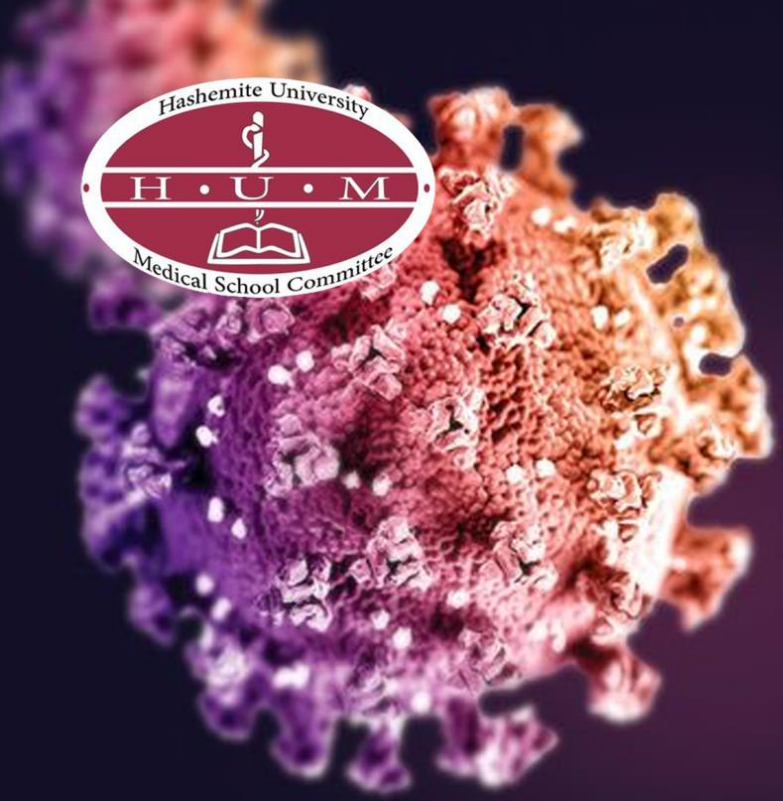


MICROBIOLOGY

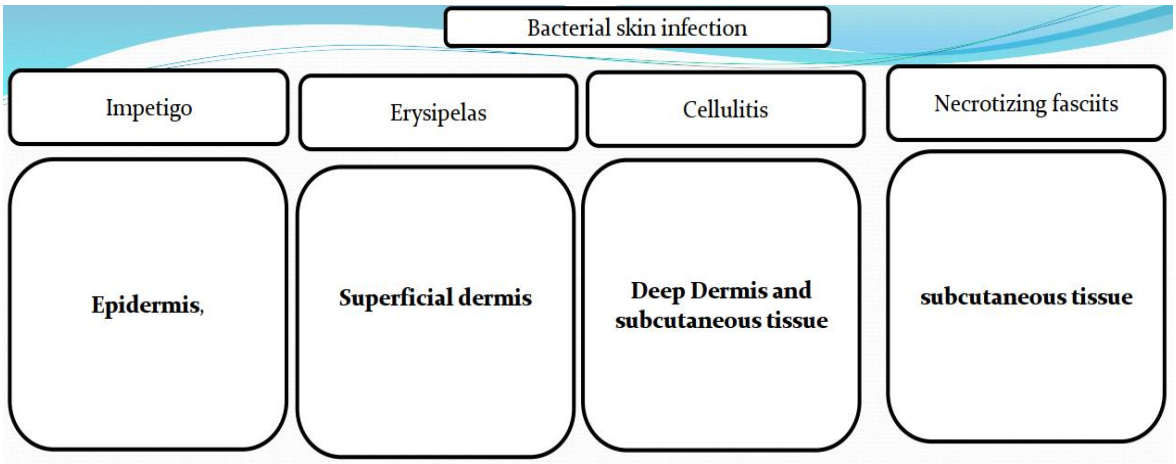
DONE BY : Farah Bdair

Bacterial skin infection

- This lecture will talk about bacterial skin infection:

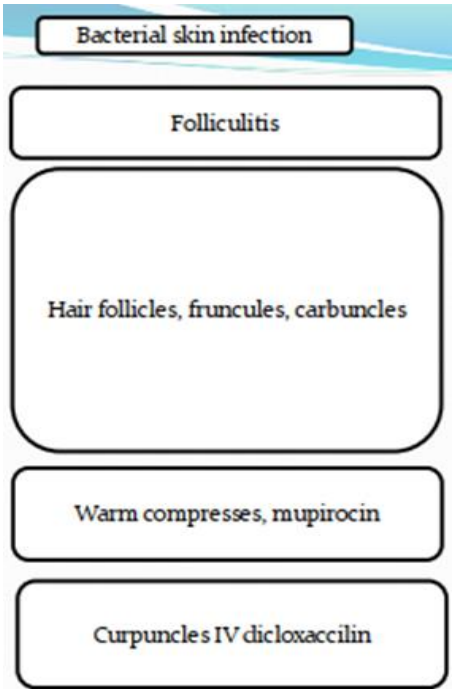
السلايدات باللون **الأسود** .. كلام الدكتور باللون الأزرق

