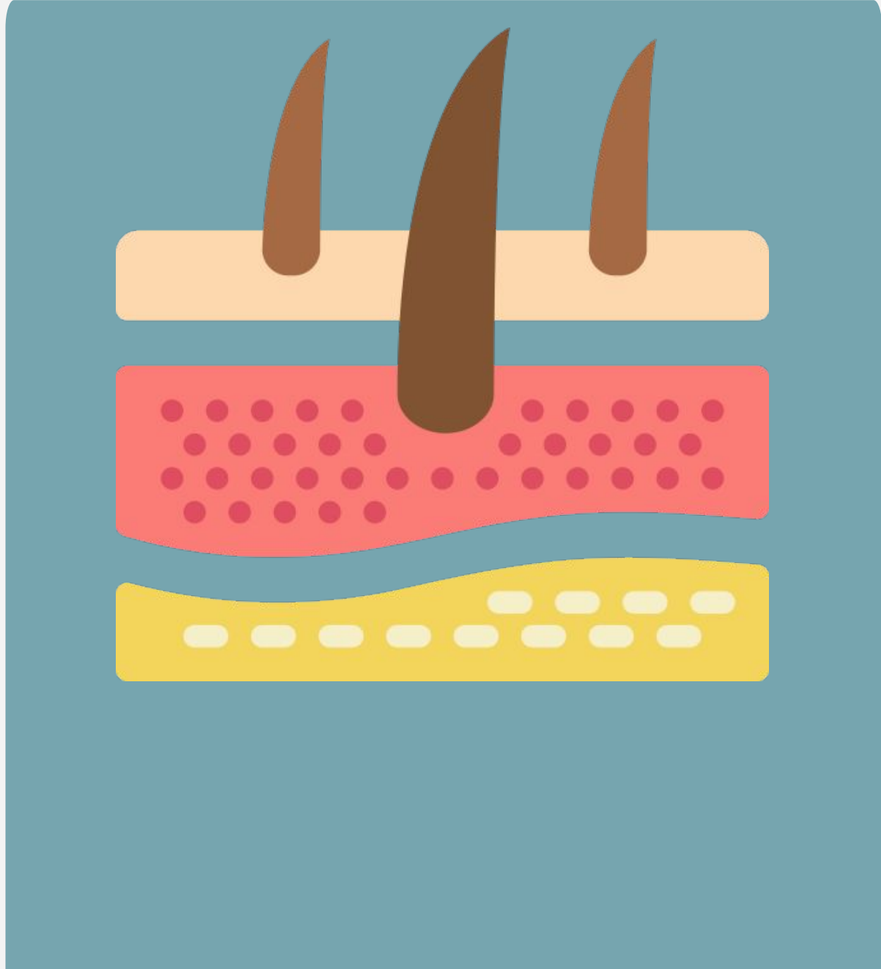


5- Skin infections

Objectives:
Not given

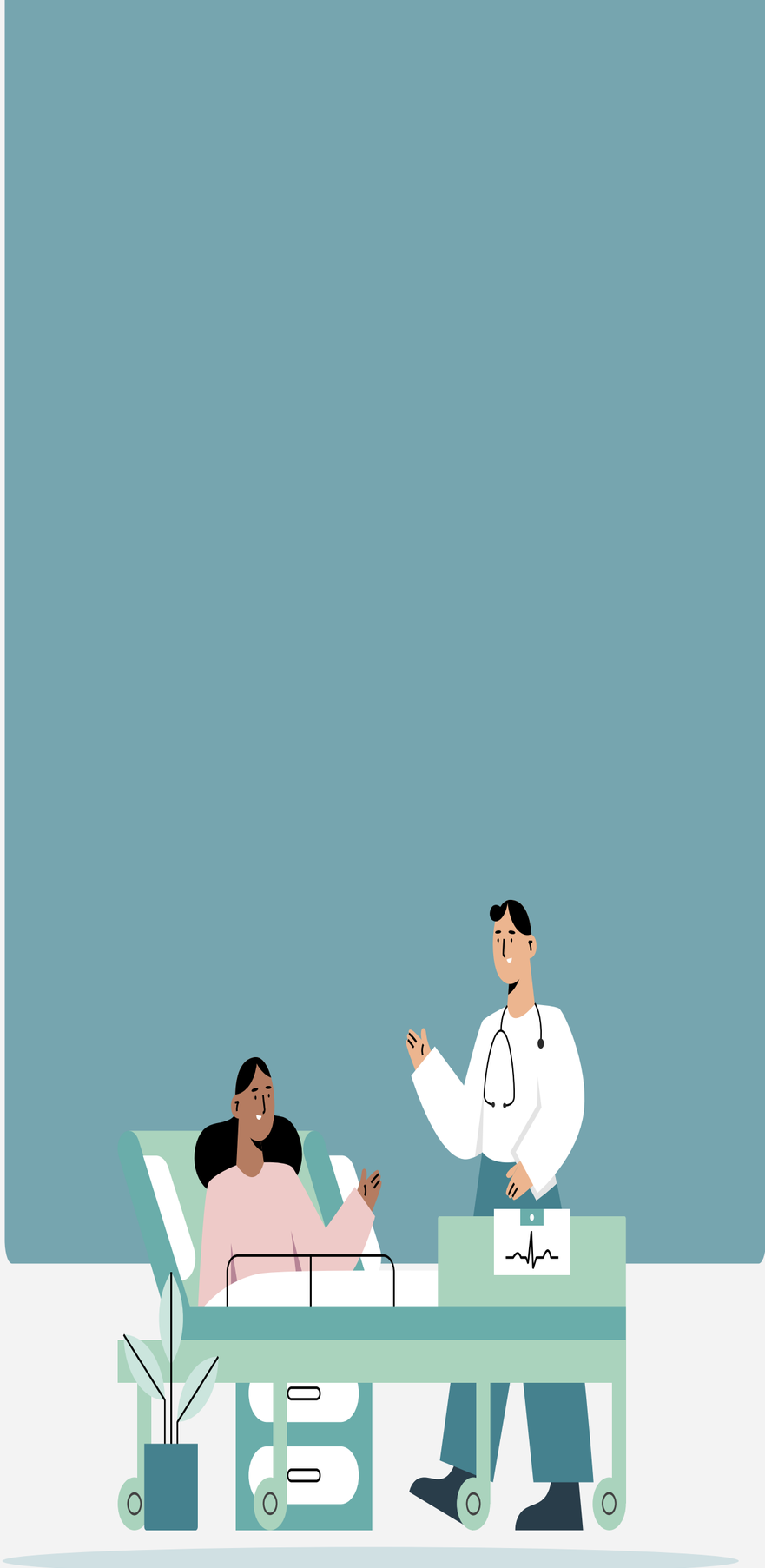
Done by:
Adnan almogbel, Maan Shukr, Rakan AlSalhi
AlAnoud AlMansour, AlHanouf AlJaloud, Noura AlBassam
Revised by: Sultan Al Nasser, Rotana Khateeb

References: Doctor slides, Team 436

Color Index:

- Important
- Doctor's Notes
- Extra

Editing File



Why does skin get infected?

