Common Cutaneous Infections And Infestations PART1

Objectives :

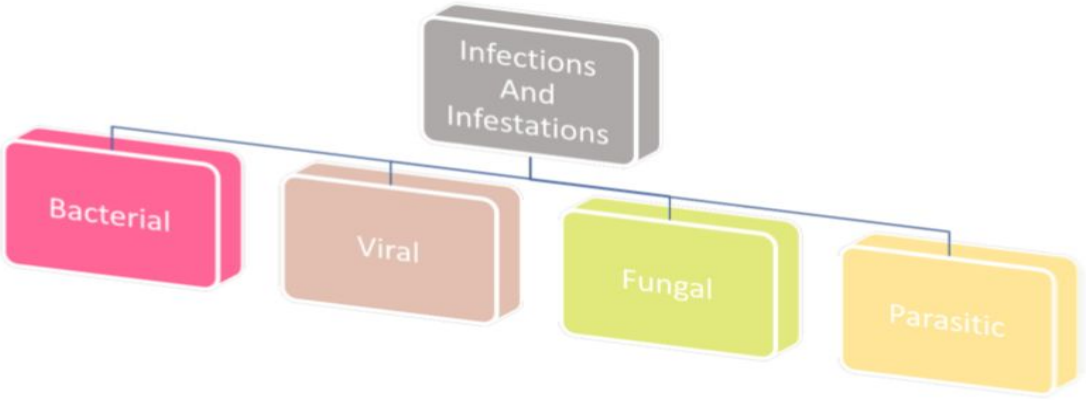
- To be familiar with common bacterial, viral, mycobacterial, fungal,and parasitic infections of the skin.
- To be familiar with presentation and management of common skin infections.
- To be familiar with skin infestation, their clinical features, prevention and treatment.
- The main reference is the TEXTBOOK..

Done by: Qusay Ajlan
Revised by:

***Sources**: FITZPATRICK color atlas +433 male + 434 + doctors slides and notes*

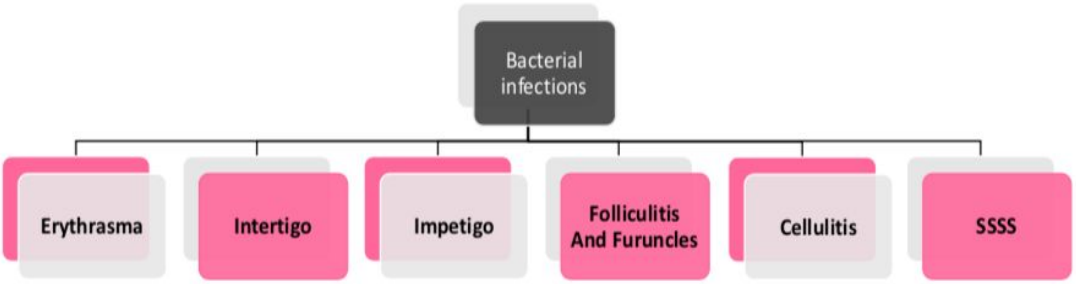
[Color index : Important | Notes | Extra]

- **This lecture consists of 100 slides**
- **The slides were filled with information which we think it is more than enough**
- **Adding any more (extra informations) would put extra strain on student**
- **And the doctor typically didn't add anything and just kept reading the slides**
- **All we can do as a team is to TRY AND SIMPLIFY THE LECTURE AS MUCH AS POSSIBLE**
- **Hopefully this will BE helpful**



Introduction

•Human body contains > 10 times more microbial cells than human cells.
•Microbial colonization of skin is more dense in humid intertriginous areas.(AXILLA)
•An intact stratum corneum is the first barrier against pathogenic invasion.
•Most common bacterial infections are caused by **staph. Aureus an streptococcus group A**



SYPHILIS + others (infections caused by mycobacterium)
1-leprosy 2-cutaneous tuberculosis

BACTERIAL INFECTIONS**

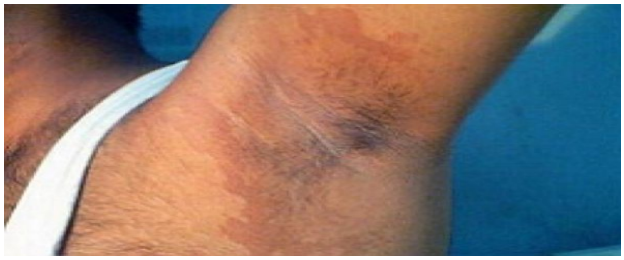
1-Erythrasma

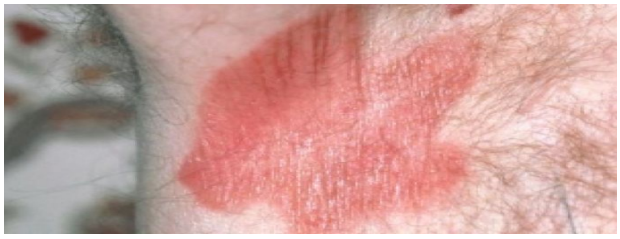
Definition	**sharply marginated brown patches** in intertriginous (an **intertriginous** area is where two skin areas may touch or rub together) skin like axillae, groins, toe web spaces.
Etiology	Corynebacterium minutissimum, gram-positive bacillus
Investigations	**1-Woods lamp: shows coral red fluorescence.** 2-KOH to exclude dermatophytosis
Clinical Presentation	•Mainly in intertriginous areas like axilla, groin •**Web space is the most common site.** ما بين الأصابع
DDx	inverse psoriasis, tinea, Hailey-Hailey disease epidermal dermatophytosis.
Treatment	**benzoyl peroxide,** clindamycin and erythromycin lotion.

