Dermatology PP& Notes Summary

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References

DWorld

Normal Skin & Skin Lesions

1. Cells of the Epidermis

- **Keratinocytes:** The primary cells of the epidermis. Mitosis (cellular division) is strictly limited to the basal layer (stratum basale). The outermost layer, the stratum corneum, consists of dead cells devoid of nuclei.
- **Melanocytes:** Located in the basal layer, these cells synthesize melanin. A single melanocyte supplies melanin to approximately 36 surrounding keratinocytes, forming an epidermal melanin unit. Under a microscope, they appear larger and clearer than neighboring cells.
- **Langerhans Cells:** These are dendritic, antigen-presenting immune cells originating from the bone marrow, found primarily in the stratum spinosum.
- **Merkel Cells:** Found in the basal layer, these act as mechanoreceptors for light touch and sustained pressure. (Note: They are *not* dendritic cells).

2. Skin Pigmentation (The Rule of Melanosomes)

- Differences in skin color across races are **not** caused by the number of melanocytes, which is generally similar across populations.
- Instead, pigmentation differences are determined by the size of melanosomes, the number/packaging of melanosomes, the degree of melanosome dispersion within keratinocytes, and the overall rate of melanin synthesis.

3. Sensory Receptors

- **Meissner Corpuscles:** Superficial receptors that mediate fine/light touch and tactile discrimination.
- **Pacinian (Vater-Pacini) Corpuscles:** Deep receptors located in the dermis/subcutis that detect deep pressure and vibration.
- **Free Nerve Endings:** Mediate pain and temperature.

4. Skin Glands

- **Sebaceous Glands:** Use holocrine secretion (cells disintegrate to release sebum) and are driven by androgens. They are absent on glabrous skin (palms/soles) and true mucosal surfaces (like buccal mucosa). *Meibomian glands* (eyelids) and *Montgomery glands* (areola) are specialized, modified sebaceous glands.

- **Eccrine Glands:** Produce watery sweat for thermoregulation and are controlled primarily by sympathetic cholinergic nerves.
- **Apocrine Glands:** Characterized by "decapitation" secretion.

5. Glabrous Skin (Palms & Soles)

- Characterized by a thick epidermis (prominent stratum corneum/granulosum), dermatoglyphics (fingerprints), and a high density of encapsulated sensory organs. It entirely lacks hair follicles and sebaceous glands.

6. Key Pathology: Disorders of Keratinization

- **Ichthyosis Vulgaris (Simplex):** The most common inherited ichthyosis. Usually presents in early childhood (not at birth) with fine scaling on *extensor* surfaces, characteristically *sparing the flexures*. It is highly associated with atopy and keratosis pilaris.
- **X-Linked Ichthyosis:** Caused by steroid sulfatase deficiency. Unlike vulgaris, it can have congenital systemic associations like cryptorchidism or corneal opacities.
- **Bullous Ichthyosiform Erythroderma:** Inherited in an *Autosomal Dominant* pattern.
- **Non-Bullous Congenital Ichthyosiform Erythroderma:** Inherited in an *Autosomal Recessive* pattern.

7. Other Clinical Presentations & Tools

- **Acanthosis Nigricans:** Presents with clinically thickened, velvety, hyperpigmented plaques in skin folds. It is heavily associated with insulin resistance.
- **Herald Patch:** The initial lesion of Pityriasis Rosea—a single, well-demarcated scaly plaque (often with a collarette of scale).
- **Macule:** A flat (non-palpable) skin lesion representing a color change that is <1 cm in size.
- **Dermatoscope:** A non-invasive hand-held tool for viewing subsurface structures of pigmented lesions and hair disorders. It is *not* used to see fungal hyphae/spores (which require a KOH prep under a microscope).

Eczema (Dermatitis)

1. Atopic Dermatitis (AD)

- **The Core Issue:** A chronic, intensely itchy inflammatory condition caused by a skin barrier dysfunction (like a filaggrin defect) and immune dysregulation, primarily driven by a Th2 helper cell predominance.
- **Labs:** Routine labs aren't usually necessary, but if checked, you will characteristically see elevated **IgE** levels.
- **The "Age-Shifting" Distribution:** This is highly tested!
    - *Infants/Young Children:* Usually affects the cheeks/face and **extensor** surfaces. Note: It is *not* typically present immediately at birth.
    - *Older Children/Adults:* Shifts primarily to the **flexures** (e.g., popliteal fossa, antecubital fossa).
- **Clinical Picture:** Characterized by severe pruritus (itching). Acute flares in kids can present as red, weeping, or oozing vesicular rashes.
- **Associations:** Commonly linked to a positive family history of the "atopic triad" (asthma, allergic rhinitis, and atopic dermatitis).

2. Seborrheic Dermatitis (SD)

- **The Core Issue:** A chronic, relapsing, but generally milder eczema linked to the lipid-dependent yeast ***Malassezia***.
- **Distribution:** Strictly favors sebum-rich areas. Look for it on the scalp (dandruff/cradle cap), eyebrows, nasolabial folds, and post-auricular (behind the ear) areas.
- **Clinical Picture:** Presents with erythema and characteristic **greasy scales**. Unlike AD, the itching is usually mild.
- **Age Pattern:** Biphasic—it can occur very early in infants (<3 months) as "cradle cap" and also in adults.

3. Allergic Contact Dermatitis (ACD)

- **The Core Issue:** A **Type IV (delayed, cell-mediated) hypersensitivity** reaction driven by sensitized T lymphocytes.

- **Timing:** Rashes typically develop **48 to 96 hours** after exposure to the allergen.
- **Diagnosis: Patch testing** is the pathognomonic and gold-standard test used to identify the specific allergen.
- **Classic Triggers & Presentations:**
    - ***Nickel:*** The most common classic allergen overall (think jewelry, watches, belt buckles).
    - ***Primula (plant):*** A classic cause of severe, even *bullous* (blistering) contact dermatitis.
    - ***Fragrances/Perfumes (in cosmetics):*** Classically causes "Pigmented Contact Dermatitis" (Riehl melanosis), where hyperpigmentation is prominent.
    - ***Nail Varnish:*** Often causes dermatitis not on the nails, but on "touched sites" like the **neck** and eyelids due to hand-to-skin transfer.
    - ***Clothing:*** Typically affects areas of friction, occlusion, and sweat, such as the **body flexures**.

