NOTES
SKIN & SOFT TISSUE INFLAMMATION & INFECTIONS

GENERALLY, WHAT ARE THEY?

PATHOLOGY & CAUSES

- Inflammation of epidermis, dermis, underlying tissues

CAUSES
- Infections, autoimmune response

RISK FACTORS
- Impaired skin barrier
- Pressure
- Friction
- Exposure to infectious agents
- Immunosuppression

COMPLICATIONS
- Infection
  - Local or systemic disease

SIGNS & SYMPTOMS

- See individual disorders

DIAGNOSIS

LAB RESULTS
- Tissue cultures, Gram stain

OTHER DIAGNOSTICS
- Mostly clinical, based on presentation

TREATMENT

MEDICATIONS
- *Infections:* antimicrobials

OTHER INTERVENTIONS
- Dressings, ointments
- Cryotherapy
- Curettage

ACNE VULGARIS

osms.it/acne_vulgaris

PATHOLOGY & CAUSES

- Common inflammatory skin disorder affecting hair follicles, sebaceous glands
- May involve comedones, papules, pustules, cysts, scars
- Especially affects individuals who are
  - Biologically male, with hormonal disorders producing androgens (e.g. polycystic ovarian syndrome), adolescent
  - Symptoms decrease with age

TYPES

Mild

- Occasional, comedones, inflammatory papules, pustules

Moderate

- Multiple pustules, nodules occur on trunk

Severe

- Cystic, large nodules predominant, with persistent involvement of trunk; scarring

CAUSES

- Androgen stimulates sebaceous follicles to overproduce sebum → follicles become blocked
- Hyperkeratinization of epithelium → accumulation → follicular blocking → debris accumulates further → skin follicles rupture
- *Propionibacterium acnes* replicates within follicle → releases lipase → sebum converted to free fatty acids → release of cytokines → inflammation

RISK FACTORS

- Genetic predisposition
- Oil-based skin products
- Hormonal imbalance
- Infection
  - Follicle colonization by *Propionibacterium acnes*
- Medications
  - Lithium, glucocorticoids, anabolic steroids
- Polycystic ovarian syndrome
- Stress

COMPLICATIONS

- Cosmetic
  - Scars, hyperpigmentation, pyogenic granulomas, osteoma cutis
- Psychiatric
  - Low self esteem, depression

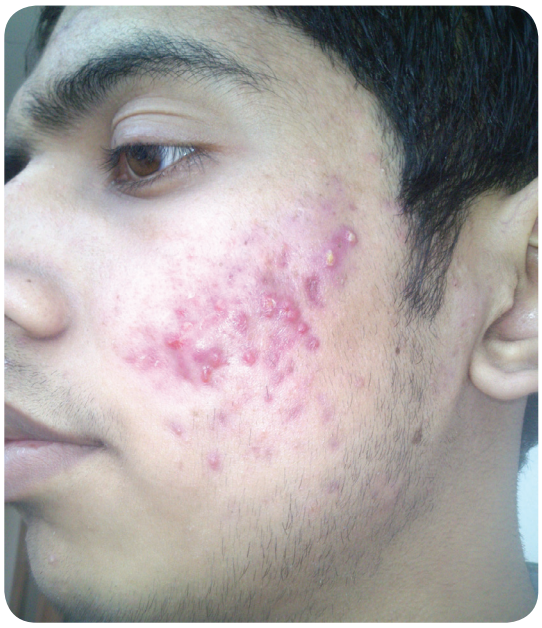

Figure 8.1 Acne vulgaris affecting the face of an adolescent male.

SIGNS & SYMPTOMS

- Papules, pustules, painful nodules (cysts) on face, neck, chest, back
- Closed comedones
- Erythema

DIAGNOSIS

OTHER DIAGNOSTICS

- Clinical presentation

TREATMENT

MEDICATIONS

- Target sebum production, inflammation, bacterial/follicular proliferation

Mild

- Benzoyl peroxide treats *Propionibacterium acnes*
- Add topical retinoids to prevent follicular proliferation
- In case of comedonal acne, use azelaic acid, salicylic acid as anti-inflammatory, antibacterial, antiproliferative agent

Mild to moderate

- Benzoyl peroxide, retinoid
- Add topical antibiotic (clindamycin) for synergistic effect with benzoyl peroxide

Severe

- Oral isotretinoin decreases sebum production, bacterial proliferation, inflammation
- Avoid using tetracycline due to sun sensitivity

