

Common skin infections

Objectives: Not given

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Based on Dr. Eman slides and notes

[Color index: Important | Notes | Extra]

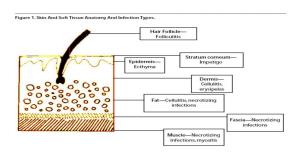
Skin infections

Bacterial	Viral	Fungal	Protozoal	Infestations
 Impetigo Erysipelas Cellulitis Furuncle Folliculitis Carbuncle Ecthyma Ecthyma gangrenosum Erythrasma SSSS 	 Warts Molluscum contagiosum Herpes simplex Varicella Herpes zoster 	 Candida Dermatophyte Pityriasis versicolor 	• Leishmaniasis	ScabiesPediculosis capitis

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 infection?
 - Colonisation: Bacteria are present, but causing no harm
 - 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.

■ Bacterial skin infections:



1) <u>Impetigo:</u> It is the most common skin infection

- Acute superficial cutaneous Infection
- 2 forms (bullous 30%, non-bullous **70%**)
- The causative organism is usually Staphylococcus Aureus (>90% cases), but less often can be strept. Pyogenes (group A beta hemolytic streptococci).
- Very contagious, autoinoculation is common.
- Can cause systemic symptoms (fever, LAD lymphadenopathy)
- Children especially if they have atypical dermatitis, Adult.
- Prognosis: Scarring is unusual, but postinflammatory hyperpigmentation or hypopigmentation.
- Complications: Post-strept GN (Rare):
 - Nephrogenic syndrome associated strains 49,55,57, 59.
- Investigations: Swab : Gram stain and culture show gram positive cocci





Non-bullous impetigo (70%)

- 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 crust (Honey-colored crust). Crust = secondary lesion.
- Predisposing factors:
 - Warm, humid climate, poor hygiene, trauma, insect bites and immunosuppression.





Bullous impetigo (30%)

- Due to staph aureus. Phage group 2 toxins not directly caused by staph itself > caused by staph toxins (staph aureus group 2 exotoxins) which cleave desmoglein-1 in the epidermis which lead to the formation of bulla. Desmoglobin function: connect the keratinocytes together. They are the target of staph exotoxins.
- Newborn and in renal patients. & immunocompromised
- Face, hands, diaper area.
- Bullae (flaccid) on grossly normal skin easily ruptured
- Manifests as clusters of vesicles or pustules that enlarge rapidly to form bullae. The bullae burst and expose large bases which become covered with Honey-colored varnish or crust





Treatment

- Remove crust
- Localized:
 - Topical Abx (bactroban, bacitracin).
 - Compresses to loosen crusts.
 - Intranasal mupirocin for periodic decolonization in carriers.
- Recurrent impetigo(BIDx 5d/month) do nasal swab + give prophylactic Abx
- Severe or widespread: use systemic antibiotics (must cover both staph/GABHS) such as:
 - Penicillinase- resistant PCN, 1st/2nd generation cephalosporin, clindamycin,or erythromycin (esp if penicillin allergic)

2) 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 if upper lip, nose or eye affected. Dangerous zone of the face
- 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 broad spectrum antibiotics."Gram -ve folliculitis: give isotretinoin"
- Pseudomonas ("Hot tub folliculitis") after swimming / jacuzzi
- Yeasts (candida and pityrosporum) very itchy
- Demodex usually occurs in pts with rosacea
- Treatment:
 - Topical antiseptics such as Chlorhexidine
 - Topical antibiotics, such as Fusidic acid, Mupirocin or clindamycin.
 - More <u>resistant cases</u> may need oral antibiotics (similar to impetigo). Always swab the nostrils
 - Hot tub folliculitis (P.aeruginosa) usually self limited (ciprofloxacin in severe cases).
 - Gram negative trimethoprim, isotretinoin.

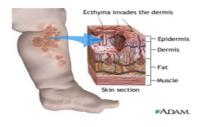


3) Furunculosis (boils) and carbuncles:

- Deeper **Staphylococcal** abscess of the hair follicle
- Coalescence of boils leads to the formation of a carbuncle
- presents as red tender nodule common in diabetic/immunocompromised pts
- Treatment is with systemic antibiotics and may need incision and drainage.
- Consider looking for underlying causes, such as diabetes.