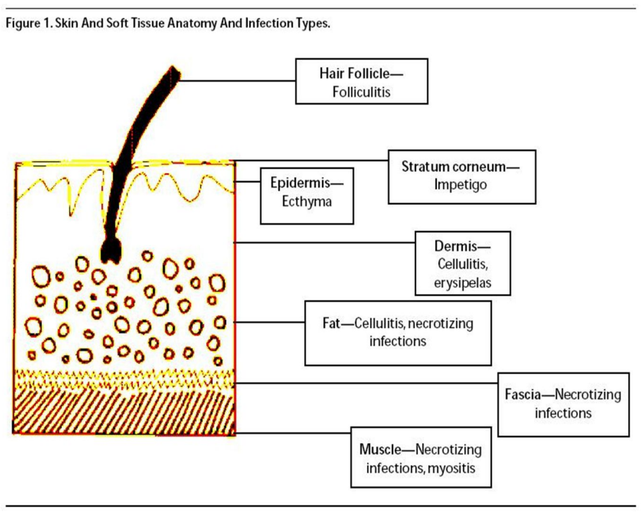
- There are multiple types of organisms which are normally present on the skin as normal flora such as: Staphylococcus epidermidis and yeasts.
- The presence of bacteria does not automatically lead to a skin infection.
- What is the difference between colonisation and infections?

  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.

Types of infections:

- BACTERIAL INFECTIONS: (commonest)
  - Impetigo, Erysipelas, Cellulitis, Furuncle, Carbuncle, Folliculitis , Ecthyma , Ecthyma gangrenosum , Erythrasma , SSSS
- VIRAL INFECTIONS:
  - Warts, Molluscum contagiosum, Herpes simplex, Varicella, Herpes zoster
- FUNGAL INFECTIONS:
  - Candida, Dermatophyte inf., Pityriasis versicolor
- Protozoal: Leishmaniasis
- INFESTATIONS:
  - Scabies, Pediculosis capitis

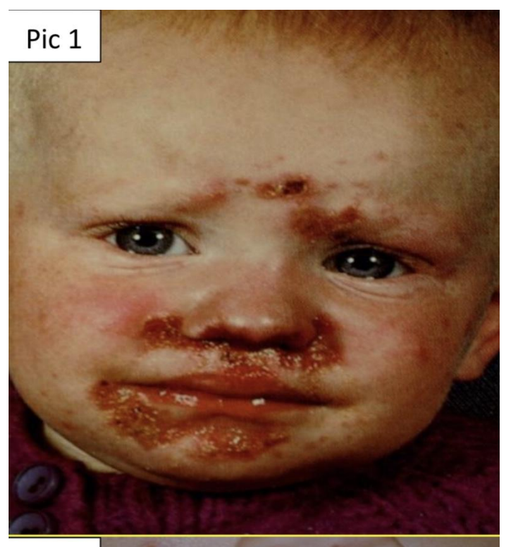


1- Bacterial infections:

Impetigo: (commonest skin infection)

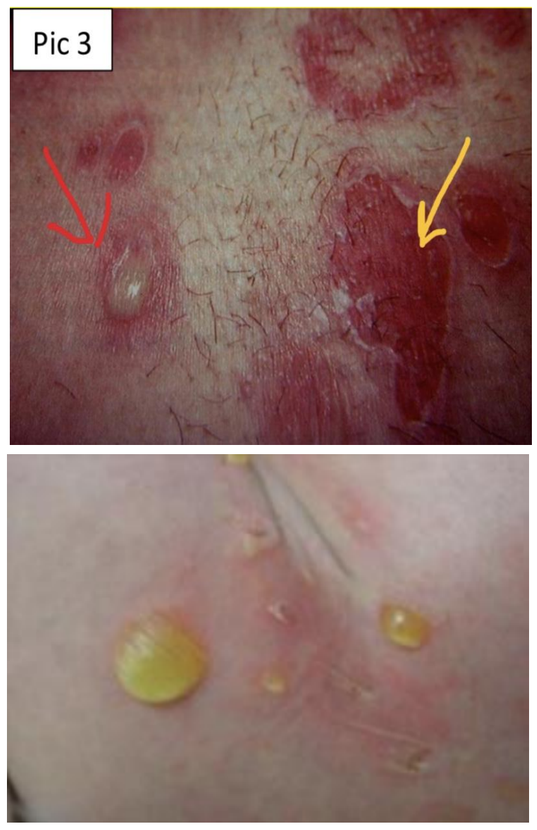
- Not serious in immune competent pt , tx by topical steroid.
- Acute superficial cutaneous Infection (limited to the stratum corneum)
- Very contagious, auto-inoculation is common.
- Can cause systemic symptoms (fever, lymphadenopathy)
- Children, Adult.
- It is mainly affect children
- The causative organism is usually Staphylococcus Aureus (>90% cases), but less often can be strept. Pyogenes (gp A beta-hemolytic streptococci).
- Most common organism: streptococcus, The most important complication caused by this bacteria is post-streptococcal glomerulonephritis
- 2nd most common: staphylococcus
- 2 forms (**bullous** 30%, **non-bullous** 70% commonest).
- **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 bullae or pustule))
  - Predisposing factors:
    - Warm, humid climate, poor hygiene, trauma, insect bites, immunosuppression. (anything that affect the skin's integrity like eczema)
  - **Pic1&2:** Perioral peri-nasal crusted honey colored papules

Pic 1



Pic 2

- **Bullous Impetigo**. (less common)
  - Due to staph aureus. Phage group 2 toxins (exotoxins) which cleave desmoglein-1
    - Desmoglein-1 is located in the upper part of the epidermis (spinous cell layer) in the desmosomes which connect the cells together, thus if there's any toxin breaking down the desmoglein, the desmosomes will break as well which will affect the intergrity of the skin leading to bullae formation
  - Newborn and in renal patients.
  - Face, hands, diaper area or any part.
  - Bullae **(flaccid)** on grossly normal skin. (we have 2 types of bullae, flaccid & tens)
  - **Pic 3:** Bullous, Erupted bullous.



- **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-stept.
  - (it is usually clinically diagnosed)
- **Treatment:**
  - Topical unless if the patient has:
    - Fever, constitutional symptoms, immunocompromised.
  - Then give oral antibiotics: amoxicillin, Cephalosporin
  - Wound care
  - Localized:
    - Topical Abx (Mupirocin)
    - compresses to loosen crusts.
    - Intranasal mupirocin for periodic decolonization in carriers: 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) such as: Penicillinase- resistant PCN, 1st/2nd generation cephalosporin, clindamycin,or erythromycin (esp, if penicillin allergic)

b. Cellulitis:

- **Adults: s. Aureus , children: h. Influenza**
- **Deeper involvement of the SC. It Can be superficial (in the dermis), or in the deep tissue (SC) .**
- Acute, raised, hot, tender, erythematous plaques (leg),
- **If superficial then it is called: erysipelas. Affect adult and children.**
- **Most common organism:** streptococcus**, 2nd:** staphylococcus.
- Preceded by abrasion, insect bite or trauma
- Cutaneous abrasion or ulcer.
- There should be a port of entry for the organism either 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