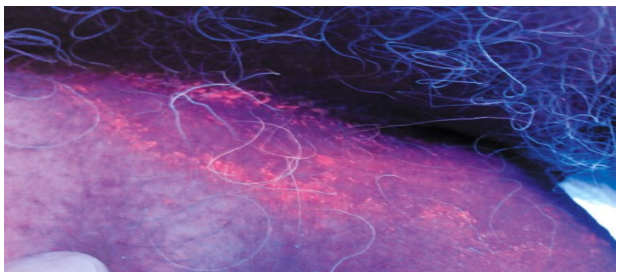
2-Intertrigo

DEFINITION	**Inflammation** of body folds.
ETIOLOGY	could be caused by staph aureus, streptococcus group A,B,G, or pseudomonas aeruginosa.
INVESTIGATION	Identify pathogen by bacterial culture, Wood's lamp examination, or KOH preparation.
CLINICAL PRESENTATION	May represent inflammatory dermatosis or superficial colonization or infection.
DDx	inverse psoriasis, Erythrasma, Hailey-Hailey disease, tinea, Langerhans Histiocytosis.
TREATMENT	**1-treat the underlying cause.** 2-Topical mild corticosteroids 3-topical antibacterial or antifungal.

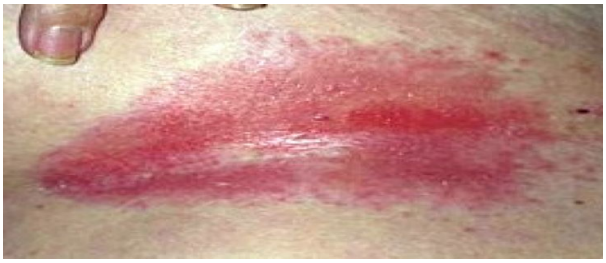
Erythrasma (patches)

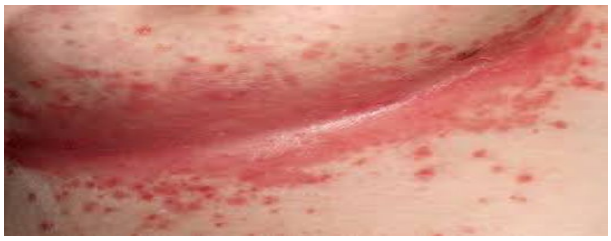


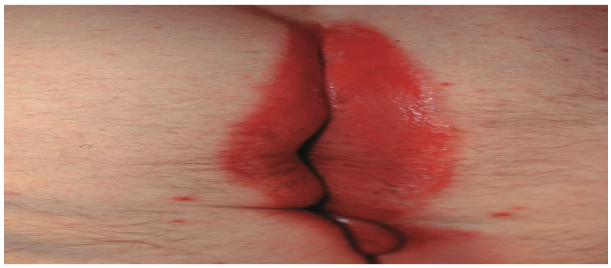




intertrigo (inflammation)





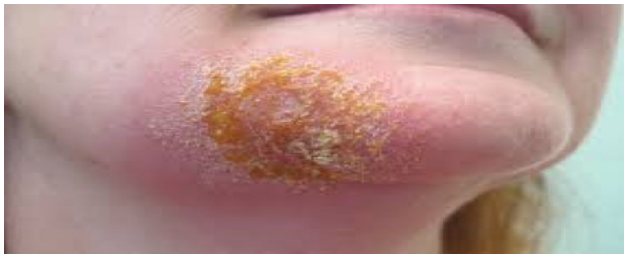


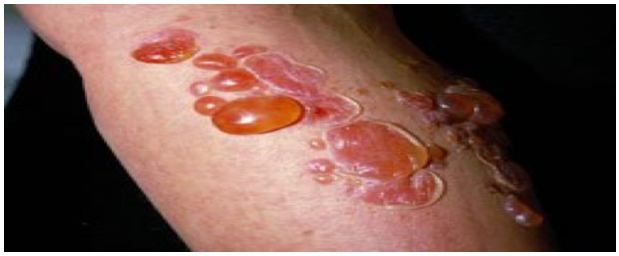
3-Impetigo/Ecthyma

ETIOLOGY	Mostly caused by staph aureus and GAS (group A streptococci).Could be primary mostly in children.or Secondary that arise next to area of colonization, wounds or site of trauma, eczematous lesions and other skin dermatosis (impetiginization).
INVESTIGATION	Diagnosis confirmed by culture but not usually necessary.
CLINICAL PRESENTATION	**Honey colored crusted erosions.** Mostly asymptomatic.
DDx	excoriation, contact dermatitis, herpes simplex, tinea, and scabies.
TREATMENT	**1-Topical antibiotic** topical fusidic acid or mupirocin. **2-Systemic antibiotics in severe cases.** (ECTHYMA)

CONT..

IF UNTREATED ?	If untreated it becomes more extensive and may develop into **ecthyma** (read below) or **cellulitis**.
1- ECTHYMA	•Ecthyma is **ulceration** with thick adherent **crust** at occluded sites in patients with poor hygiene. Lesions may be tender **•Ecthyma might heal with scarring.**
2-BULLOUS IMPETIGO	Bullous impetigo with superficial bullae with erythematous halo mostly in intertriginous areas.

