Bacterial skin infections

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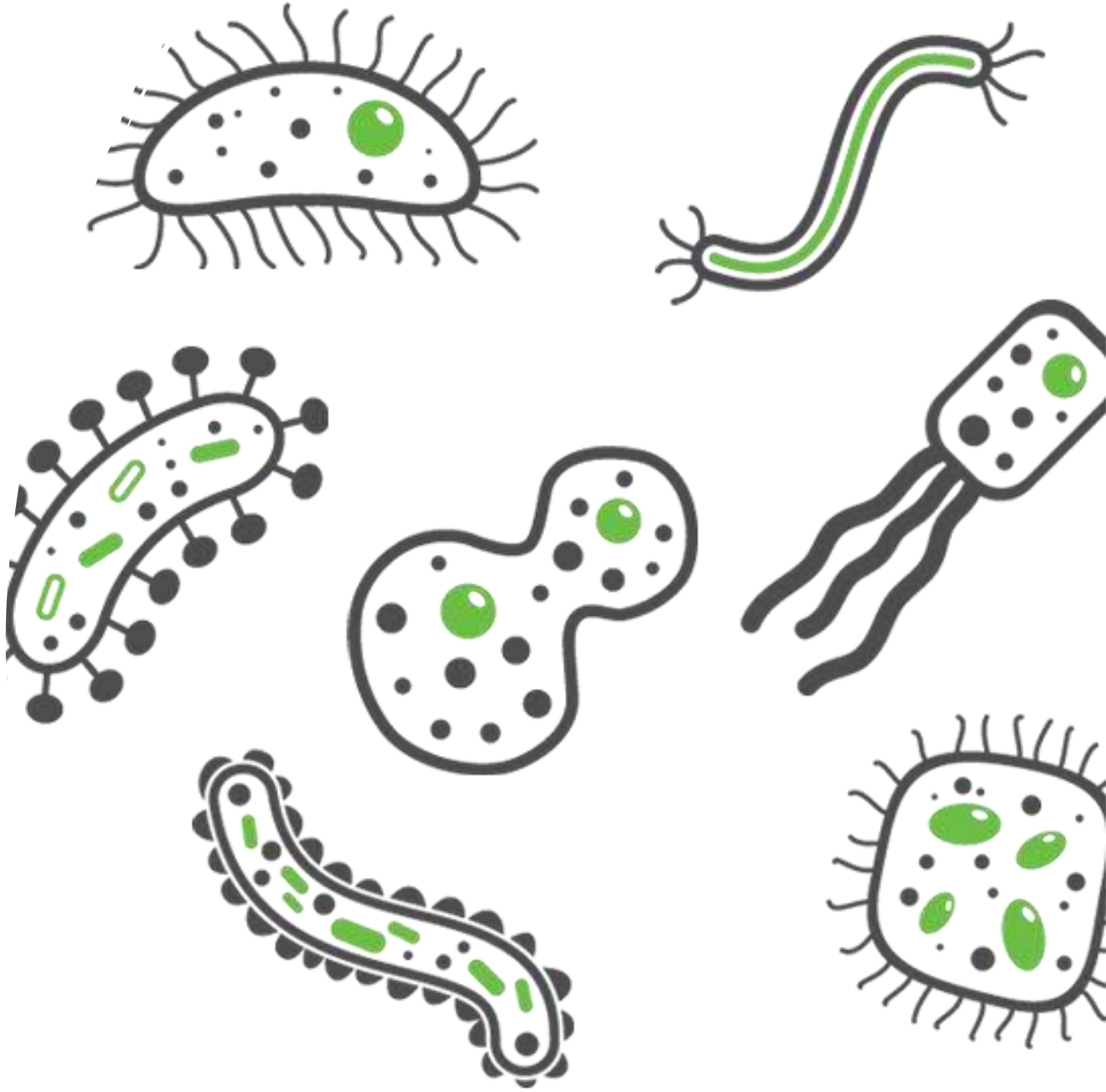


INTRODUCTION

- Intact skin forms a highly effective barrier against invading pathogenic bacteria. Many micro-organisms come into contact with the skin and some live there as part of the normal skin flora, but they rarely cause disease. Normal skin flora consists of:

❖ coagulase-negative Staphylococcus, Corynebacterium, diphtheroids and α-haemolytic Streptococci in the epidermis.

❖Propioni- bacterium in the pilosebaceous unit.

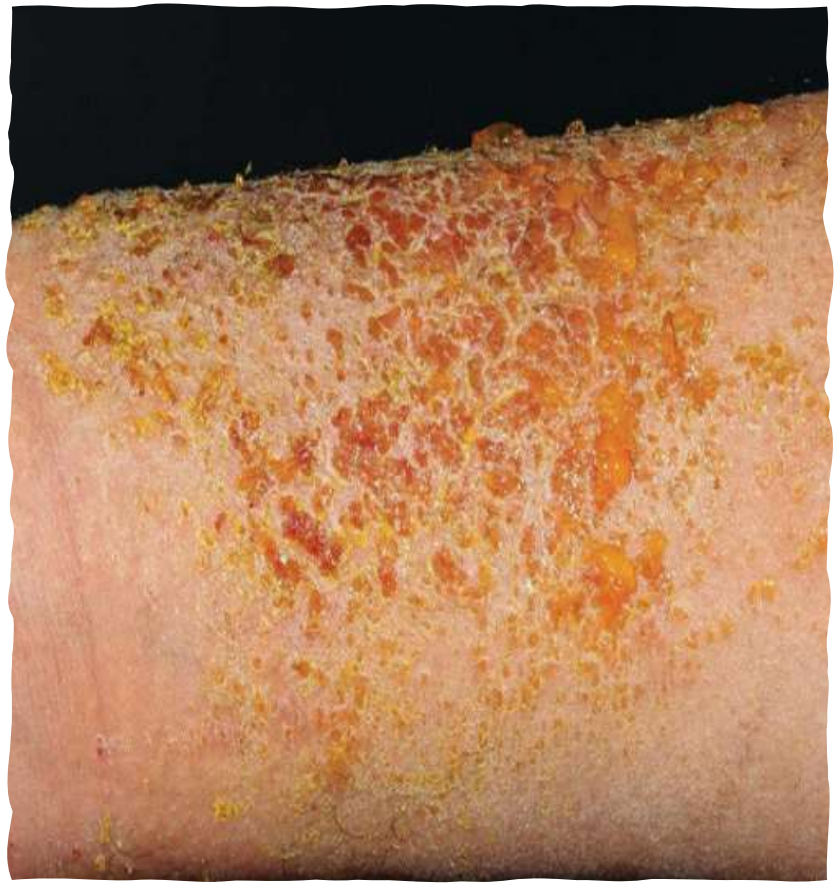
- Normal flora competes with invading pathogenic micro-organisms, thereby acting as a ‘biolog- ical shield’. However, if the host immune system weakens or there is a change in the micro-environment (such as an underlying skin disease) this may allow such bacteria to become pathogenic.

INTRODUCTION

- Bacterial skin infections may be acquired from the external environment (from plants, soil, fomites, animals or other humans) by implantation, direct contact, aerosols or water-borne transmission.

- Patients with a bacterial skin infection may recall an episode of trauma to the skin such as a graze, laceration, insect bite or implantation of foreign material, or they may have a history of ongoing skin disease. A more detailed history may reveal contact with potentially contaminated water via bathing, animal contact, travel abroad or other family members/close contacts similarly affected.

Superficial Infections



1. **Impetigo:** is a common and highly contagious skin infection that causes sores and blisters.

- How it comes?

Impetigo occurs when the skin becomes infected with bacteria, usually either

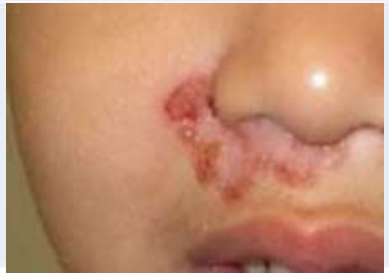
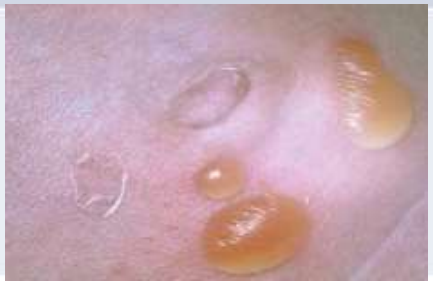
Staphylococcus aureus or Streptococcus pyogenes

Impetigo

The bacteria can infect the skin in two main ways:

- through a break in otherwise healthy skin such as a cut, insect bite or other injury – this is known as primary impetigo
- through skin damaged by another underlying skin condition, such as head lice, scabies or eczema – this is known as secondary impetigo

Impetigo

	Non-bullous lesions	Bullous impetigo
Presentation	clusters of vesicles or pustules that rupture and develop a honey-colored crust over the lesions.	is similar except that vesicles typically enlarge rapidly to form bullae and expose larger bases, which become covered with honey-colored crust.
Causative Agent	Staphylococcus Aureus or Steptococcus Pyogenes	Staph.Aureus (produce epidermolytic toxins A/B)
	Streptococcus infection is associated with regional lymphadenopathy.	
How it looks?		

