- المحاضرات المكتوبة باسم زميلتينا أنوار جابر و فرح زياد عبارة عن محاضرات جديدة كتبت في 2015\2014
- باقي المحاضرات من دوسية 2015\2014

بسم الله الرحمن الرحيم

NORMAL SKIN

Done by: Mohammad Al-Madani د. محمد شرف

There are 2 types of skin:

A) Glaberous skin:

- Presents on the palm of the hands & sole of the feet.
- Thick keratin layer.
- Presence of dermatoglyphics, which is specific for each person.
- Presence of special nerve organs.
- Lacks the presence of hair follicles or sebaceous glands.

B) Hairy skin:

- Presents on all over the body except the palms & the soles.
- Cauterized by wide variation according to anatomical site, examples:

Scalp→Large sebaceous glands & hair follicles.

Axilla → large sweat glands (apocrine).

Embryology:

Epidermis originates from ectoderm.

Dermis & sub-cutaneous tissue originate from mesoderm.

Melanocytes from neural crest & migrate along the neuron.

Functions of the skin:

1) Protection against physical & chemical trauma.
2) Heat regulation: through the glomus bodies and sweating
3) Perception of sensation.
4) Secretion of sebum & sweat.
5) Pro-vitamin D synthesis.
6) Mirror of the body because the skin act an organ of expression:

Anxiety → sweating.

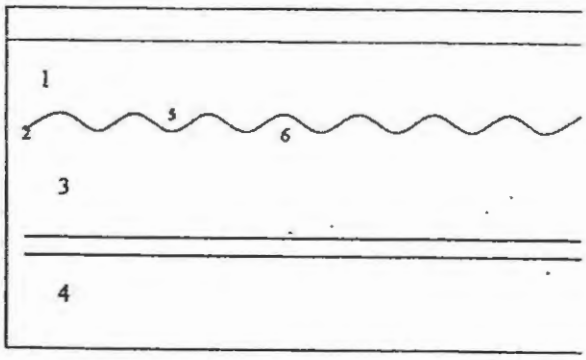
Fear → pallor.

Anger → redness.

Reflection of many systemic diseases.

Layers of the skin:

1 → epidermis
2 → Basement membrane
3 → Dermis
4 → sub cutis
5 → rete ridge
6 → dermal papilla



Epidermis.

Contains 4 major types of cells:

A) Keratinocytes B) Melanocytes
C) Langerhans cell D) Mickle cell.

A) Keratinocytes: organized in 4 layers

1) *Basal cell layer:*

Consists of one raw of cells, columnar in shape with their longitudinal axis perpendicular to the basement membrane.

The mitotic activity is restricted to these cells to form the cells of other layers.

Between the cells of this layer are the melanocytes that appear as clear 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.

2) *Prickle cell layer (spinous, squamous, Malpighian):*

Polygonal cells separated by spaces.

Between these cells there are intracellular cement (tonofilaments) & inter cellular cement (Desmosomes).

3) *Granular cell layer:*

Diamond in shape filled with keratohyaline granules.

4) Keratin layer (stratum conium):

Keratinization: the process of formation of keratin.

Normally it takes 4 weeks, but in certain diseases like psoriasis it takes 3 days.

Keratin is the normal barrier between the environment & the body.

B) Melanocytes: dendritic cells that form & secret melanin by the mean of dendrites (for more details go to the lecture about pigmentations).

C) Langerhans cells: They are Ag presenting cells.

D) Mickle cells: Bell-like, & below it we have free nerve endings related to touch.

Epidermal appendages: structures originating from the epidermis but lye in the dermis & they are:

1) Keratinous: hairs & nails.
2) Glandular:
   - Apocrine sweat glands: large sweat glands present in the axilla & groin.
   - Eccrine sweat glands: small sweat glands all over the body.
   - Sebaceous glands: holocrine glands (their secretions are formed by destruction of their own cells), secret sebum along hair follicles leading to skin, present all over the body except palms & soles.
   - Both apocrine & eccrine sweat glands are controlled mostly by nerves while sebaceous glands are controlled by androgens

Dermis.

Composed of:

1) CT fibers: collagen elastin & reticulin fibers.
2) Ground substance of mucopolysaccarides.
3) Cellular elements: Fibrocytes, fibroblasts, mast cells.
4) Nerves:
   a) Sensory nerve organs:
      - Miessner corpuscles: mediate the sense of touch, so they are concentrated in the tips of fingers.
      - Pacinian corpuscles: deep in the dermis, they mediate the senses of pressure & vibration. They are large onion-shaped, on palms, soles, areola & genitalia.
      - Muco-cutaneous nerve organs: loops of nerves without capsules, mediate general sensory reception.
      - Free nerve endings: mediate heat, itch, and pain.
   b) Autonomic: for sweat glands, muscles & vessels.
5) Blood vessels: there are 2 plexuses
   - Superficial: capillaries + venuoles + lymphatic vessels with lymph fluid circulating between cells.
   - Deep: artery + vein + lymphatic vessels.
6) Muscles: voluntary (skeletal) & involuntary (smooth)
7) Lymphatic vessels.

Skin lesions:

1) Macule: circumscribed FLAT lesion, < 1 cm, of normal texture, only change in color (Black, blue, red, pink, white).
   Example: Pitichea.

2) Papule: circumscribed raised lesion, < 1 cm, of any color.
Example: purpura.
3) Vesicle: visible accumulation of clear fluid in the skin.
Circumscribed, < 1 cm, contains clear fluid, could be in dermis or epidermis.
Examples: Herpes facialis, Herpes labialis, Herpes zoster, varicella zoster.
4) Pustule: accumulation of visible puss in the skin.
Circumscribed, < 1 cm.
Example: characteristic lesion of folliculitis.
5) Nodule: large indurated hard, varying in size from 1- several cm, could be flat as in erythema nodosum which is highly painful seen in conditions like TB, streptococcal infections, or raised as in cancer mets.
6) Scar: replacement of normal tissue with fibrosis. Could be atrophic, hypertrophic or keloid.
7) Ulcers: Loss of normal tissue. Could be deep (ulcer) or superficial (erosion).
8) Bullae: large accumulation of visible fluid in the skin (clear Vs hagic), can be dermal or epidermal.
Examples: in burns & bullous diseases of the skin.
9) Atrophy: thinning of the skin so that the skin becomes transparent & you see the blood vessels.
Caused by strong topical steroids.
Note → do not give strong topical steroids for babies & ladies because they cause atrophy. (CI to use topical steroids on face)
10) Crust: dried serum. When the vesicles rupture, they give out serum that dries to give the crust.
Example: seen in impetigo (bacterial infection of the skin) that forms honey-colored crust.
11) Wheal (weal): center of edema surrounded by a hallow of erythema, hyperemic well circumscribed compressible lesions, very itchy, of different shapes & sizes, the single lesion lasts for 10 minutes.
Example: Characteristic lesion of urticaria.
12) Cyst: it is a cavity filled with fluid & lined by epithelium.
Example: sebaceous cyst.
13) Scales: accumulation of excess normal or abnormal keratin of the skin.
Examples: seen in psoriasis & icthiosis vulgaris.
14) Lichenifaction: thick skin due to prolonged scratching.
15) Plague: raised group of lesions> 1 cm.
16) Patch: flat group of lesions> 1 cm.
17) Comedons: Black heads/ inactive follicular plug.

They are the primary lesions for acne vulgaris.
Destruction of the follicles by sebum

Morphology of lesions:

- Discoid lesions: eczema.
- Linear lesions: psoriasis, lichen planus, warts.
- Herpitiform lesions.
- Zosteriform lesions: distribution of zoster.
- Annular lesions.
- Arciform lesions.
- Polycyclic lesions.
- Targeted lesions (iris lesions): erythema multiformis, Grouped lesions.

Target lesions seen in erythema multiforme after herpes simplex with 2 weeks. It's an abnormal response to virus

Management of a patient with dermatological disease:

- *History:*
  1) Chief complaint.
  2) Duration: constant Vs periodic.
  3) Location of symptoms.
  4) Progression of lesions: beginning, evolution, alteration, natural or produced by therapy.
  5) Occupation: especially in cases of hands eczema.
  6) Habits & hobbies.
  7) Garments (clothes).
  8) Cosmetics.
  9) Drugs.
  10) Allergy history.
  11) Seasonal variation: for example psoriasis decreases in summer & pregnancy but increases in winter.
  12) Relation to other diseases.
  13) Past history: skin diseases, other diseases, medication, and hx of allergy (asthma).
  14) Family Hx: allergic diseases, skin diseases, other diseases.
- *Physical examination:*
  1) The patient should be seen naked and with good illumination.
  2) Do not forget the mucous membrane.
- *Diagnosis:* coordinate the Hx + P/E + lab findings

- *Treatment*
    1) Systemic.
    2) Local:
        a) Lotion: water + powder.
        b) Creams: water + powder + oil.
        c) Ointment: oil + powder.
        d) Pastes: wax + powder.
        - For acute oozing lesions we use creams & lotions to dry them.
        - For chronic old lesions we use ointments & pastes.
    3) Other modalities: UV light, surgery, laser, cryotherapy.

THE END

بسم الله الرحمن الرحيم

Sub-Medicine, Dermatology

Lecture topic: Bullous Diseases

Date : 2009/2010

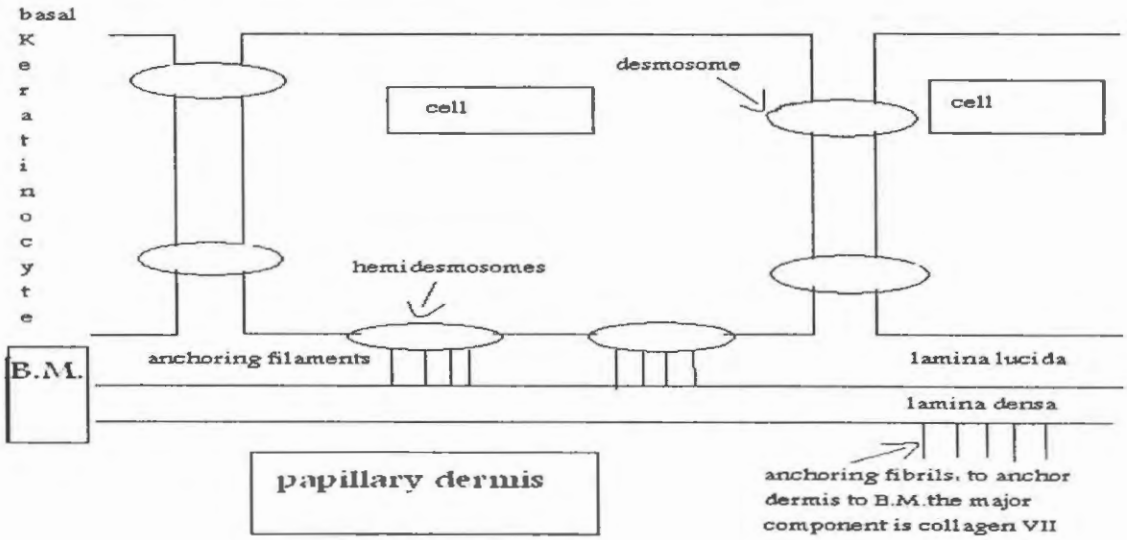
Modified by : Dr. Muna

Printed by : Mohammad Ghalib Riziq

Bullous Diseases (الأمراض الفقاعية)

Introduction:

- Blister: accumulation of fluid within or under the epidermis
- Blisters:
  - Vesicles: < 1cm in diameter
  - Bullae: > 1cm in diameter
- Spongiosis: mild intercellularedema in the acanthous layer , but it's not sufficient to destroy the desmosomes. It occurs in acute eczema.
- Acantholysis: edema + destruction of the desmosomes → bullae formation



**** Bullous diseases classified into:**

- Intraepiderma & subepidermal
- Or into: congenital & acquired

Congenital type (epidermolysis bullosa)

- There is a gene mutation (i.e. autoimmune disease)
- Simplified classification of epidermolysis bullosa:

Type	Mode of inheritance	Level of split	Mutations in
Simple epidermolysis bullosa	Usually autosomal dominant	Intraepidermal	Keratins 5 and 14
Junctional epidermolysis bullosa (epidermolysis bullosa letalis)	Autosomal recessive	Lamina lucida	Components of the hemidesmosome-anchoring filaments (e.g. laminins, integrins and bullous pemphigoid 180 molecule)
Dystrophic epidermolysis bullosa	Autosomal dominant	Beneath lamina densa	Type VII collagen
Dystrophic epidermolysis bullosa	Autosomal recessive	Beneath lamina densa	Type VII collagen
Acquired epidermolysis bullosa	Not inherited	Dermal side of lamina densa	Nil

- There are 4 types of epidermolysis bullosa congenita:
  1- Simplex (cytolysis): no mucosal involvement, no nail involvement, blisters heal without scarring. Weber-cockayne is a subtype
  2- Junctional (hemidesmosome defect): AR, more sever, may cause scarring, nail, teeth & mucosal invovment
  3- Dominant (anchoring fibrils defect): mouth is not involved
  4- Recessive (anchoring fibrils)
- 3 +4: they are of the dystrophic type, they cause teeth, nail & mucus involvement also it cause scarring.

Acquired Epidermolysis Bullosa (Epidermolysis Bullosa Aquisita –EBA-)

- All are Autoimmune diseases, subepidermal
- Target Ag: anchoring fibrils (collagen VII)
- Blisters are usually a response to trauma
- More resistant to treatment than bullous pemphigoid
- Distinguishing features of the three main immunobullous diseases:

	Age	Site of blisters	General health	Blisters in mouth	Nature of blisters	Circulating antibodies	Fixed antibodies	Treatment
Pemphigus	Middle age	Middle age, flexures & scalp	poor	common	Superficial & flaccid	IgG to intercellular adhesion proteins	IgG in intercellular space	Steroids Immunosupressives
Pemphigoid	old	Often flexural	good	rare	Tense & blood filled	IgG to basement membrane region	IgG at basement membrane	Steroids Immunosupressives
Dermatitis herpetiformis	Primarily adults	Elbows, knees, upper back, buttocks	itchy	rare	Small, excoriated & grouped	IgG to the endomysium of muscle	IgA granular deposites in papillary dermis	Gluten-free diet Dapson sulphapyridine

A- Pimphigus vulgaris:

- Race: jews
- Age: middle age
- Ag here is desmosome intercellular
- C.C:
  1- Painful erosion in the mouth for several months
  2- Classicallesion on the trunk: flaccid bullae from healthy looking skin
  3- Bad genral condition: because the serum will dry → crust → infected → bad general condition
- Site of predeilection: almost lways the umbilicus is involved
- Dx:
- History & physical examination
- Nikolsky's sign: if you press on the skin between 2 bullae, it will be yielded
- Tzank's sign: acantholysis in the smear
- Bx:
  1- Intraepidermal bullae
  2- Acantholytic keratinocytes + RBC's
- Pathology: it's an autoimmune disease in which Ab's are formed directed against intercellular cement (desmosomes).
- Direct immune-fluorescence: intercellular IgG & C3, intraepidermal, in a net-like pattern
- Treatment:
- Admission for tests & treatment (sometimes)
- High dose sysytemic prednisolone ≈ 100 mg/day + azathioprine 100 mg/day(Imuran)
- How long? Until the absence of all existing bullae, & no more bullae appear (+/- 1 month)
- Then taper the steroid → for e.g. 20 mg & continue with azathioprine also

B- Bullous pemphigoid:

- Hemidesmosome are the Ag's here
- Race: no specific race
- Old age (>70 years)
- C.C.:
- On the trunk & extremities
- Itchy, urtecaria-like skin rash for one month → tense bullae not from healthy looking skin
- Good general condidition
- Nickolisky's sign is negative
- Bx: subepidermal bullae, eosinophils is very apparent
- Pathology: autoimmune disease in which Ab is directed against the dermoepidermal junction (DET) (healthy epidermis)
- Direct immune-fluorescence: IgG & C3 in linear pattern at the DET
- Treatment:
- No need for admission
- +/- 50 mg/day prednisolone usually with tapering for few years, as spontaneous relapses may occur
- ± immunosuppersives

C- Dermatitis herpitiformis (DH):

- Target Ag: transglutaminase enzyme
- Race: no specific race
- Age: middle age
- C.C.:
  - GI: gluten sensitivity enteropathy
  - Skin: highly itchy grouped tense vesicles at the sites of predilection (shoulder, elboe, back of the neck, sacral area, kness)(it's an eczema like rash).
- Bx: sub-epidermal vesicles, but above the dermis, with neutrophilic infiltration at the tips of dermal papillae
- Direct Immune-fluorescence: IgA & C3 in granular pattern because it's above the dermal papillae
- Treatment:
  - Luten free diet
  - Dapsone 100 mg/day
  - Important note: dapsone causes hemolytic anemia in patients with G6PDdeficiency, so do the enzyme assay before giving the drug
  - Not steroid responsive

"تمت بحمد الله"

Bullous Diseases

- Could be congenital or acquired: acquired are more common:
- According to the level of the defect the shape of the bullae: if higher in the epidermis it will be flaccid and more fragile (easily ruptured) if lower in the dermis it will be more tense and harder to rupture.

