Special Signs & Tests
- Darier sign → rubbing a lesion causes an urticarial flare
- Auspitz sign → pinpoint bleeding after scale is removed
- Nikolski sign → pushing a blister causes further separation of the dermis
- Photopatch test → documents photoallergy
- Patch test → demonstrates hypersensitivity reaction
- Koebner phenomenon → minor trauma leads to new lesions at site of trauma
- Shaqreen skin → an oval shaped nevoid plaque – associated with tuberous sclerosis

Diagnostic techniques
- Diascopy → a glass slide or diascopy is pressed against skin, blanching indicates intact capillaries, extravasated blood (purpura) does not blanch
- KOH (potassium hydroxide) prep → skin scrapings, readily identifies dermatophyte infection
- Scrapings & smears → various staining techniques and visualization methods (tzanck smear, dark field microscopy)
- Wood’s light examination → used to assess changes in pigment or to fluoresce infectious lesions
- Acetowhitening → using acetic acid to examine warts
- Biopsy → excisional, incisional, shave, punch (for pathologic confirmation)

Size
- Papule → <5 mm
- Macule → <10 mm
- Patch → >10 mm (non-palpable)
- Plaque → 10 mm (plateau-like lesion)

Eczematous Eruptions

Atopic Dermatitis
- Most common = eczema
- TRIAD: asthma, allergic rhinitis (atopy), eczema
- Chronic relapsing skin disorder that begins in childhood, type I Ig E-mediated hypersensitivity rxn
- Stress may worsen condition
- No cure
- Genetic predisposition
- Clinical features
  - Papules and plaques with or without scales
  - Pruritis, dry skin, lichenification, fissures
  - Secondary infection most commonly caused by staph aureus
  - Rash is most common on flexural surfaces, neck, eyelids, forehead, face, and dorsum of the hands and feet
  - Extensor surfaces in infancy
  - Can get infected atopic dermatitis (impetiginization), usually strep/staph
- Treatment
  - Must stop itch-scratch cycle to successfully treat
  - Antihistamines help reduce itching
  - Topical steroids are the mainstay, try to avoid systemic steroids
  - Tacrolimus & pimecrolimus are topical calcineurin inhibitors (immunomodulators) approved for moderate to severe atopic dermatitis – these may cause a risk of malignancy
  - Hydration and topical emollients are key to management
  - Avoid soaps, vigorous rubbing, frequent bathing, and irritating clothing (wool)
  - Ultraviolet B phototherapy is effective
  - Severe systemic cases may require cyclosporine

Contact Dermatitis
- Allergic type IV cell mediated hypersensitivity rxn
- Inflammatory reaction precipitated by an exogenous chemical (poison ivy)
- Irritant or allergic subtypes
- Occurs in distribution of contact
  - poison ivy – linear
  - leather - shoe area
  - nickel - earlobes, neck, wrist, peri-umbilical
- Mangos & cashews have same oil that causes rxn
- ***Irritant contact dermatitis/diaper rash = prolonged contact with urine, feces, or detergents, often associated with superimposed candida (satellite lesions)
- Clinical features
  - Itching & burning in affected areas
  - Acute → erythema, exudative lesions, vesicles, crusts
  - Chronic → plaques, lichenification, excoriations
- Treatment
  - Remove offending agent, wet dressings with Burrow’s solution (aluminum acetate), and topical corticosteroids
  - For diaper rash, a barrier of petrolatum or zing odize is helpful (keep area clean and dry
  - Severe cases may need systemic steroids
  - Chronic lesions can be treated with topical steroids
  - Can clean with mild soaps, oatmeal prep, or antihistamines
<table>
<thead>
<tr>
<th>Seborrhea</th>
<th>Nummular dermatitis (discoid eczema)</th>
</tr>
</thead>
</table>
| ▪ Seborrheic dermatitis is common during infancy and puberty and in young to middle aged adults  
▪ It occurs where sebaceous glands are most active (body folds, face, scalp, genitals)  
▪ Clinical Features  
  o Scattered yellowish/gray, scaly macules and papules with a greasy look  
  o Sticky crusts and fissures are found behind the ears (especially in infants)  
  o On the scalp it manifests as cradle cap in infants and dandruff in adults  
▪ Treatment  
  o Ultraviolet radiation is helpful (lesions improve during the summer and are worse in the winter)  
  o Cradle cap: treat with olive oil compress and baby shampoo, or ketoconazole shampoo with hydrocortisone  
  o Dandruff: use shampoos containing selenium or zinc and ketoconazole for acute flare-ups, tar shampoos or topical steroids can be used for severe cases  
  o Other areas → use ketoconazole shampoo or topical steroids |
| ▪ This is a pruritic inflammatory disorder  
▪ Effects young adults and the elderly  
▪ Typically occurs during the fall and winter  
▪ Clinical features  
  o Small, grouped vesicles coalesce to form coin shaped plaques with an erythematous base  
  o Clearly demarcated boarders, most commonly on the extremities  
  o Crusting and excoriations occur  
▪ Treatment  
  o Chronic disorder that responds to moisturizers or topical steroids  
  o Tar baths or UVB phototherapy is helpful for refractory cases |

