Dermatology Summary

Summary For ABC of Dermatology

V 1.1

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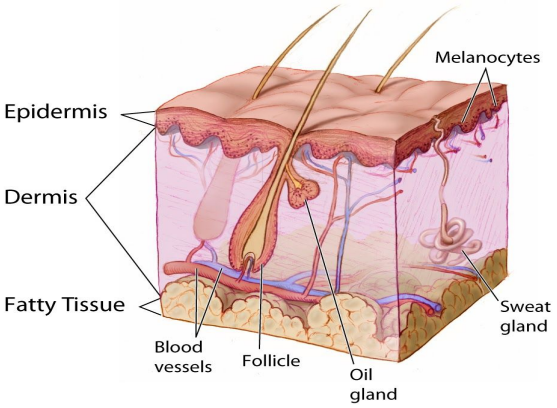
Summary for ABC of Dermatology → Ch 1-5, 7-10, 12-14, 16-17, 19-22
Additional topic commonly tested → STD, Ichthyosis, Hx and PE
This summary is for the theory exam and for the theory part of the mini OSCE (use pictures in the book also for the mini OSCE)

Normal skin

- The skin is the largest organ of the body, forming the outermost surrounding layer; separates the body from the surrounding environment
- Types of skin
- Glabrous skin:
    - Present on the palms and soles
    - Has thick keratin layer
    - Dermatoglyphics “skin markings or patterns”
    - Specific nerve organs
    - Lack of hair follicles or sebaceous glands
- Hairy skin:
    - Present all over the body except palms and soles
    - Wide variations according to anatomic site:
        - Scalp: large sebaceous glands and hair follicles
        - Axilla : large apocrine sweat glands
- Embryology
    - Epidermis → ectoderm
    - Dermis & SQ → mesoderm
    - Melanocytes → neural crest
- Functions
    - Protection,Heat regulation,Perception of sensation
    - Secretion of sebum, sweat,Pro-vitamin D synthesis
    - Mirror of the body because the skin act an organ of expression:
        - Anxiety - sweating.
        - Fear - pallor.
        - Anger -7 redness.
- Basic structure → 3 components
    - **Epidermis** – stratified squamous epithelium, consists of mainly **keratinocytes**
    - **Dermis–** forms the structural foundation of the skin, supporting its superficial + deep layers
        - The Dermis determines the thickness of skin, the epidermis thickness is the same in all body
    - **Subcutaneous-adipose layer**; acts as a fat + heat store



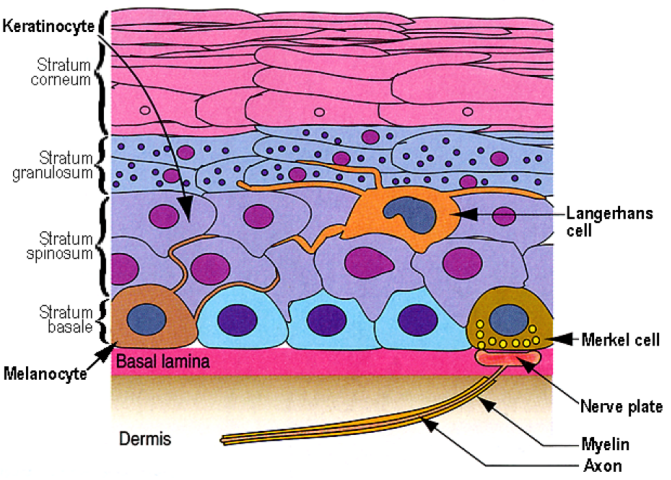
Epidermis → 4 major types of cells

- Keratinocytes
- Melanocytes
- Langerhans cells
- Merkel cell

Melanocytes → neural crest \ Langerhans cells ,Merkel cell → bone marrow

Keratinocytes → 4 layers

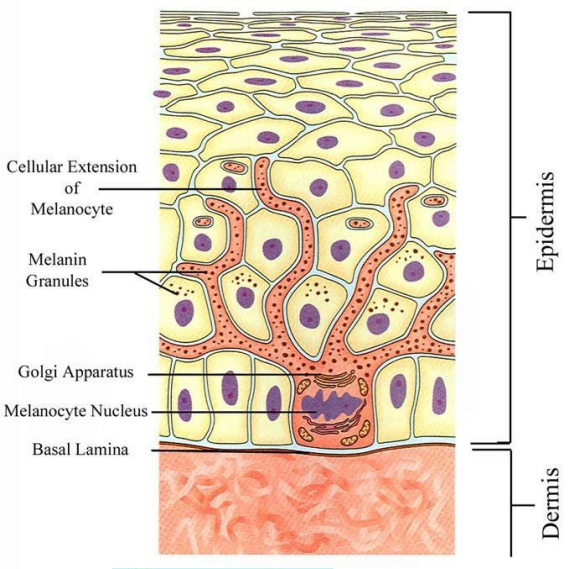
1. Basal cell layer
    a. 1st row of cells, columnar
    b. Mitotic activity restricted to these cells, and form the cells of other layers
    c. **Melanocytes** are found between the basal cells
2. Prickle cell layer (spinous, **squamous**):
    a. Polygonal cells
    b. Intercellular cement (**desmosomes**) and intercellular cement (**tonofilaments**)
3. Granular cell layer:
    a. Contain **keratohyalin** granules
4. keratin layer (S. **corneum**, horny layer):
    a. Results from keratinization → no nucleus in cells
    b. Function as a normal barrier



The Epidermis

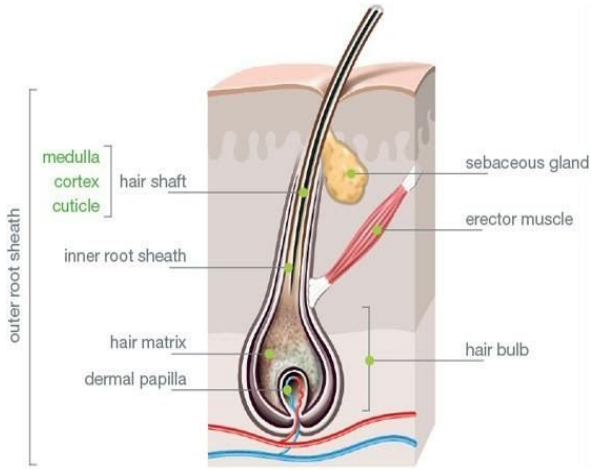
- Constituted by keratinizing, stratified squamous epithelium
- Keratinocytes form 95% of the epidermal cells; the other 5% form the melanocytes, Langerhans cells + Merkel cells
- The basement membrane has upward projections into the epidermis formed of the dermis called dermal papillae.

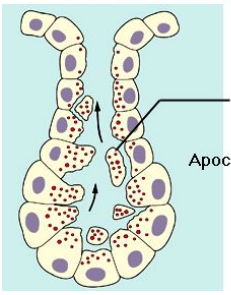
- The downward parts of the epidermis are called rete ridges
- Keratinocytes:
    - Cells that act as skin stem cells; they are located on the basal lamina
    - Here they divide, migrate upwards acquiring a large amount of cytoplasm and many desmosomes
- Melanocytes
    - Dendritic cells that form and secrets melanin by the mean of dendrites to the surrounding keratinocytes
- Langerhans cells
    - Dendritic Antigen presenting cells
    - Supra-basal layer
- Merkel cell
    - Bell-like shaped cells, at the basal layer
    - Free nerve endings below it, related to touch

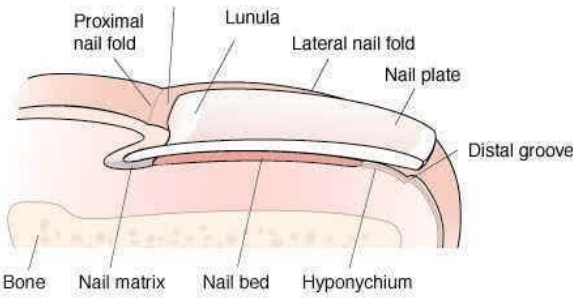


Epidermal appendages

- These are structures originating from the epidermis but present in the dermis:
- Keratinous structures: hair/ nail
- Glandular structures:
    - Apocrine sweat glands
    - Eccrine sweat glands
    - Sebaceous sweat glands





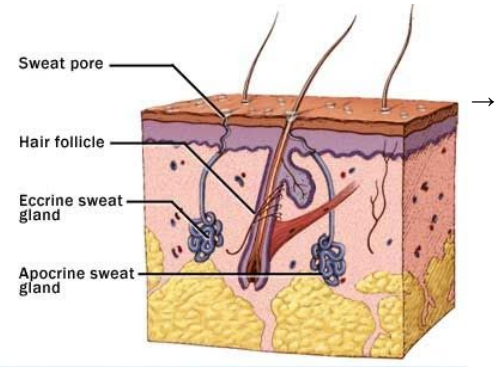


Apocrine sweat glands

- Large sweat glands, present at specific locations in the body e.g: axillae, groin..
- **Decapitation secretion**: the apical portion of the secretory cell of the gland pinches off and enters the lumen.
- Composed of a coiled secretory portion located at the junction of the dermis and subcutaneous fat, from which a straight portion inserts and secretes into the infundibular portion of the hair follicle

Eccrine sweat glands

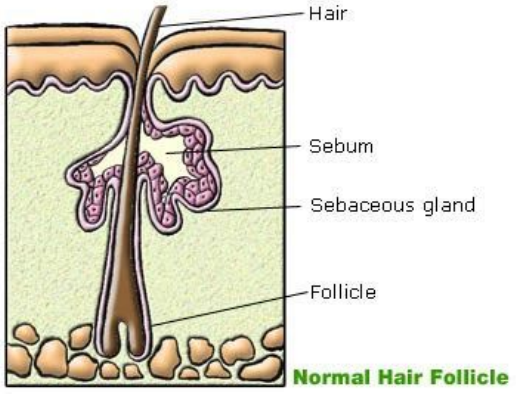
- Are the major sweat glands in the body
- Almost present everywhere on the human body
- Produce clear, odorless fluid containing mainly water and electrolytes **Merocrine** secretion
- Eccrine glands are composed:
    - intraepidermal spiral duct, the "acrosyringium";
    - dermal duct, comprising a straight and coiled portion
    - secretory tubule, coiled deep in the dermis or hypodermis.
- Eccrine glands are innervated by the **sympathetic** nervous system, primarily by cholinergic fibers



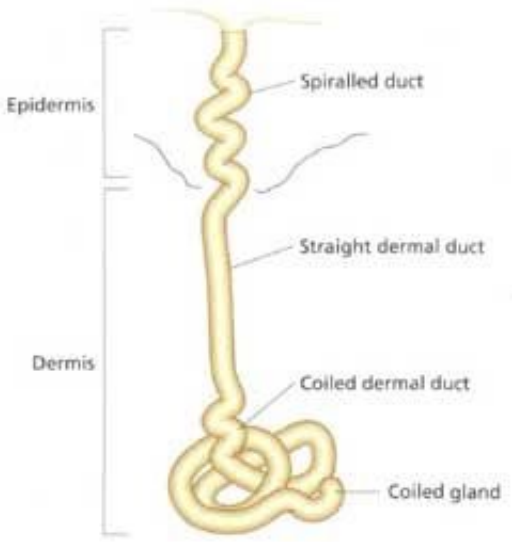
Sebaceous glands

- Related to hair follicles, and their ducts open in it.
- Present all over the body except the palms and soles.
- **Holocrine** secretion.

- Secretion controlled by **androgens** mainly.



Normal Hair Follicle



Dermis

1) Connective tissue fibers: collagen, elastin, reticulin
2) Ground substance of mucopolysaccharides
3) Cellular: fibroblasts, fibrocytes, mast cells
4) Nerves:
   - Sensory: **Messner**, **Pacinian**, Muco-cutaneous n. organs,Free nerve endings
   -Autonomic
5) blood vessels: superficial and deep
6) Muscles → voluntary (skeletal) & involuntary (smooth)
7) Lymphatics

Meissner corpuscle

- Mediates touch
- In the papillary dermis
- More at the tips of fingers

Pacinian corpuscle

- Deep in dermis
- Mediates pressure and vibration
- Onion shaped
- Palms, soles, areola and genitalia

Psoriasis

- Characterised by plaques of diseased skin often at sites of minor trauma (elbows/knees), which occur next to areas of clear ‘normal’ skin.
  - well-demarcated and are erythematous with white surface scale
- Associated with seronegative arthritis, type 2 diabetes ,cardiovascular disease.
- Pathophysiology
  - Genetic predisposition + Precipitating factors “Stress, infection, local trauma Alcohol, drugs,childbirth.”
  - Hyperkeratosis “increased thickness + Thick keratin scale ”, parakeratosis ”keratocytes retain their nuclei”, poorly adherent and easily scraped off keratocytes (‘Auspitz sign’)
  - Vasodilation, angiogenesis → Inflammation → swelling (oedema) and erythema
  - sterile pustules “accumulation of inflammatory cells” → in palmo-plantar pustulosis
- Psoriatic nail dystrophy
  - onycholysis,subungual hyperkeratosis, pitting, Beau’s lines (transverse lines, groove), splinter hemorrhages (longitudinal black lines)
- Clinical appearance
  - Plaques → well-defined raised areas ,large or small, few or numerous and scattered over the trunk and limbs
  - Scaling
  - Erythema or redness
    - erythrodermic psoriasis (who have >90% of their body surface involved).
  - Pustules → esp. In palmo-plantar pustulosis
- Clinical presentation
  - Lesions improve in the sun, and are mildly itchy

    - Distribution → elbows, knees and scalp \\ trunk “annular” \\ palms and plantar surface of foot
        - At minor skin trauma – Koebner’s phenomenon.
- Types
    - plaque psoriasis.
    - Guttate → widespread small plaques scattered on the trunk and limbs, preceded by sore throat with GBS
    - Inverse.
    - Palmo-plantar pustular psoriasis (PPPP) → yellow then brown
    - Acute generalised pustular psoriasis (Von zumbusch) → severe and unstable, precipitated by systemic steroids
    - Acropustulosis → around the nails and the fingertips
    - Flexural psoriasis → axilla, groin, natal cleft, beneath the breasts and in skin folds. No scaling
    - Napkin psoriasis → exudative
    - Erythrodermic → all skin, no scaling, triggers → steroid withdrawal, infection, alcohol, antimalarials, lithium and low calcium
        - Complications → heart failure, hypothermia, dehydration
    - Psoriatic arthritis.
        - Five types
            - **DIP** joints (80% have nail changes, enthesitis not synovitis)
            - **Asymmetrical oligoarticular** arthropathy (MC type, hands and feet, ‘sausage-shaped’ digits)
            - **Symmetrical polyarthritis** (hands, wrists, ankles, ‘rheumatoid pattern’, female predominance)
            - Arthritis **mutilans** (digits, resorption of bone → ‘telescoping’ of redundant skin)
            - Spondylitis (asymmetrical vertebral involvement,male preponderance, HLA-B27 associated)

Management of Psoriasis

- The treatment ladder starts with avoidance of known exacerbating factors (smoking, alcohol), topical therapy, then Ultraviolet treatment (phototherapy and photochemotherapy) and finally systemic medication (tablet, S/C, IV).
- In general, combination therapy is more effective than monotherapy, and change of therapy is superior to continuous usage.
- Combination products→ containing salicylic acid, vitamin D, tar and antibiotics.
- Systemic corticosteroids should not be used to treat psoriasis
- **Topical treatment**
    - Emollients “Moisturizer”
    - Coal tar → keratoplastic,antipruritic,antimicrobial ⇒for stable chronic plaque
        - Used in combination with salicylic acid for thick plaques
    - Ichthammol (ammonium bituminosulfonate) → anti-inflammatory ⇒ for unstable’ or inflamed psoriasis
    - Dithranol (anthralin, Goa powder)
    - Calcipotriol and tacalcitol, vitamin D analogues, are calmodulin inhibitors → for mild or moderate plaque psoriasis.
    - Corticosteroids “topical” → anti-inflammatory
        - relapse usually occurs on cessation and tachyphylaxis “tolerance”
        - Mild/moderate topical steroids → face and flexural skin, and in erythrodermic disease
        - Moderate or potent → chronic stable plaques on the body.
- Scalp psoriasis
    - combinations of tar, salicylic acid, sulphur and emollient
    - steroid/salicylic acid/vitamin D-containing scalp applications/gels
- **Ultraviolet treatment** – phototherapy and photochemotherapy
    - For extensive disease not cleared with topical therapy
    - Contraindications
        - history of previous skin malignancy and photosensitive diseases such as lupus,porphyria, albinism and xeroderma pigmentosum
    - Types
        - **phototherapy** “short wavelength” → **Broadband** ultraviolet B (UVB) and **narrowband** ultraviolet B (TL01;more effective) ⇒ used in children and preg.
        - **photochemotherapy** “long wavelength”→ ultravioletA plus psoralen (PUVA)
            - in combination with oral or topical psoralen
            - for recalcitrant widespread thick plaque psoriasis

