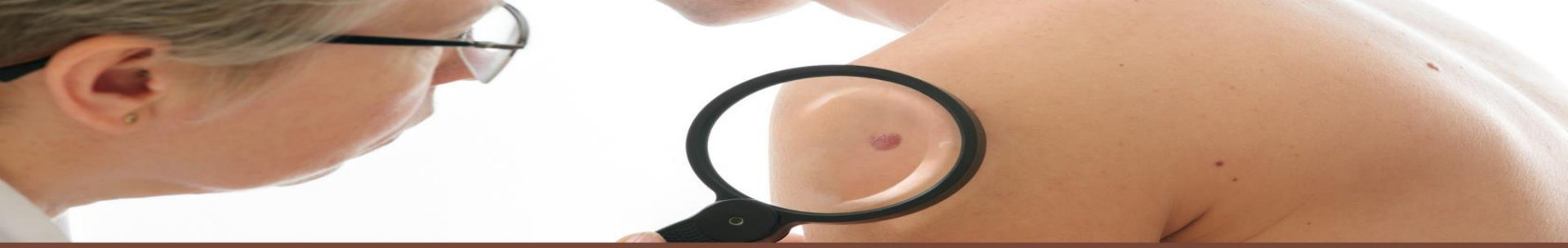

433 Teams

DERMATOLOGY

L5- common skin infections

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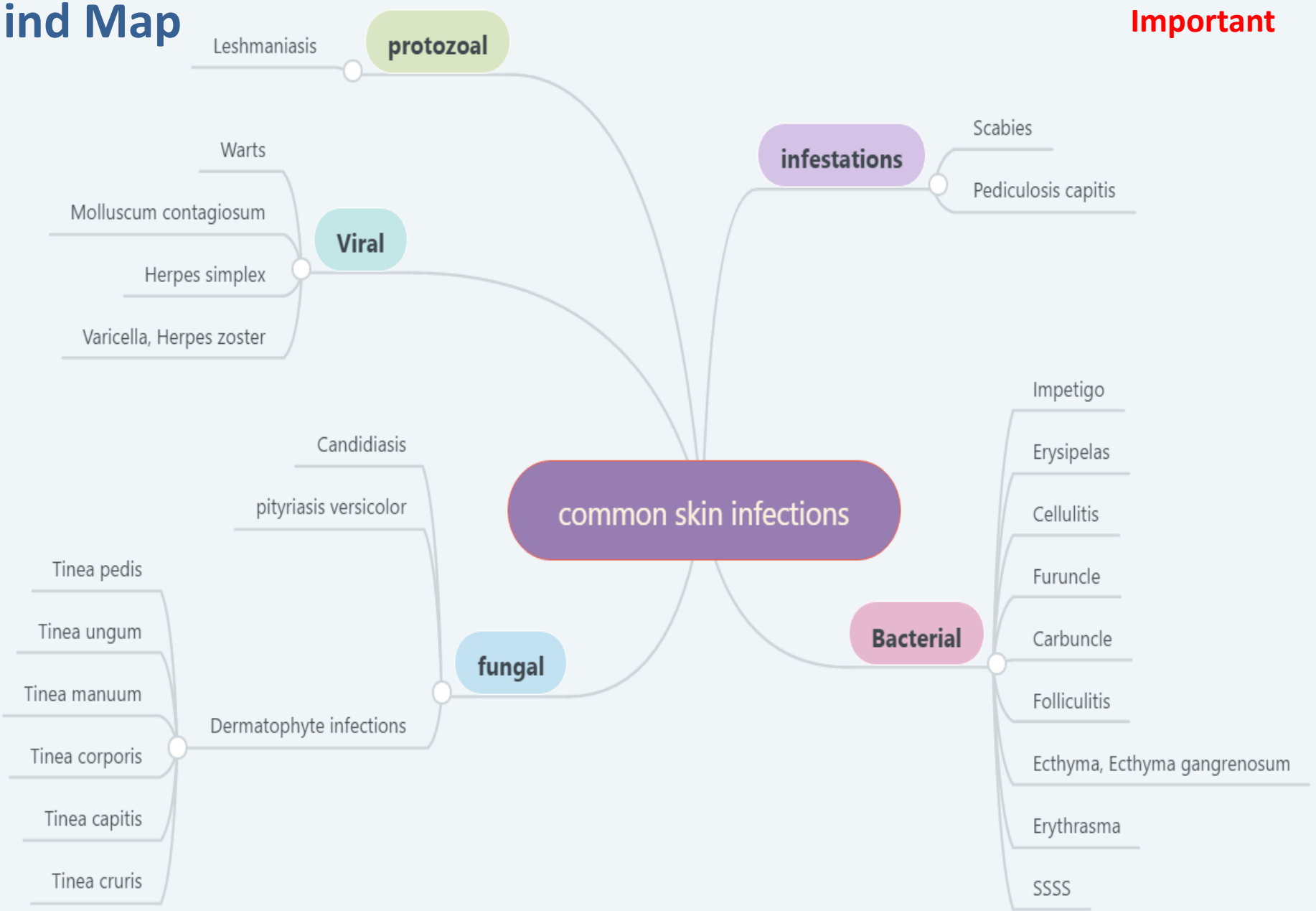
جامعة الملك سعود
King Saud University