<table>
<thead>
<tr>
<th>Stasis Dermatitis</th>
<th>Dyshidrosis</th>
</tr>
</thead>
</table>
| ▪ Chronic venous insufficiency due to valvular incompetency leads to edema, dermatitis, hyperpigmentation, fibrosis, and ulceration  
▪ Varicose veins, superficial phlebitis, and DVT commonly occur before skin changes  
▪ Women are affected 3x more than men, worse with pregnancy  
▪ Clinical features  
  o Heaviness or aching in legs, aggravated by standing and relieved with walking  
  o Dermatitis manifests with inflammatory papules, scales, crusts  
  o Stippled pigmentation develops and excoriations are common  
  o Ulcerations will occur in 30% of patients  
▪ Lab studies  
  o Doppler studies, sonography, or venography will confirm venous insufficiency  
  o Biopsy of lesions shows dilated vessels, tortuous veins, edema, and fibrin deposition  
▪ Treatment  
  o Chronic venous insufficiency is treated with compression stockings  
  o Sclerosis of varicose veins helps to prevent further dermatitis, but recurrence is common  
  o Vascular bypass, endothelial thermal ablation, or angioplasty/stenting of obscured veins may benefit severely compromised areas, but results are not great  
  o Ulcers demand chronic treatment |
| ▪ This dermatitis generally develops in people younger than 40 yrs of age, half of those have an atopic background  
▪ Eruptions follow stress or occur in hot, humid weather  
▪ Clinical Features  
  o Early → pruritis, pain if secondarily infected, small vesicles in clusters are seen, occasionally bullae form on the fingers, palms, and soles  
  o Late → papules, scaling, lichenification, and erosions from ruptured vesicles are seen  
  o Predilection for hands and feet (palms and soles)  
▪ Lab studies  
  o Culture to rule out secondary infection  
  o KOH to rule out dermatophytosis  
▪ Treatment  
  o Use wet dressing (Burrow’s solution), large bullae should be drained by kept intact  
  o Fissure are treated with topical collodion  
  o Topical steroids are used for localized lesions and systemic steroids for severe cases  
  o PUVA is recommended for generalized disease  
  o Treat secondary infection with abx |
### Pityriasis Rosea

- **Clinical Features**
  - Characterized by a **herald patch** (round oval pink plaque), which precedes a widespread, symmetrical, popular eruption
  - The cause is unknown, but might be herpes virus 7
  - It is most common in teens, incidence is high in spring and fall
  - May be preceded by a mild URI before rash
  - Rash begins as oval, salmon colored, raised macules, followed by a natural skin fold giving a **Christmas tree like pattern** on the trunk
  - Collarettes around lesions

- **Treatment**
  - No treatment needed, can use emollients for scales, UVB light may be helpful at first week, can use lotions, antihistamines etc

### Psoriasis

- **Clinical Features**
  - Affects 2% of the population
  - Most patients have localized psoriasis, can be more severe
  - A genetic predisposition exists, although only in about 1/3
  - It is a chronic, inflammatory, scaling condition of the skin that may also involve mucous membranes
  - The earlier the onset of the disease, the more severe it will be
  - Psoriasis in HIV patients can be severe and resistant to treatment

- **Treatment**
  - Most patients have localized psoriasis, can be treated with topical steroids, tar, and UVB light
  - Moderate psoriasis may respond to tazarotene gel (topical retinoid)
  - Cyclosporine may be effective in very severe cases

### P Papulosquamous Eruption

- **Drug Eruptions**
  - Occur in 2-3% of hospitalized individuals
  - Majority of rxns are mild, accompanied by pruritus, resolving promptly after offending drug is d/c’d → however severe life-threatening ACDRs do occur
  - Caused by immunologic or nonimmunologic mechanisms and provoked by systemic or topical administration of drug
  - Findings indicating life-threatening ACDR: confluent erythema, facial edema, skin pain, skin necrosis, + Nikolsky’s sign, mucous membrane erosions, urticarial, swelling of the tongue, high fever, difficulty breathing, arthralgias, enlarged lymph nodes
  - Drugs w/ high probability of rxn: PCN, allopurinol, gold salts, carbamazepine

- **Classifications**
  - Type I = immediate-type immunologic rxns
    - IgE-mediated resulting in urticaria and angioedema
  - Type II = cytotoxic rxn
    - Drug + cytotoxic antibodies = lysis of cells
  - Type III = serum sickness, drug-induced vasculitis
    - IgG usually; mediated by deposition of immune complexes in small vessels, activated by complement and recruitment of granulocytes
  - Type IV = Morbilliform (Exanthematous)
    - Cell-mediated immune rxn

### Lichen Simplex Chronicus

- **Clinical Features**
  - Lichenification due to long term atomic dermatitis (repetitive scratching and rubbing)
  - Well circumscribed plaques that are highly pruritic

- **Treatment**
  - Solid, firm, thick plaques with little to no scaling
  - Light touch makes it very itchy
  - Lesions can be single or multiple, common areas include nuchal area, scalp, ankles, lower legs, upper thighs, external forearms, or genital areas

- **Lab studies**
  - KOH to rule out fungal infection, biopsy shows hyperplasia/hyperkeratosis