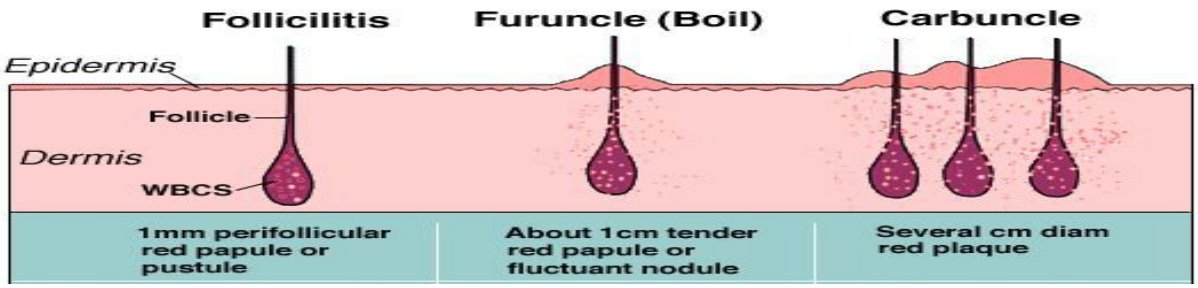




4-Folliculitis and furuncles

DEFINITION	**Infection in hair follicle.**
ETIOLOGY	•caused by staph aureus. can be caused by fungi, viruses, and mites. •Shaving
TREATMENT	•Antibiotics for 7-10 days •Furuncle and abscess treated by **incision and drainage** with antibiotics. •if Recurrent then give prolonged antibiotic therapy rifampicin + clindamycin

VARIANTS	
1-Gram negative folliculitis •occur in patients with acne treated by long term systemic antibiotics. •Treatment: stop offending agent isotretinoin	**2-Hot tub folliculitis** •pseudomonas aeruginosa. •Occurs on the trunk following immersion in spa water



5-cellulitis

DEFINITION	Acute, spreading infection of dermal and subcutaneous tissues
ETIOLOGY	Staph aureus, group A streptococcus.
CLINICAL PRESENTATION	•Fever, chills can develop before cellulitis, •Lower legs most commonly in adults. •Start as spreading and expanding erythematous lesion at the site of entry like trauma, tinea pedis, leg ulcers. •Presents as red, hot, edematous plaque with sharp irregular borders, sometimes with vesicles and erosions.
INVESTIGATION	•Diagnosis is clinical, culture is positive in 30% only. •Skin biopsy could help.
DDx	DVT, urticaria, fixed drug eruption, insect bite, erythema nodosum, erythema migrans
TREATMENT	Systemic high dose antibiotic treatment according to type and sensitivity of microbial organism.

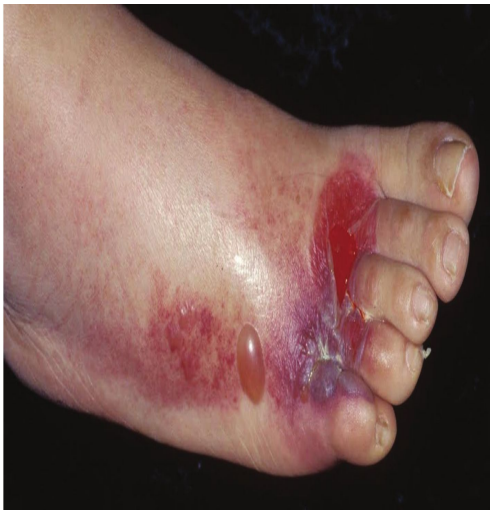
VARIANTS

1-ecthyma gangrenosum

caused BY Aeruginosa IT'S a necrotizing soft tissue infection in immunocompromised patients mostly in axilla, groin or perineum.

2-Erysipelas (acute severe)

•caused by streptococcus group A.Erysipelothrix rhusiopathiae
•Spread rapidly along superficial cutaneous lymphatic vessels.
•Tender red edematous expanding plaque on leg or face.



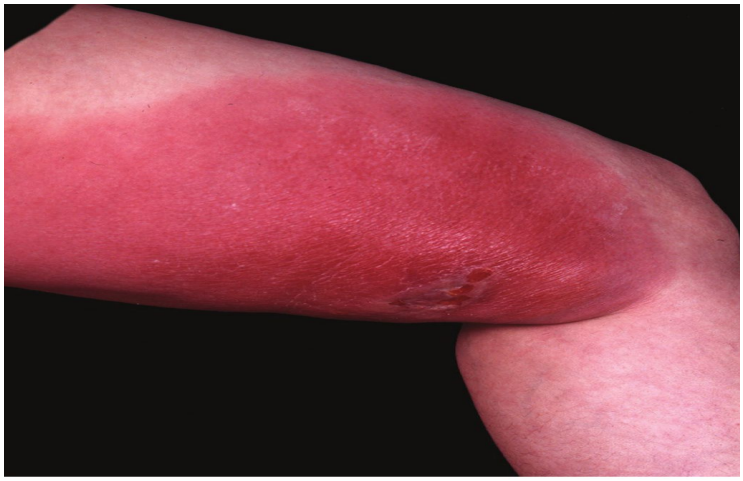
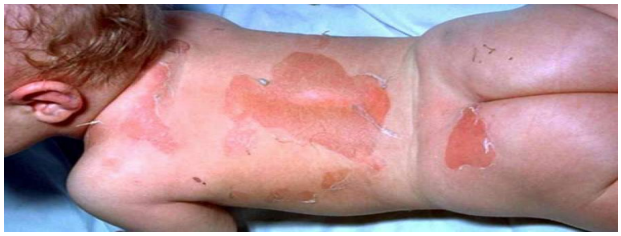
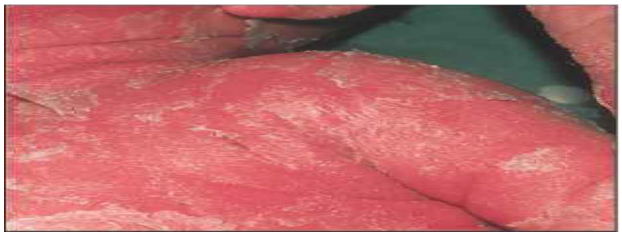


FIGURE 25-29 Erysipeas of thigh: group B streptococcus 52-year-old

6-Staphylococcal Scalded Skin Syndrome (SSSS)