OTHER INTERVENTIONS

- Clean skin with gentle agents
- Comedones extraction
- Pigmentation
  - Topical retinoid, azelaic acid, chemical peels
  - Photodynamic treatment with lasers (removes superficial layers of skin)
  - Dermabrasion (treatment of scars; may irritate skin)

CELLULITIS

osms.it/cellulitis

PATHOLOGY & CAUSES

- Non-necrotizing inflammation of dermis, subcutaneous tissue, typically caused by streptococci (*S. aureus*, *S. pyogenes*); usually unilateral
- *Skin breach:* trauma; dry, fissuring skin; ulcers → bacteria invade skin, subcutaneous tissue
- *Common locations:* face, legs; may affect arms
- *Other locations:* causes vary
  - *Orbital cellulitis:* originates from trauma, sinuses, hematogenous spread
  - *Abdominal wall cellulitis:* morbid obesity causes bacteria to enter skin sores
  - *Buccal cellulitis:* spread from tooth infection
  - *Perianal cellulitis:* affects all demographics

TYPES

- Purulent
  - Furuncles (inflamed follicles), carbuncles (accumulation of furuncles), abscesses, cysts
- Non-purulent
  - Superficial cellulitis, erysipelas

RISK FACTORS

- Skin inflammation
  - *Abrasion:* wounds, eczema, radiation, broken skin between toes
- Lowered immunity
  - Diabetes mellitus, alcohol abuse, HIV, older age
- Skin infection
  - Tinea, impetigo, varicella, rash
- Edema
  - Lymphatic obstruction, venous insufficiency
- Obesity

COMPLICATIONS

- Recurrence, abscess formation, necrotizing fasciitis, osteomyelitis, sepsis

SIGNS & SYMPTOMS

- Fever, chills
- Localized inflammation
  - Swelling
  - Warmth
  - Erythema with unclear borders (contrast to erysipelas—clear demarcations)
  - Often painful
- Enlarged lymph nodes
- Purulent cellulitis associated with *S. aureus* infection

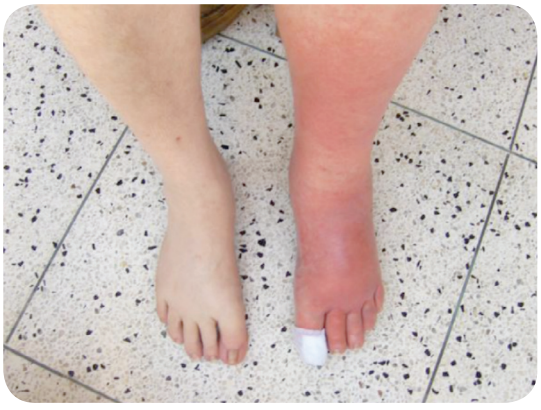

Figure 8.3 An individual with cellulitis of the left leg.

DIAGNOSIS

DIAGNOSTIC IMAGING

Ultrasound

- Subcutaneous fat separates into lobules
  - Cobblestone appearance

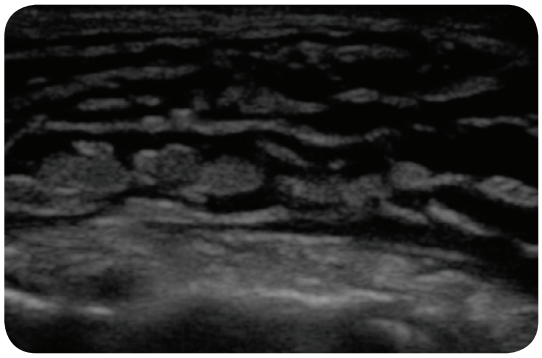

Figure 8.2 An ultrasound scan of the right lower limb demonstrating the cobblestone appearance of subcutaneous edema, in this case secondary to cellulitis.

LAB RESULTS

- Complete blood count (CBC)
  - ↑ *inflammatory markers:* ↑ C-reactive protein (CRP), ↑ erythrocyte sedimentation rate (ESR), ↑ WBC count
- Wound, blood cultures
  - Identify causative microbe

OTHER DIAGNOSTICS

- Clinical presentation
  - Spreading inflammation of skin/subcutaneous tissues

TREATMENT

MEDICATIONS

- *Antibiotics:* second generation penicillins, first generation cephalosporins; vancomycin for MRSA

OTHER INTERVENTIONS

- Immobilization, elevation, dressings
- Drain abscess

ERYSIPELAS

osms.it/erysipelas

PATHOLOGY & CAUSES

- Acute, non-necrotizing infection of upper dermis, superficial lymphatics; usually unilateral
- Well-defined demarcation between normal, infected tissue; non-purulent
- Usually caused by streptococci; most often *Streptococcus pyogenes*

