Bacterial Infections of the Skin
Lecture 2

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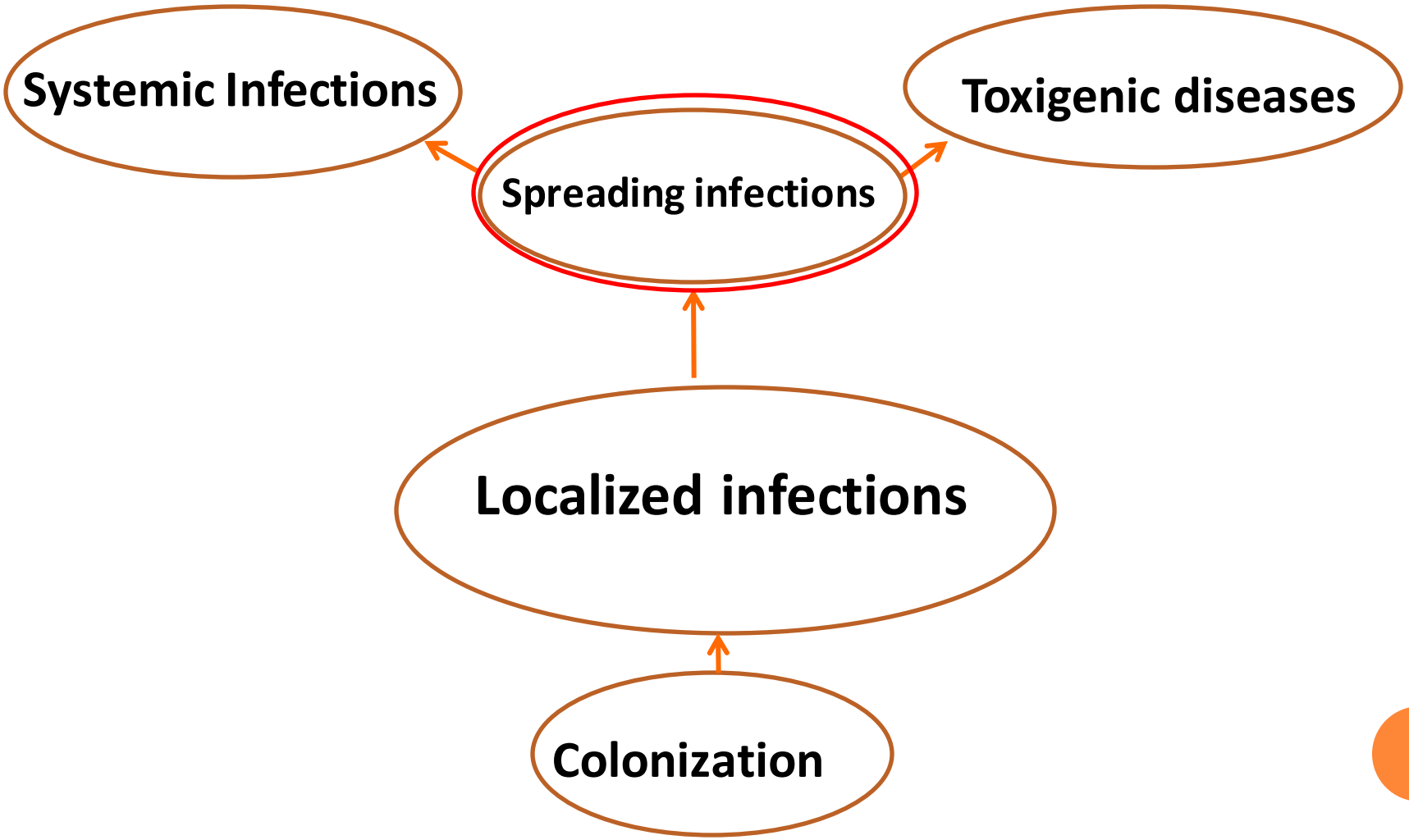
Levels of skin infections



Levels of skin infections

- **Spreading infections:**
  - Impetigo
  - Ecthyma
  - Erysipelas
  - Cellulitis

S. Pyogenes
(Some are caused by S. aureus)

- **Necrotizing fasciitis**

Streptococci

Pathogenic Streptococci

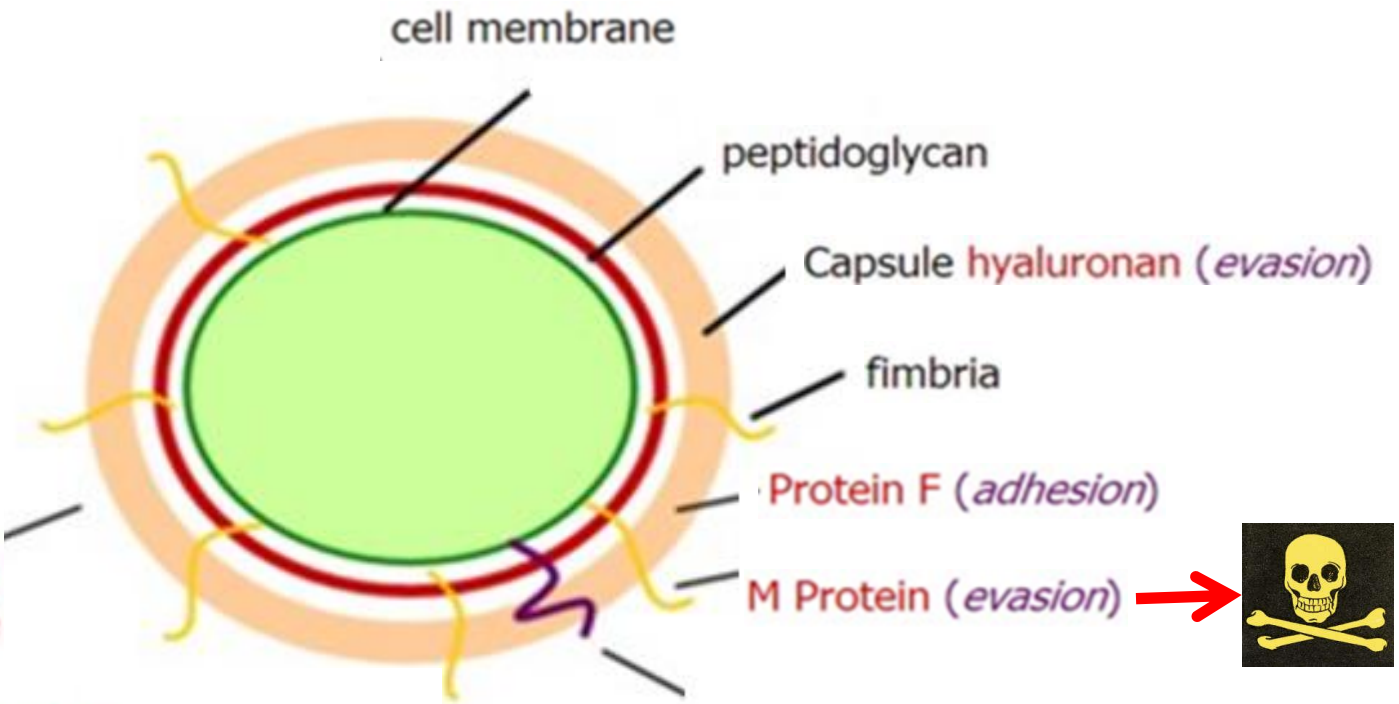
- *S. pyogenes (group A)*
- *S. agalactiae (group B)*
- Viridans streptococci
- *S. pneumoniae*
- *Enterococcus faecalis*

S. Pyogenes

- Group-A streptococci (GAS)
- β-hemolytic
- Most serious streptococcal pathogen
- Strict parasite
- Inhabits throat, nasopharynx, occasionally skin

