Dermatology Exam Notes

Introduction to Dermatology

☐ Structure of the Skin

I.) Epidermis: outermost
- layers:
  • stratum corneum: flat dead cells that are 8-15 layers thick
  • stratum granulosum: transitional layer
  • stratum spinosum: differentiating tissue
  • stratum basale: mitotic tissue
- cells:
  - most are keratinocytes that produce keratin and have immune function role
  - melanocytes: also found in hair follicles, brain, and eyes
    - same number in all races
  - Langerhans cells: APCs

II.) Dermis
- layers:
  • papillary dermis:
  • reticular dermis: contains blood vessels, hair follicles, sebaceous glands, muscle, sweat glands
    - eccrine glands for thermoregulation
    - apocrine glands for scent
- cells: fibroblasts for collagen synthesis, mast cells, macrophages

III.) Subcutaneous fat

☐ Lesions

<table>
<thead>
<tr>
<th>Primary lesion type</th>
<th>Info</th>
<th>Example</th>
<th>Picture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Macule</td>
<td>Flat, nonpalpable, &lt; 1 cm</td>
<td>vitiligo</td>
<td><img src="image1" alt="Vitiligo Image" /></td>
</tr>
<tr>
<td>Patch</td>
<td>Macule &gt; 1 cm</td>
<td></td>
<td><img src="image2" alt="Patch Image" /></td>
</tr>
<tr>
<td>Papule</td>
<td>Raised, palpable, &lt; 1 cm</td>
<td>BCC, psoriasis, seborrheic keratosis</td>
<td><img src="image3" alt="Papule Image" /></td>
</tr>
<tr>
<td>Plaque</td>
<td>Papule &gt; 1 cm</td>
<td></td>
<td><img src="image4" alt="Plaque Image" /></td>
</tr>
<tr>
<td>Vesicle</td>
<td>Raised, contains clear liquid, &lt; 1 cm</td>
<td>Chicken pox</td>
<td><img src="image5" alt="Vesicle Image" /></td>
</tr>
<tr>
<td>Bulla</td>
<td>Vesicle &gt; 1 cm</td>
<td></td>
<td><img src="image6" alt="Bulla Image" /></td>
</tr>
<tr>
<td>Pustule</td>
<td>Raised, contains inflammatory cells and fluid, variable size</td>
<td>acne</td>
<td><img src="image7" alt="Pustule Image" /></td>
</tr>
<tr>
<td>Nodule</td>
<td>Raised, solid, deeper than a papule, &lt; 1 cm</td>
<td>Metastatic melanoma</td>
<td><img src="image8" alt="Nodule Image" /></td>
</tr>
<tr>
<td>Tumor</td>
<td>Nodule &gt; 1 cm</td>
<td></td>
<td><img src="image9" alt="Tumor Image" /></td>
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</tbody>
</table>
**Wheal**
Firm, edematous papule or plaque that contains bound fluid, flat-topped elevations, transient

<table>
<thead>
<tr>
<th>Secondary lesion type</th>
<th>Info</th>
<th>Example</th>
<th>Picture</th>
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</thead>
<tbody>
<tr>
<td>Scale</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Crust</td>
<td>Collection of serum, blood, or pus</td>
<td></td>
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<tr>
<td>Erosion</td>
<td>Focal loss of epidermis that heals without scarring</td>
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<tr>
<td>Ulcer</td>
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<tr>
<td>Fissure</td>
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<tr>
<td>Atrophy</td>
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<table>
<thead>
<tr>
<th>Special skin lesions</th>
<th>Info</th>
<th>Example</th>
<th>Picture</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comedone</td>
<td>Blackheads and whiteheads; pathognomonic for acne</td>
<td></td>
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</tr>
<tr>
<td>Milia</td>
<td>Small, superficial keratin cyst from sun damage</td>
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<tr>
<td>Cyst</td>
<td>Has a visible opening</td>
<td></td>
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<tr>
<td>Burrow</td>
<td>Narrow, elevated tunnel from a parasite</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lichenification</td>
<td>Thickening of the skin</td>
<td></td>
<td></td>
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<tr>
<td>Telangectasia</td>
<td>Dilated superficial skin vessels</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Petechiae</td>
<td>Nonblanchable blood deposit &lt;1 cm</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Purpura</td>
<td>Petechiae &gt; 1 cm</td>
<td></td>
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</table>

- **Diagnostic Procedures**
  - Magnification
    - Diascopy: vascular lesions
  - Special preps
    - mineral oil: good for scabies
    - KOH: good for fungus & yeast, always scrape border of lesion
    - Tzanck smear: helpful for virus detection in vesicle fluid, looks for multinucleated giant cells
    - Gram stain
  - Biopsy
  - Patch testing: for suspected contact allergies (type IV delayed-onset hypersensitivity)
  - Wood’s lamp: UV light with nickel oxide filter detects certain porphyrin-producing organisms
    - some tinea capitis agents fluoresce green
    - erythrasmas (Corynebacterium minutissimum) fluoresce coral red
    - vitiligo fluoresces white
    - porphyria cutanea tarda fluoresces pink or orange
  - Acetowhiteness: vinegar turns dead skin white
Common Bacterial Skin Infections

CA-MRSA
- Infections by his are on the rise
- Clinically and microbiologically different from hospital MRSA
  - Presentation is dermatologic
  - More commonly in pediatric and non-white patients (Native Americans, blacks), homeless, populations in close quarters, daycare kids, competitive athletes
- Investigation:
  - Lesions may look more purpley
  - Must culture and do sensitivities
- Treatment: I&D, +/- antibiotics for large lesions, peds, pts with systemic symptoms (Septra, clinda, linezolid)

Common Bacterial Skin Infections
I.) Superficial infections:
  • Impetigo: scabbing eruption with lesions that can be bullous (clear or turbid fluid) or non-bullous (honey-crusted erosions with erythema)
    - Agents: caused by Strep pyogenes or Staph aureus
    - Subtypes:
      • Ecthyma: a deep type of impetigo that extends into the dermis; ulcerative with a thick, tender yellow-gray crust
        - Agents: Strep, Pseudomonas, Staph
      - Children and elderly at risk
    - Risk factors: trauma, underlying dermatosis (atopic dermatitis or herpes), poor hygiene, previous antibiotics, warm temps, high humidity
    - New lesions by auto-inoculation
    - Treatment: topical mupirocin (MRSA) or Altabax (MSSA), systemic cephalosporins or dicloxacillin
  • Intertrigo: nonspecific infection of rubbing skin surfaces
    - Agents:
      - Bacterial: Strep pyogenes, Strep agalactiae, Pseudomonas
        - Borders are more defined, no satellite lesions
        - Neck folds in babies = Strep
        - If caused by Strep it will really stink!
      • Erythrasma: a type of bacterial intertrigo caused by Corynebacterium minutissimum
        - Common in the groin, also in feet webs, axillae
        - Brownish color
        - Diabetics at increased risk
        - Bright red with Wood’s lamp
        - Treatment: topical benzoyl peroxide, mupirocin, +/- imidazoles, systemic doxycycline, macrolides
      - Yeast: highly inflammatory
        - Confused with psoriasis, seborrheic dermatitis, atopic dermatitis
        - Treatment: topical antibiotics, steroids?

