

CHAPTER 12

Disorders of the Skin

Acne Vulgaris

A polymorphic skin disorder characterized by comedones, papules, and pustules. Acne vulgaris may be exacerbated by steroids and anticonvulsants; food has not been demonstrated to be a contributing factor.

Etiology and Demographics

- Precise cause unknown
- Pathogenic events:
 - Plugging of the infundibulum of the follicles
 - Retention of sebum
 - Overgrowth of *Propionibacterium acnes*
 - Release of fatty acids
- More common and severe in males overall
- More common in women between ages 25–44
- Most common of all skin conditions

Risk Factors

- Genetic predisposition
- Male
- Conditions characterized by hyperandrogenism
- Anticonvulsants
- Systemic or topical steroid use on the face

Signs and Symptoms

- Comedones are hallmark sign
- Closed comedones—tiny, flesh colored noninflamed bumps
- Open comedones—“blackheads”

- Papules, pustules, and/or cysts on face and or upper trunk
- Perioral dermatitis—papular lesions on the chin and around the mouth—most common in women aged 30–50
- Mild soreness, pain, or itching
- Depressed or hypertrophic scars
- Occasionally mild discomfort from swollen lesion
- In women, may exacerbate just prior to menses

Differential Diagnosis

- Acne rosacea
- Folliculitis

Diagnostic Studies

- Typically a clinical diagnosis
- Bacterial culture may be indicated to rule out folliculitis
- Serologic testing as indicated when systemic causes suspected:
 - Hyperandrogen states
 - HIV (sudden onset widespread folliculitis)

Treatment

- Patient education:
 - Avoid picking the lesions
 - Improvement not generally seen for at least 4 weeks
 - Avoid topical exposure to oils, make-up
 - Clean face and affected areas before bed
- Comedonal acne (mild acne):
 - Topical retinoids—titrate to use nightly
 - Topical benzoyl peroxide as needed
 - Topical antibiotics—clindamycin, erythromycin
- Papular inflammatory acne (moderate acne):
 - Antibiotics are the mainstay of treatment for mild and moderate forms
 - Mild forms typically respond to a regimen of topical antibiotics and topical inoids
 - Moderate forms require oral antibiotics—choice of antibiotic depends primarily upon issues in cost, compliance, and tolerance:
 - Tetracycline and erythromycin bid typically first choice
 - Minocycline bid in those who do not respond to tetracycline or erythromycin—more expensive, commonly causes vertigo when initiated
 - Severe forms typically managed with isotretinoin (Accutane):
 - Absolutely contraindicated in pregnancy—two negative pregnancy tests should be obtained prior to use and assess monthly for duration of therapy
 - Two forms of contraception should be used

- Informed consent is required
- Liver function tests should be monitored
- Adverse effects include:
 - Depression
 - Extreme drying of mucus membranes
 - Decreased night vision
 - Bowel symptoms
 - Myalgias
 - Hypertriglyceridemia and hypercholesterolemia
- Acne does not usually recur after a course of isotretinoin—cases that do usually respond to second course
- Be alert to psychosocial issues related to acne.
- Acne managed with antibiotics typically resumes when therapy completely discontinued—successful response to therapy allows for titration of antibiotics to maintenance doses.

Acne Rosacea

A chronic facial disorder characterized by acneform inflammation of the central third of the face and characterized by four subtypes; erythematotelangiectatic type, papulopustular rosacea, phymatous rosacea, and ocular rosacea.

Etiology and Demographics

- Cause is unknown
- Occurs primarily in middle-aged and older patients

Risk Factors

- Fair skin
- Family history
- More common in those with migraine headache

Signs and Symptoms

- Rosy coloration to nose, chin, cheeks—at time involves the whole face
- Papules and pustules
- No comedones
- Occasional burning with flushing
- Telangiectasias
- Comorbid ophthalmic disease:
 - Blepharitis
 - Keratitis
- Rhinophyma—more common in men

Differential Diagnosis

- Acne vulgaris
- Folliculitis
- Cellulitis
- Rash of systemic lupus erythematosus

Diagnostic Studies

- None indicated
- Clinical diagnosis

Treatment

- Avoidance of triggers; may include cold, alcohol, wind, heat, and spicy food
- Use of daily sunscreen
- Erythema, papules, pustules:
 - Topical metronidazole 0.75% gel is first choice
 - Topical erythromycin or clindamycin for those who cannot tolerate metronidazole
 - Systemic tetracycline or erythromycin in more severe cases
 - Minocycline in those unresponsive to other therapies
 - Isotretinoin in extreme and refractory cases
- Rhinophyma—surgical debulking
- Nonablative laser therapy
- Immunosuppressants in severe cases

Allergic Contact Dermatitis

An acute or chronic dermatitis resulting from direct contact with and subsequent irritation by chemicals, plant oils, medications, or latex.

Etiology and Demographics

- 80% of cases due to universal irritants:
 - Soaps
 - Detergents
 - Organic solvents
- True contact allergens:
 - Poison ivy
 - Poison oak
- Topical irritants:
 - Antimicrobials
 - Adhesive tape
 - Latex

Risk Factors

- Exposure to irritants
- Prior sensitization to allergens
- Less common in African-American patients

Signs and Symptoms

- Pruritus
- Scaling, erythema, and edema
- Tiny vesicles or bullae
- Thickened skin and weepy, encrusted lesions in chronic phase
- Location will suggest cause
- Affected areas hot and swollen

Differential Diagnosis

- Atopic dermatitis
- Impetigo
- Scabies

Diagnostic Studies

- No testing in the acute phase—typically a clinical diagnosis
- Patch testing after episode is resolved
- Gram stain and culture to rule out secondary bacterial infection if clinical presentation warrants
- Scraping for mites if pruritus is generalized and distribution consistent with scabies

Treatment

- Prevention:
 - Identification of the irritant
 - Barrier creams when risk of exposure is high
- Local involvement:
 - Acute phase
 - Compresses
 - Potent topical steroids
 - Wet dressings
 - Residing phase—mid- to high-potency steroids—taper
- Widespread distribution:
 - Prednisone taper—60 mg for 4–7 days, taper at 20 mg increments ending with 20 mg for 4–7 days
 - Medrol Dosepak inappropriate for this condition

Atopic Dermatitis (Eczema)

Chronic skin condition characterized by intense itching along a typical pattern of distribution. Presentation frequently varies according to the age and race of the patient.

