Musculoskeletal System

Microbiology

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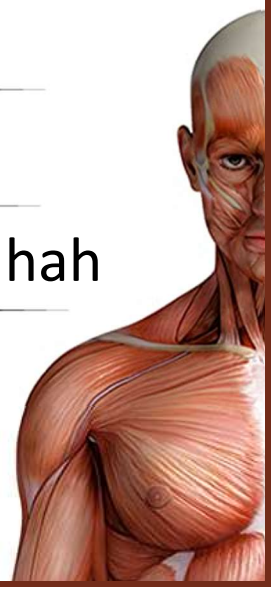

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Necrotizing Fasciitis

Necrotizing fasciitis (flesh-eating bacteria as it has been called). It's usually caused by the streptococci. There is also a staphylococcal variant caused by usually MRSA. Why MRSA and not sensitive staph? Since a resistant staph would have accumulated more virulent and antimicrobial factors in its quest of retaining more genetic material. Necrotizing pneumonia, for example, is caused only by MRSA.

Etiology:

- Streptococcal gangrene: S. pyogenes.

- Fournier's gangrene: Mixed infection between aerobic and anaerobic bacteria, we will recall that you need facultative anaerobes the ones that can utilize oxygen and live anaerobically (Like the Staph and Strep). They will destroy the tissue and once the tissue has become devitalized (is no longer perfused by blood). So, no oxygen will reach this tissue, the tissue will become necrotic. Therefore, this tissue will be an ideal for anaerobic growth (because there is no oxygen reaching the tissue from the blood). This can happen e.g., in a tooth abscess or biofilm forming bacteria where they can make things ideal for anaerobes in the gut. Generally, it can happen anywhere else, and the skin is not an exception.

DR018 Sheet:

Facultative anaerobes are the first to invade the area to prime it for stronger and prepared soldiers (obligate anaerobes). Because of the difference in their oxygen requirements, facultative anaerobes will consume all the oxygen in the area for obligate anaerobic bacteria to survive and destruct the area.

- Staphylococcal necrotizing fasciitis: Methicillin-resistant S. aureus.

You must be worried if you suspect necrotizing fasciitis, why?

Because it's **RAPIDLY** progressing infection in the area between the fascia and deep subcutaneous tissue.

Fibrous bands in some areas prevents spread of infection. **These bands are present in the head and maybe in the trunk but not in the extremities (thus extremities are more susceptible).**

So, you will find the spread of necrotizing fasciitis to be more common in the extremities.

– >50% in extremities.

– 20% in perineum or buttocks (especially seen in diabetics (DM) or alcoholics).

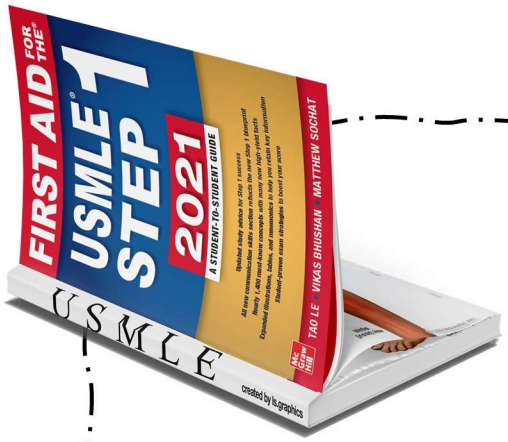
– 18% in trunk.

– 9% head and neck.

- Necrotizing fasciitis (GAS - Group A Strep) and gas gangrene (anaerobic clostridia infection) also induce **bulla formation**.

Let us recall what is a bullous? It is a fluid filled lesion.

- In the USA, the estimated incidence of invasive GAS infection is 3.5 cases per 100,000 persons—necrotizing infections account for 6% of these.



Necrotizing fasciitis:

Deeper tissue injury, usually from anaerobic bacteria or S pyogenes. Pain may be out of proportion to exam findings. Results in crepitus from methane and CO2 production. "Flesh-eating bacteria."
Surgical emergency.

Causes bullae and skin necrosis → violaceous color of bullae, surrounding skin.

- Many risk factors increase the risk:

Risk factors associated with Necrotizing Fasciitis:

1- ***Malnutrition:***
   A. Hypoalbuminemia: A malnourished patient is more likely to not have the ability to produce enough immunoglobulins to combat infections. So, immunity will decline because the body isn't synthesizing enough proteins.
   B. Alcoholism and liver cirrhosis: These will reduce the liver's ability to produce acute phase reactants and all these innate and adaptive immunity proteins that you need in your defense as well as perhaps even maybe energy.

2- ***Patient conditions:***
   - >50-year-old: declined immunity, an older person is more likely to have reduced circulation (reduced blood perfusion).
   - Obesity: an obese person is likely to have again poor blood supply to the extremities or to the extremes of the skin. (Skin will not be perfused as in a person who is in a healthy weight)

3- ***Immune compromised:***
   A- Cancer.
   B- Steroid therapy.

4- ***Poor blood supply:***
   A- Heart disease.
   B- PVD (peripheral vascular disease) نقص التروية in limbs.
   *remember that they are the most common sites for necrotizing fasciitis.
   C- DM: Diabetes itself as a syndrome reduces immunity and the arteries that supply nerves and arteries. So, the patient will have neuropathy. Hence, all of these will reduce the signals or the ability for the immune system to fight infections. A diabetic person may not sense a penetrating trauma or a pinprick or anything that may introduce bacteria and then may not even feel pain, or redness, or hotness. So, might have an infection that can spread quicker before being treated or addressed.

5- ***Skin trauma in last 3 months:***

* The patients who suffered from these cases will never have after healing a complete sterility in that area, that area will continue to harbor small number of bacteria and because it was introduced in the deeper layers of the skin, then the bacteria if it happens to have the opportunity to cause an infection, it will cause it. E.g., Group A Strep it will be a risk factor for necrotizing fasciitis.