Congenital types: *25* variants u should only know what's in the table
- Simplex epidermolysis bullosa congenita: could be generalized or localized. Characterized by flaccid superficial bullae. Abnormal desmosomes seen in the granular layer diagnosed by biopsy.
- The localized type is autosomal dominant, non-fatal and presents late in life. On walking for 1-2 km, The patient will develop palmoplantar herpitiform bullae
- Dystrophic types: associated with dysmelia like syndactyly. Teeth abnormalities occur due to retained epidermal cysts due to ruptured bullae

Acquired Types: more common and more important clinically:
- All are autoimmune diagnosed by immunofluorescence: Direct: pt's skin with dye or indirect: pt's serum and animal skin

A- Pemphigus vulgaris:
- intraepidermal bullae therefore flaccid and may present as erosions. IgG against intercellular desmosomes
- Two thirds of patients present with oral lesions that never heals. Discovered during visits to the dentist
- Blisters are seen in the face, scalp oropharynx, genitalia
- Associated with thymoma and autoimmune diseases
- First line of Treatment: high dose steroids with introduction of immuran (azathioprine) to reduce the amount of steroids used
- Tapering of steroids after *5* years usually of treatment. Individual variations in this step decide the rate u may need to decrease 2.5-5mg/month according to risk of recurrence of lesions
- Granular appearance on immunofluorescence

A similar condition occurs in pregnancy known as gestational pemphigus

B- Bullous pemphigoid:
- minor mucosal involvement
- linear appearance on immunofluorescence
- tense bullae
- no association with autoimmune disease

Other treatment options include anti-neutrophilic drugs like dapsone and colchicine.

Gestational pemphigus:
- bullae appears around the umbilicus and may be generalized
- associated with autoimmune diseases
- TTx is steroids only during pregnancy

Pemphigus vollaceous: another variant with very superficial bullae

C- Paraneoplastic pemphigus:
- Occurs in both adults and children. Occurs with lymphoproliferative disorders
- Usually generalized affecting the palms, soles and severe mucositis

- You should raise your suspicion with this disease when treatment with high dose steroids is not effective.
- Diagnosed by immunoflourescence

D- Dermatitis Herpitiform:
- Papulovesicular lesion, Symmetrical itchy vesicles
- Associated with gluten-sensitivity enteropathy and lymphomas
- Dapsone is drug of choice. Doesn't respond to steroids
- Give test dose of dapsone to diagnose
- G6PD deficient patients should not be given dapsone. Give vit. E to counteract hemolysis.

** Porphyria: may present with bullae at dorsum of hand and sun exposed areas.

THE END

Papulosquamous Squamous Diseases

- Papulosquamous disease : plaque covered with scales
- 3 types :
  - Psoriasis
  - Para-psoriasis
  - PityriasisRosea
- Some other dermatological diseases that present with erythematous papules with scales
  - Eczema
  - Lichen Planus

Psoriasis

- Common chronic inflammatory disorder of skin , of an immunological origin , on a background of genetics , currently is considered more as a systemic disease rather than skin disease
- Why systemic ?
  - Skin & joints
  - Realtion between it & systemic diseases due to an inflammatory conditions
    - DM
    - IHD
    - Hyperlipidemia
    - Metabolic Syndrome
- Immunological origin :
  - Abnormal activation of T-lymphocytes leading to release of certain cytokines & interleukins that are responsible for the changes observed in psoriasis
    - IL-12
    - IL-17
    - IL-23
- Genetic background :
  - Association with HLA
  - Increase risk of developing psoriasis in cases with +ve family history

Pathophysiology :

- Normal skin layers are :
  - Epidermis :
    - Stratum basale
    - Stratum spinosum = squamous cell layer

        - Stratum granulosum = acanthus layer
        - Stratum cornum = brick cell layer
    - Dermis
    - Subcutaneous tissue
- Granular cell layer is 1-3 cell layer thick

Abnormalities in psoriasis :

- Excessive proliferation of cells in basal cell layer
- Decreased differentiation

Histopathology :

- Stratum corneum :
    - Hyperkeratosis : increased thickness of stratum corneum
    - Parakeratosis : presence of nucleated keratocytes in stratum corneum
    - Munro's microabcesses : accumulation of neutrophils & neutrophil debris in stratum corneum
- Stratum granulosum :
    - Absence of granular cell layer in some area with basophilic duo keratocytes
    - Called : agranulosis or hypogranulosis
- Stratum spinosum :
    - Acanthosis : increased thickness of acanthus cell layer
    - Psoriasiform acanthosis : dermal papillae are getting more up & rete ridges are getting more deep
    - Opposite to psoriasiform acanthosis you can see suprapapillary plate thinning
- Stratum basale
    - Free
- Dermis
    - Mixed inflammatory cells in the upper dermis & epidermal papillae
    - Dilated & tortuous capillaries in the dermal papilla
        - Represents a clinical sign : Auspit's sign = pinpoint bleeder

Clinical types of psoriasis :

1. Plaque type psoriasis
    a. Most common type
    b. Appears as well-defined erythematous plaque covered by thick silvery sticky dry scales
    c. Color of erythema is peculiar = dull red = deep red = beefy red

   d. Sites of predilection
      i. Extensor aspects of limbs especially elbow & knee
      ii. Lumbosacral spine
      iii. Scalp
   e. Variants of this type are called annular psoriasis
      i. Ring-shaped lesion with healing center
2. Guttate psoriasis = نقطي
   a. Erythematous papule with fine scale
      i. Lacks the characteristic thick silvery sticky dry scales
   b. Sites of predilection
      i. Trunk
      ii. Upper limb
   c. Mostly affects children & adolescents
   d. Usuallay follow group A strep. Infection of URT
   e. Lesions are sterile with immunological reaction due to strep. A infection
3. Psoriasis inversa
   a. Called flexural psoriasis
   b. Flexor sites : cubital , popliteal & wherever you're having 2 opposing skins like axilla, sub-mammary site , inguinal region & abdominal fold of obese patient
   c. Well-defined erythematous plaque with minimal to no scales due to nature of these areas
   d. DDx
      i. Seborrheic dermatitis
      ii. Fungal infections
4. Pustular psoriasis
   a. Two forms
      i. Localized palmoplantar pustular psoriasis (4Ps)
         1. Acral skin of palms & soles
         2. Pustules on erythematous base covering the acral skin
      ii. Generalized pustular psoriasis (GPP)
         1. Second most severe type
         2. Most of body surface area
         3. Pustule on an erythematous base
   b. In both types : pustules may coalesce together & leak pus
5. Erythro-dermic psoriasis (psoriatic erythroderma)
   a. Erythroderma : erythema & scaling covering more than 90% of total body surface area
   b. Causes

i. Psoriasis
ii. Eczema
iii. Drugs
iv. Congenital disorders

c. Skin has lost its function as a barrier so these patient are at high risk of developing :
   i. Thermal disorders : hyper/hypothermia
   ii. Dehydration
   iii. Electrolyte imbalance
   iv. Risk of infection is less than burn patient due to natural release of β-defensins in psoriatic patients which confers immunity

d. Most severe type

Plaque psoriasis in scalp	Seborrheic dermatitis
Dry thick silvery-whitish scale	Yellowish greasy scale
Well-defined sharp demarcation	Ill-induce margin

Psoriatic arthritis:

- It's psoriasis affecting the joints
- Clinical forms are
  - Asymmetrical oligoarticular (<5) arthritis of small joints (most common type)
  - RA-like psoriatic arthritis
  - Arthritis of large weight-bearing joints (elbow , knee & ankle)
  - Sacroilitis
  - Arthritis mutilans (least common)

Psoriasis affecting nails : (the changes are in order)

- Pitting
- Thickening of nail plate
- Subungual hyperkeratosis (hyperkeratotic area between nail bed & plate)
- Onycholysis
  - Oil-drop sign
  - Salmon patch
  - Separation between nail bed & plate
- Nail destruction

Precipitating & aggravating factors in Psoriasis :

- Smoking
  - Only delays healing
- Infections
  - Aggravate any clinical type
  - +ve relationship between group A strep. &guttate type may aggravates other types
- Trauma or scars at any site of trauma
  - If psoriasis occurred here , this is called Kobner's phenomenon
- Hypocalcemia
- Hormonal factors
  - Majority of cases improve during pregnancy & flare up postpartum &vice versa
  - Some cases doesn't change with or after pregnancy
- Drugs
  - Systemic steroid and/or very potent topical steroid on large surface area and/or abrupt withdrawal
  - β-blocker
  - NSAIDs
  - Lithium carbonate
  - Penicillin
  - Anti-malarial drugs
- Seasonal variation
  - Majority worsen during winter & improve during summer

Treatment :

- Factors that determine what medication to use are :
  - PASI : psoriasis area severity index
  - DLQI : dermatology life quality index
- Modalities of treatment are
  - Topical
  - Systemic
  - Phototherapy
- Topical
  - Keratolytics : salicylic acid
  - Topical steroids : avoid very potent steroids on large surface area
  - Vit.d analogs : calcitriol , calcipotriol

  - Anthralin or dithranol : plant extract of unknown mechanism , regulate hyper-proliferative states , irritant to skin so start with low doses & concentration , can pigment skin & clothes
  - Tar preparation : better to be avoided due to carcinogenicity
  - Calcineurin inhibitors : tacrolimus , pimecrolimus
  - Topical retinoid : topical tazarotene
- Systemic
  - MTX
  - Fumaric acid
  - Cyclosporine A
  - Systemic retinoid : acitretin
  - Biological agents
    - TNF inhibitors
    - Anti-IL (12,17,23)
- Phototherapy
  - Use of UV light (UV-A long wave = 320-400 nm)
  - Requires photosensitization (psoralen)
    - Oral PUVA
    - Cream PUVA
    - Bath PUVA
  - UV-B doesn't need photosensitization because it's a narrow band wave 311 nm
  - Laser (eximer)

Lichen Planus LP

- Unknown etiology but association with Hep.B&C are well documented
- Many clinical variants but classical pattern is
  - Violaceous pruritic polygonal polyhedral papule on striated surface called Wickham's striae (flat&topped)
- Most common site is flexor aspect of limbs especially wrist & ankle
- Sometimes eruptions of LP that are widespread called eruptive LP
- LP papules may coalesce together forming plaques giving the appearance of papulosquamous disease
- Itching is the most common symptom
- Can affect the mucosa , most commonly buccal mucosa forming whitish nits-like plaques
- If longstanding , it may transform to SCC

- Can affect hairy area causing a patchy scarring alopecia (lichen planopilaris)
- Can affect nails producing the following changes :
  - Longitudinal ridging of nail plate
  - Thinning of nail plate
  - Onycholysis
  - Pterygium : distal hypertrophy & proliferation of cuticle of nail plate
  - Nail dystrophy
- Course & Prognosis
  - Relapses & remissions over a period of time from 1-7 years
  - This is classical
- After resolving the lesion leaves a post-inflammatory hyperpigmentation scar (ugly)
- Treatment
  - Topical
    - Steroids
    - Calcineurin inhibitors
  - Systemic
    - Steroids
    - Retinoid
    - Cyclosporine A
    - PUVA

PityriasisRosea :

- Benign self-limited papulosquamous disease of skin of unknown etiology
- Increment factors are
  - Infections with human herpes HHV-6 , HHV-7
  - Allergens
- Presentation
  - Single erythematous plaque
  - Asymptomatic or mildly itchy
  - Most patients will ignore it because it's asymptomatic
  - Few days up to 2 weeks of the first lesion : multiple small similar lesions start erupting , those lesions are erythematous plaques with Callorette scales
- Distribution : from neck to mid-thigh
- In the back : it follows ribs forming Christmas tree appearance sparing the spine
- Symptoms : from asymptomatic to itchy
- Prognosis & course
  - More lesions are predicted to erupt 2-4 weeks after onset

- Another 2 weeks are needed to resolve completely without any sequelae

- Treatment
  - Unnecessary
  - Mild topical steroids & anti-histamine may be used to decrease symptoms

The end

Done By Anwar Jaber

Printed By Ahmed Sabri

Eczema

- Eczema : aka dermatitis:

A distinct inflammatory response to exogenous or endogenous factors , presented clinically as an irritable papulo-vesicular rash and histologically as spongiosis (Edema).

- It can be classified according to chronicity or to the causative agent.

→According to Chronicity : Acute / subacute / chronic

Acute eczema :

- Clinically present as Erythema ,Scales and vesicular eruption
- Histologically manifested as : Hyperkeratosis, parakeratosis , spongiosis with intraepidermal vesicles and intradermal inflammatory infiltrate.

SubacuteEczema :

- Histologically: Smaller and fewer vesicles.

Chronic Eczema :

-Clinically present as lichenifaction and hyperpigmentation. (darkening and thickening of skin due to chronic scratching).
-Histologically : acanthosis (thickening of the skin), no vesicles at all.

→According to the causative agent: Endogenous / exogenous

-- Exogenous factors cause:

1. Primary irritant contact dermatitis :
   E.g: -- Ascorbic acid on skin for long time on specific area .
   pathophysiology : cell necrosis , irritation is limited to area of contact.

   -- Perfume on lateral side of neck may cause erythematous itchy scaly vesicular eruption called berlock dermatitis.

2. Allergic contact dermatitis :
   E.g-- nickle , eczema affect the site of irritation and other sites
   pathophysiology : antigen antibody rxn .

3. Infective contact dermatitis : bacterial infection

4. Photocontactdermatitis : UV light .

Treatment of Exogenous dermatitis ::

1. Avoid contact with incriminating agents
2. Mild :: topical steroid to decrease inflammation.
   Mild to Severe :: presence of excoriation marks :: give topical steroids with topical antibiotics
   Very severe :: oral steroids .. if imptiginization presents give oral antibiotics \
3. For severe itching give antihistamine.
4. Patch test for allergy.

-- Endogenous Eczema ::

1. Atopic dermatitis

- It appears after 3 months of age, on different sites according to age;

Infantile	Childhood	Adults
Extensors Face and scalp	Flexors area	Nothing specific

- Manifests as Very itchy , erythematous scaly vesicles.
- Usually with a positive past or family Hx of atopy , Atopic dermatitis, Allergic Rhinitis , asthma.
-T-helper cells play the major role in pathophysiology.

