Chapter2
Function & structure

Skin is:

- The Largest organ in the body.
- Weighs 4 kg & covers area of 2 m^2.

Skin functions are:

1. Protection: chemical (horny Layer), UV radiation (melanocyte), antigens & Haptens (Langerhans cells).
2. Preservative of internal environment: horny layer.
3. Shock absorber: dermis & SC Fat.
4. Temperature regulation: blood vessels, eccrine sweat glands.
5. Body odors\ pheromones: apocrine sweat glands.
6. Insulation: SC fat.
7. Sensation: nerve endings.
8. Lubrication: sebaceous glands.
9. Protection: nails.
10. Calorie reserve: SC fat.
11. Vit D synthesis: keratinocytes.
12. Psychosocial\display: lips, skin hair & nails.

Skin has two layers:

outer epithelial epidermis & underlying dermis, below dermis, a loose CT contains abundant fat.

1- The Epidermis:

Superficial layer is flattened & filled with Keratins (stratified squamous epithelium) adhere to dermis by interlocking of its projections (epidermal ridges of pegs) with dermal papillae.
No blood vessels, thickness from 0.1mm on eyelids to 1mm on palms & soles thickness kept although of the shedding by constant dividing of cells in the deepest germinative or basal layer.

Basal layer--- prickle layer--- granular layer--- horny layer

Journey from the basal layer to the surface last 60 days.

Basal layer (deepest layer) rest on BM which attach it to dermis, it is single layer of columnar cells that sprout many fine processes & hemidesomosomes anchoring them to lamina densa of BM.

In normal skin, 80% of basal cells are preparing for division (growth fraction) & after replication, one of the daughter cell move into the suprabasal layer.

Keratinocytes:

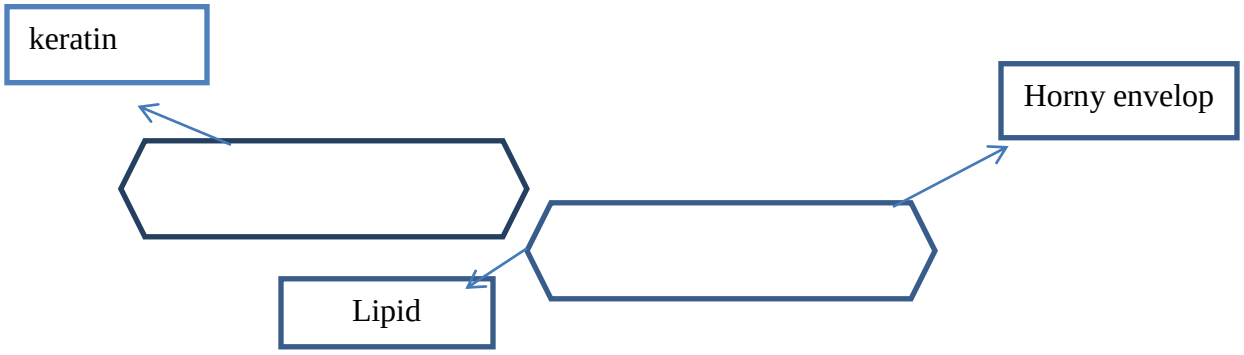
Spinous or prickle cell layer composed of keratinocytes which synthesize keratin & larger than basal cells.
Keratinocytes attached to each other by:

- Small interlocking cytoplasmic processes
- Desmosomes.
- Intracellular cement of glycoproteins & lipoproteins.

These bonds (prickles) contain desmoplackins, desmogleins & desmocollins &autoantibodies to these proteins responsible for detachment of keratinocytes & so interepidermal blister formation (pemphigus).

- Cytoplasmic continuity between keratinocytes occur at gap junctions.
- Tonofiliments small fibers running from the cytoplasm to the desmosome. More numerous in Spinous layer cells than of basal layer & packed into tonofibrils bundles.
- Lamellar granules (Odland bodies or keratinocytes) derived from Golgi apparatus appear in superficial keratinocytes of this layer. Contain polysaccharides, hydrolytic enzymes & stacks of lipids lamellae which all discharged into intracellular space of granular cell layer to become precursors of lipids in the intercellular space of horny layer.
- Granular layer consist of 2-3 layers of cells that are flatter than Spinous layer with more tonofibrils.

- Contains large irregular basophilic granules of keratohyalin, which merge with tonofibrils.
- As keratinocytes migrate out, their keratohyalin granules break up &their content are dispersed throughout the cytoplasm leading to keratinization & formation of thick and tough peripheral protein coating called the horny envelop.
- Horny layer (stratum corneum) made of piled-up (كالنسيج) layers of flattened dead cells (corneocytes) which stuck together by lipids in intercellular space.



- Lipid (lipid lamellae),horny envelop (keratinohyalin granules)
- Skin withstand all sorts of chemical & mechanical insults due to horny envelop & aggregated keratin
- No nuclei or intracytoplasmic organelles which destroyed by hydrolytic &degrading enzyme.

Keratinization:

- Keratins are the main intermediate filaments in epithelial cells
- Family or > 30 proteins produced by different genes separate into two gene families which are basic and acidic keratins
- Keratin polypeptide has central helical portion with non-helical N-terminal head &C-terminal tail. Individual keratins exist in pairs so that their double filament always consists of one acidic & one basic keratin polypeptide.
- Different keratins found at different levels of epidermis & diseases:

- Keratins 5&14: normal basal cells.
- Keratins 1&10: suprabasal cells.
- Keratins 6&16: hyperproliferative states as Psoriasis.

Cell cohesion & desquamation:

- Firm cohesion in Spinous layer ensured by stick &grip mechanisms
  - **Stick:** glycoprotein intercellular substances
  - **Grip:** intertwining of the small cytoplasmic processes with desmosomal detachments.

- Cells deep in horny layer stick tightly together &only those at the surface flake off which in part caused by activity of cholesterol sulphatase that is deficient in X- linked recessive.

  Ichthyosis (congenital disorder of keratinization characterized by dryness and fishskin-like scaling of the skin).

- Desquamation normally responsible for removal of harmful exogenous substances from the skin surface.

Epidermal Barrier :

- Horny layer prevent loss of interstitial fluid from within , and act as barrier to penetration of potentially harmful substances from outside.
- Essential fatty acid deficiency , hydration , dehydration , detergents causes poor cutaneous barrier function.
- Rate of substance penetration through epidermis is proportional directly to concentration difference across the barrier layer , & indirectly to thickness of horny layer.

- water penetrate slightly , Ions impermeable [Na^+, K^+, glucose , urea], but aliphatic alcohols pass through easily .
- Rise in skin temperature aids penetration.

Epidermopoiesis & it's regulation :

- Locally produced [1]polypeptides (cytokines), [2]growth factors &[3] hormones stimulation or
inhibit epidermal proliferation , interact in complex way to ensure homeostasis (new & lost
cells).

- cytokines & growth factors produced by [1] keratinocytes , [2] langerhans cells , [3] fibroblasts
and [4] lymphocytes within the skin.

- steroid hormones influence transcription.

Vitamin D synthesisi:

The steroid 7-dehydrocholesterol found in keratinocytes converted by sunlight to cholecalciferol & then become active after 25-hydroxylation in the kidney.

Melanocytes:

- Keratinocytes make up 85% of cells in epidermis, but other 3 types of cells present which are
Melanocytes , Langerhans cells & Merkel cells.
- Melanocytes are only cells that synthesize melanin & found also in the hair bulbs , retina & pia
arachnoid .
- Each dendritic melanocyte associate with # of keratinocytes forming " epidermal melanin unit " .
- their cytoplasm contains discrete organelles, the melanosomes , containing varying amount of the
pigment melanin.

Melanocytes:

1- Denderitic
2- mostly basal
3- No desmosomes.
4- contain melanosomes.

Langerhans cells :

- Denderitic cells , no desmosomes or tonofibrils , but has lobulated nucleus.
- Come from bone marrow , 800 cellss per mm^2 in human skin.
- Possess surface receptors for C3b and Fc portions of IgG & IgE , MCH class Ⅱ antigens.
- Play a role in immune reactions against exogenous antigens , viral & tumor antigens.
- Skin tumor causes mutations in epidermal cells & decrease # of langerhans cells.
- Langerhans cells are the principal in skin allografts & steroids reduce their density.