A- Burns
B- Penetrating trauma
C- IV drugs
D- Surgery

6- ***Breaks in the mucosa of the GI or GU tracts (where anaerobes gain access):***

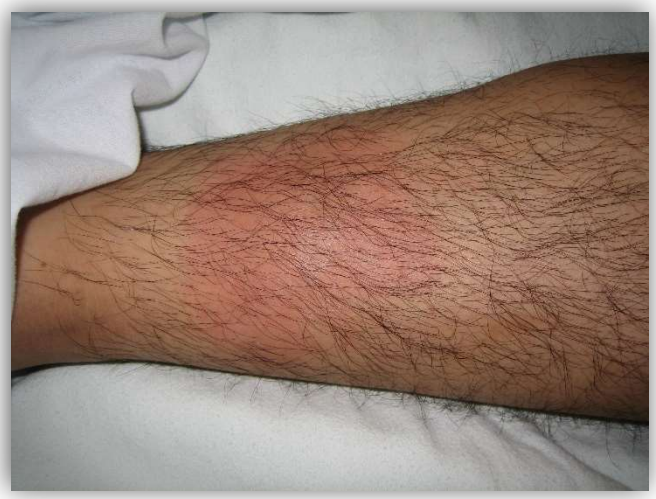
A - Colon cancer (can cause ulcer)
B – Diverticulae
C - Hemorrhoids of fissures (A person who had hemorrhoids or fissures will introduce a bacterium in the anus, that will have a free access into the skin around the anus. Hence, we may have the gangrene that may happen around the anus or the buttocks.)
E- Urethral tears

Summary of the risk factors associated with necrotizing fasciitis.

Malnutrition	*Patient conditions*	*Immune compromised*	*Poor blood supply*	*Skin trauma in last 3 months*	*Breaks in mucosa of GI or GU tracts (anaerobes)*
-Hypo-albuminemia -Alcoholism -Cirrhosis	- >50 Year olds -Obesity	-Cancer -Steroid therapy	-Heart disease -PVD -DM	-Burns -penetrating trauma -IV drugs -surgery	-colon cancer -diverticulae hemorrhoids or fissues -urethral tears

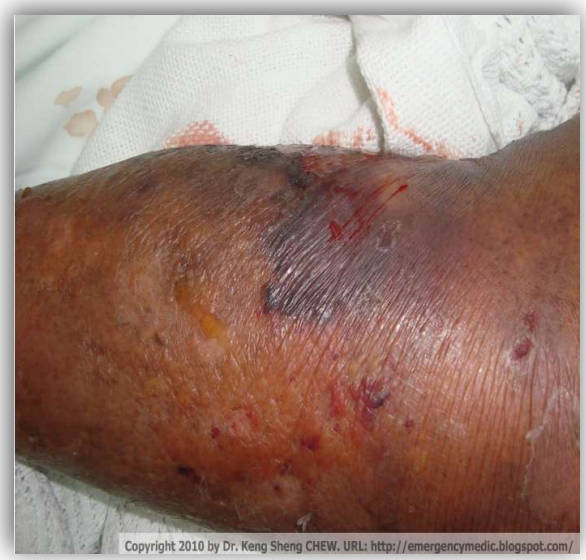
SIGNS & SYMPTOMS (occur in order) :

1- **Pain/tenderness**: In the beginning, we don't have a swelling, or redness or hotness.



* You are under the nerves that supply pain and what's going to happen is that the bacteria are growing and causing tissue destruction until they reach the top layers which harbor the pain receptors at that point you start having pain. Once we reach this point, it is almost too late because the infection has been growing for a while before it has reached the pain receptors.

2- **Unexplained fever** (Early diagnosis may be difficult when pain or unexplained fever is the only presenting manifestation, remember infection is deep, might not present with pain yet). Why this fever occurs? Because now it has caused enough destruction to gain access into the systemic circulation. Teichoic acid and LPS will be released as endotoxins and then cause fever.

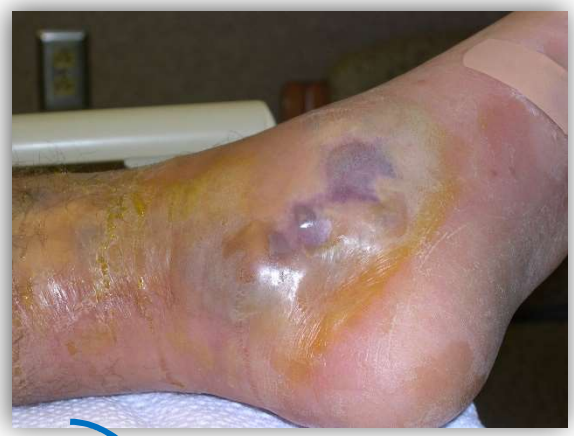


Dark red induration /swelling

3- Later: **Swelling** (infection at this point is severe).

4- Dark red induration (indicates he hemorrhage and early necrosis). it's being hard as in congealing.

Progressing to bullae /thrombosis

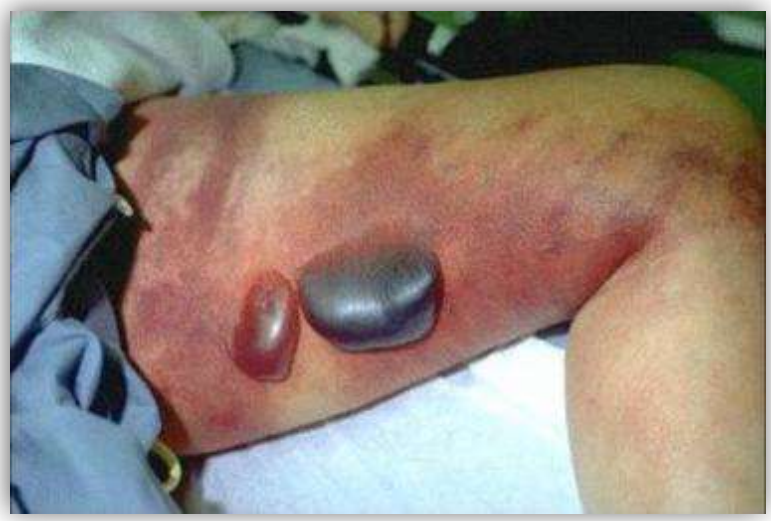


5- **BULLAE**, filled with blueish or purple fluid, which is basically all the dead cells and the debris that is coming out of the deep fascia. At this point, you are beyond necrosis.