Treatment:

- Swab + blood cultures. The diagnosis is clinical.
- Semisynthetic penicillin group 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.
- **if you suspect MRSA (Methicillin Resistant Staph A):** clindamycin, vancomycin, doxycycline or 3rd generation cephalosporin**.**

Treatment always: oral antibiotics**.**

BUT if the patient:

- **has signs of septicemia.**
- **not improving after 24 hours of Oral antibiotics**

EMERGENCY with IV antibiotics

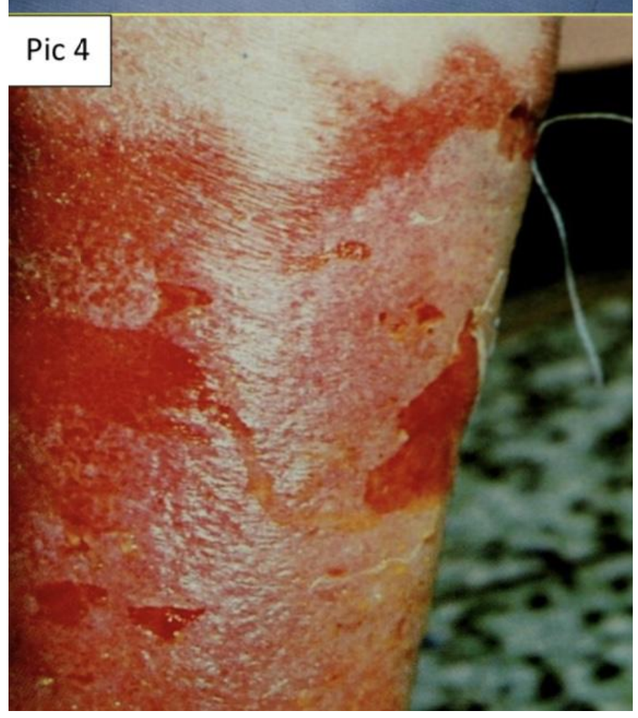
Ill defined erythematous plaque

Pic 1

Pic 2

Pic 3

Pic 4



Pic1: red and swollen, feel painful and warm
Pic2: More swollen edematous
With blisters
Pic3: Most common site for cellulitis is the leg because the leg is more prone to traumas.
Trauma > crack of skin > entry for bacteria,
Pic 4: More severe cellulitis erupted bullae
Dx clinically, biopsy will not see the organism most of the time, do blood culture for septicemia
Cellulitis can complicate tinea pedis or any skin infection, and it can lead to septicemia .

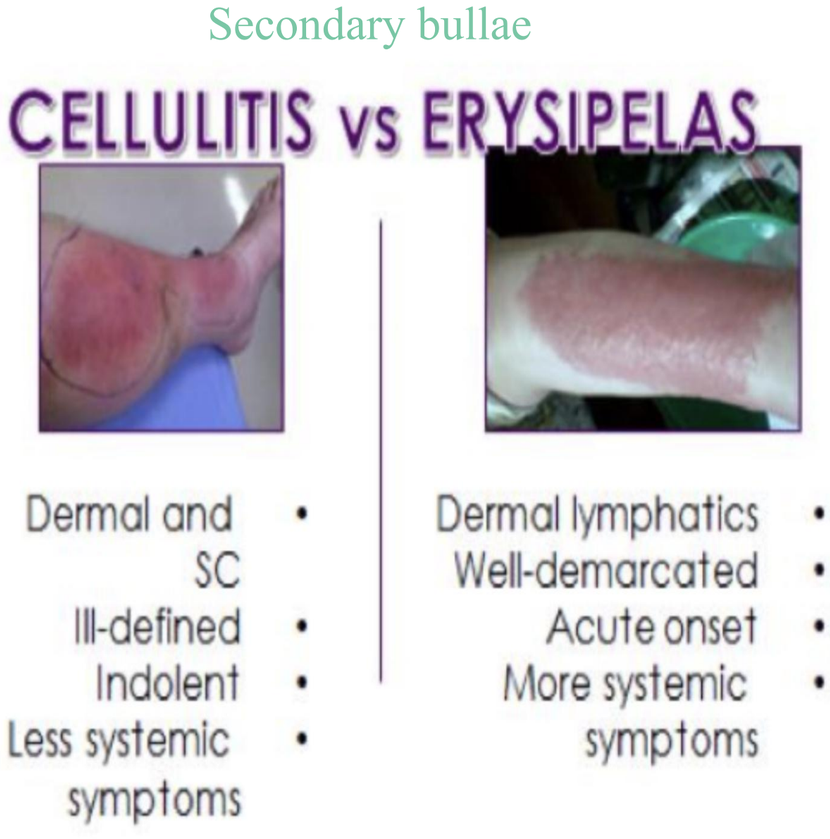
Secondary bullae

C. Erysipelas

- Superficial infection with marked lymphatics involvement.
- Sharply demarcated (distinguishing feature) unilateral, hot, red, tender & edematous plaques. They might have oozing or crust.
- Infants, young children, & elderly patients **(most commonly..)**
- Face, leg
- Beta hemolytic gp A Strept or staph.
- Preceded by abrasion, insect bite or trauma
- **Risk factor;** Minor abrasion/lymphatic dysfunction
- sup. Lymph vessels
- Might be associated with Leukocytosis & fever

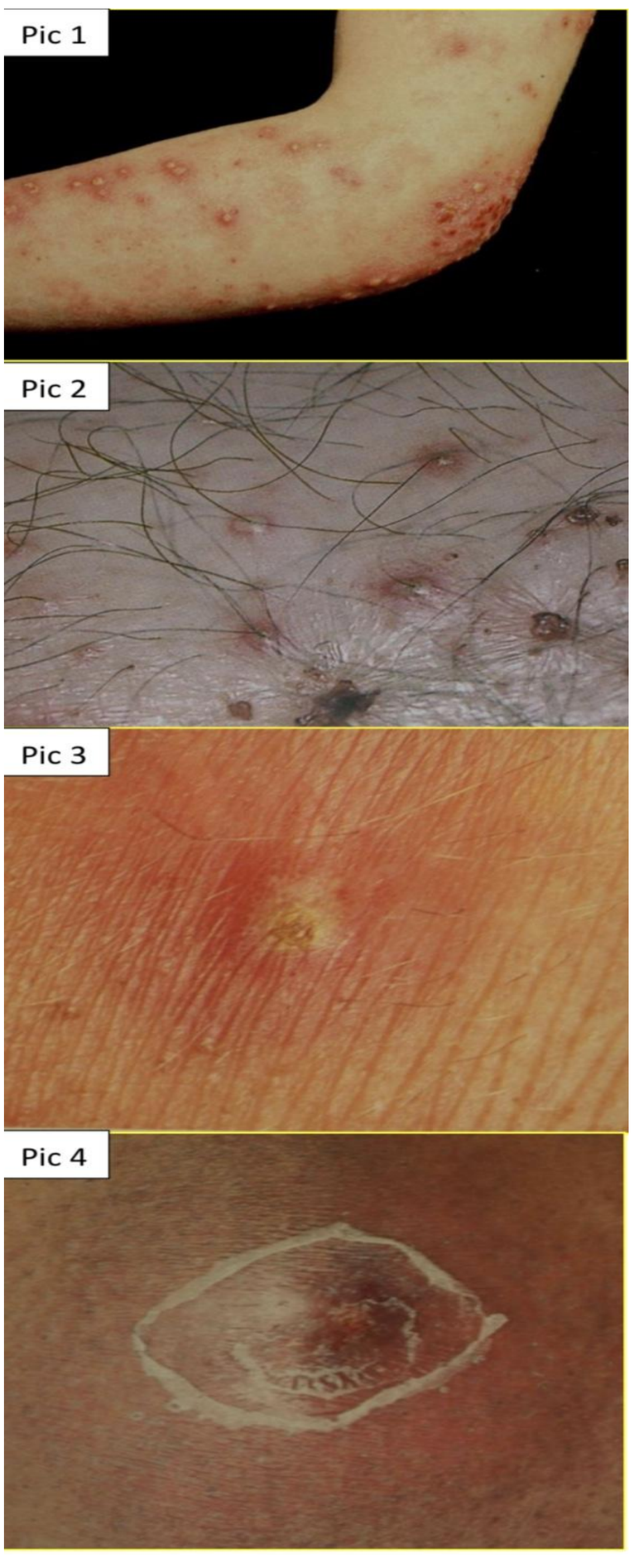
Management:

- 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.



D. Folliculitis:

- Seen after shaving
- Inflamation of hair follicles. (upper part of the hair follicle)
- **Most common organisms**: **staphylococcus** Most common is Staph Aureus.
- Presents as itchy or tender papules and pustules at the follicular openings.
- Superficial infection of follicle ostium
- 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 bc they developed resistance thus we'll give them 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 very itchy & mono-morphus.
  - Demodex (usually seen in association with rosacea over the face).
- **Divided into:**
- **Acute:** oral antibiotics resolve within 7-14 days
- **Chronic:** **different tx, common sites: nasopharynx, axilla, groin, scalp.**
  Patients are usually carriers of the bacteria in nasal, oropharyngeal, axilla or genital areas we give them oral Rifampicin (for 2-3 months) and clindamycin to clear the bacteria from their body + topical (topical especially in chronic) (need long term AB for months) .
- Complications include abscess formation and cavernous sinus thrombosis, carbuncles or furuncles if upper lip, nose or eye affected.



Pic1,2: Pustules in hair follicles: folliculitis.
Pic 3: Furuncle: deeper and wide infection in one hair follicle.
Pic 4: Carbuncle: deep and wide folliculitis in more than one hair follicle.

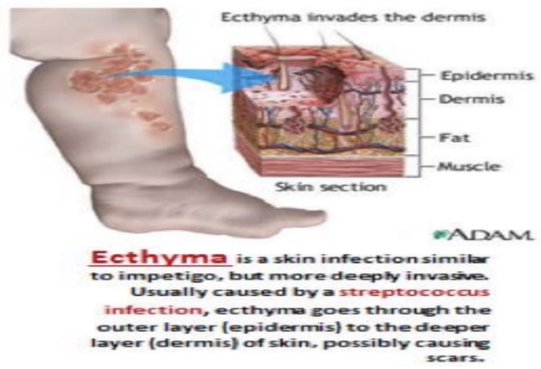
- Topical antiseptics such as Chlorhexidine.
- Topical antibiotics, such as Fusidic acid, Mupirocin or clindamycin.
- More resistant cases may need oral antibiotics (similar to impetigo).
- Hot tub folliculitis (P. aeruginosa)– usually self limited (ciprofloxacin is used in persistent severe cases).
- Gram negative – trimethoprim , isotretinoin

Tx : systemic ab that cover s. Aureus or other gram + bacteria .
1- amoxicillin 2- doxycycline (commonly used) 3- cephalosporins (3rd generation) 4- vancomycin (given iv) 5- clindamycin .
4-5 given in more severe cases
-if there is pus drain it before then give abx .

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.
Can occur in both immunocompromised & immunocompetent pts
Mostly caused by streptococcus & may heal with scarring.

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

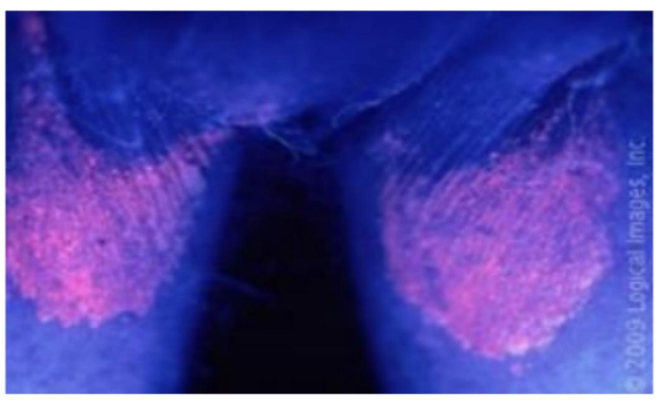
ICU-setting, immunocompromised patients with pseudomonas sepsis & as a consequence they develop these ulcerations usually with black eschar (black necrosis in the center).
Ask them to do blood culture to rule out pseudomonal

E. Erythrasma

- Colonisation of axilla or groin (flexures) with Corynebacterium Minutissimum.
- Gram positive bacilli (not caused by staph or sterpt)
- Red, brown
- Present with minimally 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).



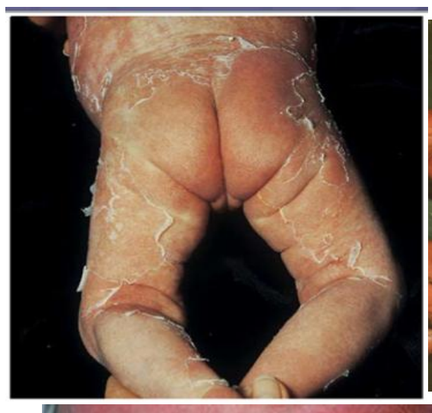
F. Staph Skin Scalded Syndrome (SSSS):

Generalized skin infection, very serious , happen in children .

Widespread infection of staphylococcus, we need immediate I.V antibiotics to avoid septicemia

important because a dr might see it in the ER and think its just an Eczema may need referral to burn center or ICU.

- A superficial blistering condition caused by exfoliative toxins of certain strains of Staph Aureus (which break desmoglein-1 just like what happens in impetigo)
- 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; it happens due to toxin breaking down the keratinocytes), 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 for wound care or ICU
- we should admit the pt and take blood culture and start him on anti staph ab (mentioned above) like vancomycin and clindamycin (depend on the case but these two has less resistant than others) .



Generalized erythroderma with scales .

