Dermatology Final

🔊 **Podcast Style Review (Experimental Feature)**

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- **NOTE:** Highlighted in **bold** are the important key info!
- Topics are arranged in order of most to least commonly tested
- Check the table of contents below for easier navigation
- Good luck 🍀

1. Connective Tissue Diseases / Cutaneous Manifestations of Systemic Diseases

- **Lichen Planus (LP)**
  - Nail changes:
    - **Pterygium** (hypertrophy and distal proliferation of nail fold).
    - Other changes include thinning, dystrophy, longitudinal ridging.
    - Onycholysis can occur; pitting is less typical than in psoriasis.
    - **Nail thickening is NOT typical** (usually thinning).
  - Mucosal involvement: **Buccal mucosa is the most common site** if mucosa is involved.
  - Symptoms: **Itching is a characteristic feature**.
  - Histology: **Hypergranulosis**, sawtooth rete ridges, band-like lymphocytic infiltrate, Civatte bodies. (Hypogranulosis is NOT typical).

    - Primary lesion: **Papule** (not macule).
    - Course: Often self-limiting; 50% of cases may clear within 18 months, but chronicity can occur, especially with mucous or hypertrophic lesions.
    - Features: **Wickham's striae** are characteristic.
- **Lupus Erythematosus**
    - Discoid Lupus Erythematosus (DLE):
        - **Can cause patchy scarring alopecia**.
        - Histology: **Destruction of basal cell layer** (liquefaction degeneration).
        - Progression to Systemic Lupus Erythematosus (SLE) occurs in **5-10% of cases**.
    - Systemic Lupus Erythematosus (SLE):
        - Commonest cutaneous eruption: **Malar (butterfly) rash**; photosensitive erythema is also common.
        - Neonatal lupus: Only a small percentage of babies born to mothers with Ro/La antibodies develop SLE later in life.
    - Photosensitivity: A girl with photosensitivity and an ANA titer of 1:32 may require further investigation (e.g., repeat ANA, anti-dsDNA, ENA) and photoprotection.
- **Dermatomyositis**
    - Associations:
        - **Frequently associated with underlying malignancy in adults**.
        - Childhood dermatomyositis is frequently associated with **Calcinosis**.
    - Clinical Signs:
        - **Heliotrope rash (pathognomonic)**.
        - **Gottron's sign/papules**.
        - Proximal muscle weakness.
        - **Ragged cuticles** and nail fold telangiectasias.
    - Demographics: More common in females.
    - Risk: **Calcinosis can occur in adults**, though more common in juvenile form.
- **Morphea (Localized Scleroderma)**
    - Tissue involvement: Can include epidermis, subcutaneous tissue, muscles, and bones. **All layers can be affected** depending on type/depth.
    - Muscular atrophy: Can be associated with **linear morphea** (e.g., en coup de sabre).
    - Course: May or may not improve with time; can progress or stabilize.
    - Features: Presents with well-defined patches; not caused by UV light.
- **Erythema Multiforme (EM)**
    - Most common cause: **Herpes simplex virus (HSV)**, especially for EM minor.
    - Other causes: Drugs, Mycoplasma.
    - Ulceration: Can occur, particularly in **EM major / Stevens-Johnson Syndrome**.
- **Erythema Nodosum (EN)**
    - Causes: Streptococcal infections, sarcoidosis, drugs, TB, pregnancy.
    - Association with malignancy: Can occur.
    - Ulceration: **Erythema nodosum leprosum (Type 2 Lepra reaction) can ulcerate**.
        - Typical EN lesions classically do *not* ulcerate. This distinction should be clearer to avoid confusion
- **Erythema Gyratum Repens**
    - Strongly associated with **underlying internal malignancy (often lung cancer)**; considered pathognomonic.
    - NOT typically precipitated by streptococcal throat infection.

- **Dermatitis Herpetiformis (DH)**
  - Associations: **Chronic autoimmune disease, frequently associated with celiac disease (gluten-sensitive enteropathy)**.
  - Histopathology: **Subepidermal blisters with neutrophilic and eosinophilic microabscesses at the tips of dermal papillae**. The prickle cell layer is NOT the primary abnormal layer.
  - Treatment: **Dapsone (diamino-diphenyl sulfone)**.
- **Vitiligo**
  - Pathophysiology: **Results from the destruction of melanocytes**, leading to DEpigmented (not hypopigmented) patches.
  - Melanocytes: **Number of melanocytes is decreased or absent** in affected areas.
  - Associations: Significantly associated with **autoimmune thyroid disease (hypo- or hyperthyroidism)**, pernicious anemia, Addison's disease, alopecia areata. Reticulosis is NOT a typical association.
  - Demographics: Affects males and females equally; onset usually in 20s and 30s.
- **Piebaldism**
  - Inheritance: **Autosomal dominant**.
- **Post-Inflammatory Hypopigmentation**
  - Causes: Can occur after psoriasis, lichen planus, and other inflammatory conditions.
- **Chloasma (Melasma)**
  - Treatment of choice: **Topical hydroquinone-containing ointments** (e.g., Eldoquin).
- **Hodgkin's Disease**
  - Commonest cutaneous lesion: Often **secondary to pruritus** (e.g., excoriations, lichenification); specific skin infiltrates are less common.

