Bacterial Skin and Soft Tissue Infections 2

Dr. Hala Mahmoud Altarawneh

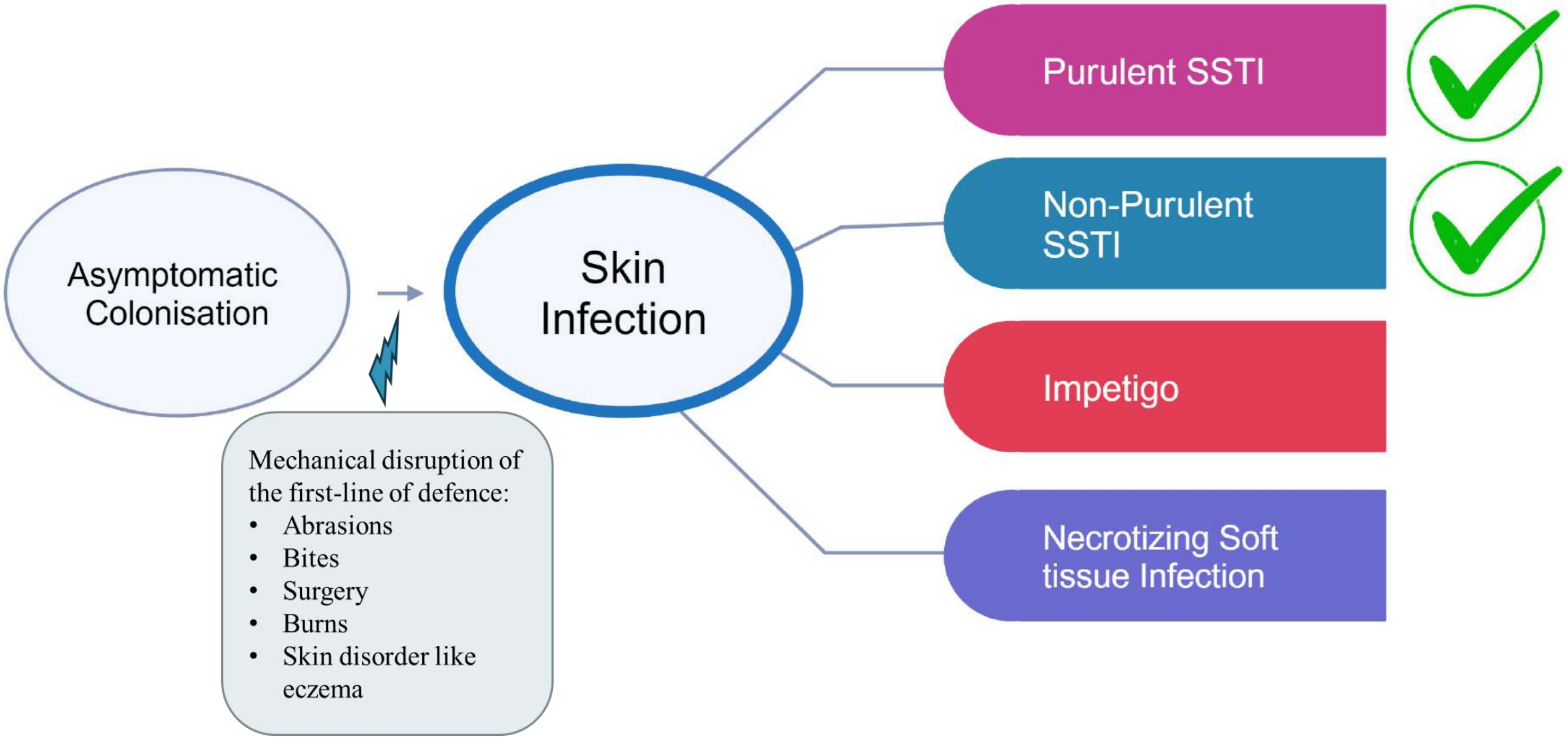
Bachelor degree in Medicine and Surgery - Mutah university
MSC Medical Microbiology – University of Manchester
PhD Medical Microbiology - University of Manchester

Outlines

- Skin and soft tissue infections (SSTIs)
  - Impetigo
  - Necrotizing fasciitis
- Toxin-mediated diseases- Staphylococcal scalded skin syndrome.
- Osteomyelitis
- Septic arthritis.

Progression of Bacterial Skin and Soft Tissue Infections

Skin Infections

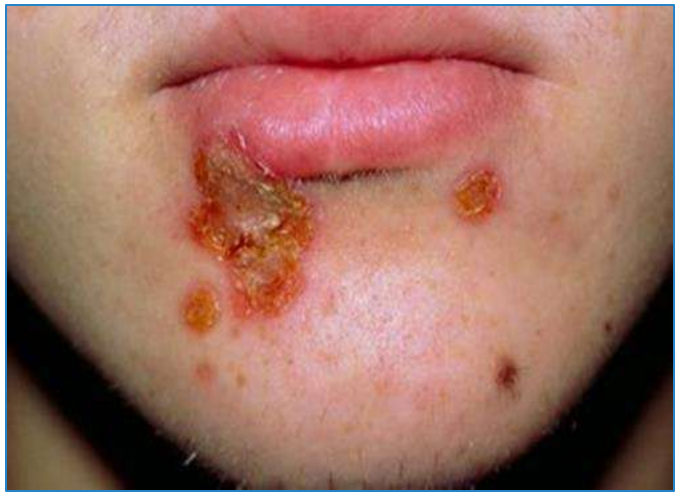


Impetigo

- A **contagious**, **superficial, purulent** bacterial skin infection involving the epidermis.
- Risk factors: poor hygiene, overcrowding, skin diseases (e.g dermatitis), warm/humid climate.
- Impetigo occurs mostly in children.
- Etiology:
  - *S. aureus*: causes 80% of cases, both bullous and non-bullous forms
  - *S. pyogenes*: causes approximately 10% of cases, non-bullous forms only
- In sever cases the infection invades deeper layer forming ecthyma