Mind Map

Important



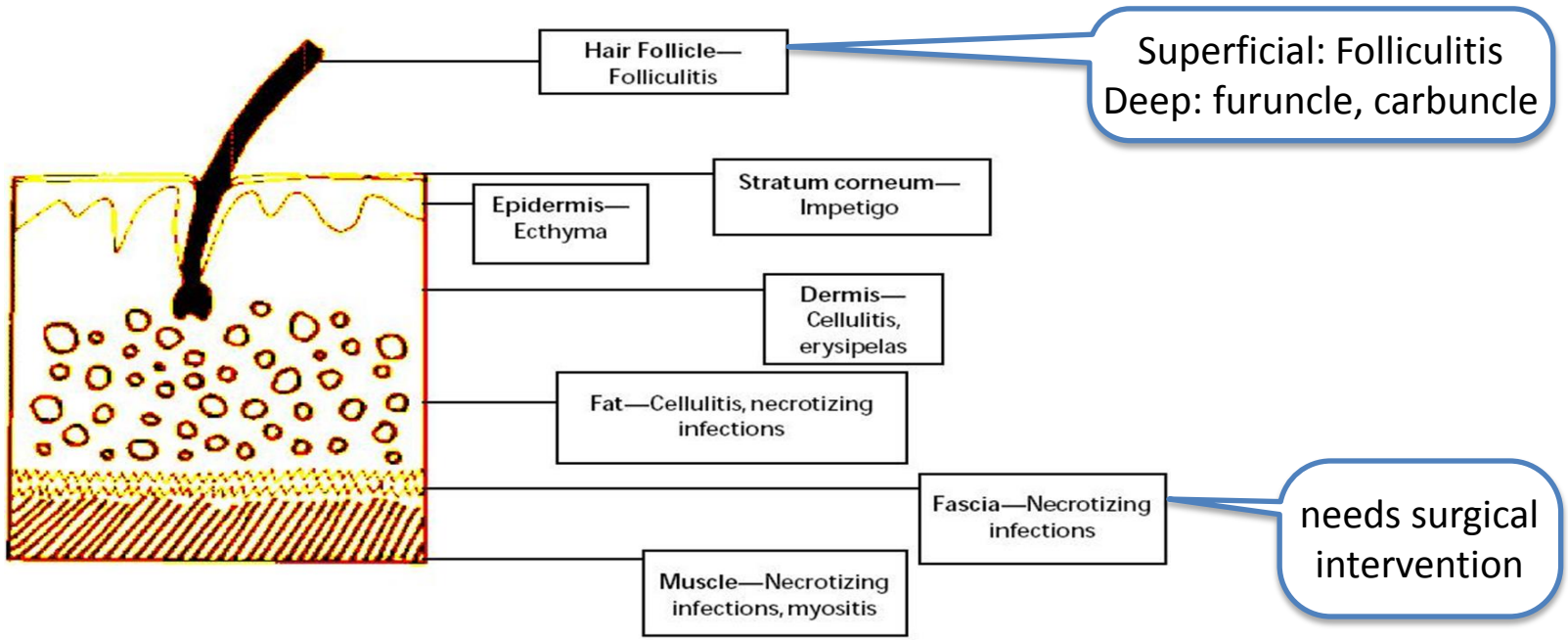
Why does skin get infected?

- There are multiple types of organisms present normally on our skin as normal flora Staphylococcus epidermidis and yeasts
- .The presence of these organisms does not lead to skin infection. However, a break in the epidermal integrity can allow organisms to enter and become pathogenic. This can occur as a result of trauma, ulceration, or skin disease such as eczema.

The difference between colonisation and infections:

Colonisation:	**Infection:**
Bacteria are present, but causing no harm	Bacteria are present and causing harm.

Skin & soft tissue anatomy and infection type:



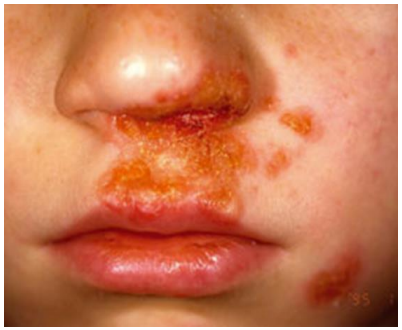
Bacterial

1. Impetigo

- Acute superficial cutaneous infection. Very contagious, auto-inoculation is common.
- The causative organism is usually **Staphylococcus Aureus (>90% cases).** Less often caused by **strept. Pyogenes (group A beta-hemolytic streptococcus)**, which may cause **complications such as, Guttat psoraisis & post-streptococcal glomerulonephritis** (Nephritogenic strains 49,55,57, .(59
- **Staphylococcus aureus attack the cells desmosomes, which lead to disruption of skin integrity and development of Nikolsky Sign.**

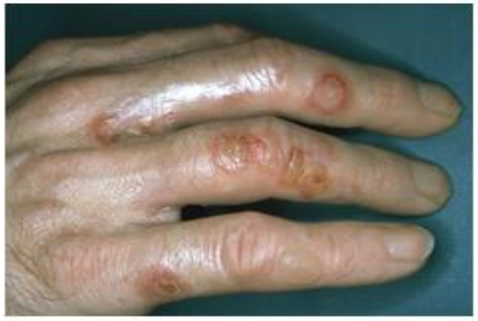
Impetigo (non-bullous)

- Manifest as clusters of vesicles or pustules with rim of erythema that rupture and develop **golden-yellow (Honey) crust.**
- **Predisposing factors:** Warm humid climate, poor hygiene, trauma, insect bites, immunosuppression and **eczema**.



Impetigo (bullous)

- Face, hands , diaper area
- **Flaccid bullae** on grossly normal skin.
- Due to staph aureus of phage group II
- Newborn and in renal patients. **(Toxins clearance is reduced)**
- staph.aureus are found on normal skin and Associated with nasal or perianal carriage.



Impetigo

Prognosis:

Scarring is unusual, but May cause post-inflammatory hyper-pigmentation or hypo-pigmentation.

Investigation:

Swab : Gram stain and culture show gram positive cocci.