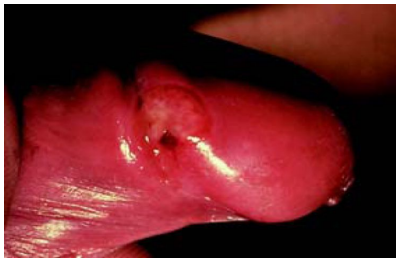
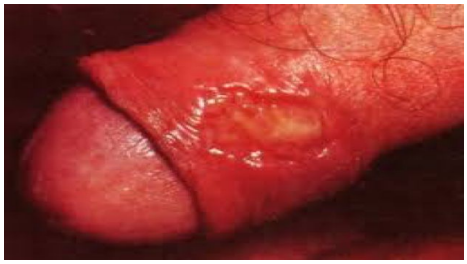
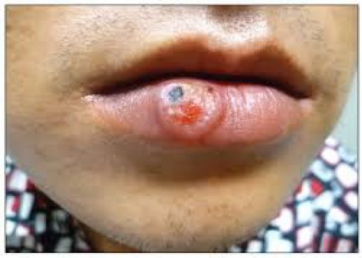
DEFINITION	DISORDERS CAUSED BY TOXIN-PRODUCING BACTERIA (s.aureus)
ETIOLOGY	S. aureus producing exfoliative toxins Exfoliatin is a *Staphylococcus aureus* exotoxin that causes a blistering of the skin
CLINICAL PRESENTATION	•Macular scarlatiniform rash or •**diffuse ill defined erythema** and a fine sandpaper rash initially that rapidly become more erythematous **tender**. The skin can be removed by gentle pressure **(Nikolsky's sign)** •No mucus membrane involvement.
INVESTIGATION	culture
DDx	Kawasaki syndrome, drug eruption, scarlet fever.
TREATMENT	•**Desquamation**(skin peeling) occurs with healing •Heals without scarring. •Systemic antibiotic to treat infection and stop toxin production.





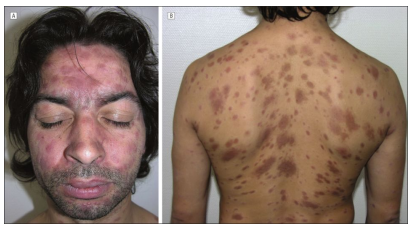
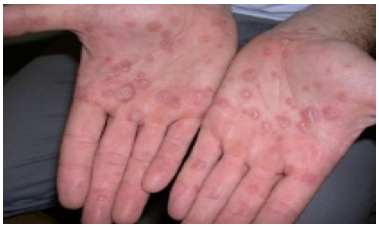
7-Syphilis

DEFINITION	•Chronic **systemic** infection by **spirochete T.pallidum.**
TRANSMISSION	•sexual contact, perinatal in congenital syphilis.
TYPES	•**Primary** syphilis is the **most contagious stage.** •**Later syphilis is a vascular disease** , lesions are secondary to obliterative endarteritis of terminal arterioles and small arteries with the resulting inflammatory and necrotic changes.
CLINICAL PRESENTATION	•Chancre is single button like papule develops at site of inoculation into a painless erosion and then ulcerate with raised border and minimal exudate on penis or vulva, vagina, cervix in women. Lymph nodes are discrete, firm, nontender and unilateral appear within 7 days.
INVESTIGATION	•**dark field microscop**y positive in primary chancre and papular lesions of secondary syphilis(condylomata lata), •**DFA** test in exudate. •**Serologic** Test for Syphilis STS: always positive in secondary syphilis, VDRL is non reactive in 25% of primary syphilis.
TREATMENT	•IM **benzathine penicillin** G 2.4 million units single dose. •**Doxycycline** 100 mg twice daily for 2 weeks.

B-Secondary Syphilis

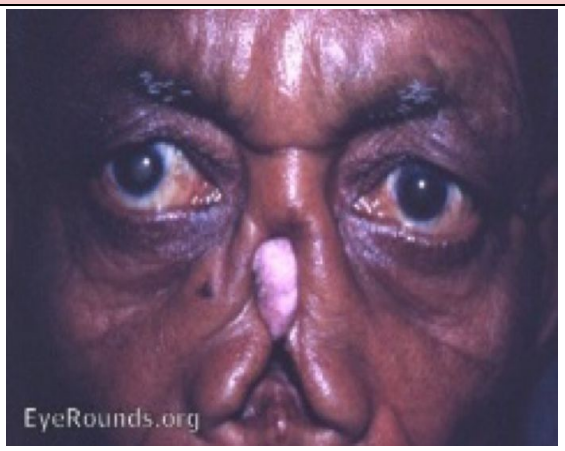
CLINICAL PRESENTATION **Systemic**	•Appear 2-6 months after primary infection. •Fever, sore throat, weight loss, malaise, headache, and meningismus. •**Oval pink-brownish macules and papules**, scaly papules, pustules, that could be annular or polycyclic, usually scattered and symmetric. •Condylomata lata in the anogenital area and mouth. •**Diffuse hair loss**, patch moth-eaten alopecia, loss of lateral eyebrows. •Small asymptomatic flat-topped papules on the oral and genital mucosa, split papules at the angle of the mouth. •Generalized **lymphadenopathy**. •**Peri-osteitis** of long bone mostly tibia, arthralgia, •Acute iritis, optic neuritis, uveitis. •Diffuse pharyngitis, hypertrophic gastritis, hepatitis, ulcerative colitis. •Glomerulonephritis, nephrotic syndrome, cystitis, prostatitis.
INVESTIGATION	•**Histopathology**: epidermal hyperkeratosis, capillary proliferation with endothelial swelling, perivascular infiltration by monocytes, plasma cells, and lymphocytes. Spirochete is present in many tissues like skin, eye, CSF(abnormal in 40% of patients). •**Elevated liver enzymes.** •**Renal function:** immune complex-induced membranous glomerulonephritis.
TREATMENT	•as primary syphilis.