S. pyogenes

Virulence factors

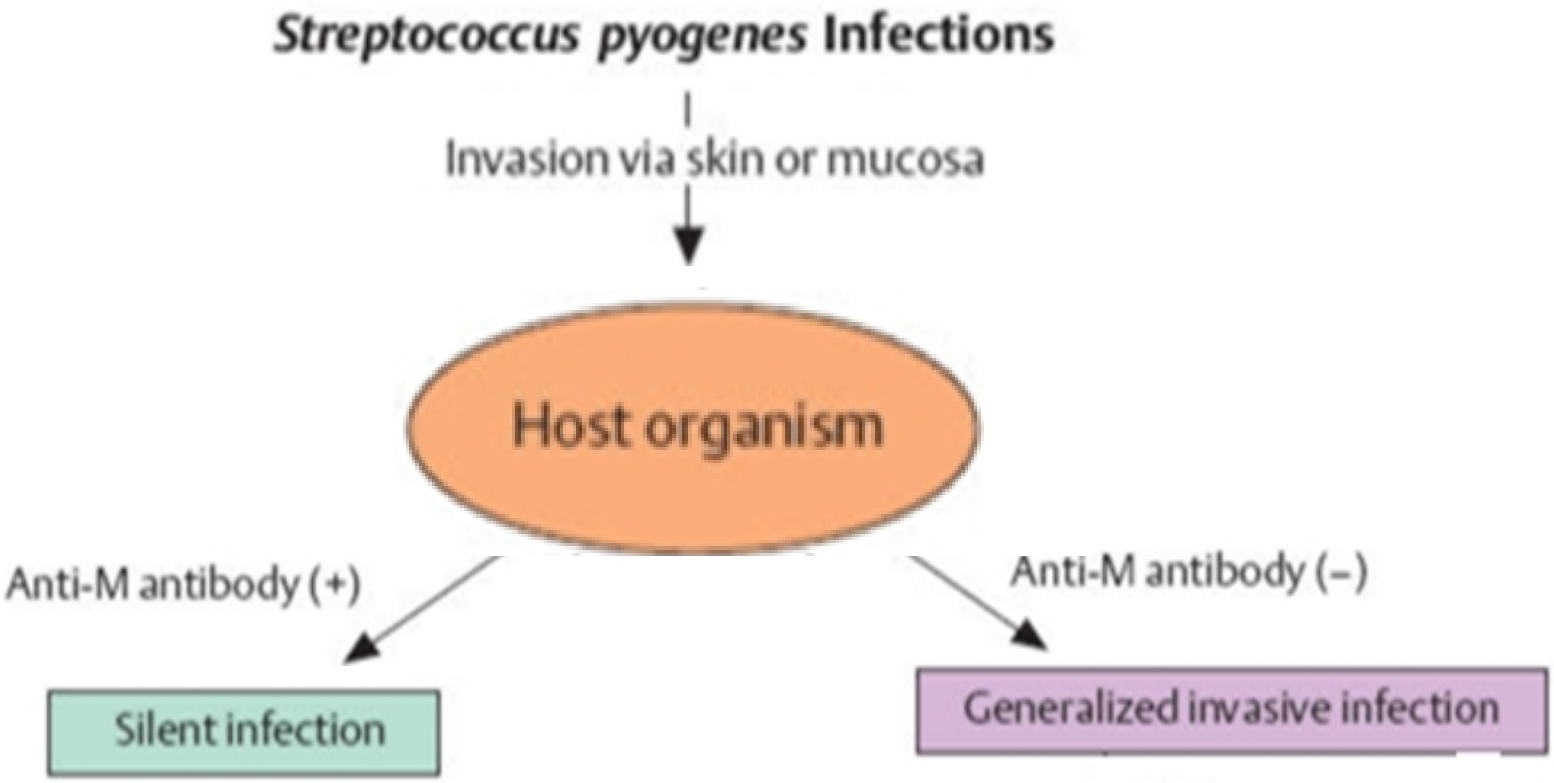


invasion:
Streptokinase
Hyaluronidase

evasion:
Streptolysin O and S
Streptodornase
C5a Peptidase
Streptococcal Chemokine Protease

toxin:
Streptococcal Pyrogenic Exotoxins

S. pyogenes


Streptococcus pyogenes Infections
Invasion via skin or mucosa
Host organism
Anti-M antibody (+)
Anti-M antibody (–)
Silent infection
Generalized invasive infection

Spreading skin infections

1- Impetigo

- Impetigo : eruption
- Called school sores
- Impetigo =Transient colonization + minor skin injury (ex. Insect bites)
- Most common in summer (increase insect numbers and the general hygiene is low)
- Contagious (if the fluid that oozes from the blisters touches an open area of the skin)

Spreading skin infections

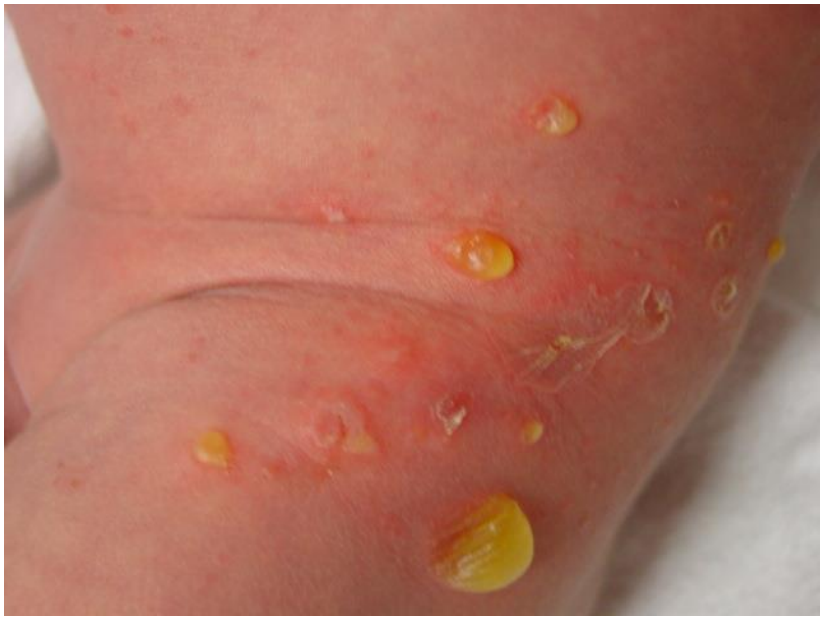
1- Impetigo

Features

- One or many vesicles
- The blisters become oozing , break, expose moist and red skin
- Plasma dry after few days forming crusted area
- In sever cases the infection invades deeper layer forming ecthyma
- Three types:
  - Bullous
  - Non-bullous
  - Ecthyma

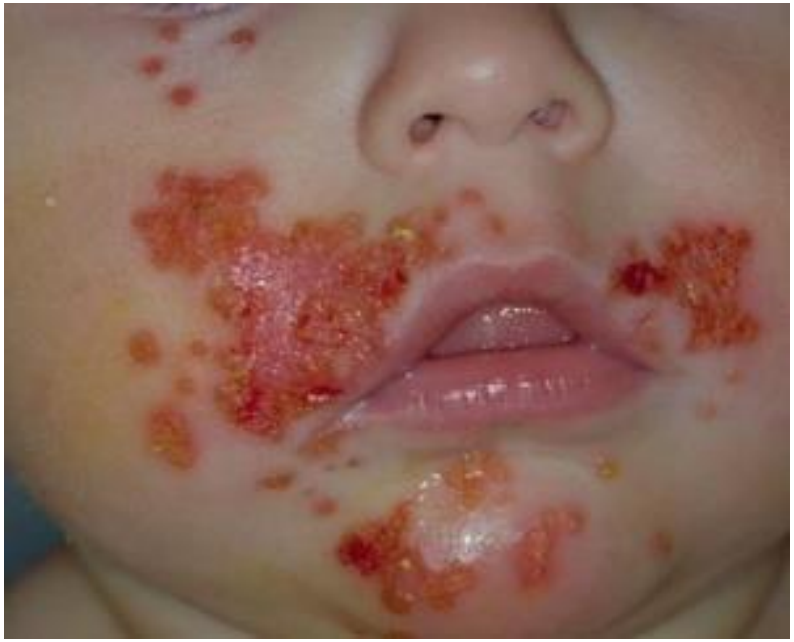
Spreading skin infections

Bullous Impetigo (S.aureus)

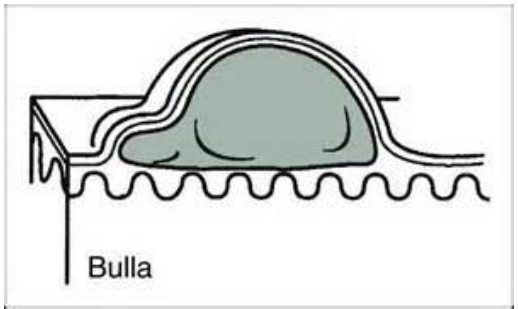


Bullous impetigo is a superficial skin infection that manifests as clusters of blisters that enlarge rapidly to form bullae. The bullae burst and expose larger bases, which become covered with honey-colored crust.
(Localized scalded skin syndrome)

Non-Bullous Impetigo (*S. pyogenes*)