- **Systemic** treatment
    - Indications → unstable inflamed psoriasis,widespread disease that have failed to respond to topical/phototherapy, psoriatic arthropathy.
    - first-line systemic → Acitretin ,Cyclosporin A,Methotrexate
    - 2nd line → Mycophenolate mofetil (MMF), hydroxyurea, azathioprine
    - Agents
        - Methotrexate
        - Acitretin “vitamin A derivative”
        - Cyclosporin A “immunosuppressant”
        - Mycophenolate mofetil (MMF), hydroxyurea, azathioprine
- Biological therapy
    - Agents
        - anti-TNF→ Etanercept ,Infliximab, Adalimumab
        - Ustekinumab (Anti (IL-12) and IL-23))

This table is **NOT** important for the OSCE

drug	indication	Adverse effect	monitoring	other
Methotrexate	-unstable erythrodermic/ pustular Psoriasis → acute and maintenance -psoriatic arthritis	-hepatotoxic -Myelosuppression	- LFT procollagen III “liver fibrosis” -FibroScan -CBC	Give Folic acid supplements -reduce dose in renal impairment
Acitretin	chronic plaque psoriasis synergistic effect with PUVA	-mucocutaneous symptoms -Hepatotoxicity and raised lipid -teratogenic	-LFT and lipid profile	Contraception during tx and for 3 years
Cyclosporin A		-renal impairment and hypertension -gum hypertrophy and hypertrichosis	-KFT	
Mycophenolate mofetil (MMF)		-gastrointestinal upset and myelosuppression, haematological malignancies and opportunistic infections		
Biological therapy	1.PASI>10,DLQI score >10, 2.high-impact sites – hands/genitals/scalp 3. failed systemic agents/phototherapy(contraindicated,non-response or stopped due to unacceptable side effects) 4. In psoriatic arthritis → at least three tender and swollen joints	-Infections → TB, heb b, septicaemia, -gastrointestinal symptoms, hypersensitivity -blood disorders -lupus-like antibody driven syndrome		continuous therapy is more effective
Etanercept	anti-TNF			Onset is slow
Infliximab	anti-TNF			rapid onset,80% effective
Adalimumab	anti-TNF			rapid onset,70% effective
Ustekinumab	(Anti (IL-12) and IL-23))			

Eczema (Dermatitis)

Definition

- Eczema includes atopic dermatitis, contact allergy, varicose eczema, pompholyx and discoid eczema
- Inflamed, dry, occasionally scaly and vesicular skin rashes

Clinical features

- Acute or chronic
    - Acute → erythema, vesicular/bullous lesions and exudates
        - Golden crusting → staph or strep
    - Chronic → scaling, xerosis (dryness) and lichenification
- Itchy → scratching → excoriation marks, loss of skin surface, secondary infection, exudates and **lichenification** (thickening of skin where surface markings become more prominent).
- Post-inflammatory hyper/ hypo pigmentation

Types

- Endogenous eczema (constitutional)
    - Atopic dermatitis "AD"
        - Facial then flexural limb, onset → Childhood
        - 90% of the cases spontaneously remit by puberty
        - Poor prognostic factor (AD continue to adult life)
            - strong family history, present at a very young age with extensive disease and have associated asthma/multiple food allergies
        - Symptoms→ Itchy, red, swollen, cracked skin
            - Intensely itchy → poor feeding → failure to thrive
        - Exacerbating factors → stress, infections, teething, food allergies,Skin Irritation,Climate, increased Sweating, House dust mite,vaccination
        - Associated asthma/multiple food allergies
        - **Pityriasis alba** → pale patches of hypopigmentation on the face
        - **Juvenile plantar dermatosis**→ dry cracked skin on the forefoot in children
    - Discoid eczema → S.Aureus
    - Pompholyx eczema → painful itching vesicles on the fingers, palms and soles
    - Venous (stasis) eczema → lower legs , venous insufficiency, brown hemosiderin pigmentation esp. on the medial ankle+ulcers, Tx→ compression.
    - Seborrhoeic eczema
    - Secondary changes
        - Eczema herpeticum → HSV, Tx → aciclovir
        - Lichen simplex
        - Asteatotic eczema → older people with dry skin
    - Unilateral eczema of the areola → DDx;Paget's disease of the nipple
    - Investigations of eczema
        - Skin swabs, Nasal swabs, scrapings (if fungal infection is suspected)
        - Eosinophilia and raised IgE
        - Skin prick, RAST (radioallergosorbent testing)→ to determine specific allergies
        - Skin biopsy
        - ABPI (ankle brachial pressure index) → Varicose eczema
- Exogenous (external factor)
    - Contact dermatitis
        - **Allergic** contact dermatitis
        - **Irritant** contact dermatitis

	Irritant contact dermatitis	**Allergic** contact dermatitis
People at risk	Anyone, esp if repeated exposure	Genetically predisposed + previously sensitized
Mechanism	Direct tissue trauma → chemical or physical irritant.	Type IV delayed hypersensitivity
Previous contact	Not required	Required

Conc of agent	High, dose related,severity varies with the quantity, concentration and length of exposure	May be low, threshold dose; all or nothing
Risk if atopic	Increased	Decreased
Histology “skin infiltration”	Neutrophils	Lymphocytes
Symptoms	Itchy or burning	Intensely itchy
Morphology	Erythema, Slight scaling, and fissuring	Erythema, oedema and vesicles. Lichenified (if chronic).
Demarcation + Distribution	Sharp, limited to area of exposure	Sometimes sharp -Activation of previously sensitised sites at a distant skin site
Onset	No predictable time interval between contact and dermatitis Typically → minutes to hours	48–96 h between contact and dermatitis -Typically → hours to days -Persistence of the allergy
Dx test	Patch test -ve	Patch test +ve

    - **Photodermatitis** →interaction of light and chemicals (topical or systemic drugs)
    - **Occupational** dermatitis
        - Can be Allergic or Irritant (acute or chronic)
    - **Contact urticaria**
        - Immediate-type sensitivity reaction to certain food proteins and latex glove allergy
    - Investigations of contact dermatitis
        - Patch testing

General **management** of eczema

- Emollients (moisturising creams) → for dry skin
- Cleansers →
    - Normal soap not used
- Topical steroids → mainstay of treatment for active eczema
    - Applied once or twice daily to the affected skin only.
    - Ointments are better than creams
    - Mild eczema or face and groin → Very low potency steroids (hydrocortisone)
    - Moderate to severe → potent topical steroids (betamethasone)
- Immunomodulators “calcineurin inhibitors“→ Tacrolimus
- Occlusion→ Covering topical therapy with bandages, ‘wet-wraps’ and dressings
- Antibiotics → topical or systemic , for infected eczema
- Phototherapy → for generalised eczema
- Systemic therapy “immunosuppressants”
    - Oral prednisolone → for acute management
    - Azathioprine, cyclosporine,mycophenolate mofetil and methotrexate → for long-term management

Classification of eczema

Endogenous (constitutional) eczema	Exogenous (contact) eczema	Secondary changes
Atopic	Irritant	Lichen simplex
Discoid	Allergic	Asteatotic
Pompholyx	Photodermatitis	Pompholyx
Varicose		Infection
Seborrhoeic (discussed later)		

Pruritus

Definition

- Itching of the skin that is an unpleasant sensation that triggers rubbing or scratching
- Localised or generalised, with or without skin changes

Pruritus with skin changes

- Localised pruritus
    - Eczema, psoriasis, lichen planus , dermatitis herpetiformis , insect bites/stings, head lice, contact dermatitis, polymorphic light eruption, urticaria or angioedema, fungal infections (particularly tinea pedis of the feet), pruritus ani and pruritus vulvae.

- Generalised pruritus
    - Widespread eczema/psoriasis, scabies, allergic drug eruptions , graft versus host disease (following bone marrow transplantation), pre-bullous pemphigoid , cutaneous lymphoma , body lice or pubic lice, viral exanthems , urticaria (generalised 'hives') and xerosis (dry skin)

Pruritus with normal skin

- Endocrine – diabetes, hyperthyroidism, menopause and pregnancy.
- Metabolic – hepatic failure, biliary obstruction and chronic renal failure.
- Haematological – polycythaemia and iron deficiency anaemia.
- Malignancy – lymphoma, leukaemia, myeloma and carcinomatosis.
- Neurological/psychological – neuropathic pruritus, multiple sclerosis and anxiety.
- Infection – filariasis, hookworm and HIV.
- Drugs – opioids.

Investigations (pruritus with normal skin)

- CBC, ESR, LFT,KFT, TFT
- Serum iron /// Fasting glucose

Management

- Simple soothing emollients→ Menthol
- Topical local anaesthetics (ex→ lidocaine)
- Topical and systemic antihistamines → H1 blockers

Urticaria and Angioedema

Introduction

- **Urticaria** transient **pruritic** swellings of the skin, often referred to as wheals, hives
    - edema in the **superficial** layers,well-demarcated erythematous lesions
    - Common, self limiting, controlled with antihistamine.
    - Onset → Minutes to hours, lasting minutes or hours (usually less than 24 h)
    - Primary lesion → wheal surrounded by flare
- **Angioedema** → **painful** rather than itchy
    - diffuse swelling "edema " in **deeper** layers of the skin (subcutaneous); can occur rapidly and may involve the mucous membranes. Laryngeal oedema is the most serious complication.
    - Onset → Minutes to hours, last hours or days.
    - may occur in the face "eyelids, lips and tongue", larynx, abdomen, or arms and legs
- Pathophysiology → histamine & bradykinin from mast cells "degranulation" ⇒ vessels leak and dilatation → edema
- urticarial vasculitis → skin eruption for more than 24h, painful and resolves with bruising
- Ask about associated respiratory distress, triggers (food "N\V", heat/cold, sun, medications, insect stings,animal contact, physical stimuli, infections, family history

Classification of urticaria

- Acute (<6 wks), chronic (>6 wks) or according to the underlying cause
- Ordinary urticaria /idiopathic (acute/chronic)
    - MC, Blanchable, raised, palpable wheals, which can be linear, papular,annular (circular), or arcuate (serpiginous)
    - Possible triggers → infections, vaccinations,medications and food
- Cholinergic urticaria → following warm shower, or after exercise, rarely after cold exposure
    - pinhead-sized wheals with a red flare around them.
- Solar urticaria → stinging, burning and itching cause by sunlight exposure, resolve rapidly (minutes to hours) when exposure ceases
    - DDx → Photosensitive drug eruptions, porphyria , polymorphic light eruption (resolve within days)
    - Dx → Light-testing
- Pressure urticaria → immediately or delayed up to 6 h, Dx → pressure challenge test
- Angioedema
    - Hereditary angioedema → deficiency in C1 (esterase) inhibitor.Serum complement C4 levels are low following attacks
- physical urticaria
    - Causes → Heat. Sunlight. Cold. Pressure. Water.
    - +ve dermatographism
- urticarial vasculitis
- contact urticaria

- non-physical urticaria causes
    - Food allergies or additives, Infections, Salicylates, Contact urticaria, Papular urticaria (insect bites)

General investigations

- Hx+PE
- Dermatographism → physical urticaria
- skin biopsy → urticarial vasculitis
- RAST (radioallergosorbent test) or skin prick testing (not in anaphylaxis)
- patch testing→ contact urticaria
- C3 level and C1 esterase → low in Hereditary angioedema
- pressure challenge test
- heat or cold challenge test

General management

- Avoid trigger
- Oral antihistamines → mainstay of treatment/prevention
- Severe resistance cases
    - H1-receptor blockers + H2-receptor blockers +leukotriene receptor antagonists (montelukast)
    - Oral corticosteroids → urticarial vasculitis
- angioedema with respiratory distress → adrenaline intramuscularly “epipen”

Drug Rashes

Intro

- Cutaneous adverse reactions account for a third of all adverse reactions to drugs
- Drugs can cause adverse reactions in several ways: by changing **normal skin function**, by **exacerbating** an existing dermatosis, by causing an **idiopathic** dermatosis such as urticaria, by causing a **specific drug eruption** (lichenoid drug rashes) or by precipitating a **severe drug reaction** (toxic epidermal necrolysis).
- Tx → identification and withdrawal of the culprit medication
- Hx → Eliciting the temporal association of the ingestion of the drug and the onset of the eruption is key, drugs within 3 months prior to rash , previous exposure to suspected culprit drugs
- PE → morphology of the rash, distribution, mucosal – eyes, mouth, genitalia –involvement
- Investigations → skin biopsy for confirmation of Dx, blood tests (white cell counts and CRP levels) for exclusion of infection ,IgE, patch testing

Classification

- immune-mediated or non-immune mediated
    - **Immune-mediated** rashes → most common ,include hypersensitivity reactions from types I to IV.
        - **Type I** reactions(immediate reactions, mediated by **IgE** or drug-specific receptors bound to **mast cells**) tend to manifest in the skin as **urticaria** or **angioedema**.
        - **Type II** reactions (**cytotoxic** reactions) result in **cutaneous** purpura.
        - **Type III** (**immune complex**-mediated) reactions lead to **cutaneous vasculitis.**
        - **Type IV** **delayed hypersensitivity** reactions are by far the **most common** , resulting in generalized exanthems, phototoxic rashes and severe drug reactions such as toxic epidermal necrolysis (TEN).
    - **Non-immune-mediated** rashes include accumulation of medications in the skin (causing pigment changes), instability of mast cells (causing histamine release), slow acetylators (metabolism of drugs affected) and photosensitivity reactions (increased susceptibility to UV light).
- Clinically
    - **alter normal** skin function.
    - **exacerbate** an existing dermatosis.
    - **Common** drug-induced rashes – maculopapular exanthem, urticaria, vasculitis, lichenoid drug reaction and fixed drug eruption.
    - **Severe** drug-induced rashes – Stevens–Johnson syndrome (**SJS**) and **TEN**, acute generalised exanthematous pustulosis (**AGEP**) and drug reaction with eosinophilia and systemic symptoms (**DRESS**).

Drugs which alter normal skin function

Photosensitivity

- **Phototoxic reactions** → more common, sunburn, may blister , confined to light-exposed sites and have sharp demarcation between covered and uncovered skin , Rapid onset and recovery on withdrawal (hours)
- Photoallergic reactions → eczematous,delayed onset and recovery on withdrawal (wks-months)

Pigmentation

- OCP → melasma

- facial blue-black pigmentation → amiodarone.
- slate-grey pigmentation → Tetracycline

Skin reaction	Drugs
Phototoxic Rxn	**Amiodarone**, **NSAIDs**, **tetracyclines**, chlorpromazine
Photosensitive Rxn	**Amiodarone**, **tetracyclines**, calcium channel blockers, diuretics, voriconazole, itraconazole, terbinafine, ritonavir, saquinavir
Photoallergic Rxn	**NSAIDs**, antibiotics, thiazides, anticonvulsants, allopurinol, quinolones, nelfinavir
Pigmentation changes	Chlorpromazine, phenytoin, hydroxychloroquine, cyclophosphamide, bleomycin, **amiodarone**, clofazimine, minocycline, mepacrine
Urticaria/angioedema	**NSAIDs**, opioid analgesics, ACE inhibitors, antibiotics, anti-retrovirals (nelfinavir/zidovudine), infliximab, PPI, IV contrast media
Drug-induced lupus Esp. sub-acute cutaneous lupus (SCLE)	**Terbinafine (MC)**, hydralazine, procainamide, quinidine, isoniazid, diltiazem, and minocycline
Drug-induced vasculitis	Antibiotics, **NSAIDs**,Anticonvulsants (ex phenytoin), ramipril, PPI , allopurinol, thiazides, adalimumab, indinavir
Lichenoid drug eruption	Gold, mepacrine, tetracyclines, diuretics, CCB(ex;**amlodipine)**, carbamazepine, propranolol, **NSAIDs**, ACE inhibitors, PPI , statins
Erythema nodosum	Oral contraceptives, antibiotics, gold, sulphonylurea
Fixed drug eruption	Antibiotics, **NSAIDs**, oral contraceptive, barbiturates
SJS/TEN	Antibiotics, anticonvulsants, **NSAIDs**, anti-retrovirals (indinavir/saquinavir) , allopurinol , barbiturates, ramipril, diltiazem
DRESS	**Allopurinol**, **anticonvulsants**, antibiotics, anti-retrovirals, imatinib (Gleevec), **NSAIDs**, ACE inhibitors, calcium channel blockers, terbinafine
AGEP “Acute generalized exanthematous pustulosis”	**Antibiotics** “MC”, anticonvulsants, antitubercular medications
Antibiotics: most commonly sulfonamides, penicillins, ampicillin, tetracyclines and vancomycin. **Anticonvulsants**: most commonly phenytoin, carbamazepine, sodium valproate and lamotrigine. **Calcium channel blockers**: most commonly diltiazem, nifedipine and amlodipine. **NSAIDs**: most commonly aspirin and ibuprofen	

Excessive hair

- Hypertrichosis is the growth of hair at sites which are not normally hair-bearing; hirsutism is excessive growth of hair in the male pattern of hair growth, especially in women.
- MC → cyclosporine and phenytoin

Hair loss

- Cytotoxic agents interrupt the anagen (‘growth’) phase of the hair cycle, and so loss is rapid and complete; delayed, insidious hair loss generally results from interference with the telogen (‘shedding’) phase of the hair cycle.
- acitretin, statins and anti-thyroid drugs, Androgenic drugs “testosterone”

Nails

- Discoloured → mepacrine or hydroxyurea
- Onycholysis → cytotoxic agents.

