Bacterial Infections and Infectious Dermatologic Emergencies

Learning Objectives

- Common Bacterial Infections
  - recognition
  - treatment
  - complications

- Infectious Dermatologic Emergencies
  - Necrotizing Fasciitis
  - Toxic Shock Syndromes

Normal Skin Flora

Major function is to prevent skin infections

- Provides ecological competition for pathogens
- Hydrolyzes the lipids in sebum into free fatty acids which are toxic to many bacteria- linoleic and linolenic acid are more inhibitory of Staph Aureus
- Antimicrobial Peptides from lamellar bodies, Cathelicidins, and Defensins function to control overgrowth of pathogens

Normal Skin Flora

Aerobic Cocci

- *Staphylococcus epidermidis*
  - Most common coccus on human skin
  - All body sites, especially intertriginous areas
- *Staphylococcus aureus*
  - More common in Atopic Dermatitis, Diabetes Mellitus, Hemodialysis, IVDU, Liver Disease, and HIV
  - resident or contaminant?
  - anterior nares- 20-35%
  - perineum- 20%
  - axillae and toe webs- 5-10%

Normal Skin Flora

Aerobic Coryneform Bacteria

- Corynebacterium minutissimum- intertriginous sites
  - Erythrasma

Anaerobic Coryneform Bacteria

- Propionibacterium acnes- sebaceous glands, hair follicles
  - Acne vulgaris

Gram Negative Bacteria

- Acinetobacter species- axillae, perineum, antecubital fossae

  - Requires moisture and maceration which increases pH and CO_2 levels

Yeast

- Pityrosporum ovale/Malassezia furfur- sebaceous sites
  - Tinea Versicolor

Common Bacterial Infections

Introduction

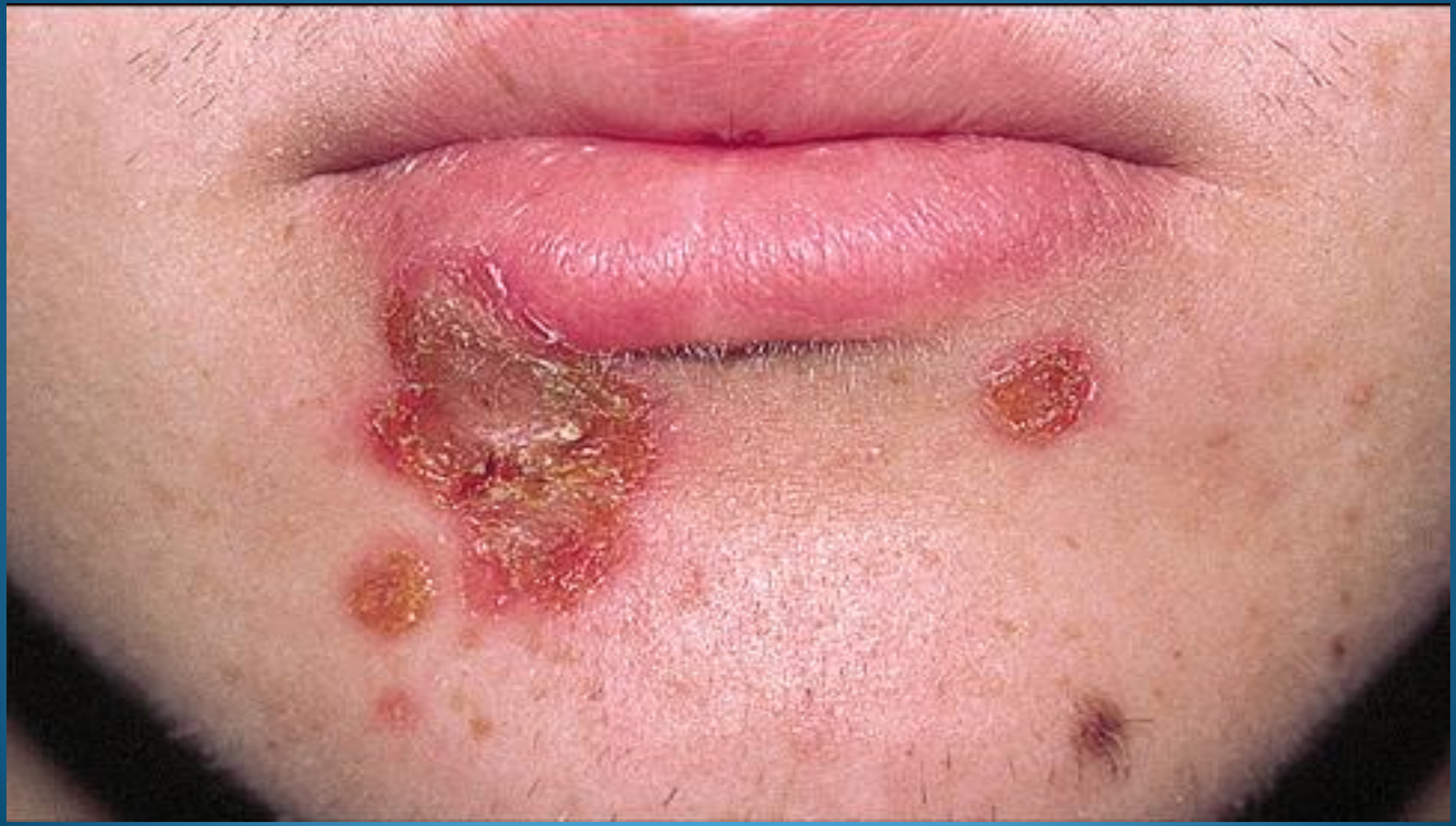
- *Strep* and *Staph* cause the majority of skin infections in immunocompetent patients

- Immunodeficiency and underlying systemic disease result in severe infections which tend to be refractory to treatment

Skin Disease related to bacterial pathogens may be:

1. Direct skin infection
2. Toxin mediated cutaneous eruption
3. Secondary skin infection of a primary skin disease
4. Manifestation of primary infection of another organ system
5. Reactive skin condition from antigen/immune mediated process

Impetigo Contagiosa

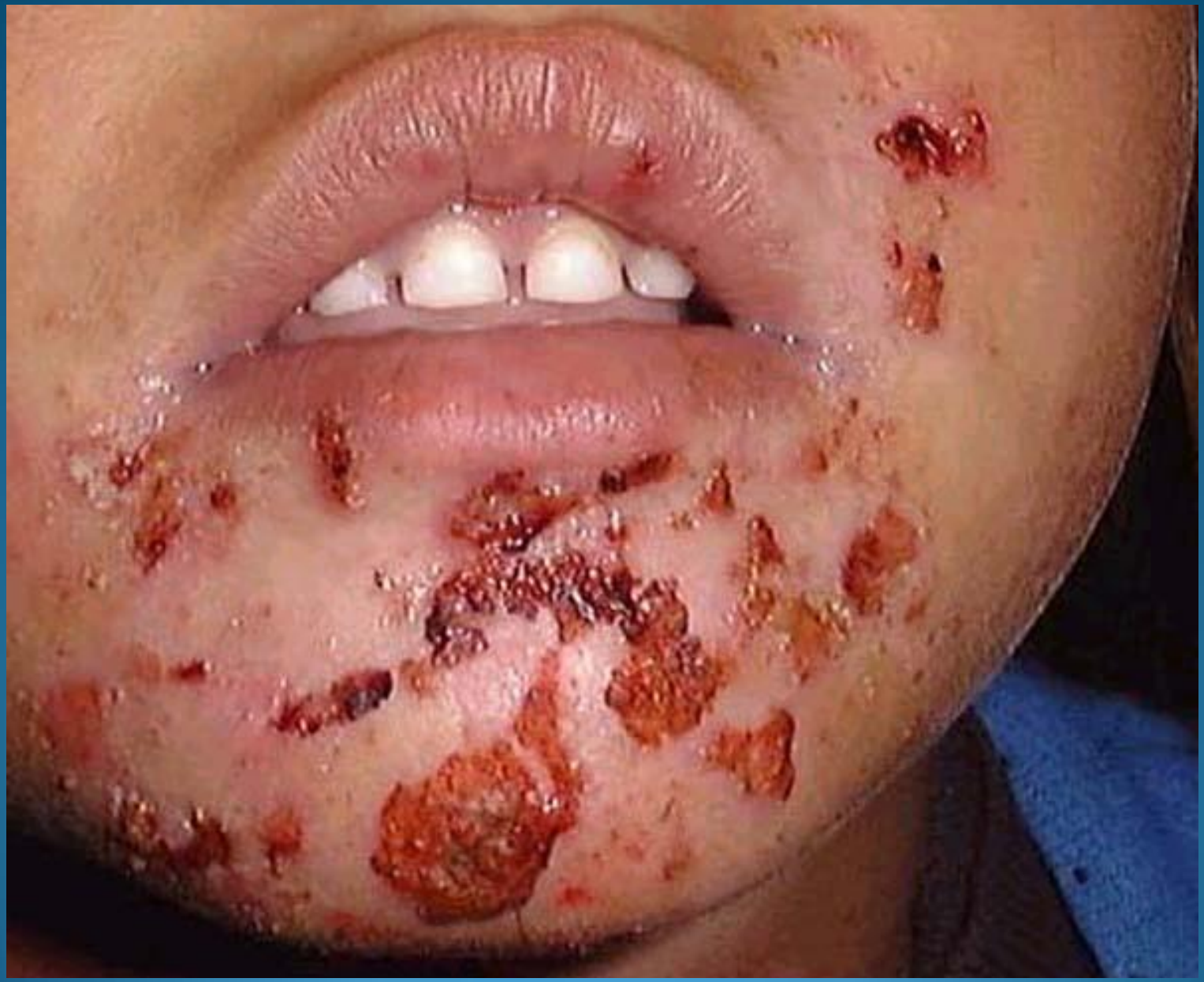


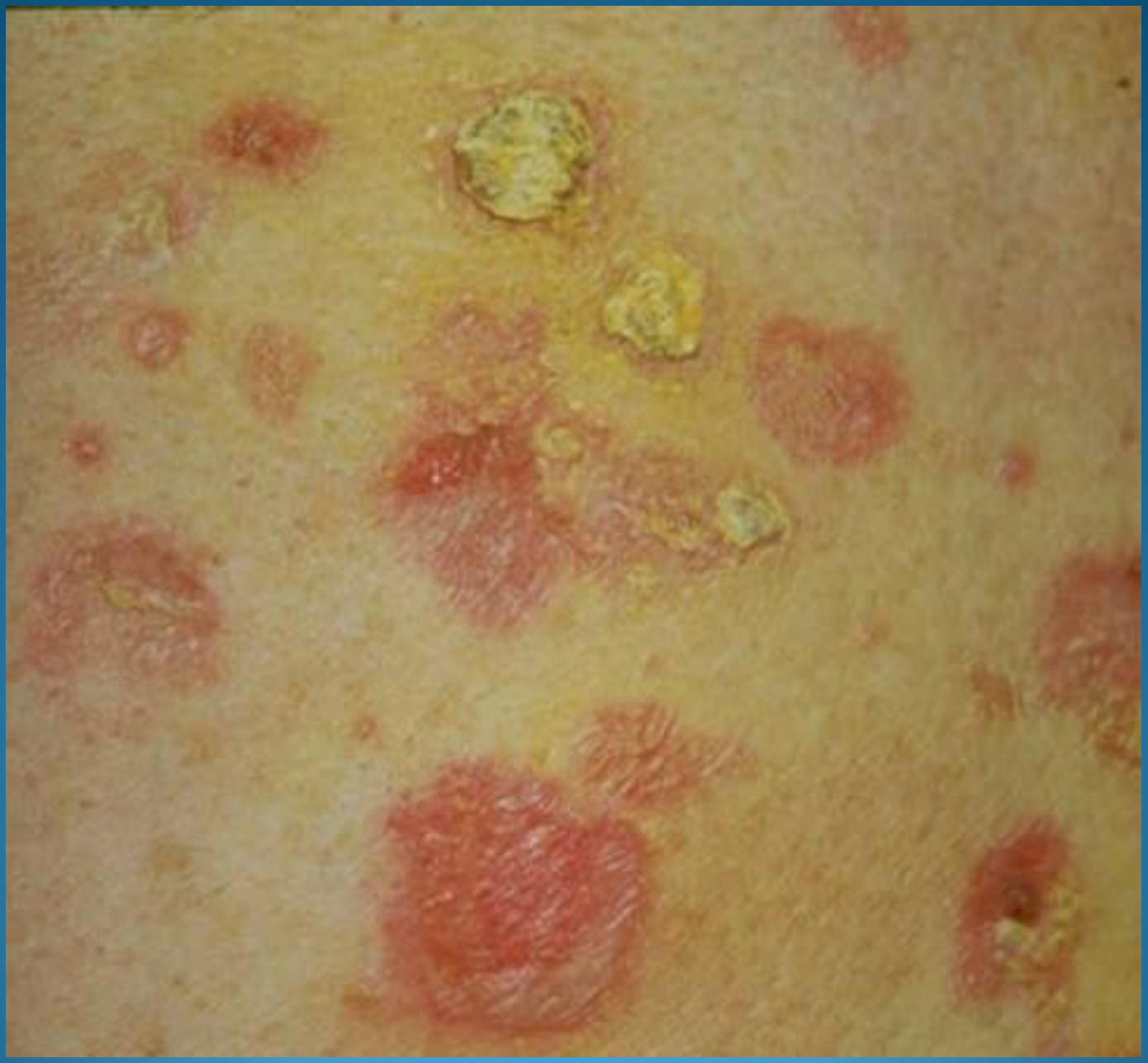
Impetigo Contagiosa

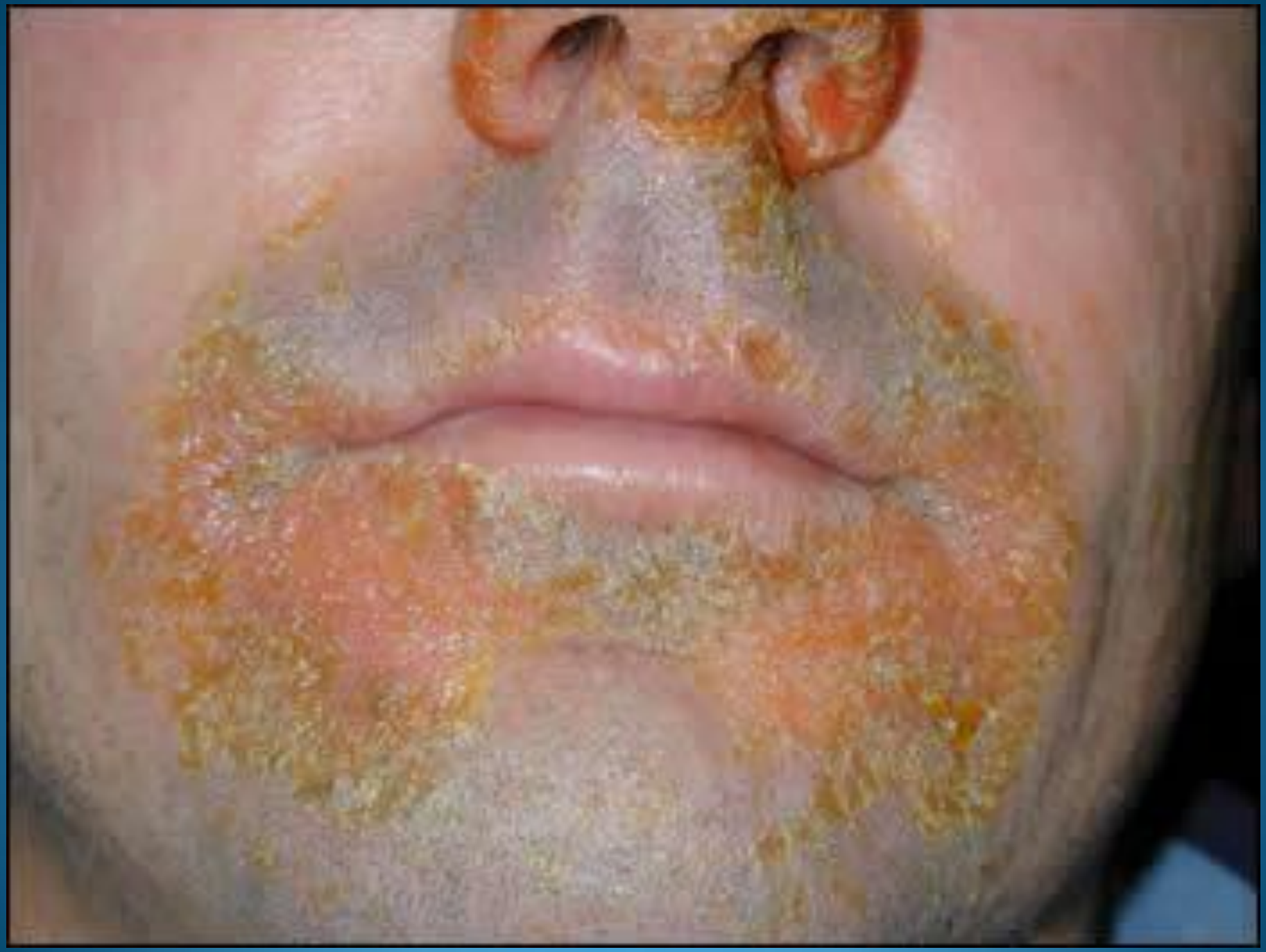
- Most common bacterial skin infection in kids
- Highly contagious via direct contact
- Incidence increased in late Summer and early Fall
- Non-bullous and bullous forms
- Predisposing factors
  - warm temps
  - high humidity
  - poor hygiene
  - *skin trauma - tinea pedis, varicella, HSV, scabies, insect bites*
  - nasal and/or perineal colonization

Non-bullous Impetigo

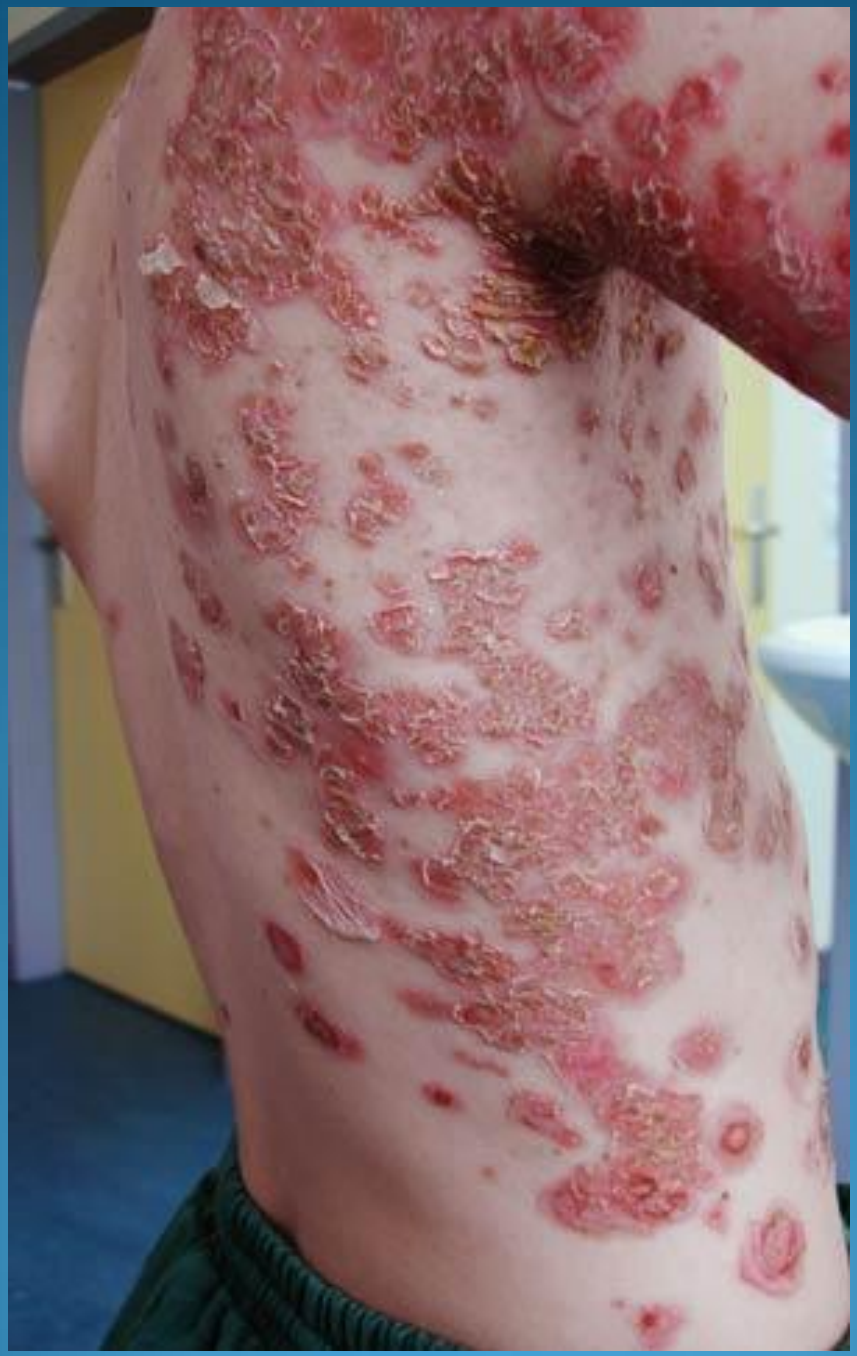
- *Staphylococcus aureus* primarily (developed countries)
- Previously *S. pyogenes (continues to be in developing countries)*
- Bacteria adhere to proteins exposed by trauma or breaks in the skin
- 70% of all impetigo

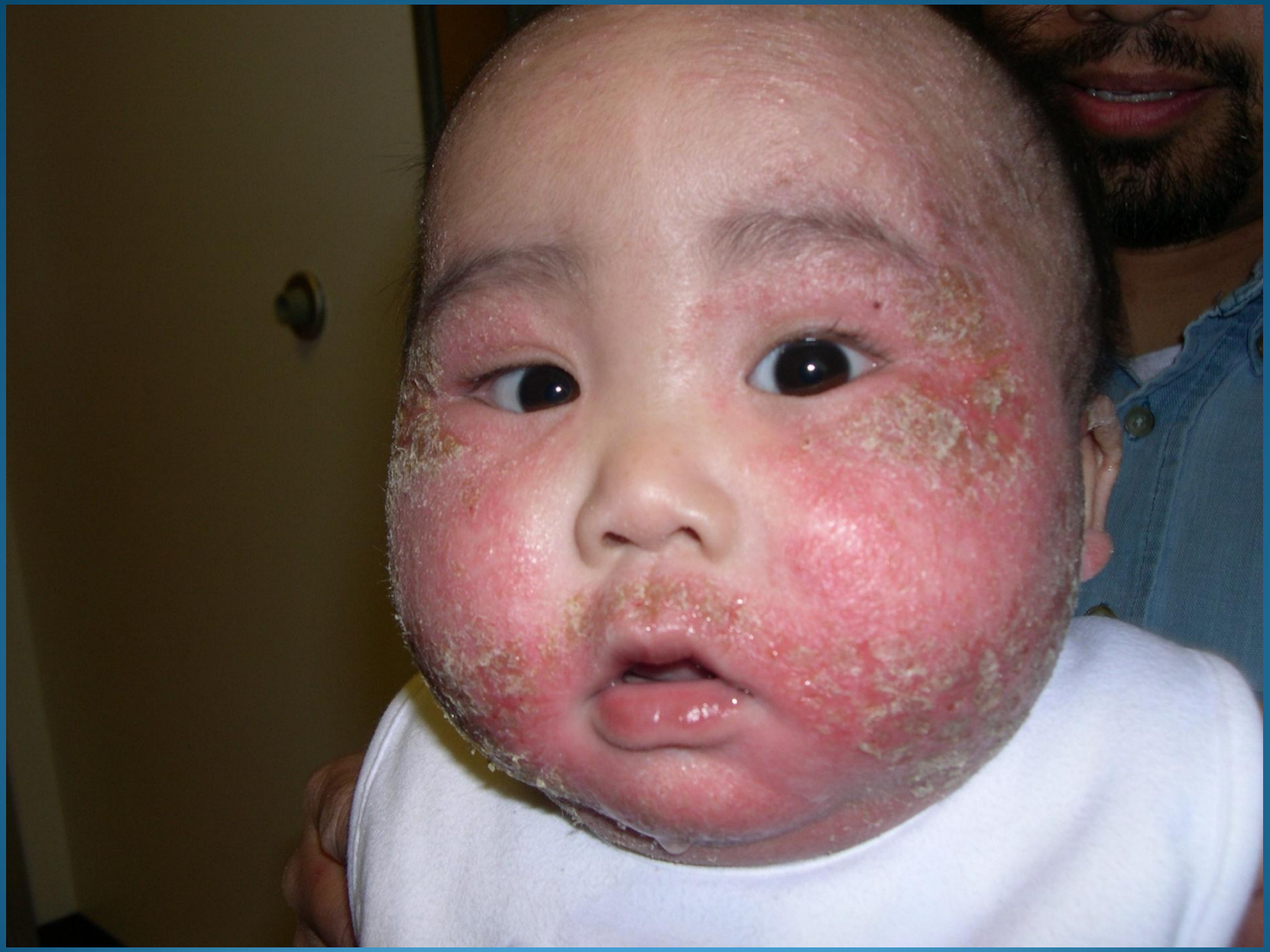


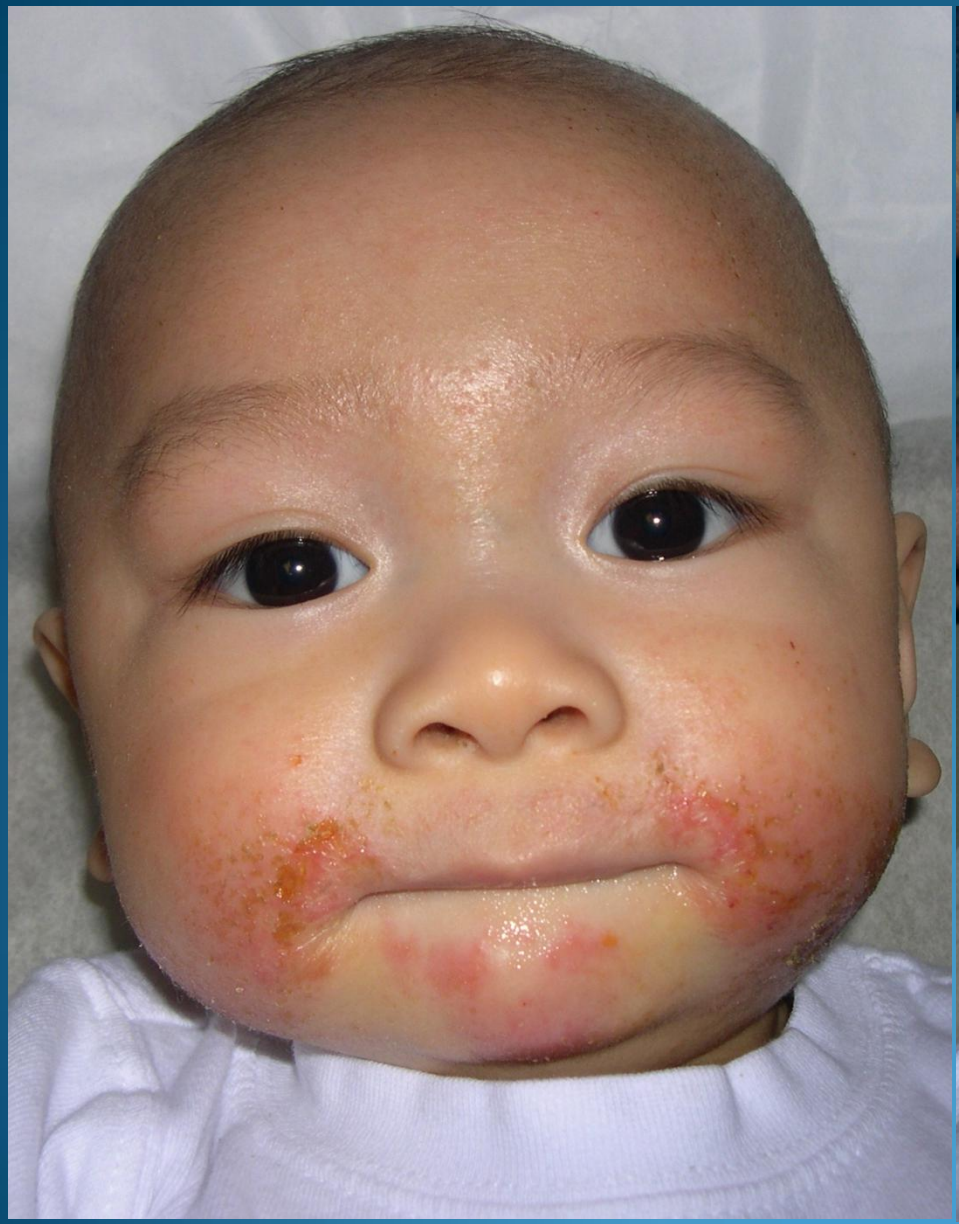


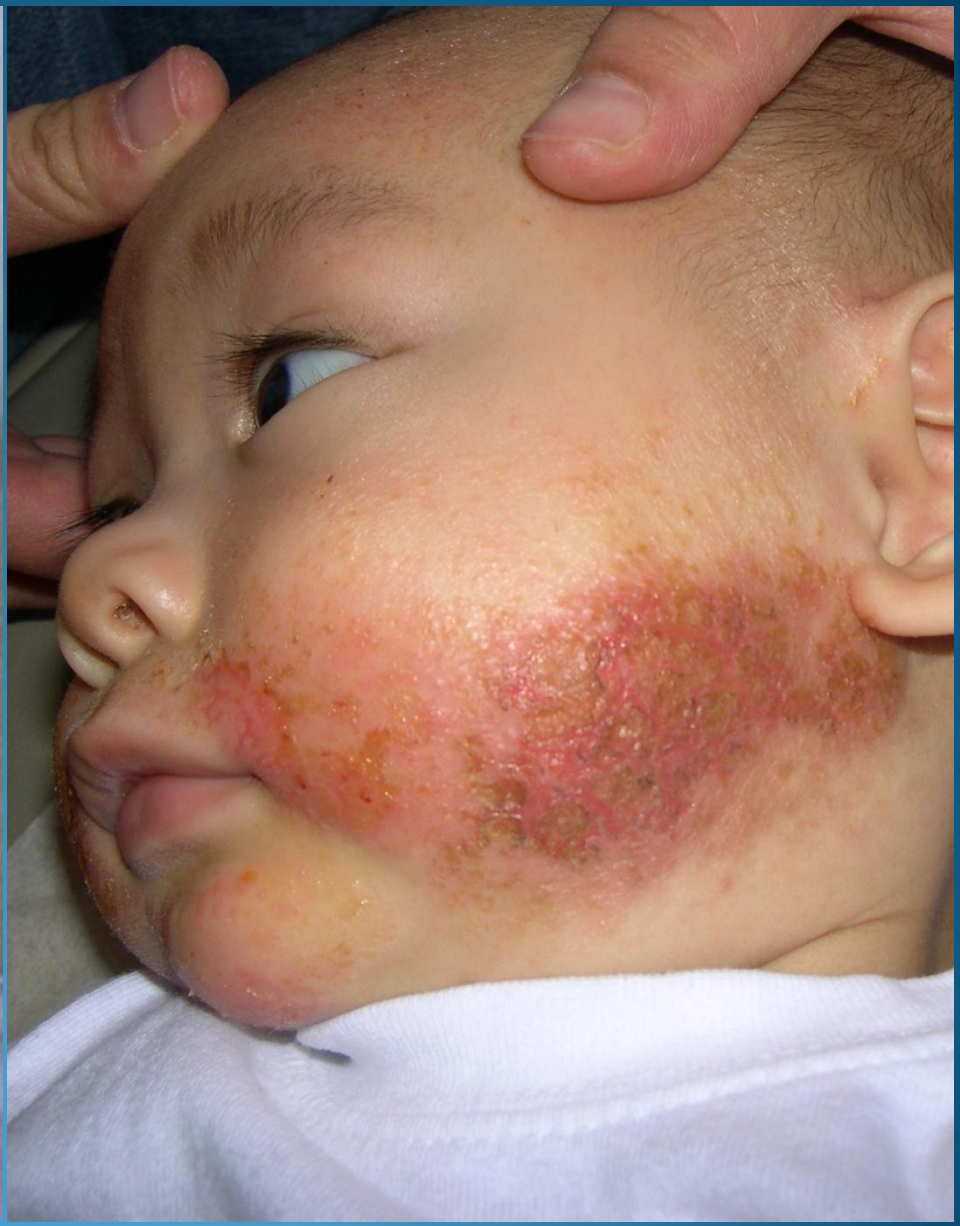


Impetiginization



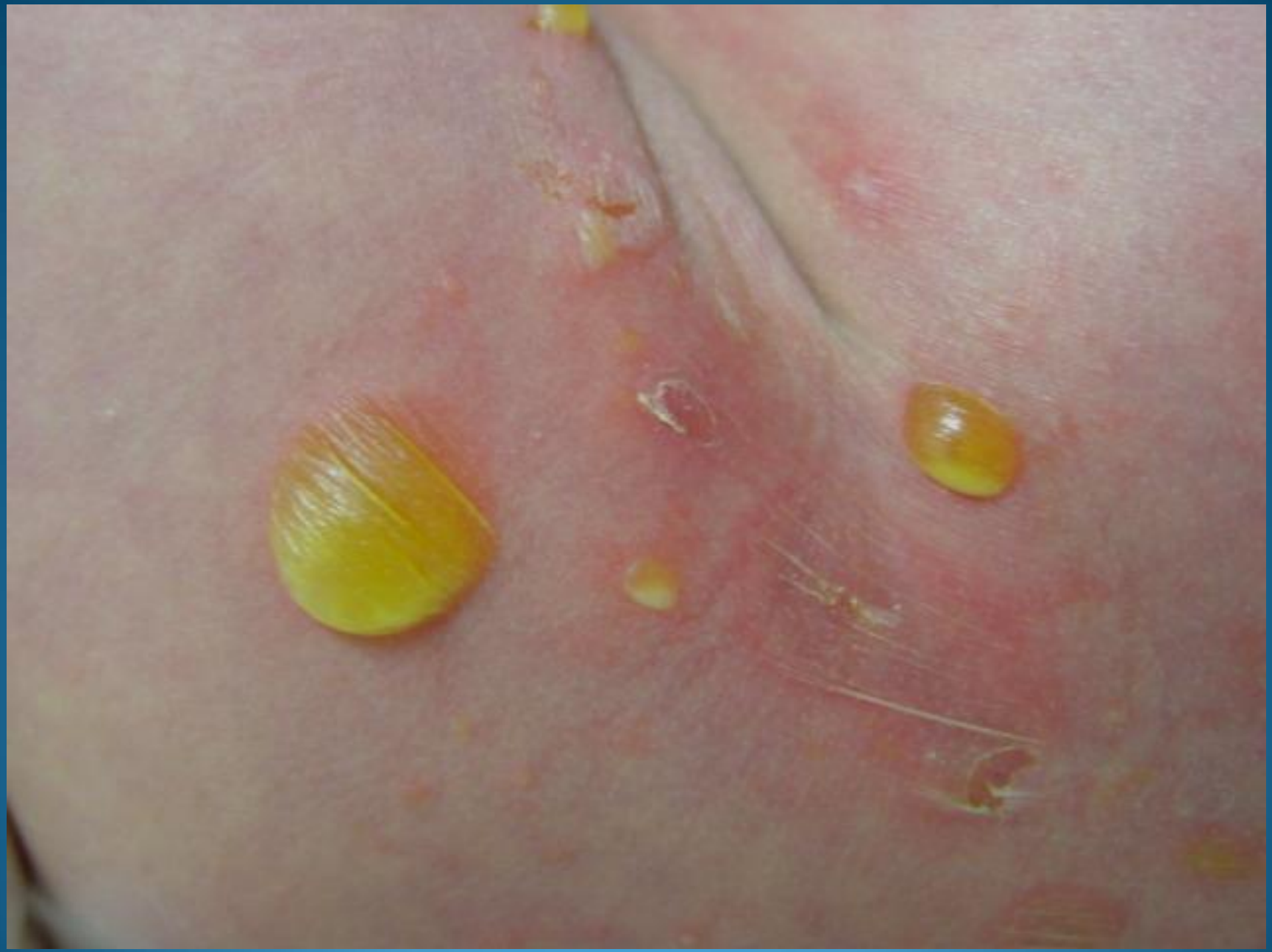




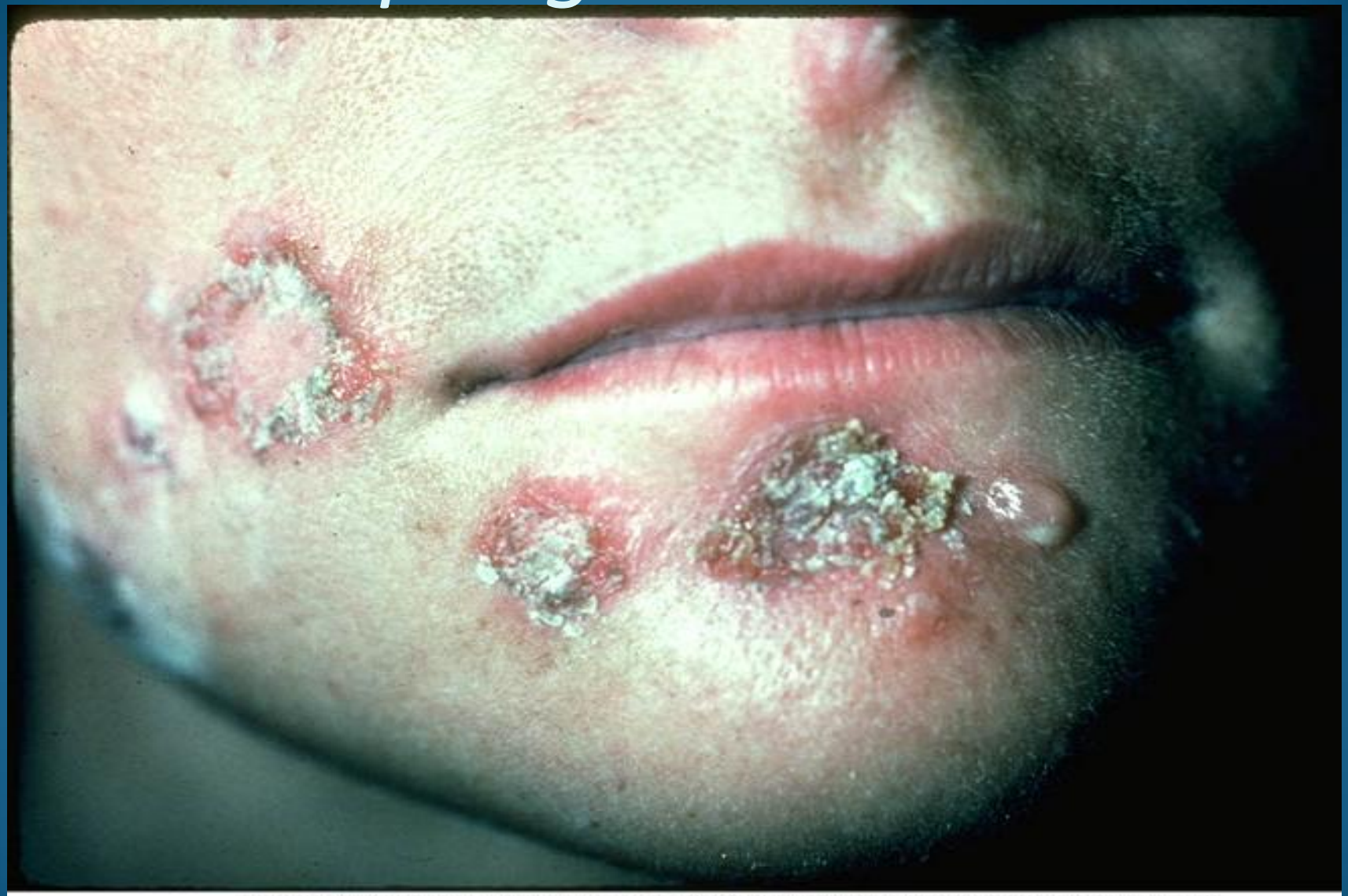


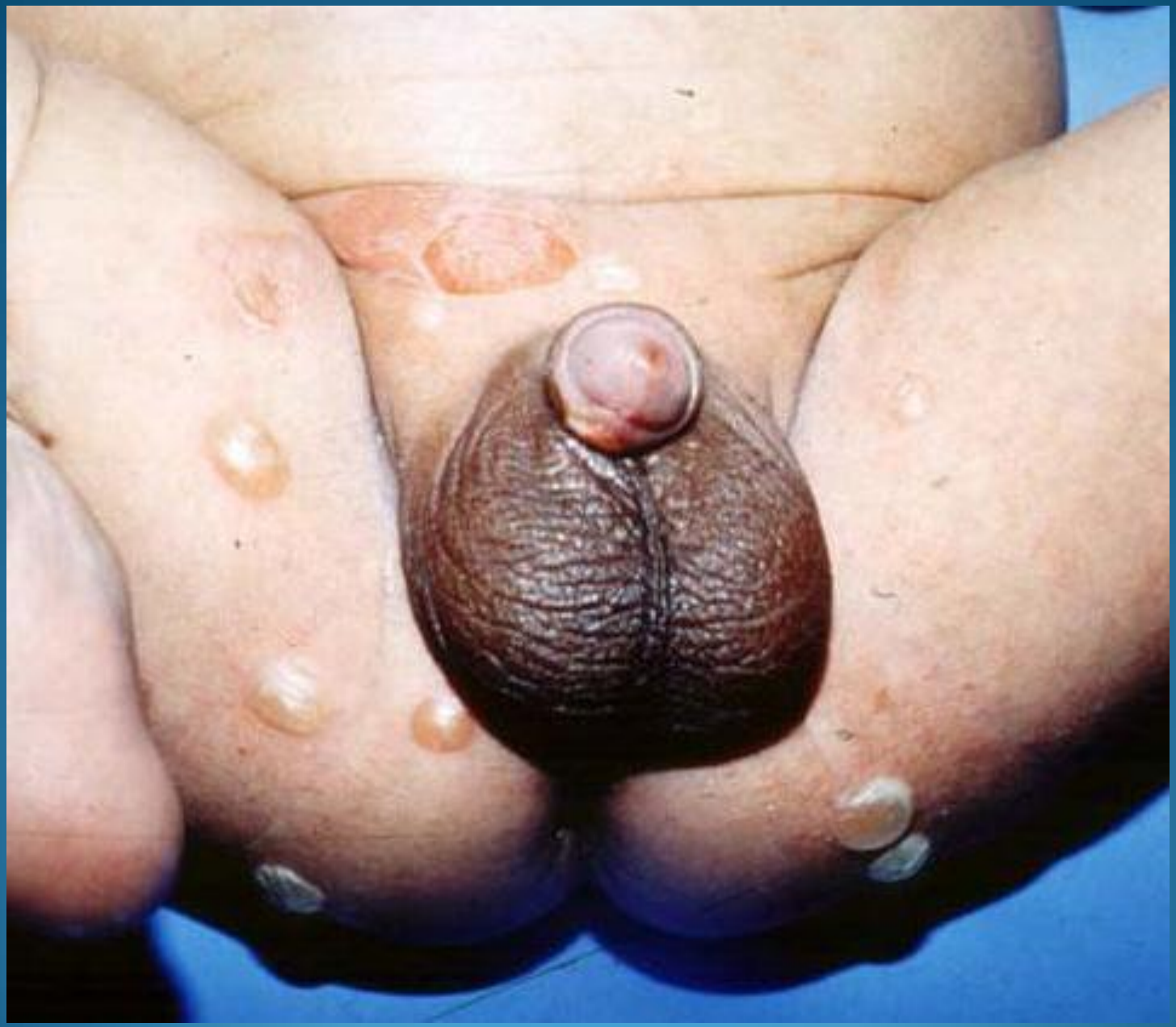
Bullous Impetigo (hx pemphigus neonatorum)

