Staphylococcus aureus and the group A β-hemolytic streptococci account for the majority of uncomplicated skin and soft tissue infections.

Impetigo

- Common, highly contagious bacterial infection in the epidermis usually caused by Staph aureus and, to a lesser degree, Group A β-hemolytic streptococcus (Strep pyogenes).
- Two variants include: non-bullous or crusted and bullous impetigo.
**Non- Bullous Impetigo**

- Most common variant usually caused by *S. aureus*
- Infections occur at minor sites of trauma (e.g. insect bite, abrasion, laceration)
- Seen around the nose, mouth, and extremities
- Vesicle or pustule develops that evolves into a honey-colored crusted plaque
- +/- regional lymphadenopathy with strep

**Bullous Impetigo**

- Less common than non-bullous impetigo
- Considered a localized form of staphylococcal scalded skin syndrome
- Lesions can occur on intact skin
- Exotoxin produced by staphylococci produce vesicles which rapidly progress to flaccid bullae
- Bullae contain clear yellow fluid that subsequently becomes cloudy and ruptures leaving a rim of scale around an erythematous moist base
- No surrounding erythema or lymphadenopathy

**Impetigo - Diagnosis**

- Often based on clinical findings
- Bacterial culture
- Anti-DNase B titer (antideoxyriboonuclease B)
- Gram stain as needed
Impetigo - Treatment

- Mild and localized
  - treat topically with Bactroban (mupirocin 2%) or Altabax (retapamulin 1%)
- Extensive
  - consider a 5-10 day course of cephalexin, dicloxacillin, or amoxicillin-clavulanate
- Recurrent disease often occurs in individuals who are nasal carriers of the organism

Ecthyma

- Considered an ulcerated form of non-bullous impetigo in which an abscess will slough into the dermis to produce a shallow ulcer
- Mixed infection:
  - usually due to primary infection by group A beta-hemolytic streptococci or a streptococcal superinfection of a preexisting ulcer
  - often contaminated with staphylococci
- Mostly seen in individuals with poor hygiene

Ecthyma

- Vesicles and vesiculopustules rupture forming crusted ulcers with a "punched out" appearance and purulent necrotic base with indurated, violaceous margins
- Lesions slow to heal and cause scarring
- Treat with 14-day course of oral antibiotics (same agents as impetigo except penicillin VR may be used)
- Compresses and topical antibiotics may be helpful
Infectious Folliculitis

Inflammation of the hair follicle resulting from infection:
- Staphylococcal
- Gram-negative
- Pseudomonas
- Candidiasis
- Pityrosporum
- Herpetic
- Tinea barbae
- Others

Staphylococcal Folliculitis

- S. aureus is the most common infectious cause
- Small fragile dome-shaped pustule or papule occurs at the opening of the hair follicle
- May rupture resulting in a crusted papule

Folliculitis of the beard area due to Staphylococcus aureus. Discrete papulopustules are seen posteriorly. Centrally, there is deeper involvement with plaque formation (sycosis barbae).

Staphylococcal Folliculitis

- Consider bacterial culture
- Mild cases can be treated with wet Burow’s compresses or topical antibacterial agents – oral antibiotics are frequently required
- Refer to treatment of impetigo
Furunculosis
(Deep Folliculitis, Boil)

- Painful, deep-seated, circumscribed perifollicular nodule with central plug becoming fluctuant over several days
- *S. aureus* is the most common pathogen
- Systemic symptoms usually absent

Carbuncle

- Collections of furuncles
- Extend deep into the subcutaneous tissue
- Surface usually displays multiple draining sinus tracts
- Occasionally ulcerates

Furuncles and Carbuncles

- Diagnosis is primarily on clinical appearance
- Bacterial culture
  - central face, muscle, or fascia is involved
  - systemic toxicity
  - treatment failure
  - an immunocompromised host
  - the possibility of MRSA
  - crepitus present
- Warm compresses, MD, packing, antibiotics in nonhealing case

Treatment of carrier state when needed
Is This Abscess MRSA?

- An abscess is a walled-off infection resembling a furuncle.
- Patient history often "spider bite".
- Occurs in the absence of risk factors for hospital-acquired disease.
- Genetically distinct organism carrying genes which bestow resistance to methicillin but not to other antibiotics (in contrast to HA-MRSA).

CA-MRSA - Treatment

- I&D fluctuant lesions, submit fluid for culture and sensitivity and consider Gram stain.
- Oral antibiotics usually not necessary but treat lesions larger than 5.0 cm or with surrounding cellulitis with an oral antimicrobial for 10-21 days.
- MRSA usually susceptible to trimethoprim-sulfamethoxazole, clindamycin, gentamicin, and minocycline; add rifampin to the oral regimen in recurrent cases.
- Also consider mupirocin ointment intranasally, daily bathing with an antimicrobial agent, and treatment of culture-positive household contacts.

