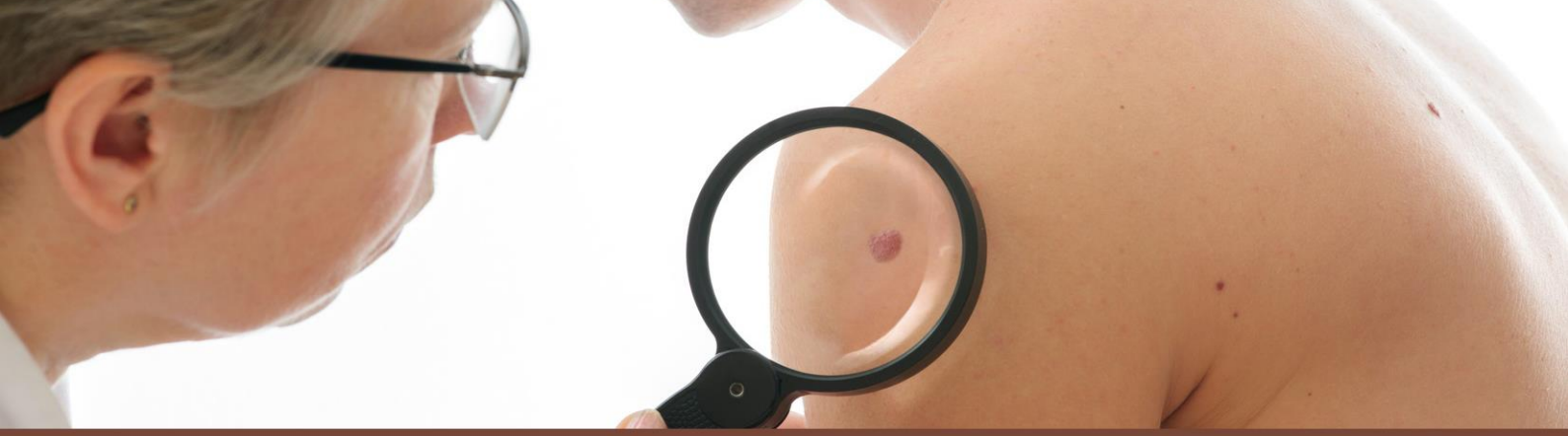

433 Teams

DERMATOLOGY

Lecture (6)

Hair disorders & pigmentary disorder of skin

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1-Hair disorders

Objectives:

- **Normal anatomy of hair follicle and hair cycle**
- **Causes, features and management of non-scarring alopecia, Particularly:**

-Alopecia areata

-Androgenetic alopecia

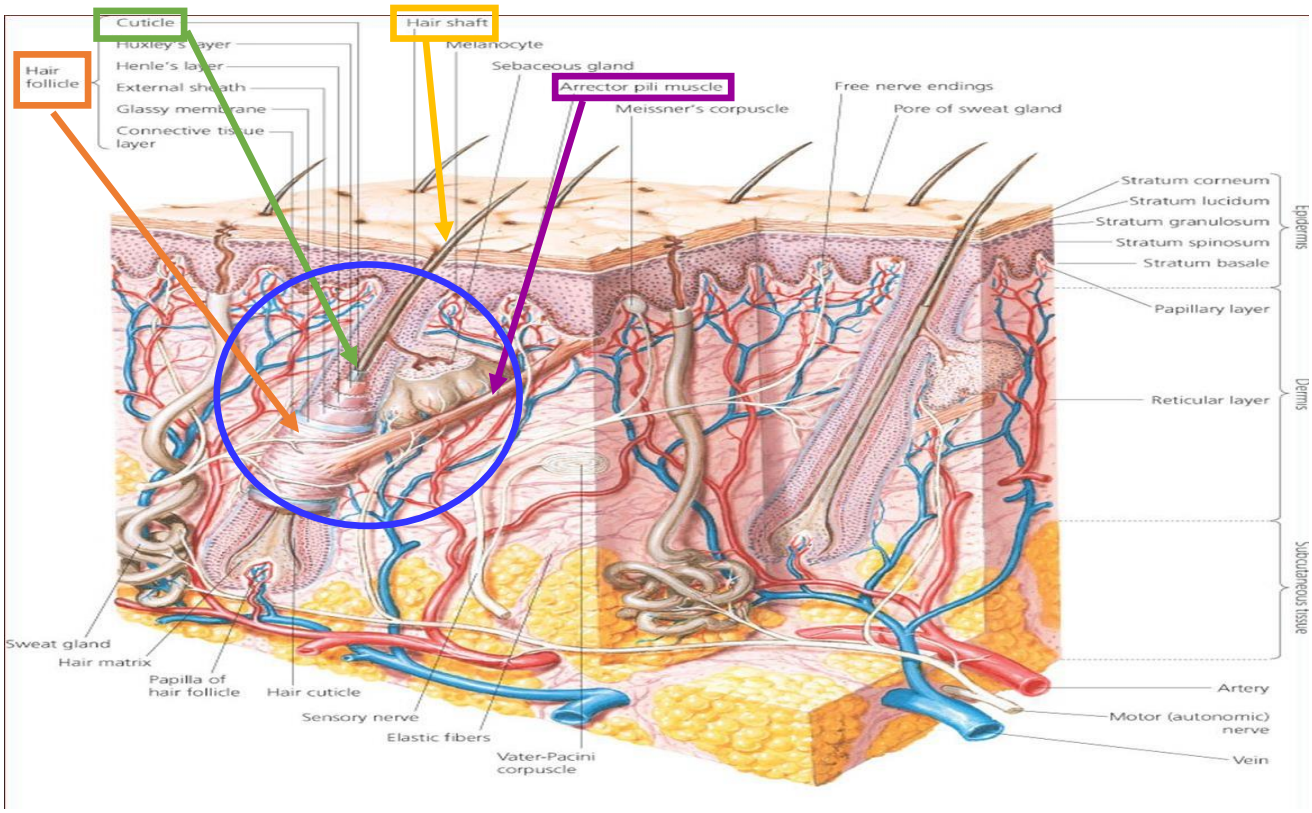
-Telogen effluvium

-Anagen effluvium

- **Causes and features of scarring alopecia**

Color index: **slides,** **doctor notes,** **432 notes** **Doctor's notes (groupF)**

• Anatomy of hair follicle:



*The pilosebaceous unit is the affected part in acne.

- The Arrector pili muscle is attached to the hair follicle the contraction of this muscle causes the hairs to stand on end (goose bumps).
- The cuticle is the outermost part of the hair it has 7 layers, it protects the cortex of the hair and gives hair the healthy appearance.

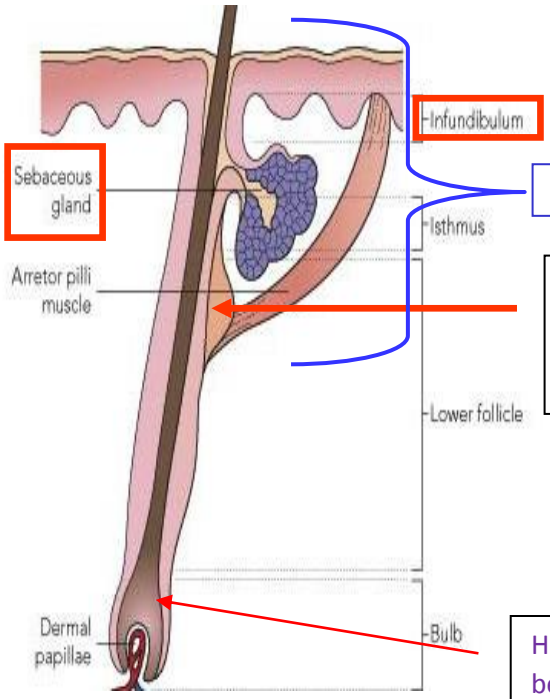
-Dyes, heat etc. can cause holes in the cuticle which later on will damage the hair. Conditioners, Oils &creams work on closing these holes to make the hair appearance healthy.

