MSS | lec. 4 micro sum.
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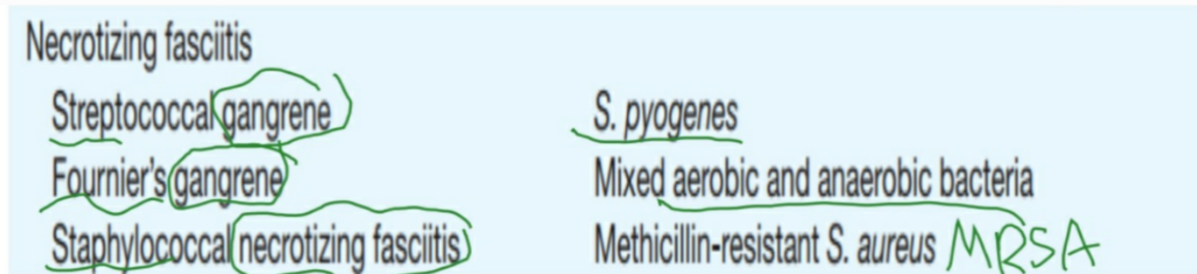
Necrotizing fasciitis: التهاب اللفافة الناخر

Also called (flesh-eating disease/متلازمة البكتيريا آكلة اللحم)



-induce **bulla** formation.

• between the fascia and deep subcutaneous tissue.

Fibrous bands in the head prevents spread of infection, These bands are not in the extremities (thus extremities are more susceptible).

Symptoms (occur in order):

1. Pain/tenderness.
2. fever.
3. Later on: Swelling (severe).
4. Dark red induration (indicates hemorrhage and early necrosis)
5. BULLAE, filled with blueish or purple fluid.
6. Thrombosis of dermal blood vessels (The affected area becomes anesthetic as a result of small vessel thrombosis and destruction of superficial nerves).
7. Extension to deep fascia with rapid spread.
8. Most progressed symptoms: toxicity, shock and multi organ failures.

causes:

A) Polymicrobial (**Type I** necrotizing fasciitis involves at least one anaerobic species (Bacteroides or Peptostreptococcus spp.)), as well as one or more facultative anaerobic species (e.g. non-GAS, E. coli, Enterobacter, Klebsiella, Proteus spp.)

• Usually a mix of aerobes and anaerobic bacteria (clostridium perfringens) that begins with a breachفتحة in the integrity of a mucous

membrane barrier.

- Symptome:

• 1 -Break in GI or Genitourinary mucosa, typically on trunk and extremities
• 2- Fournier's Gangrene (in genitalia/perineal area)
• 3- Mixed infection (in DM, PVD(Peripheral vascular disease), immunecompromised).
• Leakage into the perineal area results in a syndrome called Fournier's gangrene, a form of necrotizing fasciitis affects the male genitals and is usually polymicrobial characterized by massive swelling of the scrotum and penis with extension into the perineum or the abdominal wall and legs.

B) **Type II** necrotizing fasciitis is usually caused by GAS alone or with other species (e.g. S. aureus). (***S. pyogenes +- S. aureus***)

• MRSA that produce the **Panton Valentine leukocidin (PVL) toxin** cause necrotizing fasciitis (also necrotizing pneumonia).

- **Craniofacial necrotizing fasciitis** is usually associated with trauma and caused by GAS (skin).

- Symptoms:

1) Usually following trauma in drug abusers (**skin popping**تفرقع الجلد).
2) it progresses to **skin contusions**كدمات due to seeding by transient bactermia
3) **Gas production** if mixed infections occurs (you need anaerobes)
4) **Severe toxicity** and **renal impairments** → **shock**
5) **Myositis** (destruction of muscle tissue markedly increases CPK)

- Other predisposing factors: **peripheral vascular disease, diabetes mellitus, surgery, and penetrating injury to the abdomen.**

- In the newborn, necrotizing fasciitis may complicate **omphalitis** التهاب السرة and spread to involve the abdominal wall, flanks, and chest wall.

Diagnosis:
Clinical + surgical:
a) Altered mental status (systemic involvement)
b) redness/swelling/pain.
Bullae Pain is typically exaggerated out of exam
Tenderness is outside the red erythematous borders
a) Fever.
b) Low BP
c) Crepitationفرقعة (feeling of air pockets under skin upon examination).