RISK FACTORS

- Very young/old age
- Breaks in skin
  - Abrasions, trauma, eczema, radiation, bites
- Lowered immunity
  - Diabetes mellitus, alcohol abuse, HIV, older age
- Skin infection
  - Tinea, impetigo, varicella, rash
- Edema
  - Lymphatic obstruction, venous insufficiency
- Obesity

COMPLICATIONS

- Lymphedema due to impaired lymphatic drainage
- Necrosis
- If spread hematogenously to other areas
  - Arthritis, osteomyelitis, necrotizing fasciitis, glomerulonephritis

SIGNS & SYMPTOMS

- Initially, general infection symptoms
  - Fever, chills, headache, fatigue
- Lesions
  - Mostly on legs, but may be found on face, arms, fingers, toes
- Elevated, warm, painful rash called "forest fire rash" (because it's reddest at border)
- Vesicles may be present; may be bright, salmon red
- Inflammation of regional lymph nodes, lymphangitis in chronic infection

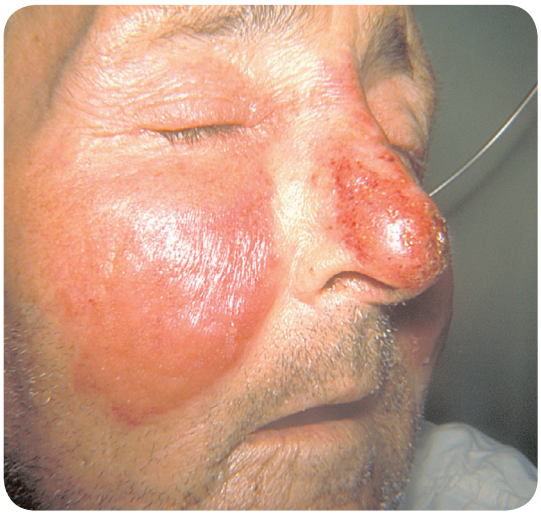

Figure 8.4 Erysipelas affecting the face of an elderly individual.

DIAGNOSIS

LAB RESULTS

- Blood test
  - CBC: ↑ CRP, ↑ ESR, ↑ WBCs; antistreptolysin titer O shows streptococcal involvement
- Wound, blood culture

OTHER DIAGNOSTICS

- Clinical presentation

TREATMENT

MEDICATIONS

- *Antibiotics:* oral penicillins/macrolides, vancomycin for MRSA, intravenous (IV) route in severe infection

FOLLICULITIS

osms.it/folliculitis

PATHOLOGY & CAUSES

- Hair follicle inflammation (pyoderma), usually infectious cause
- May also be due to persistent trauma (mechanical folliculitis)
- Pathogen enters hair follicle → inflammatory inflammatory response → infection causes a perifollicular infiltrate of lymphocytes, neutrophils, macrophages → pustule formation

CAUSES

- Bacterial
  - *S. aureus*, *Pseudomonas aeruginosa* (hot-tub folliculitis)
- Viral
  - Herpes simplex virus (HSV)
- Fungal
  - Tinea barbae
- Rarely
  - Autoimmune; oily skin in factory workers

RISK FACTORS

- Swimming pools, hot tubs
- Shaving against hair growth, tight clothes causing friction, profuse sweating (hyperhidrosis)
- Use of antibiotics, acne medication, topical corticosteroids
- Upper respiratory presence of *S. aureus*

COMPLICATIONS

- Recurrence
- Furunculosis
  - Deep infection of hair follicle → evolves into swollen nodule, may coalesce into carbuncles

SIGNS & SYMPTOMS

- Many small pustules, papules in areas of hair growth (e.g. face, legs, arms, back)
  - Typical in groin, armpits
  - Gram-negative infections more common on the face (areas of acne)
  - Methicillin resistant *S. aureus* (MRSA) more common on the front trunk
- Itching, redness; often tender
- Does not appear in areas without hair growth (palms of hands, soles of feet)
- Sycosis vulgaris
  - Multiple pustules on chin, upper lip; caused by S. aureus infection after shaving

DIAGNOSIS

LAB RESULTS

- Gram stain, wound culture performed for treatment-resistant individuals
- Skin biopsy
  - Differentiation from other skin disorders in persistent folliculitis

OTHER DIAGNOSTICS

- History
  - Risk behavior/predisposition
- Clinical presentation

TREATMENT

MEDICATIONS

- *Topical antibiotics:* mupirocin, clindamycin
- *Oral antibiotics:* tetracycline, cephalosporin
  - Used in extensive involvement
- *MRSA treatment:* trimethoprim/sulfamethoxazole, clindamycin, tetracycline
- *Fungal treatment:* fluconazole, itraconazole
- *Viral treatment:* acyclovir, valacyclovir, famciclovir