Drugs which exacerbate pre-existing dermatoses

- Psoriasis → beta blockers, lithium and antimalarial medications
- Eczema → statins and diuretics
- Acne → OCP, particularly progesterone-only pills, Corticosteroids, cyclosporine and anti-epileptics such as phenytoin
- Urticaria → NSAIDs, ACE inhibitors and angiotensin receptor blockers

Common drug-induced rashes

Drug-induced exanthems

- most common cutaneous reaction to a drug is an exanthem, meaning a widespread rash.
- burning, itch or discomfort

- Onset is typically within 7–10 days " delayed-type hypersensitivity", subsequent reactions → faster "memory T cells"
- If it exceeds 90% → erythrodermic.
- MC → Abx,antihypertensive agents and cholesterol-lowering drugs

Urticaria/angioedema

- raised, red itchy weals, if in head or neck → angioedema
- anaphylaxis, occurring rapidly after drug ingestion (type I drug hypersensitivity) or delayed by a number of days (type IV hypersensitivity).

Drug-induced lupus

- Antihistone antibodies are present in >95% of cases, but dsDNA is usually negative and complement levels are normal

Lichenoid drug eruptions

- resemble idiopathic lichen planus, but may not be confined to the classic sites of predilection of the latter
- Resolution following drug withdrawal can be slow and take up to 2 months, and there is post-inflammatory hyperpigmentation

Fixed drug eruption

- This is peculiar phenomenon whereby one or more inflammatory patches appear at the same cutaneous or mucosal site on each occasion that the patient ingests a culprit drug
- Develop within 2 to 24 h.

Severe drug reactions in the skin

Stevens–Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN)

- life-threatening drug-induced hypersensitivity reactions in the skin and mucous membranes
- The terms SJS and TEN represent points along a spectrum of severity,
    - SJS → <10% BSA detachment,
    - TEN → >30% BSA,
    - 'SJS–TEN overlap' →between 10% and 30% loss.
- Mortality from TEN may be as high as 90% (score >5) and is estimated using the SCORTEN tool
- Tx → stop the drug and supportive care and intravenous immunoglobulin or corticosteroids

Drug reaction with eosinophilia and systemic symptoms (DRESS)

- Rash, head and neck oedema, fever, lymphadenopathy, eosinophilia and involvement of one or more solid organs (usually the liver)
- latency period → 15–60 days. For this reason, the diagnosis is often overlooked, and symptoms of rash, fever and lymphadenopathy attributed incorrectly to infection
- Tx → corticosteroids oral or IV

Bullous diseases

Intro

- Blisters "large bullae or small vesicles" arise from the destruction or separation of epidermal cells by trauma "burn", viral infection "herpes", immune reactions, edema as in eczema or inflammatory causes such as vasculitis.
- Immune reactions at the dermo-epidermal junction (pemphigoid) and intraepidermally (pemphigus) cause blisters.
- Pathophysiology
    - Bullous pemphigoid → IgG autoantibodies that target the basement membrane cells (hemidesmosome proteins)
    - Pemphigus Vulgaris → autoantibodies directed against desmosomal cadherin desmoglein 3 (Dsg3)
    - Pemphigus foliaceous (PF) → desmoglein 1 (Dsg1)
    - DH → IgA deposits in the papillary dermis, IgA against dietary gluten
- Susceptibility is inherited; trigger factors include drugs, foods, viral infections, hormones, and ultraviolet radiation.

DDx

- Bullous pemphigoid, pemphigus, dermatitis herpetiformis (DH) and linear IgA.
    - Superficial blisters → sloughs off easily "rupture"
        - pemphigus vulgaris
    - Subepidermal blisters → stronger roof and usually remain intact
        - bullous pemphigoid, linear IgA and erythema multiforme
    - Distribution
        - Widespread → Bullous pemphigoid, Pemphigus vulgaris, DH, Erythema multiforme

        - Localised → DH, Pemphigus gestationis (abdomen), PF (upper trunk, face & scalp), Porphyria (sun-exposed sites), Pompholyx eczema (hands & feet).
- Other causes of cutaneous blistering.
    - Erythema multiforme
    - Stevens-Johnson syndrome/toxic epidermal necrolysis
    - Chickenpox /// Herpes simplex/varicella-zoster virus
    - Staphylococcus impetigo
    - Insect bite reactions
    - Contact dermatitis /// Phytophotodermatitis
    - Porphyria //// Fixed drug eruption

Clinical features

Table 8.2 Clinical features of immunobullous disorders.

Immunobullous disorder	Typical patient	Distribution of rash	Morphology of lesions	Mucous membrane involvement	Associated conditions
Bullous pemphigoid	Elderly	Generalised	Intact blisters	Common	None
Mucous membrane pemphigoid	Middle aged or older	Varied	Erosions, flaccid blisters, scarring	Severe and extensive	Autoimmune disease
Pemphigoid gestationis	Pregnant	Periumbilical	Intact blisters, urticated lesions	Rare	Thyroid disease
Pemphigus vulgaris	Middle aged	Flexures, head	Flaccid blisters, erosions	Common	Autoimmune disease
Dermatitis herpetiformis	Young adults	Elbows, knees, buttocks	Vesicles, papules, excoriations	Rare	Small bowel enteropathy (gluten-sensitive), lymphoma
Linear IgA	Children and adults	Face and perineum (children) Trunk and limbs (adults)	Annular urticated plaques with peripheral vesicles	Common	Lymphoproliferative disorders

Bullous pemphigoid

- on a background of dermatitis or normal skin
- Prolonged prebullous period
- flexural sites on the limbs and trunk.
- Mucous membrane involvement (20%)
- Blisters heal without scarring
- If in children → after vaccination, face, palms and soles

Mucous membrane pemphigoid (cicatricial pemphigoid)

- painful sores, ulceration that heals with scarring
- Cutaneous lesions (30% of patients)
- Scalp → scarring alopecia
- Ocular → symblepharon, synechiae, lacrimal duct fibrosis (dry eyes),fixed globe, blindness.

Pemphigus Vulgaris

- acute, widespread, painful
- oral lesions (70%),
- Mucous membrane involvement may precede cutaneous signs
- rubbing apparently normal skin causes the superficial epidermis to slough off (Nikolsky sign positive).
- Paraneoplastic pemphigus → non-Hodgkin's lymphoma or chronic lymphocytic leukemia

Pemphigus foliaceus (PF)

- middle age, flaccid small bullae on the trunk, face and scalp that rapidly erode and crust
- May be drug induced

Dermatitis herpetiformis (DH)

- intensely pruritic autoimmune blistering
- Most patients do not report any bowel symptoms unless prompted but may experience bloating and diarrhea
- small bowel investigation reveals abnormalities (villous atrophy, raised lymphocyte count) in 90% of patients. Increased risk of small bowel lymphoma
- Tx →strict lifelong gluten-free (no wheat,rye & barley),if failed → Dapsone & sulphapyridine

Linear IgA

- acute onset of Intact blistering to insidious pruritus before chronic tense bullae
- Mucous membrane involvement
- around the periphery of annular lesions ('string of beads sign') or in clusters ('jewel sign').
- Children → lower abdomen and perineum
- adults→ limbs and trunk

- Tx → as DH

Investigation

- direct immunofluorescent analysis of perilesional skin. → gold standard
- Skin biopsies → lesional part(histopathology), adjacent skin(immunofluorescence)

Table 8.3 Skin biopsy findings in immunobullous disorders.

Immunobullous disorder	Histology features	Immunofluorescence features
Bullous pemphigoid	Subepidermal blister containing mainly eosinophils	Linear band of IgG at the basement membrane zone
Pemphigoid gestationis	Subepidermal blister containing mainly eosinophils	Linear band of C3 at the basement membrane zone
Mucous membrane pemphigoid	Subepidermal blister with variable cellular infiltrate	Linear band of IgG/C3 at the basement membrane zone
Pemphigus vulgaris	Suprabasal split (basal cells remain attached to basement membrane, looking like 'tombstones')	IgG deposited on surface of keratinocytes in a 'chicken-wire' pattern
Dermatitis herpetiformis	Small vesicles containing neutrophils and eosinophils in the upper dermis	Granular deposits of IgA in the upper dermis (dermal papillae)
Linear IgA	Subepidermal blisters with neutrophils or eosinophils	Linear deposition of IgA at the basement membrane zone

Mgt

- If tense → deflated using a sterile needle
- Liquid paraffin for eroded areas
- topical treatment, immunosuppressive drugs (potent topical steroids, systemic corticosteroids, methotrexate,rituximab, azathioprine)
- pemphigoid gestationis → high doses of systemic corticosteroids
- Mucous membrane pemphigoid
    - Oral → topical steroids and tetracycline mouthwashes
    - Ocular → Topical steroid drops and mitomycin, systemic immunosuppression
- pemphigus vulgaris → rituximab "anti-CD20"
- Gluten-free diet

Connective Tissue Disease, Vasculitis

Investigations might include the following

1. Hematology
    - 1.1. Complete blood count (CBC) /// ESR or CRP
    - 1.2. Factor V Leiden, antithrombin III, proteins S and C
    - 1.3. Coagulation screen, lupus anticoagulant
2. antibodies
    - 2.1. Antinuclear antibodies (ANAs)
    - 2.2. Extractable nuclear antibodies (ENA), (Ro, La)
    - 2.3. antineutrophil cytoplasmic antibodies (ANCA)
    - 2.4. Streptococcal serology (ASOT)
    - 2.5. Rheumatoid factor /// Antiphospholipid antibodies,Anticardiolipin antibodies
3. KFT+LFT
    - 3.1. Urine dipstick and microscopy
    - 3.2. Angiotensin-converting enzyme (ACE)
    - 3.3. Blood pressure
    - 3.4. Hepatitis serology
4. TFT, serum glucose, creatinine kinase
5. Chest X ray or lung CT, lung function tests
6. skin biopsy

Cutaneous Vasculitis

- **Symptoms** → pain in the skin, general malaise, fever, abdominal pain, weight loss , neuropathies and arthropathy.
- **Morphology** → non-blanching skin eruption and erythema (mostly in lower limbs) .
    - macular or palpable purpura, blistering, ulcerated and necrotic
- Possible causes
    - Drug hypersensitivity
    - Hepatitis,Endocarditis,Inflammatory bowel disease
    - Connective tissue disease
    - Coagulopathies /// Behçet's syndrome /// Kawasaki disease

    - Sarcoidosis
- Dx → skin biopsy for histology and immunofluorescence (IMF)
- Tx → topical steroids (mild-moderate cases),systemic steroids (severe cases)

Polyarteritis nodosa (PAN)

- systemic vasculitis of small- to medium-sized arterioles → MC in skin and joints
- +ve ANCA
- Morphology → subtle lacy/mottled (net-like) pattern (livedo reticularis), purpura, tender subcutaneous nodules, ulceration and necrosis, particularly on the lower limbs
- Investigations → angiography and tissue biopsy
- Management→ oral steroids with the addition of cyclophosphamide in severe cases

Henoch–Schönlein purpura

- preceding upper respiratory tract symptoms and a positive ASOT.
- The skin, kidneys (IgA nephropathy,haematuria), GI tract (abdominal pain) and joints are mainly affected.
- Dx→ Skin/renal biopsy → deposition of IgA
- Tx → supportive, resolve spontaneously (if not , systemic steroids are used , which do not treat renal disease)

Raynaud's phenomenon

- If with systemic disease → Raynaud's disease
    - systemic sclerosis, mixed connective tissue disease (MCTD), SLE and cryoglobulinemia
- cold exposure → white (vasospasm), then blue (cyanosis) and finally red (hyperemia)
- Distribution → mostly fingers → bilateral and symmetrical
- Tx→ keep warm, nifedipine and iloprost (prostacyclin analogue).

Systemic sclerosis (SSc)

- Extensive sclerosis (collagen deposition and fibrosis) of the subcutaneous tissues in the fingers and toes "tethering of skin " + around the mouth (scleroderma)+ lung and kidneys.
- Raynaud's phenomenon (fingers) and telangiectasia (mouth and fingers)
- limited (lSSc) or disseminated (dSSc)
- 90% → have at least one positive ANA
- CREST syndrome
    - C Calcinosis cutis → painful chalky-white material at fingertips
    - R Raynaud's phenomenon
    - E Esophageal dysmotility
    - S Sclerodactyly → localized thickening and tightness of skin of fingers or toes
    - T Telangiectasia
- Morphea → benign localised systemic sclerosis, discoloured firm skin . typically in abdomen, chest or back
    - If in head "frontoparietal area → ('en coup de sabre'), alopecia and groove
- MX ⇒ Calcitriol → sclerodactyly, laser → facial telangiectasia.

Lichen sclerosus (LS)

- itchy eruption which mainly affects the genital and perineal regions in women
- well-demarcated atrophic patches and plaques with a distinctive ivory white colour
- loss of normal genital architecture
- associated with the development of **squamous cell carcinoma (SCC).**
- Tx → intermittent potent topical steroids

Lichen planus (LP)

- itchy eruption, shiny purple-coloured flat-topped papules , on the wrists and ankles.
- White lines called **Wickham's striae** may appear on the surface of the lesions
- clusters or in linear scratches/surgical scars (**Koebner phenomenon**)
- black skin → hyperpigmentation.
- May affect the mouth, labia minora, nails"linear ridges" or scalp "scarring alopecia"
- Can cause bullous lesions
- Variants → hypertrophic , bullous ,oral LP
- Tx → potent topical steroids

Lupus erythematosus (LE)

- four main clinical variants → systemic"SLE", subacute, discoid and neonatal
- 75% → skin involvement, most commonly an erythematous 'butterfly' distribution rash on the face

- Tx of SLE→ Prednisolone, or immunosuppressant drugs such as azathioprine
- SLE Dx (four of the following)

malar rash	discoid plaques	photosensitivity
serositis	arthritis	mouth ulcers
neurological disorders	haematological changes	renal changes
immunological changes	antinuclear antibodies	

Subacute cutaneous lupus erythematosus (SCLE)

- white women aged 15 to 40, less systemic involvement
- skin lesions that are scaly and evolve as polycyclic annular lesions or plaques ,in sun-exposed areas
- Risk of developing SLE→ 5%
- Tx → sun avoidance and sunscreen and topical corticosteroids

Discoid lupus erythematosus (DLE)

- photosensitive disorder , well-defined coin shaped erythematous lesions with atrophy, scaling and scarring occur on the face , scalp (alopecia, follicular plugging)
- Tx → potent and super-potent topical steroids to limit scarring

Neonatal lupus erythematosus

- transplacental passage of anti Ro/La
- Annular scaly lesions on the face/scalp
- Risk congenital heart block

Dermatomyositis

- skin, muscle “discomfort and weakness in proximal limbs” and blood vessels.
- Signs
    - **Heliotrope rash** → purple hue on the upper & lower eyelids, cheeks and forehead.
    - **shawl sign** & **v sign**→ anterior ‘V’ and posterior “shawl “aspect of the neck
    - **Gottron’s papules** → dorsal surface of the fingers (esp. joints) may be affected by the erythematous eruption and purplish papules
    - **Ragged cuticles** and dilated nailfold
- Investigations
    - creatine phosphokinase (CK), ESR,anti-Jo-1 antibody, and skin and muscle biopsy.
    - Electromyography and MRI→ myositis.
- Treatment with high dose systemic corticosteroids ⇒ or cyclophosphamide, azathioprine, methotrexate and mycophenolate mofetil

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The Skin and Systemic Disease

Intro

- Reduced numbers of melanocytes can be genetic or associated with autoimmune disease or hormonal changes.
- Increased pigment in the skin → hormonal changes or underlying neoplasia.
- Generalised pruritus without a skin rash is a strong indicator of an underlying systemic disease – such as renal/hepatic dysfunction.
- Gastrointestinal disease may be associated with skin conditions such as dermatitis herpetiformis and pyoderma gangrenosum
- Clues to a possible underlying systemic disease
    - Rash associated with other symptoms
    - not responding to topical treatments
    - inflammation around the blood vessels (migratory or fixed)
    - Vasculitis (non-blanching,palpable purplish, fixed, painful and blistering)
    - Palpable dermal lesions
- Characteristic rashes associated with underlying systemic disease
    - Erythema multiforme
    - Pyoderma gangrenosum → IBD
    - Erythema nodosum
    - Vasculitis

Skin reactions associated with infections

1)Toxic erythema

- Symmetrical reactive rash,maculopapular erythema,'morbilliform' (measles-like).
- starts on the trunk and then spreads to the limbs and is blanching
- Triggers → most commonly triggered by viruses "measles, rubella, Epstein–Barr virus"
    - bacterial infections such as Scarlet fever
    - parasitic infections
- Tx → No treatment is usually required, mild topical steroid if symptomatic, Tx of the underlying condition