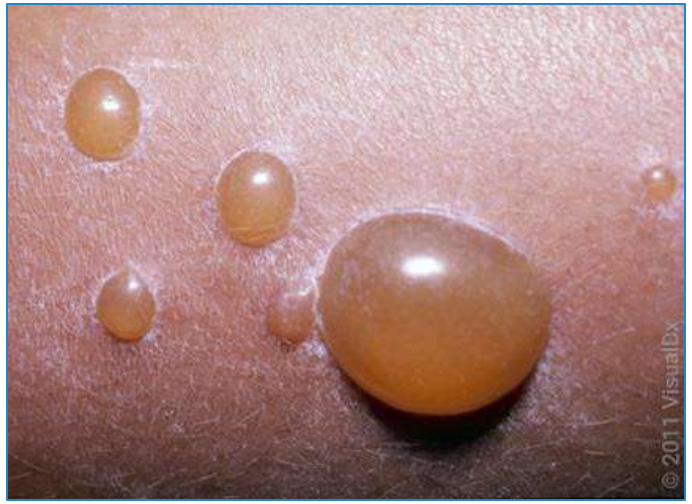
Impetigo: Subtypes - Non-bullous Impetigo

- Most common: approximately 70% of impetigo cases
- Begins as a rash with **papules** → **vesicles** surrounded by erythema → pustules, which rupture and ooze exudate (pus and serous fluid) that dries → pruritic **honey-coloured crusts** that heal with no scarring
- Lesions usually occur around the mouth and nose and/or on the hands
- Lesions may be pruritic, but non-tender



Impetigo: Subtypes - Bullous Impetigo

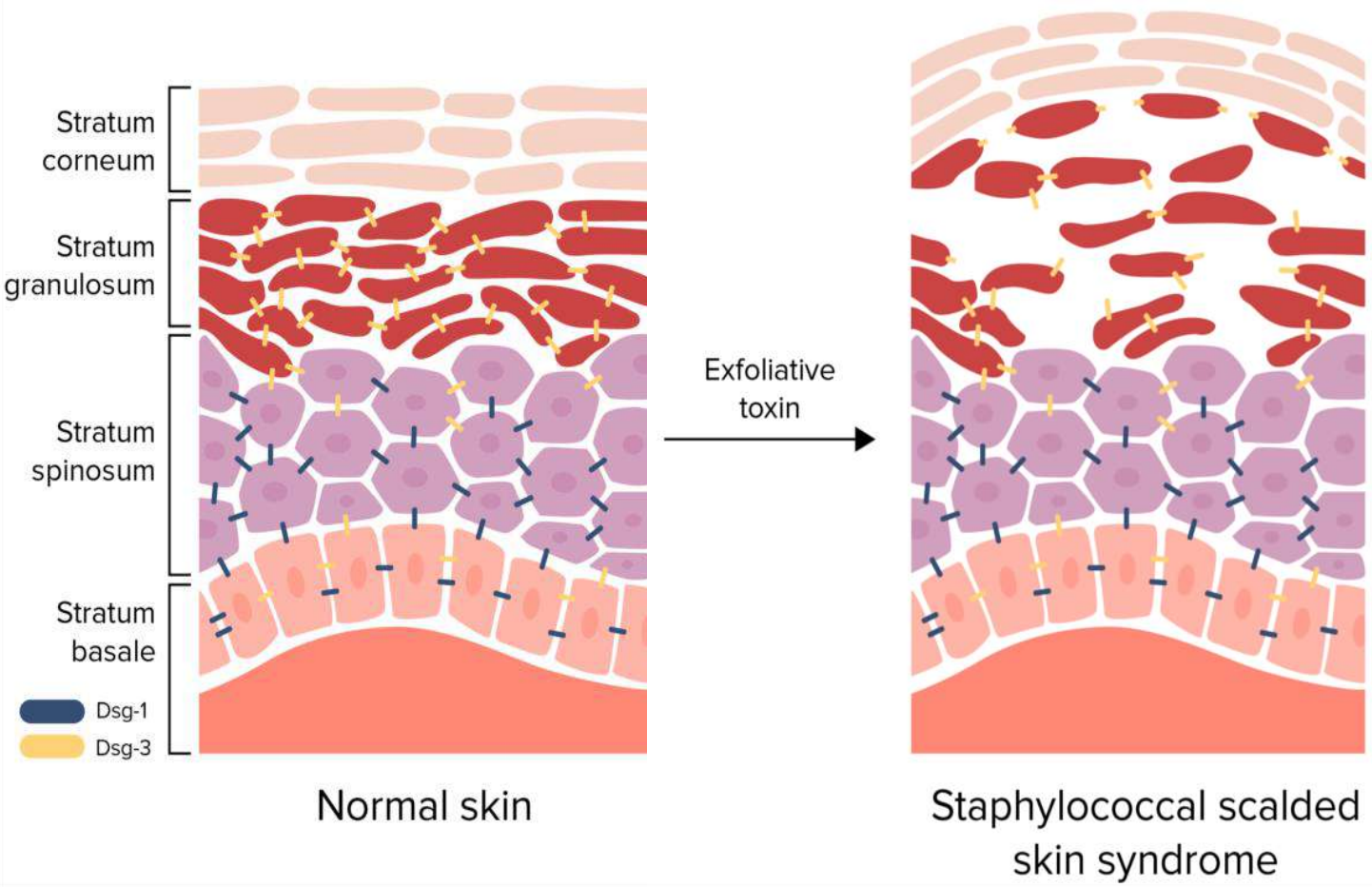
- 30% of impetigo cases
- Begins as a rash with papules → vesicles → large, flaccid bullae, which are pruritic and rupture, oozing cloudy or yellow fluid (pus) → dries into brown crusts → may lead to scarring in severe infections
- Lesions usually occur on the trunk
- May also present with systemic symptoms (fatigue, fever, weakness, general malaise)