2. Fungal Infections

- **General Principles**
  - Tinea capitis: Usually requires **systemic antifungal treatment**.
  - Tinea pedis: Usually treated topically.
  - Pityriasis versicolor: **Can recur**.
  - Chronic paronychia: Often caused by mixed yeast and bacterial infection.
- **Tinea Capitis**
  - Epidemics: Often caused by anthropophilic fungi like **Microsporum audouinii**.
  - Causative organisms: Microsporum species (e.g., M. audouinii, M. canis), Trichophyton species (e.g., T. tonsurans, T. schoenleinii, T. violaceum, T. verrucosum).
  - Fluorescence: **Apple green fluorescence under Wood's lamp is seen with some Microsporum species** (e.g., M. canis, M. audouinii).
    - **T. rubrum does NOT fluoresce**.
    - T. schoenleinii may show **dull blue** under Wood’s-lamp examination.
  - Inflammatory Tinea Capitis (Kerion): Often caused by zoophilic fungi like **T. verrucosum and T. mentagrophytes**.
  - Ectothrix infection: Caused by fungi like **T. mentagrophytes**, M. canis. (T. schoenleinii is typically endothrix/favic).
  - Acquisition from cattle: **Trichophyton verrucosum**.
  - Treatment: **Oral Griseofulvin is a first-line treatment**.
- **Griseofulvin**
  - Pharmacokinetics: **Absorption is better after a fatty meal**.
  - Side effects: **Headache is a common side effect**.
  - Contraindications: **Contraindicated in pregnancy**.

- Interactions: Phenobarbitone may reduce its effectiveness.

- **Tinea Pedis**
  - Prevalence: **Commonest fungal infection in adults**.
  - Causative organisms: T. rubrum, T. mentagrophytes, E. floccosum.
- **Tinea Corporis**
  - Lesion appearance: Typically **annular** with slightly elevated scaling margins and central clearing.
- **Tinea Versicolor**
  - Causative agent: **Malassezia species**.
  - Clinical features: Hypo- or hyperpigmented patches or plaques with fine scaling.
  - Wood's lamp: Shows **yellowish-gold or coppery-orange fluorescence** (NOT apple green or cherry red).
- **Onychomycosis (Tinea Unguium)**
  - Infection site: Primarily affects the **nail plate**.
  - Clinical features: Nail discoloration, onycholysis.
  - Treatment: **Oral antifungal agents are usually required for toenail onychomycosis**.
  - Causative organisms: T. rubrum is common.
  - Duration of treatment: **Prolonged treatment is often necessary**.
- **Candidiasis**
  - Cutaneous candidiasis: Presents as erythema with **satellite pustules**, especially in body flexures.
  - Sites: Interdigital areas, paronychial (proximal nail fold), angular cheilitis (corners of mouth), oral mucosa (thrush), genital area.
  - Skin colonization: Candida albicans is a commensal but not as ubiquitously colonizing healthy skin as Staphylococci.
  - Affected tissues: Skin, mucous membranes, nails. **Hair is not typically affected**.

3. Eczema (Dermatitis)

- **General Histology**
  - Acute eczema: Characterized by **spongiosis** (intercellular edema in the epidermis).
  - Subacute/Chronic: Parakeratosis, acanthosis. **Normal or thickened granular layer** (unlike psoriasis where it's reduced/absent).
- **Atopic Dermatitis (AD)**
  - Pathophysiology: **T helper cells (Th2 predominant in acute) play a major role**.
  - Infantile AD:
    - Onset: **Typically begins after 2 months of age**, not at birth.
    - Distribution: **Extensor surfaces and face** are common sites in infants. (Flexural involvement is more typical in older children/adults).
  - Adult AD Associations: Pruritus ani, asthma. Hair fall is NOT a direct association.
  - Investigations: **Elevated serum IgE levels** are common.
  - Lymphocytes: Bear greater than normal amounts of **IgE** on their surface.
- **Seborrheic Dermatitis**
  - Age of onset: Can occur in infants (cradle cap) and adults.
  - Distribution: Scalp, **face (nasolabial folds, eyebrows)**, post-auricular areas, presternal. **Flexural/intertriginous areas**, not extensors, are common body sites.
  - Association: Linked to **Malassezia yeast**.
  - Prognosis: Generally **better prognosis than atopic dermatitis**.
- **Contact Dermatitis**