Staphylococcal Scalded Skin Syndrome (SSSS)
Most common in children under 6 with renal immaturity
- Begins with a minor or inapparent S. aureus infection
- Often prodrome of malaise, fever, irritability, sore throat and where a fragment of the skin is peaced through the air
- Skin subsequently develops and forms vesicles or bullae, eventually peeling off in sheets leaving a moist, red surface
- Nikolsky’s sign positive
- Yellow crust forms and de weted surface dries and cracks
- Diagnosis involves skin biopsy and bacterial cultures from nose, throat, bullae, eyes, etc...
- Treatment includes hospitalization and IV antibiotic therapy, fluid rehydration, and topical wound care

Pseudomonas

*P. aeruginosa* is a gram-negative aerobic rod infecting warm, moist areas. Severe infections occur in immunocompromised patients.
Pseudomonas Folliculitis

- Infection of follicles and skin breaks following exposure to a contaminated water source
- Lesions begin as pruritic, erythematous macules that progress to papules and pustules
- Clears in 7-14 days often with red-brown, postinflammatory hyperpigmentation
- 5% acetic acid wet compresses; if severe treat with oral fluoroquinolone and/or topical gentamicin

Acute and Chronic Paronychia

- Paronychia is an inflammatory reaction of the nailfolds
- Moisture induced trauma leads to erythema, swelling, or abscess formation in the periungual skin
- Pus may be expressed from beneath the proximal nail fold
- Acute pyogenic cases are usually staphylococcus or streptococcus
- Chronic paronychia is most frequently C. albicans but pseudomonas is the most common bacterial cause
- Green Nail Syndrome is often associated in chronic cases with pseudomonas overgrowth
- Treatment consists of keeping the nail areas dry, acetic acid soaks, avoiding trauma, I&D of abscesses, and topical or oral antibiotics when necessary

Cutaneous Fungal Infections

Superficial infections of stratum corneum, hair, and nails caused by three genera of fungi known as the dermatophytes.
- *Trichophyton* species cause lesions of all keratinized tissue; *T. rubrum* most common dermatophyte pathogen
- *Microsporum* species principally invade the hair
- *Epidermophyton* species invade the intertriginous skin and are only transmitted by humans
Tinea Capitis

- Infection of the scalp in children and infrequently in adults
- Most often caused by T. tonsurans followed by M. Canis
- Alopecia is the most common presentation
- Many patients have lymphadenopathy
- Oral antifungals agents needed but selenium sulfide and ketoconazole shampoos are helpful to decrease carrier state and spread

Inflammatory Tinea Capitis

Kerion Formation

- Severe pustular eruption with alopecia resulting from advanced disease
- Lesions are boggy, purulent plaques with abscess formation
- Pruritus, fever, pain, lymphadenopathy, and scarring alopecia may be associated

Noninflammatory Tinea Capitis

Black dot presents as patches of short broken hairs along with normal appearing hairs
Noninflammatory Tinea Capitis

- Scaling without significant alopecia resembling seborrheic dermatitis
- Solitary or multiple patches of alopecia with fine scale

Tinea Corporis

- Infection of the skin, excluding the hair, nails, palms, soles, and groin
- Caused by most of the dermatophyte species with T. rubrum, T. mentagrophytes, and M. canis being the most common
- Erythematous, scaly papule or plaque that spreads centrifugally as it clears centrally
- Border may be scaly, pustular, or vesicular
Eczema

Tinea corporis

Eczema
Eczema

Majocchi’s granuloma
May develop due to penetration of organisms along the hair follicle

Tinea Cruris
- Infection of the groin
- Usually caused by *E. floccosum, T. rubrum, or T. mentagrophytes*
- Scaly, erythematous patch with vesicles or scaling along an advancing border
- Wear loose cotton underwear, treat tinea pedis if present
- Antifungal powders may be helpful
Tinea pedis & Tinea manuum

- Tinea pedis - infection of the toe webs and soles of the feet
- Tinea manuum - infection of the palmar and interdigital areas of the hand
- Most commonly caused by T. rubrum, T. mentagrophytes, and E. floccosum (same as tinea cruris)
- Avoid occlusive footwear, dry between the toes after bathing, and use an absorbent antifungal powder for prophylaxis

Tinea Pedis - classic “ringworm” presentation

Tinea Pedis – Interdigital Form
Tinea Pedis - Moccasin Type

- Presents as fine scaling or hyperkeratosis
- Often present in patients with tinea manuum
- May present as two feet and one hand syndrome

Tinea Pedis - Inflammatory Form

Presents with erythema and vesicles which may fuse into bullae

Tinea barbae & Tinea Sycosis

- Fungal infection of the coarse hair-bearing beard and moustache area of men
- Most commonly caused by T. verrucosum, T. mentagrophytes
- Annular plaques or patches resembling tinea corporis
- Pustular folliculitis
Tinea Sycosis