2)EM

- Raised erythematous macules, 'target lesions'
- acral sites (palms, soles, digits, elbows, knees and face)
- immunologically mediated hypersensitivity reaction
- EM Major. → mucous membranes + cutaneous EM rash (<10% of body surface area)
- Most common infectious trigger is herpes simplex virus (HSV 1 or 2) or cold sore
    - Or Mycoplasma pneumonia or haemolytic Streptococcus
- Adverse reactions to medications are also a common trigger for EM
- Tx → treat the underlying disease (e.g. aciclovir, penicillin) and apply topical steroid to skin lesions if painful or blistering; systemic steroids may be needed

3)Gianotti–Crosti Syndrome (GCS) – papulovesicular acrodermatitis of childhood

- viral exanthema
- vesicular in young children (age <12 years)
- Limbs then face , (spares the trunk).
- Viral triggers /// may also be triggered by vaccinations
- More common in children with atopic dermatitis (AD)

4)Erythema nodosum (EN)

- tender/painful subcutaneous erythematous nodules on the shins
- inflammation in the adipose tissue
- Causes
    - Infectious causes of EN include Streptococcus, Mycoplasma pneumoniae, TB
    - non-infectious triggers include medications, inflammatory bowel disease, sarcoidosis, pregnancy, Behcet and Hodgkin disease.
- Tx → underlying cause, elevation and compression, NSAIDs

5) Erythema annulare centrifugum (EAC)

- single/ multiple erythematous expanding rings
- on the thighs or trunk, asymptomatic
- hypersensitivity reaction, triggers → Epstein–Barr virus, HIV, Escherichia coli, Streptococcus, leukemia/lymphoma, solid tumors

6)Erythema chronicum migrans

- migrating erythema
- Borrelia burgdorferi (Lyme disease)

Sarcoidosis

- atypical mycobacterium may be the trigger
- skin disease in 40% of cases most
- common skin changes
    - EN → feature of early pulmonary disease;
    - papules, nodules, and plaques → acute and subacute forms of the disease
    - scar sarcoidosis, with papules;
    - lupus pernio

Skin changes associated with hormonal imbalance

- **Hyperpigmentation**
    - Increase in melanocyte-stimulating activity that occurs in hyperthyroidism, Addison's disease, and acromegaly.
    - In pregnancy or in those taking oral contraceptives → localized increase in melanocytic pigmentation of the forehead and cheeks known as melasma (or chloasma)
- **Hypopigmentation**
    - Widespread partial loss of melanocyte functions with loss of skin color
    - Hypopituitarism, absence of melanocyte-stimulating hormone.
- **Acanthosis nigricans** AN

    - asymptomatic velvety thickening of the skin, posterior and lateral aspects of the neck, axillae and arm flexures
    - MCC → obesity, and with weight reduction the AN resolves.
    - Type A → insulin resistance in young black women with hirsutism and polycystic ovarian syndrome
    - Type B → autoimmune conditions such as diabetes, thyroid disease and lupus.
    - Tx → underlying cause
- **Diabetes**
    - diabetic dermopathy' due to a microangiopathy → erythematous papules ,resolve to leave a scaling macule on the limbs.
    - Ulceration due to neuropathy (trophic ulcers) or impaired blood supply may occur, particularly on the feet.
    - increased susceptibility to cutaneous infections → staph, strep, coliforms, Pseudomonas and C. albicans.
- **Necrobiosis lipoidica**
    - between 40% and 60% of patients with this condition may develop diabetes, but it is actually uncommon in the diabetic population (0.3%), but checking of fasting glucose is recommended.
    - Replacement of degenerating collagen fibers with lipid material. It usually occurs over the shin but may appear at any site
- **Granuloma annulare**
    - localized papular, hands/feet and limbs
    - seen more commonly in women under the age of 30.
    - insulin-dependent diabetes
    - Self-limiting

Thyroid disease

- increases in TSH → pretibial myxoedema
- In autoimmune thyroid disease → associated vitiligo and other autoimmune conditions

Clinical signs of thyroid disease	
Hypothyroidism	Hyperthyroidism
Dry skin	Soft, thickened skin
Edema of eyelids and hands	Pretibial myxoedema
Absence of sweating	Increased sweating (palms and soles)
Coarse, thin hair; loss of pubic, axillary & eyebrow hair	Thinning of scalp hair
Pale 'ivory' skin	Diffuse pigmentation
Brittle poorly growing nails	Rapidly growing nails
Purpura, bruising and telangiectasia	Palmar erythema, Facial flushing

Skin changes associated with disorders of the **GI system and liver**

- Polyarteritis nodosa (medium vessel vasculitis) and connective tissue diseases (such as scleroderma), part of a metabolic disease such as porphyria
- **Malabsorption** → deficiency of iron, zinc, vitamins
    - dry pruritic skin, superficial eczema, Increased pigmentation, brittle hair and nails
    - Vitamin C deficiency (scurvy) → Malabsorption
        - perifollicular hyperkeratotic papules, anemia
        - Tx → Vitamin C supplementation
    - Zinc deficiency (acrodermatitis enteropathica)
        - Neonates (genetic condition,or breast milk deficient in zinc or malabsorption)
        - erythematous inflamed scaly skin around the mouth, anus and eyes as well as acral sites (hands, feet, elbows and knees
        - not itchy, well-demarcated, localized
        - Low zinc levels
        - Tx → Zinc supplementation
- Pyoderma **gangrenosum**
    - Rapid onset painful area of necrotic skin ulceration, hypertrophic undermined purplish margins
    - ulcerative colitis and Crohn's disease, rheumatoid arthritis, abnormal gamma globulins, and leukemia
- Crohn's disease (regional ileitis)

    - Glossitis and granulomatous thickening of the lips and oral mucosa
- Dermatitis herpetiformis
    - intensely itchy, erythematous and blistering papules, elbows, knees and buttocks
    - gluten-sensitive enteropathy, small bowel lymphoma
- Congenital disorders of the bowel
    - Peutz–Jeghers syndrome (hereditary intestinal polyposis syndrome)
        - pigmented macules on the oral mucosal membranes, lips and face, hands/feet
    - neurofibromatosis (intestinal neurofibromas)
    - pseudoxanthoma elasticum (arterial GI bleeding), purpuric vasculitis (bleeding from GI lesions)
- Liver disease and the skin
    - Obstructive → Jaundice, Pruritus. deposition of bile salts “Tx; cholestyramine”
    - Liver failure → Multiple spider nevi “esp in men”, Palmar erythema, White nails “hypoalbuminaemia”, yellow nails
        - Porphyria cutanea tarda → bullae, scarring & hyperpigmentation in sun-exposed areas of the skin, more in men “genetic”, excessive intake of alcohol, deficiency of uroporphyrinogen decarboxylase in the haem synthesis pathway
            - DDx → Pseudoporphyria “ chronic renal failure on haemodialysis”, no porphyrins are found in urine/blood.
        - Porphyrias → accumulation of intermediate metabolites of haem synthesis.
            - hepatic porphyrias → skin fragility
            - Erythropoietic & erythrohepatic photoporphyrias → intense photosensitivity
    - Cirrhosis → Xanthomas (primary biliary cirrhosis,Alagille syndrome), Asteatosis.
        - Xanthomas are lipid-laden macrophages, hyperlipidemia
        - Diabetes → eruptive Xanthomas

Pigmentation disorders

Hypopigmentation

- **Albinism** → recessive gene, diminished or loss of pigment in the skin, hair and eyes, Other genetic conditions with loss of skin pigment include piebaldism , phenylketonuria and tuberous sclerosis.
- **Localised depigmentation** → vitiligo; family history “ in one-third of the patients”, In the sharply demarcated, symmetrical macular lesions, loss of melanocytes and melanin
    - Autoimmune associations → Thyroid disease, Myasthenia gravis, Pernicious anemia, Alopecia areata, Hypoparathyroidism, Addison’s disease, DM
- Post Inflammatory conditions such as psoriasis, eczema, lichen planus and lupus erythematosus; infections, tuberous sclerosis (‘ash leaf’ macules).

Hyperpigmentation

- increase in the normal pigment melanin or to the deposition of bile salts(liver disease), iron salts (haemochromatosis), drugs or metallic salts from ingestion.
- In agyria, ingested silver salts.
- Medications→ chlorpromazine, other phenothiazines & minocycline (in sun-exposed areas)
    - Phenytoin (local hyperpigmentation of the face and neck). AZT(cutaneous and nail hyperpigmentation)
- Acanthosis nigricans AN
- Acromegaly
- Post-inflammatory pigmentation → acute eczema, fixed drug eruptions & lichen planus.

Skin manifestations of underlying malignancy

- Skin markers of internal malignancy
    - Acanthosis nigricans→ gastric adenocarcinoma.
    - Figurate erythemas → bronchial/ esophageal/breast carcinoma.
    - Pruritus can be associated with lymphoma.
    - Dermatomyositis → lung/breast/ovarian/testicular carcinomas.
    - Acquired ichthyosis→ Hodgkin’s disease, sarcoma, lymphoma
- Non-specific skin changes associated with malignant disease
    - Secondary deposits, Secondary hormonal effects, Acne (adrenal tumors)
    - Flushing (carcinoid), Pigmentation (pituitary tumors)
    - Generalized pruritus (particularly lymphoma)
    - Figurate erythema
    - Superficial thrombophlebitis.
- Mycosis fungoides→ T-cell lymphoma of cutaneous origin
    - erythroderma in Sezary syndrome

- Parapsoriasis → well-defined erythematous scaly patches and plaques that slightly resemble psoriasis, Dx → Bx
- Poikiloderma → may precede mycosis fungoides or after radiotherapy

Pregnancy and the skin

- Pruritus (skin appears normal in 15–20% of women (prurigo gestationis), more severe in the first trimester
- Polymorphous eruption of pregnancy (PEP) → 3rd trimester, does not affect the baby, striae of the abdomen, resolves postpartum, Sx Tx→ Topical (occasionally systemic) steroids
- Pemphigoid gestationis (PG) → pemphigoid-like vesicles, autoimmune, cross-reactivity between placental tissues and the skin, more premature babies

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Acne

Definition

- Sebaceous glands associated with hair follicles: face, back, chest and anogenital area
- Changes
    - Thickening of the keratin lining → obstruction of the sebaceous duct
        - Closed comedones (**'whiteheads'**)
        - Open comedones (**'blackheads'**)
    - Increase in sebum → greasy skin
    - Propionibacterium acnes
    - Inflammation around (redness, papules and pustules → larger cysts and nodules.)

Underlying causes

- Hormones
    - Androgenic hormones,Virilising tumours
    - PCOS, CAH
    - Cushing's syndrome (Steroids)
- Medications
    - Steroids , OCP
    - Phenytoin(antiepileptics), Isoniazid, Lithium.
- Fluid retention, Sweating
- Stress
- Diet → chocolate, nuts, coffee
- Seasons → improves with natural sunlight
- External factors→ oils

Types of acne

- **Acne vulgaris**
    - Common type, more in males, during puberty, in comedogenic areas of the face, back and chest, familial
    - **Acne keloidalis** → scarring acne on the neck (nuchae) in men
    - Symptoms and signs
        - Excessive greasiness, 'spots', 'blackheads' or 'pimples'.
        - Inflammatory papules and pustules → larger cysts and nodules.
        - Resolve into post-inflammatory **pigment changes** and **scarring**.
            - Atrophic and pitted
            - Deep → ice pick, rolling and boxcar
            - Hypertrophic or even keloid .
            - Hyper/hypopigmented and erythematous.
- **Acne excoriée**
    - Picker's acne,disfiguring erosions
- **Infantile acne** → on the face in the first few months
- **Acne conglobata/fulminans**
    - Severe, more in boys and tropical climates
    - Extensive, nodulocystic acne and abscess formation
    - **Fulminans** → associated with systemic symptoms of malaise, fever and joint pains
    - **Pyoderma faciale** → necrotic lesions
    - **Gram-negative folliculitis** → Klebsiella, Proteus, Pseudomonas & E.coli

Tx

- Comedones → Topical **retinoid**, Salicylic acid, Azelaic acid
- Inflammatory papules/pustules → **Benzoyl peroxide**

- Nodulocystic→ Oral **antibiotic** + topical retinoid
- Mixed picture → Topical retinoid ± benzoyl peroxide ± topical antibiotic "Combination"
- Residual lesions,scars→ triamcinolone, topical retinoids, dermabrasion, dermaroller
    - If severe → pinch biopsy and graft
- Mild acne → Cleansers, keratolytics
- **Topical treatments**
    - **Benzoyl peroxide** → bacteriostatic against P. Acnes, comedolytic.
    - **Salicylic acid** → comedolytic
    - **Azelaic acid**→ anti keratinising and antibacterial effects
    - **Topical retinoids** "vitamin A derivatives" → anti-inflammatory and comedolytic.
        - **Tretinoin**
    - **Topical antibiotics**→ against P. Acnes ⇒ erythromycin &\or clindamycin
    - **Phototherapy** with ultraviolet or visible light (Red-Blue)
- **Systemic treatments**
    - **Hormone** therapies
        - OCPs "higher oestrogen and lower androgen potential" that increase sex hormone-binding globulin → reduce free testosterone.
        - Antiandrogens → in the form of contraceptive (alone can be teratogenic)
            - Cyproterone acetate with ethinyl estradiol (Dianette).
    - Oral **antibiotics** → Tetracyclines (above 12 years) or doxycycline
        - If preg. Or <12 years → Erythromycin and trimethoprim
    - Oral **retinoids** → **Iso-Tretinoin**
        - For resistant cases
        - SE → teratogenesis,drying of the lips & skin, elevated liver enzymes and lipids → do baseline and follow up LFT and lipid profile
        - Daily dose → 1mg/kg/day . Cumulative target dosage → 150 mg/kg

Rosacea

Definition

- Facial flushing, persistent erythema, Telangiectasia (permanent facial erythema), inflammatory papules, pustules and oedema. (**NO** comedones)
- Mostly in cheek (uni or bilateral) , or localized to nose, If chronic → rhinophyma
- Conjunctivitis, blepharitis and eyelid oedema
- Exacerbated by heat, exercise, hot food/drinks, spicy food, emotion, alcohol and sunlight.

DDx

- Acne → comedones,improvement with sunlight, overlap → acne rosacea
- Seborrhoeic eczema → no pustules
- Dermatomyositis,Lupus erythematosus → no pustules
- Perioral dermatitis

Tx

- Avoid triggering factors
- Topical **metronidazole** or azelaic acid → mild cases, for papules and pustules
- Oral **antibiotics** → tetracycline, doxycycline, erythromycin
- Low-dose oral **isotretinoin**
- α 1&2 -agonists for diffuse facial erythema, Laser ablation of dilated telangiectatic

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Bacterial Infections

Intro

- Damage to the epidermis/dermis results in reduced barrier function, which enables bacteria to invade the skin.
- Normal skin flora consists of coagulase-negative Staphylococcus, Corynebacterium, diphtheroids and α-haemolytic Streptococci in the epidermis, and Propionibacterium in the pilosebaceous unit
- Cutaneous bacterial infections produce signs of acute inflammation: erythema, swelling/edema, heat/warmth and pain/discomfort.
    - Hx of trauma "graze, laceration", contaminated water, animal contact, travel abroad
    - systemic symptoms such as fever and malaise
- Localized superficial skin infections can be managed with antiseptic washes and topical antibiotics.

- Systemic antibiotics are needed for widespread, deep and persistent bacterial skin infections.
- Clinical signs of bacterial infections include folliculitis, boils/abscesses, blistering, crusting, erosions, ulcers, and cellulitis.
- Mycobacterial skin infections most commonly arise from implantation/trauma and are usually localized in immunocompetent patients.
- Atypical mycobacteria can result in localized suppurative lesions, persistent granulomas and sporotrichoid spread via lymphatics.