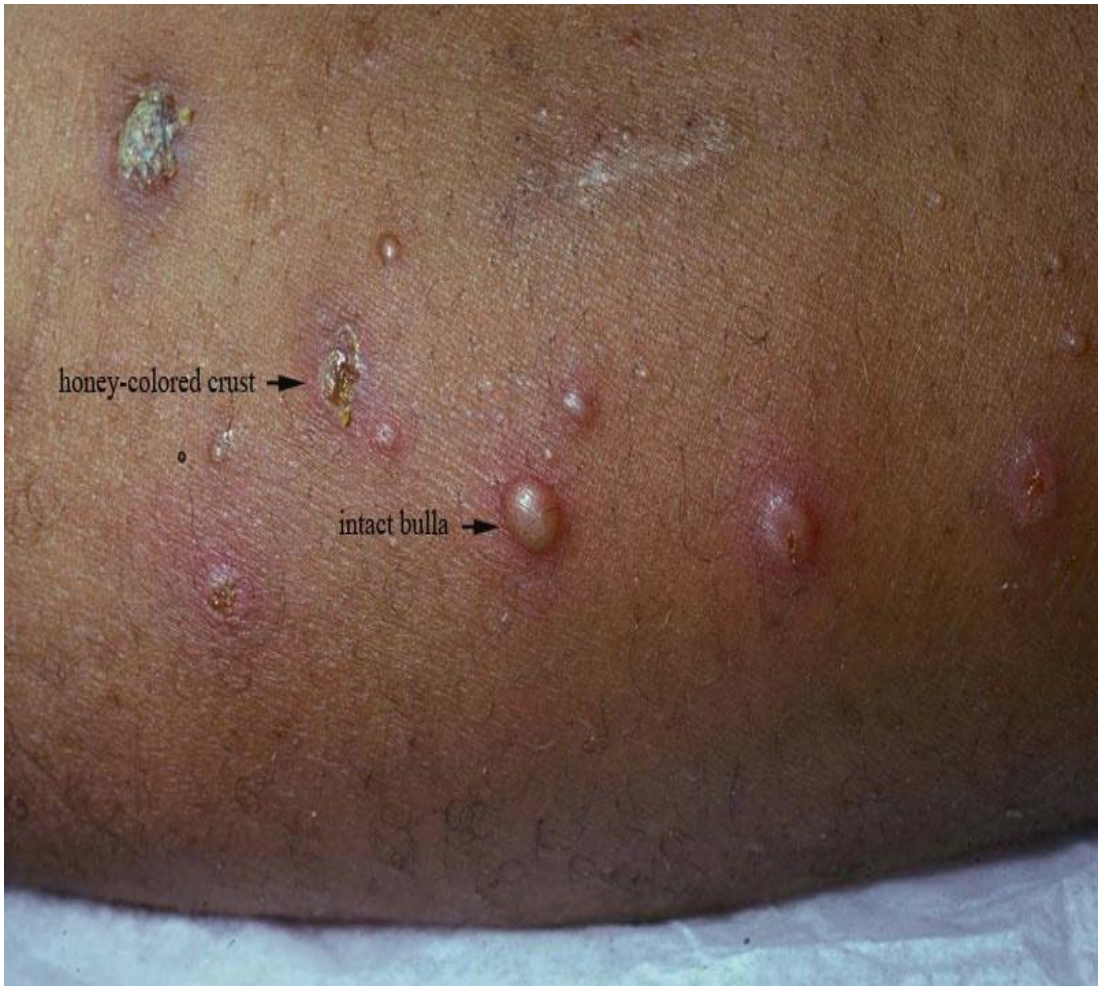


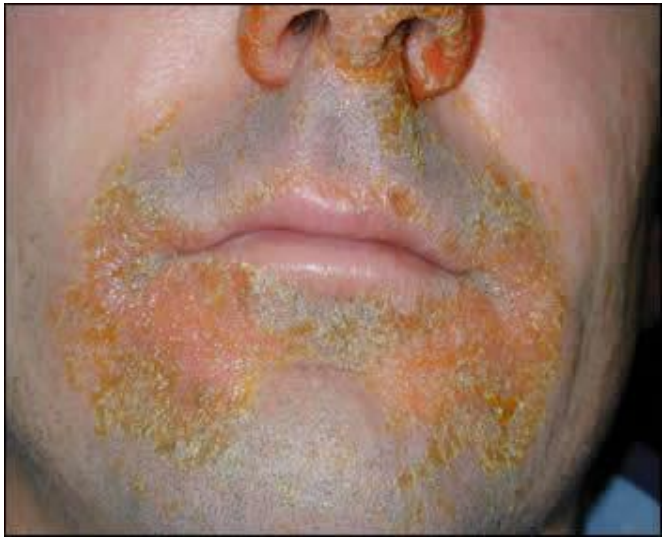
Similar to bullous except that blisters are slight and transient

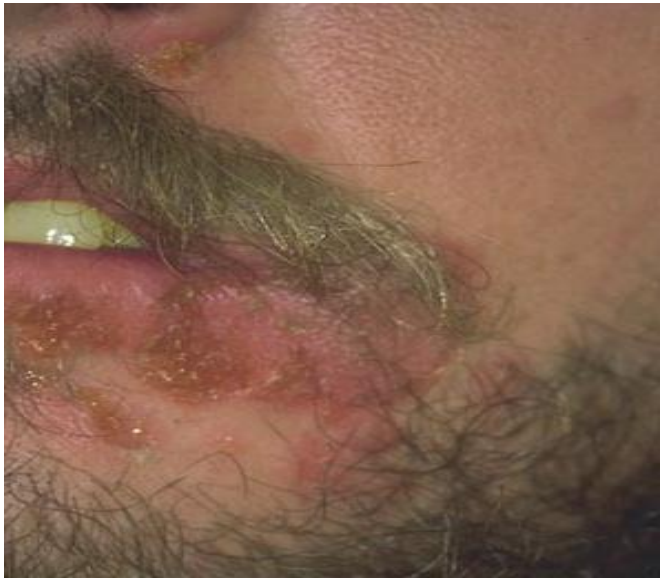


Spreading skin infections

Impetigo







Spreading skin infections

Impetigo

Diagnosis

Diagnosing impetigo is generally straightforward and based on the clinical appearance

Treatment

- Local antibiotics in mild and localized infection

- Removal of infected crusts and washing with soap and water help in treatment

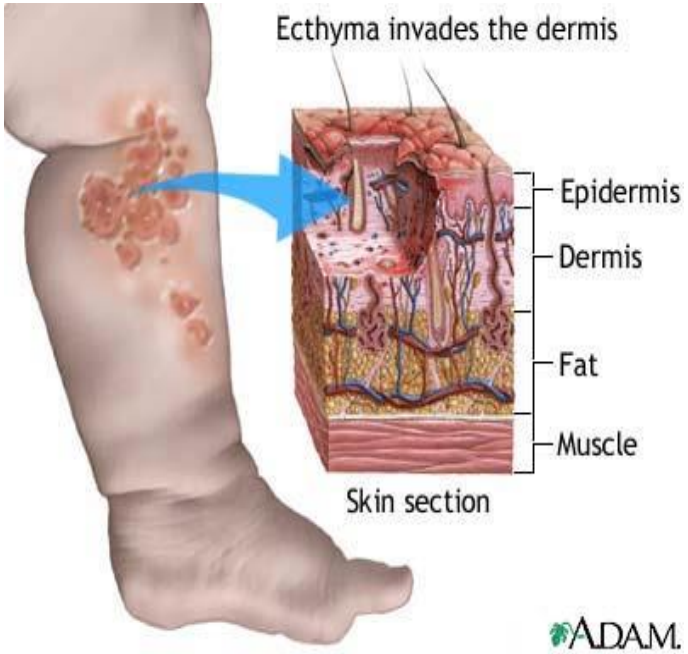
- In widespread sever infection or when accompanied by lymphadenopathy an oral antibiotic is recommended

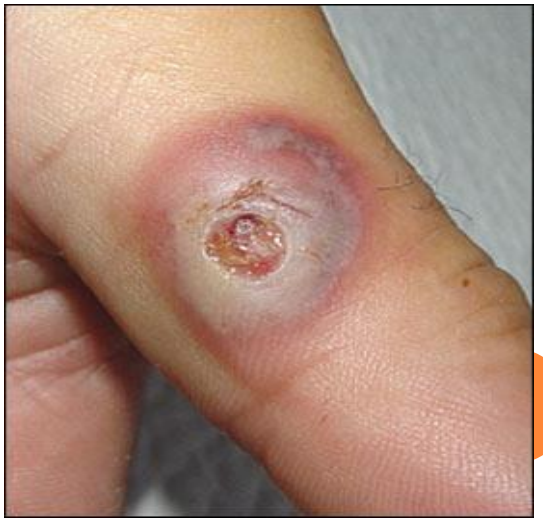
S. pyogenes

Spreading skin infections

Ecthyma

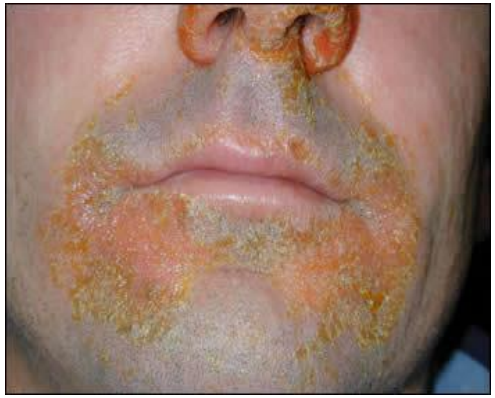
- Ecthyma is a skin infection similar to impetigo, but more deeply invasive to the deeper layer (dermis)
- The crust is much darker, thicker than of impetigo beneath which ulceration occurs
- Healing occurs after a few weeks with scars
- Treated by improved hygiene and antibiotics covering staphylococci and streptococci