6- **Thrombosis of dermal blood vessels** (The affected area becomes anesthetic because of small vessel thrombosis and destruction of superficial nerves)

What you can see in the figure are the fluids that can leak and then harden or crust over.

Last Stage/ **More bullae formation**



7- Extension to deep fascia with rapid spread.

Now it will gain access and start spreading, it can spread to the muscle, the bone, superficially but at this point the easiest path with least resistance is to keep spreading horizontally.

Most progressed symptoms : Toxicity , Shock and Multi organ failures (has progressed beyond local infection site). All these obviously carry a high risk for mortality. However, if the patient survives, he will have lasting damage in the limb or the area.

Microbiology causes:

It can be either: A. Polymicrobial (and its type A) or B. Monomicrobial (Which is type B)!

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.).

What connects all these bugs? All have representatives in the enteric tract!

Notice that all of them are enteric bacteria!

The connection here is from the lumen (inside) to the skin to the deep fascia.

• Usually, a mix of aerobes and anaerobic bacteria (clostridium perfringens)

• 1 -Break in Gastrointestinal or Genitourinary mucosa, typically on trunk and extremities.

• 2- Fournier's Gangrene (it happens in genitalia/perineal area).

• 3- Mixed infection usually have comorbid states (DM, PVD, immune-compromised) .

• 21% mortality rate with optimal treatment.

B) **Type II necrotizing fasciitis** is usually caused by GAS alone or in combination with other species (Skin flora) (e.g. S. aureus). Group A , Beta hemolytic strep (GAS), S. pyogenes +- S. aureus (connection here is these are skin representatives)

• Strains of MRSA that produce the Panton Valentine leukocidin (PVL) toxin (which has been recorded with necrotizing pneumonia). Also, have been reported to cause necrotizing fasciitis.

- The connection from the top rather than the bottom. E.g., from the air to the skin.

1) Usually following trauma in otherwise healthy individual or IV drug abusers (skin popping), they keep injecting themselves on the same area over and over until it becomes indurated (common case), not like the previous picture of co-morbid patient with gastric, genitourinary tract disruption.

2) Fasciitis progresses to skin contusions due to seeding by transient bacteremia (That means in other areas).

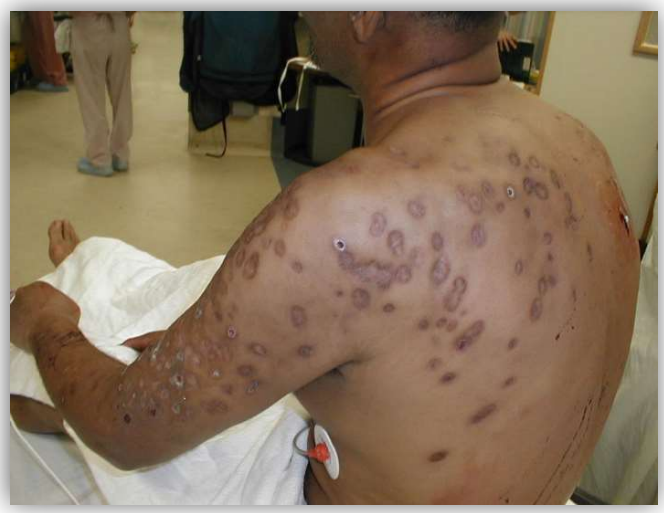
3) Gas production if mixed infections occur!!! (You always need anaerobes to produce gas). The patient will not have gas production in type II unless it progressed to anaerobic infection. As you are using other molecules rather than the oxygen as the final electron acceptor in your electron transport chain, you will then have a gas byproduct. If you see gas production under the skin, you will feel the crepitation by your hand. Hence, you can do filming (To see the gas presence), or you can smell it (If the gas gets out, it will have a distinctive smell).

4) Severe toxicity and renal impairments → Shock (caused by lipoteichoic acid).

5) Myositis (destruction of muscle tissue markedly increases CPK (Usually CPK is unique for muscle, but you can see it in other tissues e.g., elevated after the heart muscle destruction)). As a physician, it will give you an indication to know that muscle cells are being involved and being destroyed.

6) Mortality is high for type 2 (up to 50%! Even with optimal treatment)

Skin popping/drug abuse = basically SubQ injections in same area over and over



As drug abuse is becoming a common case, you should be familiar with this skin popping, they do a subcutaneous infection in different areas over and over and that introduces a bacterium to different layers of the skin and with each time it will take a very long time to heal and it may not never come back to the previous level of content of bacteria, which should be almost nil.

Necrotizing fasciitis caused by mixed aerobic-anaerobic bacteria **begins with a breach in the integrity of a mucous membrane barrier**, such as the mucosa of the gastrointestinal or genitourinary tract.

The portal can be a malignancy, a diverticulum, a hemorrhoid, an anal fissure, or a urethral tear.

Other predisposing factors include peripheral vascular disease, diabetes mellitus, surgery, and penetrating injury to the abdomen (see above table).

Leakage into the perineal area results in a syndrome called Fournier's gangrene, characterized by massive swelling of the scrotum and penis with extension into the perineum or the abdominal wall and legs.

Other forms:

• **In the newborn**, necrotizing fasciitis may complicate **omphalitis** and spread to involve the abdominal wall, flanks, and chest wall. (E.g., cutting the cord and the tool it was not clean. So, they had a lot of gangrene in this form and a lot of deaths)

The problem in this case is with aseptic techniques after delivery, when the umbilicus becomes colonized with many different types of bacteria. A study shows that using chlorhexidine as a topical antiseptic will reduce neonatal mortality and omphalitis even greater.

• Fournier's gangrene is a form of necrotizing fasciitis that affects the male genitals and is usually polymicrobial (Part of GIT).

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

• Cervical necrotizing fasciitis is usually associated with dental or pharyngeal infections and is **polymicrobial (Part of (Oral Mucosa) GI tract).**

Dx of necrotizing fasciitis:

Clinical findings are suggestive + surgical exploration/sample:

a) **Altered mental status (systemic involvement)**

b) **Soft tissue infection signs** (redness/swelling/pain) 70-80% of cases.