Bacterial investigations

- bacterial swabs for microscopy and culture
    - bacterial species, antibiotic resistance/sensitivity patterns and bacterial toxin production
- Nasal swabs → staph
- Methicillin-resistant S. aureus (MRSA) may be community or hospital-acquired
- Panton-Valentine Leukocidin (PVL) test → multiple/recurrent boils not settled with flucloxacillin
- skin biopsy and PCR → if suspect mycobacterial infections

General approach to management

- Antiseptic skin washes or creams
- Topical antibiotics→ Fusidic acid, mupirocin, neomycin and metronidazole
    - Topical antibiotic/steroid combinations are useful in treating infection and inflammation simultaneously
- Systemic antibiotics (extensive infection)
    - Staphylococcal cover → flucloxacillin, erythromycin,clarithromycin, azithromycin, co-amoxiclav, clindamycin, fusidic acid,
        - For MRSA, use vancomycin
    - Streptococcal cover → amoxicillin,erythromycin, clarithromycin

Superficial infections

- Impetigo
- Bacterial folliculitis
- Deeper follicular infections
    - sycosis barbae in the beard area
    - Hot-tub folliculitis caused by Pseudomonas aeruginosa
- Pseudofolliculitis → occlusion of the follicular openings by heavy emollients rather than bacterial infection. monomorphic, sterile pustules
- Pseudofolliculitis barbae → beard area, perifolliculitis.Coarse curly hair punctures the skin adjacent to the hair follicle
    - acne keloidalis nuchae results from folliculitis and perifolliculitis → alopecia and keloid scarring
- Erythrasma → flexural skin sites, Corynebacterium minutissimum,
    - Wood's ultraviolet light → fluoresces pink.
    - Tx → oral erythromycin

Deeper infections

- Erysipelas
- Cellulitis
    - Poorly defined margin and marked regional lymphadenopathy
    - S. pyogenes
    - lower leg is the most common site affected
    - Tx → intravenous benzylpenicillin
- Necrotising fasciitis
    - deep fascia leading to gas formation
    - Recent trauma or surgery
    - severe pain initially at the site followed by anaesthesia.
    - Tx → surgical debridement and broad spectrum antibiotics
- Staphylococcus scalded skin syndrome (SSSS)
    - exfoliative toxinsA/B
    - If localized → bullous impetigo
    - If <5 years → + conjunctivitis, otitis media or a nasopharyngeal infection,
    - widespread superficial blistering (Nikolsky sign positive)
    - systemic antibiotics for staph then if failed for MRSA
- Ecthyma → deeper form of impetigo

Mycobacterial disease

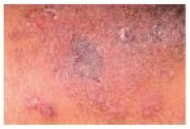
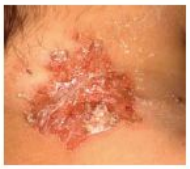
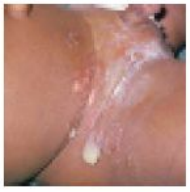
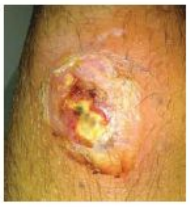
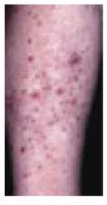
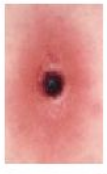
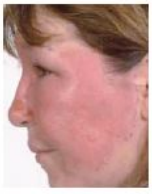
- Mycobacterium tuberculosis → miliary TB or lupus vulgaris

    - Cutaneous M. tuberculosis (TB) → secondary manifestation of the primary (respiratory), MC → lupus vulgaris "head and neck, apple-jelly nodules"
    - Allergic-type hypersensitivity reactions called tuberculids for dead bacilli
        - erythema induratum (Bazin's disease) → tender nodules & plaques
- Mycobacterium leprae → lepromatous (multibacillary) or tuberculoid (paucibacillary
- Atypical mycobacteria (ATM)
    - Fast-growing Mycobacterium chelonae complex/M. abscessus
    - M. avium complex (MAC) → lymphadenitis in children and disseminated disease in HIV patients. MC in Immunocompromised, DM, chronic renal failure, connective tissue disease
    - Tx → clarithromycin ± rifampicin for 4–6 months
- Mycobacterium marinum or 'fish tank' or 'swimming pool granuloma' → MC in hand or fingers
- Mycobacterium ulcerans → extensive non-painful ulceration (Buruli ulcer)

Other → Cat-scratch→ bacterium B. henselae.

- Crusted nodules within 3–12 days at the site of a scratch (usually by a kitten) + regional painful lymphadenopathy 1 or 2 months later. The disease usually undergoes spontaneous remission within 2–4months. A 5-day course of azithromycin can speed up recovery.

Table 13.1 Common patterns of bacterial infection in the skin.

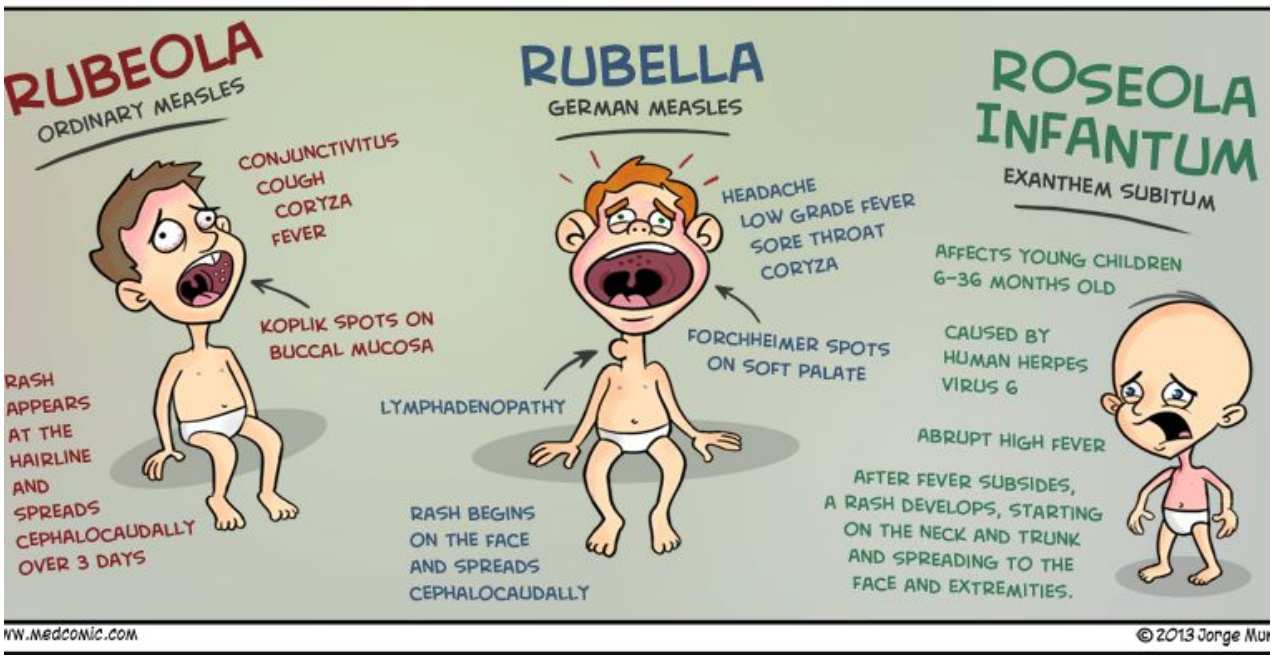
Infection	Clinical photograph	Clinical presentation	Organisms	Management
Infected eczema		Background inflammatory atopic dermatitis with excoriations and marked crusting and exudate	*Staphylococcus aureus* *Streptococcus pyogenes*	Antiseptic wash Topical antibiotic/steroid combination cream. Oral flucloxacillin or erythromycin
Impetigo		Mainly children, especially face and limbs. Highly contagious. Yellow crusted lesions surrounded by normal skin	*S. aureus* *S. pyogenes*	Antiseptic wash Topical antibiotic Oral flucloxacillin or erythromycin
Bullous impetigo		Children and adults. Face, limbs and flexures affected. Erythema with bullae which rupture leaving superficial erosions and crusts	*S. aureus* with exfoliative toxins A/B (may become generalised – staphylococcal scalded skin syndrome)	Oral flucloxacillin or erythromycin
Boils (abscesses)		Tender, inflamed indurated nodules with central pus may be single or multiple. If recurrent and recalcitrant consider toxin producing bacteria	*S. aureus* Consider Panton valentine leukocidin Toxin producing *S. aureus*	Antiseptic wash Oral flucloxacillin or erythromycin If PVL positive give nasal bactroban and consider giving clindamycin plus rifampicin for 4–6 weeks
Bacterial folliculitis		Hair-bearing sites particularly legs, beard area and scalp. May result from shaving damage to skin. In recurrent infections look for *S. aureus* nasal carriage	*S. aureus* *Pseudomonas aeruginosa* (differential diagnosis *Malassezia* spp)	Topical antibiotics Acetic acid cream EarCalm® for *P. aeruginosa* Oral flucloxacillin or erythromycin Avoid shaving if possible
Ecthyma		Children, the elderly/debilitated. Mainly on the legs. Initially small bullae with necrotic dry adherent crust and underlying ulceration Heal slowly with scarring	*S. pyogenes* *S. aureus*	Antiseptic wash Oral penicillin V or erythromycin
Erysipelas		Face or lower leg. Portal of entry is broken skin (trauma and tinea pedis) well-demarcated bright erythema	*S. pyogenes* (group A *Strep.* but also B, C, G) *S. aureus* (less common)	Intravenous benzyl penicillin or erythromycin

Viral diseases with rashes

Disease	Age	Incubation period	symptoms	Skin manifestation	Dx +Tx	other
Measles	<5 years	7-14 days	Prodromal symptoms include fever, malaise, upper respiratory symptoms, **conjunctivitis** and photophobia	**Koplik's spots** (white spots with surrounding erythema)on the oral mucosa **macular rash** appears, initially behind the ears and on the face and trunk, and then on the limbs haemorrhagic or vesicular papules → brown patches	PCR supportive care two doses of live-attenuat ed MMR	highly contagious
Rubella German measles or three-day measles,	childre n and young adults	14–21 days	prodromal fever, malaise and upper respiratory tract symptoms	erythema of the **soft palate** and **lymphadenopathy** pink macules appear on the face, spreading to trunk and limbs over 1–2 days . The rash clears over 1–2 days (occasionally no rash develops).	serum antibody titres. two doses of live-attenuat ed MMR	
Erythema infectiosu m (fifth disease)	2–10 years	5–20 days	prodrome of mild fever	hot erythematous eruption on the cheeks – hence the **'slapped cheek syndrom**e'.maculopapular eruption develops on the limbs and trunk	serology for parvovirus B19-specific IgM antibody	**parvovirus B19**,
Roseola infantum (sixth disease)	< 2 years	10–15 days	High grade fever.	rose-pink maculopapular rash appearing on the neck and trunk	serology	human herpesvirus type 6 (**HHV6**)
Gianotti–Crosti syndrome	< 14 years	unknown;	malaise, lymphadenopathy	acral eruption. erythematous papules on the face, neck, limbs, buttocks, palms and soles Itchy papular viral exanthem	Serology topical steroid for the exanthem	**Epstein–B arr virus (EBV) and hepatitis B**
Hand, foot and mouth disease	childre n and adults	3–6 days	fever, headache and malaise alongside the rash	intense erythema surrounding yellow-grey vesicles 1–1.5mm in diameter on palms/soles and lips	Serology	**Coxsackie virus A** highly contagious

Disease	symptoms	Skin manifestation	other
Measles	Prodromal symptoms, **conjunctivitis** and photophobia	**Koplik's spots** **macular rash** move cephalocaudally	
Rubella German measles or three-day measles,	Prodromal symptoms	erythema of the **soft palate** and **lymphadenopathy** **macular rash** move cephalocaudally	
Erythema	prodrome of	**'slapped cheek syndrom**e'	**parvovirus**

infectiosum (fifth disease)	mild fever		**B19**,
Roseola infantum (sixth disease)	High grade fever.	rose-pink maculopapular rash appearing on the neck and trunk	human herpesvirus type 6 (**HHV6**)
Gianotti–Crosti syndrome	malaise, lymphadenopathy	acral eruption. erythematous papules Itchy papular viral exanthem	**Epstein–Barr virus (EBV) and hepatitis B**
Hand, foot and mouth disease	fever, headache and malaise alongside the rash	intense erythema surrounding yellow-grey vesicles 1–1.5mm in diameter on palms/soles and lips	**Coxsackievirus A**



Fungal Infections

Introduction

- RF → hot climates, immunosuppressed pts.
- Tinea capitis (scalp), tinea corporis (body), tinea cruris (groin) and tinea pedis (feet).
- Principles of diagnosis
    - Clinical → itchy, dry, scaly lesions,asymmetrical distribution
        - Raised scaly margin with inflammation
    - Wood's light (UV light) → Microsporum infections "green-blue fluorescence"
    - Microscopy(faster)+culture →skin scrapings or Bx /scalp brushings/Nail clippings
        - + KOH
    - Rapid PCR tests and ELISA

Scalp and face

- **Tinea capitis (scalp ringworm)**
    - Ringworm → scaling margin with clear center → annular or ring-shaped
    - MC → Trichophyton tonsurans > Microsporum canis
    - More in prepubertal children
    - Single or multiple patches of alopecia, minimal scaling and inflammation.
        - Black dots (broken-off hairs), multiple pustules
        - Extensive alopecia with inflammation
        - Kerion + occipital lymphadenopathy → inflamed, boggy, pustular lesion on the scalp
    - Tx ⇒ systemic antifungals → Oral griseofulvin or terbinafine
        - Id reaction → papular/pustular widespread cutaneous eruption after systemic antifungal treatment
    - Ddx → scalp eczema/psoriasis, folliculitis, alopecia areata and seborrhoeic dermatitis
- **Tinea incognito** → atypical fungal infection because of use of topical\systemic steroids
- **Seborrhoeic dermatitis (SD)**
    - Allergic contact dermatitis due to the yeast **Malassezia furfur**
    - Hair-bearing skin
    - Itchy, adherent greasy scales
    - DDx → atopic eczema/psoriasis
    - Tx → Ketoconazole shampoo or topical steroids (± miconazole)

Feet (and hands)

- **Tinea pedis** or **athlete's foot** → More in adults
    - Public swimming pools or showers
- Very itchy, frequently occurs between the toes (esp the fourth toe web)
- Dry, scaling rash with vesicles at the active margins
- Tx→ topical antifungal (Terbinafine)

Trunk

- **Tinea corporis** in body
    - Erythematous with a well-defined scaly edge
- **Tinea cruris** in groin → more in men
    - Symmetrical dry scaling ,may spread to the upper inner thighs
    - Ddx
        - Candida ⇒ intense erythema and satellite lesions
        - Erythrasma, psoriasis\eczema, mycosis fungoides
    - Tx → topical Terbinafine
- **Pityriasis versicolor** → M. Furfur
    - Upper back, neck, chest & arms. Areas fail to tan
    - Well-defined macular lesions of variable colour ('versicolor') → hyper or hypo pigmented → darker brown to pale tan
    - Ddx → seborrhoeic dermatitis, pityriasis rosea, guttate psoriasis and vitiligo
    - Tx → ketoconazole

Nails

- **Onychomycosis** → mostly adult toenails
    - Mostly dermatophytes.May be caused by Candida albicans
    - Nail plates → thickened, brittle and white to yellow/brown, from distal to proximal
    - Ddx ⇒ Psoriasis → from proximal to distal, symmetrical, pitting
    - Tx → oral terbinafine (16 wk for toenails, 8 wks for fingernails)

- **Chronic paronychia**
    - Around the nails , ‘wet-work’ individuals
    - Erythema and swelling of the nail fold, on one side ,brownish discolouration

Yeast infections

- **Candida infection**
    - Mucous membranes and flexures, under the breasts and abdominal skin folds
    - Ddx → psoriasis, seborrhoeic dermatitis and contact dermatitis
    - Candida intertrigo → symmetrical and ‘satellite’ pustules or papules outside the outer rim of the rash
    - C. Albicans → mouth”white plaques or erythema”, vagina “thrush”
    - RF → diabetics, immunosuppressed”HIV, steroids”
    - Tx → topical antifungals “miconazole” or systemic “fluconazole”

Deep fungal infections

- RF → diabetics, neutropenic, immunosuppressed, skin trauma

Scabies and head lice

Intro

- Bites can cause a local skin reaction and/or systemic disease in humans through the transmission of parasites, bacteria or viruses.
- Biting insects (including mosquitoes, midges, bedbugs, fleas, sandflies, mites, ticks and lice) cause erythematous pruritic lesions on exposed skin, usually in groups/clusters.
- A generalized anaphylactic reaction to an insect sting can be life-threatening. Pts known to be at risk should carry a preloaded syringe of adrenaline to use when reactions occur.
- Worldwide, scabies is the most common infestation. Female mites burrow through the skin, resulting in intense itching.
- Other infestations include lice (which may transmit trench fever and typhus). Pubic lice may be associated with other sexually transmitted diseases.
- Cutaneous larva migrans occurs when larvae from the dog/cat hookworm penetrate human skin, causing a superficial creeping eruption, Mgt → albendazole or ivermectin

Infestations by parasites

1)Scabies (Sarcoptes scabiei)

- intense itching, keep pt awake /// female mite burrows into the epidermis and lays eggs
- close personal contact (at least 15 min of skin-to-skin contact)with an infected individual.
    - within a family, infants in playgroups, & through regular nursing of elderly Pts .
    - Sx after 2 weeks (immune Rx to proteins in the mites), carry 10 adult mites
- There may be very few burrows, though the patient has widespread itching.
- Distribution → fingers, wrists, nipples, abdomen, genitalia, buttocks and ankles.
- Itching may persist even after all mites have been eliminated;
- itching papules on the scrotum and penis are particularly persistent
- Dx → several individuals in the same household/institution, affected simultaneously by rash that is intensely itchy at night + widespread papular rash
    - burrows can be seen (in fingers or genitals), linear palpable ridges with black speck
    - Scratch by sterile needle → microscopy
- Scabies in children → erythematous cutaneous papules and nodules in the axillae and on the soles of the feet, Classic burrows not seen
- Crusted scabies → dry scaly skin rashes, immunosuppressed or elderly, hundreds of mites and no itch (week immunity)
- Mgt
    - First-line treatment for scabies is 5% permethrin (not available in Jordan) cream left on overnight, two applications 7 days apart. Adults apply from the neck downwards; babies/infants apply to all the skin.
    - Second-line → malathion (lotion), Ivermectin (oral), Benzyl benzoate (available in Jordan)

Lice

Head lice

- Children are the most common hosts. Girls > boys
- transmitted by head-to-head contact, and on combs
- Sx → Mild itching
- Dx → inspection of the scalp using Fine-toothed combs → adult lice and nits
- Mgt → First-line treatment is permethrin 1–5% crème rinse applied to dry hair and left on overnight. This should be repeated after 7 days

Body lice

- Vector for → Bartonella Quintana (agent of trench fever, bacillary angiomatosis and endocarditis) and Rickettsia prowazekii (agent of typhus)
- Affects poorer people, lice live in the host's clothes and bite the skin.(Dx → inspect skin and clothes)
- papular eruption with excoriations
- Mgt → Wash clothing in hot water then dry in sun
    - potent topical steroid plus topical antibiotic (if infected)
    - Refer to cardiologists if endocarditis is suspected.