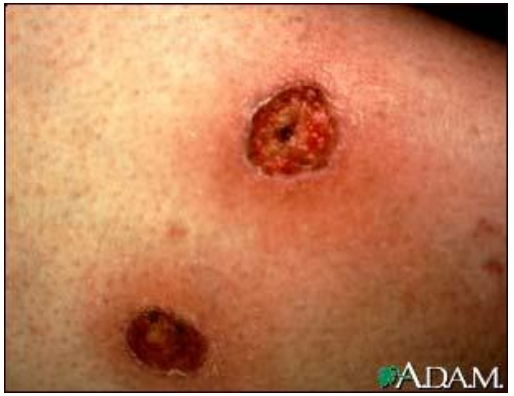




Spreading skin infections

Ecthyma vs. Impetigo





Impetigo (staph.aureus and strep.pyogenes)	Ecthyma (strep pyogenes)
superficial	deep
Honey or varnish color crusting	Chocolate color crusting
Crust is loosely adherent	Crust is tightly adherent
No ulcer on removal of crust	Ulcer on removal of crust
Common site: face	Common site: lower limbs
Heals without scarring	Heals with scarring

Spreading skin infections

Cellulitis and Erysipelas

Definition

Erysipelas (Red skin, Holy fire):

- Rapidly spreading painful bacterial infection of the dermis
- Blocking of dermal lymphatics and presents as well–defined spreading, edematous erythematous inflammation
- Erysipelas are caused by *S. pyogenes.*
- Symptoms including high fevers, shaking , chills, headaches, vomating, and general illness within 48 h of the initial infection.

Cellulitis:

- Cellulitis and erysipelas are similar. The main difference is that cellulitis affects the dermis and the layer of fat just underneath it. Doctors can't always distinguish between them and they are both treated in the same way
- Cellulitis is caused by *S. pyogenes* and less commonly *S. aureus*

Spreading skin infections

Both Cellulitis and Erysipelas

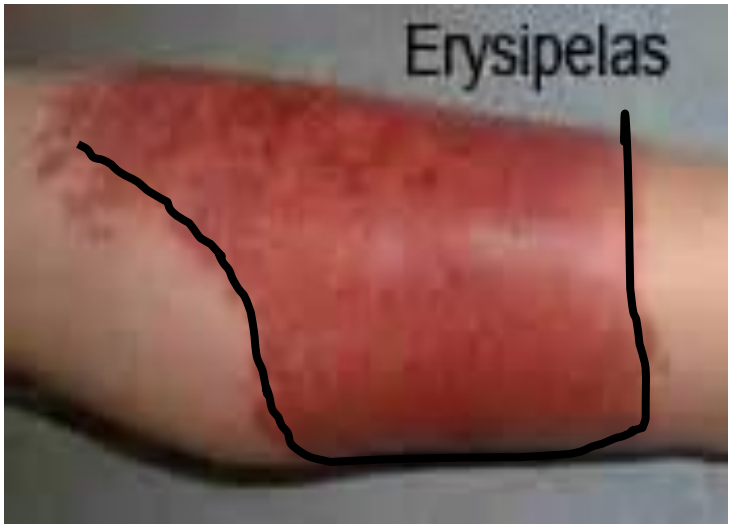
- Infections can enter the skin through minor trauma, insect bites, dog bites, eczema, athlete's foot, surgical incisions and ulcer
- The leg is commonest site because it is exposed to skin injuries
- The face is the second most frequent site (butterfly distribution on the cheeks and bridge of the nose)

Spreading skin infections

Cellulitis and Erysipelas

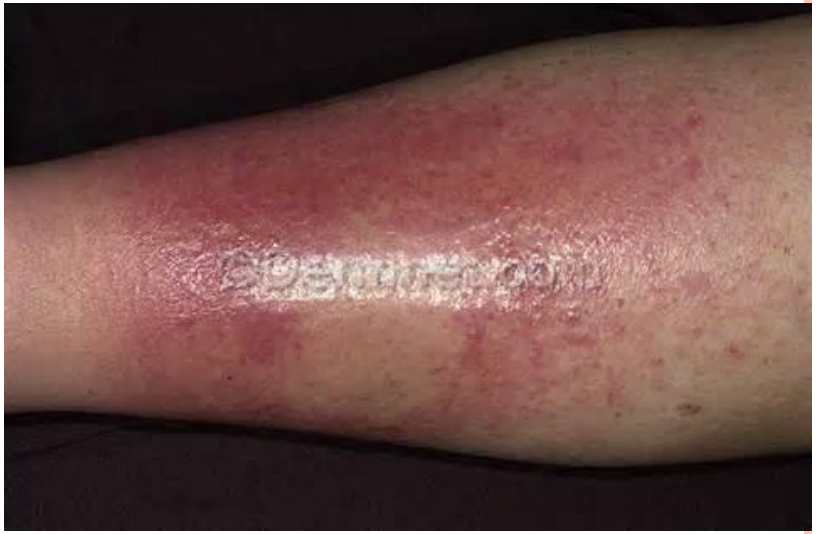
Clinical features

Erysipelas



The main feature is a well defined raised edge (demarcated) reflecting the more superficial (dermal) involvement.

cellulitis



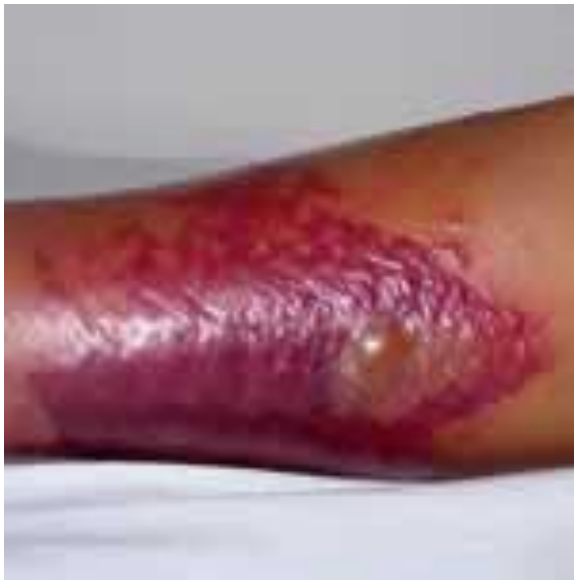
In cellulitis it is diffuse not demarcated edge.

Bacteria is carried away from the site of infection by the proteolytic enzymes as Pyrogenic exotoxin B produced By the bacteria itself in addition to the role of lymphatic vessels in the site of infection

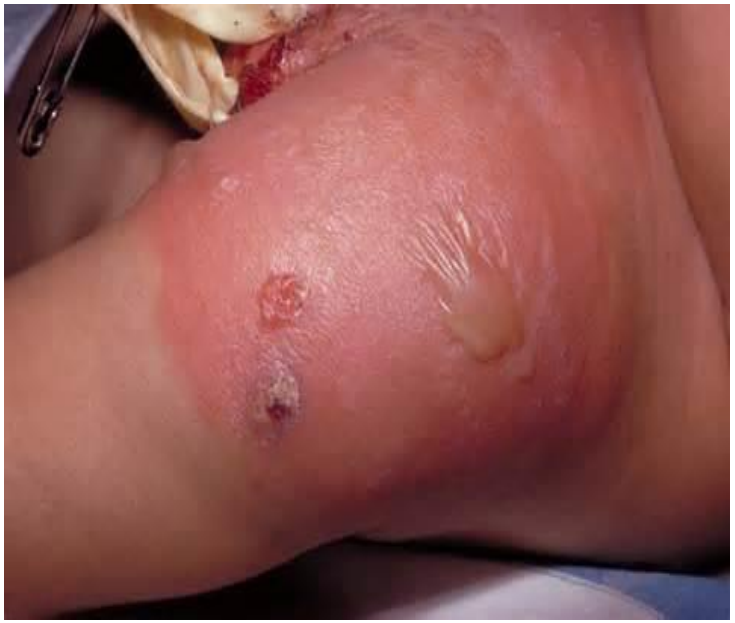
Spreading skin infections

Cellulitis and Erysipelas

Clinical features



In erysipelas blistering is common associated with hemorrhage into the blister or in intact skin.



Sever cellulitis may show bullae and can progress to dermal necrosis. Frequent Lymphangitis and lympadenopathy.

