**Dermatology Notes** (GOOGLE ALL THE NECESSARY PICTURES)

* ***General terminology:***
  + **Primary skin lesions:**
* **Papule**
* **Solid elevation** of skin, **<1 cm wide,** well-circumscribed
* **Macule**
* **Flat** skin discoloration **<1 cm wide**
* **Plaque:**
* **Solid elevation** of skin, **>1 cm wide**, usually well-circumscribed
* When compared to a nodule, it is wider vs. higher
* **Nodule:**
* Solid elevation of skin, with its height much more obvious than its width (clinically)
* When histologically you find an epithelial lining around a cavity, then it is a cyst
* **Patch:**

**Epidermis:**

**C**alifornians **L**ike **G**irls in **S**tring **B**ikinis

> Stratum **C**orneum

> Stratum **L**ucidum [in thick skin]

> Stratum **G**ranulosum

> Stratum **S**pinosum

> Stratum **B**asale

--- --- --- Basement membrane ---

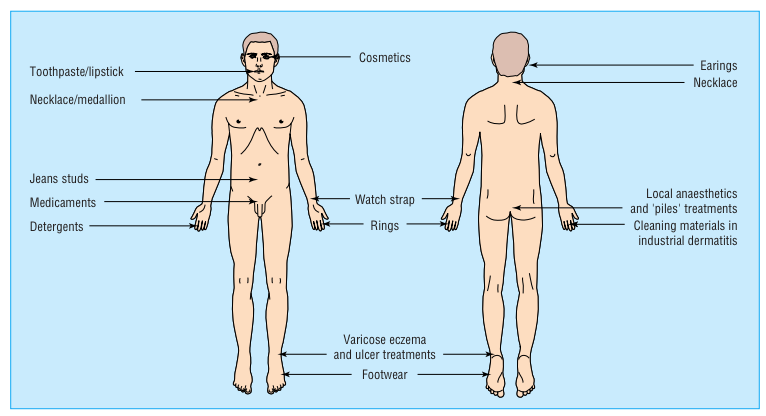
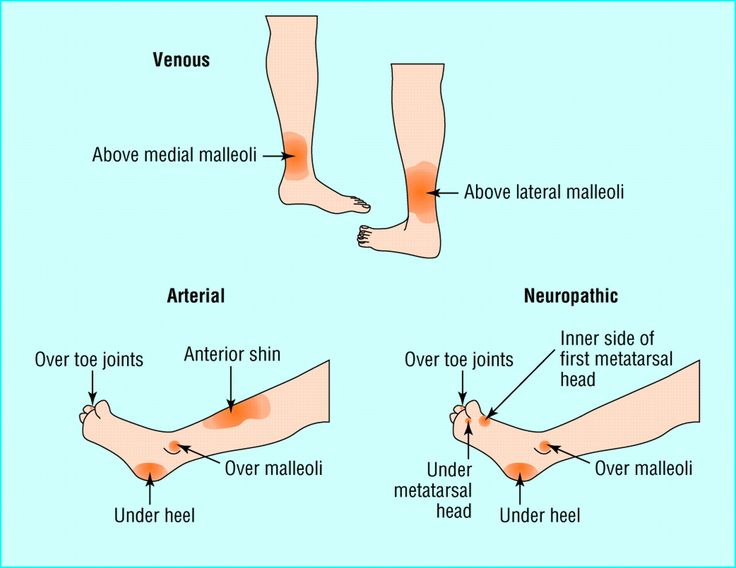
**Dermis:** consists of connective tissue (collagen and elastic fibers), nerve endings, hair follicles, sweat and sebaceous glands, and tiny blood vessels (& lymphatic vessels)

--- --- --- --- --- --- --- ---

**Hypodermis** (SQ tissue or superficial fascia): contains SQ fat mostly (but also has nerves, vessels that cross to and fo the dermis), in the breast it contains the mammary glands.

* Flat skin discoloration **> 1 cm wide**
* **Blister:**
* **Fluid filled** (non-pustular) **skin elevation**
* If **< 1cm = vesicle**
* If **> 1cm = bullae**
* **Pustule:**
  + Pus-containing skin elevation
* **Wheal:**
  + **Rounded** and **flat topped *transiently*** appearing **papules and plaques** (disappears in **24 – 48 hours**)
* **Erosion:**
  + **Discontinuity** of skin that only involves the **epidermis/epithelium** (vs. ulcer)
* **Ulcer:**
  + **Discontinuity** of skin that is deep enough to involve the **dermis or below** (SQ fat)
* **Secondary lesions:**
  + ***Crust***(ruptured blister and secondary infection 🡪 brownish crusting as in impetigo) – **dry exudate**
  + ***Scale*** (laminated **masses of keratin** of the **stratum corneum**)
  + ***Lichenification*** (**epidermal thickening** visible as **thickened skin**)
  + ***Atrophy*** (**loss of tissue**, which can present with **depressed or loose skin/wrinkling** or you can see the underlying vessels as **telangiectasia**)
  + ***Excoriations*** (punctate/linear **abrasions** secondary to **scratching** – if on back, it will spare the center producing the **butterfly sign** because the patient can’t reach there)
* **Other terms:**
  + **Configuration:**
    - **Annular** (ring-shaped)
    - **Arcuate** (arch-shaped)
    - **Discoid** or **nummular** (round/disc-shaped)
    - **Guttate** (resembling drops)
    - **Herpetiform** (resembling herpes –vesicles and/or in clusters)
    - **Linear**
    - **Reticular** (resembling a net/lace-like)
    - **Serpiginous** (wavy border)
    - **Stellate** (Star-shaped)
    - **Target or targetoid (**resembling bullseye)
    - **Verrucous** (wart-like)
  + **Distribution:**
    - Generalized
    - Flexural or extensor
    - **Intertriginous** (in areas where two skin areas rub on each other)
    - Morbilliform (like measles)
    - Palmoplantar
    - Perioficial
    - Periungual (under a fingernail or toenail)
    - Confluent (where individual skin findings merge the most)
* ****Dermatological conditions:
  + **Acanthosis nigricans – HIGH YIELD**
    - Related to **insulin resistance** (IR)
    - Seen in conditions associated with IR, such as **obesity, DM**, Cushing syndrome, acromegaly, **PCOS**
    - Can be secondary to **occult malignancies** (**GI** for example), the patient **would be wasted** (cachexic)
    - Can be **drug-induced** (GH, steroids)
    - Or **idiopathic**