-The hair is composed of medulla, cortex and cuticle.

Q/ How many hairs in the body?

Ans/ 5 millions hairs in the body,
100,000 in the scalp.

• Growth rate: 0.3mm/day for scalp hair
1cm/month.



If the hair loss happened from the bulge or above this is will be permanent.

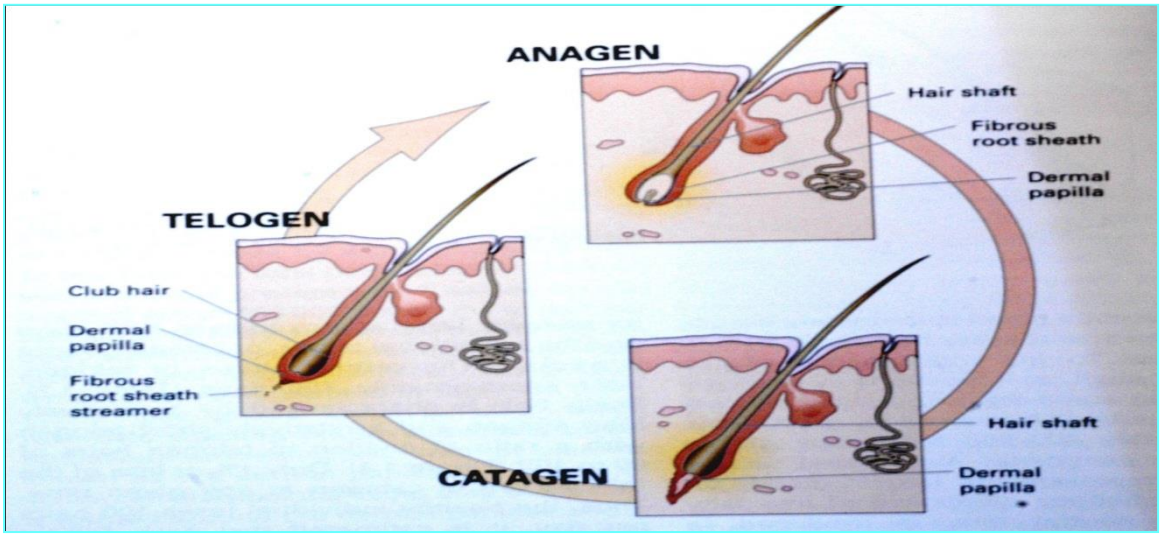
- This is the hair bulge it contains stem cells. This part is affected in Lupus.
- Laser hair removal acts by damaging this part.

Hair loss happen below the bulge this will be temporary as in Diet, Alopecia.. etc.

Hair types:

- Lanugo: Covering fetus and newborn baby.
- Vellous: Thin, less in color, light and short it covers the whole body, has no Medulla.
- Terminal: Thick and dark color, seen for example, on scalp, eyebrow or axilla. It has both Cortex and Medulla.
- Androgenic hair : Grow during & after puberty in males & females (e.g. axilla, pubic area and crown area in scalp specially in males).

Hair Cycle:



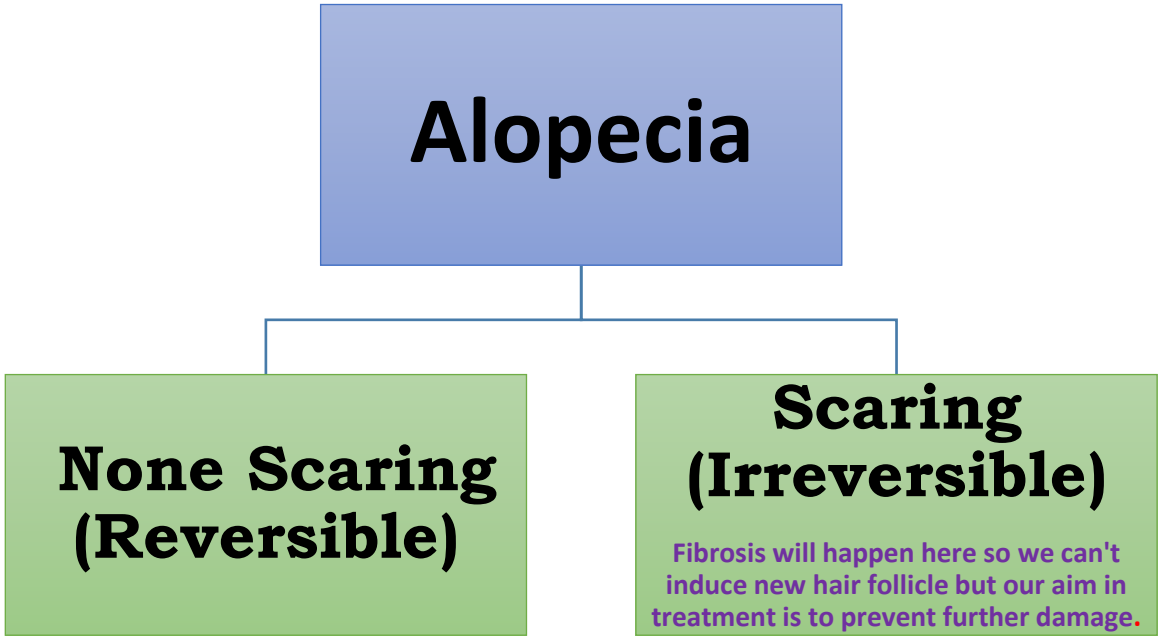
Phase	Region	Time	Description
Anagen	Scalp	2-5 years	Growing of hair. The length of this phase determines the length of the hair
Catogen	Scalp	2 weeks	A short phase of conversion from active growth to the resting phase with degradation of hair follicles.
Telogen	Scalp	2-3 month	A resting phase at the end of which the hair is shed and new hair grow.

Anagen (growth phase): 80% of the hair is in this stage. People recorded in Guinness World Records with a very long hair is because they have a long Anagen phase.

Catagen (transition phase): represent 2-5% of the hair.

Telogen (resting phase): represent 15% of the hair, before the hair goes out we call it exogen.