OTHER INTERVENTIONS

- May resolve spontaneously
- Warm compress with antiseptic use
- Loose fitting clothing; avoiding shaving

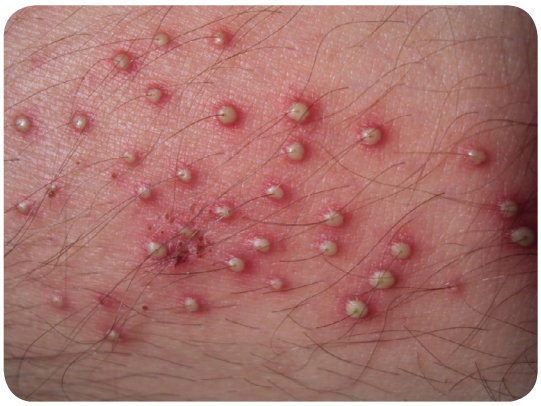

Figure 8.5 The clinical appearance of folliculitis.

HIDRADENITIS SUPPURATIVA

osms.it/hidradenitis-suppurativa

PATHOLOGY & CAUSES

- Chronic, pus-producing dermatological disorder
  - AKA acne inversa
- Dysfunctional hair follicles/apocrine sweat glands → pore clogging → inflammation → painful abscesses

CAUSES

Environmental

- Skin/clothes friction, hormonal changes, sweating, humidity

Genetic

- Apocrine gland dysfunction, cellular disorders

RISK FACTORS

- Obesity, tight clothes, smoking, deodorant use, shaving
- More common for biologically-female individuals

COMPLICATIONS

- Scarring, bacterial infection, interstitial keratitis, sinus formation, fistula formation; squamous cell carcinoma (chronic lesions); depression

SIGNS & SYMPTOMS

- Red inflamed areas, painful bumps that drain with pus
- Presence of double comedones
- Mostly in axilla, groin, under breasts

DIAGNOSIS

OTHER DIAGNOSTICS

Clinical presentation

- Sartorius/Hurley's staging systems (determines severity; guides treatment)
  - *Stage I:* solitary/multiple isolated abscess formation without scarring/sinus tracts
  - *Stage II:* recurrent abscesses; lesions may be single/multiple, widely separated; sinus tract formation
  - *Stage III:* diffuse, regional involvement across; multiple interconnected sinus tracts, abscesses

TREATMENT

MEDICATIONS

- Corticosteroids, anti-androgen medication, oral antibiotics, tumor necrosis factor (TNF) inhibitors to ↓ inflammation

SURGERY

- Incision, drainage; sinus tract opening

OTHER INTERVENTIONS

- Clean affected area
- Laser treatments remove lesions, scarring

Behavioral

- Smoking cessation, weight loss

IMPETIGO

osms.it/impetigo

PATHOLOGY & CAUSES

- Highly infectious skin infection; affects superficial epidermis
  - Commonly affects children
  - Skin-to-skin spread possible
- Contact with carrier → pathogen enters intact/non-intact skin → incubation → lesion formation, spread over body through scratching
- Commonly caused by *S. aureus*, *S. pyogenes*

TYPES

- Nonbullous
- Bullous
- Ecthyma

RISK FACTORS

- Higher incidence in warm, humid climates
- Eczema, HSV, diabetes mellitus, malnutrition
- School, daycare

COMPLICATIONS

- Cellulitis, poststreptococcal glomerulonephritis

SIGNS & SYMPTOMS

- Nonbullous
  - Most common
  - Red bump → blister → blisters rupture, ooze, form crusts → characteristic yellow scab formation
- Bullous
  - Bullae on limbs, trunk
  - Not painful
  - Ruptured bullae become covered with thin, brown crust
- Ecthyma
  - Deeper nonbullous impetigo appears on limbs
  - Painful
  - Evolves into yellow scabs
- Fever (rare); blisters may be painful, itchy

DIAGNOSIS

LAB RESULTS

- Lesion culture
  - Identify pathogen, adjust treatment

OTHER DIAGNOSTICS

- History
- Physical exam

TREATMENT

MEDICATIONS

- Topical antibiotic
- Penicillins
- In case of MRSA, use trimethoprim/sulfamethoxazole

OTHER INTERVENTIONS

- Clean with antiseptic to prevent spreading
- Topical antibiotic
  - Penicillins
  - In case of MRSA use trimethoprim/sulfamethoxazole