****

* + **Lichen *P*lanus (Disease of Ps) – HIGH YIELD**
    - Flat-to***PP***ed
    - ***P***olygonal
    - ***P***urple
    - ***P***apules (that coalesce)
    - ***P***ruritic
    - It is an **idiopathic** condition, with the CLASSICAL TYPE involving the **dorsal surfaces of the hands, wrists, ankles, lower shins, lower back**
    - **Oral involvement** (50%) produces a white net-like distribution on the inner cheek, known as **Wickham striae** (it is painful, itchy)
    - ASSOCIATED WITH **HCV (hepatitis C)**
    - Lichen planus can show **Koebner phenomenon** (occurs in area of trauma)
    - **Eczema vs. lichen planus:**
      * Eczema typically begins with dryness, which is itchy 🡪 scratches 🡪 rash with excoriations (an itch that becomes a rash)
      * In lichen planus, the itchiness is also painful (pruritic) and the patient will typically rub on in it
    - **Management:**
      * **Potent topical steroids** (long term)
      * Oral findings might require **steroid mouth wash**
      * If not responding, you need to outweigh benefit and risk for use of systemic steroids
* **Psoriasis (“sadafiya”) – HIGH YIELD:**
  + **Autoimmune** systemic disease involving **T Cells** that most commonly affects the skin
  + Occurs in people with a **genetic predisposition** exposed to **environmental triggers**
  + **Triggers** in psoriasis includes:
    - **Stress**
    - **Trauma** (**Kobner phenomenon**) – **extensor** surfaces of elbow and knees
    - **Infections** (GABHS)
    - **Drugs** (antimalarials, ACEI, beta blockers, **LITHIUM**, systemic steroids when stopped is associated with rebound psoriasis)
    - Increased in winter month, patients **present in spring**
  + TYPICAL LESION IS:
    - **Erythematous (salmon-colored)** **well-demarcated** **plaques** and papules
    - Overlying **silvery adherent scales**, that when removed, will show **pinpoint bleeding (Auspitz sign)**
  + Types of psoriasis:
    - **Psoriasis vulgaris** (“common psoriasis”/non-pustular)
      * ***Chronic plaque psoriasis*** (MC subtype ~ >90%)
* Favorite locations are scalp, elbow, knees, foot, hands, lower back, and nail involvement (see below for specific entities)
  + - * ***Guttate psoriasis***
* Mostly papular, typically secondary to infections (GABHS) or other triggers (trauma/scratch sites)
  + - * ***Inverse psoriasis***
        + Involves the **flexures instead** (**inframammary**, **axilla**, inguinal area)
      * ***Nail psoriasis***
* Suspect it in **bilateral** limbs nail damage
  + - * + **Nail pitting** (MC feature)
        + **Onycholysis** (with “**oil spots**”) – separation of nail from nailbed; if you see it on a single nail or nails of one hand, suspect onychomycosis (fungal infection of nail)
        + **Subungual hyperkeratosis** (buildup of keratin in the space created by onycholysis)
        + **Beau’s lines** (deep grooved transverse lines that is thought to be a result of temporary cessation of nail growth)
        + **Splinter hemorrhages**
        + **Crumbling of the nails**
        + A person with nail psoriasis has a **high likelihood of having psoriatic arthritis**
        + **Psoriatic arthritis** (which is part of the **seronegative spondyloarthropaties**) is associated with involvement of the **DIP and PIP** (vs. RA) and results in the image of digits known as **SAUSAGE DIGITS** – if left unmanaged, it is can be **destructive** causing **arthritis mutilans**
      * ***Scalp psoriasis***
        + Easily confused with seborrheic dermatitis
        + It has the typical features of a psoriatic lesion, prefers the hairlines
      * ***Erythrodermic psoriasis***
        + The end point of any severe psoriasis
        + This occurs when **>90% of skin is involved** with psoriatic lesions (typically erythematous)
        + **Medical emergency**
    - **Pustular psoriasis**
      * Localized (**palmoplantar pustulosis**, acrodermatitis continua)
      * Generalized
      * Can progress to **erythrodermic psoriasis**
      * **Pustules are sterile** (other cause of sterile pustules? Drug-induced pustules)
  + **Other important systemic features of psoriasis:**
    - Psoriatic arthritis
    - Uveitis
    - Metabolic syndrome and related conditions (DM, HTN, heart disease and hyperlipidemia)
    - Depression
    - Note that because of increased antimicrobial peptides in skin, they are at a **decreased risk to develop skin infections** because of the lesions (unlike in eczema, where we would need to give topical antibacterial coverage)
  + Diagnosis:
    - Typically clinical diagnosis, but confirmation is by biopsy of skin lesion showing histological findings
  + **Management:**
    - **Localized disease** (monotherapy for localized disease):
      * **Topical steroids + topical vitamin D** +/- topical retinoids
      * **Topical calcineurin inhibitors (tacrolimus)**
        + If unresponsive to topical steroids
        + More useful in **INVERSE psoriasis** (use of steroids in inverse psoriasis is difficult because too much penetrates because of the moisture and coverage in flexural creases increases absorption of steroids 🡪 more ADR)
        + Associated with less ADR
      * If unresponsive, you can switch to systemic drugs
    - **Systemic disease**
      * **Phototherapy + topical agents** (as above, for tough areas) and PUVA (UVA + psoralen)
        + No phototherapy for erythrodermic
      * **Methotrexate** (MUST check LFTs and CBC because of hepatotoxicity and bone marrow suppression; close monitoring, and is associated with derangements in glucose and TFTs – also increased risk of fatty liver disease and DM)
      * **Cyclosporine** (especially for those who want to get pregnant)
      * **Systemic steroids** and **retinoids**
      * **Monoclonal antibodies** (IV, expensive; includes the anti-TNF agents and anti-IL-17… Problem with them is might increase resurgence of latent infections including viruses and TB as well as unmask the symptoms of LUPUS and is associated with MS)
      * Alternative agents: tar
  + On a final note, the erythrodermic conditions that is important to know are:
    - Eczema
    - Psoriasis
    - PRP (pityriasis rubra pilaris)
* **Pityriasis Rubra Pilaris**
  + Very similar to psoriasis, but distinguishing features include:
    - **SMALLER SIZED** plaques, but **MORE WIDESPREAD**
    - It has a more **ORANGE-LIKE COLOR**
    - Associated with **ISLANDS OF SPARING**
    - It is **PHOTOSENSITIVE** (unlike psoriasis) and therefore unresponsive/worsens with phototherapy
  + It is associated with increased COP secondary to widespread capillary vasodilation beneath the skin (causing the erythroderma)
  + Management:
    - Like psoriasis, BUT ***no* phototherapy** (it is a photosensitive disease) – **avoid sun exposure**
      * Other conditions associated with photosensitive skin (porphyria, lupus, dermatomyositis)
* **Pityriasis Rosea**
  + Pitryriasis = flaking/scaling skin condition
  + HHV7 is thought to play a role
    - Note: HHV5 🡪 fifth’s disease/slapped cheek/erythema infectiosum; HHV6 🡪sixth disease/roseola/exanthema subitum; HHV8 🡪 Kaposi’s sarcoma
  + Begins with a HERALD PATCH:
    - This is a patch with a ***central*** scaling and which peels in a sort of collar pattern
    - This patch may be noticed or go unnoticed by the patient; the patient may present later, so ask them if they had seen a patch that went away or not
  + After 1 – 2 weeks of having the Herald patch, a rash breaks out
    - The rash follows the lines of cleavage in the back, producing a CHRISTMAS TREE PATTERN
    - The rash is papular with a central clearing and itchy
    - The distribution has been called a “1920s bathing suit” distribution (proximal extremities and trunk)
  + It has a benign course and typically goes away without Rx in weeks to months (give topical anti-histamine for itchiness, promote sunlight exposure,
  + DDx? Syphilis (secondary syphilis) – but it is typically on the palms and soles (vs. 1920s bathing suit in pitryriasis rosea)
* **ACNE VULGARIS**
  + Pathophysiology of **inflammatory acne**:
    - **Non-inflammatory phase**:
      * **Excess keratinization** (puberty, androgens, specific foods are all associated with **excess sebum** and keratinization) in the ducts of **pilo-sebaceous glands** of **hair follicles** become clogged and there is a build of excess sebum behind the obstruction (**closed/white comedone state**), promoting **bacterial overgrowth** (of **Propionobacterium acnes**)…
      * Eventually there is connection between the hair follicle and the exterior, resulting a **open/black comedone** state (the black spot is the keratin plug)
    - **Inflammatory phase:** 
      * The following stage involves the formation of **papules and pustules**, which can rupture or progress to **nodulocystic acne** (most severe form; which can lead to **pitted scars** that with primary intention will produce hypertrophic scars or even keloids in those predisposed) or heal (and may result in **post-inflammatory hyperpigmention**)
      * Severe form is called **acne fulminans**
  + **Management:**
    - **Comedone phase:**
      * **Topical retinoids** (**ALL OVER FACE**, not just the pimples) ~ isotretinoin
    - **Pustular phase:**
      * **Topical antibiotics** (on the pimples/pustules)
      * **Benzoyl peroxide** cream (the best initial thing to start with; no resistance; antibiotic and anti-inflammatory agent)
    - **With increased severity:**
      * **Topical retinoids + oral antibiotics (tetracyclines)**
      * **Oral retinoids** alone
    - ADR of retinoids:
      * Dry skin (exacerbates eczema)
      * **Teratogenic** and results in early closure of epiphyseal plates (female patients must sign consent, must do a pregnancy test and be on two OCPs) – MUST WAIT 3 MONTHS till trying to get pregnant (in reality only 1 month is needed, but we need to tell them 3 months anyway)
      * Hair loss, hypercholesterolemia, liver disease (do LFTs)
* **Rosacea** 
  + **Red-flushed skin** over the face secondary to dilated blood vessels (Demodex **mite** has been implicated in its pathogenesis)
    - The lesions are **itchy** and often produce a burning sensation
  + Thought to be due to **vascular hyper-responsiveness** to triggers such as:
    - **Sunlight**
    - **Hot foods**
    - **Alcohol**
    - **Exercise**
    - **Steroids**
    - **Hot or cold environments**, stress
  + The patient is **typically old** (vs. acne) ~ imagine a patient who is being Rx for acne and it doesn’t help at all
  + **Types:**
    - **Erythematotelangiectatic** (basically permanent erythema with flushing that is easily apparent with simple triggers/sun exposure
    - **Papulopustular rosacea** (often confused with acne)
    - **Ocular rosacea**
    - **Phymatous rosacea**
      * Complication of rosacea
      * Typically manifests on the nose as rhinophyma, which appears as an irregularly enlarged nodular looking nose