Etiology and Demographics

- Inherited IgE-mediated hypersensitivity
- Slightly more common in males
- 95% of cases appear by age 5

Risk Factors

- Personal or family history of atopic disease:
 - Asthma
 - Allergic rhinitis
 - Atopic dermatitis
- Extremes of climate
- Emotional stress

Signs and Symptoms

- Initial presentation in infancy or early childhood
- Intense pruritus
- Exudative, rough red patches in a characteristic distribution:
 - Face
 - Neck
 - Upper trunk
 - Antecubital and popliteal fossae
- Dry, leathery, lichenified skin
- Pigmented persons:
 - Papules
 - Poorly demarcated hypopigmented areas
- African-American patients may lose pigmentation in lichenified areas
- History of recurring skin infection

Differential Diagnosis

- Seborrheic dermatitis
- Allergic contact dermatitis
- Impetigo

Diagnostic Studies

- None usually performed—diagnosis made based on history and clinical presentation
- Eosinophilia
- Elevated serum IgE
- As indicated to rule out differential diagnosis:
 - Culture for bacterial infection
 - Patch testing for suspected allergy

Treatment

- General skin care and prevention:
 - Bathing no more than once daily
 - Soap use should be minimal and washed off immediately
 - No wash brushes or cloths
 - Pat skin dry—no rough drying with towel
 - Daily use of emollients after bathing
 - Avoid irritating clothes—wool, acrylics
- Local measures:
 - Acute weeping lesions:
 - Soothing soaks or dressings (e.g., saline, aluminum subacetate solution, colloidal oatmeal)
 - High-potency steroid creams or lotions (avoid irritating ointments) after soothing soaks
 - Subacute lesions:
 - Mid- to high-potency steroids in ointment form (if tolerated)
 - Taper from bid to qd application—alternatively, switch to low-potency steroid
 - Chronic lichenified lesions:
 - High- to highest-potency topical steroids
 - Night occlusion for 2- to 6-week periods
 - Tar preparations in those unresponsive to steroid
- Systemic therapy—only in extensive and severe cases:
 - Oral prednisone—begin 60 mg, taper over 2–4 weeks
 - Oral antihistamines
 - Phototherapy—UVB, UVA, PUVA
 - Immunomodulators—tacrolimus, pimecrolimus

Psoriasis

A common benign, acute, or chronic skin disease based on genetic predisposition, characterized by reduced epidermal turnover time; normal maturation of the skin cells cannot take place, and hyperkeratinization occurs.

Etiology and Demographics

- One of several factors triggers a reduction in epidermal turnover time
- Triggers include:
 - Injury or irritation
 - No obvious cause—may be genetic predisposition
 - Extensive involvement and abrupt onset may be linked to HIV infection
- Slightly more common in men
- Occurs in 1%–2% of the population

Risk Factors

- Trauma (Koebner's phenomenon)
- Streptococcal pharyngitis—may precede eruption known as guttate psoriasis
- Genetic predisposition

Signs and Symptoms

- May be asymptomatic
- Occasional pruritus
- Sharply defined red plaques with characteristic silvery scales
- Scalp, elbows, knees, palms, and soles are common sites
- Stippling of the nail beds (pitting)
- Pink or red intergluteal fold
- Auspitz sign—droplet of blood when scales removed

Differential Diagnosis

- Atopic dermatitis
- Seborrheic dermatitis

Diagnostic Studies

- None—diagnosis based on clinical examination
- As indicated to rule out HIV if presentation warrants

Treatment

- Limited involvement:
 - Topical steroids—high- to mid-potency
 - Tar preparation
 - Occlusion therapy with Duoderm or Tegaderm for at least 7 days
 - Anthralin (Drithocrema)
 - Calcipotriene (Dovonex) ointment 0.005%
 - Tazarotene gel for mild to moderate disease

- Generalized disease—> 30% body involvement:
 - Treatment of choice—UVB light three times weekly
 - PUVA for those failing UVB
 - Systemic steroids not indicated—may induce pustular psoriasis
 - Acitretin—synthetic retinoid
 - Cyclosporine
 - Sulfasalazine
 - Immunomodulators—alefacept, methotrexate, efalizumab, etc.

Pityriasis Rosea

A common, self-limiting inflammatory skin disorder that follows a typical course of presentation and resolution.

Etiology and Demographics

- Believed to be of viral origin
- 50% more common in women
- Young adults most commonly affected
- More prominent in spring and fall months

Risk Factors

- Sharing a household with an affected individual
- Female ages 10–35

Signs and Symptoms

- Mild pruritus
- Herald patch—initial lesion preceding general eruption by 1 to 2 weeks, usually larger than subsequent lesions
- Classic lesions:
 - Oval shape
 - Fawn-colored plaques to 2 cm diameter
 - Crinkled center
 - Collarette scale
- Distributes along cleavage lines (Christmas tree distribution)
- Variant presentations:
 - Inverse pityriasis—lesions affect axilla and groin
 - Papular eruptions—more common in African-American patients
- Spontaneous resolution in 4 to 8 weeks

Differential Diagnosis

- Secondary syphilis
- Tinea corporis
- Tinea versicolor
- Seborrheic dermatitis

Diagnostic Studies

- None to make the diagnosis
- Serology to rule out syphilis
- Potassium hydroxide (KOH) scraping to rule out tinea

Treatment

- None necessary
- Topical steroids for pruritus
- UVB treatment for residual hyperpigmentation (more common in dark-skinned patients)
- Prednisone for severe pruritus
- High-dose acyclovir (800 mg tid) trial being conducted to shorten duration

Abscesses (Boils, Furuncles)

A bacterial infection involving an entire hair follicle with involvement of surrounding subcutaneous tissue; characterized by autoinoculable spread. Carbuncle is the term used to represent multiple furuncle development along contiguous follicles that forms a large abscess with multiple peaks.

Etiology and Demographics

- Gram-positive bacteria—usually staphylococci, may be community-acquired methicillin-resistant *Staphylococcus aureus* MRSA (ca-MRSA)
- Most common in hairy areas exposed to friction, pressure, and moisture

Risk Factors

- Diabetes mellitus
- Injection drug use
- HIV infection
- Close contact with affected individual

Signs and Symptoms

- Significant pain
- Gradually enlarging rounded furuncle

- Erythema of involved surrounding tissue—clear line of demarcation between affected and unaffected tissue
- Increasing fluctuance

Differential Diagnosis

- Epidermal inclusion cyst
- Tinea profunda
- Deep mycotic infection
- Hidradenitis suppurativa

Diagnostic Studies

- *Staphylococcus aureus* typically the cause
- Culture and sensitivity of purulent drainage, particularly in immunocompromised patients or recurrent cases—rule out methicillin-resistant strains or other bacteria
- Culture anterior nares for carriers

Treatment

- Incision and drainage is mainstay of treatment
- Beta lactamase-resistant antibiotic with gram-positive spectrum:
 - Dicloxacillin (Dynapen)
 - First-generation cephalosporin
 - Erythromycin for penicillin-allergic patients
 - Ciprofloxacin for resistant cases
 - Recurrent cases—dicloxacillin (Dynapen) and rifampin (Rifadin)
 - Clindamycin (Cleocin) as suppressant therapy
 - 2% mupirocin (Bactroban) to nares, axillae, and anogenital regions for carriers

Impetigo

A bacterial infection of the skin characterized by its autoinoculable nature. Impetigo may present as vesiculopustular or bullous.