Treatment: empiric
• 3 drug combo/ 2 drug combo/1 drug (each +MRSA coverage)
3 drug combo (mnemonic: CCAA)
1- **Clindamycin** (anaerobic coverage and inhibits ribosomal production of toxins)
2- **Ampicillin**-sulbactam *or* Piperacillin-tazobactam→ **Antipsuedomonal** (G +ve coverage)
3- **Ciprofloxacin** (G-ve coverage)
2 drug combo (mnemonic: CCM)
Metronidazole or **Clindamycin** (anaerobic coverage) +
Cefotaxime (covers G+ and G bacteria)
1 drug combo
Carbapenem/**Imi**penem, **mero**penem, **erta**penem.
•**Van**comycin or **Line**zolid (MRSA coverage-added to any chosen empiric regimen)
•**Doxy**cycline (for Hemorrhagic bullae, may indicate presence of vibrio vulnificus).
- Surgical debridement
1-confirm the diagnosis 2-mainstay of therapy 3-Reducing compartment pressure in extremities.
•**Penicillin**, **rif**ampin, **clinda**mycin or **azir**thromycin (Prophylaxis).

Gas gangrene (Clostridium infection)

- Gas production due to **anaerobic** bacteria.
- C. perfringens is the predominant cause (its pathological effects are mediated by α and λ toxins)
- **in** contaminated DEEP wounds -no oxygen- usually following muscle injury.

progress: fasciitis → toxemia → organ failure

Alpha toxin:

- phospholipase C (PLC) with sphingomyelinase and lectinase activity.
- responsible for intravascular hemolysis, platelet aggregation, and capillary damage → loss of blood supply = **loss of oxygen**

-It **stops leukocytes and oxygen** from getting to the site of infection

-It **helps immune evasion** by interfering in neutrophil migration to the infected tissue, minimizing the number of mature cells in the bone marrow, and **causing the accumulation of neutrophils in adjacent vessels.**

- **Spontaneous or non-traumatic gas gangrene** may occur in the absence of an obvious wound, This form is usually caused by C. septicum and associated with intestinal abnormalities: colonic cancer, diverticulitis, bowel infarction, necrotizing enterocolitis.

Clinical features:

- 2-3 days incubation period
- then, acute onset of excruciating pain and signs of shock (fever, tachycardia, hypotension, jaundice, renal failure).
- Local edema and tenderness

Rapid progression, and death may occur within hours

Diagnosis:

-clinical, may be confirmed by Gram stain of the wound or aspirate.

-Liquid anaerobic cultures.

-Plain radiographs may show gas in the affected tissues.

Management: Emergency surgical exploration and debridement

piperacillin–tazobactam + vancomycin (if risk of MRSA)

- Definitive treatment for clostridial myonecrosis is with penicillin and clindamycin

Hyperbaric oxygen therapy is not recommended المعالجة بالأكسجين عالي الضغط

Cellulitis

Cellulitis	*Staphylococcus* spp., *Streptococcus* spp., various other bacteria

- acute inflammatory condition of the skin
- Has the inflammation signs: localized pain erythema, swelling, and heat.

caused by S. aureus and S. pyogenes (NF) *MRSA is appear rapidly. or other exogenous bacteria like Enterobacteriaceae, L. pneumophila, A. hydrophila, V. vulnificus, and C. neoformans.

Note: Cellulitis due to S. pyogenes is more rapidly spreading, & associate with **lymphangitis** and fever.

-Also recurrent streptococcal cellulitis is seen among patients with chronic lymphedema resulting from elephantiasis, lymph node dissection, or Milroy's disease, In both cases is due to poor drainage of limb, This is all due to the fact that streptococci use the lymphatic system in their spread.

- H. influenzae typically causes **periorbital cellulitis in children** in association with sinusitis, otitis media, or epiglottitis.

*- Cellulitis and abscesses associated with dog bites and human bites also contain a variety of anaerobic organisms, including Fusobacterium, Bacteroides, aerobic and anaerobic streptococci, and Eikenella corrodens.

- Cats bites, dog bites → Pasteurella multocida and Staphylococcus intermedius and Capnocytophaga canimorsus

Pasteurella is resistant to dicloxacillin and nafcillin, however, it is sensitive to all other β‐lactams, quinolones, tetracycline, and erythromycin.

