Musculoskeletal system

Skin and soft tissue infections 4 Harrison's infectious disease 2nd Ed Oxford handbook of ID and MM

Necrotizing Fasciitis

An indication that results in the death of parts of soft tissues (usually by streptococci)

Necrotizing fasciitis	
Streptococcal gangrene	*S. pyogenes*
Fournier's gangrene	Mixed aerobic and anaerobic bacteria
Staphylococcal necrotizing fasciitis	Methicillin-resistant *S. aureus*

Necrotizing fasciitis

- RAPIDLY progressing infection in the area between the fascia and deep subcutaneous tissue.
- Many risk factors increase the risk (see table next slide)
- Fibrous bands in this area prevents spread of infection

These bands are present in the head –and trunk- but not in the extremities (thus extremities are more susceptible)

The scary thing about necrotizing fasciitis is that it is rapidly progressing

- **>50% in extremities**
- 20% in perineum or buttocks (esp in DM and alcoholics)
- 18% in trunk
- 9% head and neck

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

- 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.

It's not that common but it's still common enough.

Risk factors associated with necrotizing fasciitis

Usually by skin flora

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 **HIV patients**	-Heart disease -PVD (**Peripheral vascular diseases**) -DM	-Burns -penetrating trauma **You will never have completely sterilization in the area of trauma up to 3 months from it happens.** -IV drugs -surgery	colon cancer -diverticulae hemorrhoids or fissues **Will introduce bacteria in the anus that will have a free access into the skin around it** urethral tears

EXPLANATION FOR THE PREVIOUS TABLE

Malnutrition: All these will reduce liver's ability to produce immunoglobulins, acute phase reactants, innate and adaptive proteins that needed in our defense.

Patient conditions:

AGE - The immunity decreases in elderly people + they are more likely to have reduced circulation (atherosclerosis and reduce artery flow)

OBESITY - Poor blood supply to the skin

POOR BLOOD SUPPLY: DM- Diabetes itself reduces immunity, and causes neuropathy, so signals to fight infections will be reduced.
Also, because neuropathy patients may not sense the penetrating trauma, then the infection might spread quicker before being treated.

Signs and Symptoms – occur in order

Pain/tenderness In the area, we don't have swelling, redness and hotness **because**

1. It's under the nerves that supply pain 2. It's deep

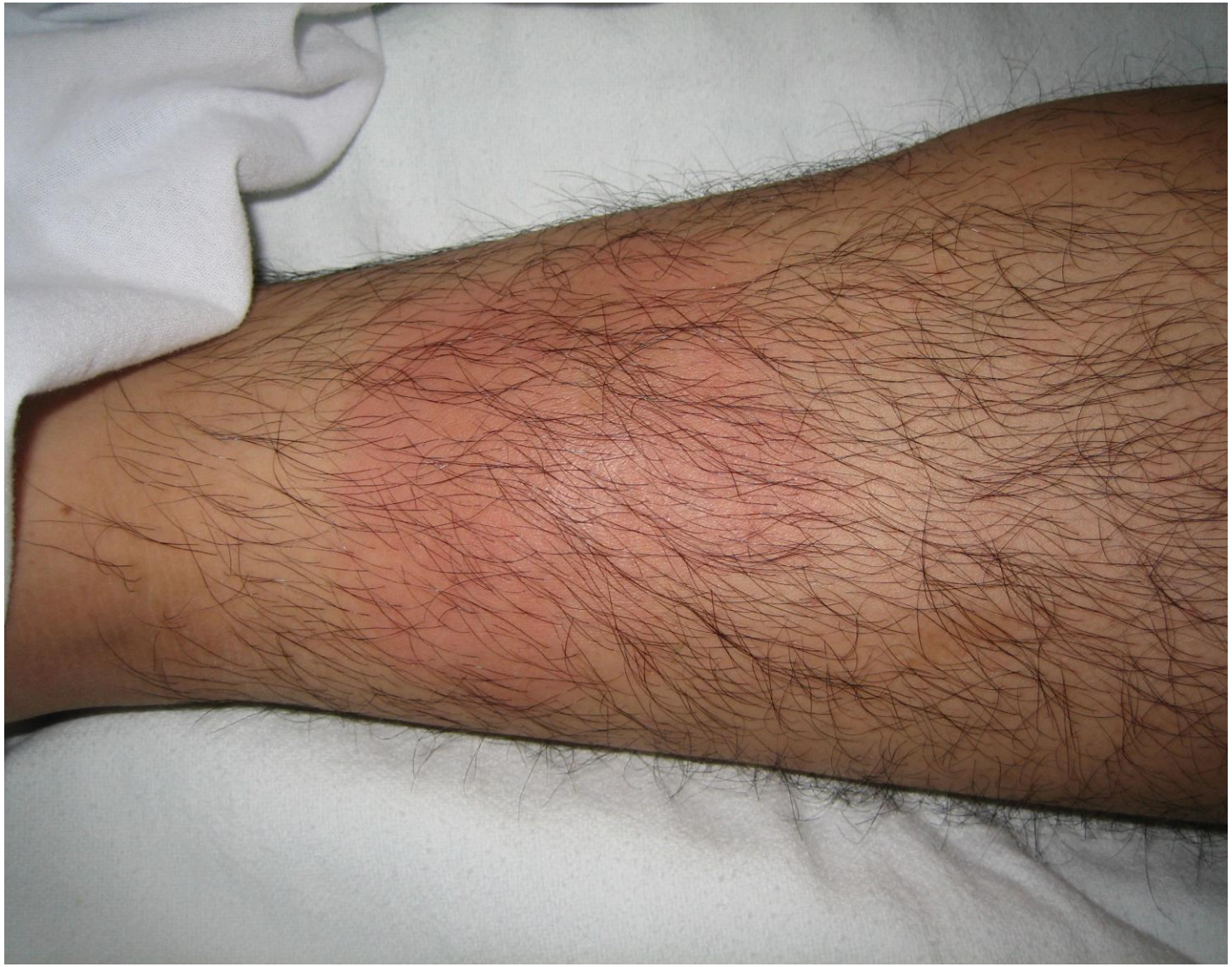
- Unexplained fever (Early diagnosis may be difficult when pain or unexplained fever is the only presenting manifestation, remember infection is deep, might not present with pain yet)

  Because it has caused enough destruction to gain access into the systematic circulation (trichoic acid and LPS will be released)

- Later on: Swelling (infection at this point is severe)
- Dark red induration (indicates hemorrhage and early necrosis)
- BULLAE, filled with blueish or purple fluid
- Thrombosis of dermal blood vessels (*The affected area becomes anesthetic as a result of small vessel thrombosis and destruction of superficial nerves)*
- Extension to deep fascia with rapid spread **-The least resistance is to keep spreading horizontally but it can spread to anywhere else-**
- Most progressed symptoms : toxicity , shock and multi organ failures (has progressed beyond local infection site)

High risk of mortality and morbidity

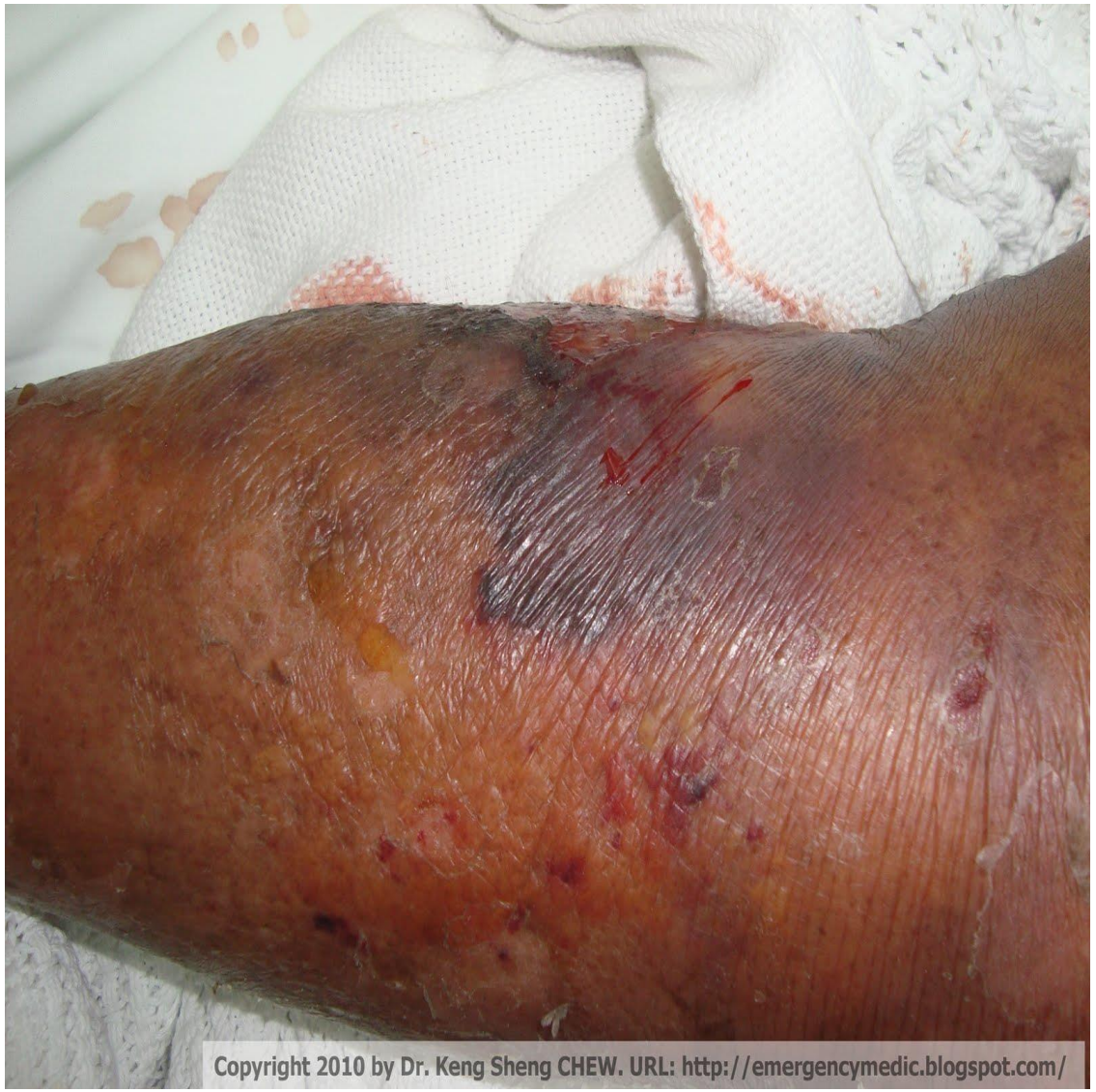
Pain / redness



This patient has necrotizing fasciitis but it almost looks normal - only pain and redness -