Etiology and Demographics

- Vesiculopustular—*Staphylococcus aureus* or group A β -hemolytic streptococcus
- Bullous—phage group II *Staphylococcus aureus*
- More common in urban or overcrowded areas
- More common in young children
- May be endemic in day-care centers or schools

Risk Factors

- Young age
- Contact sports
- Poor hygiene
- Crowded living conditions

Signs and Symptoms

- Pruritus
- Polymorphic lesions:
 - Macules
 - Vesicles
 - Bullae
 - Pustules
 - Honey-colored gummy crusts
- Face and exposed parts most frequently affected
- Ecthyma—a deeper form characterized by ulceration with thick adherent crusts and scarring; occurs on the legs and other covered regions

Differential Diagnosis

- Contact dermatitis
- Herpes simplex virus
- Perioral dermatitis
- Scabies
- Excoriations

Diagnostic Studies

- Typically a clinical diagnosis
- Gram stain and culture confirm gram-positive bacterial infection
- Viral culture or Tzanck smear to rule out herpes virus

Treatment

- Crusted areas can be treated with soap and compresses—washcloths and towels should be laundered separately
- 2% mupirocin (Bactroban), tid, for 10 days in limited cases
- Systemic antibiotics for widespread infection or in the presence of fever or other signs of toxicity—coverage for β -lactamase-producing gram-positive bacteria:
 - Dicloxacillin (Dynapen) 250 mg qid for 10 days
 - Cephalexin (Keflex) 250 mg qid for 10 days

- Erythromycin 250 mg qid, in penicillin-allergic patients for 10 days
- In recurrent cases with colonized nares, add topical mupirocin to the nares or rifampin (Rifadin) 600 mg qd, for 5 days
- Ensure all linens washed separately

Cellulitis

A bacterial infection of the skin usually resulting from some invasion of bacteria into an area of impaired integrity and spreading in a rapid, diffuse manner.

Etiology and Demographics

- Bacterial infection:
 - Usually gram-positive cocci
 - *Escherichia coli*
 - *Haemophilus influenzae*
- May be predisposing web tinea pedis with fissuring
- Most common in children < 3 years and older adults

Risk Factors

- Breaks in the skin from trauma (lacerations, abrasions, excoriations)
- Contact sports
- Underlying dermatosis
- Diabetes mellitus
- Hematologic malignancies
- Injection drug use
- Chronic lymphedema (post mastectomy or coronary artery grafting)
- Immunocompromise

Signs and Symptoms

- Initial skin break may be apparent—pre-existing fungus, laceration, puncture wound, insect bite, or other trauma
- Tenderness or pain
- Diffuse, ill-defined borders
- Classic signs of infection:
 - Heat
 - Edema
 - Erythema
 - Pain
 - Purulent drainage—particularly in more severe cases
- May be systemic toxicity—fever, malaise, anorexia

- Regional lymphadenopathy
- Red streaking or tracking

Differential Diagnosis

- Necrotizing fasciitis—must consider if:
 - Patient appears very ill
 - Cellulitis includes bullae
 - Crepitus or anesthesia of involved area is noted
 - Skin necrosis is visible
 - Rhabdomyolysis or disseminated intravascular coagulation is apparent
- Deep vein thrombosis
- Superficial vein thrombosis

Diagnostic Studies

- None—clinical diagnosis
- Injection and subsequent aspiration of saline into affected area in an effort to culture organism is rarely successful

Treatment

- Systemic antibiotics—parenteral vs. enteral as well as coverage depend upon clinical assessment.
- Patients with systemic toxicity may require initial or full course of parenteral antibiotics.
- Mild or localized cases may be managed enterally or with an initial parenteral dose followed by complete enteral course.
- In most cases, coverage for gram-positive β -lactamase-producing bacteria as noted for impetigo is appropriate.
- Broad spectrum or gram-negative coverage is appropriate in patients with increased risk of gram-negative infection or those who have been unresponsive to initial choices:
 - Patients with HIV or other forms of immunosuppression
 - Patients with chronic disease
 - Patients with increased risk of fecal contamination (bedridden)
- Nonsteroidal anti-inflammatory drugs (NSAIDs) for discomfort.
- Antipyretics when fever presents significant catabolic costs.

Hidradenitis Suppurativa

Traditionally considered a chronic suppurative disease of the of apocrine gland region of the axillae, anogenital region, breasts, and scalp with development of sinus tracts and scarring; more recently recognized as an acneform disorder that begins with follicular occlusion leading to an abscess of the apocrine gland; also known as acne inversa.

Etiology and Demographics

- Etiology unknown
- Follicular occlusion resulting in abscess formation
- More common in the African-American population

Risk Factors

- Family history
- Nodulocystic acne
- Obesity and hirsutism
- Apocrine duct obstruction (antiperspirant use)

Signs and Symptoms

- Recurrent lesions in apocrine-rich regions (axillae, anogenital area, scalp, under breasts)
- Fluctuant, abscess-like lesions
- Spontaneous seropurulent or purulent drainage
- Scarring or sinus tract formation
- Regional lymphadema
- Significant pain

Differential Diagnosis

- Furuncle or carbuncle
- Cat-scratch disease
- Ruptured inclusion cyst

Diagnostic Studies

- None indicated—clinical diagnosis
- Aspirate may be cultured to rule out secondary bacterial infection

Treatment

- Weight loss to reduce incidence of recurrence
- Avoid occlusive antiperspirants/deodorants
- Warm compresses for smaller lesions; encourage spontaneous drainage
- NSAIDs for pain
- Incision and drainage is treatment of choice—typically extensive, should be referred to surgeon
- May require skin grafting
- Empiric antibiotic coverage of gram-positive organisms is controversial

Tinea Infection

Superficial fungal infection of the nonliving, keratinized portions of the skin, including the stratum corneum (epidermomycosis), nails (onychomycosis), and hair (trichomycosis). Individual tinea infections are named according to the part of the body they infect. Treatment for all types requires antifungal therapy, either topical or systemic.