-slide 1:

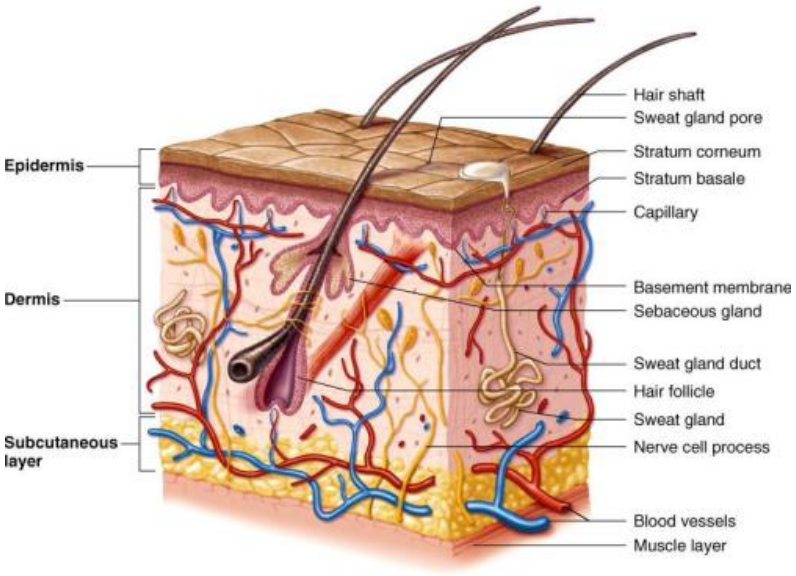


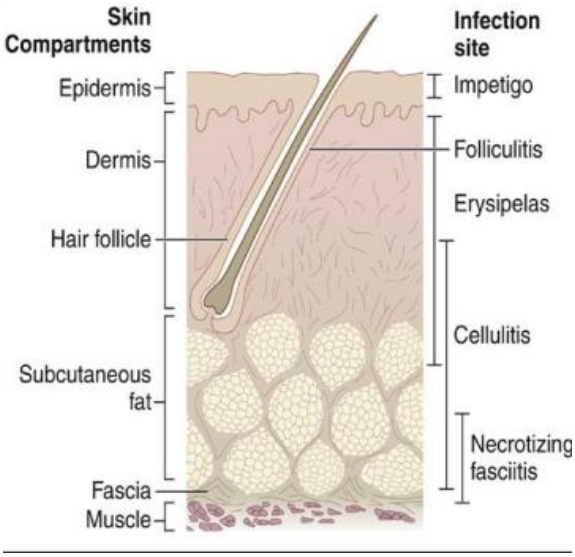
- This slide summarize the 4 main type of bacterial skin infection, in addition to folliculitis (involvement of hair follicles).
- The difference between these types of infection related to which type of skin is get infected, for example:
  - ✓ Impetigo: involve only the epidermal layer.
  - ✓ Necrotizing fasciitis which is very severe and life threatening infection involve the subcutaneous tissue, etc…

-سيتم شرح ما داخل الجداول في السلايدات القادمة.



- The anatomical structure of the skin:



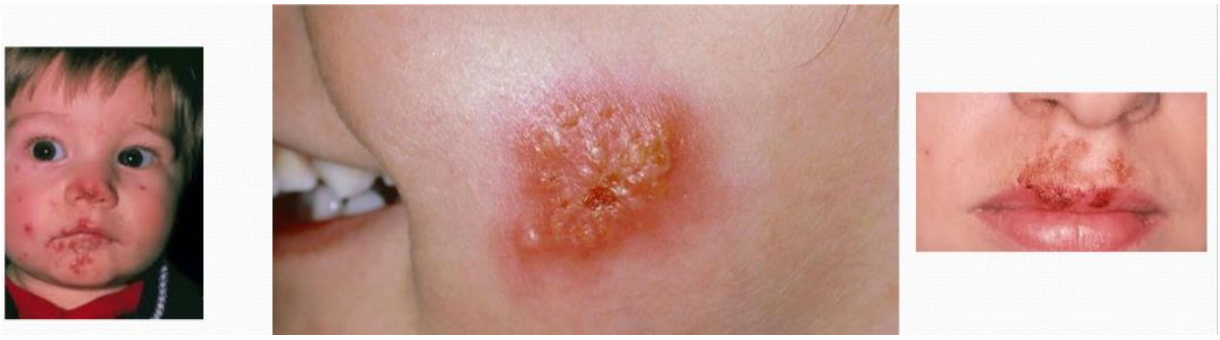


- The skin has 2 main layers: 1- the epidermis on the top. 2- the dermis. Bellow the dermis you have subcutaneous tissue (hypodermis or superficial fascia), and bellow the superficial fascia we have a deep fascia which is a connective tissue layer that surrounds the organ such as the muscle.

- In dermal and hypodermal layers you found the skin artery and vein which project capillary all the way up to the base of the epidermis, we also find different types of sensory nerve fibers which also go all the way up to the base of the epidermis as well.