- **Treatment**
  - Stop the itching, occlusive dressing (with or without topical steroids/tar), can use antihistamines
<table>
<thead>
<tr>
<th>Lichen Planus</th>
<th>Molluscum contagiosum</th>
</tr>
</thead>
<tbody>
<tr>
<td>▪ Acute or chronic inflammatory dermatitis that occurs in dults</td>
<td>▪ Common viral disease, commonly affects skin and mucous membranes, caused by a poxvirus, common in kids but can affect adults</td>
</tr>
<tr>
<td>▪ Females are more commonly affected than males</td>
<td>▪ In adults the lesions are usually in the groin areas and on lower abdomen</td>
</tr>
<tr>
<td>▪ May occur in graft vs host disease, malignant lymphoma, and drug reactions</td>
<td>▪ Can be sexually transmitted</td>
</tr>
<tr>
<td>▪ Clinical Features</td>
<td>▪ Immunocompromised patients (HIV) can have a much more widespread presentation</td>
</tr>
<tr>
<td>o The 4 Ps → purple, polygonal, pruritic, papule</td>
<td>▪ Clinical features</td>
</tr>
<tr>
<td>o Flat topped, shiny, violaceous papules with fine white lines on the surface (wickham striae), typically grouped on flexor surfaces, koebner phenomenon</td>
<td>o Lesions are discrete, flesh colored, waxy, dome shaped, umbilicated papules over the face, trunk and extremities</td>
</tr>
<tr>
<td>o Mucosal lesions occur on the vagina, glans, and penis, and in the mouth, they are usually painful and ulcerate</td>
<td>o Usually 3-6 mm, a white curd-like material can be expressed</td>
</tr>
<tr>
<td>o Lesions may affect hair (scarring alopecia) or nails</td>
<td>▪ Lab studies</td>
</tr>
<tr>
<td>▪ Oral lichen planus</td>
<td>o Biopsy may be needed with HIV to rule out fungal dissemination</td>
</tr>
<tr>
<td>o 30% of pts with mucosal involvement (includes penis and vulva) have antibodies to Hep C</td>
<td>▪ Treatment</td>
</tr>
<tr>
<td>o Increased risk of oral cancer (SCC)</td>
<td>o Not usually necessary since the disease is self-limited</td>
</tr>
<tr>
<td>o White lacy patches of the oral mucosa</td>
<td>o Can do local excision of lesions (curettage, cryotherapy, electrodessication, acid or exfoliative peel – tretinoin, imiquimod), these treatments can be painful</td>
</tr>
<tr>
<td>▪ Lab studies</td>
<td>▪ Treatment of choice: Ammonium lactate 10% twice daily (cost-effective, easy to apply)</td>
</tr>
<tr>
<td>o Biopsy and immunofluorescence, screen for Hep C</td>
<td>▪ Oral lichen planus: ▪ Oral lesions occur on the tongue, buccal mucosa, and in the mouth, they are usually painful and ulcerate</td>
</tr>
<tr>
<td>▪ Treatment</td>
<td>▪ Biopsy and immunofluorescence to determine if positive for HPV</td>
</tr>
<tr>
<td>o Topical steroids, occlusive dressings, translesional steroids, topical tretinoin, cyclosporine mouthwash, systemic therapy (cyclosporine, corticosteroids, retinoids)</td>
<td>▪ Check BGL, Hgb A1C</td>
</tr>
<tr>
<td>o Psoralens plus UVA (PUVA) can be helpful</td>
<td>▪ Acyclovir (w/in 72hrs of acute vesiculation)</td>
</tr>
</tbody>
</table>

**Herpes Zoster**

- Acute dermatomal infx asx with reactivation of varicella zoster virus (VZV)
- Characterized by unilateral pain & vesicular or bullous eruption progressing to crusts limited to a dermatome(s) innervated by a corresponding sensory ganglion
- RFs: diminishing immunity to VZV w/ advancing age (>55y/o), malignancy, immunosuppression
- Once crust develops, person is no longer contagious
- Susceptible contacts contract varicella (shingles would not develop) by direct contact with fluid from the blisters
- -shingles--reactivated form of varicella-zoster
- **Sx:**
  - Grouped clear vesicles on an erythematous base in a dermatomal distribution
  - Prodromal prickling pain
  - Post-herpetic neuralgia
  - Tzanck smear (multinucleated giant cells ), viral cx, DFA test
- **Tx:**
  - Acyclovir (w/in 72hrs of acute vesiculation)
  - Analgesics for pain control
  - Gabapentin

**Melasma**

- Acquired brown macular pigmentation of the face & neck in genetically predisposed women
- Forehead, malar cheek, upper lid, chin are most common
- Occurs usually after pregnancy or w/ OCPs
- **Tx:** d/c OCP, sunblock, hydroquinone, tretinoin, Tri-Luma, azelaic acid (Finacea), kojic acid (OTC), peels

**Acanthosis nigricans**

- (cutaneous manifestation of internal dz)
- Elevated velvety hyperpigmentation of the flexural skin: neck, axillae, groin
- Most commonly asx with obesity, DM; less commonly w/ meds (estrogen, nicotinic acid), malignancy (usually w/ sudden onset)
- Usually asx, Cutaneous marker of insulin resistance
- Check BGL, Hgb A1C
- **Tx:** ammonium lactate, tretinoin