Langerhans cells:

1- Denderitic
2- Suprabasal
3- No desmosomes
4- contains characteristic cytoplasmic organells.

Merkel cells :

- Transducers for fine touch , non-denderitic cells lying in or near the basal layer & of same size of keratinocytes .
- Concentrated in localized thickenings of epidermis near hair follicles (hair disc), and contain membrane – bound spherical granules.

- Fine unmyelinated nerve ending are often associated with Merkel cells.

Merkel cells :

1- No dendritic
2- Basal
3- contain Desmosomes
4- contain neuro-secretory granules needed For fine touch.

The Dermo-epidermal junction :

- Basement membrane lies at the interface between the epidermis & dermis.
- figure 2.9 page 15.

2- The Dermis:

- Lies between epidermis & SC fat , support the epidermis structurally & nutritionally .
- Greatest thickness in palms & soles , least in eyelids & penis with lost of elasticity in old age.
- Cells of dermis & their functions:

Cell	Function
Fibroblast	Synthesis of collagen , reticulin , elstin, fibronectin
Monocular phagocyte	Mobile , phagocytosis , secrete cytokines
Lymphocyte	Immunity
Langerhans & dermal denderitic cells	In transit between L.N & epidermis , antigen presentation
Mast cells	Release inflammatory mediators

Fibers of the dermis :

- Dermis largely made of collagen packed in bundles , which prevent tearing of skin when strongly stretched , & then with elastic fibers return skin to it's unstretched state.
- Collagen make 70-80% of dry weight of the dermis . collagen chains stabilized by cross-links involve lysine & hydroxylysine.
- Elastic fibers account for 2% of the dry weight of adult dermis.
- Defects in enzyme needed for collagen synthesis cause some skin disease as Ehler-Danlos syndrome .
- Abnormalities in the elastic tissue cause cutis laxa & pseudoxanthoma elasticum .

*** Collagen :** prevent tearing of skin when stretched ,,,,,,, 75% of dermis weight.

*** Elastin**: return skin to unstretched state ,,,,,,, 2% of dermis weight.

Ground substance of the dermis:

- Consist largely of two glycosaminoglycans , hyaluronic acid & dermatan sulphate with smaller
  amounts of heparin sulhate & chondroitin sulphate.

- Functions of ground substance are:

1. binds water , allow nutrients , hormones & waste products to pass.
2. Lubricant between the collagen & elastin fibers networks during skin movement.
3. provide bulk allowing the dermis to act as shock absorber.

Muscles :

- Muscles found are smooth arrector pili muscle , dartos muscle (scrotum) , platysma ,
  some of facial expressions.

Blood vessels:

- Lies in two main horizontal layers :
  1. **Deep plexus :** above SC fat & it's arterioles supply [1] sweat gland & [2] hair papillae .
  2. **superficial plexus :** in papillary dermis with arterioles supply inverted cone of tissue with it's
     base at the epidermis.
- important in thermoregulation which under sympathetic nervous, control a blood shunt to venous
  plexus at the expense of the capillary loops ,so reduce surface heat loss.

Nerves :

- Most nerve fibers of skin found in the face & extremities . their cell bodies lies in the dorsal root ganglia .

- Most free senory nerves end in the dermis & a few non-myelinated nerve endings penetrate
into the epidermis which some associated with Merkel cells.
* free nerve endings detect damaging stimuli of heat & pain (nocioceptors).

* specialized end organs as pacinian & meissner corpuscles register deformation of the
skin caused by pressure (mechanoreceptors) as well as vibration & touch.

Type of Collagen	Tissue
I	85% of skin + tendons & bones
II	Cartilage
III	15% of skin Collagen + blood vessels
IV	Skin (lamina densa) + Basement membrane
V	Placenta
VII	Fetal membrane , anchoring fibrils

The End

Chapter 5:
Psoriasis:

1-3% of most population have psoriasis.
It is a chronic non- infectious inflammatory skin disorder charechtarized by well-defined erythamatous plaque up and went home and bearing large adherent silvery scales.
It has the following characters:

Benign condition	There is remission and relapse
No cure	Unpredictable
Htperproliferative and inflammatory process	Salmon pink plaques & rare under 10

- **Causes:**

1- genetic predisposition 2- environmental trigger
They are two key abnormalities in psoriatic plaque:

Hyperproliferation of keratinocytes	An inflammatory cell infiltrates in which neutrophils and TH-17 type lymphocytes predominate.

1- Genetics:
- a child with one parent affected has 16% chance.
- if both parents are affected we have 50% chance
-monozygotic 70%, dizygotic 20%, other brother 10%
The mode of inheritance is polygenic inheritance.
- Those with HLA-CW6 genotype have 20 times risk to develop the disease.

2- Epidermal cell kinetics:
- The increase of epidermal proliferation of psoriasis is caused by an excessive number of germinative cells entering the cell cycle.
- The epidermal turnover time is shortened to less than 10 compared to 60 days in normal skin & proliferation 100% instead of the normal 30%
- Maybe that psoriasis is caused by genetic defect of retinoid signaling and that's why it improves with retinoid treatment.

3- Altered epidermal maturation

4- Inflammation

5- Dermis:
- The dermis in psoriasis is abnormal.
- The dermal capillary loops in psoriatic plaque are abnormaly dilated and tortuous.

-Precipitating factors:

Trauma and sunlight	Infection	Emotion
Hormonal (Improves during pregnancy)	Drugs (Anti-malarial, B-Blockers, Lithium, and IFN-α)	Cigarette smoking and alcohol

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- **Histology:**

1- Parakeratosis (nuclei are retained in the horny layer)
2- irregular thickening of epidemis, but thining over dermal papillae is apparent clinically when bleeding is caused by scratching and removing the scales (Auspitz sign)
3- Polymorphic nuclear leukocytes micro abscesses: Munro.
4- Dilated and tortuous capillary loops in dermal papillae.
5- T-lymphocyte infiltrates in upper dermis.

- -Presentation:

Common pattern:

1- Plaque pattern:-

-Most common type.
- Well demarcated lesion, pink or red with large silver-white polygonal scales.
- Site: Elbow, knee, lower back, scalp.

2- Guttate pattern:

- Seen in children and adolescents.(drop shape)
-Triggered by Streptococcus tonsillitis.
- It presents as numerous small rounded red macules which come suddenly on the trunk and then become scaly.
- The rash often clear in few months but plaque psoriasis may develop later.

3- Scalp:

- Areas of scaling are interspersed within the normal skin.
- Lumpiness is easily felt than seen.
-Significant hair loss is rare.
- overflow skin margin

4- Nails:

- Involvement of the nails s common, with thimble pitting, onycholysis (separation of the nail bed from the nail) and sometimes subungal hyperkeratosis.

5- Flexures:

-Psoriasis of sub mammary, axillary and anogenital folds.
- Not scaly but it is glistening, sharply-demarcated, red plaque often with fissuring in the depth of the fold.
- More common in women and elderly patients and hiv

6- Palms or soles:

-Palmar psoriasis, here the lesions are poorly-demarcated and bearly erythematous.
-associated with palmoplanter pustiolosis
The fingers may develop painful fissures.

- Less common patterns:

1- Napkin psoriasis:

- The psoriasis form spreads outside the napkin area.
- Clear quickly but there is an increased risk of ordinary psoriases that develops later on in life

2- Localized pustular psoriasis (palmo-planter pustulosis):

- It is recalcitrant (متمرده) , often painful.
- Affect soles and palms
- Here we have numerous sterile pustules 3-10 mm in diameter lying on erythematous base.
- The pustules change to brown macules or scales(brownish spots).
- Have –Ve auspitz sign (also the flexures pattern have –Ve sign).

-Acute generalized pustular psoriasis = associated with fever and pustiolosis

3-erythrodermic psoriasis:

- the skin become universally and uniformly red with variable scaling.
- Malaise is accompanied by shivering and skin feels hot and uncomfortable.
- Have the following DDx: pitryasis rubra, atopic or irritant dermatitis, withdrawal of potent steroid, coetaneous T-lymphoma.

➢ Complication:

- Psoriatic arthropathy: arthritis occur in about 5% of psoriasis, could be as distal arthritis involve terminal interphalangeal joints of the toe & fingers.
- Metabolic syndrome.
- IHD.

