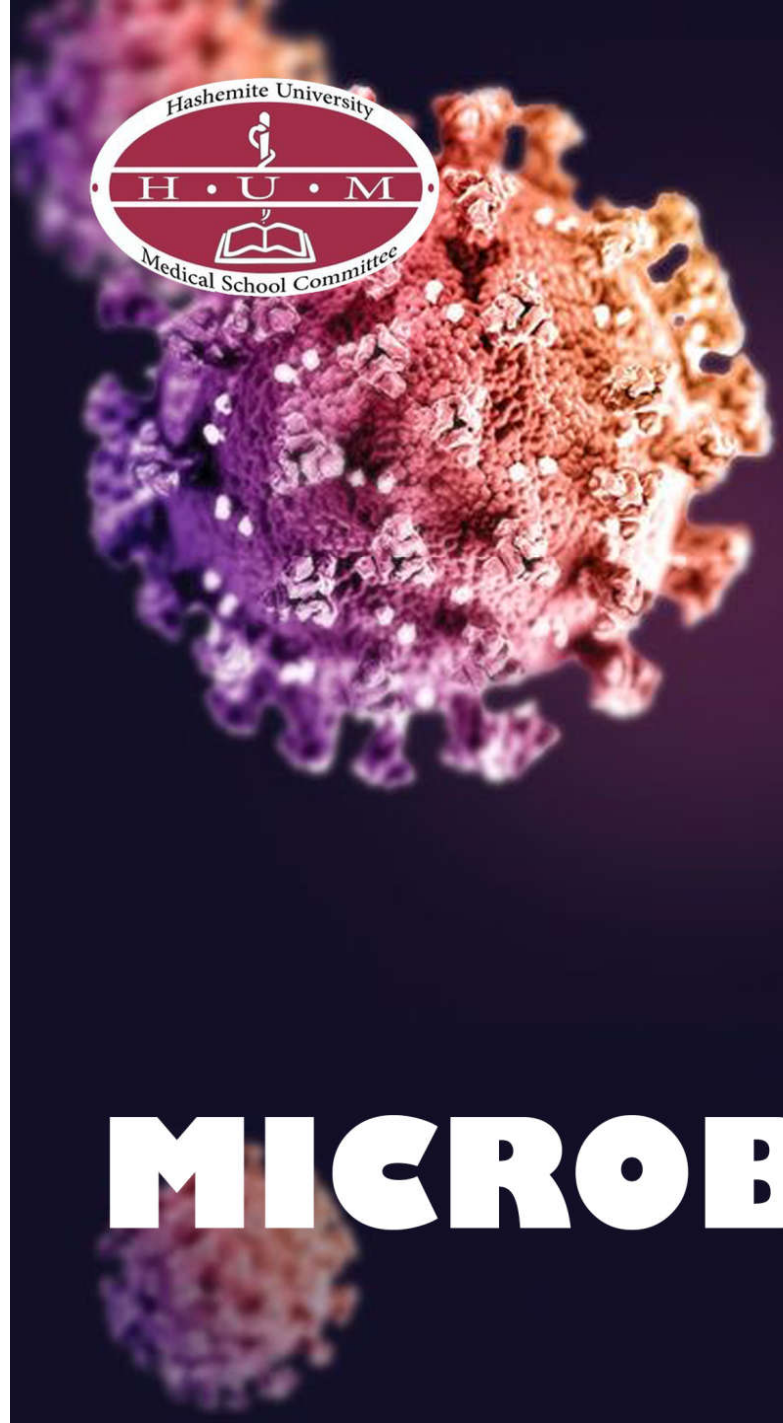

MICROBIOLOGY

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BACTERIAL SKIN INFECTIONS

Skin lesion ⟶	IMPETIGO	Erysipelas (Superficial cellulitis)	Cellulitis
Affected layer from skin	Epidermis	Superficial dermis	Deep dermis and the subcutaneous tissue
Causative MOs	1 [Staphylococcus aureus] 2 [Group A beta hemolytic] streptococcus 3 [P.aeruginosa] (usually occurs in hot bath)	Streptococcus A (sterp pyogenes)	1 [Staphylococcus aureus] 2 [Group A beta hemolytic] streptococcus 3 [H.influenza]
Description of the lesion	A contagious local, superficial bacterial blistering skin infection. Two types: a.Irritating Bullous which is mainly caused by staph aureus & b. Painless non-bullon impetigo with honeycomb appearance • ETs produced by staph aureus cause disruption of cell to cell adhesion (Target: Desmolgin Type I).	Swelling inflamed butterfly appearing rash with systemic features such as : - Fever chills rigors - LN involvement.	Inflammation & only a little bit of swelling in the lower limbs such as around the tibia. - Periorbital Cellulitis with cardinal signs of inflammation.
Classical Clinical presentation	Child presents with crusting lesions around the mouth or in the upper limb	Cardinal signs of inflammation in the lower limbs causing skin to be raised with **DISTINCT** borders.	Since the infection has occurred in the deep dermal structure of skin, it is only slightly raised with **INDISTINCT** borders. (Using a marker is useful)
Risk Factors	Poor hygiene Overcrowding Other skin diseases such as dermatitis	Patients having their own risk factor.	Patients having their own risk factor such as immunodeficiency, diabetes, lymphedema, obesity or venous insufficiency. Other skin lesions such as tinea pedis or dermatitis.

Main complications	Cellulitis, Sepsis and Bacteremia, glomerulonephritis (particulary with strep infection) and OM or septic arthritis	-	-
Diagnosis	Clinically based on history & physical examination	Clinically based on history & physical examination	Clinically based on history & physical examination