**Seborrheic Keratoses**

- Common, benign epidermal lesion in older pts
- Can be confused with other cutaneous malignancies (MM)
- Flesh colored, pink, white, tan, brown, black stuck-on warty
- Retained keratin cysts may be seen – “horn cysts”
- Usually asymptomatic but can become irritated or inflamed
- Anywhere, but lips, palms, soles
- Dermatosis papulose nigra is a type, usually seen on the face in AA (Morgan Freeman)
- The sign Lester-Trelat – sudden explosive onset of numerous SKs is associated w/ an internal malignancy (still benign but can mean they have CA)
- **Tx:** cryosurgery (liquid nitrogen), shave/snip removal, cautery, curettage (can scar)
<table>
<thead>
<tr>
<th>Acneiform</th>
<th>Rosacea</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Acne Vulgaris</strong></td>
<td><strong>Chronic acneiform disorder, most commonly affects females age 30-50, it is a disease of the pilosebaceous units → leads to telangiectasias, flushing</strong></td>
</tr>
<tr>
<td>▪ Acne affects all age groups – most common in adolescents and more severe in males</td>
<td>▪ Outbreaks are episodic and typically respond to heat, alcohol, sun, hot, spicy foods</td>
</tr>
<tr>
<td>▪ Pathology includes plugged follicles, retained sebum, bacterial overgrowth, and release of fatty acids (androgens stimulate sebum production)</td>
<td>▪ Clinical features</td>
</tr>
<tr>
<td>▪ Clinical features</td>
<td>▪ Insidious onset, face appears red or flushed</td>
</tr>
<tr>
<td>▪ Comedones, either open or closed, non-inflammatory</td>
<td>▪ There is a symmetric distribution, can cause telangiectasias, hyperplasia, and lymphedema</td>
</tr>
<tr>
<td>▪ Open = blackheads (keratin plug), closed = whiteheads</td>
<td>▪ Rhinophyma (enlarged nose), eyelids, forehead, chin, ears</td>
</tr>
<tr>
<td>▪ Sinus tracts occur with nodular acne, inflammatory lesions can lead to hyperpigmentation and scarring</td>
<td>▪ Treatment</td>
</tr>
<tr>
<td>▪ Lab studies</td>
<td>▪ Reduce triggers such as alcohol and hot beverages</td>
</tr>
</tbody>
</table>
| ▪ Testosterone, FSH, LH can be measured if endocrine disorder is suspected, but the majority are not | ▪ Topical *metronidazole*, *sodium sulfacetamide*, or *erythromycin*
| ▪ Treatment | ▪ If topical tx fails, can use oral abx |
| ▪ Mild acne → topical preps (retinoids, azelaic acid, salicylic acid) | ▪ Very severe cases may need oral isotretinoin |
| ▪ Inflammatory lesions → topical benzoyl peroxide, tretinoin, erythromycin, clincamycin, or sodium sulfacetamide | |
| ▪ Cystic acne → use oral abx & topical prep | |
| ▪ Tetracyclines, erythromycin, doxy, mino, Bactrim, and clindamycin are freq used | |
| ▪ Bacteria are becoming resistant, treat conservatively, recurrence is common | |
| ▪ Oral isotretinoin – can only be prescribed by providers approved by Roche, side effect are very dangerous → dry yes, nose, lips, epistaxis, joint pains, mood swings, suicidal thoughts, premature closure of long bones, visual changes, hepatic enzyme elevation, leukopenia, triglyceridemia, teratogenicity | |
| ▪ Furuncle (single)/Carbuncle (multiple) | **Folliculitis** |
| ▪ “Boils”, deep infections of the hair follicle | ▪ Inflammation of the hair follicles, most commonly caused by *staph aureus*, but hot tub folliculitis is most commonly caused by *pseudomonas* |
| ▪ Staph aureus is the most common | ▪ Non-infectious folliculitis is common among people working in hot, oily environments |
| ▪ Clinical features | ▪ Occlusion, perspiration, tight clothes can also cause |
| ▪ Red, hard, tender lesions in hair bearing regions | ▪ Pseudofolliculitix barbae (razor burn) |
| ▪ Carbuncles have multiple drainage points | ▪ Clinical features |
| ▪ Lesions progress to become fluctuant, can rupture and drain pus and necrotic tissue | ▪ Usually not painful, may burn |
| ▪ Treatment | ▪ Sycosis is severe, deep seated, recalcitrant folliculitis with surrounding eczema and crustung |
| ▪ Warm, moist compress | ▪ Abscess may form at site |
| ▪ Antibiotics and I&D if severe | ▪ Treatment |
| Be careful of spreading infection | ▪ Gentle cleansing, mild compress, protection from offending agent, drying agent |
| | ▪ Topical application of clindamycin or erythromycin |
| | ▪ Mupirocin (bactroban) may also help |
| | ▪ If severe, might need oral abx |
| | ▪ Hot tub folliculitis will resolve on its own (may need fluoroquinolone) |
### Lice
- **Pediculus**
  - Humanus capitis – head louse (not a vector for infectious disease)
  - Humanus – body louse
  - Pubis – genital louse (“CRABS”)
- Lice can be transmitted through direct contact & via sharing towels, combs, bed, clothing
- Moves from hair to hair by gripping it with their legs → do not fly or jump
- Allergic rxn to lice saliva may cause itch & irritation
- Dx is confirmed by the visualization of live lice
  - Nits (eggs) also be seen with the naked eye or under wood’s lamp which makes them look pale blue in color
  - “They attach to the hair shaft; looks like little pearly balls”
- Lice treatment
  - Permethrin, pyrethrin, and malathion are first line
  - Lindane or ivermectin are alternaties
  - Petroleum jelly can suffocate
  - Must tx household members and close contacts

### Scabies
- Infestation by the mite *Sarcoptes scabiei*
- Usually spread by skin-to-skin contact
- Characterized by **intractable pruritus**; itch is mediated by delayed type IV hypersensitivity rnx to the mites, eggs, feces; worse at night, Dx if often missed
- Complications: secondary bacterial infections
- Incubation period: **1st episode of scabies – pruritus usually begins within 1 mo after exposure & the generalized hypersensitivity eruption 1-2wks after**
- Reinfestation: pruritus begins immediately
- Finding the mite: highest yield in identifying a mite is in burrows on the finger web, flexor aspects of the wrists, and penis
- Scabies prep: drop of mineral oil placed over burrow → burrow scraped off with 15 blade + placed on slide → drop of oil placed on scraping, which is then covered by coverslip
  - 3 findings: mite, their eggs, & their fecal pellets (scybala)
  - Norwegian scabies = very contagious form of scabies
    - Generalized distribution (even head & neck in adults), Lesions are often markedly hyperkeratotic and/or crusted
    - More common in immune-compromised individuals
- Treatment
  - Scabicides – Permethrin 5%, Crotamiton 10%
    - Apply thin layer to entire body from neck down, wash off after 8-24hrs
    - Repeat a week later (to kill the eggs that might have hatched)
  - Lindane 1% (avoid in young kids, and pregnant women), Ivermectin
  - Household members and close contacts should be treated, after eradication of scabies, pt may develop persistent itch & dermatitis