- Deep folliculitis presenting with kerion-like plaques and nodules
- Abscesses, sinus tracts, and bacterial super-infection may develop
- Patients may develop constitutional symptoms

Tinea unguium
(dermatophytic onychomycosis)

Onychomycosis – includes all fungal infections of the nail due to dermatophytes and non-dermatophytes
- men > women
- frequently associated with chronic tinea pedis
- toenails > fingernails

Distal subungual onychomycosis (DSO)

- Most common variant
- Usually caused by T. rubrum and T. mentagrophytes
Proximal Subungual Onychomycosis (PSO)

- Least common variant
- *T. rubrum* is the most common cause
- Immunocompromised host - screen for HIV

White superficial onychomycosis

Most infections due to *T. mentagrophytes*

Dx & Tx of Dermatophytosis

Diagnostic aids to clinical exam
- Wood’s lamp examination, KOH preparation, fungal culture, biopsy for PAS staining

Treatment:
- *Tinea capitis, unguium and barbae*: require oral treatment; dosage and length of treatment depend on age, weight, location of infection, and drug used
- *Tinea corporis, cruris, and pedis*: topical treatments highly effective; most applied 1-2/day for up to 4 wks depending on site of involvement and agent used
Candidal Infections

- Most common mycotic infection ranging from superficial disease to invasive infections
- Candida albicans is commonly responsible for candidiasis
- Yeast is frequently a part of normal body flora acting as a pathogen only when given the opportunity
- Confirm diagnosis with KOH (elongated pseudohyphae and budding spores) and culture

Chronic Mucocutaneous Candidiasis

Candidal Onychomycosis
- Total nail dystrophy usually seen with chronic mucocutaneous candidiasis
- Nail plate thickens and turns yellow-brown resembling DSO; paronychial inflammation may be present
- Oral fluconazole or itraconazole combined with nail lacquers useful for onychomycosis

Candidal Paronychia
- Candida species are the most common cause of chronic paronychia
- Paronychia typically involves the periungual areas causing redness, swelling, and pain
- Control DM if present
- Avoid trauma and water exposure
- Topical antimycotic agent +/- topical steroid
- Oral fluconazole or itraconazole in resistant cases
Candidal Vulvovaginitis

- White plaques on erythematous mucosa and a creamy white discharge
- May be associated with severe itching, burning, and dysuria
- May produce inflammation of the external genitals with moist eroded patches, scaling, and satellite pustules
- Treatment:
  - Topical treatments include Miconazole, Tioconazole, Butoconazole, Clotrimazole, Terconazole
  - Mycostatin (nystatin) 100,000 U vaginal tab
  - Oral Diflucan (fluconazole)

Candida balanitis

- Infection of the penis; uncircumcised penis most susceptible
- Tender red papules and pustules rupture leaving 1-2 mm white umbilicated lesions
- White exudative plaques under foreskin
- Similar treatment as Candidal vulvovaginitis

Candidal Diaper Dermatitis

- Occluded diaper area provides a warm, moist, ideal environment
- Erythematous, macerated plaque with a scalloped border involving the perianal area, inguinal folds, lower abdomen, scrotum, penis, nipples, hands, or toes
- Satellite pustules are a hallmark
- Topical antifungal agents and improved hygiene – do NOT use Lotrisone or Mycolog II
- Oral antifungal therapy for recurrent infections
Intertriginous Candidiasis

- Axillae, groin, umbilicus, gluteal cleft, finger and toe web spaces, under breasts or pendulous abdominal fat folds, mouth angle
- Large, confluent areas of moist, eroded, erythematous skin with macerated, scaly border
- Small vesicles or pustules on an erythematous base occur as satellite lesions
- May become dry and scaly over time
- Topical antifungal agents, cool wet Burow's compresses, powder to act as a dry lubricant

Treatment: Topical and Oral Antifungals

- Azoles are mostly fungistatic but some are also fungicidal; may be used for dermatophytes and yeasts
  - Topical: econazole, ketoconazole, clotrimazole, sertaconazole, oxiconazole, miconazole, sulconazole
  - Systemic: itraconazole, fluconazole, voriconazole
- Amines are fungicidal and work best against dermatophytes
  - Topical: terbinafine, butenafine, naftifine
  - Systemic: terbinafine
- Hydroxypyridone is fungicidal/fungistatic and may be used for dermatophytes and yeasts
  - Topical: ciclopirox
- Polyenes are fungicidal/fungistatic and are effective against Candida
  - Topical: nystatin
  - Systemic: amphotericin B
- Others
  - Topical: selenium sulfide, sulfuric acid, lactic acid
  - Systemic: caspofungin, flucytosine, Griseofulvin (actually an antibiotic)