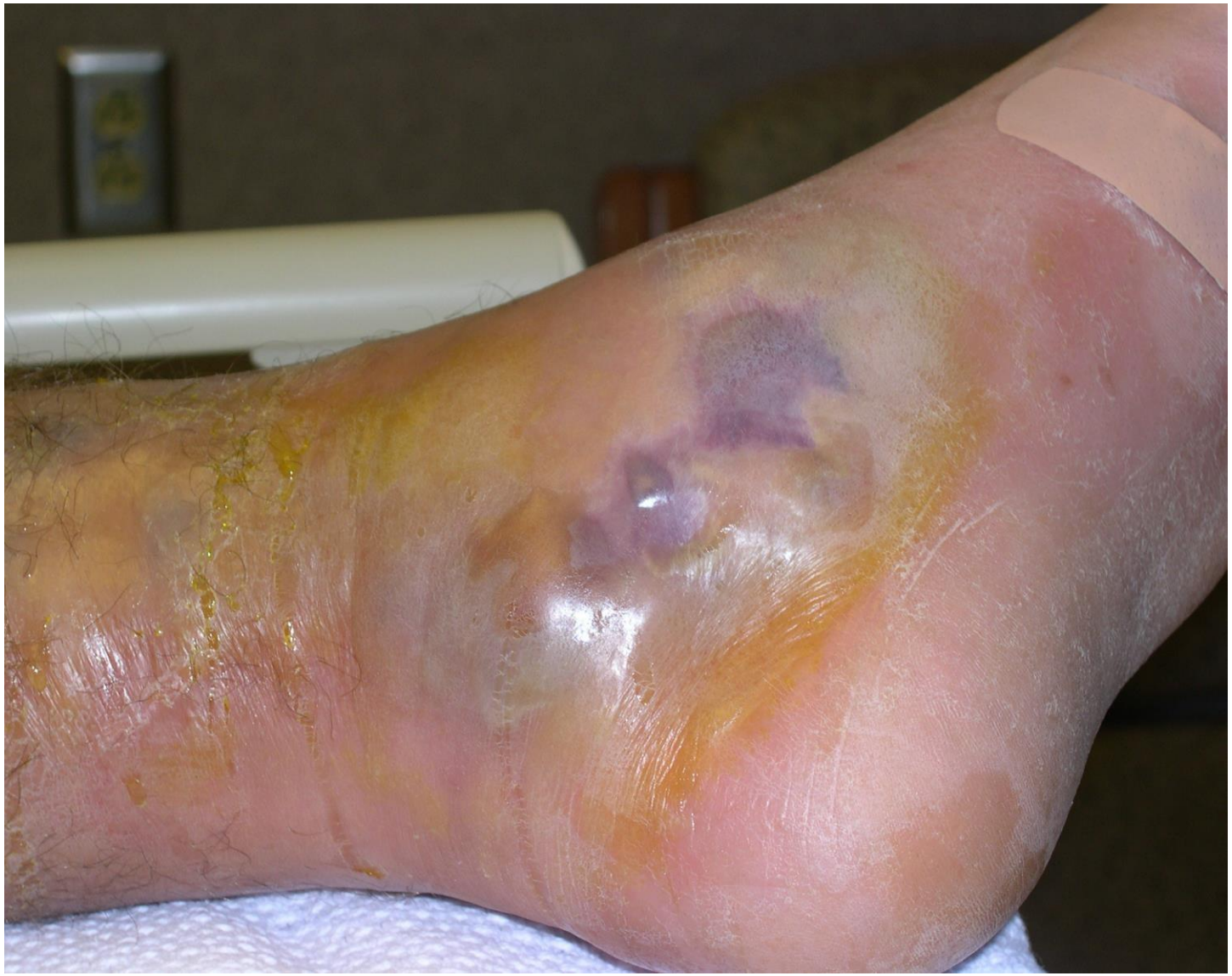
http://www.antimicrobe.org/new/printout/e11printout/e11cause/e11cau9.jpg

Dark red induration /swelling



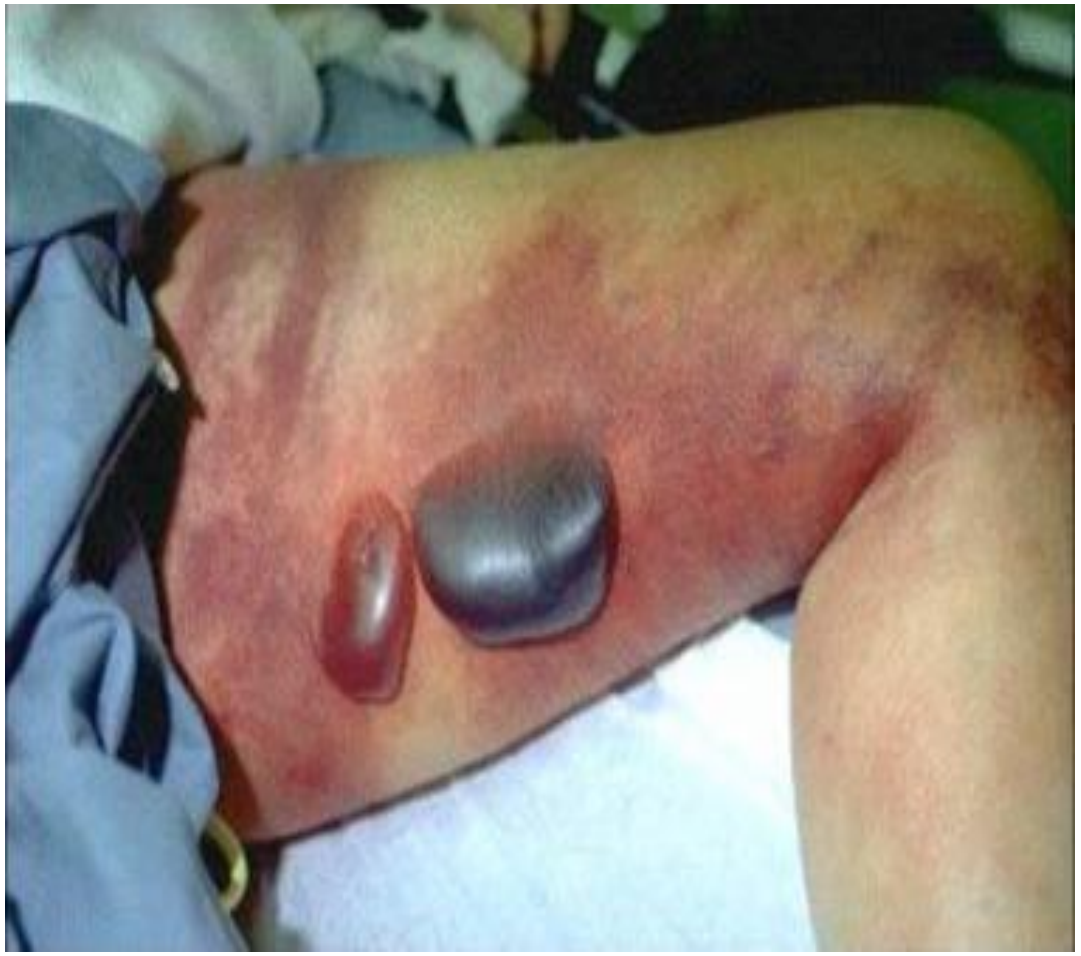
After that you'll get to the stage with the dark red induration
- severe-

Progressing to bullae /thrombosis



https://osuemed.files.wordpress.com/2012/09/diff-patient-with-blebs.jpg

Bullae formation then thrombosis of the dermal vessels then it will be harden or and get crusted



More bully formation, purple fluid

http://img.medscapestatic.com/pi/meds/ckb/55/37455tn.jpg

Microbiology causes:

- A**) Polymicrobial** • Type I necrotizing fasciitis involves at least one anaerobic species (Bacteroides or Pepto streptococcus 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!
- You would want facultative anaerobic bacteria to pave the way for the aerobic bacteria to come in (the polymicrobial cases are likely to be due to contamination or have a connection to the enteric tract)
- 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 (in genitalia/perineal area)*

- *3- Mixed infection usually have comorbid states (DM (type 1) , PVD (Peripheral vascular diseases) , immunocompromised)*

- *21% mortality rate with optimal treatment*

Microbiology causes. . .cont

Mono microbial

- B) **Type II necrotizing fasciit**is is usually caused by GAS alone or in combination with other species (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 have been reported to cause necrotizing fasciitis.(also necrotizing pneumonia)

1) Usually following trauma in otherwise healthy individual or IV drug abusers (skin popping) not like the previous picture of co morbid patient with gastric, genitourinary tract disruption.