❖ Impetigo:

- A contagious local superficial infection involving the epidermis.
- Risk factors: children, poor hygiene, overcrowding, skin diseases (e.g dermatitis)
- The classical presentation of impetigo is child presents with lesions crusting lesions around the mouth or in the upper limb.



❖ Classical feature of impetigo:

- A small blisters forming vesicles actually it can also be pustule which are usually thick walled but these can rupture and when they rupture they leave a yellow brown crusting lesion around it.
- An epidermal bacterial blistering skin infection (the definition of impetigo).

❖ The causative agents of impetigo:

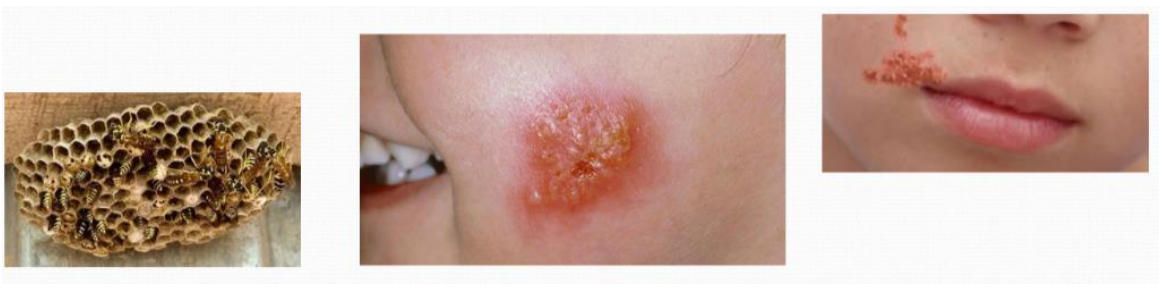
- Staphylococcus aureus (the main causative agent).
- Group A streptococcus.
- Pseudomonas aeruginosa which usually occur in **hot bath**.

❖ Impetigo is Classified into two types:

- Non-bullous impetigo and bullous impetigo
- ➢ Non bullous

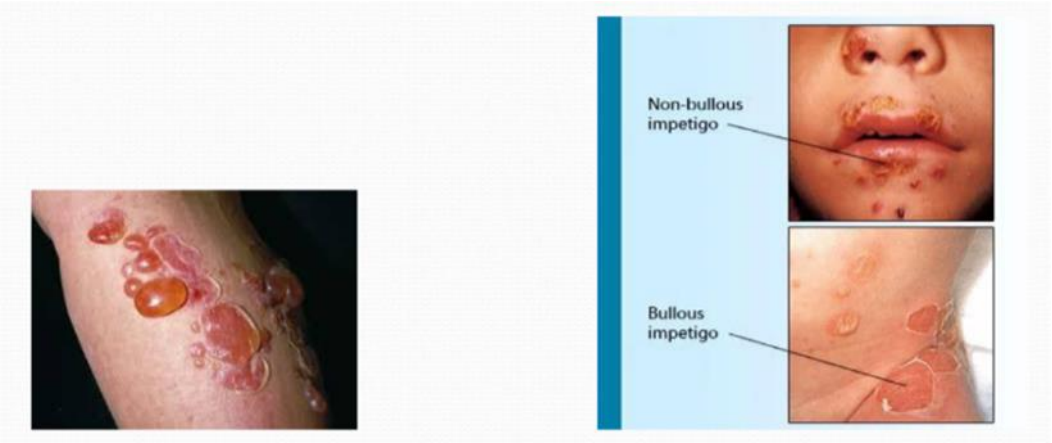
The picture of the child with crusting lesion around his mouth is an example on non-bullous impetigo because it involves small blister.

- The honeycomb crusting appearance (its feature).
- No bullous present (big blisters).
- It is not being painful because it's more in a superficial part of the epidermis (where it doesn't get contact with sensory nerves).



➢ Bullous impetigo:

- Bullous impetigo involves formation of bulla which is essentially a big blister but again this is only confined to the epidermis.
- □ Because it is so big the blister can actually cause irritation because it's very close to the sensory nerve fibers.
- □ The main causative organism of bullous impetigo is Staphylococcus aureus.