      * It can occur in other locations of the face (Chin, ears, eyelids, forehead, cheeks)
  + Management:
    - **Lifestyle changes** 
      * **Avoid sunlight exposure**, avoid spicy food and all above triggers
    - Topical agents
      * **Topical antibiotics** for their **ANTI-INFLAMMATORY** effects (topical metronidazole, clindamycin and benzoyl peroxide)
      * **Topical retinoids**
    - In more severe forms, oral antibiotics can be used (oral doxycycline, erythromycin)
    - For phymatous changes (e.g. rhinophyma), surgical options are considered
* **Eczema/Dermatitis**
  + **Inflammation of the skin** (histologically ~ spongiosis)
  + The underlying pathophysiology is described as an “**itch that becomes a rash**” and typically secondary to DRY SKIN:
    - **Dry skin** (which is exacerbated by certain triggers) becomes **itchy** causing the person to scratch, resulting in an **erythematous** swollen lesions that becomes secondarily excoriated from scratching (**acute dermatitis**)
    - This can then transform into a plaque with secondary scaling (**subacute dermatitis**) – which can be confused with psoriasis
    - Further scratching can result in **lichenification** of the lesions (**chronic dermatitis**), which can result in **lichen simplex chronicus**
    - The lesions result in damage in skin and are **prone to infection** (impetigo, folliculitis, HSV), so topical antibiotics are given when managed
  + **Types of eczema:**
    - **Atopic dermatitis/eczema *(MC type)***
      * Results from an **IgE mediated** allergic response to a trigger/allergen ~ the hygiene hypothesis has also been implicated (i.e. lack of exposure to allergens early in life)
      * **Ask for history of atopic conditions** in the patient and his/her family + unusual exposures to **identify trigger**
      * Look in P/E for **signs of other atopic conditions** (**allergic salute**, **dennie-Morgan lines**/folds, **allergic shiner**, **pityriasis alba** or **post-inflammatory hyperpigmentation**)
      * Pityriasis alba is a pale patch in areas which has had dermatitis previously, typically seen in children (it can also occur secondary to topical steroid use)
      * **Allergy to food uncommonly manifests as eczema** (only in 20% you’ll find a food related allergy)
      * **Triggers include:** 
        + Hot and cold weather
        + Dust mites
        + Sweating
        + Stress
        + Certain foods (20% of cases; eggs, nuts, shellfish)
      * In **infants** (50% are diagnosed in the first year of life), it **favors the face** but can occur anywhere but typically spares the diaper area
      * **In children**, the **flexor areas** of the upper and lower limb become more involved (but can still affect other parts of the body)
      * In **adults**, it typically affects the **flexor areas**, but high distribution in the **wrists, hands and feet**
    - **Contact dermatitis** (divided into **allergic** and **irritant** types)
      * Occurs through a **type 4 HSR** (cell-mediated)
      * **Allergic contact dermatitis** varies from person to person
        + Nickel, jewelry, hair dyes, rubber gloves, creams, clothing, watches, scissors, belt, preservatives, perfume, cement (chronic exposure), posion ivy
        + ALWAYS CORRELATE THE LOCATION OF SKIN LESION TO WHAT NORMAL HUMAN BEINGS WEAR/HAVE ON THIS LOCATION
        + Reaction occurs within **2-4 days of SECONDARY exposure to allergen**
      * **Irritant contact dermatitis** results from repeated exposure to chemicals (irritation) that typically cause a reaction in any person;
        + Typically occurs in exposed areas (face, hands, feet)
        + Always ask about occupation in DETAIL (do you come in contact with chemicals, plants)
        + The higher or longer the exposure the more likely it will be appear and be worse (no previous contact needs to be present for it to occur)
        + Examples include acids/alkalis and chemicals, detergents
      * Lesions can appear **erythematous, swollen with vesicles** (distributed in a **herpetiform manner**) and they are **very itchy** and the **vesicles may pop** before you get to see them
    - **Asteatotic dermatitis**
      * Prototype of the eczema pathogenesis
      * Typically produces a **“dried riverbed”** or cracked appearance
    - **Discoid/nummular dermatitis**
      * As the name says, it is discoid in shape; typically occurs in adults and in the lower limbs
      * **Looks like tinea corporis**
    - **Dyshidrotic dermatitis** (AKA **pompholyx**/palmo-plantar dermatitis)
      * Typically manifests as vesicles on the lateral aspect of **fingers**
      * R/O contact dermatitis first
    - **Autosensitization**
      * Reaction to infection and occurs far from the area of original infection (the eczematous lesion bears no infections) ~ fungal infections should be excluded too
      * Rx underlying infection 🡪 stop eczema
    - **Seborrheic dermatitis**
      * Typically shows little inflammation, but it is an eczematous reaction to increased oil secretion and overgrowth of **Malassezia furfur** fungus
      * Results in **flaking of the skin** typically on the face and **scalp** (this is what we call DANDRUFF)
      * In infants it is called cradle cap if it occurs on the head
    - **Stasis dermatitis**
      * Occurs in older patients secondary to **venous stasis** (on the overlying skin of varicose veins)
      * Because of the lack of blood flow through dilated veins, some blood is leaked out with resultant iron from Hb depositing in skin as a **brown pigment** (**hemosiderin**) – at the same time, a dermatitis like picture develops (it is **red, itchy**)
      * May indicate **impending venous ulcer** development
    - **Neurodermatitis (lichen simplex chronicus)**
      * The end product of long-standing lichenification of eczematous lesions results in lichen simplex chronicus
      * This is characterized as **thick leathery skin**
    - **Viral dermatitis** (eczema herpeticum or vaccinatum)
      * Damage to the skin in eczematous lesions allows viruses like HSV to gain access to the body easily, resulting in a severe disseminated herpes virus infection, which is detrimental (**life-threatening**) in infants
      * Eczema vaccinatum is a reaction to the chickenpox vaccine (although rare, it is very severe ~ avoid such vaccines in infants with a history of eczema)
  + **Tests you can do?**
    - Diagnosis is typically clinical, but you can perform:
      * CBC (eosinophilia)
      * Ig levels (elevated IgE levels)
      * Patch test (skin allergy test) and RAST tests for specific IgE
  + Management:
    - General measures
      * **AVOID THE TRIGGERS**
      * AVOID DRYNESS using ***MOISTURIZERS***
      * **Antihistamines** for itchiness (sedating ones at night)
    - **Atopic eczema**
      * **Topical steroids + topical antibiotics** (in all cases, because the lesions may increase risk of infections, especially by S. aureus ~ impetigo 🡪 crusted, weeping)
      * Topical antibiotics ~ **fusidic acid, mupirocin**
    - **Contact dermatitis**
      * Avoid the triggers
    - **Seborrheic dermatitis**
      * **Antifungal shampoo** or topical antifungals
      * Topical steroids can be used as well
    - **Pompholyx/dyshidrotic eczema**
      * Potent topical steroids
    - **Discoid eczema**
      * Topical steroids + topical antibiotics
    - **Stasis dermatitis**
      * Compression stockings, avoid prolonged standing, raise legs + moisturizers, topical steroids
    - **Eczema herpeticum** ~ oral or IV acyclovir
    - In secondary infections (e.g. impetigo), give oral antibiotics
    - Extra notes:
      * Strengths of topical steroids:
        + **Mild ~ hydrocortisone** (1%; 2.5%)
        + **Moderately potent** (**clobetasone** butyrate, betamethasone valerate, fluocinolone acetonide)
        + **Potent** (increased % of the moderately potent)
        + **Very potent** (0.05% clobetasol propionate, 0.3% diflucortolone valerate)
      * **DO NOT USE HIGH POTENCY STEROIDS ON FACE** ~ only mild steroids (hydrocortisone is ok)
      * Potent steroids are used only for short courses (7 – 10 days)
      * USE **WEAKER STEROIDS IN FLEXURES** (occluded and moist so it increases the absorption)
      * ADR ~ **skin atrophy** (alternative is topical tacrolimus, which has less ADR and CAN be applied to the face)
* **BULLOUS DISEASE** (Blistering skin conditions)
  + DDx:
    - **Eczema** (contact dermatitis, anhidrotic)
    - **Autoimmune** (rarer causes):
      * Pemphigus vulgaris and bullous Pemphigoid
    - **Systemic disease** (***dermatitis herpetiformis*** in **celiac disease**; porphyrias like PCT)
    - **Infections** (*Bullous* impetigo, SSS, herpes, impetigo)
    - ***Burns***, trauma, friction, *edema blister*
    - Congenital (hereditary epidermolysis bullosa)
* Recall that the **most common causes of blistering skin are infections** (**chickenpox, herpes, impetigo** and eczema)
* The only ones I’ll explain here are autoimmune and dermatitis herpetiformis (the others are either self-explanatory or will be explained at a later time)
* **Autoimmune blistering conditions**
  + **Bullous Pemphigoid:**
    - Auto-Ab against hemidesmosomes (the one that anchors the epidermal cells to the dermis)
      * On IF, there is a linear pattern
    - This results in collection of fluid between the epidermis and dermis, producing blisters that are large, tense and less easily breaks than the blisters in pemphigus vulgaris (e.g. the patient will present with intact blisters – then suspect bullous pemphigoid)
      * Large, tense, intact blisters (which may be hemorrhagic)
      * Favors limbs first; with increased severity, it begins to involve the trunk (increased severity 🡪 central)
      * It can be itchy
      * Typically spares the mucosa (vs. pemphigus vulgaris)
      * Patients are typically old (vs. pemphigus)
  + Pemphigus vulgaris
    - Auto-Ab (IgG4 ~ anti-desmoglein) against desmosomes (between keratinocytes)
      * On IF, there is a net-like reticular pattern (indicative of acantholysis)
    - This results in easy collection of fluid between the cells, producing blisters
      * The blisters easily break with gentle pressure (Nikolsky’s sign) and so patients typically present with erythematous, weeping EROSIONS where the blisters used to be
      * AFFECTS the MUCOSA (vs. pemphigoid)
      * Distribution typically begins in the head (mucous membranes included), neck, upper trunk 🡪 extremities (with increased severity) ~ OPPOSITE to pemphigoid (which begins distally and progresses proximally)
      * Age group is 2nd/3rd decade of life (vs. old age in pemphigoid)
  + **Management:**
    - **Diagnosis:**
      * Clinical, but confirm with **skin biopsy** (see pattern of blistering + immunofluorescence)
      * You never suspect this condition first (diagnosis of exclusion) ~ typically you consider it in the patient with a blistering condition not responding to your standard Rx