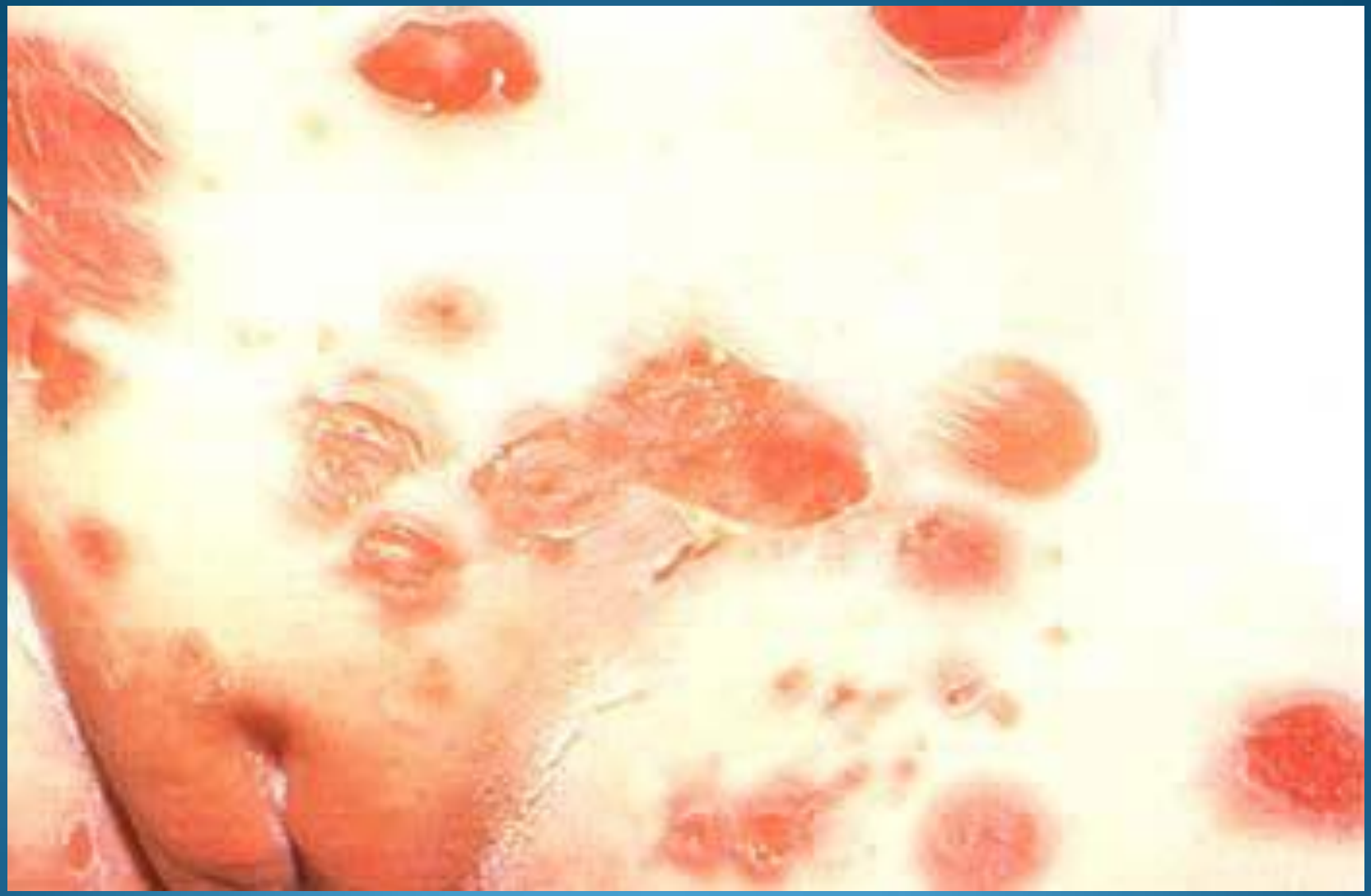
- *Staph aureus* phage types 55 and 71 or related group 2 phage type
- Flaccid blisters mediated by Staph toxins
  - Toxins attack cell adhesion molecule desmoglein 1
  - "localized Staph Scalded Skin"
  - Nikolsky sign negative
- Lesions appear on intact skin
- Neonates primarily
  - Present with weakness, fever, diarrhea

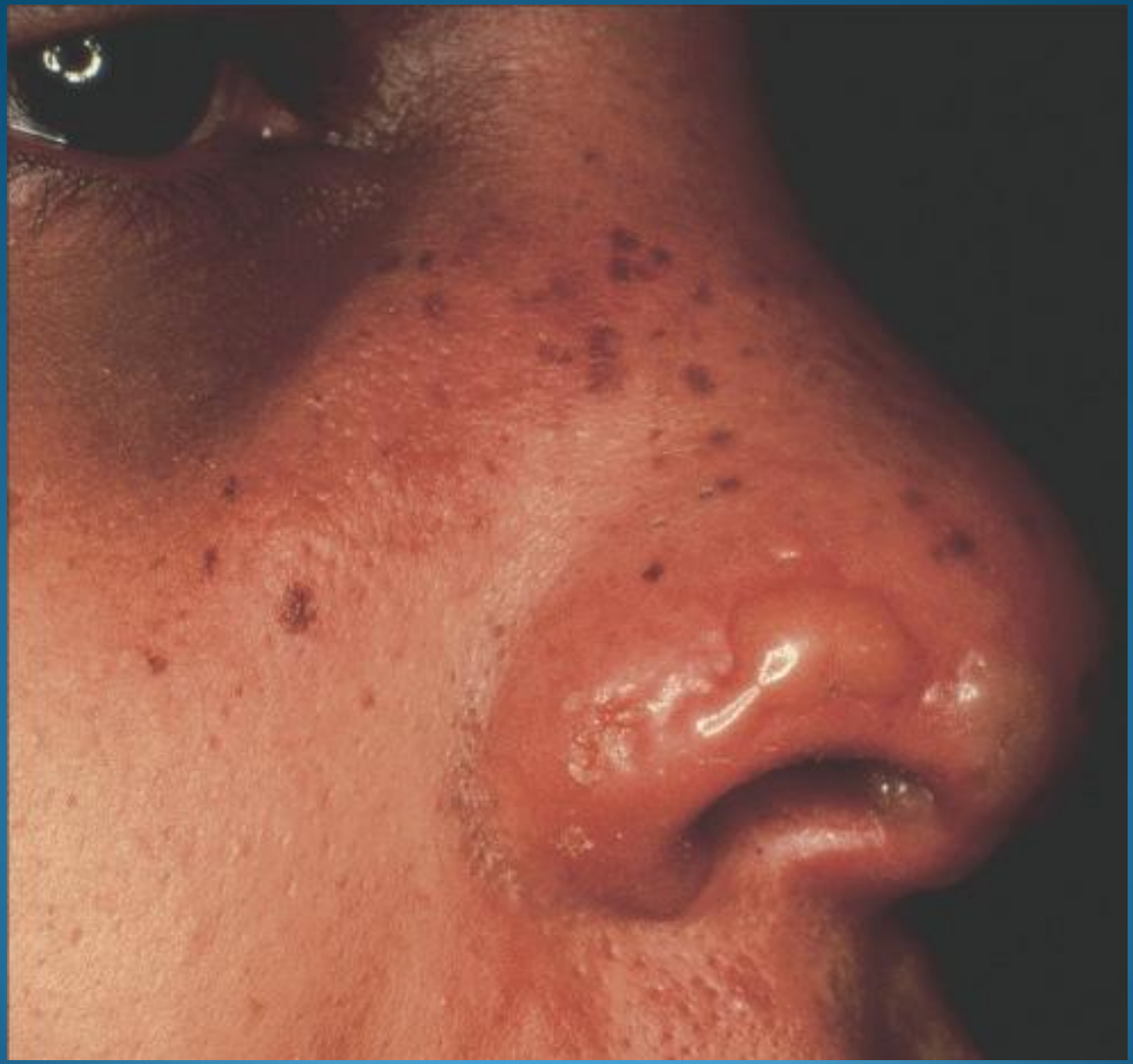


Impetigo Circinata



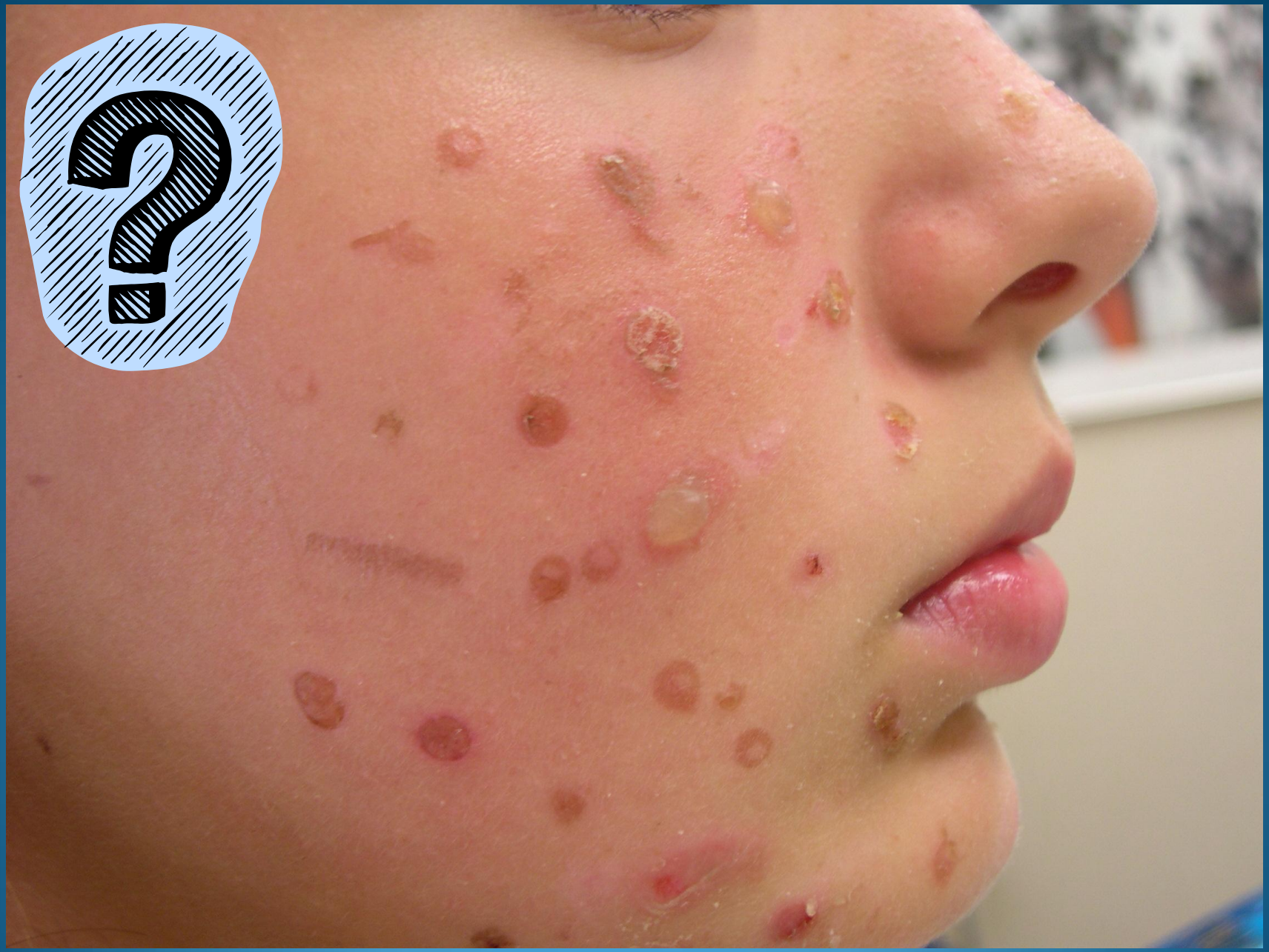






Impetigo: Work-up

- Gram stain of exudate
  - neuts with Gram + cocci in chains or clusters
- Culture
  - *S. aureus* is cultured consistently from fluid
  - Streptococci are found only occasionally
    - Usually in combination with Staph
  - Must rule out resistant organisms (MRSA)
- Biopsy is indicated in some cases



Impetigo: Treatment

- Saline compresses to remove fluid and crust
- Mild, localized cases:
  - Remove crust, topical mupirocin to lesions and nares/anus
- Extensive and/or bullous cases
  - systemic antibiotics are required 7 days for Staph, 10 days for strep
  - dicloxacillin or cephalexin
  - Erythromycin for PCN allergic
- If recurrent, culture nares and treat for carriage

Impetigo: Complications

- Post-streptococcal GN (all age groups)
- Meningitis or sepsis (infants)
- Ecthyma, erysipelas, cellulitis
- Bacteremia
- Osteomyelitis, septic arthritis
- Pneumonia

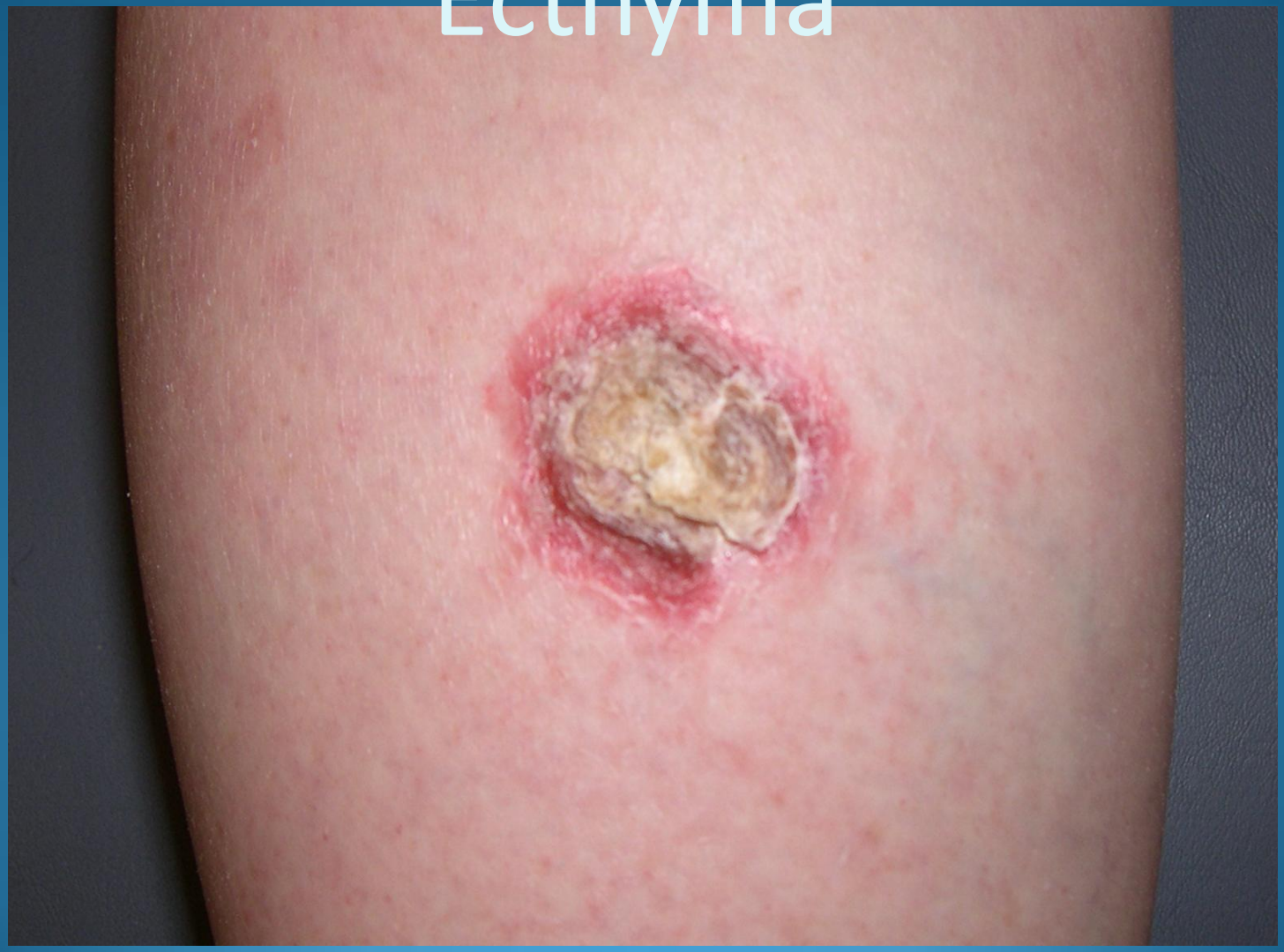
Post-Streptococcal Glomerulonephritis

- Not prevented with antibiotic treatment
- 2-5% of impetigo cases, 10-15% if nephritogenic strains
- Nephritogenic strains include: serotype 49, 55, 57, 60 and M type 2

Impetigo: Prognosis

- Early, appropriate therapy results in complete recovery within 7-10 days
- Early treatment limits the probability of scarring and complications
- Neonates have increased incidence of generalized infection and meningitis
- Antibiotic treatment will neither prevent nor halt glomerulonephritis
- Culture is mandatory if the lesions have not resolved within 7-10 days

Ecthyma



Ecthyma

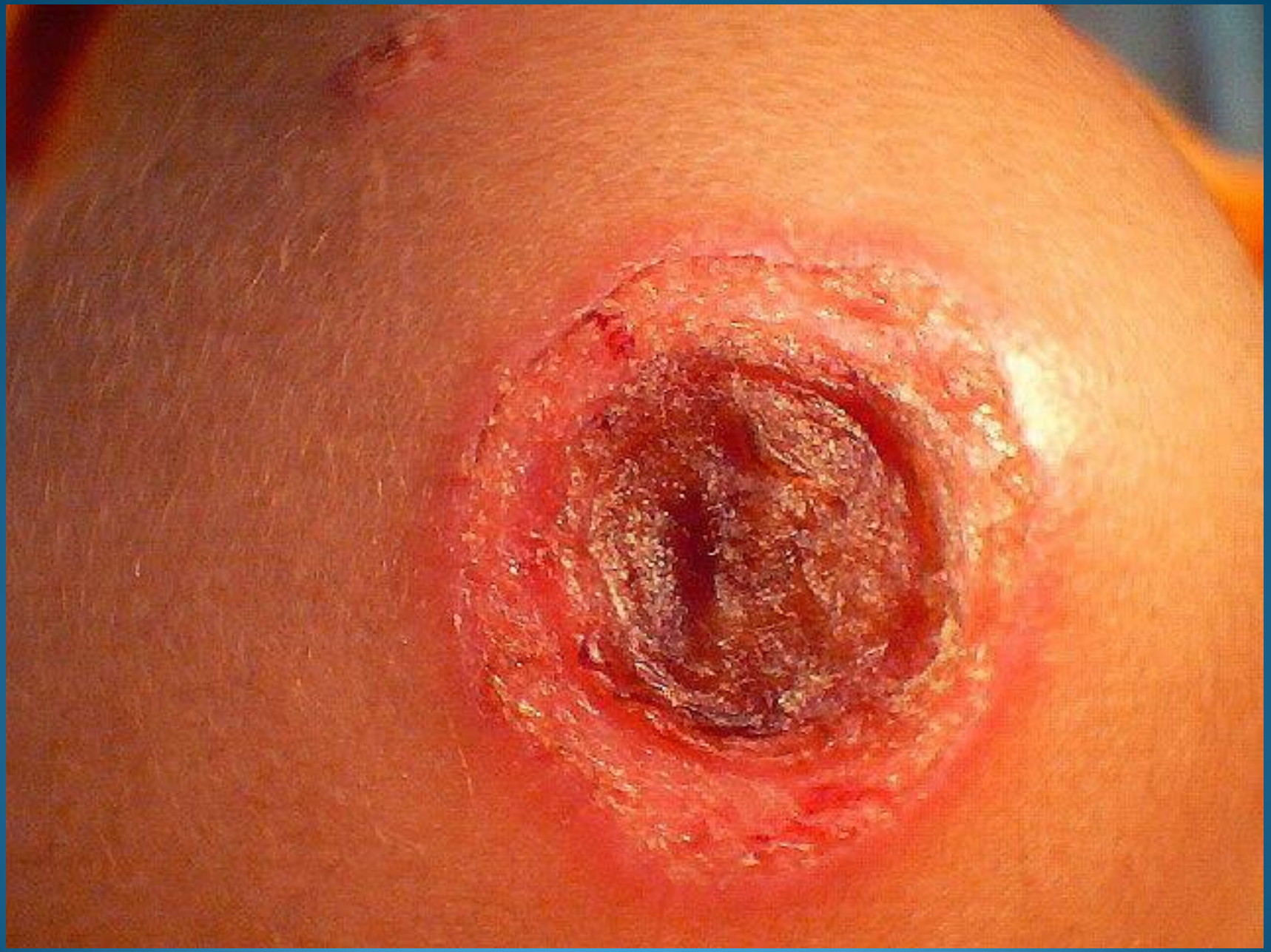
- Initially described in wartime on covered feet
- Deeper form of impetigo
- Thickly crusted erosions or ulcerations
- *Streptococcus pyogenes*: primary agent
- Rapidly superinfected with *Staph aureus*
- Lower extremities, usually < 10 lesions

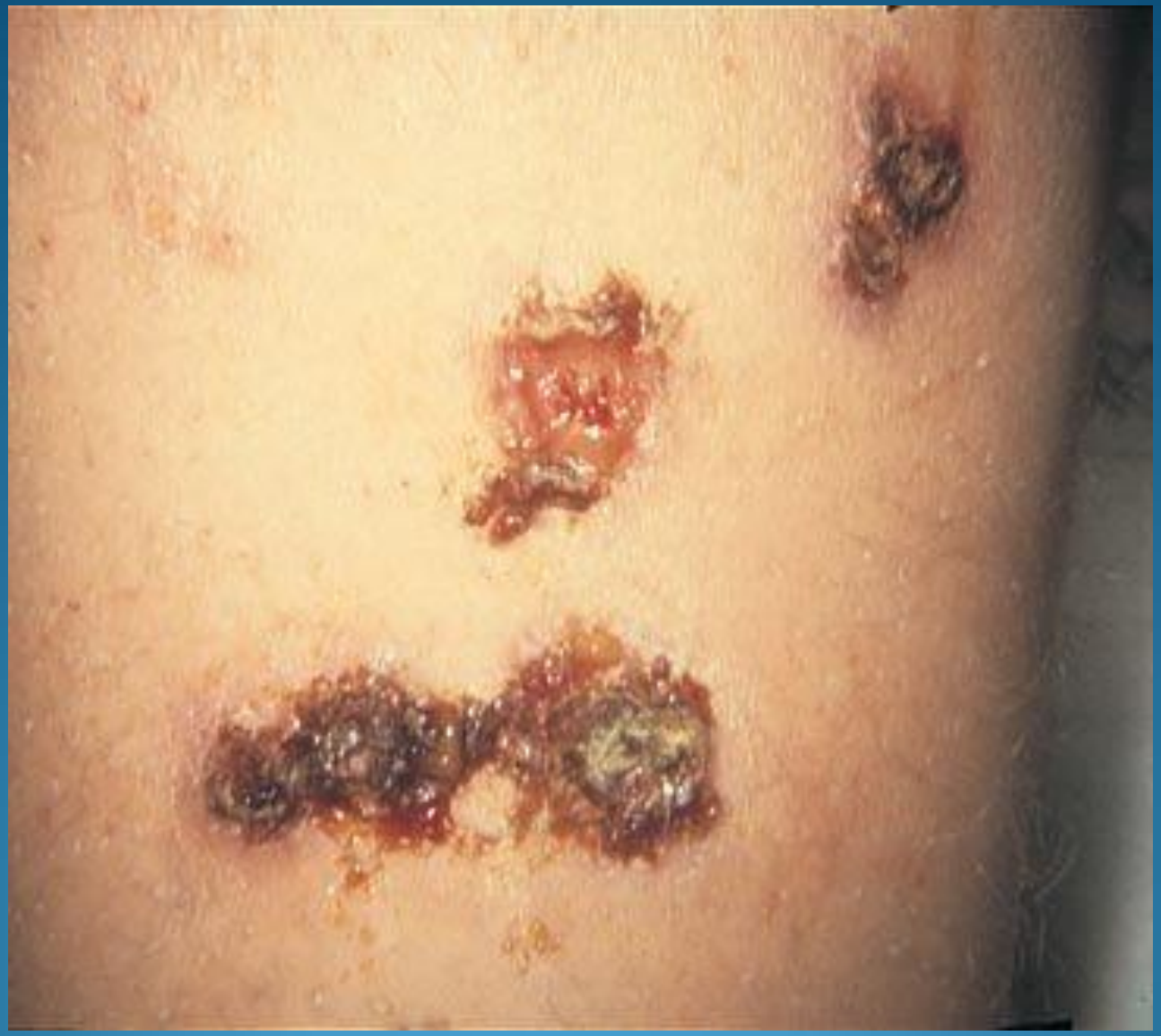
Ecthyma

High risk populations

- poor hygiene
- minor trauma
- children
- neglected elderly
- lymphedematous limbs
- immunocompromised

- Vesicule → pustule → 'punched out' crusted ulcer
- Purulent, necrotic base
- Heals with a scar
- Rare complications
  - bacteremia
  - cellulitis
  - osteomyelitis

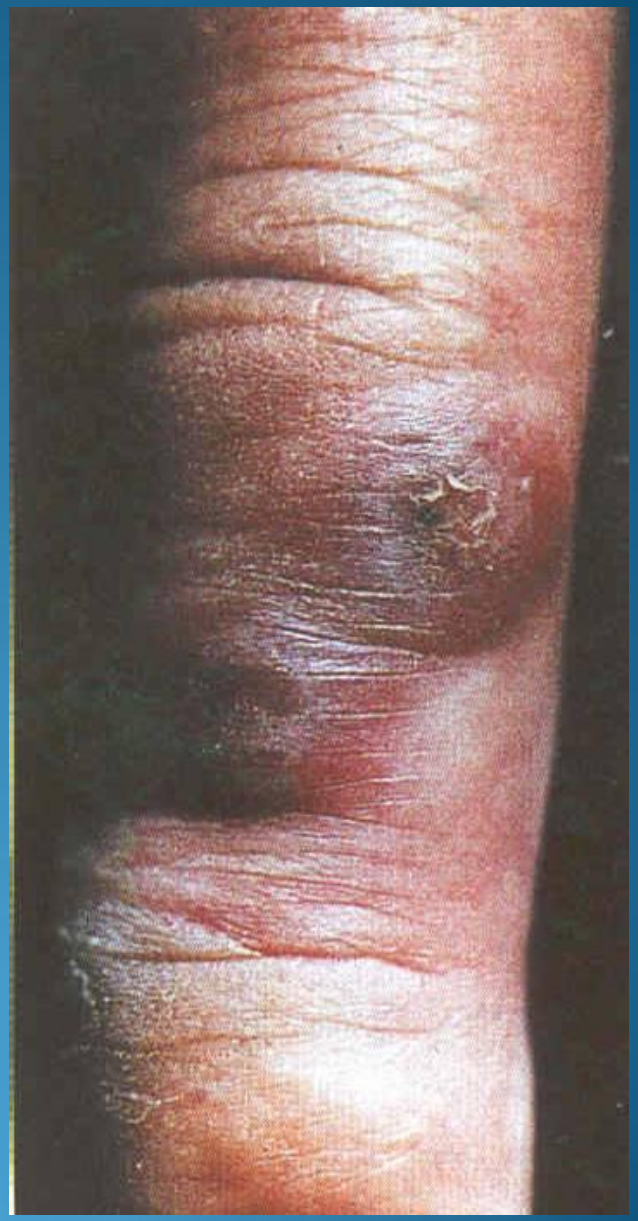




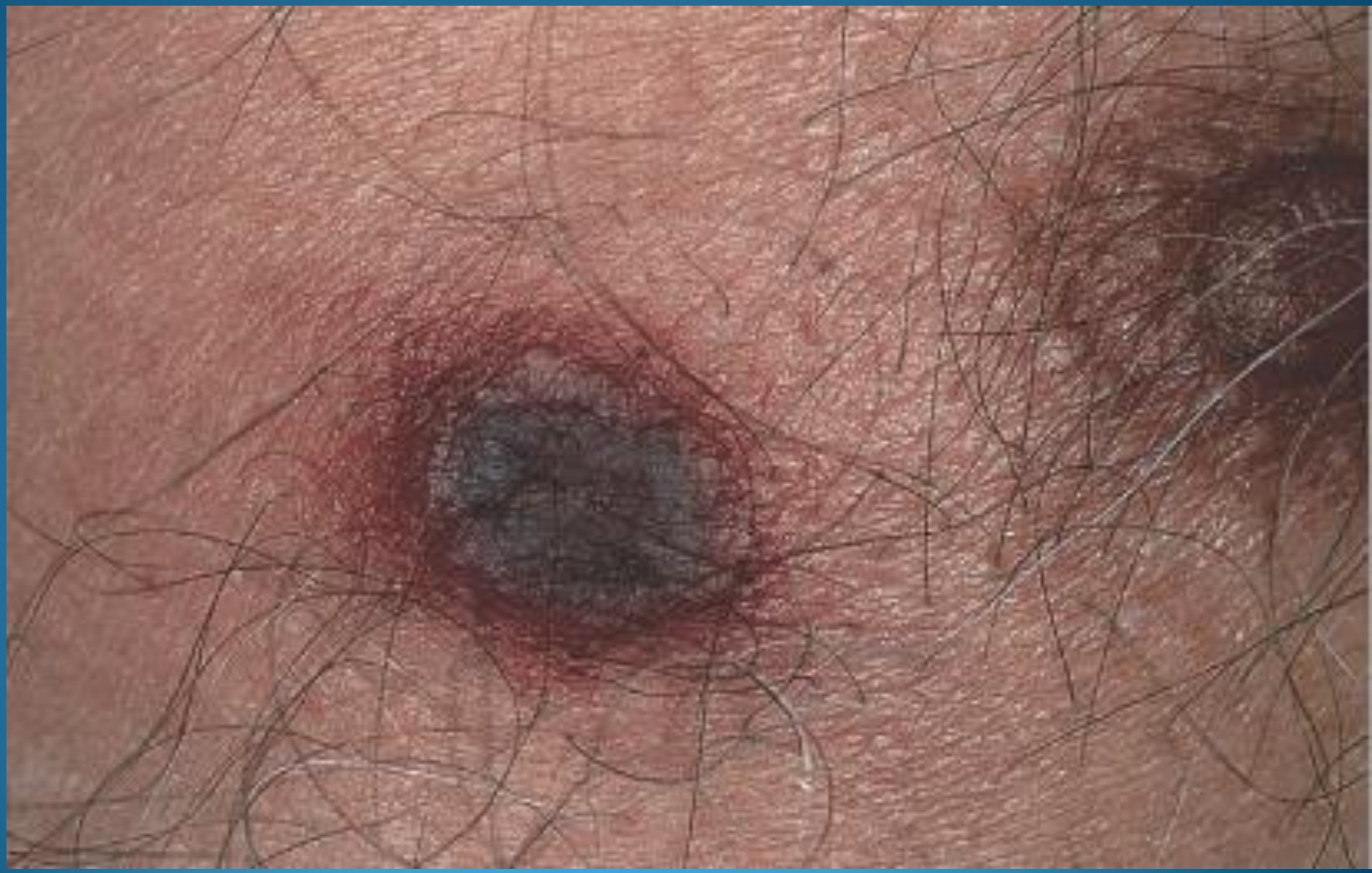
Ecthyma

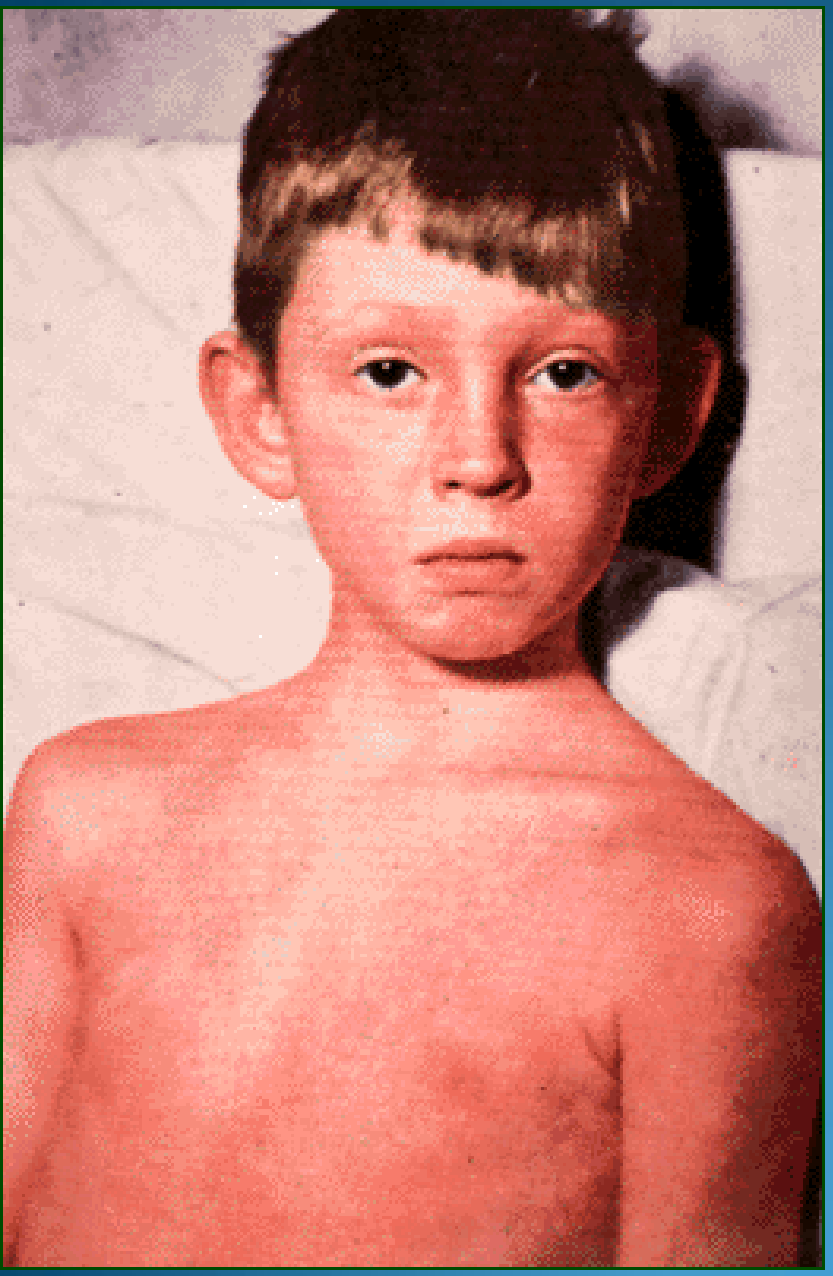
- Diagnosis
  - deep tissue biopsy for Gram stain and culture
- Treatment
  - dicloxacillin or cephalexin for at least 10 days usually requires several weeks of treatment