Impetigo: Subtypes - Bullous Impetigo

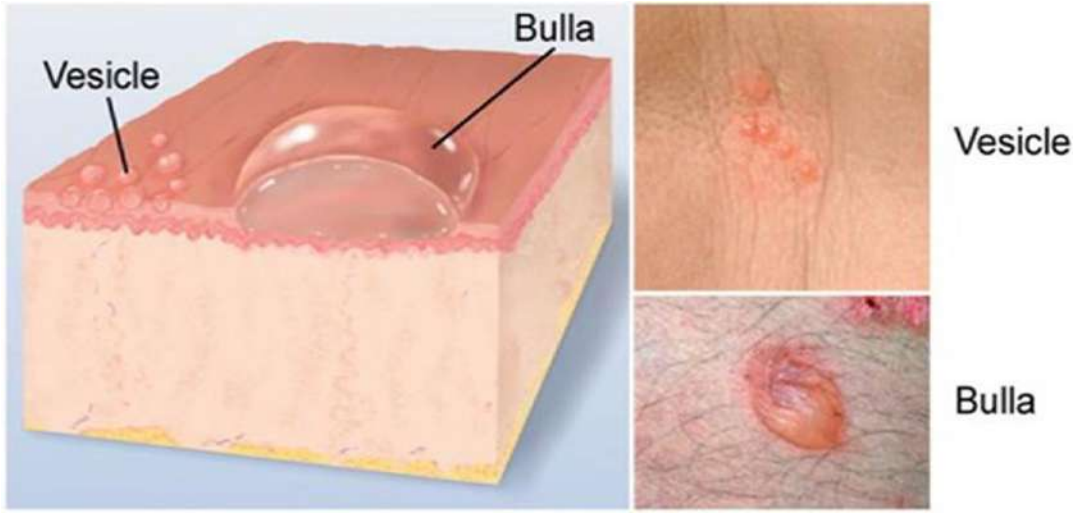
Pathophysiology

- Staphylococcus produces exotoxin (Exfoliative toxin A&B).
- Exfoliative toxin cleaves **desmoglein (Dsg) 1**, disrupting the cell-to-cell adhesion of the stratum granulosum. This detachment of the superficial epidermis causes bullae formation and desquamation.



Question

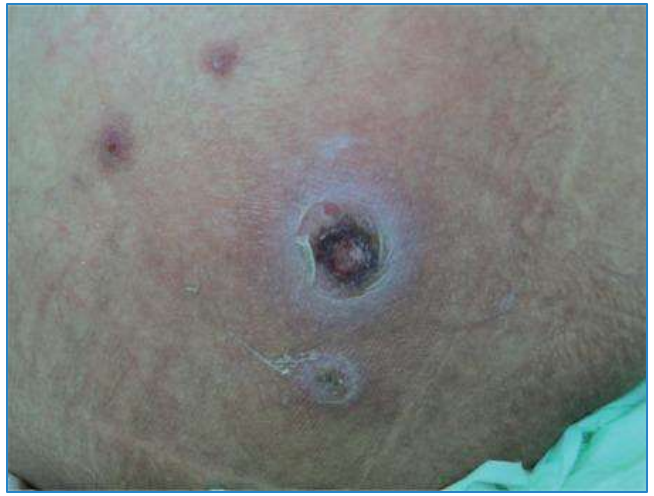
Why is bullous impetigo typically painful, whereas non-bullous impetigo is usually not?

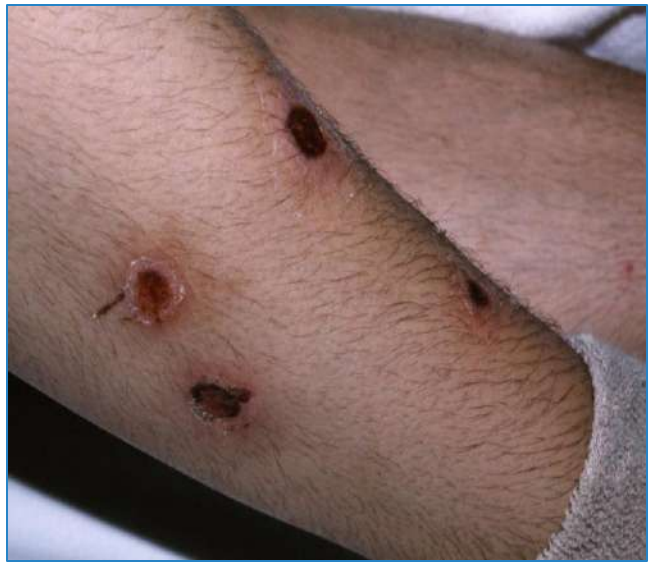


- Bullous impetigo form large bullae that extends deeper into the skin **affecting more sensitive layers** that contain a higher density of pain receptors.
- Non-bullous impetigo primarily involves the superficial layers of the skin, causing minimal disturbance to deeper, more sensitive tissues.

Impetigo: Subtypes - Ecthyma

- Rare
- Ulcerative impetigo that extends into the dermis (also known as “deep or ulcerative impetigo”).
- Begins as a rash with papules → vesicles → sores that are painful, erythematous, and fluid- or pus-filled → coin-sized ulcers with a “punched-out” appearance covered with thick gray-yellow scabs → usually lead to scarring
- Lesions usually occur on the extremities.





Impetigo: Diagnosis and Treatment

- Diagnosis: clinical diagnosis based on typical manifestations of impetigo.
- Treatment:
  - Topical antibiotics: indicated any form of impetigo with a limited area affected
    - Options: Mupirocin
  - Oral antibiotics: Indicated for impetigo with large bullae or numerous lesions, or ecthyma.
    - Options: Targeting both *S. aureus* and GAS like penicillin's (flucloxacillinas)
  - Supportive care: Measures to reduce contagion: e.g., wound care, handwashing, contact precautions

Necrotizing Fasciitis

- Necrotizing fasciitis (NF) is an aggressive life-threatening infection involving rapid and extensive necrosis of the fascia and subcutaneous tissues that can develop into a life-threatening condition within hours.
- It is associated with a high mortality rate of approximately 20% - 80% → surgical emergency
- Incidence: ≤ 1 case per 100,000 individuals per year