The connection is added from the top rather than the bottom (air to skin) Otherwise type1 from inside to outside (lumen to skin)

2)Fasciitis progresses to skin contusions due to seeding by transient bactermia -In other areas-

3) Gas production if mixed infections occurs!!! (you need anaerobes)

As you are using molecules other than oxygen in electron transport chain as final acceptor you will then have a gas by product -You can see a gas by product under the skin by feeling the precipitation with your hand so you know that there are gas production and do filming, x-ray or CT scan can also show you, or you can smell it because it has a distinctive bad smell-

The rest of the previous slide

4) Severe toxicity and renal impairments shock

Reduction of vital signs - low respiratory and heart rate, low blood pressure (we do some actions such as increasing alpha 2 receptors, norepinephrine or epinephrine)

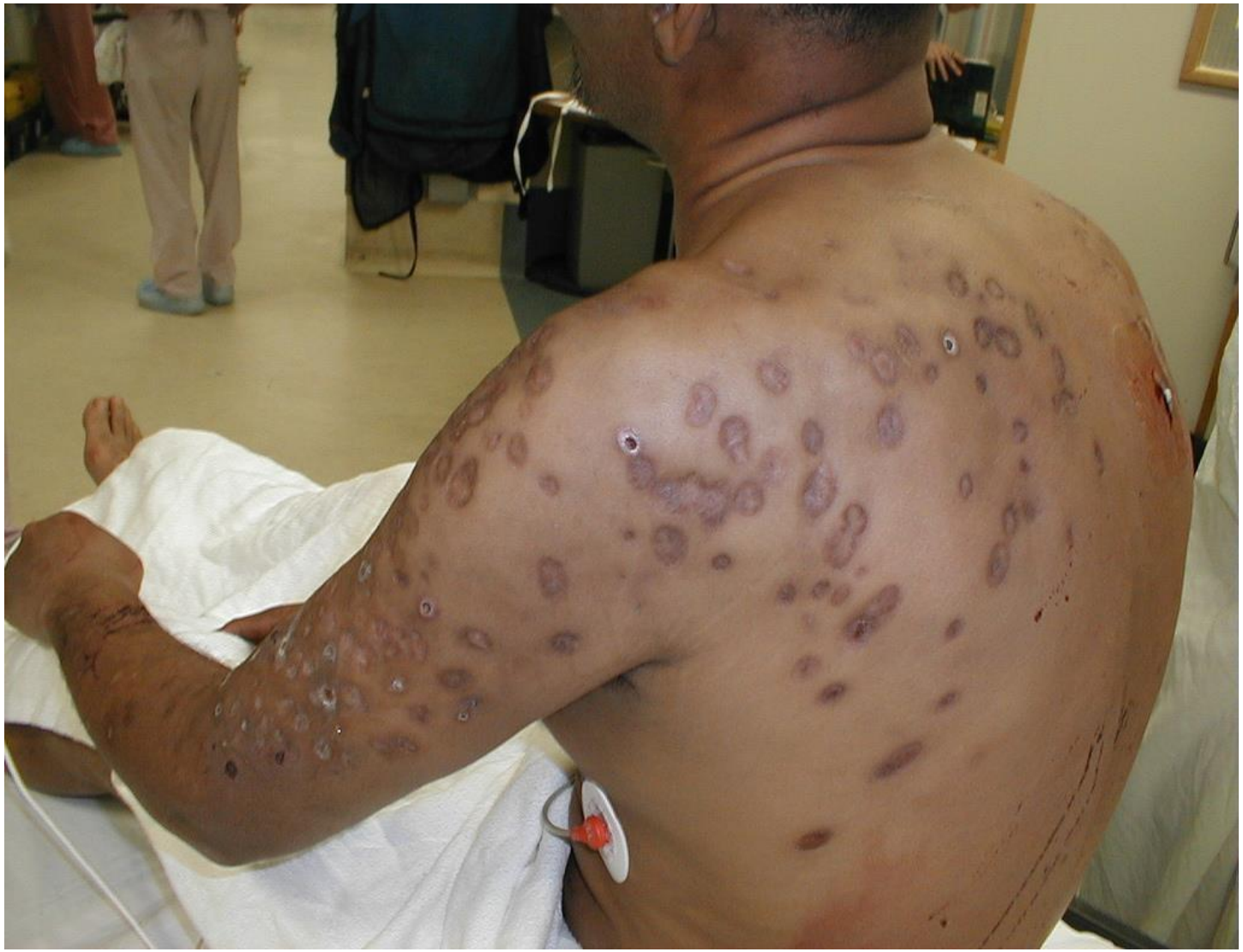
5) Myositis (destruction of muscle tissue markedly increases CPK)

CPK -unique for muscles but we can see it in other tissues-(if CPK increases we should know that muscle cells are being destroyed and are releasing their contents into the blood,

6) Mortality is high (upto 50%! Even with optimal treatment)

Mortality here is higher than type 1

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



Bacteria will never end with this area and it will take a very long time to heal then it returns to previous level but never come back to its previous state

- *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*.

-This appears in children-

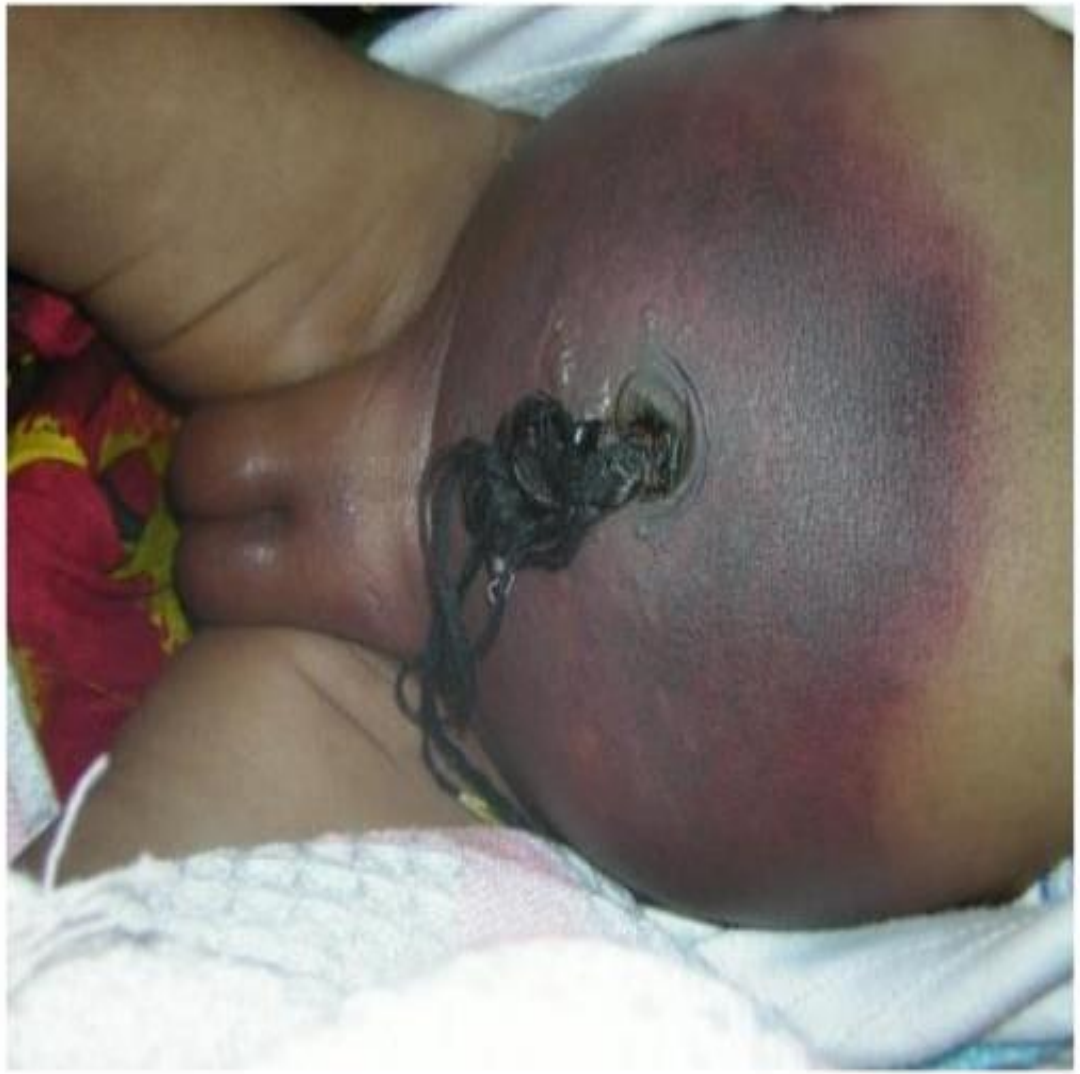
Other forms

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

For example when you cut the cord and the tool wasn't clean

- 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 -Group A streptoccocus- (skin).
- Cervical necrotizing fasciitis is usually associated with dental or pharyngeal infections and is

polymicrobial (part of GI tract -part of oral mucosa-).



Gangrene in new-borns

https://image.slidesharecdn.com/omphalitis2-120928165437-phpapp01/95/omphalitis-2-11-728.jpg?cb=1348851384

Dx of necrotizing fasciitis

Not the redness area is effected but it's beyond that

Clinical findings are suggestive + surgical exploration/sample:

a) Altered mental status (systemic involvement) -Not always-

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

Indicates systemic involvement and an overwhelming no. of bacteria being destroyed in the system, also the heart and respiratory rate will increase, vitals are easily affected by dead organisms in the blood

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

b) Low BP in 21%

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

Rx - empiric

We have 3 cocktails here :p
And we add to each one of them chocolate (MRSA)
and this depends on the patient..
1. Which regimen can the patient tolerate
2. Which has be a clinical scenario to occur

- 3 drug combo/ 2 drug combo/ 1 drug (each +MRSA coverage)

3 drug combo :

- 1- anaerobic coverage (and inhibits ribosomal production of toxins)= Clindamycin
- 2- G +ve coverage (Ampicillin-sulbactam) or (Piperacillin-tazobactam ❼*antipsuedomonal*)
- 3- G-ve coverage (Ciprofloxacin)

+ MRSA coverage

- 2 drug combo (Cefotaxime covers G+ and G- bacteria) + (anaerobic coverage by metronidazole or clindamycin)
- 1 drug combo (Carbapenem / Imipenem, meropenem, ertapenem)

- 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 is a MUST but why?
Because we need to remove the devitalized tissue to promote the reperfusion of the tissue

- Surgical debridement, and *treatment in hospital*
  Emergency surgical exploration and debridement
  1-confirm the diagnosis and are the
- *2- mainstay of therapy.*
- 3- Reducing compartment pressure in extremities

After that if you don't recall something called compartment syndrome which might occur in the extremities, where there's excessive swelling that the it is bound by the borders of compartments in the limbs, these borders are usually made by fibrous tissue. These borders form a chamber like structure and the swelling inside the chamber or compartment may be elevated high enough to prevent atrial supply to the underlying structures.