4. Histology & Morphology (The "Look" of Eczema)

- **Acute Eczema (Histology):** The hallmark microscopic finding is **Spongiosis** (intercellular epidermal edema). This edema is what leads to clinically visible vesicles, blisters, and weeping.
- **Chronic Eczema (Clinical):** Dominated by **Lichenification**, which is the thickening and hardening of the skin with exaggerated normal skin markings, caused by chronic rubbing and scratching. (Blisters are *not* typical of chronic eczema). Parakeratosis can also be seen histologically.

5. High-Yield Clinical Pearls

- **Unilateral Hand Eczema:** If a patient has eczema on only *one* hand, your immediate next step should be a **KOH scraping** to rule out a fungal infection (tinea manuum). Giving potent steroids to a misdiagnosed fungal infection will make it worse.
- **Pityriasis Alba:** A mild eczema variant in children that causes hypopigmented (lighter) patches. Importantly, these patches will appear **non-fluorescent** under a Wood's light, which helps differentiate it from the bright white depigmentation of vitiligo.

Acne & Rosacea

1. Acne Vulgaris: Pathogenesis & Lesion Progression

- **The 4 Steps of Pathogenesis:** 1. Follicular hyperkeratinization and plugging (this is the very first step). 2. Increased sebum production (sebum stagnation). 3. Proliferation of the bacteria *Propionibacterium acnes* (Note: *not* Staph. albus). 4. Inflammation. (Note: Epidermal edema is not a feature of acne).
- **Lesion Timeline:**
    - **Comedone:** The very first, non-inflammatory lesion of acne. An open comedone is a blackhead, and a closed comedone is a whitehead.
    - **Papule:** The earliest inflammatory lesion. This is the direct precursor to larger inflammatory lesions.
    - **Pustules, Nodules, & Cysts:** Later, more severe inflammatory stages.
    - *Exam Trap:* **Vesicles** do *not* occur in acne.

2. Acne Treatment & Triggers

- **Mild Acne:** First-line treatment is topical benzoyl peroxide (which has antibacterial and comedolytic effects) and/or topical retinoids.
- **Moderate/Severe Acne:** If a patient has moderate acne that is completely unresponsive to oral antibiotics (like tetracycline) for 6 months, the next step is **Isotretinoin**.
- **Triggers/Modifiers:** * *Worsens acne:* Systemic steroids (steroid acne) and Vitamin B12.
    - *Improves acne:* Estrogen (reduces androgen effects on sebaceous glands).

3. Isotretinoin / Systemic Retinoids (Highly Tested!)

Isotretinoin is a heavy-hitter drug for severe, nodulocystic, or resistant acne. You must know its profile perfectly:

- **Dosing & Labs:** The total cumulative dose depends on the patient's body weight. Baseline and follow-up blood tests are strictly mandatory.
- **Pregnancy:** It is highly teratogenic (Pregnancy Category X). Strict contraception is required during therapy and for **1 month** after stopping (not 1 year).
- **Classic Side Effects:** * Mucocutaneous dryness (dry lips happen in nearly 100% of patients).
    - Hyperlipidemia (elevated triglycerides).
    - Elevated liver enzymes (LFTs).
    - Diffuse hair thinning / hair loss.
    - Nail changes like paronychia and nail thinning.
    - Rare: Increased intracranial pressure (pseudotumor cerebri).
- **What it does NOT cause:** Isotretinoin does *not* cause infertility , thrombocytopenia , renal failure , or scarring alopecia.

4. Rosacea vs. Acne

- **Demographics:** Rosacea primarily affects middle-aged adults, whereas acne is more common in teenagers.
- **Clinical Picture:** Rosacea presents with facial flushing, persistent erythema, and **telangiectasia** (visible tiny blood vessels, which are characteristic of rosacea, not acne). Severe cases can progress to rhinophyma (a bulbous, enlarged nose).
- **The Golden Rule:** Both conditions can have papules, pustules, and redness. However, **Rosacea NEVER has comedones.** If you see comedones, it is acne.
- **Treatment Note:** Topical metronidazole is commonly used to treat rosacea; it is *not* a systemic treatment for acne.

Cutaneous Manifestations

1. The "Erythemas" (Highly Tested!)

- **Erythema Nodosum (EN):** * *What it is:* A panniculitis (inflammation of subcutaneous fat, *not* the dermis).
    - *Looks like:* Tender, red, subcutaneous nodules classically on the shins. **It does NOT ulcerate.**
    - *Triggers:* Pregnancy, Streptococcal throat infections, sarcoidosis, IBD, and certain drugs. (Note: *Herpes Simplex is NOT a cause*).
- **Erythema Multiforme (EM):**
    - *Looks like:* Classic "target" lesions. EM minor has little to no mucosal involvement.
    - *Triggers:* **Herpes Simplex Virus (HSV)** is the absolute most common cause. Mycoplasma is another documented trigger.
- **Erythema Gyratum Repens:**
    - *Looks like:* Rapidly migrating, concentric red bands that look like **"wood-grain."**
    - *Association:* This is a classic paraneoplastic rash strongly associated with **internal malignancy**. (It is *not* triggered by Strep).
- **Erythema Nodosum Leprosum (ENL):**
    - A severe inflammatory reaction in leprosy. Unlike standard EN, this *can* become necrotic and **ulcerate**.

2. Dermatitis Herpetiformis (DH)

- **The Core Issue:** A chronic, intensely itchy autoimmune blistering disease.
- **Clinical Picture:** Grouped papules and vesicles strictly on **extensor surfaces** (elbows, knees, buttocks).
- **The Big Association:** Strongly linked to **Celiac disease** (gluten-sensitive enteropathy).
- **Histology:** Starts with **neutrophilic microabscesses in the dermal papillae**, leading to a subepidermal blister. (It is *not* a prickle cell layer defect).
- **Treatment: Dapsone (DDS)** for rapid itch relief, plus a strict lifelong **gluten-free diet**.