Necrotizing Fasciitis: Etiology

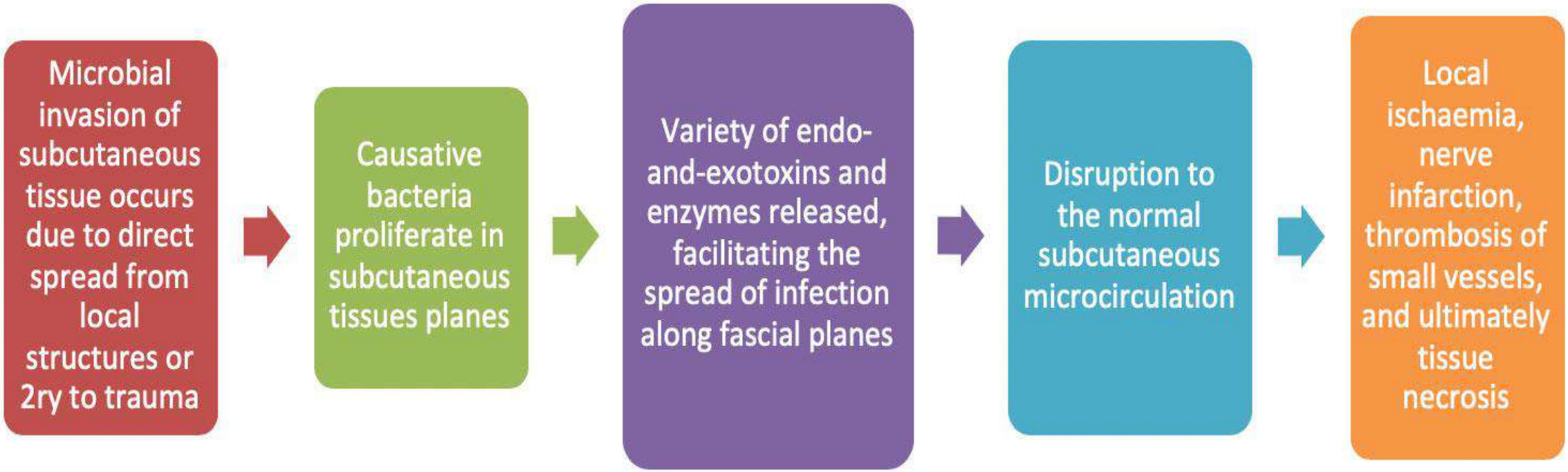
NF is divided into microbiologic categories based on the causative organism(s):

- Type I:
  - Most common type
  - Polymicrobial infection containing anaerobes and aerobes: *S. pyogenes*, Bacteroides, *E. coli*, Enterobacter, Klebsiella.
  - Often seen in older adults with comorbidities, particularly diabetes mellitus
- Type II:
  - Monomicrobial infection: Group A Streptococcus (most common), *S. aureus*
  - Occurs in any age group
  - Frequently found in individuals with no significant risk factors

Necrotizing Fasciitis: Pathophysiology

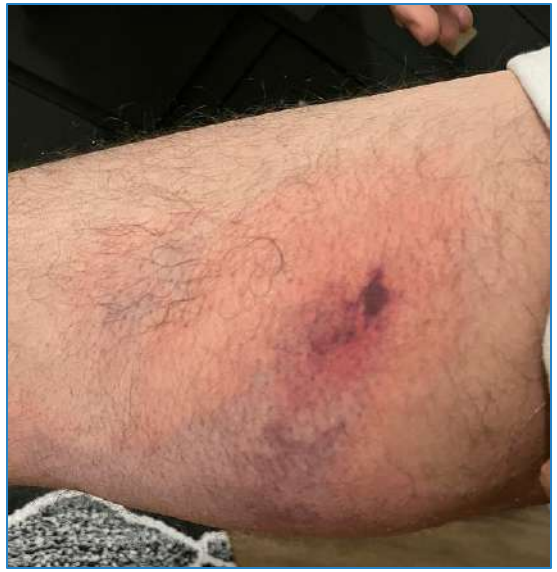
- Bacteria extend into the subcutaneous tissue from: Nearby ulcer or superficial infection, Trauma, Bloodstream (most often *S. pyogenes*)
- Infection causes occlusion of subcutaneous vessels → tissue and fascial ischemia → necrosis
- Damage occurs to superficial nerves → localized anesthesia
- Hypoxic conditions → ↓ neutrophil function → proliferation of bacteria
- Infection and necrosis can rapidly travel along fascial planes, possibly due to bacterial enzymes and toxins.

Necrotizing Fasciitis: Pathophysiology



Necrotizing Fasciitis: Clinical features

- Only 15 to 34 % of patients with NF have an accurate diagnosis at admission
- Necrotizing fasciitis first spreads along the fascia before spreading to the superficial cutaneous tissue. Local findings may, therefore, be unremarkable, with patients experiencing a disproportionate level of pain.
- Skin and soft tissue findings:
  - Common sites of infection: Extremities (most common)
  - Early signs:
    - Acute, severe **pain out of proportion disproportinate to skin signs**
    - Erythema that **quickly spreads** over hours to days.
    - Warmth
    - Tense, indurated skin

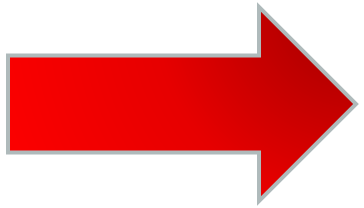


Necrotizing Fasciitis: Clinical features (Cont.)

- Late signs:
  - Crepitus
  - Bullae, or skin necrosis
  - Anesthesia or paresthesia, ulceration
- Evidence of systemic toxicity
  - High fever, tachycardia, hypotension, and/or **altered mental status**.

Necrotizing Fasciitis: Diagnosis

- A definitive diagnosis of necrotizing fasciitis is made by **surgical exploration and debridement**.