### Spider Bites
- Most important venomous spider in the US is the brown recluse (loxosceles reclusa)
- Most bites occur while patient is in bed
- Clinical features
  - Pain occurs 3 hrs after bite, sx 4-6 hrs after – rapid blood coagulation, sinking macule, pale gray, slightly eroded l center, tender, inflammation & hemorrhage
  - An acute necrotic skin injury lasts 10-15 days
  - Black widows can cause neurologic sx: aches/spasms
- Treatment
  - Most can be managed with analgesics and local care
  - Neurologic manifestations of black widow bits can be managed with diazepam and calcium gluconate
  - Antivenin rarely is indicated

### Flea Bites (pulicosis)
- In some cases, 48 to 72 hours after being bitten, a more severe rash-like irritation may begin to spread across the body.
- Symptoms include swelling of the bitten area, erythema, ulcers of the mouth and throat, restlessness. In extreme cases, within 1 week after being bitten, the condition may spread through the lymph nodes and begin affecting the central nervous system. Permanent nerve damage can occur.
- Dx is confirmed by the visualization of live lice
  - Nits (eggs) also be seen with the naked eye or under wood’s lamp which makes them look pale blue in color
  - “They attach to the hair shaft; looks like little pearly balls”
- Lice treatment
  - Permethrin, pyrethrin, and malathion are first line
  - Lindane or ivermectin are alternaties
  - Petroleum jelly can suffocate
  - Must tx household members and close contacts

### Bee Stings
- **Symptoms**
  - Immediate pain, swelling at the site of the sting,
  - A sensitive victim may experience swelling, urticaria, coughing, wheezing, coma, and respiratory arrest.
  - *Most serious reactions to bee stings occur in the first 30 minutes; however, the local effects of a sting may persist for 2 or 3 days. Delayed hypersensitivity may occur 7 to 10 days after the sting.*
- **Presentation:** Urticaria, Dyspnea, Hypotension
- **Treatment:**
  - Epinephrine, diphenhydramine intravenously, ranitidine intravenously
  - Repeat Epinephrine IM every 5 minutes until symptoms resolve
- **Disposition:** First, observe in ER
  - After 1 hour, these individuals should be totally free of symptoms. A biphasic allergic reaction can occur in up to 20% of patients, any patient requiring epinephrine should be watched for at least 6 hours and may require 23-hour observation.
  - There is a possibility of recurrence of the reaction up to 72 hours, and patients should be warned of this in their discharge instructions.
<table>
<thead>
<tr>
<th>Bacterial Infections</th>
<th></th>
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<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cellulitis</strong></td>
<td><strong>Impetigo</strong></td>
<td><strong>Erysipelas</strong></td>
<td><strong>Hidradenitis Suppurativa</strong></td>
</tr>
<tr>
<td>▪ Acute, spreading infection of the dermis and subcutaneous tissue</td>
<td>▪ Usually <strong>staph aureus or GAS</strong> causing superficial infx involving epidermis (impetigo) or extending into dermis (ecthyma)</td>
<td>▪ Usually caused by <strong>GAS</strong></td>
<td>▪ Chronic abscess of apocrine sweat glands</td>
</tr>
<tr>
<td>▪ Start empiric abx for <strong>h. flu, strep, and staph</strong></td>
<td>▪ Characterized by small vesicles or pustules that break easily forming crusted erosions (honey-colored crusts) with moist erythematous base</td>
<td>▪ Most common cause of virulent soft tissue infx in healthy host</td>
<td>▪ <em>I&amp;D</em>, systemic abs, surgical resection if recurrent</td>
</tr>
<tr>
<td>▪ Clinical features</td>
<td>▪ Bullous or nonbullous</td>
<td>▪ Face is most common area</td>
<td>▪ Unknown etiology, F&gt;&gt;M (between puberty and menopause)</td>
</tr>
<tr>
<td></td>
<td>▪ May have LAD, fever, chills, or malaise</td>
<td>▪ Tx: abx (PCN 1st line, if allergy use erythromycin or azithromycin)</td>
<td>▪ Typically, in axillae and anogenital region</td>
</tr>
<tr>
<td>▪ Treatment</td>
<td>▪ Predisposing factors: obesity, acne, DM, FH</td>
<td>▪ Hallmark is <strong>double comedone</strong> w/ tender inflammatory nodules/abscesses; progress to cystic suppurative lesions and sinus tracts; fibrosis and scarring may occur</td>
<td>▪ Cx for secondary bacterial infection</td>
</tr>
<tr>
<td></td>
<td>▪ If there is a poor response to antimicrobial therapy or a necrotizing, soft tissue infection is suspected, surgery is necessary</td>
<td>▪ Cx for secondary bacterial infection</td>
<td>▪ Tx: BPO/triamcinolone, abx, intralesional steroids, I&amp;D, surgery, isotretinoin</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Fungal infections</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Candidiasis</strong></td>
<td><strong>Dermatophyte (tinea) infections</strong></td>
<td><strong>Tinea Versicolor</strong></td>
<td><strong>Tinea Versicolor</strong></td>
</tr>
<tr>
<td>▪ Yeast (candida albicans m/c)</td>
<td>▪ <strong>Trichophyton, microsporum, epidermophyton</strong></td>
<td>▪ Nondermatophyte, lipophilic yeast <strong>Malassezia furor</strong></td>
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</tr>
<tr>
<td>▪ Sx</td>
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<tr>
<td></td>
<td>▪ Redness, itching, discharge</td>
<td>▪ Well-demarcated scaling plaque</td>
<td>▪ Hypopigmented or erythematous macules with fine scale, no itch, predominantly on trunk</td>
</tr>
<tr>
<td></td>
<td>▪ Candida intertrigo (skin folds)</td>
<td>▪ Tinea capitis, tinea faciei, tinea barbae, tinea corporis, tinea cruris, tinea manuum, tinea pedis, tinea unguium</td>
<td>▪ KOH → spaghetti &amp; meatball pattern w/ short hyphae &amp; yeast</td>
</tr>
<tr>
<td></td>
<td>▪ Thrush (cottage cheese)</td>
<td>▪ 10% KOH → <strong>hyphae</strong></td>
<td>▪ <strong>Tx:</strong></td>
</tr>
<tr>
<td></td>
<td>▪ Perleche (angular cheilitis)</td>
<td>▪ <strong>Tx:</strong></td>
<td>▪ <strong>Fx:</strong></td>
</tr>
<tr>
<td></td>
<td>▪ Balantitis, Paronychia</td>
<td>▪ Terbinafine (Lamisil) x1-2wks</td>
<td>▪ <strong>Fx:</strong></td>
</tr>
<tr>
<td></td>
<td>▪ <strong>Tx</strong></td>
<td>▪ (allyamines)</td>
<td>▪ <strong>Fx:</strong></td>
</tr>
<tr>
<td></td>
<td>▪ Nystatin</td>
<td>▪ Ketoconazole x2wks</td>
<td>▪ Griseofulvin PO for tinea capitis &amp; unguium</td>
</tr>
<tr>
<td></td>
<td>▪ Ketoconazole x2wks</td>
<td>▪ Griseofulvin PO for tinea capitis &amp; unguium</td>
<td>▪ Avoid steroids and nystatin</td>
</tr>
<tr>
<td></td>
<td>▪ Fluconazole (Diflucan)</td>
<td>▪ Griseofulvin PO for tinea capitis &amp; unguium</td>
<td>▪ Avoid steroids and nystatin</td>
</tr>
<tr>
<td></td>
<td>▪ Ketoconazole</td>
<td>▪ Griseofulvin PO for tinea capitis &amp; unguium</td>
<td>▪ Avoid steroids and nystatin</td>
</tr>
<tr>
<td></td>
<td>▪ Selenium sulfide</td>
<td>▪ Griseofulvin PO for tinea capitis &amp; unguium</td>
<td>▪ Avoid steroids and nystatin</td>
</tr>
</tbody>
</table>
### Neoplasms