4) Ecthyma and Ecthyma gangrenosum

- Ecthyma is a skin infection similar to impetigo but more deeply invasive. Usually caused by a streptococcus infection and might be caused by staph, ecthyma goes through the outer layer (epidermis) to the deeper layer (dermis) of skin, possibly causing scars. Not a serious infection
- Ecthyma gangrenosum is a bacterial skin infection caused by pseudomonas aeruginosa that
 usually occurs in immunocompromised individuals. Necrotic ulcerated lesions, a serious infection. Do
 blood culture (sepsis).





Ecthyma

Ecthyma gangrenosum

5) Erysipelas:

- Superficial infection with marked extending to the other dermis lymphatics involvement.
- Sharply demarcated unilateral, red edematous plague.
- Infants, young children, & elderly patients (most commonly..).
- Face, leg
- Beta hemolytic group A Strept.
- Risk factor; Minor abrasion / lymphatic dysfunction sup. Lymph vessels.
- Might be associated with Leukocytosis & fever.
- Management :
 - Smear for gram stain and culture (fluid, blood)
 - Cold compressor
 - Oral antibiotics or I.V. for severe infection
 - Oral penicillin or Erythromycin

6) Cellulitis:

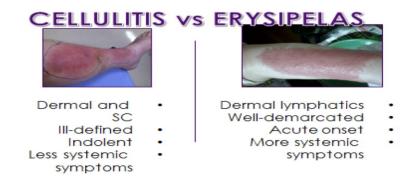
- Deeper involvement of the SC
- Acute, raised, hot, tender, erythematous (leg)
- Strept. Pyogenes or staph.aureus
- Cutaneous abrasion or ulcer
- Palpable, tender LN
- Fever, leukocytosis
- Risk factors: DM, HTN, obesity, immunocompromised patients, vascular insufficiency.
- Complicated by lymphedema if recurrent.







- Management: Swab + blood culture or skin biopsy
 - Semisynthetic Penicillin or Erythromycin if allergic. Oral: immunocompetent
 - 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.



7) Erythrasma:

- Colonisation of axilla or groin with Corynebacterium Minutissimum (Gram positive bacilli).
- Red, brown patch.
- Asymptomatic, flexural may be itchy. DDx: inverse psoriasis, contact dermatitis,tinea (fungal).
- Management:
 - Swab
 - Wood's lamp: Coral-red fluorescence because of porphyrin
 - Topical: erythromycin, fusidic acid, clindamycin.
 - Oral erythromycin x7 days. Resistant cases





8) Staphylococcal Scalded Skin Syndrome (SSSS): Quadri S syndrome

★ Emergency bacterial infection

- A superficial blistering condition caused by <u>exfoliative toxins</u> of certain strains of Staph Aureus Same as bullous impetigo (staph exotoxins which act on desmoglein-1 which lead to cleavage and bullae formation "flaccid")
- Usually in children less than 5 years old. Does not affect the mucous membranes, skin only
- Characterised by blistering and desquamation of the skin and Nikolsky's sign (shearing of the
 epidermis with gentle pressure), even in areas that are not obviously affected.
- Begins with a prodrome of pyrexia and malaise, often with signs and symptoms of an upper tract infection. You should take a throat swab
- 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 (causes of death) include hypothermia, dehydration and secondary infection.
- Treatment: ABC, admit for IV antibiotics and fluids, may need referral to burn center.









■ Viral skin infections:

1) 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). The most common
- Plantar warts (verruca plantaris).
- Plane (flat) warts(verruca plana).
- Genital warts (condyloma acuminata and bowenoid papulosis).
- Mucosal warts.

• Management:

- Involute spontaneously.
- Cryotherapy
- Topical keratolytics: Salicylic acid, TCA
- Electrocautery, curettage
- Laser
- Topical retinoids in flat warts
- Others: bleomycin, cantharidin.
- PPD, Candidal antigen.