    - **Treatment:**
      * **Immunosuppressants:**
        + **TOPICAL very potent steroids**
        + In **severe cases**, **ORAL steroids** (prednisolone ~ 30 – 60 mg) and **steroid-sparing agents**
        + Typically long-term Rx (2 – 3 years), so watch and consider ADR
      * **Pemphigus requires HIGHER doses** of oral steroids (60 – 100 mg daily!) and Rx **may be needed LIFELONG!**
        + Consider other immunosuppressants including **Rituximab (anti-CD20)**
        + Patients either die from the disease or from ADR of medications ☹
* **Dermatitis herpetiformis**
  + Associated with **celiac disease (NOT IBD)**
  + **Herpetiform like rash** (**vesicles** grouped together)
    - It is very deep (at the dermal-epidermal junction)
    - **Extremely itchy** (all the vesicles are opened – crusted and eroded - by the time you see the patient, because the patient scratches it!)
    - Occurs in **extensors** (**elbows, knees** and back of neck)
  + Management
    - Gluten-free diet (control of celiac disease)
    - Dapsone (Avoid in G6PD deficiency)
* **ULCERS**
  + DDx:
    - **Venous ulcer (MC type)**
    - **Arterial ulcer**
    - **Neuropathic ulcer** (DM, leprosy, syphilis, syringomyelia)
    - Pressure sores (**decubitus ulcers**)
    - **Neoplastic ulcer** (SCC > BCC)
    - **Vasculitic** (beurger, PAN, temporal arteritis)
    - **Infectious** (cutaneous leishmaniasis, amebiasis)
    - Hematological
    - Others (including drugs, **pyoderma gangrenosum** and trauma)
  + **Venous ulcer**
    - Results from sustained venous hypertension in the superficial veins (secondary to incompetent valves in perforator or deep veins, resulting in varicose veins, or previous DVTs)
    - **Associated findings:**
      * **Lower limb edema**
      * **Stasis dermatitis**
      * Hemosiderin (brown) pigmentation
      * **Varicose veins**
      * Lipodermatosclerosis (induration, reddish brown pigmentation and inflammation)
    - **Features of the ulcers** (vs. arterial ulcers):
      * Not as bad as it actually looks (**large,** exudative**, sloping gradual edges**)
      * It is a **superficial ulcer**, that is typically **painless** (but it CAN be painful), and its base contains **slough** or **granulation tissue** (red)
      * Typically found in the lower limbs, **above the ankles**
    - Management:
      * **Duplex US** to study for venous insufficiency and DVTs AND to **EXCLUDE arterial disease** (note: DM can have both arterial and venous problems)
      * For the ulcer: **ulcer dressings**, **antibiotics if 2o infection**; in severe cases, **skin grafting**
      * **Compression stockings**, **raise the lower limbs**, refrain from long term standing
      * Rx underlying venous insufficiency (anticoagulants)
  + **Arterial ulcers**
    - Peripheral vascular disease in arteries can result in ulcers secondary to ischemia and necrosis of tissue ahead of the blocked vessel
    - **Ulcers:**
      * Occur at **pressure points** and distal extremities (**toes**, foot, **under heel**, **over malleolus**, or on the anterior **shin**)
      * They are **well-demarcated**, **smaller sized**, **PUNCHED OUT** ulcers that are **PAINFUL**
      * The base is covered with necrotic tissue
      * The digit or distal extremity (in which it is contained) may show **signs of gangrene**
      * Look for other signs on P/E such as **lack of peripheral pulses, loss of hair, cold and pale appearance** of the limb; and on history, such as being a **smoker**, history of HTN, CAD and **intermittent claudication**
    - **Diagnostic studies:**
      * **Duplex US** can confirm it, but the best is angiography
      * Ankle-brachial index < 0.8 indicates arterial disease
    - **Management:**
      * Keep ulcer **clean and covered**; provide **analgesia**; if necessary, vascular reconstruction; **Rx underlying arterial insufficiency**
      * YOU MUST MAKE SURE THAT AN ULCER IS NOT ARTERIAL BECAUSE IF YOU GIVE COMPRESSION STOCKINGS IT WILL MAKE THINGS WORSE!
  + **Neuropathic ulcers:**
    - Occur over **pressure areas** of the feet, **where repeated trauma** occurs (it is **well-demarcated** and **punched out**)
    - Commonly seen in **peripheral neuropathy** (DM patients don’t feel the injury and so the site of trauma progresses to an ulcer, which can also become secondarily infection and even progress to osteomyelitis)
    - Causes: **DM, leprosy, neurosyphilis**, syringomyelia, peripheral neuropathies
    - P/E ~ ***reduced peripheral sensation*** & ***reflexes***and ***normal*** peripheral pulses, ***surrounding callus***; dry, warm foot
      * Ulcer is usually on the **PLANTAR side of foot**
      * Bones and tendons can be seen through the ulcers ☹
    - Investigations that can help? Nerve conduction studies
    - **Management:**
      * **Keep ulcer clean,** prevent further pressure or trauma to the affected site (note: if severe damage occurs to cause necrosis, you might need to consider amputation)
      * **Preventative measures:** proper control of DM, **daily foot inspection**, **wear appropriately sized shoes** and go see a podiatrist
  + **Pressure sores/Decubitus ulcers**:
    - **High risk individuals:**
      * Elderly, immobile, unconscious or paralyzed patients, morbidly obese paitents
      * The **majority occurs in people in hospitals**
    - Results from skin ischemia from sustained pressure over a bony prominence (**heel, SACRUM**)
    - Can be graded (1 – 4)
    - Early sign = red-blue discoloration of skin 🡪 ulcers (in 1 – 2 hours!)
    - Management:
      * Prevention 🡪 pillows to **keep pressure off bony areas**, prevent friction, **frequent turning**
      * Rx 🡪 adequate nutrition, **occlusive moist dressings**, analgesia, **debridement** and grafting may be necessary
      * Rx underlying cause (tight blood glucose control in DM)
  + **Vasculitic:**
    - Causes: temporal arteritis, PAN, systemic scleorisis
  + **Infectious ulcers:**
    - Bacterial ~ gummas (3rd stage syphilis), mycobacteria
    - Viral ~ HSV
    - Fungal and parasitic (cutaneous leishmania)
    - Make sure to do a **wound swab for gram stain and microscopy** + **C&S**
  + **Tumors:**
    - SCC (which can ulcerate or keratinize)
      * **Non-healing ulcers are SCC until proven otherwise**
    - Basal cell carcinoma, melanoma, Kaposi’s sarcoma
* **URTICARIA AND ANGIOEDEMA**
  + **Urticaria** (UR-TI-CARIA ~ because I used to call it uritcaria ☺)
    - AKA Hives
    - Characterized by acute development of itchy wheals as a result of swelling of the upper dermis secondary leaky dermal vessels
      * As opposed to angioedema, which is due to leaky SUB-DERMAL vessels
    - Pathophysiology
      * Cutaneous mast cell degranulation releasing histamine and other inflammatory mediators
    - **Typical culprits:**
      * Drugs (Radiocontrast dyes, **ACE inhibitors, penicillin, NSAIDs**)
      * **Allergies** (food allergies)
      * Autoimmune
    - **Skin manifestations:**
      * **Erythematous, very itchy wheals** (temporary macules and papules)
      * Physical urticarias are caused by physical stimuli (cold, deep pressure, stress or heat, sunlight, water, chemicals)
      * If it lasts longer than 24 hours and leaves a bruise, consider urticarial vasculitis
    - **Management:**
      * **Oral antihistamines** (non-sedating [cetirizine, loratidine] or sedating)
      * **Avoid triggers** or Rx underlying cause if any
      * Patients with idiopathic urticaria can develop chronic urticaria
  + **Angioedema**:
    - Swelling secondary to leaky **SUB-DERMAL vessels** with similar pathophysiology as urticarial
    - Etiology:
      * **Similar to urticaria** (more commonly ACE inhibitors)
        + ACEI increases bradykinin by inhibiting its breakdown
      * **Hereditary angioedema** (Don’t give ACEI to people with this!)
      * **Allergies, anaphylaxis**
    - **Skin features:**
      * **SQ involvement** (not as obvious as urticaria, but you should always watch out for angioedema and anaphylaxis with urticarial patients)
      * This manifests **periorbital swelling**, **lip swelling**, **hand swelling** (BURNING instead of ITCHY)
      * **Soft-tissue swelling of the larynx and mouth** can **obstruct airflow** and this can be **life-threatening**
    - **Management:**
      * **Antihistamines**
      * Might require urgent **IM adrenaline or IV steroids** if there is laryngeal or mucosal involvement
    - **Hereditary angioedema:**
      * AD; C1 esterase inhibitor deficiency
      * Sx like angioedema but also **abdominal pain** secondary to **intestinal edema**
      * Rx of typical angioedema is ineffective (they **need FFP** for the C1 esterase inhibitors)
* **Hypersensitivity rashes:**
  + Those rashes/conditions that develop secondary to drugs or infections mainly
    - Urticaria
    - Maculopapular rash
    - Erythema multiforme
    - Stevens Johnson syndrome (SJS)/ Toxic epidermolytic Necrosis (TEN)
    - Drug-induced lupus (INH, hydralazine, quinidine, procainamide)
    - DRESS syndrome (lamotrigine)
* **Erythema multiforme (EM)**
  + **Hypersensitivity rash** of acute onset
  + Divided into **EM major** and **minor**
    - **EM major** involves more **severe mucosal involvement** (mouth, genitalia, conjunctiva) in addition to skin
      * In reality, 2 or more mucosal membrane involvement
    - **EM minor** – **without mucosal involvement** (only skin)
      * In reality, just 1 mucosal membrane involved
  + Frequently caused by **DRUGS or INFECTIONS**
    - 50% of time no identifiable cause
    - Infections:
      * **HSV** (MC identifiable cause; causes **EM minor**)
      * **Mycoplasma** (causes **EM major**)
      * Other viruses (EBV, HIV)
    - **Drugs** (TAKE A NOTE OF THESE):
      * Aspirin/NSAIDS
      * Sulfa drugs
      * Beta-lactams
      * AEDs (**Carbamazepine [MC],** lamotrigine)
      * Antivirals
      * Allopurinol
      * Anti-malarials
    - Other causes:
      * Autoimmune rheumatic diseases (SLE, …)
      * Vasculitidis (PAN, …)
  + **Clinical features:**
    - Erythematous annular **(“targetoid”) rash**
    - Symmetrical, commonly affects the limbs
    - Mucosal involvement 🡪 **ulceration (necrotic) ~ EM major** (mycoplasma)
    - The rash **resolves in 2 – 4 weeks** (but in HSV, it can recur again and again)
  + **Management:**
    - **Symptomatic Rx**/Rx underlying cause
    - **Stop offending agents**
    - In the case of recurring EM, oral acyclovir can help or immunosuppressants (azathioprine)
* **Stevens-Johnson Syndrome (SJS) & TEN:**
  + Triggers:
    - Same as that of drug-induced EM
    - **Focus is AED** (carbamazepine, lamotrigine)
    - 2 - 3 weeks after drug exposure
  + Clinical features:
    - **High fever**, patient is toxic-looking or unwell
    - **Widespread** atypical, asymmetrical **targetoid lesions**, **BLISTERS, BULLAE,** **NECROSIS**
      * **<10%** body involvement is SJS (**10 – 30% is SJS+TEN**; **>30% is TEN**)