➢ Differential diagnosis:

1- Discoid eczema	Here the lesion is less well defined and maybe exudative& crusting &lacking candle grease scaling, maybe itchy, favor trunk and proximal part of extremities and don't favor scalp and the extensor aspect of elbow and knee.
2- Seborrhoeic eczema	Here the scalp involvement is more diffuse &less lumpy. There is a sign of seborrhoeic eczema elsewhere (eyebrow, chest, nasolabial fold).
3- Pitryasis rosea	Here the lesion is oval rather than round and run along rib lines. The scaling is of collarette type (peripherally attach & centrally not attached), and there is herald plaque may precede the rash. The lesion usually confined to the upper trunk.
4- Secondary syphilis	Here there is a history of primary chancer& the scaly lesion is brownish. There is oral changes , secondary alopecia, condyloma lata &lymphadenopathy &mucus patchy.
5- Cotaneous T-lymphocyte	Persist lesion not in typical site and are often annular, arcuate or bizarre outline and atrophy or poikloderma may present.
6- Tinea ungium	Confused with nail psoriasis, but here it is asymmetrical and ther is obvious tinea of neighboring skin. Pitting not seen here and the nails tend to have discolored at their free edge and tend to be crumbly(متفتت).

➢ Investigation:

1- Biopsy: seldom needed.
2- Throat swab for B-hemolytic streptococcus in guttate psoriasis.

3- Skin scraping & nails clipping maybe required excluding tinea.
4- Radiology & test for rheumatic factor to asses arthritis.

- **Treatment:**

1-explanation & reassurance.
2- main types of treatment:

a) Local treatment:

1- vitamin D	2- calciportriol	3- tacalcitol	4- Local retinoid (tazarotene)
5- dithranol	6- Coal tar preparetion		

- Topical corticosteroids: which we can use it in the following circumstances:
  - in limited choice (area like face, ear ,genital & flexure)
  - for patient who cannt use vitamin D analogue.
  - for unresponsive psoriasis in scalp ,palms & soles.
  -for patient with minor localized psoriasis.

b) ultraviolet radiation:

-the main risk of UVR is acute phototoxicity &induction of skin cancer.
- both the broadband UVB & narrow band UVB (311) can be used.
- the treatment should be given 2-3 times weekly for 8 weeks.

c) Systemic treatment:

- Recommended when > 20% of the body surface is affected.
- The most systemic treatment used:

1- Phototchemotherapy with psoralin & UVA(PUVA)	2- Retinoid (acitretin)	3- hydroxyurea	4- methotrexate (folic acid antagonist)
4- sulfasalazine	5- cyclosporine		

- never use systemic steroids.
- Scalp psoriasis : oily preparation containing 3% salicylic acid.
- Guttate psoriasis: penicillin V or erythromyosin.

Chapter 6
Papulosquamous disorder

Pityriasis rosea:

- A self -limited eruption of macules or papules involving the trunk and extremities and rarely the face; the lesions are usually oval and follow the lines of cleavage of the skin; the onset frequently preceded by a single larger lesion known as the herald patch or mother plaque.
- It may cause by reactivation of human herpes virus 7 or 6.
- It is common especially during winter and mainly effect children and young adult with rare 2nd attack.
- Most patients develop 'herald ' or 'mother' plaque before the others which is larger -2 cm in diameter- and is rounder, redder and more scaly.
- After several days, many smaller plaques appear mainly on the trunk but some on the neck and extremities.
- Oval plaques, salmon pink show delicate scaling run down and out from the spine in a 'sfir tree' pattern along lines of the ribs.

Course:

- Herald plaque preceded the generalized eruption by several days then the subsequent lesions enlarge over the 1st or the 2nd week with minority patients having systemic symptoms like aching and tiredness.
- Eruption last 2-10 weeks then resolve spontaneously leaving hyperpigmented patches that fades more slowly.

DDx:

- Herald plaques are often mistaken for ringworm (tinea corporis).
- Guttate psoriasis.
- Secondary syphilis.
- Pityriasis versicolour.
- Gold and captopril most likely to cause pityriasis rosea-like drug eruption but barbiturate, penicillamine and antibiotics can also do.
- No curative treatment but potent topical steroid or calamine lotion can help the itching, 1% salicylic acid in soft white paraffin can reduces the scaling and sunlight or artificial UVB can relieve the pruritis.

Lichen Planus:

- non-infectious
- eruption of flat- topped, shiny, violaceous papules on extensorsurfaces, male genitalia and buccal mucosa; may form linear groups; individual lesions may be angular or umbilicated; hypertrophic lesions may form on legs.
- Unknown cause but seems to be mediated immunologically with indication that CD8+ cytotoxic T cell recognize antigen on basal keratinocyte.
- Chronic graft vs host disease cause eruption like Lichen Planus that histocompatibility make lymphocyte attack the epidermis.
- Associated with some autoimmune disease as vitiligo, alopecia ariata, and ulcerative colitis.
- Contact allergy to mercury seems can cause oral lichen planus.
- Some drugs can cause lichen planus and some of lichen planus, patients also have hepatitis B or C infection.
- Drug as gold, heavy metals may often cause reaction like lichen planus. Others are antimalarial, beta blockers, NSAIDs, thiazides, penicillamine and some chemicals used to develop colour films.

Presentation:

- Violaceous or lilac-coloured intensely itch, flat topped papules that usually arise on the extremities especially on the volar aspect of the wrist and legs
- Close look shows white streaky pattern on surface of these papules (Wickham striae).
- White asymptomatic lacy lines dots also found in the mouth especially inside the cheeks in about 50% of patients and can be the sole manifestationof the disease. The genital skin can be similarly affected.
- As in psoriasis, Köbner phenomena may occur. Normal nails but in 10% of patients show changes from grooves to destruction of entire nail fold and bed. Scalp lesion can cause patchy scarring alopecia.
- Individual lesions last for many months and eruption of the whole tends to last 1 year but hypertrophic variant (thick warty lesion around ankle) may last for many years.
- As lesion resolve, it become darker, flatter and leave discrete brown or grey macules.
- 1/6 of patients have recurrence.

Variant of Lichen Planus:

1. Annular
2. Hypertrophic
3. Atrophic
4. Bullous
5. Follicular
6. ulcerative

Complication:

- nails and hair loss may be permanent. Ulcerative form if lichen planus in the mouth may lead to squamous cell carcinoma.

Treatment:

- difficult but as to stop the cause if it is a drug
- potent topical steroids can be used to relieve and flatten the plaque. Systemic steroids is used in case of extensive involvement, nail destruction or painful and erosive oral lichen planus.
- Antihistamine for the itch, photochemotherapy with psoralen and PUVA or with narrowband UVB may reduce pruritis.
- Oral ciclosporin or acitretin can be uses on stubborn lesion.
- Corticosteroid or calcineurin inhibitor (tacrolimus) is use on symptomatic mucous membrane lesion.

Chapter 7
Eczema & dermatitis

-contact eczema = contact dermatitis
-eczema subdivided into :
a- exogenous (contact)
b-endogenous (constitutional)

- Classification of eczema :
  1- Caused mainly by exogenous (contact) factors :

Irritant	allergic	Photo-dermatitis

  2- Other type of eczema:

Atopic	Seborrhoiec	Pompholys
gravitational	Asteatotic	Neuro-dermatitis
Juvenile planter dermatitis	Napkin dermatitis	Discoid

- Histology:
  -acute stage : here there is edema in epidermis (spongiosis :edema bet the cells) , progress to the formation of intra-epidermal vesicle, which may coalesce into large blister or rupture>
  - chronic stage : show less spongiosis & vesication, but here there is more thickening of prickle cell layer (acanthosis) and horney layer (hyperkeratosis & parakeratosis) .

- Clinical presentation:
  1- Absence of sharp margin (ill-defined margin): this how we differentiate eczema from other papulosquamous eruptions.
  2- If dose not itch, it is not eczema.
  3- Scaly

  Acute eczema:

1- Weeping & crusting	2- Blistering
3- redness& papule& swelling with ill defined border.	4- Scaling

  Chronic eczema:

1- less vesicular& exudative	2- more scaly, pigmented and thickened
3- show lichenification (dry ,lethary, thickened state with increase skin marking secondary to repeated scratching.	4- More likely to fissure

- Complication :
  1- Heavy bacteria colonization is common in all type of eczema.
  2- Overt(علني) infection is common in seborrhoiec & Atopic dermatitis.
  3- Local superimposed allergic rxn to medication.
- Differential diagnosis :

1- psoriasis	It is sharply marginated, strong color, very scaly, points of elbow & knee involved.