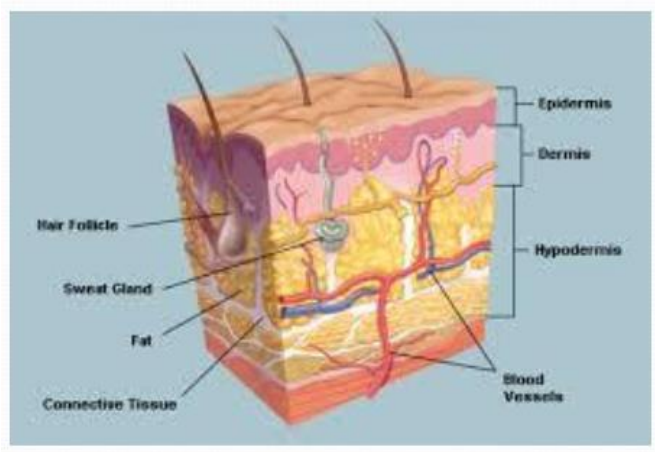
❖ Pathophysiology

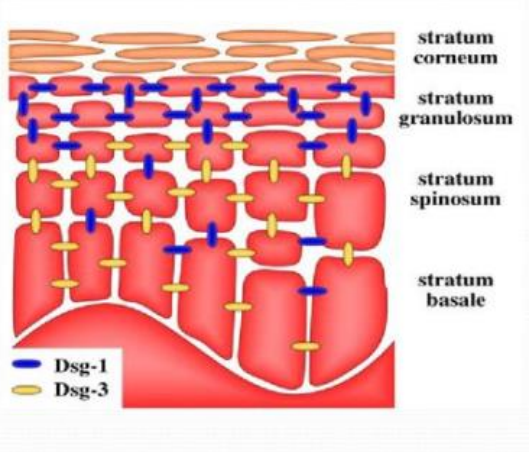
By focusing on bullous impetigo an an example, the main cause of it is S.aureus which is part of our bacterial skin microflora.

- □ Staphylococcus aureus which is your normal microflora.

Now, let's zoom into the epidermis to see how it works.

Remember that basement membrane lies between the epidermis and dermis. Above the basement membrane there is stratum basale layer, its cells continually divide, and new cells constantly push older ones up toward the surface of the skin and lose its blood supply.





- Staphylococcus aureus can actually begin to invade the skin epidermal layer.
- □ because it releases a toxin called exfoliative toxins (ETs) also known as epidermolytic toxins which allow to go deeper into the epidermis.
- □ Disrupting (damaging) the cell to cell adhesion of our skin.

- Normally, your skin cells (keratinocytes) are bounded together by adhesion proteins called desmosomes (in blue and yellow – picture A). In top layers of epidermis (stratum corneum and stratum granulosum) the cells are bounded together by desmoglein 1 (type of desmosome). However, the cells of stratum basale and stratum spinosum are bounded by another type of desmosome called (desmoglein 3).
- What staphylococcus does is that it produces exfoliative toxin, this toxin breaks apart the adhesion protein (desmoglein 1), thus disrupting the cell to cell adhesion allowing the bacteria to infiltrate to invade deeper into the epidermis and cause inflammatory reaction with neutrophils and fluid and formation of bullous and blistering in this region.
- Important information: this exofoliative toxin cannot damage type 2 of desmosomes (desmoglein 3) and that's one of the reasons why impetigo doesn't involve the dermal layer.

- وهذا في الوضع الطبيعي، لكن في بعض الحالات بتكون الS.aureus قوية أو يكون عند الشخص نقص مناعة، هاي ال S.aureus ممكن توصل الdermal layer والsubcutaneous tissue، ممكن تعمللنا complication مثل: erysipelas و cellulitis، ممكن توصل للblood والcapillaries وتعمل sepsis و bacteremia وأيضا ممكن تروح على أي جزء من أجزاء الجسم وتعمل فيه infection.
- وبالتالي الimpetigo تصيب الepidermis فقط الا في بعض الحالات اللي ذكرناها سابقا ممكن أن تصبح أكثر خطراً.

- however if the bacteria is able to go past the epidermal area it can cause some complications and these complications include c**ellulitis** which is infection of the dermal and subcutaneous tissue.
- If the bacteria that causes impetigo can sort of invade further it can also enter the vessels such as the capillaries using **sepsis** and **bacteremia**.
- Other complications of impetigo include **glomerulonephritis** which is particularly caused by Group A streptococcus.
- If bacteria reaches the bones or joins it will cause **Osteomyelitis** or **septic arthritis**.

❖ Diagnosis

- Impetigo is diagnosed clinically (from the history of the patient and physical examination by noticing the swelling, redness and tenderness).
- if complications are suspected investigations can include blood cultures and a blood workup.

يعني اذا لاحظت انه المريض مبين انه very ill وعنده high grade fever وخايف يكون عنده bacteremia ممكن انه نطلب بعض الinvestigations مثل الx-ray ,imaging ,CBC ,blood culture .. ولكن غالبا بتم تشخيصه عن طريق الphysical examination.

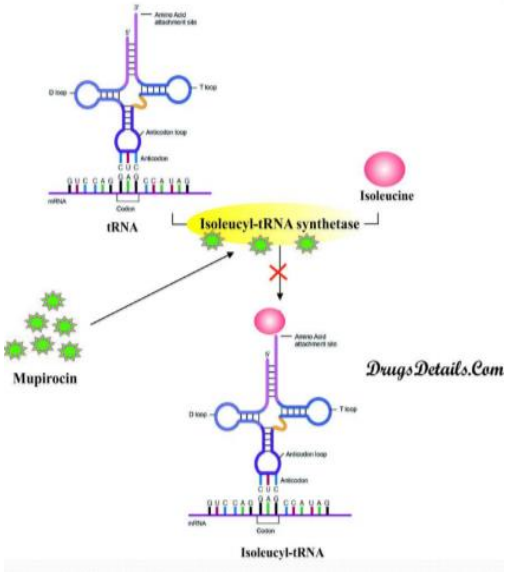
- The management of impetigo is twofold because impetigo is contagious it's important to wash hands with soap.
- If the child goes to school for example it's probably a good idea to keep them at home

- Usually impetigo is self resolving and you don't need any Pharmacological management

فقط نلجأ للphysical examination اذا كان عند المريض low immunity وخفنا من حدوث المضاعفات.
وكونه منعرف انه الmicroorganisms اللي بتسبب الimpetigo هي bacteria (+) gram منستخدم antibiotic يغطيهم.