Thus, for animal or human bites the treatment is **Ampicillin/clavulanate, ampicillin/sulbactam, and cefoxitin**

Aeromonas hydrophila → aggressive cellulitis in injuries sustained in freshwater (lakes, rivers, and streams).

Treatment according to known sensitivity of this organism→ **fluoroquinolones, chloramphenicol, trimethoprim‐sulfamethoxazole, and third-generation cephalosporins**

otherexogenous causes: Physical activities - trauma - water contact -animal, insect, or human bites -immunosuppression.

***Recurrence is seen in patients with eosinophilia**

Recurrent streptococcal cellulitis of the lower extremities may be caused by

organisms of group A, C, or G in association with chronic venous stasis or with saphenous venectomy for coronary artery bypass surgery.

Clinical features:

-spreading ,erythematous, hot, tender lesion.

-accompanied by systemic symptoms.

-ulcer+cellulitis is the common case

Diagnosis: - clinically.

- bacterial numbers are low and local to tissue, so culturing isn't effective, the inflammatory effect is exaggerated due to **toxins**.
- Can do culture if there is drainage or a site of entry is seen

Treatment: empiric

- IV **flu**cloxacillin *or* **clinda**mycin.
- **Van**comycin, teico**plan**in, **line**zolid, or **dapto**mycin are for MRSA cellulitis.

The affected limb should be immobilized and elevated.

P. aeruginosa

Causes 3 types of infections in MSS:

1→ **Ecthyma gangrenosum** in neutropenic patients حمى نقص العدلات، قلة الخلايا المتعادلة

2→ **Hot-tub folliculitis** التهاب جريبات حوض الاستحمام الساخن

3→ **Cellulitis** following penetrating injury (usually stepping on a nail)

- seen in hospital

Treatment: surgical inspection and drainage/debridement

-Aminoglycoside

third-generation cephalosporin: (**cef**tazidime, **cef**operazone, or **cef**otaxime)

-semisynthetic penicillin (ticarcillin, mezlocillin, or piperacillin), or a fluoroquinolone (not in pediatric patient)

ANTIBACTERIAL AGENTS

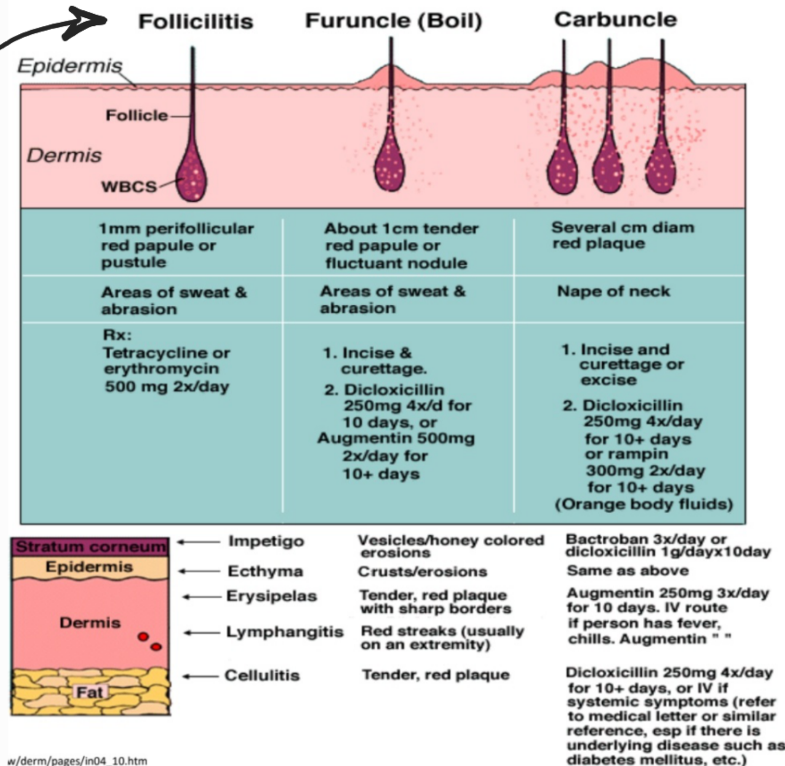
Table 5.1 Principal types of antibacterial agent (other than agents used exclusively in mycobacterial infection)