Spreading skin infections

Cellulitis and Erysipelas

Complications

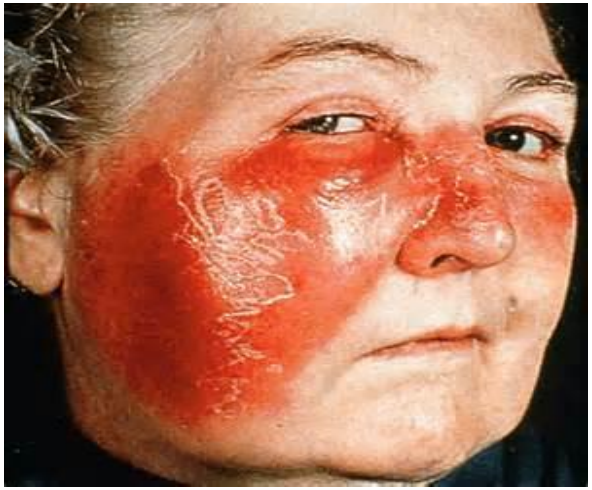
Without treatment = fasciitis, myositis, subcutaneous abscesses, and septicemia.

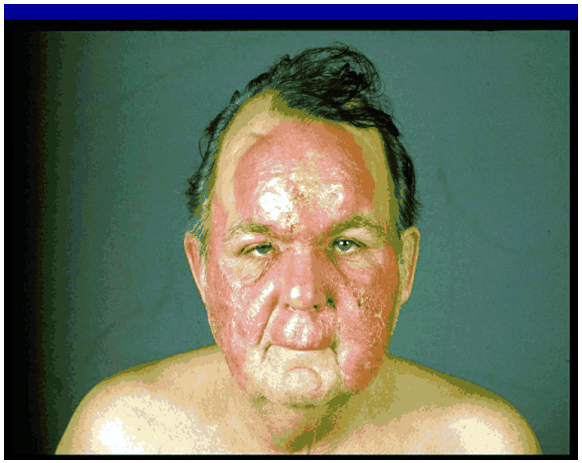
Treatment

- Should be treated with antibiotics that cover *S. pyogenes* and *S. aureus.*
- Oral or intravenous penicillin is the antibiotic of first choice.
- Vancomycin is used for facial erysipelas caused by MRSA.
- Treatment is usually for 10–14 days

Spreading skin infections

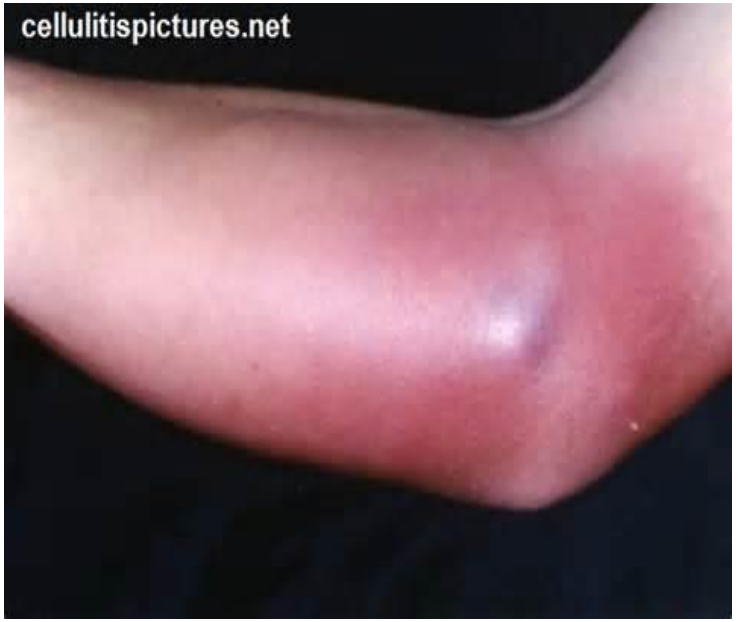
Erysipelas



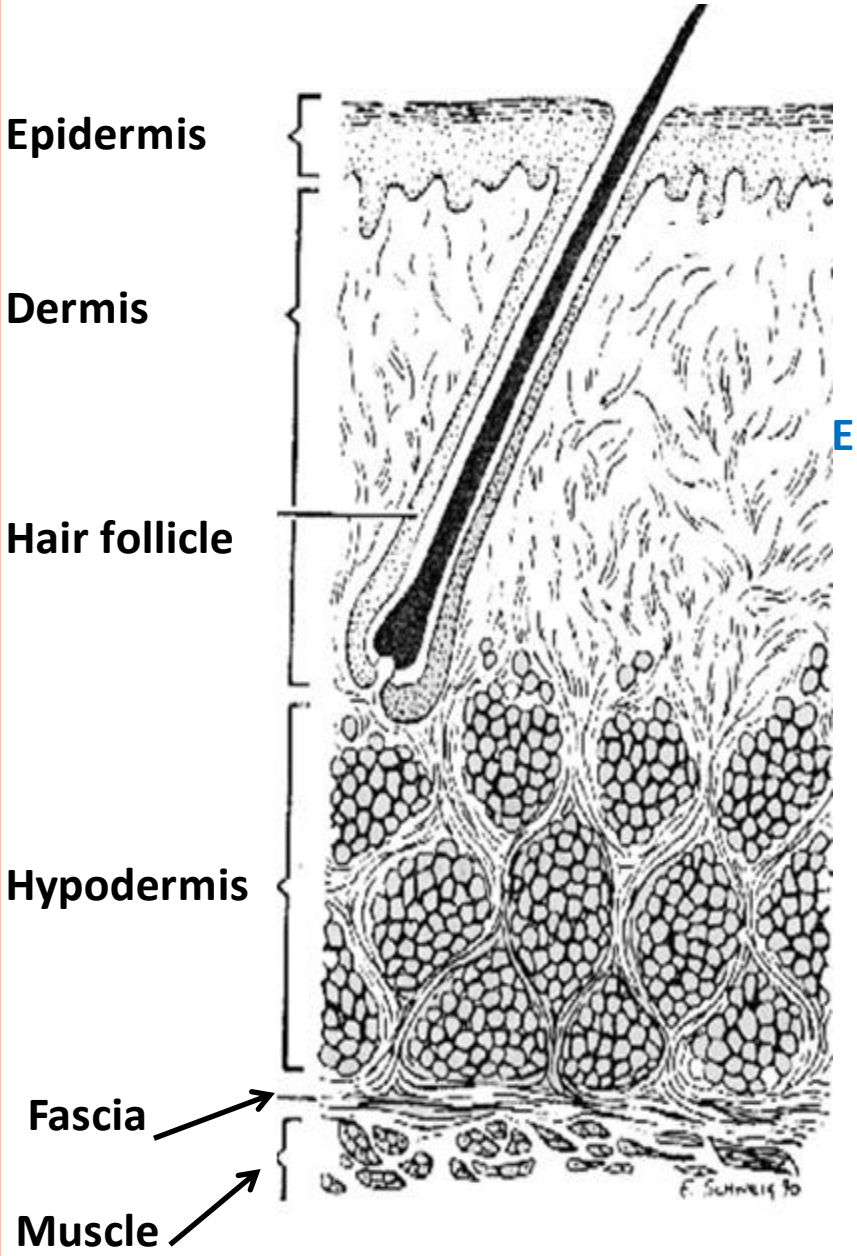


Erysipelas is characterized by shiny, raised, indurated, and tender plaque-like lesions with distinct margins.