Etiology and Demographics

- Causative organism may be any one of several dermatophytes with regional predominance
- Three common dermatophytes in the United States:
 - Trichophyton
 - Epidermophyton
 - Malassezia furfur
- Can be spread by direct contact with active lesion of an animal or another human.
- Fomites such as clothing, linens, or gym mats may also facilitate spread.
- Soil is an unusual source of fungal infection.
- More common in tropical climates, during warmer months in temperate climates.
- More common in immunocompromised patients.
- More common in peripheral nerve disease—due to reduced blood flow to periphery.
- Subtypes of tinea based upon distribution:
 - Tinea facialis—face
 - Tinea capitis—scalp
 - Tinea corporis—trunk and limbs
 - Tinea manuum—hands
 - Tinea barbae—beard
 - Tinea pedis—feet
 - Tinea cruris—groin
 - Tinea unguium—nails
 - Tinea versicolor (named for its multicolored appearance)—trunk and neck

Risk Factors

- Prolonged topical or systemic steroid use
- Heat and humidity
- Obesity—fosters warm, humid conditions
- Exposure to fungal infections of animals and humans with whom the person has close physical contact (pets, household members, classmates, teammates)
- Exposure to fomites in hot, humid environments such as wrestling or gymnastic mats
- Mechanical pressure from shoe—predisposes susceptible individuals to tinea unguium

Signs and Symptoms

- May be asymptomatic or pruritic.
- Physical findings depend upon specific type.
- Tinea corporis, manus, facialis (epidermomycosis of an exposed area):
 - Scaling erythematous plaque ranging from < 1 cm up to 20 cm
 - Varying shapes: round, arciform, or polycyclic
 - With or without pustules/vesicles
 - Usually has an elevated, sharp border with central clearing; annular configuration (“ringworm”)
 - Color is erythematous or hyperpigmented
- Tinea cruris (jock itch):
 - Similar to presentation described above, but usually arciform or polycyclic and is more dull in color
 - Often coexists with tinea pedis; infection transferred from feet to groin by hands
 - Maceration common in intertriginous areas
- Tinea pedis (athlete’s foot):
 - Erythema
 - Diffuse desquamation with superficial white scales and possible bulla formation
 - Hyperkeratosis of soles
 - Painful fissuring/cracking along lateral borders of the soles and toe webs
 - Usually bilateral foot involvement
 - Maceration seen in intertriginous areas between toes
- Tinea manuum:
 - Distribution more often unilateral but may present as bilateral
 - Chief finding may be flaking or scaling of palmar surface of hand
 - Frequently coexists with tinea pedis or tinea cruris
- Tinea versicolor:
 - Clinically significant only in some individuals
 - Distribution on trunk and neck
 - Hypo- or hyperpigmented nummular macules
 - Discrete, scattered, or confluent patches
 - Usually asymptomatic but may be mildly pruritic
- Tinea capitis and tinea barbae:
 - Inflammation of the hair follicle
 - Painful, boggy, suppurative nodules with crusting/scabs
 - Alopecia
- Tinea unguium:
 - Nails become white, brown, yellow, or black
 - Nails thicken and surface becomes roughened
 - Nails eventually separate from the nail bed

Differential Diagnosis

- Pityriasis
- Psoriasis
- Candidiasis
- Atopic dermatitis
- Contact dermatitis
- Impetigo
- Carcinoma

Diagnostic Studies

- Typically a clinical diagnosis
- “Spaghetti and meatball” hyphae apparent with KOH stain
- Wood’s lamp examination—some tinea fluoresce yellow-green
- Fungal cultures will grow but take weeks—only indicated in invasive fungal infection not responsive to treatment

Treatment

- Antifungal treatment required—topical vs. systemic dependent upon type and extent of infection
- Tinea manuum, pedis, and cruris—topical antifungal agents
- Tinea capitis, versicolor, and unguium—enteral antifungal agents
- Topical applications may be used for as little as 7 days (tinea cruris) to 6 weeks (tinea pedis)
- Systemic applications continued for 2 weeks (tinea corporis) to 12 months (tinea unguium) depending upon area infected—nail bed infection typically requires the longest duration of therapy
- When treating with prolonged oral therapy, liver function tests should be monitored
- Affected areas should be dried thoroughly after washing
- Avoid tight or occlusive clothing
- Control risk factors as possible

Candidiasis

Yeastlike superficial fungal infection that may affect any moist cutaneous or mucosal site in susceptible individuals when local immunity is disturbed.

Etiology and Demographics

- *Candida sp*
- No demographic preference; can occur in all gender, age, and ethnic groups

Risk Factors

- Predisposing factors that impair local immunity
- Immunocompromised states:
 - HIV
 - Chronic debilitation
 - Chemotherapy
- Diabetes or polyendocrinopathies
- Systemic broad-spectrum antibiotic therapy
- Moisture from repeated immersion in water
- Obesity with redundant skin folds
- Occlusive clothing that traps moisture (e.g., diapers, rubber boots)
- Hyperhidrosis (excessive sweating)
- Corticosteroid use
- Pregnancy
- Oral contraceptives

Signs and Symptoms

- Typical sites of infection include skin folds, anogenital region, axillae, intertriginous areas
- Pruritus—may be intense
- Burning sensation
- Superficial “beefy-red” appearance
- White, curdlike discharge or secretions
- Peripheral “satellite” vesicular borders
- Paronychia
- Fissuring

Differential Diagnosis

- Variety of differentials may be ruled out dependent upon site of infection.
- Mouth:
 - Hairy leukoplakia
 - Bite irritation
 - Pernicious anemia
- Genital involvement:
 - Bacterial vaginosis
 - Scabies
- Intertriginous areas:
 - Eczema
 - Contact dermatitis
 - Tinea

Diagnostic Studies

- None typically indicated
- Hyphae visible with KOH preparation
- Culture on Sabouraud's medium—takes several weeks

Treatment

- Affected area should be kept dry and exposed to open air as possible.
- Skin and nail surfaces:
 - Many topical antifungals available
 - Gentian violet 1%
 - Castellani's paint (carbol-fuchsin paint)
- Oral involvement:
 - Nystatin swish and swallow
 - Clotrimazole troches
 - Systemic antifungal agents
- Anogenital involvement:
 - Topical or intravaginal antifungal agents
 - Single-dose 150 mg fluconazole orally
 - Itraconazole 200 mg BID, for 2–4 weeks for refractory cases

Herpes Simplex Virus (HSV)

Recurrent viral mucocutaneous infection spread by skin-to-skin, skin-to-mucosa, or mucosa-to-skin contact. Only known to affect humans. HSV-1 has an oral-labial predilection, HSV-2 has a urogenital predilection; while cross-contamination may occur, outbreaks tend to be less severe and less likely to recur.