3. Vitiligo & Pigmentary Disorders

- **Vitiligo:**
    - *Mechanism:* Caused by the **destruction and absence of melanocytes** (it is *not* an abnormal melanin synthesis or tyrosinase defect).
    - *Looks like:* Well-demarcated **depigmented** (milk-white) patches, not just "hypopigmented."
    - *Demographics:* Affects males and females equally (1:1), usually starting in the 20s–30s.
    - *Associations:* Autoimmune diseases! Always screen the **thyroid** (hypo- or hyperthyroidism/Hashimoto's). Also linked to **Pernicious anemia** and **Addison's disease**.
- **Piebaldism:** Congenital depigmented patches with a white forelock. Inherited as **Autosomal Dominant**.
- **Chloasma (Melasma):** Facial hyperpigmentation. Treatment of choice is strict sun protection + **topical hydroquinone (Eldoquin)**.
- **Post-Inflammatory Hypopigmentation:** Can occur after the resolution of inflammatory diseases like Psoriasis or Lichen Planus.

4. Cutaneous Leishmaniasis

- **The Bug:** A protozoal infection transmitted by the bite of a sandfly. The organism lives strictly inside **macrophages (R.E. cells)** in the skin.
- **Clinical Picture:** Common in the Jordan Valley. Starts as a **painless** papule at an insect bite site that enlarges into an **ulcer**.
- **Treatment: Antimonials** (like sodium stibogluconate). Antibiotics do not work.
- **Leishmaniasis Recidivans:** A chronic, relapsing form classically caused by ***L. tropica***.

5. Systemic Clues & Quick Hits

- **Generalized Pruritus with Normal Skin:** Think systemic! In biliary obstruction, the itch is directly related to retained **bile salts**.
- **Hodgkin's Lymphoma:** Commonest cutaneous manifestation is intense pruritus leading to **secondary excoriations** (scratch marks).
- **Erythroderma:** Defined as generalized erythema and scaling over **>90%** of the body surface area. Can cause severe complications like hyperthermia and dehydration. Causes include Psoriasis, drugs, cutaneous lymphoma, and congenital ichthyosis.
- **Acanthosis Nigricans:** Velvety, hyperpigmented thickened plaques in skin folds/creases. Strongly associated with **insulin resistance**.
- **Henoch-Schönlein Purpura (HSP):** An **IgA-mediated** small-vessel vasculitis (Not IgG).
- **Necrobiosis Lipoidica:** Yellow-brown atrophic plaques with telangiectasia on the shins. Strongly associated with **Diabetes Mellitus (DM)**.
- **Placental Transfer:** Only **IgG** can cross a normal placenta to give passive immunity to a fetus.

Connective Tissue Diseases

1. Lichen Planus (LP)

- **The "6 Ps":** Pruritic (intensely itchy), Purple, Polygonal, Planar (flat-topped), Papules, and Plaques . (Note: The primary lesion is a papule/plaque, not a macule).
- **Classic Signs:** Wickham's striae (lacy white lines) and Koebner phenomenon (spreading along scratch lines) .
- **Histology:** Classically shows **Hypergranulosis** (thickened granular layer), *not* hypogranulosis .
- **Nail Involvement:** Inflammation of the nail matrix leads to thinning, longitudinal ridging, and in severe cases, **pterygium** (scarring and overgrowth of the nail fold) . It does *not* typically cause paronychia, onycholysis, or nail thickening .
- **Mucosal Involvement:** Very common, classically appearing on the **buccal mucosa** rather than the gingivae .
- **Disease Course:** Often self-limiting (clearing in 1-2 years), but mucosal and hypertrophic forms can be chronic .
- **Association:** Highly tested link to **Hepatitis C** infection .

2. Lupus Erythematosus (LE)

- **Systemic Lupus Erythematosus (SLE):** The most classic common cutaneous eruption is the malar ("butterfly") rash, which is highly linked to photosensitivity .
- **Discoid Lupus Erythematosus (DLE):** A chronic, **scarring** cutaneous form. It causes destruction of the basal cell layer . On the scalp, it leads to **patchy scarring alopecia** with follicular plugging . It progresses to SLE in <5% of cases .
- **Subacute Cutaneous Lupus (SCLE):** Presents with photosensitive lesions but typically heals **without** scarring .
- **Neonatal Lupus:** Caused by the transplacental transfer of maternal autoantibodies (anti-Ro/La). The infant's rash will **resolve** as the maternal antibodies drop; it does *not* typically transform into SLE .

3. Dermatomyositis

- **The "Look":** A rare disease affecting skin, muscles, and vessels. Look for a **Heliotrope rash** (purple hue on eyelids/face), **Gottron's sign/papules** (over dorsal finger joints), and proximal muscle weakness .
- **Nail Clues:** Features include **ragged cuticles** and dilated nailfold capillaries .
- **Demographics:** It affects both children and adults, but is *more common in females* .
- **The Two Big Associations:**
    - **Adults:** Strongly associated with an underlying **malignancy** .
    - **Juvenile (Children):** Frequently associated with **calcinosis cutis** (calcium deposits in soft tissues) .

4. Morphea (Localized Scleroderma)

- **Clinical Picture:** Presents with localized sclerosis/fibrosis, resulting in firm, well-defined plaques . Because of the sclerosis, hair in the affected patches is usually reduced (it is *not* a "hairy" lesion) . It often improves over time .
- **Linear Morphea:** A specific variant that can extend beyond the skin into subcutaneous tissue, muscle, and even bone. It is classically associated with **muscular atrophy** .

5. Lichen Sclerosus

- **Classic Presentation:** This is a major cause of an intensely itchy, painful, and white (ivory, atrophic) vulva, most commonly seen in **postmenopausal women** .