C.Tertiary Syphilis

CLINICAL PRESENTATION	**1-Gumma**: nodular or scaly plaques that may ulcerate and form circles. a-May expand rapidly causing local destruction and heal by scarring. b-Could be in skin, bones, liver, stomach, upper airway. **2-Nodular ulcerative syphilides:** like gumma but flatter with central healing. **3-Meningeal syphilis:** headache, nausea, vomiting, stiff neck, cranial nerve palsies, seizures. **4-General paresis:** after 20 years of infection, hyperactive reflexes, Argyll-Robertson pupil, delusions, hallucinations. **5-Tabes dorsalis:** 25-30 years after infection, ataxic wide based gait, foot slap, paresthesia, bladder dysfunction, impotence, optic atrophy **6-endarteritis obliterans.**

Tertiary Syphilis



Mycobacterial Infections**

Mycobacteria are rod-shaped or coccobacilli acid-fast bacilli (AFB). More than 120 species identi ed. Relatively ew associated with human disease:

1-Leprosy (hansen disease)

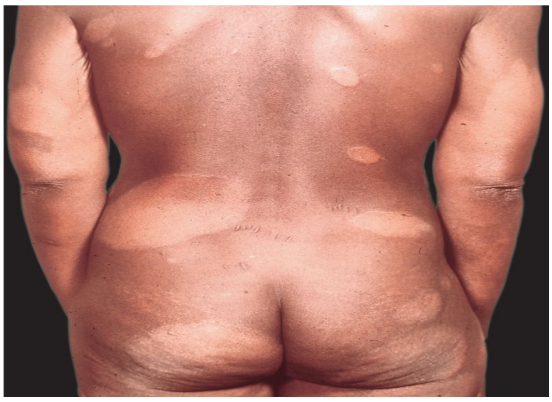
https://www.youtube.com/watch?v=YnGvLVk2BD0 (RECOMMENDED)

DEFINITION	Chronic granulomatous disease principally acquired during childhood or young adulthood.
ETIOLOGY	•**Mycobacterium leprae.**
ONSET	•Onset of leprosy is insidious and painless, **affects nerve initially** eventually muscle weakness, atrophy and contracture.
TRANSMISSION	•Mode of transmission is **unknown**,**due to the long Incubation period 5-7 years**
CLINICAL PRESENTATION	•**VARIABLE** •**Affects skin, peripheral nerves, upper respiratory tract, eyes, testes.**
INVESTIGATION	**1-slit skin smears** A small skin incision is made; the site is then scraped to obtain tissue fluid which is stained by Ziehl-Neelsen stain. **2-PCR.** **3-Skin biopsy:** •**TL shows :**epithelioid granuloma with sparse or **absent bacilli.** •**LL shows** a-extensive cellular infiltrate, b-skin appendages is destroyed, c-and macrophages filled with M.leprae **(virchow cells.)** **Dont culture**
DDx	•DDx: sarcoidosis, Leishmaniasis, syphilis, granuloma annulare, lymphoma.
TREATMENT	Leprosy Management •**TL: dapsone + rifampicin** •**LL: dapsone + rifampicin + clofazimine** •Immune reactions: prednisolone, thalidomide. •Prevention and rehabilitation after nerve damage.

TYPES	
1-Tuberculoid Leprosy TL <u>mild</u>	•localized skin and nerve involvement. **FEW** well defined **hypopigmented** annular plaques with raised borders and anesthetic center. May resolve spontaneously and **is not associated with immune reaction.**
2-Borderline Leprosy BL	•**has both features from Tuberculoid and Lepromatous**, anesthesia and decreased sweat is prominent, does not heal spontaneously and could be associated with **type 1 immune reaction.**
3-Lepromatous Leprosy LL <u>severe</u>	•**generalized involvement of skin** and other tissues.(More Systemic Involvement) •skin colored papules, nodules, might coalesce with diffuse dermal infiltration with **loss of hair** of lateral brows and eyelashes and leonine face.

Immune responses:

- <u>Type 1</u> acute tenderness and pain along affected nerves with loss of function and skin lesions become inflamed and may ulcerate. Edema on face, hands, and feet.
- <u>Type 2</u> erythema nodosum leprosum which is seen in 50% of patients with Lepromatous leprosy (LL) after starting treatment. With painful red skin nodules on the face and extensor limbs.
- <u>Type 3</u> Lucio reaction in Mexico, manifests as erythematous plaques with shallow large polygonal sloughing ulcerations on the legs in patients with LL.



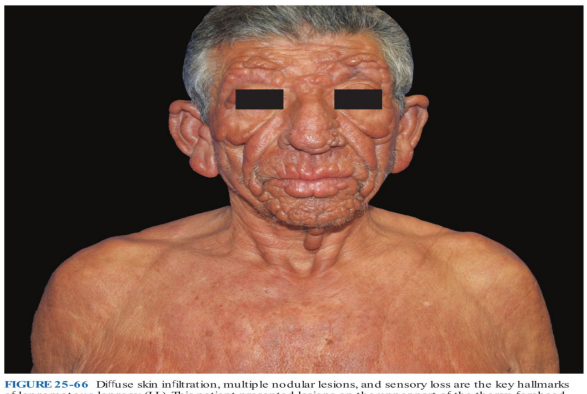


FIGURE 25-66 Diffuse skin infiltration, multiple nodular lesions, and sensory loss are the key hallmarks of lepromatous leprosy (LL). This patient presented lesions on the upper part of the thorax, forehead,