Treatment	1st line : mupirocin (Bactroban) 2nd line : penicillins such as dicloxacillin & flucloxacillin. If there is hypersensitivity Clindamycin is an alternative.	1st line : penicillins such as dicloxacillin & flucloxacillin If there is hypersensitivity Clindamycin is an alternative.	1st line : penicillins such as dicloxacillin & flucloxacillin If there is hypersensitivity Clindamycin is an alternative.

Skin lesion →	Folliculitis	Furuncle (Boil)	Carbuncle
Description of lesion	Bacterial infection in the upper portion of the hair follicle that causes a papule, pustule of erosion at the base of the hair follicle. Hot tub Folliculitis is caused by P.aeruginosa.	Acute Superficial skin infection result from *S.Aureus* invasion of hair follicles making small abscess. It affects the entire hair follicle and the adjacent subcutaneous tissue.	Extensive infection of a group of contagious follicles, dermis and subcutaneous tissues. It is simply a group of interconnected furuncles.
Signs & Symptoms	papules/pustules on scalp, arms or legs	painful nodules, commonly on neck, face, axillae, or buttocks. Nodules enlarge and then rupture	Extremely painful, deep abscesses draining through multiple openings onto the skin surface, around several hair follicles (painful hard lump)

Complications:		Involvement of adjacent sebaceous or sweat gland.	Large deep ulcer. Fever and malaise. Low immunity is the main predisposing factor.
Main causative MOs:	Staphylococcus Aureus. P.aeruginosa can cause condition known as Hot tub folliculitis	Staphylococcus Aureus.	Staphylococcus Aureus.
Treatment	Thorough cleaning of the infected area with antibacterial soap and water several times per day. Warm, wet compresses to promote VD and drainage. Topical antibiotics, such as mupirocin ointment or clindamycin or erythromycin solution	warm, wet compresses -incision and drainage - systemic antibiotic therapy	incision and drainage / systemic antibiotic therapy.