Pubic lice "crab lice"

- pubic, axillary and eyelash areas ⇒ check for sexually transmitted diseases
- Mgt → topical permethrin 5% to skin from the neck downwards, left on overnight, repeated after 7 days
    - If eyelashes involved → petrolatum only

Hair and Scalp

Hair Types→ Lanugo hair "fetus", Vellus hair "whole body,shorter non-pigmented", Terminal hair "eyebrows, lashes & scalp, axillae, pubic region & on the central chest in men, darker and longer"

Hair cycle

- Anagen → active growth phase, 1000 days, 85% of hair
- Catagen → short growth arrest phase,10 days
- Telogen → resting phase,100 days. 15% of hair

Hair loss "alopecia" → generalised or localised, with or without inflammation.

Non-scarring alopecias

- Androgenetic alopecia → MC in male and females, male-pattern baldness,female pattern hair loss (FPHL), caused by dihydrotestosterone (DHT)
    - RF → age, genetic predisposition
    - Men → temples (frontotemporal recession) or vertex → horseshoe distribution
    - Women → thinning (shorter and finer hairs) over the central scalp, often presenting with a widening of their parting. preservation of the frontal margin
    - Tx →
        - Males → oral finasteride "5α-reductase inhibitor" and topical minoxidil
        - FPHL → oral finasteride and topical minoxidil,OCP, Anti-androgens (spironolactone & cyproterone acetate ⇒ for hyperandrogenism)
        - Hair transplantation
- Alopecia areata "AA"
    - alopecia totalis → total scalp hair loss
    - alopecia universalis → total body hair loss
    - Ophiasis → localised hair loss along the scalp margin
    - AA → smooth round or oval patches of non-scarring hair loss on the scalp.
    - Exclamation mark hair " narrower closer to the scalp" is diagnostic for AA
    - Poor Px → childhood onset, atopic, ophiasis, nail dystrophy, family history
    - Associated autoimmune→ vitiligo, thyroiditis
    - Tx → Topical/intralesional corticosteroids "diluted with local anaesthetic", Systemic immunosuppression,PUVA, Contact sensitisation,Topical minoxidil
- Telogen effluvium
    - Diffuse synchronous shedding approximately 2 months after the triggering event
    - Causes
        - Hormonal (e.g. pregnancy and post-partum)
        - Nutritional (e.g. iron deficiency,hypo-proteinaemia),Acute weight changes
        - Drugs (e.g. β-blockers, anticoagulants, retinoids, immunisation)
        - Systemic disease (e.g. chronic inflammatory diseases,IBD, malignancy)
        - Stress (e.g. pyrexia, major surgery or trauma)
    - Investigations → TFT, ferritin, vitamin B12, folate and zinc
- Tinea capitis
    - patchy non-scarring hair loss short broken off hairs, scaling and erythema

- In children.
- MCC → Trichophyton tonsurans,Microsporum canis (green under Wood's light)
- DX → microscopy (spores) + culture
- Kerion formation → inflamed, boggy, pustular lesion
- Tx → systemic antifungal "griseofulvin "

Scarring alopecia → permanent hair loss due to replacement of hair follicles by scar tissue.

- Primary causes
  - **Lymphocytic** disorders
    - Inflammatory → Discoid lupus erythematosus ,lichen planopilaris,
    - Non- Inflammatory → Pseudopelade of Brocq,Central centrifugal cicatricial alopecia (CCCA),traction alopecia→ caused by pulling force being applied to the hair. Trichotillomania → hair pulling psychological disorder. Hair styling
    - Tx → include topical or systemic corticosteroids
  - **Neutrophilic** disorders
    - Dissecting folliculitis (multiple painless boggy nodules+pustules)
    - decalvans Folliculitis (patches of scarring. Crusting and tufting of the hairs, S. aureus)
- Secondary causes
  - Post-traumatic,Burns
  - Radiotherapy
  - Neoplasia (e.g. squamous cell carcinoma, lymphoma and sarcoma)
  - Infection
    - Bacterial (e.g. folliculitis, acne keloidalis and syphilis)
    - Viral (e.g. herpes zoster)
    - Fungal (e.g. kerion formation)
- Dx → Skin biopsy with direct immunofluorescence

Excessive hair → hirsutism and hypertrichosis

Hirsutism

- increased growth of the terminal hairs in androgen-sensitive areas such as the beard and moustache regions in females
- Causes
  - Ovarian → PCOS "MCC",Ovarian tumours
  - Adrenal → CAH,Cushing's disease
  - Idiopathic (racial and familial, with a wide spectrum of normal variation)
- Management
  - Normal menstrual cycle → normal hormone levels
  - serum testosterone for screening
  - androgen antagonists (Cyproterone acetate) and cosmetic (laser)

Hypertrichosis

- excessive growth of hair in any part of the body and may be localised (e.g. Becker's nevus) or generalised
- Causes → congenital or acquired
  - Hyperthyroidism, porphyria and anorexia nervosa
  - Drugs. → Ciclosporin ,Minoxidil,Psoralens,Phenytoin,Penicillamine

Scalp

- Seborrhoeic dermatitis → Malassezia furfur yeast, erythema and scaling, Tx → antifungal agents" topical ketoconazole", topical steroids for the inflammation
- pityriasis amiantacea

Diseases of the Nails

Intro

- Nails are **ectodermal** derivatives composed of **keratin** which grow forward from a fold of epidermis over the nail bed. Fingernails grow at approximately 1mm per week and toe nails 1mm per month.Nail keratin is derived mainly from the matrix
- The attachment of the nail plate to the underlying nail bed can be affected by excess keratin, inflammatory changes or infection, which causes the nail plate to lift: **onycholysis**.

- Thickening of the nail plate may occur as a result of inflammatory, traumatic and infective conditions.
- **Transverse ridges** are seen in psoriasis and eczema.
- **Beau's line** is a transverse depression affecting most of the nails due to a severe illness or physiological stress.
- Changes in the **shape** of the nail include **clubbing**, which is due to swelling and increased vascularity of the tissues surrounding the nail. **Koilonychia** is a 'spoon-shaped' deformity of the nail that may be associated with **iron deficiency**.
- Infection and inflammation adjacent to the nail results in **paronychia** that may be acute or chronic.
- **Colour** changes in the nail can arise through alteration of the nail bed or the nail plate and sometimes both. These include **leukonychia** (whitening of the nails) and **black** discolouration from **subungual bleeding. ⇒** use **Dermoscopy**
- **Longitudinal brown streaks** often occur in those with racially pigmented skin. In Caucasians, isolated brown streaks in the nail may be due to a **dysplastic nevus**, and involvement of the nail fold suggests a subungual melanoma

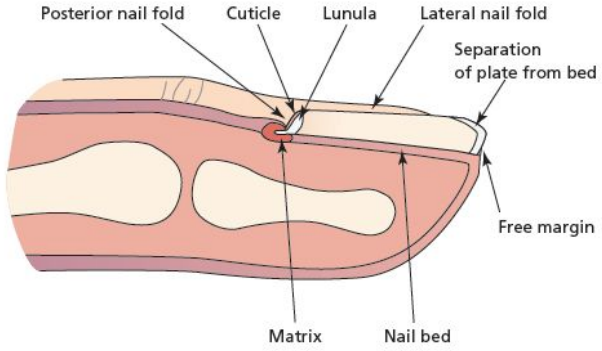


Figure 20.1 Section through finger.

Changes of shape and attachment

- Pitting
  - small surface depressions, loss of the parakeratotic scale
  - **Psoriasis, eczema**, lichen planus and alopecia areata ⇒ scales → Pitting
- Subungual hyperkeratosis
  - scaling occurs beneath the nail in the distal nail bed → scales build up
  - **psoriasis, eczema** and fungal nail disease.
  - In toes → reaction to trauma or generalised hyperkeratosis.
- Oily spot → **psoriasis**, discolouration with oily appearance.
- Onycholysis
  - painless separation of the nail from the nail bed
  - inflammatory, traumatic or infective
  - Acute trauma → re-adherence \\ Chronic trauma → manicure
  - Psoriasis or thyrotoxicosis.
  - If chronic ⇒ secondary infection → (if wet and warm) Candida spp. Or Pseudomonas → persistence of the split
- Nail plate thickening
  - thickens and becomes yellow
  - as part of normal ageing on the toes.
  - **Eczema**, **psoriasis**, lichen planus and yellow nail syndrome
  - Fungal infection
  - Chronic trauma → ex; hammer toe
- Transverse ridges
  - psoriasis and eczema
  - Isolated digital trauma → solitary
- Irritant dermatitis (eczema)
  - significant cause of chronic paronychia
  - Seen in atopy or occupational irritation
  - disposition to secondary infection with Candida spp
- Beau's line
  - A substantial general physiological disturbance can result in a solitary episode of reduced nail matrix function
  - Partial thickness transverse break in the nail plate, in many nails
  - **eczema or psoriasis,**severe illness or physiological stress.
- Nail loss (onychomadesis)
  - nail matrix inflammation is global and severe

    - inflammatory skin disease or an episode of trauma (bleeding beneath the nail)
- Longitudinal splits
    - If single → focal matrix damage,acute trauma, chronic trauma, dysplastic process ⇒ close examination and imaging and may require surgical exploration
    - If multiple → generalised inflammatory (lichen planus) or degenerative process (ageing) or both (Darier's disease)
    - Ageing → loss of nail substance and increased fragility → distal few mm splits
- Transverse (lamellar) splits
    - in childhood, in the big toes and where there is thumb sucking
    - loss of adherence between the lamellae of the nail plate
- Pustules in the periungual skin
    - sterile or reflect infection(S.aureus), but assume infection until proven otherwise.if red and swollen digit → acute paronychia
    - Herpes simplex → pustules (vesicular) are smaller,more numerous and clustered
    - Candida → indolent
    - Sterile pustules → psoriasis, multiple and less pain
- Koilonychia ('spoon-shaped' deformity) → **iron-deficiency anaemia** or lichen planus
- Clubbing
    - chronic swelling of periungual tissues with increased vasculature.
    - Increase in the transverse and longitudinal curvature
    - loss of the angle between the proximal nail fold and the base of the nail.
    - systemic or inherited factors→ affects all nails
    - More in digits of the hands > toes
    - If cyanotic heart disease or fibrotic or cavitating pulmonary disease→ cyanosed nail bed
    - If Idiopathic variants, or IBD and liver disease → normal pink colour nail bed

Nail bed changes

- Leukonychia "Whitening"
    - Apparent leukonychia → vascular "oedema", loss of normal vascular pigment, as in hypoalbuminemia with cirrhosis of the liver., old age
    - True leukonychia → fungal infection,trauma and autoimmune nail disease
        - Mees lines → white transverse band ⇒ chemotherapy
- Red lunula is→ inflammatory joint disease, cardiac failure, blue cyanotic heart or respiratory diseases.
- Grey colour → mepacrine.
- Purple/black discolouration results from subungual bleeding

Nail plate changes

- Patchy brown discolouration → 'yellow nail syndrome', fungal infection and psoriasis
- Tetracycline → yellow \ antimalarials a **blue** discolouration and chlorpromazine a **brown** colour.
- Yellow nail syndrome → respiratory and sinus disease.
- Thickened nail → trauma or fungal infection
- Brown colour or melanonychia refers to a brown streak in the nail due to pigment production in the nail matrix
    - Single → Melanocytic naevi or lentigo,Subungual melanoma,Trauma
    - Multiple → Racial, Addison's, Minocycline and zidovudine
- Hutchinson's sign in which pigmentation extends into the surrounding tissues → sign of subungual melanoma

Common dermatoses and the nail unit

- Psoriasis → common, 80% have nail involvement, pitting, transverse ridges, onycholysis, oily spots, subungual hyperkeratosis
- Eczema → brittle nails, split, Thickening and deformity, transverse ridging. Pitting
- Lichen planus → atrophy of the nail plate, pterygium "cuticle may be thickened and grow over the nail plate"
- Alopecia areata is associated with changes in the nails in about 30% of cases. Features include **ridging**, **pitting**, **leukonychia** and **friable** nails. Where the nails are friable, it is referred to as **trachyonychia** and may involve any or all of the nails – known as 20-nail dystrophy
- Discolouration,Pterygium and friability are associated with **lupus erythematosus**

Infection

Bacterial infection of periungual tissues

- proximal and lateral nail folds are typically affected by S. aureus → tx systemic antibiotics

Fungal nail infection

- Dermatophyte, Associated with trauma, more in toenails
- Tx → terbinafine (systemic)

Nail changes in systemic illness

Acute illness

- transverse line of atrophy → Beau's line
- Shedding of the nail, onychomadesis

Chronic illness

- Clubbing
- Cyanosis
- Splinter hemorrhages → subacute bacterial endocarditis and severe rheumatoid arthritis.

Lesions adjacent to the nail

- Viral warts → most common tumour arising in the nail folds
- Myxoid pseudocysts "Mucoid cyst" → secondary to osteoarthritis
- Nevi
- Melanoma → arise from matrix ⇒ dark longitudinal streak, pigmentation of the cuticle, Hutchinson's sign.
- Subungual exostosis → painful, confirmed by X-ray examination
- Glomus tumours → painful, worse in the cold and at night. → surgical excision
- Periungual fibrokeratoma → if multiple; suspect tuberous sclerosis

Treatment of nail conditions

- Keep nails short, Dry hands and feet
- Wear gloves when undertaking wet work
- Ensure Well-fitting footwear with high 'box' (the space at the end to accommodate the toes).
- emollients to prevent drying
- Tumours require surgical management

Benign Skin Tumours

Differential diagnosis of common benign skin tumours

Clinical features	Differential diagnoses
Pigmented	Seborrhoeic keratoses, dermatosis papulosa nigra, freckles (lentigines), solar lentigo, melanocytic nevus, blue nevus, Mongolian blue spot, dermatofibroma, apocrine hidrocystomas
Vascular	Nevus flammeus, strawberry naevus, port-wine stain, spider naevi, Campbell de Morgan spots, pyogenic granuloma
Papules	Skin tags (fibroepithelial polyps), milia, sebaceous gland hyperplasia, dermatosis papulosa nigra, syringomas, trichoepitheliomas, apocrine hidrocystomas
Nodules	Dermatofibroma, lipoma, angiolipoma, epidermoid cyst, pilar cyst, pilomatrixoma, poroma, intradermal naevus, apocrine hidrocystomas
Plaques	Nevus sebaceous, epidermal nevus, inflammatory linear verrucous epidermal nevus(ILVEN), seborrhoeic keratoses

Definitive Dx → skin biopsy

Malignancy development in benign lesion is suspected if → change in size, colour, border and new satellite lesions ⇒ surgical excision

Pigmented benign tumours

- Seborrhoeic keratoses → trunk, face and neck
    - well-defined edge , color → pale tan to dark brown
    - warty, dull papillary surface, with keratin plugs
    - raised above surrounding skin → 'stuck on,protuberant or pedunculated'.
- Dermatosis papulosa nigra (DPN)
    - multiple small pigmented papules seen on the face of adults with **black** skin
    - very common, familial
    - cheeks, forehead, neck and chest
    - No Tx is needed
- Skin tags (fibroepithelial polyps)
    - at sites of occlusion where the skin may be rubbed by skin or clothing
        - axillae, neck, groin and under the breasts
    - Tx → removed by shave under local anaesthetic
- Lentigines (freckles)
    - solar-induced freckles as 'sun spots' or 'liver spots'.
    - small macular well-demarcated pigmented lesions on sun-exposed skin
    - More in **fair** skin
- Melanocytic naevi → majority are benign
    - **Congenital** melanocytic nevi ('birthmark')
        - Small < 2 cm, medium (2-20cm), giant > 20cm
        - grow in proportion to the growth of the child, color → pale brown to black.
        - hair on it
        - Giant lesions → most likely to undergo malignant change (~5%).
        - Mongolian blue spots→ macular blue-gray and large lesion on the back.
    - **Acquired** melanocytic naevi
        - RF → solar radiation and a genetic susceptibility.
        - Junctional nevi "dermoepidermal junction" → flat brown macules
        - Compound nevi " epidermis and dermis" → raised and pigmented
        - intradermal nevus "only in the dermis" → raised,non-pigmented, on the face
        - Blue nevus → deeply pigmented melanocytes, dark blue
        - Spitz nevus → fleshy pink or pigmented papule in children.
        - Halo nevus → nevus with a surrounding halo of depigmentation
        - Becker's nevus → increased pigmentation and hair
- Dermatofibroma → firm discrete nodules arising in the dermis, on the legs of women.
    - red or light brown ⇒ firm brown papule with a ring of darker peripheral pigment

- itchy or painful.

