

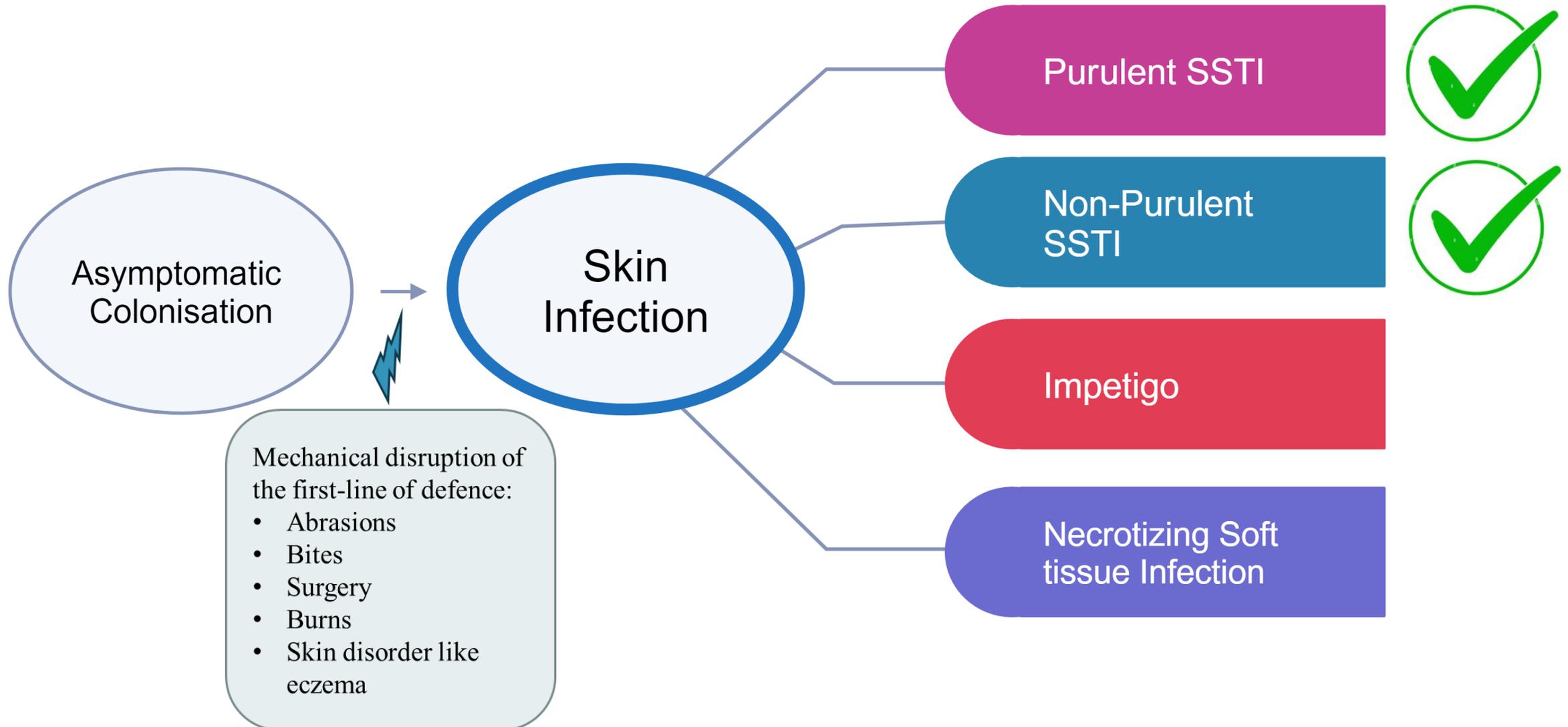
Bacterial Skin and Soft Tissue Infections 2

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Outlines

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

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
 - *S. pyogenes*: causes approximately 10% of cases



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 → 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)