Manegement:

- Remove crust & use localised topical antibiotics (**bactroban, bacitracin**(

Recurrent impetigo:

- periodic decolonization by using **Mupirocin** (BID for 5d every month) **over the colonising sites (intra-nasal & perianal).**

Severe or widespread :

systemic antibiotics (must cover both staph/group A beta-hemolytic streptococci (**GABHS**)) such as :

- Penicillinase resistant penicillins.
- 1st/2nd generation cephalosporin .
- Clindamycin or erythromycin (if allergic to penicillin(

2-Folliculitis

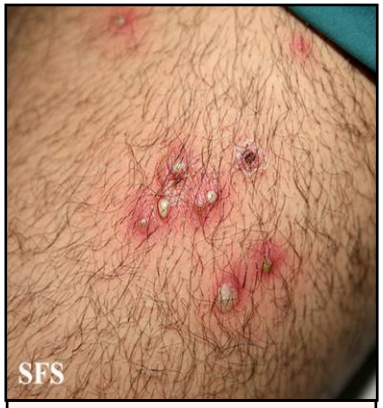
- **Superficial** hair follicle infection by;
  - ✦ **Staph Aureus (common)**
  - ✦ Gram-negative (acne patients on broad spectrum antibiotics.(
  - ✦ Pseudomonas ("Hot tub folliculitis") **over areas covered with the bathing suit.**
  - ✦ Yeasts candida and pityrosporum; **itching, has a abrupt onset and on wood's lamp appears yellowish in color(**
  - ✦ Demodex (mites that live in or near hair follicles)

Inflammation of the hair follicle.
Presents as itchy or tender papules and pustules at the follicular openings

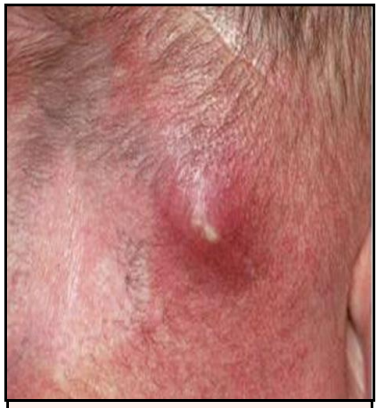
- **Complications**: abscess formation and cavernous sinus thrombosis if upper lip, nose or eye are affected.
- **Management :**
  - ✦ Topical antiseptics; **Chlorhexidine**
  - ✦ Topical antibiotics; **Fucidin**, **Mupirocin** or **clindamycin** .
  - ✦ **Oral antibiotics** in resistant cases.
  - ✦ P. aeruginosa: usually self limited (**ciprofloxacin** in severe cases).
  - ✦ Gram negative: trimethoprim, isotretinoin (**Roaccutane**).

3-Furunculosis (boils)

- **Deep** Staphylococcal abscess of the hair follicle.
- **Carbuncle**: coalescence (cluster) of boils
- **Presents as** red tender nodule
- Consider looking for underlying causes, such as diabetes
- **Management:** systemic antibiotics and may need incision and drainage.



Folliculitis



Boils

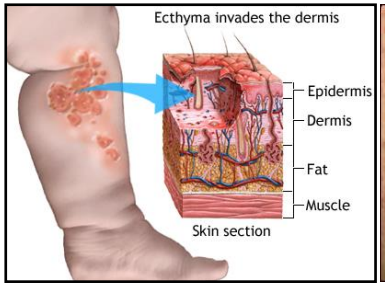
4-Ecthyma

Ecthyma

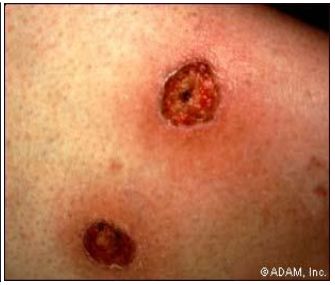
- a skin infection similar to impetigo. It is often called deep impetigo, but more deeply invasive .
- Most often caused by the **streptococcus** bacteria .
- Ecthyma goes through the epidermis and the dermis, possibly **causing scars.**

Ecthyma gangrenosum

- skin infection caused by **pseudomonas aeruginosa**
- usually occurs in **immunocompromised individuals**.
- Indurated plaques with **black eschar** and rim of erythema.



Ecthyma gangrenosum

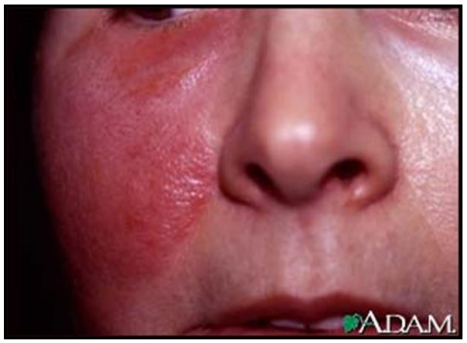


5-Erysipelas

- Superficial infection with **marked lymphatics involvement**.
- **Sharply demarcated** unilateral, red edematous. Occurs in face & leg.
- **Affects** infants, young children, & elderly patients (most commonly)
- **Beta hemolytic group A Strept.**
- Risk factor; Minor abrasion/lymphatic dysfunction .
- Might be associated with Leukocytosis & fever.

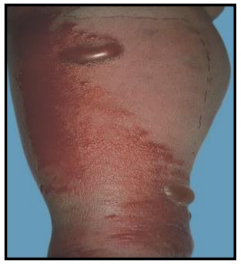
Management:

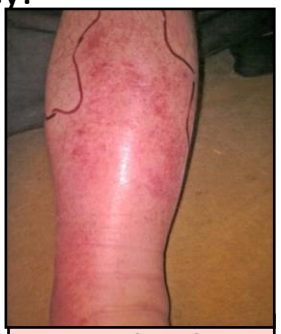
- ✤ Smear for gram stain and culture (fluid, blood)
- ✤ Cold compressor & Oral antibiotics or I.V. for severe infection.
- ✤ Oral penicillin for 10 days or Erythromycin



6-Cellulitis

- **Deeper** involvement of the **dermis and subcutaneous tissue**.
- Acute, raised, hot, tender, erythematous.(leg)
- caused by **strept. pyogenes**, **staph.aureus**
- Could be due to cutaneous abrasion or ulcer.
- **Systemic manifestation:** **Palpable, tender lymph nodes**, Fever & leucocytosis.
- **Risk factors:** DM, HTN, obesity, immunocompromised patients , vascular insufficiency.
- Complicated by **lymphedema** if recurrent.