Psoriasis

1. The Classic Clinical Signs

- **Auspitz Sign:** Pinpoint bleeding that occurs when a psoriatic scale is peeled off . This happens because the epidermal layer directly above the dermal papillae (the suprapapillary plate) is extremely thin, and the capillaries right below it are dilated .
- **Koebner Phenomenon:** The development of new psoriatic plaques at the exact sites of skin trauma, such as scratch lines or scars .
- **Demographics & Genetics:** It can affect anyone at any age, including children. It is a complex, polygenic/multifactorial disease (it is *not* a simple autosomal recessive trait) .

2. Histopathology (Highly Tested!)

If you see these terms together on a pathology report, think psoriasis:

- **Acanthosis:** Epidermal hyperplasia (thickening) .
- **Hyperkeratosis & Parakeratosis:** A thickened stratum corneum with retained nuclei in the dead skin cells .
- **Munro's Microabscesses:** Characteristic collections of neutrophils trapped in the stratum corneum .
- **Suprapapillary Plate Thinning:** The anatomical reason for the Auspitz sign.
- *Exam Trap:* Psoriasis does **not** show epidermal atrophy, spongiosis (which is eczema), hypergranulosis (which is lichen planus), or acantholysis (which is pemphigus) .

3. Nail Psoriasis

- **Pitting:** The absolute most common nail manifestation . It is caused by disease in the **nail matrix**. Abnormal keratinization creates parakeratotic foci in the nail plate; when these grow out and reach the surface, they break off, leaving tiny pits .
- **Other features:** Onycholysis (separation of the nail from the bed), subungual hyperkeratosis (thick scale under the nail), and "oil drop" or "salmon patch" discoloration .
- *Exam Trap:* **Clubbing** is *not* a feature of psoriasis; it points to systemic cardiopulmonary issues .

4. Subtypes & Exacerbating Factors

- **Guttate Psoriasis:** Presents as sudden-onset, small "drop-like" plaques widespread on the trunk and limbs . It is classically triggered by a **Streptococcal sore throat** .
- **Erythrodermic Psoriasis:** A severe form involving most of the body surface area. Life-threatening complications include temperature dysregulation, dehydration (due to fluid/protein loss), and an increased risk of sepsis .
- **Pustular Psoriasis Triggers:** Can be linked to **hypocalcemia** (not hypercalcemia) .
- **Drug Triggers:** Psoriasis can be provoked or worsened by **antimalarials** . (Macrolide antibiotics are not a classic trigger).

5. Treatment Rules & "What Not To Do"

- **Systemic Therapies:** Standard options include Methotrexate, Cyclosporin, Acitretin (a systemic retinoid), and Biologics (e.g., TNF-alpha blockers) . (Note: Isotretinoin is primarily for acne; Acitretin is the retinoid used for psoriasis).
- **Avoid Antimalarials:** They are strictly avoided as a treatment because they aggravate the disease .
- **Avoid Systemic Steroids:** Oral corticosteroids are generally avoided in routine management. Withdrawing them can cause a severe rebound flare, precipitating unstable forms like pustular or erythrodermic psoriasis .

Urticaria

1. Pathophysiology & The Primary Lesion

- **The Main Cell:** The **Mast cell** is the core initiating cell in urticaria .
- **The Mechanism:** Mast cell degranulation releases **histamine**, which increases capillary permeability, leading to transient superficial dermal edema .
- **The Wheal:** This is the primary lesion of urticaria . It is a well-demarcated, edematous, erythematous swelling that **blanches with pressure** and is intensely itchy .
- **The 24-Hour Rule:** A classic, simple urticarial wheal lasts for minutes to hours (usually <24 hours) and resolves completely **without leaving any scars or hypopigmentation** .
- *Red Flag:* If a wheal lasts >24 hours, is painful rather than just itchy, or heals with bruising, you must suspect **urticarial vasculitis** and perform a biopsy .

2. Treatment Protocol (Know the sequence!)

- **First-Line Treatment:** The absolute treatment of choice for acute and chronic urticaria is **oral Antihistamines** (H1 blockers), taken regularly .
    - *Daytime Use:* Prefer 2nd-generation, non-sedating antihistamines (like the **piperidines** group, e.g., loratadine/fexofenadine) to avoid impairment .
- **What NOT to use:** * **Topical antihistamine ointments** are *not* standard or effective because the pathology is systemic mediator release causing dermal edema .
    - **Systemic Steroids** are *not* first-line . They are strictly reserved for short courses in very severe eruptions or when urticarial vasculitis is suspected .
- **Anaphylaxis:** Adrenaline is reserved for severe, life-threatening reactions involving respiratory distress or angioedema .

3. Specific Subtypes of Urticaria

- **Chronic Urticaria:** Defined as symptoms lasting more than 6 weeks. In up to 90% of these cases, the cause remains **unknown (idiopathic)** .
- **Cholinergic Urticaria:** * *Trigger:* A rise in core body temperature and sweating (e.g., exercise, hot showers) .
    - *Looks like:* Characteristic **pinhead-sized wheals** surrounded by a large red flare .
    - *Diagnosis:* The most reliable diagnostic test is provocation with **exercise and heat** .
- **Cold Urticaria:** * *Trigger:* Exposure to cold. Usually acquired, but can be familial or transferred by serum .
    - *Danger:* Extensive cold exposure (like swimming in cold water) can lead to massive systemic mediator release, resulting in hypotension, syncope, and **unconsciousness** .

Pruritus & Scabies

1. Systemic Pruritus

- **The Golden Rule:** If a patient has generalized itching but their skin looks completely normal, you must suspect a systemic cause and run labs .
- **Biliary Obstruction:** In cholestasis or biliary obstruction, the intense generalized itching is most directly caused by retained **bile salts** .

2. Scabies: The Basics & Transmission

- **The Bug:** Caused by the mite *Sarcoptes scabiei hominis*.
- **Transmission:** It requires **prolonged, close skin-to-skin contact** (a simple, brief handshake is not enough to spread it).
- **Incubation:** In a first-time infestation, the itching does *not* start immediately. It takes about **2 to 4 weeks** for the immune system to become sensitized to the mite's proteins, eggs, and feces before the itch begins .