- Prophylaxis for exposed house hold 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), and these spores will then germinate
- 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 toxin zinc-dependent, phospholipase C (PLC) with sphingomyelinase and lectinase activity and approximately 42.528 kDa
- Alpha toxin is responsible for intravascular hemolysis, platelet aggregation, and capillary damage ➏ loss of blood supply = loss of oxygen.
- These factors stop leukocytes and oxygen from getting to the site of infection ➏ 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

https://www.ncbi.nlm.nih.gov/pmc/articles/PMC5127136/

Cont.. 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.
- If it travels through the blood, it can seed into the deep fascia causing gas gangrene without trauma.

It's not really an obvious wound but it's usually caused by this type of clostridium. So, there is a link but it's not as clear a link as the RTA method for traffic accidents.

Clinical features

- The incubation period is usually 2–3 days but may be **shorter.**
- 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

Why we're talking about short period here is that the short time for spores to germinate so it needs about one day to germinate and takes day to two to start replicating enough to be damaged

The shorter the dirtier of the wound the shorter the incubation period

Surface and subsurface discolouration

http://blog.almawiclinic.com/wp-content/uploads/2015/07/Foot-with-gangrene.jpg

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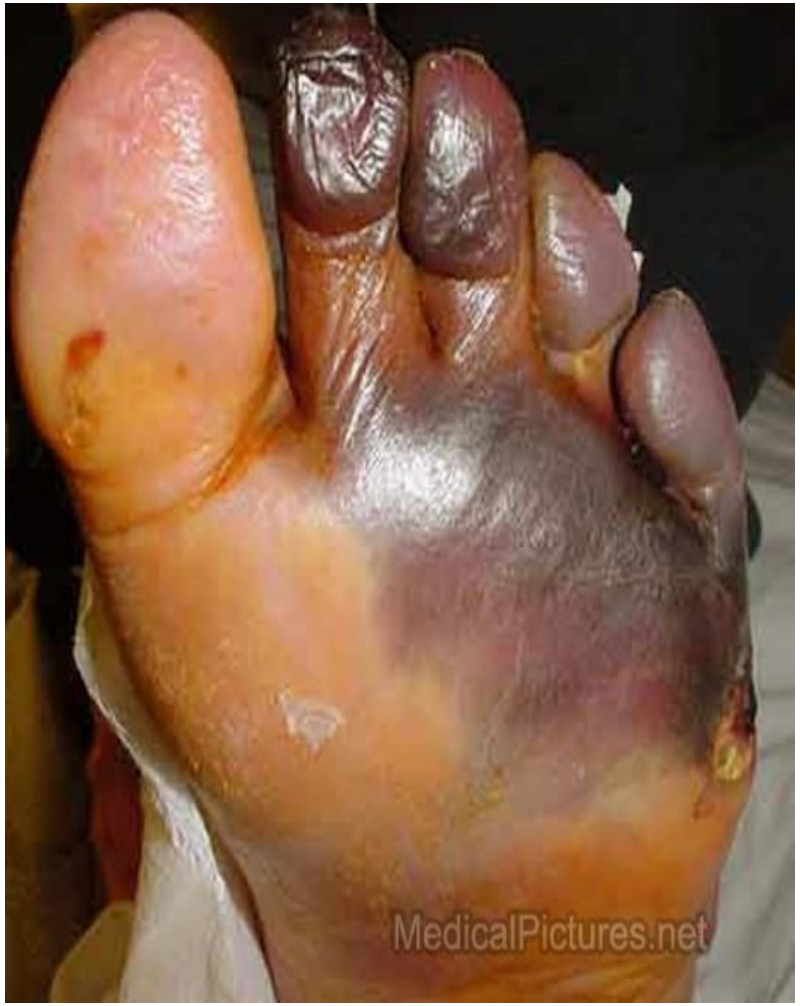


02/gangrene-pictures-3.jpg

http://healthooze.com/wp-content/uploads/2012/12/wet-gangrene.jpg

Diagnosis •

- The diagnosis is usually clinical but may be confirmed *by Gram stain of the wound or aspirate.*
- *Liquid anaerobic cultures may be positive within 6h.*
- *Plain radiographs may show gas in the affected tissues*

Management •

- Emergency surgical exploration and debridement of the affected area should be performed.
- • Empirical antibiotic therapy with piperacillin–tazobactam plus vancomycin (if risk of MRSA) is appropriate, pending cultures.
- • Definitive treatment for clostridial myonecrosis is with **penicillin and clindamycin.**
- Hyperbaric oxygen therapy is not recommended, as it has unproven benefits, may also delay resuscitation/surgery treatment

Explanation for the previous slide

- Surgical: the doctor should remove the bacterial food (destroyed tissue) that is making the bacteria viable

- Anti-MRSA : because these drugs will cover your pseudomonas and cover your G+ rods

- Penicillin + Clindamycin: because we're targeting the peptidoglycan cell wall of the bacteria and anaerobic mechanism as well.

- Hyperbaric oxygen: because the bacteria is anaerobic, we can give it high oxygen pressure which will penetrate the wound and then kill the bacteria. This is in theory, but in practice it is not as easy.
- WHY? Because if you aerate the wound, it should be enough. So, why would it increase the pressure, increase the production of oxygen and how well the doctor can introduce oxygen into the tissue. This is why the benefits are unproven. If you can really pump enough oxygen into the tissue, you will do well, but what we are scared of is that if we have a lot of formation of oxygen species which will go through the system and cause other problems, we don't need

Cellulitis "not bullus forming"

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

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

❼ l o c a l i z e d 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.

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

Explanation for the previous slide

- These organisms are introduced into the dermis, and since we have nerves and blood vessels there, the main symptoms are pain, erythema and swelling (inflammation signs). So, the keyword for cellulitis is ACUTE INFLAMMATORY CONDITION.

- To detect the *source of the exogenous bacteria involved in cellulitis a thorough history (+ epidemiologic data) is needed, as these bacteria occupy small niches in nature.*

Cellulitis

- Supporting data which gives clues to otherexogenous 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

- ❼ **spreading ,**erythematous, hot, tender lesion
- ❼ usually accompanied by systemic symptoms.
- • The Dx is usually clinical, as cultures are rarely positive (only 20%)- this suggests bacterial numbers are low and local to tissue but the inflammatory effect is exaggerated due to toxins.
- Can do culture if there is drainage or a site of entry is seen
- • 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.

*** 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.

Explanation of the previous slide

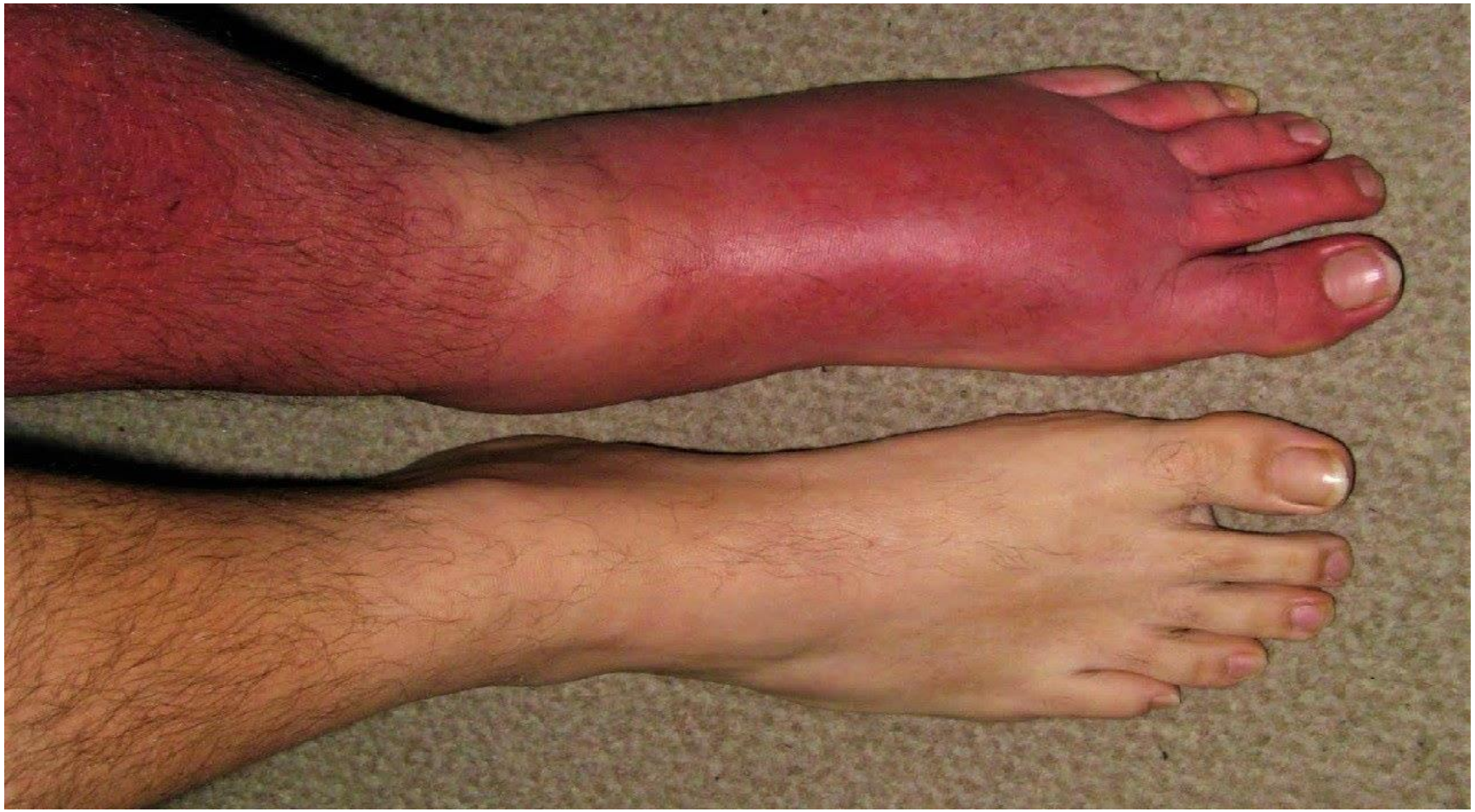
- Rarely positive WHY? The problem is you have an acute inflammation with lots of swelling, so the bacteria is diluted with the amount of fluid that it is in the cells and if you take a small needle, you may not capture enough bacteria for the culture

2. The culture would show you small colonies of staphylococcus and streptococcus, so it is sometimes be confused with a contamination.