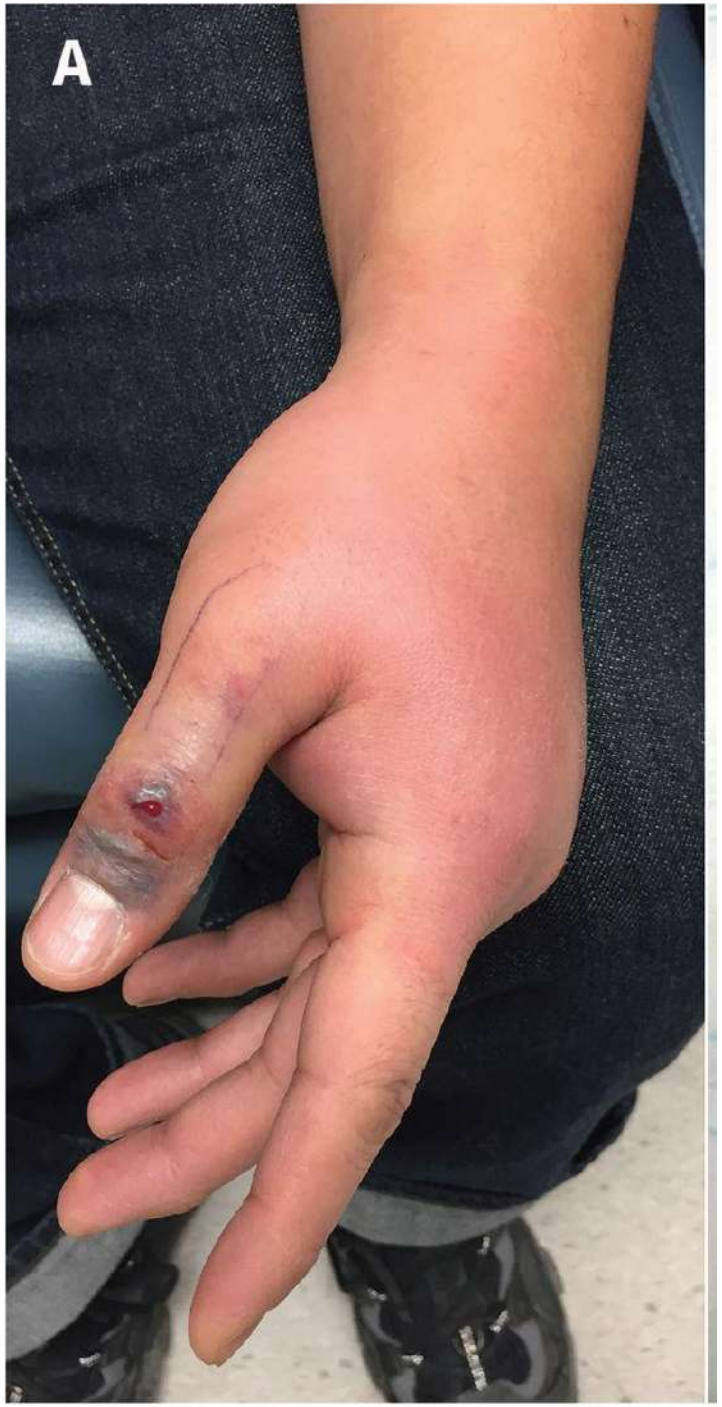
Surgical exploration **should not be delayed** to obtain diagnostic information, if the clinical suspicion is high.

Necrotizing Fasciitis: Management

- Admit all patients with suspected or confirmed NF to hospital for treatment.
- Surgical debridement is the mainstay of treatment.
  - Necrotic tissue is removed.
  - Amputation may be required for severe disease affecting an extremity.
- Antibiotic therapy:
  - Start systemic, broad-spectrum antibiotic therapy immediately after blood cultures have been obtained.
  - Intravenous antibiotics should be given for coverage of gram-positive, gram-negative, and anaerobic bacteria

Necrotizing Fasciitis: Case Report

- A 44-year-old pathologist presented to the emergency department after sustaining a scalpel injury during a postmortem examination 16 hours previously. He had stabbed the dorsum of his left thumb and immediately irrigated the wound with water.
- At the time of presentation, he had erythema and severe pain in his thumb.
- Upon examination, he was afebrile and had a 0.5-cm laceration oriented obliquely over the dorsum of the of his thumb. There was a haemorrhagic blister distal to the laceration and minimal purpuric discolouration around the laceration.



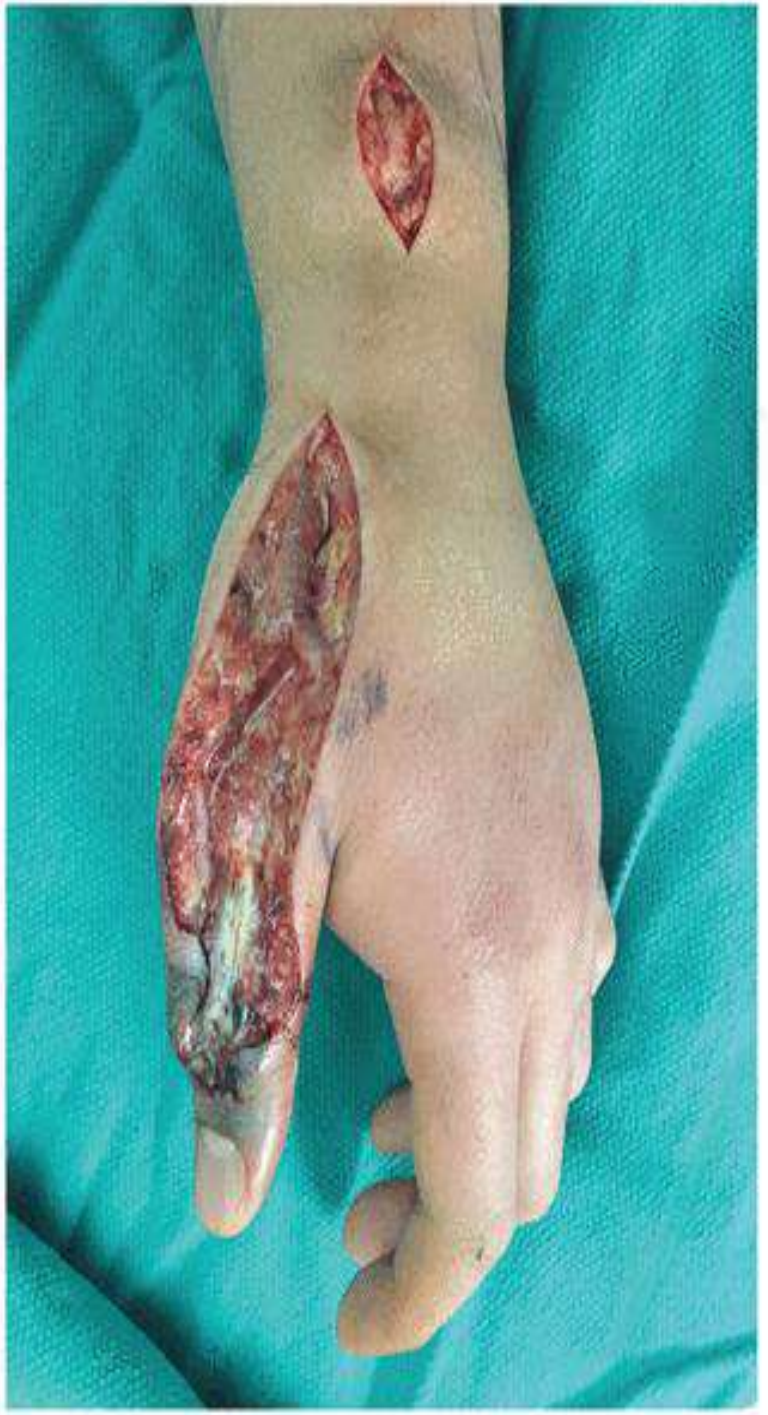
(Brichacek et al., 2017)

Necrotizing Fasciitis: Case Report (Cont.)