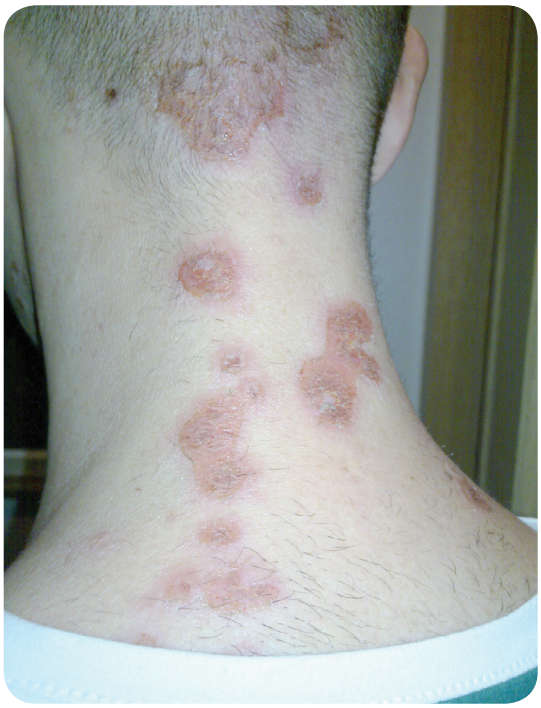

Figure 8.6 Impetigo on the back of the neck of an adult male.

NECROTIZING FASCIITIS

osms.it/necrotizing_fasciitis

PATHOLOGY & CAUSES

- Potentially life-threatening infection
  - Progressive destruction of deep soft tissue (subcutaneous fat, muscle fascia)
- Bacteria spread via subcutaneous tissue → release exotoxins → tissue destruction spreads along fascial planes

TYPES

Type I: polymicrobial

- *Causes:* combination of aerobic, anaerobic bacteria
  - *Most common anaerobes:* *Bacteroides, Clostridium, Peptostreptococcus*
  - *Enterobacteriaceae:* *Escherichia coli, Klebsiella, Proteus, Enterobacter*
  - Facultative anaerobic streptococci
- Common sites
  - *Perineum (Fournier's gangrene):* impaired gastrointestinal/urethral mucosal integrity → spreads to anterior abdominal wall; gluteal muscles; scrotum, penis (in biological male); labia (in biological female)
  - *Cervical (head, neck):* impaired oropharynx mucosa (often related to dental/odontogenic infection) → spreads to face, neck, mediastinum

Type II: monomicrobial

- *Causes:* Group A *Streptococcus*, other beta-hemolytic streptococci, *Staphylococcus aureus*

Type III: saltwater infection

- *Cause:* *Vibrio vulnificus*
- Rare

Type IV: fungal infection

RISK FACTORS

- Immunosuppression
  - HIV, diabetes, cirrhosis, corticosteroid use
- Peripheral vascular disease
- Trauma
  - Injury, surgery
- IV drug abuse
- Childbirth
- Exposure of open wound to fresh/salt water, swimming pools, hot tubs
- Ludwig's angina (submandibular region infection)
- Lemierre syndrome (septic thrombophlebitis located in jugular vein)

COMPLICATIONS

- Shock
- Organ failure
- Potentially fatal

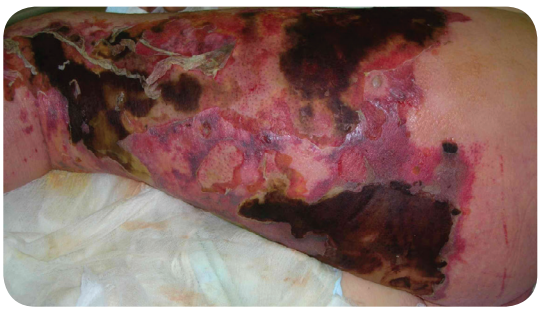

Figure 8.7 Necrotizing fasciitis on the left leg. The dark areas represent progression to necrosis.

