MSS | Lec 3 micro sum.
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Hand foot and mouth disease (HFMD):

-Affect large groups of people in small space = outbreak
-typically in schools
-**caused by:**
1. Coxsackie virus A16 (an enterovirus)
2. Enterovirus 71 (HAS BEEN ASSOCIATED WITH ENCEPHALITIS AND MYOCARDITIS)

- Atypical disease: is the disease that is caused to a Higher age groups (adults and teenagers), immune system is more competent and mounts a more aggressive response and this → severe presentation.
-caused by: coxsackie A6
-fever, arthralgia and flu like symptoms, rash as vesicles that affect (the nose, cheeks, extensor arms, elbows, thighs, buttocks, groin)

*Both typical and atypical disease are transmitted by fecooral route, or direct close contact to rash.

HFMD **Symptoms**:
• URT symptoms.
• fever, malaise and pharyngitis.
• rash:
• ORALLY: football shaped (eye shaped) painful vesicles, involves the buccal mucosa and tongue. (spares posterior pharynx as opposed to herpangina that spares anterior pharynx)
• ON SKIN: red papules that **progress to gray vesicles** on the soles and palms and buttocks

-**Treatment**:
-Only symptomatic treatment (treat fever, pain)
• Must maintain hydration, (essential as in all fevers)

Smallpox:

-caused by variola virus, and orthopoxvirus
-incubation period: 10–12 days, followed by a prodromal period of 1–2 days.
•rash is initially maculopapular and progresses to vesicles, pustules, and scabs over 1–2 weeks.
• Death may occur with fulminant disease (sudden and escalates quickly, and is intense and severe)
• **Diagnosis**: may be confirmed by EM or PCR (to differentiate it from other poxviruses).
• no specific **treatment** (supportive)

Orf disease: (soremouth infection)

-**caused by** parapoxvirus.
transmit between animal to human (petting, feedings, harnesses,bite) and human to human.
-**no treatment.**
Six stages: each about a week
• Small papule → nodules → ulcerate and crust

Molluscum contagiosum virus فيروس المليساء المعدية

-induces flaccid vesicles on the skin
- Mcc 2-11 year olds, in adults as part of STD.
- **transmission** by direct contact
• Single or small clusters of vesicles (<30) raised papules
- not erythematous!, with central umbilication
• On face and trunk, pubis and rarely mucosa
• Resolve on their own
- may be present for YEARS
• If associated with HIV → SEVERE
• Complications: Scarring especially if manipulated, bacterial infections, and conjunctivitis if happens near the eyes.

Recall من المحاضرة الأولى:

Crusted lesions : bullae that do not remain closed for long, fluid released crusts over/or lesion that crust during the course of infection.

Infections Associated with Crusted Lesions

Crusted lesions	
Bullous impetigo/ecthyma	*S. aureus*
Impetigo contagiosa	*S. pyogenes*
Ringworm	Superficial dermatophyte fungi
Sporotrichosis	*Sporothrix schenckii*
Histoplasmosis	*Histoplasma capsulatum*
Coccidioidomycosis	*Coccidioides immitis*
Blastomycosis	*Blastomyces dermatitidis*
Cutaneous leishmaniasis	*Leishmania* spp.
Cutaneous tuberculosis	*Mycobacterium tuberculosis*
Nocardiosis	*Nocardia asteroides*

Impetigo:

-Caused by S. aureus and/or GAS (group A strep) ((SKIN NF))
prevalent in warm weather.

• It is the most common bacterial skin infection in children age 2-5.

• 70% appear as crusted legions

• highly contagious معدي (scratching, towels, clothing, autoinfection and spread in daycares)

- occurs on the face and extremities.

• start as small macule or papule → small vesicles (with erythema)
→ flaccid bullae → rupture, releasing a yellow discharge which forms thick crusts.

• Usually seen with regional LAP (lower abdominal pain)

• Can cause cellulitis (deeper infection) Or PSGN (post strep glomerulonephritis)
- can progress to ecthyma.

• **Treatment**: mupirocin (a topical agent).

- in numerous lesions/no response to topical treatment ➡️ oral antibiotics: flucloxacillin or cefalexin.

- In MRSA cases: doxycycline, clindamycin, or co-trimoxazole.