II.) Pyodermas
- Types:
  • Abscess: localized, wall-off pus collection that progresses from firm to fluctuant
    - Can develop at any cutaneous site
  • Furuncle: deep-seated erythematous nodule with a rim of fine scale that develops a central necrotic plug
    - Aka epidermal inclusion cyst (technically NOT a sebaceous cyst)
    - Common in hairy areas or areas of friction
  • Carbuncle: large area of coalescing abscesses or furuncles
    - Risk factors: trauma, chronic Staph carrier, diabetes, obesity, poor hygiene, minor immunologic deficits
-prevention: antibacterial soaps daily, monthly Betadine or Hibiclens showers, control of predisposing conditions
-treatment: I&D with removal of loculations (may need to wait and do compresses until it is soft), +/- systemic antibiotics (shouldn’t need them)

III.) Soft tissue infections
-types:
  • cellulitis: infection of deep dermal and subcutaneous layers with indistinct borders
    -agents: *Strep pyogenes*, *Staph aureus*
    -affects any cutaneous site
  • erysipelas: a more superficial type of cellulitis characterized by sharp, raised borders with clear demarcation from uninvolved skin
    -caused almost exclusively by *Strep pyogenes*
    -LE and face most affected
-risk factors: trauma, surgery, mucosal infection, underlying dermatoses, immunodeficiency
-presentation: acute, diffuse skin inflammation with warmth and tenderness, may have systemic symptoms
-treatment: therapy must be systemic!
  -penicillinase-resistant synthetic pencillins (cloxacillin, dicloxacillin) or macrolide if allergic
  -supportive: rest, elevation, warm compresses

IV.) Infectious folliculitis: infection of upper portion of hair follicle
-subtypes:
  • pseudofolliculitis barbae: aka barber’s itch, a result of foreign body reaction to ingrown hairs
  • keloidal folliculitis: found at nape of neck, often coalesces into furuncles over months to years
    -more common in blacks
    -treat with cyclic antibiotics
  • hot tub folliculitis: caused by *Pseudomonas*, short incubation of 1-5 days
    -lesions clear spontaneously
-agents can be fungal, viral, or bacterial
  -bacterial: most commonly *Staph aureus*, also gram negatives, *Pseudomonas*
    -evolves into pyodermas
-risk factors: shaving, friction, immunosuppression, topical steroids
-prevent with antibiotic soaps
-presentation: single, scattered papules or pustules that are not tender or pruritic
  -predilection for the face, scalp, neck, legs, trunk, and buttocks
-treatment: usually a 7-10 day course of oral antibiotics that cover *Staph = 1* gen cephalosporin or macrolide if allergic

Less Common Bacterial Skin Infections
A.) Erysipeloïd: single plaque with sharp borders usually seen on the hand
- caused by *Erysipelothrix rhusiopathiae* and usually seen in poultry/fish/animal carcass handlers
B.) Necrotizing soft tissue infections: continuum of disease that begins with soft tissue infection and progresses to multi-organ failure
-agents: usually polymicrobial, can be *Strep pyogenes*, *Staph aureus*, *Clostridium*, *Bacteroides*, *Peptostreptococcus*, *Enterobacter*, *Proteus*, *Pseudomonas*
-types:
  • erythroplakia gangrenosum: caused by *Pseudomonas*
  • Fournier’s gangrene: perineum, caused by *E. coli*, *Klebsiella*, *Proteus*, or *Bacteroides*
    -male predominance
-infection initiated by an insect bite or surgical wound, then spreads hematogenously
  -common sites: perineum, extremities, trunk
-risk factors: PVD, impaired cellular immunity (DM), IVDU, smoking, alcohol, HTN, CAD, chronic steroids, lymphedema, varicella lesions, genital trauma or infection
-presentation: pain out of proportion to physical findings

C.) Acute lymphangitis: infection of subcutaneous lymphatic channels usually from trauma → erythematous linear streaks extending from the wound to site of skin break
- agent: usually *Strep* *pyogenes*, less commonly *Staph* *aureus*, rarely *Pasteurella multocida*, mycobacteria, *Sporothrix*
- treat with antibiotics to cover *Strep* and *Staph*

D.) Cutaneous anthrax
- acquired from infected animals or their products, contaminated feed, soil
- begins as papulovesicular lesion → necrosis → eschar-covered ulcer
- treat with cipro or doxycycline

E.) Cutaneous mycobacterial infections
- single nodule that ulcerates or crusts, joined to other nodules by satellite lesions
- lesions usually clear spontaneously but excision may help
- anti-TB drugs not helpful, these are different species

F.) *Vibrio* infections
- macular area develops into bullous lesions that are often symmetric → necrotic ulcers
- initial cellulitis doesn’t look any different from regular cellulitis
- skin disease can develop secondary to enteric infection
- at risk: immunocompromised, high serum Fe, occupational exposure to fish, seafood, brackish water

**Fungal and Viral Infections of the Skin**

- **Superficial Fungal Infections**
  1.) Dermatophytoes: infections of the hair/nails by keratin-loving fungi
  - dermatophytes: *Microsporum*, *Trichophyton*, *Epidermophyton*
  - the most common skin fungal infections
  - risk factors: atopy, steroid use, dry skin, occlusion, high humidity
  - transmission: person-to-person, fomites, animal-to-human, environmental
  - infections are known clinically as “ringworm” or “tinea + ____”
    - **tinea pedis**: foot infection characterized by erythema, scaling, vesicles, maceration in web spaces
      - risk factors: increased sweating, shoes, contaminated public floors
      - more common in males
      - multiple presentations:
        - can have involvement of toenails or bacterial 2° infection
        - interdigital
        - “moccasin” with involvement of heels, soles, lateral foot
        - inflammatory/bullous with erupting vesicles
    - treatment:
      - topical: 2-4 weeks of imidazoles or allylamines
      - oral: for severe or refractory cases; 2-6 weeks of same agents
      - exposure to air
    - **tinea cruris**: aka jock itch, subacute or chronic infection of the groin and medial thighs with erythematous, scaling, well-demarcated plaques
      - risk factors: obesity, tight clothing
      - investigation: differentiate from candidiasis and erythrasma
      - treat topically
    - **tinea corporis**: subacute infection of the neck, trunk, or extremities with sharp-bordered plaques of varying sizes possibly with smaller pustules or vesicles within the border
      - lesions enlarge peripherally and may have an area of central clearing
      - treat topically
    - **tinea capitus**: scalp infection; ecto/endothrix depending on penetration of hair shaft, can see broken-off hairs resembling black dots or *kerion* (swollen, painful nodule)
      - most common in kids 6-10
-treatment: the only dermatophytic infection that must be treated systemically, needs both steroids and 6-12 weeks of antifungals (DOC terbinafine- dermatophytes only, not Candida) if kerion is present
-can add topical ketoconazole or selenium sulfide to reduce transmission