3. Clinical Presentation & Diagnosis

- **The Itch:** Intensely pruritic, and characteristically **worse at night**.
- **Family History:** Because it spreads in households, a history of family members or close contacts itching is a massive diagnostic clue .
- **The Primary Lesion:** The pathognomonic sign is the **burrow** (a linear ridge with a tiny dark speck at the end) .
- **Microscopy:** To get the highest diagnostic yield under a microscope, you must take skin scrapings specifically from the **burrows**, not from the secondary papules or scratch marks (excoriations) .

4. The Distribution Trap (Adults vs. Infants)

This is the most frequently tested concept in this section!

- **Adults:** Classically affects the finger webs, flexures, and genitalia. In adults, scabies **spares the face and the back** .
- **Infants & Young Children:** The distribution is much wider. In babies, it **does NOT spare the face and back**. It also frequently involves the **palms and soles** . Furthermore, infant lesions can blister or appear as acral pustules .

5. Treatment Protocol

- **First-Line Drug: Topical Permethrin 5% cream** . (Alternative options include benzyl benzoate . Note: Benzoyl peroxide is for acne, not scabies).
- **Application:** Applied overnight and washed off. It must be **repeated after 7 days** to break the life cycle .
    - *Adults:* Apply from the neck down.
    - *Infants:* Apply to all skin, **including the head and neck** .
- **The Golden Rules of Management:**
    1. You **MUST treat all members of the household** and close contacts at the exact same time, even if they have no symptoms, to prevent re-infestation.
    2. Warn the patient that **itching can persist for weeks** after successful treatment. The immune system is still reacting to dead mites in the skin. Do not keep re-treating with permethrin, as it will cause irritant dermatitis .

Bacterial Infections

1. Skin Flora vs. Carriers (Highly Tested!)

- **Normal Flora:** Healthy skin is normally colonized by coagulase-negative Staphylococci (like *Staph. albus*) and diphtheroids (like *Corynebacterium*).
- **The Carrier State: *Staphylococcus aureus*** is **not** considered normal skin resident flora . Instead, it frequently acts as a pathogen or a colonizer. The main local source for *Staph. aureus* contamination and recurrent skin infections is carriage in the **nose (anterior nares)** .

2. The "Depth" of Infection (Superficial to Deep)

You must know these three infections and how to tell them apart based on how deep they go:

- **Impetigo (Most Superficial):** * *Depth:* The absolute most superficial bacterial infection.
    - *Looks like:* Vesicles/pustules that rapidly break down into classic **golden crusts**.
    - *Demographics:* Highly contagious and very common in infants and children.
    - *Bug: Staph. aureus* and/or *Strep. pyogenes*.
- **Ecthyma (Deeper Impetigo):**
    - *Depth:* A deeper form of impetigo where the bacteria (usually *Streptococcus*) invade down into the dermis .
    - *Looks like:* It causes adherent crusts with underlying **ulceration**, and unlike impetigo, it **heals with scarring** .
- **Erysipelas (Dermis & Lymphatics):**
    - *Depth:* Deeper dermal and lymphatic involvement .
    - *Looks like:* A **well-demarcated**, shiny, raised, and tender erythematous plaque. (If the question describes a *poorly defined* margin, it is cellulitis, not erysipelas).
    - *Treatment:* The classic cause is *Streptococcus* (Group A), and the drug of choice is **Penicillin**.

3. Erythrasma (The Wood's Lamp Clue)

- **The Bug:** A superficial infection caused by *Corynebacterium minutissimum* .
- **Clinical Picture:** Superficial scaling and mild redness in the flexures (axilla, groin).
- **The Key Test:** It fluoresces a classic **coral-red (or pink)** under a Wood's light. This perfectly distinguishes it from fungal groin infections.

4. Leprosy (Hansen's Disease)

- **Lepromatous Leprosy:** This is the severe, multibacillary form. A highly tested complication of this specific form is significant **eye involvement** (lids, cornea, uvea) .
- **Treatment:** Requires prolonged multidrug therapy (MDT). Classic exam teaching dictates treatment should continue for **2 years** for this multibacillary form .

5. Secondary Syphilis

- The characteristic rash of secondary syphilis is a widespread, generalized **maculopapular** eruption .
- Key features: It typically includes **scale**, frequently involves the **palms and soles**, and classically does **not itch** . It is *not* a vesicular rash .

Viral Infections

1. Warts (Human Papillomavirus- HPV)

- **The Basics:** Warts are caused by HPV, a **double-stranded DNA** virus . Most common warts (especially in children) will eventually resolve spontaneously as immunity develops; they do *not* typically turn into skin cancer . You should **never** use topical steroids for viral warts .
- **Types of Warts (Highly Tested!):**
    - **Common Warts:** The most common type overall. They are **rough, dry, and hyperkeratotic** papules .
    - **Plane (Flat) Warts:** These are **smooth, flat-topped**, flesh/pink/brownish papules, most commonly found on the **face** . Because they are smooth, do not confuse them with spiky warts. They can exhibit the Koebner phenomenon (spreading along scratch lines).
    - **Filiform Warts: Spiky, finger-like** projections that commonly affect the face, neck, and body flexures .
    - **Plantar Warts:** Found on the soles. The pressure of walking makes them look **smooth** and flat, but they are often painful and show black dots (thrombosed capillaries) .
- **Genital Warts (Condylomata Acuminata):** Treatment of choice includes **Podophyllotoxin** (patient-applied) or **Podophyllin** resin (provider-applied) .