2- Viral infections:

a. Warts:

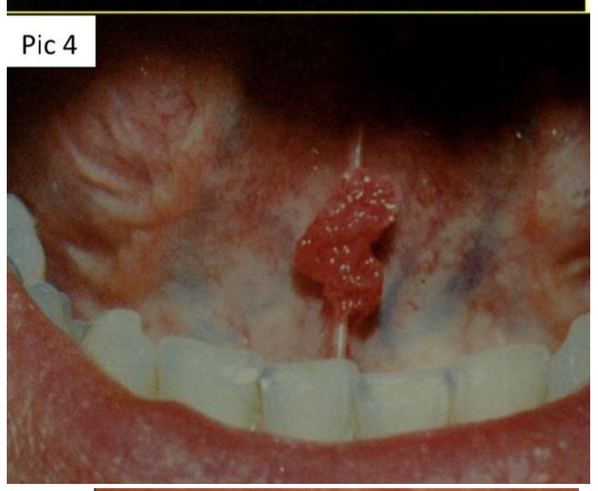
- Caused by Human papillomavirus HPV (DNA virus)
- More than 100 subtypes of HPV have been identified with different epithelial preferences (skin vs mucosa) and different clinical patterns
- **Common warts (verruca vulgaris):**
  - Caused by HPV 1, 2,3,4,7 and 54.
  - Affects the hands, could be peringuinal.
  - Common in children, presents as hyperkeratotic (verrucous) papules.
  - Koebner phenomenon d.t autoinoculation.
- **Pic 1,2,3**: Hyperkeratotic verrucous papules.
- **Plane warts (Verruca plana):**
  - Affects face, back of hands caused by HPV 3,10 and 28
  - Flat skin colored papules
  - **Pic 4**: plane wart caused by HPV type 1 mainly
  - **Pic 5,6**: Plane warts caused by HPV 1 and 5(Affects Face, back of hands) (salmon like).
    - Pic (6) : multiple well-defined skin color papule, no scaling
- **Plantar warts (Verruca plantaris):**
  - Affects the soles caused by HpV 1,2,4,60 and 63
  - To be differentiated from plantar corn
  - **Pic 7:** Plantar wart. (affect sole) calcity caused by many things but one of the ddx is hpv.
    - MCQ : patient has calcity and when we do shaving we find pinpoint bleeding, answer is most likely warts.
- **Management:**
  - Involute spontaneously
  - Cryotherapy (using liquid nitrogen)
  - Topical keratolytics : Salicylic acid , TCA
  - Electrocautery, curettage, Laser
  - Topical retinoids in flat warts
  - Others : bleomycin , cantharidin, PPD , Candida antigen to stimulate the immune response

Pic 1

Pic 2

Pic 3

Pic 4



Pic 5

Pic 6

Pic 7

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 trials[67].

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)

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	

Multiple skin colored papules with hyperkeratotic or verrucous surface.

Skin colored flat surface papules

Skin colored papules over the sole of the foot

Pin-point hemorrhage indicates presence of wart

In genital warts its painful to use the previous treatments so Imiquimod (cream) is the treatment of choice.

- **Genital wart (Condylomata Acuminata):**
  - Present with multiple verrous Cauliflower like plaques & nodules over the vulvar skin, mucous membrane and perianal area.
  - Most common STD
  - Caused by HPV type 6,11, 16 and 18
  - Sexual Partner
  - Child ? Sexual abuse
  - Oncogenic strains :16, 18
    - Cervical squamous cell carcinoma
    - HPV 16,18 (31 , 33, 51 ,52) are the most aggressive to causing genital cancer
  - Vaccination
    - There is vaccine for people when they reach 2 years , polyviralent vaccine.
    - Vaccine (GARDASIL-9) provides immunity against 9 HPV type: 6,11,16,18,31,33,45,52,58.
  - Diagnosis of the cancer by skin biopsy
  - To know what subtype of Human Papilloma virus is by PCR
  - Once child comes wth giniral waerts think about sexual abuse.
  - **Pic 8**: **Condyloma acuminate:** (most common is 6-11 hpv) it's a type of genital wart, commonly sexually transmitted **description**: Multiple Soft convoluted papules.
    - Pic (8) : multiple skin colored plaques and papules in genital area,
  - What is the next step after encountering a patient with genital wart?
    - Screen for other STDs, Screen the partner, Pap smear.
  - **Pic 9,10,11**:
  -

- **Mucosal Warts**

b.Molluscum contagiosum

(Poxvirus) (DsDNA): Dome shaped shiny yellowish- whitish papule with central umbilication (central punctum mostly with children rarely with adults,

in adults: immunocompromised or as sexually transmitted disease(**pic11**).

In genital warts in Children it can be cause by Autoinoculation or abuse.

Face, neck

- Koebner phenomenon d.t autoinoculation when a person transfers a disease from one part of their body to another

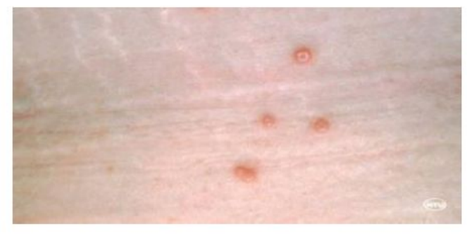
H/P: Hunderson-patterson bodies

Management:

Involute spontaneously

curettage, cryotherapy

Other : Salicylic acid



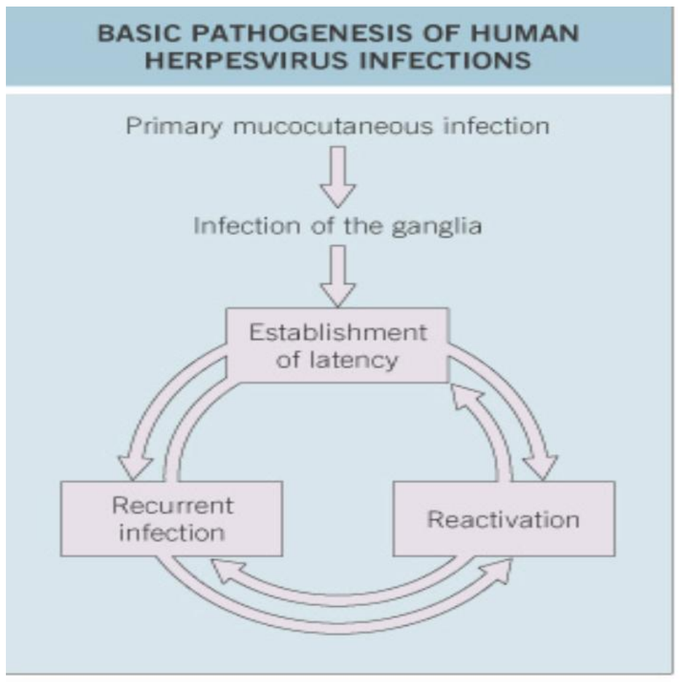
© 2003 Elsevier - Bologna, Jorizzo and Rapini: Dermatology - www.dermtext.com

Raised papule <1 cm with central umbilication (characteristic)

c. Herpes Viruses:

Group of small blisters.

- HSV-1 (H. labialis)
- HSV- 2 (genital herpes)
- Herpetic whitlow
- Eczema herpectium: Infection with HSV in patients with previous skin
- disease (eg: atopic dermatitis, pemphigus, Darrier disaese)



1- HSV1:

- mostly affects Orolabial mucosa.
- 80% of general population aged 18 had hsv1 whether it was clinical or subclinical. But if you test them with IGG for HSV will be positive
- Hsv1 is normally self-limiting, but sometimes it causes Primary severe Gingivostomatitis and you should give systemic antiviral.
- Stay latent at trigeminal ganglia

Pic 1,2: Multiple grouped erythematous vesicles

Pic 3,4: **Eczema Herpeticum**: IMPORTANT

is a serious complication that needs admission and systemic antiviral.