**Causes of hemorrhagic crusts in mouth:**

- Pemphigus vulgaris

- Erythema multiforme

- SJS/TEN

- Herpes

* + - * **Nikolsky sign +ve** (skin sloughs off upon slight rubbing)
      * **≥ 2 mucous membrane involved** with necrotic ulcers
      * The **skin is PAINFUL!!!** (just like in SSSS and pustular psoriasis)
    - **Mortality risk is 5%** (It can be **FATAL**) in SJS and **>30% MR in TEN!**
      * Increased risk of **SEPSIS, DEHYDRATION, ELECTROLYTE DISTURBANCES** (imagine the involved skin is burned)
  + **Prognostic factors:**
    - % of skin involvement
    - Age (very young or very old ~ not good)
    - Tachycardia
    - BUN
    - Comorbidities
  + **Management:**
    - **A, B, Cs**
    - Stop offending agents
    - Admit to ICU/Burn unit
    - Continual monitoring
      * Vitals
      * RFTs, lactic acid, electrolytes, sepsis screen
    - Make sure there is proper wound care and mucosal hygiene is maintained
    - Medical therapy?
      * Controversial, but IVIG has shown to improve survival (if given early)
* Maculopapular rash drug reaction
  + MANY CAUSES of maculopapular rash:
    - Viral exanthems (measles, rubella, EBV [IM], etc.)
    - Other infectious causes (2nd stage syphilis, RMSF)
    - Vasculitic or autoimmune conditions
    - Drug-reaction
  + Rash:
    - The most confluent area is generally where the rash began
    - In the classical viral exanthems like measles and rubella, it begins in the head and neck and then progresses to involve the trunk and extremities
      * CBC shows lymphocytosis
      * Serology can help to determine the viral cause
    - In the drug-related maculopapular rash, it begins in the trunk and then distributes
      * It starts 1 – 3 weeks after drug initiated
      * It is extremely ITCHY
      * CBC shows eosinophilia
      * Sx goes away when drug is stopped and antihistamines are given
* **ALOPECIA:**
  + Hair loss that can result from a disorder in the hair follicle (non-scarring alopecia) or in the scalp skin resulting in permanent loss of follicle (scarring/cicatricial alopecia)
  + **Non-scarring alopecia DDx**:
    - **Androgenic alopecia** (MCC)
    - **Autoimmune** (alopecia areata)
    - Endocrine (hypothyroidism, androgens)
    - Micronutrient deficiencies (iron, zinc)
    - Toxins (heavy metals, anticoagulants, chemotherapy, vitamin A)
    - Trauma to the hair follicle (**trichotillomania, traction alopecia** [in long term cases it can be come scarring])
    - Others (**syphilis**, severe illness)
    - **Post-partum** or after severe stress (e.g. **Telogen effluvium**)
  + **Cicatricial (Scarring) alopecia DDx:**
    - **Discoid lupus**, follicular lichen planus
    - A lot of other scary sounding conditions
    - **Infectious causes** (post-cellulitis, tinea capitis, neoplasms like BCC, SCC)
    - **Physical agents** (mechanical trauma, burns, radiotherapy, caustic chemicals)
  + Specific entities:
    - **Androgenic alopecia (male pattern baldness)**
      * Most common type of non-scarring hair loss
      * Depends on genetic factors and **abnormal sensitivity to androgens**
      * **Receding frontal hair lining** and **thinning of the crown**
      * Rx ~ topical 5% minoxidil; oral finasteride (5aR inhibitor), anti-androgen therapy in women
    - **Alopecia areata:** 
      * **Immune-mediated hair loss** (occurs with other autoimmune conditions)
        + Can be precipitated by stress
      * **Patches of baldness**
      * Presence of broken **exclamation mark hairs** (narrow at the scalp, wider and more pigmented at the tip) ~ basically diagnostic
      * Regrowth of hair can occur slowly over months (initially can be white hair!)
      * Distribution:
        + All of scalp hair + eyebrows? Totalis
        + All of body hair? Universalis
      * Rx ~ **potent topical steroids** or immunomodulators; PUVA/UV light
    - Other notes:
      * Traction alopecia (hair tied and pulled for a long time)
      * Causes of increased hair growth in females (certain races are more likely to have hirsutism, but if virilizing features are present consider an endocrine screen);
      * Hypertrichosis = excessive hair growth in both sexes (seen in anorexia nervosa and certain drugs like cyclosporine or minoxidil use)
      * **Discoid lupus** 
        + Cheeks, forehead and nose
        + Lesion are hypopigmented, atrophic, and thick - but **borders are hyperpigmented**
        + **Avoid sunlight exposure**; use **super potent topical steroids (dermovate)** or oral steroids if topical agents fail; anti-malarials and dapsone can also be tried
* **Vitiligo**
  + Autoimmune disorder whose effects mainly fall on the skin causing **DEpigmentation** (*NOT* hypopigmentation)
    - **Cell-mediated destruction of melanocytes** (vs. reduced melanosomes and melanin because of tyrosinase deficiency in albinism)
    - Melanosomes are located in the stratum basale and stratum spinosum & in the hair follicles; the first ones to get destroyed (permanently) are those in the stratum basale; if disease is controlled early enough, the pigments in the hair follicles are spared and the areas of depigmentation will have speckled spots of dark pigment representing hair follicle melanin (“**follicular pigmentation” = GOOD SIGN**)
    - Vitiligo is associated with **Koebner’s phenomenon**
    - The depigmented sites might not be obvious in white people unless they go tanning or when it involves the follicular pigments (as in the eyelashes)
      * UV light can help contrast the depigmented skin from pigmented skin ~ SO USE **WOOD’S LAMP** IN VITILIGO
    - It frequently affects the hands and face and genitalia
    - Depigmented skin (for 1 year or more or with hair depigmentation) generally is irreversible
      * Note that **depigmented skin are more prone to sunburns and skin cancers**
  + **Types:**
    - **Localized**
    - **Generalized** (which includes universal vitiligo)
  + Investigations:
    - R/O other autoimmune conditions
    - Wood’s lamp (Bedside)
  + Management:
    - Very difficult to reverse, our hope is to prevent progression
    - Medical Rx:
      * **Topical steroids**
      * **Intralesional steroids**
      * For the face (around the eyes), **topical tracolimus**
    - Cosmetic procedures (skin grafts, depigmentation of whole body in the case of universal vitiligo ~ as in Michael Jackson)
    - “REMISSION”
      * **No new depigmentation for >6 months**
* Oculocutaneous albinism
  + Autosomal recessive disorder
  + Deficiency of tyrosinase 🡪 low melanin production 🡪 hypopigmented skin, hair and irises of the eyes
    - **NORMAL melanocyte NUMBER** (vs. vitiligo)
    - Pale skin, eyebrows and eyelashes and hair; iris colored typically pinkish
    - Lack of ocular pigment can result in photophobia and visual disturbances
    - The skin is **more prone to develop sunburns and skin cancer**
  + Management:
    - General measures
      * Avoid sunlight exposure
* **Hypertrophic scar (HS) and Keloid**
  + Healing of a scar can sometimes be unpredictable
  + In the case of **hypertrophic scar**:
    - The scar **remains within the boundary of the original scar**, however it is red and obviously raised
  + In the case of **keloid:**
    - The scar grows well **beyond the boundary of the original scar**
    - It is the result of **excess collagen** secondary to a multitude of factors including **genetic and racial background** (African americans more likely to get it) and possibly environmental factors
    - It can be **severely disfiguring, continue to grow** (vs. HS, which can fade in a year or so), and it regularly recurs after removal
    - Predilection sites include **earlobes, chest, upper back, shoulders, chin**
  + Management:
    - **Intralesional steroids**
    - Compression
    - Surgical excision can be tried, but always always do medical Rx after it or else it will come back and be much worse than before
    - Radiation and other options are considered
* **SKIN MANIFESTATIONS IN SYSTEM DISEASES:**
  + Psoriasis (see above)
  + **Dermatomyositis:**
    - Autoimmune condition resulting from **humoral inflammation** of the perimysium of muscle
    - Myalgias and proximal muscle weakness is classical
    - Dermatologic manifestations:
      * **Grotton papules**
        + Bluish red nodules or plaques on the knuckles
      * **Heliotropic rash** 
        + Purplish/violaceous rash over the eye lids
        + Often edematous
      * **Shawl sign**
        + Erythematous rash over neck, upper chest and shoulders
      * **Periungual erythema and telangiectasia**
    - It may be associated with other rheumatological conditions and it can also have **CARDIAC** (Arrhythmias, CHF, conduction defect) and **LUNG** (ILD) manifestations
    - Remember that in older adults it is **associated with occult malignancies** (GI, ovary, breast, lungs)
    - **Investigations**:
      * **Creatine kinase (CK) levels** (elevated ~ correlates with extent of muscle damage)
      * **Serology** (**ANA, Anti-Jo1, anti-Mi2** [better prognosis])
      * **EMG, MRI**
      * **Muscle biopsy**
      * Screen for occult malignancies (malignancy surveillance)
    - **Management:**
      * **High dose steroids**, immunosuppressants
      * **Hydroxychloroquine** are helpful for the skin conditions
  + **Reactive arthritis**
    - Typically **following infection** (e.g. GI or GU infection)
      * Reaction to infections most commonly **SSYCC** (salmonella, shigella, yersinia, campylobacter [MC] and chlamydia)
    - Reiter’s syndrome ~ can’t see, can’t pee, can’t climb a tree
    - Arthritis is typically asymmetrical and oligoarthritic (LL> UL)
    - Dermatological manifestations:
      * **Keratoderma blenorrhagica**
        + Vesico-pustular waxy lesion; brownish color
        + Desquamating edges
        + Soles and palms commonly affected
      * **Circinate balanitis** 
        + Serpiginous annular dermatitis of the glans penis
  + **Ehler-Danlos syndrome**
    - Loose skin, easy bruising, hypermobile joints
  + **Scleroderma/Systemic sclerosis**
    - Now called **systemic sclerosis** and divided into:
      * **Limited type** (**CREST syndrome**)
      * **Diffuse type**
    - In all cases, **Raynaud’s phenomenon** can occur
      * Vasospasm of vessel walls of the digits
      * White (no blood) 🡪 blue ( deoxyHb) 🡪 red (reactive hyperemia)
      * Raynaud’s disease if primary (idiopathic, benign, in females) and Raynaud’s syndrome if secondary (CREST, systemic sclerosis, CTD, SLE)
    - **Cutaneous fibrosis** 
      * **Tightening of skin of hands** (fingers ~ **sclerodactyly**) and **face** (**fish-mouth appearance**; bird-like facies cuz of **microsomia** and **pointed nose**) making it shiny
      * The tightening of skin of hand results in a **claw hand** appearance and **lack of creases**
      * There may also be **finger pitting**
    - In **CREST syndrome** (associated with **anti-centromere Ab**), there is **telangiectasias** (on the nails, fingers and face)
    - Don’t forget they also have **visceral involvement** (which is typically severe and can be fatal):
      * **GI involvement** (on top of the list is esophageal dysmotility)
      * **Pulmonary involvement** (**ILD and pulmonary HTN**) ~ **MCC of death**
      * **Renal involvement**
      * **Cardiac involvement**
  + **Erythema nodosum**
    - Tender blue-red nodules commonly over the shins or lower limbs
      * Will fade over 2 -3 weeks leaving a bruised appearance
    - Causes:
      * Sarcoidosis
      * Inflammatory bowel disease, reactive arthritis
      * Behcet’s disease
      * Lymphomas
      * Infections (bacterial gastroenteritis, chlamydia, TB, streptococcus and some fungal infections)
      * Drugs (OCPs, sulfonamides)
      * Idiopathic
    - Management:
      * Symptomatic (NSAIDs, bed rest, bandaging)
      * Rx underlying condition
  + **Pyoderma gangrenosum** 
    - Ulcerative condition associated with:
      * **IBD** (UC > CD)
      * Gammopathies (IgA plasma cell dyscrasias)
      * Behcet’s disease
      * Leukemias and lymphomas
    - **Ulcers:**
      * Rapidly growing, **PAINFUL**
      * **Jagged borders** that are **purplish**
      * **Necrotic, dirty ugly base** with overlying purulent surface
      * Similar to venous ulcer but **PAINFUL**
  + **SLE:**
    - 4/11 criteria of SLE is dermatological (4/11 also needed to diagnose):
      * **Malar/Butterfly rash**
      * **Discoid rash**
      * **Oral ulcers**
      * **Photosensitivity**
      * Arthritis
      * **Serositis**
      * ANA +ve
      * Neurological findings (cerebral lupus)
      * Immunological findings (ant-dsDNA, anti-Sm)
      * Renal findings (lupus nephritis)
      * Hematological findings (leukopenia, AIHA, ACD)
  + **Behcet’s disease**
    - Autoimmune vasculitic disease associated with genetic component (**HLA-B51**)
      * Please don’t be stupid Mohamed ☺
      * B51 = BEHCET; B27 = SERONEGATIVES
    - Associated with PAINFUL oral and genital ulcers
    - **Pathergy:**
      * **Needle prick site** shows **papulo-pustular lesions** within 48 hours (similar to Koebner phenomenon)
    - **Pyoderma gangrenosum** and **EN** can be seen
    - Other findings:
      * Arthritis
      * Eye involvement (uveitis, optic neuritis, etc.)
      * CNS findings (**meningoencephalitis, brain atrophy syndrome**)
      * GIT findings
  + **Vasculitides** 
    - Small vessel vasculitis
      * Petechiae or purpura (non-blanching)
      * HSP, hypersensitivity vasculitis and urticarial
    - Small-to-medium sized vessels
      * Wegener’s granulomatosis (granulomatosis with polyangitis)
      * Microscopic polyangitis
      * Churg-Strauss syndrome (eosinophilic granulomatosis with polyangitis)
    - Medium vessels:
      * Polyarteritis nodosa (PAN)
      * Kawasaki’s disease (mucocutaneous lymph node syndrome)
    - Large vessel vasculitis:
      * Giant cell (temporal) arteritis
      * Takayasu’s arteritis
  + **Livedo reticularis**