Benign vascular tumours

- Nevus flammeus neonatorum
  - 'Salmon patches' present at birth most commonly at the (glabella, eyelids→ disappear by the age of 2 years) and nape of the neck (persist for life)
- Port-wine stains + Capillary malformations
  - capillary malformations (not tumor),present at birth, on the head and neck
  - pale pink ⇒ red to purple.
  - increase in size proportionally with the growth of the child and tend to persist
  - unilateral with a sharp midline border
  - epilepsy – Sturge–Weber syndrome
  - Tx → pulsed-dye laser
- Cavernous haemangiomas/strawberry nevi
  - true benign vascular neoplasms → grow out of proportion to the growing neonate
  - Appear at the 1st 3-4 wks , rapidly enlarge at around 6 months of age
  - single (80%) or multiple
  - Soft vascular swelling on the head and neck
  - resolve spontaneously in time and do not require intervention unless recurrently bleeding or interference with visual development.
  - Tx → oral beta blocker (propranolol)
- Spider nevi
  - central vascular papule with fine lines radiating from it
- Campbell de Morgan spots (cherry haemangiomas)
  - Discrete red papules 1–5mm in diameter, in adults, on trunk
- Pyogenic granuloma
  - At digits, capillary haemangioma, not infectious, does not resolve spontaneously
  - easily bleeds → profuse and recurrent
  - removed surgically by curettage and cautery under lignocaine local anaesthetic

Benign tumour papules

- Syringomas
  - **eccrine** glands, multiple, slow-growing, small and **flesh coloured**
  - on the face around the eye around puberty
- Trichoepitheliomas
  - **hair follicle**, multiple, slow-growing, small and flesh coloured
  - On face and scalp
- Milia
  - clog of the eccrine sweat gland.small keratin-filled cyst , **white** papules,on cheek and eyelids of newborns
- apocrine hidrocystomas
  - apocrine secretory glands, papules or nodules around the eyes.dome-shaped
  - solitary or multiple , **translucent** or pale or black (lipofuscin pigment).
- sebaceous gland hyperplasia "SGH"
  - benign hamartomatous enlargement of the sebaceous glands" not a tumour".
  - yellowish, soft, small papules on the face (particularly nose, cheeks, and **forehead**). umbilicated bumps "cauliflower-shaped", swell with sweating (pathognomonic)
  - confused with benign skin tumours and BCC
  - adults of middle age or older. Or in newborns
- Skin tags (fibroepithelial polyps)
- dermatosis papulosa nigra "DPN"
- DDx
  - syringoma and trichoepithelioma are flesh coloured
    - if in the eyelids → syringoma
    - if on cheek around the nose → trichoepithelioma
  - milia is white in color , more in neonates
  - Yellowish umbilicated bumps "cauliflower-shaped" on forehead→ SGH
  - If translucent and on eyelid → apocrine hidrocystomas

Benign tumour nodules

- Lipoma
  - common slow-growing benign subcutaneous tumours of fat
  - soft, movable, and painless
  - congenital or acquired, single or multiple
  - Common locations include upper back, shoulders, and abdomen.

    - Angiolipoma
        - painful.
- Epidermoid cysts (sebaceous cyst)
    - soft, well-defined, mobile swellings usually on the face, neck, shoulders or chest
    - central punctum
    - Mostly asymptomatic , if inflamed or infected → discomfort and discharge (thick yellow , bad odour)
- pilar cyst
    - on the scalp ,very common and frequently multiple. hair follicles
    - No central punctum
- Pilomatrixoma
    - Hair matrix, very hard slow-growing lump,on the head/neck of a child
- Poroma
    - apocrine- or eccrine-derived, painful
- Keloid scars
    - dermal fibroblasts, at the sites of skin trauma, proliferate beyond the site of the injury and they do not regress,unlike hypertrophic scars
- intradermal naevus
- apocrine hidrocystomas
- Dermatofibroma

Benign tumour plaques
- Nevus sebaceous
    - warty, well-defined hairless yellow plaque of 0.5–2 cm in diameter , on the scalp
    - trichoblastoma
- epidermal nevus
    - congenital , linear or clustered ,warty brown papular lesions
- inflammatory linear verrucous epidermal nevus(ILVEN)
    - at birth or appear during the first 5 years of life, most commonly on the lower limb or trunk. warty ,brown , linear or clustered.
    - may become red and inflamed
- seborrhoeic keratoses
- DDx of plaques
    - Scalp + warty → Nevus sebaceous
    - warty brown papular
        - not inflamed → epidermal nevus
        - inflamed → inflammatory linear verrucous epidermal nevus(ILVEN)

Benign painful tumours in the skin: ‘BENGAL’
- Blue rubber bleb nevus
- Eccrine spiradenoma
- Neurilemmoma/neuroma
- Glomus tumour
- Angiolipoma
- Leiomyoma

Premalignant and Malignant skin lesions

Premalignant skin tumours

- uncontrolled proliferation of differentiated cells , whereas, Malignant → uncontrolled proliferation + complete undifferentiation “dysplastic”
- Actinic keratoses
    - Distribution → over sun exposed skin→ face (including the lip), dorsal hands, distal limbs and bald scalp
    - RF → fair/sun-damaged skin and increasing age
    - Morphology → patch of thick, scaly, or crusty skin. dark, light, tan, pink, red, a combination of all these, or have the same color as the surrounding skin.
        - irregular edge,less than 1 cm in diameter
        - Classic AKs → white, scaly macules, papules or plaques of various thickness, with surrounding erythema
    - asymptomatic
    - Px → 20% risk of progression to squamous cell carcinoma if untreated
    - Management
        - Procedures → liquid nitrogen (**cryotherapy**,70% cure rate), Photodynamic therapy (PDT)
        - **Medication → 5-Fluorouracil (5-FU)** 5% cream, Imiquimod 5%,Topical NSAID diclofenac
        - Surgical techniques → Excision ,Shave excision & curettage,Dermabrasion

        - If no response → Bx to check for invasive malignancy
- Bowen's disease "SCC in situ"
    - elderly ,on the trunk and limbs
    - RF → solar radiation,HPV 16 , radiotherapy, arsenic ingestion
    - Morphology →well-defined, erythematous patches with slight crusting
    - If in the glans penis or prepuce → Erythroplasia of Queyrat
    - Ddx → superficial BCC, eczema
    - Dx → **Skin biopsy**
    - Management → excision, curettage and cautery, cryotherapy, 5-FU,imiquimod 5% and PDT

Malignant skin tumours

- Basal cell carcinoma (BCC)
    - most common cancer in humans.lifetime risk of around 30%
    - RF → age, fair skin, high-intensity UV exposure, radiation, immunosuppression, previous BCCs
    - Sun-exposed skin in the 'mask area" of face , Painless
    - Morphology →shiny,translucent pearly skin nodule or red patch or 'rolled edge' ulcer
        - Colour → clear to deeply pigmented.
    - Clumps of dysplastic basal cells form nodules that expand and break down → rolled edge ulcer
    - BCC types
        - Nodular → small pearly papules or nodules, rolled edge ulcer, telangiectasia. On sun-exposed areas of the head and neck
        - Superficial → erythematous patch, on trunk
        - Pigmented
        - Morphoeic or sclerosing → superficial atrophic scar, loss of the normal skin markings and the indistinct edge
        - Rodent ulcer→ central necrosis.
        - Management
            - Depends on Histology, Bx before Tx
            - Face or Morphoeic or sclerosing → Mohs' surgery "'gold standard"
            - excision (including Mohs' surgery), excision and grafting, curettage and cautery, radiotherapy, cryotherapy, imiquimod 5% and PDT for large superficial BCCs
- Squamous cell carcinoma (SCC)
    - develops in previously normal skin or pre-existing lesions such as actinic keratoses or Bowen's disease or in chronic wound or scar (Marjolin's ulcer)
    - 2nd most common skin CA, HPV and chronic scar is a RF, mostly in head and neck
    - Symptoms → rapidly growing,painful and hyperkeratotic
    - Morphology →nodular,crusting, ulceration (everted ulcer) or cutaneous horn
    - On sun-exposed areas (dorsal hands, scalp, lip, and superior surface of pinna)
    - DDx→ Keratoacanthoma, actinic keratosis, melanoma, warts, basal cell cancer
    - SCC types
        - Keratoacanthoma (central crater keratin filled within the nodule)
    - Management of SCC
        - Surgical excision (4–6mm margin) with or without Skin grafting, or curettage and cautery or radiation therapy, chemotherapy
    - Good Px
- Moles/nevi: benign or malignant?
    - The **ABCDE** acronym→ assessing the malignant potential of a mole: asymmetry, border (irregular), colour (irregular,variation), diameter (>0.5 cm), evolving
        - Symptoms → pain, crusting, ulceration or bleeding or discharge
- Dysplastic "atypical" nevi
    - deeply pigmented ,irregular margin, ('funny-looking moles').
- Melanoma
    - Incidence → increase
        - 4% of skin tumours,75% of skin cancer deaths
        - white adults, >30 years
        - males→ trunk, females → legs
    - RF ⇒ Sun exposure (major) , fair-coloured hair, light-coloured eyes, female sex, older age, a personal or family history of melanoma and congenital defect of DNA repair (xeroderma pigmentosum).

- Pre-existing moles
- Types of melanoma
  - Superficial spreading melanoma → most common,back in men,legs in women, irregular margin, brown to black pigmentation , surrounding inflammation or pale, nodules (worse Px)
  - Lentigo maligna melanoma → on face,elderly
    - single or multiple solar lentigos → irregular ,larger pigmented macule (lentigo maligna,tx;imiquimod) → if darker\nodule → Lentigo maligna melanoma
  - Nodular melanoma → dark nodule from the start, vertical growth (worse Px)
  - Acral melanoma→ palm and soles and near/under the nails
    - Hutchinson's sign → Melanonychia ; black or brown pigmentation of nail fold adjacent to the nail
  - Amelanotic melanoma → non-pigmented nodules
  - Dysplastic malignant melanoma
- Prognosis → depends on the depth of invasion + LNs, ulcers, mets
  - If not Tx → melanoma satellites (small islands of melanoma nearby) and local Mets → distant mets "hematogenously, lymphatics"
- Treatment
  - Excise with 2 mm margin → Breslow thickness
  - lymph node involvement → fine needle aspiration or lymph node removal for cytology/histology
  - If Breslow thickness >1mm→ Sentinel lymph node biopsy (SLNB)
  - wide local excision
  - Adjuvant therapies→ for stage 4 (mets or satellites)
    - Chemotherapy
    - Immunotherapy "interleukin 2 (IL-2)"
    - Targeted therapy for the gene mutation in BRAF
    - Melanoma vaccines

- Cutaneous lymphoma
  - Cutaneous T-cell lymphomas (CTCLs) → 80%
    - Mycosis fungoides (MF) → MC form , increase with age, male, blacks
      - scaly erythematous patches and plaques on trunk and buttock, don't respond to topical steroids or antifungal ⇒ nodules
      - Sézary syndrome "exfoliative erythroderma with lymphadenopathy" → aggressive form of MF
    - Management
      - Early → potent topical steroids, topical nitrogen mustard and (PUVA).
      - Plaque or tumour → localised radiotherapy, rexinoid
  - cutaneous B-cell lymphomas (CBCLs)→ 20%
    - Firm indurated papules, nodules or plaques . erythematous, violaceous or brown.
    - Management
      - low grade solitary lesions → surgical excision, localised radiotherapy, intralesional interferon alpha or intralesional rituximab
      - Multiple lesions → systemic rituximab
      - High grade → doxorubicin or CHOP chemotherapy
- Paget's disease of the nipple
- Cutaneous metastasis → Metastases from internal organs
  - breast, lung,GI tract, renal tract, oral pharynx, larynx and melanoma (originating from the retina and leptomeninges

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Sexually Transmitted Diseases

Bacterial Infection	Viral infection
1- Gonorrhea. سيلان 2- Chancroid القُرَيْح أو قرحة لينة 3- Lymphogranuloma Venereum 4- Granuloma Inguinale 5- Syphilis الزهري	1- HSV 2- HPV

Viral Infection :

Herpes infection (HSV 1 + HSV 2)

- Enveloped dsDNA virus .
- Usually the genital infection caused by HSV 2 more than HSV 1
- Clinical pattern :

Primary infection	latent infection	2nd or reactivation infection
First time of infection	-Asymptomatic stage -Virus dormant in **dorsal ganglion.** - positive serology	A- **A**symptomatic (virus in shedding phase, most of transmission happens during this stage) B- **Symptomatic** .

Primary infection	Recurrent infection
1. More in number 2. constitutional symptoms. 3. More painful	1. Less in number. 2. NOT associated with constitutional symptoms. 3. Less painful. 4. Preceded by burning and tingling sensation

- HSV1 transmitted by body fluid ,HSV2 transmitted through epidermis layer with abrasion.
- Incubation period of most STD is less than 7-10 days EXCEPT:
    - 1- Syphilis up to 3 months 2- Granuloma inguinale up to 1 year .
- Site of infection :

Males	Females
Penile ,Glans “head” penis Pubic area	Vulva,Labia majora Pubic area,Buttocks

- Clinical scenario :
  1- Constitutional symptoms. → fever, fatigue, HA
  2- Tingling sensation at the site of incubation of virus.
  3- Skin lesion (Herpetic form vesicle)
  4- Within few days- weeks erosions appear forming (Painful, superficial erosions with scalloped borders) .
- Dx
  1-Tzanck smear (multinucleated giant cells, inclusion bodies called Cowdry A body)
  2- DFA (direct fluorescent Ab, green fluorescence sensitive for serotype HSV 1 & 2)
  3- **PCR**: Most sensitive , most specific
  4- Viral culture .

- Tx
    - Goals of tx
      1- limits viral shedding. 2- Decrease pain.
      3- shortens the duration of clinical manifestation
    - Acyclovir or Valacyclovir
        - primary infection for 10 days
        - recurrent infection for 5 days

Human papillomavirus HPV

- Non-enveloped dsDNA virus .
- Transmitted mainly by close contact and sexual intercourse.
- HPV classified according to the risk of malignancy :

low risk virus	High risk virus
HPV 6 HPV 11	HPV 16, 18, 31, 33 E6 & E7 are the carcinogenic proteins in high risk group

- Clinical presentation of genital warts :

1- Red, Brownish , Erythematous papules with rough keratotic surface (Typical presentation)

2- condyloma acuminatum → Rough keratotic skin or hyperpigmented brownish plaque on skin fold

- Condolymata acumninata → HPV
- Condylomata lata → 2nd syphilis
- 75% of genital warts are self-limiting within 2 years.

Tx of HPV

- Destruction
  - Physical destruction by **cryotherapy** (very painful)
  - Cytotoxic agent → Podophyllotoxin, imiquimod 5%
  - Green tea catechins
  - Cidofovir gel
  - IFN- alpha (painful)
- Systemic "oral" → if extensive or failed topical Tx
  - Isotretinoin
  - Cidofovir

Bacterial Infections

Syphilis :

-Causative agent is spirochete Treponema Pallidum

- Incubation period up to 3 months

-transmitted through sexual intercourse, transplacental spread and unscreened blood transfusions

-Types:

1- Primary syphilis 2- Secondary syphilis 3- Latent syphilis

4- Tertiary syphilis 5- Congenital syphilis

types	manifestation
Primary syphilis	Chancre ;painless genital ulcer at the site of inoculation
Secondary syphilis	widespread eruption of red-brown scaly patches and macules that affects the trunk and limbs (particularly palms and soles)
Tertiary syphilis	Gumma

Primary syphilis :

- Solitary, painless, firm, indurated ulcer on genital area or extra genital area ;lips and fingers
- Number 3 → appears in 3 wks, heals in 3 wks, 2nd in 3 wks-3 months
- Lesion in this stage called **Chancre** , which appears within the first 3 weeks of infection and heals within another 3 weeks.even in the absence of treatment
- Within the next 3 weeks- 3 months hematogenous dissemination → 2nd syphilis.
- Diagnosis:
  1- Dark field microscopy (most sensitive)
  2- RPR /VDRL → Rapid Plasma Reagin\ Venereal disease research laboratory test
  3- Treponemal Ab (less sensitive +ve only in 80%)

Secondary syphilis

- Early sign of secondary syphilis is **Rose colored macules** usually on **shoulders** and flanks
- Then papulosquamous lesions appear.
- **palmoplantar** manifestation (Copper- pin spots , bronze colored ,surrounded by collarette scales) this stage is highly contagious.
- **Condylomata lata** one of the forms of secondary syphilis (moist colored skin)
- patchy alopecia may complicate the infection .
- **Necklace of venus** may be seen (Hypopigmented area on neck) → background hyperpigmentation with superimposed white macules on neck associated with syphilis

Latent syphilis :

- After complete resolution of secondary syphilis asymptomatic period with +ve serology starts called latent period

Early latent	Late latent
1-Starts **within** one year after resolution of 2nd syphilis 2- Most of the **relapses** of 2nd syphilis happen during this period.	1-Starts **after** one year after resolution of 2nd syphilis 2- Proceed either into complete **cure** or into **tertiary** syphilis.

