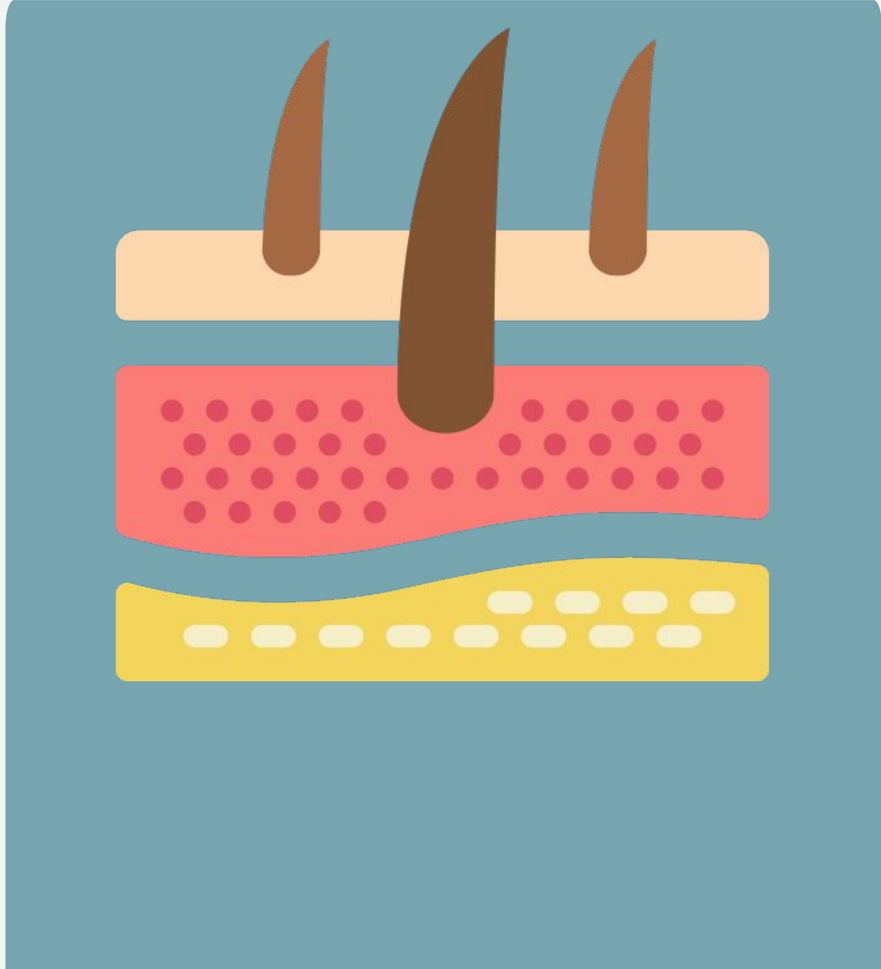


10-Pigmentary Disorders of the skin & Hair

Objectives:

- Physiology of melanocytes and skin color.
- Common cutaneous pigment disorders, pathophysiology, clinical presentation and treatment
- Physiology of hair follicle
- Common hair disorders, both acquired and congenital, their presentation, investigation and management

* Reference is the both the lecture and the TEXTBOOK*

Done by:
Saleh almoaiqe , Abdulaziz Almuhanna, Fahad Al Hussein, AlHanouf AlJaloud, Fatima Balsharaf, Shahad AlTayash.
Revised by: Sultan Al Nasser, Rotana Khateeb.

References: Doctor slides, Team 436

Color Index:

- Important
- Doctor's Notes
- Extra

Editing File

Skin Pigment:

- Reduced hemoglobin: blue
- Oxyhemoglobin: red
- Carotenoids: yellow
- Melanin: brown

Human skin color is classified according to Fitzpatrick skin phototype.

THERE ARE 6 DIFFERENT SKIN TYPES				
PHOTOTYPE	HAIR	SKIN	TENDENCY TO BURN	TANNED
I	Red Hair	Milky	Constant high	Null
II	Blonde Hair	Light	Constant medium	Mild
III	Brown	Light	Frequent	Clear
IV	Dark Brown	Matt	Infrequent	Dark
V	Very Dark Brown	Matt	Exceptional	Very dark
VI	Black	Black	No	Black

Type 1: red melanin
Other types: eumelanin (normal melanin)

This classification is important. We divide skin color to 6 phototypes.

- 1 being the fairest and 6 is the darkest.
- 1 and 2 usually fair people like Europeans.
- Most Saudis are between 3,4,5.
- **The majority is skin type 4.**
- Tendency to burn is very high in fair skin type
  - Fair skin is more prone to sunburn, freckles, wrinkles, and skin cancer.
- Tendency to burn is low in dark skin type.