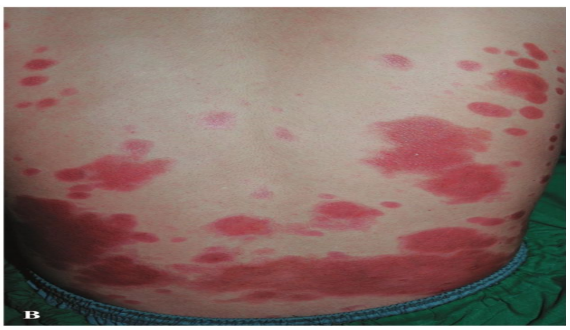
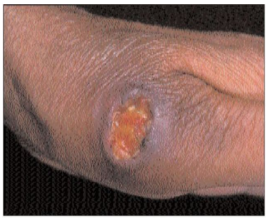


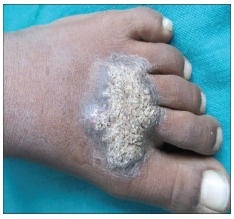
FIGURE 25-65 **Leprosy: borderline-type** A 26-year-old Vietnamese male. **(A)** Well-demarcated, infiltrated, erythematous plaques on the face. **(B)** Identical red plaques on the lower back.

2-Cutaneous Tuberculosis

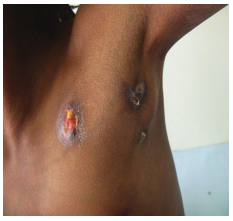
ETIOLOGY	•**Mycobacterium tuberculosis.**
TYPES **Based on** 1-route of inoculation 2-And patient immunologic status **Chancre = painless ulcer**	**1-Tuberculous chancre:** exogenous inoculation into skin in non immune patient. **2-Tuberculosis verrucosa cutis:** exogenous inoculation into skin in immune patient. **3-Scrofuloderma:** direct extension from joints, bone or lymph nodes. **4-lupus vulgaris** Lymphatic spread to skin **5-acute miliary TB** Hematogenous dissemination
CLINICAL MANIFESTATIONS **LOOK AT PICS BELOW**	**1-Tuberculous chancre:** starts as papule at inoculation site after 2-4 weeks then enlarge to form a painless ulcer and regional lymph node. **2-Tuberculosis verrucosa cutis:** papule with violaceous border that evolve to hyperkeratotic warty plaque. Usually single on hands in adults, and on legs, knees in children with no lymph nodes. **3-Scrofuloderma:** firm subcutaneous nodule that liquefies and perforate leading to undermined ulcer with pus or caseous discharge. On the parotid, submandibular, supraclavicular, lateral neck. **4-Lupus vulgaris:** papules evolve to well defined irregular scaly plaque, that is soft and friable, mostly on nose, ear and scalp. Scarring is prominent. **5-Acute miliary TB:** disseminated minute macules and papules or purpuric lesions **all over body** especially trunk.
INVESTIGATION	PPD(TUBERCULIN SKIN TEST), skin biopsy, PCR, culture.
TREATMENT	•**isoniazid+rifampicin** •Tuberculosis verrucosa cutis **if small surgical excision.**



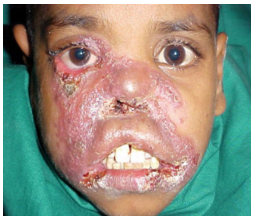
TYPE 1



TYPE 2



TYPE 3



TYPE 4

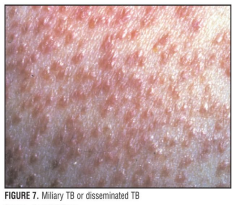


FIGURE 7. Miliary TB or disseminated TB
Source = DermNetNZ.org

TYPE 5

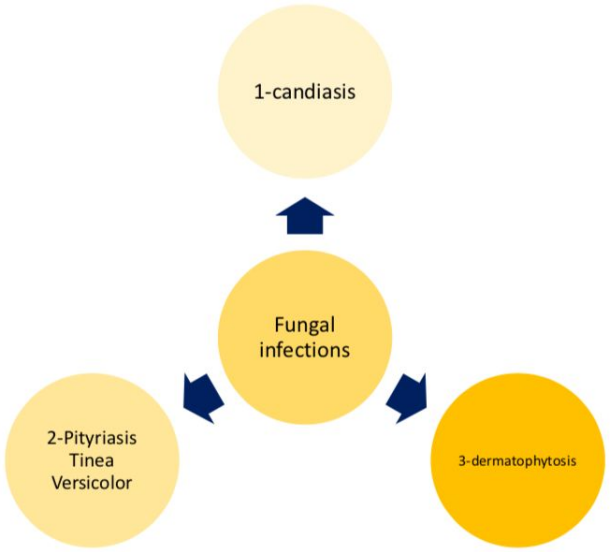
FUNGAL INFECTIONS**

divided into:

1-superficial (our topic): fungi that affect skin, hair, nail and mucosa, like candida, malassezia, and dermatophytes.

2-Deep chronic cutaneous: after percutaneous inoculation like eumycetoma, chromoblastomycosis and sporotrichosis.

3-Systemic with cutaneous dissemination like cryptococcosis, histoplasmosis, blastomycosis, coccidioidomycosis.



1-Candidiasis

ETIOLOGY	•**Candida albicans,** C.tropicalis, C.parapsilosis,C.krusei, C.kefyr,C.glabrata.
CLINICAL MANIFESTATIONS	**1-Cutaneous candidiasis:** intertriginous and occluded skin.LOOK BELOW **2-Mucosal candidiasis**: oropharynx and genitalia. **3-Disseminated candidemia:** host defense defects especially **neutropenia**. Usually after invasion of GIT.
RISK FACTORS	•Immunocompromised, DM, obesity, hyperhidrosis, warm climate.
PREVENTION	keep intertriginous area dry, anti fungal powder.
TREATMENT	•Topical antifungal: nystatin, azoles, imidazole. •Systemic antifungal: fluconazole, itraconazole, voriconazole.