II.) Candidiases
a.) cutaneous/intertriginous
- infections involve sites where maceration and occlusion create a warm, moist environment
- breasts, abdominal folds, axillae, groin, web spaces, angular cheilitis, diapers
- risk factors: obesity, diabetes, hyperhidrosis, steroids
- treatment: nystatin (Candida only), imidazoles, steroids sparingly
b.) mucocutaneous
c.) nail
d.) systemic

III.) Tinea (pityriasis) versicolor: opportunistic infection caused by Malassezia furfur → multiple well-demarcated, hyper- or hypopigmented macules with fine scaling
- white, pink, shades of brown
- upper trunk, axillae, groin, thighs, face, neck scalp
- stimulated by oils, greasy cosmetics
- investigation: characteristic “spaghetti and meatballs” under microscope
- treatment: topical selenium sulfide, imidazoles, oral ketoconazole (single dose sweaty workout style)
- lesions may persist for some time even after treatment

Invasive/Subcutaneous Fungal Infections
- Background:
  - usually transmitted via traumatic implantation
  - agents are saprophytes living in soil and vegetation in warm climates
    - Sporothrix, Exophila, Fonsecaea, Madurella, Pseudallescheria
  - infections progress slowly
  - suspect in patients with outdoor habits with persistent lesions unresponsive to antibiotics

Viral Infections of the Skin and Mucous Membranes
- Viral exanthems: skin eruptions secondary to systemic infection
  - agents: rubeola (measles), rubella (German measles), varicella, roseola (sixth disease), erythema infectiosum (fifth disease)
    - similar manifestations can also be seen in Strep pyogenes, Staph aureus, and meningococcal infections
  - common in kids and adolescents, whereas adults have immunity to many of these infections
  - presentation:
    - rash may be preceded by a prodrome of fever, malaise, sore throat, nausea, vomiting, abdominal pain
    - may be accompanied by oral lesions (“enanthems”)
- Hand-foot-mouth disease: common systemic viral illness characterized by oral lesions and a vesicular exanthem limited to the distal extremities
  - agent: Coxsackie virus and some other enteroviruses
  - epidemics every 3 years
- Non-genital herpes simplex
  - herpes labialis: aka cold sores, fever blisters; grouped vesicles on an erythematous base
    - usually HSV-1 but can be HSV-2
    - usually perioral but can be anywhere on the face and auto-inoculate to other areas
    - treatment: penciclovir, acyclovir, valacyclovir
  - herpes whitlow: herpes on the fingertip
  - herpes gladiatorum: herpes anywhere else on the external body
    - complications: systemic symptoms, conjunctival autoinoculation, Bell’s palsy, erythema multiforme, eczema herpeticum, severe infection in immunocompromised
- Varicella-zoster virus
  - prevention: Zostavax vaccine

- Varicella-zoster virus
  - prevention: Zostavax vaccine
-single dose indicated for patients over 60
-primary infection is varicella, secondary is zoster (shingles)
treatment: high dose acyclovir within 72 hours of onset
-prednisone if over age 50
-analgesics
-complications: post-herpetic neuralgia, pain at site of shingles for years, ophthalmic complications, hemiplegia
-Molluscum contagiosum: distinct flesh colored or pearly white papules with umbilicated centers
-agent is a poxvirus
-common in children and sexually active adults
-transmission is skin to skin
-treatment: lesions usually resolve spontaneously but can take a year, otherwise cryotherapy
-HPV warts: see warts lecture

**Contact Dermatitis**

-**Background**
- An eczematous dermatitis caused by exposure to environmental substances
  - substances are either irritants (not immunologically mediated) or allergens (type IV hypersensitivity)
    - irritants: organic solvents, soaps
      - high concentration required for response
      - onset is gradual
      - rash borders are indistinct (unless acute?)
    - allergens: low molecular weight haptns
      - low concentration
      - onset is rapid with sensitization
      - defined borders
-May require sunlight acting on substance to cause the dermatitis
-Presentation:
- inflammation is acute, subacute, or chronic
- occurs in characteristic distribution
-Investigation:
- distinguish irritant from allergic dermatitis by provocation tests
  - apply substance to antecubital fossa twice daily for a week
    - contact urticaria 15-30 min after application suggests allergic etiology
  - patch testing
    - indicated when dermatitis is chronic, recurrent, or deters work or life activities
    - test site should be free of dermatitis
    - patch stays on for 48 hours and is read at 72-120 hours:
      + = doubtfull reaction: mild redness only.
      + = weak, positive reaction: red and slightly thickened skin.
      ++ = strong positive reaction: red, swollen skin with individual small water blisters.
      +++ = extreme positive reaction: intense redness and swelling with coalesced large blisters or spreading reaction.
      IR = irritant reaction. Red skin improves once patch is removed.
      NT = not tested.
- these test for type IV hypersensitivities rather than type I (which scratch skin tests check for)
-complcated by people with “angry back syndrome” (skin hypersensitivity → nonreproducible testing results)
- re-test with serial dilutions
  - dilution of irritants will reduce reaction while dilution of allergens will not
-histology is not helpful
-Treatment:
- allergen avoidance
- topical or systemic steroids
- emollients or other barriers
-oral antihistamines
-UVB and PUVA to help response subsite

- Irritant Contact Dermatitis
- Accounts for 80% of cases of dermatitis
- Common irritants: water, soaps, detergents, wet work, solvents, greases, acids, alkalies, fiberglass, dusts, humidity, chrome, trauma (chronic lip licking)
- Acute: occurs within minutes to hours of exposure, accompanied by pain, burning, stinging, or discomfort exceeding itching
  - acids, alkalies
  - involves tissue destruction
  - presentation: bullae and erythema with sharp borders
- Chronic: onset within 2 weeks of exposure, with many people in the same environment similarly affected
  - low-level irritants
  - presentation: poorly-demarcated erythema, scale, pruritus
- Presentation:
  - dry, fissured, thickened skin, usually palmar
  - macular erythema, hyperkeratosis, or fissuring predominating over vesicular change
  - glazed, parched, or scalded-appearing epidermis
  - not likely to spread
  - vesicles juxtaposed closely to patches of erythema, erosions, bullae, or other changes
- Investigation:
  - differential: dermatophytosis, psoriasis, pustular psoriasis, dyshidrosis-pompholyx, atopic dermatitis, pustular bacterid, keratoderma climactericum
  - negative patch test
  - healing process proceeds without plateau upon removing of the offending agent

- Atopic Dermatitis
  - an inflammatory, chronically relapsing, non-contagious and pruritic skin disorder
  - Cause uncertain, could be a variety of factors
  - Seen in patients with a history of environmental allergies, asthma, eczema
  - Investigation:
    - patch test to look for pustular reactions