- **Treatment :**
  1. Prevention by : cotton clothes ,emollients, bathing for a period no longer than 5-10 minutes and avoidance of very hot water. (hot water dries the skin even more).

  2. Mild → topical steroids / topical Abs, after 3-4 weeks stop steroids and start on calcineurine inhibitors (tacrolimus and pemicrolimus)

  3-Very severe → oral steroids / oral Abs .

  4- you can use antihistamine for its sedative effects to relieve itching.

2. Seborrhoeic Dermatitis :

- In areas where sebaceous glands are numerous and highly active ;Eye brows, forehead, chin and nasolabial folds , scalp , anterior chest and upper back.

- On scalp it is either dandruff or greasy scales.

-In infants it may appear from day one of life called cradle cap (unlike Atopic dermatitis that doesn't appear before 3 months) -- Tx by olive oil.

-On face : erythema and scales

-Anterior chest and upper back : well demarcated erythematous patch with fine scales

-Itching is very mild or nothing at all

-Usually, no family history usually

-Tx for dandruff : ketoconazole shampoo twice weekly.

Seborrheic Dermatitis	Atopic Dermatitis
1- Negative family history 2- Appear within 1st week of life 3- Site : scalp , nasolabial fold, forehead, eyebrows , anterior chest, upper back. 4- Creasy scales with erythema. 5- Mild or absent itching . 6- Better prognosis .	1- Positive family history. 2- Appears after 3 months of life . 3- Site of presentation according to age(check the previous pages) 4- Erythematous papules and vesicles. 5- Severe itching. 6- Poor prognosis.

* Leiner's syndrome: is diffuse seborrheic dermatitis in infants manifesting as Erythroderma and requires hospital admission.

3. Discoid dermatitis :

-Mainly in the elderly, present as Disc like erythematous scaly lesion (discoid description always implies a well demarcated lesion) that is caused by xerosis or dryness.

- Manifests mainly on the extensor surfaces.

-Treat by emollients to prevent dryness , if severe give topical steroids.

4. **Dyshydrosis / pompholyx**

-Deep seated nodules mainly on palms and soles that are very very itchy

-Give potent topical steroids.

5. **Pitryasis alba :**

-More prominent in black skin, manifests as white macules or patches mainly on the face, while in children it mainly on upper or lower limbs

-Increases with sun exposure.

- Wood's light examination can differentiate between it and vitiligo :: vitiligo looks milky white depigmented lesion . while pitryasis alba appears as a hypopigmented lesion.

-Tx by : sun block , emollient and if itchy give steroids.

The End

Done by Anwar Jaber

Printed by Saja Majed

Fungal Infections.

Fungal infections are classified into
Inflammatory vs. non-inflammatory and,
superficial, subcutaneous, or disseminated

- **Superficial noninflammatory**

1. **Tinea versicolour**

- Brownish hyperpigmentation with scales, without erythema. (Asymptomatic)
- Appears on areas where sebaceous glands are present; anterior chest, back, axillary pits and neck.
- Caused by *Malassezia furfur* which is also known as *Pityrosporum orbiculare* or *Pityrosporum ovale*

Responsible fungi is part of the normal flora, but becomes pathogenic in cases of increased humidity and bad hygiene. It is lipophilic, therefore it doesn't affect children because their lipid profile is not yet well developed.

- Dx: 1. wood's lamp (remember wavelength used is 365 nm UVA1 light) where it appears as yellowish fluorescence

2. Scrape the skin and soak in KOH where it shows spaghetti (hyphae) and meat balls (spores) pattern under the microscope.

- Causes post-inflammatory hypopigmentation.
- Rx: topical antifungal agents., Systemic if widespread.

2. **Tinea nigra**

- Caused by *Hortoea werneckii*
- Affects the palms
- lesions appear as annular, brown, hyperpigmented patches.
- DDx includes Nevi
- Rx: topical antifungal agents

3. **Piedra**

- Presents as palpable, grossly visible yellow nodules on the hair shaft.
- white piedra caused by *Trichosporon beigelii*
- Black piedra caused by *Piedraia hortae*
- Usually associated with low hygiene standards
- Rx: shower + antiseptics

- **Inflammatory superficial – Dermatophytes**

Capitis = scalp hair
Corporis = Skin
unguium = nails
pedis = interdigital webspace
Cruris = Groin
Barbae = beard

1. Tinea capitis

Hair shaft/ nails are made up of keratin (dead tissue) , Systemic agents are used because topical agents are not absorped .

- divided into

a. inflammatory

Ectothrix (outside the hair shaft)
fluorescents, woods +ve -- apple green
short broken hair
most commonly caused by *Microsporum canis* followed by *M. audini.*
Dx: KOH

b. non-inflammatory

Endothrix
appears as black dots on affected areas
patients present with patchy hair loss
most commonly caused by *Trichophyton tonsurans/ Trichophyton violoceum*
woods –ve.
Dx: KOH

-Rx: systemic antifungal drugs.

*Favus is the most severe form of tinea capitis, caused by *Trichophyton schoenleinii* and appears as white to blue fluorescence on using wood's light. It is a dermatological emergency because it can cause scarring and permanent hair loss. Or may spread causing sepsis.
*Kerion is a clinical sign for tinea capitis where severe inflammation and a boggy appearance can be seen.

2. Tinea corporis

- Description: annular, erythematous, scaly patch with active (pustules on rim)

borders and a healing centre.
- Affects children and young adults.
- Organisms: *Trichophyton rubrum* and *Trichophyton mentagrophytes*
- Treatment: topical antifungal agents. / Griseofulvin?

3. Tinea barbae

- Caused by *Trichaphyton verrucosum*
- appears as inflammatory lesions with erythema, pustules and a boggy appearance it should be treated asap. As It migh cause loss of hair follicles --> Scarring
- Treatment: Topical Antifungals
  Systemic Antifungals
  Antibiotics.

4. Tinea pedis

- Divided into 4 subtypes. All are most commonly caused by *Trichophytin rubrum*, Epidermophyton Floccosum and *Trichopyton mentagrophyte* except for the inflammatory subtype which is more commonly caused by *T. mentagrophytes var mentagrophytes.*

It is the most common fungal infection in adults.

a. interdigital webspace (fourth and fifth spaces)
most common type and appears as white macerated skin, common in diabetic patients.
b. inflammatory
extension to soles and appareas as vesicles and bullae.
c. Ulcerative with exfoliation.
d. Mucisin
Treatment: - Frequent washing and immediate drying.
-Topical management unless the patient in diabetic or immunocompromised.

5.Tinea Ungum

onychomycosis is divided into 3 subtypes – (all are most commonly caused b *T. rubrum* and treated with systemic antifungal drugs.)
1. Distal and lateral type – which is the most common (Onycholysis + subungal hyperkeratosis)
2. Superficial white
3. Proximal (in this subtype it's important to rule out HIV infection)

Treatment : Systemic treatment for 2 months for finger nails, 3 months for toe nails.

6. Tinea cruris

- appear as erythematous, scaly pruritic patch
- Affects the inner upper thigh, may also extend to the genitalia and buttocks
- caused by *t.rubrum, t.mentagrophyte* and Floccosum.
- Affects the diabetic, pregnant and obese people.

* Cruris vs. candidia?

1. Scrotum is not involved in tinea cruris whereas it might be involved in candidial infection.
2. Shiny surface in candidia
3. Satellite lesions are peculiar to candidia (specially in bed-ridden patients)

- Risk factors for mucocutaneous candidia
  1. HIV infection 2. Immunesupression
  3. Intranasal or oral steroids 4. Antibiotics abuse
- Angular stomatitis is seen in
  1. Candidial infections
  2.Contact dermatitis
  3. Iron deficiency
  4. B12 deficieny

- Candidal infections:
  1. Oral thrush : Lesions are friable crusts.
  2. Flexural candidiasis: Bright red pustules with satellite lesions.
  3.Anal cleft: Pseudomembranes.
  4.Vulvovaginitis: Milky discharge, itchy
  5. Ring and middle finger.
  6.Paronychia.
  7. Chronic mucocutaneous Candidiasis.

Good luck :)
Farah Ziad

Reactive Erythema

Erythema Nodosum :

One of the reactive erythema , it represents a pathology called paniculitis (inflammation of fat or subcutaneous tissue) .

Typical Presentation :

- Acute eruption of erythematous tender deep seated subcutaneous nodules over the shin ,bilateral , symmetrical .

- last for 2-3 weeks then disappear

- It repeats again if ause is still there

More common in females

Causes of EN :

A) Most common

1- Idiopathic

2- Infection (streptococcus pharyngitis)

3- Drugs : Estrogen , OCP , Sulfonamide

4- Sarcoidosis

5- TB

6- IBD

Pathogenisis

due to deposition of Ag-AB complex at the septae between fat cells in subcutaneous tissue .

It's the prototype of septal paniculitis

- Skin over the nodules is streatch , erythematous , shiny , might be associated with generalized symptoms like fever , myalgia , weakness .

- Ulceration is NOT a feature of EN

Diagnosis :

1) Clinical diagnosis in most of the time (Hx + PE)

2) Biopsy → * In Debate / * for medicolegal cases

3) ESR 4) Culture (Blood) 5) CXR → for hyperlymphadenopathy

6) Intradermal Skin test → for T.B

Treatment :

1- Bed Rest and elevation of leg

2- stop the incriminating factor

3- NSAID

4- In severe cases give oral steroid

5- Potassium Iodide (anti-inflammatory)

Erythema Multiforme : multiple forms or presentations

One of the reactive erythemas which comes with acute attacks , the lesions appear and progress over 24 hours then it subsides after 10-14 days , it can reappear if the cause is still there .

If can be divided into two types :

1- EM minor → EM of skin alone

2- EM major → EM of the skin and one mucus membrane (oral mucosa / Conjunctival Mucosa)

Causes :

1) Idiopathic

2) Infection 90% (Most common one is HSV1 , 50% of patients)

3) Drugs 10% → NSAID/Sulfonamides / Antiepileptic / Antibiotic

Causes severe EM (major)

The most common cause of recurrent EM is recurrent HSV

The more mild the EM is , the more likely the cause is infection

The more severe the EM is , with involvement of mucosa , the more likely the cause is drugs

Presentation :

1) Target Lesions → Typical

2) Iris lesions

3) Uritcaria lesions

Involvement of the mucosa causes erosions of M.M especially oral mucosa (pain , difficulty in swallowing)

Involvement of the Eye mucosa , it will heal with scar formation called Synchia , So you should take it seriously and send the patient for urgent consultation to an ophthalmologist .

Treatment :

1) stop the incriminating cause (drug/ infection)

2) Symptomatic Treatment : Itching → Antihistamine , Skin lesions → topical Steroid , Oral mucosal lesions → antiseptic , Eye mucosal involvement → Urgent opthalmology consultation

Steven Johnson Syndrome (SJS)

EM of the skin and 2 or more mucous membranes

Cause usually drugs → 1) allopurinol 2) NSAID 3) antiepileptic 4) Sulfa drugs

TEN Toxic Epidermal Necrolysis :Seperation of epidermis from the dermis in more than 30% of body surface area

Drugs that may cause TEN : 1) Allopurinol 2) Antiepileptic 3) Sulfa Drugs 4) NSAID

It's Fatal in 50-75% of patients , sometimes one dose of drug can cause it

Pathophysiology : Failure of detoxification of toxic byproducts of drugs due to enzymes deficiency (genetics)

It heals with scars and post inflammatory hyperpigmentation

We admit those patient into burn unit (we consider them as 3rd degree burn)

Risk of 1) Dehydration 2) Electrolyte Imbalance 3) Infections

Which are the main causes of death

Risk of 1) DVT and PE 2) DIC

Which are minor complications

Minimal manipulation should be done in order not to cause further sloughing .

Treatment :

1) TEN

*Admit the patient in the burn unit

*Stop the incrimination factor *Supportive care , electrolyte care , fluid care , eroded surface area

* Don't give systemic Steroid

* IVIg decrease mortality rate by 50%

2) SJS

* Admit the patient in ICU

* stop the incriminating drug

* There is a debate in using systemic steroid (most → not to give)

* Supportive care

Urticaria :

*Appears in the form of wheals lesions – Red , erythematous , edematous lesions –

* Each wheal lasts for less than 24 hrs then it reappears again (Not the same lesion)

Types :

1) Acute < 6 weeks

2) Chronic > 6 weeks

* Physical , Hypersensitivity , Drug induced , Autoimmune , Idiopathic , Contact

*Main complaint is Itching

Treatment : 1) Eliminate the cause 2) Antihistamine
3) Steroid → in Resistant cases , and cases which are associated with edema angioedema (lips / under lower eyelid)

Note : *Angioedema is dangerous if it is in the larynx * Hereditary angioedema is an AD , recurrent attacks of angioedema with abdominal pain and diarrhea is associated with enzyme deficiency called (CI esterase Inhibitor)

Figurate Erythema :

A) Erythema annulare (ring) Centrifugum : started from center then expands peripherally , associated with drugs , fungal infections , cancer , blue cheese .

B) Erythema gyratum repens : Very rare type , associated with lung cancer

C) Erythema Chronicum migrans : chronic , migrates , associated with lyme disease

D) Erythema Marginatum Rheumatican : Rheumatic fever

E) Erythema Elevatum Diutinum : represent leukocytoclastic vasculitis

The End

Done by Anwar Jaber .
Printed by Majed Ftaiha.