Suspected organisms

- **Impetigo:** Group A *Streptococcus, Staphylococcus aureus*
- **Folliculitis:** *Staphylococcus aureus, Pseudomonas aeruginosa*
- **Furuncles:** *Staphylococcus aureus*
- **Carbuncles:** *Staphylococcus aureus*
- **Cellulitis:** Group A *Streptococcus, Staphylococcus aureus, Haemophilus influenzae*
- **Erysipelas:** *Group A Streptococcus*
- **Necrotizing fasciitis:** *Group A Streptococcus, Clostridium perfringens and other species, Bacteroides fragilis, ther anaerobes, Enterobacteriaceae, Pseudomonas aeruginosa*

VIRAL SKIN INFECTIONS

Viral skin infection:	Viral Warts	Molluscum Contagiosum	Varicella	Herpes Zoster
The causative virus	- High & low risk HPVs (non-enveloped virus).	-Molluscum Contagiosum virus. - Pox virus - it is one of the most common childhood skin conditions.	Herpes Virus	Herpes Virus infecting dorsal root ganglion & Trigeminal ganglion of (5th cranial nerve)
Resulting lesions and clinical presentation	-The virus infect epithelial cells of skin & mucous membranes. -HPV types such as 6/11 and others cause benign proliferations of the skin (hands, nails, soles, face) & mucosa HPV types: 16 & 18 can cause malignant transformations in genital region or upper respiratory tract (mainly SCCa). Genital warts = Condyloma Acuminata	-Chronic localized skin infection with vesicular shaped papules. -Risk factors include: Immunodeficiency, sexually active individuals and having other skin conditions.	-Chicken pox -98% of human populations by adulthood. -rash on the face and scalp which progress downwards to trunk and extremities. - Macules & Papules & Fluid filled vesicles & crust over forming scabs & eventually the scab fall without scarring. -Painful lesions in the oral mucosa can also be seen. - remember that it is possible to see lesions in different phases and different sites on the body at the same time. -lesions often are accompanied by mild fever , malaise or secondary bacterial infection due to scratching by the infected individual.	-Shingles that results from reactivation of VZV when the immunity of infected individual drops. -Lesions appear posteriorly then progress in anterior direction then to peripheral locations. -presents as grouped papules, vesicles, pustules and crusts on erythematous base. -pain itching tingling. -Single stripe of vesicles around left or right side of body or at one side of face.
Pathogenesis	HPV enter the basal cell layer of epidermis through trauma or abrasion and there can produce viral DNA &	-It can be distributed anywhere in the body except in the palms and soles.	-Getting infected with a virus (e.g. through droplets) will cause the virus to enter the	As the VZV infects the keratinocytes in chicken pox, it also can infect the

	proliferation of keratinocytes & BVs and that will lead to formation of warts. Koilocyte (which is cytoplasmic vacuolization & producing perinuclear halo) can be seen in some lesions.	-It is often asymptomatic but pureitis can occur. - Itching can cause autoinoculation and result in mollscum dermatitis.	and replicate there. The regional LNs then can take the virus by means of circulating immune cells. -Primary viremia takes place when the virus reaches the reticuloendothelial system (RES). -within 2 weeks the virus start to infect T-cells themselves resulting in secondary viremia and the infected T-cells will produce viral proteins that bind with specific receptors on the epithelium of skin causing the apparent lesions of Chicken pox.	on the skin. Retrograde transport of viruses through theses neurons will cause the virus to enter the dorsal root ganglion of that neuron allowing the virus to enter a long period of latency (10-20 years). -Reactivation can occur resulting in the previously described lesions. - PHN can occur as a complication of shingles.
Transmission	Direct contact with infected epithelial cells. Activities which increase getting infection include: -Sex, Child birth, Sharing clothes & Auto-inoculation.	1.direct skin to skin contact 2.Sexullay transmitted 3.Autoinculation 4.infected fomites 5.Swimming pool	By respiratory contact with airborne droplets AND by direct Skin-To-Skin contact as the virus is highly contagious.	Direct contact with fluid
Diagnosis	-***Clinically*** (when pare the surface the surface of wart it shows small black dots, that is , thrombosed capillaries) -***Dermoscopy & Endoscopy*** help in recognizing of warts. Definitive diagnosis is made by molecular testing for viral DNA or RNA.	By two ways : -***Clinically*** by observing dome-shaped papule having belly button or umbilicus appearance -***Histologically*** by observing mollscum bodies or Henderson Patterson Bodies	Clinically	-Clinically -when in doubt we can do viral culture for VSV -Histopathology, Tzanck smear & Ab studies all are useful.