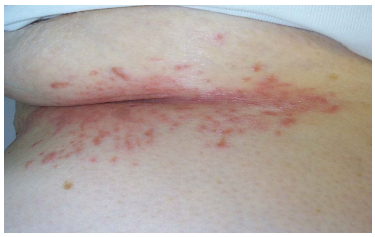
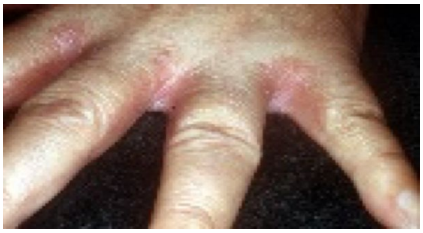
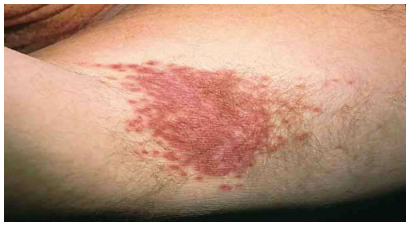
INVESTIGATIONS: CULTURE +KOH preparation

Cutaneous candidiasis subdivided into:

1-Candidal Intertrigo: sharply demarcated, polycyclic, erythematous, eroded patches with satellite pustules affect body folds.

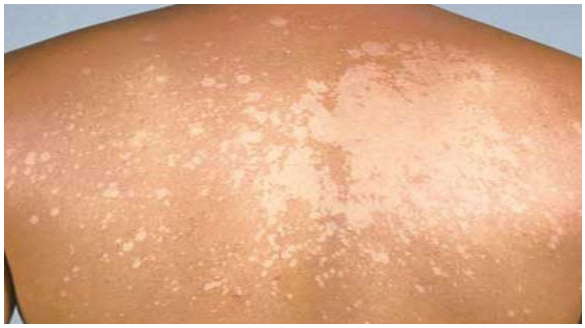
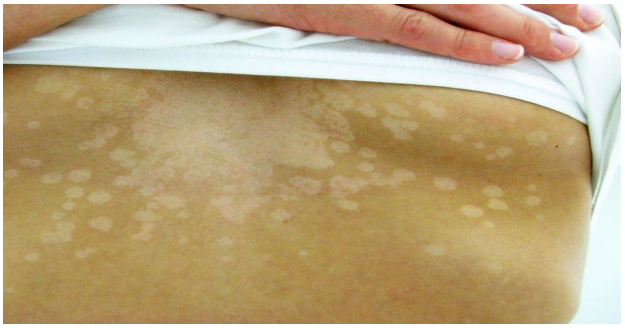
2-Interdigital: in obese elderly, webspace between 3rd and 4th toes.

3-Diaper dermatitis: beefy red plaques with papular and pustular lesions, erosions on genital and perianal area, inner thighs and buttocks

2-Pityriasis (Tinea) Versicolor

ETIOLOGY	•Superficial overgrowth of **Malassezia** furfur and M. globosa, both resident cutaneous flora. •**Not contagious**. More with sweating, warm weather, oily skin
CLINICAL MANIFESTATIONS	•Asymptomatic tan-light brown macules with fine scales, on dark skin could be hypopigmented macules mostly on trunk, also on neck, upper arms axillae and groin. •Malassezia changes from a yeast to hyphal form leading to inflammation of the skin. •Hypopigmentation is caused by **dicarboxylic acid produced by Malassezia species which inhibits tyrosinase in melanocytes.**
INVESTIGATIONS	•Woods lamp: blue green fluorescence of scales. •KOH: filamentous hyphae and globosa yeast forms (spaghetti and meatballs) •Histopath: budding yeast, and hyphal forms in the stratum corneum by PAS stain, hyperkeratosis, hyperplasia and chronic inflammation.
TREATMENT	•**ketoconazole shampoo**, azole creams. •**IF SEVERE OR RECURRENT** **Systemic fluconazole** for 3 weeks or itraconazole single dose.

3-Dermatophytosis

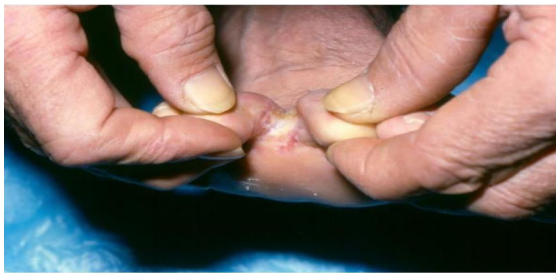
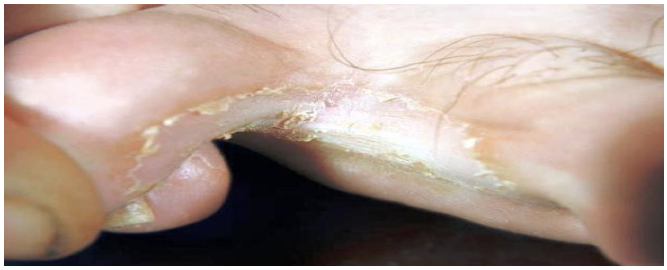
•Dermatophytes FUNGAL INFECTIONS
•3 genera of dermatophytes: **trichophyton**, microsporum, and epidermophyton.
•**Trichophyton rubrum** is the most common cause of epidermal dermatophytosis.
•**Children have scalp infections, adults have intertriginous infections.**

A.Tinea pedis

AREA	Infection of feet
CLINICAL MANIFESTATION	•Usually asymptomatic, with hyperkeratotic macerated plaques between **toe webs**. Could be seen with tinea unguium.
RISKS	•Hot, humid, occlusive footwear, hyperhidrosis.
DDx	**1-interdigital type:** Erythrasma **2-moccasin type:** psoriasis, chronic eczema **3-inflammatory type:** bullous impetigo, allergic contact dermatitis.