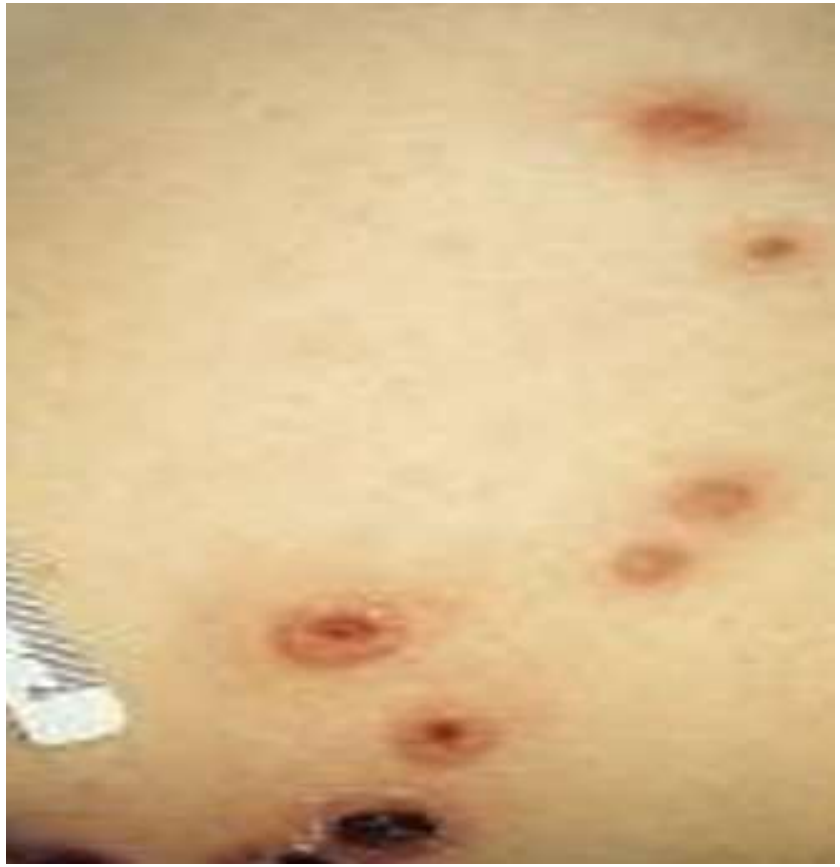
Treatment and Complications

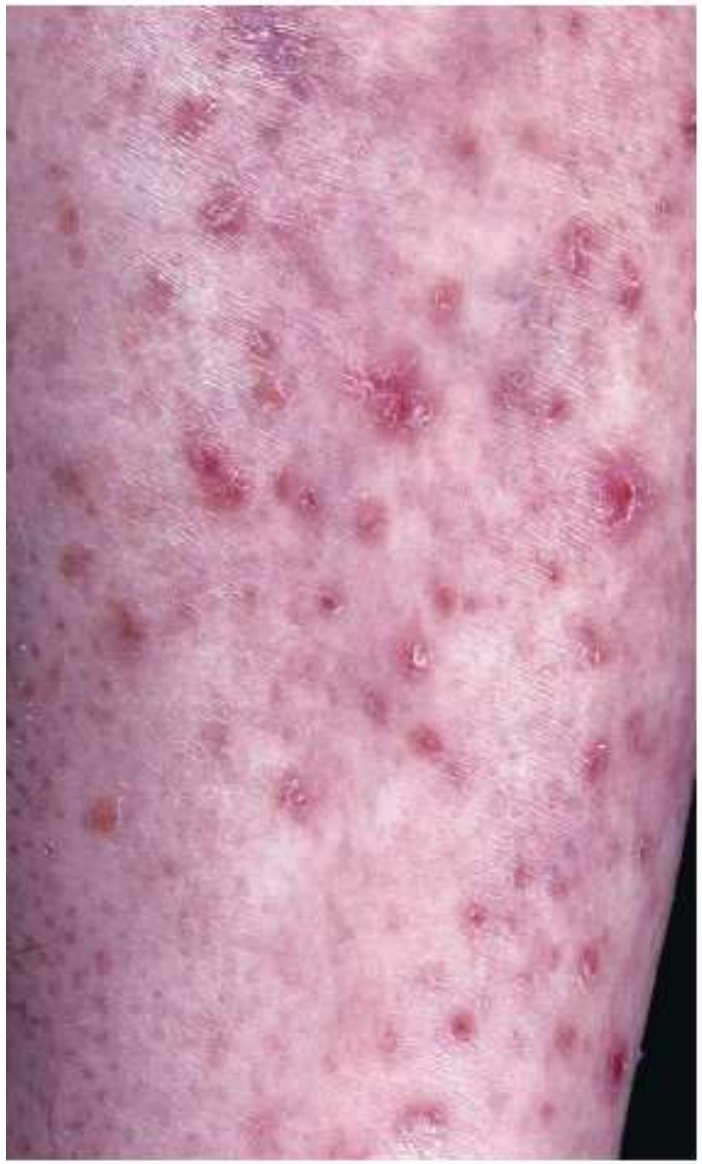
-Topical treatment includes **antiseptic washes**, **fusidic acid, mupirocin, and polymyxins**. Oral antibiotics most frequently used include **flucloxacillin and erythromycin**

-Ecthyma is an ulcerative form of impetigo. Treatment is with topical and sometimes oral antibiotics.

Ecthyma

- **Ecthyma** is a form of impetigo characterized by small, purulent, shallow, punched-out ulcers with thick, brown-black crusts and surrounding erythema.





Folliculitis

- **Folliculitis is an infection of hair follicles.**
- **The etiology** is often unclear, but perspiration, *trauma, friction, and occlusion of the skin* are known to potentiate infection.
- Bacterial folliculitis is usually caused by *Staphylococcus aureus*, but **occasionally** *Pseudomonas aeruginosa* (hot tub folliculitis).

Folliculitis

- The majority of those are frequently mild and self-limiting.
- **Hot-tub folliculitis** caused by Pseudomonas aeruginosa appears within 2 days of exposure to contaminated water or water accessories
- Deeper follicular infections are characterized by abscess formation (which is termed sycosis barbae in the beard area), boils, and furunculosis.
- When several furuncles coalesce, they form a carbuncle.
- Treat most staphylococcal folliculitis with **topical mupirocin**

or topical clindamycin.

Pseudo-folliculitis Barbae

- **Pseudo folliculitis barbae** (‘razor bumps’) is has a similar clinical appearance but this is caused by occlusion of the follicular openings by heavy emollients rather than bacterial infection.

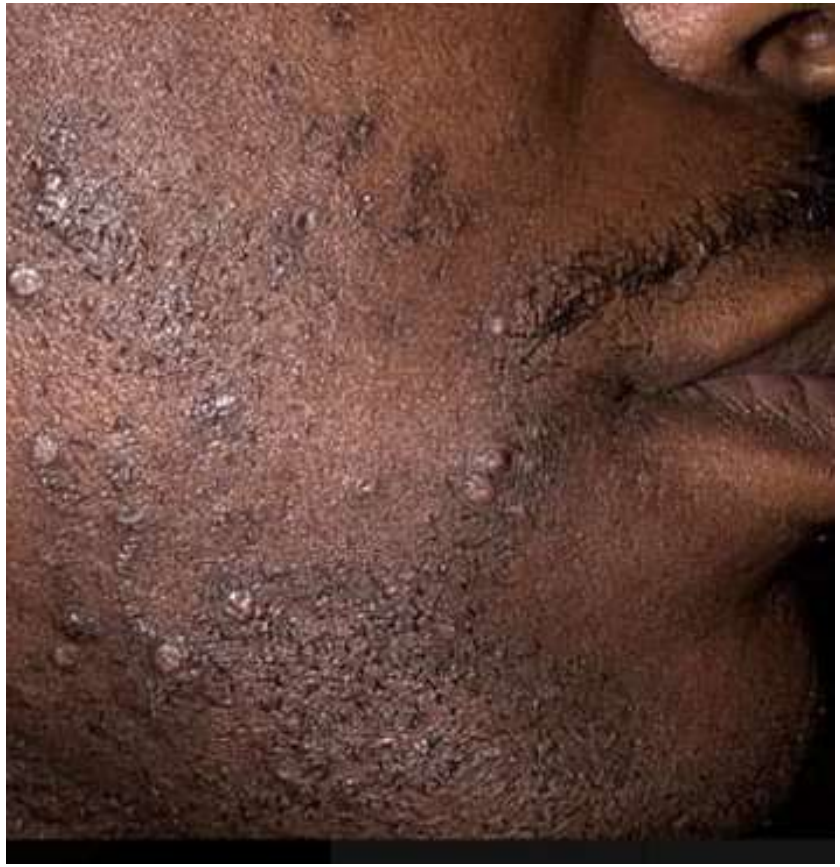
- In pseudo-folliculitis the lesions are all at the same stage of development and are clinically very monomorphic, and the pustules are sterile

Risk factors

- Pseudo folliculitis barbae predominantly **affects Black men.**
- tightly curled hairs
- certain keratin gene variations (*KRT75*, this gene is on the long arm of chromosome 12 and is associated with the synthesis of type II keratin.).
- It typically results from shaving.

Pseudofolliculitis barbae

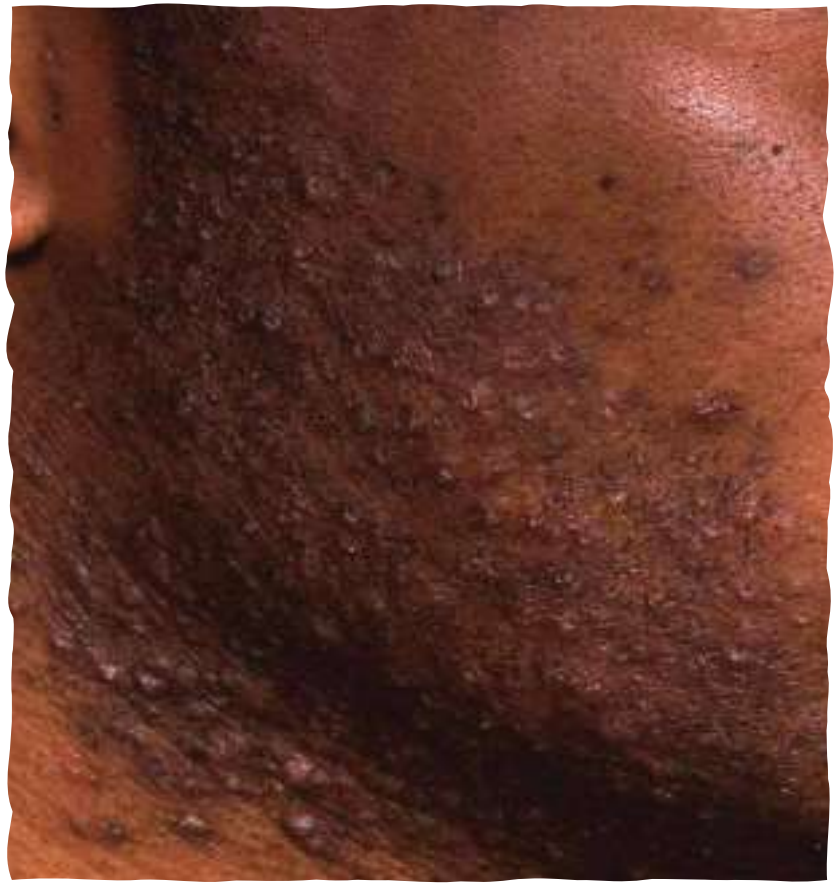
- Coarse curly hair punctures the skin adjacent to the hair follicle (from which it has arisen), resulting in a foreign body reaction with inflammation which can become chronic and lead to scarring



Pseudofolliculitis Barbae Treatment

- Cessation of shaving
- Warm compresses and retraction and release of ingrown hair tips
- Topical or oral drugs as needed for inflammation and secondary infection
- Sometimes hair follicle removal
- Sometimes prednisone

Acne Keloidalis Nuchae



- Happens in **the occipital area of the scalp and in beard**
- It results from **folliculitis and peri folliculitis** with resultant **alopecia and keloid scarring from chronic inflammation.**
- The cause is unknown but it occurs almost exclusively in black males who shave their hair very short.

Erythrasma

Erythrasma is an intertriginous infection by ***Corynebacterium minutissimum*** that is most common **among patients with diabetes** and among people living **in warmer climates**.