SIGNS & SYMPTOMS

Overlying skin

- May appear normal initially
- Later
  - Warmth, erythema/violet/purple (violaceous), woody induration, edema, necrosis
- Bullae; may fill with serosanguinous fluid
- Subcutaneous emphysema (if infection with anaerobes)

Systemic findings

- Pain
  - Often out of proportion to exam findings
- Fever
- ↑ pulse
- ↓ perfusion (motling, pallor, altered level of consciousness)
- Hemodynamic instability (↓ BP)

DIAGNOSIS

DIAGNOSTIC IMAGING

CT scan

- Subcutaneous gas visualized in fascial planes

LAB RESULTS

Blood

- ↑ WBCs, left shift; ↑ creatine phosphokinase; ↓ hemoglobin, ↑ glucose, ↓ sodium

Urine

- Proteinuria

Gram stain, cultures of skin

- Debrided tissue identifies organism(s)

OTHER DIAGNOSTICS

- Laboratory Risk Indicator for Necrotizing Fasciitis (LRINEC) score
  - *Score = 6:* ↑ suspicion of necrotizing fasciitis
  - *Score = 8:* strongly predictive

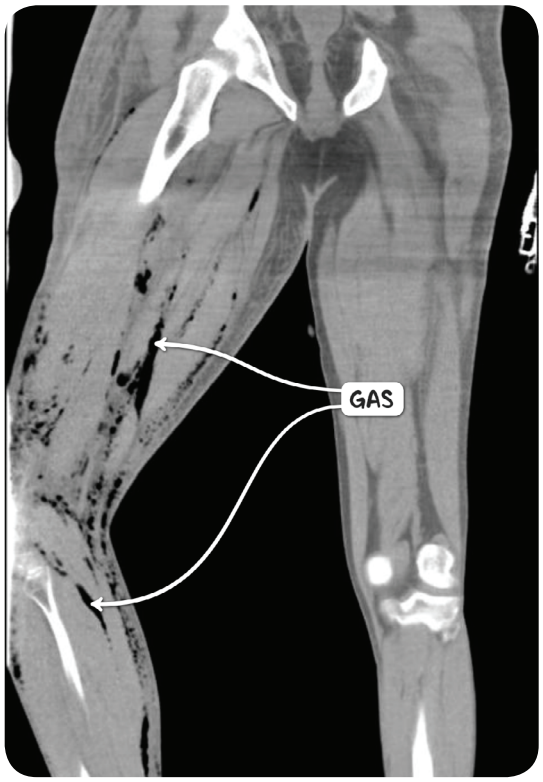

Figure 8.8 A CT scan in the coronal plane demonstrating the presence of gas in the fascial planes of the leg, consistent with a diagnosis of necrotizing fasciitis.

LRINEC

TEST	RESULT	SCORE
C-REACTIVE PROTEIN (mg/L)	< 15 ≥ 15	0 4
WBCs (mm^3)	< 15 15-25 > 25	0 1 2
HEMOGLOBIN (g/dL)	> 13.5 11-13.5 < 11	0 1 2
SODIUM (mEq/L)	≥ 135 < 135	0 2
CREATININE (mg/dL)	≤ 1.6 > 1.6	0 2
GLUCOSE (mg/dL)	≤ 180 > 180	0 1

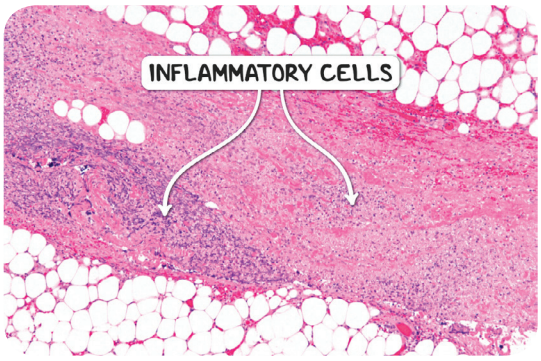

Figure 8.9 A histological section of subcutaneous tissue in a case of necrotizing fasciitis showing an inflammatory infiltrate in the fascia leading to necrosis.

TREATMENT

MEDICATIONS

- Empiric IV antibiotics
  - Carbapenem/beta-lactam-beta-lactamase inhibitor + vancomycin/linezolid + clindamycin
- Hemodynamic support
  - Fluids, vasopressors

SURGERY

- Direct surgical examination of skin, subcutaneous tissue, fascial planes, muscle → debridement of all devitalized, necrotic tissue
- Fasciotomy

OTHER INTERVENTIONS

- Hyperbaric oxygen

ONYCHOMYCOSIS

osms.it/onychomycosis

PATHOLOGY & CAUSES

- Chronic fungal infection
  - Nail bed, matrix, plate of toes/fingers
- Infection spread by direct contact from people, animals, soil, fomites (upholstery, hairbrushes, hats)
- Causative agents
  - *Dermatophytes (tinea unguium):* most commonly *Trichophyton rubrum*
  - *Nondermatophyte molds:* *Aspergillus* spp.
  - *Yeasts:* most commonly *Candida albicans*
- Dermatophyte hyphae penetrate stratum corneum of skin, nails → manufacture keratinolytic proteases → invade living cells
- Spores of nondermatophyte (e.g. *Aspergillus*) lodge under nail/at lateral nail folds → colonization, spread toward cuticle *Candida spp.* → infect soft tissue around nail, penetrate nail plate

