposed pustules

- Trunk



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Tinea capitis

Well circumscribed pruritic (itchy) scaling area of hair loss

- Black dot endothrix (*T. tonsurans*).
- Ectothrix (*M. canis*, *M. audouinii*).
Affect the outer part of the hair shaft, usually present with alopecia, itching & scaling. *M. canis* appear green on lamp's wood which is characteristic.
- Kerion (*T. verrucosum*).
Inflammatory form of tinea.
- Favus (*T. schoenleinii*).



Tinea Capitis – commonest in children. Presents as non-itchy patches of hair loss with broken hairs.

Hairless patch
The hair shaft is broken down because the dermatophytes are invading the hair shaft

12 YRS old boy with 2 weeks hx of very itchy skin lesions?

Describe the lesion: multiple annular erythematous scaly plaques.



Tinea cruris

It affects the flexures (the axilla & groin).



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KERION



Inflammation with crust



Endothrix



Ectothrix



Kerion



Favus characterized by large yellowish crust.

Investigation:

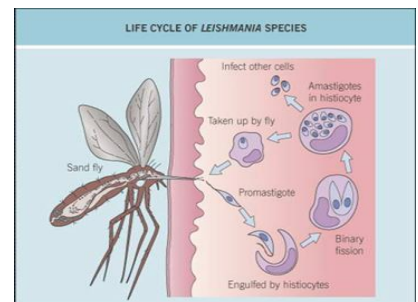
- Scraping, hair plug, nail clippings---KOH and culture.
- Wood's light.

Rx:

- Topical (terbinafine, daktarin)
 - Oral (Griseofulvin for peds, terbinafine, itraconazole): in extensive cases **Hair, nail involvement.**
- Tinea capitis & onychomycosis you MUST treat with oral antifungal.**

Protozoal Leishmaniasis

- Includes a spectrum of chronic infections in humans and several animal species.
- There are three major clinical patterns:
 - (1) Cutaneous (localised or diffuse): **the common pattern in KSA.**
 - (2) Mucocutaneous, which affects both the skin and mucosal surfaces.
 - (3) Visceral (post kala-azar), which affects the organs of the mononuclear phagocyte system, e.g. liver, spleen.
- **Transmitted by sand fly.**
- L.tropica, L. major.
- Sand fly (promastigote).
- Macrophage (Amastigote).
- **Leishman-Donovan bodies.**
- Painless papule slowly enlarge over several weeks into nodule or plaque then become ulcerated or verrucous.
- Exposed sites such as face, neck, arms, and legs are most



commonly involved.

Dx:

- Confirmed by demonstrating the presence of amastigotes in dermal macrophages within skin biopsy specimens, tissue impression smears (touch preparations), and smears of dermal scrapings.
- Giemsa stain.
- Ulcer is the location of choice for dermal scrapings, a biopsy specimen or a needle aspirate; the latter two types of samples may be used for culture and PCR.
- The leishmania culture is rarely done.
- Leishmanin test (rarely done).
- PCR-based methods are the most sensitive & specific diagnostic tests.

Management:

- Resolve spontaneously leaving a scar thus need treatment.
- Intralesional pentavalent antimony.
- Parenteral pentavalent antimonials (Sodium stibogluconate) are the treatment of choice for cutaneous and mucocutaneous leishmaniasis.
- Liposomal amphotericin B for visceral leishmaniasis.
- Topical Paromomycin sulfate.
- Fluconazole or itraconazole.
- Cryotherapy.

Complication: disfiguring scarring



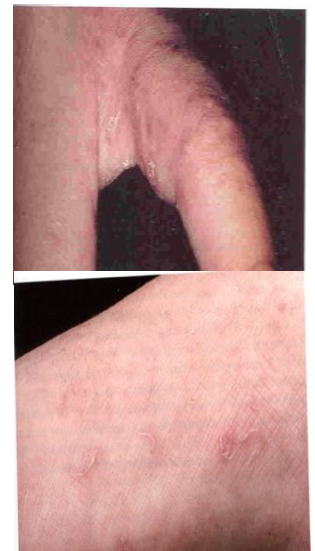
Infestations

Scabies

- The female *sarcoptes scabiei var hominis* mite lays 60-90 eggs in her 30-day lifespan, although less than 10% of the eggs result in mature mites.
- Mite: *Sarcoptes scabiei var. hominis*.
- It resides in burrows in the stratum corneum laying eggs then dying and the eggs will mature in 2 weeks period and the cycle repeated.
- Severe itching worse after bathing and at night.
- Skin lesions are secondary eczematous eruption due to immune reaction to the mite and eggs.
- Sites: finger webs, flexor of the wrist, axillae, areolae, umbilicus, lower abdomen and scrotum.
- Linear burrows are a pathognomonic sign that represent intraepidermal tunnel.
- Small erythematous papules are present in association with a variable degree of excoriation vesicles, indurated nodules or crusting.
- Might be complicated by secondary bacterial infection.

When to suspect scabies?

1. Pruritus mainly at night.
2. Other member of the family also having severe pruritus.
3. Pruritus and skin eruption is more severe in the flexors.