#### Basal Cell Carcinoma
- Most common skin cancer in US, slow growing, locally invasive, very low incidence of mets
- **Sx**
  - Flat, firm area with small, raised, translucent pearly papule with central ulceration and raised rolled borders
  - Most common on face, nose, trunk, often friable
  - May have telangiectastic vessels
- **Dx**
  - Punch or shave biopsy looking for basophilic cells
- **Tx**
  - Electric dissection/curettage
  - Moh's for difficult cases
  - Imiquod & 5FU

#### Squamous Cell Carcinoma
- 2nd most common, often preceded by actinic keratosis or HPV*, also sun and envt
- Bowen's disease = squamous cell carcinoma in situ (slow growing)
- Rarely metastasize
- **Sx**
  - Most common on lips, hands, neck and head
  - Red, elevated nodule with adherent white scaly or crusted bloody margins
- **Dx**
  - Biopsy
  - Epidermal or dermal cells with large, pleomorphic, hyperchromatic nuclei
- **Tx**
  - *Excision*, can do Moh's, XRT

#### Melanoma
- 80% caused by UV radiation, aggressive, high mets
- Increased risk if light skinned or xeroderma pigmentosum
- Most common cause of skin cancer death
- **Sx**
  - Diameter >6mm
  - Thickness is most important prognostic factor for mets*
- **Dx**
  - Full thickness wide excision biopsy with LN biopsy*
- **Tx**
  - Excision, possible LN dissection
  - High risk = a-interferon

#### Actinic Keratosis
- Pre-skin cancer
- Light skinned individuals
- Hx of excessive sun exposure
- Scaly red macules on sun exposed areas
- +/- tender
- Progression to squamous cell carcinoma variable (0.025-16% risk)
- Viewpoint: AK is histologically early SCC-in situ
- Common sites: face, ears, hands, forearms, chest, scalp
- May be pigmented and mimic melanoma
- Spares the adnexa (hair follicles and eccrine ducts)
- Treatment
  - Cryotherapy – liquid nitrogen freeze
  - Medical
  - 5-fluorouracil cream – topical chemo
  - Aldara cream – enhances local lymphocytic inflammation
  - Photodynamic therapy “blue light”
- Actinic Chelitis = AK on the lips
  - Can advance to SCC
  - Has ↑ risk of metastatic dz bc lips very vascular

### Hair and Nails

#### Alopecia Areata
- Non-scarring hair loss in sharply defined oval or round areas, typically of rapid onset
- Likely immunologic/T lymphocyte mediated
- May be associated with thyroid disease, pernicious anemia, Addison's disease, vitiligo, lupus, ulcerative colitis, DM, down syndrome
- **Tx:** topical steroids, intradermal injections of kenalog

#### Onychomycosis
- Dermatophyte infection of the nail
- Distal subungual infection most common
- Pt. may also have yeast (Candida) or mold infection

#### Paronychia
- Bacterial infection of proximal and lateral nail fold
- Pus accumulates behind cuticle or deeper in lateral nail folds
- Causes rapid onset of pain and swelling
- **Tx:** I&D, ABX treatment for stoph infection

#### Paronychia caused by Candida
- More assoc w/ chronic paronychia (dish washes, surgeons, dentists),
- Infx of the proximal and lateral nail folds causes erythema, pain, swelling
- Oral fluconazole 150mg qd 1-2wks; avoid irritation; wear cotton under vinyl glove