Cutaneous manifestations of C.T. diseases

Contents of C.T.:

1. Cellular elements: fibroblasts, macrophages, mast cells
2. fibrillar elements: elastin, collagen, reticulin
3. ground substance: mucopolysaccharides& prtns

Factors affecting C.T. dis's:

1. Nutritional : prtns, vit.C
2. hormonal : GH, TSH, steroids
3. aging, inheritance & dis's

C.T.dis's r either:

a) Inherited (not imp.):

1. Pseudo xanthoma elasticans
2. Ehler Danlos syn.
3. Marfan's syn.
4. Osteogenesis imperfecta

b) Acquired:

1.SLE
2. Scleroderma (SCM)
3. Dermatomyositis

I. Lupus erythromatosis:

- Chronic disorder affecting c.t. of most organs & mucous membranes, associated with major vasculitig & other immune abnormalities & +ve *ANA*
- Classified into: SLE & chronic discoid Lupus erythromatosis (CDLE)
- It's an *AI* dis, the *AG* is an altered DNA, the *AB* is IgG, IgM & complement
- Immune complexes are found in all tissues
- In CDLE skin only is affected, lesions r n patches & immune complexes r found only @ the dermoepidermal junxn

CDLE:

* Relatively benign
* Only skin & MM's
* C:E 8:1
* Peak incidence : 4th decade
* histology : hyperkeratosis, keratin spikes obstructing hair follicles (follicular plugging) ,atrophic epidermis hydropic degeneration of basal cell layer , lymphocytic infiltrates around bid *vessels,* hair follicles n the dermis

* clinical features: (variants)

1. Plaque type:
   - → occurs on areas exposed to sunlight (face ,neck ,hands)
   - → a plaque of sharp erythema containing follicular plugs
   - → adhesive scales
   - → later on , atrophy
   - → warty like overgrowth n chronic lesions
2. lupus pernio: in people with poor circulation & raynaud's phenomenon +ve
3. disseminated CDLE
4. lupus profundus : deep involvement
5. subacute CDLE

* On the scalp CDLE causes permanent scarring

Rx of LE:

1. Strong topical steroids
2. dermo jet: intradermal injection of steroid
3. topical sun block
4. systemic sun block (antimalarial : hydroxychloroquine)

Cutaneous lesions n SLE: They r common, occurring n >80% of cases

1. Discoid lesions
2. urticarial oedematous *rash*
3. diffuse erythema
4. photosensitivity
5. MM involvement ii both SLE & CDLE
6. popular telangiectasia (pathognomonic)
7. ulceration of the skin
8. necrotizing vasculitis
9. livedo reticularis
10. erythema multiforme

II. Scleroderma (sclerosis)

- Induration of the skin
- Occurs in:
  - as a part of systemic sclerosis
  - dermatomyositis
  - SLE

→ Scleroderma confined to the skin (morphea)
→ C:E 3:1
→ Children &adults r affected

→ Histology:

1. early inflammatory stage
2. hypertrophy of collagen bundles
3. infiltrate around bulges
4. late sclerotic stage; no hair follicles, anhidrotic patch of morphea , v. sparse infiltrate

→ The first evidence of plaque type morphea is induration of the skin, later on; waxy ivory shiny hard patches attached to the underlying tissues, anhidrotic & maybe preceded by hyperpigmentation

Systemic sclerosis:

1. Small mouth
2. Dysphagia
3. hard skin all over

Generalized morphea:

1. all skin is involved
2. might turn into systemic sclerosis
3. stiff fingers
4. occurs n elderly *C*
5. no curative Rx

Lichen sclerosis ET atrophicans:

- in children & adults
- when u touch the skin btn 2 fingers u c wrinkles
- atrophic silvery shiny patches
- serious when C genitalia is involved : burning sensation & severe itching ,small vague genitalia& coitus is impossible, give topical progesterone 2 relieve burning& itching
- rarely ,there's balanitis xerotica obliterans (unknown etiology): v. hard skin of penis & coitus is impossible

Linear (frontoparietal) morphea:

- Begins as a groove
- Common on extremities, esp. L.L.
- A band of hard skin extending along the limb, attached to the underlying tissues
- May result n shortening of the limb
- On the head , it leaves permanent scarring
- May result n hemiatrophy of the face

III. Dermatomyositis:

→ Chronic dis. Affecting skin & proximal muscles

→ Characterized by erythematous & oedematous patches on the face & extensor surfaces

→ C/C: difficulty n combing hair & rising from sitting position

→ In children its *ass.* With calcinosis

→ In adults with underlying malignancy
→ More n C
→ Heliotrope rash on the upper & lower eyelids (pathognomonic)
→ Diffuse non-scarring alopecia
→ MM involvement (mouth, eyes , nostrils &genitalia)
→ Sclerosis in 25% of cases
→ Sensitive 2 sunlight
→ Telangiectasia all over the erythematous patches
→ Knuckles erythema (characteristic but not pathognomonic)
→ Sclerosis of the cuticles of the nails (pathoghomonc)
→ Ischemic changes n post. nail folds.

THE END

Cutaneous Manifestations of CT diseases

SLE:
Risk of lupus: (F:M -6:1)
Ethnicity: African Americans has increased risk of nephritis, pneumonitis, DLE,
less photosensitivity
Earlier age ass. With higher mortality.

Cutaneous manifestations:
-Acute:
Erythema in Epidennis and dermis inflammatory cells, look for systemic manifestations, not scarring
Acute manifestation:

-poikiloderma: areas of increased and decreased pigmentation, prominent blood vessels, and thinning of the skin. (telangectasia, atrophy, hyper+hypopigmentation)
-Malar rash: Bilateral erythemay also known as butterfly rash. Transient resolves without scarring. Associated with Anti-ds-DNA and nephritis

-Subacute:
Photosensitive rash due to sun exposure with sparing of mid face
It affects epidermis and upper dermis. It appears as an annular raised erythematous lesion with red borders and central clearing.
Involves V area of the neck ears and extensors of extremities
Associated With Anti-Ro antibodies
10-15% association with SEE
Not scarring

-Chronic: discoid lupus erythematosis
-CDLE: most common very rarely develop SCC. 5- 10% will develop SEE later on.
-Brown red or violet scaly well-demarcated plaque with follicular plugging. T-helper infilterate.
-Scarring produces a depressed scar
-Dyspigmentation: hypopigmented center surrounded with hyper pigmented lesion.
-Causes Scarring alopecia
It affects mucosal membranes.
-Affects lower dermis and adnexal structures
Plaque type: can be generalized (widespread hard indurated seen in SLE) or localized (usually in head and neck).
Atrophic DLE and Veroccus DLE are other forms too
* * Add tacrolimus to the treatment options of DLE and note that anti-malarial drugs cause diffuse disfiguring.

Others:

- Chill bling: appear on extremities aggravated by cold
- Lupus tumidus: affects only the dermis not in epidermis or adnexal structures.
- Panniculitis of subcutaneous fat.

Systemic sclerosis (SS):
* * Limited or diffuse skin involvement
[SS----CREST----Morphea]
** To diagnose S5 u need 1 major or two minor:

-Major criterion: symmetric cutaneous sclerosis starting at distal limb ends
-Minor criteria: sclerodactylyl, pitting scars on digits, bibasilar pulmonary fibrosis
** Morphea: scleroderma limited to skin and blood vessels.
-Types of morphea: plaque-generalized-linear (paramidline)- guttate
-Stages of morphea: edematous plaque - indurated plaque - atrophic scar
-Salt and pepper appearance: area of hypopigmentation except around hair follicles (diagnostic for scieroderma)
** In systemic sclerosis u might also find telangectasias of lips and palms, capillary abnormality of nail fold and alternating loss and dilation of capillaries.
Calcinosis cutis, raynauds, cutaneous ulcers at tips of digits, beaked nose and puckered lips are also manifestations of SS.

Lichen sclerosis ET atrophicus:
Whitish hypopigmented cigarette paper like atrophic patch, affecting upper dermis.
Unknown etiology, occurs at *40-45* yrs. itching soreness and irritations in genital area, vulva perineal skin and in males balanitis and xerotic obliterans
TTx: topical steroids. Here we may use potent topical steroids in genital area.

Dermatomyositis:
It's an autoimmune disease of uncertain etiology. Proximal myopathy
Juvenile form: F=M, risk of vasculitis, calcinosis and cutaneous manifestaions but no risk of malignancy.
Adult form: F>M, age: 45-52, *25%* have occult malignancy
TTx: Immunosuppression with topical steroids and systemic steroids if myositis is present.

Skin infections

Viral infections of the skin

Herpes viruses , they are 8 types :

1- HSV1 and HSV2 " those are the common herpes that we all know , orolabial/ genital" HSV1 seen more in orolabial while HSV2 seen more in genital hepes
2- HHV 3 : this is the varicella zoster virus
3- HHV4 : this is the EBV that cause infectious mononucleosis and Burkitt's lymphoma.
4- HHV5 : this is the CMV one of the congenital viruses (TORCH)
5- HHv6+7 :
6- HHV8 : Kaposi sarcoma

Now we will talk about herpes simplex virus 1 and 2 :

Description of the herpetic lesion:
grouped vesicles with scalloped erythematous borders , superficial erosions or ulcers with/out secondary bacterial infection.

presentation :
1- primary infection :
usually present with prodromal symptoms , it present in one of the following :

** orolabial herpes

** genital herpes " one of the STD'" , this may cause :
1- neonatal herpes
2- keratoconjunctivitis
3- herpetic whitlow (lesions on fingers)

** Eczema herpeticum :
start as orolabial lesions then it will disseminate because patients have impaired skin barrier ,
usually present with systemic symptoms.

2- latent infection :

virus in this phase stay dormant in dorsal ganglia.

3- secondary infection or reactivation :

in this phase there are no or just mild prodromal symptoms , most common presentation is burning sensation at the site of infection.

Presentation either :
1- herpes labialis
2- herpes facialis " present with tengling sensation at the site of infection then vesicles erupt, usually present with recurrent lesions at the same site.

Triggering factors of herpes:
1- stress
2- any infections or illness
3-ultraviolet radiation

treatment of herpes infections :

Topical antiviral :	Systemic antiviral:
- Acyclovir 5 times / day - Pincyclovir every 2 hrs	- Acyclovir -Valcyclovir given IV in immunocompromized patients or if there is no response to oral medications

Note: give treatment in the prodromal period or in the first three days of the appearance of lesions.

Warts :

-Causative agent HPV dsDNA virus .

- Types of warts :

1- Common wart :

- most common type .
-Description: Dome shaped verrucous hyperkeratotic with or without fissures on the surface
- Verroucous means : benign skin lesion that is raised, firm and rough to touch.
- Exophytic lesions

2- Planter wart:

- Second most common type .

Planter wart	Corn مسمار اللحم
- Present at planter surface of the soles . - Endophytic lesions . - Painful lesions. -Multiple lesions at pressure and non-pressure areas. -It may cause destruction of surrounding dermatoglyphics . -Pinpoint bleeders .	-It is hyperplasia of sratum corneum -Present only at pressure areas -No pinpoint bleeders .

3- Plain or Facial wart:

- Usually present in children on their face however it may present in other age groups and on other sites .
- Description : Flat top ,brownish, pinkish, greenish , smooth surface papules appear on face.

4- Genital wart :

- One of the STD.
- Risk for cervical cancer.

Note :

Kobner phenomenon :

- It is the appearance of a lesion at the site of trauma .
- Causes : 1- Warts
  2- Psoriasis
  3- Lichen planus .
  4- Vitiligo .

Treatment for all types of warts :

-No benefit from giving Antiviral for warts .

- Treatment of choice is Destruction By :

1- Physical destruction by Cryotherapy.
2- Chemical destruction by Salicylic acid / Keratolytic agents / Lactic acid .

3- Cytotoxic agents by Podophyllotoxin .
4- Immunomodulators.

Molluscum Contagiosum :

- Causative agent is poxvirus
-**Description** : Firm umbillicated pearly shiny papules .
- Contagious by direct contact .
- If it presents on the child face it is not considered to be one of the STD
- If it presents in adults on genital area or on lower abdomen then it is considered to be one of the STD.
- Potato bodies appears under the microscope .
- May disappear without any treatment .

- **Treatment** :
1- Don't use antiviral drugs.
2- treatment of choice is destruction :
- physical destruction by Curette destruction.
- Chemical destruction by keratolytic agents.

Infestation:

- **Definition** : infection of the surface of the skin usually without invasion , caused by Mites
- **Types :**
1- Scabies مرض الحكاك / الجرب
2- Pediculosis القمل

1- Scabies :

- Acute disease .
- Highly contagious .
- Caused by mites called Sarcoptes Scabiei Hominis

- Main complaint is ITCHING :
1- more at night .
2- history of itching in other family members .
3-Distribution of lesions : all over the body more in interiginous areas with sparing to Back and Face HOWEVER they may be involved in :

A- Immunocompromised patients
B- Infants and Elderly patients.

-Incubation period 2 weeks – 2 months .

- Characteristic lesions may be seen in scabies :

1- Burrows lesion : slightly raised , linear, whitish to light brownish lesions in interweb spaces where the mites put their eggs.

2- In infants palmoplanter Vesicles are characteristic .

- Rule : Itchy papule on glans penile this is scabies until proven other wise .

-Tratment :

Instructions	Topical Tx	Systemic Tx
-كل العائلة لازم تتعالج - غسيل الملابس والأثاث على درجة حرارة 60 أو يغلقوهم بإحكام لمدة 10 أيام - قص الأظافر -give antihistamine for itching - Give antibiotic if there is 2ed infection.	- 1st line is Permithrin 5% one application only from neck to toes - Benzoyl peroxide 25% three applications over 8 hours . - Both choices : repeat another application after one week. - For infants <1 year use half the concentration .	- Use systemic tx for: - patient with severe impairment of the skin barrier (Ex: patients with atopic dermatitis , Icthyosis) - Immunocompromized patients. - patients who are not responding to topical tx give orar Ivermectin.

2- Pediculosis القمل :

A- Pediculosis Capitis (scalp lice)
B- Pediculosis Corporis (Body Lice)
C- Phthirus Pubis (Carb Lice)

A- Pediculosis Capitis :

- Causative agent mite that can be seen with naked aye.
- Specific for scalp.

Pediculosis	Dandruff
-Sticky , adherent to hair shaft -More proximal to hair shaft - Distribution : 1- Occipital areas. 2- Behind ears. - You can see mites under the microscope	- Not sticky - More distal to hair shaft.

Treatment :

Examine other family member but don't treat all of them if they are not affected (Not like scabies we treat all family members).

Topical	Systemic
-Permithrin Shampoo 1% do one application then the second one after one week. -Benzyl alcohol lotion -Malathion for resistant pediculosis.	- Oral Ivermectin

B- Phthirus Pubis (Carb Lice).

- One of the STD .
- May affect body hair , scalp hair, eyelashes ,eyebrows .

-Treatment :

1- permithrin

2- Malathion

3- Oral Ivermectin (use it if the eyelashes are involved because permithrin is very irritant)

- Post lice folliculitis is very common .

Bacterial infection of the skin:

- We classify them acoording to depth of infection in to :

1- Impetigo : داء جلدى يصيب الأطفال يسمى الحَصَف

- Most superficial infection .
- Epidermis bacterial infection.
- Most commonly caused by **staph** but maybe caused by step.
- Types : 1- Bollous Type caused only by staph. 2- Non-Bollous type .
- Highly contagious lesion .
- Description : Macule ⟶ superficial vesicles disappear within few days ⟶ Erosions of vesicles ⟶ crusts (Honey crust appearance)

- **Treatment :**

Topical	Systemic
-Topical Antibiotic (Fucidine)	-Usually we don't use systemic treatment except : 1- widespread area. 2- Lesions not responding to topical Tx. - Use either B-lactamase antibiotic , 1st generation cephalosporin or chlindamycin

2- Ecthyma :

- Deeper form of Impetigo (Epidermis + superficial dermis)
- Most common cause is **Strep**
-Description : eroded plaque with necrotic black center مثل حرق السيجارة.

- **Treatment :**

Topical	Systemic
-Topical Antibiotic (Fucidine)	-Usually we don't use systemic treatment except : 1- widespread area 2- Lesions not responding to topical Tx. 3- Lesions on face - Use either B-lactamase antibiotic , 1st generation cephalosporin or chlindamycin

3- Erysipelas :

- Deeper lesion (Epidermis + Dermis up to lymphatics)
- Most common cause Strep
- Description : sharp demarcated plaque.
- Associated with signs of inflammation 1- Hotness 2- Redness. 3-Oedema. 4-Tenderness
- Associated with prodromal symptoms 2-5 days after infection .
- Associated with systemic manifestation (fever, , enlarged fatigue , chills lymph nodes) .
- Classic site : face of elderly .
- Most common site : lower limb.

- Treatment :

1- No role for topical antibiotic.
2- Use oral penicillin for 10-14 days .
3- Use IV penicillin if no response to oral.

4- Cellulitis:

- Deeper lesion (Epidermis + Dermis + Subcutaneous tissue)
- Most common cause **Staph & Strep .**
- In children the most common type Staph .
- In diabetic patient mixed infection strep + pseudomonas
- Description : NOT well demarcated plaque
-Associated with signs of inflammation 1- Hotness 2- Redness. 3-Oedema. 4-Tenderness.
- Associated with systemic manifestation (fever, , enlarged fatigue , chills lymph nodes) .
- Types : 1- Bollous type 2- Non –bollous type.
- DDx : DVT

-Treatment :

1- cold compressors.
2- Leg elevation.
3- Systemic Antibiotic for 10 days.