- Allergic Contact Dermatitis
  - Common allergens:
    - metallic salts
    - plants
      - allergens from *Compositae* plant family: parthenolide, tansy, feverfew, pyrethrum
      - can also be airborne
    - fragrances: balsam of Peru, fragrance mix, benzyl alcohol, benzaldehyde
    - nickel has cross-reactivity with chocolate, tea, and whole wheat
    - cross-reactivity between similar antigens: poison ivy ~ mango ~ poison oak ~ lacquer tree ~ cashew shell ~ Indian marking nut ~ black varnish tree
    - preservatives: methylchloroisothiazolinone, quaterium-15, bronophol, imidazolidinyl urea, diazolidinyl urea, DMDM hydantoin
    - formaldehyde in wrinkle-free clothing, especially cotton and rayon
    - propylene glycol: also in brownie mix
    - oxybenzone: in moisturizers, cosmetics, shampoos, nail polish, lip sticks
    - bacitracin and neomycin (and possibly gentamycin and other –mycins)
    - thiuram: in rubber gloves, insoles, box toes, lining, adhesives, disinfectants, germicides, insecticides, soaps, shampoos
    - mercaptobenzothiazole: in rubber shoes, cutting oil, antifreeze, greases, cements, detergents, flea and tick sprays
    - bleached rubber: plain rubber is ok, but once it gets bleached you better watch out!
    - chrome: leather tanning agents
    - sorbic acid (food preservative)
-Causes a T-cell mediated response to kill cells
-Accounts for 20% of cases of contact dermatitis
-Presentation:
  -acute: macules, papules, vesicles, bullae
  -chronic: lichenified, scaling, fissured lesions
  -uncommon on scalp, palms, soles, or other thick-skinned areas that allergens can’t get through
  -uncommon to have mucous membrane involvement
  -systemic with an oral or IV agent on top of previous topical sensitization
  -painful, dry, fissured, hyperkeratotic eczema beginning at nail margin
  -associated with tuliaposide A allergen from *Alstroemeria* (Peruvian lily)
  -nickel can penetrate rubber gloves
  -parabens can be used on unaffected body parts without producing a dermatitis reaction on affected body parts
  -sorbic acid allergy will not improve with topical steroids because the acid is present in the steroid cream
-Investigation:
  -nickel allergy: dimethylglyoxime test using sweat, pressure, and friction with a nickel-plated object
  -parabens can have a false negative on patch test
  -bleached rubber allergy will have a negative patch test to plain rubber

- **Contact Urticaria**
  -Strongest inciting agents: benzoic acid, sorbic acid, cinnamic acid or aldehyde, foods (chicken, fish, veggies), topical meds
  -Can lead to contact dermatitis with repeat exposures

- **Latex Hypersensitivity**
  -Not a contact dermatitis because it is a type I (immediate onset) hypersensitivity, with symptoms within 1 hour of exposure
  -Risk factors: atopy (previous sensitization), frequent glove use, hand eczema, childhood surgeries
  -Presentation: skin as well as respiratory and mucosal symptoms

- **Common Inflammatory Dermatoses**

- **Papulosquamous Diseases**
  A. **Psoriasis**: a systemic, immunologic, genetic disease manifesting in the skin +/- joints
  -a hyperproliferation of the epidermis with altered differentiation → inflammation of the epidermis and dermis with accumulation of T-cells and cytokines
  -flared by streptococcal infections, injury, trauma, drugs (Li, beta-blockers, antimalarials, systemic steroids), low humidity, emotional stress, overtreatment
  -presentation:
    -lesions are most commonly on the scalp, elbows, legs, knees, arms, trunk, lower body, palms and soles
    -occur at sites of trauma
    -red scaling papules coalesce into round-oval plaques with a silvery white adherent scale
    -pustules may border lesions
    -variable pruritus
    -extracutaneous manifestations: nails (onycholysis, yellowing, pitting), geographic tongue, joints
      -arthritis: destructive polyarthritis, ankylosing spondylitis, DIP arthritis
      -asymmetric or symmetric
      -associated with cardiovascular disease, depression, lymphoma
  -types of psoriasis:
    i. **chronic plaques**: sharply defined erythematous scaling plaques in ~symmetric dist
        -most common type
        -lasts months to years
        -can have nail involvement
ii.) **erythrodermic psoriasis**: generalized erythema with scaling and exfoliation
   - accounts for 10% of cases
   - can occur at any age
   - presentation: patients are very sick, with hypo- or hyperthermia, protein loss, dehydration, renal and cardiac failure
   - investigation: may need to do a punch biopsy to differentiate from contact dermatitis

iii.) **pustular psoriasis**: individual or coalescing sterile pustules 1-10 mm that are generalized or localized

iv.) **guttate psoriasis**: small papules of short duration
   - usually in kids and young adults
   - associated with recent streptococcal infection
   - can also see the “typical” plaque lesions on the knees and elbows

v.) **intertriginous**

- treatment:
  - strategy is to control disease, decrease area involved, decrease erythema, scaling, and lesion thickness, maintain remission, avoid adverse effects, and improve QOL
  - topical therapies:
    - anthralin
    - steroids
    - tars
    - vit D analogs: very expensive
    - retinoids such as tazarotene
    - Taclonex ointment
  - phototherapy: UVB (vs tanning beds that are mostly UVA)
  - systemic therapies:
    - retinoids: acitretin
    - anti-cytokines: methotrexate
    - immunomodulators:
      - cyclosporine
      - biologics: adalimumab, alefacept, etanercept, infliximab, ustekinamab
  - treating depression can help the psoriasis

B.) **Seborrheic dermatitis**: a chronic inflammatory papulosquamous dermatosis with genetic and environmental causes

- possible agent: *Pityrosporum ovale*

- types:
  - infants: cradle cap
    - treatment: remove scale, treat infection, reduce inflammation
  - young children: tinea amiantacea and blepharitis
  - adolescents and adults: classic seborrheic dermatitis
    - causes: inflammation, bedridden, chronic illness, neurologic disease
    - presentations:
      - fine, dry, white scalp scaling
      - involvement of eyebrows, nasolabial folds, external ear canals, postauricular fold, presternal area
      - less common: involvement of the axillae, groin, umbilicus
    - investigation:
      - persistent or resistant cases should be cultured for dermatophytes
- AIDS-associated seborrheic dermatitis
  - may precede AIDS symptoms
  - severity often parallels clinical deterioration

- treatment:
  - frequent washing of all involved areas
  - use anti-dandruff shampoos
  - topical steroids
  - maintenance therapy may be needed

C.) **Pityriasis rosea**: a common, benign, usually asymptomatic self-limited dermatosis
may have viral origin (HSV-7 or 8)
most patients are between 10 and 35
-presentation:
-may have recent history of infection with fatigue, headache, sore throat, lymphadenitis, fever
-initially just one 2-10 cm herald lesion that is raised with a fine scale
-7-14 days later diffuse eruptions show up on trunk dermatomes that are salmon pink in white patients and hyperpigmented in black patients
-fine ring of scale known as collarette scale
-can mimic rash of secondary syphilis although rash is in a different location
-lesions are asymptomatic or pruritic at night or with heat
-treatment: most beneficial within first week
-oral antihistamines and topical steroids
-oral prednisone
-UVB phototherapy
-prognosis: eruption clears in 1-3 months