- Bullae Pain is typically exaggerated out of exam

- Tenderness is outside the red erythematous borders (indicates further progress)

Are only seen in ¼ of cases.

a) **Fever in less than 50% of the cases!**

b) **Low BP in 21%.** (indicates systemic involvement and overwhelming number of bacteria being destroyed in the system/ releasing bacterial protein into the blood stream is the cause of decreased blood pressure) If you have low blood pressure, it will indicate the beginning of shock.

c) **Crepitation** (feeling of air pockets under skin upon examination) **in 20%.**

Rx – Empiric: (We start with the empirical treatment)

3 Choices depending on the patient and depending on the scenario:
3 Drug combo/ 2 Drug combo/ 1 Drug (each + MRSA coverage).

3 Drug combos:	2 Drugs combos:	1 Drug.
Anaerobic coverage (and inhibits ribosomal production of toxins) = Clindamycin	**(Cefotaxime covers G+ and Gbacteria)**	**(Carbapenem / Imipenem, meropenem, ertapenem)**
G +ve coverage (Ampicillin-sulbactam) or (Piperacillin-tazobactam → antipsuedomonal)	**(anaerobic coverage by metronidazole or clindamycin)**	
G-ve coverage (Ciprofloxacin)		

- The MRSA coverage to be added to any chosen empiric regimen includes = Vancomycin or Linezolid
- Hemorrhagic bullae may indicate presence of vibrio vulnificus, in which case doxycycline is used.

Rx.

Surgical debridement and treatment in hospital ER, surgical exploration, and debridement >> Removing the devitalized tissue that anaerobes use to cause necrosis which might improve systemic drug delivery as well as the immune system.

1- Confirm the diagnosis.

2- Mainstay of therapy.

3- Reducing compartment pressure in extremities.

Prophylaxis for exposed household members (penicillin, rifampin, clindamycin or azirthromycin).

Gas gangrene (Clostridium infection)

Gas production due to anaerobic bacteria

• Typically, due to **contaminated DEEP wounds** - no oxygen - (surgery, car crash...etc.) to introduce spores of G+ve clostridia into the wound (from environment). Common scenario (غرغرينا ومن ثم البتر): There is a car crash and there is a severe devitalization of the tissue due to the destruction of the arterial supply + this wound is contaminated.

• Also progresses similarly to other types: **fasciitis → toxemia → organ failure**

• Gangrene usually occurs following muscle injury and contamination of the wound by soil or foreign material containing clostridial spores. (typical scenario)

• **C. perfringens** is the predominant cause (80–95%), and its pathological effects are mediated by α and λ toxins.

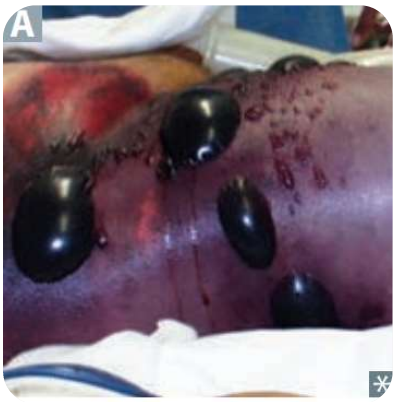
Alpha toxin

Alpha toxins are zinc-dependent, phospholipase C (PLC) with sphingomyelinase and lectinase activity (All of these are destroyed by Alpha toxin) and approximately 42.528 kDa.

• Alpha toxin is responsible for intravascular hemolysis, platelet aggregation, and capillary damage → loss of blood supply = loss of oxygen.

• **These factors stop leukocytes and oxygen from getting to the site of infection → Favorable for the proliferation of C. perfringens.**

• Alpha toxin, **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.



Clostridium perfringens:

Produces α-toxin (lecithinase, a phospholipase) that can cause myonecrosis (gas gangrene A ; presents as soft tissue crepitus) and hemolysis.

If heavily spore-contaminated food is cooked but left standing too long at < 60°C, spores germinate → vegetative bacteria → heat-labile enterotoxin → food poisoning symptoms in 10-12 hours, resolution in 24 hours..

Etiology and pathogenesis:

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, e.g., colonic cancer, diverticulitis, bowel infarction, necrotizing enterocolitis.

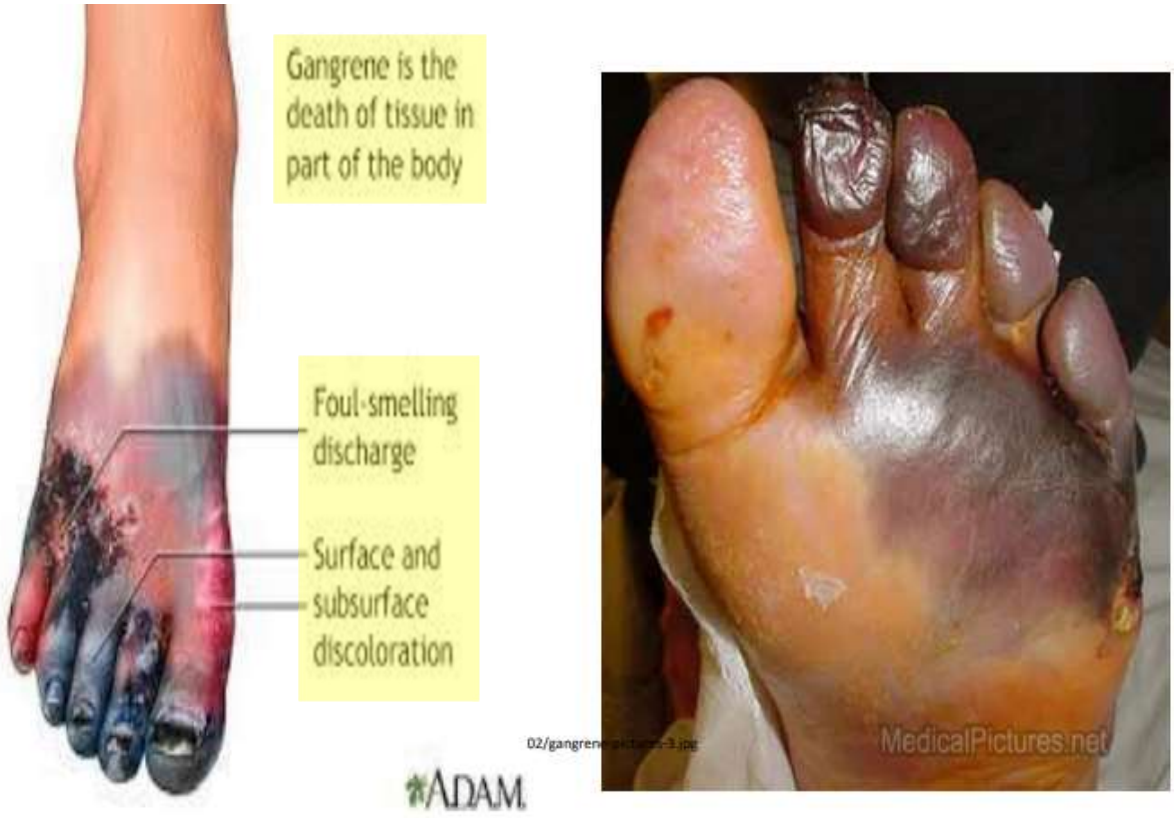
Clinical features:

The incubation period is **usually 2–3 days but may be shorter** because this is the time it's required for spores to germinate. The spores need about a day to germinate and then takes a day to two to start replicating enough and causing enough damage. (The dirtier the wound, then the shorter the incubation period).

• Patients present with acute onset of excruciating pain and signs of shock (fever, tachycardia, hypotension, jaundice, renal failure).

• Local edema and tenderness may be the only early signs, or there may be an open wound, herniation of muscle, a serosanguinous and foul-smelling discharge, crepitus, skin discoloration, and necrosis.

• Progression is rapid, and death may occur within hours.



Diagnosis:

• The diagnosis is **usually clinical** but may be confirmed by Gram stain (you can see the G+ve rods) of the wound or aspirate.

• Liquid anaerobic cultures may be positive within 6 hours.

• Plain radiographs may show gas in the affected tissues

Management:

• **Emergency surgical exploration and debridement** (Removing the destroyed tissue) of the affected area should be performed.

• Empirical antibiotic therapy with piperacillin– tazobactam plus vancomycin (if risk of MRSA)(It will cover the G+ve rods and Pseudomonas) is appropriate, pending cultures.

• Definitive treatment for clostridial myonecrosis is with **penicillin and clindamycin. You are now targeting the glycan cell wall and the anaerobic mechanism (G+ve anaerobe).**

• Hyperbaric oxygen therapy is not recommended (Introducing a high oxygen pressure inside to the tissue), as it has unproven benefits, may also delay resuscitation/surgery treatment. (We may fear ROS, which will travel to the system circulation if you penetrate the tissue)

Cellulitis

Cellulitis is an **acute inflammatory condition** of the skin that is characterized by:

→ localized pain erythema, swelling, and heat (inflammation signs).

Usually caused by indigenous flora colonizing the skin (S. aureus and S. pyogenes) or by a variety of non-colonizing exogenous bacteria.

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

To detect the source of the exogenous bacteria involved in cellulitis a thorough history (+ epidemiologic data) is needed, you need to ask the patient where he was working or what he was doing e.g. working in a marine life, swimming,…etc. As these bacteria occupy small niches in nature.

Supporting data which gives clues to other exogenous causes include:

• Physical activities - trauma - water contact - animal, insect, or human bites - immunosuppression.

• Examples of exogenous bacteria : Enterobacteriaceae, L. pneumophila, A. hydrophila, V. vulnificus, and C. neoformans.

Clinical features:

• Spreading, erythematous, hot, and tender lesion.

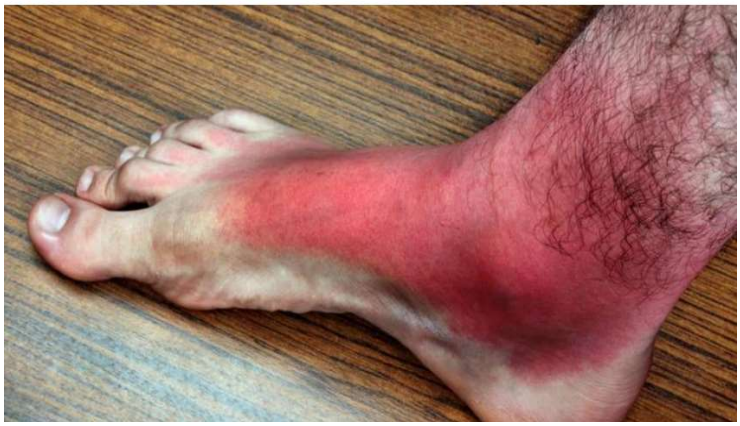
• Usually accompanied by systemic symptoms.

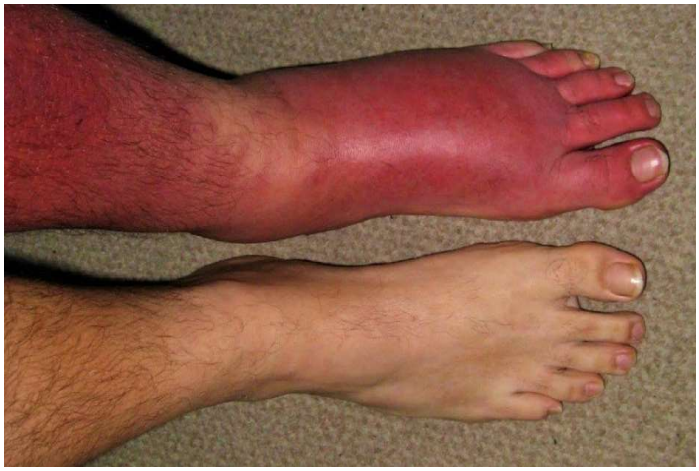
• The Dx is usually clinical, as cultures are **uncommonly positive** (only 20%) - this suggests bacterial numbers are low and local to tissue, but the inflammatory effect is exaggerated due to toxins.