Hypopigmentation:

Decrease of melanin in the epidermis:

- Melanopenic hypomelanosis:
  - A decrease of the **production** of melanin only (an example is albinism).
- Melanocytopenic hypomelanosis:
  - A decrease in the **number** or absence of melanocytes in the epidermis producing no or decreased levels of melanin (an example is vitiligo). Destruction of melanocytes

Hyperpigmentation

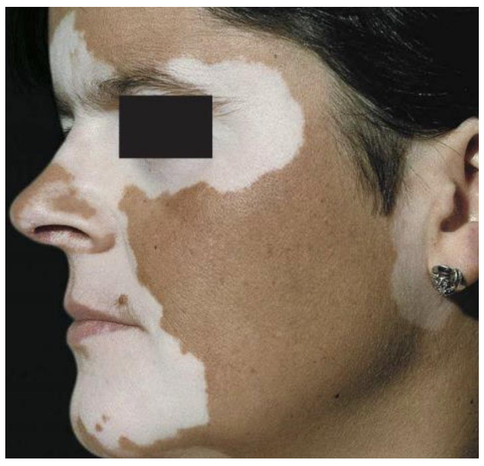
Increase of melanin in the epidermis:

- Melanocytic hypermelanosis:
  - An increase in the **number** of melanocytes in the epidermis (melanin) (an example Is lentigo).
- Melanotic hypermelanosis:
  - No increase of melanocytes but an increase in the production of melanin only (an example Is melasma).

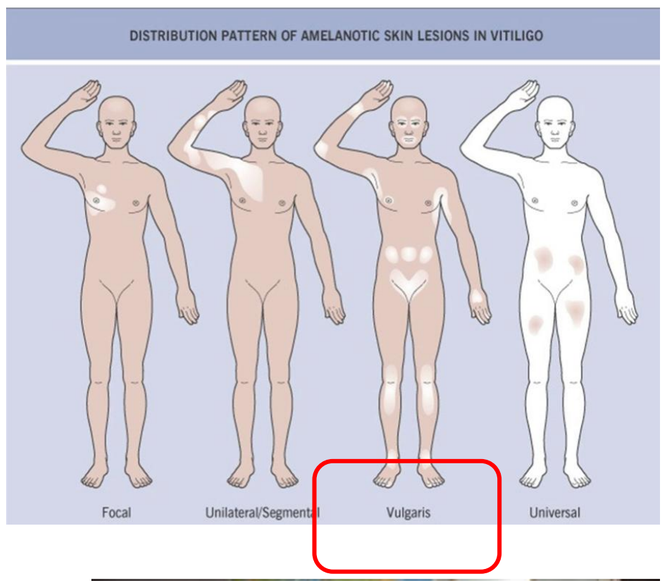
Hypopigmentation:

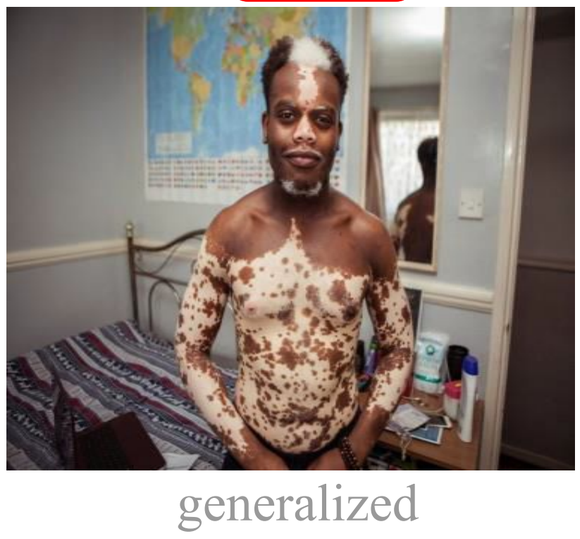
Vitiligo:

- A chronic **autoimmune disease** with genetic predisposition.
- Complete **absence** of melanocytes.
- **Incidence: 1%**
- Early onset (10-30 year of age).
- Could affect skin, hair, retina, but Iris color no change.
- Rarely could be associated with:
  - Alopecia areata
  - Thyroid disease
  - Pernicious anemia
  - Diabetes mellitus.
  - In patient with vitiligo it is important to ask about family history and to do workup to rule out other associated autoimmune diseases.
- Koebner phenomenon
  - If u scratch the skin in the active phase you will get new lesion. In trauma, surgery, inflammation
  - Diseases that have koebner phenomenon like:
    - Psoriasis, lichen planus, and vitiligo.
  - Classic vitiligo can be found on knees and elbows lip and tip of fingers as they are more prone to trauma.
- Ivory white macules and patches with sharp **convex** margins.
- Slowly progressive or present abruptly then stabilize with time.