D.) Lichen planus: inflammatory, pruritic, planar, polyangular purple papules
-possible causes:
exposure to gold, chloroquine, methyldopa, penicillamine
-exposure to film processing chemicals
-secondary syphilis
graft vs. host disease s/p bone marrow transplant
-usually occurs in 40s
-risk factor: liver disease
-presentation: varies from a few chronic plaques to generalized eruption
-various patterns: actinic, annular, atrophic, follicular, guttate, hypertrophic, linear, localized, vesiculobullous
-may need to do histology because there are so many presentation forms
-primary lesion is 2-10 mm flat-topped papule with an irregular angulated border
-surface of lesions have lacy-recticulated pattern of white lines (Wickham's stria) correlating to areas of epidermal thickening
-most commonly on wrist flexors, forearms, legs above the ankles
-variable pruritus
-treatment:
topical steroids
-intralesional steroids
-systemic steroids
cyclosporine
-retinoids
-methotrexate
-PUVA
-antihistamines
-prognosis: eruption can clear in one year but may recur; unpredictable course

E.) Tinea corporis
F.) Syphilis
G.) Drug reaction

Cutaneous Manifestations of Systemic Diseases

Autoimmune Disorders
A.) SLE: > 85% of patients will have skin findings, and some only have skin findings
i.) acute cutaneous SLE: localized or generalized rash of the face, scalp, upper extremities
-malar or “buttefly” rash
-papules, papular urticaria
-scaly plaques (more subacute)
discoid lesions (more chronic)
-bullae
-palmar erythema

ii.) subacute cutaneous SLE: lesions of the shoulders, forearms, neck, upper trunk
-usually no facial lesions
-not strictly associated with SLE; can be other diseases
-lesions are annular or papular (psoriaform)
 -begin as small erythematous papules with scale
 -can also uncommonly resemble erythema multiforme

iii.) chronic cutaneous SLE (discoid lupus): face, neck, and scalp
-localized or generalized
-lesions are classically discoid
 -begin as well-defined scaling plaques that extend into hair follicles
 -expand slowly and heal with scarring, dyspigmentation, or atrophy
 -can resemble nummular eczema, psoriasis

iv.) other manifestations: alopecia, oral ulcers, photosensitivity, lupus profundus (panniculitis; subcutaneous nodules), vasculitic lesions, livedo reticularis (lacy purple), urticaria

B.) Dermatomyositis: one of the idiopathic inflammatory myopathies
-classic lesion is heliotrope: erythematous or violaceous macular rash of eyelids and periorbital area, often accompanied by edema
-other lesions:
- Gottron’s papules: slightly raised pink, dusky red, or violaceous papules over the dorsal MCP/PIP or DIP joints, or over the wrists, elbows, or knees
- Gottron’s sign: macular rash over same areas as papules
- V sign: macular photosensitivity over anterior neck, face, or scalp
- shawl sign: macular rash over posterior shoulders and neck
- poikiloderma: mottled red-brown discoloration from previous dermatomyositis lesions
 -calcifications on the elbows, trochanteric, and iliac areas
 -linear erythema over the extensor surfaces of the hands
 -nail changes: periungual erythema, telangiectasias, cuticle overgrowth

C.) Scleroderma
-localized lesions: rarely progress to systemic
- morphea: localized plaques in a band-like distribution
- linear scleroderma: line of thickened skin that can affect muscle and bone underneath
 -most common up through 20s
-systemic: sclerodactyly (tight hands), Raynaud’s, sclerosis of face, scalp, and trunk, periungual telangiectasia, pigmentation abnormalities, calcinosis cutis (subdermal calcifications)

D.) Vasculitides
 -dermal manifestations are usually a result of disease secondary to infection (Strep pyogenes, Hep B, Hep C), drugs (sulfonamides, penicillins), connective tissue disease
 -hallmark is palpable purpura
 -primarily on the Les
 -can progress to ulceration and necrosis

☐ Endocrine Disorders
A.) Diabetes
 -cutaneous manifestations:
- acanthosis nigricans: thickened, hyperpigmented, velvety plaques on neck, axillae, or body folds that are associated with obesity and insulin resistance
- diabetic dermopathy: atrophic, < 1cm brown lesions on lower extremities
 -a result of microangiography
 -most common cutaneous manifestation of DM
 -lesions are asymptomatic and resolve after 18-24 months
• **diabetic bullae**: appear spontaneously, usually on the hands and feet
  - may be associated with peripheral neuropathy or longstanding DM2
  - three types: sterile (heal without scarring), hemorrhagic (scar), and non-scarring (triggered by sun exposure)
• **necrobiosis lipoidica diabeticorum**: flesh-colored or red-brown papules that evolve into waxy plaques
  - center of lesion becomes yellow and atrophic +/- telangiectasias
  - from degeneration of collagen
  - usually on shins
  - treatment: topical or intralesional steroids

- infections
- lesions secondary to peripheral neuropathy or PVD
  - diabetic ulcers
    - can lead to osteomyelitis
• **granuloma annulare**: uncommon benign skin disorder of papules and plaques in an annular distribution
  - hands, feet, knees, and elbows
  - localized, generalized, or perforating
  - more common in women

**Immune Disorders**

A.) **Dermatitis herpetiformis**: chronic skin disorder associated with Celiac disease; erythematous papules or plaques studded with vesicles
  - due to IgA deposition in the skin in response to gluten
  - classically located on the elbows, knees, buttocks, scapular areas, scalp
  - symmetric and intensely pruritic
  - treatment: gluten-free diet, dapsone, sulfapyridine

B.) **Urticaria & angioedema**: localized swelling of skin or mucous membranes
  - immunologic and non-immunologic causes
  - more common in females
  - characteristic lesion is a transient, pruritic wheal that is warm
    - not cellulitis because it doesn’t hurt!
  - urticaria:
    - mediated by IgE, cytotoxic/immune complex deposition, physical factors (heat, cold, pressure, light), direct mast cell release, malignancy, infection, or emotional stress
    - classification:
      • **acute urticaria**: occurs once and lasts days up to 6 weeks
        - triggers: foods, drugs, infection, stress, latex, environmental agents
      • **chronic urticaria**: recurrent or constant urticaria of 6 weeks or greater
        - usually due to autoimmunity or chronic disease
        - investigation:
          - labs: CBC, liver panel, thyroid panel, renal panel, ESR or CRP
          - biopsy if vasculitis is present
          - patients can have systemic symptoms such as fever
          - angioedema is urticaria that extends into the subcutaneous tissue
          - most commonly involves the face but other sites can be affected
        - treatment:
          - H-1 or H-2 blockers
          - doxepin
          - glucocorticoids if acute (not if chronic!)
          - epinephrine
          - around-the-clock antihistamines if chronic

C.) **Sarcoidosis**: chronic multisystem granulomatous disease
  - skin involvement in 25% of patients
  - lesions can be diverse and non-specific, but some are specific
-usually asymptomatic
-predilection for scarred/tattooed areas
-macules, papules: brown, yellow, or purple on face or extremities
-nodules: brown or purple on face, trunk, and extremities
-plaques: annular or serpiginous +/- scales on butt, trunk, extremities
-classic lesion is lupus pernio: infiltrating violaceous plaque on the nose, cheeks, ears, or lips