Explanation:
The cultures are rarely positive since the infection happens in the dermis, so we need to access it, and even if we did , the area is so swollen and diluted to the point where the bacteria won't appear. The 2nd reason is the culture would also show the staph and the strep, sometimes small enough that it may be confused with a contamination. So, the dx doesn't depend on the culture.

• Can produce cultures if there is drainage or a site of entry is seen.

Remember; acute and spreading





Notice that cellulitis is a spreading, erythematous, hot & tender lesion; very painful!

Treatment—empiric treatment:

• IV flucloxacillin or clindamycin.

• Vancomycin, teicoplanin, linezolid, or daptomycin are for MRSA cellulitis.

• Gram-negative and anaerobic cover may be required for cellulitis in the context of diabetic ulcers (ulcer + cellulitis is the common case).

• The affected limb should be immobilized and elevated.

Pathogenesis

• Cellulitis caused by S. aureus spreads from a central localized infection (abscess, folliculitis, or an infected foreign body such as a splinter, a prosthetic device, or an IV catheter).

• MRSA is rapidly replacing methicillin-sensitive S. aureus (MSSA) as a cause of cellulitis in both inpatient and outpatient settings (increasing in the outpatient).

• Recurrence is seen in patients with eosinophilia.

• Cellulitis due to S. pyogenes is more rapidly spreading, diffuse process that is frequently associated with **lymphangitis and fever. This is all because streptococci use the lymphatic system in their spread**.

• 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.

• 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**.

• Cellulitis caused by group B Streptococcus occurs mostly in elderly patients (usually patients with diabetes mellitus or peripheral vascular disease.

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

• It is unclear if this form of cellulitis will become less common because of the efficacy of the H. influenzae type b vaccine.

• Cats bites, dog bites → *Pasteurella multocida and Staphylococcus intermedius* and Capnocytophaga canimorsus (more in dog bites).

• 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.

• Pasteurella is known to be resistant to dicloxacillin and nafcillin however, it is sensitive to all other βlactams as well as to quinolones, tetracycline, and erythromycin.

• Thus, for animal or human bites the treatment is usually →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 (ampicillin doesn't work).

P. aeruginosa

Causes 3 types of infections in MSS

• 1→ Ecthyma gangrenosum in neutropenic patients

• 2→ Hot-tub folliculitis

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

• **Commonly seen in hospital setting/immune compromised patients.**

Rx:

Surgical inspection and drainage/debridement (recall biofilm of pseudomonas).

Empirical treatment :

- Aminoglycoside - a third-generation cephalosporin (ceftazidime, cefoperazone, or cefotaxime) -semisynthetic penicillin (ticarcillin, mezlocillin, or piperacillin), or a fluoroquinolone(not in pediatric patient) **pseudomonas is notoriously hard to treat.**

What you can see in the following table:

- The dominant bacterial species and the activity of the antimicrobials against them.
- (+) Represents: activins, (-): inactive, (V) variable among different agents of the group, (+R) indicates that acquired resistance is very common.

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.

ANTIBACTERIAL AGENTS

As you can notice:

- Staphylococci are notoriously weak, Streptococci in a later degree is also weak.

- All of the antimicrobials is negative (inactive) against pseudomonas aeruginosa.

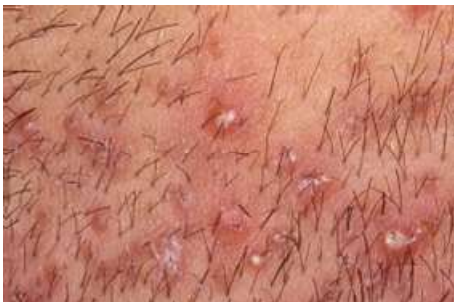
- 2 antimicrobials (one of them we don't use it) only work (active) against TB.

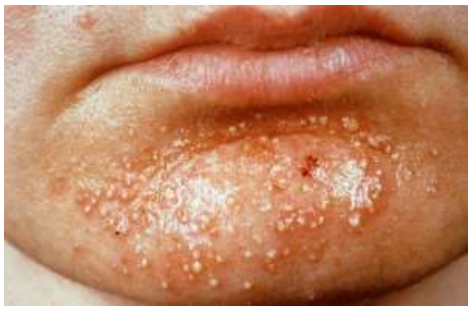
Folliculitis:

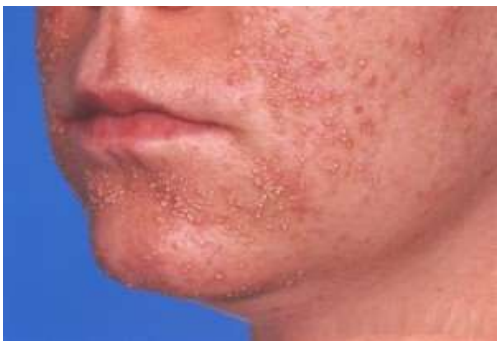
Folliculitis	
Furunculosis	*S. aureus*
Hot-tub folliculitis	*Pseudomonas aeruginosa*
Swimmer's itch	*Schistosoma* spp.
Acne vulgaris	*Propionibacterium acnes*

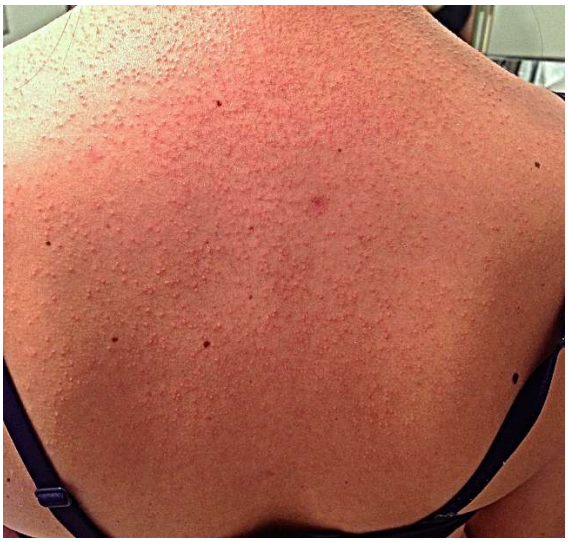
• A superficial infection of the hair follicles and apocrine structures.
• Causative organisms: **S. aureus** (commonest), **P. aeruginosa** ('hot tub' folliculitis), Enterobacteriaceae (complication of acne), Candida spp., and M. furfur (in patients taking corticosteroids).
• Eosinophilic pustular folliculitis occurs in AIDS patients.