Pityrosporum Infections

- Caused by Malassezia furfur growing in a hyphal phase
- In its yeast phase, known as Pityrosporum orbiculare, it exists as an element of normal follicular flora
- Acts as an opportunistic to cause Pityrosporum folliculitis and tinea versicolor, and possibly implicated in seborrhoeic dermatitis
- Overgrowth in susceptible individuals occurs due to multiple factors, but especially common in warm humid environments or in the physically active
- KOH demonstrates groups of thick-walled spores and short angular hyphae (spaghetti and meatballs)
Pityrosporum Folliculitis

- Infection of the hair follicle
- Slightly itchy follicular papules and pustules on upper torso, arms, and neck
- Oral ketoconazole best

Pityriasis (Tinea) Versicolor

- Superficial infection of the stratum corneum
- Hypo- or hyperpigmented, scaly, irregular macules and patches most commonly on the upper trunk
- Involved areas do not tan after sun exposure (metabolites of yeast cause depigmentation in inhibiting tyrosinase)
- Fluoresces yellow-gold

Tinea Versicolor Treatment

- Selsun, Exsel (selenium sulfide 2.5% lotion) applied daily for 10 minutes x 7 days
- Spectazole crm (1% econazole), Nizoral crm (2% ketoconazole), Loprox (Ciclopirox) cream, gel or lotion: qd-bid x 2 wks
- Consider oral treatments for extensive disease (Nizoral, Diflucan, Sporanox)
Deep Mycoses (Mostly Disseminated Fungal Infections)

- Most systemic mycoses come from inhalation of contaminated dust, animal droppings, or contamination from other sources.

- Nodules and ulcerations that are not chancriform should be considered to arise from an internal focus, usually the lungs or upper respiratory tract.

Deep Mycoses

Today’s focus will be on cutaneous features rather than the systemic manifestations.

Sporotrichosis

- Sporothrix schenckii typically causes disease localized to skin and subcutaneous tissues; rarely disseminated

- Most often seen in gardeners, florists, farmers, and laborers following injuries from plants or straw

- Dusky red, crusted plaque or nodule evolves to a painless chancre

- Draining lymphatics become inflamed and a chain of 2-5 "crusted nodules develops which may also ulcerate
Sporotrichosis

- Biopsy for histology not often helpful
- Submit tissue specimen for fungal culture
- Itraconazole 100-200mg/d for 3-6 months
- Amphotericin B for disseminated and meningeal forms

General Diagnostic Aids and Treatment Measures

- Serology for antibodies (blood, urine), KOH (potassium hydroxide), fungal culture (skin, blood, urine), biopsy (histology, fungal culture), CSF, lumbar puncture (India ink stains of cerebrospinal fluid)
- Systemic disease occurs most frequently in individuals with underlying immunocompromise; conduct appropriate workup as dictated by history and exam findings
- Generally speaking, treatment consists of oral itraconazole, ketoconazole, fluconazole, and intravenous amphotericin B
- Consultation with an infectious disease expert is recommended, with the exception of lymphocutaneous sporotrichosis

Cutaneous Viral Infections
Human papillomavirus (HPV)

- More than 150 genotypes are known to infect humans
- Cause disease ranging from warts to SCC of the skin and mucous membranes
- Incidence is highest in children and adolescents
- Direct contact, autoinoculation, and fomites spread HPV
- Warts obscure normal skin lines, may spread along lines of trauma, and have brown-black dots (thrombosed dermal capillary loops) - important diagnostic features

Verruca Vulgaris

- Caused most commonly by HPV types 2 and 4
- Well-defined keratotic papules with an irregular surface
- Most frequently on the fingers, dorsum of hands, paronychial areas, face, knees, and elbows

Plantar Warts

- Caused most commonly by HPV type 1
- Hyperkeratotic papules, often surrounded by callus
- Coalescing warts (HPV type 4) may form a large plaque referred to as a mosaic wart
Verruca Plana

- Most commonly caused by HPV types 3 and 10
- Slightly elevated, flat-topped, 1-4 mm pink to tan papules
- May occur in profusion on the face, arms, dorsum of the hands, knees, and lower legs
- May be very resistant to treatment; consider tretinoin 0.1%, Aldara, or cryotherapy

Treatment options

Observation – >60% or more disappear spontaneously within 2 years

OTC remedies
- salicylic acid and lactic acid preparations, home freeze kits
- Duofilm, Occlusal-HP, Trans-Ver-Sal, Mediplast
- Home freeze kits have no proven benefit

Home-Based Prescription Treatments
- Imiquimod (Aldara)
- podofilox 0.5% gel or soln (Condylox)
- fluorouracil 5% cream (Efudex)
- tretinoin crm or gel (Retin-A)

Office-Based Treatments
- Immunotherapy - Squaric acid dibutylester (SADBE), diphenylcyclopropenone (DCP), and dinitrochlorobenzene (DNCB)
- Destructive - Canthacur-PS or Verrusol, Trichloroacetic acid 85%, Podophyllum resin, Cantharidin 0.7% solution, cryotherapy, electrodesiccation and curettage, laser ablation, surgical excision, intralesional bleomycin