TYPES

Distal lateral subungual

- Initially affects distal corner of nail; eventually spreads toward cuticle
- Most common

Proximal subungual

- Affects nail plate near cuticle; extends distally
- Sign of severely immunocompromised state

White superficial

- Affects nail surface; may spread to cover entire nail

Endonyx

- Affects interior of nail plate

Total dystrophic

- Nail plate is completely destroyed

Mixed pattern

- Combination of other types

RISK FACTORS

- ↑ age
- Biological male > female
- Communal showers, swimming pools
- Contributory/predisposing factors
- ↑ warmth, humidity
- Occlusive footwear
- Occupational
  - Jobs that involve frequent hand washing; dishwashers, housekeepers
- Immunocompromised state (HIV, diabetes)
- Living with others affected by onychomycosis
- Chronic mucocutaneous fungal infection

COMPLICATIONS

- Pain
- ↑ risk of bacterial coinfection, cellulitis
- Nail disfigurement
- Recurrence

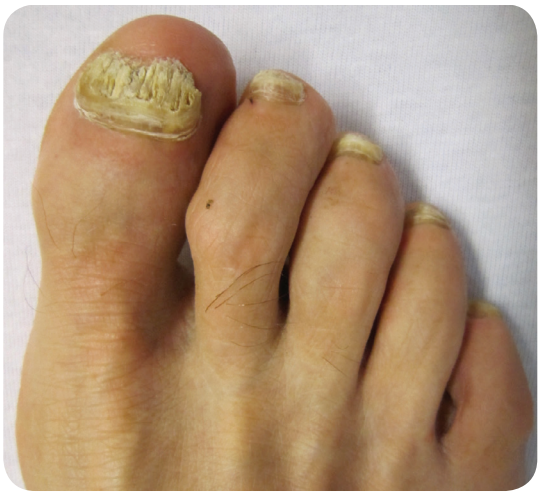

Figure 8.10 Onychomycosis of the toe nails.

SIGNS & SYMPTOMS

- Distal lateral subungual
  - Yellow, brown, white discoloration; subungual hyperkeratosis; mild inflammation; onycholysis
- Proximal subungual
  - Diffuse patches/transverse striate of white to yellow patches on nail plate
- Endonyx
  - Discoloration, onycholysis
- White superficial
  - Soft white spots on nail surface
- Total dystrophic
  - Keratotic debris on thick, rigid nail bed
- Other associated features
  - Coexisting tinea pedis infection (common)
  - Chronic paronychia (proximal/lateral fold inflammation)
  - Dermatophytoma (linear, yellow/white band of dermatophyte hyphae)
  - Fungal melanonychia (black/brown discoloration; caused by pigment-producing molds, fungi)

DIAGNOSIS

LAB RESULTS

- Potassium hydroxide (KOH) microscopy
  - Identifies fungal elements (e.g. fungal hyphae, pseudohyphae, yeast)
- Histopathological analysis using periodic acid-Schiff (PAS) stain
  - Identifies fungal elements
- Fungal culture (e.g. dermatophyte test medium/DTM)
  - Identifies organism

OTHER DIAGNOSTICS

- History, physical examination with characteristic findings

TREATMENT

MEDICATIONS

- Topical triazole
  - Efinaconazole
- Systemic
  - Terbinafine (dermatophyte infections); itraconazole (yeast, non-dermatophyte infections)
- Coexisting tinea capitis
  - Griseofulvin
- Keratolytic (urea)
  - Reduces nail thickening

SURGERY

- Nail removal in some cases (nail avulsion)

OTHER INTERVENTIONS

- Laser (Nd:YAG)
- Photodynamic therapy

PRESSURE ULCER

osms.it/pressure-ulcer

PATHOLOGY & CAUSES

- Localized skin, underlying-tissue injury
  - Caused by unrelieved pressure/pressure in combination with friction, shearing forces
- AKA bedsores/decubitus
- Blood flow diminishes
  - Pressure → ischemia → necrosis
- Bony prominences most commonly affected
  - Sacrum, heels, hips, elbows

RISK FACTORS

- Reduced mobility
  - Chronic/acute disease (e.g. hip fracture, stroke, Parkinson disease)
  - Central/peripheral neural damage, altered level of consciousness, advanced age
- Reduced perfusion
  - Atherosclerosis, peripheral vascular disease, hypotension, smoking
- Factors affecting skin structure
  - Malnutrition, protein deficiency, skin moisture (incontinence, sweating)
- Diabetes mellitus