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







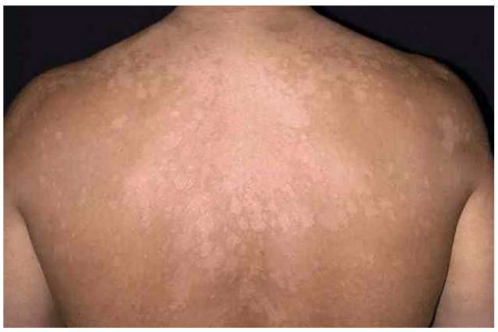


Hot tub folliculitis. (Remember: p.aeruginosa)

Each follicle is inflamed, so obviously the water was contaminated and insufficiently chlorinated.
Notice: All follicles are affected
Patient is young

Hot tub folliculitis:
Usually self-limited In waters not sufficiently Chlorinated and maintained At 37 Celsius

Tinea veriscolor –*M. furfur*



This is not folliculitis, its Tinea versicolor, blanching of the skin (loss of the pigment).
– M. furfur.
(fungal infection)

Treatment—empiric treatment:

is with oral flucloxacillin.

• If the clinical response is slow → consider other pathogens.

Development of folliculitis

Folliculitis: 1 mm of perifollicular red papule or pustule, small that can be treated with just antimicrobials.

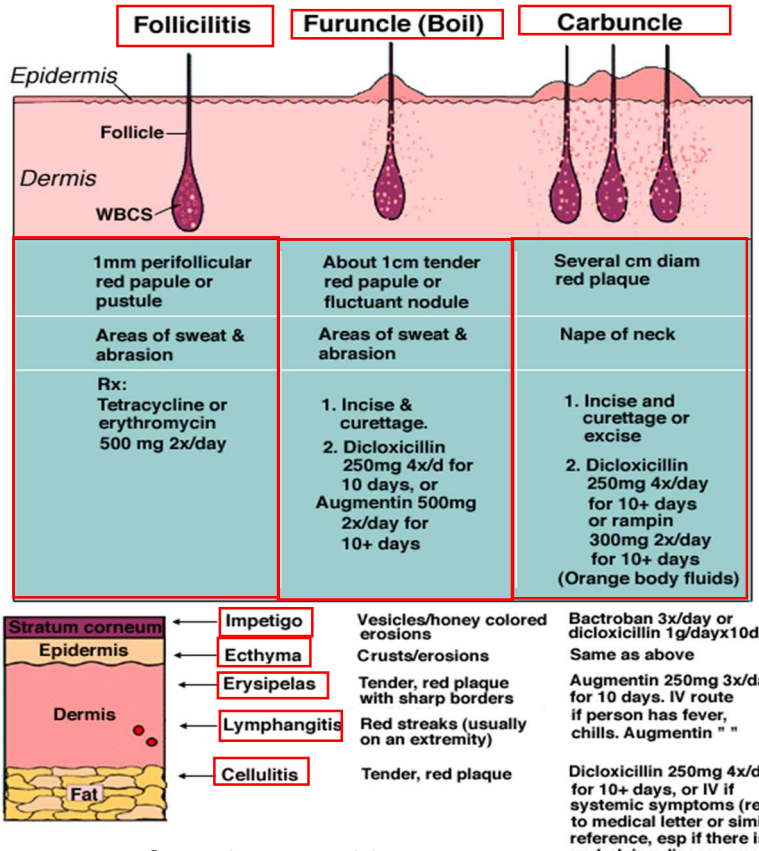
Furuncle: more than one folliculitis joined together, also called boil. furuncle, it's a Folliculitis that has progressed now it has a boil, you must incise and curettage.

Carbuncle: several furuncles.

-Under the cellulitis it will be the necrotizing fasciitis.

- Both Furuncle and Carbuncle need drainage and then antimicrobials.

Bacterial Infections



What was mentioned during the lecture, it is found in a red box.

Cutaneous abscesses

• Collections of pus within the dermis and deeper skin structures.

• Usually polymicrobial containing skin/mucous membrane flora; S. aureus is the sole pathogen in 25% of cases.

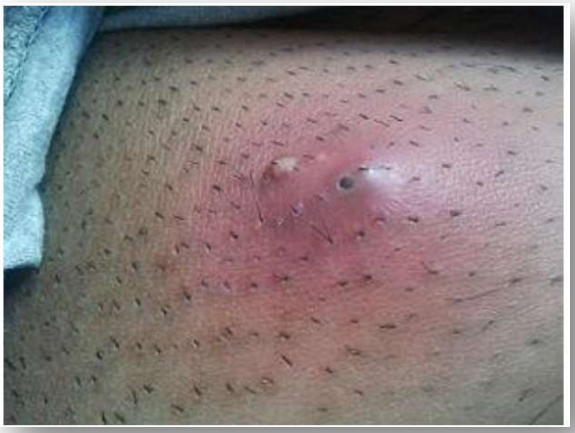
• Clinical features—painful, tender, fluctuant nodules, usually with an overlying pustule and surrounded by a rim of erythematous swelling.

• Treatment is (incision and drainage) I&D → Antibiotics are rarely necessary (except in extensive infection or systemic toxicity, or immunocompromised).

Cutaneous abscesses

Notice: raised lesion, white head Hair follicle might be port of entry, the pus is pushed to the outside rather than to the inside because of pressure.

(There is no amount of antibiotics that you can give to remove an abscess anywhere in the body. (You must drain))



Furuncles and carbuncles

• A furuncle (boil) is a deep inflammatory nodule that usually develops from preceding folliculitis.