- two hours later the patient was reassessed and, although he remained afebrile with normal blood pressure, he had a sinus tachycardia.
- Erythema had progressed past our previous markings to involve the entire hand. His pain had increased and the area of purpura surrounding the initial laceration had progressed.
- Given this rapid change, the patient was taken to the operating theatre for urgent incision and débridement of suspected necrotizing fasciitis.

Necrotizing Fasciitis: Case Report (Cont.)

- Incision and drainage that was performed in the operating theatre.
- Frank purulence was found to track along the fascia overlying the extensor pollicis longus tendon.
- The fascia appeared nonviable in many areas, and the distal skin overlying the interphalangeal joint also appeared nonviable.
- The entire wound was irrigated with normal saline. Skin tissue overlying the dorsal interphalangeal joint was nonviable and required débridement.



Necrotizing Fasciitis: Case Report (Cont.)

- This case, although uncommon, is a reminder that even a minor scalpel injury can result in a life-threatening infection. Substantial cutaneous infections that progress over a short period of time should alert clinicians to necrotizing fasciitis. Patients should be reassessed frequently; expert advice should be requested early, and imaging should not delay surgical treatment.

Progression of Bacterial Skin and Soft Tissue Infections

Toxin-mediated diseases: Staphylococcal scalded skin syndrome

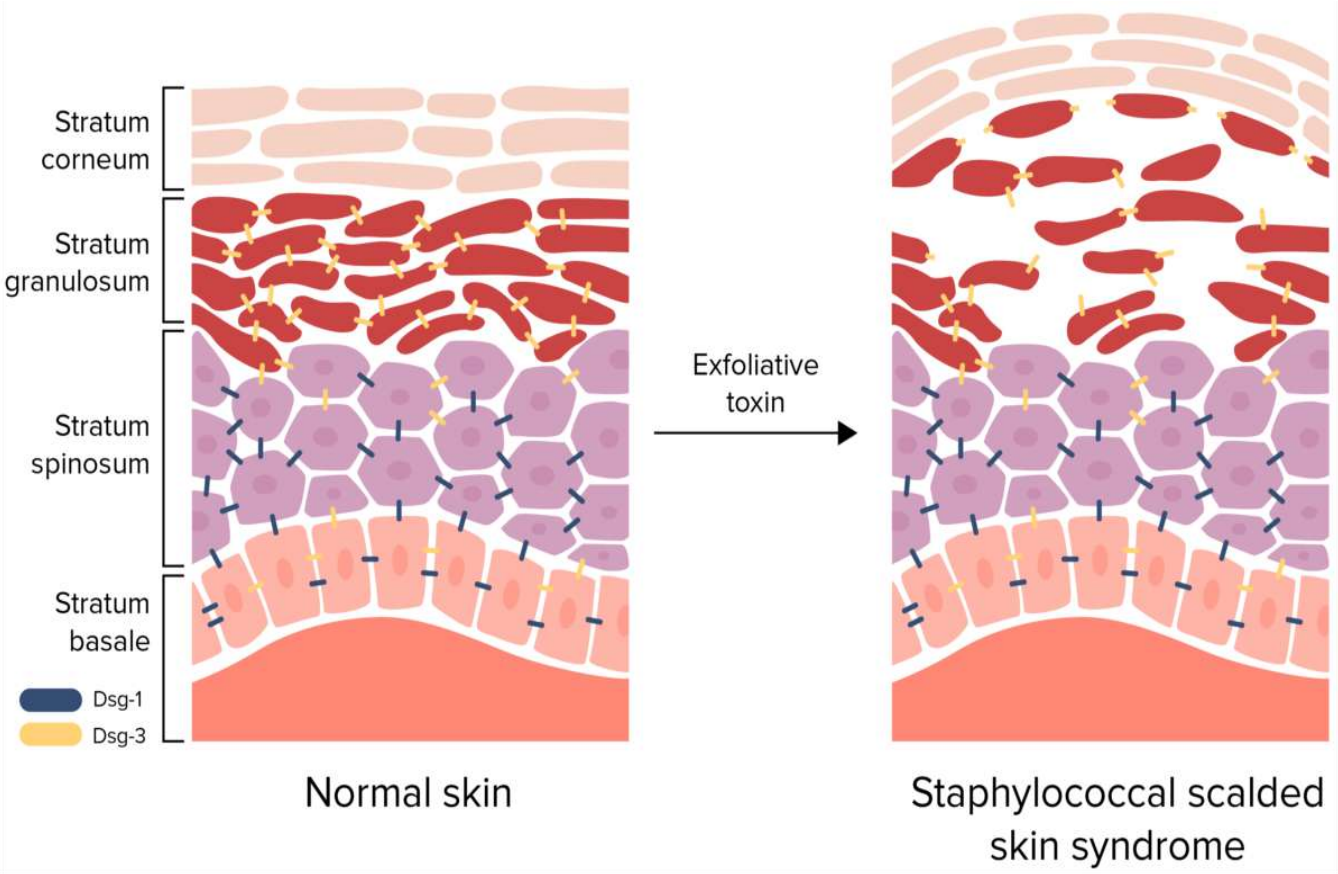
- Staphylococcal scalded skin syndrome (SSSS) is an acute skin condition caused by **exfoliative toxins** from *S. aureus*.
- SSSS primarily affects infants and young children and most often follows a staphylococcal infection.

Toxin-mediated diseases: Staphylococcal scalded skin syndrome-

Pathophysiology

- Starts as a localized, staphylococcal infection: Skin wounds, Conjunctivitis, Pharyngitis, Pneumonia.
  - The primary site of infection is not always evident.
- Staphylococcus produces exotoxin → spread haematogenously
  - Two types of exotoxins: Exfoliative toxin A and Exfoliative toxin B

Toxin-mediated diseases: Staphylococcal scalded skin syndrome-Pathophysiology (Cont.)



Exfoliative toxin A& B Cleave desmoglein (Dsg) 1 complex in the stratum granulosum

↓

Disrupts keratinocyte-to-keratinocyte adhesion

↓

Causes separation and detachment of the superficial epidermis

Quick Check:

The widespread blistering in SSSS is caused by the direct spread of *Staphylococcus aureus* bacteria throughout the body.