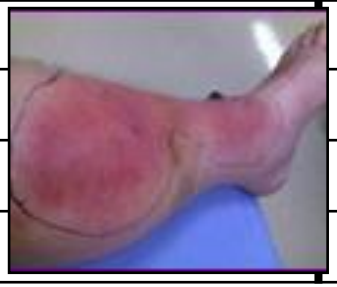
vague borders

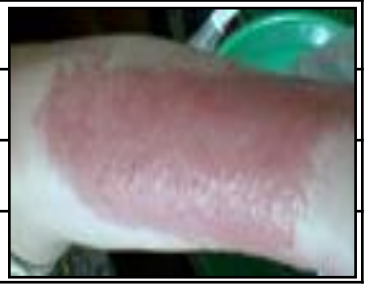
Management :

- Swab + blood culture
- **Oral 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 underlying cause

Cellulitis VS Erysipelas

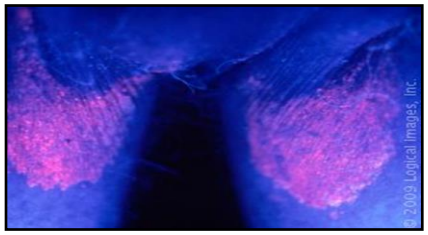
Cellulitis	Erysipelas
Dermal & SC	Dermal & lymphatics
ill-defined	well-defined
Indolent	Acute onset
Less systemic symptoms	More systemic symptoms

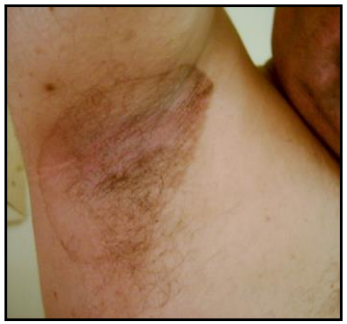




7-Erythrasma

- Dray scaly brownish erythematous plaque **commonly affect flexures (groin & axila**).
- Caused by gram (+) bacilli **Cornebacterium Minutissimum .**
- asymptomatic, **may cause itching**



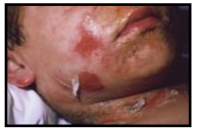


Management:

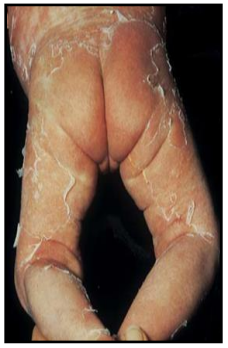
- Swab & wood's lamp: **coral-red fluorescence**
- Topical erythromycin, fucidic acid or clindamycin
- Oral erythromycin X 7d

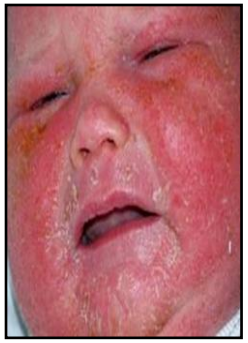
8-Staphylococcal Scalded Skin Syndrome (SSSS)

Superficial blistering and desquamation of the skin caused by exfoliative toxins of certain strains of **Staph Aureus (the strains that cause bullous impetigo)**



- Usually in **children less than 5 years old**
- **Nikolsky's sign** (shearing of the skin with gentle pressure), even in areas that are not affected.
- Begins with pyrexia and malaise, often **with signs and symptoms of an upper respiratory tract infection**
- **Generalised erythema** often **worse in the flexures** with **sparing of the mucous membranes**. large, fragile **bullae form in the erythematous areas** and then rupture.





Complications: hypothermia, dehydration and secondary infection .

Management: ABC, admit for IV fluids & antibiotics, may need referral to burns center .

Viral

A) Warts

1-Plane warts (Verruca planae)

- Caused by Human papilloma virus HPV (DNA virus).
- Affects Face, back of hands .
- Caused by HPV type **3,10**, .28
- Flat skin colored papules.

2-Common warts (verruca vulgaris)

- Caused by HPV type .**1,2**,3,**4**,7,54
- Affects the Hands, could be periungual (**around & under the nails**).
- Common in Children.
- **Presents as hyperkeratotic (verrucous) skin color papules**
- **Koebner phenomenon** due to **autoinoculation.**

3-Plantar warts (verruca plantares)

- affects the soles.
- caused by HPV type **1,2,4**, .60,63
- Often painful.
- To be differentiated from plantar corn(**"مسمار القدم" baring of the skin over the Wart will expose pin point haemorrhages).**

Management

1. Involute spontaneously (**may lead to autoinoculation**).
2. Others: **cytotoxtic agent** bleomycin , cantharidin.

3. **Destructive treatment:**

★Cryotherapy **liquid nitrogen.**

★Topical keratolytics: Salicylic acid, TCA

★Electrocautary, curettage

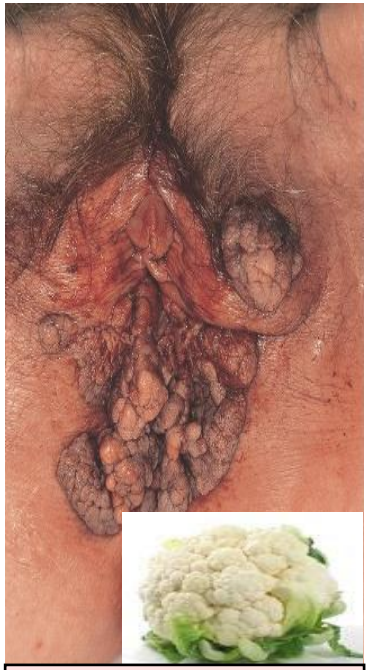
4. Laser
5. Topical retinoids in flat warts

4-Genital wart (Condylomata accuminata and bowenoid papulosis)

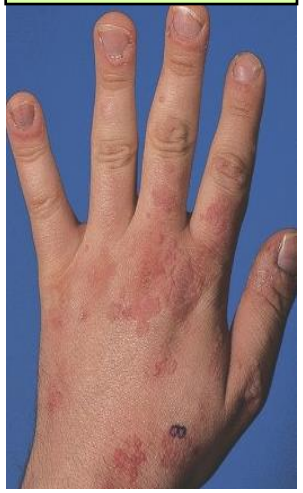
- Most common STD
- Caused by HPV type 6,11, 16, 18
- **Oncogenic strains :16, 18 (has a vaccine)**
- **Cauliflower like**
- Penile, vulvar skin, mm, perianal area
- In Child; sexual abuse or **transmitted from the mother when changing the diaper.**

Management :