Investigation:

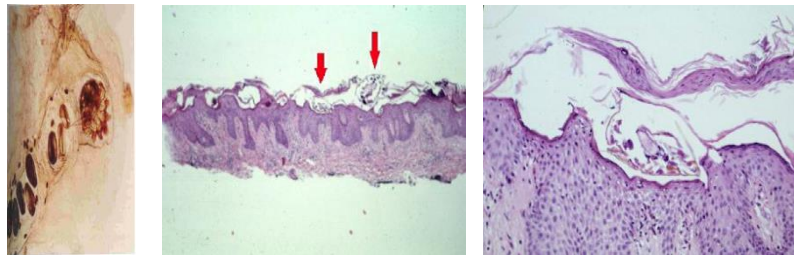
- India ink or gentian violet then removed by alcohol to identify the burrows.
- A drop of mineral oil on the lesion then scraped away with a surgical blade.
- Demonstration of the mite under the microscope.

Management:

- Treatment of family members and contact even if asymptomatic!
- Washing clothing and bed linen in hot water (60 c).
- Permethrin 5% cream (standard topical scabicide).
From neck to toes for 8 hours & repeat it after a week.
Permethrin is safe for children & pregnancy.
- Lindane 1% lotion or cream (not safe in children d.t neurotoxicity).
- Crotamiton 10% cream for 5 days.
- 2.5% Sulfur preparation (safe in children and pregnancy).
- Oral ivermectin.
- Itching may persist for up to a month, even following successful treatment.



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Mite under the stratum corneum

Pediculosis capitis

- Common in school children.
- Caused by head louse (pediculus humanus var capitis).
- A mature female head louse lays 3-6 eggs (nits) per day. Nits are white and less than 1 mm long. Nymphs (immature lice) hatch from the nits after 8-9 days, reach maturity in 9-12 days, and live as adults for about 30 days.
Look for the nits in the occipital scalp & post auricular areas.
- Severe itching of the scalp.
- Posterior cervical LN.
- Secondary bacterial impetigo.
- The diagnosis is clinical.
- **Management:**
- Examination of other family members and treated simultaneously.
- Wash all fomites (combs, hats, scarves) in hot water (louse dies at temp. 53.5 c).
- Combing with a metal nit comb.
- Pyrethrin and Permethrin lotion or cream or shampoo 1% and 5% for 10 min then rinsed off. Repeat after 1 week
- Malathion 0,5% lotion.
- Lindane (neurotoxicity).
- Topical Ivermectin 0.5%.



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Questions:

What is common between scabies and dermatitis:

- A. Pruritus
- B. Xerosis
- C. Inflammation
- D. Infection

Answer: A

A young patient presented with chronic hypopigmented and hyperpigmented scaly patches in immunofluorescence showed orange deposition, what is the most likely organism?

- A. Scabies
- B. Malassezia furfur
- C. Dermatophytosis
- D. Staph. Aureus

Answer: B

A 35-year-old male presented to the clinic complaining of intense itching over the body for the last two weeks that prevent him from sleeping. On skin examination he was found to have inflammatory papules and eczematous dermatitis over wrists, axillae and genitalia. The dermatologist thinks the patient has scabies. Which of the following will be part of the management of this patient?

- A. Using fine-toothed comb to remove the hair nits
- B. Finding the mite by Tzanck smear from the lesions
- C. Treatment of the family members and contacts
- D. Using Imiquimod cream over the lesions

Answer: C

Woods lamp is helpful in diagnosing which one of the following?

- A. Lichen planus.
- B. Tinea capitis.
- C. Atopic dermatitis.
- D. Psoriasis.

Answer: B

Under Wood's lamp, what is the color of "Tinea Versicolor"?

- A. Red
- B. Blue
- C. Yellow green
- D. Milky white

Answer: C

Boy came with his parents complaining of grey patch on his scalp what's the diagnosis?

- A. M. Audouinii.
- B. T. Schoenleinii.
- C. T. verrucosum.
- D. T. tonsurans

Answer: A (tinea capitis)

What is the test which helps in the diagnosis of an annular itchy in the face

- A. Gram's stain
- B. Potassium hydroxide test KOH
- C. Tissue smear
- D. Wood's light test

Answer: B Tinea corporis

Post herpes zoster neuralgia is associated with?

- A. Lumbar
- B. Cervical
- C. Thoracic
- D. Ophthalmic

Answer: C

A 5 year old boy is brought to the clinic with lesions on neck and trunk. On examination there are several smooth reddish elevated papules with a central punctum. What is the most likely diagnosis?

- A. Melloscum contagiosum
- B. Herpes simplex
- C. Warts
- D. Varicella zoster

Answer: A

A case diagnosed with Varicella. What is TRUE about varicella?

- A. Mainly a disease of Adulthood
- B. Adulthood disease carry more risk of complications
- C. Caused by reactivation of Varicella.Zoster virus
- D. Does not cause scarring

Answer: B

Q86: A 29-years-old female, in her second trimester of pregnancy, she gave history of contact with her nephew who has chicken pox today, she is worried that she might get the infection as she never had varicella before. What will be your management?

- A. Reassurance
- B. Start varicella zoster immunoglobulin
- C. Start Acyclovir only if she develops signs and symptoms of chickenpox
- D. Give her sick leave to avoid further contact with that student

Answer: B