Molluscum Contagiosum

- Self-limited, benign epidermal viral infection caused by molluscum contagiosum virus (MCV), a poxvirus

- Spread by direct contact with an infected person, autoinoculation, or less commonly from fomites
- Discrete, dome-shaped, skin-colored or pearly papules often with central umbilication
- 10% KOH demonstrates a mass of homogeneous cells, often with identifiable lobules and is diagnostic
Molluscum Contagiosum - treatment

- Observation (usually resolves in 6-9 months; but scarring may occur)
- Physical modalities: cryotherapy, electrodecoagulation, manual extraction
- Topical & intralesional: cantharidin, imiquimod, salicylic & lactic acids, podophyllotoxin, retinoids
- Laser Therapy: CO2, pulsed dye
- Systemic Therapy: cimetidine, acyclovir

Nongenital Herpes Simplex (herpes labialis/cold sore/fever blister, herpes gladiatorum, herpetic whitlow)

- Most nongenital infections caused by HSV-1
- HSV-1 is transmitted chiefly by contact with infected saliva or other infected secretions (whereas HSV-2 is usually transmitted sexually or from mother to newborn)
- Readily inactivated at room temperature and by drying
- Some sources report up to 90% of 20-40 year olds have antibodies against HSV-1

Herpes Simplex

- Many first episodes are asymptomatic but localized pain, lymphadenopathy, fever, H/A, ulcers may occur within several days of exposure to viable virus
- Most common presentation is grouped, uniform vesicles on an erythematous base preceded by burning or tingling
- Virus enters the nerve endings and ascends to the dorsal root ganglia where it remains in a latent stage until reactivated
Laboratory

- Detection of HSV DNA with polymerase chain reaction of dried or fixed tissue, direct fluorescent antibody testing, viral culture, Tzanck smear.

Treatment:

- Oral antivirals (acyclovir, famciclovir, valacyclovir) - dose by age and primary vs. recurrent infection.
- Topical antiviral ointments (penciclovir oint 1%, docosanol oint 10% and acyclovir oint 5%) minimally beneficial.
- Comfort measures include NSABAs, wet dressings with 5% aluminum acetate (Burow's solution), or cool tap water.
- Suppressive therapy for > 6 recurrences/year.

Herpes Zoster

- During the course of varicella, varicella-zoster virus (VZV) passes from lesions into the cutaneous nerves and is transported to the dorsal root ganglia where a latent infection is established.
- Reactivation associated with HIV infection, local tumor or trauma, Hodgkin's disease, immunosuppressive drugs, emotional stress, and age (due to gradual decline in cell-mediated immune responses to VZV).
- Pain and paresthesia in the dermatome may precede the eruption by 2-3 weeks.

Herpes Zoster

- Erythematous macules develop into closely grouped vesicles and bullae on an erythematous base over 2-3 days; these dry and crust in 7-10 days, may be hemorrhagic.
- Lab: Tzanck smear, direct fluorescent assay (DFA), polymerase chain reaction (PCR).
Zoster - Treatment

- Oral antiviral in zoster doses
- Bedrest for several days in older patients
- Prednisone or tricyclic antidepressants if pain is severe
- Local comfort measures (see herpes simplex)

Zoster - Complications

Postherpetic neuralgia

- Pain persisting in a dermatome for months or years
- Tricyclic antidepressants
- Gabapentin 100-300 mg po tid
- Acetaminophen, NSAIDs, and opiate analgesics as required

- Topical agents may be helpful and include lidocaine 5% patches, EMLA, Zonalon crm, Zostrix (capsaicin) crm after healed completely
- Epidural and sympathetic blocks
- Surgery (rhizotomy) in extreme cases

Zoster - Complications

Ramsay Hunt syndrome (herpes zoster oticus)

- Infection of the geniculate ganglion of the seventh cranial nerve
- Syndrome includes unilateral facial paralysis, hearing deficits and vertigo, taste loss in the anterior 2/3 of the tongue, dry mouth and eyes, vesicles on the ear canal, tongue, and/or hard palate
Herpes Zoster Ophthalmicus

- Involvement of the ophthalmic branch of the trigeminal nerve
- >20% develop ocular complications
- Vesicles on the side or tip of the nose (Hutchinson’s sign)
- Treat with oral antiviral
- Consultation with Ophthalmologist required

Viral Exanthems

Roseola Infantum

- Age group 0-4 years
- Caused primarily by human herpes virus 6
- Prodromal symptoms include high fever for 3-5 days, diarrhea, cough
- Pale pink almond-shaped macules and papules on trunk, neck, and proximal extremities
Roseola Infantum