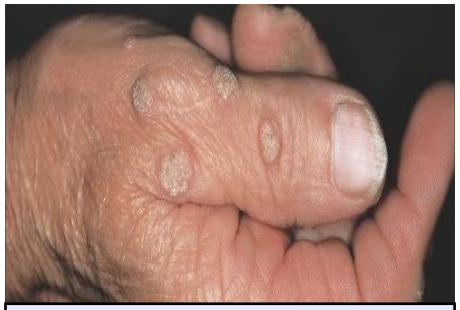
- **If female examine the vagina & cervix.**
- **perform PAP smear .**
- **examine for other STDs.**
- **examine sexual partners.**



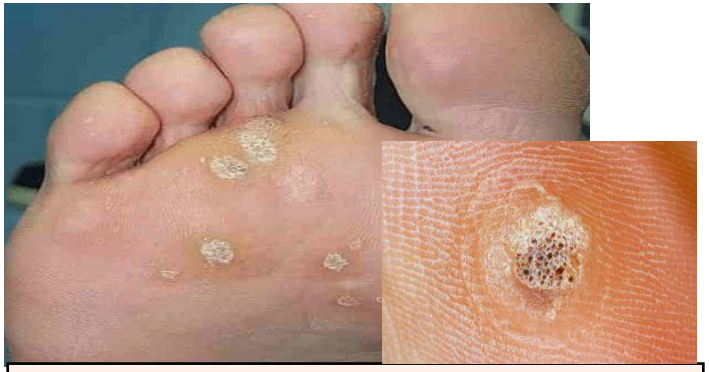
Genital wart



Plane warts



Common warts



Plantar warts

Management of Genital Worts & Clinical Manifestation of HPV

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)

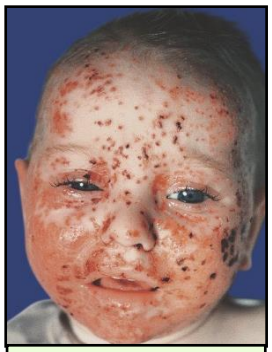
CLINICAL MANIFESTATIONS AND ASSOCIATED HPV TYPES		
	Frequently detected	**Less frequently detected**
Skin lesions		
• Common, palmar, plantar, myrmecial and mosaic warts	1, 2, 4	26, 27, 29, 41, 57, 60, 63, 65
• Flat warts	3, 10	28, 29
• Butcher's warts	7, 2	1, 3, 4, 10, 28
• Digital squamous cell carcinoma 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	

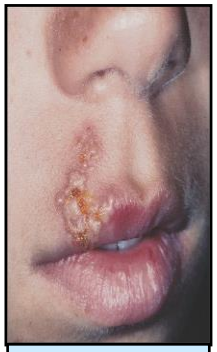
B)Herpes simplex

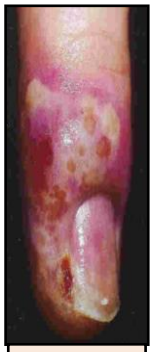
- Group of small blister
- **HSV-1 (H.labialis) & HSV-2 (genital herpes).**
- Herpetic whitlow (very painful infection of terminal phalanx).
- **Eczema herpeticum: Infection with HSV on top of previous skin disease (atopic dermatitis, pemphigus, Darier's disease).**

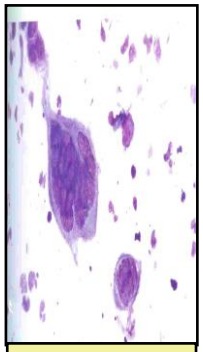
Diagnosis:
- Tzanck Smear.
- Direct fluorescent antibody (DFA).
- **Viral culture**- **most definitive**.

Treatment:
- Oral / IV acyclovir.
- Genital, Recurrent, immune suppressed, neonatal, Eczema Herpeticum.


Eczema herpeticum


Herpetic blisters

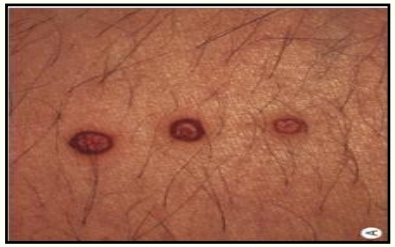

Herpetic whitlow

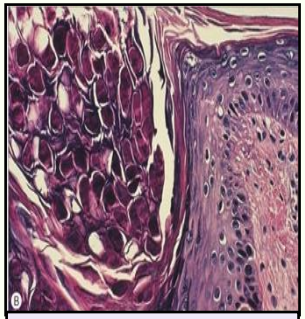

Tzanck Smear

C)Molluscum contagiosum

- Caused by **Poxvirus** (DNA virus)
- Common Children by contact
- In adults : immunosuppression , STD
- Face, neck
- **Skin colored papules with Central punctum (umbilication)**
- **Kobner phenomenon due to autoinoculation**
- **H/P**: **Hunderson-patterson bodies** (very characteristic)

Management:
- Involute spontaneously(possibility of autoinoculation)
- curettage, cryotherapy
- Other : Salicylic acid




Hunderson-patterson bodies

D)Varicella (chicken pox)

- initial infection with varicella zoster virus (VZV)
- Incubation period : 2 weeks
- Prodrom of respiratory coryza followed by disseminated red macules with central vesicles or pustules .
- The whole illness : 3 weeks
- The patient contagious 5 days before and 5 days after skin eruption
- Children
- Adults : consider immunosupression , beaware of varicella pneumonia.

Diagnosis : tzanck smear , DFA , Viral culture

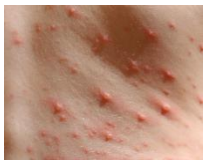
Treatment : symptomatic for itching , Systemic antiviral in immunocompromised patients.

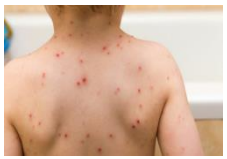
- Vaccination

- Varicella in pregnancy :

- 1st and 2nd trimester : risk of varicella embryopathy syndrome , abortion.
- 3rd trimester congenital varicella.

pregnant patients with varicella should receive VZ immunoglobulin and antiviral therapy.