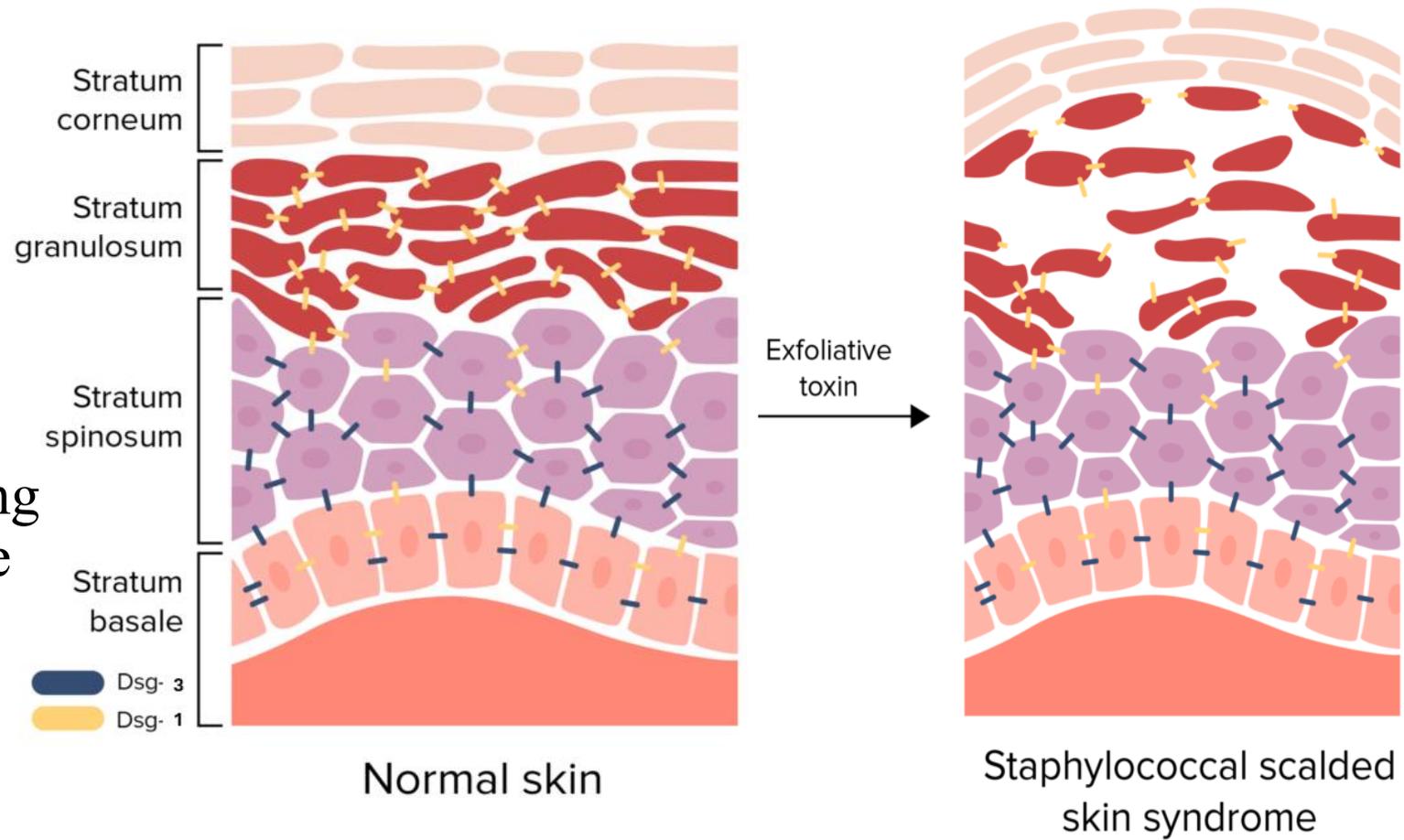
Impetigo: Subtypes - Bullous Impetigo



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.



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 15 to 20% → **surgical emergency**
- Incidence **rare**, with an incidence of 0.3 to 15 cases per 100,000 individuals