    - Vertical arteries that are stagnated (due to whatever cause) and slow drainage of veins 🡪 resulting in skin that has a mottled reticulated/lace-like purplish pattern
    - Causes:
      * Idiopathic (MC)
      * **Drugs (Amantadine)**
      * **SLE**
      * PAN
      * Thromboembolic (**cholesterol embolization** also)
      * SO MANY DAMN CAUSES!
  + **Renal diseases with skin manifestations**
    - **Uremic frost** in CRF and AKI
    - **Chronic severe pruritis** in CRF (MCC of chronic itching)
    - **Half-and-half nail** (and also terry’s nails) in CRF
    - **Calciphylaxis** in CRF
      * Can be preceded by livedo reticularis
      * **Metastatic calcification** of blood vessels near the skin surface results in damage and ischemia 🡪 necrotic ulcers form
      * Fatal condition
  + **Sarcoidosis:**
    - Chronic systemic granulomatous disease characterized by non-caseating granulomas (with lungs almost always involved)
    - The condition is most frequently seen in women, particularly in African Americans
      * Age group **20 – 40**
    - **Skin manifestations (25%):**
      * **Lupus pernio** (chronic, raised/indurated purplish skin lesion on nose, ears, lips, cheeks and forehead)
        + Associated with a poorer prognosis
        + Another is pernio (chilblains)
      * **Erythema nosodum**
    - Don’t forget about other manifestations and notes:
      * Cardiovascular involvement (arrhythmias, RCM, HTN)
      * CNS involvement (neurosarcoidosis 🡪 facial palsy, hypopituitarism, meningism?)
      * Arthritis and eye manifestations
      * Most patients get complete recovery, some of them progress
  + **Wilson’s disease:**
    - AR disorder in copper metabolism in the body, resulting in copper accumulation in various organs
    - Signs:
      * **Kayser Flescher rings** (also seen in PBC)
        + Best visualized with **slit lamp**
        + **Does NOT interfere with vision**
      * Copper in lunula of nails resulting in BLUE NAILS (AZURA LUNULA)
  + **Primary biliary cirrhosis**
    - Autoimmune condition that primarily affects the intrahepatic bile ducts resulting in cholestatic liver disease and eventually cirrhosis
    - Skin findings? Jaundice, **xanthomas, pruritis** (bile salts) and skin findings relevant to fat-soluble vitamin deficiencies (ADEK)
  + **Inflammatory bowel disease**
    - Dermatologic manifestations:
      * Erythema nodosum (CD>UC)
      * Pyoderma gangrenosum (UC > CD)
      * Oral ulcers (CD > UC)
  + **Hemachromatosis:**
    - Inherited (AR) disorder of iron metabolism, resulting in decreased excretion of iron as well as its deposition in various tissues, mainly the liver (but also the pancreas, brain, testis, and heart)
    - **Bronze skin color** (“Bronze DM”)
  + **Cirrhosis:**
    - Skin manifestations:
      * Nail findings ~ **Terry’s nails** (distal nail has an arc of darkened color – note that if this was half way through the nail, it would called half and half nails or Lindsay’s nails ~ seen in CRF); **leukonychia and clubbing**
      * **Duputyren’s contracture**
      * **Palmar erythema**
      * **Petechia**
      * **Gynecomastia**
      * **Spider angioma**
  + **Tuberous sclerosis**
    - Key = hamartomas, **AD disease**
    - Dermatological manifestations:
      * **Angiofibromas** (**adenoma sebaceum**)
        + Look like acne
        + Much smaller than neurofibromas
      * **Subungual fibromas**
      * **Phakomas** in the eyes
      * **Shagreen patches**
        + Roughened patches of skin
      * **Ash leaf spots** (seen with **Wood’s lamp**)
        + Depigmented patches
    - Other manifestations:
      * Seizures (West Syndrome) – cortical tumors (subependymal astrocytomas are common)
      * Rhabdomyoma
      * PKD
      * Angiomyolipoma
* **Neurofibromatosis**
  + Autosomal dominant
  + NF type 1:
    - Diagnosis requires 2 of the following:
      * 6 or more **café au lait spots** (> 5mm before puberty, >15 mm after puberty)
      * 1 or more **neurofibromas** (unsightly dermatological overgrowth that are firm, nodular and represent distal nerve ending abnormalities)
      * **Axillary freckles**
      * **Lisch nodules in iris**
      * **Optic gliomas**
      * First degree relative with NF
  + **NF type 2:**
    - Bilateral acoustic neuromas (schwannomas) causing cerebellopontine syndrome (CN7, CN8 palsy and cerebellar ataxia)
    - Increased risk of meningiomas
* **Sturge-Weber syndrome**
  + Port-wine spot (nevus flammeus) – non-neoplastic hemangiomas birthmark in CNV1 or CNV2 distribution
* Endocrine conditions with skin findings:
  + **Cushing syndrome:**
    - Moon facies, lemon-on-sticks appearance
    - Acne and plethora
    - Supraclavicular fat pads
    - Truncal obesity
    - Abdominal striae that are purplish
    - Easy bruising
  + **Hypothyroidism**
    - Loss of lateral 1/3rd of eyebrows
    - Dry and cold skin (dry skin is ITCHY)
  + **Hyperthyroidism**
    - **Graves disease:**
      * Graves acropathy (clubbing), wet moist skin
      * Ocular 🡪 exophthalmos, lid lag, lid retraction
      * Pretibial myxedema
      * Don’t forget about tremors, tachycardia,…
  + **Addison’s disease:**
    - Primary adrenocortical insufficiency (high ACTH) 🡪 hyperpigmented (Also seen in Nelson syndrome and Whipple disease)
  + **DM:**
    - Acanthosis nigricans (insulin resistance)
    - Neuropathic ulcers (painless but looks like venous ulcer) has a hard callus
* **Metastatic skin lesions:**
  + Paget’s disease of the nipple or extramammary paget disease
  + Paraneoplastic:
    - Dermatomyositis
    - Seborrhoeic keratitis
    - Hypertrichosis
* **Metabolic skin lesions:**
  + Hyperlipidemia:
    - Don’t forget to think of the different inherited hyperlipidemias, cholestatic diseases and nephrotic syndrome
    - Findings:
      * **Xanthelesma** around the eyes
      * **Xanthomas** (Tendon and/or palmar xanthomas, eruptive xanthomas)
  + **Hypovitaminosis C**
    - **Bleeding gingiva**
    - **Perifollicular hemorrhage**
    - **Cork-screw hair**
    - **Easy bruising**
  + **Vitamin B3 deficiency (pellagra)**
    - 3Ds:
      * **Dermatitis** (\*) on SUN-EXPOSED SKIN
      * **Diarrhea**
      * **Dementia**
  + **AMYLOIDOSIS:**
    - **Macroglossia**
    - Skin deposits
    - **Pinch purpura**
* **INFECTIONS of the SKIN:**
  + Let’s divide them into:
    - Bacterial
    - Viral
    - Fungal
  + **Bacterial:**
    - It’s best to remember it according to involvement of superficial layers of the skin to deeper layers:
      * **Impetigo** (epidermis)
      * **Erysipelas** (upper dermis)
      * **Cellulitis** (deep dermis and upper SQ fat)
      * **Necrotizing fasciitis** (deep tissue and muscle)
      * **Folliculitis** = superficial infection of hair follicle
      * **Furuncle** (boil) & **carbuncle** = deep infection of hair follicle
      * Others (**erythrasma**, scarlet fever, SSSS, chancre, chancroid)
    - **Impetigo:**
      * Superficial infection of the **epidermis**
      * Causes:
        + **Staphylococcus aureus**
        + **GABHS**
      * Common locations:
        + **Around the mouth and nose** (especially in children); **sites of eczema**
      * Description:
        + Vesicles rupture produce a **honey-colored crust** that represent dried exudate
        + **Spreads by direct contact**
        + In severe cases (**S. aureus** related toxin A), large blisters form resulting in **BULLOUS IMPETIGO**
      * Things to do?
        + Skin **swab** of affected area (send for **gram stain + C&S**)
      * Management:
        + **Topical antibiotics** (fusidic acid, **mupirocin**)
        + In severe cases, **oral antibiotics** are given for 7 – 10 days (anti-staph antibiotics ~ **flucloxacillin**)
        + Avoid school for 1 week
    - **Folliculitis:**
      * ***Superficial*** inflammation/infection of the **hair follicles**
      * Causes:
        + **S. aureus**
        + **Pseudomonas** (hot tub folliculitis)
        + Other gram –ve bacteria
      * Description:
        + Distribution is that of hair follicle architecture
        + There are **tender papules/pustules** (expect pus) at **site of hair**
        + Head, neck and trunk
        + The one involving the beard area = sycosis barbae
      * Management:
        + Same as impetigo
    - **Furuncle (boils) and carbuncles:**
      * **Deep infection** of the **hair follicles** (starts as folliculitis but then involves lower part of the follicles)
      * A **carbuncle are multiple furuncles** that have **grouped together** into one lesion, which involves mainly the back of the neck and posterior thighs
      * Causes:
        + **S. aureus**
      * Description:
        + Painful firm red swellings
        + May or may not release pus
        + Typically occur in occluded areas of the body (armpits, hip)
        + **High risk of progressing to an ABSCESS** (felt like a fluid containing bag on palpation)
      * **Investigations:**
        + **Swab of lesion** (and send for C&S, gram stain etc.)
        + If febrile, consider blood culture, CBC
      * **Management:**
        + **Incision and drainage** of abscess/large boils
        + If afebrile ~ trial of topical antibiotics
        + If **febrile ~ oral antibiotics** (anti-staph penicillins like cloxacillin flucloxacillin)
    - **Erysipelas**
      * Infection of the **SUPERFICIAL DERMIS** + **LYMPHATICS** (important, to distinguish cellulitis which is a lower dermis and SQ tissue infection)
      * Cause:
        + **GABHS (S. pyogenes)**
      * **Description:**
        + **Well-demarcated** **erythema** that is **hot, tender** (vs. cellulitis, unclear borders)
        + Can occur at **site of trauma/cuts** (think of venous ulcer)
        + Prefers the **FACE** > lower extremities (**cellulitis likes the LL**)
        + Fever can be present, but remember that the site of infection is very hot
        + It is non-purulent
      * DDx?
        + Venous stasis dermatitis/DVT?
      * **Investigations:**
        + **CBC** (**neutrophilia**)
        + If systemic symptoms are present: blood culture, ASO titer
      * **Management:**
        + Empirical antibiotics (oral or in systemic symptoms and signs, IV)
        + Choice of oral antibiotics: oral penicillin or amoxicillin
        + Choice of IV antibiotics: cefazolin, ceftriaxone, flucloxacillin
    - **Cellulitis** 
      * Infection of the **deep dermis** and **SQ tissue**
      * Causes: **S. aureus and GABHS**
      * Description:
        + **NOT well demarcated**
        + Area of **hot, tender erythema**
        + **LL** > face
        + Can be purulent or non-purulent
      * Investigations:
        + No need unless systemic features (fever, toxic-appearance, etc.) 🡪 blood culture, ASO titers, imaging (MRI or US to look for abscess or distinguish osteomyelitis in high risk patients)
      * Management:
        + If no systemic features 🡪 oral antibiotics
        + If systemic features 🡪 IV antibiotics
        + If there is evidence of an abscess 🡪 incision and drainage
    - **Necrotizing fasciitis:**
      * **Life-threatening** infection of the **deep tissues** (including muscles)
      * Not to be confused with gas gangrene (myonecrosis, C. perfringes)
      * **Causes:**
        + **POLYMICROBIAL (80%)**
        + GABHS, S. aureus
      * Description:
        + The patient appears **very ill, toxic-looking**, **high fever**
        + Patient has **PAIN OUT OF PROPORTION TO P/E** FINDINGS (this classical statement it also seen in acute mesenteric ischemia)
        + Typically **misdiagnosed as cellulitis** and **patient is not responding to antibiotics**
        + The infection **follows the plains of the muscle and tissue**
      * Management:
        + **IV broad spectrum antibiotics** (meropenem, + clindamycin)
        + **WIDE surgical debridement**
        + In severe cases, if in limbs, amputation may be considered
    - **Ecthyma gangrenosum**
      * Cause:
        + Pseudomonas aeruginosa
        + “Ecthyma” is by staph and strep
      * Seen in immunocompromised patients or those with sepsis (e.g. pseudomonas sepsis)
      * Results from perivascular invasion of dermis and SQ tissue
      * Description:
        + Round lesion with a necrotic center and a surrounding halo of erythema
        + DDx ~ cutaneous anthrax
      * Heal with scarring
    - Scarlet fever:
      * Cause:
        + GABHS (erythrogenic toxin)
      * Clinical features:
        + Pharyngitis followed by a systemic reaction to the presence of GABHS
        + Fever, sore throat
        + **Pastia’s lines** (red streaks) in the armpits
        + **Strawberry tongue, circumoral pallor**, forscheimer spots in mouth
        + Diffuse **SANDPAPER erythematous rash** with tiny papules **followed by desquamation** (occurs 7 – 10 days later)
        + Can it lead to GN? Yes (the pharyngitis can increase risk of RHD)
      * Management:
        + Same as strep throat (penicillin)
    - **Scalded Skin Syndrome (SSS)**
      * Causes:
        + Due to exfoliatin toxin (erythogenic toxin) spread into blood (NOT the bacteria itself… i.e. the blood and skin are sterile)
        + **S. aureus** (classically; SSSS)
      * Clinical features:
        + Fever, **erythematous rash that desquamates**
        + Note that the **superficial epidermis** is involved
        + It has a high mortality rate in children
        + Nikolsky sign +ve, multiple blisters may form
      * Rx ~ **anti-staph antibiotics**
    - **Toxic shock syndrome (TST)**
      * **Causes:**
        + **S. aureus** (classically ~ TSST)
        + **GABHS** (yes!)
      * **Clinical features:**
        + Fever, diffuse erythematous rash, mucous membrane involvement, blisters can form
        + Hypotension and organ failure ensue
        + Can occur secondary to use of tampons in women
      * Management:
        + A, B, Cs
        + Remove agent that causes it (e.g. tampon)
        + IV vancomycin, consider IVIG
    - **Erythrasma** 