E)Herpes zoster

-Following initial varicella infection VZV remain latent in sensory ganglia when reactivated it will affects skin dermatome supplied by this ganglia.
-Most affected dermatomes are thoracic.
- Adult , immunocompromised.
- Prodromal pain—dermatomal (grouped blisters and vesicles on background of erythema)—post-herpetic neuralgia.

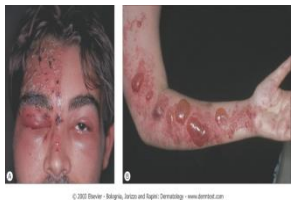
- Diagnosis :

- Tzanck Smear.
- Direct fluorescent antibody(DFA).
- Viral culture.

- Treatment :

- Analgesia,
- Antiviral : start within first 72 hrs.

immune-suppressed, wide spread → IV
Immunocompetent → oral



Pic on the left is herpes zoster ophthalmicus
May cause conjunctivitis and keratitis → so, admission, IV antiviral, and ophtha referral.
If facial nerve was affected → Ramsay Hunt Syndrome.

Fungal 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	*Malassezia furfur* (*Pityrosporum ovale*)
	Tinea nigra	*Exophiala werneckii*
	Black piedra	*Piedraia hortae*
	White piedra	*Trichosporon beigelii*
Inflammatory response common	Tinea capitis, barbae, faciei, corporis, cruris, manuum, pedis	*Trichophyton, Microsporum, Epidermophyton* spp.
	Cutaneous candidiasis	*Candida albicans*

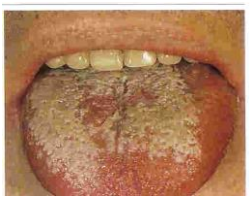
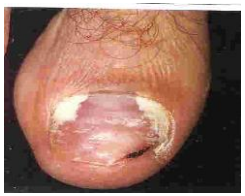
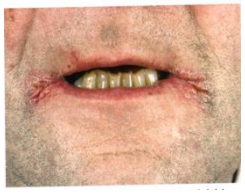
1-Candidiasis

- **physiological** (old age , neonate and pregnancy).
- **pathological** (DM, HIV and organ transplant, on immunosuppression).
- **Itrogenic** (long course of Antibiotics).

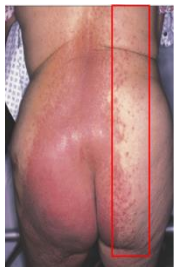
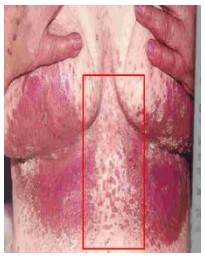
Candida albican (normal commensal of GIT):

- Napkin candidosis & Intertrigo can be differentiated bet. Other types by→(satellite lesions)
- Paronychia
- mm→ oral,urogenital and oesophagus.
- Vulvovaginitis---irritation, discharge
- Candida folliculitis
- Generalized Systemic infection
- Chronic mucocutaneous candidiasis → consider endocrinopathy (adrenal suppression, hypothyroidism).

Management :

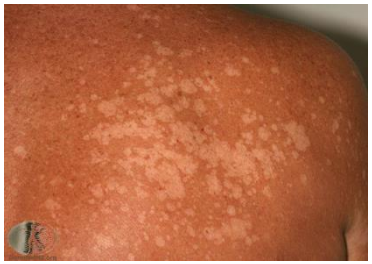
- ✓ Swab and KOH.
- ✓ Alter (avoid) moist warm environment.
- ✓ Nystatin-containing cream.
- ✓ Imidazole (Daktarin, canastein).
- ✓ Oral antifungal (itraconazole): immune suppressed, persistent infection.

*Satellite lesions → small pustules away from the main border of lesion.

2-pityriasis versicolor النخالة المبرقشة

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



Malassezia can cause seborrheic dermatitis, pityrosporum folliculitis, and pityriasis vesicolor

pityriasis versicolor -

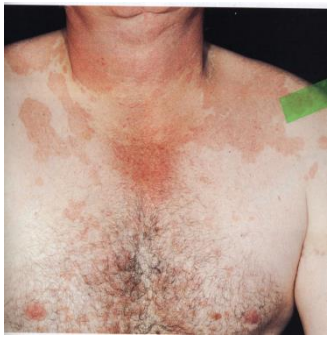
-Malassezia furfur (hyphea)

Pityrosporum orbiculare (yeast)

- Upper Trunk (seborrheic areas like chest and back).
- Might be Asymptomatic.
- Yellowish- brown or hypopigmented scaly patches.
- 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.
- Oral Antifungal (azoles).
- Recurrence is common in athletes bcuz of sweating.

3-Dermatophyte infections

3 main genera:

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

Can be:

- Anthopophilic – contracted from humans.
- Zoophilic – contracted from animals. Cause more inflammation
- Geographic – contracted from soil.

Clinical appearance depends on the organism involved, the site and the host reaction.

May invade:

- Skin: the keratin layer.
- Hair
- Nails

3-Dermatophyte infections

1. **Tinea pedis**
2. **Tinea ungum**
3. **Tinea manuum**
4. **T.Cruris**
5. **Tinea capitis**
6. **tinea corporis.**

Next slides

1- Tinea pedis

If tinea infected the sole of foot called tinea pedis, has three types:

1.Erosive interdigitalis → most common, macerated whitish patch over web space, atheletes foot, because they wear shoes all time.