These patients had eczema (or any condition break the integrity of skin) and herpes at the same time, the herpes autoinoculated the area of the eczema, so any patient with active eczema and herpes should be given systemic anti-viral to prevent **Eczema Herpeticum**.

2- HSV2:

- mostly affects genital.
- pregnant lady with HSV we better treat her .
- Sexually transmitted.
- patients need counseling to teach them not to spread the virus.
- You should treat the partner.
- Stay latent at sacral ganglia

Any patient treated for acne by laser, we must give them one of the acyclovir drugs.

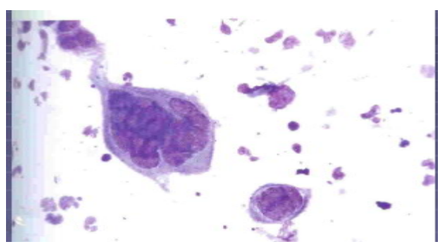
Treatment:

Oral (immunocompetent) / IV (immunocompromised) acyclovir for Genital/ Recurrent/ neonatal /immunosupressed , eczema herpeticum - **If recurrent more than 6 times a year:** the patient should be given suppressive systemic Acyclovir for 6 months to one year.

Herpetic whitlow

Diagnosis:

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



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 within **multiple vesicle on erythematous base (dew drops) قطرات المطر**
- 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.
- **Self limited in Children.**
- **More aggressive in adult.**
- Adults as a primary disease: immunosuppression, pneumonia, individuals never infected during childhood,
- **Complication of varicella in adult:**
  - **Varicella pneumonia**
  - **Varicella encephalitis**
- Diagnosis: tzanck smear ;doesn't differentiate between varicella & HSV), DFA, Viral culture.
- Treatment: symptomatic for itching, Systemic antiviral in immunocompromised patients (2x the dose of HSV 1) to aviod the complications..
- 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

Describe the lesion? Multiple vesicles on erythematous base (new drops) قطرات المطر

Herpes zoster (shingles)

- Following initial varicella infection VZV remain latent in sensory ganglia when reactivated it will affect skin dermatome supplied by this ganglia.
- Usually come to people 50 Yo and older
- It is a disease of elderly and immunocompromised.
- Prodromal pain.
- dermatomal (grouped blisters and vesicles on background of erythema)
- post-herpetic neuralgia (pain in dermatomal distribution) need painkiller with antiviral as early as possible.
- Diagnosis:
  1.Clinical diagnosis
  2.Tzanck Smear.
  3.Direct fluorescent antibody (DFA).
  4.Viral culture
- Treatment:
  1.Analgesia.
  2.ANTIVIRAL (within 72 hours of skin eruption).
  We give it to minimize the disease and may help to decrease **post herpetic neuralgia** Oral for immune-competent. Admission & IV acyclovir for immunocompromised & patients who develop ophthalmic zoster or Ramsay hunt syndrome

Ophthalmic zoster ;
trigemnial nerve. (opthalmic branch of trigeminal V1)
Admit and IV Acyclovir

When tip of the nose and outer ear canal = Ramsey Hunt Syndrome
IV acyclovir and Admt

3- Fungal infections

Candida infection (intertrigo)

- Candidiasis is a normal flora in the skin especially in the moist, humid & flexural areas.
- Physiological (old age, neonate and pregnancy).
- **Pathological; Higher risk in (DM, HIV and organ transplant, on immunosuppression, obese and bedridden patient).**
- Iatrogenic (long course of Antibiotics).

Candida albican (normal commensal of GIT).

• Napkin candidiasis & Intertrigo (satellite lesions: pastulus that are away from the main lesion). (in intertigo it affects the body folds (flexures)

•Paronychia. (infection of the nail fold)

•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, ketoconazole, miconazole, lamazine): immune suppressed, persistent infection.+ steroid if they have eczema

Well defined erythematous scaly eroded patches with stellate lesions (very classic for candida infection) .

Pityriasis versicolor

- Most common fungal infection in adults and it is chronic affecting the truncal area
- 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).

- Comes during the summer usually after sweating and exercise

-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: Dx is clinical

-Wood's lamp: coppery-orange fluorescence).

-Scraping for KOH and fungal c/s.

- Skin biopsy for PAS stain.

- Treatment:
  Topical antifungal (eg.ketoconazole shampoo), imidazole (nizoral) creams or shampoo. From the neck to the waist for 10 mins then washed away.
  - Oral Antifungal (itraconazoles).
  - Recurrence.

Present with macerated red lacerated erythematous plaques with satellite pustules

Candida Intertrigo

Napkin Candidosis

Cheilitis

Another form of candidiasis that is common in bed ridden patients

Oral thrush

Onychomycosis

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:

- Anthopophilic: 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 or 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

Dermatophyte infection:

- skin
- Hair
- Nails

Tinea pedis (athlete feet)

- Erosive Interdigitalis (Web space) fungal infection caused by superficial dermatophytes, It affects mainly adults, (patients with moist skin in between the toes like athletes bc they wear tight shoes most the time making it a good environment for the dermatophytes to grow and diabetics are at higher risk).
- Hyperkeratotic type (T. rubrum) affecting the sole.
- Inflammatory type (T.mentagrophyte) forming blisters on the sole of the foot.
- treatment:
  1-Education to dry up the skin.
  2-Topical antifungal.
  3-In severe cases systemic antifungal .
- Come to adults usually

Can be complicated by cellulitis .


Pic 3

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

Present with fissuring, vesicles and erythematous scaly plaques over the sole of the foot (inflammatory type)

Tinea corporis

- caused by superficial dermatophytes.
- Comes from pets usually .
- Two 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
- If it is limited (few plaques), it can be treated topically. If extensive it needs systemic anti-fungal. (mainly systemic) .

Describe lesions on left?:scaly annular lesions Resemble psoriasis

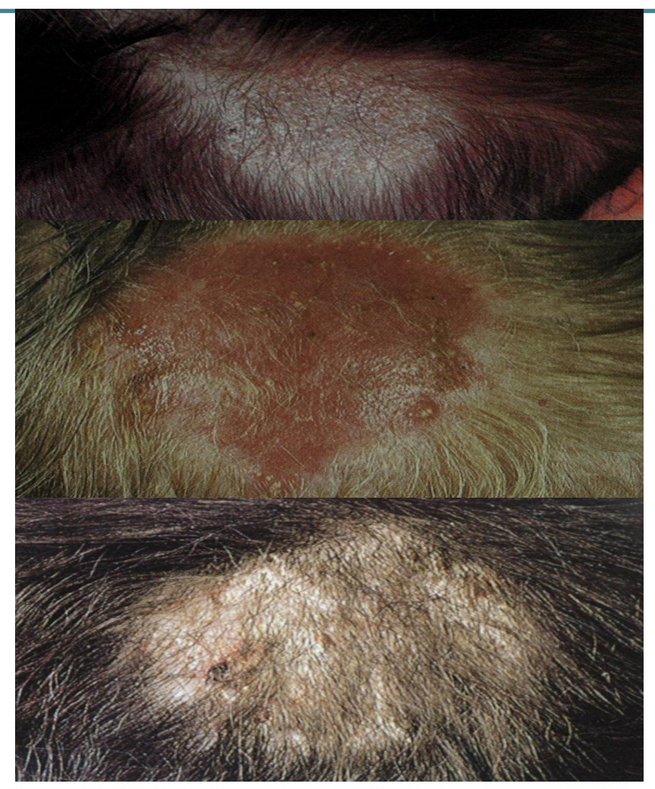

Pic 8

12 years old boy with 2 weeks history of very itchy skin lesions? Describe the lesion: multiple annular (ring like with clear centers) erythematous scaly plaque affecting his trunk