It affects the flexural skin sites,particularly the **axilla and groin**

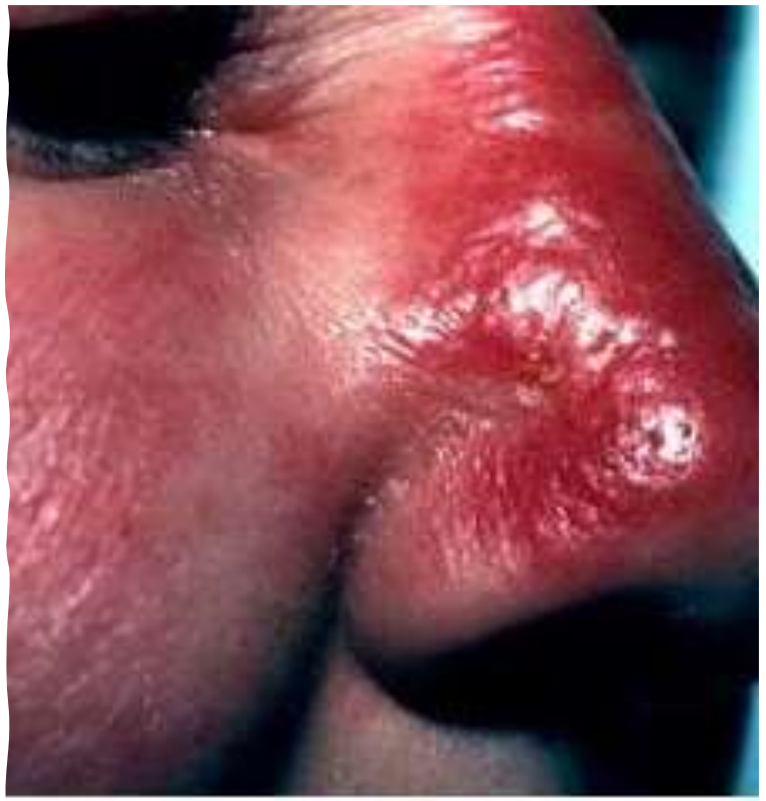
Diagnosis : Coral-red color under Wood's ultraviolet light the affected skin (bacteria) fluoresces pink.

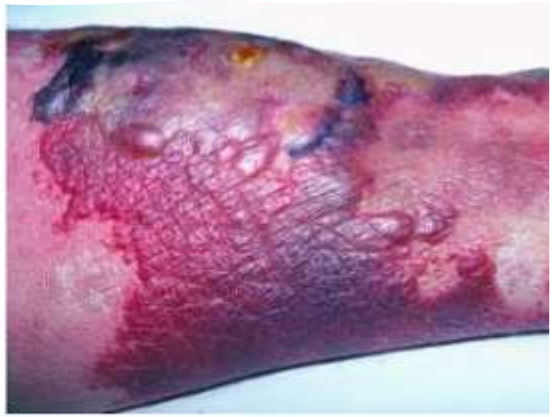
First-line treatment is usually oral **erythromycin**, but if topical treatment is preferred, then **clotrimazole, miconazole, fusidic acid, or neomycin** can be effective

Erysipelas

- Erysipelas infection involving upper dermis and superficial lymphatics, usually from **S.pyogenes.**

- Erysipelas is characterized clinically by red, shiny, raised, indurated, and tender plaques with distinct margins. High fever, chills, and malaise frequently accompany erysipelas.





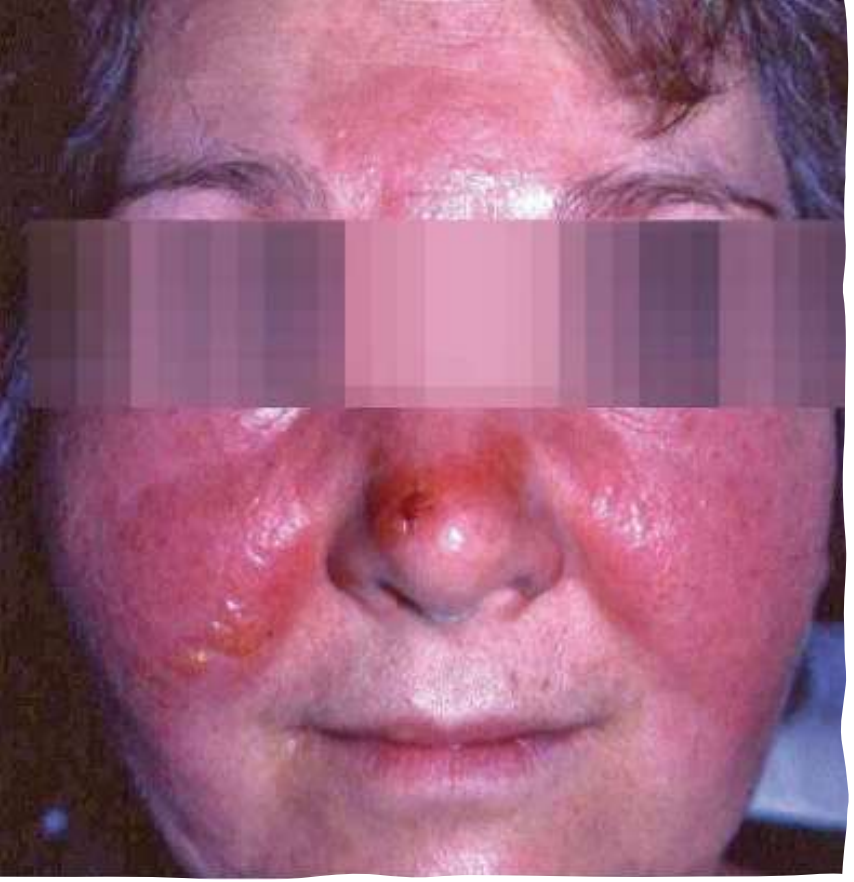
Erysipelas (Bullous)

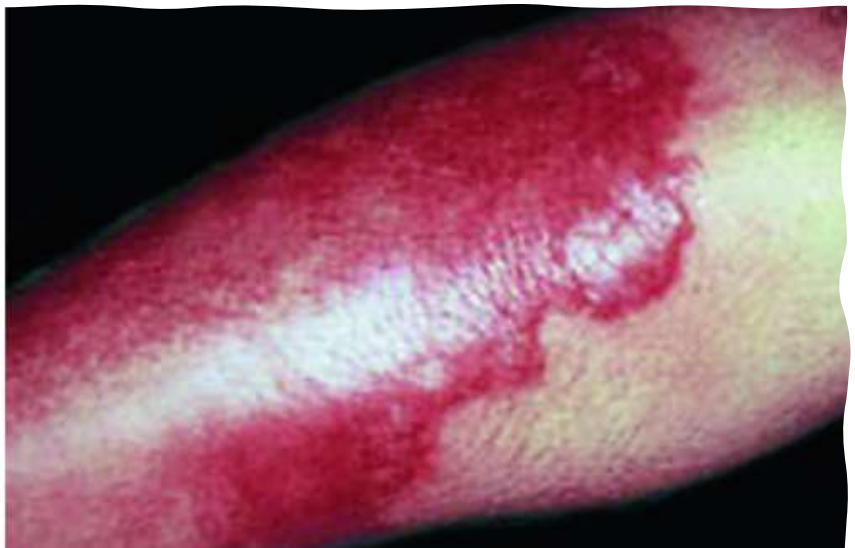
This image shows the bullous form of erysipelas.

Erysipelas

- Differential diagnosis of erysipelas on the face includes **contact dermatitis, photodermatitis, rosacea, systemic lupus erythematosus** and **fifth disease or 'slapped cheek'**

- The **face** (S. pyogenes from throat colonisation) and **lower legs** are most frequently affected.





Erysipelas

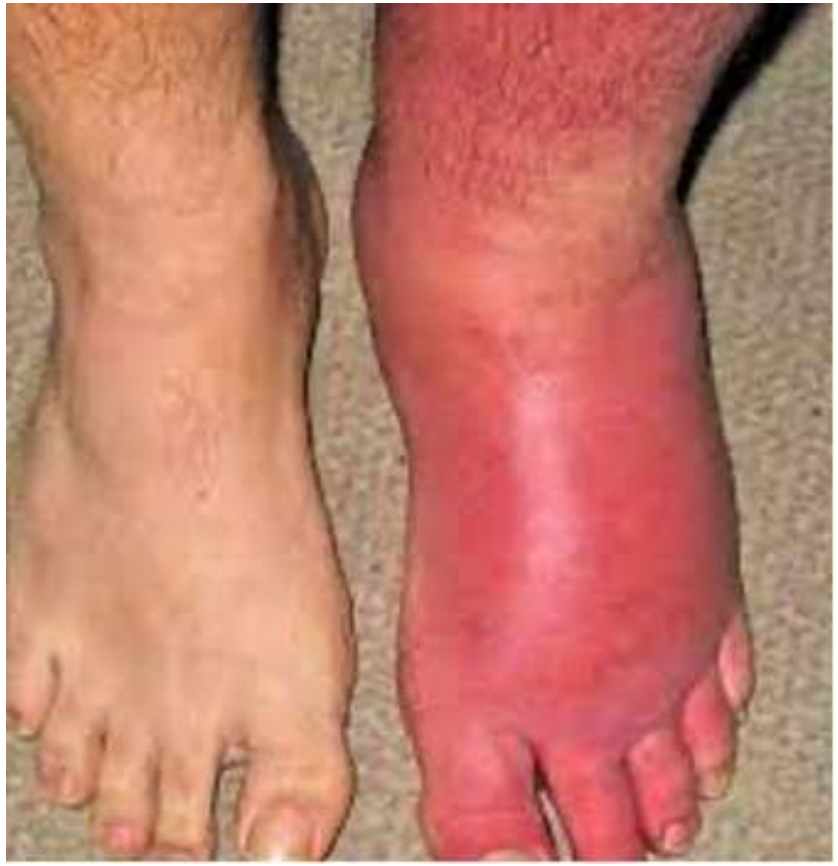
- If the infection is severe, treat **with intravenous benzylpenicillin** or orally with **amoxicillin, roxithromycin, or pristinamycin** for one to two weeks.
- **Complications** of erysipelas commonly include **thrombophlebitis, abscesses, and gangrene.**

Deep Infections

- **Cellulitis**
  develops more slowly than erysipelas, it is a local non necrotizing infection of the deep dermis and subcutaneous tissue and has a poorly defined margin and marked regional lymphadenopathy.

- **Clinical features**
  Local signs: erythema, edema, warmth, tenderness
  Systemic symptoms (in moderate/severe infections): fever, chills, confusion, nausea, headache, muscle and joint pain.

  The lower leg is the most common site affected . Patients may have underlying dermatoses such as a diabetic foot ulcer, tinea pedis or stasis dermatitis which act as a portal of entry for the bacteria
  Bilateral cellulitis is exceedingly rare. Patients presenting with bilateral leg erythema should also be evaluated for alternative diagnoses, including stasis dermatitis and lymphedema.