Organism	Type of Pathogenesis	Typical Disease	Main Site of Disease (D), Colonization (C), or Normal Flora (NF)
S. pyogenes (group A)	1. Pyogenic		
	a. Local	Impetigo, cellulitis	Skin (D)
		Pharyngitis	Throat (D)
	b. Disseminated	Sepsis	Bloodstream (D)
	2. Toxigenic	Scarlet fever	Skin (D)
		Toxic shock	Many organs (D)
	3. Immune-mediated (poststreptococcal, nonsuppurative)	Rheumatic fever	Heart, joints (D)
		Acute glomerulonephritis	Kidney (D)

Organism	Type of Pathogenesis	Typical Disease	Predisposing Factor	Mode of Prevention
S. aureus	1. Toxigenic (superantigen)	Toxic shock syndrome	Vaginal or nasal tampons	Reduce time of tampon use
		Food poisoning	Improper food storage	Refrigerate food
	2. Pyogenic (abscess)			
	a. Local	Skin infection (e.g., impetigo, surgical-wound infections)	Poor skin hygiene; failure to follow aseptic procedures	Cleanliness; handwashing; reduce nasal carriage
	b. Disseminated	Sepsis, endocarditis[1]	IV drug use	Reduce IV drug use

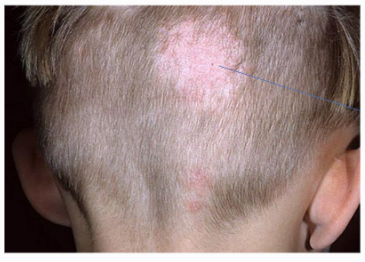
Ecthyma:

- A deeper form of impetigo.
• it is punched-out ulcers surrounded by raised deep red/violet margins.
- invades into the dermis.
• **Caused by:** S.aureus or GAS, other similar lesions (ecthyma gangrenosum) may occur with P.aeruginosa in neutropenic (reduced neutrophils in blood) patients.
• **treatment:** flucloxacillin or cephalexin (unless cultures yield streptococci alone, in which case penicillin is appropriate).
- In the deeper form, topical treatment isn't used
• Antipseudomonal agents, e.g. piperacillin-tazobactam, should be given for P. aeruginosa infections.

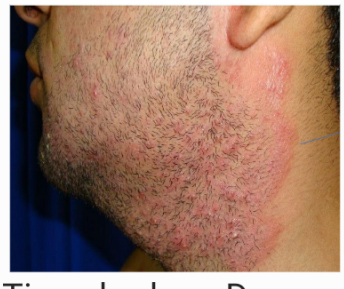
Dermatophytes:

A group of fungi (more than one), the connection between this is that they are capable of invading and feeding off of the dead keratin of skin, hair, and nails (require keratin for their growth).
• **spread by:** direct contact with patients or animals or soil.
-**Clinical classification** is by age group:

Children:
-tinea capitis (scalp hair, the commonest in children).
-tinea corporis (trunk and limbs).
-tinea faciale (face).
Mnumonic: CACOFA
Adolescents:
-tinea manuum and pedis (palms and soles -athletes foot-, the commonest worldwide).
-tinea unguium (nail—also known as onychomycosis).
Adults:
-tinea cruris (groin), called as jock itch.
-tinea barbae (beard area and neck).
-Tinea corporis Gladiatorum (wrestlers).



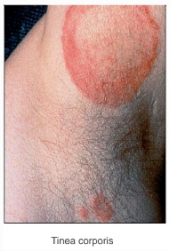
Tinea capitis: Feeding off of Keratin, present In hair and skin



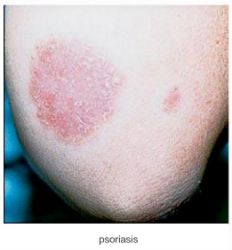
Tinea barbae: Demarcation lines hint at A possible dermatophyteInfection, rather than acne vulgaris

DIAGNOSIS:

• KOH mounts of skin scrapings and infected hairs demonstrate hyphae.
• Some species fluoresce by a U.V. lamp.
• Culture is used when KOH preparations are negative.
• NOTE: Infections caused by bacteria, other fungi, and noninfectious disorders (psoriasis, contact dermatitis) may have similar features.



Tinea corporis



psoriasis

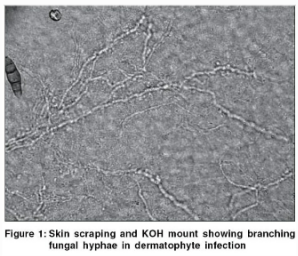


Figure 1: Skin scraping and KOH mount showing branching fungal hyphae in dermatophyte infection

TREATMENT AND PREVENTION:

- tolnaftate, allylamines, or azoles (topically)
- griseofulvin or itraconazole and terbinafine (forNail bed and more extensive skin infections)
- Note: Many local skin infections resolve spontaneously without chemotherapy.