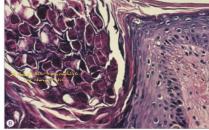
- FFD, V	- PPD , Candidal antigen.				
Common warts (verruca vulgaris)	 Caused by HPV type 1,2,3,4,7,54 a DNA virus Affects the Hands, could be periungual Common in Children Presents as hyperkeratotic (verrucous) papules. Koebner phenomenon d.t autoinoculation 				
Plane (flat) warts (verruca plana)	 Affects Face, back of hands. Caused by HPV type 3,10, 28. Flat skin colored papules. Slightly erythematous 				
Plantar warts (verruca plantaris)	- Affects the soles. 1ry lesion: plaque or papule - Caused by HPV type 1,2,4,60,63 To be differentiated from plantar corn. Corn مسمار القدم is more painful on pressure and it is not caused by HPV + not infectious. If you see black dots (dilated blood vessels) = it is wart not corn. May take 2 yrs to resolve spontaneously. To avoid infecting other people we prefer tx (eg.: salicylic acid, curettage, vascular laser or topical retinoid).				
Genital warts (condyloma acuminata and bowenoid papulosis)	 Most common STD. Condylomata acuminata. Cauliflower like Penile, vulvar skin, mm, perianal area Sexual partner Child: sexual abuse Caused by HPV type 6,11(nononcogene) 16,18 (Oncogenic strains). Vaccination even if she is already infected to protect from other strains. Management: Screen the sexual partner, screen other STD (syphilis, HIV, 				

Hepatitis), perform a pap smear and then decide which tx to give.

2) Molluscum contagiosum:

- Caused by Poxvirus (DNA virus)
- Common Children by contact Molluscum contagiosum has 2 types, 2 is sexually transmitted
- In adults : immunosuppression , STD
- Face, neck or genitalia
- Skin colored papules with Central punctum (umbilication).
- Koebner phenomenon d.t autoinoculation.
- H/P: Henderson patterson bodies
- Management:
 - Involute spontaneously
 - Curettage, cryotherapy
 - Other: Salicylic acid

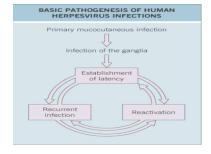






3) Herpes simplex:

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











*Multiple vesicles on an erythematous background, painful.

*Eczema herprticum

*Whitlow

• Diagnosis:

- **Tzanck Smear** (Multinucleated giant cell)
- Direct fluorescent antibody (DFA)
- Viral culture (most definitive).

• Treatment:

Oral / IV acyclovir for Genital, Recurrent, immune suppressed, neonatal, Eczema Herpeticum.



4) Varicella (chicken pox):

- Initial infection with varicella zoster virus (VZV) HSV-3
- Incubation period : 2 weeks
- Prodrome of respiratory coryza followed by disseminated red macules with central vesicles or pustules. Polymorphic: vesicles, crustation or papules.
- The whole illness: 3 weeks.
- The patient contagious 5 days before and 5 days after skin eruption.
- Children.
- Adults : immunosuppression, pneumonia
- **Diagnosis:** Tzanck Smear, DFA, Viral culture.
- **Treatment:** symptomatic for itching, systemic antiviral in immunocompromised patients.
- Vaccination
- Varicella in pregnancy:
 - 1st and 2nd trimester: risk of varicella, embryopathy syndrome, abortion
 - 3rd trimester congenital varicella (cardiac, skin and skeletal deformities).
 - Pregnant patients with varicella should receive VZ immunoglobulin and antiviral therapy. Within 36 h

5) Herpes zoster:

- Following initial varicella infection VZV remain latent in sensory ganglia when reactivated it will affects skin dermatome supplied by this ganglia.
- Adult, immunocompromised.
- Prodromal pain <u>dermatomal</u> (grouped blisters and vesicles on background of erythema) - post herpetic neuralgia. 1st & 2nd branch of trigeminal
- Diagnosis: Tzanck Smear, DFA, Viral culture.
- **Treatment:** Analgesia, antiviral: immunosuppressed, wide spread.

▼ Fungal (Superficial mycosis):

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

1) Candidiasis:

- Physiological (old age, neonate and pregnancy).
- Pathological (DM, HIV and organ transplant, on immunosuppression).
- latrogenic (long course of Antibiotics)
- Candida albican (normal commensal of GIT):
 - Napkin candidosis & Intertrigo(satellite lesions).
 - Paronychia
 - Mucous membrane candidiasis: oral,urogenital and oesophagus.
 - Vulvovaginitis: irritation, discharge
 - Candida folliculitis.
 - Generalized Systemic infection
 - Chronic mucocutaneous candidiasis



















• Management:

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

2) Pityriasis versicolor:

- Malassezia furfur (hyphae)/Pityrosporum orbiculare (yeast)
- Upper Trunk
- Asymptomatic
- Yellowish-brown or hypopigmented scaly patches. Once the rash has gone it leaves hypopigmented macules which take time to tan.