Bacterial infections of hair follicles

All infections of hair follicles are caused by **STREP**

1- Folliculitis :

- Mild vs Severe / Superficial vs Deep.
- Description : Papules + Pustules on every hair follicles , seen on hair bearing area .
-Note : Acne (papules + pustules + COMEDONES specific for acne) .
- Treatment : topical antibiotic.

Psedo-folliculitis	Folliculitis
-Papules + Pustules caused by irritation (post shaving) - Negative culture . - Tx : Topical steroid .	- Papules + Pustules caused by Bacterial infection. - Positive culture. - Tx: topical Antibiotic.

2- Furuncles : الدمّل .

-Same as abscess But have follicular base.
- Description : Erythematous nodules (collection of pus) on follicular base .
- Treatment :
1- Incision and drainage .
2- Give Antibiotic oral and topical if it presents on face .

3- Carbuncles :

- Multiple furuncles with discharging sinuses .
- Present in immunocompromized patients / diabetic patients / post trauma.
- Treatment :
1- Incision and drainage (Mandatory)
2- Systemic Antibiotic .

The End

Done by : Anwar Jaber

Sexually Transmitted Diseases

Bacterial Infection	Viral infection
1-Gonorreha . 2- Chancroid . 3-Lymphogranuloma Venerum . 4- Granuloma Inguinale . 5- Syphilis .	1-HSV 2- HPV

Viral Infection :

Herpes infection (HSV1 + HSV2)

- Enveloped dsDNA virus .
- Usually the genital infection caused by HSV2 more than HSV1
- Clinical pattern :

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

Primary infection	Recurrent infection
1-More in number 2- Associated with 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 while 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 inguinali up to 1 year .

- Site of infection :

Males	Females
-Penile - Glans pennes -Pubic area	- Vulva -Labia majora -Pubic area -Buttocks

- **Clinical scenario :**
1- Constitutional symptoms .
2- Tangling 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) .

- **Diagnosis of Herpes :**
1-Tzank smear (you see multineucleated giant cells , inclusion bodies called Cowdry A body)
2- DFA (direct florescent Ab , green flurescence sensitive for serotype HSV1 &2)
3- PCR: Most sensitive , most specific
4- Viral culture .

- **Treatment :**
Goals of treatment :
1- Limits viral shedding .
2- Decrease pain.
3- shorts the duration of clinical manifestation.

Tx of primary infection	Tx of recurrent infection
-Acyclovir 200mg *5 for 10 days - Valcyclovir 1g *2 for 10 days	- Acyclovir 800 mg *2 for 5 days - Valcyclovir 1 g *1 for 5 days

Human papilloma virus 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- Rough keratotic skin colored or hyperpigmented brownish plaque on skin fold called (Condylomata acumninata).

Note :

Condolymata acumninata caused by HPV Condylomata lata cauded by 2ed syphilis.

- Treatment :

Destruction	Systemic
1-Physical destruction by cryotherapy (very painful) 2-Cytotoxic agent like : - Podophyllotoxin cream 0 .15% - Imiquimod cream 5% for 3-4 months. -Green tea catechins cream 15 %. - Cidofovir gell - INF- alpha (painful)	Use it only if genital warts are extensive or if topical treatment failed 1- Oral Isotretinoin . 2- Oral Acitritin. 3- Oral Cidifovir .

- 75% of genital warts are self-limiting within 2 years.

Bacterial Infections :

Syphilis :

- Causative agent is Treponema Pallidium
- Incubation period up to 3 months
-Types :

1- Primary syphilis 2- Secondary syphilis 3- Latent syphilis
4- Tertiary syphilis 5- Congenital syphilis

Primary syphilis :

-Description : Solitary , painless , firm , indurated ulcer on genital area or extra genital area like lips and fingers .

-Lesion in this stage called Chancre , which appears within the first 3 weeks of infection and heals within another 2-3 weeks.

- Within the next 3 weeks – 3 months hematogenous dissemination occurs causing secondary syphilis .
- Diagnosis : 1- Dark field microscopy (most sensitive)
  2- RPR / VDRL .
  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 papilosquemous lesions appear.
- Has palmopanter manifestation (Copper- pin spots , bronze colored , surrounded by collarette scales) this stage is highly contagious.
- Codylomata lata one of the forms of secondary syphilis (moist colored skin)
- patchy alopecia may complicate the infection .
- You may see Necklace of venus (Hypopigmented area on neck)

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 2ed syphilis 2- Most of the relapses of 2ed syphilis happen during this period.	1-Starts after one year after resolution of 2ed syphilis 2- Proceed either into complete cure or into tertiary syphilis.

Tertiary syphilis :

- Most common cautaneous 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.

Congenital syphilis :

Early congenital syphilis	**Late congenital syphilis**
1-Age less than 2 years 2- Median age 3 months 3- Present with : - Snuffles - Bullae - Perioral fissures .	1-Age preschool 2- Present with skeletal deformities . 3- Hutchinsan's triad : - Intestinal keratitis - Teeth abnormalities . - Deafness .

Treatment :

Primary + Secondary + Early latent : Benzathine penicillin 2-4 IU *1 IM

Late latent + tertiary : Benzathine penicillin 2-4 IU *1 / week*3

If you have neuro or ocular manifestations : Benzathine penicillin 4 IU Q 4 hrs for 14 days .

Gonorrhea :

- Gram negative diplococi , 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.

- **Diagnosis :**

1- Gram stain 2- Culture 3- PCR 4- DNA probes

- **Treatment :**

1- Ceftriaxone 250 mg *1 IM

2- Azithromycin 1g *1 IM to prevent infection by clamydia trachomatis .

Chancroid :

- Causative agent is Hemophilus duceri
- Incubation period 3-10 days .

-Always symptomatic. (start as multiple painful purulent ulcer with undermined edges.

- In gram stain you can see chaining pattern characteristic to hemophilus duceri

Single lesion on chancroid	Chancre lesion of secondary syphilis
1-Painful. 2- Multiple . 3- Enlarged tender L.N	1-Painless. 2- Solitary . 3- Enlarged Non-tender L.N

- Treatment :

1- Azithromycin 1g single dose.

2- Ceftriaxon 1g IM single dose.

Lymphogranuloma Venerum :

- Causative agent is Chlamydia trachomatis serotype L1-L3 (L1-L3 / Lypmhogranuloma).
- 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 .
- **Diagnosis** : 1- Serology 2- PCR
- **Treatment :**

1- Doxycyclin 100 mg *2 for 3 weeks .

2- For pregnant ladies give erythromycin 500 mg .

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
- **Diagnosis :** Geimsa / wright / Leishman stain for Danovan Bodies .
- **Treatment :**

1- Doxycyclin 100mg *1 1st line

2- TMX .

The End

Done by Anwar Jaber

Acne Vulgaris

Acne Vulgaris:Chronic inflammatory condition of the pilosebaceous unit .
* Pilosebaceous unit is composed of a pilosebaceous gland, duct and a hair follicle.

- Pathophysiology:

1. Increase in sebum production
2. Abnormal Follicular keratinization and desquamation due to inflammatory mediators (Blockage) .
3. Bacterial proliferation "propionbacterium acne" .

- Stages :

A) Non inflammatory:
comedones; either open (black heads), or closed (white heads).

B) Inflammatory :
Papule—papulopustule – Nodulocystic..

- Causes ::

1. Increase in sebum production , which occurs at puberty .
2. Hormonal abnormalities : testosterone causes increased mitotic activity and cell proliferation , that's why we order hormonal profile for pts complaining of acne.
3. Bacterial proliferation convert the triglyceride component of sebum into free fatty acids which causes chemotactic increase in inflammatory cells. (prostaglandin- like substances production)

- Types :

1. Adolescent acne :
   a. Premenstrual acne -- 1 week before menses, mostly on the chin.
   b. Tropical acne .– in hot humid areas.
   c. Acne excoree : linear hyperpigmentation due to scratching

2. Occupational acne :
   (usually affecting unusual sites) either due to friction , or chlorinated hydrocarbon

3. Cosmoticacne : caused by obstruction of pilosebaceous duct.(No increase in sebum secretion).

4. Acne medicintosa :related to drugs -- androgen , steroids, anti-epileptic, anti-TB,vitb12, progesterone (OCPs),vit D overdose. Lithium.

5. Acne fuliminants : Associated with systemi symptoms (severe joint pain, low grade fever, High ESR, probability of MOF)

- How to take Hx ?

1. Ask about menses , if it regular or not.
2. Ask about drugs : vit b12 injections , steroids :: here clinically acne looks monomorphic w/out comedones
3. Ask about previous Rx for the acne
4. Ask about occupation :: E.g acne on lateral side of neck is common in عازفي الكمان

- Treatment ::

1. Comedones : keratolytic Agents (Retinoid Family) ::

   - **Differen** (adapalene) given once daily at night , NO SUN EXPOSURE , may cause dryness and irritation .
   - **Benzac AC** (benzoyl peroxide) = Keratolytic and anti-inflammatoy.
   - **Isotrexin gel** = topical tretinion and erythromycin
   - Optimal (tretinion gel)

2. Erythematous papules ::

   - **Topical antibiotic** (clindamycin solution twice daily)
   - Topical **erythromycin with zinc.** (Zineryt solution)
     *Clindamycin and benzac ac have synergistic effect together.

3. Papulopustular acne : **systemic abs** ::
   - doxycycline 100mg /day:
   - If so severe give doxycycline 200mg/day for one month then 100 mg/day
   - Duration is 6-7months before we call it failure of Rx.
   - Side effects : 1. Irritation to the esophagus; that's why you should tell your pt to take it with his big meal and to drink a cup of water after it and not to lie down for at least half an hour after the pill
     2. increase intracranial pressure.

- 3. Discoloration of teeth and bone and so never give it for a child <2 yrs or a pregnant lady.

4. For PIH , give **skinoren**(azelaic acid) twice daily

5. 13-cis Retinoic acid (**isotretinoin**) aka Roaccutane : (Important)

   * Indications :

   - 1. Nodulocystic acne
   - 2. Acne with scarring tendency
   - 3. Acne refractory to other methods of treatment
   - 4. Pts with psychological stress due to acne (relative indication)

   * Dose :0.5-1mg/kg/day

   - When to stop Tx? If cumulative dose reaches 120-150 mg/kg
   - E.g : 60kg pton 40mg/day dose for 5 months , cumulative dose would be 40*30days*5month divided by 60 kg .

   - Side effects :
   - 1. Xerosis in skin and mucous membranes including lips , nose and eyes
   - 2.Epistaxis due to xerosis
   - 3. Increases sensitivity to sun →use sunblock
   - 4.increase liver enzymes→ obtain a base line before the start of tx and then monitor monthly.
   - 5.cholesterol and triglycerides may also increase.
   - 6. Increased ICP, DON'T use it with doxycycline
   - 7. Pregnancy category X :absolute contraindication and pt should be on 2 methods of contraception and after she stopped no pregnancy is allowed for 2-3 months later on.

6. Post acne scar treatment :subcision scar surgery , ablation (CO_2 laser), and dermal needling .

The End

Done By Anwar Jaber

Printed By Summer Rashid

Icthyosis(داء السماك)

Definition: Excessive generalized dryness and scales of the skin.

Classification of Icthyosis :

1- congenital Icthyosis
2- Icthyosis associated with syndromes.
3- Acquired Icthyosis.

CONGENITAL ICTHYOSIS :

1- Icthyosis Vulgaris
2- X- linked recessive Icthyosis.
3- NBIE (Non-Bollous Icthyosis Erythroderma)
4- Bollous Icthyosiform Erythroderma .
5- Lamellar Icthyosis.

Icthyosis Vulgaris :

- Autosomal dominant disorder.
- Most common type of all types of Icthyosis.

-Presentation :

- starts after the 2ed or 3ed 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.
-Involves the extensors WITH sparing of flexors.
- Minimal itching.
- You may find perioral and ear pinna scales.

DDX :

- Other types of Icthyosis.
- Atopic dermatitis.
- Asteatotic eczema.

Description of scales : Small, Whitish, Branny semiadherent scales.

X-linked recessive icthyosis.

- Starts at the 1st 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.

NBIE	Lamellar Icthyosis
-Autosomal Recessive.	- Autosomal Recessive.
- Presentation at birth.	-Presentation at birth.
- Present as Collodion baby +++.	- Present as Collodion baby ++.
- Excessive scales ++++.	- Excessive scales ++++.
- Erythema & Erythroderma ++++.	-Erythema & Erythroderma ++.
- Hyperkeratotic palms and soles.	- Hyperkeratotic palms & soles.
- Ectropion (everted Lid) & Eclabium	-Ectopion & Eclabium (everted lip)

NOTE :

Collodion Baby :

collodion is a membrane surrounds the fetus during the intrauterine fetal life and during the early days of life.

-Transient appearance of generalized glistering, taut yellow film at birth (dipped in wax)

Bollous Icthyosis Erythroderma .

- Autosomal dominant disorder.
- Present at birth.
- Characterized by erythroderma that is associated with flaccid blisters , once they ruptured they are replaced by very thick hyperkeratotic area called (Epidermolytic Hyperkeratosis) .

Icthyosis associated with syndromes:

1- Netherton's syndrome:

- Autosomal recessive
- Includes: a- Atopic dermatitis b- Icthyosis linearis circumflexa c- Trichorhexis invaginatum or nodosa (brittle hair with so called bamboo deformity)

2- SjÖgren-larsson:

- Autosomal recessive
- Includes: a- Neurological deficit b- Mental retardation c- Icthyosis

3- Refsum's syndrome:

- Autosomal recessive
- Includes: a- Retinal degeneration b- Neurological deficits c- Icthyosis

4- Rud's syndrome:
- Includes: a- Icthyosiform b- Erythroderma c- Epilepsy d- Mental retardation

5- KID syndrome:
- Includes: a- Keratitis b- Icthyosis c- Deafness.

6- IBIDS:
- Includes: a- Icthyosis b- Brittle hair & nail c- Impaired intelligence d- Decreased fertility e- Short stature

7- Kallman's syndrome:
- Includes: a- Hypogonadism b- Anosmia c- Icthyosis (X-linked) d- Neurological deficit

Acquired Icthyosis :

-May be considered as paraneoplastic features (lymphoma , Leukemia) .
- Asoociated with chronic 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.

How to manage patients with Icthyosis :
1- For those of congenital icthyosis and icthyosis associated with syndromes , the only thing you can do is supportive Tx .

2- Acquired icthyosis : do supportive care + treat the underlying cause.
Supportive care :

Topical treatment	**Systemic treatment**
-Emollient.	Systemic Retinoid
Keratolytics : don't use high concentration on large surface area because of risk of systemic absorption.	
Urea containing preparation : 1- prevents transepidermal water loss 2- Hydrophilic agent (absorbs water inside the epidermis) 3- Improve the epidermal hydration. 4- Has some antiseptic effect. 5- Has keratolytic effect at high concentration.	
Air modifier for baby in NICU	

DONE BY : Anwar Jaber

MAZEN AL-MANSOUR

DISORDERS OF PIGMENTATION

Normally the skin owes its color to four pigments:

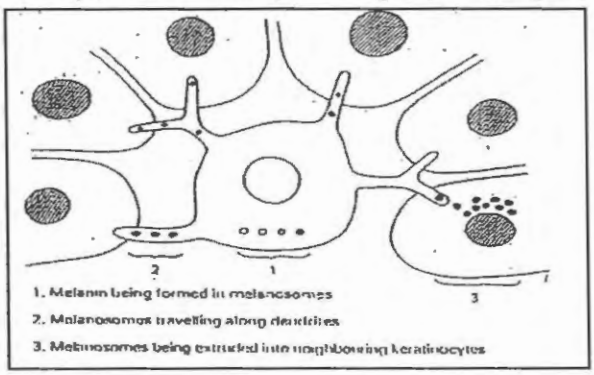
- Melanin: the most important pigment.
- Oxyhemoglobin: excess oxyhemoglobin results in plethora.
- Reduced hemoglobin: excess reduced hemoglobin results in cyanosis.
- Carotene: excess carotene as in people who eat large amounts of carrots (e.g. special diets) develop orange discoloration of the palms.