  - First line is a topical antibiotic called mupirocin (bactroban).
  - Second line are penicillin's such as that dicloxacillin and flucloxacillin. Oral antibiotics.

❖ Mupirocin: mode of action: block protein synthesis



- Ribosomes which are these machinery workshops which make protein.
- They make protein by reading mRNA
- Mupirocin works by reversibly inhibiting bacterial isoleucine tRNA synthetase.

❖ Penicillin : mode of action: damaging of call wall.

- Penicillin's work by binding on to penicillin binding proteins on the cell wall and thus interrupts cell wall synthesis which eventually leads to auto lysis of the bacteria.
- If someone is hypersensitive to penicillins such as they get some form of reaction you use cephalosporin's as an example but interestingly cephalosporin are also being lactams and actually work he same way as penicillin's.

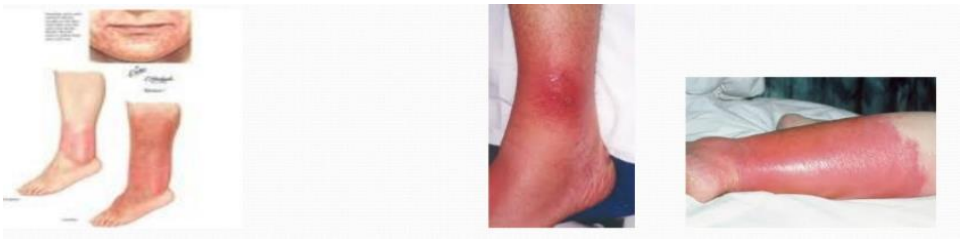
وممكن يكون عنا cross reaction بين الpinicillin والcephalosporin، فممكن نتجه لخيار ثالث اللي هو الclindamycin.

❖ Conclusion

- Impetigo which gain is a contagious skin infection involving the epidermis
- The main causative organisms are staph aureus and Group A streptococcus
- It is usually self resolving but if concern you can use antibiotics

❖ Cellulitis and Erysipelas:

- Cellulitis is a bacterial skin infection of the dermis and the subcutaneous tissue
- The classical presentation of cellulitis is an infection in the lower limb such as near the tibia.
- Erysipelas which is actually known as superficial cellulitis because it is infection of the superficial dermal layer.



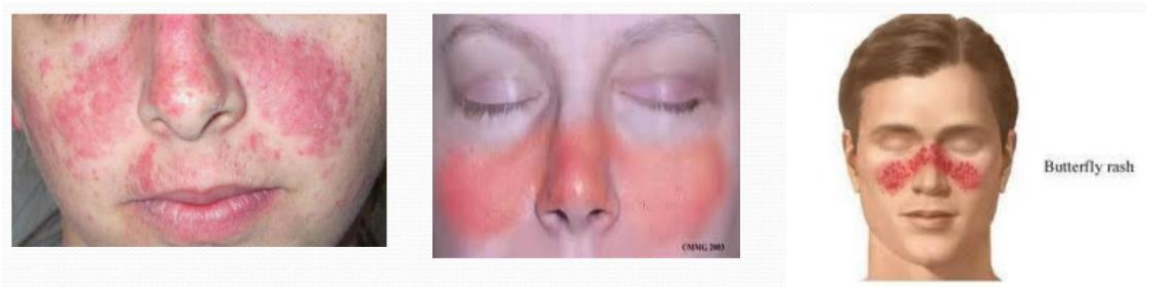
❖ Erysipelas:

Is a good deferential for cellulitis.

- superficial dermal layer infection.
- The main causative agent is usually Group A (beta haemolytic) streptococcus which is also known as strep pyogenes.

❖ Classical presentation:

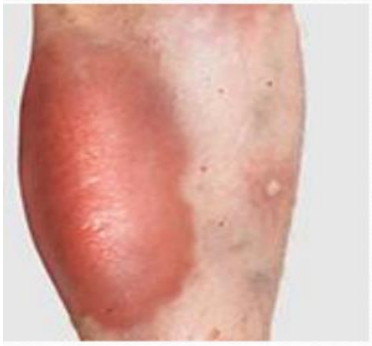
- Patient coming in with sort of a swelling inflamed butterfly appearing rash with systemic features such as fevers chills rigors well as lymphatic involvement.

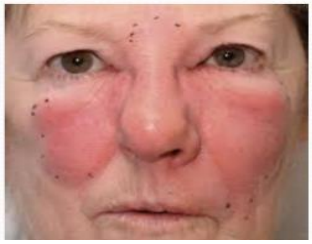


- Erysipelas can also present in the lower limbs such as in around the tibia
- It has the cardinal features of inflammation (signs of inflammation)

Because the erysipelas involves the superficial dermis, the inflammation pushes everything up (it's very important to differentiate it from cellulitis)

- The whole skin area is markedly raised
  - Distinct borders.
  - Inflammation of the superficial dermis which pushes everything up.