    - Mechanism: Usually a **Type IV cell-mediated hypersensitivity reaction**.
    - Diagnostic test: **Patch test is pathognomonic** for allergic contact dermatitis.
    - Common allergens:
        - **Nickel** is a very common cause of allergic contact dermatitis.
        - **Primula** (plant) can cause severe, even bullous, contact dermatitis.
        - Perfumes and hair dyes can cause **pigmented contact dermatitis**.
    - Site specific:
        - Nail varnish dermatitis: Commonest site is the **face and neck** (transfer from fingers).
        - Clothing dermatitis: Commonest site is **body flexures**.
    - Timing: Develops **12-72 hours after exposure** to the allergen.
- **General Eczema Features**
    - Lichenification: **Thickening and hardening of the skin with exaggerated skin markings**, seen in chronic eczema.
    - Blisters (vesicles/bullae): Can occur in acute eczema. **Chronic eczema typically does not cause blisters**.
    - Unilateral hand eczema: **KOH examination of skin scrapings** is important to rule out tinea manuum.
    - Pityriasis alba: Appears **hypopigmented** under Wood's light.

4. Psoriasis

- **Histopathology**
    - Key features: **Parakeratosis** (retention of nuclei in stratum corneum), hyperkeratosis, acanthosis with elongated rete ridges, suprapapillary epidermal thinning, Munro's microabscesses (neutrophils in stratum corneum). **Epidermal atrophy is NOT a feature** (epidermis is thickened).
- **Nail Psoriasis**
    - Commonest manifestation: **Pitting**.
    - Other signs: Onycholysis, subungual hyperkeratosis, discoloration ("oil drop" sign).
    - Cause of nail pitting: **Loss of parakeratotic cells from the nail surface** due to involvement of the proximal nail matrix.
    - **Clubbing is NOT a characteristic feature** of psoriasis nails.
- **Clinical Types and Triggers**
    - Guttate psoriasis: Often **triggered by streptococcal infection**.
    - Exacerbating factors: Infections, stress, certain drugs (e.g., lithium, beta-blockers, **antimalarials**, NSAIDs, withdrawal of systemic corticosteroids), hypocalcemia, alcohol, smoking. **Hypercalcemia is NOT an exacerbating factor**.
- **Psoriatic Erythroderma**
    - Complications: Temperature dysregulation, dehydration, sepsis, high-output cardiac failure.
- **Treatment Considerations**
    - Systemic treatments: Methotrexate, cyclosporine, acitretin (systemic retinoid), biologic agents (TNF-alpha blockers). PUVA (Psoralen + UVA). Azathioprine is a second-line agent.
    - **Isotretinoin is NOT typically used for psoriasis** (used for acne).
    - **Antimalarials are generally NOT used and can exacerbate psoriasis**.
    - Topical Vitamin D analogues are used.
    - **Oral steroids are generally avoided for chronic plaque psoriasis** due to risk of rebound flares; may be used short-term for erythrodermic or pustular psoriasis.
- **Inheritance**
    - Pattern: Complex, polygenic. Often described as **autosomal dominant with incomplete penetrance**, not autosomal recessive.

- **Associated Conditions**
  - Psoriatic arthritis.
  - Metabolic syndrome.

5. Viral Infections

- **Herpes Simplex Virus (HSV)**
  - Recurrent HSV: **Herpes labialis (cold sores) is the commonest form**.
  - Herpes genitalia: **Viral shedding and contagion can occur even when asymptomatic**.
- **Varicella-Zoster Virus (VZV) - Herpes Zoster (Shingles)**
  - Clinical features:
    - Pain may precede the rash.
    - Rash is vesicular, typically **unilateral and dermatomal** (NOT commonly bilateral).
    - More serious in elderly and immunocompromised individuals.
  - Complications: **Postherpetic neuralgia** can last for months.
  - Treatment: Systemic antiviral treatment (e.g., acyclovir, valacyclovir, famciclovir) is indicated for patients >50 years, immunocompromised, or with severe/ophthalmic involvement. **Topical acyclovir has limited efficacy for acute zoster**.
- **Pityriasis Rosea**
  - Etiology: Associated with **Human Herpesvirus 6 (HHV-6) and/or HHV-7**.
  - Clinical features:
    - **Herald patch** (a single larger lesion) often precedes the generalized eruption.
    - Generalized rash consists of oval, erythematous, slightly raised patches with fine scales, often arranged in a "Christmas tree" pattern on the trunk. Collaret scales.
    - Itching is variable, usually mild to moderate.
    - **Self-limiting**, typically resolving in 6-8 weeks.
  - Diagnosis: Primarily clinical; family history is not typically relevant.
  - Appearance: Primarily a papulosquamous eruption, not macular.
- **Molluscum Contagiosum**
  - Causative agent: **Poxvirus**.
- **Warts (Verrucae)**
  - Causative agent: **Human Papillomavirus (HPV)**, a double-stranded DNA virus. (NOT HHV-6).
  - Plain warts (Verruca plana):
    - Flat-topped papules, often on the face and hands.
    - **Koebner phenomenon** can occur.
    - Often resolve spontaneously.
    - **Flat-topped, not spiky**.
  - Common warts (Verruca vulgaris):
    - Rough, hyperkeratotic papules.
    - **Do NOT typically transform into skin cancer**.
    - Often resolve spontaneously.
  - Filiform/Digitate warts: Often affect body flexures, face.
  - Plantar warts: On soles of feet, can be painful. Surface can be rough/hyperkeratotic.
  - Treatment: Cryotherapy, salicylic acid, 5-fluorouracil (5-FU). **Topical steroids are NOT a treatment for viral warts and can worsen them**.