Ecthyma Contagiosum (Orf)



Ecthyma Gangrenosum (Pseudomonas Sepsis)





Scarlet Fever

Scarlet Fever

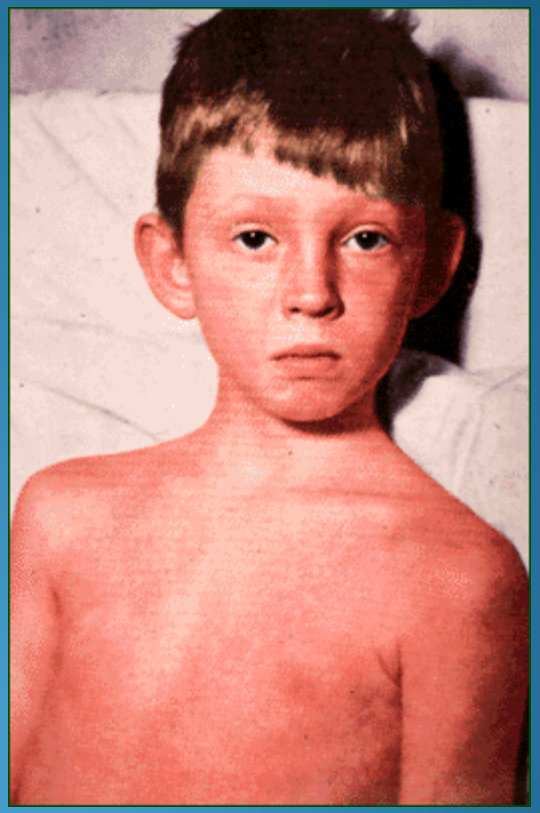
- Group A β-Hemolytic Streptococcus
  - Erythrogenic Toxins A, B and C
  - Strains B and C are a more common cause but A is more virulent
- Majority of cases occur in kids aged 1-10
- Winter and Springtime
- Clinical settings:
  - Following Strep tonsillitis or pharyngitis
  - Following surgery “surgical scarlet fever”
  - Pelvic and puerperal infections
  - Burns

Staph may cause a similar presentation:

- Scarlatiniform erythroderma
- Scarlatiniform rash
- No pharyngitis
- No strawberry tongue
- More tender

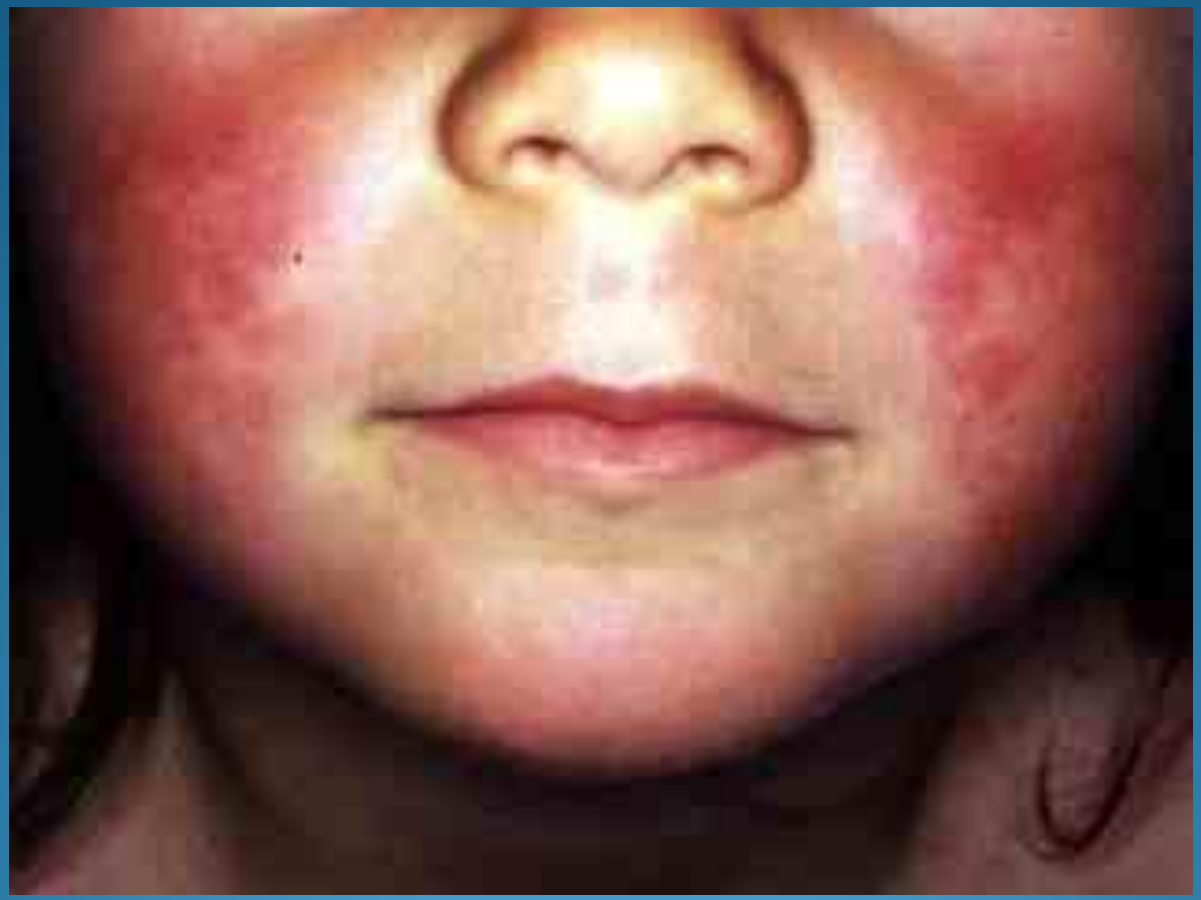
Scarlet Fever

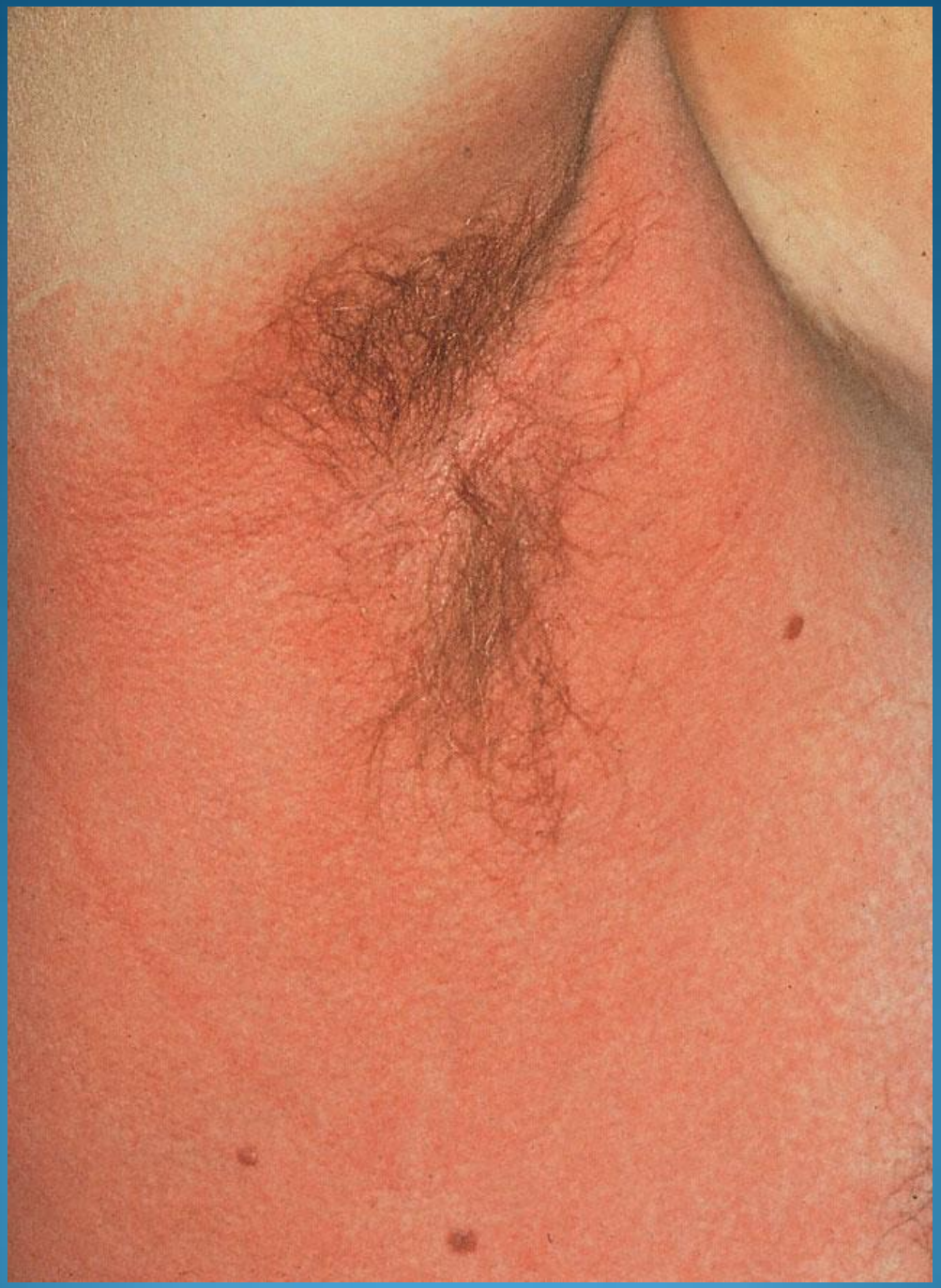
- Eruption is produced by a toxin- mediated delayed-type hypersensitivity reaction
- Antibodies against the toxins are protective
- 80% of the population has protective Ab by age 10

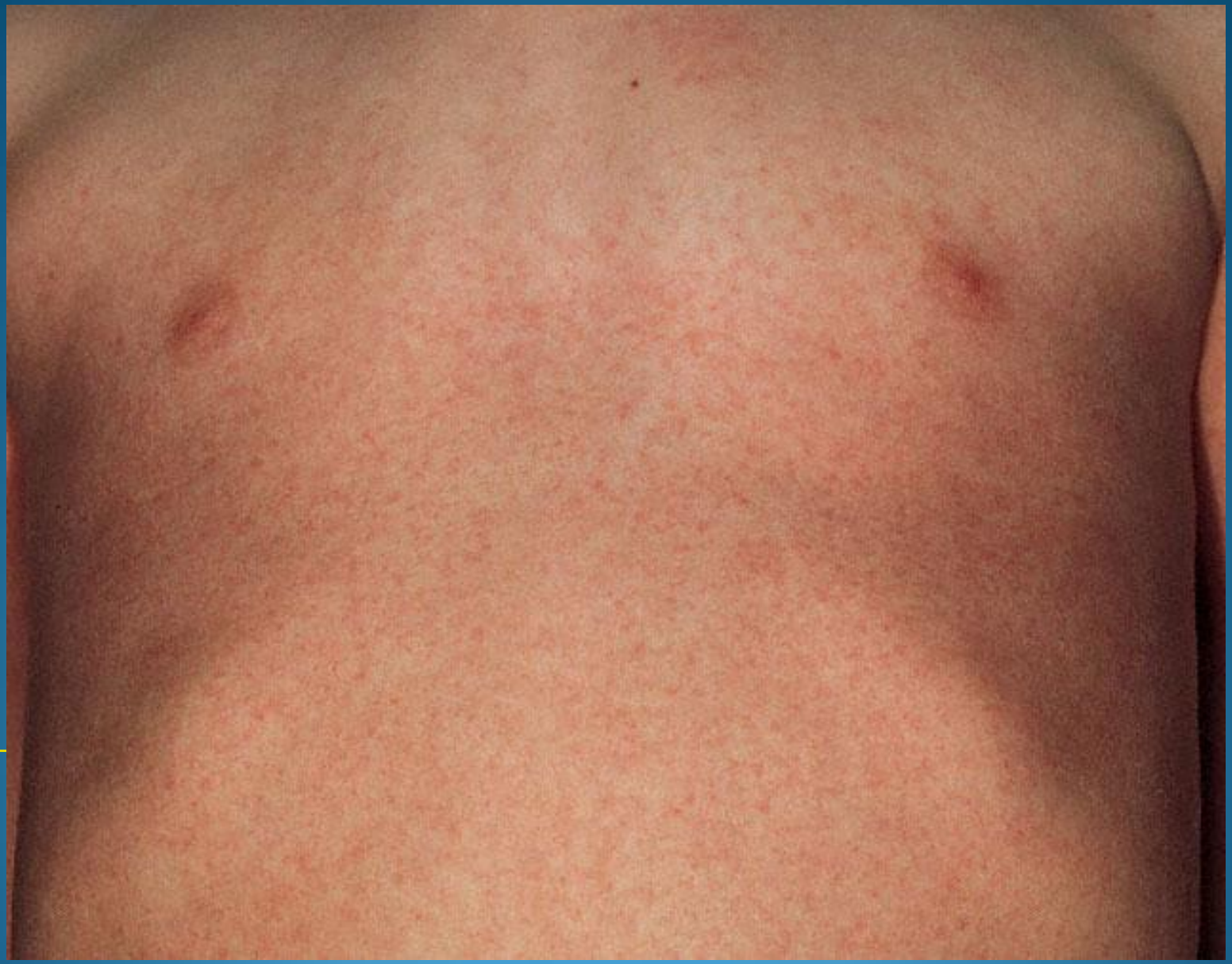


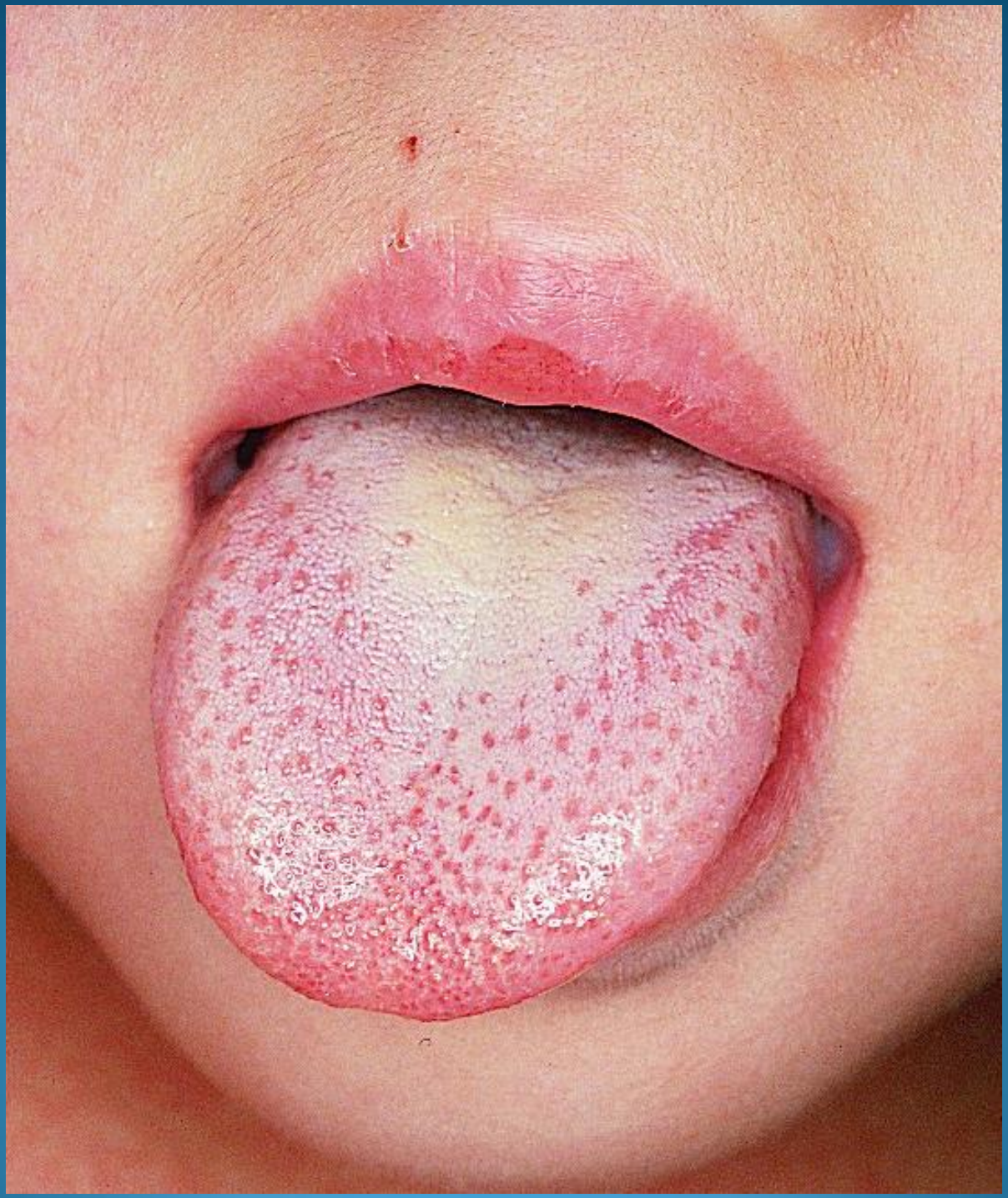
Scarlet Fever: Clinical Features

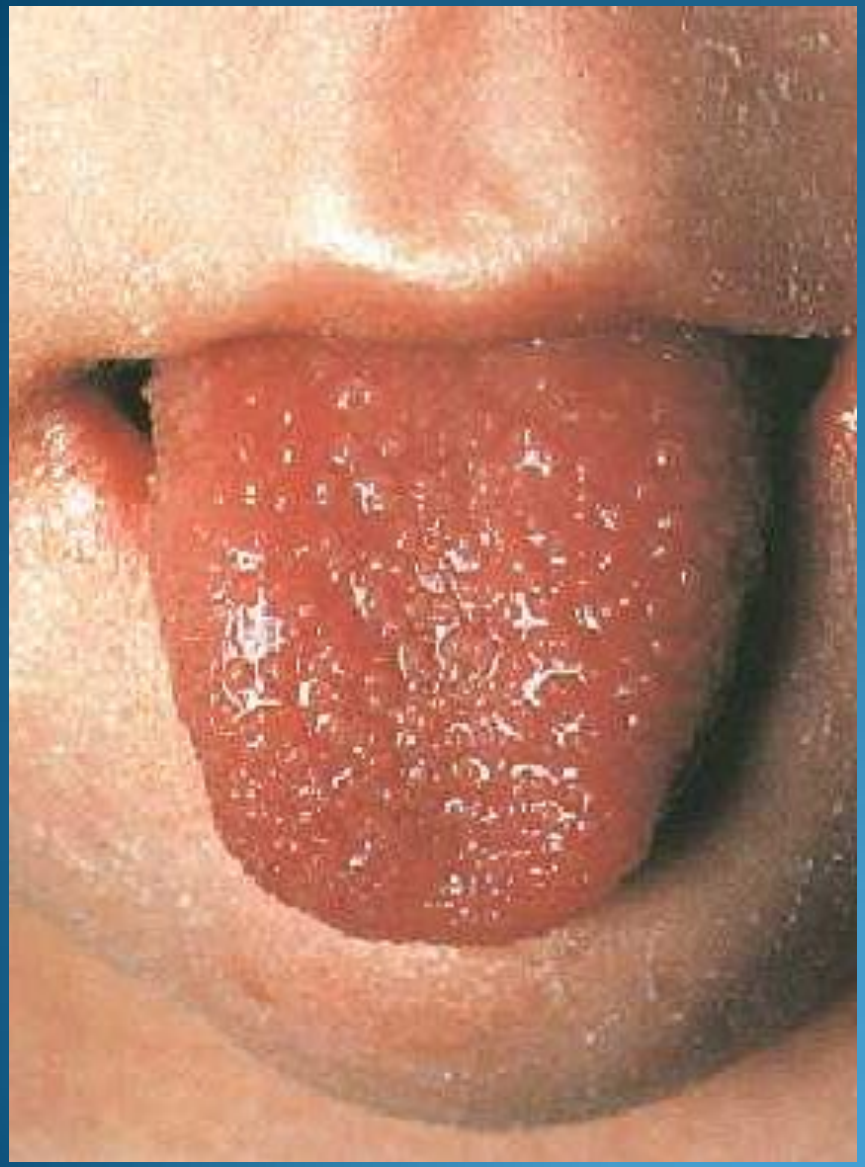
- Abrupt onset sore throat, F/C/N/V/HA, malaise
- Rash begins 24-48 hours later
- Proximal erythema of neck, chest and axillae
  - generalizes within 6 hours
  - Spreads from neck down
  - Spares the palms and soles
- Red, sandpaper-like rash blanches with pressure
- Flushed cheeks with circumoral pallor
- Pastia's Lines: linear petechiae in flexures
- White, then red strawberry tongue
- Desquamation in 7-10 days, lasts 4 weeks

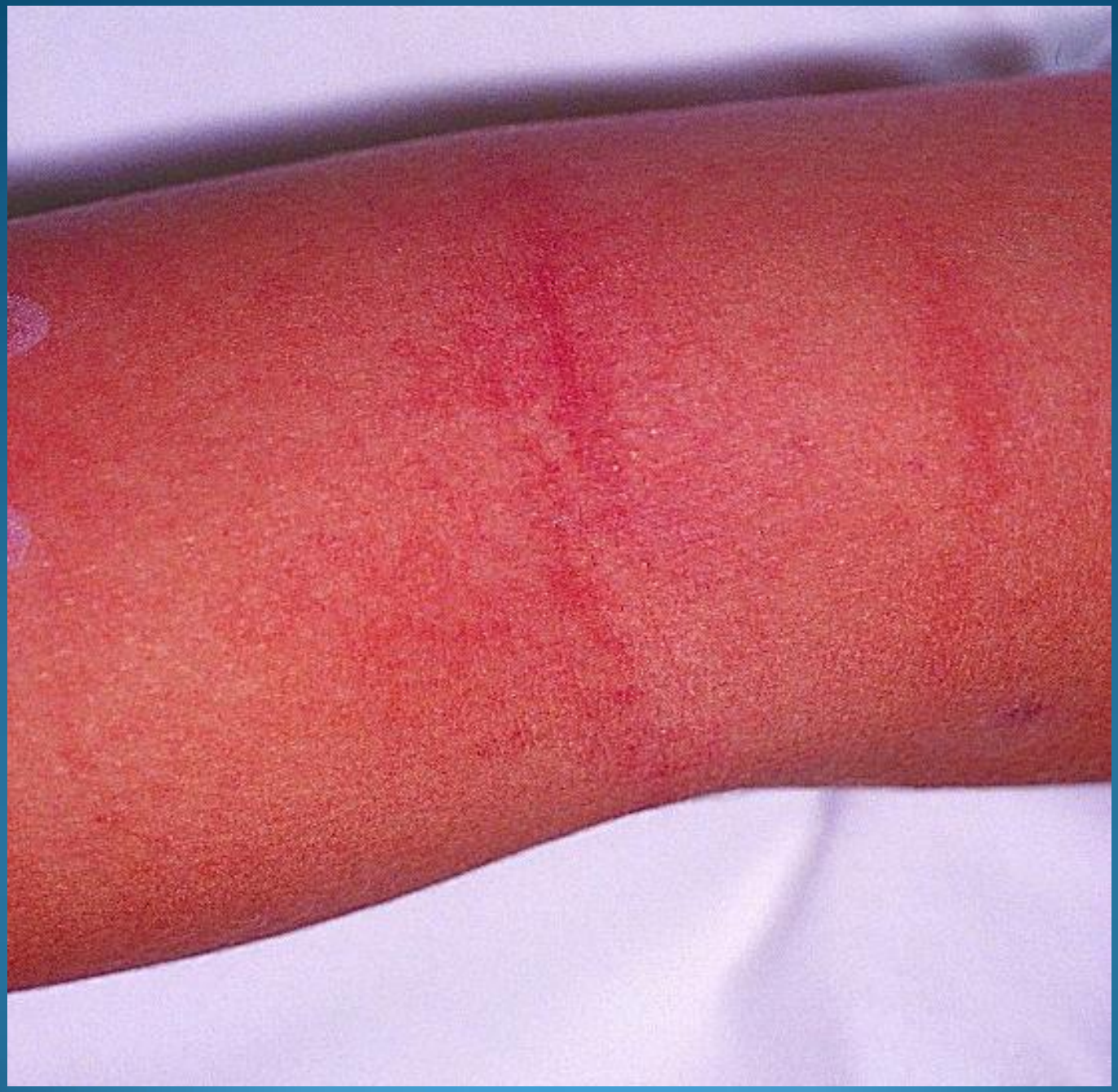


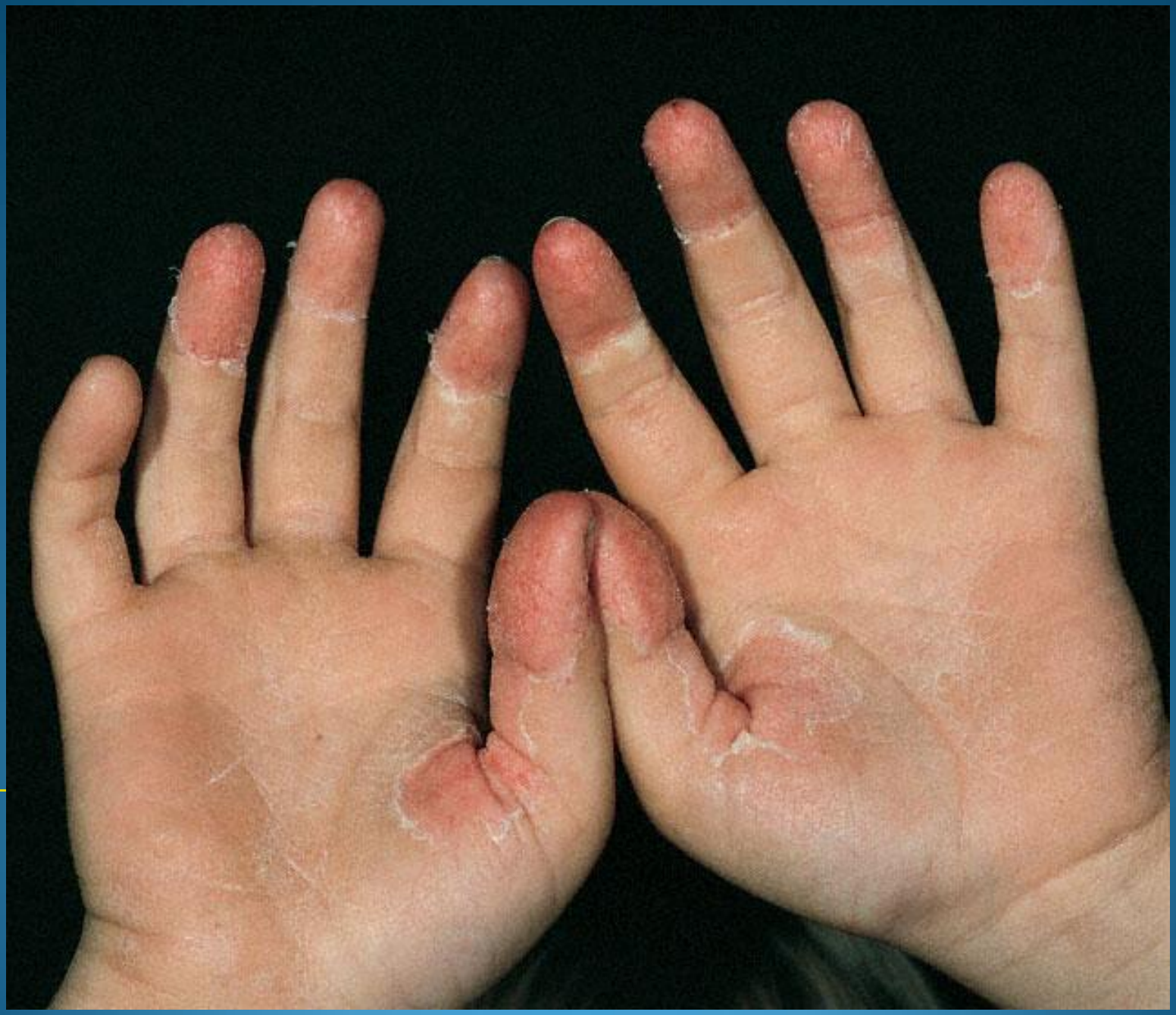


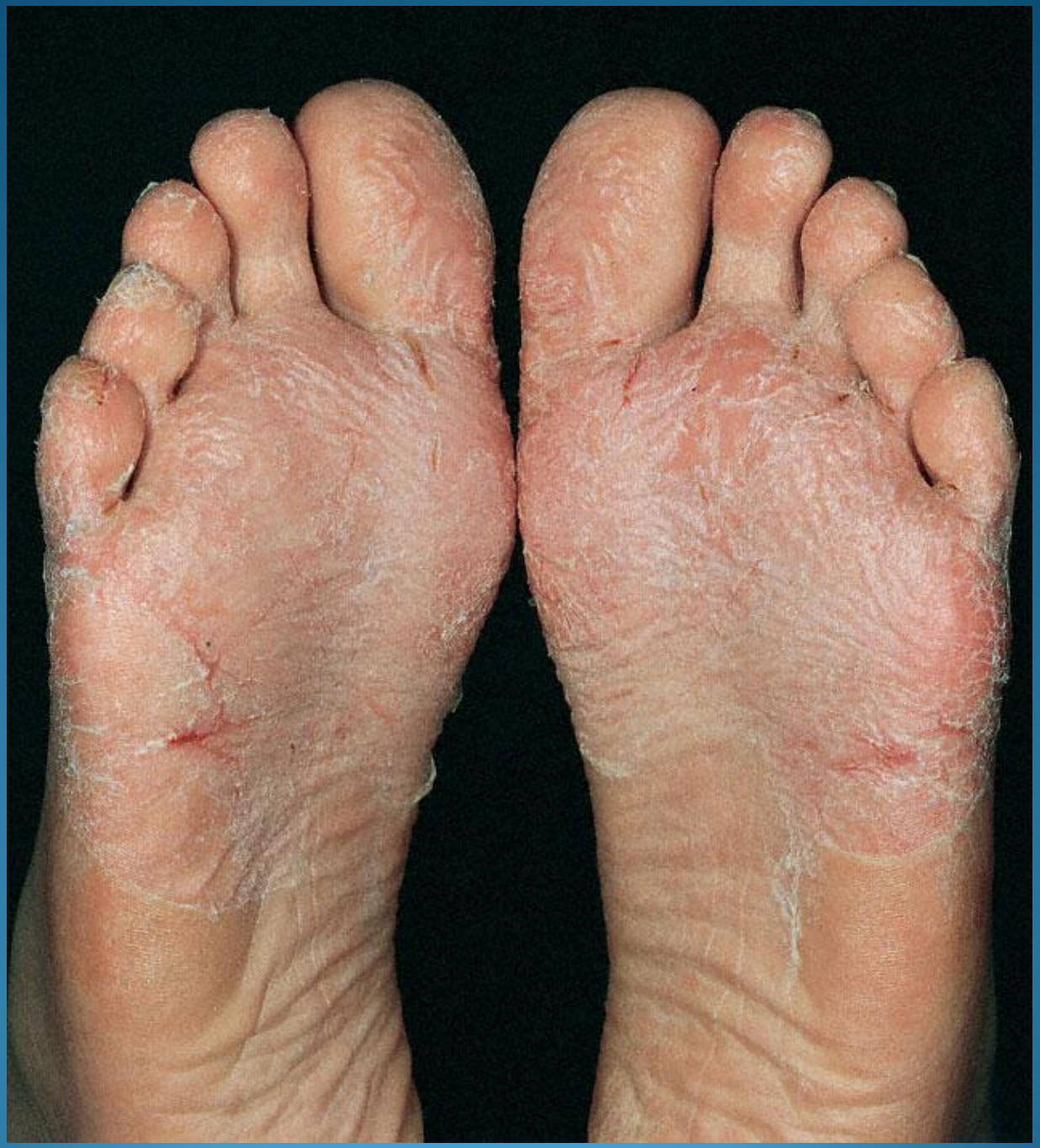












Scarlet Fever

Complications

- Otitis
- Mastoiditis/Sinusitis
- Pneumonia
- Myocarditis
- Meningitis
- Arthritis
- Hepatitis
- Glomerulonephritis
- Rheumatic fever

Diagnosis/Treatment

- Clinical
- Throat culture
- Anti-Streptolysin O Ab
- Anti-DNase B Ab

- Penicillin x 10-14 days
- Erythromycin if pen allergy
- **Treatment prevents rheumatic fever**

Staph Scalded Skin Syndrome

Ritter's Disease

Dermatitis Exfoliativa Neonaturum

Staph Scalded Skin Syndrome

- Staphylococcal toxin-mediated infection
  - Staph Scalded Skin Syndrome
  - Toxic Shock Syndrome
  - Bullous Impetigo
- Staph aureus phage group II strains
- Exfoliative exotoxins ET-A and ET-B
  - Serine protease which attack cell adhesion molecule-Cadherins (Desmoglein-1)
  - results in superficial skin cleavage as seen in Pemphigus Foliaceous

Staph Scalded Skin Syndrome

High Risk Populations

- Neonates (nursery outbreaks)
- Children less than 6 years old
- Adults with chronic renal insufficiency
- Immunocompromise

Why?