2- scabies	Itchy social contact, face is spared, burrow is found& genitalia an nipples is affected.
3- lichen planus	Mouth lesion , violaceouse tinge shiny flat-topped papule.
4- palmo-planter pustulosis	Localized to palms & soles & there is obvious pustules .
5- angioedema	Usually swollen in the face.
6- fungal infection	Annular lesion with active scaly edge.

- Investigation:

-to confirm your diagnosis is by biopsy.

1- exogenous eczema: patch test to confirm a suspected allergy.

2-other type of eczema:

a- Prick test : in atopic dermatitis.

b- RAST (radio-allergyo-sorbent-test): measurement of total serum IgE and IgE antibodies specific to certain antigen.

3-Malabsorption should consider in otherwise unexplained widespread pigmented atypical pattern of eczema (endogenous).

- Treatment :

1- Rest & liquid application.

2- Non-steroidal preparations are helpful.

3- Wet-wrap dressing : atopic dermatitis in children.

4- Sub-acute eczema: topical steroid & fusidic acid or neomycin.

5- Chronic eczema: topical steroid (ointment) & systemic antibiotics if secondary infection is suspected.

6- Systemic treatment : short course of systemic steroid in severe eczema.

Common pattern of eczema:

- **Irritant contact dermatitis :**

- Account for > 80% of all cases of contact dermatitis.
- **Causes:**

1- strong irritant which elicit an acute rxn after brief contact.

2- patient with dry or fair skin are more vulnerable.

3- patient with atopic dermatitis double the risk for irritant hand eczema.

- **Common irritants are:**

detergents	Alkalosis	Acidic materials
solvents	Cutting oil	Abrasive dusts

- **Complication:**

Loss of work.

- **DDx :**

a- Allergic contact dermatitis b- atopic dermatitis.

- **Investigation :**
  - patch test to a battery of common allergens if an allergic element is suspected.
  - the allergens in our battery are: **MRCP**:

M: medication & metals (nickel, chrome)	**C:** cosmetics
R: rubber& resins	**P:** plants

- **Treatment:**
  1- Avoidance of the irritant by protective gloves.
  2- Moderate topical steroids & emollients (مرطب).

- **allergic contact dermatitis :**
  - **Causes:**
    The mechanism is of delayed type 4 hppersensitivity.
  - **Characteristic of all allergic contact dermatitis:**

1- Previous contact is needed to induce allergy	2- Specific to one chemicals.	3- All area of skin will react to allergen
4- Sensitization persist indefinitely.	5- Desensitization is seldom possible.	

  - **Presentation & course :**
    - the origin site of eruption give a clue to the likely allergen but secondary spread may confuse that.
    - **when to suspect allergic contact dermatitis:**

1- Certain area are involved.	2- Known contact with ana allergen
3- The individual wprk carry a high risk.	

  - **Investigation:**
    - patch test to a battery with common allergen.
    - **treatment: avoidance & topical steroids.**

- **Occupational dermatitis :**
  - for men : chemical plant worker , metal worker , spray painter.
  - for women : hairdresser , nurse , biological scientists.

- **Atopic dermatitis :**
  - atopy : is a sate in which an excessive production of IgE , occur as a response to common environment allergen .
  - early infection may lower the risk of allergy by increasing the production of INF-ϒ.
  - Atopic eczema in monozygotic 86 % and in zygotic 21%.
  - One gene for inheritance of atopy lies on chromosme 11q13, which encode E subunit of high affinity IgE receptor.

***Presentation and course :**

-75% >>begin before age of 6 months.
-80-90% >> begin before age of 5 years.

-***in infancy***: it tend to be vesicular and weeping which start on face, with patchy else where, sparing napkin area.
-***in childhood***: the eczema become lethary,dry and excoriated ,affecting elbows , knee flexures, wrist and ankle.
-***in adult*** : the distribution is like in child hood with marked tendency toward lichenification and more wide spread but low grade involvement of the trunck,face and hands.

What are the cardinal features of atopic eczema?? 1. Itching 2. Scratching

Diagnostic criteria:

1. must have a chronically itchy skin (scratching or rubbing).
2. plus 3 or more of the following :
a. history of itchiness in skin creases (fold of elbow and behind knee)
b. history of asthma or hay fever or history of atopic disease in first degree relative in children <4 years.
c. general dry skin in past year .
d. visible flexural eczema.
e. onset in the first 2years of life.

Complications :

1. bacterial super infection.
2. viral infection (by herpes simplex eczema herpeticum).
3. disturb sleep and grow poorly (GH increase during sleep).

Investigation :

1. prick test **2.** high IgE level .

Treatment :

1. Explanation and reassurance and encouragement.
2. Avoidance of exacerbating factors.
3. Using topical steroids in the following principle :
-using the weakest steroid that control the eczema.
-review their use regularly .
-be wary of repeat prescription
-In primary care avoid using potent steroid.
4. using tacrolimus :which is macrolide immunosuppressant used systemically for kidney ,heart, liver transplantation. Topicaly used for atopic eczema.
-common side effect : transient burning sensation.

- **seborrhoiec eczema (endogenous):**

***Presentation and course :**
-mainly affect hairy area and ogten showing characterstic greasy-yellowish scales.
***Common sites :**
A .red scaly or exudative eruption of scalp ,ear , face and eyebrow .
b. dry scaly lesion of presternal and interscapular area.
c.i ntertiginous :lesion of armpits ,umbilicus and groins.
***Causes :**
- Not related to seborrhea. - Overgrowth of yeast.

- Run in family, affecting those with tendency to dandruff. - Often an early sign of AIDs.

***Complications :**

1. association with furnculosis.

2. in intertriginous type :superadded candida infection may occur .

***Investigation :**

None is done , but put in your mind HIV infection and Parkinson .

***Treatment :**

- Suppressive rather than curative - Topical imidazole
- Topical Li preparation

Chapter 8
Reactive erythema & vasculitis

- **Urticaria:**
  - **Definitions:**
    - it is a common reaction pattern in which pink, itchy or burning swelling (wheal) can occur anywhere in the body.
    - divided into : 1- acute (if the duration < 6 week)
      2- chronic (if the duration > 6 week)
  - **Causes:**
    - the sign & symptoms of urticaria are caused by mast cell degranulation, with the release of histamine.
    - and the degranulation may caused by:

Spontaneously	Chemicals(aspirin)& trauma
Type 1 hypersensitivity (IgE mediated)	Autoimmune (antibodies directed against IgE receptor on mast cell)

- these causes will release chemicals like: histamine , heparin, protease, 5-hydroxytryptamine & inflammatory mediators (PG, IL, neutrophilis &esinophilis)
-these chemicals will lead to increase the capillary permability → transient leakage of fluid → developing wheal.

- **classification:**

Physical (cold, solar, heat, cholinergic, delayed pressure & dermographism(immediate pressure urticaria)	Hypersensitivity	
Autoimmune	Pharmacological	contact

1- **Physical urticaria:**

a- Cold urticaria: patient develop wheal n area exposed to cold(face).

b- Solar urticaria: wheal occur within minute of sun exposure, some patient may have erythropoietic protoporphyria (autosomal dominant condition caused by mutation in ferrochelatase gene, and most of our patient have IgE mediated urticarial reaction to sun light>

c- Heat urticaria: wheal arise in area after contact to hot object.

d- Cholinergic urticaria: it is response to anxiety , heat, sexual excitement, exercise. Here the vessels over-react to acetylcholinelibrated from sympathetic nerve in the skin. It is transient 2-5mm follicular macule or papule.

e- Dermographism: the most common type of physical urticaria; here the skin mast cell release extra-histamine after scratching, leave linear wheal.

f- Delayed pressure uricaria: here sustained pressure cause edema of the underlying skin and subcutaneous tissue 3-6hr later. And the

swelling last up ot 48hr. and it is PG mediated. Common site for this type: feet after walking, hand, buttocks>

2- **Hypersensitivity urticaria:**

- the commonest type of urticaria.
- caused by IgE mediated (type 1) hypersensitivity .
- the ten Is of antigen encounter in hypersensitivity urticaria:

ingestion	inhalation	instillation	injection	Insertion
Insect bite	Infection	infusion	infestation	Inunctions(contact)

3- **Autoimmune urticaria:**

-patient with chronic urticaria has autoimmune disease of IgG antibodies to IgE or to Fc IgE receptor on mast cell ▶ leading to mast cell degranulation.