- Genital warts (Condylomata acuminata):
  - Treatment options include **podophyllotoxin**, trichloroacetic acid, cryotherapy, imiquimod.

- **Viral Exanthems**
  - Roseola infantum (Exanthem subitum - HHV-6/7), Slapped cheek syndrome (Erythema infectiosum - Parvovirus B19), Rubella are viral.
  - **Scarlet fever eruption is caused by a bacterial toxin (Streptococcus pyogenes)**.

6. Skin Tumors

- **Nevi (Moles)**
  - Nature: Developmental disorders resulting from abnormal proliferation of melanocytes.
  - Malignant degeneration: **Junctional nevi** (and compound, dysplastic nevi) have a higher potential for malignant transformation to melanoma.
  - ACTH: Nevi do **not typically increase in size or number after ACTH injection**.
- **Actinic Keratosis (AK)**
  - Nature: **Premalignant lesion** that can progress to squamous cell carcinoma (SCC). It is not malignant itself.
  - Clinical features: Fine scaled erythematous plaques on sun-exposed skin, especially in fair-skinned individuals.
- **Seborrheic Keratosis**
  - Nature: **Benign epidermal tumor**.
- **Basal Cell Carcinoma (BCC)**
  - Most common type: **Nodular (or noduloulcerative) BCC**.
  - Clinical features (Nodular BCC): Pearly papule or nodule with telangiectasia, often on the face (e.g., nose).
  - Prognosis: Generally good, as BCC is slow-growing and rarely metastasizes. **Not always associated with a bad prognosis**.
  - Metastasis: **Rare**, but if it occurs, regional lymph nodes are the most frequent site.
  - Demographics: More common in Caucasians.
- **Squamous Cell Carcinoma (SCC)**
  - Commonest site: Sun-exposed areas like the **face, lower lip, ears, hands**.
  - Growth rate: Can be faster growing than BCC.
  - Origin: Can arise from **actinic keratosis**.
  - Location: Approximately **75% of SCC lesions occur on the head and neck**.
- **Melanoma**
  - Prognosis:
    - **Nodular melanoma generally has the worst prognosis** and is associated with early metastasis.
    - Key prognostic factors: **Breslow thickness** (measured from granular layer or ulcer base to deepest point of invasion), ulceration, mitotic rate, lymph node status.
    - Gender can be a factor (females often have better prognosis).
  - Origin: Can arise de novo or from a pre-existing nevus (estimates vary, **around 20-40%** from nevi).
  - Clinical features (ABCDEs): Asymmetry, Border irregularity, Color variegation, Diameter >6mm, Evolving.
  - Commonest type: Superficial spreading melanoma.
- **Eccrine Sweat Gland Tumors**
  - Example: **Syringoma** (benign).
- **Paget's Disease of the Breast**
  - Presentation: Unilateral, eczematous rash of the areola.
  - Next step: **Skin biopsy is crucial** to confirm diagnosis and rule out other conditions.

- **Metastasis to Skin**
  - Most frequent primary tumors metastasizing to skin: **Breast cancer** in women, lung cancer in men. Melanoma itself can also metastasize to skin.

7. Normal Skin Structure and Function

- **Epidermis**
  - Stratum corneum: **Devoid of nuclei**.
  - Basal layer: **Mitotic cells (cell division) are primarily limited to this layer**.
  - Melanocytes:
    - Located in the basal layer.
    - Connect to approximately 36 surrounding keratinocytes (epidermal melanin unit).
    - Appear as clear, larger cells relative to surrounding keratinocytes under a microscope.
    - **Number of melanocytes is similar across different skin phototypes**; differences in skin color are due to the size, number, and packaging of melanosomes, and rate of melanin degradation.
  - Langerhans cells: **Dendritic, antigen-presenting cells** found in the epidermis.
  - Merkel cells: Mechanoreceptor cells, dendritic in appearance, located near nerve endings in the basal layer.
- **Dermis and Subcutis**
  - Pacinian corpuscles: Mediate sensation of **deep pressure and vibration**.
  - Meissner's corpuscles: Mediate sensation of **light touch**. Located in dermal papillae.
- **Glands**
  - Sebaceous glands:
    - Originate from **ectoderm**.
    - Holocrine glands.
    - Controlled by **androgens**.
    - **NOT normally found in buccal mucosa** or glabrous skin (palms/soles). Found on face, scalp, upper back, vermilion of lip, areola of nipple.
    - Meibomian glands of the eyelids are modified sebaceous glands.
  - Sweat glands:
    - Controlled by **neurons** (autonomic nervous system).
    - Eccrine sweat glands: Cholinergic innervation.
    - Apocrine sweat glands: Characterized by decapitation secretion; adrenergic innervation.
- **Glabrous Skin (Palms and Soles)**
  - Characteristics: Thick epidermis, dermatoglyphics (fingerprints), presence of encapsulated sensory organs.
  - **Absence of sebaceous glands and hair follicles**.