- Toxins are renally excreted
- Neonates, kids, and CRI pts cannot clear the toxins

Staph Scalded Skin Syndrome

Prodrome

- fever, malaise, irritability, skin tenderness
- purulent rhinorrhea or conjunctivitis

Staph Scalded Skin Syndrome

- Initial:

  localized tender erythema with periorificial and fexural accentuation
- Hours:

  erythema generalizes with an orange red color, skin wrinkles flaccid bullae
- 1-2 days:

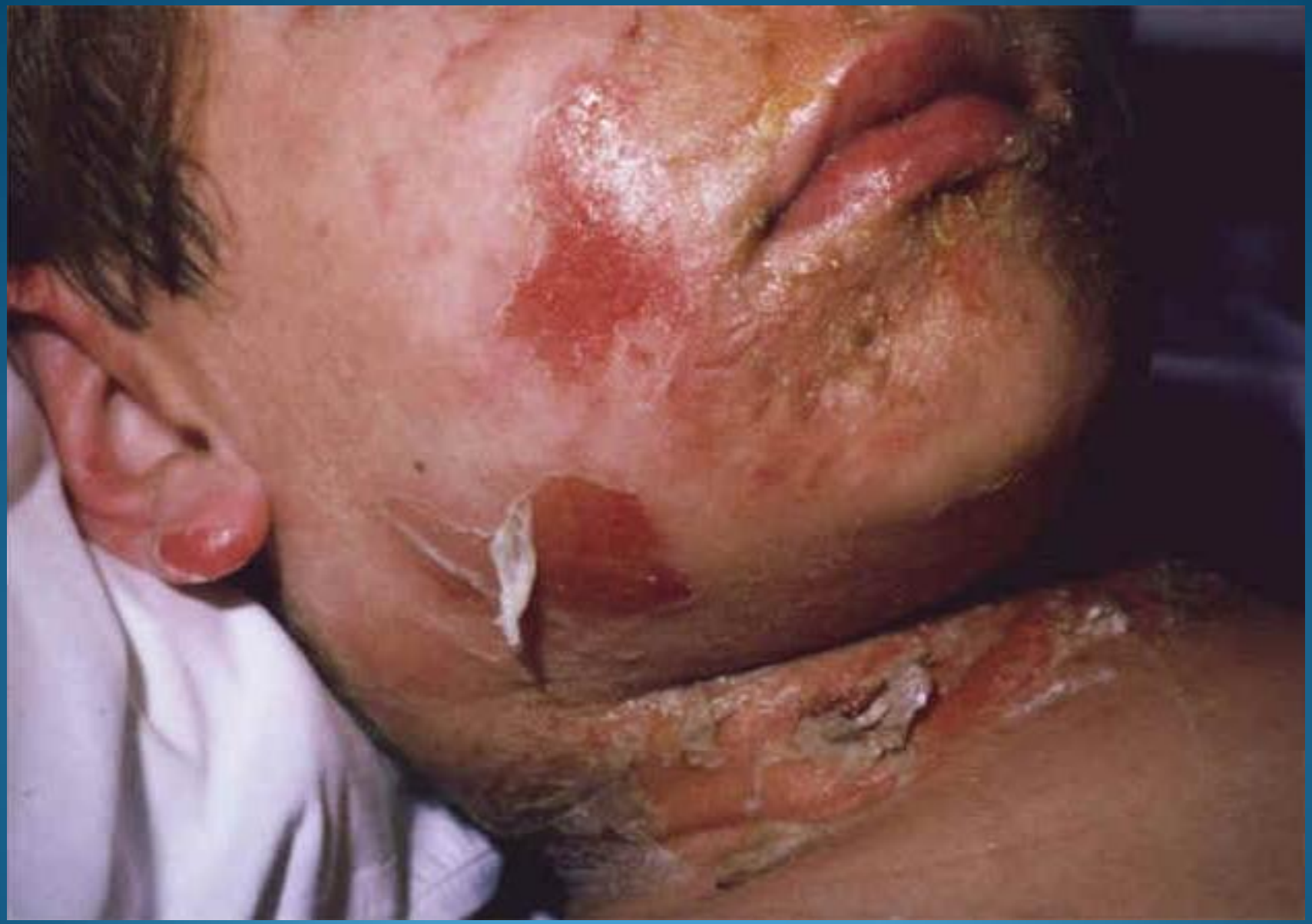
  slough results in moist, thin, varnish-like crust
- 3-5 days:

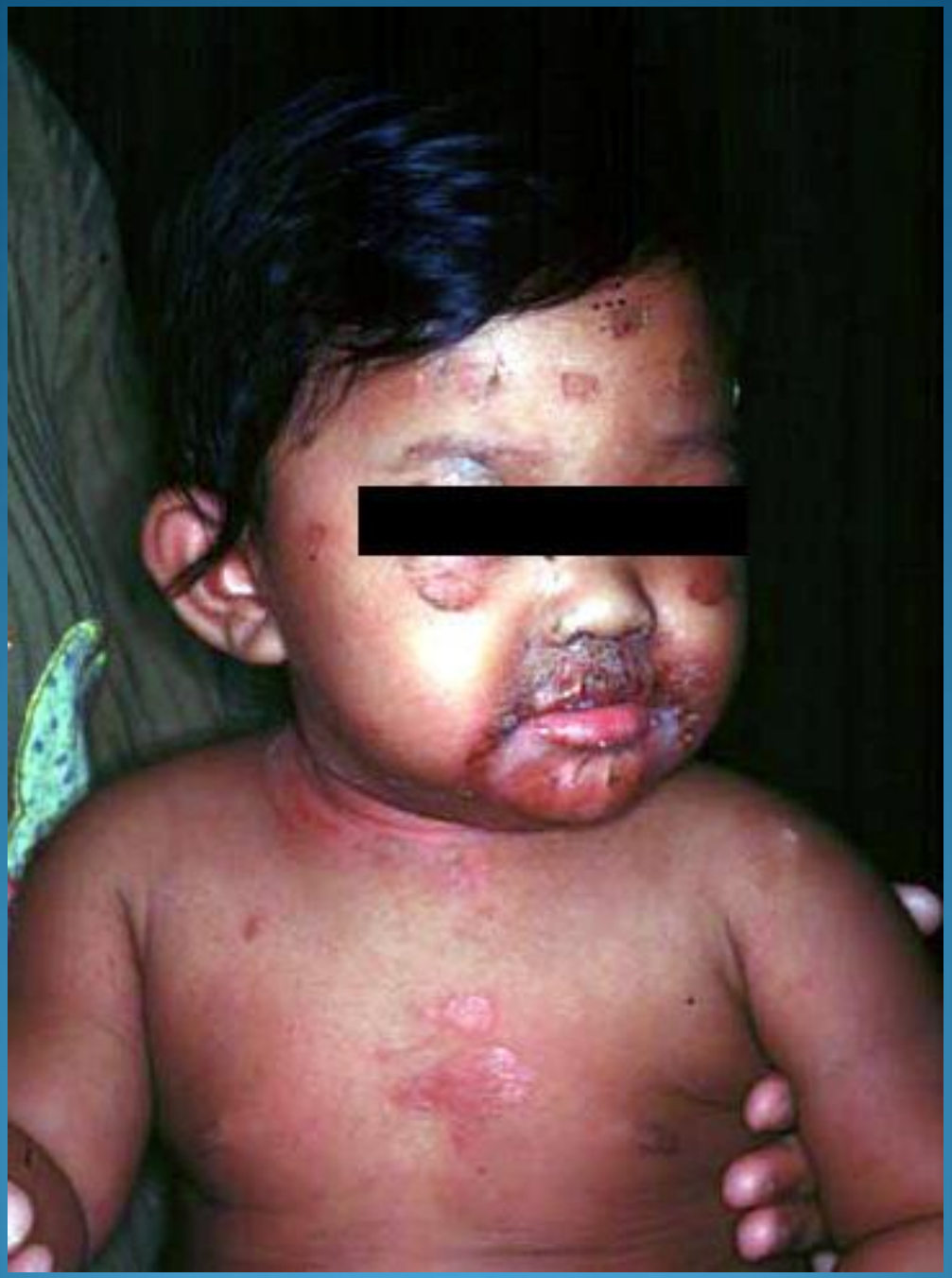
  scaling and desquamation
- 10-14 days:

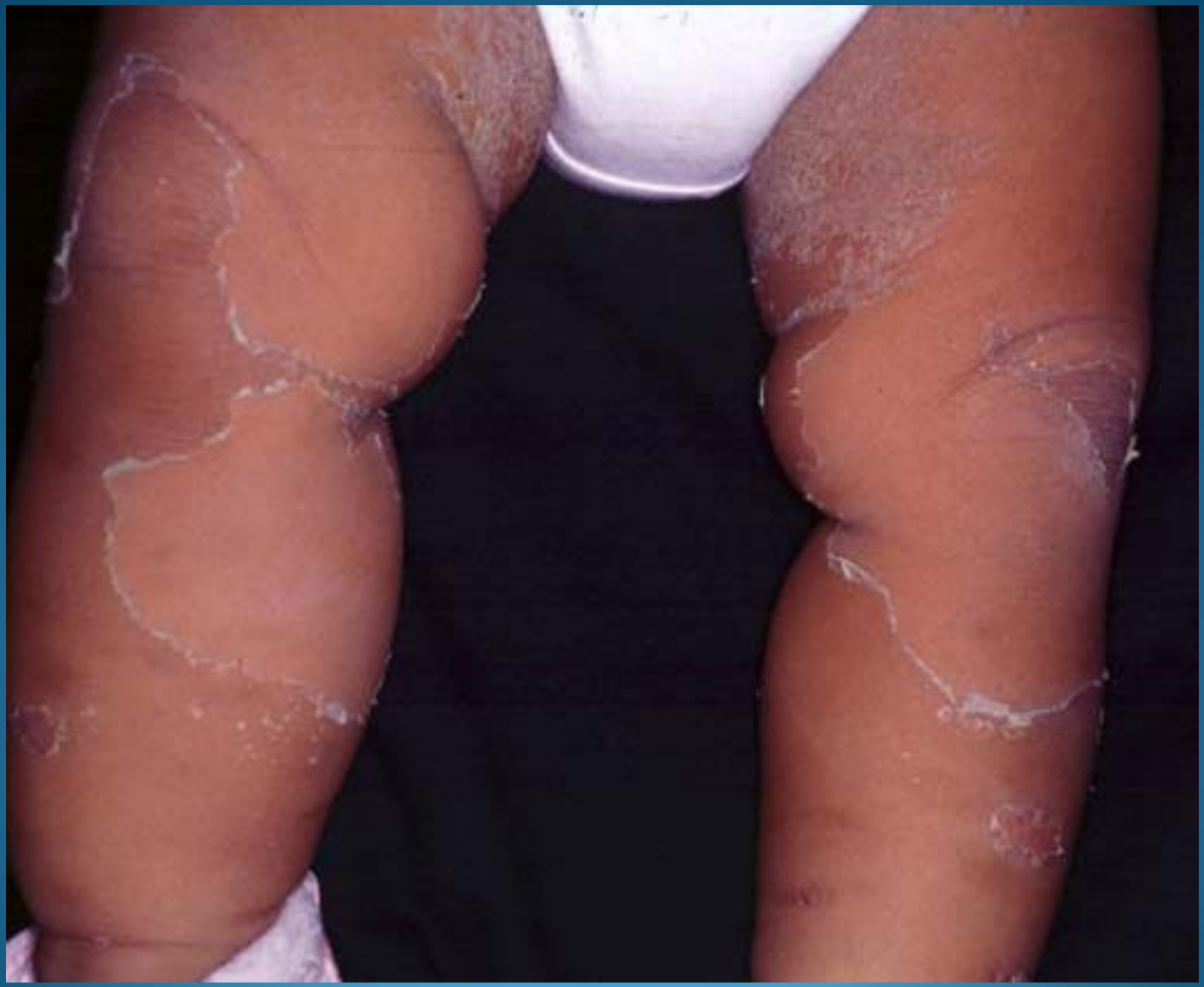
  re-epithelialization without scarring

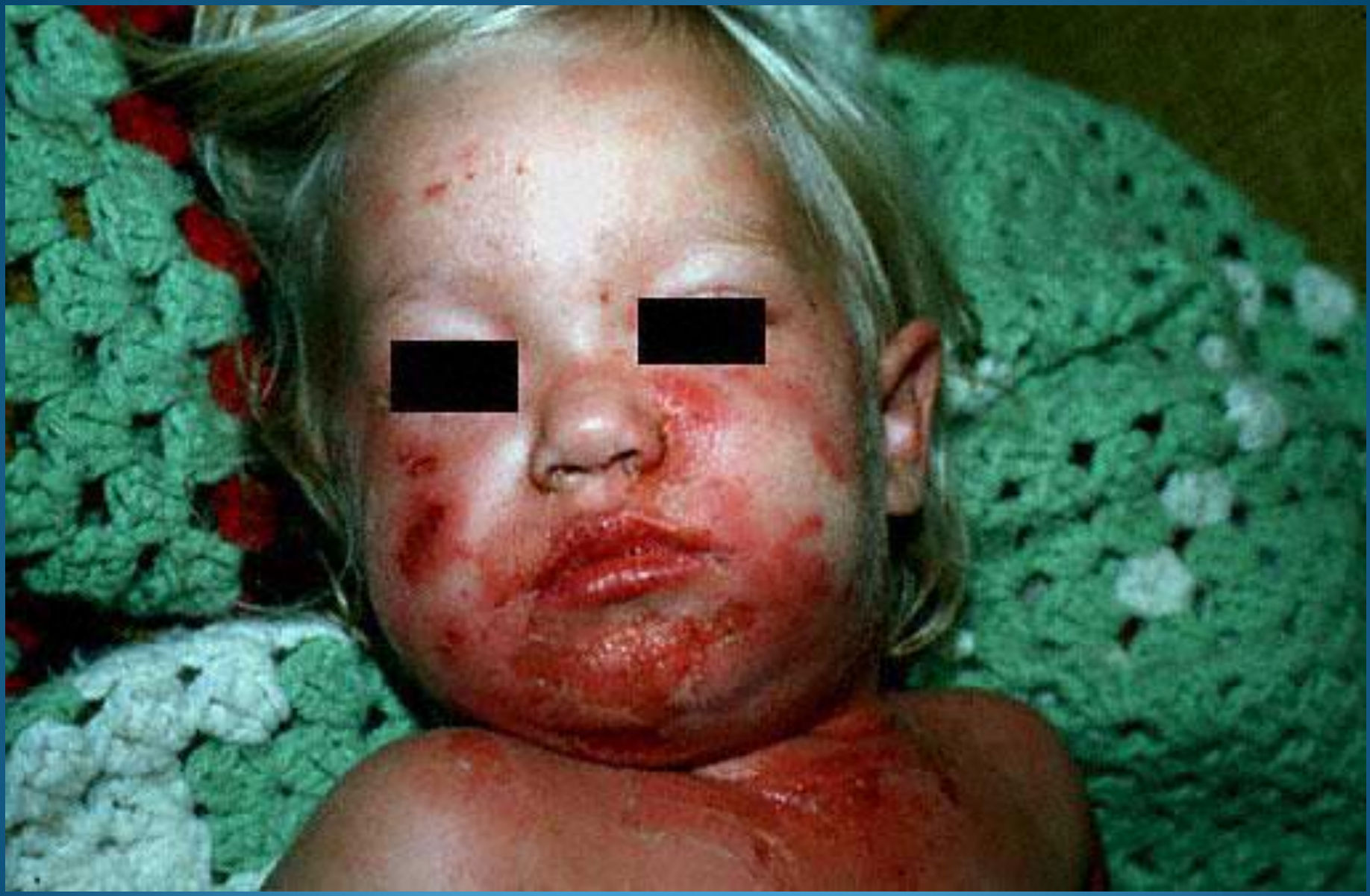
Differentiating from TEN:

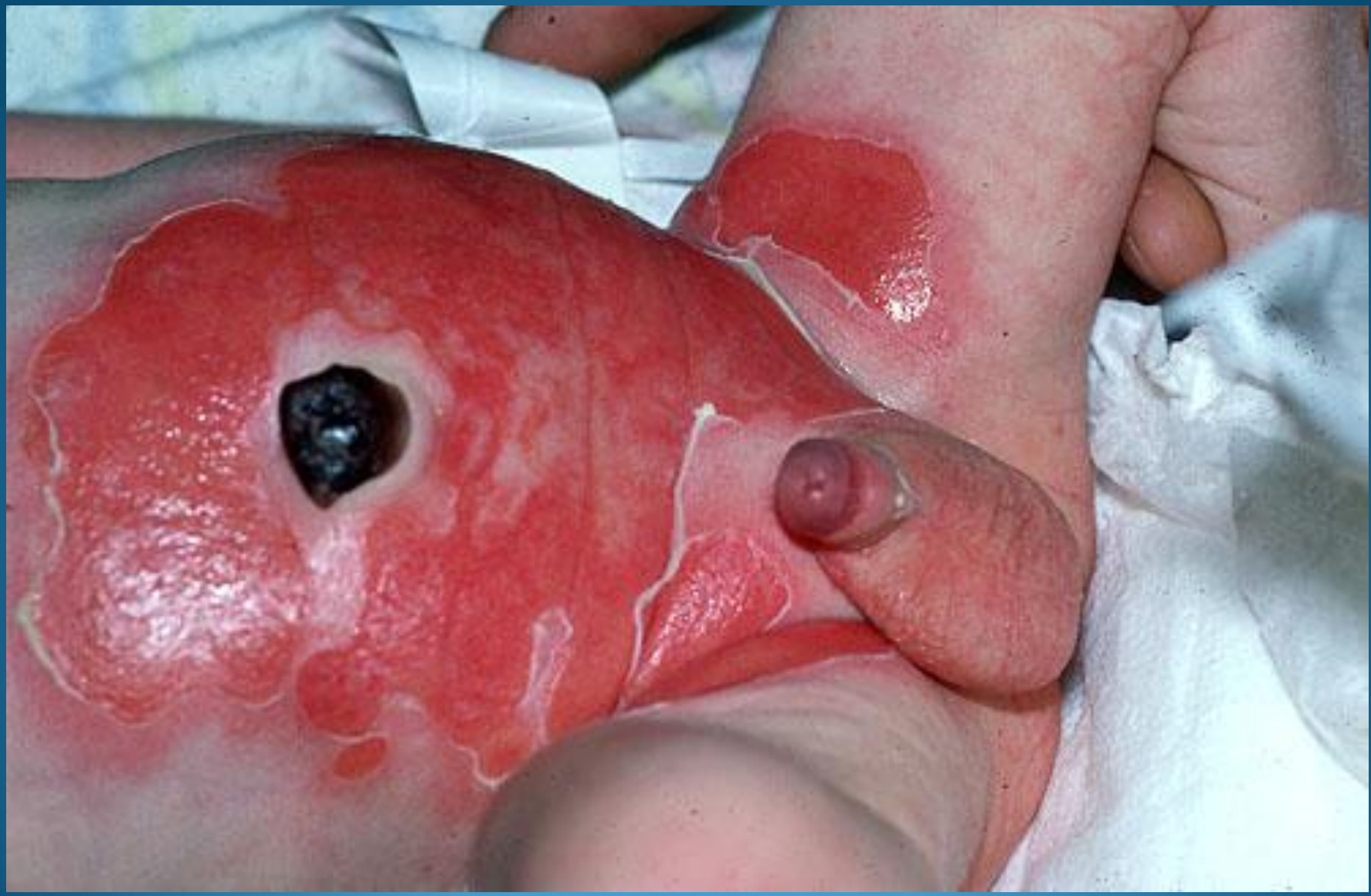
- Patients should not appear toxic
- There should be no mucosal erosions
- No palms or sole involvement
- Biopsy

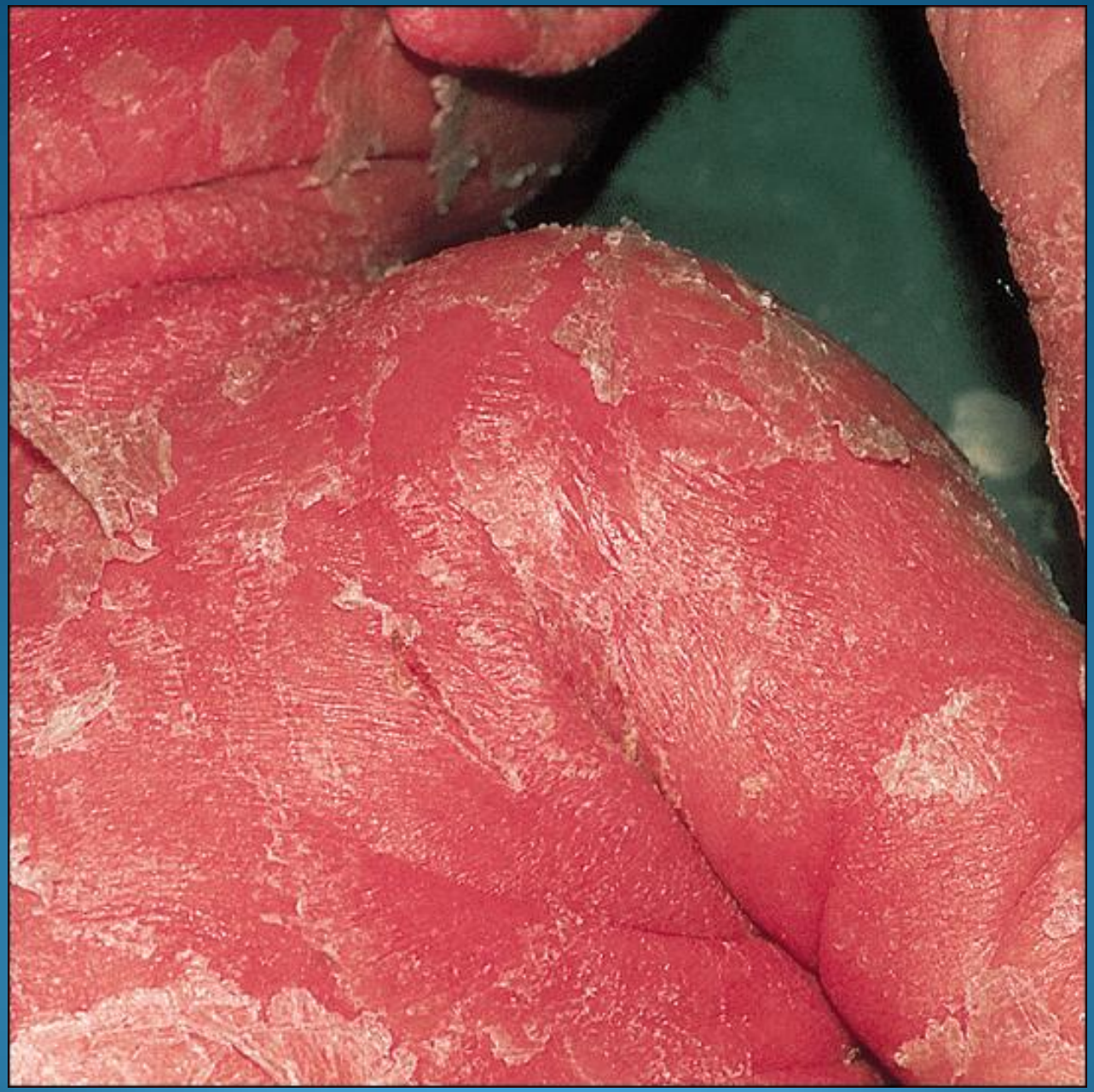












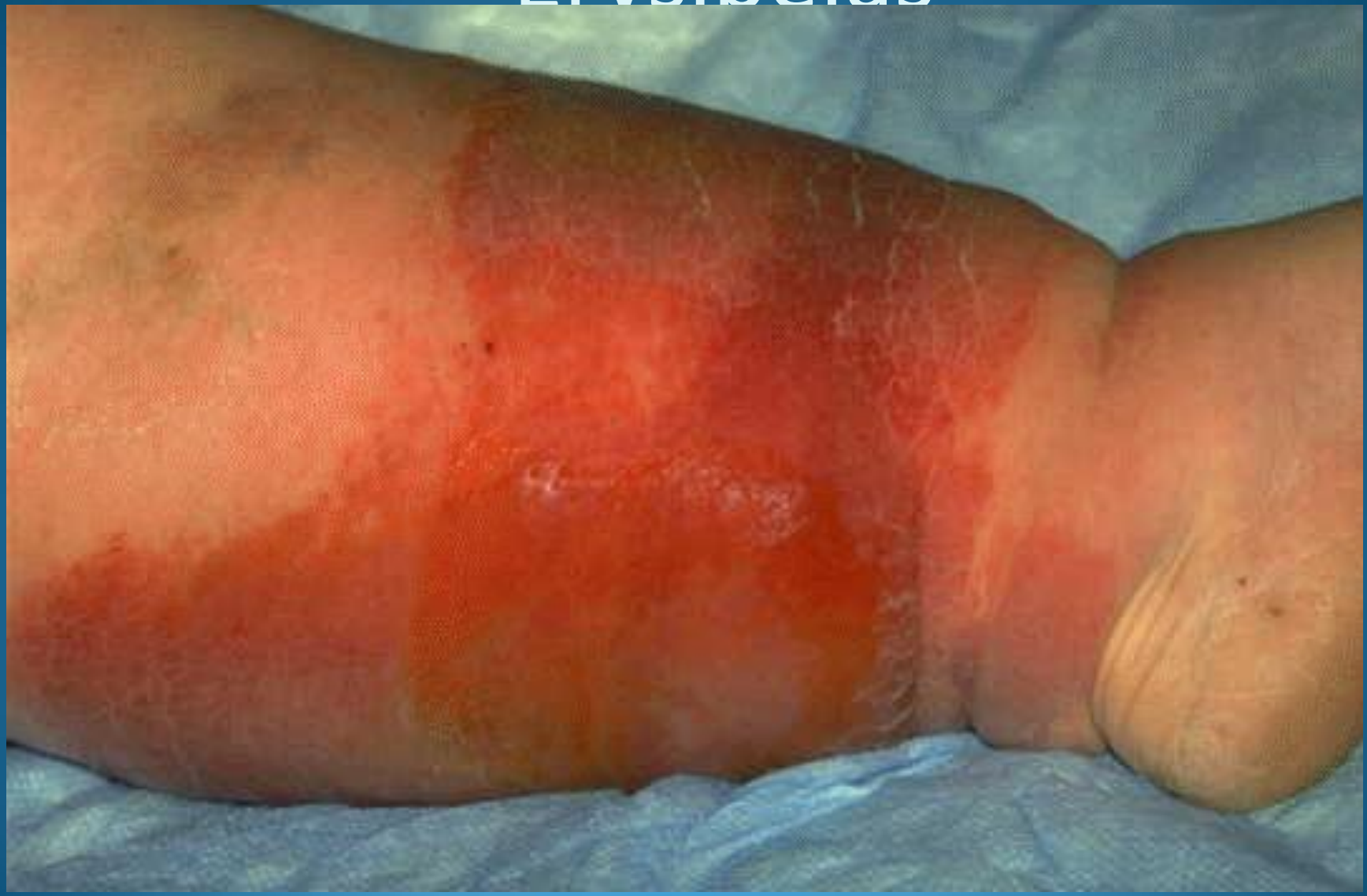
Staph Scalded Skin Syndrome: Diagnosis

- Clinical
- Culture:
  - intact bullae are always negative
  - conjunctivae, nasopharynx, feces or pyogenic foci on the skin may be positive
  - blood is negative in kids, may be + in adults
- Biopsy: superficial cleavage in granular layer, no organisms
- Toxin ID via ELISA, immunodiffusion

Staph Scalded Skin Syndrome: Treatment

- Severe
  - Hospitalization for parenteral antibiotics
  - Local wound care w/ bland emollients
- Localized
  - Oral β-lactamase-resistant antibiotic
  - Dicloxacillin, cephalexin x 7-14d
  - Local wound care
- Isolate affected newborns from other neonates
- Identify and treat asymptomatic carriers

Erysipelas



Erysipelas

(*aka St. Anthony's Fire, Ignis Sacer)*

- Group A β-Hemolytic Strep, occ Group C and G
- Group B Strep in Newborns and Postpartum
- Involves superficial dermis and lymphatics

At risk populations

- Children, elderly
- Debilitated, diabetic, alcoholic
- Immunocompromised
- Lymphedematous extremities
- Chronic ulcers

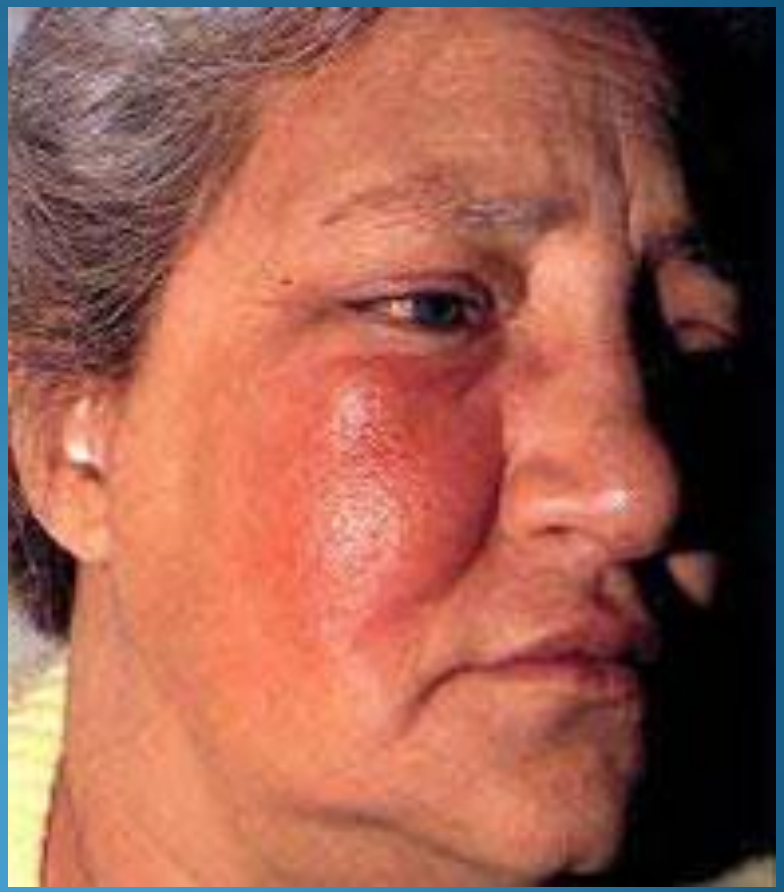
Erysipelas

A defect in the skin barrier allows bacteria to enter

- trauma, abrasions, skin ulcers
- insect bites, eczema, psoriasis

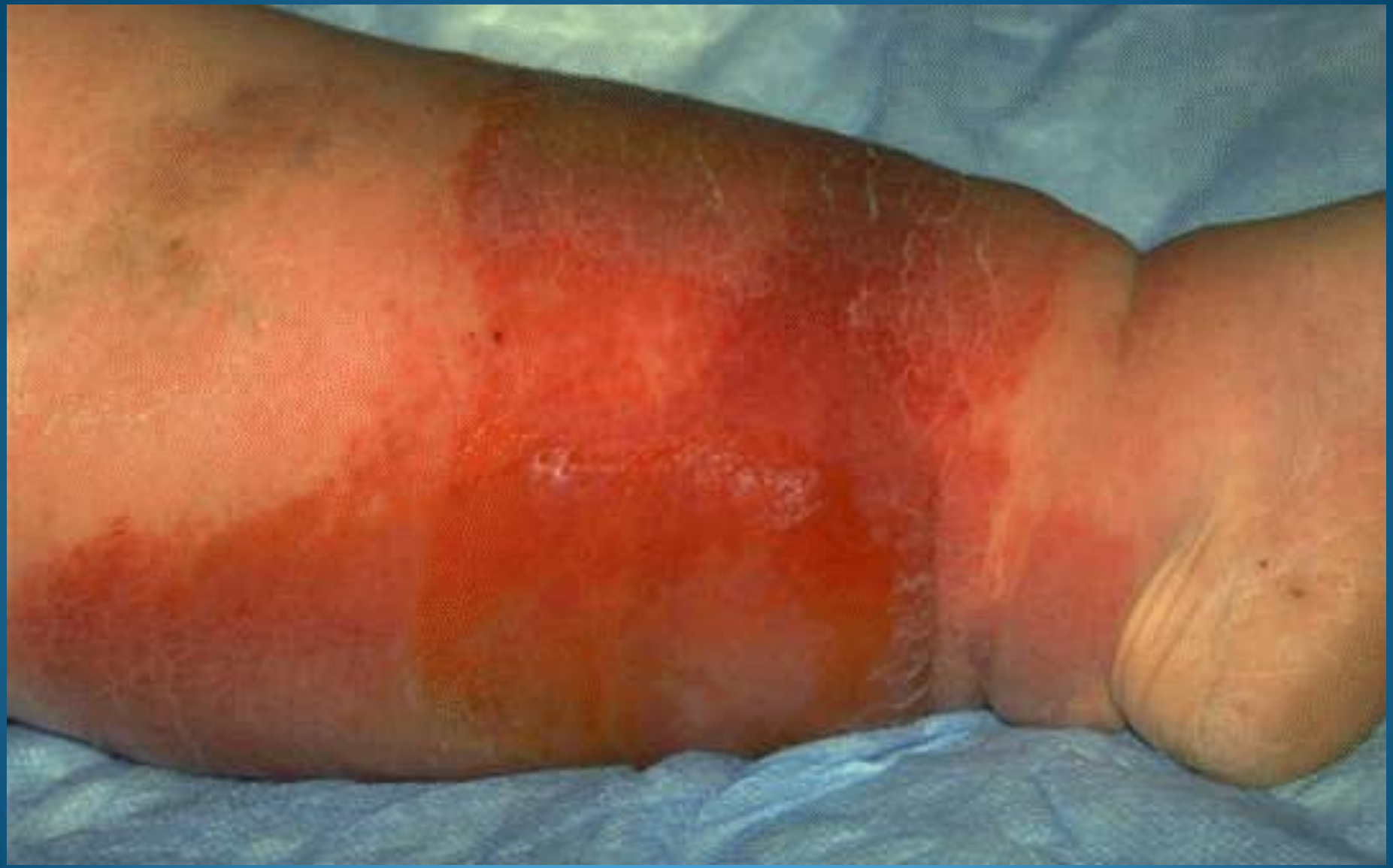
Other predisposing factors

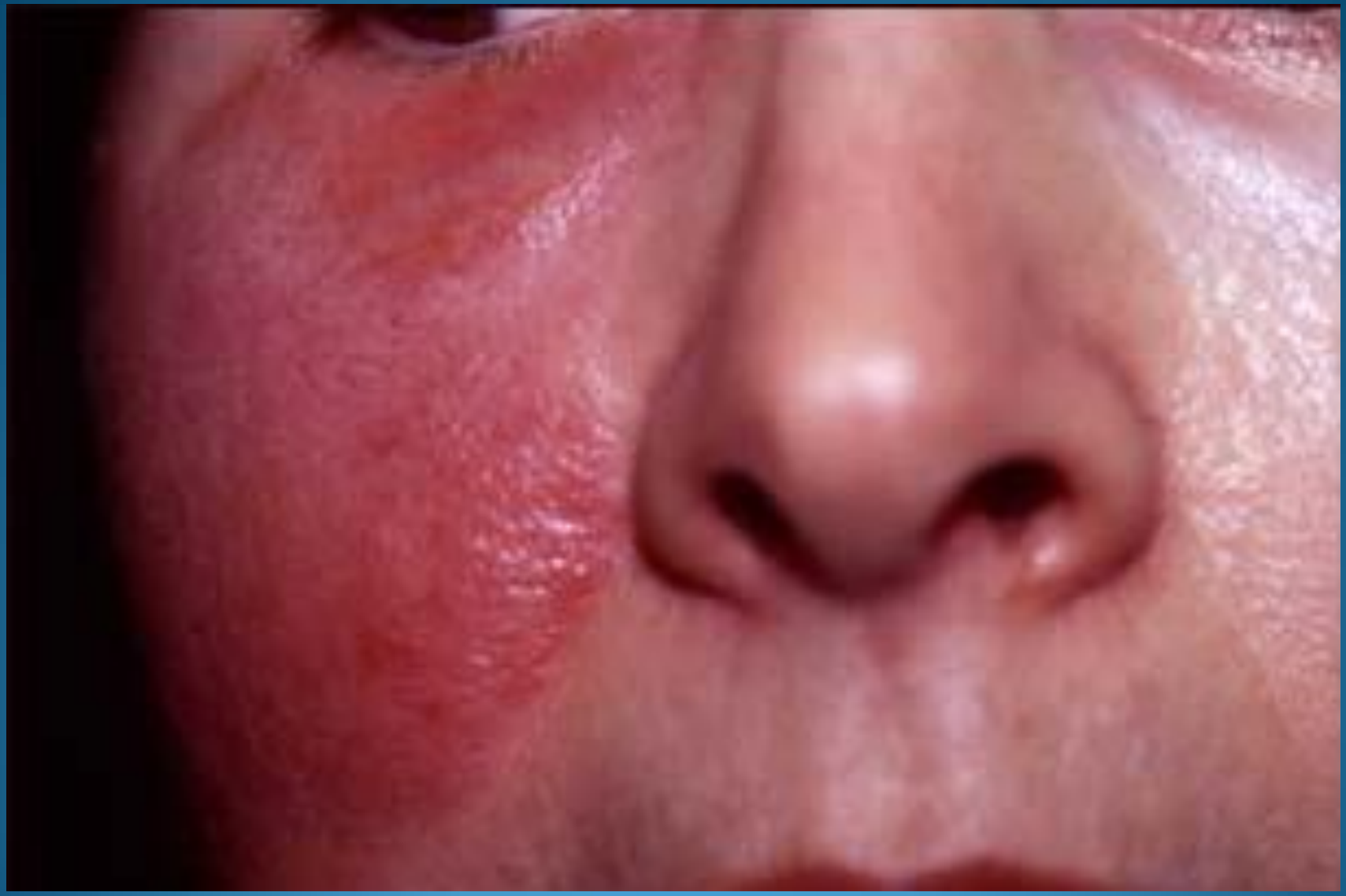
- Lymphatic obstruction or edema
- Saphenous vein grafting
- Post- radical mastectomy
- Arteriovenous insufficiency
- Paretic limbs

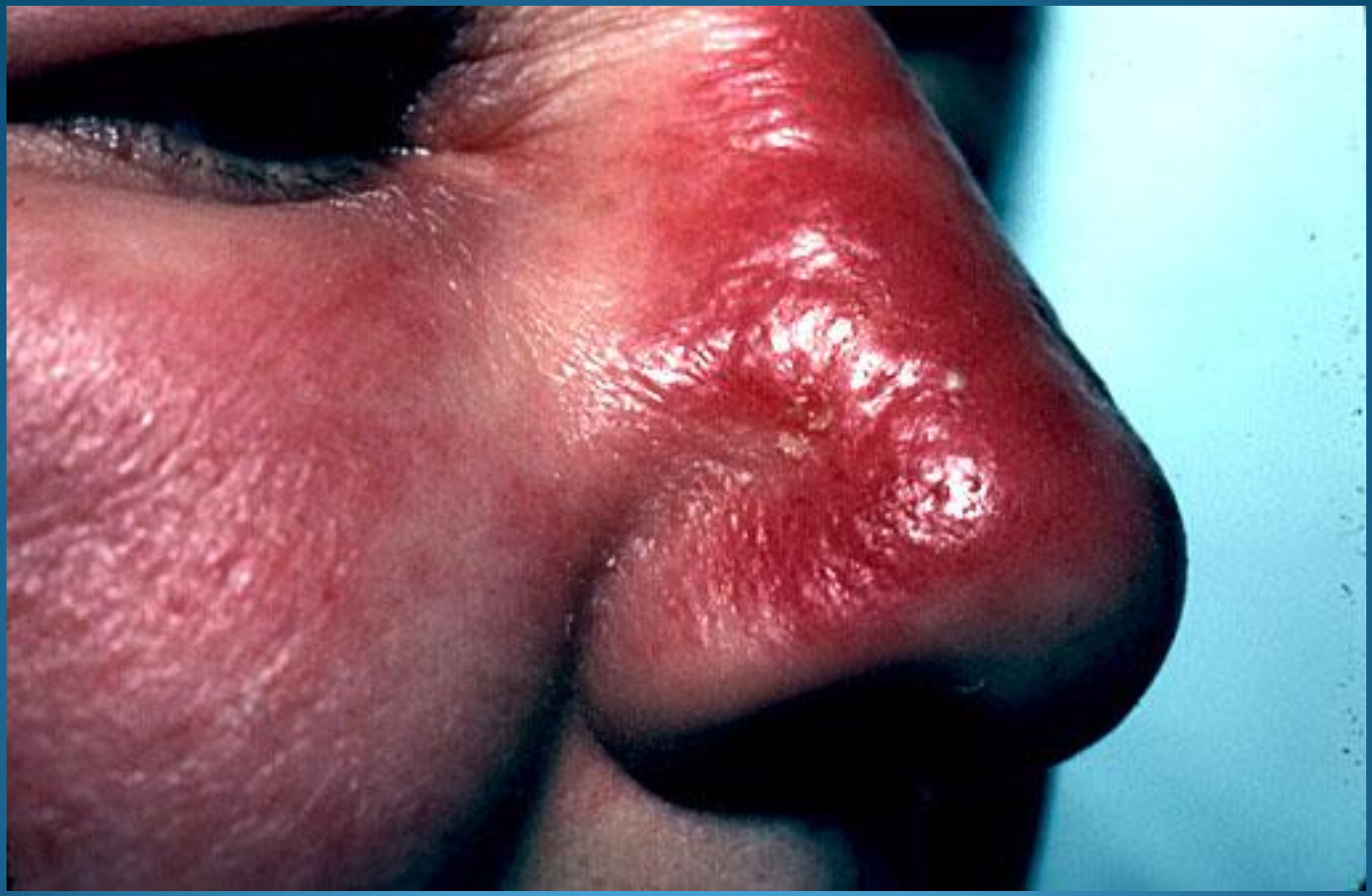


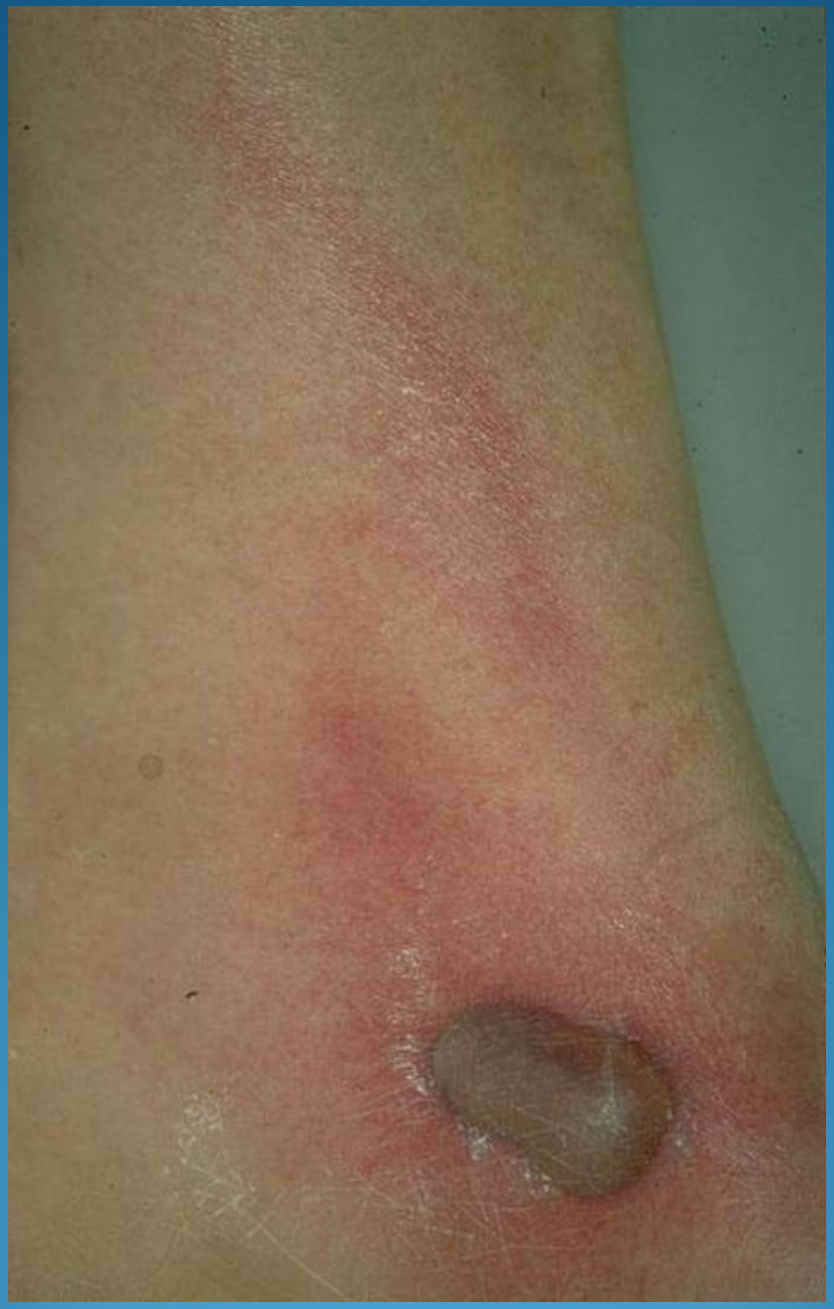
Erysipelas

- Lower extremity is most common location
- Abrupt onset of fever, chills, nausea, malaise
- Well demarcated, indurated elevated border, painful plaque
- Regional lymphadenopathy +/- streaking
- May develop vesicles, bullae, necrosis
- Resolves w/ desquamation &↑pigmentation
- Underlying disease may obscure borders