Cellulitis

Pathophysiology

Entry is commonly via a minor skin injury (e.g., interdigital tinea pedis). May also be secondary to a systemic infection

Etiology

S. pyogenes (also groups C/G β-hemolytic Streptococcus, or rarely S. aureus) organisms invade deeper tissues than those found in erysipelas.

In severe infections intravenous benzylpenicillin may be needed for up to a week as the infection settles slow

Complications

Recurrent infections

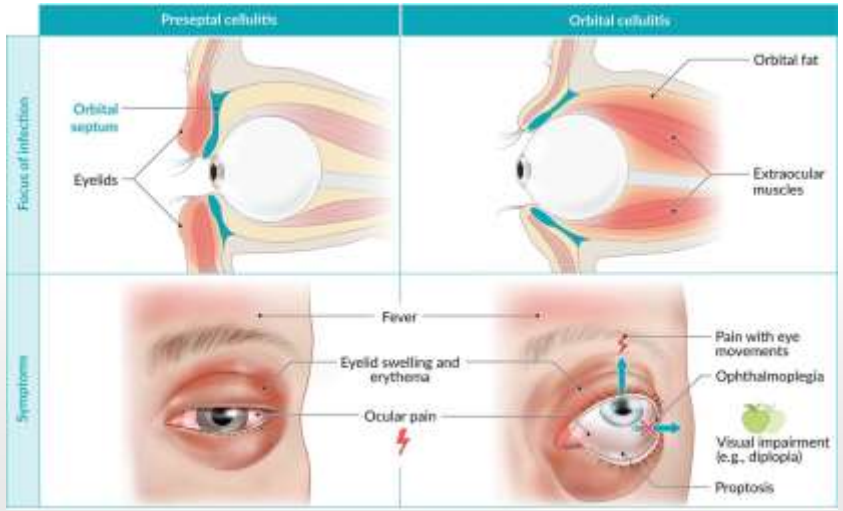
Abscess

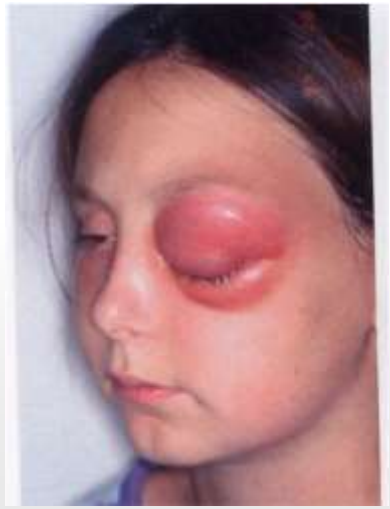
Infection of deeper tissues: cellulitis, necrotizing fasciitis, osteomyelitis

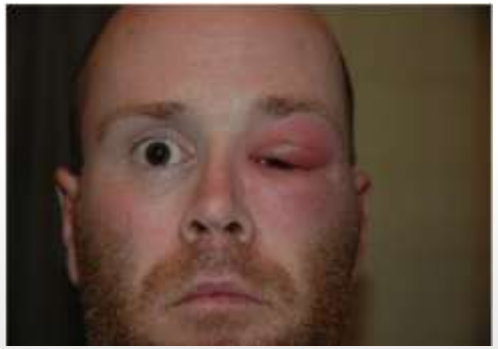
Thrombophlebitis, lymphedema

Systemic complications (e.g., sepsis, endocarditis, streptococcal toxic shock syndrome, poststreptococcal glomerulonephritis, acute rheumatic fever)

In orbital cellulitis: blindness, cavernous sinus thrombosis, intracranial abscess







Orbital Cellulitis

An **infection** involving the **contents of the orbit** (adipose and muscle)

Etiology
- Children > Adults
- Bacterial rhinosinusitis (most common)
- Orbital trauma with fracture/foreign body
- Dacryocystitis
- Teeth, middle ear, face infection
- Infected mucocele

Management
- Broad-spectrum antibiotics (e.g. vancomycin + piperacillin-tazobactam)
- Surgery

Clinical feature	Preseptal cellulitis	Orbital cellulitis
Eyelid swelling (w/w/o erythema)	Yes	Yes
Eye pain/ tenderness	May be present	Yes, deep eye pain
Pain with eye movements	No	Yes
Proptosis	No	Usually, may be subtle
Ophthalmoplegia (+/- diplopia)	No	Yes
Vision impairment	No	May be present (Afferent pupillary defect)
Chemosis	Rarely present	May be present
Leukocytosis	May be present	May be present

Necrotizing fasciitis

Characterized by dusky purplish erythema associated with extensive life-threatening necrosis of the deeper tissue because of rapidly progressive mixed (anaerobic and aerobic bacteria) infection of the deep fascia leading to gas formation in the subcutaneous tissues.

Fournier gangrene: Necrotizing fasciitis of the external genitalia that can spread rapidly to the anterior abdominal wall and gluteal muscles.

Clostridial myonecrosis: a rapidly spreading necrotizing infection caused by Clostridium perfringens or Clostridium septicum

Clinical features

Systemic symptoms: fever, chills, altered mental status

Cutaneous findings:

-Diffuse erythema (often manifests initially as suspected cellulitis that is not responding to initial antibiotic therapy)

-Extreme tenderness and pain out of proportion to the area of erythema

-Significant induration of the subcutaneous tissue

-Crepitus: due to the production of methane and CO2 by bacteria

Purple skin discoloration (skin necrosis, ecchymosis)

Bullae

Loss of sensation in the affected area (paresthesia)

Urgent surgical debridement and broad-spectrum antibiotics are indicated

Necrotizing fasciitis

Etiology

Aerobic and anaerobic, gram-positive and gram-negative bacteria are frequently isolated.

Both monomicrobial and polymicrobial causes are common.

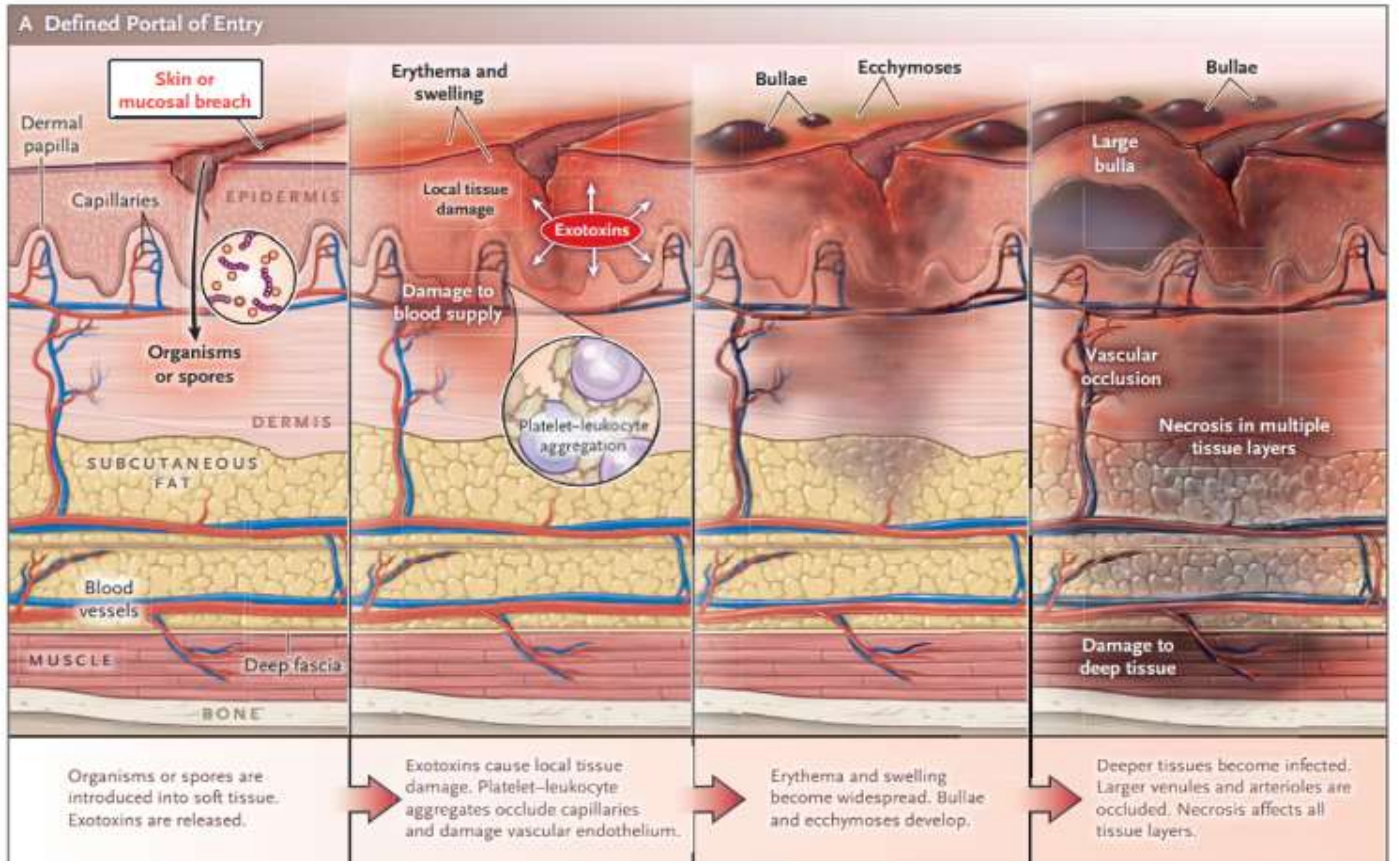
Polymicrobial: wide variety of aerobic and anaerobic pathogens, often of intra-abdominal or genitourinary origin (E. coli, Bacteroides spp.)