*** The book says that is rarely positive, but it's more uncommonly positive

- Clindamycin is for the anaerobes and it also kills G+ but not as good as Penicillin

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



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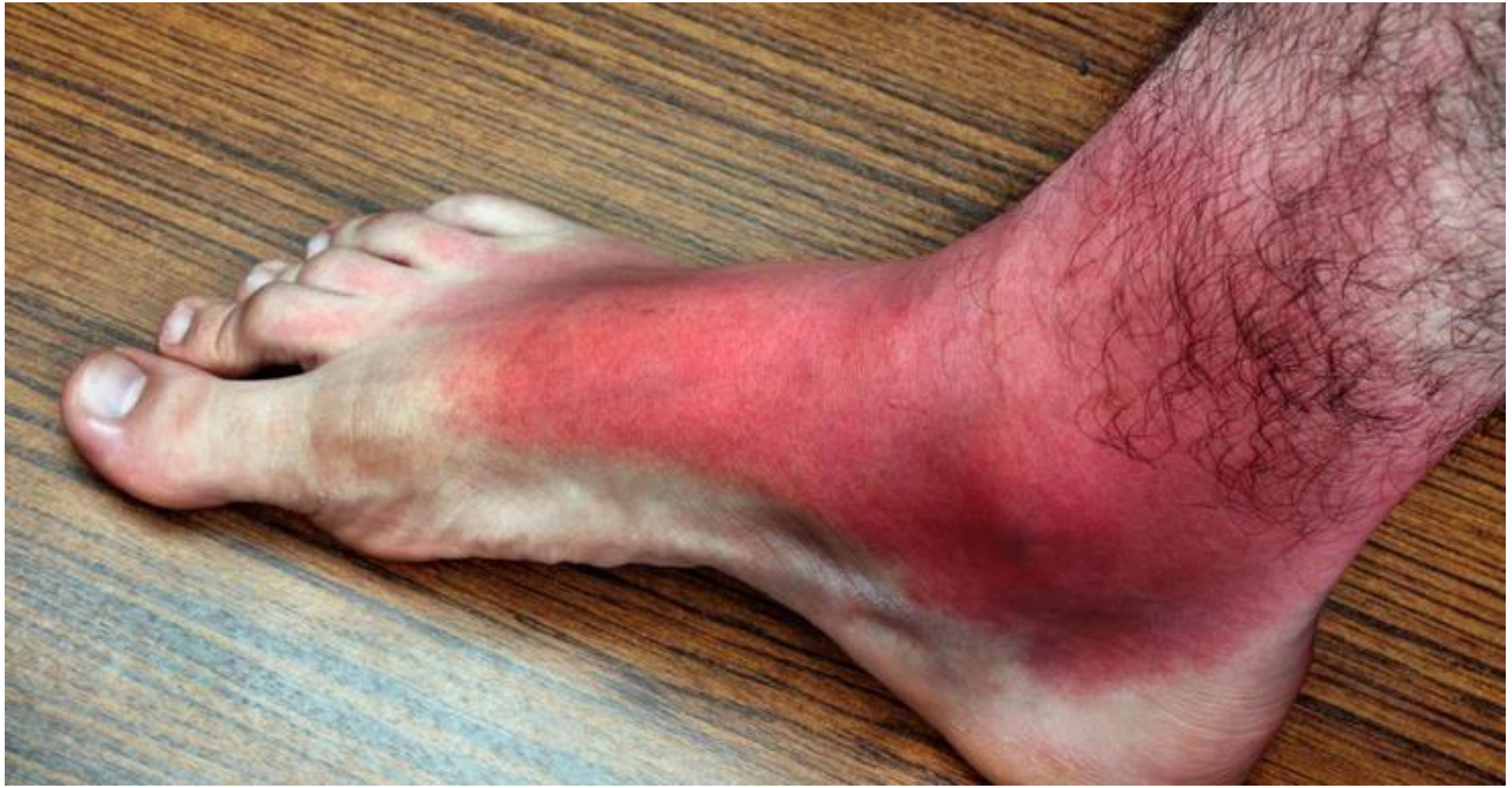
Remember; acute and spreading



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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.
- Recurrence is seen in patients with eosinophilia

MRSA , right now over time it is increasing in outpatient settings rather than inpatient settings.

- Cellulitis due to *S. pyogenes* is **more rapidly spreading**, diffuse process that is frequently associated with lymphangitis and fever.
- 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
- This is all due to the fact that **streptococci use the lymphatic system in their spread**.
- Anything related to lymph should lead us to think of GAS.

What is happening here is that you when you remove some of these superficial veins or this chronic stasis will be breeding ground for these streptococcus which the growth condition better for them and then makes cellulitis more likely to occur

- 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 as a result of the efficacy of the *H. influenzae* type b vaccine.

Explanation of the previous slide

- GBS, you know that CC is not common to be seen in the skin.
- H.inf: it may access through the pharynx, sinuses and can causes p.cellulitis.
- Again, with sinusitis substance media or epiglottitis and it is unclear if this form of cellulitis will become less common as a result of the vaccination or not.
- As we vaccinate more against type B H. influenza, other types become more dominant as they become pushed into the pressure so some of them and this is an evolutionary pressure for them to survive.

- 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 β- lactams as well as to quinolones, tetracycline, and erythromycin.
- Thus, for animal or human bites the treatment is usually ❼Ampicillin/clavulanate, ampicillin/sulbactam, and cefoxitin

Everything in the mouth will be introduced into the skin "polymicrobial"

You don't have to memorize all of these but just to know that is polymicrobial

- *Aeromonas hydrophila* ❼ aggressive cellulitis in injuries sustained in freshwater (lakes, rivers, and streams).
- Treatment according to known sensitivity of this o r g a n i s m ❼, fluoroquinolones, chloramphenicol, trimethoprim-sulfamethoxazole, and third-generation cephalosporins (ampicillin doesn't work)

P. aeruginosa

- Causes 3 types of infections in MSS
- 1 ❼ Ecthyma gangrenosum in neutropenic patients
- 2 ❼ Hot-tub folliculitis (Water associated with baths)
- 3 ❼ Cellulitis following penetrating injury (usually stepping on a nail)
- Commonly seen in hospital setting/immune compromised patients. (You need to improve the cure or the treatment)
- 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.

ANTIBACTERIAL AGENTS

This is a good table to keep your head on what drugs work on which bacteria

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

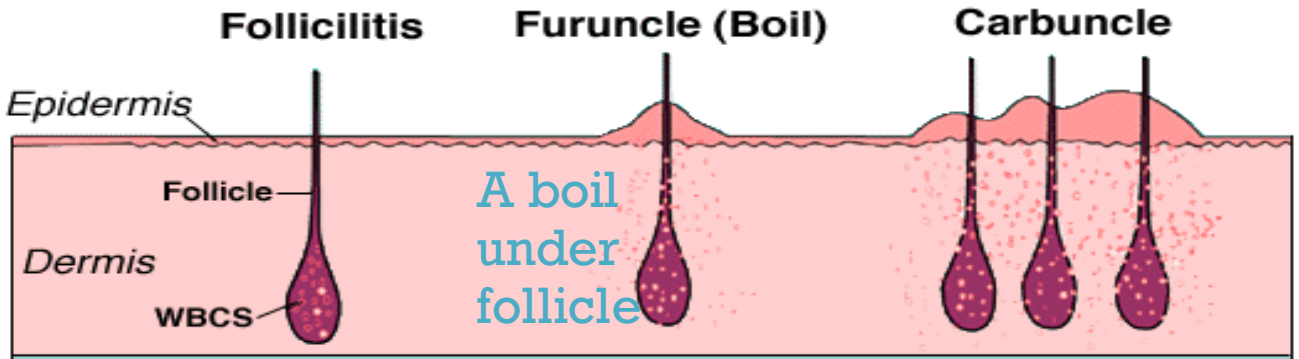
Agent	Site of action	Usual activity[a] against: Staphylococci	Streptococci	Enterobacteria	*Pseudomonas aeruginosa*	*Mycobacterium tuberculosis*	Anaerobes
Penicillins	Cell wall	+R	+	V		–	
Cephalosporins	Cell wall	+	+			–	
Other β-lactam agents	Cell wall	V	V			–	
Glycopeptides	Cell wall	+	+	–	–	–	
Tetracyclines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