#### Lipomas
- Most common benign fatty tumor
- Growth of fat cells in a thin, fibrous capsule subcutaneously
- Most often on neck, torso, upper thighs, upper arms
- One or more may be present at the same time (5% multiple)
- Mobile with soft, rubbery consistency
- Should not cause pain
- May stay same size or grow slowly over the years
- Histology: well-circumscribed mass of mature adipocytes
- **Tx:** surgical excision
**Urticaria**

- Acute (<6wks) or chronic (>6wks)
- Pruritic, common, distinctive reaction pattern; affects 15-20% of population
- **Type I IgE / complement mediated reaction**
- Transient edematous pink to red plaques with central clearing vary in size and shape; individual lesions last <24 hrs
- Etiology: usually undetermined; caused by histamine release induced by allergies (such as food, airborne allergens, meds, heat/cold, stress, infection)
- More common in atopic individuals
- Angioedema – hive-like swelling caused by increased vascular permeability in subQ tissue
  - deeper variant of urticarial & somewhat less pruritic, but burns and swelling may be painful (may affect face, lips, soles, parts of extremity)
- Mainstay of tx = antihistamines (hydroxyzine max of 100mg qd)
  - Prednisone, epi, Xolair (for CIIU only)
- Chronic cases - pt worked up for internal diseases (cancer, infections, lupus, thyroid)

**Vitiligo**

- Depigmenting dz of unknown origin that causes destruction of melanocytes
- Family hx in 30% of cases
- Increased incidence of autoimmune thyroid dz and AA
- **Wood’s lamp accentuates depigmented areas**
- Tx: sunscreen, topical steroids (caution w/ atrophy and eye exposure – glaucoma), *tacrolimus (Protopic), PUVA, narrow band UVB, later; destroy pigment w/ 20% monobenzone

**Erythema multiforme**

- Can be induced by drugs → sulfonamides, phenytoin, barbiturate, PCN, allopurinol
- Infections → herpes simplex, mycoplasma
- Or idiopathic (50%)
- Half of all cases occur in patients under 20
- Previous hx is a strong risk factor
- Clinical features
  - Target or iris lesions are characteristic
  - Lesions can be localized to the hands or feet or become more generalized
  - Mucosal lesions are a hallmark and can be painful and erode
  - Patients have fever, weakness, and malaise, lungs and eyes may be effected
- Treatment
  - Avoid precipitating substance
  - If herpes, can use acyclovir
  - Severely ill patients are treated with systemic steroids

**SJS & TEN**

- These are mucocutaneous blistering reactions most often caused by a drug reaction
- Common drugs → sulfonamides, aminopenicillins, quinolones, cephalosporins, tetracyclines, phenobarbital, carbamexapine, phenytoin, valproic acid, oxicam, allopurinol, and corticosteroids
- **SJS → is thought to be a severe form of EM, and TEN is a severe form of SJS**
- Can occur in patients of any age
- Thought to be an immune-mediated response
- Danger is mostly in secondary infection, fluid loss, electrolyte imbalance, **TEN can be life threatening**
- Clinical features
  - Fever, photophobia, sore throat, mucosal inflammation, and sore mouth
  - Progresses over 4 days, sheet like loss of epidermis
  - Flaccid blisters with positive Nikolski sign
  - TEN has a higher fever and more severe epidermal separation and loss
  - Regrowth of skin takes 3 wks, and is delayed in pressure points
  - 90% of patients have mucosal lesions in GI tract
  - Other complications → ATN, erosion in the lungs and gut, bronchitis
- Lab studies
  - Patients have anemia and lymphopenia
  - Biopsy is diagnostic
- Treatment
  - Prompt withdrawal of offending agent
  - Can be transferred to burn unit for care
  - Treat electrolyte imbalances and infections
  - Debate about whether corticosteroids are helpful
  - IVIG is commonly used
### Pressure ulcers

- **Risk factors** ➔ Advancing age, FH, increased BMI, smoking, h/o LE trauma, prior DVT, pregnancy, poor circulation, venous insufficiency, disorders of clotting, diabetes, sickle cell, neuropathy, renal failure, HTN, lymphedema, inflammatory skin diseases, smoking, genetics, malignancy, meds
- **Screening** ➔ Recommended annually with visual examination and monofilament test (checks most common sites of ulceration)
- **Signs & symptoms** ➔ Ulcers with punched-out borders with calloused surrounding skin, underlying neuropathy
  - C/o tired, heavy legs, leg pain, or leg swelling
  - Telangiectasias, reticular veins, and varicose veins
  - Edema, inflammation, pruritic dermatitis
  - Ulcers with irregularly shaped borders along the medial ankle or saphenous veins that are tender, shallow, exudative, and have a base of granulation tissue
  - Skin discoloration or redness, may appear shiny or tight
  - 20% of symptomatic patients will have no visible clinical signs
- **Workup** ➔ Ankle-brachial index with symptoms of PAD
- **Management** ➔
  - Comprehensive assessment of ulcer and patient’s overall medical condition
  - Classification of wound at each follow-up
  - **Debridement**, local wound care, pressure relief, infection control, and proper dressing selection
  - Negative pressure wound therapy following debridement after infection, necrosis, or amputation
  - **Revascularization for critical wound ischemia**
  - SCDs for patients refractory to stockings
  - Aspirin therapy accelerates healing
  - Referral to subspecialty for slowly healing ulcers, persistent dermatitis, or recurrent cellulitis
  - Negative pressure wound therapy following infection control, and proper dressing selection
  - Debridement after infection, necrosis, or amputation