STAGING

- *Stage I:* nonblanchable erythema; skin intact, localized
- *Stage II:* partial thickness dermis loss; red wound bed; serum-filled blister; no skin sloughing
- *Stage III:* full thickness tissue loss; visible subcutaneous fat; raised wound edges (epibole); skin sloughs
- *Stage IV:* full thickness tissue loss; bone, muscle, tendon exposed; raised wound edges; skin sloughs/eschar formation
- *Unstageable:* if filled with sloughed skin, scabs; diagnosis difficult
- *Deep tissue injury:* nonblanchable erythema, skin separation; no skin disruption

COMPLICATIONS

- Biofilm formation on wound → inflammation → delayed healing → wound dehiscence
- Wound, bone, joint infection; sepsis; fistulas; gangrene
- Malignant transformation rare

SIGNS & SYMPTOMS

- Ulceration (skin in contact with underlying surface)
- Fever, foul odor (if complicated by infection)
- May be painful

DIAGNOSIS

LAB RESULTS

- Swab culture
  - May help determine treatment in healing-resistant ulcers

OTHER DIAGNOSTICS

- Clinical presentation

TREATMENT

MEDICATIONS

- Topical sulfadiazine cream

OTHER INTERVENTIONS

- Debridement of biofilm, dressing replacement, negative pressure therapy

Prevention

- If bedridden, reposition at least every two hours (reduces chance of ulcer development)
- Use of special mattresses

ROSACEA

osms.it/rosacea

PATHOLOGY & CAUSES

- Chronic inflammatory cutaneous disorder
  - Usually affects face (may extend to neck, upper chest, ears)
  - *Ocular involvement:* dry, burning, itching, foreign-body sensation
- *Typical onset:* 30–50 years
- Defining features
  - Persistent central facial erythema; intensity may be intermittent
  - Phymatous changes (irregular, nodular skin), usually affecting nose (biologically-male > biologically-female individuals)
- Hyperactive vascular response, may extend to eyes (ocular rosacea)
- Triggered by warm weather, alcohol, certain foods, sun, stress

CAUSES

- Unknown
- May be innate immune system dysfunction; response to bacteria, UV → chronic inflammation

COMPLICATIONS

- Skin thickening, scarring, rhinophyma, ocular rosacea (blepharitis)

RISK FACTORS

- Genetic predisposition
- More common in biologically-female individuals, especially of Celtic/Northern-European descent

SIGNS & SYMPTOMS

- Telangiectasia with erythema, papules, pustules
- Flushing

Characteristics of different types

- Papulopustular
  - Similar to acne, no comedones
- Phymatous
  - Thick oily skin; mostly on nose, in biologically-male individuals
- Ocular (common)
  - Conjunctivitis, keratitis, tearing, burning, telangiectasias
- Granulomatous
  - Papules around eyes, cheeks
- Pediatric (rare)
  - Never phymatous
- Neurogenic
  - Pain, neurologic symptoms

DIAGNOSIS

OTHER DIAGNOSTICS

- History; clinical exam with characteristic findings

TREATMENT

MEDICATIONS

- Antibiotic (topical metronidazole)
- Topical azelaic acid
- Oral doxycycline

OTHER INTERVENTIONS

- Avoid exacerbating factors (e.g. spicy food, alcohol, sun, stress)
- Regular sunscreen use
- Laser therapy
  - Telangiectasia ablation

STAPHYLOCOCCAL SCALDED SKIN SYNDROME (SSS)

osms.it/SSS

PATHOLOGY & CAUSES

- Infectious, superficial skin disorder
  - Skin blistering, desquamation
- AKA Ritter's disease

CAUSES

- *S. aureus* produces epidermolytic exotoxin → enters skin → breaks down desmosomes between cells → peeling skin

COMPLICATIONS

- Cellulitis, sepsis

RISK FACTORS

- *S. aureus* infection
- Immunocompromised state
- Immature renal function/kidney disease
- Affects children < six years

SIGNS & SYMPTOMS

- Tender erythema with large desquamation areas, moist patches
- Nikolsky sign: skin peels at gentle touch
- Fever, malaise, appetite loss

DIAGNOSIS

DIAGNOSTIC IMAGING

Chest X-ray

- Check for pneumonia as infectious cause; lobar infiltrates

LAB RESULTS

Biopsy

- Epidermal splitting in stratum granulosum near skin surface

Blood culture

- *S. aureus*

OTHER DIAGNOSTICS

- Clinical presentation

TREATMENT

MEDICATIONS

- Antibiotics