Metabolic Disorders
A.) Xanthelasma: soft yellow plaques occurring near medial canthus of eyelid
  -more prominent on upper lid
  -relatively rare
  -more common in women in 40s-50s
  -half of cases are associated with elevated lipid levels
  -treatment: reduction of serum lipids, surgical excision

Venous Insufficiency
-Multiple skin manifestations secondary to decreased or absent return of venous blood and increased capillary pressure
-Presentation:
  -pitting edema
  -varicose veins
  -stasis dermatitis: appears eczematosus +/- papules, excoriations, pruritus
    -on LEs and ankles
    -often mistaken for cellulitis
    -can be concomitant with irritant contact dermatitis or bacterial superinfection
    -treatment: compression, oral antibiotics, topical steroids
  -hyperpigmentation: mottled blue and purple
  -skin fibrosis (lipodermatosclerosis)
  -venous ulcers: usually above the medial malleolus
    -bacterial superinfection is always present
  -atrophie blanche

Misc. Disorders
A.) Erythema nodosum: a cutaneous reaction to antigenic stimuli (infection, drugs like OCPs, IBD, malignancy)
  -erythematous nodules limited to extensor surfaces of LEs
  -very painful
  -other symptoms: fever, arthralgias
B.) Erythema multiforme: cutaneous immunologic response to varied antigens such as drugs, infection, systemic illness
  -macule → papule with vesicle or bulla in the center (iris lesion or target lesion)
  -occurs on hands, forearms, feet, face
    -usually symmetric
  -can be painful or pruritic
  -ranges from mild to severe
  -patients present very ill, with fever and high white counts
  -treat with steroids
C.) Stevens-Johnson syndrome and toxic epidermal necrolysis: widespread bullae on trunk and face with mucous membrane involvement
  -can have epidermal detachment
  -treatment: systemic steroids with monitoring of fluids and electrolytes
D.) Infective endocarditis: Osler nodes, Janeway lesions, subungual hemorrhages
E.) Meningococcemia: petechiae, purpura, necrosis
F.) Disseminated gonococcal infection: hemorrhagic pustules
G.) Lyme disease: erythema migrans, lymphocytoma cutis, acrodermatitis chronica atrophicans
Acne

Background

- Acne vulgaris is an inflammatory disease of the hair follicles and sebaceous glands of the skin
  - open comedones: blackheads
  - closed comedones: whiteheads
  - inflammatory papules, pustules, and cysts
- Pathogenesis is multifactorial, involving hormones, keratin, sebum, and bacteria
  - begins as comedones in the hair follicles
  - androgens stimulate sebaceous glands to produce more sebum
  - *P. acnes* proliferates in this atmosphere → further plugging and inflammation → foreign body reaction → rupture
  - can lead to cysts, scarring, keloids, or pyogenic granulomas
- Affects face, neck, upper trunk, and arms
- Most common in teens 15-18, with no gender preference
- Usually ends by age 25
- Can be flared (but not caused) by sweating, chocolate, cell phones, hands on face, cosmetics
- Form of acne:
  a.) comedonal acne: blackheads and whiteheads predominate
  b.) inflammatory acne:
  c.) cystic acne (acne conglobata): characterized by cysts, fissures, abscess formation, deep scarring
    - more common in men
    - associated with oily skin
    - begins in puberty and worsens with time
    - more on trunk, less on face

Acne Treatments

1.) Behavioral modification
- no picking or exfoliation
- only mild, gentle cleansing twice a day
- consider avoiding milk
- use oil free, non-comedogenic products

2.) Topical comedolytics
   a.) retinoids: increase cell turnover, prevent new comedones, chemically exfoliate
      - use a pea-sized amount all over skin once a day- not for spot treatments!
      - gels more effective than cream but more drying = best use with oily skin
      - allow 4-6 weeks for full effect; acne will get worse before it gets better
      - side effects: dry skin, irritation, sun sensitivity
      - contraindicated in pregnancy!
      - forms:
        • tretinoin:
        • adapalene:
        • tazarotene:
   b.) azelaic acid: antikeratinic, antibacterial, anti-inflammatory
      - better option for patients who want to get pregnant
   c.) glycolic acid & salicylic acid preparations: chemically exfoliate and enhance penetration of other topicals by reducing pH
      - caution: sun sensitivity

3.) Topical antibacterials
   a.) benzoyl peroxide: the workhorse against acne
      - no drug resistance
      - concentrations from 2.5 to 10%
   b.) clindamycin
      - with or without benzoyl peroxide (with decreases resistance)
      - contraindicated in patients with h/o UC, pseudomembranous colitis
   c.) erythromycin
- with or without benzoyl peroxide
- high *P. acnes* resistance
d.) sulfur-containing preparations
e.) metronidazole
- more for treating rosacea
f.) dapsone: for treatment of inflammatory acne
- usually paired with a retinoid

4.) Oral therapies
a.) oral antibiotics: allow 2-4 weeks to work
   • **minocycline**: anti-inflammatory as well as antibacterial
     - more expensive
     - side effects: vertigo, bluish-gray discoloration of skin, mucosa, teeth, and nails, lupus-like syndrome (long-term), serum sickness, hepatitis, pseudotumor cerebri
     - contraindications: pregnancy, peds
   • **doxycycline**:
     - side effects: photosensitivity, GI upset, vaginitis
     - contraindications: pregnancy, peds
   • **tetracycline**:
     - side effects: photosensitivity, GI upset, staining of teeth, photo-onycholysis, hepatitis, pseudotumor cerebri
     - contraindications: pregnancy, peds
   • **erythromycin**: used less frequently due to emerging resistance
     - good option for pregnancy and peds
     - side effects: GI upset, vaginitis
   - others: clindamycin, ampicillin, cephalosporins, Septra (2nd line due to side effects)
b.) oral **isotretinoin**: for severe, nodular, cystic, inflammatory, recalcitrant acne
   - tightly regulated by FDA via iPledge system due to strong teratogenicity
   - requires monthly visits with a registered provider
   - usually one 5 month course is sufficient
   - some patients may need additional course after a 2 month rest period
   - used alone without any other acne treatments
   - side effects: dry skin, cheilitis, headaches, myalgias, arthralgias, bone pain, osteopenia, mood changes or depression, elevated glucose, elevated TG, hepatotoxicity, decreased night vision, hearing changes
c.) oral hormonal treatments: for patients with adult acne, hirsutism, PCOS, premenstrual flares
   i.) oral estrogens (OCPs): suppress sebaceous gland uptake of testosterone and peripheral metabolism of testosterone
     - all of them will work
     - side effects: HTN, hypercoagulability, hyperkalemia
   ii.) **spironolactone**: for poor OCP candidates
     - acts as androgen receptor blocker ➔ decreased serum testosterone
     - side effects: menstrual irregularities, hyperkalemia, breast tenderness
     - contraindications: pregnancy (category X)