Etiology and Demographics

- Herpes simplex virus types 1 and 2
- Virus resides in sensory ganglia
- Reactivation occurs in response to a variety of triggers:
 - Minor infection
 - Physical or emotional stress
 - Sun exposure
 - Other unknown triggers
- Expression typically occurs initially in young adulthood

Risk Factors

- Intimate skin-to-skin or skin-to-mucous membrane contact with an infected person.
- Can be transmitted at any time—70% of infection occurs during asymptomatic viral shedding.
- In monogamous couples with one infected partner, the other partner is infected within 1 year in 10% of cases.

Signs and Symptoms

- Primary outbreak (after initial exposure) tends to be more severe
- Neuralgia—burning and stinging
- Neuralgias precede physical eruption
- Small, grouped vesicles on an erythematous base
- Sites of vesicular eruption:
 - Vermillion border of lips
 - Penile shaft
 - Labia
 - Perianal region
 - Buttocks
- Vesicles burst, crust, and dry over approximately 1 week
- Regional lymphadenopathy
- Malaise
- Myalgia

Differential Diagnosis

- Chancroid
- Syphilis
- Pyoderma
- Trauma

Diagnostic Studies

- Tzanck smear—positive for multinucleated cells, least sensitive test
- Direct immunofluorescent antibody slide test
- Viral culture
- Herpes, enzyme-linked immunosorbent assay (ELISA), or Western blot—used more for carriers of infection than diagnosis of acute ulcer eruption
- Patients with frequent outbreaks should consider HIV screening

Treatment

- Prevention:
 - Safe sex practices of limited value—condoms do not reduce risk of transmission as viral shedding occurs along a wide area of the groin/perineal region
 - Sunscreen—prevention of outbreaks in infected persons
 - Prophylactic antiviral agents when risk of triggers is high (e.g., ultraviolet light exposure, dental or oral surgery):
 - Acyclovir (Zovirax) 200 mg five times daily, beginning 24 hours prior to exposure
 - Valacyclovir (Valtrex) 500 mg bid (same regimen)
 - Famciclovir (Famvir) 250 mg bid (same regimen)
- Treatment in immunocompetent patients:
 - Oral forms are not typically treated
 - First genital episode (all regimens 7–10 days)
 - Acyclovir (Zovirax) 200 mg five times daily or 800 mg tid **or**
 - Valacyclovir (Valtrex) 1000 mg bid **or**
 - Famciclovir (Famvir) 250 mg tid
 - Recurrent genital cases typically mild and do not require therapy—therapy typically only reduces duration of outbreak by < 24 hours; if treatment desired, acyclovir at indicated dose, or one-half dose famciclovir or valacyclovir for 5 days
 - Suppressive therapy for those with frequent or severe recurrences—reduces outbreaks by 85%
 - Acyclovir (Zovirax) 400 mg bid **or**
 - Valacyclovir (Valtrex) 500 mg qd **or**
 - Famciclovir (Famvir) 125–250 mg bid
 - Topical therapies neither indicated nor effective in immunocompetent patient
- Immunosuppressed patients:
 - Treatment of attacks as noted for immunocompetent
 - 5% acyclovir ointment for initial infection—apply q2h while awake for 4 days—may reduce duration of attack

Herpes Zoster (Shingles)

Acute vesicular eruption as a result of the varicella-zoster virus, following a typical course in terms of symptoms and sign presentation, and occurring only once in the overwhelming majority.

Etiology and Demographics

- Varicella-zoster virus
- Typically contracted in childhood as chicken pox
- Lies dormant in nerve ganglion

- Viral reactivation results in typical disease course
- Cause of reactivation is unclear
- Most common in persons > 50 years of age

Risk Factors

- Advancing age
- Immunosuppression—HIV-infected patients 20 times more likely to develop zoster
- Trauma to sensory ganglia

Signs and Symptoms

- Pain along a dermatomal pathway—occurs 48 hours before eruption of lesions
- Grouped vesicles along an erythematous base
- Unilateral distribution typically:
 - Some lesions may scatter outside dermatome
 - Eruption may cross mid-line in immunocompromised patients
- Vesicles erupt, burst, and crust spontaneously over a period of 10–14 days
- Regional lymphadenopathy

Differential Diagnosis

- Poison oak and ivy
- Allergic contact dermatitis
- Herpes simplex virus
- Dermatomal pain may mimic systemic disease prior to vesicular eruption depending upon dermatome affected

Diagnostic Studies

- Tzanck smear
- Varicella-zoster virus antibodies
- Viral culture of lesions

Treatment

- All patients should be treated because treatment reduces postherpetic neuralgia:
 - Immunocompetent patient—all regimens for 7 days
 - Acyclovir (Zovirax) 800 mg five times daily **or**
 - Famciclovir (Famvir) 500 mg tid **or**
 - Valacyclovir (Valtrex) 1000 mg tid
 - Oral corticosteroids for 3-week tapering dose—begin 60 mg qd for pain

- Immunosuppressed patient:
 - Antiviral agents in doses shown for the immunocompetent patient, with duration of therapy of 14 days (or until lesions healed)
 - No corticosteroids in this population—increases risk of dissemination
 - Progression of infection may require parenteral acyclovir
 - Foscarnet (Foscavir) 40 mg/kg for acyclovir-resistant cases
- All patients with corneal involvement require ophthalmology evaluation:
 - Postherpetic neuralgia:
 - Capsaicin ointment 0.025% to 0.075%
 - Topical lidocaine
 - Tricyclic antidepressants
 - Antiepileptic drugs
 - Regional nerve blocks

Human Papillomavirus (HPV)

Discrete, benign, epithelial hyperplastic papules occurring on skin and mucous membranes—the wart.