Monomicrobial: commonly group A Streptococcus (S. pyogenes), Peptostreptococcus spp., S. aureus

Fournier gangrene: usually mixed infection with facultative pathogens (E. coli, Klebsiella, Enterococcus) and anaerobic bacteria

The only way to definitively establish the causative pathogen is by obtaining a deep tissue culture

Pathophysiology



Staphylococcus scalded skin syndrome (SSSS)

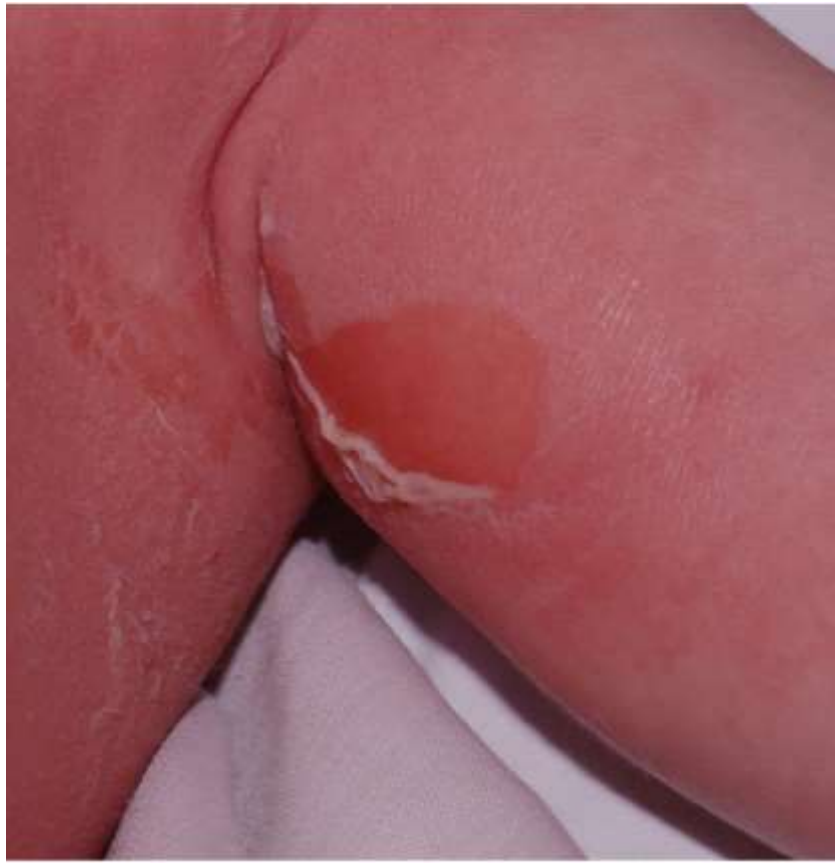
Staphylococcus scalded skin syndrome (SSSS)

It is caused by strains of S. aureus that produce exfoliative toxins A/B resulting in intraepidermal splitting (the target is desmoglein 1 which is responsible for keratinocyte adhesion).

A localized form of the disease is called bullous impetigo.

Route of infection: dissemination of toxins from a local infection Following a staphylococcal infection elsewhere (e.g., skin, mouth, nose, throat, GI tract, umbilicus). The initial infection may also be completely undetected.

Primarily affects infants and young children between 6 months and 5 years of age.



Staphylococcus scalded skin syndrome (SSSS)

Clinical features

Initially : Fever, malaise, and irritability, skin tenderness ,diffuse or localized erythema, often beginning periorally

After 24–48 hours

Flaccid, easily ruptured blisters that break to reveal moist, red skin beneath (i.e., with a “scalded” appearance) → widespread sloughing of epidermal skin

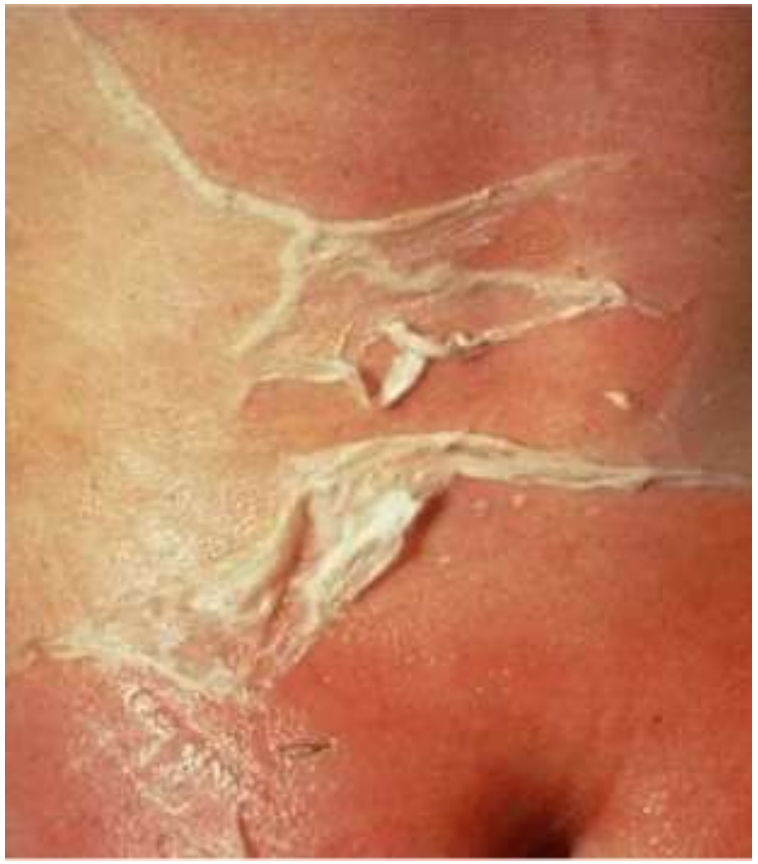
Nikolsky sign is positive (widespread superficial blistering)

No mucosal involvement

Cracking, and crusting is common

Signs of shock (hypotension, tachycardia)

Give systemic antibiotics to treat Staphylococcus. If patients fail to respond, then consider treating for MRSA which has a higher mortality rate.

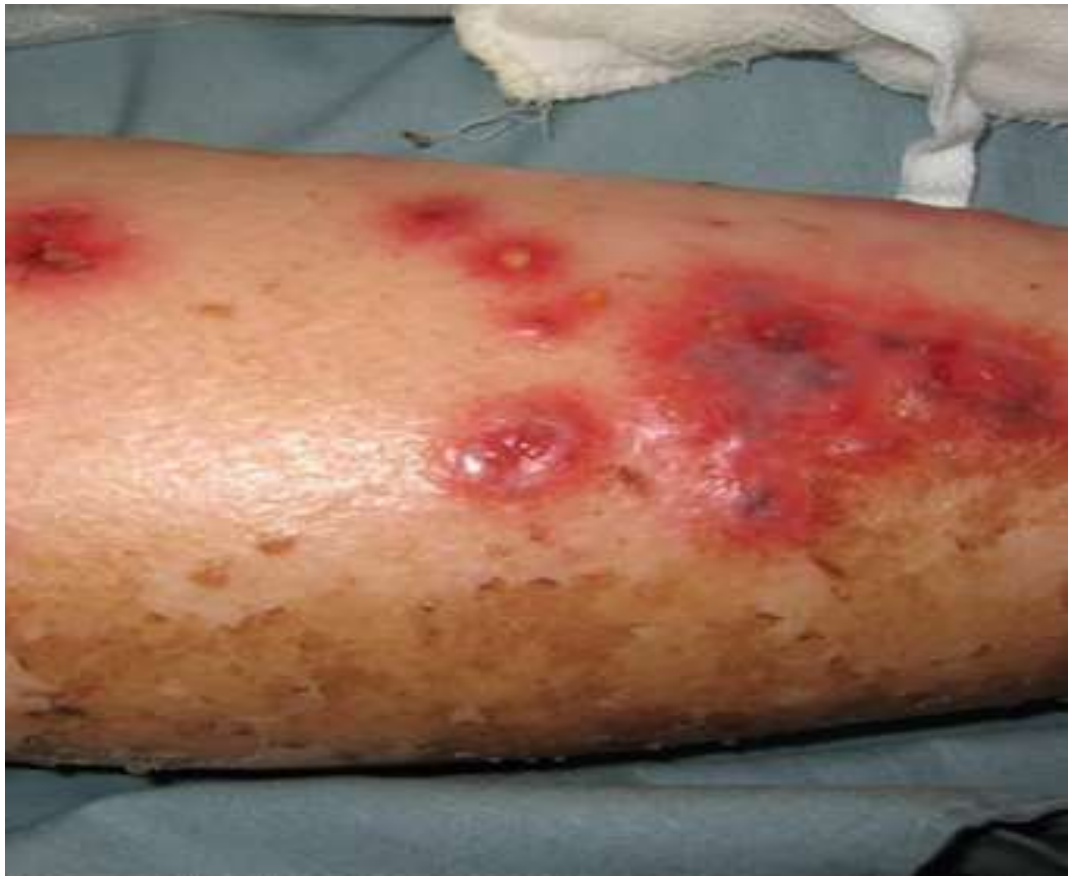


Ecthyma gangrenosum

is an uncommon cutaneous infection (ulcerative lesion extending into the dermis) usually associated with Pseudomonas aeruginosa bacteraemia, and occurs invariably in unwell and immunocompromised patients, especially when there is a prolonged spell of neutropenia.

Rapid progression (within 12–18 hours) of painless red macules → induration, development of pustules, vesicles, and/or bullae → crusted ulcers

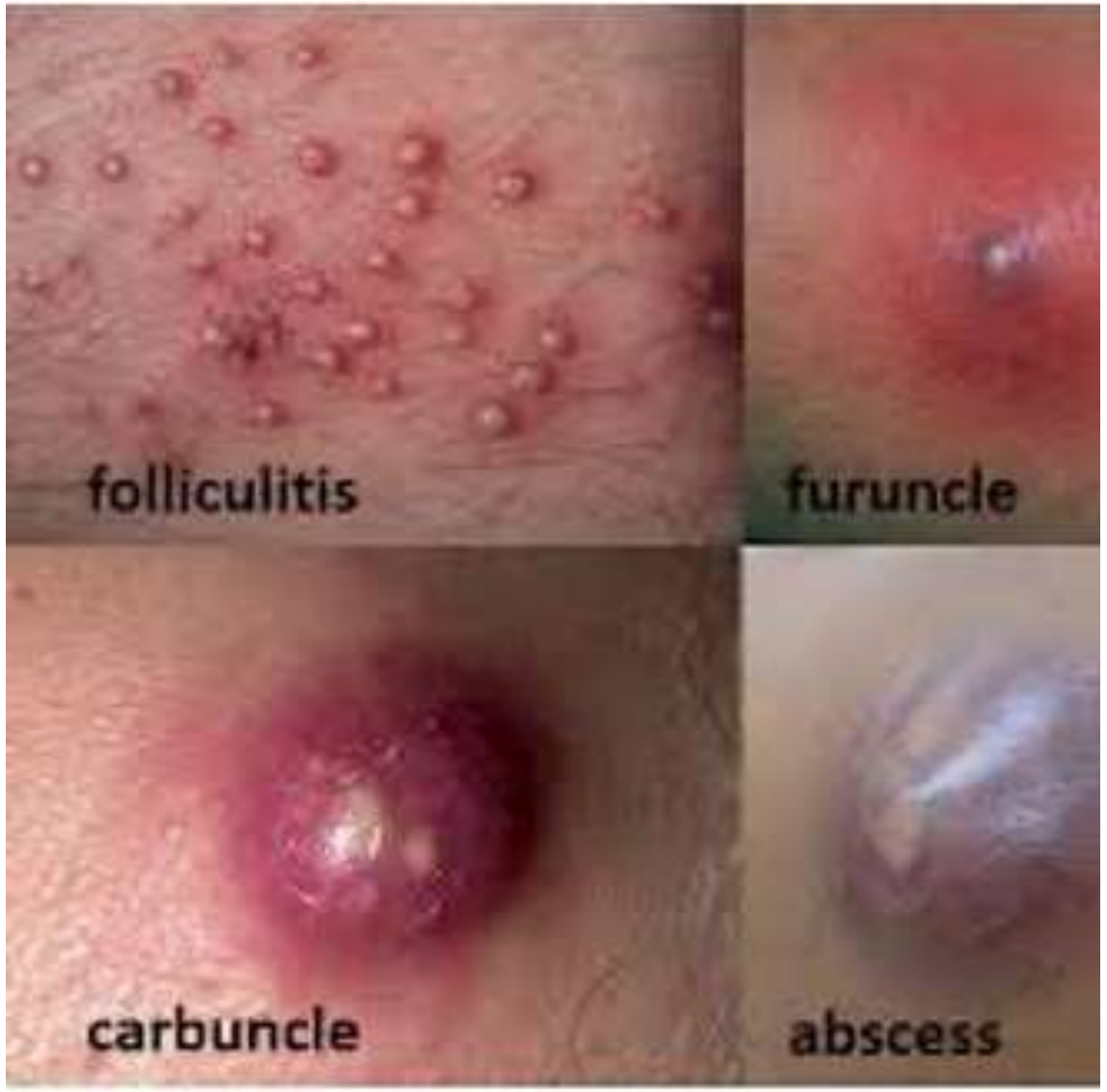
Can involve skin or mucous membranes; anogenital and axillary areas most commonly involved.