Erysipelas: Diagnosis

- **Clinical**
- Elevated WBC with a left shift
- ASLO and anti-DNase B: reasonable indicators
- Cultures
  - Blood: + in 5%
  - Skin biopsy: rarely +
  - Portal of entry, pustules or bullae: +/-
  - Throat and nose: +/-

Erysipelas: Treatment

- Penicillin x 10 days
  - Macrolide if penicillin- allergic
- Children and debilitated patients
  - May need admission for IV antibiotics
- Recurrent cases may warrant prophylaxis

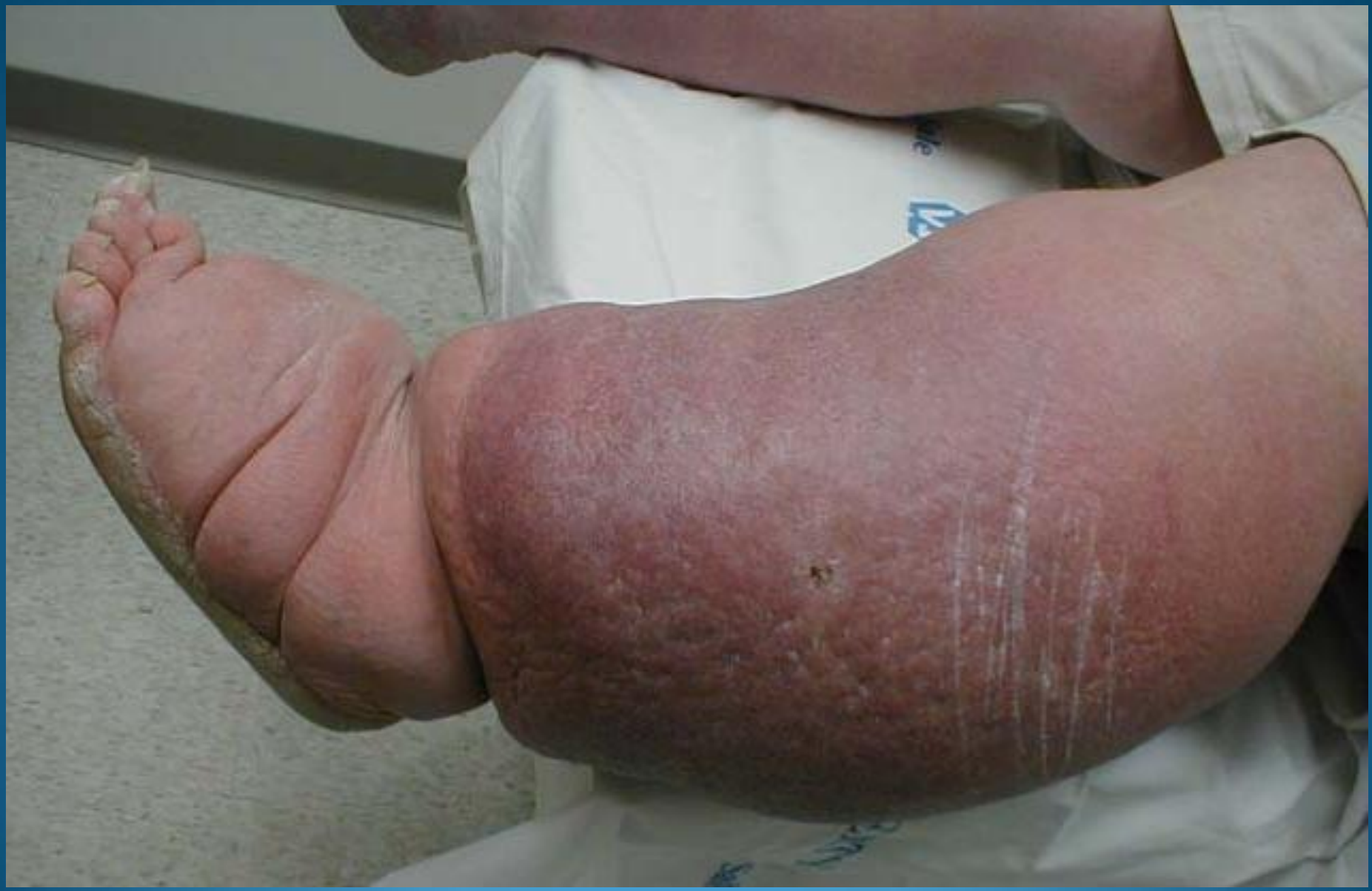
Erysipelas

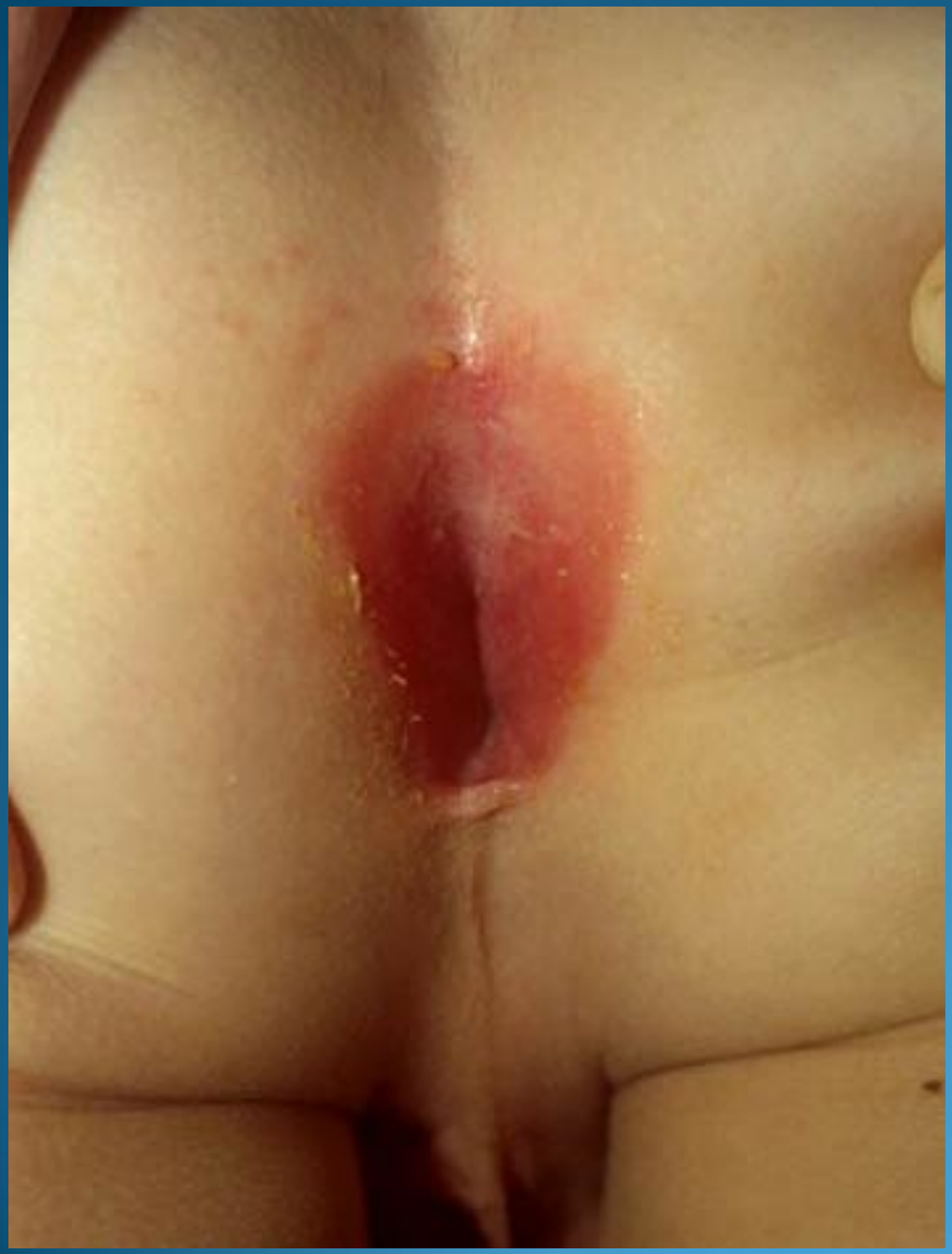
Complications

- Gangrene / amputation
- Bacteremia / sepsis
- Scarlet fever
- Pneumonia
- Abscess
- Embolism
- Meningitis
- Death

Prognosis:

- Excellent in immunocompetent patients with proper treatment
- Chronic edema
- Scarring
- Elephantiasis in chronic, recurrent cases



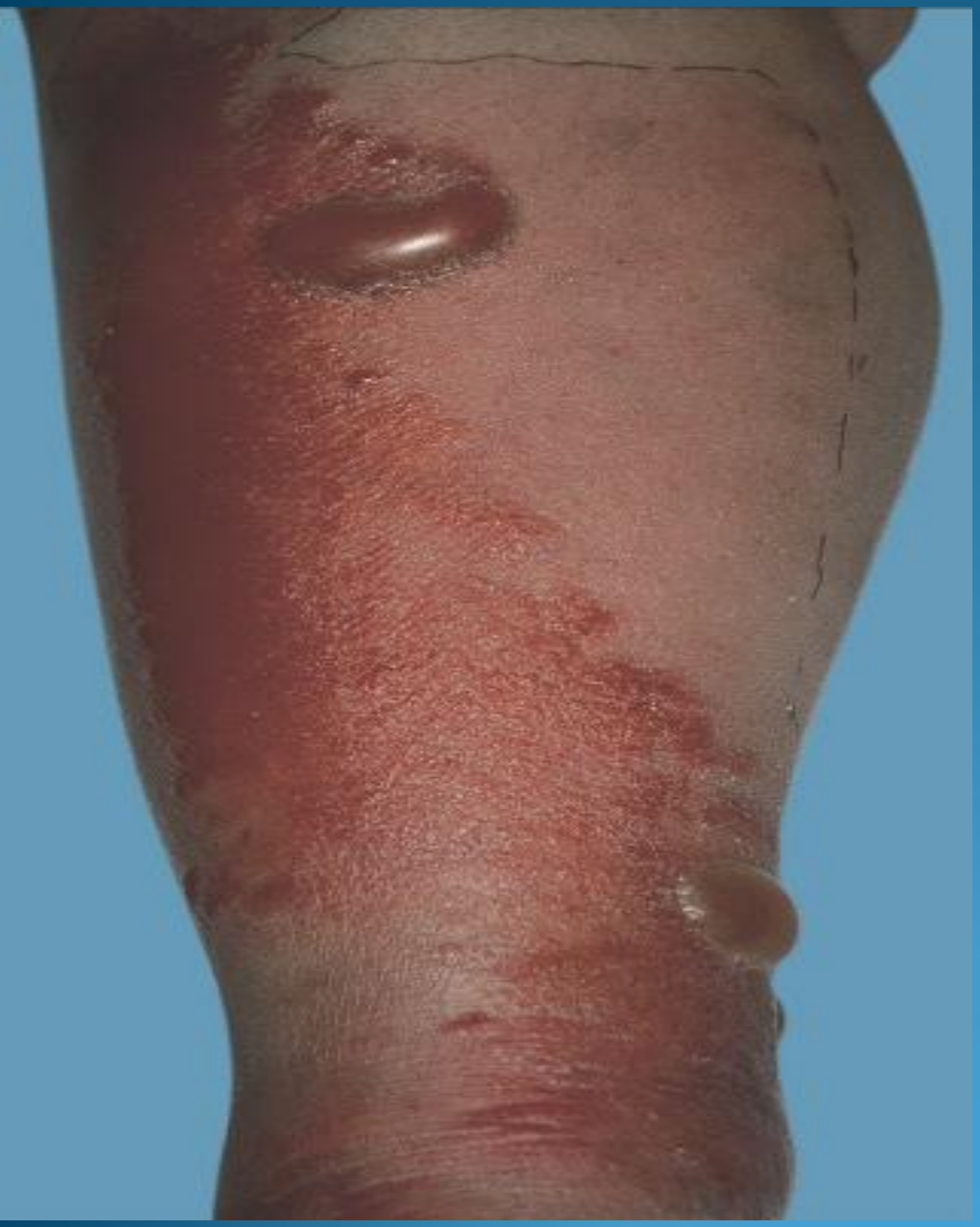


Perianal Cellulitis

S. pyogenes

Perianal Cellulitis

- Kids less than 4 years old
- Pruritus, painful defecation, bloody stools
- No systemic symptoms
- May follow/accompany pharyngitis
- May initiate guttate psoriasis
- Dx: skin culture (anus and throat)
- Rx: penicillin or erythromycin x 14-21 days



Cellulitis

Cellulitis

- Involves the deep dermis and subcut. tissues
- Adults: *S. pyogenes and S. aureus*
- Children: *S. aureus and H. flu*
- Immunocompromised
  - gram-negative bacilli
    - *Pseudomonas, Proteus, Serratia, Enterobacter*
  - other opportunistic pathogens
    - *Fungi (Cryptococcus)*
  - anaerobes

Cellulitis

Bacterial portal of entry

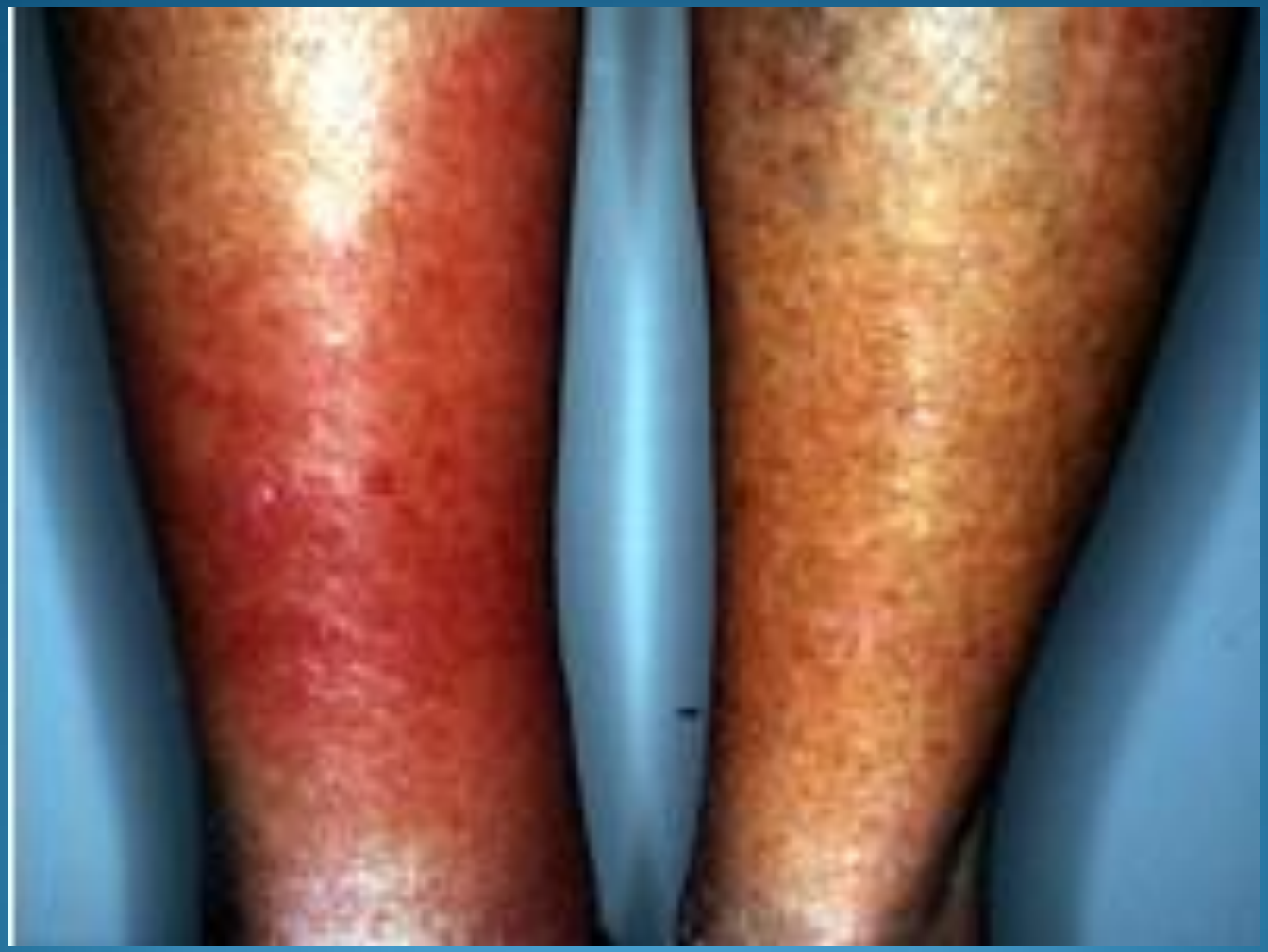
- Immunocompetent
  - External (disrupted skin barrier)
- Immunocompromised
  - Hematogenous (*S. pneumoniae*)

Predisposing factors

- Alcoholism, diabetes, malignancy, PVD, IVDA
- Tinea pedis
- Lymphatic damage
  - node dissection, vein harvest, prior cellulitis

History of preceding event:

- Surgical Wounds- Staph aureus
- Pressure Ulcers- Staph aureus, Pseudomonas aeruginosa, B. fragilis, Enterobacter
- Bites- *Cat*- P. multocida, *Dog*- P. multocida, Capnocytophaga canimoris *Human*- Eikenella

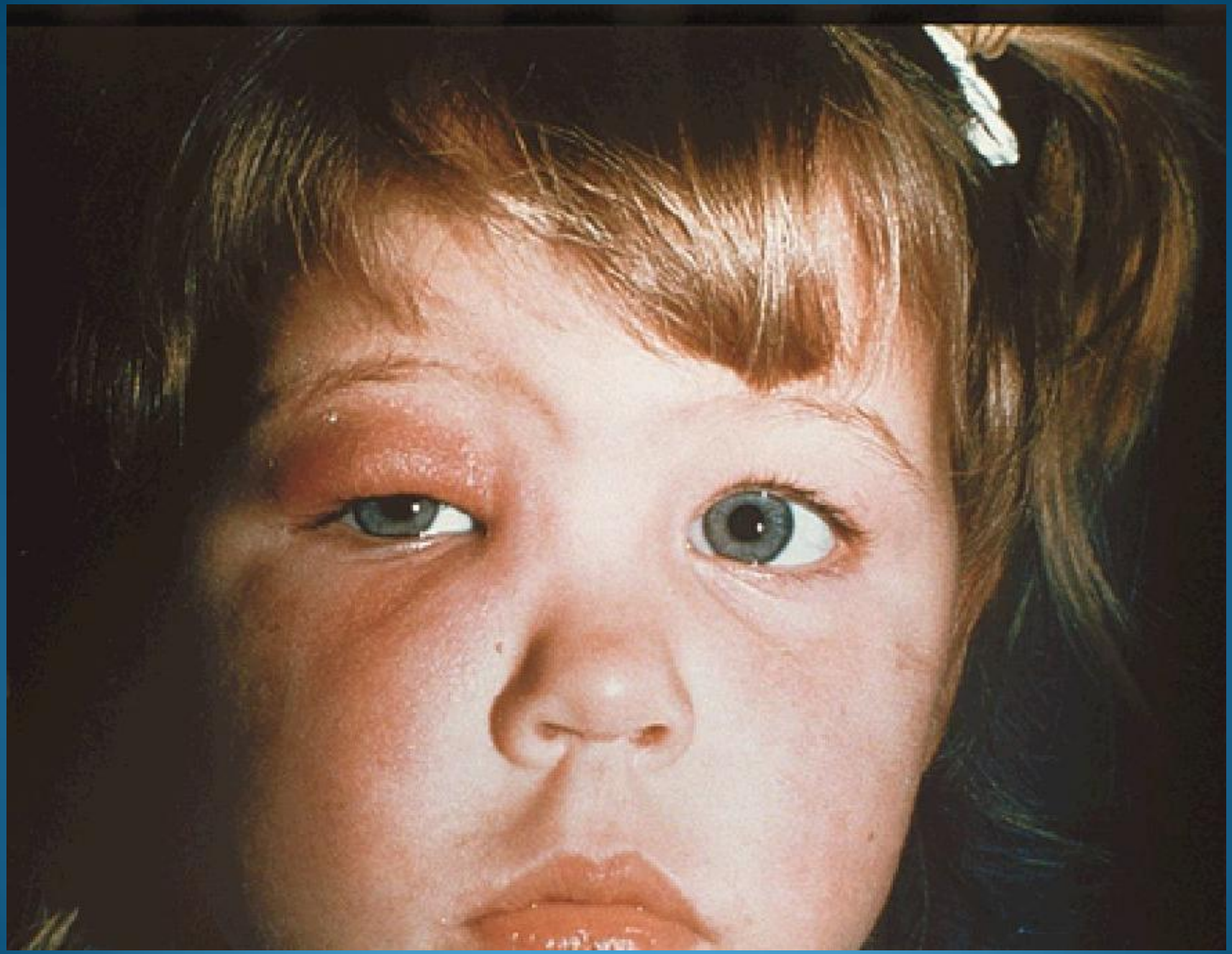


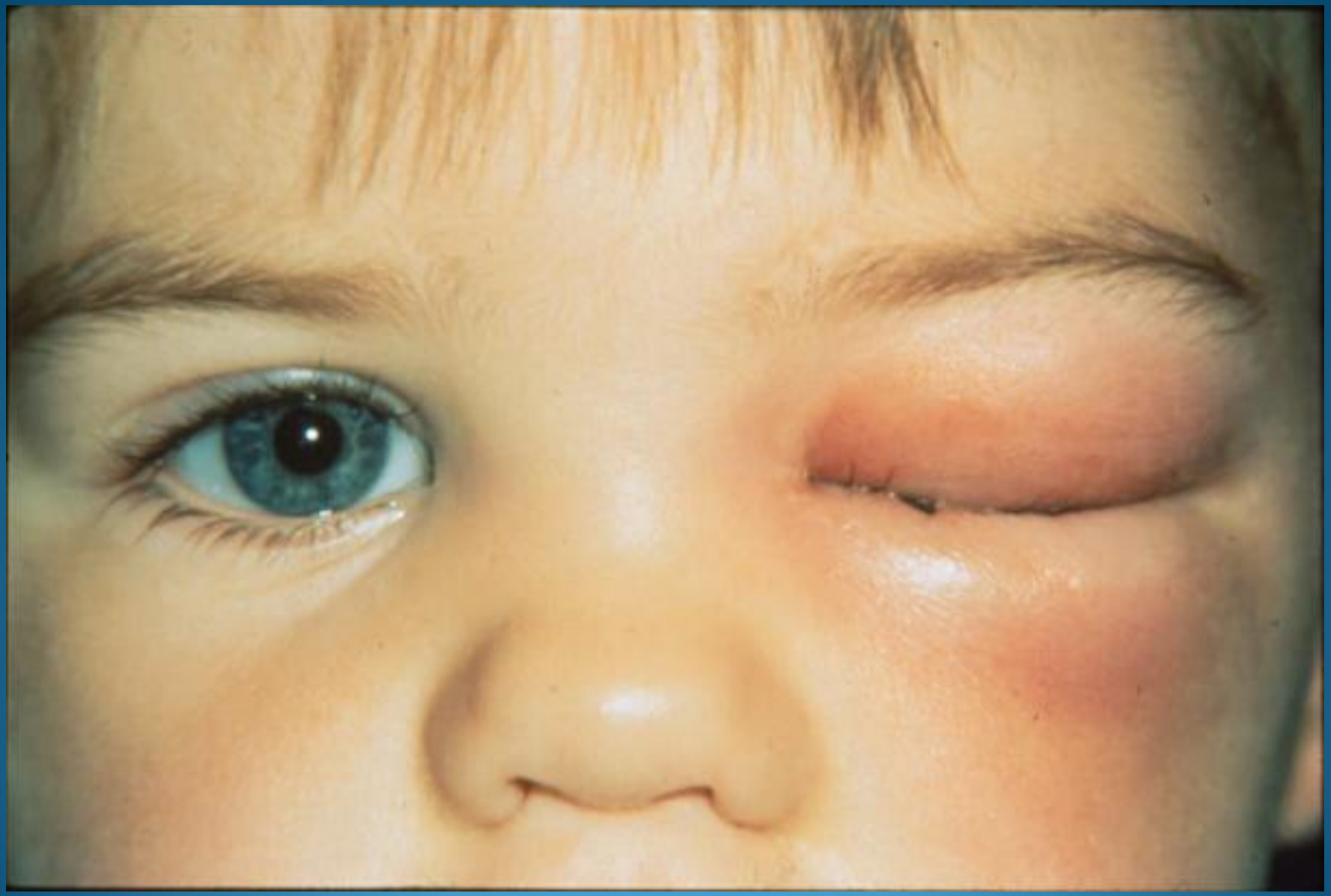
Cellulitis

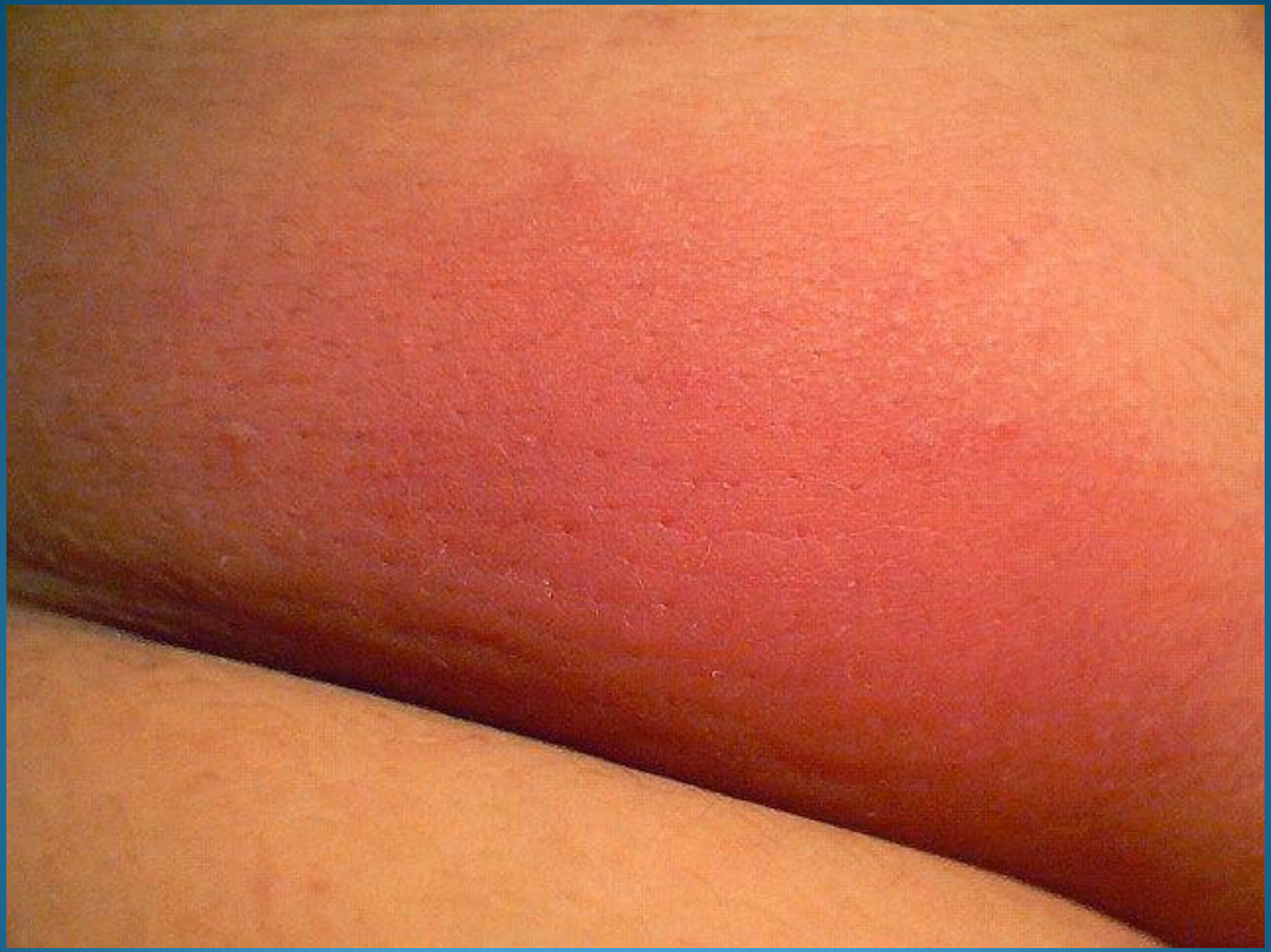
- Fever, malaise and cutaneous inflammation
- Ill defined, non-palpable erythema
- May develop vesicles, bullae and necrosis
- Ascending lymphangitis and regional LAD

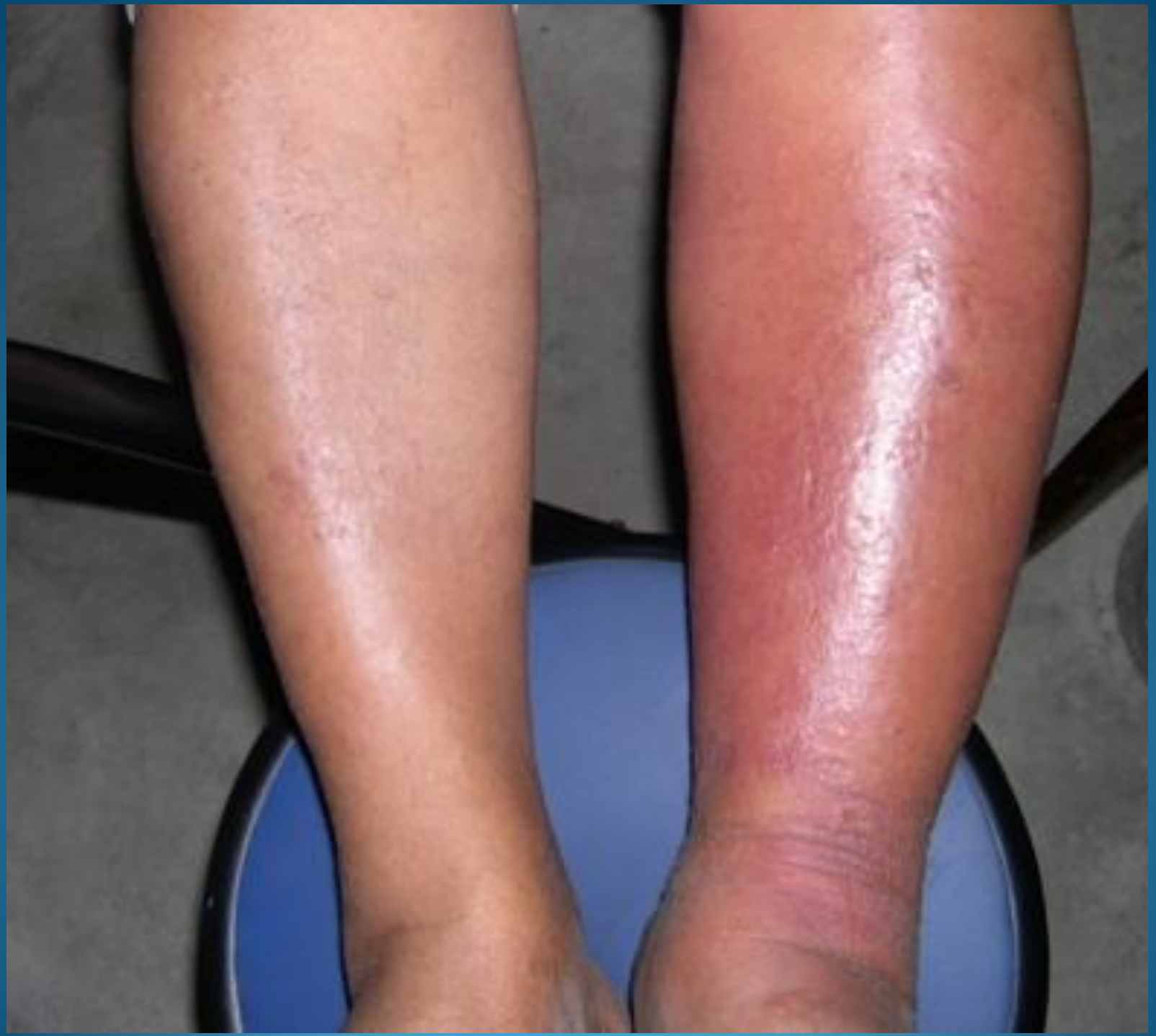
Cellulitis

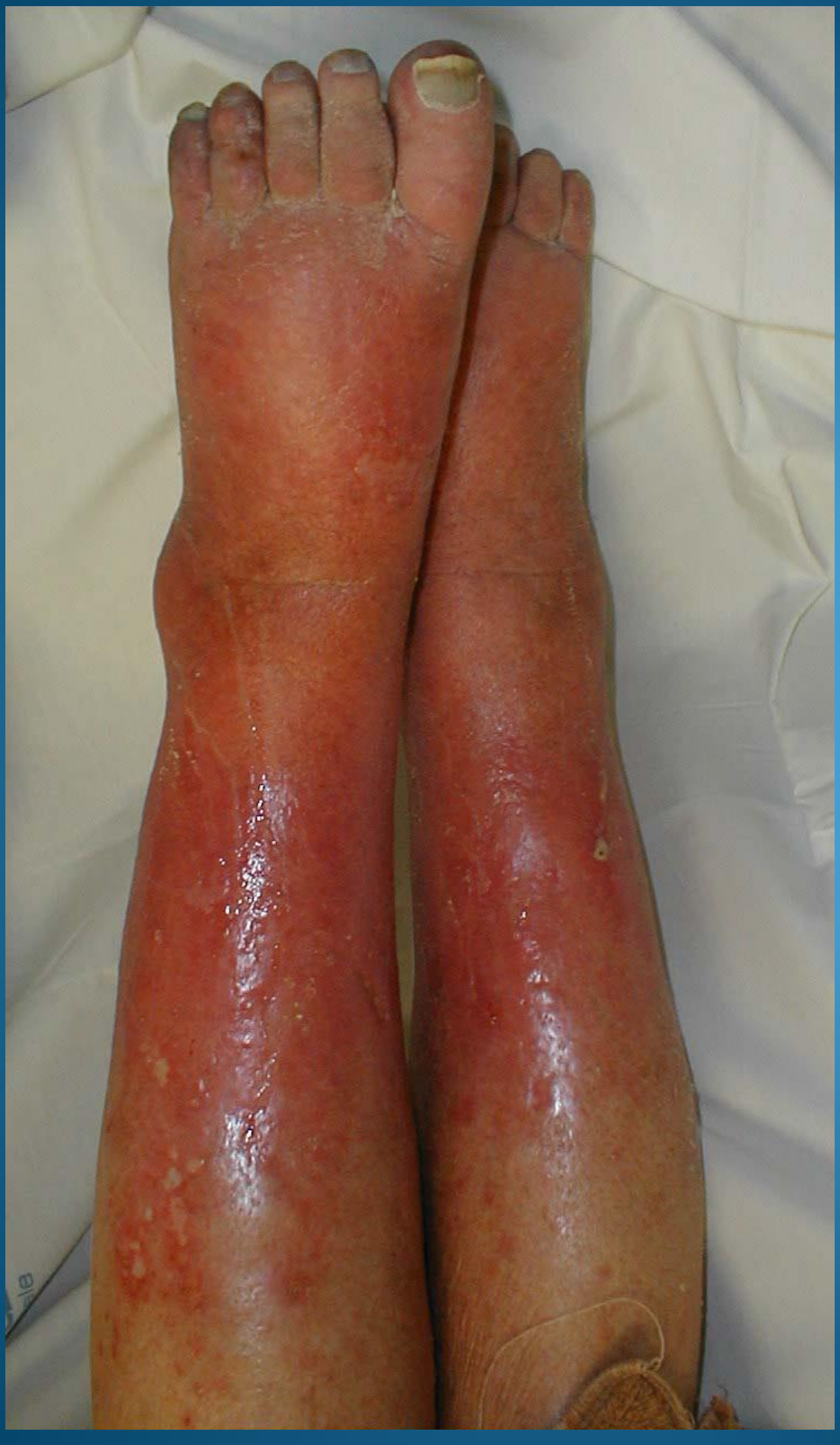
- Children- head, neck, perianal
- Adults- extremities
- IVDA- upper extremities
- Complications
  - Acute GN if caused by a nephritic strain of GAS
  - Subacute bacterial endocarditis
  - Lymphatic damage ► stasis ► recurrent cellulitis