• Occur in areas of the hairy skin, e.g. face, neck, axillae, and buttocks.

• A carbuncle is 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.

• Patients may be systemically unwell.

• Outbreaks of furunculosis caused by MSSA and MRSA have been described in groups of individuals with close contact, e.g. families, prisons, and sports teams.

Rx for furnucles:

Application of moist heat (improve treatment) promotes localization and spontaneous drainage.

• Large lesions require surgical drainage.

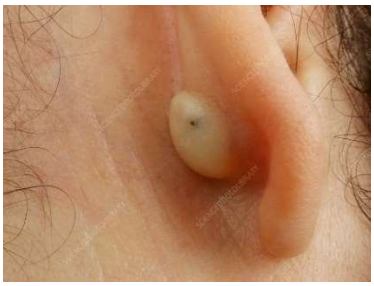
• Systemic antibiotics are indicated → 1- fever, 2-cellulitis 3-lesions are located near the nose or lip.

• Outbreaks control with chlorhexidine soaps and stop sharing of clothing articles or towels, and decolonization of staph.

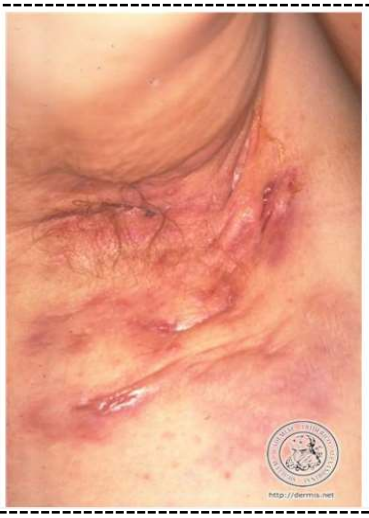
• Sebaceous glands that empty into the hair follicle maybe blocked and cause a swelling similar to an abscess (sebaceous cyst).

• Infection of sweat glands (hidradenitis suppurativa) can also mimic infection of hair follicles, particularly in the axillae.

• Chronic folliculitis is uncommon except in acne vulgaris, where constituents of the normal flora (e.g., Propionibacterium acnes) may play a role.



Sebaceous Cyst



Hidradenitis Suppurativa.

Usually in sweaty areas.

Where skin folds (axilla, Buttocks, breasts, inner thighs)

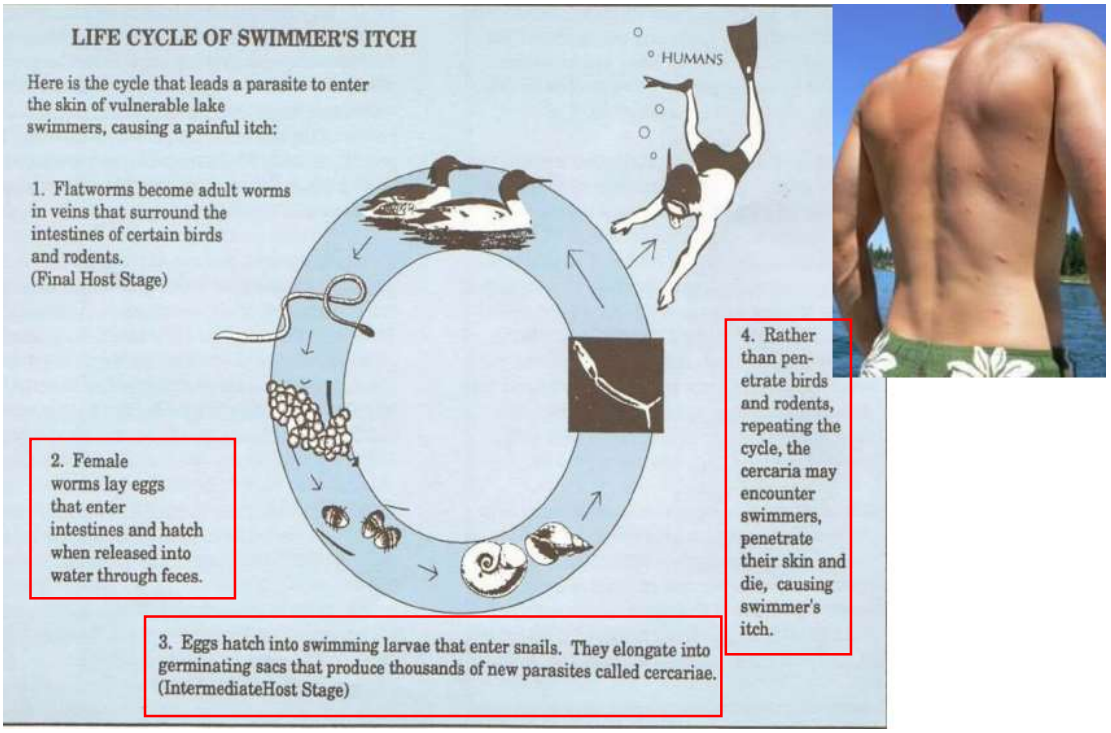
• Infection of sweat glands (hidradenitis suppurativa) can also mimic infection of hair follicles, particularly in the axillae.

Swimmer's itch

• Occurs when a skin surface is exposed to water infested with freshwater avian schistosomes.

• Warm water temperatures and alkaline pH are suitable for mollusks that serve as intermediate hosts between birds and humans.

• Freeswimming schistosomal cercariae readily penetrate human hair follicles or pores, but quickly die and elicit a brisk allergic reaction, causing intense itching and erythema.



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Erysipelas

• Erysipelas is due to S. pyogenes and is characterized by an abrupt onset of fiery-red swelling of the face or extremities.

• The distinctive features of erysipelas are well-defined indurated margins, particularly along the nasolabial fold; rapid progression; and intense pain.

• Flaccid bullae may develop during the second or third day of illness, but extension to deeper soft tissues is rare.

• Treatment : penicillin(flucloxacillin, clindamycin) or is effective

• Swelling may progress despite appropriate treatment, although fever, pain, and the intense red color diminish.

• Desquamation of the involved skin occurs 5–10 days into the illness.

• Infants and elderly adults are most commonly afflicted, and the severity of systemic toxicity varies