      * Caused by **CORNYBACTERIUM** minutissimum (*not diphtheria*)
      * Clinical feature:
        + **Axilla and groin redness** that **resembles tinea cruris**
        + On **Wood’s lamp** 🡪 **COIL RED FLUORESCENCE**
      * DDx ~ tinea cruris
      * Rx ~ topical or oral macrolide
    - **Chancre:**
      * Cause:
        + **Primary syphilis** (Treponema pallidum ~ spirochete, gram –ve but too thin to be visualized)
      * **Notes:**
        + **Primary syphilis – chancre**
        + **Secondary syphilis – maculopapular rash** (INVOLVES PALMS AND SOLES) vs. pityriasis rosea and guttate psoriasis; condyloma lata
        + **Tertiary syphilis – gummas, neurosyphilis, thoracic aortic aneurysm**
      * Clinical features:
        + **Painless ulceration** (needs direct physical contact to infect others ~ locations are commonly **mouth, anus, vagina and penis**)
        + **Diminish 4 – 8 weeks without Rx**
      * **Chancre vs. chancroid**
        + **Painless** (vs. painful)
        + **Single** (vs multiple)
        + Regional **bilateral LN enlargement** (chancroid unilateral regional LN enlargement)
        + **Hard indurated base** with **sloping edges** (vs. chancroid ~ soft base with undermined edges)
        + Heals on its own
      * Management:
        + **IM penicillin G** (2.4 million Units for primary and secondary)
    - **Chancroid**
      * Cause:
        + **Hemophilus ducreyi**
      * DDx = chancre, LGV
      * Chancroids are/have:
        + **PAINFUL**
        + **SOFT**
        + **DIRTY NECROTIC BASE**
        + **DOES NOT HEAL EASILY**
        + Smears show a **SCHOOL OF FISH PATTERN** on gram stain
  + **Viral infections**
    - **Warts:**
      * **Common wart**
        + Irregular, verrucuous, thick raised lesions
        + Typically on exposed hands and feet
        + Caused by HPV 1, 2 and 4
      * **Flat wart**
        + Thin, seen in children
        + HPV 3 & 10
      * **Genital wart (condyloma accuminata)**
        + **6 & 11 = true wart**
        + If **16 & 18 = squamous cell carcinoma in situ** (predisposed to cervical cancer)
      * **Wart vs. corn**
        + Warts have **black dots when shaved** (which represent **thrombosed vessels**)
        + Warts are **multiple** and can occur anywhere
        + **Corns** are usually **single** and on **pressure areas**, and when shaved, **leave a hole**
      * Management:
        + **Cryotherapy** (best); if very raised or large, shave it before cryotherapy
        + **Electrocautery is not favored anymore** because the fumes may transmit HPV and can be inhaled resulting in laryngeal papilloma and cancer
    - **HSV:**
      * VERY PAINFUL!!!!
      * HSV-1 🡪 orolabial herpes (herpes labialis) and herpes gingivostomatitis
        + Primary 🡪 more severe, grouped, painful vesicles which can ulcerate
        + Secondary 🡪 reactivation of dormant HSV in stressful situations (less severe); canker sores
        + If other person puts finger in affected people mouth with the lesion 🡪 herpetic whitlow or herpes gladioatorum
      * HSV-2 🡪 genital herpes
        + Multiple vesicles that ulcerate in the genitalia
      * Continual shedding occurs after episodes
      * Watch out for eczema herpeticum and herpes encephalitis
      * Investigation:
        + Gold standard now is **DFA**
        + **PCR** or **Tzank smear**
      * Rx ~ reduces viral load (acyclovir)
    - VZV
      * Rapidly evolving skin manifestations:
        + Erythema 🡪 dew-drop vesicle 🡪 pustule 🡪 crusts
      * Most people have been immunized or had chickenpox in childhood (highly contagious)
      * In adults it manifests as shingles (Zoster), but can also lead to encephalitis and pneumonia in immunocompromised individuals
        + Shingles follows a dermatomal distribution (lays dormant in dorsal root ganglia) or trigeminal nerve (in trigeminal ganglion) – VERY PAINFUL skin vesicles (looks like? Contact dermatitis)
        + **CNV1 involvement can lead to corneal damage and blindness**
        + **Post-herpetic neuralgia** = very painful, doesn’t go away, hard to relieve (gabapentin, amitriptyline and opioids are options for pain relief)
        + Rx ~ does not eradicate, only reduce activity (valacyclovir or famciclovir or acyclovir)
    - **EBV**
      * Infectious mononucleosis
        + Sore throat
        + Maculopapular rash (alone or secondary to amoxicillin administration)
        + HSmegaly (avoid contact sports)
    - **Parvovirus B19 🡪 fifth’s disease** (erythema infectiosum/Slapped cheek syndrome)
      * Note: also associated with hydrops fetalis, polyarthritis (symmetrical – like RA), aplastic anemia
      * Note: hand-foot-mouth disease is by coxsackievirus
    - HHV6 🡪 sixth disease (roseola infantum/exanthema subitum)
      * High fevers (which can result in febrile seizure)
      * Followed by a diffuse rash
    - HHV7 🡪 thought to be related to pityriasis rosea (not to be confused with rosacea)
    - HHV8 (Aka KSHV) 🡪 Kaposi’s sarcoma
      * Violaceous/purplish/bluish-black macules and patches that can become plaques or nodules
      * Occur in immunocompromised individuals, particularly HIV +ve individuals approaching AIDS (AIDS-defining illness)
    - **Molluscum contagiosum**
      * Caused by **poxvirus**
      * **Pearly white**, **flesh-colored dome shaped** skin lesions
      * **Central umbilications** can be seen
      * May itch, and when scratched or touched can be spread further within the body (“**pseudo-koebner phenomenon**”)
      * Self-limiting condition (goes away on its own), but options can include cryotherapy or topical retinoids
    - Measles and rubella (☺ fuck off)
* **FUNGAL INFECTIONS**
  + **Candida**
    - **Oral candidiasis (thrush)**
    - **Esophagitis**
    - **Diaper rash/intertrigo**;
      * Typically shows **SATELLITE LESIONS**
      * **INVOLVES THE CREASES**
      * SHOWS **INFLAMMATION** (**very red**)
      * **Involves the mucous membranes**
      * This is all in contrast to contact dermatitis (e.g. to fecal matter in kids with diaper rash ~ which does not involve the creases)
    - **Candida onychomycosis**
      * DDx – nail psoriasis
      * Thick, destructive infection (vs tinea)
      * **Oral antifungals are needed** (oral itraconazole or **terbinafine** [not so good with candida])
  + **Dermatophytes (tinea) – “ringworm**”
    - HATE oily areas and mucous membranes
    - **Tinea capitus**
      * Typically in kids (less oily scalp)
      * If seen in adults, suspect immunocompromised
      * **Types:**
        + **Endothrix** (confined to hair shaft)
        + **Ectothrix** (spread out over hair surface)
      * Spread by **close contacts or hairdressers**
      * Early on, the hair loss is reversible, but eventually it becomes irreversible (a resultant **kerion develops**, which is a **boggy swollen mass** with copius pus and exudate + alopecia)
      * DDx – alopecia areata (looks cleaner and neat)
      * Ectothrix 🡪 **fluoresce under WOOD’S LAMP**
      * NEEDS **oral antifungals** (**griseofulvin**)
    - **Tinea corporis**
      * **Asymmetrical patches** with **scaly, erythematous borders** and **central clearing** (hence “ring”worm) – but don’t always expect central clearing
    - **Tinea cruris**
      * Same as tinea corporis but in **flexures**, **arc-like borders** extending down upper thigh
      * **NO mucous membrane involvement** AND **no satellite lesions (vs. candida)**
    - **Tinea pedis**
      * Athlete’s foot
      * Typically found **between the toe clefts** (looks **white, macerated and fissured**)
      * Toe nail involvement can be seen as well
    - **Tinea mannum**
      * Hands show diffuse erythematous scaling of the palms with some skin peeling and thickening
    - **Tinea unguium**
      * Tinea of the nail
      * Less severe onychomycosis than candida
      * Requires **ORAL antifungals (Terbinafine)**
    - **INVESTIGATIONS:**
      * Wood’s lamp for tinea capitis
      * **Scrapings of lesion 🡪 KOH 🡪 microscopy**
    - TREATMENT:
      * **All 🡪 topical antifungal creams** (clotrimazole, miconazole, terbinafine) – applied daily for **1 – 2 weeks**
      * **Widespread infection or tinea capitus or tinea unguium require ORAL ANTIFUNGALS**
        + Tinea capitus 🡪 griseofulvin PO
        + Tinea unguium 🡪terbinafine PO
  + **Pityriasis versicolor (“Tinea versicolor”)**
    - Caused by **pityrosporum**
    - Clinical features:
      * Common condition in **young adults**
      * Presents as **reddish-brown scaly macules on trunk** (asymptomatic)
      * In those who go tanning (black or white), it will appear as **areas of hypopigmentation**
    - Investigations:
      * **Yellow fluorescence** under **WOOD’S LAMP**
      * **Skin scrapings 🡪 KOH 🡪 spaghetti & meatball appearance**
    - Rx ~ **selenium or ketoconazole shampoo** (apply to body and remove after 30 – 60 minutes); pigmentary changes take a long time to reverse
* OTHER FUCKING THINGS:
  + Scabies:
    - Sarcoptes scabiei
    - Sites of predilection – web spaces of fingers and toes
    - Linear burrows
    - Very severe pruritis
    - Confirm by scrapings of skin, KOH 🡪 mite or egg visualized
    - Rx ~ topical permethrin (over all skin below neck; to all close contacts); wash and clean all clothes
  + Lice:
    - Pediculosis capitis = head lice
      * Itch, scalp excoriations
      * Confirmed by nits (which are eggs) stuck to hair shaft
      * Rx ~ malathion
    - Pediculosis corporis = body lice
    - Pubic lice = crabs
  + Bed bugs:
    - 3 bites from big to small (“breakfast lunch and dinner”)
* **SKIN FUCKING TUMORS ☺**
  + **Benign tumors:**
    - **Seborrheic keratosis** (*not* seborrheic dermatitis)
      * MC benign tumor in man
      * **Dark-colored papules** that has a **“STUCK-ON” APPEARANCE**
      * Typically occurs on **sun-exposed skin**
      * Can normally be seen, especially in **African Americans** (think Morgan Freeman’s face!)
      * **Appearance of 100s of new lesions suddenly** (especially on the **back**) 🡪 **LESAR-TRELAT SIGN**
        + Possible sign of **underlying malignancy** (GI malignancy ~ stomach cancer)
      * **WILL NEVER BE MALIGNANT** (not precancerous, but can be sign of underlying cancer)
      * Rx ~ no need or **just like warts**
    - **Melanocytic nevi (moles)**
      * **Benign overgrowth of melanocytes** (common in white-skinned people)
      * **Start as flat brown macules** (proliferation of melanocytes at the **dermal-epidermal junction** ~ **junctional nevus**)
      * They continue to proliferate and **grow down into the dermis (compound nevis)**, which causes the **above skin to elevate**
      * Borders are **regular**, **symmetrically pigmented**
    - **Cherry angioma** 
      * Benign angiokeratomas that appear as tiny red papules
      * Occurs in older age (similar to strawberry nevus in children)
    - Keratoacanthoma
    - Pilar cyst and epidermoid cyst
  + **Potentially pre-malignant skin condition**
    - **Actinic keratosis**
      * **White-skinned** people + **sun-exposure** = this
      * Erythematous **silver-scaly patches** (conical surface and a red base)
      * Surrounding skin may show flat brown macules (solar lentigos)
      * **Risk to develop SCC** in many years
    - **SCC in situ (Bowen’s disease)**
      * **Intraepidermal squamous cell carcinoma in-situ**
      * Commonly on sun exposed skin
      * Presents looking like psoriasis but has an irregular edge (increase in size with time)
      * A form that occurs in the genital area (glans penis, vulva, etc.) is called erythroplasia of Queyrat 🡪 increased risk of SCC
    - **Atypical mole syndrome** (dysplastic nevus syndrome)
  + **CANCERS:**
    - **Squamous cell carcinoma (SCC)**
      * **2nd MC cancer** in the world (**BCC = number 1**)
      * ***A non-healing ulcer in sun-exposed skin is SCC until proven otherwise***
      * Risk factors:
        + **Sun exposure**
        + Being Caucasian/**White** (fair skin, blue eyes)
        + **Vitiligo, albinism**
        + **Xeroderma pigmentosa**
      * Pre-cancerous conditions:
        + **Actinic keratosis, Bowen disease**
        + **HPV infection** and related genital conditions (warts) 🡪 SCC!
        + **Keratoacanthoma** 🡪 volcano-looking nodule (looks like a humongous molluscum)
      * Presentations:
        + May **ulcerate** or **keratinize** (secondary presentation of the primary pre-malignant conditions mentioned above)
        + May have a verrucous surface and look like a wart (verrucous)
        + **Rarely metastasizes**, but you can do investigations if necessary
      * Investigation & Management:
        + Biopsy confirms whether or not it is SCC, but you should **perform an EXCISION of the lesion** and then send to histopathology
        + So surgical excision = diagnostic and therapeutic
    - **Basal Cell Carcinoma (BCC)**
      * **Most common cancer in man**
      * Same risk factors as SCC
      * Types:
        + **Nodular (MC type)**
        + Superficial BCC
        + Ulcerative BCC
        + Pigmented BCC
        + Sclerotic BCC (worst kind)
      * Clinical features:
        + Depends on which type above
        + But in general, they have a **PEARLY WHITE APPEARANCE** with **ROLLED BORDERS** and **TELANGIECTASIAS**
      * Rx ~ surgical excision
    - **Malignant Melanoma:**
      * Cancer of the melanocytes
      * RF:
        + Same as all + **many moles** + **atypical nevi/familial dysplatic nevus syndrome** + immunosuppression
      * Types:
        + **Superficial spreading (70%)**
        + **Nodular (15%)**
        + **Lentigo maligna** (10 – 15%)
        + **Acral** (seen in **blacks** – nails – **melanonychia**)
        + **Amelanotic melanoma** (worst type, you think it’s BCC and it turns to be a melanoma)
      * Clinical features:
        + Most serious skin cancer (**metastasizes early**)
        + Always suspect it in an **UGLY DUCKLING MOLE** which follows the **ABCDE rule**