MELANIN PRODUCTION

- The skin is formed of 3 layers: epidermis, dermis and hypodermis (the subcutaneous layer). The epidermis is separated from the dermis by a basement membrane.
- The epidermis is formed of 4 layers:
  - * basal cell layer
  - * acanthous cell layer[1]
  - * granular cell layer
  - * keratin layer

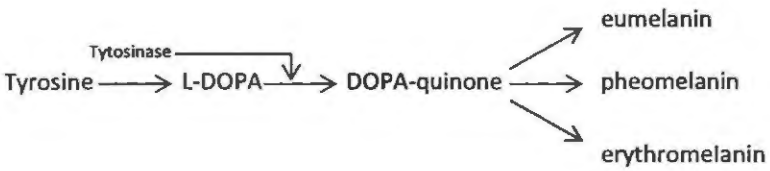
The epidermis is derived from the ectoderm, the dermis and subcutaneous tissue from the mesoderm and melanocytes from the neural crest. Since neurons and melanocytes share a common origin it is not surprising to share a few characteristics in common. Melanocytes have a similar cell shape to neurons since both of them have dendrites.

Melanocytes are interspersed between basal layer keratocytes in the epidermis. They rest on the basement membrane. They form dendrites which reach the surrounding basal cell keratocytes. A term was applied to one melanocyte and the basal cell keratocytes to which it is attached to by dendrites, **epidermal melanin unit.** On average one melanocyte is attached to 36 cells in an epidermal melanin unit. Melanocytes are also present in hair follicles, in the choroid of the eye, inner ear and leptomeninges. (See the figure)



[1] Other names of this layer include: stratum spinosum, prickle cell layer, squamous cell layer and malpighian layer.

Melanin the most important skin pigment is synthesized in melanocytes in specialized membrane-bound organelles called melanosomes. Inside melanosomes melanin is synthesized from tyrosine.



The first step is catalyzed by an important enzyme which is tyrosinase.
Tyrosinase has an enhancing effect on the second step as well. The end products of this reaction is melanin.

There are 3 types of melanin:

1. Eumelanin: which is deep brown-black in color.
2. Pheomelanin: which is light brown in color.
3. Etythromelanin: which is red and it is present mainly in hair.

The amount of each type of melanin produced in different from one person to another and is genetically determined. And this gives the different colors of the skin and hair.

The melanosomes pass in 4 different stages to reach its mature form. After that they pass through the dendrites to reach the basal cell keratocytes to which they are injected into. The melanosomes cluster around the nucleus of the keratocyte. The basal cell keratocytes then divide, one of the daughter cells move up to replace the lost keratocytes. Melanosomes in the keratocytes disintegrate at a certain rate which differs from one person to another.

The importance of melanin is in its ability to absorb ultraviolet radiation from solar rays. These rays have a harmful effect on the nuclei of keratocytes. When these rays reach the keratocytes they produce free radicals which can damage the DNA and result in mutations which contribute to carcinogenesis. So melanin has an essential role in prevention of malignant transformation of keratocytes.

However, melanin has some disadvantages. The absorption of UV light results in decrease in the transformation of 7-dehydrocholesterol to cholecalciferol. This accounts for the increase of incidence in rickets in black children. Also black skin absorbs more light than fair skin. This increases the heat load. In order to avoid an elevation in body temperature negroids have a more efficient heat loss system this explains the larger amount of sweat a black individual produces after certain effort compared to a fair skinned individuals after the same effort.

The ratio of melanocytes to basal cell keratocytes (1:10) was found to be equal in males and females and in people of different races and skin colors. So how can the different skin colors be explained?

In addition to the different amounts of each type of melanin produced in each individual there 5 more difference between individuals of different skin colors:

1. *Number of melanosomes inside keratocytes.* It was found that black individuals have a larger number of melanosomes inside keratocytes than white individuals.
2. *Size of melanasomes inside keratocytes.* It was found that black individuals have larger melanosomes inside keratocytes than white individuals.
3. *Packaging of melanosomes inside keratocytes.* It was found that the melanosomes inside keratocytes in black individuals do not cluster unlike those of white individuals.
4. *Distribution of melanosomes inside keratocytes.* It was found that the melanosomes inside keratocytes in white individuals are aligned eccentric in the cell in the side of the cell most subjected to UV light. While they are distributed evenly in black individuals.
5. *Disintegration of melanosomes inside keratocytes.* The rate of disintegration is higher in white individuals than black individuals. So that the superficial layers of the epidermis do not contain melanosomes in white individuals.

HYPERPIGMENTATION DUE TO MELANIN:

Localized hyperpigmentation:

1. ***Freckles (النمش):*** benign hyperpigmented macules with no increase in the number of melanocytes. It is an autosomal dominant disease mainly occurring in children and young adults especially fair-skinned. It increases by exposure to sunlight.
2. ***Lentigo:*** benign hyperpigmented macules with an increase in the number of melanocytes. There are several types of lentigo, some of which are premalignant (lentigo maligna) but the common senile lentigo is absolutely benign. Senile lentigo usually presents in elderly especially fair-skinned on sun exposed areas like dorsum of hands, bald salps of elderly males and face in order of frequency. Clinically, lentigo cannot be differentiated from freckles. They can be differentiated under the light microscope.
3. ***Chloasma (melasma) (الكلف)*** : benign hyperpigmented patches mainly occurring on the face. It is precipitated by sun exposure. The infraorbital area is usually spared since it is protected from the sun by cheek bones and sunglasses. It may occur in both males and females but it is far commoner in females due to hormonal factors. Both estrogen and progesterone are involved (but estrogen is more incriminated). This explains the association between chloasma on one hand and pregnancy and oral contraceptive pills on the other hand. Chloasma is treated by bleaching agents (e.g. derivatives of hydroquinones).
4. ***Pigmented nevi*** (these will be discussed in the lecture about skin tumors)

Generalized hyperpigmentation:

1. ***Addison's disease:*** In Addison's disease the diminished levels of cortisol in serum will result in loss of its feedback inhibitory effect on the pituitary and resultant increase in ACTH production. ACTH is the end product of the breakdown of a larger molecule; *proopiomelanacortin.* A byproduct of the breakdown of this molecule is melanocyte stimulating hormone (MSH), which has a stimulatory effect on melanocytes. Thus, the elevated levels of MSH result in hyperpigmented lesions particularly in the oral mucosa,

palmar creases, and sites of previous scars[2].

2. ***Systemic sclerosis:*** one of the stages of the cutaneous involvement of this disease is the stage of hyperpigmentation.

3. ***Thyrotoxicosis.***

4. ***Chronic hepatic insufficiency.***

5. ***Chronic renal failure.***

6. ***Drugs:*** *chlorpromazine* (which is an anti-psychotic) and ***hydontoin*** (anti- epileptic).

7. ***Postinflammatory hyperpigmentation:*** this is a common result of inflammatory skin disorders particularly the itchy disorders due to scratching. An example is eczema.

HYPERPIGMENTATION NOT DUE TO MELANIN:

In this type other pigments are responsible for the skin color.

A. Normal pigments

- Oxyhemoglobin: increase in oxyhemoglobin in polycythemia for example results in plethora.
- Reduced hemoglobin: increase in reduced hemoglobin results in cyanosis
- Carotene: increase in carotene as happens with people on special diets containing large amounts of carrots leads to orange pigmentation of palms.

B. Abnormal pigments

' ***Endogenous***

- methemoglobin
- sulphahemoglobin
- carboxyhemoglobin
- bilirubin and biliverdin (most common endogenous abnormal pigment)
- hemosiderin

' ***Exogenous***

- metals (gold, silver, mercury...)
- bismuth
- mepacrine (atabrine)
- dinitrophenol
- tattoos (most common exogenous abnormal pigment)

Hypopigmentation and depigmentation disorders

- albinism
- vitiligo
- drugs (chloroquine, monobenzene ether of hydroquinone[3])
- postinflammatory hypopigmentation

---

[2] Dr Azmi Taleb once said that recently it was discovered that the hyperpigmentation is mainly due to the action of ACTH itself which has a direct effect on melanocytes.

[3] This compound was used by Michael Jackson to change his skin color.

Albinism

• It is a group of genetically determined disorders of melanocytes characterized by a decrease or absence of melanization in skin, hair, and eyes, with associated defects in other tissues. It is inherited as an *autosomal recessive* disease in most cases but other modes of inheritance were also reported.

• **There are two clinical categories of albinism:**

1. *Oculocutaneous albinism:* this can be subdivided into:
    - *tyrosinase positive:* which gives a positive result in tyrosinase test. This test is carried out by impregnation of the hair follicles in a solution having a high level of tyrosinase enzyme. If the hair gets some stain this implies a tyrosinase positive result and this indicates *partial defect of melanization due to the absence of tyrosinase enzyme only.* This is the *commonest type* of oculocutaneous albinism. This type is characterized clinically by very fair skin, golden-yellow hair, a prominent red reflex (using a fundoscope) and risk of developing freckles upon sun exposure.
    - *tyrosinase negative:* Gives a tyrosinase negative result, that is the hair stays unstained. This indicates *complete defect in melanization due to defects in other steps.*
      Clinically this type is characterized by pink skin (reflecting the underlying vasculature), white hair, and a very prominent red reflex.
2. *Ocular albinism:* this is a rare type of albinism.

• **Associated defects:**

- Photophobia
- Squinting
- Errors of refraction
- Abnormalities in the optic pathway
- Horizontal and/or rotatory nystagmus

• **Treatment:** photoprotective creams and regular medical follow up to detect any precancerous or cancerous conditions: (e.g aktinic keratosis, squamous cell carcinoma, basal cell carcinoma and malignant melanoma). These individuals tolerate temperate climates better, but visual impairment will constitute the greatest disability.

Vitiligo:

• condition in which pigment disappears from the skin in a bilateral symmetrical pattern. The skin becomes white (i.e. depigmented) due to destruction of melanocytes.

• **Epidemiology**: It affects all races. The world incidence is around 1%. Positive family history in 30-40%. The peak age of incidence *is* the 2 and 3 decades.

• **Etiology**: The strongest theory is autoimmunity supported by the association of other autoimmune diseases such as Hashimoto thyroiditis, pernicious anemia, type I diabetes mellitus, Addison's disease, myasthenia gravis, halo navus and alopecia areata.

• **Clinically**: It is a slowly progressive disease. The characteristic lesions are depigmented macules lacking scalling. The lesions are usually bilateral and symmetrical. Sometimes they have a hyperpigmented rim. The hair is usually spared except in long standing lesions.

- **Sites of predilection:**
  a. Hyperpigmented areas: face, genitalia, areola, axillae and groins.
  b. Areas of friction: dorsa of hands, elbows, knees, ankles and dorsa of feet.

GOOD LUCK

Hypopigmentation and Depigmentation

Vitiligo appears under woods light to be ivory white in color (Depigmented well defined lesion). If it is not defined and gets vague then it's a hypopigmented lesion think of healing eczema or scar...

- Lichen planus, eczema and acne cause hyperpigmentation
- Post-inflammatory hypopigmentation occurs in eczema and psoriasis

Vitiligo

It's an Acquired idiopathic autoimmune disease which is Common in our population.

Koebner' s phenomenon: eruption of original disease at site of injury! bum as in psoriasis or lichen planus is also seen in vitiligo

Classification:

1. Diffuse: >90% of body surface area
2. Localized
3. Segmental at dermatome distribution seen in children which supports the neurogenic theory
4. Generalized: the most common type
    - Universal
    - Vitiligo vulgaris
    - Acrofacial

Treatment: according to percentage of diseased skin to surface area

<2S%: topical steroids, topical tacrolimus

25-50%: phototherapy UVA,UVB, PUVA, Solar PUVA, melanocyte grafting, monobenzyl hydroxyl, total depigmentation

>95%: total depigmentation: excision of normal skin. This is what Michael Jackson did!

Facial spots respond beautifully to steroids. While generally treatments are not satisfactory to patients.

Disorders of pigmentation

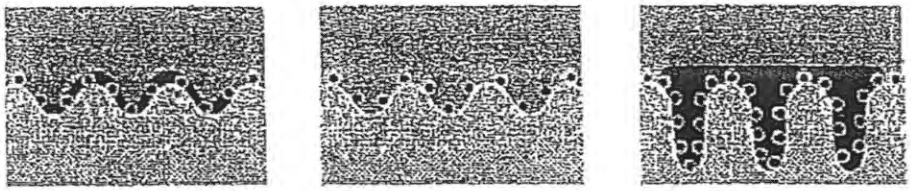
Vitiligo

1. Generalized vitiligo
2. Segmental vitiligo
    - Is restricted to one part of the body, but not necessarily to a dermatome. It occurs earlier than generalized vitiligo, and is not associated with autoimmune diseases.
    - Trauma and sunburn can precipitate both types.
    - Spontaneous repigmentation occurs more often in this type than in generalized vitiligo

    - Sometimes leprosy must be excluded by sensory testing and a general examination.
    - Treat with cosmetic cover and sunscreens or sun avoidance.
    - Do not promise a cure.

Freckles and lentigo

- Increased melanin is seen in the basal layer of the epidermis without any increase in the number of melanocytes, and without elongation of the rete ridges
- In contrast to freckles,
- Lentigines have increased numbers of melanocytes and elongation of the rete ridges. They should be distinguished from freckles, from junctional melanocytic naevi and from a lentigo maligna.



Freckle Normal Lentigo

Skin Tumors

Any of skin layers (Epidermis , Dermis , Subcautanerous tissue) may develop tumor either benign or malignant tumors.

We classify skin tumors in to :

1- Epidermal and its appendages tumors.
2- Melanocyte tumors.
3- Cautaneous T. cell lymphoma.
4- Skin metastasis .

Tumors originating from epidermis layer and its appendages :

1- Benign tumors of epidermal origion:

- Seborrheic Keratosis :
Present clinlcally as slightly elevated papules or plaque with dry , rough , hyperkeratotic , pigmented surface , and characteristically they have stuck on appearance.

2- Premalignant tumors of epidermal origion:

- Actinic Keratosis :
Present clinically with erythematous plaque with adherent scales , mostly affecting fair skinned people on sun exposed area like dorsum of the hand , forearm, face, forehead, dorsum of the nose.
it has a risk of malignant transformation in to SCC up to 20%

Note :
signs of chronic sun damage :
1- Actinic Keratosis .
2- Mottled hyperpigmentation.
3- Skin atrophy.
4- Loss of elasticity .

3- Carcinoma in situ :

- Bowen's disease : SCC in situ of the skin .

- Erythroplasia of queyrat : SCC in situ of genitalia , clinically it looks like an eczematous or psoriatic plaques.

4- Malignant tumors of epidermal origin :

A – Basal Cell Carcinoma (BCC):

- Originate from basal cell layer of epidermis .
- Slowly growing tumors that is locally invasive and destructive BUT rarely metastasize.
- Most commonly affects people who have fair skin with history of heavy sun exposure .
-Mostly erupting after middle age.
- Most common site is in the upper part of the face

- Clinical types of BCC :

1- Noduloulcerative BCC :
- Present with slowly growing nodules
- Smooth shiny surface .
- Pearly pinkish indurated edges (Hard edges)
- Prominent telangectasia .
- As it progresses it becomes central superficial ulcer with rolled up edges (Rodent Ulcer)
- Best management in BCC is complete histologically controlled surgical excision then do reconstruction surgery .