Alopecia:

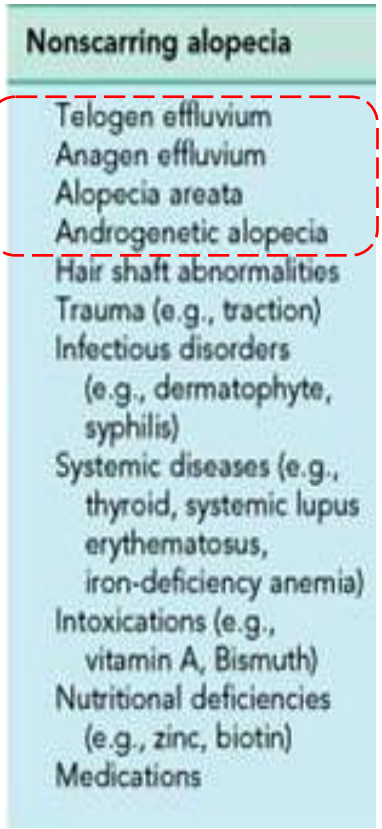


Nonscarring alopecia	Scarring alopecia
Telogen effluvium	Developmental defects (e.g., Aplasia cutis) → Baby born with focal hair
Anagen effluvium	Infections (bacterial, viral, fungal)
Alopecia areata	Trauma (irradiation, thermal or caustic burns)
Androgenetic alopecia	Neoplastic disorders
Hair shaft abnormalities	Lichen planus (lichen planopilaris), lupus erythematosus, morphea, scleroderma, sarcoidosis
Trauma (e.g., traction)	Keratosis pilaris atrophicans
Infectious disorders (e.g., dermatophyte, syphilis)	Folliculitis decalvans
Systemic diseases (e.g., thyroid, systemic lupus erythematosus, iron-deficiency anemia)	Dissecting cellulitis of the scalp
Intoxications (e.g., vitamin A, Bismuth)	Acne keloidals
Nutritional deficiencies (e.g., zinc, biotin)	Pseudopelade
Medications	Alopecia mucinosa

Very imp, it's the most common type of lichen plans that can cause scaring alopecia.

- Could be genetic from childhood or acquired in adults due to use of hair relaxer, keratin or protein treatments. These substances work by breaking the bonds in the keratin chain making the hair weaker, Pregnant ladies shouldn't use them.
- Traction of hair as in stress "like in medical students". In repeated trauma it will transform into scaring.
- Fungal infection with no inflammation as in Gray batch and black dots these wild not cause scaring, while others as Favid and Kerion which wild cause inflammation and scaring.
- Acute Lupus is non-scaring while discord lupus is scaring.
- The most common nutritional deficiencies in KSA is Vit D and iron.

Non-scarring alopecia (reversible):



1- Alopecia Areata:

- Sudden Acute hair loss (localized, generalized "the whole scalp" or universalis "the whole body").
- Alopecia Areata affects up to 2%.
- 75% Self recovery with 2-6 months.
- Etiology: 30% +ve Family history.
  Autoimmune.
  Psychological factor.

Clinical findings:

- **Well demarcated** non-scarring hairless patch.
- Exclamation point (**!**) (Pathognomoic feature).
- Nail: pitting, ridges (indicating severe alopecia).
- No scales, no scars and no Erythema.

Types of alopecia areata:

Good Prognosis
- ✓ Localized partial (1-2).
- ✓ Localized extensive (more than 2).

Bad Prognosis
- ✓ Alopecia ophiasis (occipital and paraital area). → Susceptible to fibrosis.
- ✓ Alopecia totalis (Total hair loss in the scalp).
- ✓ Alopecia universalis (whole body).

Bad prognostic signs:

- Young age.
- Atopy.
- Alopecia totalis, universalis, ophiasis.
- Nail changes.

Diagnosis:

- Clinically
- H/E: sworm bees

- In Alopecia Areata the patient will wake up and find loss of hair suddenly.

Treatment:

- ✓ Observation, in children who has 1-2 patches.
- ✓ Intralesional Corticosteroids. Used in adult 1-2 patches, Act be reducing inflammation.

- ✓ **Skin Sensitizers**: used specially in children;

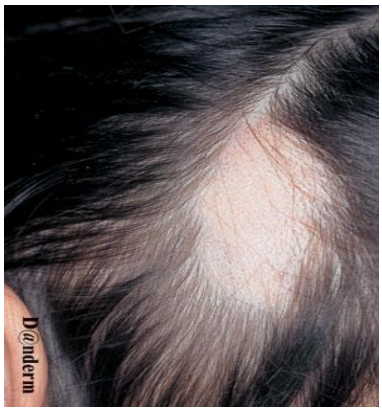
  – Anthraline. – Diphencyclopropenone (DPCP).

  Act by causing inflammation in the skin this will distract the immune system from attacking the hair follicle.

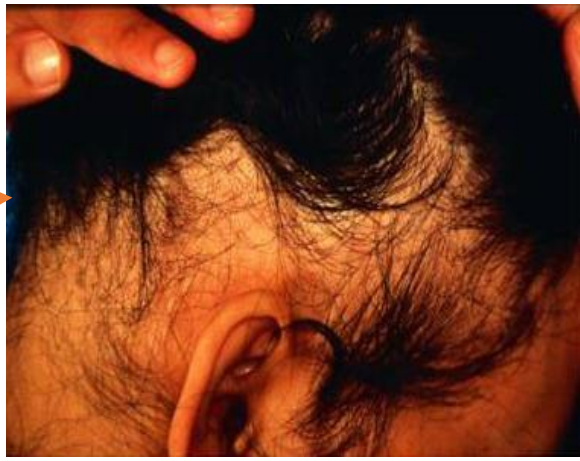
- ✓ **Others**:
  - Topical steroid (used under occlusion" plastic" with potent ointment in alopecia universalis)
  - Systemic Steroids. (In patient who has Alopecia areata and ongoing hair loss).
  - Minoxidil.
  - Cytotoxic Rx. (we can use it in late stage)
  - Phototherapy (PUVA).

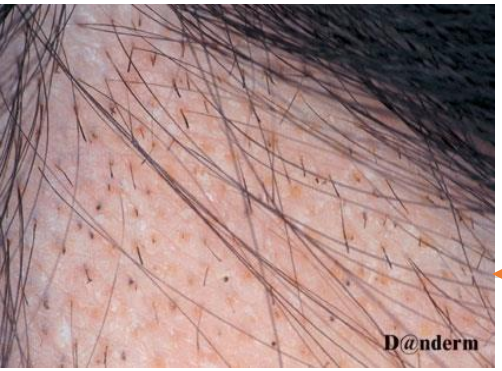
- ✓ **<u>We don't do hair transplant</u> in this case because it is an autoimmune disease.**

- ✓ **<u>We can't combine steroid and skin sensitizers</u> because they have opposite mechanism of action, so the tx will fail.**