Necrotizing Fasciitis: Etiology

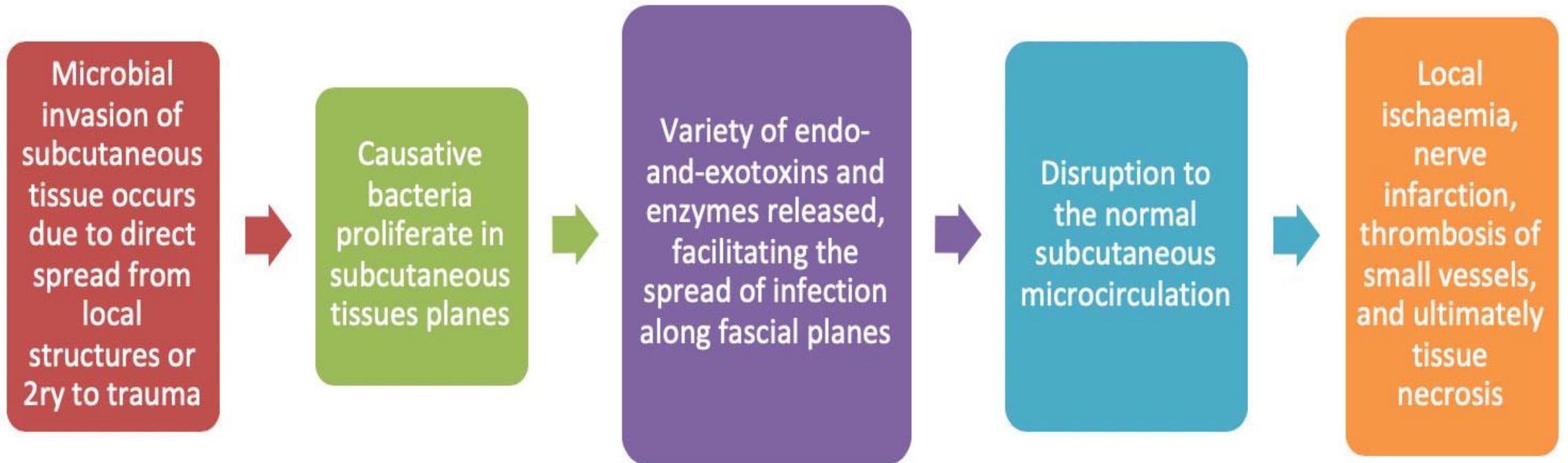
NF is divided into microbiologic categories based on the causative organism(s):

- 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 disproportionate 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

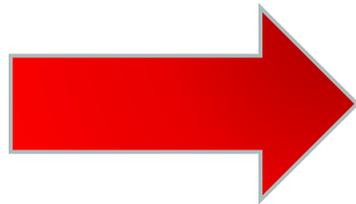


- High fever, tachycardia, hypotension, and/or **altered mental status.**



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**.



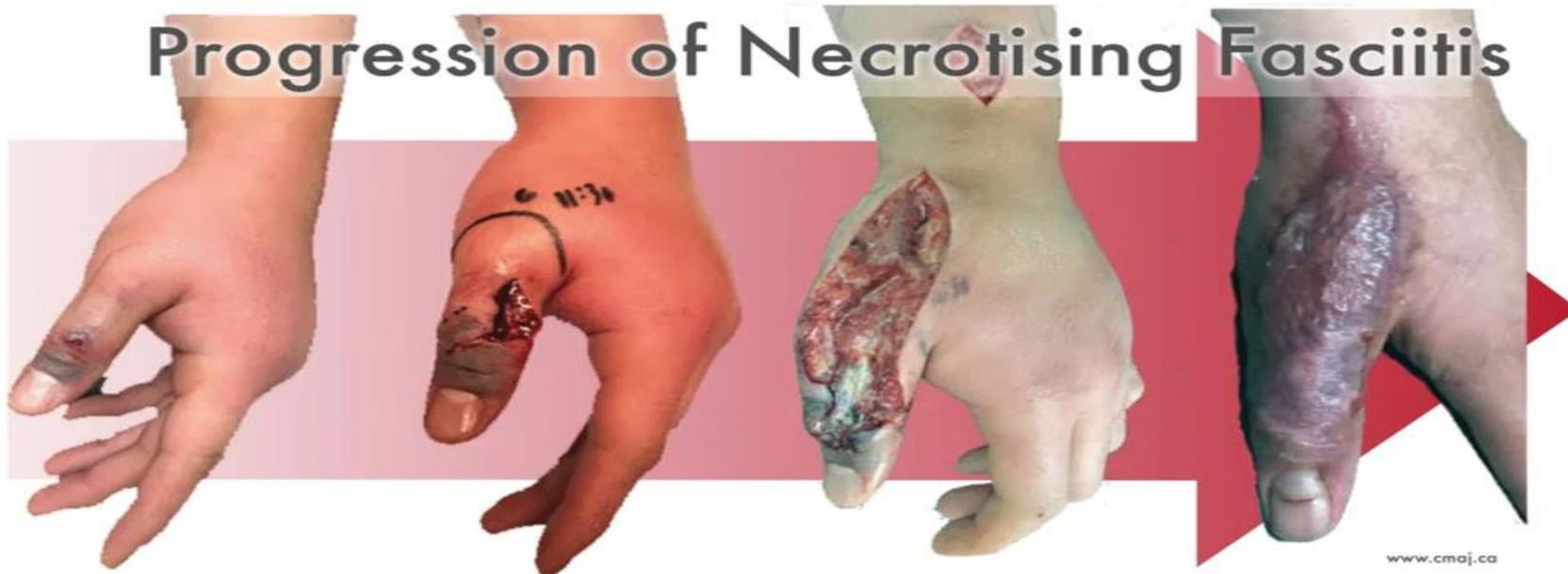
Necrotizing Fasciitis: Case Report (Cont.)