Tertiary syphilis

- Most common cutaneous manifestation :
  - 1- Benign **Gumma** in up to 50% of cases, appear in any organ .
  - 2- Cardiovascular syphilis in 25 % of cases in a form of aneurysms .
  - 3- Neurosyphilis in 25% of cases .
- Gumma : Archiform erosion with depigmented atrophic center
  - firm, necrotic center surrounded by inflamed tissue

Congenital syphilis :

Early congenital syphilis	Late congenital syphilis
1. Age less than 2 years 2. Median age 3 months 3. Present with : a. Snuffles, Bullae, Perioral fissures .	1. Age preschool 2. Present with skeletal deformities 3. Hutchinson's triad a. Interstitial keratitis b. Teeth abnormalities c. Deafness

Treatment → Benzathine penicillin

- Primary + Secondary + Early latent: → 2-4 IU *1 IM
- Late latent + tertiary→ 2-4 IU *1 \ week for 3 wks
- neuro or ocular manifestations → 4 IU Q 4 hrs for 14 days .

Gonorrhea

- Gram negative diplococci, intracellular bacteria.
- Transmission :
  - 1- In adults → by sexual contact.
  - 2- In neonates → by vertical transmission result in Ophthalmia neonatorum (bacterial conjunctivitis causes blindness)
- Incubation period: 2-5 days .

- presentation :

Males	Females	Neonates
-Anterior urethritis (Purulent discharge from anterior urethral meatus. -Epididymitis. - Orchitis. -Prostatitis.	- 50% are asymptomatic -Most common site is endocervix - Salpingitis	- Opthalmia neonatorum.

- Dx → Gram stain, Culture, PCR, DNA probes
- Tx → Ceftriaxone, Azithromycin

Chancroid

- Causative agent is **Haemophilus ducreyi**
- Incubation period 3-10 days.
- Always symptomatic. (start as multiple painful purulent ulcer with undermined edges)
- Gram stain → chaining pattern characteristic to haemophilus ducreyi
- Tx → Ceftriaxone, Azithromycin

Chancroid	Chancre lesion of primary syphilis
1. Painful. 2. Multiple. 3. unilateral Enlarged tender L.N 4. grey or yellow purulent exudate 5. soft base with undermined edges	1. Painless. 2. Solitary 3. bilateral Enlarged Non-tender L.N 4. exude serum 5. hard (indurated) base with sloping edges

lymphogranuloma Venereum

- Causative agent is **Chlamydia trachomatis** serotype L1-L3
- Infects lymphatic tissue in genital area .
- Starts as **papules** that ulcerate later on (few days- one to two weeks)
- 1-6 months later it become enlarged tender lymph nodes with pus discharge & pus draining sinuses called (PUPO) single L.N with sinus .
- Dx → Serology ,PCR

- Tx → Doxycycline . If pregnant→ erythromycin

Granuloma Inguinale

- Causative agent is **Klebsiella granulomatis** , gram negative bacilli
- Incubation period 1 day -1 year.
- Starts as painless beefy red ulcerated papules or nodules with foul smelling discharge
- Extragenital involvement in bones
- Dx → Giemsa , wright , Leishman stain for **Donovan Bodies**
- Tx → Doxycycline or azithromycin or trimethoprim-sulfamethoxazole

Lesion or disease	Causative agent	Tx
lymphogranuloma Venereum	Chlamydia trachomatis serotype L1-L3	Doxycycline, erythromycin "pregnant"
Granuloma Inguinale Donovan Bodies	Klebsiella granulomatis	Doxycycline or azithromycin
Chancroid	Haemophilus ducreyi	Ceftriaxone, Azithromycin
Chancre	Primary syphilis (Treponema Pallidum)	Benzathine penicillin
Condylomata lata	2nd syphilis (Treponema Pallidum)	
Gumma	Tertiary syphilis (Treponema Pallidum)	
condyloma acuminatum	HPV	cryotherapy or cidofovir
Gonorrhea		Ceftriaxone, azithromycin

Ichthyosis السُماك

Definition: Excessive generalized dryness and scales of the skin.

- Recent advances include the identification of two loss-of-function **filaggrin gene** defects that lead to dysregulation of keratohyalin synthesis manifesting as ichthyosis vulgaris (IV) (which affects 1 in 250 individuals)

Classification of ichthyosis :

1. **congenital** ichthyosis
2. ichthyosis associated with **syndromes**. → Netherton's syndrome, SjOgren-larsson, Refsum's syndrome, KID syndrome,Kallman's syndrome
3. **Acquired** ichthyosis

CONGENITAL ICHTHYOSIS:

1- ichthyosis **Vulgaris**
2- **X- linked** recessive ichthyosis.
3- **NBIE** (Non-Bullous ichthyosis Erythroderma) 4- **Bullous** ichthyosiform Erythroderma .
5- **lamellar** ichthyosis

ichthyosis Vulgaris :

- Autosomal **dominant** disorder.
- **Most common** type of all types of ichthyosis.
- Presentation
  - starts after the 2nd or 3rd months of age , sometimes it may delay up to or after the 1st year of life.
  - You may see dandruff on the head due to **excessive scales**
  - Commonly associated with Keratosis pilaris. "common, harmless skin condition that causes dry, rough patches and tiny bumps, usually on the upper arms, thighs"
  - Involves the **extensors** WITH **sparing** of **flexors**.
  - Minimal itching.
  - You may find perioral and ear pinna scales.
- DDX:
  - Other types of ichthyosis.
  - Atopic dermatitis. /// Asteatotic eczema.
- Description of scales :Small, Whitish, Branny "cereal" semi adherent scales.

X-linked recessive ichthyosis.

- Starts at the In week of age.
- Mostly on extensor but it may affect body flexors.
- Associated with extracutaneous manifestations
- There is a mutation on steroid sulfatase gene.

- DX : definitive dx by chromosomal analysis.

Acquired ichthyosis

- May be considered as **paraneoplastic** features (lymphoma, Leukemia) .
- Associated with c**hronic renal and hepatic** diseases.
- Associated with **drugs** (especially Lipid lowering agents)
- Associated with **thyroid** and **parathyroid** diseases in young age group
- **Malabsorption** syndromes.
- Sarcoidosis,Leprosy, AIDS.

Mx

- Congenital → supportive
- Acquired → supportive + tx underlying disease
- Supportive care
  - Topical treatment → Emollient, Keratolytics (Urea containing preparation)
  - Systemic Retinoid

Dermatology Hx taking & Physical Exam

Dermatology history-taking

1. PC.DOT
   a. Onset → sudden or gradual
   b. Duration "How long"
   c. Timing → previous infection or Contact history with infectious skin problem, sun exposure, sting or bite, food, trauma
      i. Precipitating factors, Relieving factors
      ii. Food, heat/cold, sun, medications, insect stings,animal contact, physical stimuli, infections, stress (emotional or physical)
   d. Progression "Trend" → better or worse?
   e. Course → continuous or intermittent?
2. Where "distribution" → site of initial lesion(s) and subsequent distribution
3. Symptoms
   a. Pain, Itching, burning
   b. scaling, or blisters
   c. Bleeding, Discharge
   d. Food allergy → N\V, D\C, abdominal pain
   e. Respiratory distress
   f. Systemic symptoms → fever / malaise / weight loss / arthralgia
4. PMH
   a. Previous episodes – timing? Similar/dissimilar?
      i. Recurrence
   b. Skin disease → Skin cancer, Atopy – eczema / hay fever / asthma
   c. Other medical conditions with dermatological manifestations
      i. Diabetes – acanthosis nigricans / scleroderma diabeticorum / necrobiosis lipoidica diabeticorum
      ii. Inflammatory bowel disease – pyoderma gangrenosum / erythema nodosum
5. DH
   a. Any medication for other diseases
   b. Treatment – prescription or over the counter? Frequency/time course/compliance?
   c. Drugs that trigger psoriasis → β-blockers, lithium, antimalarials
6. FH
   a. Who else – Family members/work colleagues/school friends affected?
7. SH
   a. Occupation (exact details of the patient's job)
   b. Recent emotional or physical stress
   c. Smoking, alcohol
   d. Recent → travel, animal contact, contaminated water contact, sick contact

Describing a lesion in dermatology

1. **General inspection**
   a. **distribution** and number of the lesions
      i. Generalized
      ii. **Symmetrical** or not

iii. **Periorificial** → Around the orifices of the body
iv. **Periungual**/subungual
v. Photodistributed
vi. **Acral** – affecting distal areas, hands and feet
vii. **Extensor** – extensor surfaces, elbows, knees
viii. **Flexors** – flexors surfaces, axilla, cubital or popliteal fossa,genital areas
ix. **Follicular** – arising from hair follicles
x. **Seborrhoeic** – associated with areas where there are sebaceous glands, face and scalp
xi. **Dermatomal** – corresponding with nerve root distribution

2. **Close inspection** of individual lesions → Size, shape ,discrete or confluent,Borders, Colour, Morphology " flat, raised , depressed" → primary and secondary lesions
   a. Size → width/height (if raised)
   b. shape → linear, discoid, target, annular
      i. **Linear** lesions – e.g. scratching related lesions
      ii. **Discoid** (coin shaped) – discoid eczema/discoid lupus
      iii. **Target** lesions – concentric rings of varying colour ,resembles a bullseye , multiple rings inside each other , ex erythema multiforme
      iv. **Annular** – ring like lesions
      v. **Reticulate** → net-like
   c. Discrete or confluent
      i. **Discrete** lesions – individual lesions, clearly separated from one another
      ii. **Confluent** lesions – lesions that appear to be merging together
   d. Borders → well defined vs poorly defined
   e. Colour
      i. **Red**
         1. **Erythema** → red, blanchable ,Caused by increased blood supply
         2. **Purpura** → Reddish/purple, not blanchable , bleeding into the skin
            a. **Petechiae** – small red/purple spots (< 2 mm in width)
            b. **Ecchymosis "**bruise"– larger red/purple lesions (>2mm)
      ii. **Brown**
         1. **Hyperpigmentation "Brown"**→ diffuse or focal
         2. Hypopigmentation → paler skin
      iii. White
         1. Depigmentation → absence of melanin within the skin,skin appearing completely white → ex; Vitiligo
      iv. **Yellow**
         1. Lipid deposition- xanthelasma (cholesterol deposits around the eyelids) and xanthomata (cholesterol deposits in the tendons) can occur in hyperlipidaemia disorders.
         2. **Bilirubin-** yellow discolouration due to jaundice (which is defined as bilirubin >35µmol)

f. Morphology → flat, raised , depressed
   i. Primary lesions
      1. **Non raised** "without elevation or depression"
         a. **Macule** – a flat area of altered colour **<2cm** in diameter
         b. **Patch** – a flat area of altered colour **>2cm** in diameter

      2. **Raised lesion**
         a. **Papule** – solid raised palpable lesion **<1cm** in diameter
         b. **Nodule** – solid raised palpable lesion **>1cm** in diameter
            i. Haemangioma → vascular papule or nodule

         c. **Plaque**:
            i. flat, elevated palpable lesion usually **>1cm** in diameter
            ii. مسطحه كالهضبه
            iii. most are raised, but some may just be thickened without being visible raised
            iv. circumscribed, superficial, elevated plateau area 1.0–2.0 cm in diameter
            v. Macule and patch are not palpable, only altered color
      3. **Fluid-filled lesions**
         a. **Vesicle** – raised, clear fluid filled (blisters) lesion **<1cm** in diameter

b. **Bulla** – raised, clear fluid filled (blisters) lesion **>1cm** in diameter

c. **Pustule** – superficial pus containing lesion <0.5cm in diameter
d. **Abscess** – deep pus containing lesion, >0.5cm in diameter

e. Boil / **furuncle** – staphylococcal infection around or within a hair follicle
f. **Carbuncle** – red, swollen, and painful cluster of boils (multiple boils/furuncles)

4. Wheal – oedematous papule or plaque , edema in dermis
5. Cyst: Epithelial-lined cavity containing liquid, semi-solid, or solid material
6. Telangiectasia → enlargement of superficial blood vessels to the point of being visible

ii. **Secondary lesions** → modifications of primary lesions, due to trauma to, or evolution of, the primary lesion.
i. **Atrophy** → thinning in the epidermis/dermis
1. **Excoriation** –Partial or complete loss of epidermis as a result of scratching
2. **Lichenification**:
a. **Thickening** of the **epidermis** seen with exaggeration of normal skin lines
b. It is usually due to chronic rubbing or scratching of an area
3. **Induration**:**Thickening** of the **dermis**
4. **Scales**:
5. **Visible fragments** of the stratum corneum
6. Epidermal cells produced by abnormal keratinisation of the skin which have died and then been shed.
7. Most commonly associated with psoriasis
ii. **Desquamation** → peeling of superficial scales
iii. **Crust**:
1. **Rough surface** consisting of dried serum, blood, bacteria and cellular debris
iv. **Erosion**: Discontinuity of the skin exhibiting **incomplete** loss of the epidermis, heals without scarring
v. Ulcer
1. Localised defect in the skin of **irregular** size and shape , **complete** loss "discontinuation" of the epidermis and the upper dermis
2. Healing results in a scar
3. **Arterial** ulcer- caused by ischaemia and are usually located on the lateral aspect of the ankle or distal ends of the digits of the lower limbs
4. **Venous** ulcer- due to valvular insufficiency of the veins
5. **Neuropathic** ulcer- related to sensory loss in the lower limbs, most common in diabetes
vi. **Fissure**:
1. **Sharply-defined**, linear or wedge-shaped tears in the epidermis with abrupt walls, narrow but deep
2. Usually due to excess dryness
vii. **Scar** → New fibrous tissue which occurs after skin injury
1. **Atrophic** scarring – **thinning** of the normal tissue
2. **Hypertrophic** scarring – **hyperproliferation** of scar tissue **within** the wound boundary
3. **Keloid** scarring – **hyperproliferation** of scar tissue **beyond** the wound boundary

viii. **Striae**:
1. Often referred to as **stretch marks**
2. Evolution in colour = Purple → Pink → White
3. Associated with growth spurts, excess steroid use or production and pregnancy

Pigmented lesion → ABCDE

Asy**mmetry** more suggestive of unfavorable pathology
Border irregularity
Colour variation or changes:

- **Two** or more colours within one lesion is more suggestive of unfavorable pathology

Diameter:

- change in size of the lesion?Increasing size, particularly over **6mm** diameter

Elevation/evolution:

- Changes in colour, size, symmetry, surface characteristics, and symptoms.

- Symptoms include itching, bleeding and scabbing of the lesion

Then inspect the rest of skin, and palpate local LN

Palpation

- surface characteristics
  - Texture – smooth/rough
  - Flat, raised or depressed?
  - Crust – if present, are you able to remove crust and see what is underneath?
  - Temperature – is the lesion warm?
- Assess deeper characteristics of the lesion
  - Consistency – hard/soft/firm/fluctuant
  - Mobility – is the lesion attached to the underlying/overlying tissue?
  - Tenderness

Systemic examination → Hair, elbows, hands and Nails

- Hair and scalp
  - **Loss** of hair
    - Alopecia **areata** – well defined patches of hair loss with surrounding normal hair
    - Alopecia **totalis** – loss of all hair from the scalp
  - **Excess** hair
    - **Hirsutism** – androgen dependent excess hair growth in females
    - **Hypertrichosis** – non-androgen dependent excess hair growth
  - **Scalp**
    - **Psoriasis** plaques
    - **Dandruff** – e.g. seborrheic dermatitis
- Elbows
  - **Xanthomas** → hyperlipidaemia
  - **Psoriasis** plaques on elbows
- Nail
  - Nail pitting
    - **Punctate depressions** of the nail plate
    - Associated with eczema, psoriasis and alopecia areata
  - Onycholysis
    - **Separation** of the distal end of the nail plate from the nail bed
    - Associated with psoriasis and fungal nail infection
  - Koilonychia:
    - **Spoon shaped** indentation of the nail plate
    - Associated with iron deficiency anemia, can also be congenital
  - Nail clubbing:
    - Loss of the angle between the posterior nail fold and nail plate
    - Associated with inflammatory bowel disease, cyanotic heart disease, lung cancer, bronchiectasis
- Mucous membranes
  - Inspect oral mucosa to evidence of skin disease (e.g. pigmented lesions/bullae)