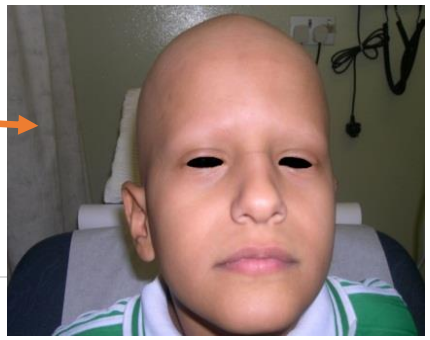
Alopecia ophiasis





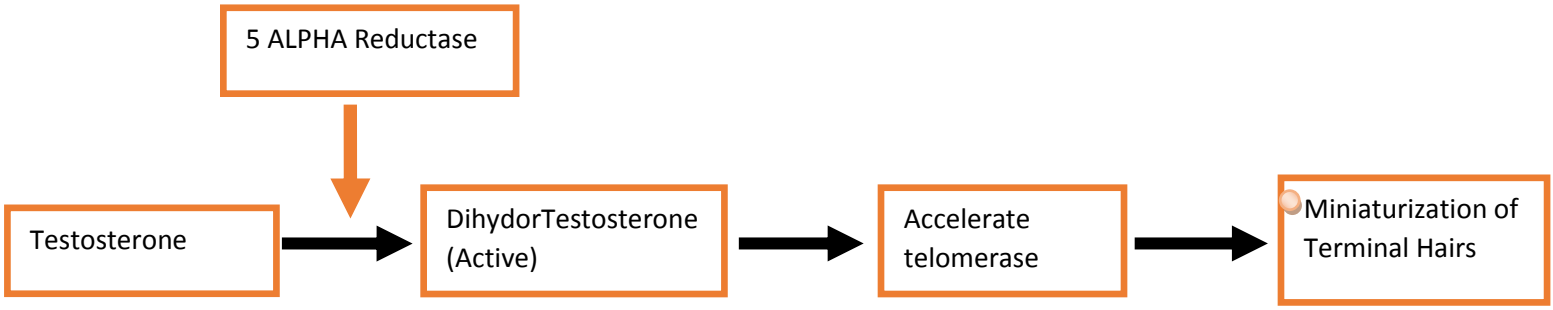
Exclamation point

Alopecia universalis

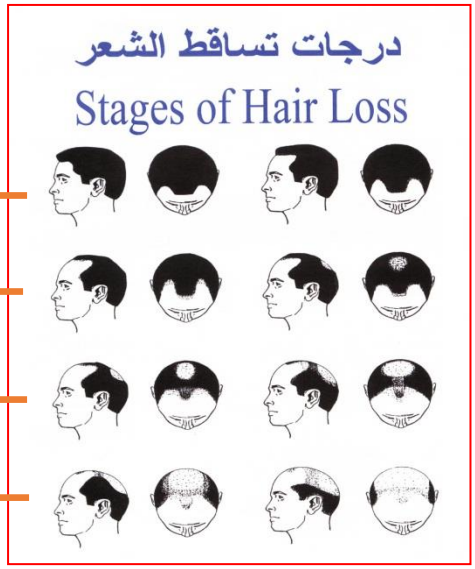


2- Androgenetic Alopecia (Male and Female Pattern Hair Loss):

- Androgen dependent loss of scalp hair, specially crown area.
- Androgenetic Alopecia affects up to 50% of males and 40% of females.
- Autosomal dominant with variable penetrance.
- 85% +ve family history.



Miniaturization: this is the last stage in which the hair will get weaker and transform into Vellous hair which is normally not present in the scalp.



Bitemporal Recession.

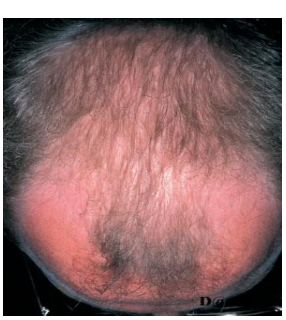
Frontal line receding.

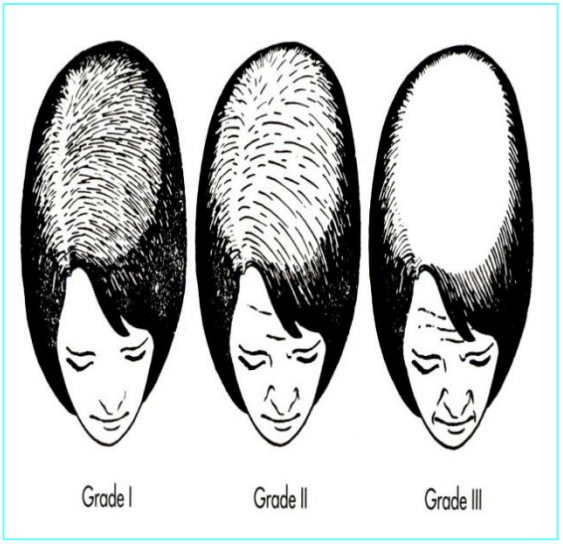
Crown shedding.

The whole crown hair lost or complete boldness.

Male Pattern Hair Loss

(Hamilton stages = 5 stages)



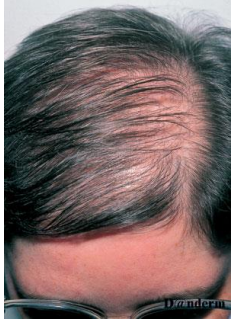


-Female doesn't usually loss her frontal hair line.

-**thinning** over **crown** in 3 stages.

Female Pattern Hair Loss

(Ludwig= 3 stages)



- ✓ **Male pattern hair loss**: It starts with thinning; it is called fronto-parietal recession and then it goes upwards. It usually spares the Temporal and occipital areas.

- ✓ **Female pattern hair loss**: There is no frontoparietal recession and no frontal recession, so the frontal hairline is preserved. There is never complete boldness, there is **thinning** only (not hair lost). It is more common in postmenopausal women

Treatment: should be used throughout life; start with topical if ineffective switch to systemic therapy.

- ✓ **Topical**:

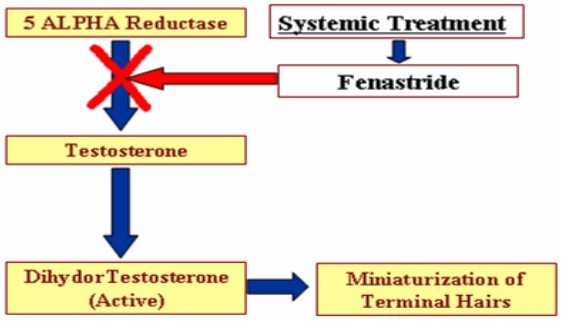
Minoxidil 2% (female) - 5%(male) solution or foam (foam is stronger and less irritant but more expensive).

Minoxidil is:

1- Anagen phase inducer.
2- Vasodilator.

- ✓ **Systemic**:

– Finastride, can't be used in pregnancy Because it can cause male genital malformation.



– Spironolactone.

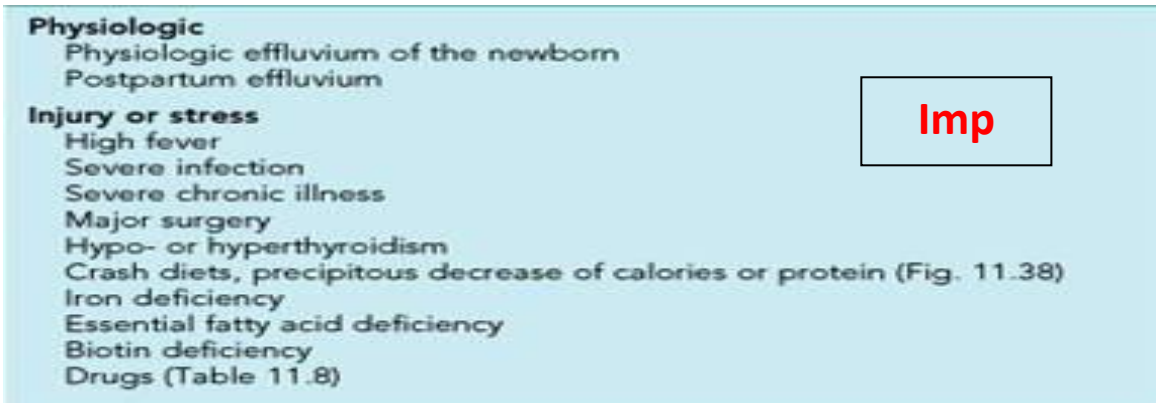
– OCP (Estrogen has antiandrogen effect, so if a female lady came with androgenic type hair loss and she was on IUD switch her to OCP "Diane or Yasmin").