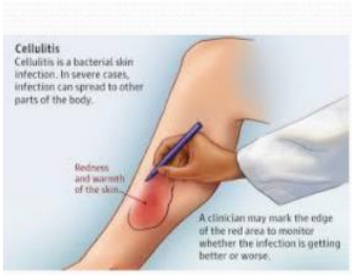
أما بالcellulitis بسبب انها deep in the dermis ما بكون عنا distinct borders.

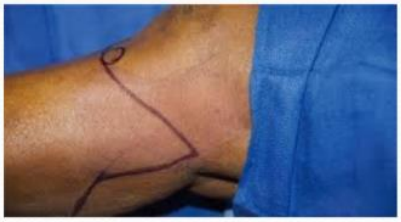
❖ Cellulitis:

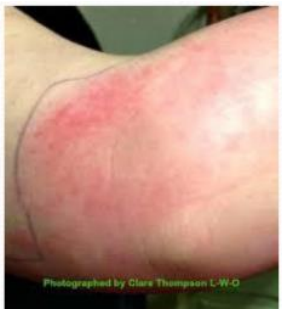
- The classic presentation of cellulitis :is in the lower limbs such as around your tibia
  - Patients with risk factors such as diabetes مهمة جدا

    بمعنى انه الأشخاص اللي عندهم سكري معرضين انه تدخل البكتيريا داخل قدمهم.
- The other common presentation **periorbital cellulitis** with cardinal features of inflammation.
- Cellulitis is bacterial infection involving the deep dermis and the subcutaneous tissue
- The appearance of the skin is only a little bit of swelling
  - it's only slightly raised
- Use a marker and draw around it and date it the next day you can see whether then infection is getting worse and this is also a good way to see if antibiotic treatment is working or not (it is used to follow up the patient to see if he is responding to the treatment or not).

النقاط مهمة.







❖ The main causative agent of cellulitis is

- Staphylococcus species such as staph aureus
- Group A streptococcus

❖ the pathophysiology of both erysipelas and cellulitis

- If Bacteria who possess vernal factors (exfoliate of toxins)
- The patient have their own risk factors

❖ Patient risk factors:

- Having a history of cellulitis, tinea pedis which is basically fungal infection in between toe webs allowing basically axis of bacteria into the dermis area
- Trauma
- Pre-existing skin conditions such as dermatitis
- Lymphedema, obesity diabetes as well as venous insufficiency

❖ Diagnosis:

Based on history and physical examination (inflammatory signs).

- Cellulitis and erysipelas can be difficult to distinguish however both are diagnosed clinically
- Investigations
- Culture of a wound foci (and give the antibiotic according to sensitivity test).
- An x-ray (osteomyelitis) to see if there is an involvement of the joint
- Blood cultures if you suspect sepsis and bacteremia
- Also if you suspect the patient has an abscess contained infection (in leg or tebia) you can order an ultrasound

❖ Management:

- The management of cellulitis and erysipelas is actually the same.
  Because the main agent in both of them is the same, both of them are involving the dermis, and the cellulitis can be happened as a complication of erysipelas.
- Antibiotics (that cover gram (+) bacteria)
- first-line penicillin such as that dicloxacillin and flucloxacillin (we begin in oral medicine)
- if the patient is hypersensitive to penicillinase they have some form of reaction you can give cephalosporin's which are also beta lactams and work the exact same way as penicillin's. (like clindamycin)
- Alternative is drug such as clindamycin
- Clindamycin works by inhibiting protein synthesis thus by disrupting protein synthesis cause stasis of bacterial function

❖ Complications of cellulitis and erysipelas are a lot common ones include sepsis as well as chronic edema (inflammation of the deeper skin layers you're essentially increasing pressure)

necrotizing fasciitis ويعمل subcutaneous layerممكن انه يوصل لل dermal layerموجود بال infectionال

، ممكن يوصل للblood ويعمل bacteremia أو sepsis.