### Burns

- **First Degree Burns**
  - Superficial burns only involving the epidermis
  - Ex. sun burns, “flash” burns
  - **No blisters or edema, skin is pink or red, dry**
  - Will heal on their own in 3-6 days
  - Are not included in burn calculations
- **Second Degree Burns**
  - Partial thickness burn that involves the dermis
  - Superficial partial = small amount of dermis involved; caused by flame, scalding, or chemicals; moist, pink/red, edema, blistering, extremely sensitive to touch; heals in 10-21 days
  - Deep partial = significant amount of dermis involved, more than 50%; caused by grease, flame, or chemicals; fewer capillaries left = appears white, dry, moderate edema, decreased sensation & circulation ➔ minimal pain; may scar; may convert to full thickness burn; healing takes > 21 days
    - Tell these apart by degree of pain and pressure sensation
- **Third Degree Burns**
  - Full thickness burn, entire epidermis and dermis is gone, extends to subcutaneous fat
  - A result of prolonged exposure to any heat source
  - Extensive edema, dry, leathery, charred skin, no sensation or circulation
  - Will not heal spontaneously, requires skin grafting
- **Fourth Degree**
  - Penetration to the bone
  - Usually requires amputation

#### Calculation of Burn Area - “Rule of 9s” for adults

<table>
<thead>
<tr>
<th>Area</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head</td>
<td>4.5</td>
</tr>
<tr>
<td>Each arm</td>
<td>4.5</td>
</tr>
<tr>
<td>Torso</td>
<td>18</td>
</tr>
<tr>
<td>Each leg</td>
<td>9</td>
</tr>
<tr>
<td>Groin</td>
<td>1</td>
</tr>
</tbody>
</table>

**Management**

- Remove clothes, irrigate with room temp saline (not cold, will cause vasosconstriction), cover wound to prevent heat loss
- Use LR for IVF using Parkland formula to estimate needed amount, keep room warm, IV morphine, NG decompression, Tetanus booster
- Assess for smoke injury: facial burn, singed nose or facial hair, carbonaceous deposits in oropharynx
- Surgical management may be needed
- Indications for referral to burn center: > 10% body surface are with 2nd degree burns, any 3rd degree burns, electric or chemical burn, inhalation injuries, trauma

### Verrucae/warts

- **HPV**
- **Lab studies**
  - Microscopy shows hyperplasia and hyperkeratosis, koilocytotic squamous cells are present
  - Immunofluorescence can show HPB
- **Treatment**
  - Spontaneous regression
  - Salicylic acid, imiquimod (aldara), intralesional interferon, anogenital warts can be treated with trichloracetic acid or topical podophyllin
  - Surgical, HPV vaccine

### Allergic reaction/anaphylaxis

- **Typically an IgE-mediated reaction**

#### Signs & Symptoms

- Cardiopulmonary arrest: occurs in 5 minutes with iatrogenic anaphylaxis, 15 minutes in stinging insect anaphylaxis, and 30 minutes in food-induced anaphylaxis

#### Workup

- Transiently elevated plasma histamine or total tryptase

#### Management

- Epinephrine, intubation, oxygen, albuterol, epi infusion, referral
<table>
<thead>
<tr>
<th><strong>Pemphigus Vulgaris</strong></th>
<th><strong>Bullous pemphigoid</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>- Serious bullous autoimmune disease – IgG antibodies induce acantholysis, resulting in loss of cell-to-cell adhesion</td>
<td>- Itches, usually occurs in 60s or older</td>
</tr>
<tr>
<td>- This disorder occurs in middle aged adults</td>
<td>- Autoantibodies, complement fixation, neutrophil, and eosinophils cause bullous formation</td>
</tr>
<tr>
<td>- It is more common in people of Jewish or Mediterranean ancestry</td>
<td></td>
</tr>
<tr>
<td><strong>Clinical Features</strong></td>
<td><strong>Clinical features</strong></td>
</tr>
<tr>
<td>o Lesions usually start at the oral mucosa, skin lesions occur 6-12 months later, there may be pain or burning, but NO ITCHING</td>
<td>o Prodrome of urticarial lesions</td>
</tr>
<tr>
<td>o Weakness and malaise are common</td>
<td>o Bullae are large, tense, oval, and contain serous or hemorrhagic fluid</td>
</tr>
<tr>
<td>o Lesions are round vesicles that contain clear liquid and easily rupture</td>
<td>o Rupture less easily than pemphigus (negative Nikolski sign)</td>
</tr>
<tr>
<td>o Nikolski sign occurs, secondary infection as well as fluid and electrolyte imbalance are common causes of morbidity and mortality</td>
<td>o Typically, bullae collapse and crust</td>
</tr>
<tr>
<td><strong>Lab studies</strong></td>
<td><strong>Lab studies</strong></td>
</tr>
<tr>
<td>o Immunofluorescence of blister serum, look for IgG</td>
<td>o Biopsy and immunofluorescence</td>
</tr>
<tr>
<td>o Biopsy proves acantholysis</td>
<td></td>
</tr>
<tr>
<td><strong>Treatment</strong></td>
<td><strong>Treatment</strong></td>
</tr>
<tr>
<td>o Systemic therapy is required, start with prednisone, and then add immunosuppressive agents (azathioprine, methotrexate as needed)</td>
<td>o Systemic prednisone, azathioprine, mild cases or localized recurrences can be treated with topical steroids</td>
</tr>
<tr>
<td>o Dapsone, gold, and cyclophosphamide may help in severe cases</td>
<td></td>
</tr>
<tr>
<td>o Support therapies are fluid and electrolyte replacement, baths, wet dressings, topical steroids, and abx as needed</td>
<td></td>
</tr>
</tbody>
</table>

**Decubitus leg ulcers**