- ✓ **Hair transplant:** can be done, but should continue on tx even after transplantation.

3- Telogen effluvium:

- Chronic alopecia. (fastening of the telogen phase)
- Reversible (but may be become chronic).
- **3-4 months** from trigger.

Causes:



Common scenario:
Description: When I wake up I found lot of hair in my pillow, when I take a shower my hair fall out.
People who are at risk: - A healthy person who underwent a crash diet after 4 months he developed hair loss.

- A *postpartum lady after 4 months from delivery she developed hair loss (postpartum effluvium) caused by multifactorial:
1- Sudden drop in estrogen level
2- Severe stress in delivery
3- Anesthesia in C-Section
4- Bleeding

*Pregnant lady will have a good hair because the increase in estrogen during pregnancy will retain the anagen phase.

Treatment:

- Remove or **treat the cause**.
- Minoxidil 2%-5% Solution.

4- Anagen effluvium:

- Always related to cytotoxic **chemotherapy.**
- Acute and severe alopecia (the whole body hair will be lost).
- Mostly reversible but not always.
- 2-3 week from trigger.

- Special tests should be performed in any hair loss:
  - ❖ Hair pull test:

(+) Telogen effluvium

(-) Androgenetic female pattern

https://www.youtube.com/watch?v=Y2u_ZPJ7n5w

❖ Central parting:

(+) Androgenetic female pattern

- Common hair complains:

❖ Shedding (hair fall as in alopecia Areata & telogen effluvium)

❖ Thinning (as in female pattern)

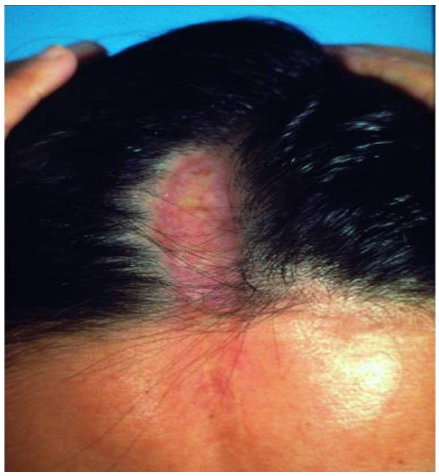
❖ Hair breakage (as in hair relaxants)

Scarring alopecia(irreversible):

- SLE—DLE.
- LP.
- Sarcoidosis.
- Leprosy.
- Kerion.
- Trauma.

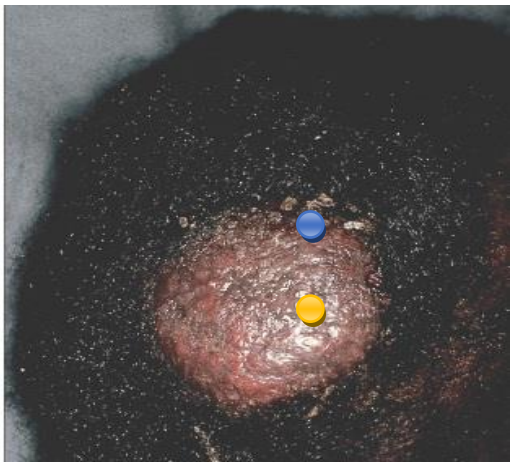
Scaring= loss the opening of hair follicle

In scaring alopecia: inflammation and scaring is present.



Localized Morphea (en coup de saber ضربة السيف)

TX: topical steroid

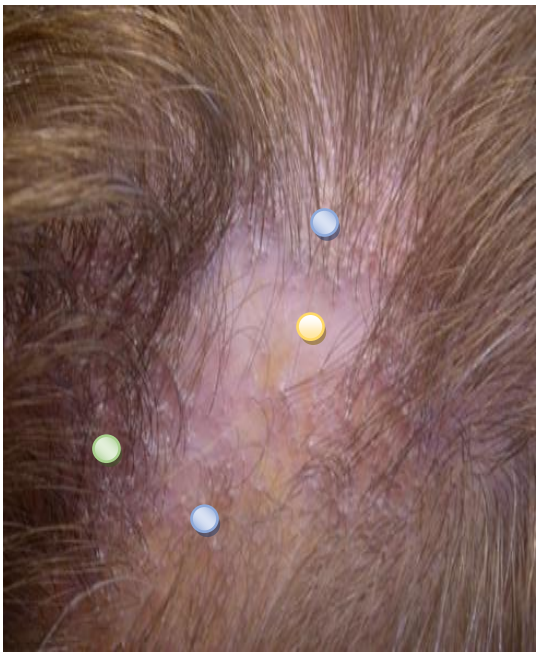


Tinea Capitis (lesion;kerion)

- inflamed and boggy scalp
- scales in the edges

Common in children who has pets as cats or can be transmitted in school.

TX: **Systemic** Antifungal

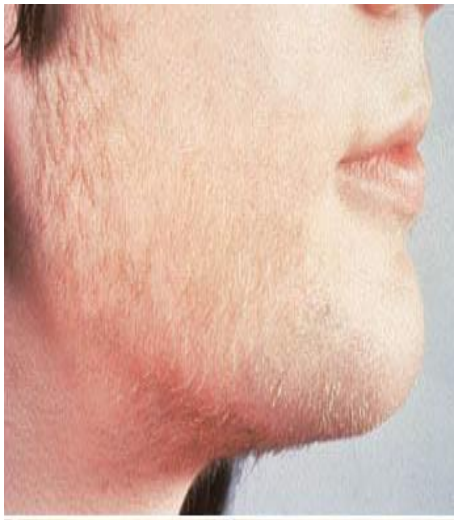
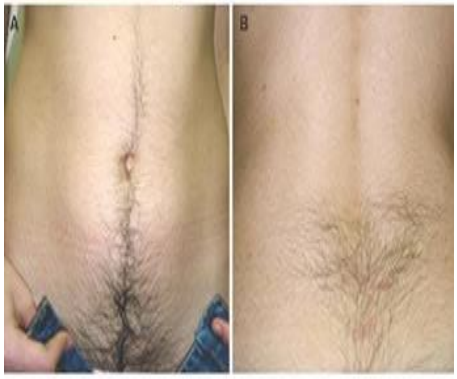
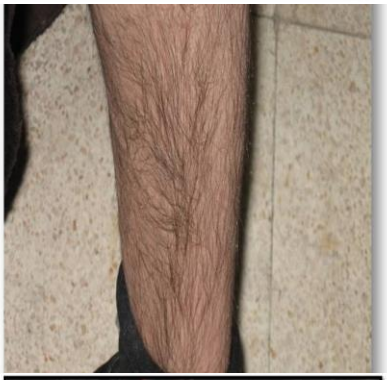
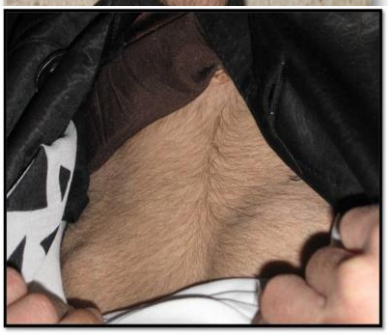


Lichen planopilaris

- Scaring
- perifollicular papule
- scales and inflammation 'redness'

TX: Intralesional steroid, topical ointment steroid and if extensive Antimalarial.