Treatment	-Most resolve spontaneously in 1 to 3 years. -Treatment may include : removal of warts , destructive treatment and agents like : keratolytics. -Cryotherapy with liquid nitrogen / for those large & annoying warts we can remove them by electrosurgery. -Appropriate expectations must be discussed first !	-destructive ttt	- Vaccines are available and are 80% effective. Symptomatic, including: -analgesic anti inflammatory. -Topical antiprutitc. - Antibiotics in cases of secondary bacterial infections. **REMEBER THAT ASPIRIN IS AVOIDED IN CHILDREN AS IT CAUSES REYE SYNDROME !!!**	Mainly Supportive & Symptomatic

FUNGAL SKIN INFECTIONS

A.Superficial infections

Skin lesions	Ringworm	Pityriasis (tinea)versicolor	Tinea nigra	Piedra
Fungus	Dermatophytes including three species: 1- Microsporum 2- Trichophyton 3-Epidermophyton.	Malassezia Furfur (body yeast cell with hyphae)	Hortaea werneckii (black pigmented fungus found usually in soil)	White : Trichosporon cutaneum Black : Piedraia hortae
Manifestations	Erythema, induration, itching & scaling	Hypo- or Hyper-pigmentation with induration and scalir	Brown to black macular lesions on the palms & soles	Infection of the hair characterized by black or white nodules attached to the hair shaft
Description of disease	-slowly progressive -mild infection -rarely invading the deep dermal layer -symptoms are attributable to fungal metabolic products during	-	-	WHITE PIEDRA: Light + Soft + loosely attached to the shaft BLACK PIEDRA Dark + Hard

	-well-demarcated lesion with minimally inflammatory border containing central clear region with scaling.	-	-	-
Transmission	Human to human transmission through close contact. Also, through intact or detached skin or hair in barber shops or other places.	-	-	-
Clinical presentation	It ranges from unapparent colonization of the skin to chronic progressive eruption with considerable discomfort & disfiguration. The course of infection is dependent on : 1-affected anatomical location (tinea capitis on scalp / tinea cruris on groin region) 2-dynamics of skin growth in that region 3- associated inflammatory processes.	-	-	-
Risk factors	Moist and sweaty skin folds as well as obesity and tight apparel	-	-	-
Diagnosis	1.KOH → 2.U.V lamp → 3.Cultute (LAB MATERIAL) →	-Dermatophytes can spontaneously resolve. -topical antifungal can be used or oral systemic therapy in case of extensive skin infections	-	-

Tinea capitis	Fungal infection	Steroids are contraindicated	fractures of shafts of hair
Alopecia	Non-fungal infection	Treated with steroids	Complete loss of hair at affected site

B. Subcutaneous Infections

Skin disease	Sporotrichosis	Chromoblastomycosis	Mycetoma
Causative fungus	Sporothrix schenkii	Multiple etiologies	Multiple etiologies
Epidemiology	Infection is acquired by traumatic inoculation through the skin of material containing the organism – Considered as occupational disease of gardeners and farmers.	-tropical disease caused by pigmented saprophytic fungi. - organisms found in the soil of endemic areas and occur mostly with individuals working barefoot	-
Manifestations	-painless papule that ulcerates within weeks and months - LN involvement with multiple ulcers on the skin	-appears as papules then develops into scaly wart-like structures under the feet. - slow painless - NO involvement of lymphatics.	-trauma to the foot with inoculation of a dozen (multiple) fungal species. - massive induration and draining sinuses

فاقنَعْ بما قَسَمَ المليكُ فإنَّما
قَسَمَ الخَلائِقَ بيننا عَلَّامُها

لبيد رضي الله عنه