4- Pharmacological urticaria:

-drugs caused the mast cell to release histamine in a non-allergic manner.

-Ex.: aspirin, NSAIDs, ACEI, morphine.

5- **Contact urticaria:**

-this is may be IgE mediated or caused by pharmacological effect.
-wheals here occur around the mouth.
-food & food additives are the most common culprits.
-latex allergy: which it is a rxn to natural rubber latex of " hevea brasiliens tree" which include : irritant dermatitis , contact allergic dermatitis & type 1 allergy(hypersensitivity urticaria).

- **Presentation of urticaria:**

1- Sudden appearance of pink , itchy wheal , last for less than 24h and most disappear within few hours.
2- Angioedema is a variant of urticaria ,affect the subcutaneous tissue, so that swelling is less demarcated and less red than urticarial wheal. Angioedema occur between the junction of skin and mucus membrane (periorbital, perioral, genitalia) .

- **Complication of urticaria:**

-normally it is uncomplicated condition but the itch may lead to disturb sleeping and decrease activity and depression.
-in anaphylactic rxn ▶ larynx edema ▶ asphyxiation.

- **DDx of urticaria:**

1- Infestation and insect bite elicit urticarial response but these have a central punctum and the lesion may last longer than 24hr.
2- Erythema multiform can mimic annular urticaria.
3- Urticarial vasculitis (form of vasculitis), but here the lesion last longer than 24hr.
4- Bullous disease (dermatitis herpetiformis, phemphigoid) begin as urticarial papules or plaque , but later th bullae make the D obvious.

5- On face: erysipelas can be distinguish from angioedema by its sharp margin, redder color and accompany pyrexia.
6- Hereditary angioedema : here there is attack of abdominal pain and vomiting or massive edema of soft tissue. It is autosomal dominant condition , in which there is deficiency of an inhibitor to C1 esterase, so to confirm the diagnosis , measure C1 esterase inhibitor level and C4 level.

- **Some endogenous cause of urticaria:**

Infection	Intestinal parasite	Connective tissue disorder
Hyper-esinophilic syndrome	hyperthyroidism	Cancer, lymphoma

- **Some exogenous cause of urticaria:**

Drugs	food	bites	Inhalants
Pollens	Insect venoms	Animal dander	

- **Treatment :**
  - we can use cetirizine + loratidine (antihistaminic)
  -chlorpheniramine, diphenhydramine : used in pregnancy
  -sympathomimetic agents can be used (epinephrine, psuedoephidrine).

- **Erythema multiforme:**
  - **Cause:**
    -here there is a rxn to : viral (herpes simplex, hepatitis A,B, orf) & bacterial, fungal, parasitic infection & pregnancy, malignancy & drugs.
  - **Presentation:**
    1- The symptom of URTI may precede the erubtion.
    2- Typically : annular non-scaly plaque, appear on palms, soles, forearm& leg. Initially it have clear center but later on become more purple and look like a target.
    3- Steven-johnson syndrome is a sever variant of erythema multiform, associated with fever & mucus membrane lesions, oral mucosa +lips+ bulbar conjunctiva are mostly affected.
  - **Course:**
    -lesion last several days (2 weeks), and the site of resolved lesion become hyperpigmented.
  - **Complication:**
    -usually no complication.
    -sever lesion in patient with steven-johnson syndrome → can cause asphyxia, blindness, corneal ulcer, anterior uvetitis & geital ulcer.
  - **Investigation:**
    -histology of erythema multiform is:

Epidermal necrosis	Dermal changes (endothelial swelling, prevascular infiltration, papillary dermal edema)

-Tzank test or culture of vesicles ➤ HSV.
-chest x-ray + serological test ➤ mycoplasma pneumonia

- **Treatment:**

Identify and remove cause	Symptomatic treatment in mild cases	Good nurse care
Steven-johnson syndrome: infusion of human ɤ-globulin & prednisolone	Prevention of secondary infection	

-recurrent or continuous erythema multiform: suspect HSV ➤ acyclovir.

- **Erythema nodosum:**
  - Inflammation to the subcutaneous fat " panniculitis".
  - It is an immunological rxn caused by bacteria , viral or fungal infection & drugs or systemic disease.
  - **Presentation:**
    - the lesion is tender, red nodules develop alone or in groups on leg & forearms.
    -some patient have painful joint & fever.
    Lesion usually resolves in 6-8 week.
  - **Complication:**
    1- Walking difficulty.
    2- Erythema nodosum leprosum occur when lepromatous leprosy patient establish cell-mediated immunity to mycobacterium leprae (sever fever . malaise, lethargy).
  - **Investigation:**
    -history & PE and chest x-ray, throat culture for streptococcus and mantoux test & ASO titer.
  - **Treatment:**
    -identify& eliminate the cause.
    -antibiotics & bed rest& NSAIDs.
- **Vasculitis:**
  - Inflammation within the vessels & endothelial cell swelling , necrosis & fibrinoid change.

1-leucocytoclastic (small vessels) vasculitis :

(syn: allergic or hypersensitivity vasculitis, anaphylactoid purpura)

  - **Cause:**
    - immune comlex may ladge(ينغرس) in the wall of the blood vessels which will activate complement and attract PMN leucocytes , which will release enzymes and then degrade the cell wall.
  - **Presentation:**
    1- Painful palpable purpura (3Ps).

2- Group of lesion arise in dependant area (in forearm &leg in walking patient or on the buttocks and flanks in bedridden patient).
3- Some have black small centre caused by necrosis.

-HSP is a small vessel vasculitis associated with palpable purpura & arthritis & abdominal pain which are preceded by URTI. Children are most common affected.
-urticarial vasculitis = small vessel vasculitis characterized by urticarial-like lesion which last longer than 24hr, leaving bruising and pigmentation.

- **Complication:**

-other organ damaged if it was systemic.

- **Investigation:**

1- Identify the cause and detect internal involvement.
2- Chest x-ray, ESR, urine analysis, skin biopsy.
3- Finding of circulating immune complexes.
4- Decrease total complement or decrease C4.
5- Test for hepatitis, RF, ANA, cryoglobulins.
6- Direct immunoflurescence.
7- HSP vasculitis is confirmed by IgA deposit in the blood vessels.

- **Treatment:**

1- Identify cause & eliminate it.
2- Bed rest & antihistamine.
3- Cholchicine & dapsone.
4- Systemic corticosteroid or immunosuppressant agent (cyclophosohamide).

Chapter12
Sebaceous and sweat gland disorder

Sebaceous gland is amultilobed cells full of lipids which shed whole during secretion give sebum that composed of :

- Remnants of lipid cells
- Triglycerides
- Fatty acids
- Wax esters
- Sequalene
- Cholesterol

Functions of sebum are :

- Lubricate and waterproof the skin
- Bacteriocidal and fungistatic
- Protect from drying

*Free sebaceous glands found in the eyelid (meibomian glands) , mucous membranes , nipple , perianal region and genitalia .
*Androgenic hormone especially dihydrotesterone stimulate sebaceous glands activity . these glands react to maternal androgens for short time after birth then lay dormant until puberty , when a surge of androgens produce a sudden increase in sebum excretion and set stage of acne .

Acne

*disorder of pilosebaceous apparatus (hair follicle and sebaceous glands) featured by comedones ,papules ,pustules cysts ,and scars .
*all teenagers have some acne (acne vulgaris) . affect both sexes and start at 12 -14 years old with tending to be earlier in females .
*Peak of severity in females 16-17 and in males 17-19 years old .

Acne vulgaris

Many factors combine to cause acne characterized by chronic inflammation around pilosebaceous apparatus :

A. **Sebum :** increase in excretion but it's n't the causative factor .
B. **Hormonal :** androgens are main stimulus for sebum secretion because the sensitivity although thyroid and GH have minor effect .The response of the sebaceous glands is the main factor because their sensitivity to androgen increase after puberty . So androgens are in normal levels but the target organ sensitivity increase(which occur after puberty). This caused by 5 alfa reductase higher activity.
C. **Poral occlusion :** both genetic and environmental factors cause epithelial to over grow the follicular surface as hyperkeratinization , occlusion (oils and tar)and genetic influence .