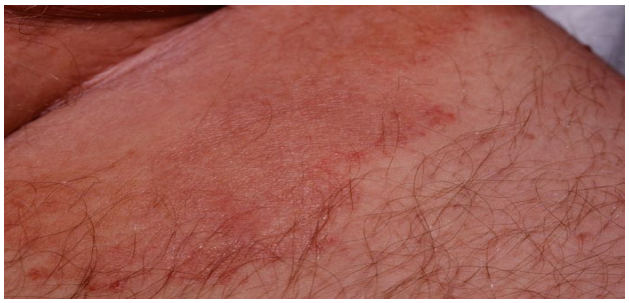
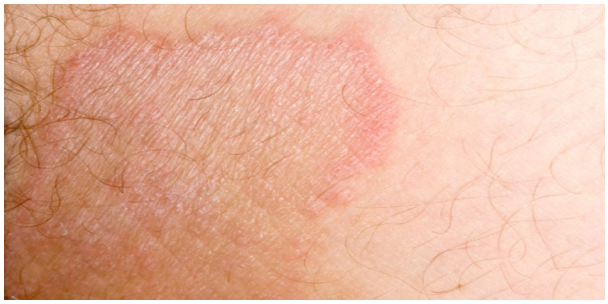
Types:

1-*Interdigital type*: dry scaling or maceration and fissuring of toe webs, hyperhidrosis is common most commonly between 4th and 5th toes.
2-*Moccasin type*: well demarcated scaling with erythema and minute papules on margin, fine white scales and hyperkeratosis on soles, heels and lateral border of feet. Bilateral involvement is common.
3-*Inflammatory type*: vesicles or bullae, may be associated with id reaction.
4-*Ulcerative type*: extension of interdigital tinea onto plantar and lateral foot ? Secondary bacterial infection

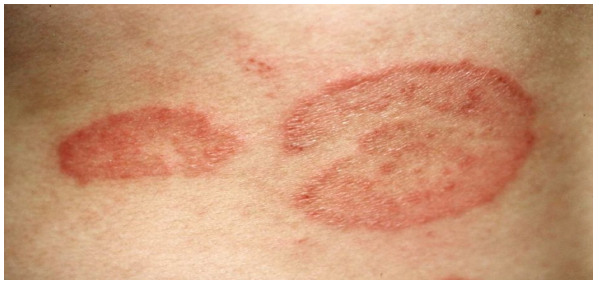
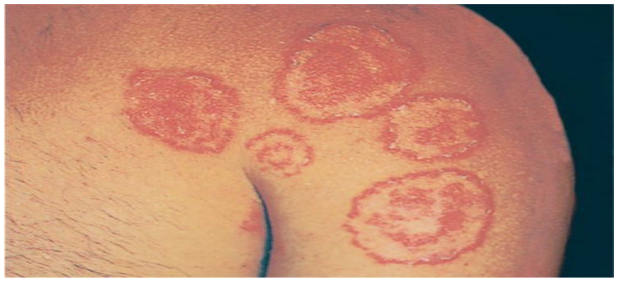
B.Tinea Cruris

AREA	**groin, thighs and buttocks.**
CLINICAL MANIFESTATION	•Large scaling well demarcated erythematous- brown plaques, with papules and pustules at the periphery
DDx	•Erythrasma, candida Intertrigo, inverse psoriasis, Pityriasis Versicolor.
TREATMENT	•**topical antifungal** imidazole, allylamines, naphthionates and pyridine. •if severe? Systemic antifungal, terbinafine, itraconazole, fluconazole.

C.Tinea Corporis

organism	•Mostly by T.rubrum
Area	•**trunk and extremities.**
Clinical manifestation	•**Well defined scaly erythematous plaques** with advanced border and central clearing, sometime it coalesce to form large polycyclic annular and concentric rings.
DDx	•DDx: allergic contact dermatitis, atopic dermatitis, annular erythema, psoriasis, Pityriasis rosea, Pityriasis Versicolor, subacute lupus, mycosis fungoides.

D.Tinea Capitis

ORGANISMS	•T. tonsurans, M.canis, M.audouinii, T.violaceum, T.schoenleinii.
AREA	•**scalp**
ONSET	•6-10 years of age
CLINICAL MANIFESTATIONS	**1-Ectothrix infection:** occurs outside the hair shaft with cuticle destruction. Caused by M.audouinii, M.canis. **2-Endothrix infection:** occurs within the hair shaft without cuticle destruction. Caused by T.tonsurans, T.violaceum. **3-Black dot:** resembling seborrheic dermatitis. **4-Kerion:** with boggy inflammatory plaques. **5-Favus:** endemic in middle east and Africa.
TREATMENT	•**Griseofulvin** 15-20mg/kg/day for 4-6 weeks. •**Terbinafine** 3-6mg/kg/day for 4-6 weeks •**Itraconazole** 3-5mg/kg/day for 4-6 weeks. •**Fluconazole** 3-6mg/kg/day for 4-6 weeks

TYPES:

1-Non inflammatory infection: gray patch type, with scaling diffuse or circumscribed alopecia. Circular alopecia with broken off hairs, fine scales, no or minimal inflammation. M.canis may show green fluorescence under woods lamp.
2-Black dot: broken off hairs near scalp, diffuse poorly circumscribed, usually caused by T.tonsurans.
3-Kerion: inflammatory mass with loose hairs, boggy, painful,purulent, inflamed nodules and plaques oozing pus with lymphadenopathy. Caused by T.verrucosum, T.mentagrophytes. Heal with scarring.
4-Favus: thick yellow adherent crust (scutula) composed of skin debris and hyphae pierced by remaining hair shaft. **Often results in scarring.**