Don’t expect many melanomas at one time

Having MANY MOLES increases the risk of melanoma that can occur in the individual as a whole, NOT necessarily on one of the moles themselves (i.e. whole skin has equal risk to develop)

* + - * + **ABCDE rules** (Fulfill it = more likely melanoma)

A = Asymmetry

B = borders that are irregular

C = Color variation

D = diameter (> 6 mm)

E = Elevation/Evolution

* + - * + Nodular melanoma and amelanotic melanoma have bad prognoses

The fact that the melanoma is nodular means that it has infiltrated deeper, raising the overlying skin

* + - * + **Acral melanoma** are seen under the nails

Rules **A** (asymmetry/age), **B** (breadth and black), **C** (color variation), **D** (digit most commonly involved ~ thumb/big toe), **E** (extension – to the surrounding skin) and **F** (Family history)

Can be discovered late if patient doesn’t watch over nails

* + - * + MELANOMA LOVES TO METASTASIZE TO THE BRAIN (relatively rare), but also LN, liver (LDH can be useful)
      * **Investigations**
        + **Biopsy** (staging, depth of invasion, malignancy)
        + **Dermatoscope** can help distinguish benign and malignant moles
        + **R/O metastasis when biopsy shows malignancy**
      * Management:
        + Prevention 🡪 sun avoidance
        + Stage 1 – 3 (papillary dermis) 🡪 **wide local excision**
        + LN biopsy and dissection are considered