Agent	Site of action	Usual activity[a] against: Staphylococci	Streptococci	Enterobacteria	*Pseudomonas aeruginosa*	*Mycobacterium tuberculosis*	Anaerobes
Penicillins	Cell wall	+R	+	V	V	–	+[b]
Cephalosporins	Cell wall	+	+	+	V	–	+[b]
Other β-lactam agents	Cell wall	V	V	+	V	–	V
Glycopeptides	Cell wall	+	+	–	–	–	+[c]
Tetracyclines	Ribosome	+R	+R	+R	–	–	+R
Chloramphenicol	Ribosome	+	+	+	–	–	–
Aminoglycosides	Ribosome	+	–	+	V	V	–
Macrolides	Ribosome	+	+	–	–	–	+
Lincosamides	Ribosome	+	+	–	–	–	+
Fusidic acid	Ribosome	+	+	–	–	+	+
Oxazolidinones	Ribosome	+	+	–	–	–	–
Streptogramins	Ribosome	+	+[d]	–	–	–	
Rifamycins	RNA synthesis	+	+	+	–	+	+
Sulphonamides	Folate metabolism	+R	+R	+R	–	–	–
Diaminopyrimidines	Folate metabolism	+	+	+R	–	–	–
Quinolones	DNA synthesis	V	V	+	V	V	–
Nitrofurans	DNA synthesis	–	–	+	–	–	+
Nitroimidazoles	DNA synthesis	–	–	–	–	–	+

[a]Usual spectrum of intrinsic activity
[b]Poor activity against anaerobes of the *Bacteroides fragilis* group.
[c]Poor activity against most Gram-negative anaerobes.
[d]Poor activity against *Enterococcus faecalis*.
+, active; –, inactive; V, variable activity among different agents of the group. +R indicates that acquired resistance is very common.

Folliculitis التهاب جريبي/التهاب بصيلات الشعر

Folliculitis	
Furunculosis	*S. aureus*
Hot-tub folliculitis	*Pseudomonas aeruginosa*
Swimmer's itch	*Schistosoma* spp.
Acne vulgaris	*Propionibacterium acnes*



• A **superficial** infection of the hair follicles and apocrine structures.

- In water that is not sufficiently Chlorinated and maintained.

• **Causative organisms**:

S. aureus (commonest),

P. aeruginosa ('hot tub' folliculitis),

Enterobacteriaceae (complication of acne),

Candida spp.,

M. furfur (in patients taking corticosteroids).

• Eosinophilic pustular folliculitis occurs in AIDS patients.

• **Clinically**: lesions consist of small, erythematous, pruritic papules, often with a central pustule.

• **Treatment:** oral flucloxacillin. (mnemonic: Foll Flu)

- Usually self limited

Cutaneous abscesses

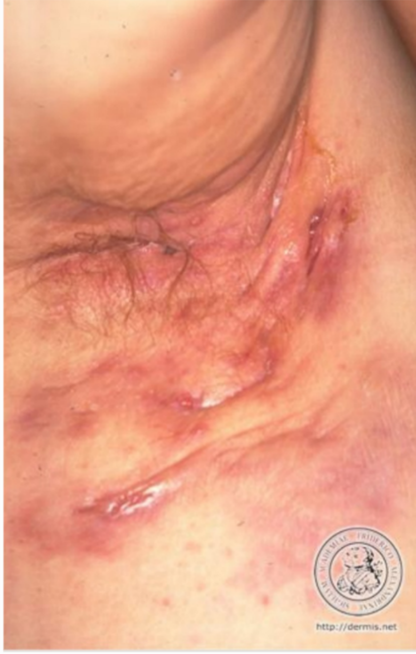
• Collections of pus within the dermis and deeper skin structures.
• **cause**: polymicrobial containing skin/mucous membrane flora;
S. aureus.
• **Clinical features**—painful, tender, fluctuantمتقلب nodules, usually with an overlying **pustule** and surrounded by a rim of erythematous swelling
raised lesion,
White head
*Hair follicle might Be port of entry.
• **Treatment** is I&D → Antibiotics are rarely necessary (except in extensive infection or systemic toxicity, or immunocompromised).