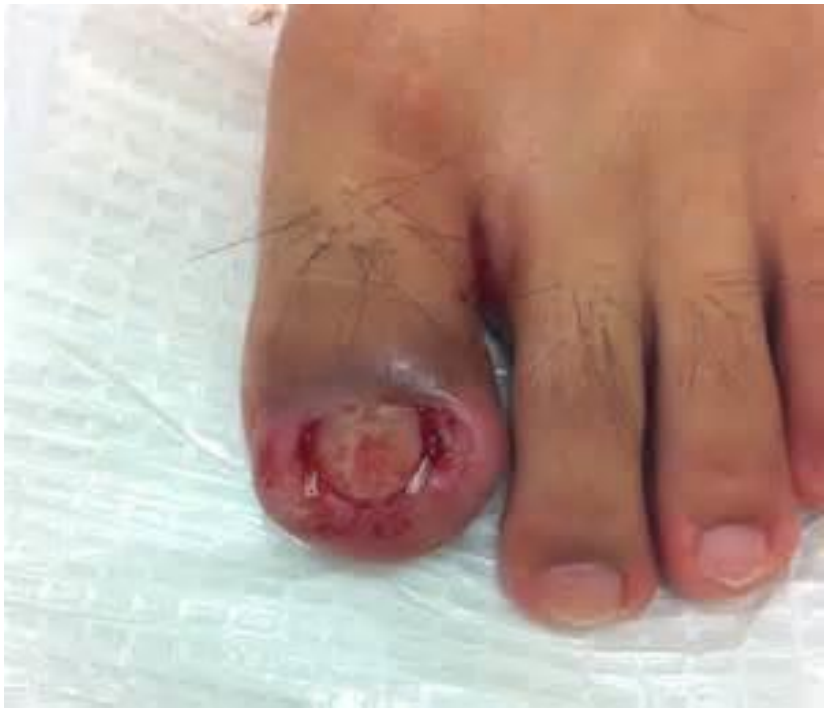
Cellulitis

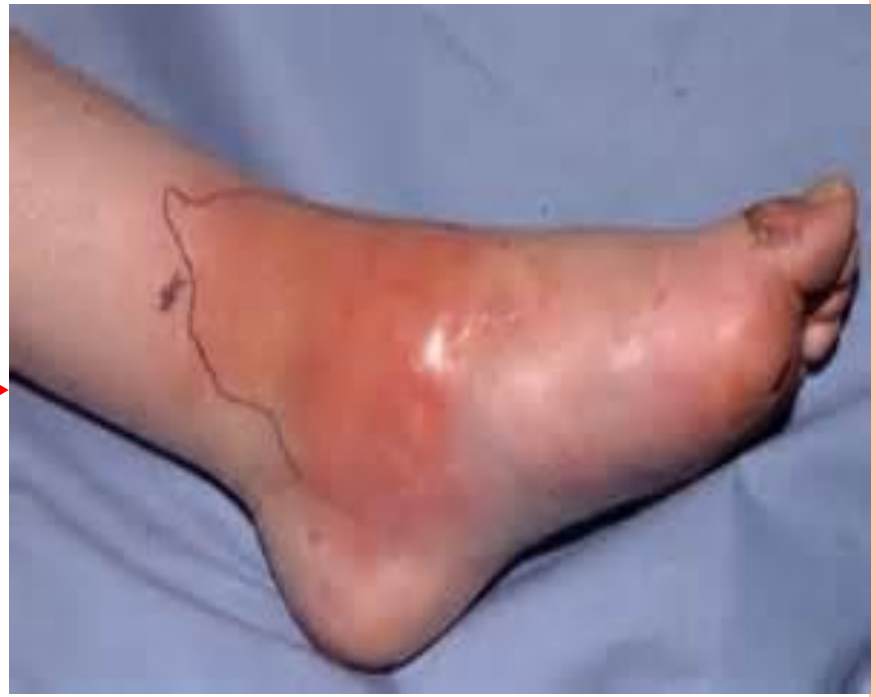


Spreading skin infections



Infection site	Etiologic agent
Impetigo	S. aureus, S. pyogenes
Ecthyma	*S. pyogenes*
Erysipelas	*S. pyogenes*
Cellulitis	*S. Pyogenes* (common) *S. aureus* (uncommon)





Case (cellulitis)

27 years old man was seen for an infection around his toe nail (paronychia). The lesion was drained and the pus cultured and gave beta-hemolytic *S. pyogenes*. The patient was not given antibiotic because the physician believed that the drainage was sufficient. Five days later the patient complained of fever and sever pain in his foot, which had become erythymatous and swollen. His temperature was 40.2C, sweaty and hot.

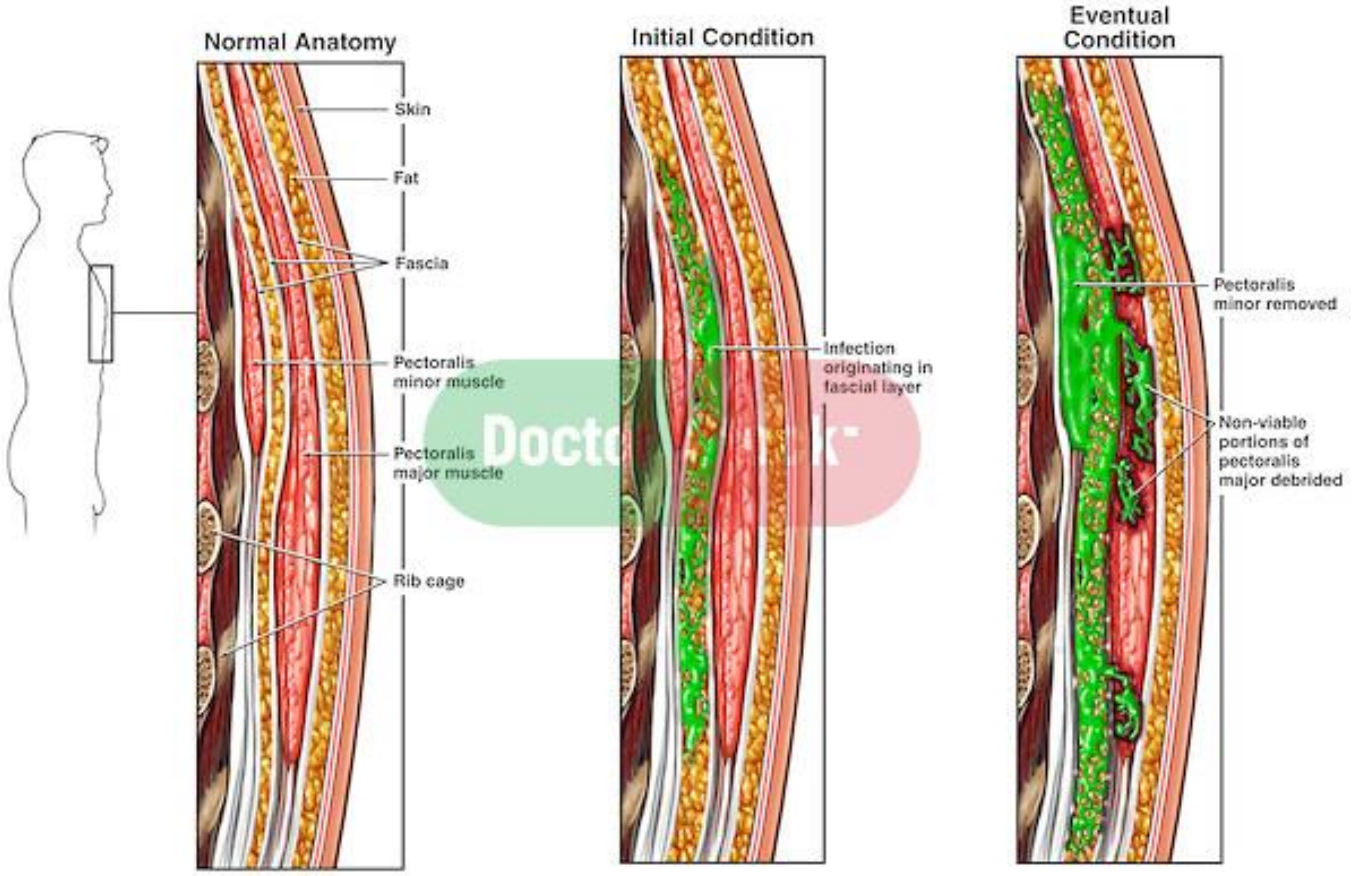
- The patient then was admitted to hospital and diagnosed of Strptoccocal cellulitis
- He was treated successfully with penicillin. Before starting therapy the blood culture also yielded *S. pyogenes*

S. pyogenes

Spreading skin infections

Necrotizing fasciitis (NF)

- Necrotizing = death of tissue
- Sever Bacterial infection that spread rapidly through superficial and deep fascia



S. pyogenes

Spreading skin infections

Necrotizing fasciitis

- Incidence 1-20/100,000
- 30-70% mortality
- Causative agents:
  - monobacterial (S. pyogenes)
  - polymicrobial (candida, E. coli, Klebsiella, vibrio vulnificus,….)
  - gas gangrene (Clostridium perfrengenes)
- **Treatment:**
  - Prompt IV anitibiotic treatemnt
  - Depridmrnt or amputation is mandatory
- **Diagnosis**:
  - Gram stain fron exudate
  - Biopsy
  - Culture and antibiotic sensitivty