Etiology and Demographics

- Human papillomavirus—more than 65 types identified
- Simultaneous infection with multiples types is common
- Most common in school-aged children
- Occurs in 5% of adult population

Risk Factors

- Skin-to-skin contact with infected person
- Household contact with infected person
- HIV infection

Signs and Symptoms

- Typically asymptomatic—rarely lesions may itch, burn, sting
- Plantar warts often tender (occurring on plantar surface of foot)
- Pruritus more common with anogenital warts
- Polymorphic appearance:
 - Macular
 - Papular
 - Fleshy
 - Hyper- or hypopigmented
 - Rough or smooth surfaces

Differential Diagnosis

- Actinic keratosis
- Seborrheic keratosis
- Condylomata lata
- Molluscum contagiosum
- Squamous cell carcinoma

Diagnostic Studies

- None—typically clinical diagnosis
- Suspicious lesions should be sent for pathology evaluation when removed

Treatment

- Warts of the hands:
 - Liquid nitrogen 20–45 seconds over a 2–4 week period—can produce scarring if used inappropriately or incorrectly
 - Keratolytic agents—variety of salicylic acid products available
- Anogenital warts—podophyllum resin
- External genitalia—imiquimod 5% has high rate of clearance
- Laser removal
- Operative removal
- Cimetidine 35–50 mg/kg daily
- Retinoids
- Due to viral nature, recurrence is not uncommon
- Sexual transmission of virus may occur even following removal of visible lesions
- Human papillomavirus quadrivalent vaccine (Gardasil) can protect against HPV types 6 and 11, which cause 90% of genital wart infections; also protects against types 16 and 18 which cause 70% of cervical cancers, indicated only for females

Molluscum Contagiosum

A dermal manifestation of infection with poxvirus, characterized by a typical popular lesion that is autoinoculable.

Etiology and Demographics

- Poxvirus (molluscum contagiosum virus—MCV-1 and -2)
- Common in children and sexually active adults
- Occurs more often in males than females

Risk Factors

- Multiple sexual partners
- AIDS infection with helper T cell count < 100/uL

Signs and Symptoms

- Single or multiple papules
- Dome-shaped
- 2–5 mm in diameter
- Firm, solid, and flesh-colored initially
- Progresses to softer, white-gray color
- Characteristic umbilication
- Occur most commonly on face, genitals, abdomen

Differential Diagnosis

- Verruca plana
- Condyloma acuminata
- Sebaceous hyperplasia
- Basal cell carcinoma

Diagnostic Studies

- None—typically clinical diagnosis
- Direct microscopy with Giemsa's stain demonstrates central inclusion bodies

Treatment

- Curettage
- Liquid nitrogen (as shown for human papillomavirus)
- Electrosurgery with fine needle

Burns

A heat injury to some level of the epidermis (known as first-degree or superficial burn), dermis and above (known as second-degree or partial thickness burn), and/or subcutaneous tissue and above (known as third-degree or full thickness burn).

Etiology and Demographics

- Exposure to intense heat:
 - Fire
 - Steam

- Chemicals
- Electricity
- 1.25 million burn injuries annually
- 51,000 acute burn hospitalizations annually
- More than 75% of burns involve < 10% body surface
- Incidence of burn injuries dropping—down 50% in recent years

Risk Factors

- Occupational exposure to chemical or fire
- Firefighters
- Unsupervised children

Signs and Symptoms

- First degree:
 - Pain
 - Erythema
 - Edema
 - Will demonstrate good capillary refill
 - No initial blistering
- Second degree:
 - Pain
 - Hair absent or easily extracted
 - Initial blistering
 - Oozing serous fluid
 - Skin appears smoother
- Third degree:
 - Charred, white skin
 - Skin appears dry and depressed
 - Feels “tight”
- Rule of nines for assessing percentage of burned area:
 - Anterior head and neck—4.5%
 - Posterior head and neck—4.5%
 - Anterior trunk—18%
 - Posterior trunk—18%
 - Anterior arms—4.5% each
 - Posterior arms—4.5% each
 - Genitalia—1%
 - Anterior legs—9% each
 - Posterior legs—9% each

- Comorbid smoke inhalation injury:
 - Singed nasal hairs
 - Dark sputum
 - Burn occurred in a closed space
- Cardiac dysrhythmia in electrical injury
- Pancreatitis in severe burns

Differential Diagnosis

- None—history of injury rules out other differentials
- Identification of specific agent in chemical injury

Diagnostic Studies

- None immediately in the outpatient setting
- Culture and sensitivity if bacterial infection suspected during the healing process

Treatment

- Immediate triage is the first priority—determine the need for emergency care.
- Stabilization of serious burns in accordance with Advanced Trauma Life Support® (ATLS®) guidelines:
 - Establish airway.
 - Rule out cervical spine and head injury.
 - Stabilize fractures.
 - Establish vascular access.
 - Fluid resuscitation.
- Irrigate eye burns with water, saline, or lactated Ringer's solution.
- Outpatient management appropriate for:
 - First-degree burns
 - Superficial second-degree burns up to 6% of body not involving functional or cosmetic areas
 - Selected, deeper second-degree burns if not on lower extremities, hands, face, areas of function or cosmetic concern, or genitals, and that probably do not cover more than 1% to 2% of the body
- Prevention of bacterial infection is of utmost priority.

Scabies

A mite infestation resulting in generalized pruritus.

Etiology and Demographics

- *Sarcoptes scabiei*
- Common in young children
- Occurs in institutional settings or crowded living quarters
- Epidemics occur in cycles

Risk Factors

- Close contact or shared bedding with infected individual
- Institutional living
- AIDS infection

Signs and Symptoms

- Pruritus—ranges from mild to severe
- Generalized excoriations with vesicles
- Occurs in burrows or runs—2–3 mm stretches
- Typical sites:
 - Web spaces of hands/feet
 - Palms
 - Wrists
 - Axillae
 - Feet
- Pruritic papules may occur
- Crusting occurs after several months

Differential Diagnosis

- Pediculosis
- Atopic dermatitis
- Metabolic pruritus
- Pityriasis rosea

Diagnostic Studies

- Scraping of lesion after oil immersion—microscopic visualization of organism, ova, or feces
- Burrow ink test—apply ink to burrow, shave, and visualize under light microscope

Treatment

- All clothing and linens should be laundered and stored in plastic bags for 14 days.
- All infected persons must be treated.
- Permethrin (Nix) 5% cream:
 - Apply for 8–12 hours
 - May repeat in 1 week
 - Safe in patients > 2 months old
 - Used in pregnant women with documented infestation
- Lindane 1% cream or lotion:
 - Safe for use in adults
 - Potential neurotoxicity—avoid in children and pregnant women
 - Do not overuse
- Resistant cases or immunosuppressed host—ivermectin (Stromectol) 200 ug/kg in a single dose or two doses 2 weeks apart.
- Pruritus may continue for several weeks after successful treatment.
- Antihistamines or steroids for pruritus.
- Systemic antibiotics for pyoderma—suggests secondary bacterial infection.

Pediculosis

A parasitic infestation of the skin occurring on the scalp, body, or pubic region causing pruritus and excoriation.