Furuncles الدمل and carbuncles الجمرة

A furuncle (boil): a deep inflammatory nodule
- develops from preceding **folliculitis**.
• in areas of the hairy skin, e.g. face, neck, axillae, and buttocks.
A carbuncle: a larger, deeper lesion made of multiple abscesses extending into the subcutaneous fat.
• Usually occur at the nape of the neck, on the back, or on the thighs.
• Outbreaks of furunculosis is caused by MSSA and MRSA.
Treatment of furnucles
• moist heat, to promotes localization and spontaneous drainage.
• Large lesions require surgical drainage.
• Systemic antibiotics are indicated→ 1-fever, 2-cellulitis 3-lesions are located near the nose or lip.
• Outbreaks control with **chlor**hexidine soaps.
- **Sebaceous glands** that empty into the hair follicle **maybe blocked and cause a swelling similar to an abscess (sebaceous cyst).**
• **Infection of sweat glands (hidradenitis suppurativa) can also mimic infection of hair follicles, particularly in the axillae.**
• in acne vulgaris → Chronic folliculitis, because constituents of the normal flora (e.g., Propionibacterium acnes) may play a role.

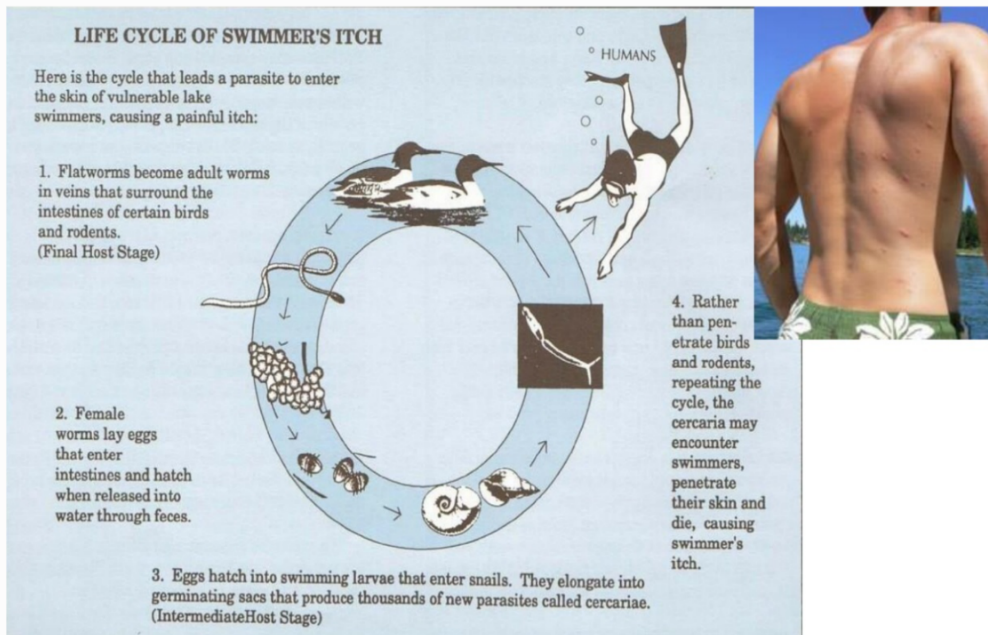
Hidradenitis Suppurativa التهاب الغدد العرقية القيحي

in sweaty areas Where skin folds (axilla, Buttocks, breasts, inner thighs).



Swimmer's itch

- when a skin surface is exposed to water infested with freshwater avian schistosomes.
- Warm water temperatures and alkaline pH are suitable for **mollusks**رخويات that are intermediate hosts between birds and humans.
- Freeswimming schistosomal cercariae readily penetrate human hair follicles or pores, but quickly die and elicit a briskحاد allergic reaction, causing intense itching and erythema.



Erysipelas: الحمرة

Cause: S. pyogenes

- characterized by an abrupt onset of fieryناري-red **swelling**

In face or extremities.

• **features:**

1. well-defined indurated margins, particularly along the nasolabial fold
2. rapid progression
3. intense pain.
4. **Flaccid bullae** may develop.
5. swelling, fever, pain, and the intense red color diminish.
6. Desquamation تقشر الجلد of the involved skin.

• **Treatment**: **pen**icillin (**flu**cloxacillin, **clinda**mycin).

دعواتكم