8. Acne and Rosacea

- **Acne Vulgaris**
  - Pathophysiology:
    - **Follicular plugging (comedone formation) is the first step**.
    - Increased sebum production (androgen-mediated).
    - Proliferation of *Propionibacterium acnes* (now *Cutibacterium acnes*).
    - Inflammation.
    - **Epidermal edema is not a primary pathogenic feature**.
  - Lesions:
    - **Comedones (open and closed) are the primary non-inflammatory lesions**.

        - Inflammatory lesions include papules, pustules, nodules, cysts.
        - **Vesicles are NOT typical lesions of acne vulgaris**.
    - Precursor of large inflammatory lesions: **Papules** (which arise from comedones).
    - Factors: Greasy cosmetics may cause/worsen acne.
    - Flare-ups: Can be caused by steroids, certain antimalarial drugs, Vitamin B12. **Estrogens (in OCPs) often improve acne**.
    - Chloracne: Comedones predominate.
    - Treatment:
        - **Isotretinoin is very effective for severe cystic acne**.
        - **Metronidazole is used for rosacea, NOT commonly for systemic treatment of acne vulgaris**.
- **Oral Isotretinoin**
    - Side effects: **Teratogenicity (Pregnancy Category X)**, dry lips (cheilitis is very common), dry skin/mucosa, elevated triglycerides, elevated liver enzymes, potential for hair loss. Increased intracranial pressure (rare).
    - Monitoring: Blood tests (lipids, LFTs, pregnancy test) are required before and during treatment.
    - Pregnancy: **Strict contraception is mandatory** during and for at least one month after stopping treatment.
    - **Scarring alopecia and infertility are NOT typical side effects**.
- **Acne Rosacea**
    - Clinical features: Persistent facial erythema, telangiectasias, papules, pustules. Rhinophyma can occur in severe, long-standing cases.
    - Age of onset: **Typically affects adults (30-50s)**, not primarily teenagers.
    - Distinction from Acne Vulgaris: **Rosacea lacks comedones**. Rosacea has more prominent telangiectasias and persistent erythema.

9. Bullous Diseases

- **Pemphigus Vulgaris**
    - Pathophysiology: **Autoimmune disease** with antibodies against desmogleins (Dsg1 and Dsg3) leading to acantholysis.
    - Blisters: **Intraepidermal (suprabasal), flaccid bullae**, often on skin and mucous membranes (painful oral erosions are common).
    - Prognosis: **Associated with significant morbidity and mortality if untreated**.
    - Demographics: More common in middle-aged to elderly individuals, and in certain ethnic groups (e.g., Ashkenazi Jews).
- **Bullous Pemphigoid**
    - Pathophysiology: Autoimmune disease with antibodies against hemidesmosome components (BPAG1/BP230 and BPAG2/BP180/Collagen XVII) at the dermoepidermal junction.
    - Blisters: **Subepidermal, tense bullae**.
    - Immunofluorescence: **Linear deposits of IgG and C3 along the basement membrane zone**.
    - Prognosis: Generally less severe than pemphigus vulgaris.
- **Differentiating Pemphigus and Pemphigoid**
    - Blister location: Pemphigus = **intraepidermal**; Pemphigoid = **subepidermal**.
    - Blister type: Pemphigus = **flaccid**; Pemphigoid = **tense**.
    - Mortality: **Pemphigus generally has higher morbidity/mortality if untreated**.
- **Epidermolysis Bullosa (EB)**
    - General: Group of inherited disorders characterized by skin fragility and blister formation.
    - Mucous membrane involvement: Can be extensive in severe forms, particularly **dystrophic EB**.

- Scarring:
  - **Dystrophic EB heals WITH scarring**.
  - Epidermolysis bullosa simplex typically heals WITHOUT scarring.

- **Other Blistering Conditions**
  - Generalized blistering can be caused by Pemphigus gestationis (Herpes gestationis).
  - Impetigo, dermatitis herpetiformis can cause epidermal bullae.
  - **Chronic eczema does NOT typically cause epidermal bullae** (acute eczema can).
- **Diagnostic Aids**
  - **Immunofluorescence (direct and indirect) is highly valuable** in differentiating autoimmune bullous diseases like bullous pemphigoid from other conditions like erythema multiforme.