Erythema Infectiosum-fifth disease
- 5-17 years of age
- Caused by the B19 Parvovirus
- Prodromal symptoms-nonspecific fever and malaise
- Macular erythema on face (1 to 4 days), erythematous macular eruption for the first week followed by lacy erythema affecting the extremities, trunk, and buttocks

Erythema Infectiosum
Erythema Infectiosum

Hand, foot, and mouth disease
- Primarily caused by coxsackievirus A16 and enterovirus 71
- Children
- Prodromal symptoms—mild low-grade fever, sore throat, and malaise for 1-2 days
- Vesicular palmoplantar eruption and erosive stomatitis

Measles (rubeola)
- 0-20 years
- Prodromal symptoms—rhinitis, cough, fever, conjunctivitis, Koplick’s spots
- Erythematous macules and papules later become confluent which begins at the hairline on neck and face, moves down and covers entire body
Measles

German measles (rubella)
- 5-25 years
- Prodromal symptoms- mild URI symptoms
- Generalized maculopapular becomes pinpoint, begins of face and migrates to trunk

Arthropod Bites and Infestations
Arthropod Assaults

- There are a number of insects that cause skin injury - mites, ticks, spiders, scorpions, centipedes, lice, mosquitoes, bedbugs, bees, wasps, caterpillars, fleas, etc.
- Injury mechanisms include trauma, invasion, contact dermatitis, granulomatous reactions, disease transmission, injection of irritating or harmful substances, and anaphylaxis
- Skin reaction depends on the species of insect as well as the reactivity of the host.

Arthropod Assaults – General Treatment Recommendations

- Oral antihistamines, cool compresses, topical steroids, topical antipruritics (e.g., camphor, menthol)
- Topical anesthetics (pramoxine)

- Severe reactions may require systemic steroids

- An effort should be made to identify and eradicate the etiologic agent, use of repellents

Flea Bite

Small urticarial papule with bite punctum, often grouped around the ankles or lower legs
Mosquitoes
- Initially minimal or no response to a bite
- Additional bites lead to a persistent papule or wheal within 24 hr (delayed hypersensitivity)
- Further exposure results in an erythematous wheal within minutes (immediate hypersensitivity), followed by a papule
- Eventually only a wheal may form with the person ultimately becoming insensitive to the bite

Bedbugs
- Blood sucking, feed at night, and produce rows of papules & wheals

Stings
- Bees, hornets, wasps
- Pain followed by erythematous papule or urticarial reaction
- Anaphylaxis in sensitized individuals
- Remove stinger by gently scraping away, wash with antibacterial soap and water, ice, elevate extremity, oral antihistamines, oral analgesics (ibuprofen), tetanus booster if needed

Harvest mites (chiggers, red bugs)
- Pruritic papules and urticarial lesions occur on the ankles, legs, and where constrictive clothing is worn
Scabies

- Caused by host-specific itch-mite Sarcoptes scabiei var. hominis, which lives its entire life within the epidermis.
- Reaction caused by host’s response to the burrowing of the female mite.

- Isolated mites die within 2-3 days.
- Night pruritus.
- Erythematous papules, threadlike burrows (classic pathognomonic lesion of scabies), vesicles, and excoriations develop in the interdigital spaces, wrist flexors, anterior axillary folds, buttocks, umbilicus, genitals in men, and areolas in women.
- Less frequently lesions consist of bullae, pustules, wheals, red-brown nodules, or a generalized eczematous dermatitis.
- Face, scalp, palms, and soles are often involved in infants.
- Diagnosis confirmed by microscopic identification of mites, feces (scybala), or eggs.

Scabies Treatment

- Permethrin 5% cream (Elimite, Acticin).
- Sulfur in petrolatum (6-10%).
- Crotamiton (Eurax).
- Ivermectin (Mectizan, Stromectol) 200 mcg/kg twice.
Scabies

- Topical corticosteroid preparations and antihistamines for severe pruritus.
- Clothing, bed linens, and towels should be hot laundered or set aside for one week in sealed containers/room.

Norwegian or Crusted Scabies

- Variant occurring mainly in debilitated individuals or those infected long term (immune response).
- Crusts and exfoliating scales of the skin and scalp, dystrophic nails may be seen.
- Generalized lymphadenopathy and eosinophilia may occur.
- Isolation measures, scale removal, repeated applications of antiscabietics (not lindane), and consideration of ivermectin.

Pediculosis

- Three types of lice are parasites of the human host:
  1. Body or clothing lice (Pediculus humanus corporis)
  2. Head lice (Pediculus humanus capitis)
  3. Pubic or crab lice (Phthirus pubis)
- Nits are glued to hairs or fibers of clothing and hatch in 1-2 weeks.
- Symptoms develop as an individual becomes sensitized.
- Hallmark of disease is pruritus.
- Pyoderma from scratching may result in crusts, lymphadenopathy, or hair loss.
**Pediculosis corporis**

- Pruritic red macule or papule with a central hemorrhagic punctum commonly found on the shoulders, trunk, or buttocks.
- Chronic infestation manifests as "vagabond's skin" (lichenified, scaling, hyperpigmented plaques).
- Lice are found on the skin only during feeding; otherwise, they inhabit the seams of clothing where they attach their nits.
- Improved hygiene and hot water laundering of clothing and bedding.