S. pyogenes

Spreading skin infections

Necrotizing fasciitis

Indications of Necrotizing Fasciitis

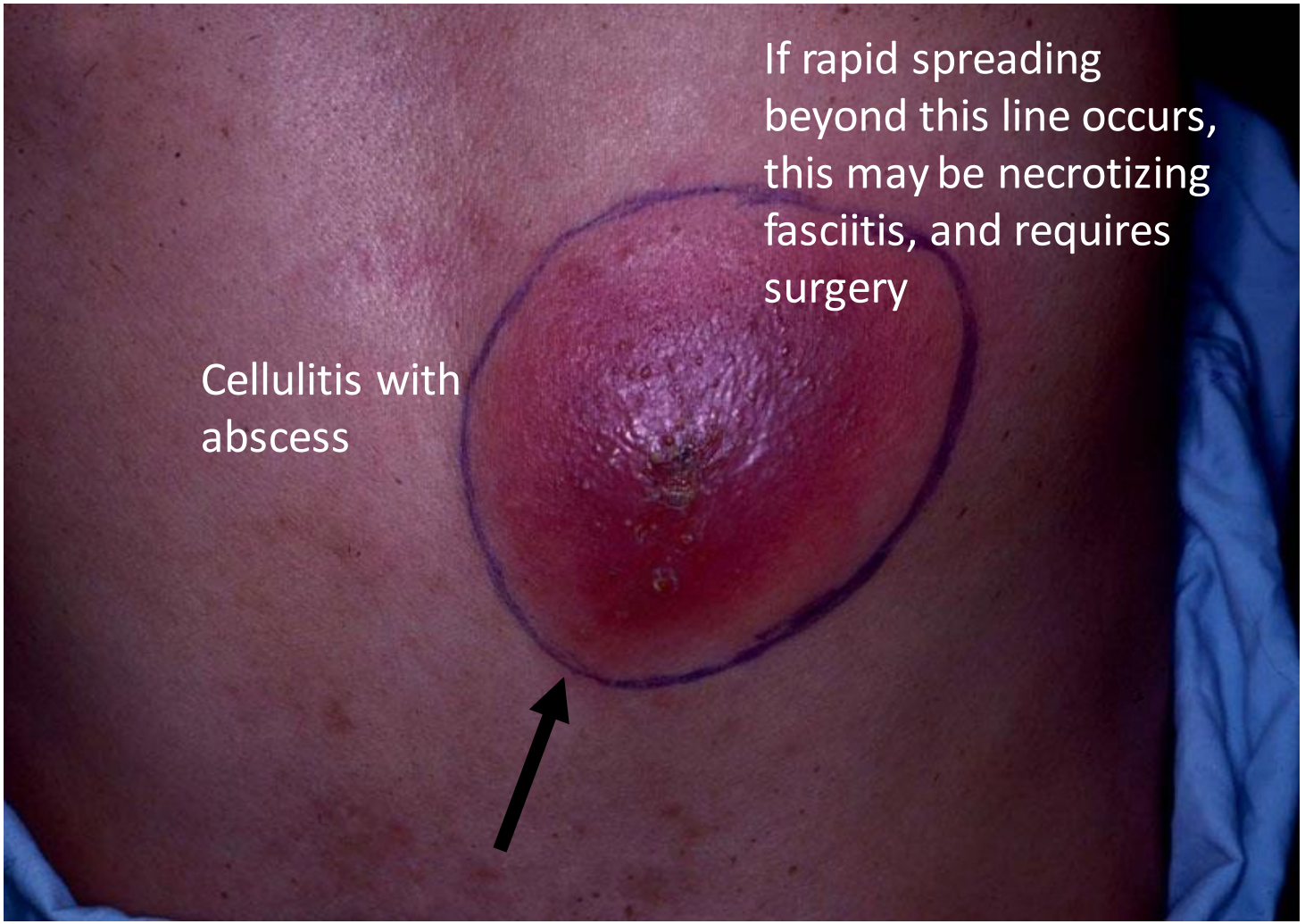
- Rapid spreading of redness area (≥ ½ inch per hour) this may be “Necrotizing Fasciitis”
- If the area is extremely painful
- If the person shows signs of bacteremia (fever, change in mental function such as delirium, profound weakness)
- Draw a line around the red area with a pen, then watch for spreading beyond the line

S. pyogenes

Spreading skin infections

Necrotizing fasciitis

Indications of Necrotizing Fasciitis



S. pyogenes

Spreading skin infections

Necrotizing fasciitis
Symptomes

- The patient develops fever, malaise, and myalgias

- Extreme pain and tenderness over the involved skin and underlying muscle which is the hallmark symptom

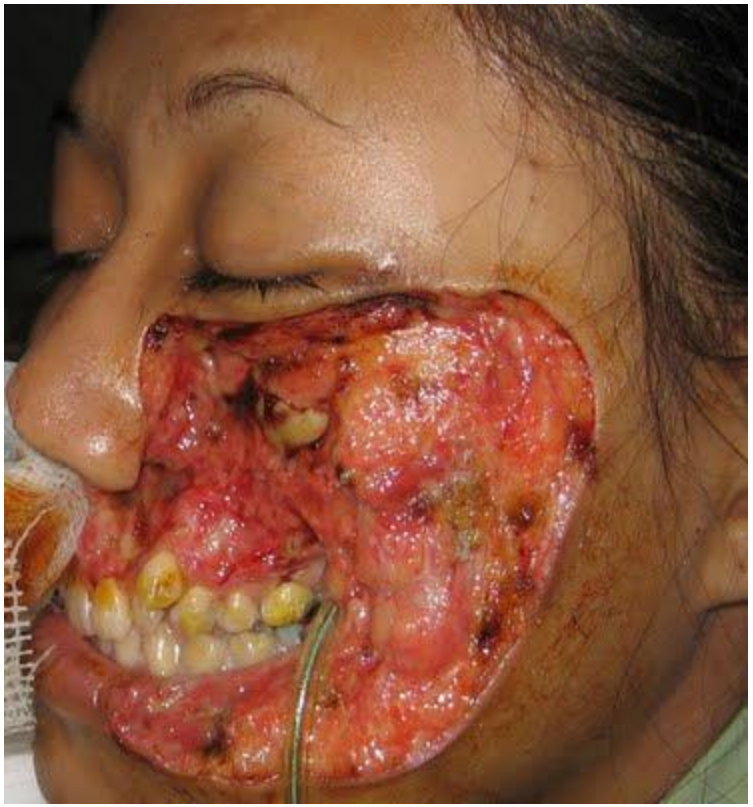
- The intensity of the pain often causes suspicion of a torn or ruptured muscle

- Over the next several hours to days, the local pain progresses to anesthesia

S. pyogenes

Spreading skin infections

Necrotizing fasciitis



Acne

Propionibacterium

- Classification
  - Two species *P. acnes and P. granulosum*.
  - Are described as anaerobic diphtheroids, though some can grow in CO_2.
  - Most clinical isolates are *P. acnes* which is part of the NF of skin.
- Morphology and cultural characteristics
  - Pleomorphic, small G+B, may have Chinese letter configurations or may be branching.

Propionibacterium

- Grow well on CBA, producing tiny translucent to opaque and white to gray colonies.
- Growth may be slow.
- Anaerobic, though occasional strains of *P. granulosum* grow in CO_2

- Biochemistry
  - Catalase +
  - Indole +/-
  - Ferment glucose
  - Produce caseinase

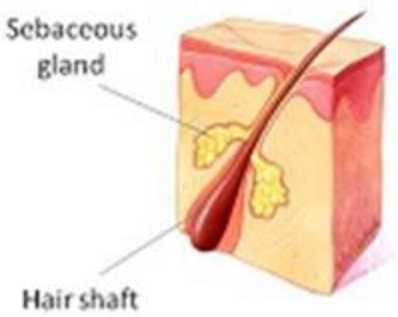
Propionibacterium

- Virulance factors
  - Protease
- Clinical significance –
  - Is part of skin NF
  - Has been implicated in causing acne –
    - During adolescence more sebum is produced, and *P. acnes* metabolizes it to produce fatty acids.
    - These may contribute to the inflammatory response seen in acne.
  - Has also been isolated from joint infections

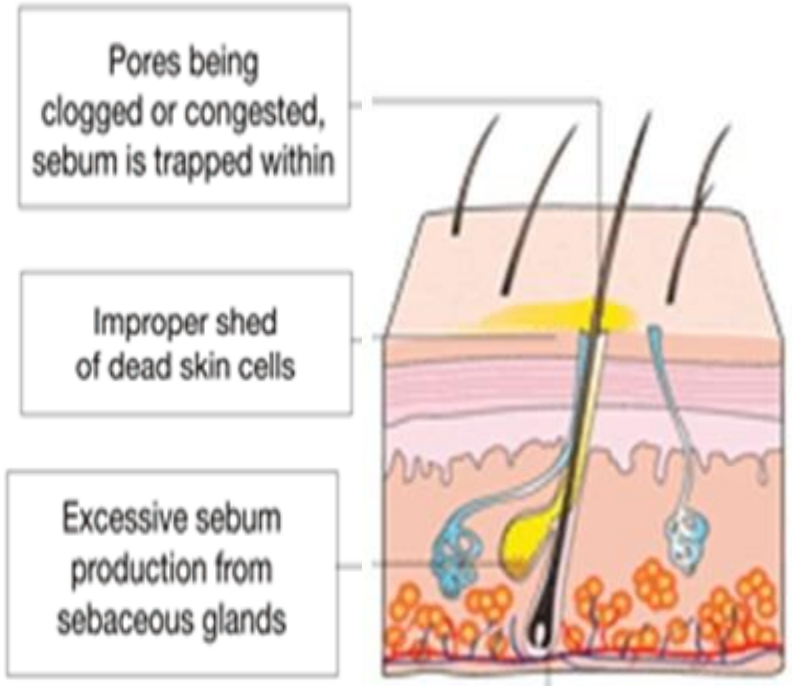
Propionibacterium

- Antibiotic susceptibility/treatment
  - Tetracycline
  - Acutane – inhibits sebum formation and is only used in severe cases of acne because there are many side effects.