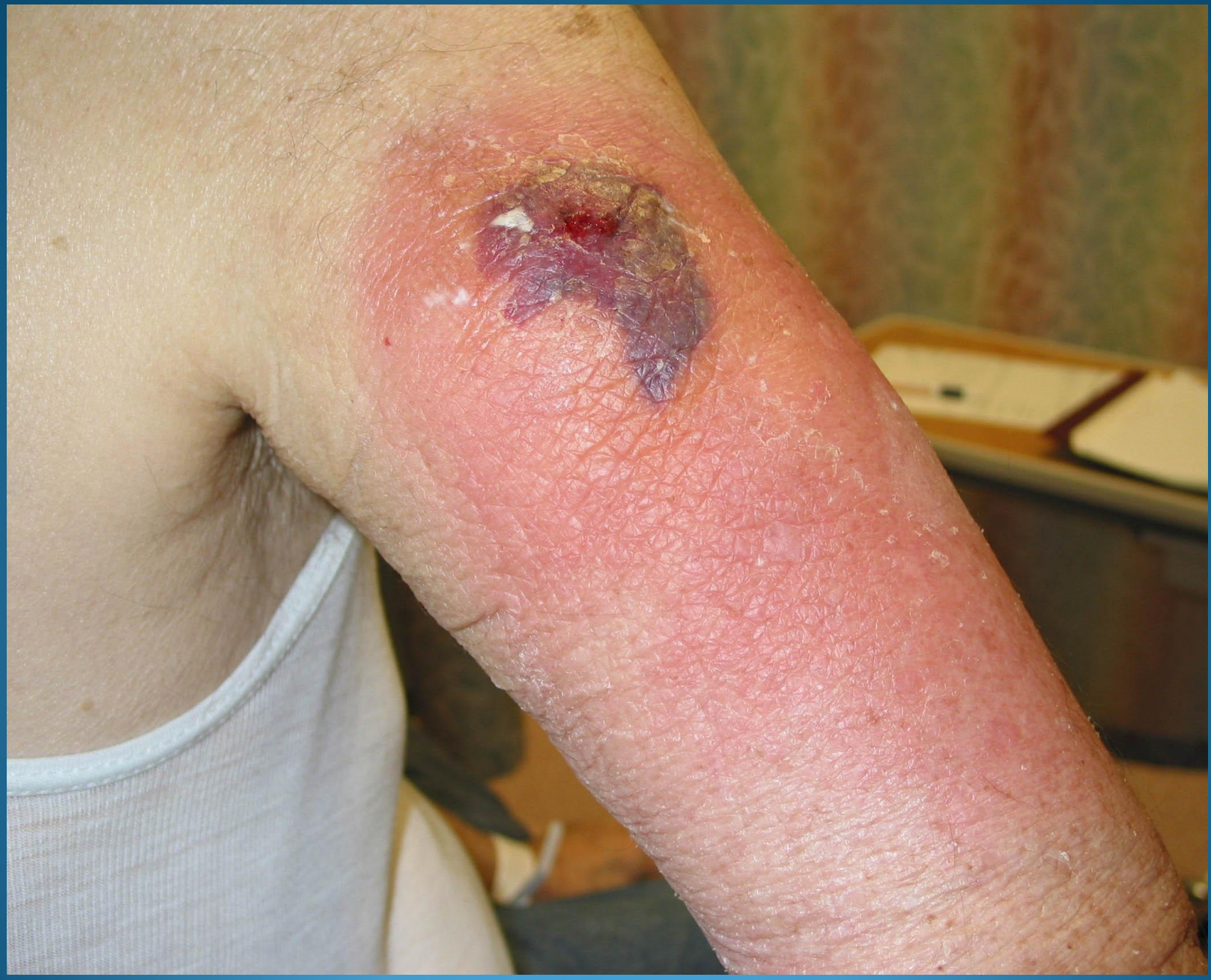












Cellulitis: Diagnosis

- Clinical
- Blood culture- only if bacteremia suspected
  - Staph and Strep cases positive in < 5%
  - H. flu may be positive
- Needle aspiration
  - Rarely obtained, ~30% yield
  - May be helpful in immunocompromised pts as high as 60% positivity
- Skin biopsy
  - Special stains can identify causative organisms

Cellulitis: Treatment

Mild cases

- Outpatient setting
- 10 day course of oral agents active against staph and strep
  - dicloxacillin, cephalexin, cefuroxime axetil, clindamycin

Severe cases

- Face, immunosuppression, or significant comorbidities
- Inpatient setting initially with IV antibiotics, wound care
  - Cefazolin, ceftriaxone, piperacillin/tazobactam

Allergic Patients

- Clindamycin, vancomycin, metronidazole + ciprofloxacin

Cellulitis: Treatment

Wound Care

- Immobilization and elevation
- Cool saline compresses to exudative areas
- Bland emollients if exfoliative
- Avoid NSAIDs
  - mask signs/sx of deeper necrotizing infections
- Control underlying disease
  - Tinea pedis, stasis dermatitis, fissures

Necrotizing Fasciitis

"Flesh-eating Bacteria"

A rapidly progressing necrosis of subcutaneous fat and fascia

Can be life threatening without prompt recognition, surgical intervention, and immediate antibiotic therapy

Necrotizing Fasciitis

Type 1: Polymicrobial

- Aerobes and anaerobes
- Strep, Staph, E. coli, Bacteroides, Clostridium
- Organisms enter at sites of trauma or surgery
- Slower pace of progression

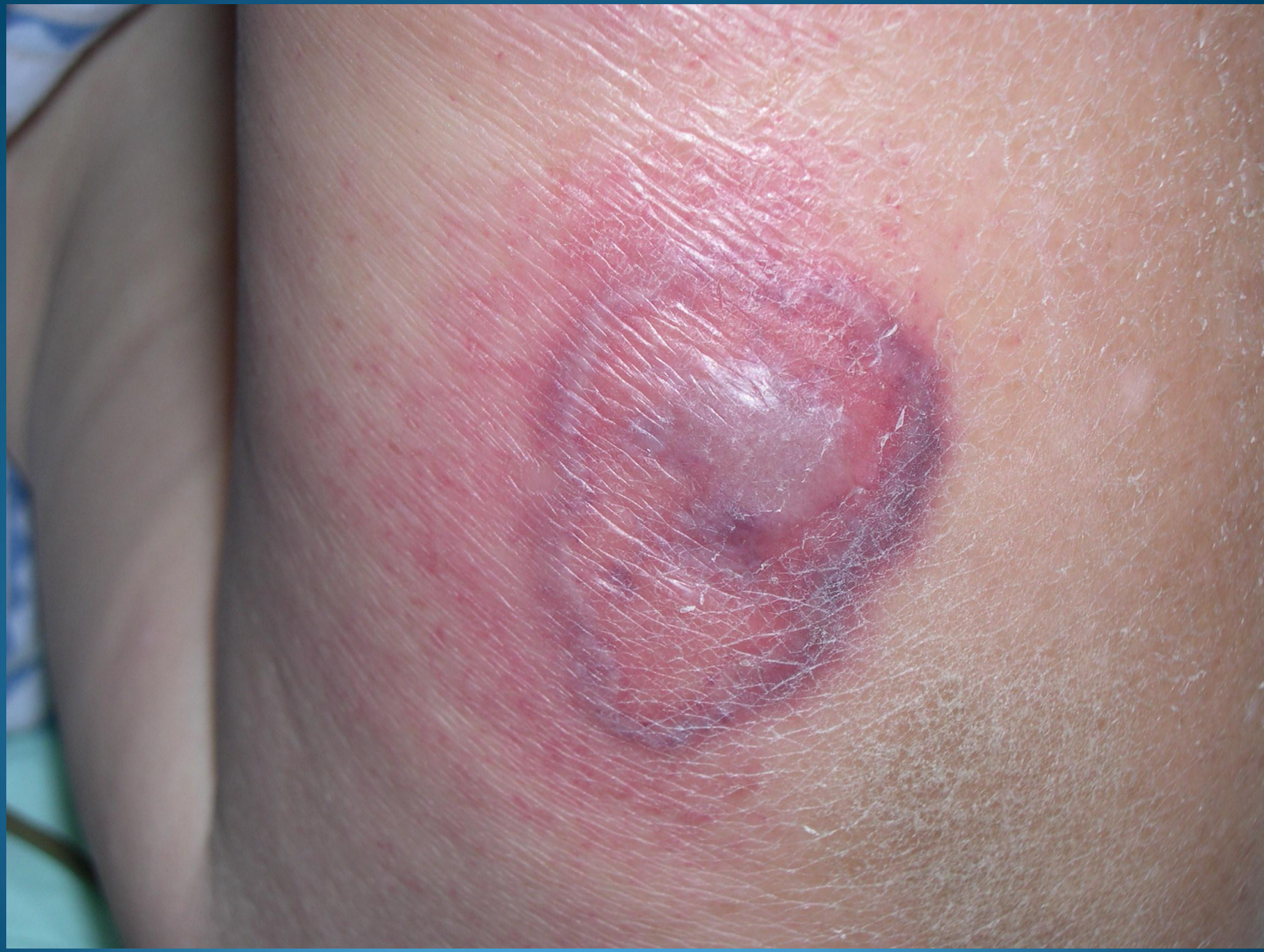
Type 2: Group A Strep (10%)

- M proteins: resist phagocytosis
- Pyogenic exotoxins: act as superantigens
  - Mediate fever, shock, and tissue injury

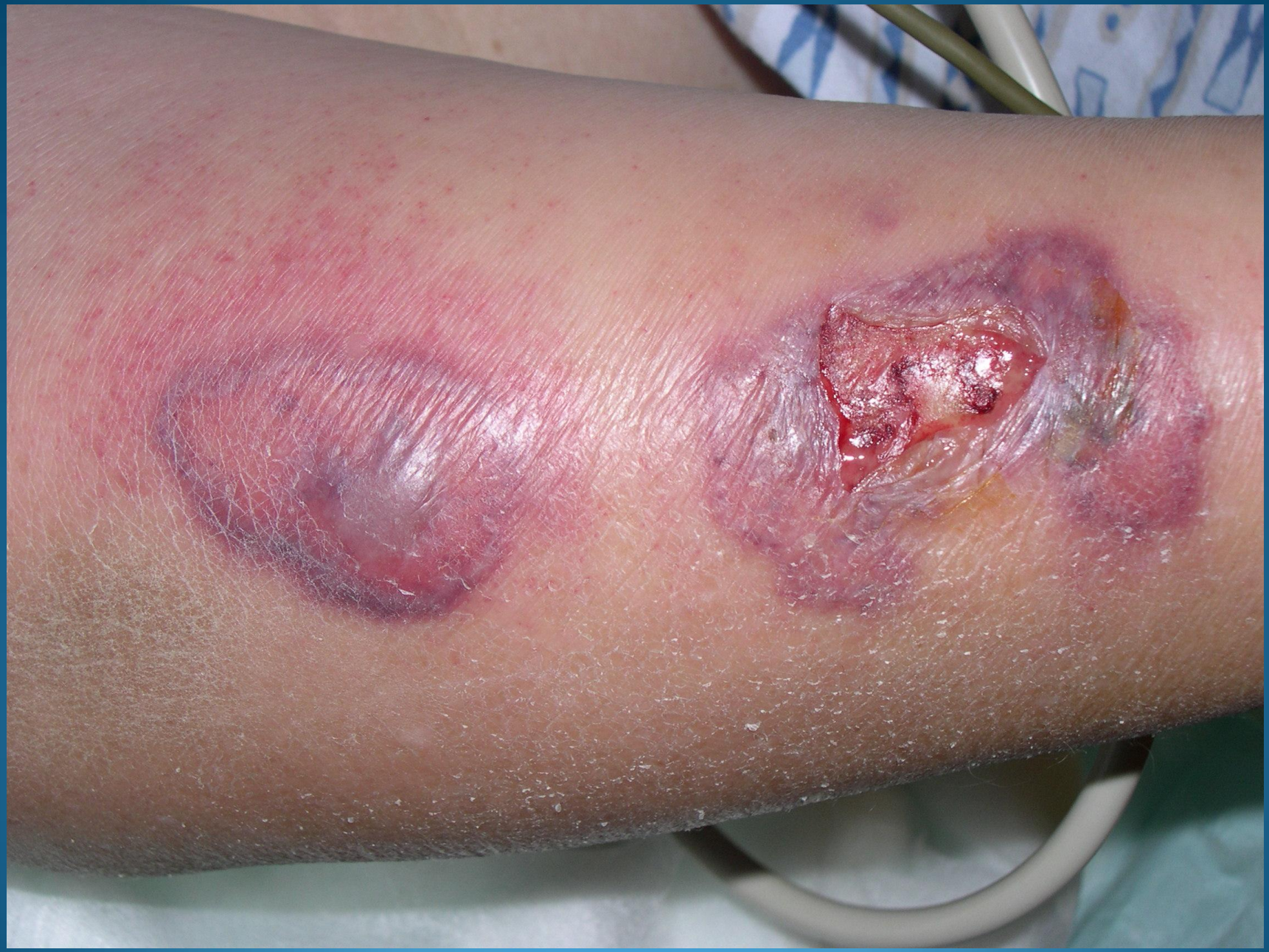
Necrotizing Fasciitis: Clinical Features

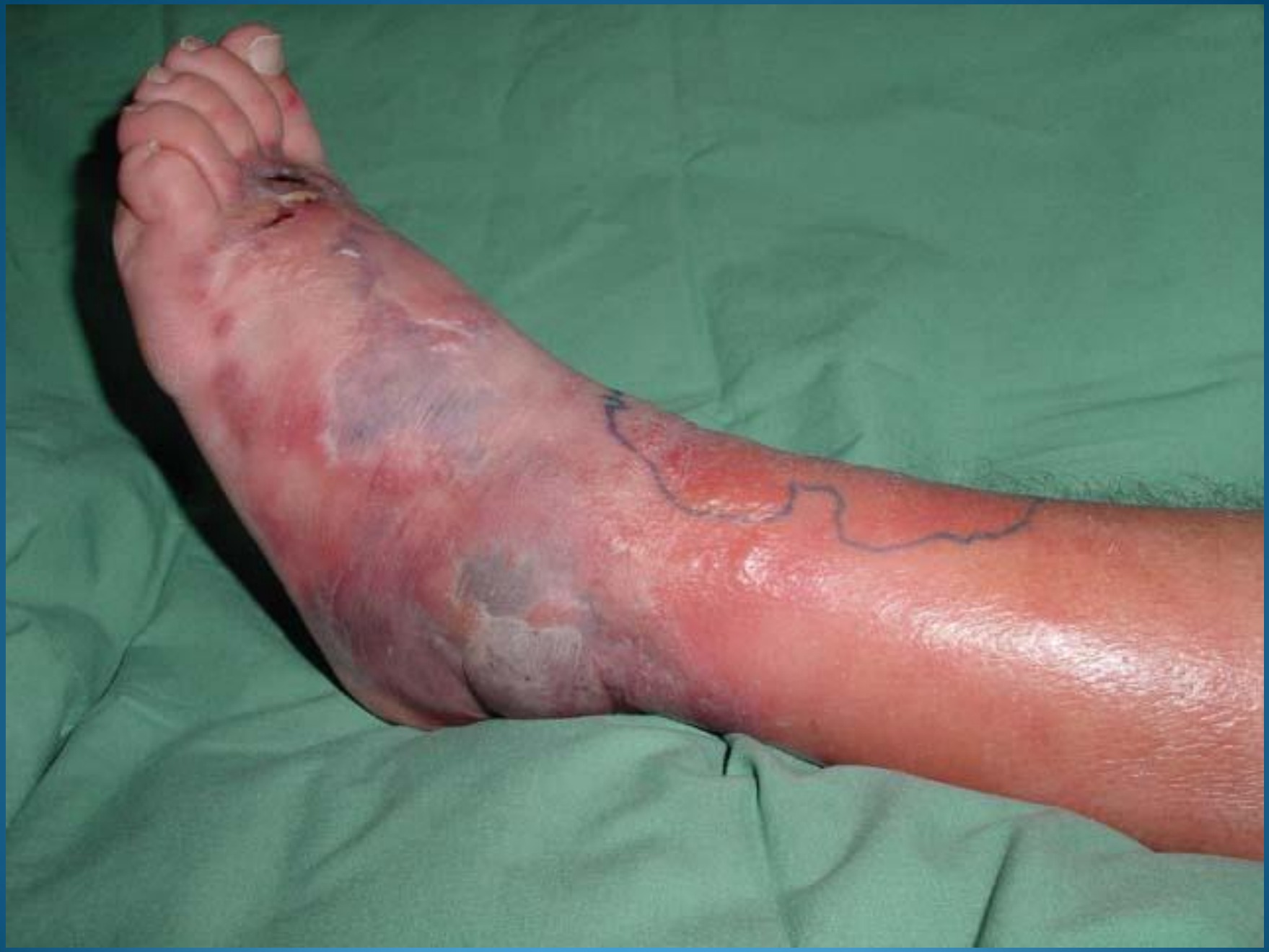
Severe, rapidly progressing cellulitis unresponsive to standard therapies

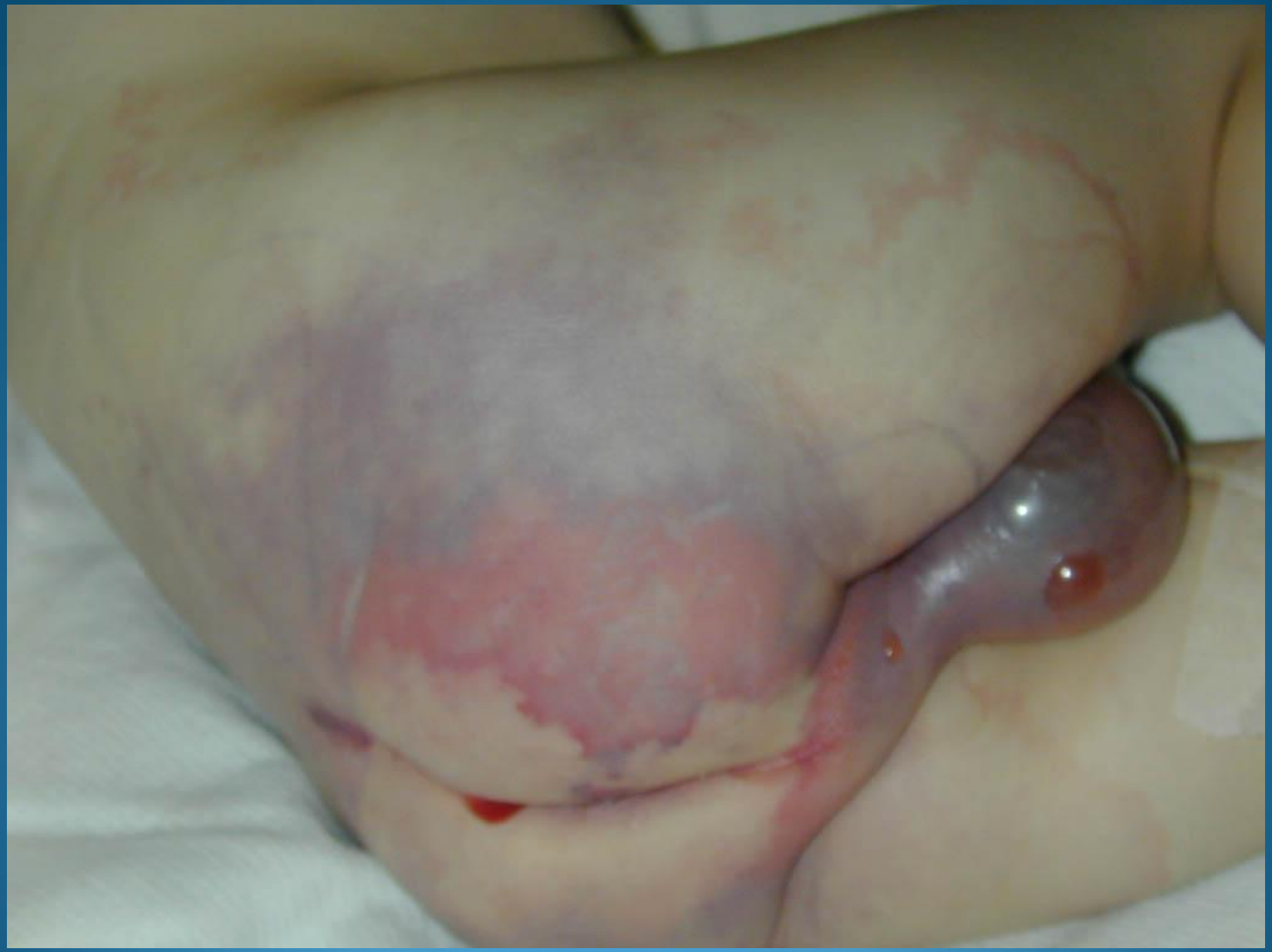
Extremities are #1 site



Shiny, tense skin changes from red-purple to a pathognomonic gray-blue with ill defined patches by 36 hours after onset

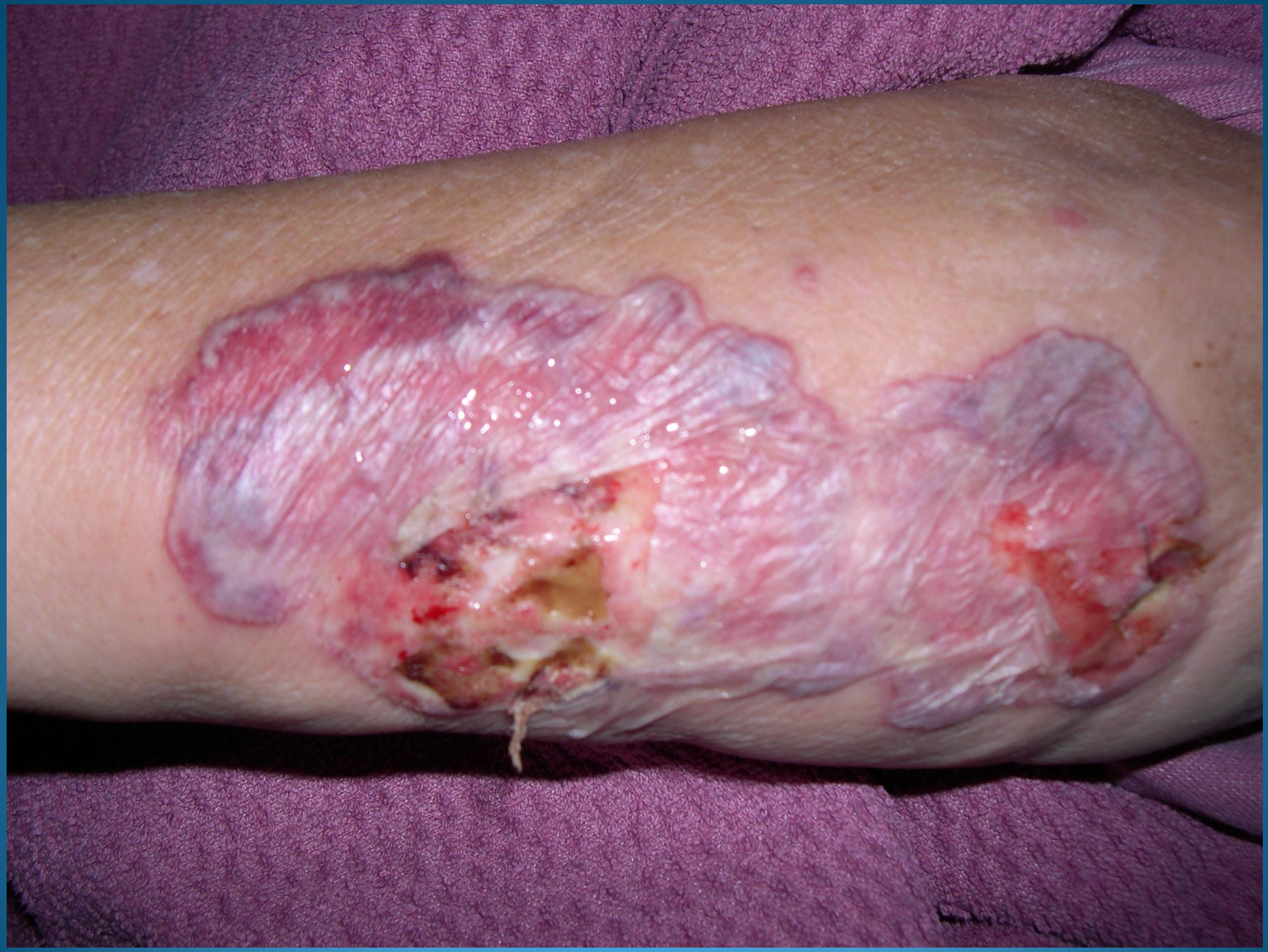


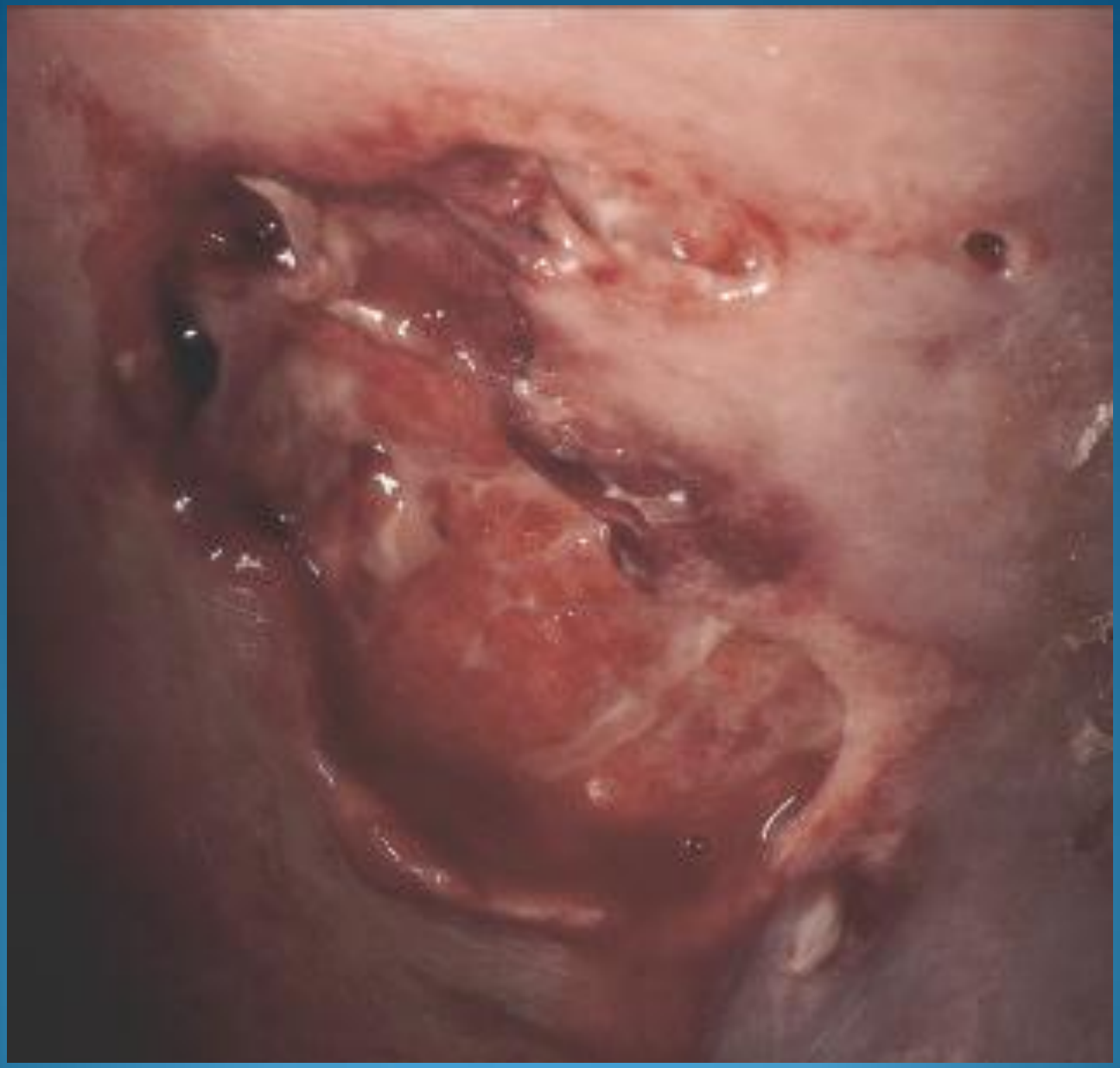




Violaceous bullae may develop.

Necrosis of fascia and fat produce watery, malodorous fluid

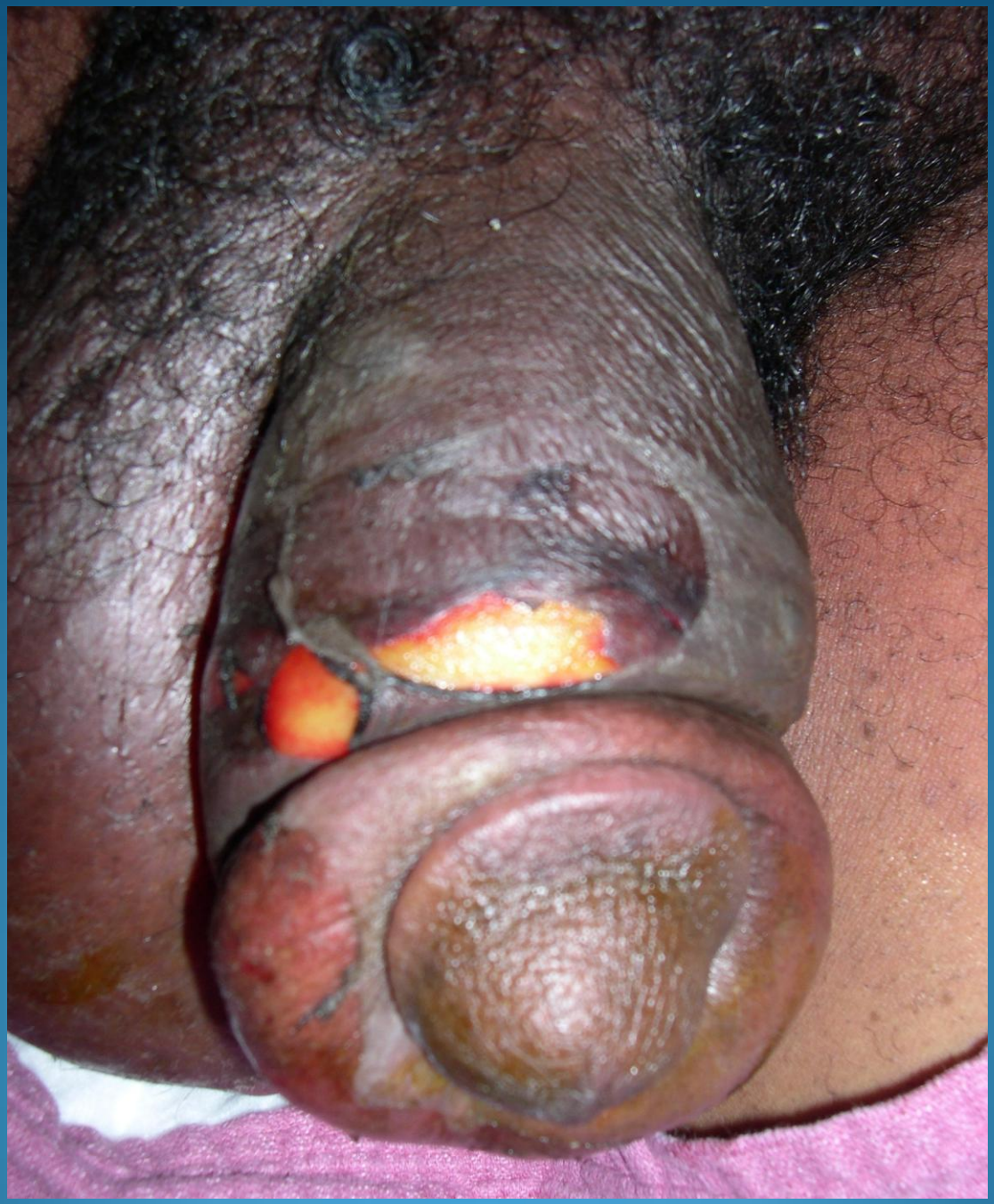


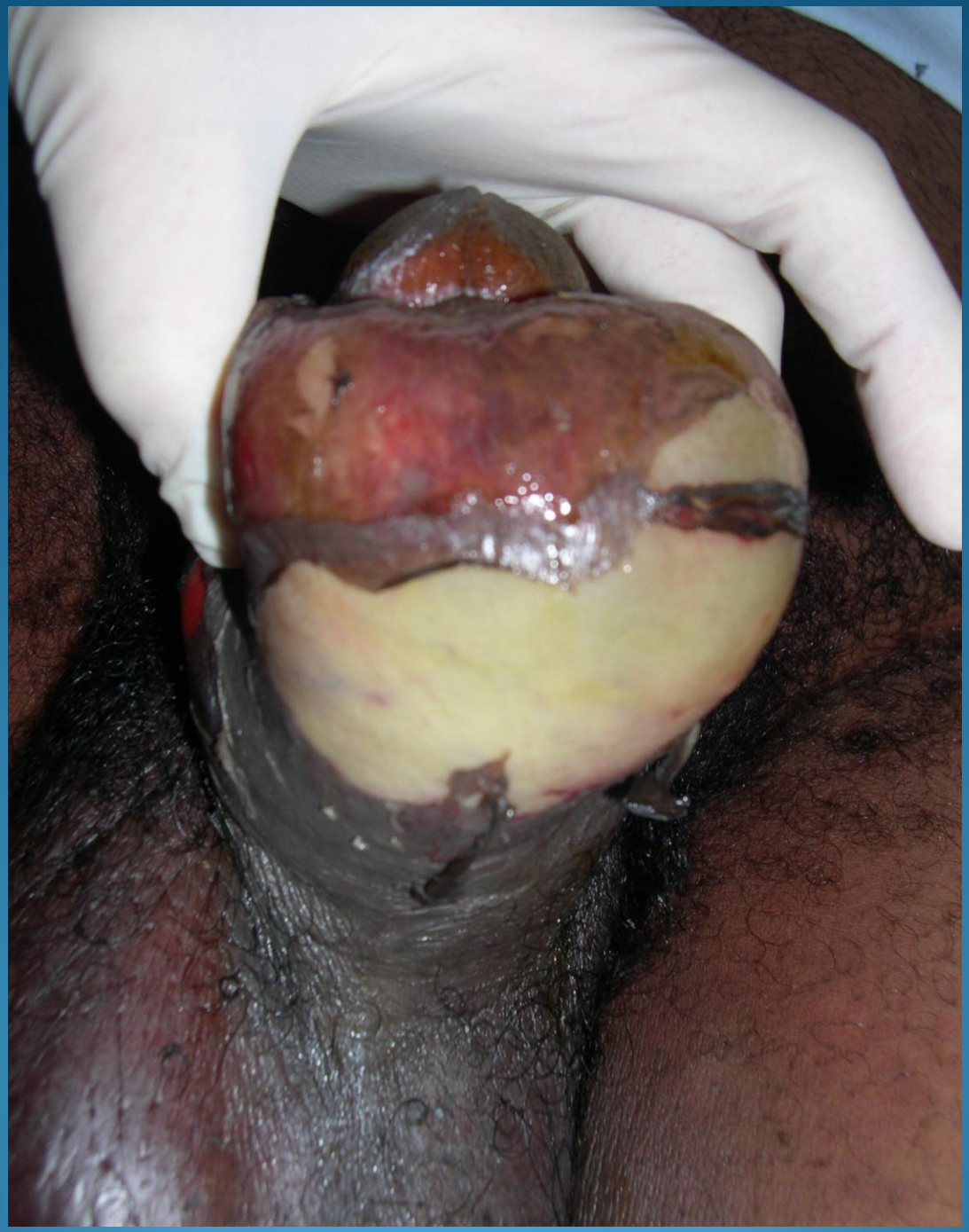


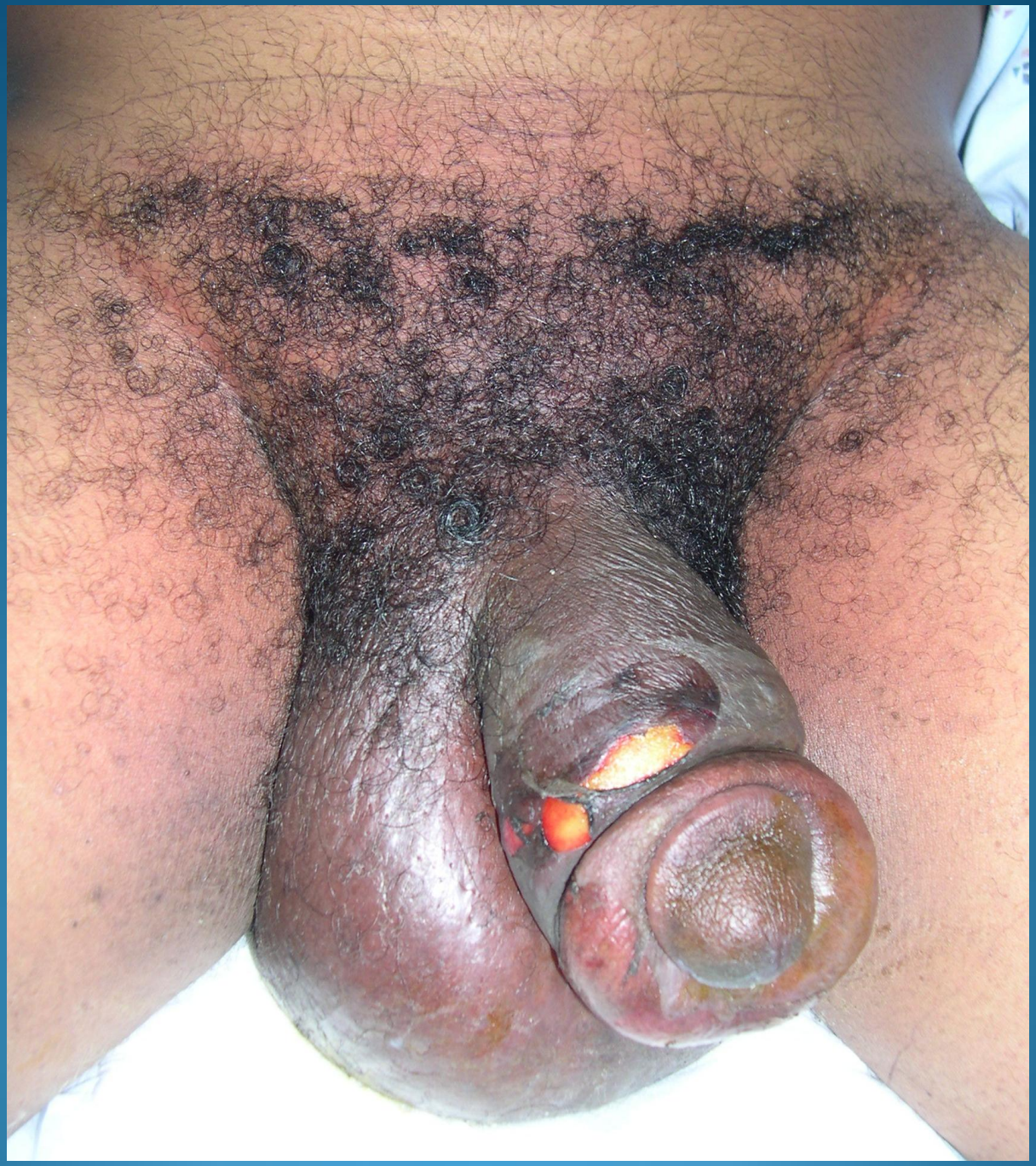
Necrotizing Fasciitis: Clinical Features