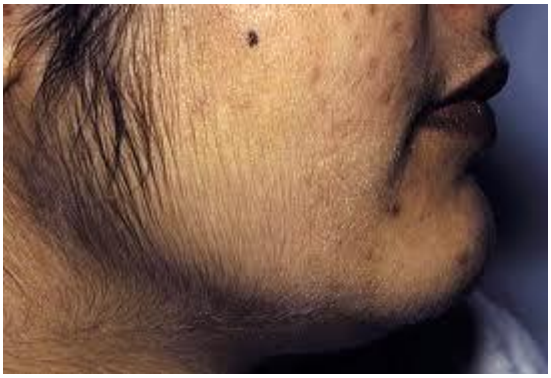
D. **Bacterial (propionibacterium acne) :** normal skin commensal plays a pathogenic part. Bacteria colonize the piolosebaceous duct ,breakdown triglycerides into free fatty acids ,produce substance chemotactic for inflammatory cells and induce cytokines secretion . inflammatory reaction of type 4 IV .

E. **Genetic** : acne is familial in 50% those with acne .

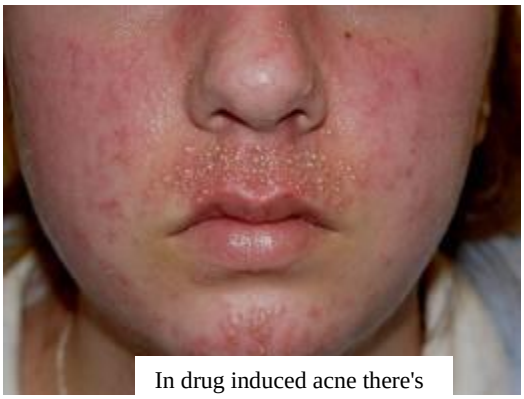
Variants of acne :

- **Infantile acne :** follow a transplacental stimulation of a child's sebaceous glands by maternal androgens .
- **Mechanical (excoriated):** by excessive scrubbing , picking ,and rubbing can rupture occluded follicles .
- **Acne associated with virilization :** include clitoromegaly that cause by androgen-secreting tumors of adrenals, ovaries, or testes .
- **Drug induced** : corticosteroids ,anabolic and androgenic steroids ,gonad ,oral contraceptive pills , lithium, iodide , bromide , antituberculosis (anti TB) , and anticonvulsent therapy .
- **Tropical :** heat and humidity are responsible for this variant which affect caucasiods with tendency to acne .
- **Acne cosmetics .**

Infantile acne



Acne with virilization



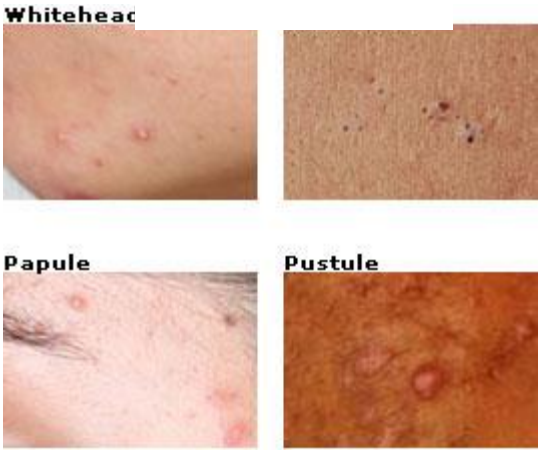
In drug induced acne there's no comedones

Presentation :

*lesions confined to face ,shoulder , upper chest and back . seborrhea (greasy skin) is often present .

*lesion can be :

- Open comedones : black head due to plugging by keratin and sebum of pilosebaceous orifice .
- Closed comedones : white due to over growth of the follicle opening by surrounding epithelium .
- Inflammatory papules ,pustules and cysts : seen with both types .
- Conglobate :the sever form of acne with all the above features as well as abscesses or cysts with intercommunicating sinuses that contain the thick



seroanguinous fluid or pus on resolution it leave deeply pitted or hyper trophic scars .

The variants features are :

Type	Features	Lesion
Infantile	Present or appear soon after birth , common in males ,last 3 years	Like common acne
Fluminants	Rare , severe with fever , joints pain & high ESR	Conglobate
Exogenous	Tars , chlorinated hydrocarbons , oils may cause or exacerbate excising acne	
Excoriated	Most common in young girls	Obsessional picking or rubbing leave discrete denuded areas
Late onset	Occur mainly in women & often limited to the chin	Nodular & cystic lesions , stubborn & persistent
Tropical	Mainly on trunk	Conglobate
Drug induced	Non-teenager , appear suddenly with drug	Papulo-pustules rather than comedones
Polycystic ovarian syndrome	Obese females with oligomenorrhea or amenorrhea or infertility	----------
Congenital adrenal hyperplasia	Hyperpigmentation, ambiguous genitalia	----------
Androgen secreting tumors	Rapid onset of virilization	----------

Course :

*Acne vulgaris clear by the age of 23 – 25 years in about 90% of the cases , but 5%of females and 1% of males still need treatment .

Investigation :

*None are usually necessary . cultures are occianally needed to exclude pyogenic infection and only few P.acne are sensitive to antibiotics .

*any acne associated with virilization need investigations to rule out malignancies .

Differential diagnosis DDX :

*Rosacea affect older older individuals , comedones are absent , papules and pustules occur only on the face .

Treatment:

Local Trearment:

a. Regular gentle cleaning.
b. Antibacterial cleansers as chlohexidine.
c. Benzoyl peroxide as antibacterial agent: applied 5% then up to 10% if necessary.

d. Retinoids: Vit A analogues to normalize follicular keratinization & especially effective against comedones. Concomitant eczema is C/I to it's use. Weakest preperations used 1st then sometimes after a week or two stopped temporarily due to irritation. As benzoyl, strength of tretinoin increase after 6 weeks if tolerated, so combining both of them has many advocates. Topical retinoids should not be prescribed for pregnant women with acne.
    - Isotretinoin: 0.05% which made in gel base & applied 1-2 times daily
    - Adapalene: retinoid-like drug indicated for mild to moderate acne.
    - Tazarotene: applied once daily.

e. Azeliac acid: bacteriocidal for pyogenic acne but not used >6 months.
f. Abrasive pastes: contain aluminium oxide.
g. Sulphur.
h. Local antibiotics: topical Clindamycin, Erythromycin & Sulfacytamide.
i. Combinations
j. Aluminium chloride.
k. Cosmetic camouflage.

Systemic treatment:

a. Antibiotics: tetracycline, Erythromycin & Trimethoprim.
b. Hormonal: combined anti-androgen-estrogen treatment. Course last 8-12 months then replaced by estrogen/progesterone oral contraceptives. <u>These drugs are not for males.</u>
c. Isotretinoin: oral retinoid inhibit sebum excertion, growth of P. acnes & acute inflammatory processes. The drug reserved for sever nodulo-cystic acne. Full blood count, liver function test, fasting lipid levels should be checked & routine urine analysis. Isotretinoin is highly teratogenic, so we need 2 –ve pregnancy tests before giving the drug. Treatment starts at the 3rd day of the patient's next menstrual cycle following –ve pregnancy test.
    - Side effects of isotretinoin include dry skin, inflamed lips & eyes, hyperlipidemia & hair loss which all are reversible with rare changes in night-time vision & hearing loss.

- Isotretinoin flares up acne at 1st but this is short lived &drug can be continued.

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Diet: avoid nuts, chocolates, dairy products & wine.

Avoid	Reason
Pregnancy	Teratogenicity
Breast feeding	Unknown effect on baby
Giving blood	Teratogenicity in recipient
Uncontrolled hyperlipidaemia	Additive side-effects
Taking vitamin A and hypervitaminosis A	Additive side-effects
Cosmetic procedures	Increased scarring
Excessive natural or artificial UVR	Photosensitivity
Oral contraceptive with low dose of progesterone—'minipills'	Ineffective contraception
Concomitant antibiotics, unless with permission of prescribing doctor	Intracranial hypertension

Physical:

- UV B radiation therapy often help with exacerbation as two months cources.
- Cysts can be incised & drained.
- Intralesional injecton of 0.1 ml Triamcinolone acetonide to resolution of stubborn cysts but can leave atrphy.
- Dermabrasion to smooth out facial scars but not for active lesion or depressed scars.
- Lasers: skin resurfacing with CO2 & erbium lasers done on equiescent (ساكنة) acne under local anesthesia.
- Collagen injection: bovine collagen can be injected into depressed scars to improve their appearance but C/I un any autoimmune disorders.