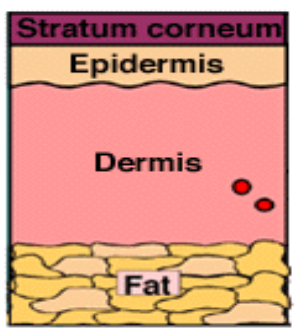
Folliculitis

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

Bacterial Infections



1mm perifollicular red papule or pustule	About 1cm tender red papule or fluctuant nodule	Several cm diam red plaque
Areas of sweat & abrasion	Areas of sweat & abrasion	Nape of neck
Rx: Tetracycline or erythromycin 500 mg 2x/day	1. Incise & curettage. 2. Dicloxicillin 250mg 4x/d for 10 days, or Augmentin 500mg 2x/day for 10+ days	1. Incise and curettage or excise 2. Dicloxicillin 250mg 4x/day for 10+ days or rampin 300mg 2x/day for 10+ days (Orange body fluids)



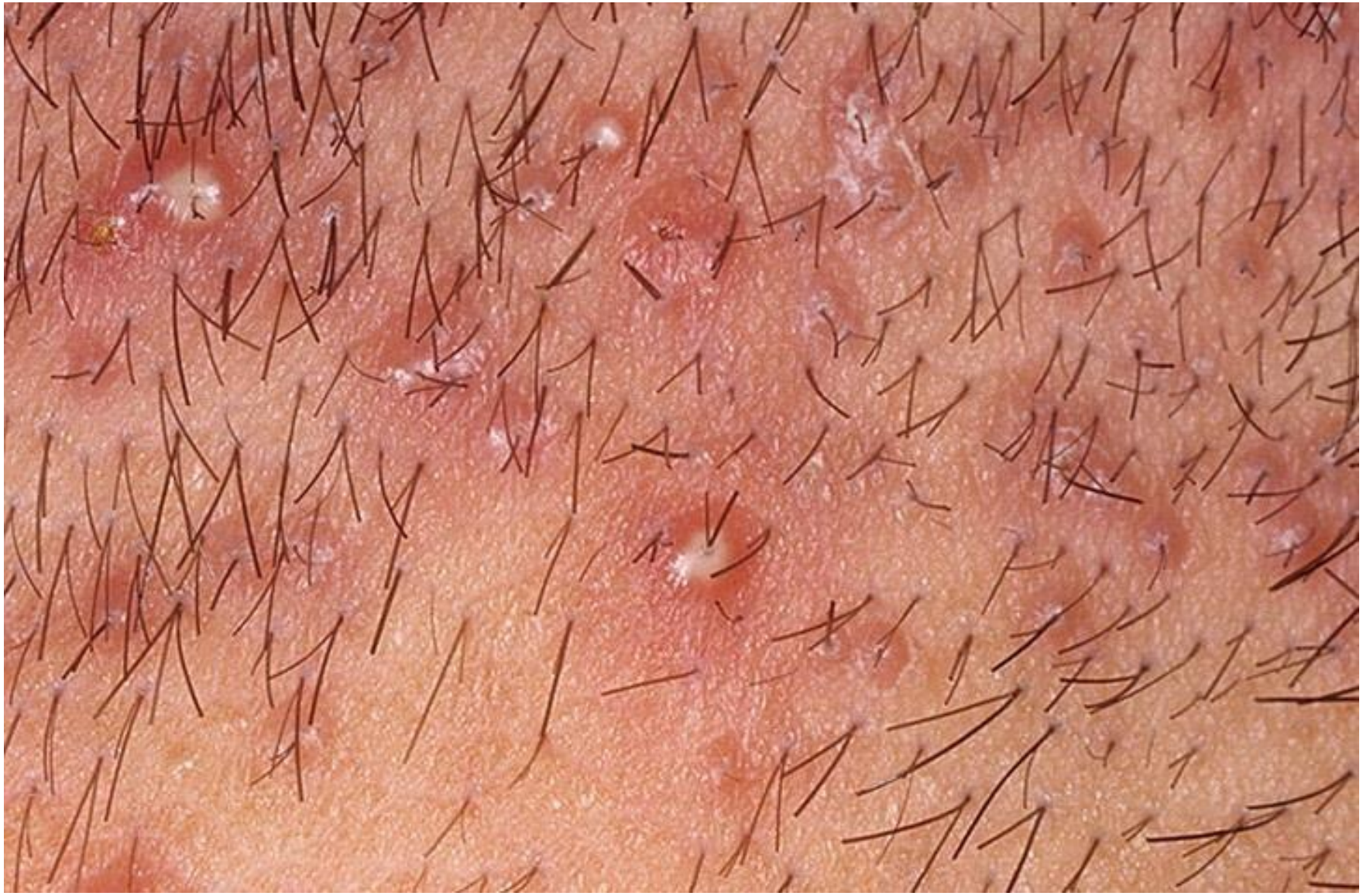
Impetigo	Vesicles/honey colored erosions	Bactroban 3x/day or dicloxicillin 1g/dayx10day
Ecthyma	Crusts/erosions	Same as above
Erysipelas	Tender, red plaque with sharp borders	Augmentin 250mg 3x/day for 10 days. IV route if person has fever, chills. Augmentin " "
Lymphangitis	Red streaks (usually on an extremity)	
Cellulitis	Tender, red plaque	Dicloxicillin 250mg 4x/day for 10+ days, or IV if systemic symptoms (refer to medical letter or similar reference, esp if there is underlying disease such as diabetes mellitus, etc.)

https://library.med.utah.edu/kw/derm/pages/in04_10.htm

Folliculitis

- • 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.
- • Treatment—empiric treatment is with oral flucloxacillin.
- If the clinical response is slow ➡ consider other pathogens.

Usually associated with shaving.



http://diseasespictures.com/folliculitis-symptoms-causes-pictures-treatment-home-remedies/

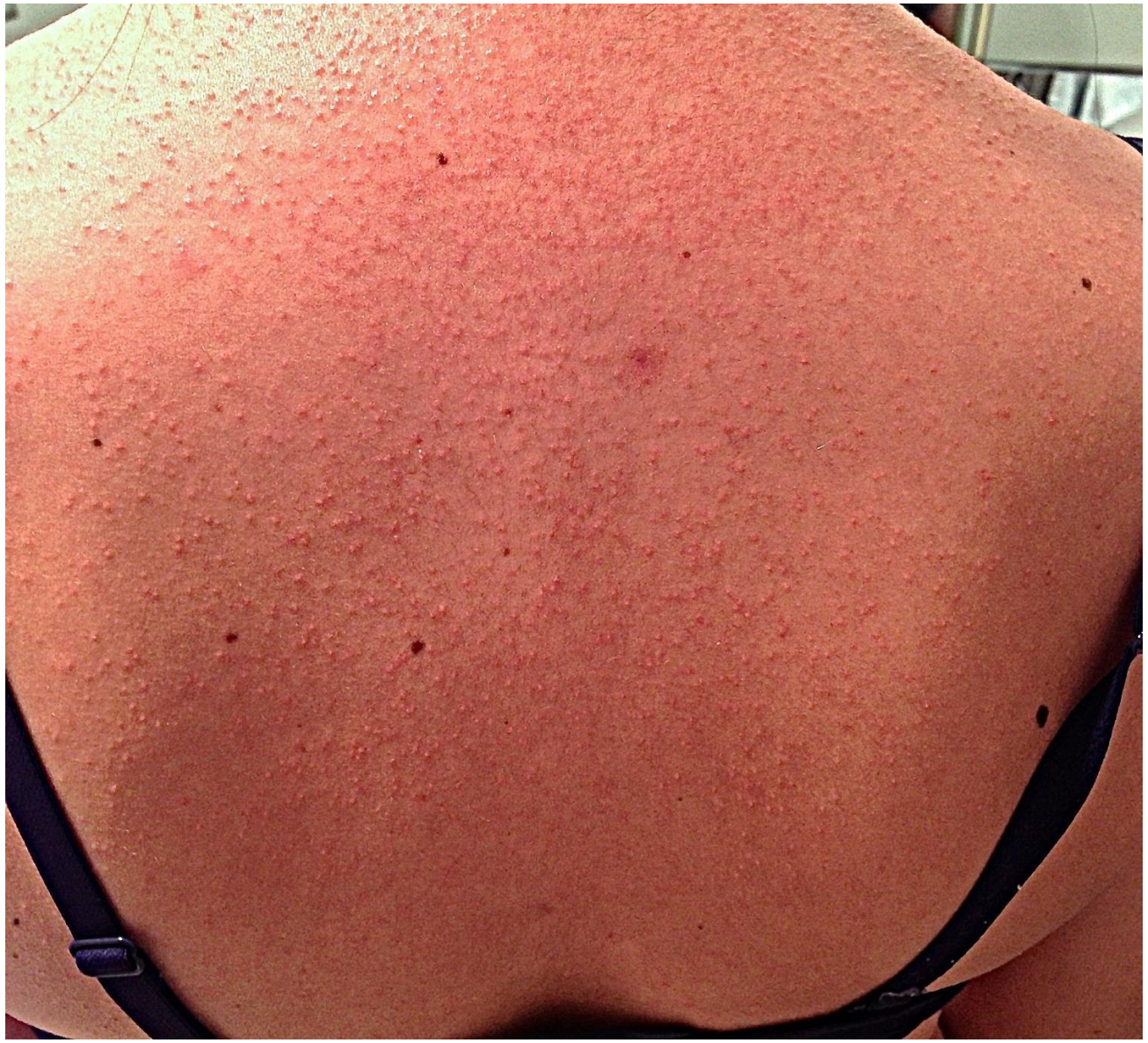
Notice:
All follicles are affected

Patient is young

Hot tub folliculitis:

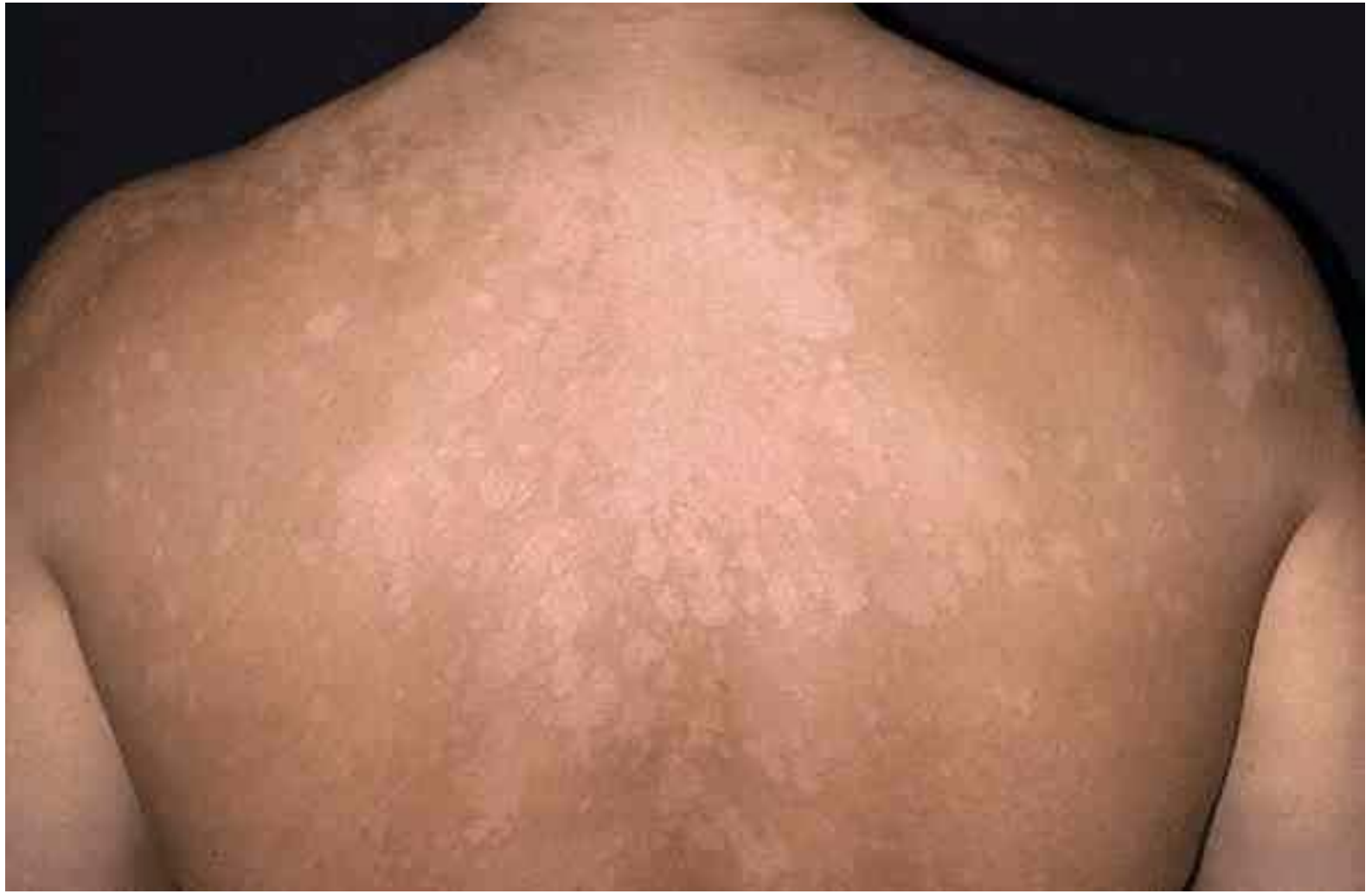
Usually self limited

In waters not sufficiently
Chlorinated and maintained
At 37 Celsius



Tinea veriscolor –*M. furfur*

Bleaching of the skin -> loss of pigment

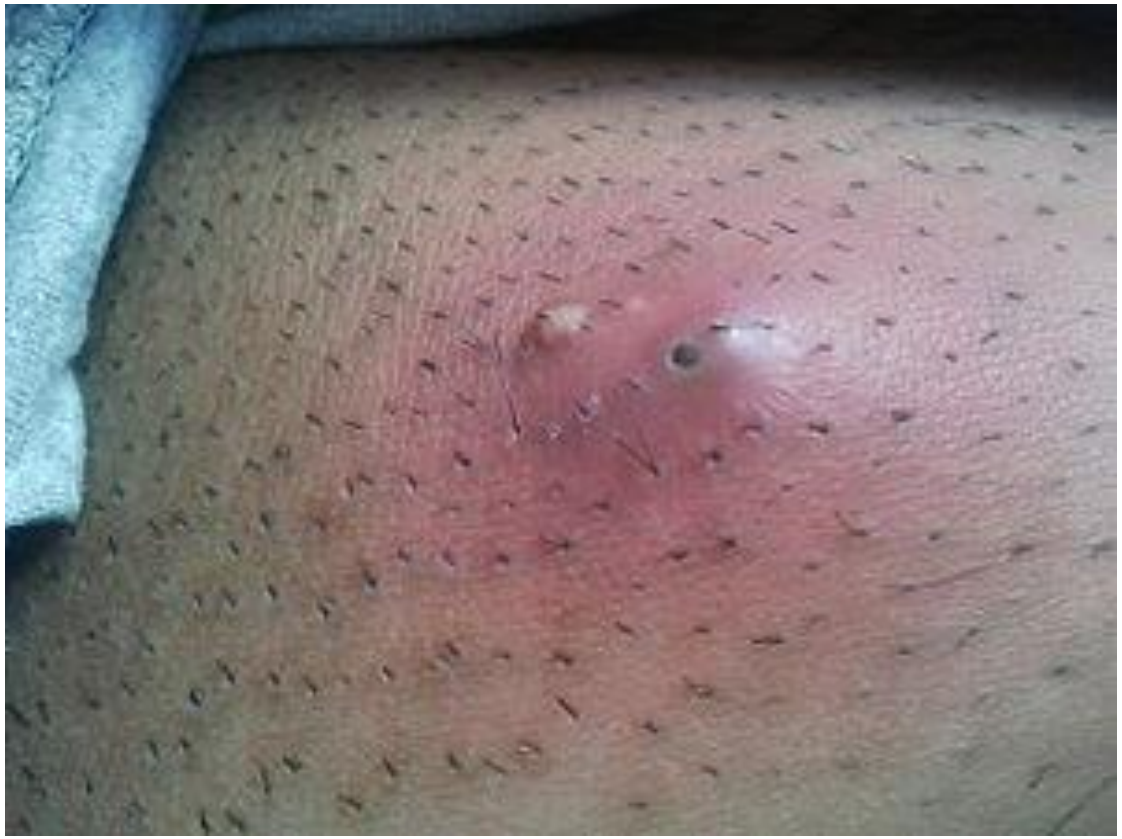


Cutaneous abscesses

- • 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 I&D ❼ Antibiotics are rarely necessary (except in extensive infection or systemic toxicity, or immunocompromised).

Notice:

raised lesion,
White head
Hair follicle might
Be port of entry



Furuncles and carbuncles

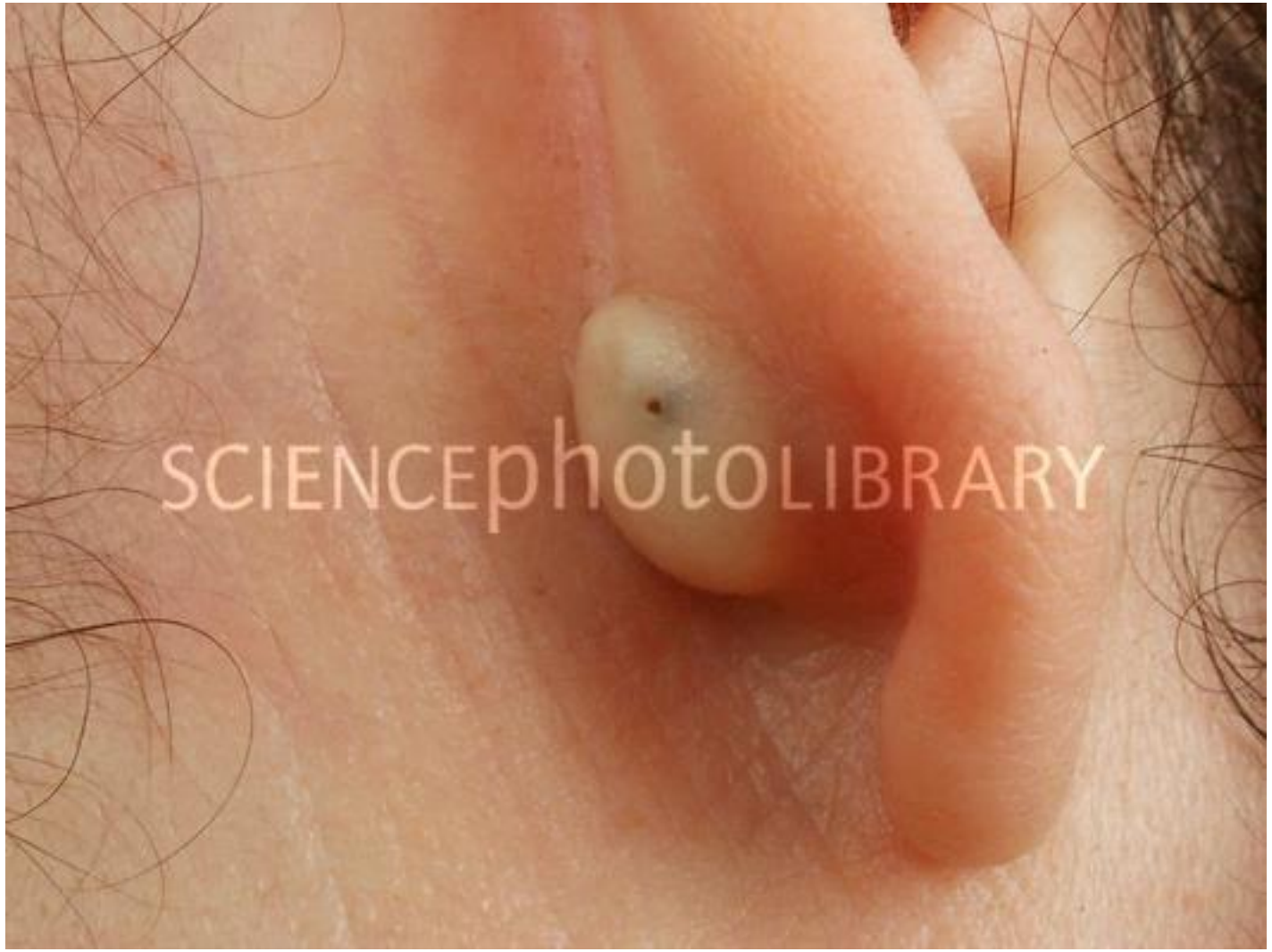
- • A furuncle (boil) is a deep inflammatory nodule that usually develops from preceding folliculitis.
- Occur in areas of the hairy skin, e.g. face, neck, axillae, and buttocks.
- • A carbuncle is a larger, deeper lesion made of multiple abscesses extending into the subcutaneous fat.
- Usually occur 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 promotes localization and spontaneous drainage.
- Large lesions require surgical drainage.
- Systemic antibiotics are indicated❼ *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.