2. Hyperkeratotic type caused by (T. rubrum).

3. Inflammatory type caused by (T.mentagrophyte).

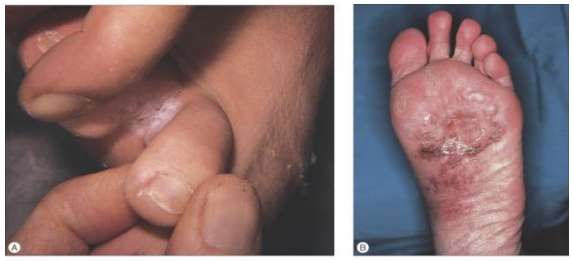


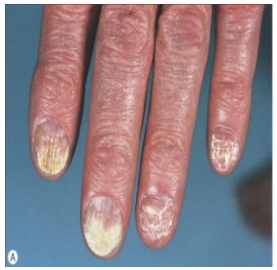
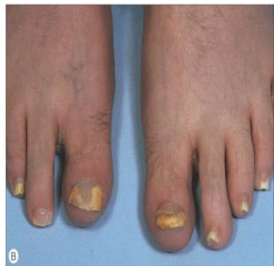
Table 77.9 The four major types of 'tinea pedis' (including dematiaceous and dermatomycoses).
*Because of the thickness of stratum corneum on plantar surfaces and the inability of *T. rubrum* to elicit an immune response sufficient to eliminate the fungus[16]. ‡Often *Pseudomonas*, *Proteus* or *Staphylococcus 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 DERMATOMYCOSES)

Type	Causative organism	Clinical features	Treatment considerations
Moccasin	*T. rubrum* **E. floccosum** *S. hyalinum* *S. dimidiatum*	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
Interdigital	*T. mentagrophytes* (var. interdigitale) *T. rubrum* *E. floccosum* **S. hyalinum** *S. dimidiatum* *Candida* spp.	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
Inflammatory (vesicular)	*T. mentagrophytes* (var. *mentagrophytes*)	Vesicles and bullae on the medial foot; associated with the dermatophytid reaction†	Topical antifungal usually sufficient
Ulcerative	*T. rubrum* *T. mentagrophytes* *E. floccosum*	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*

Tinea ungum

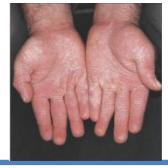



Different presentation including:

- - White Onchomycosis.
- - Oncholysis.
- - Distal or proximal Sub-ungural hyperkeratosis.
- - Thickening of nail plate.

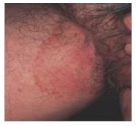
We should take nail clipping for KOH and culture.
-Caused by T. rubrum, T. mentagrophytes.

Tinea manuum



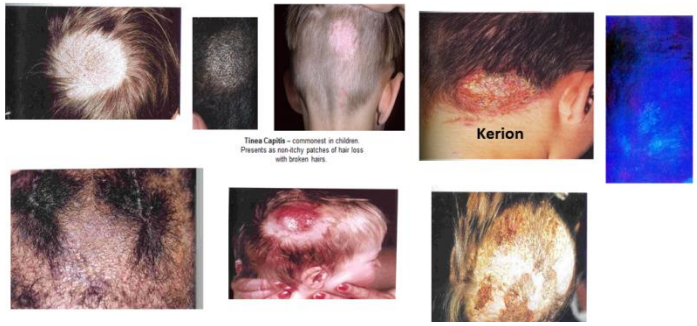
May present with hyperkeratosis or inflammation with vesicle and pustules.
diffuse dry scaling over the palm.
In pt with dermatitis (eczema) keep in mind tinea (fungal infection) as ddx.

T.cruris



Infection of genital skin

Tinea capitis



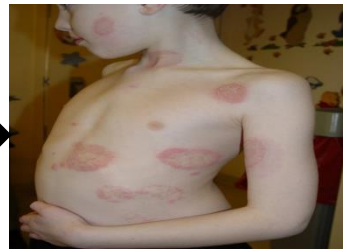
Well circumscribed pruritic scaling area of hair loss mostly affect children
Black dot endothrix (T. tonsurans)
Ectothrix affect outer layer of hair follicles or shaft (M. canis , M. audouinii).
Kerion more inflammation(T. verrucosum)
Favus (T. schoenleinii)

Mx:

Education
Scraping, hair plug, nail clippings---KOH and culture
Wood's light → greenish- yellowish.
Topical (terbinafine, daktarin)
Oral (Griseofulvin, terbinafine, itraconazole): extensive, Hair, nail→ it is self limiting but, we need to give oral rx esp. kerion because if we leave it resolve spontaneously this will leads to scarring.

Tinea Corporis

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



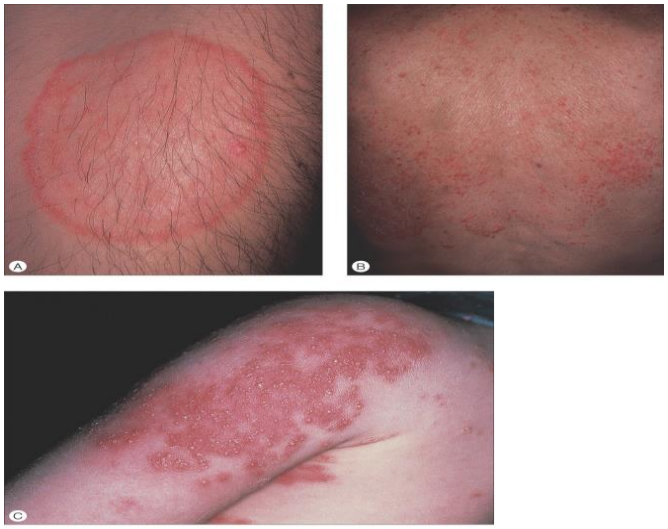
Tinea Corporis (If tinea affect trunk we called if)

2 Types :

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

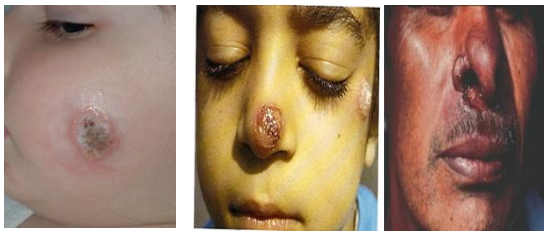
2.Inflammatory type (T.mentagrophyte) well-demarcated edematous red plaque with superimposed pustules

- Trunk



Leshmaniasis

- includes a spectrum of chronic infections in humans and several animal species. More in farms.
- **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, e.g. liver, spleen.
- **Presents as** →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.