10. Hair, Scalp, and Nail Disorders

- **Hair Cycle and Growth**
  - Anagen phase (growing): Approximately **85% of scalp hair follicles** are in this phase. Lasts 2-6 years.
  - Telogen phase (resting): **Resting stage of hair**. Lasts 2-3 months.
  - Growth rate: Approximately **1 cm/month**.
  - Hair characteristics: Genetically determined.
- **Alopecia (Hair Loss)**
  - Traumatic alopecia: Caused by traction, pressure, trichotillomania.
  - Alopecia Areata:
    - **Non-scarring alopecia**, often patchy.
    - Can occur in children and is often recurrent.
    - **Does NOT fluoresce under Wood's lamp**.
  - Telogen Effluvium:
    - Diffuse non-scarring hair shedding, occurring a few months after a trigger (e.g., childbirth, surgery, severe illness, crash diet, drugs like heparin).
    - **Wood's lamp does NOT aid in diagnosis**.
  - Anagen Effluvium:
    - Diffuse hair loss due to interruption of anagen phase, classically caused by **cytotoxic drugs (chemotherapy)**.
  - Non-cicatricial (non-scarring) alopecia causes:
    - Diffuse: Telogen effluvium, anagen effluvium, endocrine dysfunction (e.g., hypothyroidism), nutritional deficiencies, hereditary hair shaft abnormalities.
    - Patchy: Male/female pattern hair loss, alopecia areata, **secondary syphilis (moth-eaten alopecia)**.
    - **Trichotillomania causes patchy, irregular non-scarring alopecia**, not typically diffuse.
  - Cicatricial (scarring) alopecia causes:
    - **Morphea** (if scalp involved), DLE, lichen planopilaris, severe infections, sarcoidosis (if cutaneous lesions involve scalp).
- **Nail Structure**
  - Nail cuticle (eponychium): Formed by the **dorsal layer of the posterior nail fold**.
  - Nail matrix disorders: Can result in changes in nail shape, longitudinal ridging, and thickened nails.
- **Specific Hair Disorders**
  - Netherton's syndrome: Characteristic hair lesion is **trichorrhexis invaginata ("bamboo hair")**.

11. Sexually Transmitted Diseases (STDs)

- **Syphilis**

- Treatment: **Benzathine penicillin G is the treatment of choice for all stages** (except neurosyphilis, where aqueous crystalline penicillin G is used).
- Diagnosis:
  - Early diagnosis: **Dark field microscopy of chancre exudate**. Serological tests like **FTA-ABS (or other treponemal tests like TPPA/TPHA) become positive later but are very sensitive and specific**.
  - Follow-up: **Non-treponemal tests (VDRL/RPR) are used to monitor treatment response** as titers decrease. FTA-ABS typically remains positive for life.
- Primary Syphilis:
  - Chancre: Typically **painless**, single, indurated ulcer at the site of inoculation, rich with treponemes.
  - Dark field microscopy: If negative from chancre, aspirate from regionally enlarged lymph node can be examined.
- Secondary Syphilis:
  - Onset: Lesions usually appear 2-12 weeks after infection.
  - Rash: **Maculopapular or papulosquamous rash**, often involving palms and soles. Typically generalized and can be itchy (variable). **NOT commonly vesicular**.
  - **Condylomata lata**: Highly infectious, moist, flat-topped papules in intertriginous areas.
  - **Moth-eaten alopecia**: Patchy non-scarring alopecia.
  - Other: Mucous patches, generalized lymphadenopathy (usually non-painful), constitutional symptoms. Auditory neuritis, periostitis can occur.
- Treponema pallidum survival: Dies within **approximately 48-72 hours** in blood stored at normal refrigerator temperature (+4°C).

- **Gonorrhea**
  - Causative agent: *Neisseria gonorrhoeae*, a **Gram-negative diplococcus**.
  - Symptoms: Many females (up to 50%) can be asymptomatic.
  - Site of infection: **Columnar epithelium** is a predilection site (e.g., endocervix, urethra).
  - Best swab site (female): **Endocervical swab**.
  - Treatment: Penicillin resistance is widespread; current guidelines recommend combination therapy (e.g., ceftriaxone plus azithromycin or doxycycline). **Old low-dose, long-term penicillin regimens are NOT effective**.
- **Chancroid**
  - Causative agent: **Haemophilus ducreyi**.
- **Non-Gonococcal Urethritis (NGU)**
  - Treatment: **Doxycycline (a tetracycline) or Azithromycin** are drugs of choice.

12. Bacterial Infections

- **Impetigo**
  - Nature: **Most superficial bacterial skin infection**.
  - Causative agents: **Staphylococcus aureus and/or Streptococcus pyogenes**.
  - Commonly affected: **Infants and children**.
- **Ecthyma**
  - Nature: **Deeper, ulcerative form of impetigo**, extending into the dermis. It is NOT a superficial infection.
  - Common cause: Streptococcus pyogenes.
  - Predisposing factors: Immunocompromised individuals.
- **Erysipelas**
  - Clinical features: **Well-defined, erythematous, indurated plaque** with a raised border, often on the face or lower limbs. Fever and constitutional symptoms can occur.