2- Morphea- like basal cell carcinoma :
- Slightly elevated plaque with sclerotic area and ill-defined borders.

3- Superficial basal cell carcinoma:
- Superficial BCC resembles an eczematous lesion.
- Mostly occurring on trunk and in most cases lesions reach a large size due to delayed diagnosis
- No itching.

4- Pigmented basal cell carcinoma :

- Not everything pigmented must has melanocytic origin.
- Dermoscope is needed to examine the lesion to differentiate between melanocytic origin and non- melanocytic origin.

5- Nevoid basal cell carcinoma syndrome :

- Autosomal dominant disorder affects more than one organ.
-Skin features : 1- palmoplanter pits 2- multiple basal cell carcinoma.
-Can affects boney system , endocrine and neurological system.
- Eruption starts during adolescent or childhood .

Histopathology for BCC :

1- presence of tumor masses inside the dermis.
2- Retraction spaces around tumor masses.
3- Fibrovascular stroma.
4-Presence of connection between one tumor mass and the overlying epidermis (Must be seen at least in one cut)

BCC differentiate in to one of the skin appendages :

1- Sweat gland (Adenoid differentiation)
2- Hair follicles (Keratotic differentiation)
3- Sebaceous gland (BCC with sebaceous differentiation)

Treatment :

1- Complete histologically controlled surgical excision of tumor with 5-6 mm safety margin under local anesthesia . (BEST)
2-Radiotherapy successful in thin tumors .
3- Topical chemotherapy successful in thin tumors.
4- Topical immunotherapy (Imiqumod)
5- Photodynamic therapy (PDT) :
use of visible light spectrum 400-700nm in the treatment of benign and malignant tumor of epidermal origin (Not same as phototherapy that use UV light) .
6- Cryotherapy.

Squamous Cell Carcinoma :

- It originates from squamous cell layer.
- slowly growing tumor But faster than BCC
- Locally invasive and destructive, but can metastasize into regional lymph nodes.
- Most common site is lower face especially lower lip and ear pinna
- Rarely erupts in previously healthy skin , rather it erupts in damaged skin (sun exposure area, Actinic keratosis, scars , burns , chronic , dermatitis)

-Clinical presentation :

1-Ulceration (chronic dirty ,slough , non-healing ulcer with indurated base)
2-Nodules .
3- Verrucous plaque mostly in the top of actinic keratosis .

NOTE:

Keratoacanthoma :
1- Is a tumor of hair follicle that is clinically present as perifollicular nodules with slightly elevated hyperkeratotic center.
2- Rapidly growing tumor reach its maximum size in 4-6 weeks , if lift untreated it needs another 6-8 weeks to completely disappear leaving ugly scar.
3- One of the variant of SCC.

Histopathology of SCC :

1- Presence of tumor masses in the dermis.
2- Lymphatic inflammatory infiltrate between tumor masses.
3- Horn pearls : abrupt abnormal immature keratin formation.
4- Presence of pleomorphisim (variable sizes , atypical cells, mitotic figures).
Atypical cells : larger cell with large hyperchromatic nucleus
Prominent nucleoullous.
Increase N:C ratio.

Treatment : the same for BCC.

Melocytic tumors:

Malignant melanoma :

- Originates from melanocytes , highly aggressive tumor with early mets through lymphatics & hematogenous route.
- Most common site: in females : lower limb in males : trunk
- Major types of melanoma (90 % of all melanoma) .

1- superficial spreading malignant melanoma 57% :

- Most common type .
- Presentation : slightly elevated plaque with
  - A- Asymmetry (we cant divided it into two equal half)
  - B- Borders (ill-defined borders)
  - C- color (variable)
  - D- Diameter > 6mm
  - E- Extension

In top of this plaque we may find nodules or ulceration .

2- Nodular melanoma 21%:

- Presentation : nodules that is usually pigmented .
- Considered to be the most aggressive clinical type due to high vertical growth rate.

3- Lentigo maligna melanoma 3% :

- Presentation: patches with
  - A- Asymmetry B- ill-defined borders C- Variable color
- Most common site is face in elderly individual above 70 years .

4- Acral lentigionous melanoma 4 % :

- Affects acral skin .
- Presentation : plaque with ill-defined borders , variable colors, induces destruction of surrounding tissue , may erupt beneath nail plate.
- Have higher predilection in colored individual rather than white individual.

NOTE :

- In diagnosis of melanoma the best method to be used is Excisional biopsy.
- Exception : large lesion in a cosmetically important site Ex: face .

Staging :
TNM : T (thickness)
Investigation : US for L.N , Pan CT , CXR.

Treatment :
1- Surgical excision with 0.5-2 cm safety margin (depends on tumor thickness) .
2- Alpha interferon (immunotherapy)
3- Chemotherapy (regional or systemic)
4- Radiotherapy .

Poor prognostic factors of malignant melanoma :
1- Tumor thickness (clark's level of invasion histologically)
2- Presence of histological or clinical ulceration.
3- The number of mitotic figure per mm2
4- Presence of microsasatellite metastasis or in transit metastasis
- Small metastatic lesions within 2 cm from the primary tumor .
- In transit : small metastatic lesion lying within 2 cm between the primary tumor and the first draining lymph node.
5- Localization in head & neck region .
6- Gender (males have poor prognostic factor)
7- Age .

NOTE :
point 5,6,7 are not evidence based factors .

Benign Melanoma (Called Nevi)
- Nevi : developmental defect leads to proliferation and aggregation of normal melanocytes that undergo certain changes to be called nevus cells.
- Most commonly erupts during pregnancy and puberty due to ACTH spurts (melanocyte stimulating hormones)

Classification :
1- Usual nevi .
2- Unusual nevi.

Usual nevi :

1- Junctional type :
- Clinically it is a flat symmetrical macules with well defined borders and homogenous color.
- Aggregations of nevus cells which called nits present at dermoepidermal junction and from here the name junctional nevi comes.

2- Compound type :
- Slightly raised symmetrical papules, with well defined borders and homogenous color.
- Nits present at dermoepidermal junction & intradermally .

3- Intradermal type :
- Dome shaped nodules with flesh color up to dark brown .

Unusual nevi :

1- Spit'z nevus : Red – pink papules on the face of children.

2- Halo nevus : central hyperpigmentation surrounded by a halo of depigmentation.

3- Nevi with verrucus surface .

4- Congenital hairy nevi : they have high risk of malignant transformation than the rest of nevi, and it is proportional to its size.
Management : regular follow up VS excision .

5- Nevus spilus : Dotted hyperpigmentation on nevi ground of café au lait spots .

6- Becker's nevus : Associated with hypertrichosis , mostly affects males and mostly erupts at the pubertal age.

Dermal Melanosis : it occurs when the rest of melanocyte locate deep down in the dermis.

1- Blue nevus : Blue color due to error of refraction (depth)

2- Nevus of ota : Bluish – greenish discoloration involving the ophthalmic and maxillary division

of trigeminal nerve with ipsilateral involvement of the sclera.

3- Nevus of ito : Same as ota but on trunk .

4- Mongolian spot : Bluish- greenish discoloration on lumbosacral spine and buttock , it fades spontaneously during the first two years of life.

Primary cautaneous T. cell lymphoma :

1- Mycosis fungoides has three stages : 1- patches 2- plaque 3- tumerous stage.
2- Sezary syndrome .

Metastasis to skin :

1- Lymphoma.
2- Leukemia.
3- Solid tumors.

The End

Done By : Anwar Jaber

Hair

One of the skin appendages which are : 1-hair 2-nail 3-sweat gland

Types of hair :

1-lanugo hair :fine, long hair, unmedullate, covers the fetus during the intrauterine period of life, it sheds one month before birth replaced by vellous hair.

2-vellous hair: fine, short hair, non pigmented, unmedullated replaces the lanugo hair before birth.

3- terminal hair: long, coarse, pigmented and medullated.

There is a balance between vellous & terminal hair once it is disturbed :

Male: male pattern alopecia "terminal hair replaced by vellous hair "

Female : hirsutism "fine vellous hair on face & breast replaced by terminal hair "

Anatomy of the hair :

**** longtudinal cuts :**

1-bulber portion : *deepest portion.

*characterized by having dermal papillae which surrounded by germinative layer of hair called hair matrix .

*gives nutrition to hair by diffusion.

2-suprabulber portion : *just above bulber layer.

*ends at the insertion of arrector pili muscle .

3-isthmus : lies between arrector pili muscle and sebaceous duct.

4-infundibulum .

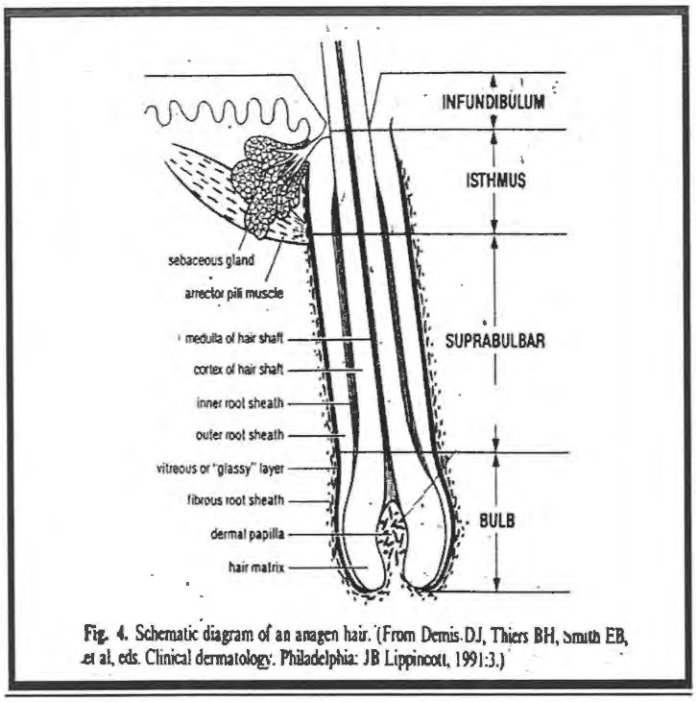


Fig. 4. Schematic diagram of an anagen hair. (From Demis DJ, Thiers BH, Smith EB, et al, eds. Clinical dermatology. Philadelphia: JB Lippincott, 1991:3.)

**cross section :

1- Hair medulla 2-hair cortex 3-hair cuticle 4-inner & outer root sheath

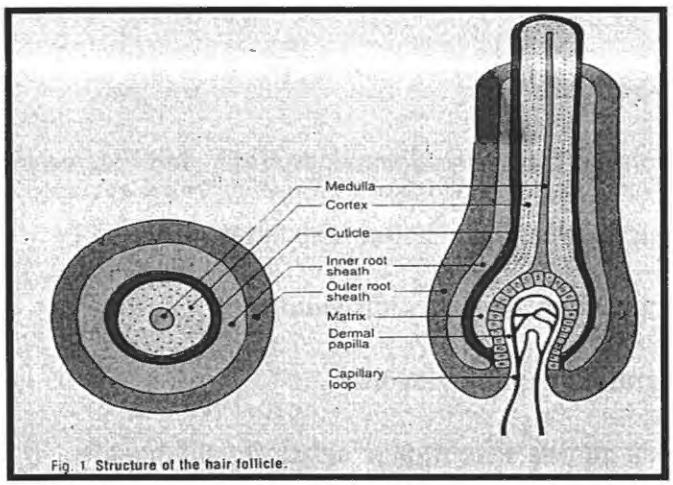


Fig. 1. Structure of the hair follicle.

Hair cycle:

Anogen	Katogen	Telogen
Growth phase of hair	Involution phase of hair	Resting phase of hair
3-4 years long	Few weeks	Few months
85% of scalp hair	1-2% of scalp hair	12-13% of scalp hair
Characterized by well formed dermal papillae	Separation between dermal papillae and hair	Hair completely dropped and replaced by new anogen hair with well formed dermal papillae

Hair loss (alopecia) classification :

1-scarring(cicatricial) :

a-diffuse

b-localized(patchy)

2-non scarring(non cicatricial):

a-diffuse

b- localized(patchy)

Diffuse non scarring alopecia :

1-telogen effluvium : premature entry of the hair into telogen phase, so hair in anogen phase decrease an in telogen phase increase.

-post febrile

-post surgery

-Postpartum and difficult child birth

-surgical shock

-drugs : anticoagulant , vitamin A

-crush diet

2-anogen effluvium : loss of hair during anogen phase by ***chemotherapy*** because it targets growth cells .

3-endocrine dysfunction : hypo/hyperthyroidism, hypoparathyroidism , hypopituitary states

4-nutritional :

Protein malnutrition, disturbing protein synthesis , iron deficiency, hemocystinuria.

5-congenital ectodermal dysplasia : hidrotic , anhidrotic

6-hereditary hair shaft abnormalities :

-pili torti (twisted hair 180° around longitudinal axis)

-monilethrix (beaded hair)

-trichorexis nodosa (frayed nodes)

-trichorexis invaginata (bamboo hair)

-wooly hair

7-very severe seborrhoic dermatitis

Patchy non scarring alopecia

1-hereditary male pattern alopecia

2-hereditary female pattern alopecia

3-alopecia areata:

-unknown mechanism but immunological origin is the most accepted.

-usually follows psychological stress.

-clinically : rounded or patches area of hair loss without any signs of inflammation.

-most common site is the scalp.

-if the extension involved the whole scalp it called *alopecia totalis* , and if involved the whole body hair *alopecia universalis* .

-on examination : exclamation mark hair (narrower along the length of the strand closer to the base)

** bad prognostic factors :

1-early age of onset

2-duration ≥ 2 years

3-involvment of the margins (ophaisis) especially at occipital margins

4-association with atopy

5-association with down syndrome

6-involvment of sites other than the scalp like beard

Treatment :

1-topical : topical steroid , topical calcinurin inhibitors, contact sensitizers (induction and periodic elicitation of an allergic contact dermatitis by topical application of Diphencyprone of high concentration)

2-intralesional : intralesional steroid

3-systemic : cyclosporine, PUVA "AVOID THE USE OF SYSTEMIC STEROID"

4-tinea capitis.

5-secondary syphilis : "in tertiary syphilis the alopecia is patchy scarring alopecia"

6-traumatic alopecia :

*physical trauma : 1-tichotillomania : in psychological disorders patients, produces hair pulling tic sign (hair broken at different layers)

2-frictional alopecia

3-marginal tractional alopecia

4-pressure and massage alopecia

*chemical trauma : hair straighter

Diffuse scarring alopecia :

Extremely rare caused by physical agents (burns, x-ray)

patchy scarring alopecia : most common, caused by :

1-physical agents (burn, x-ray, hotcomb)

2-deep infections

3-specific diseases :a-CDLE

b-LP

c-morphea

d-pseudopelada de brocq : scarring alopecia without clear cause

EXCESSIVE HAIR GROWTH

1-hirsutism :excessive hair growth in female patient on unwanted sites

Causes : 1- ovarian (benign or malignant disease) : A-PCOD

B-tumors

2-adrenal (benign or malignant disease) : A-Cushing's syndrome

B-CAH

3-exogenous : androgen therapy

4-idiopathic of familial

2-hypertrichosis: excessive hair growth usually localized

Causes: 1-congenital hairy nevi

2-Spina bifida

3-drugs : cyclosporine

Treatment of both : treat the underlying cause then laser

The End

Done by Anwar Jaber
printed by

Nails:

Dr. M.Sharaf

Function of the nails:

1- protection.
2- cosmetic.
3- Scratching.
4- Squeezing objects.
S- Gripping.
6- picking small objects.
7- Chipping objects.