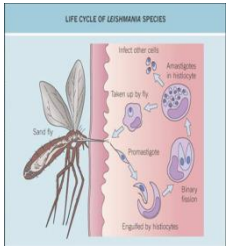


-**Transmitted** by sand fly

- L.tropica , L. major
- Sand fly (promastigote)
- Macrophage (Amastigote)
- Lieshman-Donovan bodies

Diagnosis

- 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
- Leishmanin test
- PCR-based methods are the most sensitive & specific diagnostic tests.



Management

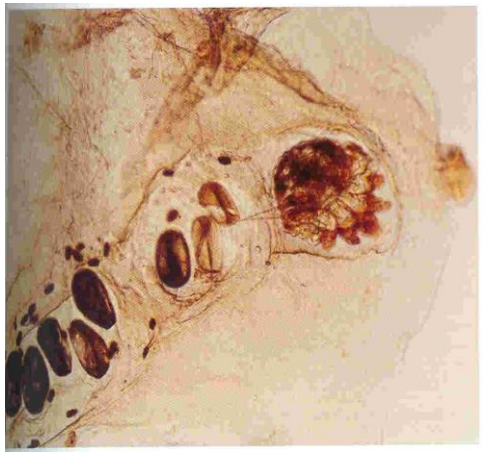
- Resolve spontaneously leaving a scar →so, we should to rx.
- Intralesional pentavalent antimony
- Parenteral pentavalent antimonials (Sodium stibogluconate) are the treatment of choice for cutaneous and mucocutaneous leishmaniasis.
- Liposomal amphotericin B for visceral leishmaniasis
- Topical Paromomycin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

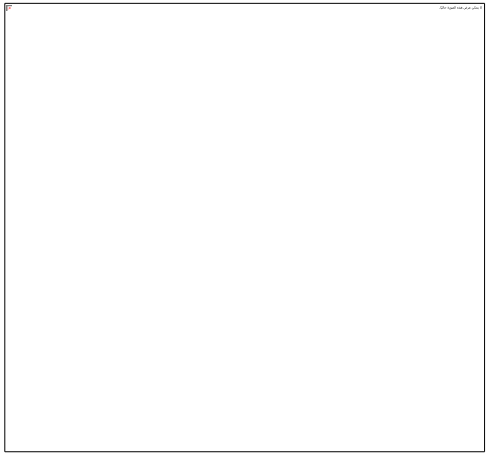
Infestation

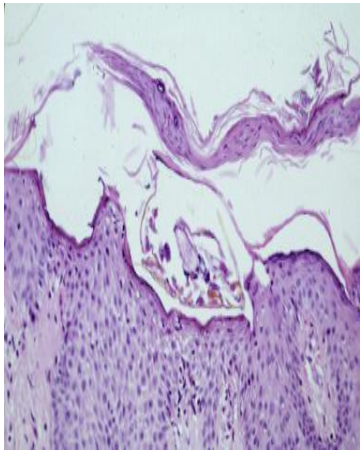
1-Scabies

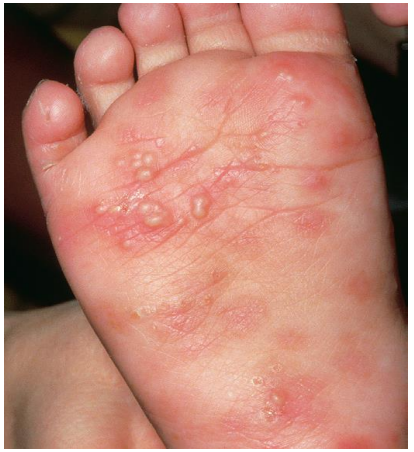
The female sarcoptes scabiei var hominis mite lays 60-90 eggs in her 30-day lifespan, although less than 10% of the eggs result in mature mites.

- Mite: *Sarcoptes scabiei var. hominis.*
- It residue in burrows in the stratum corneum laying eggs then dieying and the eggs will maturate 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 (foreign body like lesion in the epidermis) 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.









Scabies

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

- 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. Look behind ears.
- 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%.

Summary

- **Varicella zoster** is the initial infection by VZV present by diffuse vesicles or pustules, preceded by or complicated to pneumonia Rx symptomatically. **Where herpes zoster** is the recurrent infection present by vesicles and blisters on dermatomal distribution (mostly thoracic) Rx symptomatically or by oral or IV antiviral based on severity and immunity.

- **Viral infections can be diagnosed by** : tzanck smear , DFA , Viral culture.
- **Fungal infections can be diagnosed by** swab and KOH, fungal c/s, skin bipsy, and wood's lamp. **Rx** by topical antifungal creams or shampoos, or oral antifungal.
- **Candidiasis** can be physiologic, pathologic, or iatrogenic. Caused by candida albican.
- **pityriasis versicolor** by Malassezia furfur (hyphea) or Pityrosporum orbiculare (yeast) can be asymptomatic or as hypopigmented scaly patches.
- **Tinea pedis** → infect the sole of foot → can be by Erosive interdigitalis → most common, atheletes foot, Hyperkeratotic type caused by (T. rubrum), or Inflammatory type caused by (T.mentagrophyte).
- **Tinea ungum** mainly affect nails→ may present as White Onchomycosis. Oncholysis. Distal or proximal Sub-ungural hyperkeratosis. Thickening of nail plate.
- **Tinea manuum** may present with hyperkeratosis or inflammation with vesicle and pustules. diffuse dry scaling over the palm.
- **Tinea corporis** →2 types: Hyperkeratotic type (T. rubrum), and Inflammatory type (T.mentagrophyte).
- **Tinea capitis** →Black dot endothrix (T. tonsurans), Ectothrix (M. canis , M. audouinii), Kerion (T. verrucosum), Favus (T. schoenleinii) may causes scarring.
- **Tinea cruris** → affect genital skin.
- **Leshmaniasis**→ painless papule increase to ulcerated nodule or plaque, mainly on exposed areas by sand fly.
- **Scabies**→ by Sarcoptes scabiei var. hominis, present by severe itching, burrows (pathognomonic), +family hx. Affect finger webs, flexor of the wrist, axillae, areolae, umbilicus, lower abdomen and scrotum.
- **Pediculosis capitis**→ by pediculus humanus var capitis, common in school children, causes severe itching of scalp.

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