- Causative agent: Primarily **Streptococcus pyogenes**. Staphylococcus aureus is NOT the primary cause (though it can cause cellulitis or secondary infection).
- Treatment: **Penicillin is the drug of choice**.

- **Erythrasma**
  - Causative agent: *Corynebacterium minutissimum*.
  - Wood's light examination: Shows a characteristic **coral-red fluorescence**.
- **Staphylococcus aureus Carriage**
  - Main local source contaminating the skin: **Anterior nares (nose)**.

13. Urticaria and Angioedema

- **Primary Lesion of Urticaria**
  - **Wheal**: An edematous, erythematous, transient plaque that blanches with pressure. Very itchy.
  - **Does NOT typically leave a hypopigmented scar**.
- **Pathophysiology**
  - Main cells involved: **Mast cells** (release of histamine and other mediators).
- **Acute Urticaria**
  - Treatment of choice: **Antihistamines**.
  - **Oral steroids are NOT first-line treatment**; reserved for severe or refractory cases.
- **Chronic Urticaria**
  - Cause: In **up to 90% of chronic cases, the cause is unknown** (chronic idiopathic urticaria).
- **Cold Urticaria**
  - Characteristics: Can be familial or acquired, may be transferable in serum (passive transfer test), and can result in systemic reactions (e.g., unconsciousness if swimming in cold water).
- **Cholinergic Urticaria**
  - Diagnostic test: Provocation with **exercise and heat challenge** is most reliable. Intradermal methacholine test can also be used.
- **Treatment**
  - Antihistamines: Both sedating and non-sedating antihistamines are used.
    - For day-time use (less sedating): **Second-generation antihistamines (e.g., piperidines like loratadine, cetirizine, fexofenadine)**.
  - **Topical antihistamine ointments are generally NOT effective** for widespread urticaria.
- **Contact Sensitivity**
  - Degree of sensitivity is influenced by: Amount of allergen, frequency of exposure, and route of exposure.

14. Ichthyosis

- **Ichthyosis Vulgaris (Simplex)**
  - Prevalence: **Most common type of ichthyosis**.
  - Clinical features: Fine, white scales, typically involves extensors, **spares flexural areas**. Often associated with **keratosis pilaris and atopy**.
  - Onset: Usually presents **after 3 months of age**, not at birth.
- **X-linked Ichthyosis**
  - Cause: Steroid sulfatase deficiency.
  - Associations: May be associated with corneal opacities, cryptorchidism. Congenital ichthyosis with renal agenesis/hernia is a severe manifestation potentially linked.
- **Bullous Ichthyosiform Erythroderma (Epidermolytic Ichthyosis)**
  - Inheritance: **Autosomal dominant**.

- **Non-bullous Ichthyosiform Erythroderma (e.g., Lamellar Ichthyosis, Congenital Ichthyosiform Erythroderma)**
  - Inheritance: **Autosomal recessive**.
- **Ichthyosis Hystrix**
  - Clinical features: Severe form with prominent hyperkeratosis, leading to thickening of all skin layers.
- **Defective Keratinization**
  - Common feature in various ichthyoses, psoriasis, epidermolytic hyperkeratosis. Lichen sclerosus et atrophicus involves epidermal atrophy and dermal changes, less primarily a keratinization defect in the same context.

15. Pruritus and Scabies

- **Pruritus**
  - Biliary obstruction: Pruritus is most directly related to the accumulation of **bile salts** in the skin.
- **Scabies**
  - Causative agent: *Sarcoptes scabiei var. hominis*.
  - Transmission: Typically by prolonged close personal contact. Can spread by simple handshake if prolonged.
  - Pruritus onset: After initial infestation, pruritus (due to sensitization) typically develops in **2-6 weeks**.
  - Clinical features:
    - Intense itching, characteristically **worse at night**.
    - **Burrows** are pathognomonic lesions (best yield for scrapings).
    - Commonly affected sites in adults: Finger webs, wrists, axillae, areolae, umbilicus, genitalia. **Back is usually spared in adults**.
    - Infants: Can affect face, scalp, palms, and soles (sites often spared in adults). May present with acral pustules. **Family history of itching IS usually present**.
  - Treatment:
    - **Permethrin 5% cream is a first-line treatment**.
    - **All household members and close contacts should be treated simultaneously**.
    - Itching may persist for weeks even after successful treatment (post-scabetic pruritus).
    - **Benzoyl benzoate** is a topical treatment; **benzoyl peroxide is for acne and not used for scabies**. Systemic treatment is ivermectin.