2. The Herpes Viruses (HSV & VZV)

- **Herpes Simplex Virus (HSV):** * The most common form of recurrent HSV is **Herpes Labialis** (cold sores) .
    - *Golden Rule for Genital Herpes:* Patients are still contagious **even when they are asymptomatic** due to viral shedding.
- **Herpes Zoster (Shingles):**
    - *The Basics:* Reactivation of the Varicella-Zoster Virus (VZV). It presents as **unilateral** (not bilateral) vesicular rash in a dermatome, often preceded by pain .
    - *Complication:* Postherpetic neuralgia (PHN), which can last for months, especially in the elderly .
    - *Treatment Rules:* You MUST use **systemic antivirals** (like oral acyclovir) early. Do **not** treat shingles with topical acyclovir or routine systemic steroids .
    - *Who needs systemic treatment the most?* Patients >50 years old, the immunocompromised, or those with more than one dermatome involved.

3. Pityriasis Rosea

- **The Bug:** It is highly associated with **HHV-6 and HHV-7**.
- **The Sequence:** It always starts with a **Herald Patch** (a single oval plaque with collarette scale), followed days later by a widespread eruption of smaller scaly patches on the trunk/proximal limbs (a "Christmas tree" pattern) .
- **The Look & Feel:** It is primarily a **scaly** patch/plaque rash (not just macular) . The itching is usually mild or even completely absent . It is **self-limiting**, meaning it goes away on its own.

4. Other Viral Exanthems (Quick Hits)

- **Erythema Infectiosum (Fifth Disease):** Causes the classic "Slapped Cheek" appearance. The bug is **Parvovirus B19**.
- **Molluscum Contagiosum:** Presents as umbilicated papules. The bug is a **Poxvirus**.
- **Roseola Infantum:** Caused by HHV-6 .
- *Exam Trap:* **Scarlet fever** is a *bacterial* rash (Streptococcus), not a viral exanthem .

Fungal Infections

1. The "Systemic vs. Topical" Treatment Rules (Highly Tested!)

- **Tinea Capitis (Scalp/Hair):** You MUST use **systemic antifungals**. Topical creams do not penetrate the hair shaft deeply enough to cure the infection.
    - *Drug of Choice:* **Griseofulvin** .
    - *Griseofulvin Pearls:* It has better absorption with a fatty meal, commonly causes headaches, is contraindicated in pregnancy, and its effect is reduced by phenobarbital .
- **Tinea Unguium (Nails / Onychomycosis):** Infection of the **nail plate**. Also requires **systemic antifungals** for a prolonged period, especially for toenails, because topical agents penetrate the thick nail poorly .
- **Tinea Pedis (Athlete's Foot):** The most common adult fungal infection . Unlike the scalp and nails, this is usually treated successfully with **topical antifungals**.

2. The Bugs & Their Unique Clues

- **Trichophyton verrucosum:** A zoophilic fungus classically acquired from **cattle** . It causes a highly inflammatory reaction on the scalp called a **Kerion** (a boggy, inflamed, pustular plaque) . Exam Trap: Treat a kerion with systemic antifungals; do NOT surgically incise and drain it .
- **Microsporum audouinii:** The classic culprit behind **epidemic/outbreak** tinea capitis in schools due to its highly efficient spread .
- **Trichophyton mentagrophytes:** A classic example of an **ectothrix** organism (spores sit on the *outside* of the hair shaft) .
- **Candida:** A yeast that commonly infects mucous membranes (thrush), skin flexures, and nails/nail folds .
    - *Classic Sign:* Bright red flexural rash with characteristic **satellite pustules/papules** at the outer edges .
    - *Associations:* It is heavily involved in **chronic paronychia** (nail fold inflammation) in people who do chronic "wet work" .
    - *Exception:* Candida does *not* typically infect the hair.

3. Pityriasis (Tinea) Versicolor

- **The Bug:** Caused by the yeast ***Malassezia***.
- **The Look:** Fine, scaly, hypo- or hyperpigmented patches typically on the upper back, chest, and neck .
- **The Sun Clue:** The lesions do not tan, making them much more obvious after sun exposure.
- **Disease Course:** It has a very high rate of **recurrence/relapse**, especially in hot, humid weather.

4. Diagnostics: Wood's Lamp & Microscopy

- **First Step:** The absolute best initial test for any suspected fungal skin infection is a **KOH preparation and microscopy** .
- **Wood's Lamp Colors (Memorize these!):**
    - *Microsporum* (Some Tinea Capitis) = **Green / Blue-Green**.
    - *Pityriasis Versicolor* = **Yellow / Golden** (Not apple-green, not cherry-red) .
    - *Erythrasma* (Bacterial, *C. minutissimum*) = **Coral-red / Pink** .
    - *Trichophyton rubrum* = **Does NOT fluoresce** .

5. Morphology Identification

- **Tinea Corporis (Ringworm):** Classically presents as an **annular** (ring-shaped) lesion with a slightly raised, scaly active border and **central clearing** .

Bullous Diseases

1. Pemphigus Vulgaris (PV) – The Flaccid, Superficial Blister

- **The Target:** Autoantibodies attack **desmosomes** (specifically desmoglein).
- **The Depth:** Because desmosomes hold epidermal cells together, their destruction causes *acantholysis* (loss of cell adhesion), leading to an **intraepidermal (suprabasal)** blister.
- **Clinical Picture:** The blisters are very fragile and **flaccid**, meaning they rupture easily and leave behind large, painful erosions.
- **The Key Sign:** Nikolsky sign is typically **positive** (the skin sloughs off with lateral pressure).
- **Mucosal Involvement:** Very common and severe. Patients often present first with **painful oral/mouth ulcers**.
- **Demographics & Prognosis:** Usually affects middle-aged adults, carries a recognized racial/ethnic predilection (e.g., higher in certain Jewish populations), and can be fatal if left untreated.

2. Bullous Pemphigoid (BP) – *The Tense, Deep Blister*

- **The Target:** Autoantibodies attack **hemidesmosomes** (BP180/BP230) located at the basement membrane zone.
- **The Depth:** Because the entire epidermis separates from the dermis, it creates a deeper, **subepidermal** split.
- **Clinical Picture:** The blisters have a thick roof (the whole epidermis), making them **firm and tense**.
- **The Key Sign:** Nikolsky sign is usually **negative**. Mucous membranes are much less commonly involved compared to PV.
- **Diagnostics (Highly Tested!):** The most valuable test to diagnose BP (and differentiate it from mimics like erythema multiforme) is **Direct Immunofluorescence (DIF)**. It will classically show a **linear deposition of IgG and C3** along the basement membrane.