Furuncles and Carbuncles

Furuncle: deep folliculitis beyond the dermis with abscess formation in the subcutaneous tissue, it is well-demarcated, firm, painful, purulent nodule

Carbuncle: confluent folliculitis that forms an inflammatory mass; It is formed by a cluster of interconnected furuncles, which are painful, pus-filled, inflamed hair follicles, abscess and skin necrosis may be present



Furuncles and Carbuncles

Clinical features

1-Tender papules and/or pustules, often pruritic

2-Located at the site of hair follicles

3-Possible pus drainage from follicular orifices

4-Potentially multiple lesions

5-Variable locations (carbuncles most often develop on the back of the neck), (Furuncles are common on the neck, breasts, face, and buttocks).

6-Facial furuncles can result in severe complications (e.g., periorbital cellulitis, cavernous sinus thrombosis).

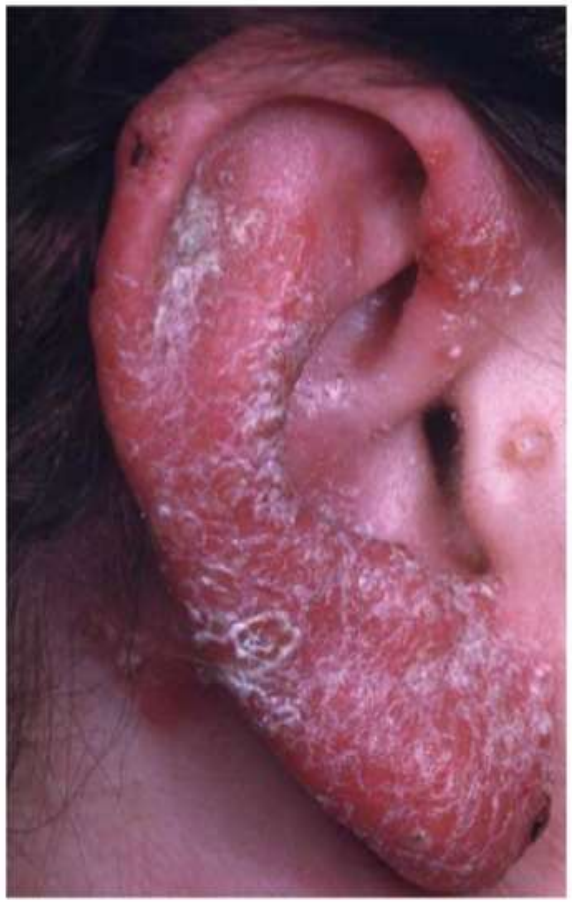


Figure 13.12 Lupus vulgaris.

Mycobacterium infections

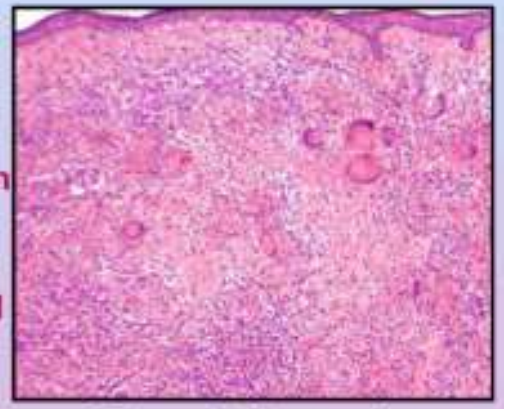
- Clinical manifestations of mycobacterial infections are largely determined by the ability of the host to mount an immune response.

➢ Cutaneous M. tuberculosis (TB) is rare even in endemic areas. TB in the skin usually occurs as a secondary manifestation of disease with its primary focus in the respiratory tract. The most common manifestation is lupus vulgaris which usually presents on the head and neck.

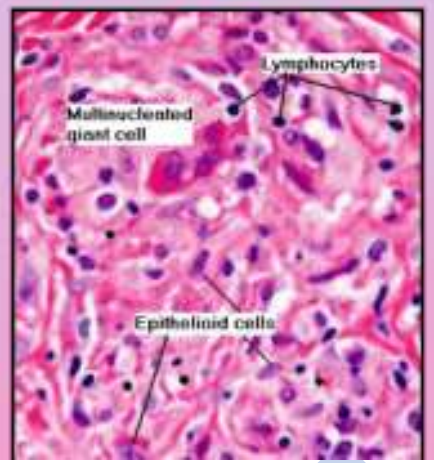
Lupus Vulgaris

Common form of cutaneous reinfection with Mycobacterium tuberculosis

Soft reddish brown nodules which enlarge to form irregularly shaped plaques



Tuberculoid granulomas

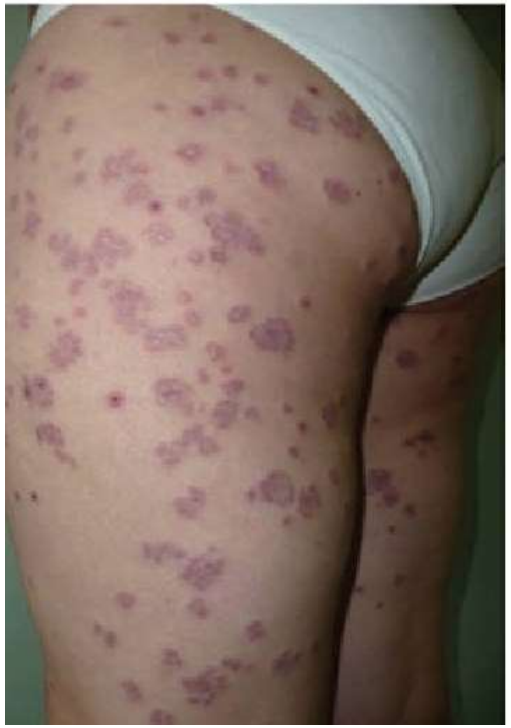


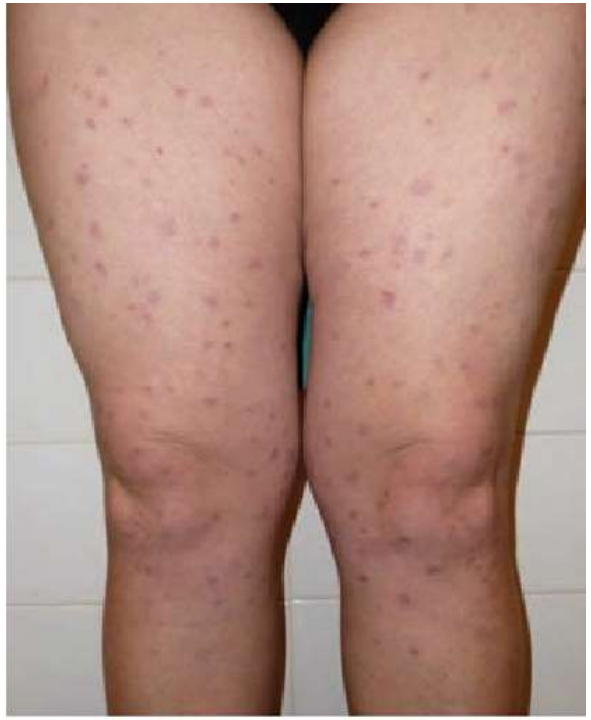
Common in young adults
90% in head and neck region
Also in extremities and buttock

Myobacterium infections

- Allergic-type hypersensitivity reactions called tuberculids can occur in the skin of patients with underlying TB. Tuberculids are thought to represent hypersensitivity reactions to antigenic fragments of dead bacilli deposited in the skin via haematogenous spread. Tuberculids include

1. erythema induratum (Bazin's disease) where patients present with tender nodules and plaques that ulcerate and heal with scarring on the lower legs.
2. Papulonecrotic tuberculid (which some authors believe to be a more superficial form of Bazin's disease)
3. lichen scrofulosorum (very small lichenoid papules over the trunk and limbs in young patients).