CASE: 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, pityriasis alba, post-inflammatory, pityriasis versicolor.



• Investigations:

- 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
- Oral Antifungal (azoles)
- Recurrence

3) **Dermatophyte infections:** the most common fungal infection

3 main genera:

- Trichophyton
- Microsporum
- Epidermophyton
- Invade the keratin of the stratum corneum

Can be:

- Anthropophilic: contracted from humans.
- Zoophilic: contracted from animals.
- Geographic: contracted from soil.
- Clinical appearance depends on the organism involved, the site and the host reaction.
- Affects: skin, hair and nails.

Tinea pedis	1.Erosive interdigitalis (web space) ,athletes due to humidity which allows the infection to grow. 2. Hyperkeratotic type (T. rubrum) 3. Inflammatory type (T.mentagrophyte)
Tinea unguium (Onychomycosis)	 Different presentation including: White Onychomycosis might be caused by candida or Dph Onycholysis Distal or proximal Sub Ungual hyperkeratosis Thickening of nail plate Caused by T. rubrum, T.mentagrophytes
Tinea manuum	Diffuse dry scaling over the palm
Tinea corporis	2 Types: 1. Hyperkeratotic type (T.rubrum) well-demarcated annular red hyperkeratotic plaque with central clearing (Ringworm) 2. Inflammatory type (T.mentagrophyte) well- demarcated edematous red plaque with superimposed pustules - Trunk CASE:12 YRS old boy with 2 weeks hx of very itchy skin lesions?
Tinea cruris	infection of the groin with a dermatophyte fungus. It is most often seen in adult men. Tinea cruris is commonly known as jock itch.
Tinea capitis	Well circumscribed pruritic scaling area of hair loss: - Black dot endothrix (T.tonsurans) Ectothrix (M.canis, M. audouinii) Kerion (T.verrucosum) Favus (T.schoenleinii). ◆ Kerion: A kerion is an abscess caused by fungal infection. It most often occurs on the scalp (tinea capitis), but it may also arise on any site exposed to the fungus such as face (tinea faciei) and upper limbs (tinea corporis). It is often misdiagnosed as bacterial infection. Management: - Education - Scraping, hair plug, nail clippings - KOH and culture - Wood's light - Topical (terbinafine, daktarin) - Oral (Griseofulvin, terbinafine, itraconazole): extensive, Hair, nail. You treat with topical except scalp & nail you give systemic(oral) tx bc it will end up with scarring alopecia.

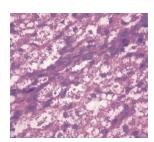
◄ Protozoal:

Leishmaniasis:

- Includes a spectrum of chronic infections in humans and several animal species.
- There are three major clinical patterns:
 - 1) Cutaneous, (localised or diffuse)
 - 2) Mucocutaneous, which affects both the skin and mucosal surfaces.
 - 3) Visceral (post kala-azar), which affects the organs of the mononuclear phagocyte system (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 a nodule or plaque then become ulcerated or verrucous.
- Exposed sites such as face, neck, arms, and legs are most commonly involved
- Diagnosis:
 - Confirmed by demonstrating the presence of amastigotes in dermal macrophages within biopsy skin specimens, tissue impression smears (touch preparations), and smears of dermal scrapings.
 - Giemsa stain
 - Ulcer is the location of choice for dermal scraping, a biopsy specimen or a needle aspirate; the later 2 types of samples may be used for culture and PCR.
 - Leishmanin test
 - PCR-based methods are the most sensitive and specific diagnostic tests.

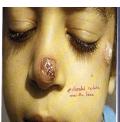
Management

- Resolve spontaneously leaving a scar
- 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











◄ Infestations:

1) Scabies: الجرب

- 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 residue in burrows in the stratum corneum laying eggs then dying and the eggs will maturate in 2 weeks period and the cycle repeated.
- Severe itching worse after bathing and at night.
- Skin lesions are immune reaction to the mite and secondary eczematous eruption due to 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 crustation.
- 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)
- 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).
- Itching may persist for up to a month, even following successful treatment.

2) Pediculosis capitis: القمل

- 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.
- Severe itching of the scalp. Posterior cervical LN Secondary bacterial impetigo.

• 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.
- Malathion 0.5% lotion / Lindane (neurotoxicity) / Topical Ivermectin 0.5%