Excessive hair growth: wasn't mentioned by the female Dr.

Type	Hirsutism	Hypertrichosis
Defination	Excess growth of androgen-dependent hair in a male pattern affecting Female	Excess growth of hair in a non-androgenic pattern affecting both sex.
cause	Idiopathic (the commonest). Adrenal, pituitary. Ovarian (PCO). Turner syndrome. iatrogenic (drug).	Congenital. Acquired: drug, porphyria, endocrine: (thyroid , anorexianervosa).
Tretment	Underline cause + laser	
Pictures	 	  

2-Pigmentary disorders

Objectives:

Pathogenesis, features and management of different pigmentary disorders including:

- Freckle
- Different types of Melanocytic naevi
- Melasma
- Vitiligo

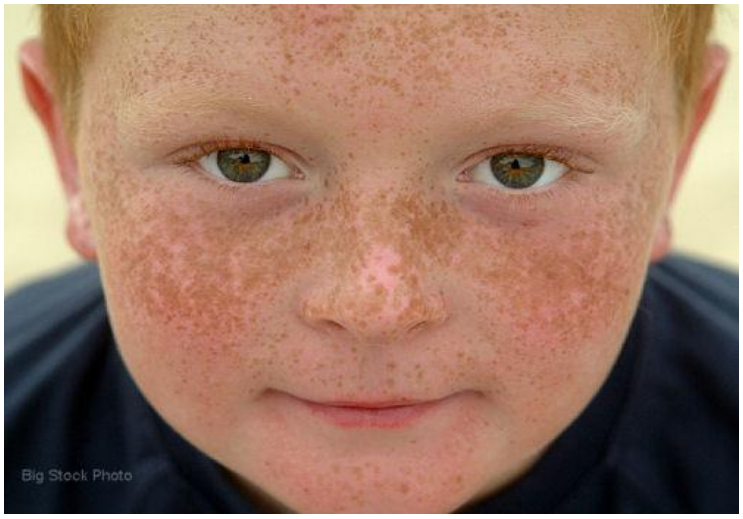
Color index:	slides,	doctor notes,	432 notes	Doctor's notes (groupF)

Freckle (Lentigo- نمش):

- Overactivity and increased no. of melanocytes.
- Fair individuals (**white people**) , common in children
- Sun exposure in genetically predisposed individuals
- Affect sun exposed area (face&forearm), so they get sun burn easily.

Treatment:

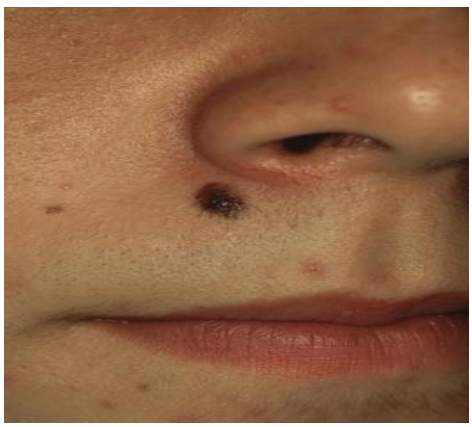
- Sun block (to prevent burns and further damage)
- Pigmented laser (recurrence might happen after sun exposure)



Melanocytic naevi (mole):

1-Acquired MN:

- very common 99%, small, uniform, **no need for treatment except** in ABCD (Change in size shape, edge, color)



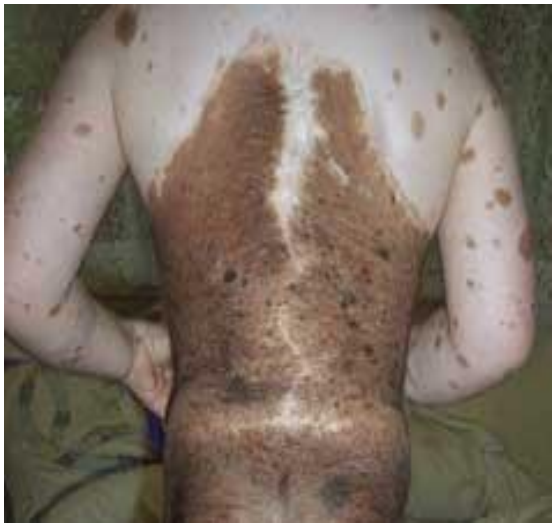
ABCD: 1-Assymetery 2-irregular Border 3-irregular Color 4-Dimeter more than 1cm or quickly enlarging 5-Bloody, ulcer or painful.

If any of ABCD present there is a chance to convert into malignant melanoma (killer)

If hair coming out from a mole don't remove it just cut it.

2-Congenital MN:

variable size could be Giant CMN (Bathing trunk) could harbor "Malignant melanoma" Higher risk of developing malignant melanoma than the Acquired MN.

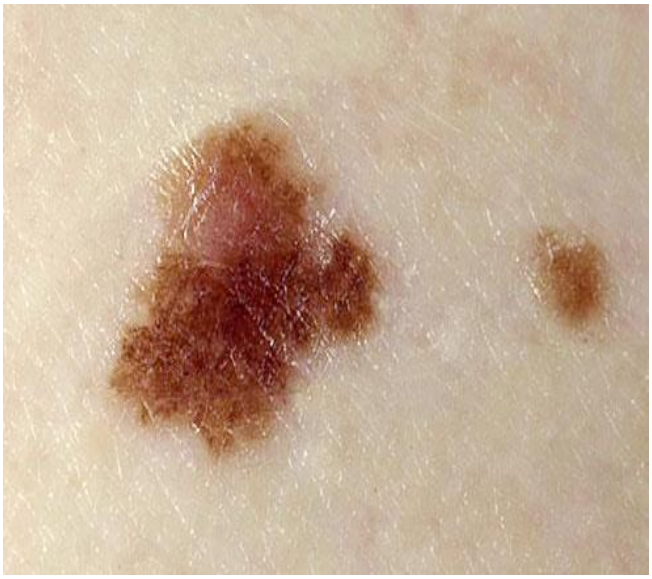


3-Atypical naevi (dysplastic, premalignant):

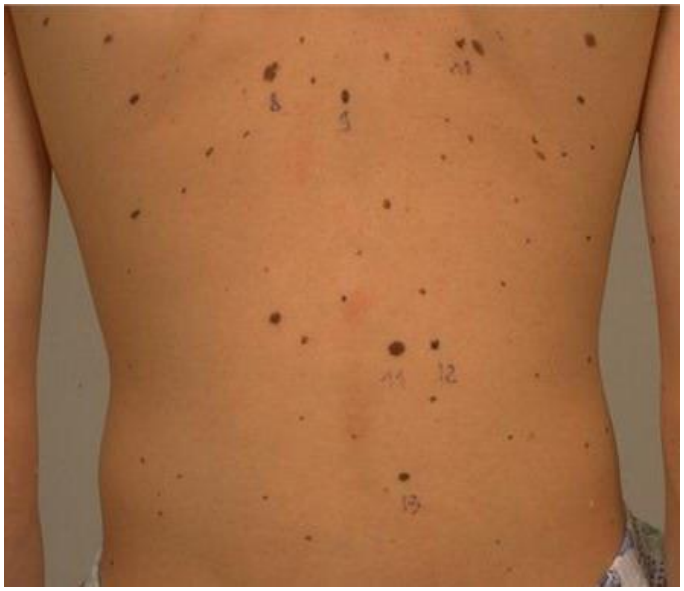
- Larger **with one or more atypical signs** (ABCD) (**4 or more: risk of malignant melanoma in the subject**).