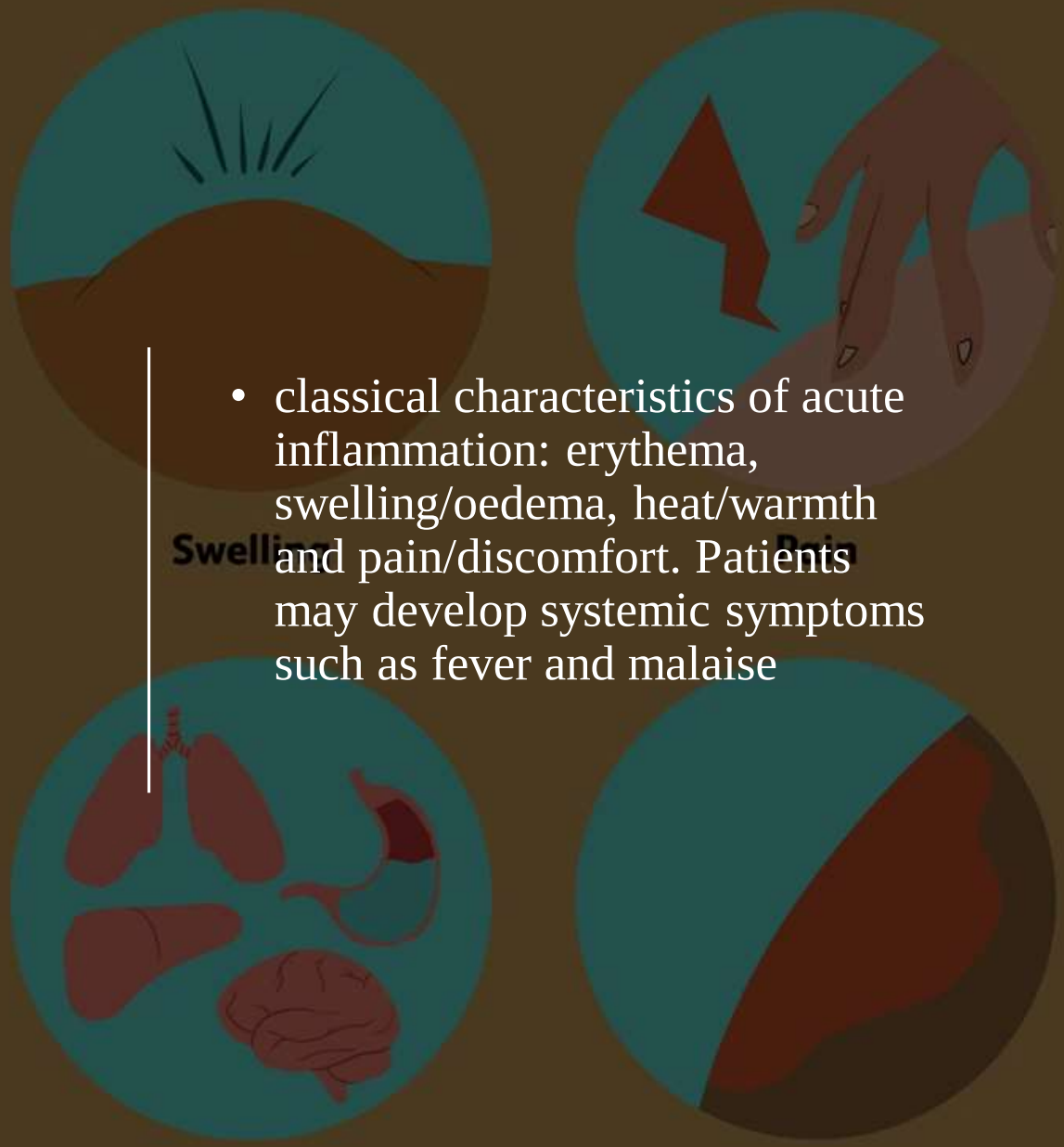




Myobacterium infections

- Mycobacterium marinum or ‘fish tank’ or ‘swimming pool granuloma’ usually occurs because of contact with infected tropical fish or contaminated water. The hand or fingers are most frequently affected; initially, a single warty nodular and occasionally pustular lesion appears with subsequent sporotricoid spread along local lymphatics, forming a chain of nodules.
- Mycobacterium ulcerans causes extensive non-painful ulceration (buruli ulcer) usually on the limbs in children/young adults living in tropical humid areas associated with minor skin trauma and contact with the mycobacterium in standing water.
- Atypical mycobacteria (ATM) are usually found in the environment in vegetation and water

Clinical presentation



- classical characteristics of acute inflammation: erythema, swelling/oedema, heat/warmth and pain/discomfort. Patients may develop systemic symptoms such as fever and malaise

OTHERS

1- Syphilis

is caused by the spirochete bacterium Treponema pallidum which is transmitted through sexual intercourse, transplacental spread and via unscreened blood transfusions.

The incidence of syphilis is steadily increasing due to coinfection with human immunodeficiency virus (HIV)

Primary syphilis manifests as a painless genital ulcer at the site of inoculation.

Cutaneous manifestations of secondary syphilis are characterised by a widespread eruption of red-brown scaly patches and macules that affects the trunk and limbs (particularly palms and soles) .

During the tertiary stage, characteristic granulomas (gumma) may appear, which can cause irreversible organ damage

In patients with HIV the rash may be florid with marked crusting. Serology is needed to know whether patients have previous or current infection, which will subsequently guide management and contact tracing.

OTHERS

2- Rocky Mountain spotted fever (RMSF)

is one of the most common rickettsial infections (Rickettsia rickettsii) in the USA and has a 4% mortality rate.

The most common vector of RMSF is the dog tick.

Pathophysiology Rickettsia species invade capillary endothelium → inflammation → small vessel vasculitis

Clinical features

Blanching maculopapular rash begins on the wrists and ankles , spreads to the trunk, palms, and soles and may become petechial and/or hemorrhagic

Ankle and/or wrist swelling

Gastrointestinal symptoms (e.g., nausea, vomiting, abdominal pain)

Meningitis, focal neurological deficits

Rapid clinical deterioration with shock and multiorgan dysfunction (e.g., DIC)

Treat adults with doxycycline 100mg twice daily for approximately 1 week and children with azithromycin for 5 days.

OTHERS

3- Cat-scratch disease

Caused by the bacterium B. henselae

One or more 5–10 mm large, erythematous, nontender cutaneous papules or vesicles develop approx. 3–10 days after exposure at the site of a scratch (usually by a kitten) associated with the development of regional painful lymphadenopathy 1 or 2 months later.

The disease usually undergoes spontaneous remission within 2–4months. A 5-day course of azithromycin can speed up recovery.

4- Bacillary angiomatosis

caused by Bartonella henselae and Bartonella quintana infections presents in HIV patients with multiple small hemangioma-like papules.

Clinical manifestations are most commonly seen in the skin and mucous membranes but underlying visceral disease (especially liver) may occur simultaneously.

Patients usually present with multiple small cherry-like hemangiomas on the skin which appear over weeks to months .

Serology rather than culture is usually used to confirm the diagnosis (indirect fluorescent assay or ELISA IgG >1:64 indicates likely current infection).

Erythromycin 500 mg for up to 12 weeks is recommended, or 4−6 weeks of azithromycin 500 mg daily.

How are bacterial skin infections diagnosed?

- Bacterial skin infection is commonly diagnosed clinically, although laboratory studies may be useful, such as:
- Full blood count: bacterial infection often raises the white cell and neutrophil count
- C-reactive protein (CRP): elevated >50 in serious bacterial infections (note there can be a delay of >24 hours between onset of symptoms and CRP rise)
- Procalcitonin: blood test marker for generalised sepsis due to bacterial infection
- Blood culture: if fever (>38ºC)
- Swab of the skin lesion/s sent for microscopy, culture, and sensitivities.

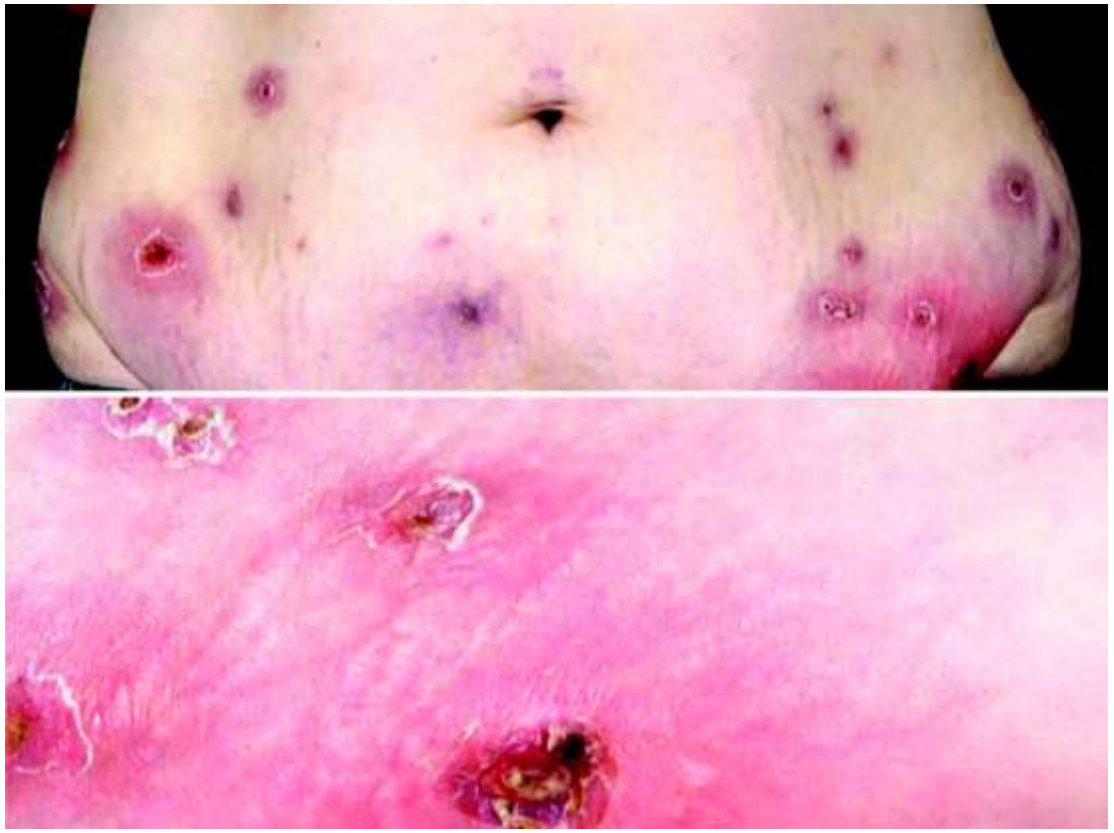
Diagnosis

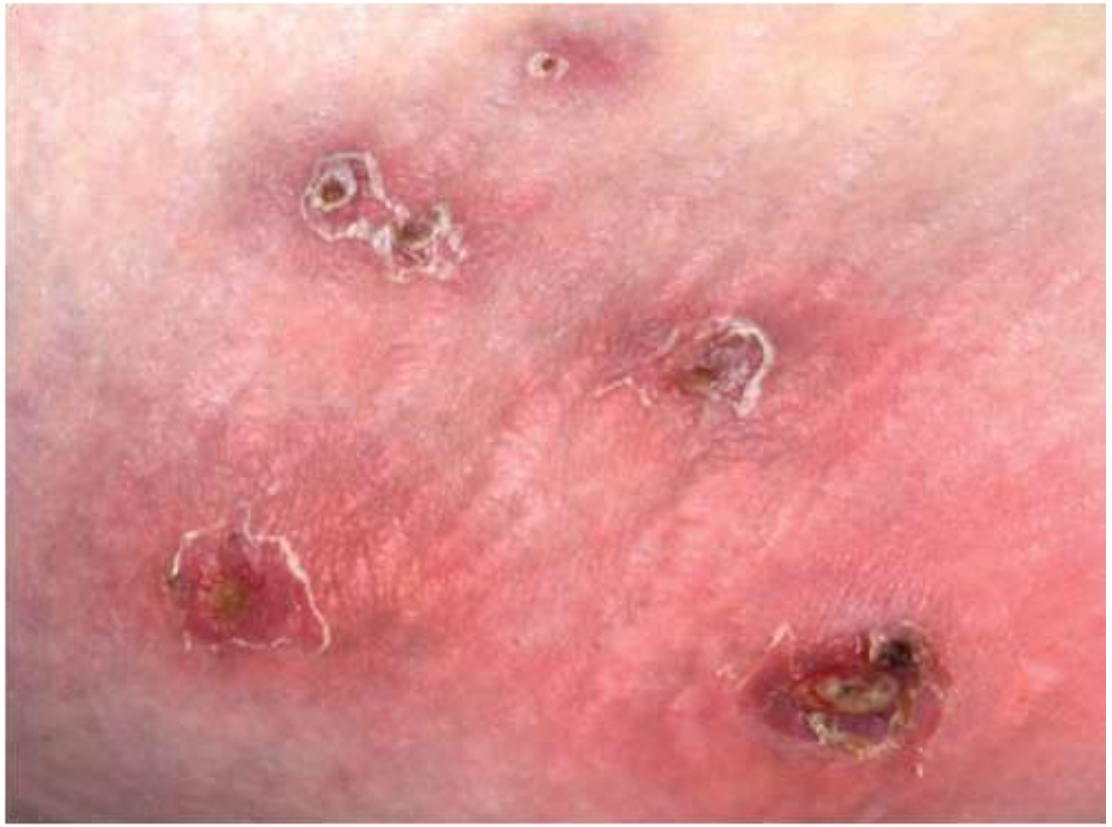
- bacterial swabs for microscopy and culture:Microbiological testing can identify the bacterial species, antibi otic resistance/sensitivity patterns and bacterial toxin production.