Rosacea:

- Vascular and follicular dilation involving the nose and contigiois portions of the cheeks, may vary from very mild but persistent erythema to extensive hyperplasia of the sebaceous glands with deep seated papules and pustules accompanied by telangiectasia at the affected erythematous sites.
- Affects face of adults usually women with peak incidence in their 30s – 40s.

- Unkown cause. It is flush in response to warmth, spicy food, alcohol or embarrassment & any psychological abnormalities are secondary to the skin condition.
- Normal sebum excretion rate & skin microbiology & no pathogenic role for hair follicle mite Demodex folliculorum.

Course & complications

- Cheeks, nose, center of forehead & chin are most commonly affected. Peri-orbital & peri-oral areas are speared.
- Intermittent flushing followed by fixed erythema & telangiectasia. Discrete domed inflamed papules, papulopustules & rarely nodules develop later. No comedones or seborrhea.
- Features of rosacea symmetrical & prolonged course. Complications are:
    1. Blepharitis.
    2. Conjunctivitis.
    3. Keratitis.
    4. Rihnophyma.
- Rhinophyma caused by hyperplasia of the sebaceous glands & connective tissue on the nose tha is more common in males.
- Some patients treated with potent topical steroids develop a rebound flare of pustules worse than the original rosacea when treatment stopped.
- Treated by Tetracyline (traditional & effective treatment). Erythromycine is 2nd choice.
- Condition recur in 50% of patients within 2 years so need repeated rather than prolonged maintenance antibiotics therapy.
- We can use topical metronidazole, isotretinoin, sulphur, icthammol, zinc & sunscreen if the sun exposure is an aggravating factor.

Chapter 13
The Hair & Nails

The hair:

- Hair follicles form before the ninth week of fetal life. Sebaceous gland is an outgrowth at the side of the hair germ, establishing early the two parts of the pilosebaceous unit.
- Melanocytes migrate into the matrix & responsible for different colors of hair. Grey or white caused by low pigment production, filling of the cells in hair medulla with minute air bubbles that reflect light.

Classification:

a- Lanugo hair: fine long hair covers the fetus but sheds about 1 month before birth.

b- Vellus hair: fine short unmedullated hair covers much of the body surface which replaces the lanugo just before birth.

c- Terminal hair: long coarse medullated hair seen in scalp and pubic regions. Its growth influenced by circulating androgen level.

Note:

- Terminal hair converts into vellus in male pattern alopecia, & vellus hair converts into terminal hair in hirsutism.
- Lips, glans penis, labia minora, pals& soles remain free of hair follicles.

The hair cycle:

- Each follicle -independently of its neighbors'- passes through regular cycle of growth & shedding with three phases:
  - a- **Anagen**: the active phase of hair production, last up to 5 years (1000 days)
  - b- **Catagen:** short phase of conversion from active to resting phase, last for 2 weeks. Growth stops in this phase & the end of hair becomes club-shaped.
  - c- **Telogen**: resting phase at the end of which the club hair is shed, last for 3 months.
- Scalp contains 100,000 hairs : 100 hairs shed daily, 85% in Anagen phase, 13% in Telogen phase , & 2% in Catagen phase.
- Length of the hair determined by the duration of Anagen phase.
- Hairs of the eyebrows have shorter cycles than those of the scalp.
- If many follicles pass into the resting phase at the same time, large number of hair will be shed at 2-3 months later as in **Telogen effluvium**.

Alopecia:

- It is hair loss. It is divided into localized of diffuse.
- Localized alopecia is caused by :

1- **Non-scarring causes**: such as alopecia areata, androgenetic, traction alopecia, & scalp ringworm.
2- **Scarring causes**: such as burns, radidermatitis , carbuncle, basal cell carcinoma, lichen planus, SLE, & sarcoidosis.

Alopecia areata:

- It affects 2% of patients seen in skin clinics with immunological basis cause, because of association with thyroid diseases, vitiligo & atopy.
- Histologically, there is clustering of T-lymphocytes around affected hair bulbs attracted & made to divide by cytokines from the dermal papilla.
- Presented as typical **un-inflamed patches, without scaling,** & with easily seen empty hair follicles.
- Pathognomonic "**exclamation mark**" hairs seen around the edge of enlarging areas which are broken off, 4mm long, narrowed, & less pigmented proximally.
- Patches are common in scalp & beard, but area as eyebrows & eyelashes can also be affected too.
- Unpredictable outcome course with re-growth within a few months in 1st attack.
- Subsequent episodes tend to be be more extensive & slower re-growth.
- Few patients lose all the hair head (called **alopecia totalis**) or from the whole skin surface (called **alopecia universalis**)
- Factors that indicate poor prognosis are:
  1- Onset before puberty.
  2- Associated with atopy or Down's syndrome.
  3- Unusually widespread alopecia.
  4- Involvement of the scalp margin **(Ophiasiform type)** especially at the nape of the neck.
- **Treatment:**
  - Reassure the patient about prospects of re-growth.
  - Using systemic steroids should be avoided , but intradermal injection of **intralesional Triamcinolone Acetonoide** can be effective.
  - PUVA therapy can help in extensive cases, but hair fall returns when stop treatment.

Androgenic alopecia (male-pattern baldness):

- Clearly familial, mainly of polygenic type of inheritance. Androgen dependent in male.
- If androgenic alopecia occur in females with normal level of circulation androgen, then seen only in those with strongly predisposed genetically.

- Presented in males as hair loss 1st from the temples, then from the crown. In women, hair loss is much more diffuse especially over the crown.
- Complications are great **anxiety** & rarely **monosymptomatic hypochondriasis**. Bald scalps burn easily in the sun & may develop **multiple actinic keratoses**.
- **Treatment:**
  - **a- Scalp surgery , hair transplant & wigs.**
  - b- **Minoxidil:** it slows hair loss & even stimulates new growth of hair in few cases, but when stop treatment new hairs fall out after 3 months.
  - **c- Antiandrogens:** help women with diffuse type of androgenic alopecia.
  - d- **Finasteride:** inhibitor of **human type II 5∝-reductase** that reduces serum & scalp skin levels of dihidrotestosterone in balding men. Not indicated in women or children with rare side effect as decrease libido, erectile dysfunction & alerted prostate specific antigen.

Traction alopecia:

- Pulling of the hair by hot combing, tight hairstyles (pony tail or corn rows) & hair rollers.
- Presented usually in girls & young women especially those hair always tented to be thin anyway.
- Hair loss being more where there is maximal tug as marginal alopecia, which applied to one common pattern in which hair loss is mainly around the edge of the scalp (sides or front)
- Regrowth is less because mainly bald areas show short broken hairs, folliculitis & scarring, so incompleted regrowth.
- Treatment is stopping the cause as rollers or others.

Patchy hair loss caused by skin disease:

Disease	Features
Scalp ringworm	Inflammation , pustulation
Psoriasis	Rough removal of scales can also remove hairs but regrowth is must.
Scarring alopecia	Damage of hair follicles with previous described cause.

Diffuse hair loss:

- Loss from whole scalp accompanied by thinning visible to others. Most common causes:
  - **Telogen effluvium.**
  - **Endocrine**: hyperpituitarisim, hyperparathyroidism, hypo/hyperthyroidism.

-**Drugs:** antimitotic agent (anagen effluvium), anticoagulants, Vit. A excess, & contraceptives.
-**Androgenetic.**
-**Iron deficiency anemia**: most common cause , especially in women.
-**Chronic illnesses & malnutrition**.
-**Diffuse type of alopecia areata**.

Telogen effluvium:

- Triggered by any sever illness, especially those with fever or hemorrhage by childbirth or sever dieting.
- All of these hairs will synchronize Catagen, so large numbers of hairs are lost at the same time.
- Diffuse hair fall, **2-3 months after provoking illness** that can be mild or sever. In latter case, **beau's lines** can be seen on the nails.
- Regrowth is not usually complete occur within few months.
- Unaffected by therapy, but can reassured that their fall will be temporary.