❖ Folliculitis

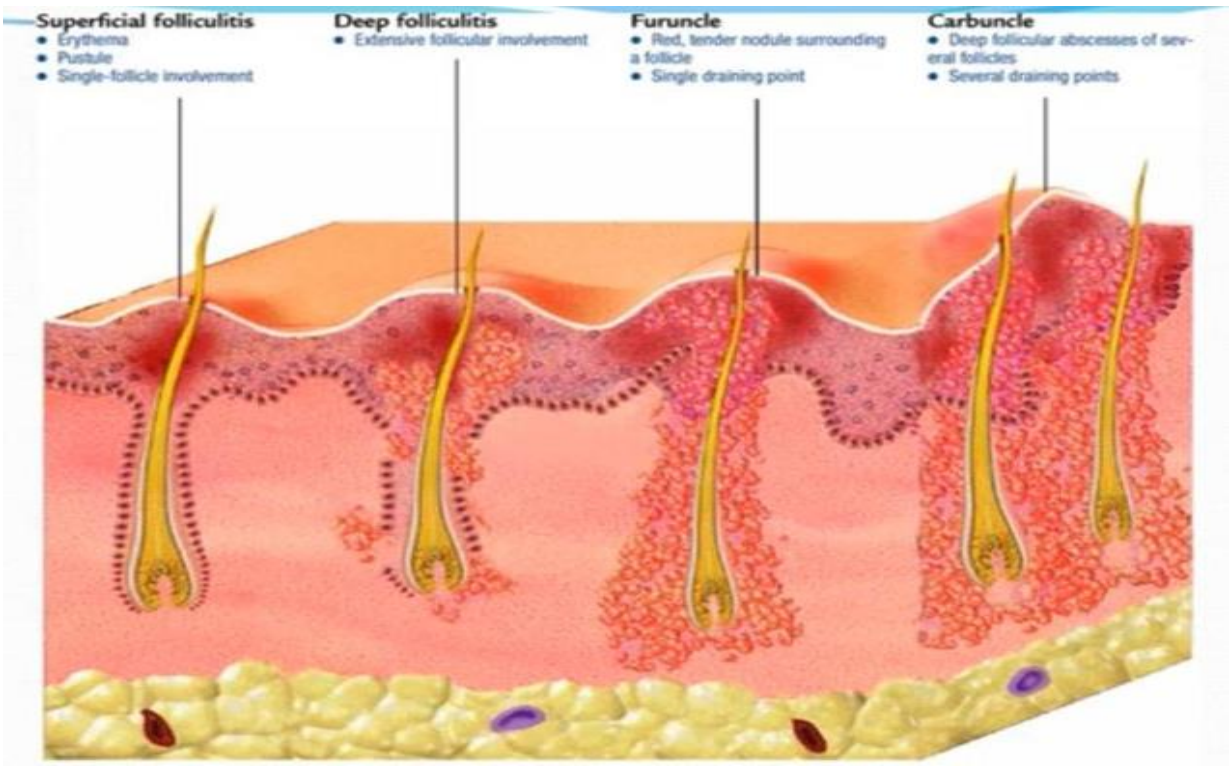
- Bacterial skin infection may involve the epidermal layer, dermal layer, hypodermis along with associated structures such as hair follicles.
- In the hair follicle infection, the single most common organism that involve pus formation is staphylococcus aureus.

- folliculitis is a bacterial infection in the upper portion of hair follicle that causes a papule, pustule, or erosion.

Folliculitis refers generally to infection or simply inflammation of hair follicle which leads to erythematous papule or pustule surrounding this follicle. The upper infection(superficial infection) may have fragile pustule that open easily or deeper inflammatory papule (nodule). وبالتالي الها نوعين

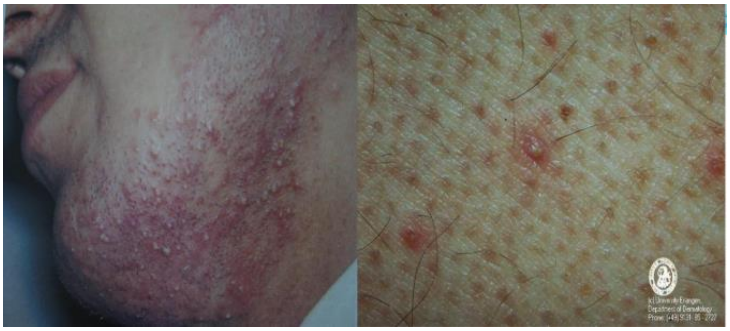
- Commonly caused by S. aureus, P. aeruginosa
  - S.aureus is the most common cause, and it's mostly methicillin resistance.
  - P.aesuginosa infection is mostly happen because of an exposure to adequately chlorinated pool (hot tub pool), so it is called hot tub folliculitis.
  - There is a non-bacterial cause of the folliculitis, fungal infection and viral infection of hair follicles.

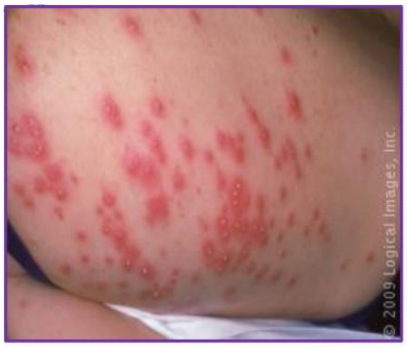
- Folliculitis: is a bacterial infection in the upper portion of a hair follicle that causes a papule, pustule, or erosion.
- Furuncles: affect the entire hair follicle and the adjacent subcutaneous tissue.
- Carbuncles: a group of interconnected furuncles (forming sever infection and severe pus formation).



❖ Signs and Symptoms:

- Folliculitis (mild and simple infection, the patient doesn't go to hospital)
  - Papule or pustules on scalp, arms, or legs.