Toxin-mediated diseases: Staphylococcal scalded skin syndrome-Clinical Presentation

- Prodromal symptoms: Fever, irritability, malaise, and poor feeding.
- Sites of primary infection:
  - Infants: umbilical stump or diaper region
  - Older children: face
  - Frequently not evident

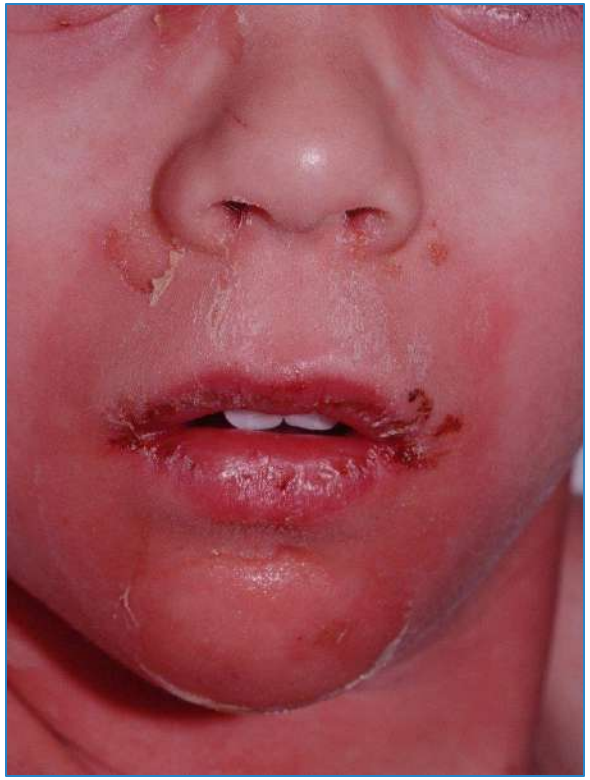
Toxin-mediated diseases: Staphylococcal scalded skin syndrome- Clinical Presentation (Cont.)

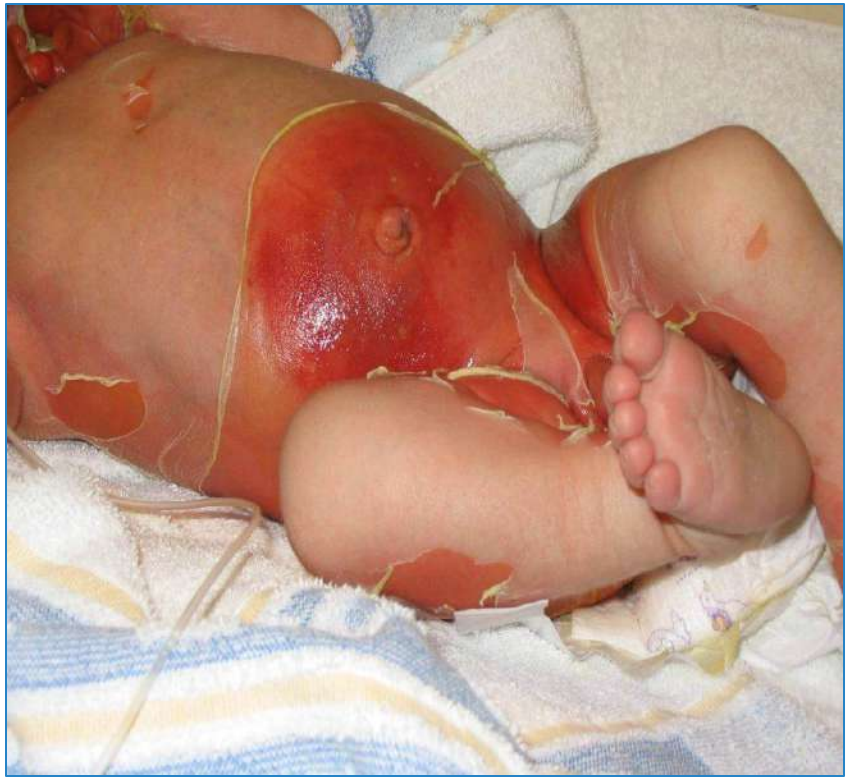
Cutaneous findings

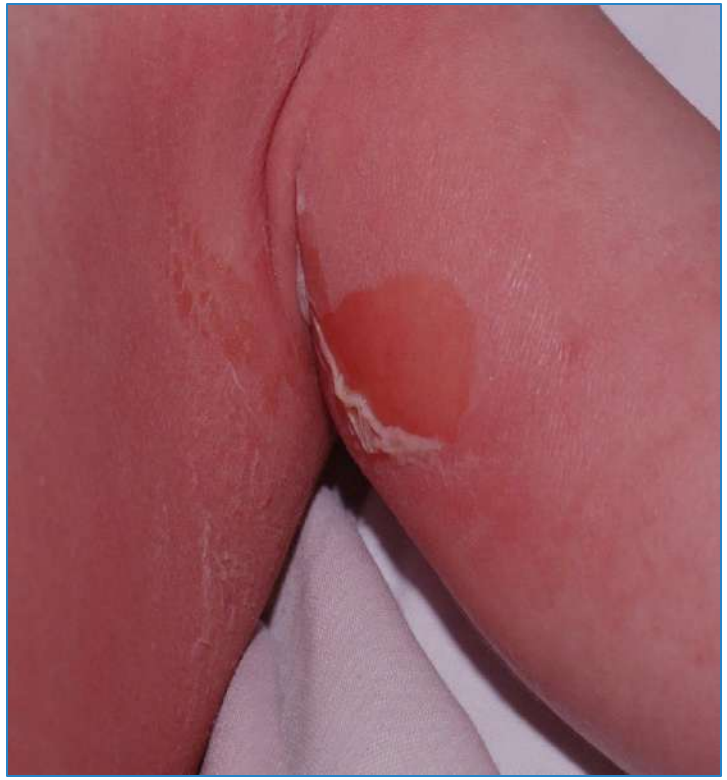
- Erythematous macules on the face and flexural surfaces (e.g., axilla, inguinal folds, gluteal cleft), and Skin pain
- Erythema spreads diffusely within 24–48 hours.
  - Resembles an acute burn
  - Skin peeling and erosions in areas of friction with red, moist skin underneath
  - Fissures and crusting around the mouth, eyes, and nose
  - Widespread desquamation may take place within 36–72 hours.
  - Healing occurs within 2 weeks.