3. Epidermolysis Bullosa (EB)

This is a group of inherited blistering disorders. You must know the difference between the healing patterns:

- **EB Simplex:** A superficial form that typically heals **without scarring**.
- **Dystrophic EB:** A deeper structural fragility that classically heals with severe **scarring and deformity**.
- *Note:* Extensive mucous membrane involvement is a hallmark of the severe/dysplastic forms, not the simplex form.

4. General Blistering Rules

- **Eczema vs. Bullous Disease:** Eczema causes vesicles due to *spongiosis* (intercellular edema). It is *not* considered a primary cause of generalized massive blistering like the immunobullous diseases or severe drug reactions.
- **Bullous Impetigo:** Remember from the bacterial section that this causes *intraepidermal* blistering due to staph toxins.

Memory Trick:

- **PemphigUS** goes with **US** (up superficial = intraepidermal, flaccid).
- **PemphigOID** goes with **DEEP** (subepidermal, tense).

Sexually Transmitted Diseases

1. Syphilis: The Stages

- **Primary (1ry) Syphilis:** * *The Lesion:* Presents as a **Chancre** at the site of inoculation .
    - *Key Features:* It is usually single, **painless**, and highly infectious (rich in treponemes) . It is *not* an allergic reaction; it is a direct bacterial infection.
- **Secondary (2ry) Syphilis:** * *The Rash:* A widespread, generalized **maculopapular and scaly** rash that characteristically involves the **palms and soles** . It typically does **not itch** (or itches very minimally) and is *never* vesicular.
    - *Other Cutaneous Signs:* Look for **Condylomata lata** (broad, moist, highly infectious lesions) and highly infectious **mucous patches** . It also causes a patchy, non-scarring hair loss known as **"moth-eaten" alopecia**.
    - *Systemic Signs:* Generalized, **non-tender (painless)** lymphadenopathy.
- **Tertiary (3ry) Syphilis:** Late organ disease (not heavily focused on in this cutaneous section).

2. Syphilis: Diagnostics & Treatment

- **The "Follow-Up" Tests (Non-Treponemal):** Tests like VDRL, RPR, and the older **WR (Wassermann reaction)** have titers that correlate with disease activity. Because the titers fall after effective treatment, they are the best tests for **following up** and monitoring the response to therapy .
- **The "Confirmatory" Tests (Treponemal):** Tests like the **FTA-ABS** are the most highly sensitive and specific tests for early diagnosis, but they usually remain positive for life (making them poor for follow-up) .
- **Microscopy Clues:** If a dark-field examination of a chancre fails to show spirochetes, you can establish the diagnosis by aspirating a regionally enlarged lymph node or using serology . *Fun fact: T. pallidum* can only survive in refrigerated blood $(+4^\circ C)$ for a few days, roughly **72-92 hours** .
- **Treatment:** The absolute drug of choice for all standard stages of syphilis is **Benzathine Penicillin** . (Note: Crystalline aqueous penicillin is reserved for neuro/ocular syphilis).

3. Gonorrhea

- **The Bug:** Caused by *Neisseria gonorrhoeae*, a **Gram-negative diplococcus**.
- **The Target:** It has a strict predilection for infecting mucosal **columnar epithelium**.
- **Diagnostics in Females:** Because it targets the columnar epithelium of the cervix, the best site to take a routine swab in females is an **endocervical swab** . Importantly, up to **50% or more of females are entirely asymptomatic**.

4. Other Key STDs (The "Match the Bug" Rules)

- **Chancroid:** Caused by ***Haemophilus ducreyi***. *Exam Trap:* Unlike the painless chancre of syphilis, the ulcer of chancroid is characteristically **painful** with tender lymphadenopathy .
- **Lymphogranuloma Venereum (LGV):** Caused by ***Chlamydia trachomatis***.
- **Non-Gonococcal Urethritis (NGU):** Often caused by *Chlamydia*. The classic drug of choice is a **Tetracycline**.
- **Genital Warts (Condylomata Acuminata):** Caused by **HPV** (Human Papillomavirus). Do not confuse this with Condylomata *lata* (which is secondary syphilis) .

Drug Eruptions

1. The SJS / TEN Spectrum

These are severe, life-threatening mucocutaneous drug reactions. They are classified purely by the percentage of Body Surface Area (BSA) that has detached :

- **Stevens-Johnson Syndrome (SJS):** Involves **<10%** of the body surface area.
- **Overlap SJS/TEN:** Involves **10% - 30%** of the body surface area.
- **Toxic Epidermal Necrolysis (TEN):** Involves **>30%** of the body surface area.

2. Clinical Presentation & Triggers

- **The Triggers:** They are almost always caused by **drugs**, primarily anti-epileptics (like phenytoin), NSAIDs, allopurinol, and sulfonamide antibiotics . (They are *not* most commonly caused by infections).
- **The Look & Feel:** Typically begins with a prodrome of fever and malaise. The skin becomes **extremely painful** and erythematous, followed by blistering and massive sloughing/detachment .
- **The Defining Tissue:** SJS/TEN is fundamentally a **mucocutaneous** disease. It involves massive skin detachment *plus* severe mucosal erosions (oral, ocular, genital) . Severe oral erosions are nearly always present .

3. Management Rules (Highly Tested!)

- **Step 1:** The absolute, most important initial step is to **immediately stop the offending drug**. Do *not* start systemic steroids or antibiotics as your first step.
- **Step 2:** Provide urgent, intensive supportive care, usually in an **ICU or burn unit**, to manage fluid loss, temperature dysregulation, and infection risk .
- **Complications:** SJS/TEN is highly fatal. Early ophthalmology involvement is critical to prevent permanent, vision-threatening eye damage .