Etiology and Demographics

- *Pediculus humanus capitis*—scalp lice
- *Pediculus humanus corporis*—body lice
- *Phthirus pubis*—pubic lice, crabs
- Head lice most common in elementary school-aged children—uncommon in African-American population
- Pubic lice most common in young adults
- Body lice most common in overcrowded living environments

Risk Factors

- Head lice:
 - School-aged children
 - Any person (adult or child) sharing comb, brush, or hat with affected individual

- Body lice:
 - Overcrowded living conditions
 - Institutional living
 - Sharing clothing/bedding with affected individual
- Pubic lice:
 - Sexual contact with affected individual
 - Poor personal hygiene

Signs and Symptoms

- Pruritus
- Scratching may result in deep excoriation
- Pyoderma may be present
- Lice can be visualized:
 - Head and body lice—3–4 mm long
 - Best visualized close to skin, at ears, or nape of neck
 - Nits may be visualized on hair shaft
 - Pubic lice clearly visible to naked eye—often seen crawling
 - Body lice—in clothing folds

Differential Diagnosis

- Seborrheic dermatitis
- Scabies
- Atopic dermatitis
- Tinea infection

Diagnostic Studies

- Culture for suspected secondary bacterial infection
- KOH scraping to rule out tinea
- Sexually transmitted infection (STI) screening as indicated in pubic lice

Treatment

- Body lice treated via disposal of infested clothing.
- Head lice—1% permethrin cream rinse (Nix)—apply for 30 minutes to 8 hours, rinse, repeat in 1 week.
 - Nits removed with a fine-toothed comb.
 - Other agents include synergized permethrin (RID) and malathion lotion 1% (Ovide).
- Pubic lice—lindane or permethrin as noted for scabies.
- In all cases—treatment of source contacts or all affected individuals.

Seborrheic Keratoses

Benign epithelial tumors that frequently appear in old age. In most circumstances they represent a normal age-related change and do not require treatment.

Etiology and Demographics

- Proliferation of keratinocytes, melanocytes, and plugged follicles
- Occurs most often in the late middle age and old age
- Most common benign tumor in the elderly
- Infrequently is associated with underlying malignancies (e.g., adenocarcinoma of the gastrointestinal tract, lymphoma, and acute leukemia)—consider when the onset is widespread and sudden (sign of Leser-Trélat)

Risk Factors

- Advancing age
- Family history

Signs and Symptoms

- Plaques ranging in size from 3 mm to 20 mm
- Color ranges—beige, brown, black
- “Fuzzy” or irregular surface
- Appear “stuck on” to skin surfaces
- Lesions not typically multicolored
- Borders may be irregular
- May occur on any skin surface except hands/feet

Differential Diagnosis

- Squamous cell carcinoma
- Malignant melanoma
- Pigmented basal cell carcinoma
- Solar lentigo

Diagnostic Studies

- None—typically a clinical diagnosis
- As indicated to rule out other causes when diagnosis is in question
- As indicated to rule out malignancy when onset is widespread and acute

Treatment

- None required—may be removed for cosmetic reasons
- Pure trichloroacetic acid
- Ammonium lactate
- Alpha hydroxyacids
- Electrodesiccation
- Curettage removal
- Liquid nitrogen freezing
- Cryosurgery (CO₂)

Actinic Keratoses

Small patches that occur on sun-exposed parts of the body in persons of fair complexion.

Actinic keratoses are considered premalignant although only 1:1000 lesions per year progress to squamous cell carcinomas.

Etiology and Demographics

- Recurrent or prolonged sun exposure
- More common in males
- Onset middle or late adulthood

Risk Factors

- History of sun exposure
- Hot, sunny geographic living
- Fair coloring

Signs and Symptoms

- Small patches (< 10 mm)
- Colors range from flesh-colored to hyperpigmented
- Rough “sandpaper” to touch
- May occur singly or with multiple presentation

Differential Diagnosis

- Seborrheic keratoses
- Basal cell carcinoma
- Human papillomavirus

Diagnostic Studies

- Any suspicious lesions should be biopsied.
- Typically a clinical diagnosis.

Treatment

- Liquid nitrogen freezing—lesions crust and resolve in < 2 weeks
- Excision
- 1%–5% fluorouracil cream—bid applications result in crusting and eroding of lesions
- Lesions not responding to therapy should be biopsied

Basal Cell Carcinoma

Slow-growing carcinoma that most commonly presents as a papule or nodule that may have central umbilication, progressing to significant ulceration. There is no risk of metastasis, but can produce significant cosmetic deformity.

Etiology and Demographics

- Proliferation of atypical basal cells with various amounts of stroma
- Most common type of skin cancer
- Occurs most in young and middle-aged adults
- Males affected more often than females

Risk Factors

- Excess sun exposure
- Fair skin with poor tanning ability
- Previous incidence of basal cell carcinoma

Signs and Symptoms

- Slow growing—achieves sizes of 2 cm over a period of years
- Waxy, pearly appearance
- Brown or gray pigment may occur in some cases
- Visible telangiectasia
- Pearly, translucent quality—diagnostic feature
- Less often may appear as red scales or plaques
- Most common on the face, but can occur on any sun-exposed surface
- Can progress to severe ulceration if not treated

Differential Diagnosis

- Molluscum contagiosum
- Intradermal nevi

Diagnostic Studies

- All suspect lesions should be shaved or punch biopsied
- As indicated to rule out malignancy or metastasis

Treatment

- Referral/consult with physician
- Goal of therapy is eradication and minimal cosmetic deformity
- Options include:
 - Excision
 - Curettage and electrodesiccation
 - Radiotherapy
 - Mohs' technique—removal of tumor with immediate pathologic examination of margins and reexcision with final closure—all at one procedure
- Instruction to patient to avoid sun exposure
- Follow-up at regular intervals for 5 years to evaluate for recurrence

Squamous Cell Carcinoma

A skin malignancy that occurs on sun-exposed areas and may arise out of actinic keratoses on exposed parts in individuals who burn easily; develops in the course of a few months. Approximately 5% have the potential for metastasis—variants include “erythroplasia of Queyrat,” which occurs on the penis, and Bowen’s disease, which is a form of squamous cell carcinoma arising de novo on any area of skin.