- Lesional skin and carrier sites can be swabbed.

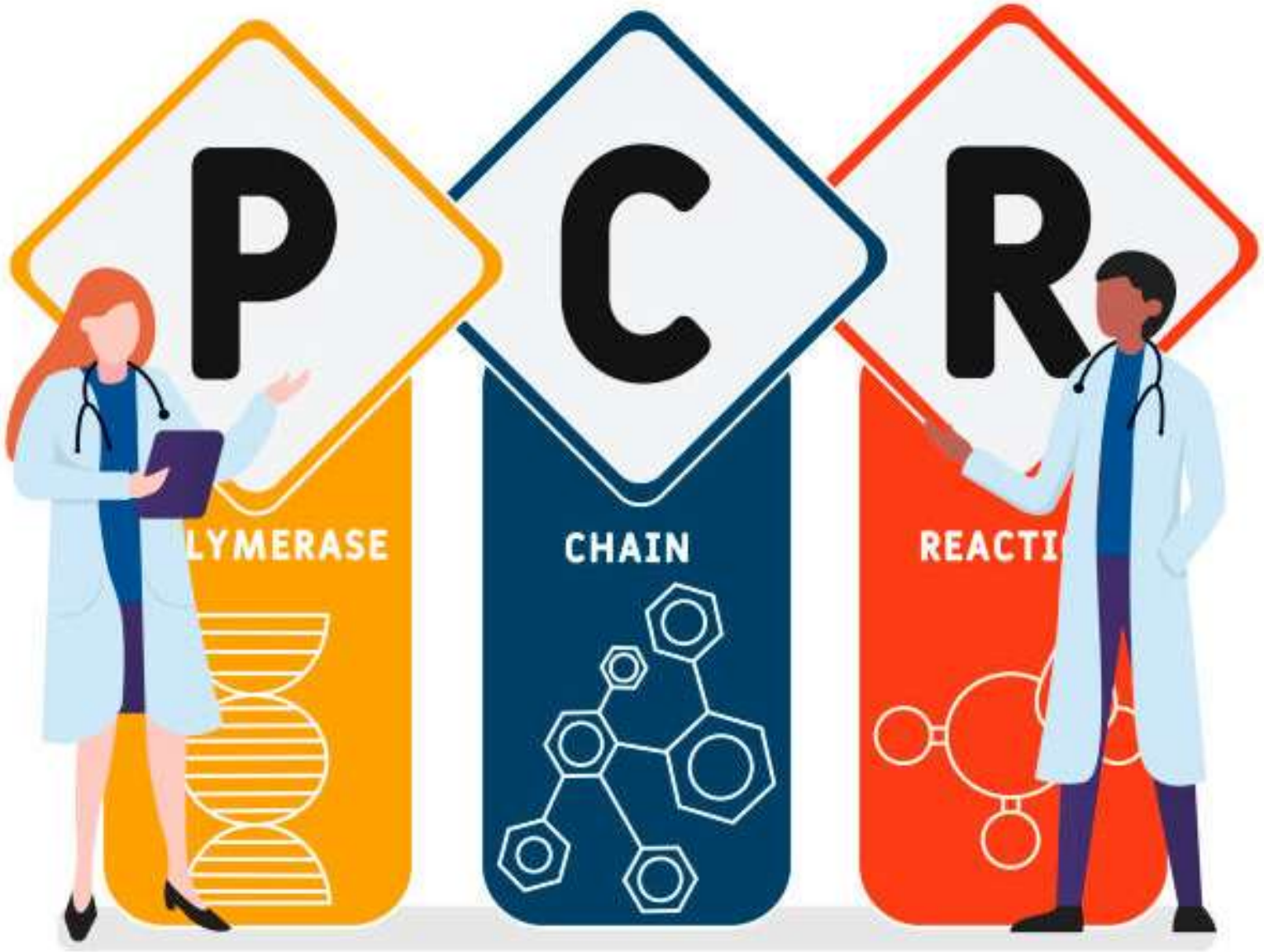
- Nasal swabs may identify Staphylococcus aureus carriers who can suffer from recurrent infections because of bacterial shedding from the nose.

- Panton Valentine Leukocidin (PVL) is a toxin produced by some strains of S. aureus which cause the bacte- ria to be highly virulent and highly transmissible. PVL-positive S. aureus often present with multiple/recurrent boils not settling with short courses of flucloxacillin





- Mulitple abscesses due to PVL Staphylococcus aureus infection.



Diagnosis

- In severe skin infections or when you suspect mycobacterial infections, take a skin biopsy for culture and polymerase chain reaction (PCR).

Treatment

- Antiseptic skin washes or creams containing chlorhexidine hydrochloride can be helpful in removing superficial bacteria, and many of the novel formulations are suitable for use in patients with sensitive skin such as atopic eczema. Potassium permanganate soaks or diluted bleach can be very effective at treating any cuta- neous infections, particularly on the lower legs.
- Topical antibiotics applied twice daily can be used alone to treat mild localised infections. Fusidic acid, mupirocin, neomycin, polymyxins, retapamulin, silver sulphadiazine and metronidazole are all available in topical formulations. Prolonged exposure to topical antibiotics leads to the selection of resistant organisms and rarely contact dermatitis.
- Systemic antibiotics are needed for more extensive cutaneous bacterial infections.

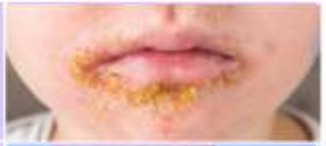
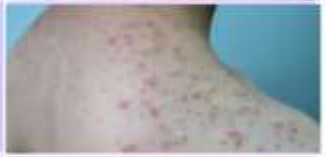
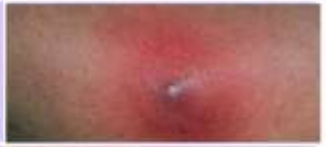
Treatment

Staphylococcal cover is provided by flucloxacillin, erythromycin, clarithromycin, azithromycin, co-fluampicil (contains flucloxacillin and ampicillin), co-amoxiclav, clindamycin, fusidic acid, cipro- floxacin, cefuroxime, dicloxacillin, cloxacillin, linezolid, pristinamycin and roxithromycin.

For MRSA, use vancomycin, nafcillin, daptomycin or tigecycline.

Streptococcal cover is provided by penicillin V, amoxicillin, flucloxacillin, erythromycin, clarithromycin, azithromycin, co- amoxiclav, cefuroxime, ceftazidime, clindamycin, pristinamycin, roxithromycin, vancomycin and levofloxacin.

CHARECTERISTIC SKIN LESION & CAUSATIVE BACTERIAS

STAPH. AUREUS	Impetigo	
	Folliculitis	
	Furuncle	
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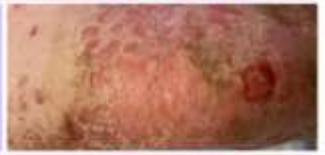
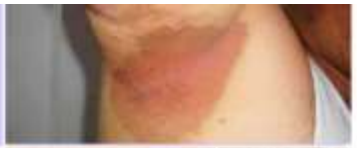
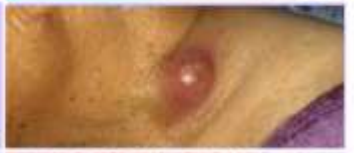
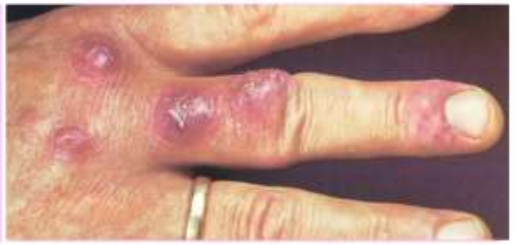
	Staphylococcus-Scalded Skin Syndrome	
STREPTO COCCI	Erysipelas	
	Cellulitis	
	Necrotizing Fasciitis	
	Blistering Distal Dactylitis	

CORYNE BACTERIUM	Erythrasma	
	Trichomycosis Axillaris	
	Pitted Keratolysis	
Erysipelothrix rhusiopathiae	Erysipeloid	
M. Tuber culosis	Lupus vulgaris	
	Verrucosa cutis	
	Scrofuloderma	
	Lichen scrofulosorum	
	Papulonecrotic tuberculid	

M. Marinum	Swimming Pool Granuloma	
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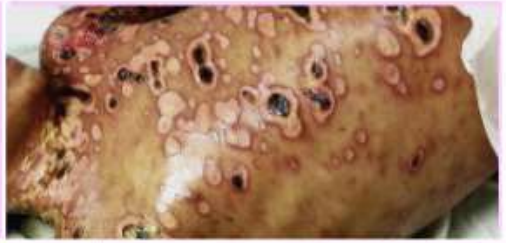
Pseudo monas	Ecthyma gangrenosum	
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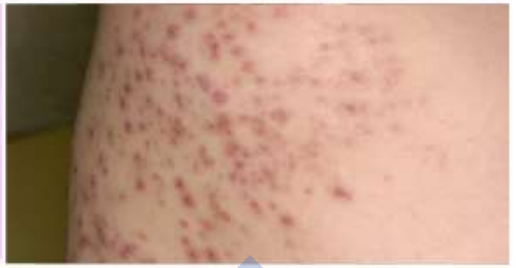
	Hot tub folliculitis	

Thank you!!