4. Acne Medicamentosa (Drug-Induced Acne)

- Certain medications can provoke or worsen acne flares. Classic triggers include **Systemic Steroids** ("steroid acne"), anti-epileptics (like Phenytoin), and **Vitamin B12** .
- *Exam Trap:* **Azelaic acid** is *not* a trigger; it is an anti-inflammatory medication used to *treat* acne and rosacea .

Skin Tumors

1. Basal Cell Carcinoma (BCC)

- **The Basics:** The absolute most common skin cancer. It has an excellent prognosis because it rarely metastasizes. If it *does* metastasize, it goes to the **regional lymph nodes** first .
- **The Look:** Classically presents as a **pearly, translucent, or glossy nodule**.
- **Key Features:** Look for surface **telangiectasia** (visible tiny blood vessels) and a central **"rolled-edge" ulcer** (also called a rodent ulcer) .
- **Location & Type:** The most common site is the sun-exposed face, and the most common subtype is **Noduloulcerative** .

2. Squamous Cell Carcinoma (SCC) & Precursors

- **The Precursor: Actinic Keratosis (AK)** is the classic **premalignant** lesion . It presents as rough, fine-scaled, erythematous patches on heavily sun-exposed areas (like the back of the hands or a bald scalp) in older, fair-skinned individuals .
- **SCC Basics:** It is more aggressive, grows faster, and has a higher metastatic risk than BCC .
- **Location:** Like BCC, it is highly associated with sun exposure and most commonly found on the **face / head / neck** . (It is *not* predominantly on the extremities).

3. Melanoma & Nevi

- **The ABCDE Rule:** 'E' stands for **Evolution** . Any changing pigmented lesion (size, color, shape) is highly suspicious .
- **Biopsy Rule:** If you suspect melanoma, the most appropriate next step is an **excisional biopsy** . (Do not do chemical peeling or just a dermatoscope exam).
- **Subtypes:**
    - *Superficial Spreading:* The most common type. Presents as an asymmetrical lesion with ill-defined borders and variable colors .
    - *Nodular Melanoma:* The **worst/most aggressive** type. It has early **vertical growth**, leading to early metastasis .
- **Prognosis:** The most critical prognostic factor is **Breslow thickness**, which measures the depth of the tumor from the **granular layer** down to the deepest point of invasion . (Gender is not a primary staging factor).
- **Nevi (Moles):** About **20-40%** of melanomas arise from pre-existing nevi . Among nevi, **junctional nevi** have the classic association with malignant potential compared to intradermal nevi . Also, acquired nevi are *not* common in infants; infants get congenital nevi .

4. Benign Tumors & Skin Manifestations (Quick Hits)

- **Seborrheic Keratosis:** A **benign** tumor of the epidermis that has a classic "stuck-on" waxy appearance .
- **Syringoma:** A benign tumor of the **eccrine sweat glands**, often presenting as multiple small flesh-colored papules around the eyelids .
- **Paget's Disease:** If a woman presents with a persistent, unilateral "eczematous" rash on the areola/nipple, you MUST do a **skin biopsy** to check for underlying breast adenocarcinoma .
- **Cutaneous Metastases:** The tumor that most frequently metastasizes to the skin is **Breast Carcinoma** .
- **Neurofibromatosis Type 1 (NF1):** The classic triad includes freckling, café-au-lait spots, and **Lisch nodules** (iris hamartomas) .

Hair & Nail Disorders

1. The Normal Hair Cycle

- **Anagen (Growth):** The active growth phase. At any given time, the vast majority (**~85%**) of scalp hair follicles are in the anagen phase . Hair grows at an average rate of **1 cm per month** and spends roughly 3–4 years in this phase .
- **Catagen (Transition):** A brief transition phase.
- **Telogen (Resting):** The resting and shedding-ready stage of the hair cycle .
- *Exam Trap:* "Male hair grows faster" is false; hair characteristics and growth are strongly determined by genetics .

2. Diffuse Shedding (The Effluviums)

- **Telogen Effluvium:** * *Mechanism:* A diffuse, non-scarring shedding disorder where a stressor prematurely pushes many follicles into the resting (telogen) phase .
    - *Triggers:* Typically happens weeks or months after **childbirth (post-partum), major surgery (surgical shock), severe fever, crash dieting/cachexia**, or certain drugs like **heparin** .
    - *Diagnostics:* Wood's lamp does *not* help diagnose telogen effluvium .
- **Anagen Effluvium:**
    - *Mechanism:* Rapid, diffuse, non-scarring hair loss caused by an abrupt interruption of the active growth phase (mitosis stops in the hair matrix) .
    - *Trigger:* Classically caused by **cytotoxic drugs (chemotherapy)**.

3. Patchy & Traumatic Alopecia

- **Alopecia Areata:** * *Basics:* An **autoimmune**, non-scarring patchy hair loss that can occur in children and is highly recurring .
    - *Look:* It produces smooth, round patches. (Remember from earlier sections that it classically features "exclamation mark" hairs). It does *not* fluoresce under a Wood's lamp.
- **Traumatic Alopecia:** Hair loss caused by physical/mechanical force. This includes traction (tight hairstyles), pressure, marginal alopecia, and **Trichotillomania** (compulsive hair pulling, which causes irregular patches of broken hairs) .

4. The "Scarring vs. Non-Scarring" Rules

You must know which diseases permanently destroy the hair follicle (scarring/cicatricial) and which ones don't.

- **Non-Scarring (Reversible):** * Telogen effluvium, anagen effluvium, and alopecia areata.
    - **Male Pattern Baldness** (androgenetic alopecia).
    - **Secondary Syphilis** (causes "moth-eaten" alopecia) .
- **Scarring / Cicatricial (Permanent loss):**
    - **Morphea:** Localized scleroderma that causes dermal sclerosis/atrophy, leading to scarring alopecia if it hits the scalp.
    - **Sarcoidosis:** Granulomatous inflammation that can destroy follicles on the scalp.
    - *(Recall from the Connective Tissue section: Discoid Lupus Erythematosus also causes scarring alopecia).*
    - *(Recall from the Fungal section: Kerion can cause scarring if severe).*