16. Drug Rashes

- **Acne Medicamentosa (Acneiform Eruption)**
  - Causative drugs: Phenytoin, B12, corticosteroids, lithium, isoniazid.
  - **Azelaic acid is a treatment for acne**, not a cause of acne medicamentosa.
- **Stevens-Johnson Syndrome (SJS) / Toxic Epidermal Necrolysis (TEN)**
  - Nature: Severe, life-threatening mucocutaneous reactions.
  - Most common cause: **Drugs** (e.g., sulfonamides, anticonvulsants, NSAIDs, allopurinol), not infection.
  - Clinical features: Prodrome of fever/malaise, followed by painful skin, widespread blistering and epidermal detachment, mucosal erosions (oral, ocular, genital).
    - **SJS involves <10% BSA detachment, SJS/TEN overlap 10-30%, TEN >30% BSA detachment**.
    - **Absence of oral erosions would NOT be typical for TEN**.
  - Management: **Requires intensive care unit (ICU) management** due to high fatality rate.

17. Miscellaneous Dermatological Conditions and Principles

- **UV Light and Skin**
  - Penetration: **UVA penetrates deeper into the skin than UVB**. UVC is mostly absorbed by the ozone layer.
  - Intensity:

        - **UVA intensity is relatively constant throughout the day**.
        - UVB intensity peaks around midday.
- **Sunscreens**
    - Mechanism: Physical sunscreens (e.g., zinc oxide, titanium dioxide) reflect UV radiation. Chemical sunscreens absorb UV radiation.
    - SPF (Sun Protection Factor): Measures protection against **UVB**.
    - UVA protection: Indicated by ratings like PA system (PA+, PA++, etc.) or UVA star rating.
    - Efficacy: The actual level of sun protection achieved is often less than specified on the bottle due to inadequate application by users.
- **Corticosteroids**
    - Topical Corticosteroids (Side Effects of Prolonged Use):
        - **Skin atrophy**, telangiectasia, striae.
        - Hypopigmentation or hyperpigmentation.
        - **Perioral dermatitis/rosacea-like eruptions**.
        - Hypertrichosis (overgrowth of hair).
        - Cataracts or glaucoma (if applied near eyes).
    - Systemic Corticosteroids:
        - Indications: Systemic vasculitis, severe drug eruptions (e.g., SJS/TEN, DRESS), severe urticaria with angioedema, widespread acute eczema.
        - **Generally NOT indicated for widespread chronic plaque psoriasis** (risk of rebound).
        - Uses in acne: May be used for severe inflammatory acne (e.g., acne fulminans) or severe acne medicamentosa. Not for ordinary or nodular acne vulgaris.
- **Phthirus Pubis (Pubic Lice)**
    - Transmission: Can be transmitted from pubic hair to **eyelashes (phthiriasis palpebrarum)**, axillary hair, chest hair. Less commonly to scalp hair.
- **Erythroderma (Exfoliative Dermatitis)**
    - Definition: Generalized erythema and scaling involving **>80-90% of the skin surface**.
    - Complications: **Hypothermia (due to heat loss)**, dehydration, electrolyte imbalance, secondary infection, high-output cardiac failure. Hyperthermia is NOT a feature.
    - Associations: Can be caused by pre-existing dermatoses (e.g., psoriasis, eczema), drugs, malignancies (e.g., Sézary syndrome, mycosis fungoides), congenital ichthyosis. **Lichen planus rarely causes erythroderma**.
    - Investigation: **Biopsy is usually done** to help determine the underlying cause.
- **Placental Transfer of Immunoglobulins**
    - **IgG is the only immunoglobulin that significantly crosses the normal placenta**.
- **Dermatoscope**
    - Uses: Examination of pigmented lesions (nevi, melanoma), alopecia areata (e.g., exclamation mark hairs), vascular patterns. It is a hand-held tool.
    - **NOT used for directly visualizing fungal hyphae and spores** (this requires KOH preparation and microscopy).
- **Cutaneous Leishmaniasis**
    - Transmission: Transmitted by the bite of infected **sandflies** (not mosquitoes).
    - Clinical presentation: Often a painless ulcer on exposed skin (e.g., face in a child from an endemic area like the Jordan Valley).
    - Causative species for Leishmaniasis recidivans: **L. tropica**.
    - Pathology: Infection of **reticuloendothelial (RE) cells (macrophages)**.

- Treatment: **Antimonials (e.g., sodium stibogluconate, meglumine antimoniate)** are drugs of choice for many forms.

- **Leprosy (Hansen's Disease)**
  - Eye involvement: Can occur in **intermediate and lepromatous leprosy**.
  - Treatment duration (Lepromatous): **Multidrug therapy is continued for at least 2 years (WHO recommendation), and often lifelong or until skin smears are consistently negative**, depending on guidelines and response.
- **Hyperpigmented Lesions**
  - Evaluation of a growing, hyperpigmented lesion on the face:
    - Initial step: **Dermoscopic examination**.
    - If suspicious: **Biopsy (excisional if possible for suspected melanoma, or incisional if very large)**. Chemical peeling is a cosmetic procedure and not for diagnosis of potentially malignant lesions.