Cutaneous leishmaniasis (CL): (Disease of the poor)

-Vector and parasite: sandflies

- Trypanosome (single flagellae)

-L. major and L. tropics is most common

• Parasite **Has two forms**: disease causing (resistant) form and infectious motile form

• Leishmania **comes in 3 forms** (Visceral or Kala Azar-serious form), mucocutaneous and cutaneous (We will discuss the cutaneous form, which has many types, other than vesceral one)

• 1) Old world in Asia, Africa and Europe → long incubation time (MONTHS 2-24)

• Skin lesion(s) on the face or leg: papules become necrotic and then pigmented scars

- papule at site of bite → small nodules→ painLESS ulcer→ crust (pigmented) → leave ugly scars after healing

• Mostly occurred in 6 countries: Afghanistan, Algeria, Brazil, Colombia, Iran (Islamic Republic of) and the Syrian Arab Republic.

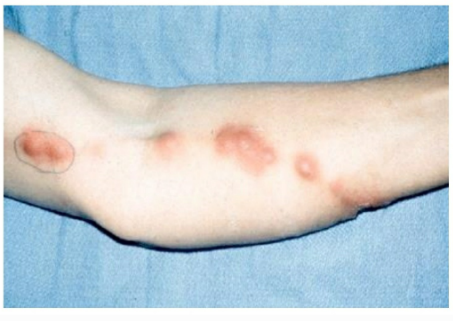
• 2) **Mucocutaeous**: leads to partial or total destruction of mucous membranes of the nose, mouth and throat. occur in Bolivia (the Plurinational State of), Brazil, Ethiopia and Peru.

• May resemble other skin lesions (nodular lymphangitis) caused by waterborne pathogens (such as Sporothrix schenckii, Nocardia brasiliensis, Mycobacterium marinum, Leishmania (Viannia).

• **Nodular lymphangitis**: granulamatous reaction to these pathogens on the path of lymphatics.

• **Diagnosis**:
• Remove crusts and take skin scrape for microbiology
• Biopsy (punch or needle aspirate) to retrieve organism and detect under microscope.
• On CBC (complete blood count) shows reduced cell count (red, white or all)
• culture
• **Treatment**:
• Local heat to area for 2-3 hours a day
• Pentavalent antimonials (group of chemicals given for CL)
• Others: (Liposomal amphotericin B, Oral miltefosine, Pentamidine)
- Given for a minimum of 20 days.

Sporotrichosis, on of many differentials



From WEB: **Sporotrichosis, also known as “rose gardener's disease”, is an infection caused by a fungus called Sporothrix. This fungus lives throughout the world in soil and on plant matter such as sphagnum moss, rose bushes, and hay.
-Spread in lymphatics.

Recall من المحاضرة الأولى:

Bullae: are large fluid filled lesions.

Infections Associated with Bullae

Bullae	
Staphylococcal scalded-skin syndrome	*S. aureus*
Necrotizing fasciitis	*S. pyogenes, Clostridium* spp., mixed aerobes and anaerobes
Gas gangrene	*Clostridium* spp.
Halophilic vibrio	*Vibrio vulnificus*

Staphylococcal scalded-skin syndrome (SSSS):

- in neonates
- **caused by:** a toxin (exfoliatin) from phage group II (bacteria is infected by this phage, acquires the gene to produce the toxin)
• By S. aureus that carry exfoliative toxins A and B.
- to distinguished between **SSSS** & **TEN** (toxic epidermal necrolysis):
Punch biopsy with frozen section is used, since the cleavage plane is the stratum corneum in SSSS and the stratum germinatum in TEN
***TEN occurs primarily in adults (not neonates), is drug-induced, and is associated with a higher mortality rate, IV γ-globulin is a promising treatment for it.
• Due to HEMATOLOGIC (not local) spread of staphylococcal EXOTOXIN.
• That means there is a point of infection somewhere else in the body:
→ Otitis media / Respiratory tract infection.
• Breaks down **desmoglein-1** resulting in **Acantholysis** (breakage of cell to cell adhesions).
• **Symptoms**:
• Preceded by a prodromal illness: URTI or Otitis Media, Pharyngitis, Conjunctivitis
• Then, actue phase: fever + malaise (**loss of fluids**) and red painful skin with bullae formation
• **Signs**:
• Paper thin (peeling) skin
• Large flaccid BLISTERS, more in the flexor creases (see image above)
• Mucous membranes SPARED!
• Positive Nikolsky's sign (detects acantholysis). (Google it💥)
• **Dx**: blood cultures (remember hematologic spread of toxin = bacteremia)
• Skin biopsy will show typical acantholysis
• exotoxin assay
• **treatment** : YOU MUST ADMIT THE CHILD (burn unit or ICU)
• Systemic IV antibiotics (anti MRSA or anti S. aureus)
• Systemic steroids (only if patient doesn't look toxic)
• IV Immunoglobulins and plasmapheresis "removal of plasma" (In severe cases).