Tinea capitis

- Well circumscribed pruritic (itchy) scaling area of hair loss, mostly affects children.
- Comes from pets .
- 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).
- Treatment: systemic antifungal mainly: Griseofulvin, then Terbinafine, itraconazole
- Erythematous and inflammation that how we differentiate between it and alopecia areata .

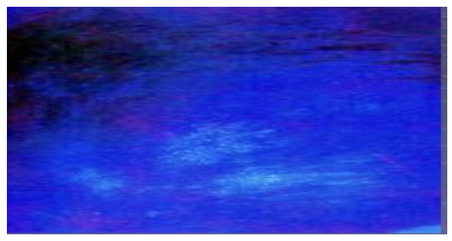
Describe lesions on right: well defined erythematous scaly alopecic patches



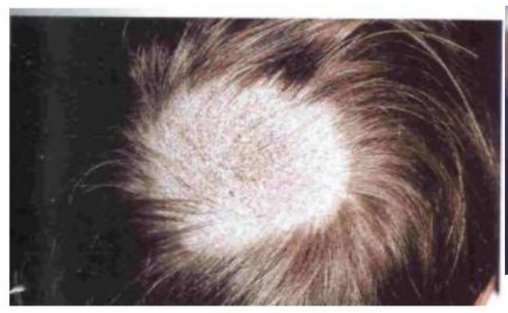
Tinea Capitis – commonest in children.
Presents as non-itchy patches of hair loss with broken hairs.

kerion

Investigation :
Scraping, hair plug, nail clippings---KOH and culture Wood's light
Rx: Topical (terbinafine, daktarin)
Oral (Griseofulvin, terbinafine, itraconazole): in extensive cases ,
Hair, nail involvement



Inflammation with crust



Endothrix

In left, Favus characterized by large yellow crust

Ectothrix

Kerion

Deeper inflammation. If left will end up with scarring alopecia. So you need to start antifungal ASAP to avoid it

Tinea ungum (onychomycosis)

Fungal infection of the nail plate
Different presentations including:

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

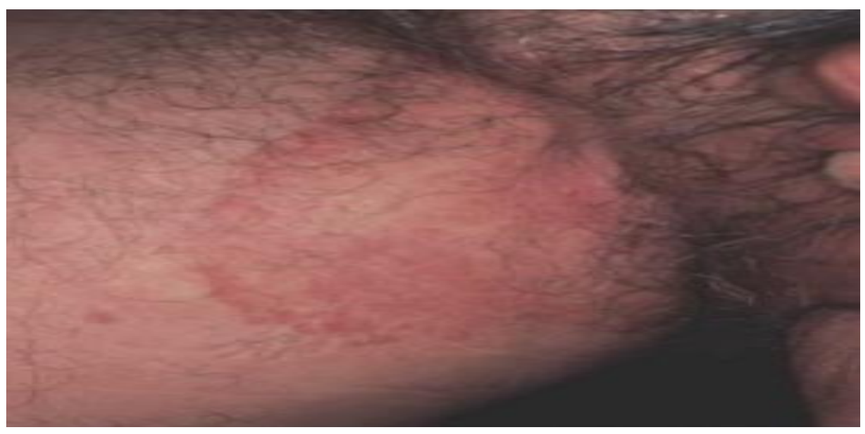
How to differentiate from psoriasis?
Psoriasis is a systemic disease affecting all nails (finger & toenails) while fungal infection usually affects only few nails (skip some nails)
How to confirm the diagnosis: Send nail clipping for KOH & fungal culture. PAS stain. Treatment: oral antifungal.

Tinea manuum

Diffuse dry scaling over the palm & itching.
You need to take scrapping and send it for KOH & fungal culture to confirm the diagnosis and differentiate it from eczema of the hand

Tinea cruris

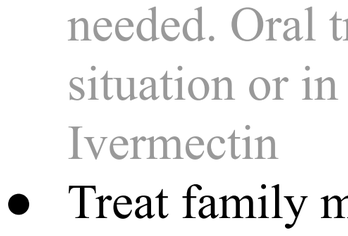
It affects the flexures (the axilla & groin).
Annular and scaly erythematous plaques.

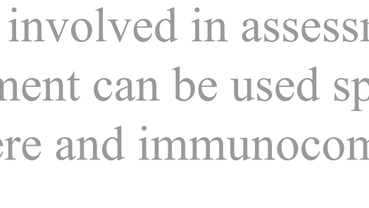


4- Infestations

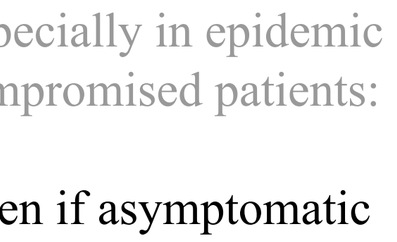
Scabies

- Causative organism: **female Sarcoptes scabiei var. Homini** lays 60-90 eggs in her 30-day lifespan, although less than 10% of the eggs result in mature mites.
- It residue in burrows in the stratum corneum laying eggs then diying and the eggs will maturate in 2 weeks period and the cycle repeated.
- Severe itching worse after bathing and at night.
- Usually in groins / body folds / around nipples /genitalia
- 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 (within the stratum corneum)
- 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.
- affects all age group and became more common in crowded close lived people like in shelters, homeless, prisoners, etc.
- 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:
  1- India ink or gentian violet then removed by alcohol to identify the burrows.
  2- A drop of mineral oil on the lesion then scraped away with a surgical blade
  3- Demonstration of the mite under the microscope.
- Treatment: Topical: Permethrin / lindane cream, and sulfate they apply it on the whole body for 8 hours. They need to apply it again after one week and one more time after two weeks, 3 in total to kill the newly hatched eggs Close contacts should be involved in assessment and treatment if needed. Oral treatment can be used specially in epidemic situation or in severe and immunocompromised patients: Ivermectin
- Treat 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)
- Oral ivermectin