-Should be examined every 6 months 'risk of transformation.'

- the most common type in our society is Acral.

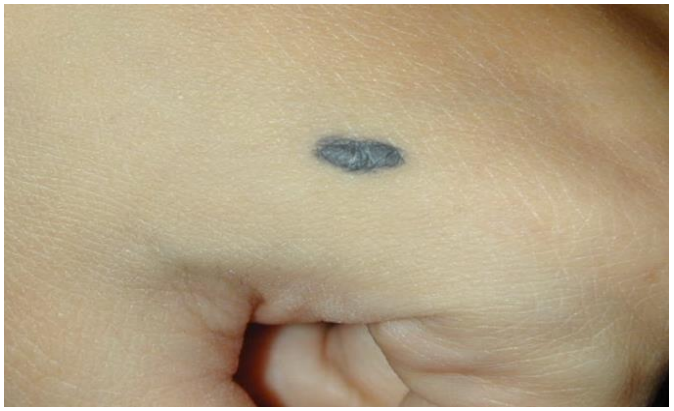


Irregular border

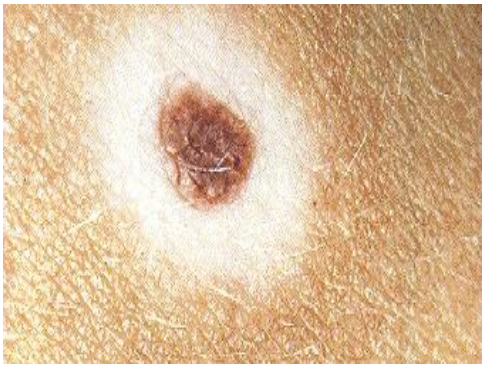


Dysplastic Nevis syndrome
-Tens of dysplastic Nevi
-have a high risk of transformation into malignant Melanoma
-should be examine every 6m or earlier.

4,5 &6 wasn't mentioned by the female Dr.

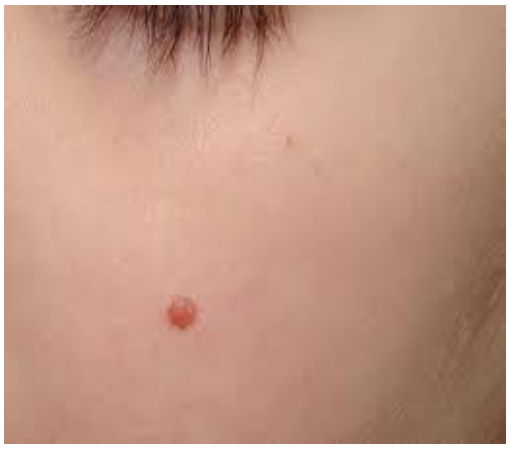


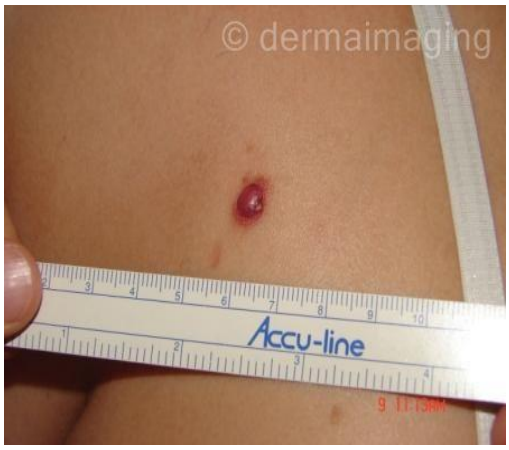
4-Blue naevi: deep-blue color and common on face, hand or feet.



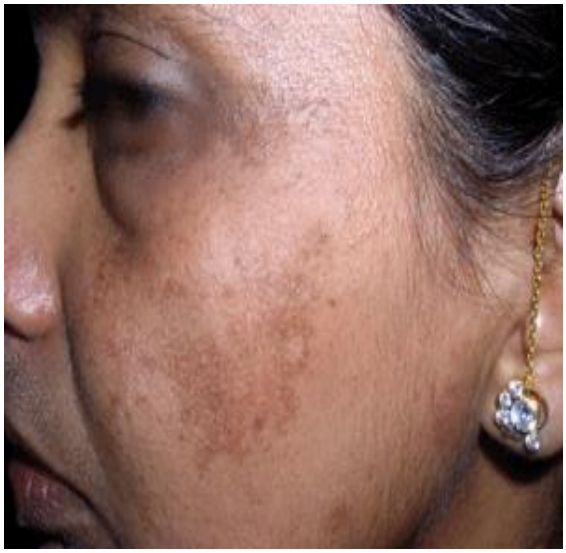
5-Halo naevi: compound naevi with halo of depigmentation.

6- Spitz naevi: common on children face with pink or pale brown color and in adult carry the risk of transformation to malignant melanoma.





Melasma (chloasma):



- Genetically programmed increase in **melanogenesis** (increase in activity not in number) caused by hormones that's why it's common in **adults**.
- Affecting the Face (an old name: mask of pregnancy)
- Could be induced by **Pregnancy**, **OCP** and excessive **Sun exposure**.

Treatment:

- sun block & bleaching cream (here the bleaching cream is more effective than in freckles).
- Change or stop OCP.

Remember:

Increase in number more than activity of melanocyte→ lentigo

Increase in activity more than number of melanocyte→ chloasma

-Hypopegmentaion: decrease in pigment

-Depigmenation: loss of pigment 'appears chalky white' this happens in vitiligo.

Vitiligo:

- **Acquired** depigmentation (loss of melanocyte)
- **Kobner phenomena**": dermatologic disease occur in the site of the trauma could be (vitiligo, psoriasis, eczema)

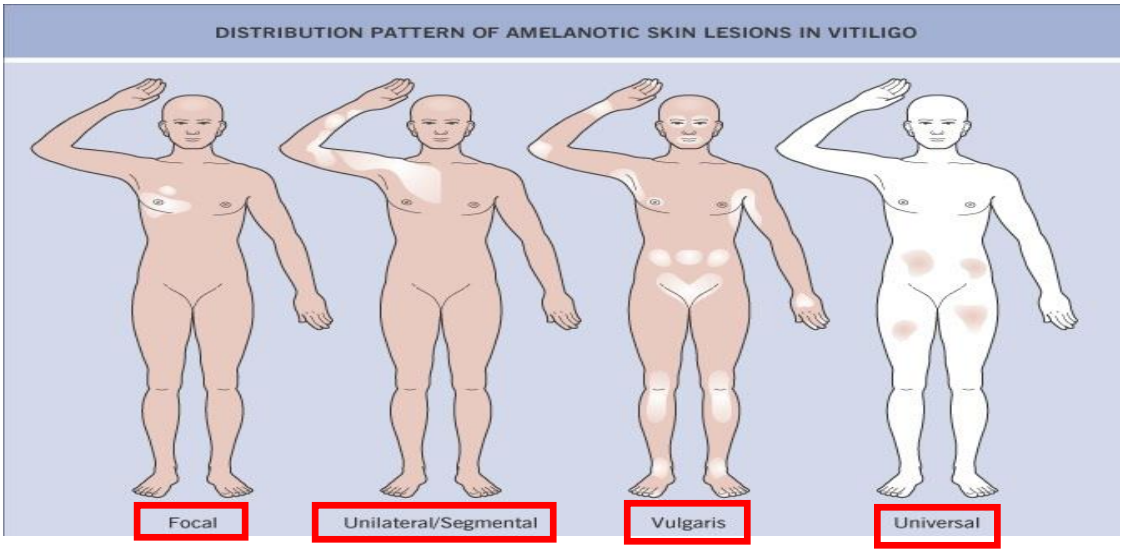
Causes:

1. Genetic 2. Autoimmune disease (**the most prominent**, so they'll be more prone to other autoimmune diseases as Hashimoto thyroiditis, DM..etc)

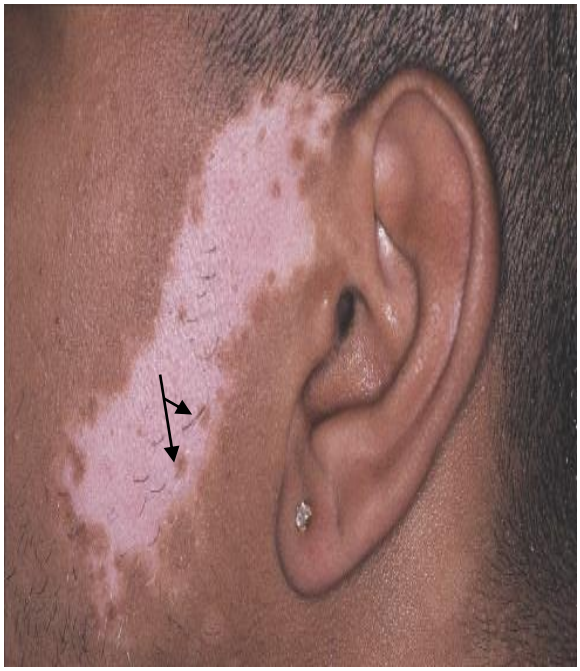
3. Neural (segmental vitiligo along the distribution of dermatome)

4. Cytotoxicity.

Natural course (imp in choosing appropriate Tx): Variable



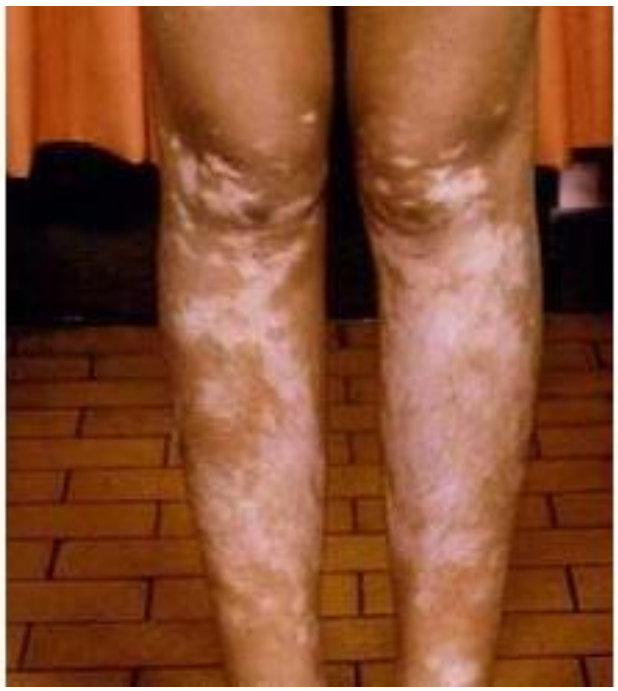
- Focal: 1-3 patches, **TX: topical tx, excimer laser, surgical if stable for 2 years.**
- Segmental: dermatomal in distribution, **TX: topical tx, excimer laser.**
- Vulgaris: most common type, happens around the eyes, around lips, fingers and in trauma sites as: knee and elbows, **TX: phototherapy; PUVA or narrow band uvb.**
- Universal: more than 90% of the body, **TX: bleaching agent to the remaining normal skin.**



Perifollicular repigmentation

-It is the most common type of repigmentation **AFTER TREATMENT.**
-Because Hair Bulb contain **melanocytes** and b.v, when it goes to the opening it will let melanocytes to go to the skin.

-(this case was focal so was treated by topical therapy).



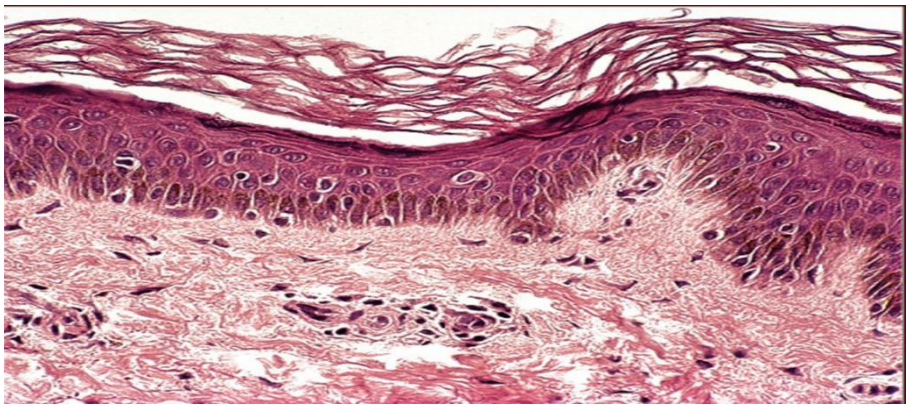
Vitiligo Vulgaris

TX: phototherapy

Diagnosis:

- Wood's lamp: - A is a diagnostic tool used in dermatology by which ultraviolet light is shone (at a wavelength of approximately 365 nanometers) onto the skin of the patient. Wood's lamps have also been used to differentiate hypopigmentation from depigmentation such as with vitiligo. A vitiligo patient's skin will appear milky white under the Wood's lamp.

- Histologically: using Dopa stain



Slide stained with Dopa Stain shows **Loss of normal melanocytes**

- Special tests:
  - T4, TSH, FBS (they'll be prone to autoimmune diseases)
  - ANA/Ro/La (prior to PUVA to rule out sun sensitivity)

Treatment (Gives good results but can't guarantee cure 'unpredictable'):

❖ Sunscreen (to prevent sunburn, koebnerization, tanning)

❖ Limited (for local lesion):

- Class 3 topical glucocorticoids.
- Topical Tacrolimus (local immune modulator)
- Excimer laser (local laser)
- Topical PUVA
- Resistant but **Stable for 2 years** (didn't develop any new lesion during 2 years even a small patch) Surgical treatment:
  - Melanocyte Transplant (only in fix inactive vitiligo because if active kobner phenomenon will be present and then induce new lesion).

❖ Generalized: Phototherapy (NBUVB, PUVA)

❖ Universalis: Bleaching agent: by Benoquin

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