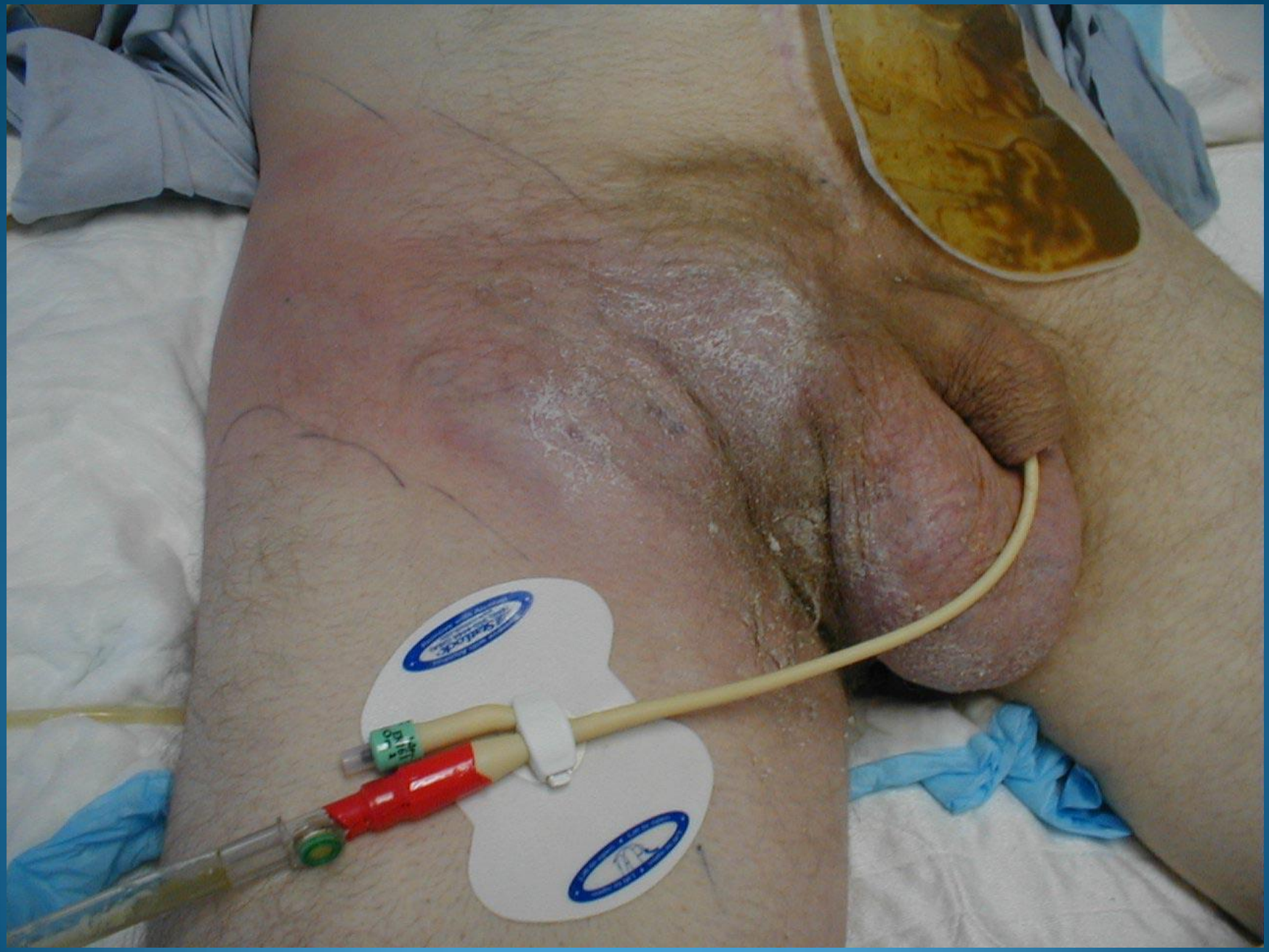
- Involved areas become anesthetic
- Tissue may develop woody induration
- Patient may become toxic +/- shock

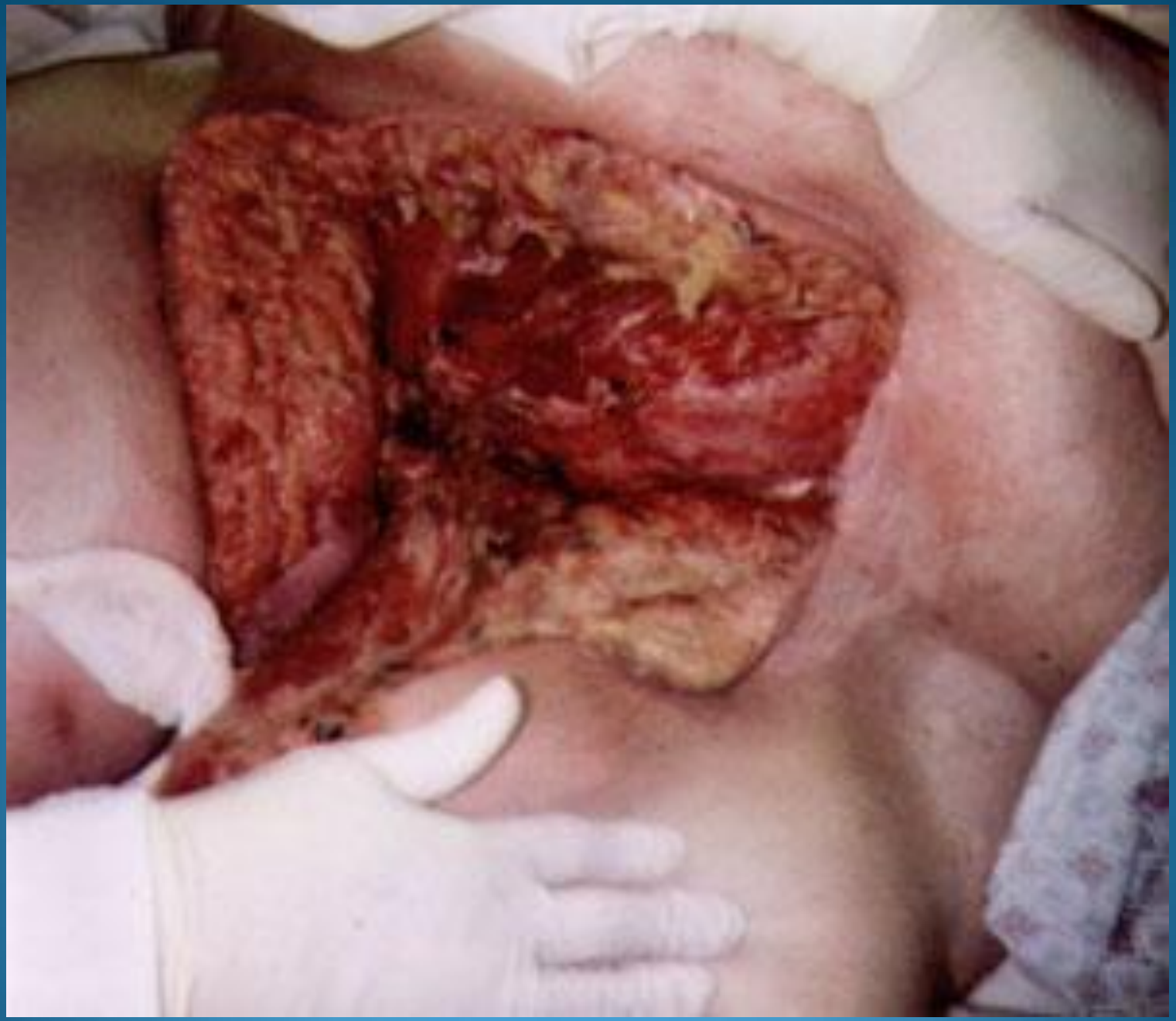
- Fournier's Gangrene- like type 1
  - Originates from the scrotum
  - Spreads to perineum and abdominal wall











Necrotizing Fasciitis: Risk Factors

- Group A Strep- Type 2
  - Young, previously healthy patients
- Polymicrobial- Type 1
  - Elderly, diabetics, alcoholics, CAD, PVD
- Penetrating or blunt traumatic injury
- Ulcers, recent surgery, disrupted skin
- Many cases occur in the absence of identifiable risk factors

Necrotizing Fasciitis

- Mortality ranges from 20-40%
- Increased mortality in:
  - Diabetics, elderly, malnourished, obese, PVD
  - Delayed diagnosis and treatment
  - Cases caused by Group A Strep
- Prognostic factors:
  - Hypotension on admission
  - WBC on admission >15.4
  - Serum Na < 135
  - MRI findings positive

Necrotizing Fasciitis: Diagnosis

Clinical exam

- Severe pain or anesthesia
- Rapid progression
- Bullae formation
- Toxic shock syndrome
- Elevated CPK
- Relevant history
  - surgery, trauma, NSAIDs, etc.

Work-Up

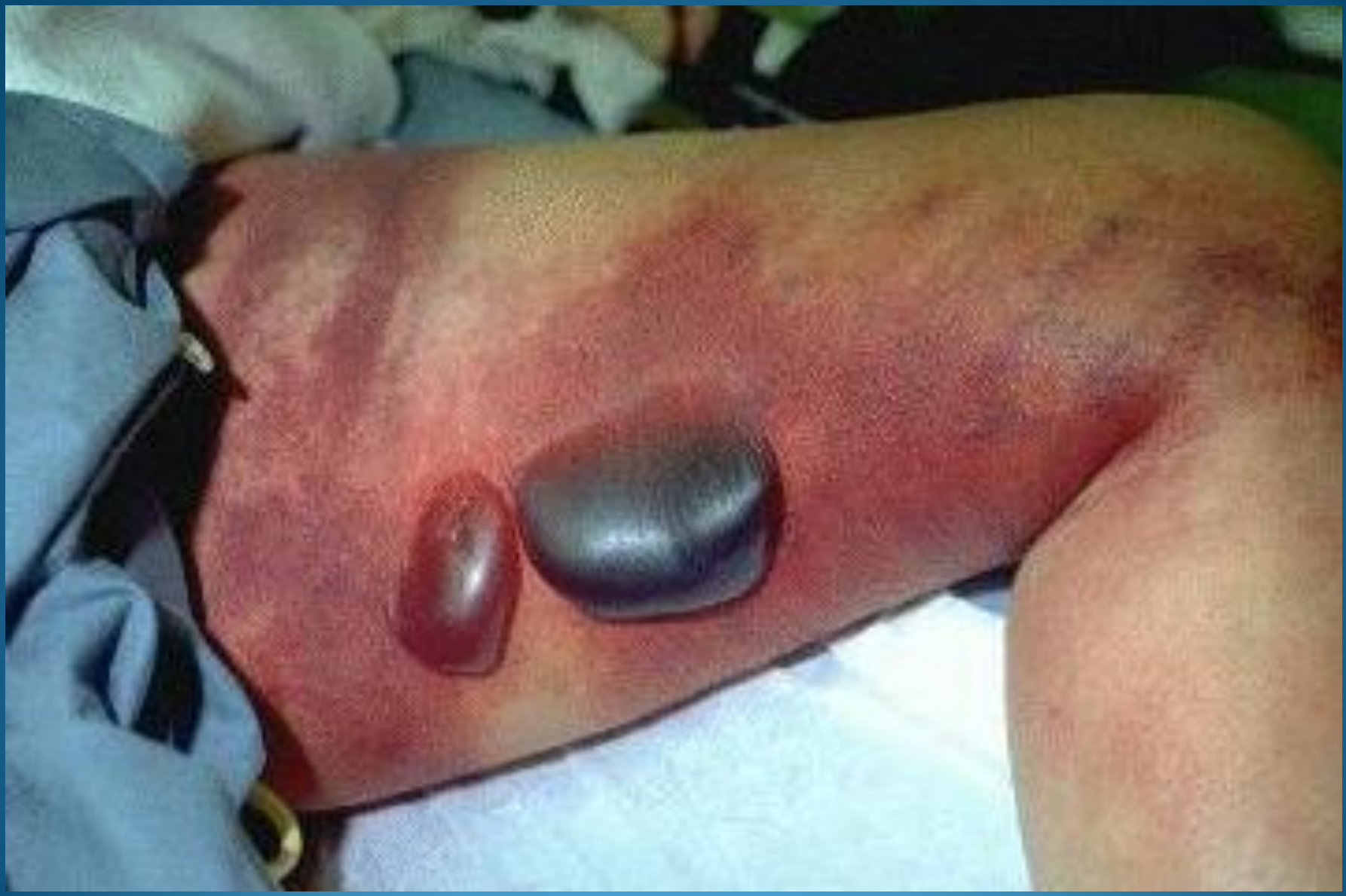
- **MRI** delineates depth
- **CT** pinpoints site
- Surgical consult
- Tissue Bx for culture
  - Advancing edge
  - Deep fascia

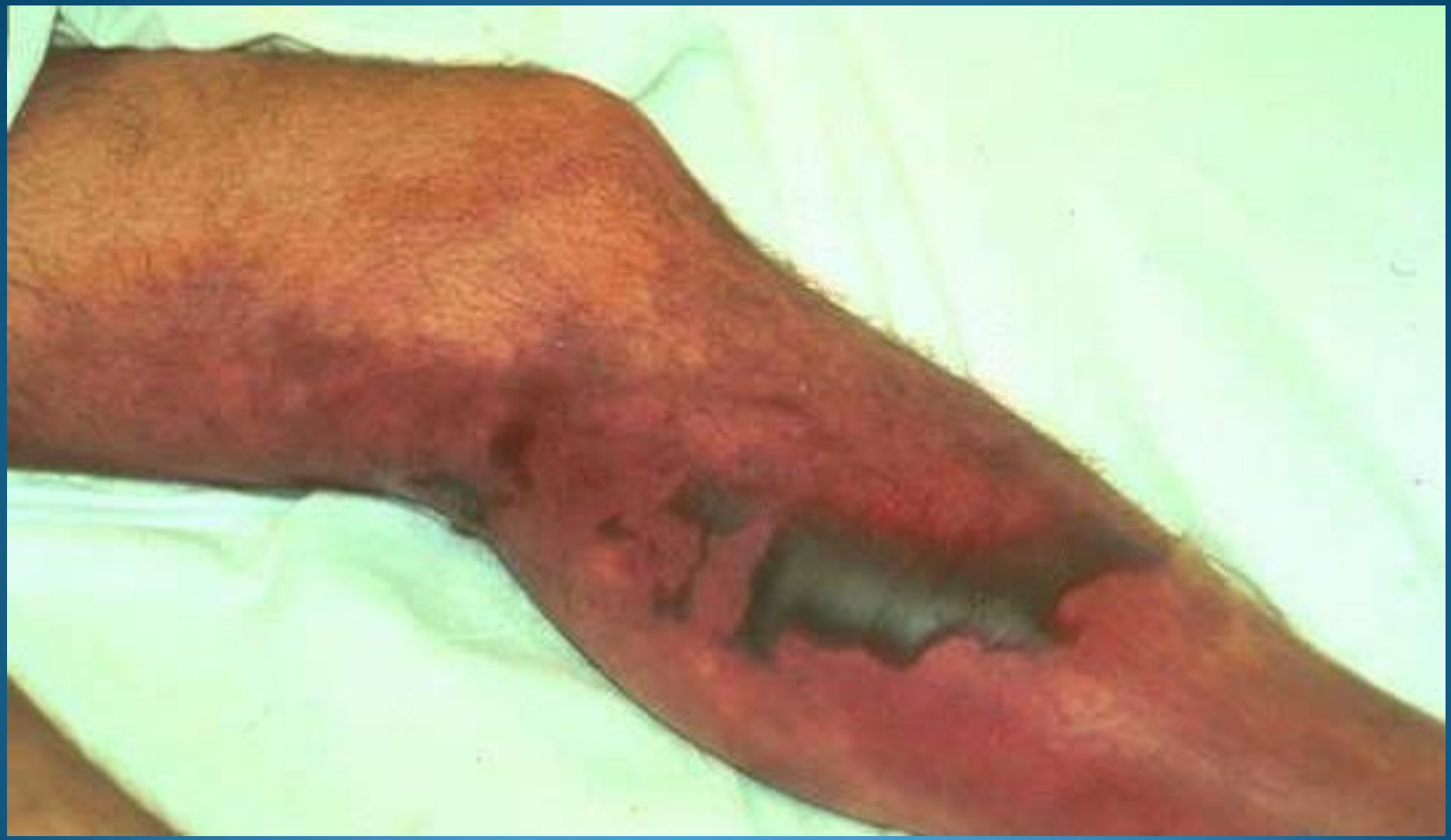
Necrotizing Fasciitis: Treatment

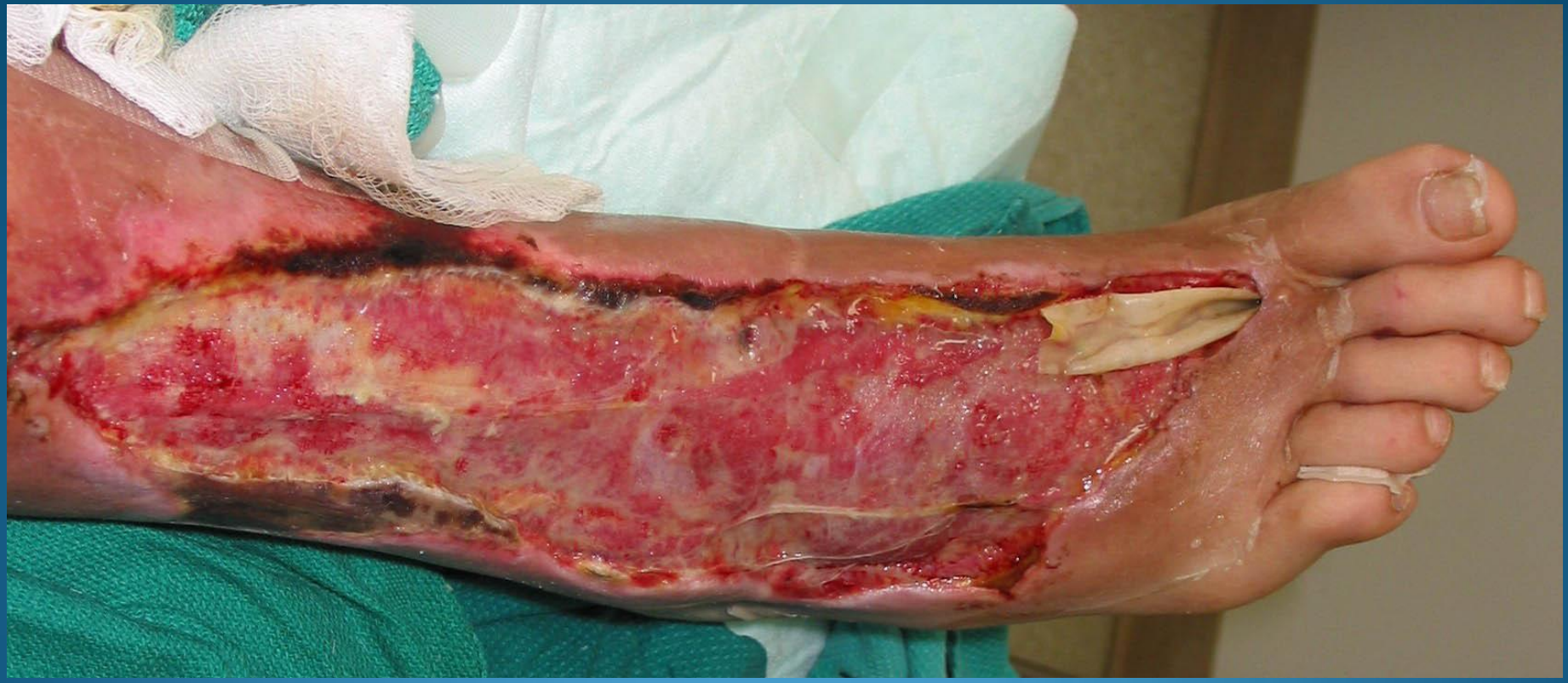
- Surgical fasciotomy
- Antimicrobials based on initial Gram stain
- B-lactam with broad spectrum coverage against GNR, staph, strep, and anaerobes
  - eg. piperacillin/tazobactam
  - If septic, add ciprofloxacin
  - If pen-allergic, consider cipro + metronidazole

Necrotizing Fasciitis: Treatment

- Hyperbaric **oxygen** is controversial- most data in anaerobic bacteria
- **IVIG** in Group A Strep cases- anecdotal
- Investigational drug **Tefibazumab**- humanized monoclonal antibody against microbial surface compounds
- Recombinant **Activated Protien C** in severe sepsis
- Intensive supportive care







Toxic Shock Syndromes

Toxic Shock Syndromes

- Toxin-mediated multisystem disease precipitated by infection with *Staph aureus* or *Strep pyogenes* (Group A Strep)
- Strep is more common than Staph
- Characterized by:
  - Sudden onset of high fever and hypotension
  - Petechial or maculopapular rash
  - Severe N/V/D/HA/ST, myalgia, confusion, coma
  - High mortality

Toxic Shock Syndromes

- Consider in anyone presenting with abrupt onset fever, rash, hypotension, renal or respiratory failure, and mental status changes

- Risk factors
  - HIV, diabetes, cancer, other chronic disease
  - Alcoholism
  - Recent varicella infection (chicken pox)
  - Patients using NSAIDs

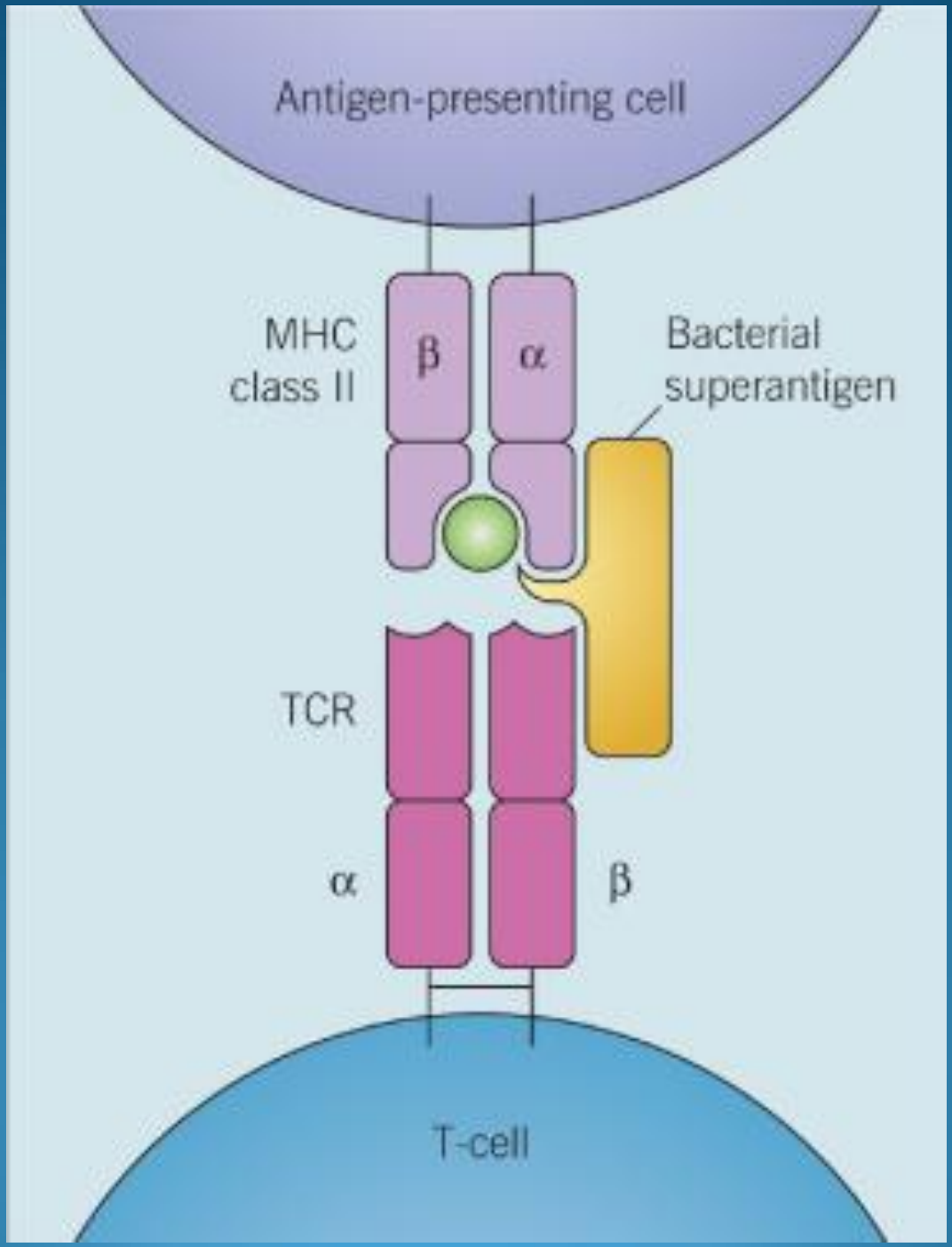
Streptococcal Toxic Shock Syndrome

Strep Toxic Shock Syndrome

- Healthy adults aged 20-50
- A clinically apparent soft tissue infection is usually present in 80%
  - Abscess; cellulitis; necrotizing fasciitis

Strep Toxic Shock Syndrome

- Direct tissue invasion and destruction
  - Cutaneous barrier disruption = portal of entry
- Streptococcal pyrogenic exotoxins, SPEA,SPEB and Strep M protein 1 and 3:
  - Act as **superantigens**
    - Stimulate T-cells
    - Massive cytokine production-TNFα, IL 1, IL6
    - Tissue injury and shock


Antigen-presenting cell
MHC
class II
β
α
Bacterial
superantigen
TCR
α
β
T-cell

Super Antigens

- Not MHC restricted
- Binds the Variable region of T cell receptor and conserved area of MHC II
- Normal antigen binds all five variable sites to activate T cells
- While antigen stimulation may case 0.1% T cell activation, superantigen is able to activate 20-30% of T cells
- Increases CLA- cutaneous lyphocyte antigen, leading to increased homing of lymphocytes to the skin
- Massive release of TNFα, IL1, IL6

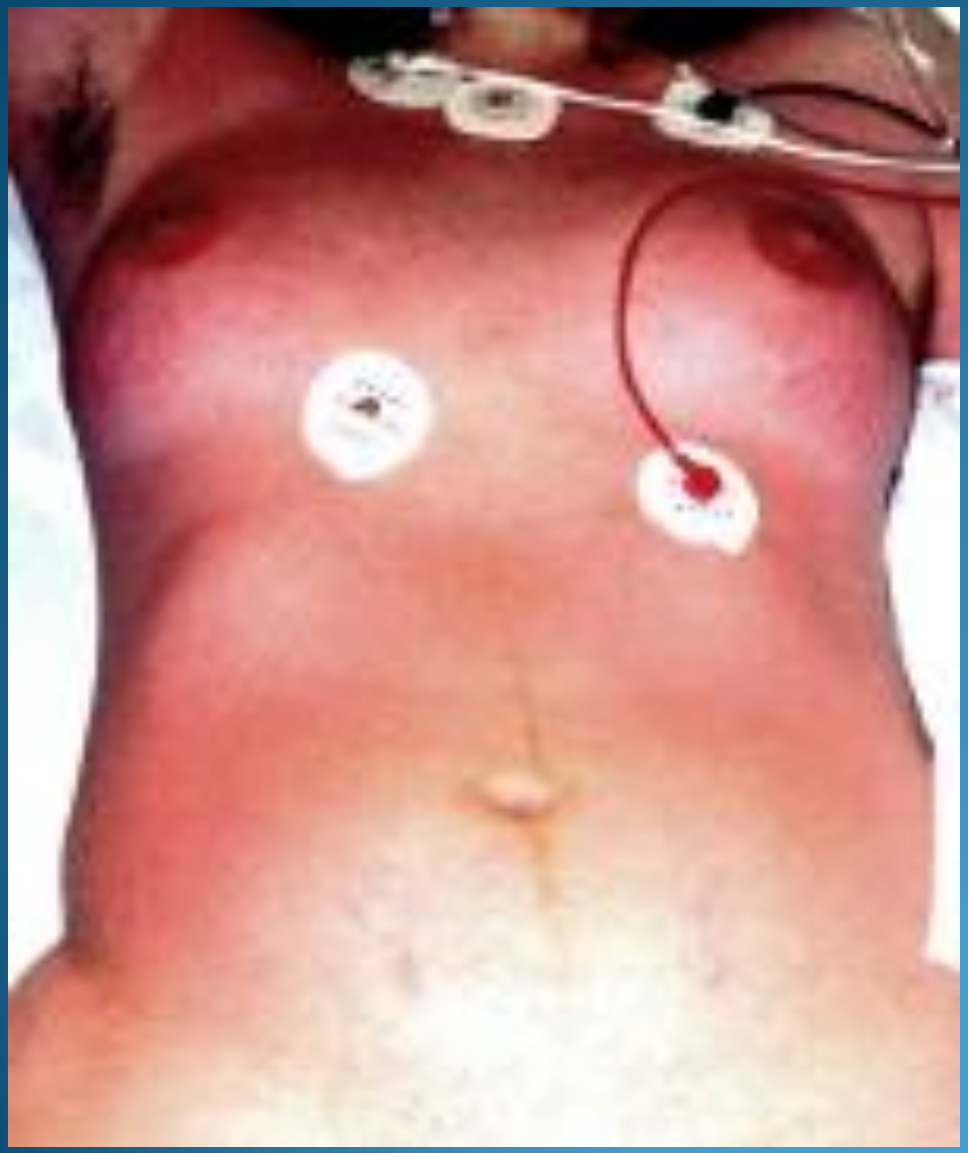
Streptococcal TSS: Clinical Features

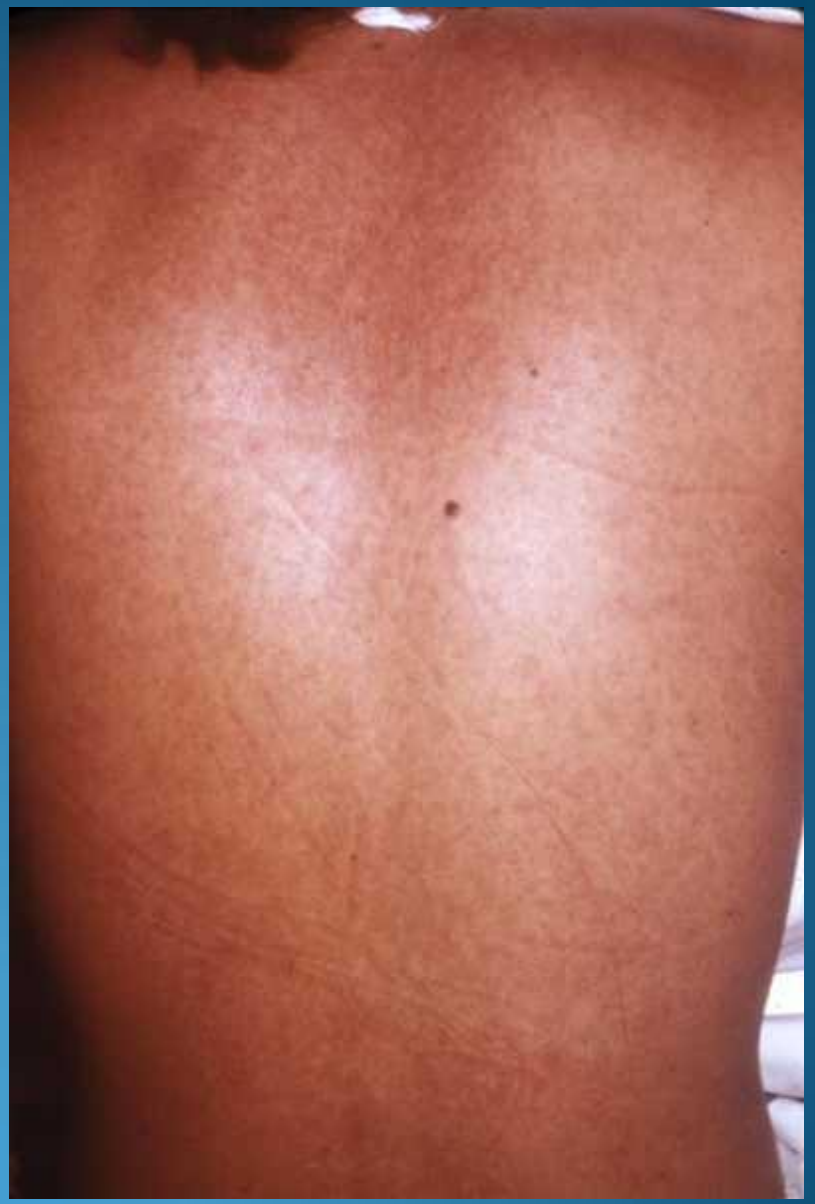
- Begins insidiously with flu-like symptoms
- *Severe pain in an extremity*
- 80% have an apparent soft tissue infection
- Not associated with tampon usage
- Blood Cultures + in greater than ½ unlike Staph 1/10
- Vesicles and bullae indicate a deeper infection and a worse outcome

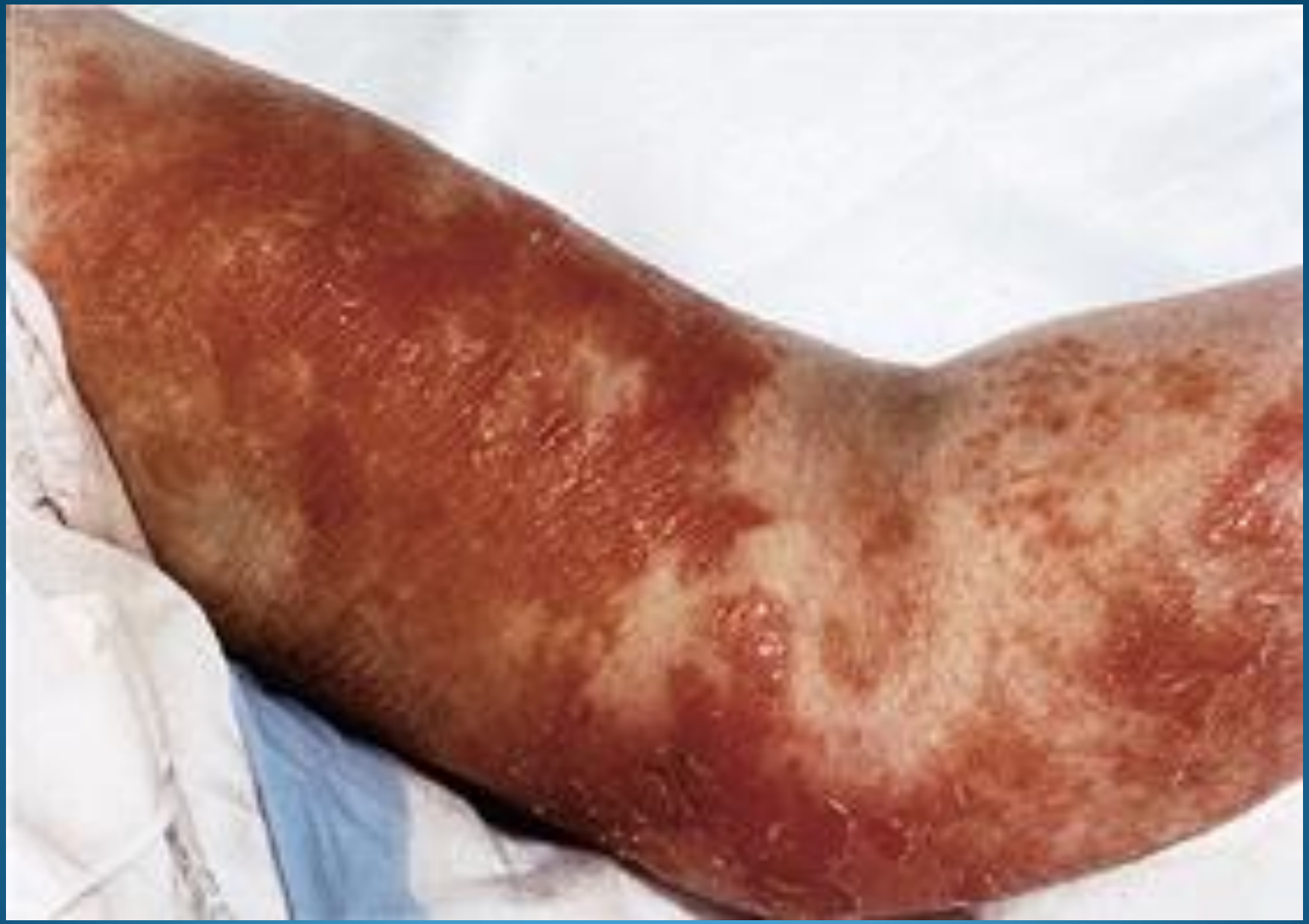
Streptococcal Toxic Shock Syndrome

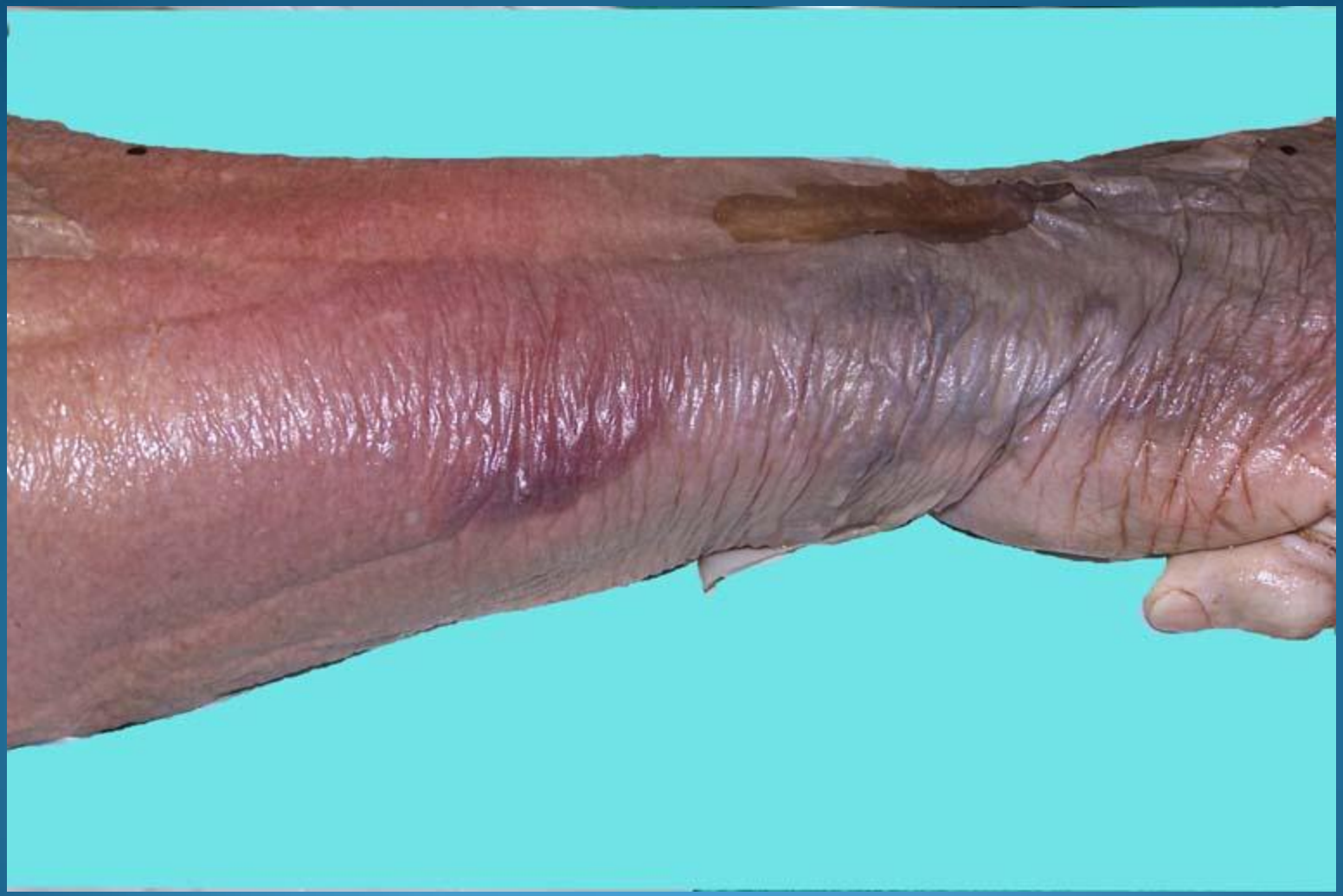
Cutaneous findings:

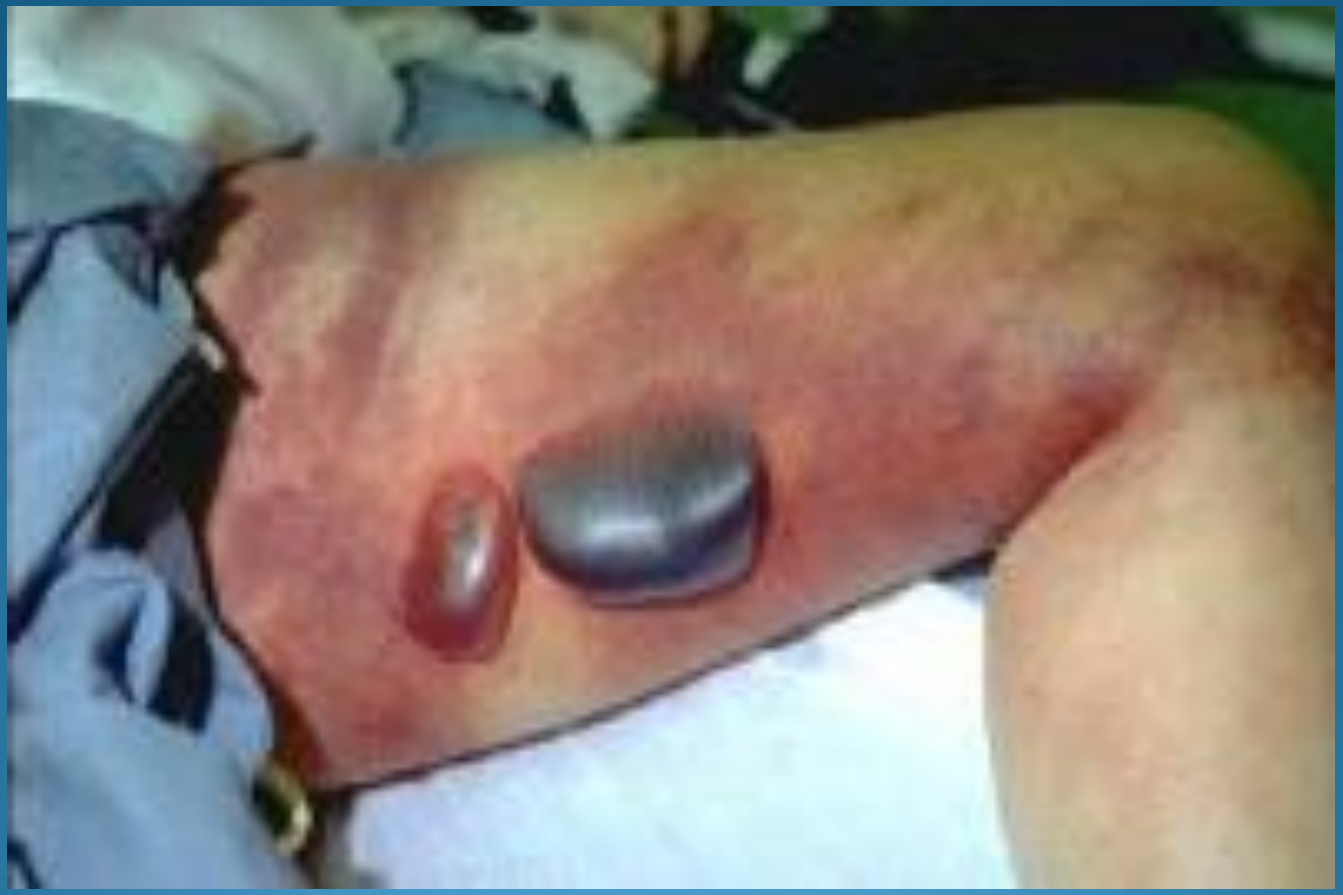
- Soft tissue infection (80%)
- Generalized erythematous eruption
- Acral desquamation in 20%
- Shock and multi-organ failure within 48-72°











Streptococcal TSS Case Definition
(JAMA 1993)

- Isolation of *S. pyogenes* from a normally sterile site
  - blood, CSF, pleural fluid
  - definite case

OR

- Isolation from a nonsterile site
  - throat, sputum, vagina, superficial skin lesion
  - probable case

Streptococcal TSS Case Definition
(JAMA 1993)

PLUS

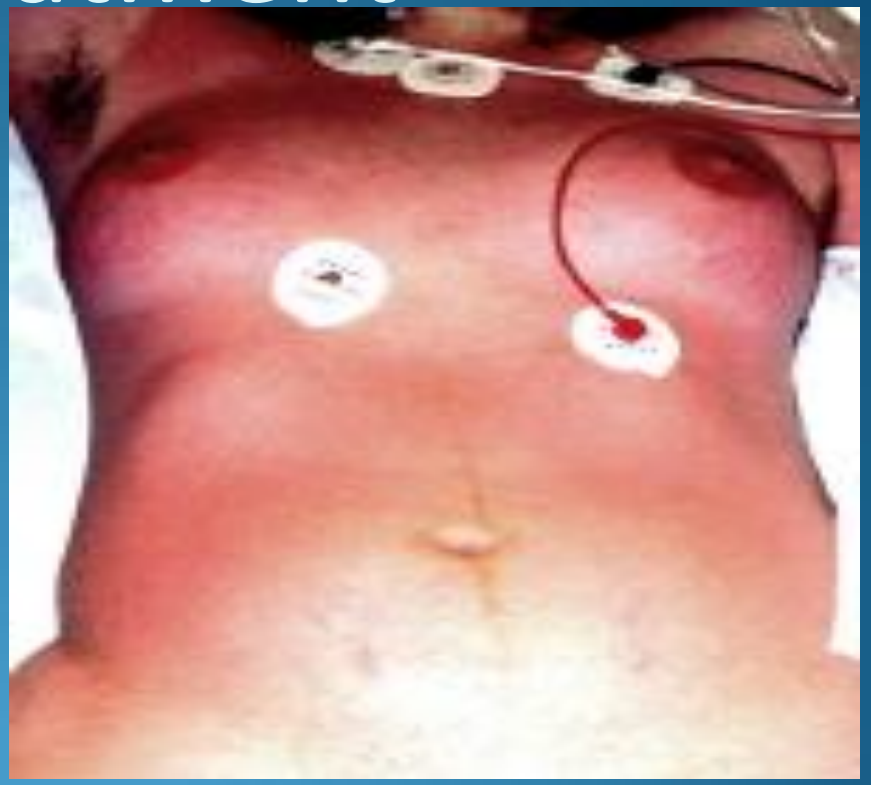
- Hypotension
  - SBP < 90 mmHg or < 5th percentile (kids)

AND

- Multi-organ involvement (2 or more systems)
  - renal, hepatic, pulmonary, hematologic, cutaneous

Strep TSS Treatment

- Intensive supportive therapy
- Intravenous antibiotics
- Surgical intervention



Strep TSS Treatment

- Clindamycin (900 mg IV q8h)
  - 1st line
  - Inhibits bacterial toxin production

- β-lactam antibiotics alone *are not* 1st line
  - aggressive S. pyogenes infections do not respond well to penicillin and are associated with high morbidity/mortality

Strep TSS Treatment

- Clindamycin 900 mg IV q8h

- Clindamycin 900 mg IV q8h

  +

  Penicillin G 4 million U IV q4h

- Mortality: high (30-70%)

Staphylococcal Toxic Shock Syndrome

Staph Toxic Shock Syndrome

- Toxin production by *Staphylococcus aureus*
- Described in 1978
- Associated with tampons in 1980
- Currently, non-menstrual cases predominate

Staph Toxic Shock Syndrome

Toxic Shock Syndrome Toxin-1- menstruation

- Exotoxin has 3 main mechanisms
  - acts as a superantigen
  - direct toxic effects on organ systems
  - impairs clearance of endogenous endotoxins derived from gut flora
  - Able to cross mucous membranes

Staph Enterotoxin B and C for nonmentruating

Staph Toxic Shock Syndrome: Clinical Settings/Risk Factors

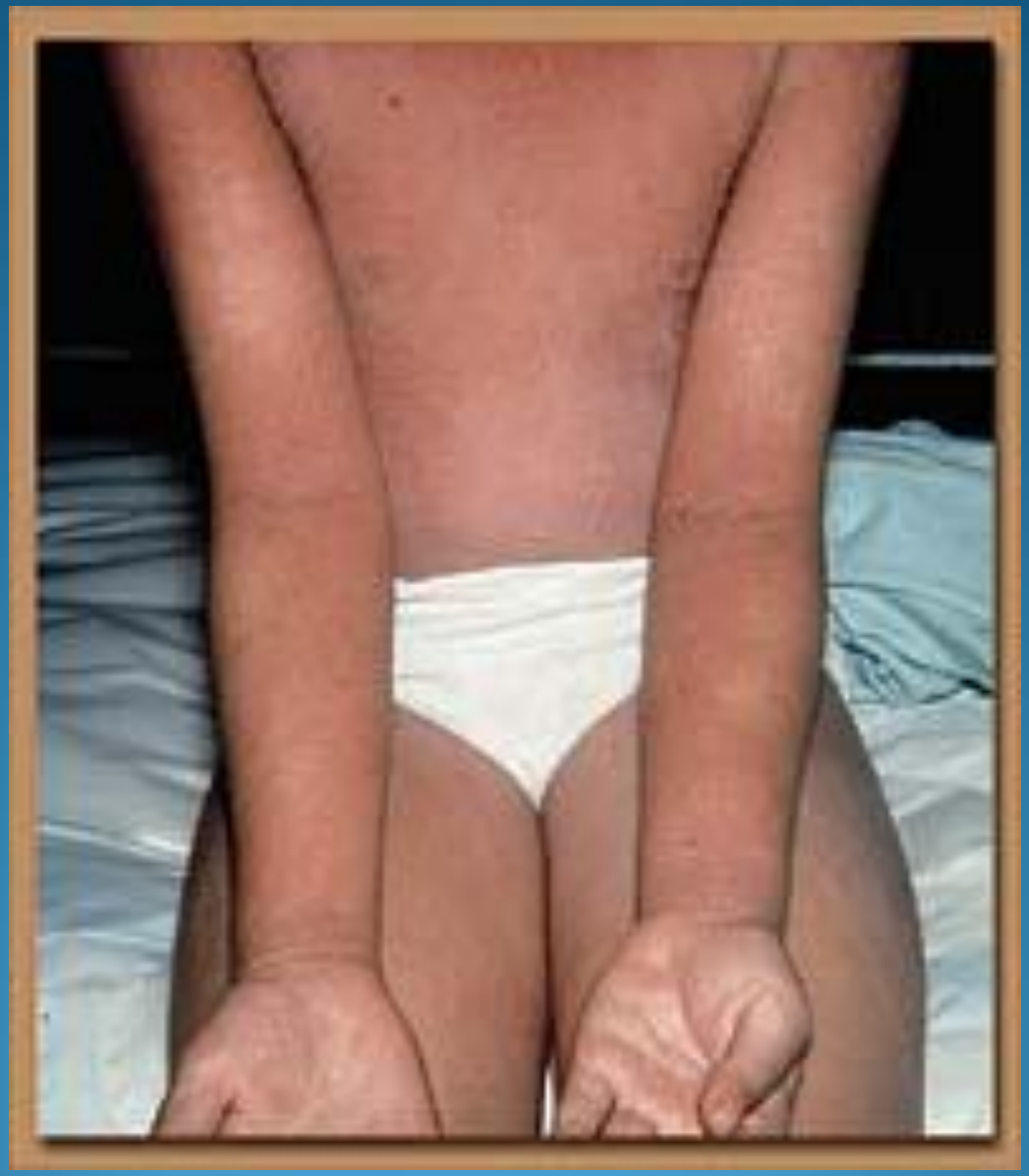
- Surgical wounds
- Postpartum infections
- Focal pyodermas
- Deep abscesses
- Nasal packing
- Tampons
- Insulin pump infusion sites
- Peritonsillar abscesses
- Sinusitis
- Osteomyelitis

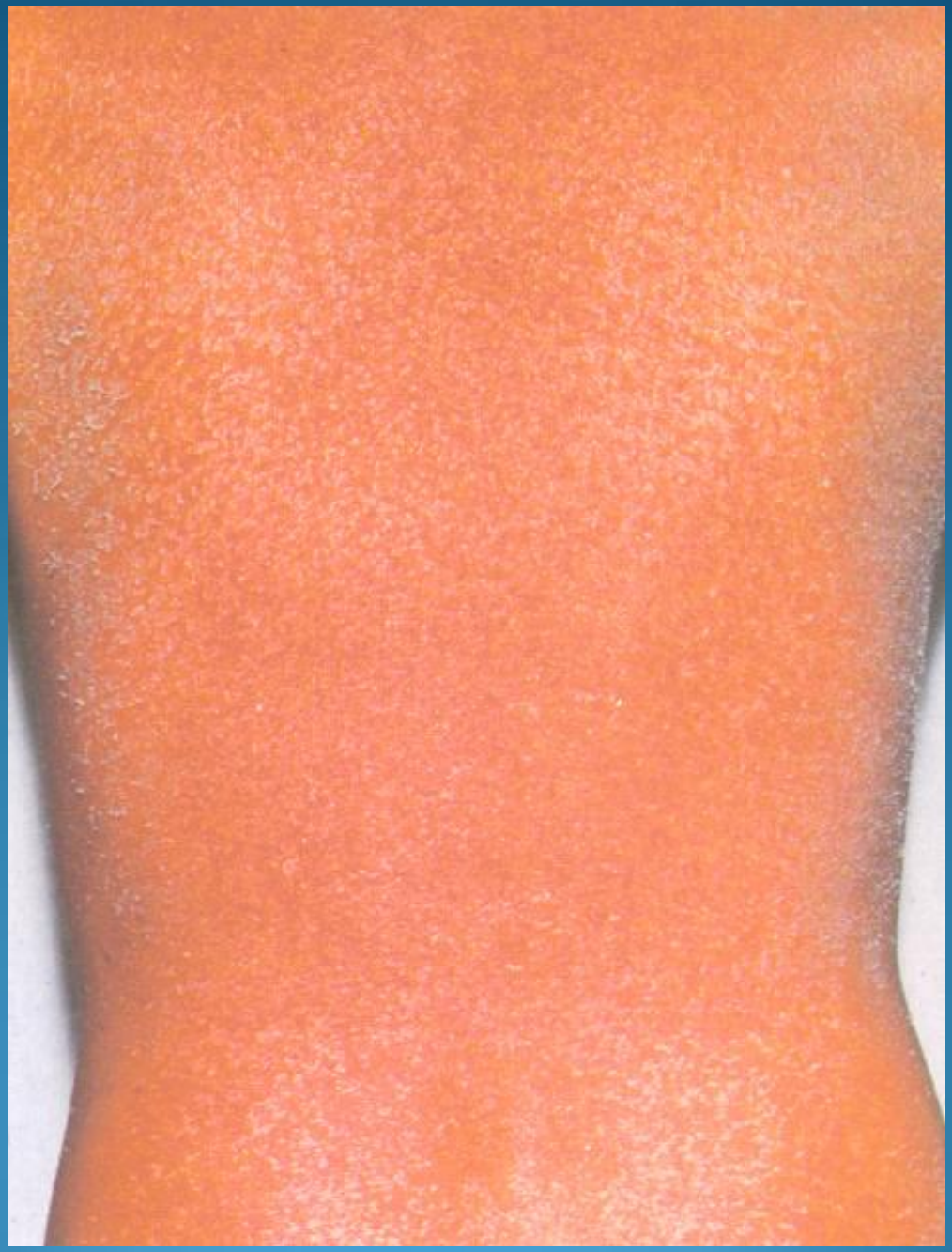
Staph Toxic Shock Syndrome: Clinical Features

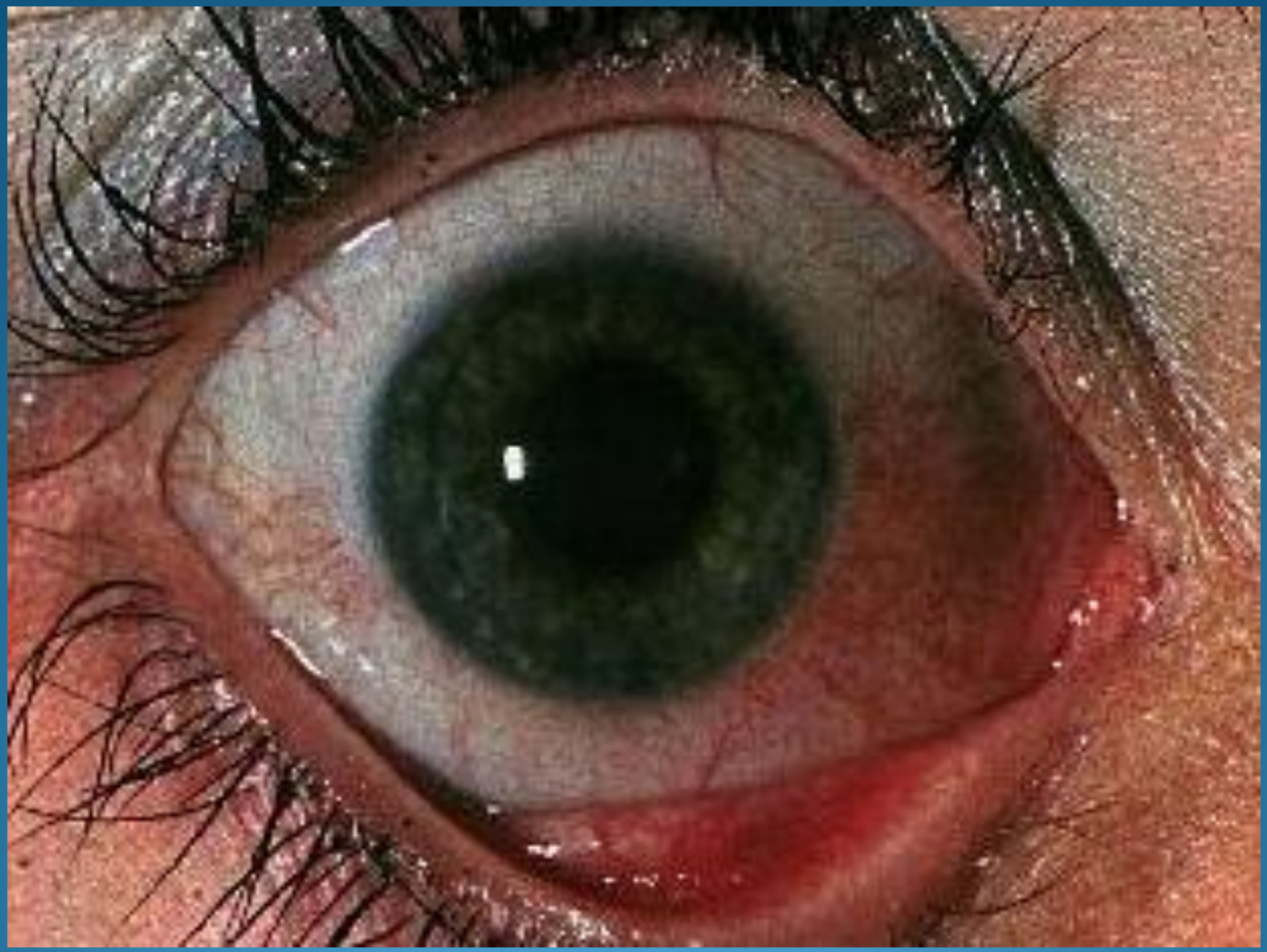
- Abrupt onset
  - F/N/V/D/HA, pharyngitis, myalgias
- Rapid progression to shock
- Spectrum: mild to rapidly fatal
- Skin findings are extensive and predictable

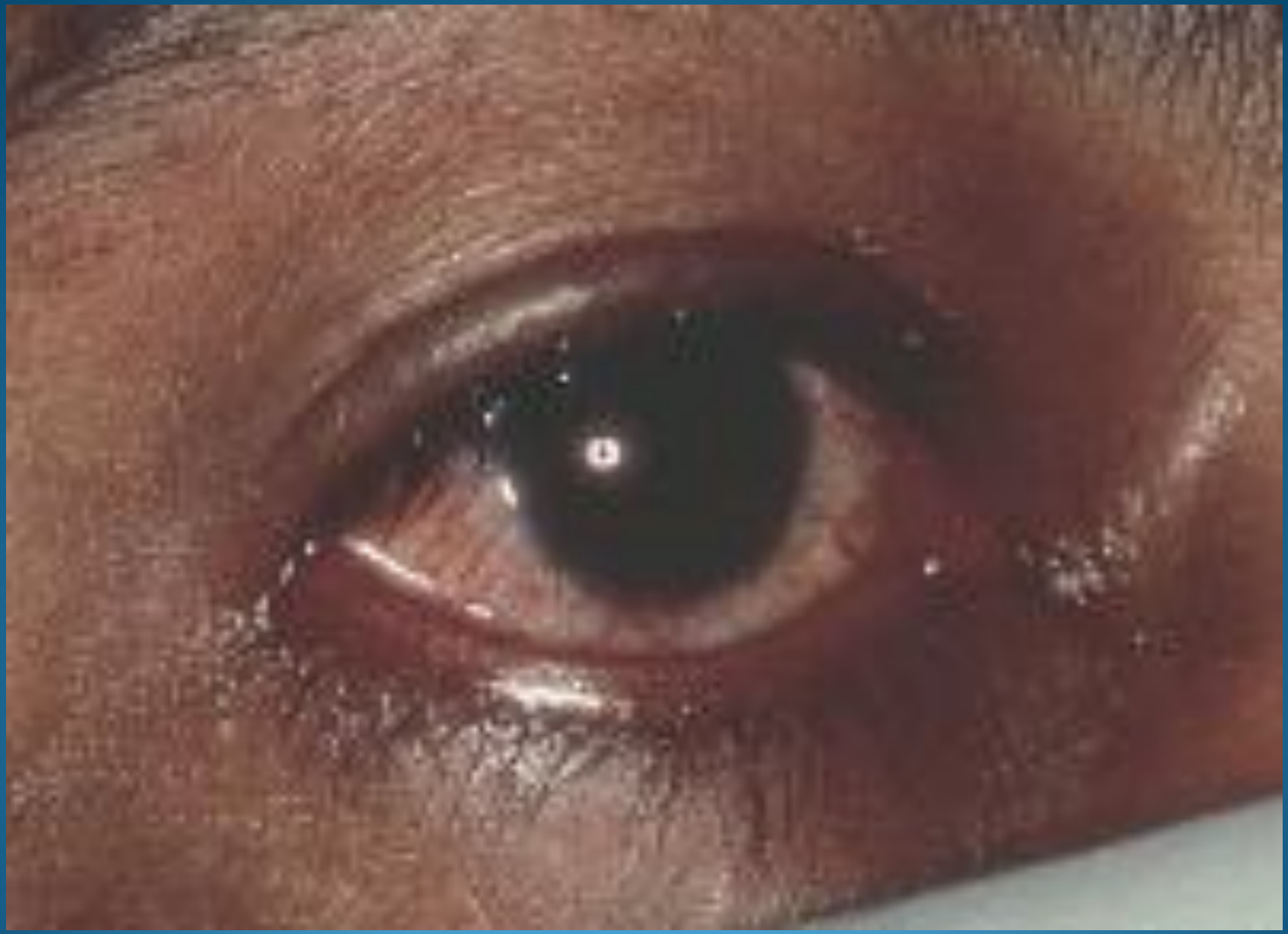
Staph TSS: Clinical Features

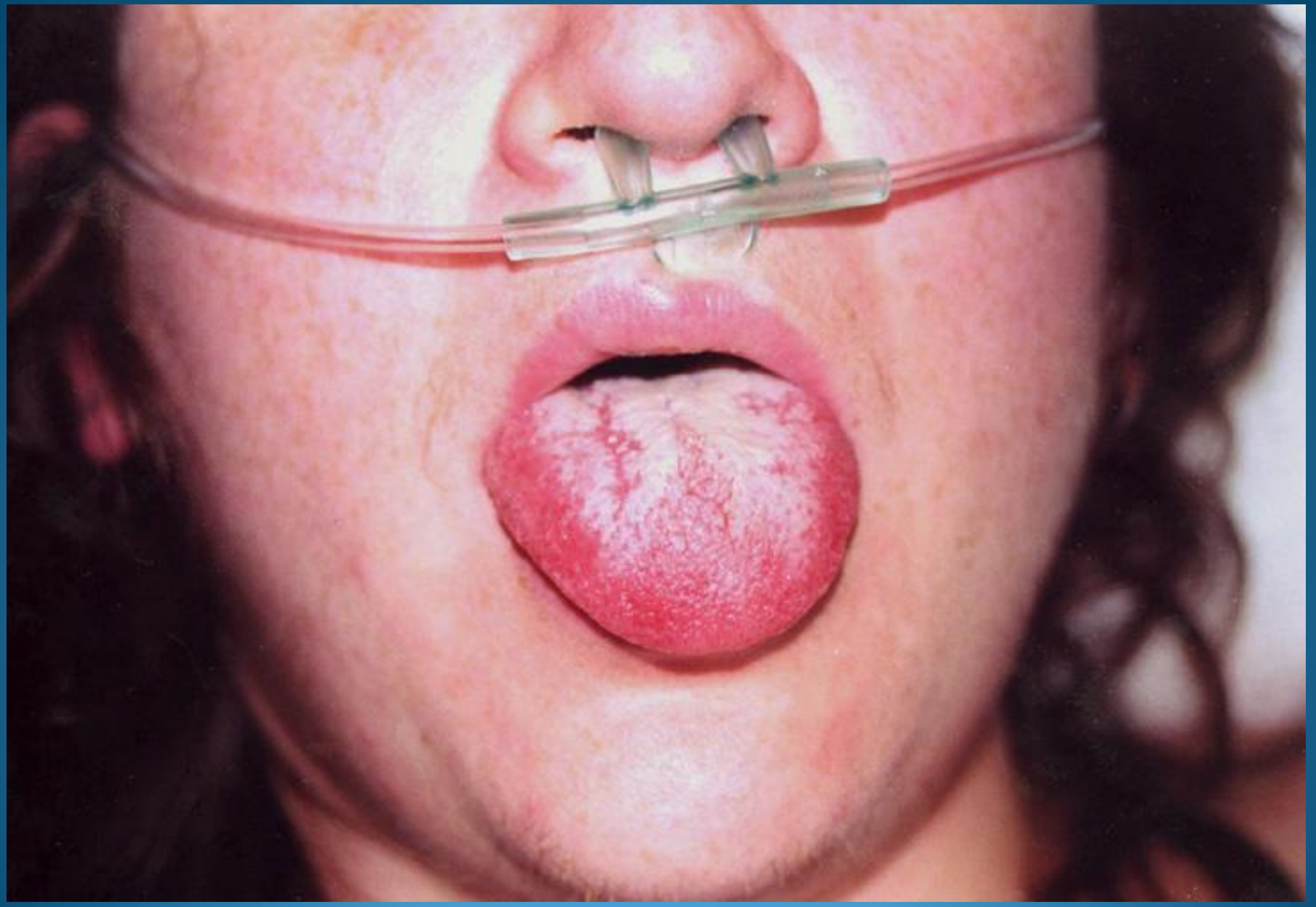
- Diffuse scarlatiniform exanthem
- Erythema and edema of the palms and soles
- Mucous membrane erythema
  - Strawberry tongue
  - Conjunctival hyperemia
- Generalized non-pitting edema
- Acral desquamation 1-3 weeks after onset

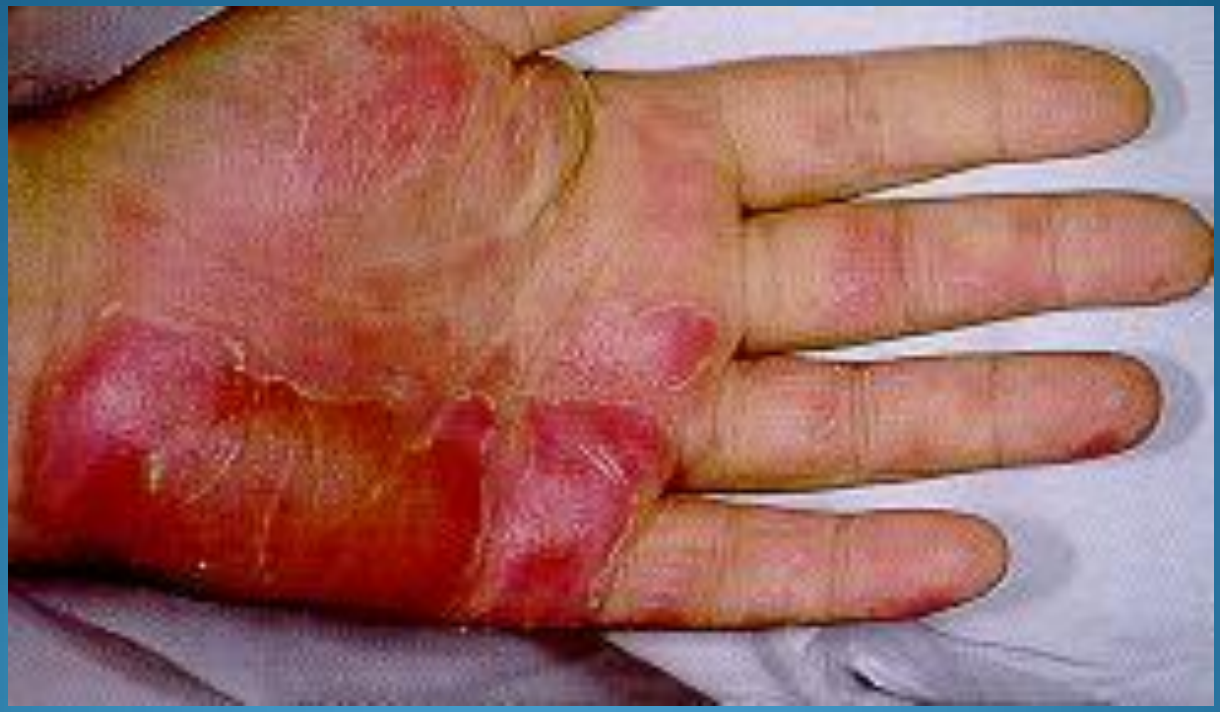


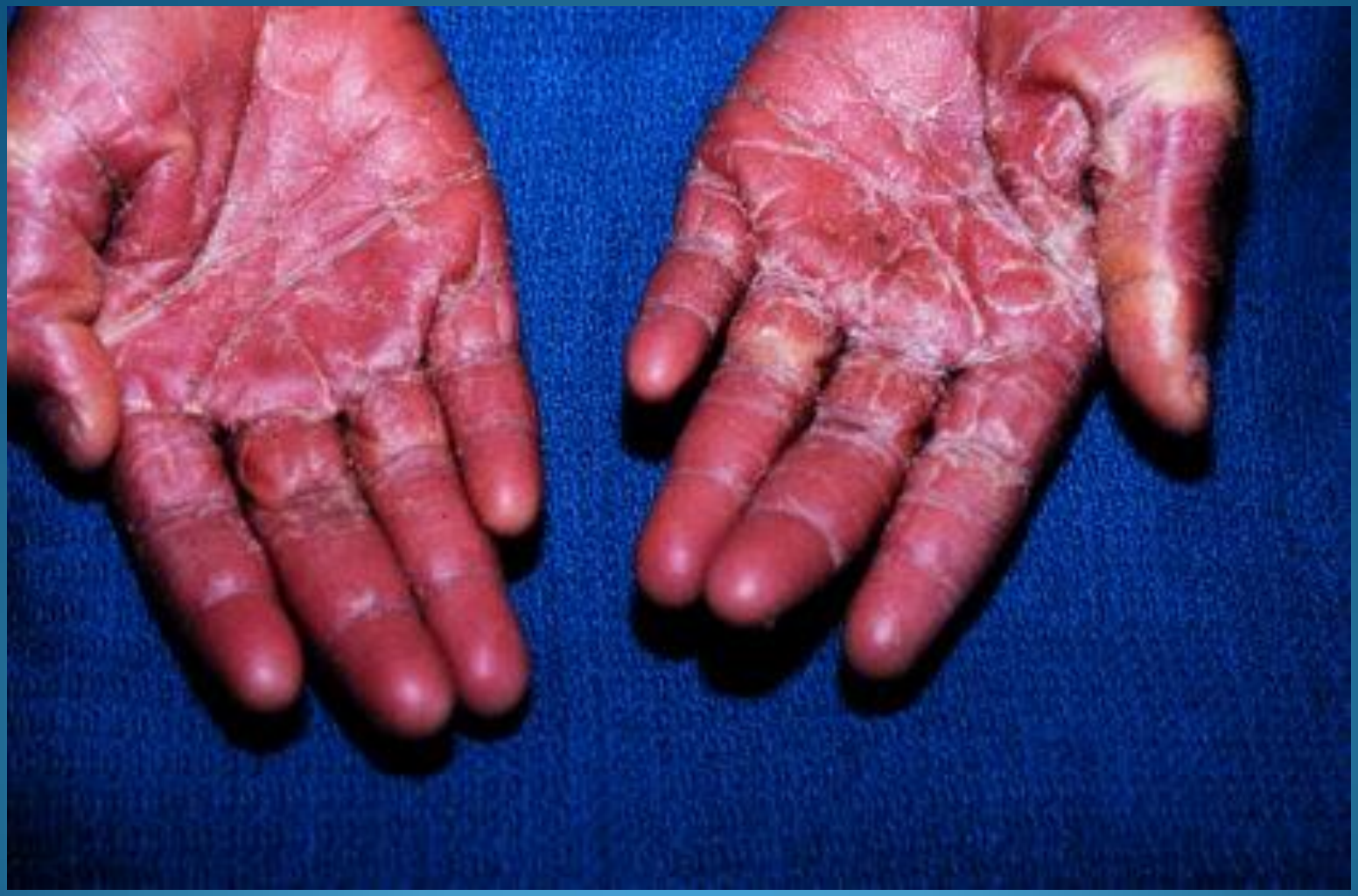












Dx: Staph Toxic Shock Syndrome

Centers for Disease Control and Prevention, 1990

- Fever: > 39.6 C / 102 F
- Rash: diffuse macular erythroderma
- Desquamation of palms/soles in 1-2 weeks
- Hypotension: SBP < 90 mmHg or < 5th %
- Involvement in 3 or more organ systems
- Absence of other causes

Rx: Staph Toxic Shock Syndrome

- Intensive supportive care
- Removal of nidus of infection
- Combination antibiotic therapy
  - eradicate Staph and suppress toxin production
- Surgical debridement
- IVIG (anecdotal; small series)
  - Demers, 1993; Stevens, 1998; Kaul, 1999

Rx: Staph Toxic Shock Syndrome

- IV β-lactamase resistant antimicrobial
  - nafcillin or oxacillin (2 g q4h)
  - 1st generation cephalosporins
  - vancomycin if penicillin allergic
- Add clindamycin to reduce toxin synthesis
- Continue antibiotics for 10-14 days
- Mortality: low (<3%)
- Recurrance in 20% -avoid triggers

THE TOXIC SHOCK SYNDROMES

	Staphylococcal	Streptococcal
Typical patient	Young (15–35 years) and healthy	Young (20–50 years) and healthy
Diffuse macular erythroderma	Very common	Less common
Vesicles and bullae	Rare	Uncommon (5%)
Localized extremity pain	Rare	Common
Soft tissue infection	Rare	Common
Hypotension	100%	100%
Renal failure	Common	Common
Predisposing factors	Surgical packing, surgical meshes, abscesses, contraceptive sponge, tampon*	Lacerations, bites, bruises, varicella
Positive blood cultures	<15%	>50%
Mortality	<3%	30–70%