Nail apparatus:

1. Nail plate.
2. Nail matrix.
3. Nail bed.
4. Nail folds.
5. Hyponychium.

Nail plate:

- located on each dorsal surface of each distal digit.
- Flat, transparent, rectangular in shape, formed slowly by the nail matrix.
- The color is pink due to heavy vascular network through the nail bed reflected the transparent nail plate.
- Nails grow continuously throughout the life and not shed.
- Finger nails growth = *0.5mm/wk.*
- Toe nails growth = 0.15- 0.2 mm/w. (so toe nail growth is slower).
- $1/4^{th}$ of the nail plate is present beneath the post nail fold.
- The half moon white area (linula) is the visible part of the matrix.

Contents of Nail plate:

1. Nail keratin.
2. Phospholipids — responsible for flexibility of the nail plate.
3. Free fats — long chain of fatly acid — extrinsic source.
4. Calcium — extrinsic source from soaps.
5. Cu, Mg, Zinc, Iron.

Nail Bed

- The surface is not smooth, there are longitudinal grooves to provide strong attachment between nail bed and nail plate.
- These grooves are the causes of linear splinter Hemorrhage.
- The epidermis of the nail bed is thin + keratinization occurs without keratohyaline granules + there is no granular cell layer.

- Connective tissue fibers normally are parallel to the surface of the skin but here they are vertical to provide strong attachment.
- No subcutaneous fat in the nail bed, also to provide stronger attachment (directly bone).

Nail Plate 3 layers:

1. Dorsal thin layer.
2. Intermediate.
3. Ventral layer (on nail bed).

Nail Matrix:

- Thick epidermis.
- Have melanocytes.
- Keratinization without keratohyaline granules + no granular cell layer.
- Nail bed is formed slowly by matrix.

Hyponychium:

- Skin present beneath the free end of the nail.
- Entry of bacteria, viruses is from here, except candida.
- Normal epidermis.

Post nail fold :

- beneath it is the matrix.
- composed of 2 surfaces of epithelium:

* Ventral layer.
* Dorsal layer.

- Dorsal layer: forms the visible skin.
- Ventral layer: nail cuticle (Eponychium) is formed by the ventral layer, which protects the nail from infections.
- Candida: basal infection, enters from the site of cuticle.

Lat folds: Attached tightly to the nail.

Bld vessels: paired digital arteries.

*Pattern of nail disturbances:

Depends on the site of pathology:

1 - Matrix:

A - destruction of the matrix:

- Anonychia (no nails).
- Inherited Door syndrome.
- Complete or partial.

Pg. 97

-Anonychia in sever scleroderma.

B - Atrophy of the matrix.

- Decrease in the size of the matrix.
- Thin nail, the nail may
- Show thin band (Partial atrophy) Or the entire nail becomes thin (complete atrophy).
- Atrophy of nails in lichen planus.
- Retinoids (in psoriasis or Acne) cause atrophy.

C- Hypertrophy of the matrix:

-Thick nails, as in psoriasis, fungal infection.

D - Abnormal keratinization of the matrix:

1- The nail may change its appearance:
white spots and pits.

2- Nails exhibit fragility:
- break off at free edge.
- splitting of the nail into layers.

3- Longitudinal ridging of nails.
- common in old people.
- radiation damage.

4- Unusual nails shapes:
- spoon shaped (koilonychia).
- clubbed nail.

* in enzems pits are large in size.
* nail atrophy in onychotillomania.
*people who destroy their nail.

Leukonychia:

- White opaque nails.
- Congenital.
- Could be spots in abnormal keratinization.

Leukonychia: with acquired liver disease + anemia.
Leulconychia + spoon shaped koilonychias nails : in deafness disease.
Leukonychia : band across the nail in hypoalbuminemia.
Striate leukonychia : striae.
Leukonychia due to onycholysis in hyperthyroidism.

Old people have:

- thin nails.
- Longitudinal ridges.
- Ragged cuticles.

Causes of lamellar splitting:

Proximal: psoriasis.
Lichen planus.
Retinoid Tx.

Distal: Chemical injury.
Old age.
Repeated nail wetting.
Polycythemia vera.

2 - Nail folds

Connective tissue surrounding the nail plate 2 laterals + one dorsal, attached tightly to provide water proof.

1) paronychia (inflammation).
   - Acute or chronic.
   - Common in house wives + nail biters.
   Causes:
   - infective: virus, bacteria, Candida, fungus.
   - Inflammation due to skin disease.
   - Drugs — Retinoid.
   - Occupational wet works.
   - Trauma— finger sucking.
   - Cosmetics + irritants.
   - Systemic disease: collagen vascular disease, Sarcoid, leukemia, vasculitis.

2) tumors of nail fold.
3) Ingrowing toe nails cause paronychia.
4) Periungual warts. Tx: cryo therapy because laser destroys the matrix.
5) Ragged Cuticle:
   Thickened irregular + hyperkeratotic.
   Common in:
   a. Dermatomyosites.
   b. Nail biters.
   Paronychia in finger sucking cause candidal infection.

3 - Nail Bed:

1) Splinter Hemorrhage:

Trauma.
Subacute bacterial endocarditis.
Vasculitis.
Cirrhosis.
Psoriasis.

2) Tumors:

*Pitting of nails:

Common:

Psoriasis.
Alopecia areata.
Eczema.

Uncommon:

Pityriasis Rosea.
2ry syphilis.
Normal.
- Self induced periungual skin picking + ragged cutile : very painful.
- Sq. all carcinoma of the bed.

3) Onycholysis-: Separation of the nail from nail bed.

- Partial
- Total → onychomadesis

Causes:

- Trauma.
- Occupational.
- Pseudomonas.
- Phototoxic-reactions included by drugs.
- Contract allergy to nail polish.
- Thyroid dysfunction.
- Candidiasis.
- Psoriasis.

THE END

Additional notes to nails

- Lunula is visible part of nail matrix
- Hyponychia is main route of infection
- Nail changes in Retinoid treatment: atrophy, lamellar splitting, proximal nail splitting, paronychia
- Glomus tumors may occur in nails
- Paronychia occurs in housewives and nailbiters: its inflammation of nail bed that can be acute or chronic.
- Zinc deficiency in children causes paronychia

OSCE

By: Bayan Al-Khdour

Dermatology

* OSCE *

* Describe the lesion of :-

- Lichen planus (L.P)

(violaceous, polyhedral, polygonal, Flat-topped papules covered w Fine scales and very minute grooves, (wickham's striae)

⑥ sites : Flexors (around wrist + ankle)

- plaque psoriasis

④ well defined, darkly erythematous plaques, beefy in color, covered w heavy silvery scales

sites : - knee, elbow (extensor surfaces)
- scalp
- sacral area

- Guttate psoriasis (guttate : point)

⑤ numerous small rounded red macules w Fine scales (lacking dry thick silvery scales)

- mostly effect the trunk

- DLE

↳ discoid lupus erythematosis

Brown, red or violet scaly well demarcated plaque w Follicular plugging, adherent scales (T-helper Infiltration)

- superficial BCC

→ rare ① in morphology it resembles (a patch of eczema)

② mainly on the trunk

③ usually retains a large size due to delayed diagnosis

[1]

* Nodulo ulcerative BCC

slowly growing nodule, w/ shiny smooth surface, pearly indurated edges with telangiectasia

---

* poor prognostic factors of MM:

① depth of primary tumor
② sex (♂)
③ site (neck, scalp)
④ presence of ulceration (clinically or histologically)
⑤ clark level of invasion
⑥ Microsatellite, Intransit Mets
⑦ age (old age)

---

* Causes of:

① acquired ichthyosis
- chronic hepatic Ds
- chronic renal Ds
- Thyroid and parathyroid Ds
- lymphoma and Malignancies
- drugs (nicotinic acid, statin)
- AIDS, leprosy, sarcoid
- malapsorption syndrome

② Focal scaring alopecia
↳ Trauma (physical, chemical, Thermal)
↳ Infections → Fungal (Tinea capitis)
Bacterial (deep Folliculitis) / pyogenic
Viral (Herpes Zoster)
non-pyogenic (TB + leprosy)
↳ skin Ds → DLE
lichen planopilaris / rare
Scleroderma

pg. 109

Tinea capitis

① Definition ⇒ Fungal infection of scalp hair

② ttt ⟶ systemic (Griseofluvin for 1 month)
(no need for Topical antifungals لازم في الكريمات)

---

what is the most common type of MM, and describe it, plz ?

- superficial spreading MM ⇒ (75%) of cases
- characterized By
  - A ⟶ Asymmetrical patches
  - B ⟶ ill-defined Borders
  - C ⟶ variable shades of Color
  - D ⟶ diameter > 0.6 cm

(may have nodules on its top or may ulcerate)

- mention types of psoriasis:
  1. plaque pattern
  2. pustular pattern
  3. Guttate pattern
  4. psoriatic erythroderma
  5. Flexural pattern (psoriasis inversa)

- describe nail involvment in psoriasis :-
  1. pitting (most common)
  2. onycholysis describes either (oil drop sign) or (salmon patch)
  3. thickinning of nail plate
  4. sub-ungual hyperkeratosis
  5. dystrophy of nails
  6. distal splinter hemmorhage

- describe scalp involvmt ⇒ (silvery stick scale), hair loss [3]

Treatment of psoriasis :-

* Topical
  1. Keratolytics (salicylic acid)
  2. dithranol
  3. Tar preparations
  4. Topical steroids (avoid potent)
  5. vit-D analogue
  6. Topical Retinoids
  7. calcineurin inhibitors

* systemic
  1. systemic retinoids
  2. MTX
  3. cyclosporin A
  4. Fumaric acid esters
  5. Biologic agents (TNF Inhibitors)

* phototherapy
  - PUVA , UVB , UVA
  - Bath PUVA
  - cream PUVA

* laser therapy (308 - 312 nm)

---

* Seborrhoeic dermatitis Vs atopic dermatitis

no FHx	+ve FHx
greasy scales	papules + vesicles
mild pruritis	severe pruritis
!! age at birth	age > 3 months
good prognosis	chronic Fluctuate

4.

* Roaccutane

13-cis retinoic acid (isotretinoin)

- Indications :-
  1. severe nodulocystic acne
  2. moderate acne but unresponsive to conventional Rx
  3. Inflammatory acne and scars
  4. acne w severe psychological problems

- Contra-Indication :-
  - pregnancy → bec it is Teratogenic
    catagory (X)
    only in ♀

- side effects
  1. Teratogenic
  2. ↑ TG , ↑ cholesterol
  3. ↑ Liver enzymes (Transaminase)
  4. dryness of mucous membrane (cheilitis)
  5. Conjunctivitis , epistaxis
  6. headache, (pseudo tumor cerebri)
  7. paronychia

- monitering
  (TG, LFT , cholesterol)

- dose
  - 0.5-1.5 mg/kg/day
    total dose 120-150 mg

5

* definition of Acne Vulgaris
  ↳ chronic Inflammatory condition of the pilo-sebaceous ducts (unit)
  ↳ Types of Acne
    ① Inflammatory (papule, pustule, nodule, cyst)
    ② non Inflammatory
      ↳ white comedo / closed
      ↳ Black comedo / open

---

* describe pityriasis rosea or (Sic جرب) ??
- clinically starts as:
  (single oval or round well defined patch with Fine scales on the edges (herald spot)
  Then (Few days - 2wks) later ⇒ Multiple lesion well develop (scaly); collaret / (From neck to mid thigh)
- Benign papulosquamous Ds, asymptomatic
- self limiting, non-Infectious
- Unknown etiology but some studies (show relation to: HHV6, HHV7, allergy)
- excellent prognosis; no sequale behind
- no-recurrence (usually)
- on back, follow the direction of ribs giving (christmas tree appearance)

---

* classification of Bacterial Infxns of skin :-
① Impetigo → Involves epidermis
② ecthyma → Involves epidermis + upper part of dermis
③ erysipelas → Involves epidermis; dermis till level of lymphatics
④ cellulitis → Involves epidermis, dermis, and subcutaneous tissue.

6

* mention clinical types of warts :-
① common warts (≈ 70%)
② plane warts
③ plantar warts
④ genital warts
⑤ Filiform warts
⑥ digitate warts

---

* describe (plane warts) ?
- mainly in children
- mostly on the Face
- characterized by (slightly elevated papules, smooth surface, Flesh colored (Flat) warts)

---

* Tt of warts ?
① physical destruction by cryotherapy (N , ~~-169~~ -196)
② chemical destruction (Keratolytics such as salicylic acid)
③ Topical cytotoxic (5-FU)
④ Topical Immunomodulator

---

* guttate psoriasis
(he asked about its relation to something ?? ∞ ^
" Infections (of group A strep) ⇒ may trigger the Disease

---

* read page-8 , (Tabels very Imp)

7

* objectives of dermatoscope
① to know if the lesion is melanocytic or not
② if melanocytic, it is Benign or Malignant

Pityriasis versicolor الزهري
- affects adults and young adolescents
- common sites (trunk, neck, UL)
- lesions are (Macules covered w Fine scales of different colors (white, brown, black)
- Dx ⇒ by wood's light shows gold fluorescence
- lipophilic (so not common in (young + old) bc lipid profile
- non-Itchy
- could be acute or chronic ⇒ recurrent and Resistant
- fungi are part of normal Flora and became pathogenic in
① poor hygiene
② over sweating
③ prolonged course of Ab and steroids
④ swimming
⑤ Immuno compromized pts
⑥ pregnancy
⑦ Cushing Ds
- ttt by → Topical antifungal → shampoo
→ systemic

8

* Describe histopathology in psoriasis ?

① hyper Keratosis
② para Keratosis
③ Munro's abscess
④ hypo or agranulosis
⑤ acanthosis
⑥ Tortuous capillaries in dermal papillae
⑦ supra papillary plate thinning
⑧ presence of mixed inflammatory infiltrate in the upper dermis

---

* classification of hair loss :-
↳ localized vs generalized
↳ scaring vs non scaring

---

* causes of non scaring diffuse alopecia

① Telogen effluvium
↳ ⓐ post Febrile
ⓑ post partum
ⓒ emotional stress
ⓓ surgical shock
ⓔ drugs
Ⓕ ↓ Food Intake (sudden)

② anogen effluvium (cyto toxic drugs)
③ endocrine dysfunction
(hypo or hyper thyroidism, parathyroid, hypopituitarism, DM, pregnancy)

Continue ☝ 9 →

④ Congenital ectodermal dysplasia
⑤ Nutritional
(esp iron deficiency anemia
⑥ Hereditary hair shaft abnormality
⑦ very severe seborrheic dermatitis

Woods light → use in :-
① helps in differentiation btw (depigmented and hypopigmented lesion)
- depigmented ⇒ well defined, ivory white in color
- hypopigmented ⇒ disappear.
② in Fungal Infection (esp Tinea)
③ in vitiligo
④ to know if (hyperpigmented lesion is deep or superficial

---

pitting of nails :
↳ ① psoriasis
② alopecia areata
③ chronic dermatitis
④ idiopathic

nail changes in LP :
① : longitudinal striation
② thinning
③ onycholysis
④ nail dystrophy
⑤ pterygium

---

* bad prognostic factors :-(in psoriasis)
① Involvment of face
② eyebrows Involvment
③ occurrence in 1st 2 decades
④ Flexural and pustural types

enjoy with my Dishes
Bayan Alkhalaf

10

- This is a BLANK page for Notes

- This is a BLANK page for Notes

- This is a BLANK page for Notes

- This is a BLANK page for Notes

- **This is a BLANK page for Notes**