Toxin-mediated diseases: Staphylococcal scalded skin syndrome- Clinical Presentation (Cont.)

Cutaneous findings







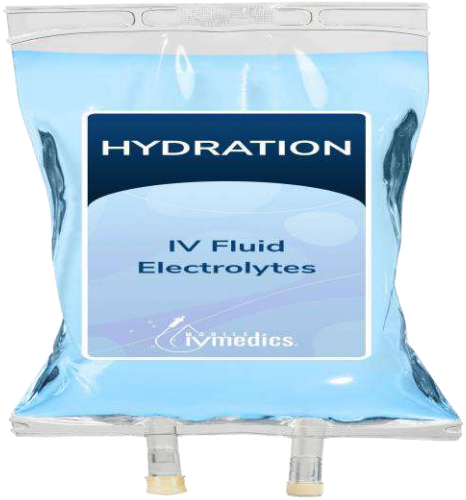
Toxin-mediated diseases: Staphylococcal scalded skin syndrome- Clinical Presentation (Cont.)

- The loss of the skin barrier predisposes patients to: dehydration, electrolyte imbalances, sepsis, or hypothermia

- Diagnosis: Usually diagnosed clinically.

Toxin-mediated diseases: Staphylococcal scalded skin syndrome-Treatment

- Antibiotic therapy
  - Patients without methicillin resistant *S. aureus* (MRSA) risk factors: Nafcillin OR oxacillin
  - Patients with MRSA risk factors: Vancomycin
- Supportive care
  - IV fluid hydration
  - Monitor and replace electrolytes
  - Gentle skin and wound care
  - Analgesia



Osteomyelitis

- Osteomyelitis is an infection of the bone that results from the spread of microorganisms from the blood (hematogenous), nearby infected tissue, or open wounds (non-hematogenous).
- Infections are most commonly caused by *S. aureus*, but a variety of organisms have been linked to osteomyelitis.

Osteomyelitis: Etiology and Classification

- Non-hematogenous osteomyelitis (80% of cases):
  - Caused by a spread of bacteria (typically multiple pathogens) from the surrounding environment.
  - Direct inoculation of bacteria due to: Surgery, Prosthetic devices, Trauma, Soft tissue infection.
  - Polymicrobial: *S. aureus* (present in > 50% of cases), *S. epidermidis*, Streptococcus.
- Hematogenous osteomyelitis (20% of cases):
  - Bacteria spread via blood supply from the primary site of infection.
  - Monomicrobial: *S. aureus* (most common), Streptococcus

Osteomyelitis: Clinical Presentation

Acute osteomyelitis

- Onset: within days or weeks; associated with acute bone inflammation
- Duration: < 2 weeks
- Signs and symptoms:
  - Localized swelling
  - Warmth
  - Erythema
  - Dull pain
  - Fever and chills

Osteomyelitis: Clinical Presentation

Chronic osteomyelitis

- Onset: develops slowly (over months or years) following acute infection
- Duration: typically > 6 weeks
- Associated with: avascular bone necrosis and sequestrum formation (necrotic bone fragment that has become detached from the original bone)
- Signs and Symptoms:
  - Similar to acute osteomyelitis
  - Intermittent bone pain
  - Draining sinus tract (pathognomonic)
  - Systemic findings: typically absent; may include low-grade fever, malaise

Osteomyelitis: Diagnosis

- Routine studies:
  - CBC → thrombocytosis, possible leukocytosis
  - inflammatory markers → ↑ CRP, ↑ ESR
  - blood cultures → May be positive in hematogenous osteomyelitis but typically negative in exogenous osteomyelitis
  - If there is Purulent wounds/sinuses: Consider culture of purulent material.
  - Suspected hematogenous osteomyelitis : Consider additional studies (e.g., urine culture, chest x-ray) based on clinical presentation.

Osteomyelitis: Diagnosis (Cont.)

- Imaging:
  - X-ray: low sensitivity and specificity for osteomyelitis
  - MRI: Most sensitive and specific modality for osteomyelitis
- Consider bone biopsy with cultures to confirm the diagnosis if imaging findings and blood cultures inconclusive: .

Osteomyelitis: Treatment

- **Antibiotic therapy:**
  - Start most patients directly on pathogen-directed antibiotics based on culture results.
    - Methicillin-susceptible S. aureus (MSSA) → oxacillin
    - Methicillin-resistant S. aureus (MRSA) → Vancomycin
  - Consider switching to oral antibiotics after an initial IV course.
  - Duration of therapy is **normally 4–8 weeks**.

Osteomyelitis: Treatment (Cont.)

- **Surgery:**
  - Chronic osteomyelitis or acute osteomyelitis refractory to antibiotic treatment → Debridement of necrotic bone and tissue and amputation may be considered in severe disease.
  - Infected prosthetic joint or foreign body → Removal to promote remission

Septic Arthritis

- Septic (infectious) arthritis is an infection of the joint space, which can occur in a native joint or a prosthetic joint.
- Patients with underlying joint diseases (e.g., rheumatoid arthritis) are at an increased risk of septic arthritis.
- Routes of infection include hematogenous spread (most common), direct inoculation (e.g., iatrogenic, penetrating trauma), and contiguous spread.
- Causative organisms: *S. aureus* (Most common in adults and children > 2 years)

Septic Arthritis: Clinical Presentation

- Patients with native joint infections usually present with:
  - An acutely swollen painful joint
  - Limited range of motion
  - Fever
- Patients with prosthetic joint infections (PJIs) usually have a milder, chronic course, which often makes diagnosis more challenging.

Septic Arthritis: Diagnosis

- Diagnosis: arthrocentesis: a diagnostic and/or therapeutic procedure in which synovial fluid is aspirated from a joint using a sterile needle.
  - Indicated in all patients with suspected septic arthritis.

Septic Arthritis: Treatment

- Joint drainage:
  - Native joints: Therapeutic arthrocentesis (drained to dryness) is indicated in all patients.
  - Prosthetic joints: Surgery to remove pus and infected tissue from the affected joint is typically required
- Antibiotic therapy: early administration of empiric antibiotic therapy then switch to culture-specific antibiotics once antibiotic sensitivities are known.
  - Gram-positive cocci → Vancomycin (empiric)
  - MSSA → Nafcillin
  - MRSA → Vancomycin

Thank you