5.) Additional therapies
- intralesional steroids: risk of causing permanent divet
- comedo extraction
- photodynamic therapy
- laser therapy
  - CO2 lasering for icepick scarring
- chemical peel

→ Algorithm:
- mild, comedonal acne: start with a topical retinoid
  - add benzoyl peroxide or topical antibiotics if needed
- moderate acne with papules and pustules: start with a topical retinoid + benzoyl peroxide (or benzoyl peroxide + topical antibiotic)
  - add oral antibiotic if needed
-severe nodular acne: start with topical retinoid + benzoyl peroxide (or benzoyl peroxide + topical antibiotic) + oral antibiotics
-refractory severe acne: oral isotretinoin

## Acne Vulgaris Lookalikes

A.) **Hydradenitis suppurativa (acne inversa):** plugged sweat duct → inflammation and bacterial growth → rupture → ulceration and fibrosis → sinus tract formation
- a chronic and relapsing condition
- seen in the axillae, inguinal folds, perianal area
- rarely on the scalp
- hallmark is double comedone
- treatment: oral antibiotics, topical antibiotics and washes, intralesional triamcinolone as needed, oral prednisone course, surgical I&D or excision

B.) **Steroid acne**

C.) **Drug acne:** Li, tetracyclines (paradoxically), phenytoin, OCPs, isoniazid

D.) **Cutting oils and other occlusives**

E.) **Infectious folliculitis**

F.) **Rosacea:** etiology not well understood
- triggers: hot or spicy foods, alcohol, exercise, sun
- resembles acne but also has flushing, telangiectasia, and lingering erythema on the forehead, chin +/- eyes
- no comedones!
- late manifestation is rhinophyma (large bulb-shaped nose)
- treatment:
  - topical metronidazole, sulfacetamide + sulfur, azelaic acid
  - time-released oral doxycycline for anti-inflammatory effects
  - laser therapy

G.) **Perioral dermatitis:** etiology not understood
- grouped 1-2 mm papules on an erythematous base that goes on for weeks or months
- no comedones
- can also be perinasal or periorbital
- treatment:
  - avoid cinnamon, tartar control toothpaste, whitening agents, heavy facial moisturizers, topical steroids
  - topical antibiotics

## Warts

### Background
- Caused by HPV infections of skin keratinocytes (cutaneous warts) or mucous membranes (condyloma acuminatum)
- occur in areas of skin trauma
- 100 serotypes = multiple infections
- Regression is dependent on cell-mediated immunity
- = occur more often in immunosuppressed patients
- Don’t have “roots” as they are confined to the epidermis
- Cause necrosis of capillaries (may only be seen after paring of lesion with surgical blade)
- Differentiate from callous by interruption of normal skin lines
- Oncogenic potential

### Clinical Subtypes of Cutaneous Warts

A.) **Verruca vulgaris:** the common wart
- common in ages 5-20
- presentation: verrucous surface, thrombosed capillaries, loss of dermatoglyphics
- can have fingerlike projections in kids
- periungual are hard to treat without damaging nail matrix
B.) **Verruca plana:** flat wart  
- common in ages 5-20  
- commonly spread by shaving  
- presentation: flat-topped pink to brown papules, usually in linear formation  
- predilection for the face, dorsal hands, wrists, knees  

C.) **Verruca plantaris:** plantar wart  
- presentation: verrucous surface, thrombosed capillaries, often coalesce into a “mosaic”  
- prefers pressure points on the feet  

**Mucous Membrane Warts: Condyloma Acuminata**  
- background:  
  - often on the genitals, most common STD  
  - cervix, vulvovaginal skin, anus, penis, perianal area  
  - highest risk for oncogenesis with subtypes 16 and 18  
- presentation: lobulated surface that is cauliflower-like, gray or pink  
- high-risk lesions are often hyperpigmented  
- investigation:  
  - can be misdiagnosed as moles or skin tags  
  - cervical exam to look for dysplasia  
- treatment:  
  - cutaneous symptoms can spontaneously resolve  
  - therapy to stimulate immune response  
- prognosis: often recur following treatment, as tissue is killed rather than the HPV itself  

**Wart Treatments**  
- physical destruction:  
  - cryotherapy: liquid nitrogen application, preferably twice  
  - repeat every 2-4 weeks as needed  
  - can cause hypopigmentation  
  - laser  
  - cautery  
  - duct tape occlusion  
  - excision  
  - cantharadin (beetle juice): causes blistering → risk of scar = not for face  
  - done in office  
  - podophyllin gel: applied at home  
  - retinoids +/- occlusion  
  - salicylic acid  
  - 5-fluorouracil  

- immunomodulation:  
  - imiquimod  
  - cimetidine  
  - squaric acid  

**Hair and Nails**  

**Background**  
- total number of hair follicles is present at birth  
- hair anatomy and physiology:  
  - lanugo is the fine, soft nonpigmented hair in utero that is shed before birth  
  - vellus hair is soft, nonpigmented, and short  
  - lacks a medulla  
  - terminal hairs are pigmented with variable length  
  - androgens trigger vellus ↔ terminal hair switch  
  - hair follicle:
-segment from skin opening to sebaceous gland opening is the infundibulum
-from the sebaceous gland to the bulge is the isthmus
-bulge formed by insertion of erector pili muscle into the follicle
-transient portion that goes through stages of telogen, anagen, and catagen is the lower follicle
  -anagen: growth phase that 85% of hairs will be in
    -average duration of 3 years for the scalp
    -max hair length determined by anagen duration
  -catagen: involution phase between growth and resting where all cell division stops
    -3% of hairs are in this at any given time
    -lasts 10-15 days
  -telogen: the resting phase where club hair is produced
    -12% of hairs
    -lasts 3 months as the old hair is pushed out
-bulb at bottom of follicle contains stem cells
-plucking hairs leads to breakage above this level
-color determined by eumelanin (brunettes, blondes, and grays) or pheomelanin (redheads)

Alopecia

- Normal daily hair loss is 75-150 hairs
- Causes:
  a. **alopecia areata**: increased lymphocytes around bulb ➔ patchy, non-scarring alopecia
    - associated with thyroid disease, stress, vitiligo, autoimmune disease, DM, atopic dermatitis
    - a common cause of hair loss
    - in men, women, and children
    - presentation: see “exclamation point hairs” or associated nail pitting
      - can involve entire scalp
    - treatment:
      - small patches will grow back on their own
      - topical and intralesional steroids speed up regrowth
    - prognosis: worse with acute onset, extensive hair loss, or hair loss beginning over the ears
  b. **androgenetic alopecia**: inherited
    - the most common form of hair loss
    - presentation:
      - men: recession of frontal hairline, decreased length and thickness of hair shaft
      - women: later onset and less progressive than males
        - advanced loss associated with hirsutism
    - treatment:
      - minoxidil (vasodilator), finasteride (5-alpha reductase inhibitor)
        - can’t stop or hair will fall out!
      - hair transplant
  c. **anagen effluvium**: loss of hair due to chemotherapy or radiation
    - because anagen is most metabolically active it is targeted
  d. **telogen effluvium**: diffuse shedding of hair as more follicles are shifted from anagen to telogen
    - occurs after stressful events, childbirth, massive blood loss, high fever, surgery, drugs, thyroid disease, crash dieting, car accident, stopping OCPs
    - more hairs are retained anagen phase during pregnancy
    - loss can occur up to 3 months after event!
    - almost always temporary with normal regrowth
  e. **trichotillomania**: pleasure or relief from pulling hair out
    - investigation:
      - see pigment casts and achordion (stretching of epithelium) on biopsy
f.) traction alopecia: caused by constant pulling on hair follicles from wearing tight cornrows and braids
  -most commonly on the frontotemporal scalp
  -initially nonscarring but can progress to scarring