Hirsutism:

- Growth of terminal hair in female distributed in the pattern normally seen in a male.
- Can be recial, familial, minor after menopause & can be idiopathic (no hormonal cause) or the patient have one of the following disorder:
  -**Polycystic ovary syndrome.**
  -**Cushing syndrome**
  -**Ovarian tumor.**
  -**Adrenal tumor**.

(تكملة التشعر)

1- Adrenal hyperplasia
2- Anabolic steroid use

- So patent with hirsutism we ask about menses to exclude hormonal abnormality like (↑androgen)
- Familial, racial or idiopathic hirsutism tend to start at puberty & worsen with age.
- Investigations needed in cases of :

1- Hirsutism in childhood.
2- Sudden or recent onset.
3- Other features of virilization (clitoromegaly)
4- Menstrual irregularity or cessation.

- Vast of cases have POS polycystic ovary syndrome that accompanied with obesity & acne. And the treatment of this case is :
  - Hair removal physically

- Drugs as Ethinylestradiol & Cyproterone acetate or Spironolactone (antiandrogen)

Treatment :

- Plucking النتفshould Avoided as it can stimulate hair roots into anagen.
- Laser can used to remove hairs if are a lot. Or electrolysis with few active hair.
- Oral antiandrogens can be used, but needed long term.
- Pregnancy must be avoided during treatment as it's carry a risk of feminizing a male fetus.

Hypertrichosis

- Excessive growth of terminal hair that doesn't follow androgen-induced pattern .
- Can be of localized type the most commonly seen:
  - Melanocytic naevi: as Becker's naevi
  - Stayr't tuff: that affects sacral area in case of spina bifida.
- Some causes of generalize hypertrichosis are:
  - Anorexia nervosa
  - Drug as minoxidil & Cyclosporin.
  - Fetal alcohol & fetal phenytoin syndrome.

The Nails:

- The hard keratin of the nail plate is formed in the nail matrix which lie in investigation of the epidermis (nail fold) in the back of the terminal phalanx of the each digit .
- Nail bed is capable to produce small amount of keratin which contribute to the nail & which resp for the " false nail" formed when the nail matrix is obliterated by surgery of injury .
- The cuticle act as a seal to protect the potential space of the nail fold from chemical & infection.
- Fingernails average growth is 0.5-1.2 mm/week while toenail grow more slowly . Nail grow faster (1)in summer , (2) if bitten & (3) in youth.

Trauma

Permanent ridges or splits in the nail plate can follow damage to the nail matrix.

- **Splinter hemorrhage** is a longitudinal ridges & grooves in the nail bed commonly seen in cases of minor trauma, psoriasis, & subacute infective endocarditis .
- Larger subungular hematoma caused by trauma as dark areas of altered blood.

- Chronic trauma from sport or ill-fitting shoes cause hemorrhage under the nail of the big toe to gross thickening of toenails known as **Onychogryphosis**.
- **Onycholysis** is a separation of nail plate from nail bed that result from minor trauma, psoriasis & thyroid disease.

Feature	Description	Disease
Koilonychias	Spoon shape	IDA
Half & half nails	White proximal & red or brown distal	CRF
White nails		Hypoalbuminemia
Discoloration		Antimalarial, antibiotic & phenothiazines
Beau's line	Transverse grooves on all nails	A few weeks after acute illness (like in telogen effluvium)
Nail fold telangiectasia or erythema		Drematomyositis, SLE & systemic sclerosis
Pitting		Psoriasis esp with arthritis
Scratched		Eczema
Pterygim	Reversible thinning that when sever lead to cuticle growth over nail base to attach to nail plate	Lichen planus

Acute paronychia:

- Usually staphylococci that enter through a break in the skin of cuticle due to minor trauma.
- Acute inflammation present with pus in the nail fold or under the nail require systemic treatment & surgical drainage
    - So the treatment will be (1) anti staph antibiotic &(2) drainage

Chronic paronychia:

- Opportunistic pathogen (yeast G+ve cocci & G-ve rods) colonize the space between nail fold & nail plate.
- Predisposing factors include poor circulation, wet work, working with flour, DM, vaginal candidosis & vigorous cutting back of the cuticles.
- Present as tender & swollen nail folds with small amount of pus . Cuticle seal is damaged & adjacent nail plate becomes ridged & discolored .
- Treated by imidazole cream or itraconazole if candida is present with keeping the hands dry.

Onchomychosis

- Fungal infection of the nail

- More in toes
- The most common is **DLSO pattern** (Distal and lateral subungual onychomycosis)
- Treated by systemic antifungal:…like…

Dermatophyte infections:

Commonly tinea pedis with early changes at the free edge (broken edge) of the nail & spread proximally.

- Nail plate become yellow, crumbly & thickened affecting one hand (to differentiate from psoriasis that affect both hands).

Chapter 15
Infestation

Infestation : *is the presence of animal parasite on or in the body.*

1-lice infestation (pediculosis)

- lice : flattened wingless insect that suck blood.
- nits : eggs of lice which attach to hair or clothes.
- main features : sever itching followed by scratching and secondary infection .
- we have 2 parasite in human :

1-pediculosis humans (capities , corporius). 2-phthirus pubis.

A-head lice :

****cause:***

- affect 10% of children.
-head louse (peduclosis humans capitis) is 3-4 length and is grayish.
-her nits are firmly stuck to hair shafts.
-spread from person to person by head-head contacts or hats.

****presentation and course :***

-main symptoms is itchy , at first around the side and back of the scalp and then more generally over.
-scratching and secondary infection follow this itching.
-in heavy infestation the hair become matted and smelly.
-the drainage L.N are often enlarge.

****complication :***

-secondary bacterial infection.

****DDX :***

-all patient with recurrent impetigo or crusted eczema on their scalp should examined for the presence of nits.

****treatment :***

-malathion , carbaryl and permethrin.
-lotions should remain on the scalp for at least 12hr.

2-scabies:

****cause*** :

-caused by mite sarcoptes scabies var.huminis.
-adult mite are 0.3-0.4 mm long , which transferred from person to person by close body contact.
-once it is on the skin , the fertilized female mite burrow through the stratum corneum at rate of about 2mm per day. And produce 2-3 oval eggs each day and these eggs turn to sexual mature mite in 2-3 week.

****presentation*** :

-for the first 4-6 weeks after infestation , there may be no itching but after this period pruritus dominate the picture.
-the most dramatic part of eruption-excoriated eczema or urticarial papule is usually on the trunk.
-the burrow must be identified to confirm your Dx.
-most brow site in scabies are:

Between fingers and on wrist 85%	Elbows points, nipple,umbilicus	Rubbery pink nodules on male genetalia	Sides of feet
buttocks	Medial aspect of thigh and arm	In babies only here you can see face burrow	

-burrows : grey -white slightly scaly tortuous line 0.5-1.5 cm.
-mite may be seen by a lens as a small black dot at the most recent least scaly end of burrow .
-in chronic stage the number of mites may be small.

Complication:

1- Secondary infection with postulation.
2- Persistent itchy lead to red nodule which may remain on genital or arm pit of children for month.
3- The use of scabicides cause skin irritation & eczema.
4- Veneral disease may be acquired at the same time as scabies
5- Crusted (Norwegian) scabies which it's not itchy and crusted eruption in which vast number of mite are found and it affect people with learning difficulty and immunosuppressive.

DDx:

- Only scabies shows characterized burrow.
- Animal scabies induce itchy rash in human but this lack burrow.

1- Late onset atopic eczema
2- Lichen plans
3- Cholinergic urticarial.
4- Dermatitis herpetiformis

Onchomychosis

- Fungal infection of the nail
- More in toes
- The most common is **DLSO pattern** (Distal and lateral subungual onychomycosis)
- Treated by systemic antifungal :…like
- Pick the mite by needle from the end of burrow and then identify it.

Treatment:

3- Malathion or permethrin.
4- Topical treatment & invermectin (200mg by mouth) is used for Norwegian scabies and unresponding scabies.
5- For babies > 2month, toddler & young children we use permthrin cream + 25% benzyl benzoate emulsion diluted with 3 part of water.

Or 6%-10% sulphar in white soft parfine (Vaseline).

6- Treat all members of the family.

Note:

- Mites die in clothing unworn for 1 weak.
- Don’t forget to look for head lice in children present with cervical adenopathy.
- Patient with atopic dermatitis + scabies the drug of choice is permthrin.