Pathophysiology of acne



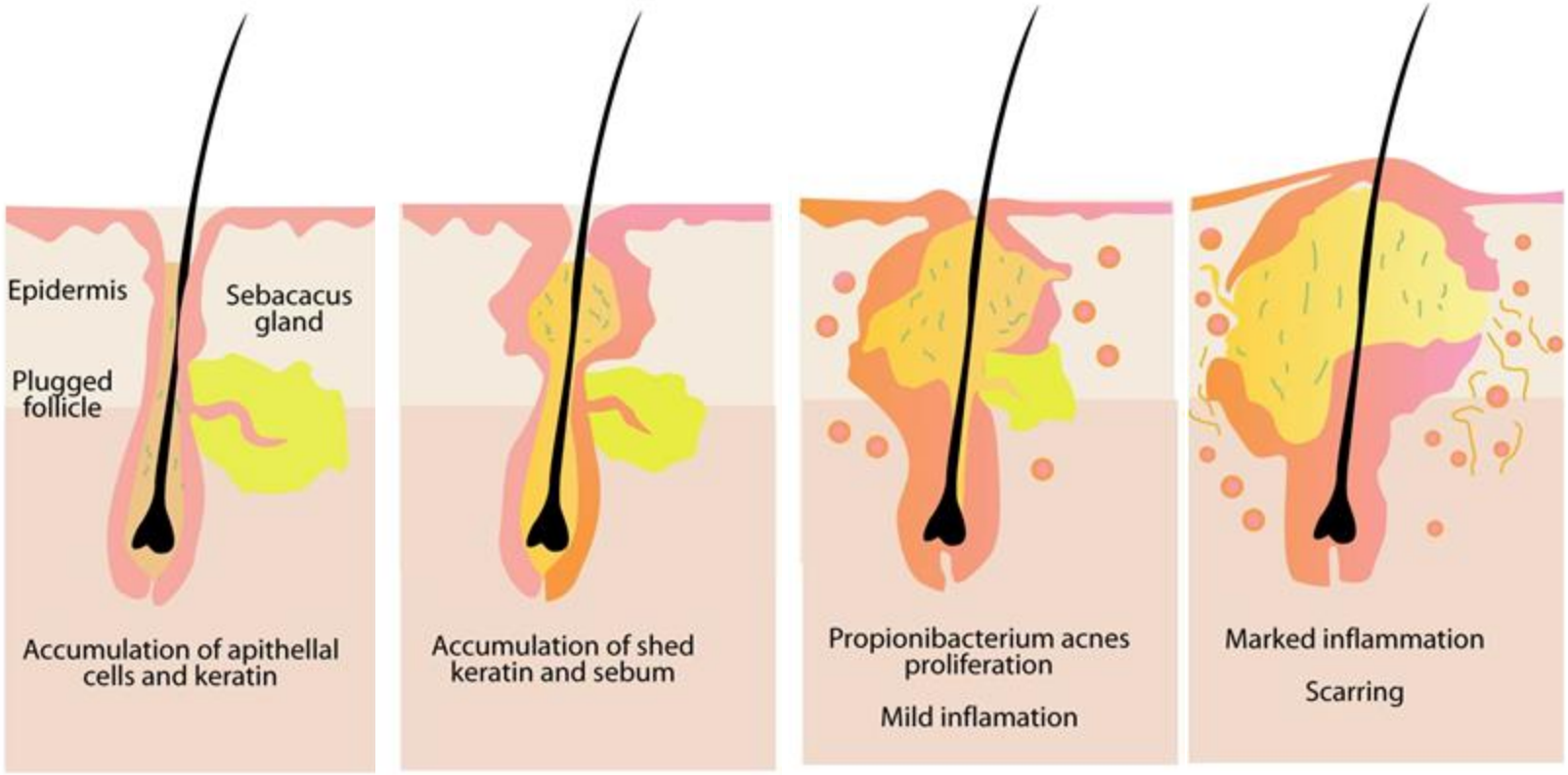
Before puberty



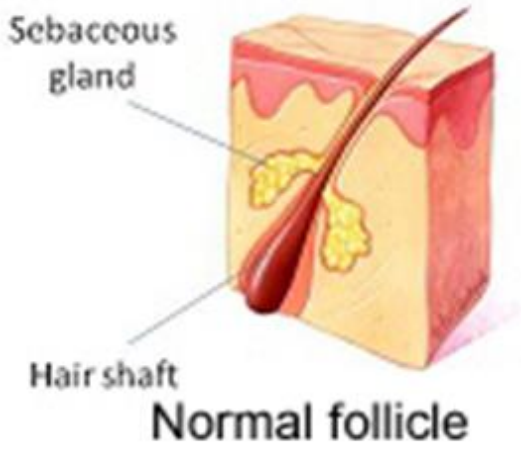
After puberty

The production of sebum is under the control of androgens. During puberty the androgens stimulate the production of sebum plus increased keratinization and desquamation in sebaceous duct. This causing blockage of ducts and this turns the gland as a sac for the multiplication of *P. acnes* and other flora (yeast, staph, micrococcus).

Acne

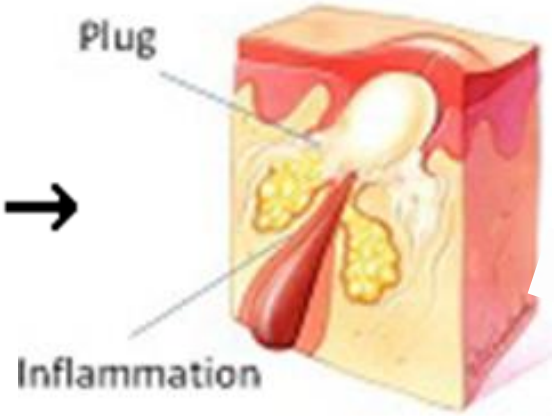

Epidermis
Sebacacus gland
Plugged follicle
Accumulation of apithellal cells and keratin
Accumulation of shed keratin and sebum
Propionibacterium acnes proliferation
Mild inflamation
Marked inflammation
Scarring

Stages of acne development



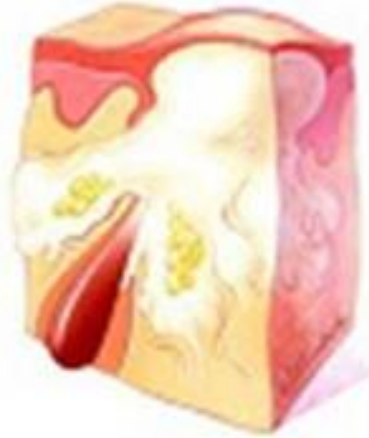
Normal follicle

If the inflammation is deeper, a papule (pimple) is formed



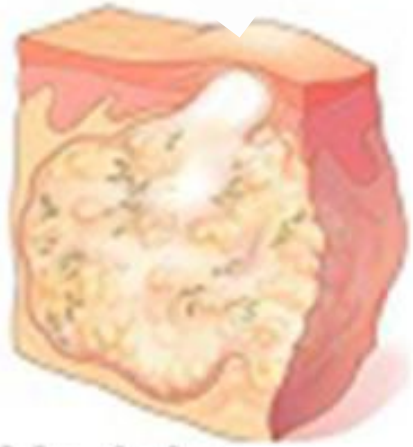
Papule

If the inflammation is right near the surface, a pustule is formed



Pustule

If the inflammation expands deeper it becomes a cyst or nodule.



Nodule

STAPH. EPIDERMIDIS

Coagulase positive staphylococci

- *Staphylococcus aureus*

Coagulase negative staphylococci

- *Staphylococcus epidermidis*
- *Staphylococcus saprophyticus*

Staphylococcal diseases

TABLE 15–2 Important Features of Pathogenesis by Staphylococci

Organism	Type of Pathogenesis	Typical Disease	Predisposing Factor	Mode of Prevention
Sta. aureus	1. Toxigenic (superantigen)	Toxic shock syndrome	Vaginal or nasal tampons	Reduce time of tampon use
		Food poisoning	Improper food storage	Refrigerate food
	2. Pyogenic (abscess)			
	a. Local	Skin infection (e.g., impetigo surgical-wound infections)	Poor skin hygiene; failure to follow aseptic procedures	Cleanliness; handwashing; reduce nasal carriage
	b. Disseminated	Sepsis, endocarditis[1]	IV drug use	Reduce IV drug use
Sta. epidermidis	Pyogenic	Infections of intravenous catheter sites and prosthetic devices	Failure to follow aseptic procedures or remove IV catheters promptly	Handwashing; remove IV catheters promptly
Sta. saprophyticus	Pyogenic	Urinary tract infection	Sexual activity	

IV = intravenous.

[1]For simplicity, many forms of disseminated diseases caused by *Sta. aureus* (e.g., osteomyelitis, arthritis, were not included in the table).