**Pediculosis capitis**

- Infestation in the scalp hair.
- Head-to-head contact is the major mode of transmission.
- Lice are not always visible, but nits are detectable on the proximal hair shaft.
- Bites result in pruritic papules and wheals.
- Nits can be removed with a fine-toothed comb after a 1:1 vinegar:water rinse.

**Pediculosis Pubis or Crab Lice**

- Transmitted by skin-to-skin or sexual contact.
- Infestation may involve other hair-bearing sites (beard, eyelashes, axillae, perianal region).
- Coexistence of other STIs should be considered.
Eyelash infestation (Pediculosis palpebrarum)

- Seen almost exclusively in children
- Can be acquired from other children or from an infested adult with pubic lice (may be a sign of sexual abuse)
- Apply petroleum bid x 10 days

Treatment

- Permethrin 1% crm rinse (Nix)
- Synergized pyrethrins (RID Mousse, RID Shampoo, R&C, A200)
- Malathion (Ovide)
- Lindane (Kwell shampoo/lotion)
- Oral ivermectin or trimethoprim/sulfamethoxazole (Septra, Bactrim DS)
- Householders' members or sexual contacts should be treated; clothing, towels, brushes, and bed linens should be washed in hot water or sealed for 2 weeks

Spider Bite

- Spiders are carnivorous arthropods with fangs and venom.
- Most venoms consist of an enzyme-spreading factor and a toxin. The majority of toxins simply cause pain and inflammation.
- About 50 species in the U.S. have been known to bite humans.
- Only the black widow, hobo, and brown recluse cause any significant morbidity.
Common Spider Bites

- Most bites cause pain and an urticarial reaction consisting of warmth and deep erythema; two puncta may be evident
- Resolves spontaneously
- Treat with cool compresses and antihistamines PRN

Black Widow Spider (Latrodectus mactans)

- Found in every state except Alaska but most numerous in the rural South
- Smooth black body with red hourglass marking on the underside of the abdomen; females reach 4 cm in size
- Neurotoxin in venom releases acetylcholine from neuromuscular junctions of sympathetic and parasympathetic nerves
- Bite usually causes immediate sharp pain followed by burning
- Produces redness and edema surrounding a small set of fang marks

Latroderctism

Systemic reaction to black widow bite characterized by:
- regional muscle spasms or generalized abdominal, back, and leg pain most common presenting complaints
- severe abdominal pain and spasm simulating a surgical abdomen
- deep tendon reflexes may be increased
- other symptoms include dizziness, HA, sweating, NV, weakness, salivation, slight rise in temperature
- convulsions, paralysis, and shock occur in 5%, mortality <1%

Treatment:
- antivenin, calcium gluconate for muscle spasms
- healthy patients between 16-60 years of age usually respond to muscle relaxants, analgesics, ice, and elevation and recover spontaneously
Brown Recluse Spider Bites

- *Loxosceles reclusa*
- Found primarily in the southern central states of the midwestern region of the US
- Tan to dark brown body with fiddle-shaped marking on the back
- Bite often produces stinging or burning
- Most reactions only cause mild swelling and erythema

Loxoscelism

Localized Reactions

- Blue-gray cyanotic macular halo (local hemolysis) with a pustule or vesicle at the bite site
- Pallor with a red ring surrounding the area gives the appearance of a “bull’s-eye” (red, white, and blue sign)

Systemic Reactions

- SIRS – sepsis, shock, coma, and fever
- Wheals – chills, fever, arthralgia, petechiae, generalized
- Skin – epidermolysis, necrosis, and hemorrhage
- Mental status: mental failure, secondary infections, shock and death

Brown Recluse Bites - Management

- Most should be treated conservatively with ice, elevation, rest, antibiotics, ASA, Tetanus toxoid if necessary
- Surgical excision only for stable lesions as needed for cosmesis
- Steroids for severe skin lesions, loxoscelism, and in small children
- Dapsone only in adult patients who have been screened for G6PD deficiency
- Topical nitroglycerin can be of value in decreasing the enlargement of necrotic skin ulcers
Acne Vulgaris

- Acne is a disease of the pilosebaceous unit
- The disease may be mild with only few comedones or papules, or it may occur as the highly inflammatory and diffusely scarring acne conglobata
- Acne is too often dismissed as a minor affliction
- Permanent scarring of the skin and psyche can result from postponing treatment