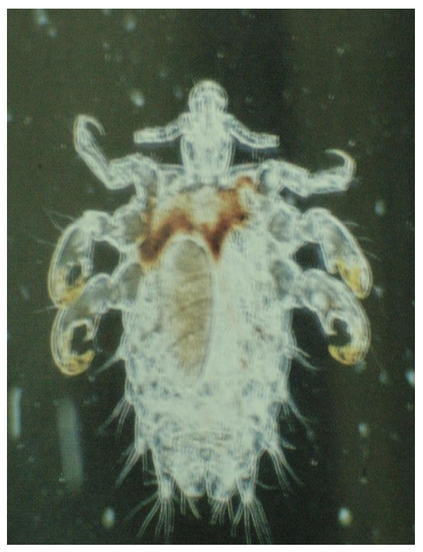


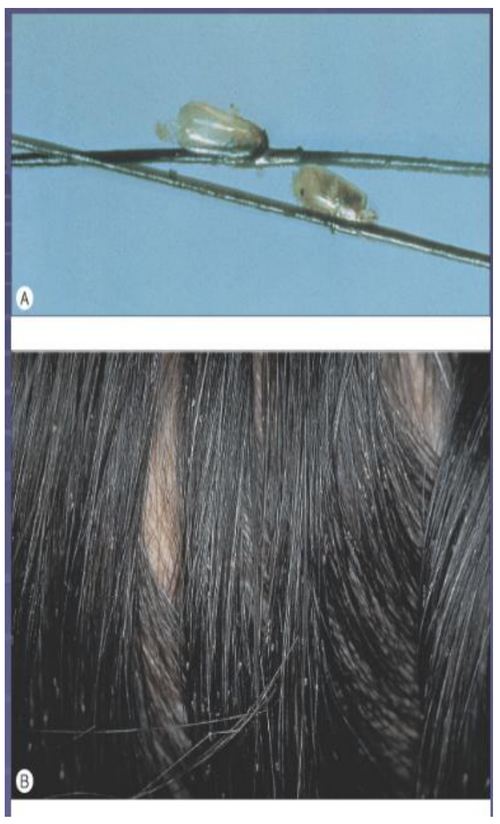
Tennels (burros) inside epidermis



Pediculosis capitis

- 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
- Affect scalp or and sometimes groin.
- Usually in children and homeless people Also, close live people might be affected Can cause Superficial bacterial infection
- Severe itching of the scalp
- Posterior cervical LN.
- Secondary bacterial impetigo.
- Treatment: same as scabies but in shampoo formulation to apply it over the scalp for 15 minutes the rains it with water, this can be repeated in 3 consecutive days to make sure for kill all insects. Close contacts should be involved in assessment and treatment if needed
- 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%.
- Shaving is the best treatment.
- More in female





Leishmaniasis

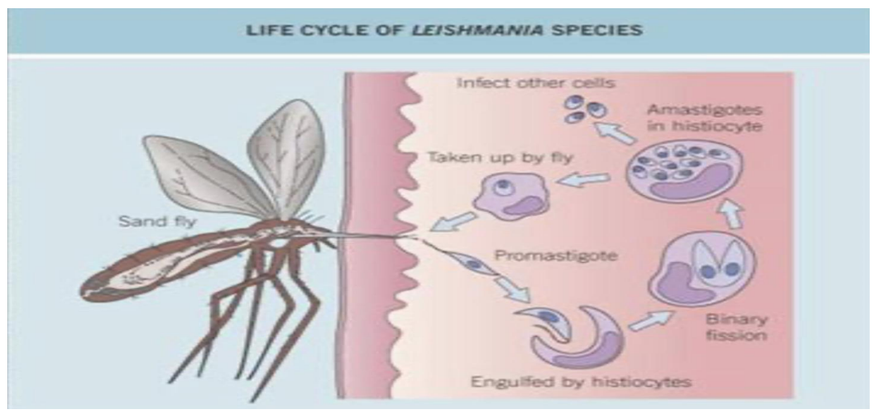
- **Includes a spectrum of chronic infections in humans and several animal species.**
- **There are three major clinical patterns:**
  (1) Cutaneous (localised or diffuse): the common pattern in KSA.
  (2) Mucocutaneous, which affects both the skin and mucosal surfaces. Cartilage -> saddle nose.
  (3) 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.**

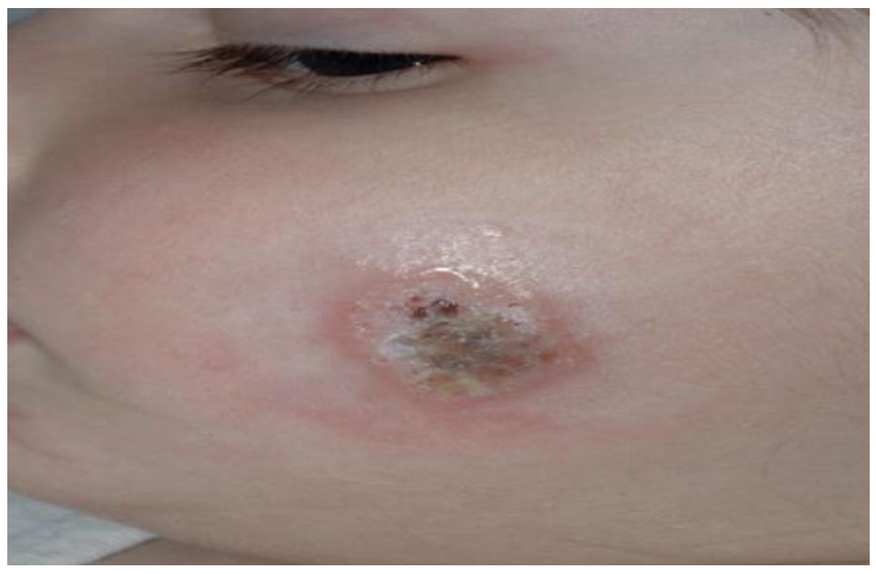
Dx:

1- Confirmed by demonstrating the presence of amastigotes in dermal macrophages within skin biopsy specimens, tissue impression smears (touch preparations), and smears of dermal scrapings.
2- Giemsa stain.
3- 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.
4- Leishmanin test (rarely done).
5- PCR-based methods are the most sensitive & specific diagnostic tests.

- **Management:**
  1- Resolve spontaneously leaving a scar thus need treatment.
  2- Intralesional pentavalent antimony.
  3-Parenteral pentavalent antimonials (Sodium stibogluconate) are the treatment of choice for cutaneous and mucocutaneous leishmaniasis.
  4- Liposomal amphotericin B for visceral leishmaniasis.
  5- Topical Paromomycin sulfate.
  6- Fluconazole or itraconazole.
  7- Cryotherapy.
- **Complication: disfiguring scarring.**

Mucocutaneous with saddle nose deformity and erosion





Questions

1-What is common between scabies and dermatitis:
A. Pruritus
B. Xerosis
C. Inflammation
D. Infection

Answer: A

2- 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

3- 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

4- Woods lamp is helpful in diagnosing which one of the following?
A. Lichen planus.
B. Tinea capitis.
C. Atopic dermatitis.
D. Psoriasis.

Answer: B

5- Under Wood's lamp, what is the color of "Tinea Versicolor"?
A. Red
B. Blue
C. Yellow green
D. Milky white

Answer: C

6- 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)

7- 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

8- Post herpes zoster neuralgia is associated with?

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

Answer: C

9- 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

10- 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

11: 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