-Investigation:
  -history: meds, family h/o hair loss, recent fever, severe illness, general anesthesia, hyperandrogenic signs
  -hair pull test: anagen:telogen ratio should be 10:1
  -scalp biopsy: one vertical section and one for horizontal

<table>
<thead>
<tr>
<th>Nail Diseases</th>
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<tbody>
<tr>
<td>Condition</td>
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<tr>
<td>Psoriasis</td>
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<tr>
<td>Nail mechanical trauma</td>
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<tr>
<td>Muercke’s lines</td>
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<tr>
<td>Paronychia</td>
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<td>Beau’s lines</td>
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<tr>
<td>Half &amp; half nails (Lindsey’s nails)</td>
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<tr>
<td>Blue nails</td>
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<tr>
<td>Pseudomonas infection</td>
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</tbody>
</table>
### Nail Tumors

<table>
<thead>
<tr>
<th>Tumor</th>
<th>Info</th>
</tr>
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<tbody>
<tr>
<td>Digital mucous cyst (myxoid cyst)</td>
<td>Translucent papule at proximal nail fold.</td>
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<tr>
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<td>Communicates with DIP and contains clear jelly.</td>
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<td>Can cause longitudinal ridge or indentation in nail plate distal to growth.</td>
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<tr>
<td>Melanoma</td>
<td>Dark pigmentation at the proximal nail fold (Hutchinson’s sign)</td>
</tr>
<tr>
<td></td>
<td>or pigmented longitudinal nail streaks. Normal in blacks but can be true melanoma in whites!</td>
</tr>
<tr>
<td>Squamous cell carcinoma</td>
<td>Can mimic a nail fold wart.</td>
</tr>
</tbody>
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### Human Ectoparasites

#### Background

- Clinically relevant arthropods are those that suck blood and transmit disease, those that bite/sting causing lesions, those that inject venom or allergens

#### CRABS: Cutaneous Reactions to Arthropod Bites

- Allergic or inflammatory
- Cause a variety of lesions: transient erythema, bullae, hemorrhagic ulcers, necrosis
  - **papular urticaria**: hallmark arthropod bite lesion; 2-8 mm erythematosus, papulovesicular lesions usually in clusters
    - common culprits are fleas, mosquitoes, bedbugs
    - kids with pre-existing atopic dermatitis will have increased sensitivity to bites

#### Spider Bites

- Most clinically important:
  a.) black widow:
    - likes dark, dry places like basements, crawl spaces
    - bite is from defensive female
    - venom is a potent neurotoxin
    - only 25% of bites evoke a serious reaction: muscle cramping, HTN, tachycardia
    - treat these rxns with antivenom
  b.) brown recluse: like dark, quiet places like shoes and clothing
    - bites are defensive
    - toxic protein in venom stimulates platelet aggregation and neutrophil infiltration → necrosis and systemic effects
    - bite lesion has a black center with minimal peripheral erythema
    - no antivenom available
- Generalized treatment:
  - RICE
  - tetanus booster
  - analgesics
  - antibiotics if there is a secondary infection
**Mites**

A.) Scabies:
- spread by skin to skin or sex
- can live for 48 hours on clothing, bedding, and furniture
- highly contagious
- make burrows and lay eggs and poop in them → intensely pruritic papules and pustular rash that is worse at bedtime
  - predilection for the fiber webs, wrist flexors, elbows, axillae, penis, external genitalia, feet, ankles
  - babies < 1 year can get scabies from the neck up
- immediate and delayed hypersensitivity rxns
- normal incubation for first infection is 21 days
  - sequential infections have immediate rxn due to memory response
- “Norwegian” scabies make severe crusting and have a heavy infestation
- infected patients usually have underlying immunodeficiency
- variable pruritus
- investigation: scraping under oil immersion for mite, feces, eggs
- treatment: permethrin cream, lindane lotion (known neurotoxin, banned in Ca)
  - apply at night and wash off in morning
  - usually only single application needed
  - can also do single dose ivermectin but it’s hard to find
  - treat all family members!
  - wash all bedding and clothing in hot water

B.) Chiggers: aka redbugs, jiggers
- larval form injects digestive fluid into host to form a feeding tube
- feeds for 3-4 days then drops off
- presentation:
  - causes papules or hives at suck site
  - predilection for the ankles, backs of knees, groin, axillae
  - severe itching within 1 day
- treatment: immediate bath in hot soapy water, topical antihistamines, topical steroids, nail polish to starve it off?

C.) Dust mites:
- feed on human scale
- associated with asthma, maybe atopic dermatitis

**Ticks**

- Lesions:
  - papule at site of bite from antigenic tick saliva
  - local reaction of swelling, erythema
    - classic lesion of *erythema migrans* 4 days to 3 weeks after bite
      - only occurs half the time and is not diagnostic of Lyme
  - can also get vesicles, malar rash, urticaria, nodules
  - hardens after a few days → pruritus, tenderness
- Uncommon lesions:
  - rare granulomatous reaction
    * lymphocytoma cutis: bluish nodule at site of bite or in remote location such as earlobe, areola, neck
    * acrodermatitis chronica atrophicans: bluish erythema + edema

**Lice**

- Body and head louse are *Pediculosis*
- lesions:
  - causes small erythematous papules on the axillae, neck, shoulders
  - hemorrhagic puncta and linear excoriations
  - vesicles
  - blueish brown macules at bite site called *maculae ceruleae* from heme breakdown
- vectors for epidemic typhus, trench fever
-hair of black children is rarely infested
-presentation: scalp pruritus, excoriations, cervical adenopathy
-treatment: permethrin cream, malathion
- must re-treat in one week to treat eggs that hatch

-Pubic louse is *Phthirus*
-can also infest eyelashes
-public lymphadenopathy
-treatment: lindane or permethrin

**Bedbugs**
-Painless bite → papular urticaria or vesicular, eczematous rash
- tend to be in linear clusters

**Moths, Butterflies, Caterpillars, Bees and other Vespids, Fire Ants**
-Caterpillars can cause dermatitis as well as systemic illness with urticaria and airway hypersensitivity
- the asp caterpillar is the most dangerous in the US
- sting produces intense pain with a train-track pattern
-Gypsy moths can cause pruritic rash
-Bees
- treatment: remove stinger, 1st gen antihistamine?, oral steroids for severe local reactions
-Fire ant stings produce papular eruptions that cause a systemic response in 16% of the population