Acne Vulgaris

- Medications –
  - Retinoids
  - Benzoyl Peroxide
  - Topical antibiotics
  - Oral antibiotics
  - Isotretinoin-Accutane
  - Antiandrogens (Oral Contraceptives, Spironolactone)
Keloids

NEVI
- Junctional- flat to slightly elevated, light brown to brown black, nevus cells at dermo-epidermal junction, generally develop after the age of 2.
- Compound- elevated, brown or flesh colored, nevus cells at the dermo-epidermal junction and upper dermis,
- Intradermal- elevated, brown or flesh colored, nevus cells in the dermis and sometimes fat cells

Compound Nevus
Intradermal Nevus

- Also referred to as atypical nevi.
- Usually larger than 5mm, flat to slightly raised, darkly or irregularly pigmented with shades of brown and pink.
- Atypical nevi are a marker for patients at an increased risk for development of melanoma.
- Dysplastic Nevi are common.

Dysplastic Nevi

- Most experts believe that dysplastic nevi are at higher risk of changing into melanoma as compared to normal moles.
- A dysplastic nevus has features that are in between a normal mole and a melanoma.
- Pathologist might classify a dysplastic nevus as having mild, moderate, or severe atypia.
Dysplastic Nevi

- These lesions should be observed for changes. If lesions are changing they should be removed.
- No data are available to assess what effect prophylactic removal has on decreasing the risk for future melanoma.
Dysplastic Nevi

Melanoma

- A malignancy of melanocytes that occurs in the skin, eyes, ears, gastrointestinal tract, oral and genital mucous membranes.
- One of the most dangerous tumors, melanoma has the ability to metastasize to any organ.
- Melanoma either begins de novo or develops from a preexisting lesion, such as a congenital or atypical mole.
Melanoma

- Risk factors include:
  - Personal history of atypical moles.
  - Family history of melanoma.
  - Previous nonmelanoma skin cancer.
  - Congenital nevus >20cm.
  - History of melanoma.
  - Immunosuppression
  - Large number of nevi.

Melanoma

- Risk factors:
  - Chronic tanning
  - Repeated blistering sunburns.
  - Freckling
  - Fair skin
  - Inability to tan

Melanoma

- The goal is to recognize melanomas at the earliest stage.
- A patient’s description of a change in a mole may be the earliest sign of melanoma.
- ABCDs- Asymmetry, Border irregularity, Color variation, and Diameter enlargement.
Melanoma

- If a suspicious lesion is found, whenever possible, excise the lesion for diagnostic purposes using narrow margins.
- A punch biopsy is appropriate when the suspicion is low, when the lesion is large, or when it is impractical to perform an excision.
- Shave biopsies are discouraged, they may not provide an accurate depth measurement.

Melanoma

- Treatment is wide excision.
- When in doubt always biopsy.
Melanoma

- The most common malignant cutaneous neoplasm found in humans.
- This tumor rarely metastasizes.
- Most tumors appear on the head and neck region.
- BCC is rarely found on the dorsal hands.
- Tumors also occur in sites protected from the sun.

Basal Cell Carcinoma
Basal Cell Carcinoma
- The tumor may occur at any age, but the incidence increases markedly after age 40.
- The course of BCC is unpredictable.
- BCC may remain small for years with little tendency to grow, or it may grow rapidly.

Nodular Basal Cell Carcinoma

Nodular Basal Cell Carcinoma
Nodular Basal Cell Carcinoma

Pigmented Basal Cell Carcinoma

Superficial Basal Cell Carcinoma
Superficial Basal Cell Carcinoma

- The second most common cancer among Caucasians.
- Unlike Basal Cell Carcinoma, cutaneous SCCs are associated with a risk of metastasis.
- Risk factors include exposure to sunlight during childhood, sunburns, light skin, hazel or blue eyes, blonde or red hair.

Squamous Cell Carcinoma

- The second most common cancer among Caucasians.
- Unlike Basal Cell Carcinoma, cutaneous SCCs are associated with a risk of metastasis.
- Risk factors include exposure to sunlight during childhood, sunburns, light skin, hazel or blue eyes, blonde or red hair.

Squamous Cell Carcinoma

- Renal transplant patients have a 253 fold increase in the risk of SCC.
- Lesions are most common on the scalp, dorsal hands, and helix of the ear.
- Basal Cell Carcinoma is rarely found on these sites.
- A Squamous Cell Carcinoma in situ is called Bowen’s Disease.
Squamous Cell Carcinoma

- Clinical Presentation will vary
- Lesions may have a thick, adherent scale with an erythematous base.
- Lesions may have more of a cutaneous horn with an erythematous base.
- Others may have a very thin scale with an erythematous nodule as the base.
- Lesions on the scalp, forehead, ears, nose, and lips are at higher risk for metastasis.
Squamous Cell Carcinoma

References
Images

- National Library of Dermatologic Teaching Slides 4.0, American Academy of Dermatology