Hot tub folliculitis

- *Pseudomonas aeruginosa*
- Appears 72 hours after hot tub use
- Resolves in 7-10 days
- Control:
  - Remove swimsuit
  - Shower
  - Clean hot tub

لا تحتاج تدخل treatment

بتروح لحالها ←

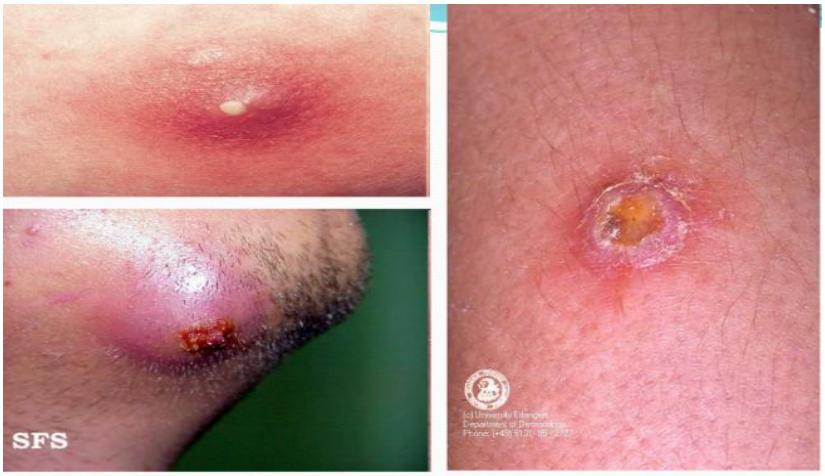
- Furuncles (larger nodules and do irritation of sensory nerve fibers)
  - Painful nodules, commonly on neck, face, axillae, or buttocks.
  - Nodules enlarge for several days, then rupture, discharging pus and necrotic material.
  - After rupture, pain subsides (erythema and edema persist for days or weeks.
  - Patients don't need treatment but we they use topical antibiotic.
- Carbuncles (large part of the skin get infected)
  - Extremely painful, deep abscesses draining through multiple openings onto the skin surface, around several hair follicles.
  - Fever and malaise. Systemic manifestation of infection
  - Need surgical intervention, oral antibiotic, and maybe need IV antibiotic.

✓ Folliculitis:

✓ Furuncle (Boil):

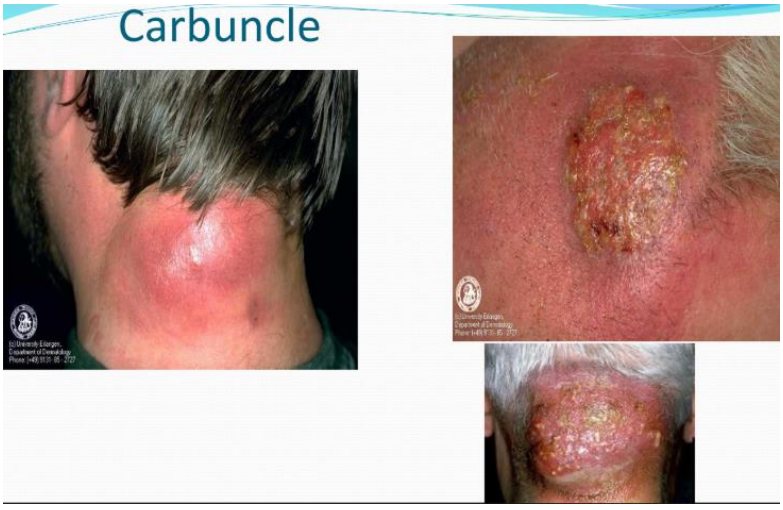
- Acute Superficial skin infection result from S. aureus invasion of hair follicles making small abscess
- It may develop also in a sebaceous or sweat gland.
- 2-4 days, folliculitis, fibrin deposit, site walled off.
- Small, follicular noduler -- Pustule--necrotic--discharge pus

- Painful, mild infection
- Heals with scar
- Site: Neck, Wrist, Waist, Buttocks, Face



✓ Carbuncle: severe infection of hair follicle and adjacent structure

- □ S. aureus
- □ Extensive infection of a group of contagious follicles, dermis and subcutaneous tissues.
- □ Extensive multilocated abscesses (painful).
- □ Middle or old age
- □ Predisposing factors
  - □ Diabetes
  - □ Malnutrition
  - □ Severe generalized dermatoses
  - □ During prolonged steroid therapy
- Painful, hard lump
- □ Suppuration begins after 5-7 days
- □ Pus discharge from multiple follicular orifices
- □ Necrosis of intervening skin
- □ Large deep ulcer



Severe and large

❖ Treatment:

مختلفة بكل واحد حسب الclinical presentation، الdiagnosis.

- If it was simple folliculitis:
  - Through cleaning of the infected area with antibacterial soap and water several times per day.
  - Warm, wet compresses to promote vasodilation and drainage.
  - But if you are thinking that the patient is in risk to get more infections you can start using topical antibiotics, such as mupirocin.
- Specific treatments:
  - Extensive folliculitis (in multiple folliculitis): systemic antibiotics, such as a cephalosporins or dicloxacillin
  - Furuncles:
    - Warm, wet, compresses.
    - Incision and drainage. (محتمل)
    - Systemic antibiotic therapy.
  - Carbuncles: incision and drainage (أكيد) / systemic antibiotics.

"استمر بالمُحاولَة ، تأتي الأحلامُ دوماً على طبَق مِن تَعب ،! 🌱❤"

The end