- 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**.



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.

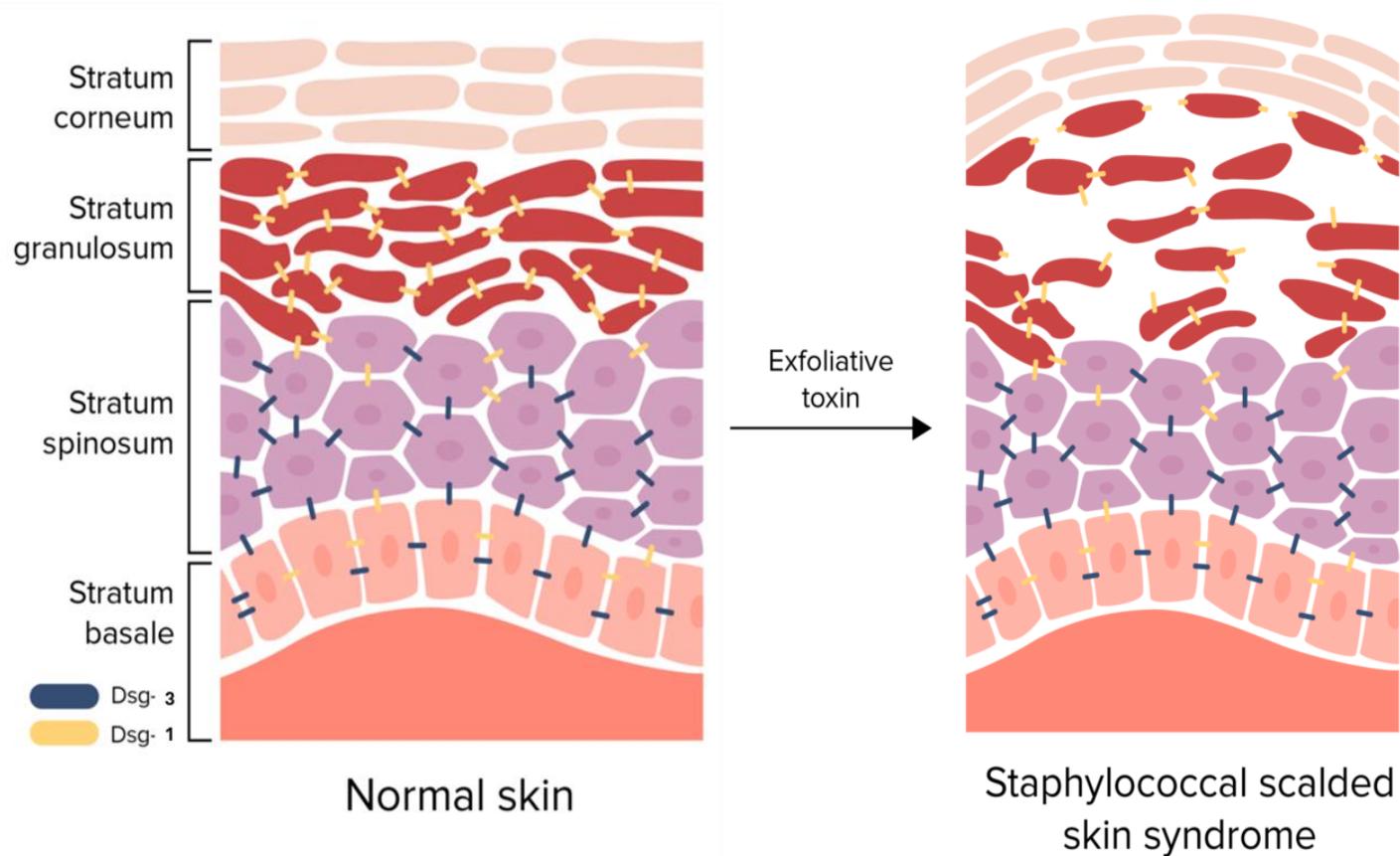
Toxin-mediated diseases: Staphylococcal scalded skin syndrome-

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



Thank you



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