Because the damage of the lymph node has already been done and it requires time to repair itself and then drain the tissue.

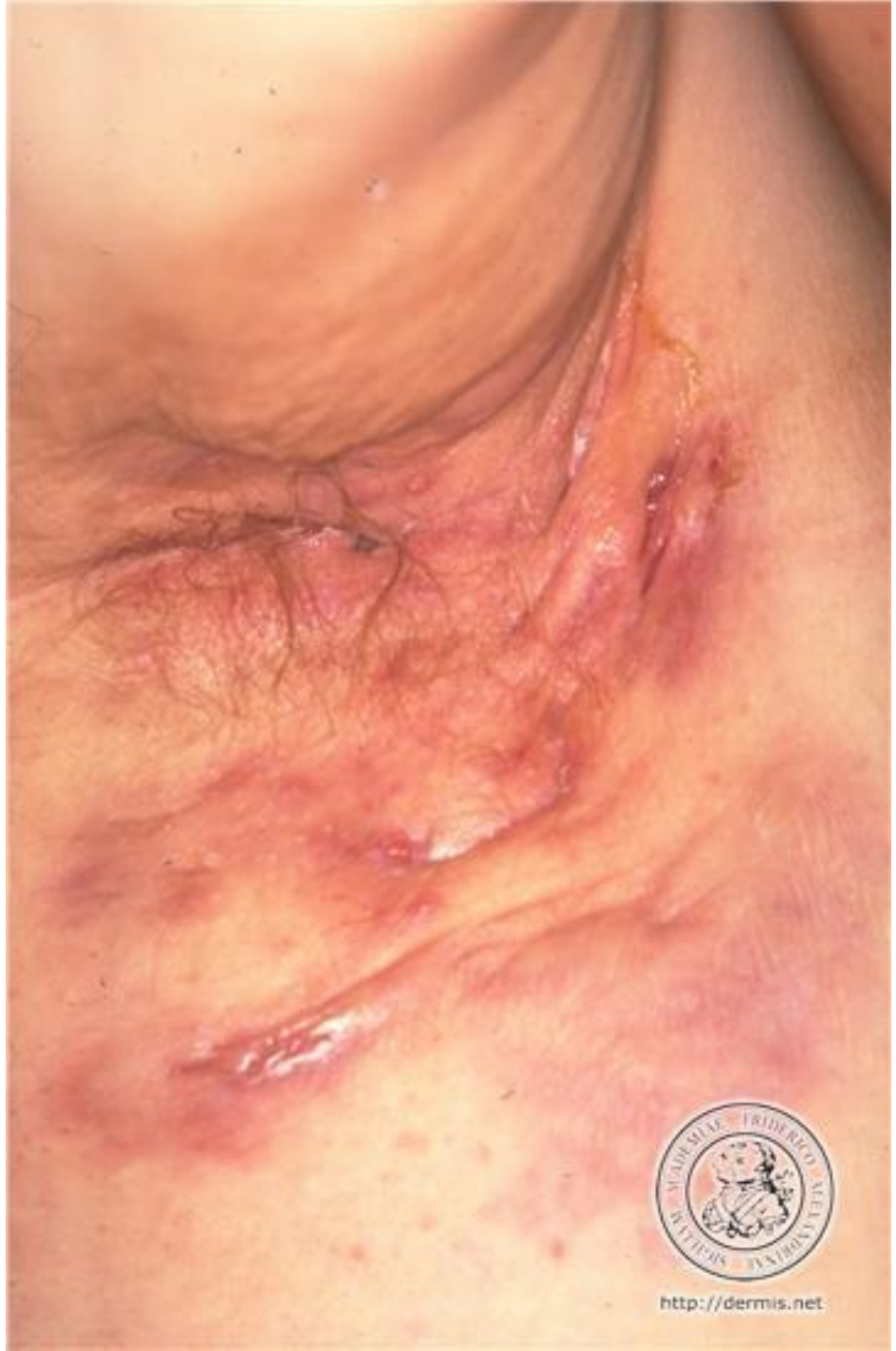
- Sebaceous glands that empty into the hair follicle maybe blocked and cause a swelling similar to an abscess (sebaceous cyst).
- Infection of sweat glands (hidradenitis suppurativa) can also mimic infection of hair follicles, particularly in the axillae.
- Chronic folliculitis is uncommon except in acne vulgaris, where constituents of the normal flora (e.g., Propionibacterium acnes) may play a role.



http://www.sciencephoto.com/media/531447/view

Hidradenitis Suppurativa

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



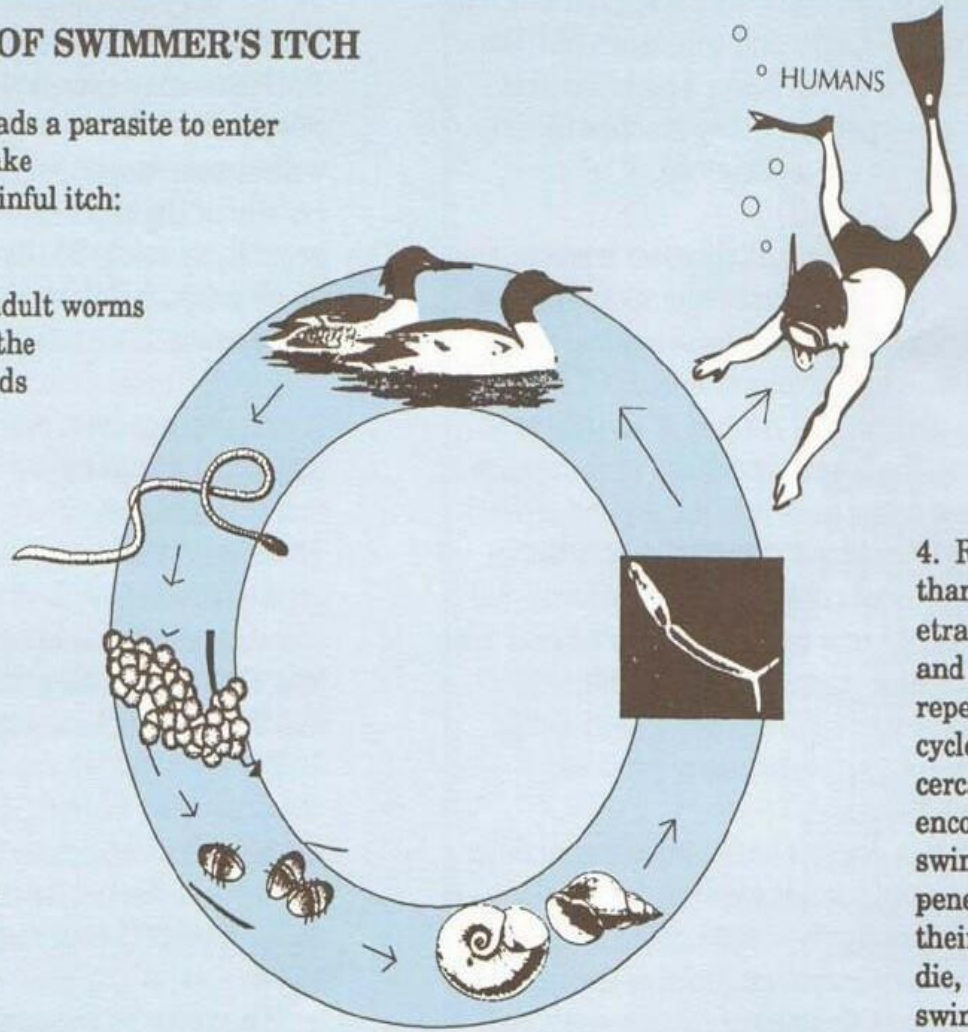
Swimmer's itch

- 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.

LIFE CYCLE OF SWIMMER'S ITCH

Here is the cycle that leads a parasite to enter the skin of vulnerable lake swimmers, causing a painful itch:

1. Flatworms become adult worms in veins that surround the intestines of certain birds and rodents. (Final Host Stage)



2. Female worms lay eggs that enter intestines and hatch when released into water through feces.

3. Eggs hatch into swimming larvae that enter snails. They elongate into germinating sacs that produce thousands of new parasites called cercariae. (IntermediateHost Stage)

4. Rather than penetrate birds and rodents, repeating the cycle, the cercaria may encounter swimmers, penetrate their skin and die, causing swimmer's itch.

https://www.google.com/url?sa=i&rct=j&q=&esrc=s&source=images&cd=&ved=2ahUKEwiG09qa6LHZAhWmsKQKHfYYDgwQjxx6BAgAEAI&url=http%3A%2F%2Flakeleelanau.org%2Fswimmers-itch%2F&psig=AOvVaw2an8V7ygIHZoLmPTWOqb5X&ust=1519124027122364

Erysipelas

Erysipelas	*S. pyogenes*

- 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.