Etiology and Demographics

- Malignant tumor of epithelial keratinocytes developing on skin and mucous membranes
- Second most common type of cutaneous carcinoma
- Most common in males
- Age distribution typically > 55 years

Risk Factors

- Fair skin with poor tanning ability
- History of significant sun exposure—outdoor occupation
- Tobacco use

- Radiation exposure
- Organ transplant patients
- Cases occurring in the oral cavity and surrounding region and the genitalia have a greater incidence of metastasis

Signs and Symptoms

- Develop relatively quickly—over a period of months
- Originate with a small red nodule, papule, or plaque
- Progresses to induration
- Thick, adherent, keratotic scale
- Honey-colored exudate extruded from periphery
- May be eroded, crusted, ulcerated, hard, erythematous, isolated, or multiple lesions

Differential Diagnosis

- Nummular eczema
- Paget's disease
- Keratoacanthoma

Diagnostic Studies

- Suspicious lesions should be biopsied

Treatment

- Immediate consult/referral with physician
- Excision if preferred treatment
- Mohs' technique for high-risk lesions
- Electrodesiccation and curettage in select lesions
- Intralesional fluorouracil or methotrexate for keratoacanthomas

Malignant Melanoma

Least common but most deadly form of skin malignancy, with a high risk of metastasis. Cases with lymph node involvement at time of diagnosis have a 5-year survival rate of 30%; those with distant metastasis, < 10%.

Etiology and Demographics

- Proliferating malignant melanocytes
- Comprise 5% of all cutaneous carcinomas in the U.S.
- More common in sunbelt states
- Most common cancer of women aged 25–29

Risk Factors

- Positive family history
- Excessive sun exposure
- Severe sunburn, particularly at an early age
- Outdoor occupations
- Fair skin

Signs and Symptoms

- Five histopathologic types:
 - Superficial spreading malignant melanoma (most common)
 - Nodular malignant melanoma
 - Acral-lentiginous melanoma (occurs on palms of hands, soles of feet, and 1 beds)
 - Malignant melanomas of mucous membranes
 - Melanomas arising from blue or congenital nevi
- > 6 mm diameter
- May be macular, papular, or nodular
- Variety of colors, may demonstrate multiple colors in one lesion
- May hurt, bleed, or itch
- Irregular margins
- Five cardinal signs of malignant melanoma via ABCDE method:
 - **A**symmetry
 - **B**order is irregular and often scalloped
 - **C**olor is mottled with haphazard display of brown, black, gray, and/or pink
 - **D**iameter is large—greater than 6.0 mm
 - **E**levation is almost always present with subtle or obvious surface distortion and best assessed by side-lighting of the lesion

Differential Diagnosis

- Benign nevi
- Pigmented basal cell carcinoma

Diagnostic Studies

- Biopsy to include margins
- Tumor thickness is single most useful prognostic indicator:
 - < 0.76 mm—96% 10-year survival
 - 0.76 mm to 1.69 mm—81% 10-year survival
 - 1.7 mm to 3.6 mm—57% 10-year survival
 - > 3.6 mm—31% 10-year survival

Treatment

- Immediate consultation with physician
- Excision to include 1–3 cm margin
- Sentinel lymph node biopsy

Resources and References

- Balin, A. K. (2006). *Seborrheic keratoses*. Retrieved October 1, 2007, from www.emedicine.com/DERM/topic397.htm.
- Berger, T. G. (2007). Skin, hair, and nails. In L. M. Tierney, Jr., S. J. McPhee, & M. A. Papadakis (Eds.), *Current medical diagnosis and Treatment* (46th ed., pp. 87–150). New York: Lange Medical Books/McGraw-Hill.
- Blount, B. W. (2002). Rosacea: A common, yet commonly overlooked, condition. *American Family Physician*, 66(3), 435–440.
- Fairhurst, D. A., Ashcroft, D. M., Griffiths, C. E. (2005). Optimal management of severe plaque form of psoriasis. *American Journal of Clinical Dermatology*, 6(5), 283–294.
- Fisher, D. (2001). Common dermatoses: How to treat safely and effectively during pregnancy, part 2. *Consultant*, 41(8), 1174–1177.
- Fitzpatrick, T. B., Johnson, R. A., Wolff, K., & Suurmond, D. (2005). *Color atlas & synopsis of clinical dermatology* (5th ed.). New York: McGraw-Hill.
- Gnann Jr., J. W. (2002). Clinical practice: Herpes zoster. *New England Journal of Medicine*, 347(4), 340–346.
- Kaplan, D. L. (2001). Dermclinic: Cutaneous conundrums, dermatologic disguises. *Consultant*, 41(4), 571–579, 523–525, 529–530.
- Marghoob, A. A. (2002). Dermatologic look-alikes: Skin cancer concerns. *Clinical Advisor*, 5, 121–122, 127.
- Monroe, J. R. (2005). Controlling rosacea: A case-based approach. *Clinical Advisor*, 8, 20–21, 25–27.
- Rees, M. T. (2002). Managing atopic eczema. *Primary Health Care*, 12(8), 27–32.
- Rosi, Y. L., Lowe, L., & Kang, S. (2005). Treatment of hidradenitis suppurativa with infliximab in a patient with Crohn's disease. *Journal of Dermatological Treatment*, 16(1), 58–61.

Supplement to Chapter 12

Medications for Disorders of the Skin

Antifungals

Systemic

- Caspofungin
- Fluconazole
- Griseofulvin
- Terbinafine
- Clotrimazole
- Nystatin
- Ketoconazole
- Itraconazole
- Voriconazole

Topical

- Sulconazole nitrate 1%
- Terbinafine 1%
- Ciclopirox 0.77%
- Clotrimazole 1%
- Miconazole 2%
- Butenafine HCl 1%
- Ketoconazole 2%
- Oxiconazole nitrate 1%
- Selenium sulfide 2.5%
- Tolnaftate 1%
- Econazole nitrate 1%

Retinoids

- Isotretinoin
- Tretinoin
- Adapalene 0.1% (topical)
- Tazarotene 0.05%, 0.1%

Topical Antibiotics

- Clindamycin 1%
- Erythromycin 2%, 3%
- Sulfacetamide 10%

Topical Steroids

Low potency

- Hydrocortisone acetate 1%, 2.5%
- Alclometasone dipropionate 0.05%
- Desonide 0.05%

Medium potency

- Prednicarbate 0.1%
- Triamcinolone acetonide 0.1%
- Fluocinolone acetonide 0.01, 0.25%
- Mometasone furoate 0.1%

High potency

- Diflorasone diacetate 0.05%
- Amcinonide 0.1%
- Fluocinonide 0.05%

Ultra-high potency

- Betamethasone dipropionate 0.05%
- Clobetasol propionate 0.05%
- Halobetasol propionate 0.05%

Immunomodulators

- Alefacept
- Methotrexate
- Cyclosporine
- Efalizumab
- Etanercept
- Infliximab