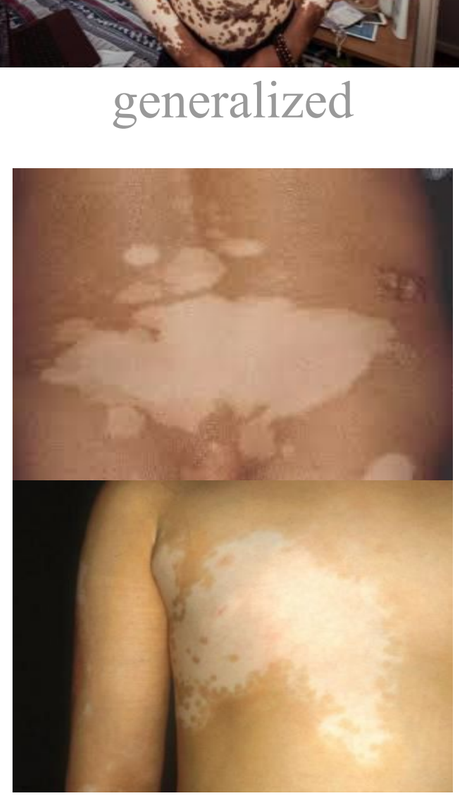


Well demarcated very white

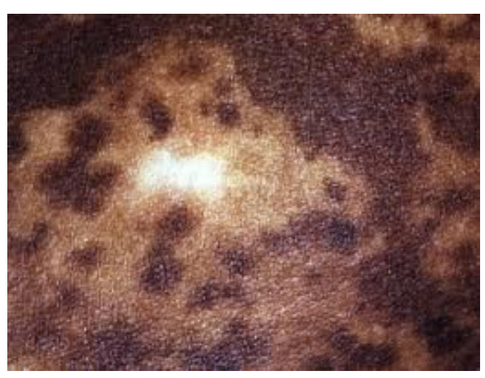




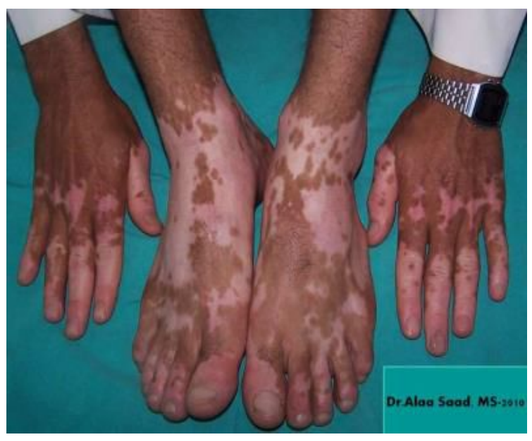
generalized



Segmental vitiligo
(like dermatomal)



Trichrome



Acral

Distribution could be:

depends on the **distribution** you will choose the **treatment**

- Focal
  - 1,2 or 3 patches.in 1 small **region**
- Segmental
  - Follows the dermatome.
- Generalized (commonest)
  - The other name is vulgaris, it is the commonest, lesions are around the eyes, lips, elbows and knees (trauma areas).
- Trichrome:
  - Light brown, hypopigmentation. depigmented area with adjacent hypopigmented area.it has 3 colors (brown , white, skin color)
- Acral
  - Depigmented patches over the **feet** and distal parts of **hands**.
- Poliosis:
  - White hair
- Universal:
  - >90% of the body is affected by vitiligo,and only 10% have normal skin type

Diagnosis:

- Usually **clinical.**
- **Wood's lamp for early vitiligo ,white person.**
- Pathology shows **normal** skin with no melanocytes.

Differential Diagnosis of Vitiligo:

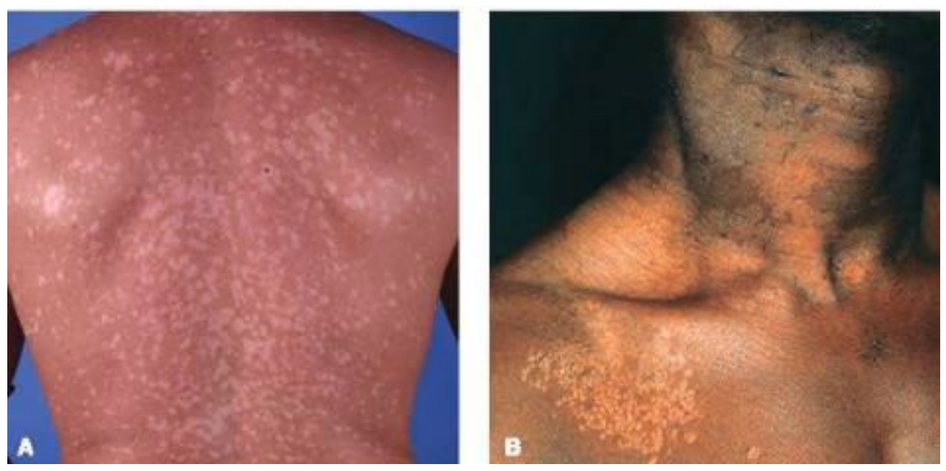
- **Pityriasis alba** Mainly in children on face
- leprosy
- Hypopigmented pityriasis Versicolor
- Discoid lupus erythematosus
- Post inflammatory hypopigmentation
- Mycosis fungoides hypopigmented
- Chemical leukoderma
- **Nevus anemicus**
- **Nevus depigmentosus**
- Hypomelanosis of Ito
- Piebaldism
- Any lesion can cause hypo or hyperpigmentation, but hyperpigmentation is more common.

Vitiligo Management:

- Sun protection:
  - Sun-avoidance, clothes, hats, sunscreens
- Make up
- Tattoo
- Psychological Support
- **Focal Vitiligo Management:** **For example 1 in 1 cm vitiligo. Non systemic**
  - Corticosteroids (topical)
  - Tacrolimus (immunomodulator)
  - 8-MOP topical phototherapy
  - Excimer laser
  - Localized NB-UVB
  - **Surgical (stable disease for 2 years): didnt get any better nor worse**
    - Melanocytes transfer, Blister graft, Punch graft
- **Generalized Vitiligo Management:**
  - NB-UVB make sure no body sensitization
  - Oral PUVA (rarely nowadays use)
  - Systemic therapy:
    - Oral corticosteroids, Methotrexate, Cyclosporine, Mycophenolate mofetil, Azathioprine
  - Depigmentation with 20% monobenzylether of hydroquinone cream
  - Depigmentation with Q-switched laser and cryotherapy if the vitiligo involve more than 90% it's easier to depigment the remaining 10%
- The most common way of repigmentation is perifollicular repigmentation (through the hair follicle). Why?
  - Because under the hair follicle there is area called **Bulge area** contains stem cells for hair and melanocytes that's why repigmentation will be through hair follicles resulting in **depigmented area** with **dots of normal skin pigment** (Reassuring sign)

Post Inflammatory Hypopigmentation:

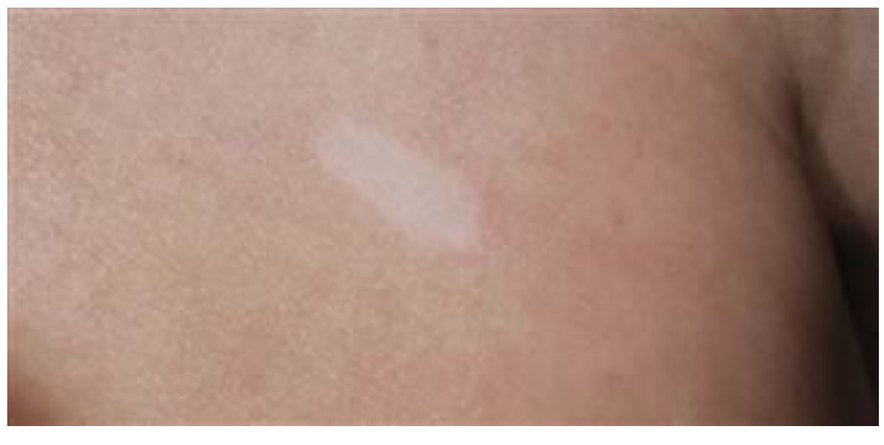
- Could happen after any inflammatory dermatosis any inflammation happened
- **Pityriasis versicolor** hypopigmentation (fungal infection)
- Post chemical peel or laser
- Post intralesional corticosteroid injection
- **Treatment:**
  - Make up, Tattoo, Excimer laser, NB-UVB



Hypopigmentation as results of inflammation

Nevus Depigmentosus

- **Congenital**, solitary depigmented **patch**.
- Cutaneous mosaicism with altered clones of melanocytes with **decreased ability to produce melanin** genetics
- Stable
- Mostly in trunk and extremities
- **Treatment:**
  - Make up, Tattoo, Melanocyte transfer no topical. Unlike vitiligo

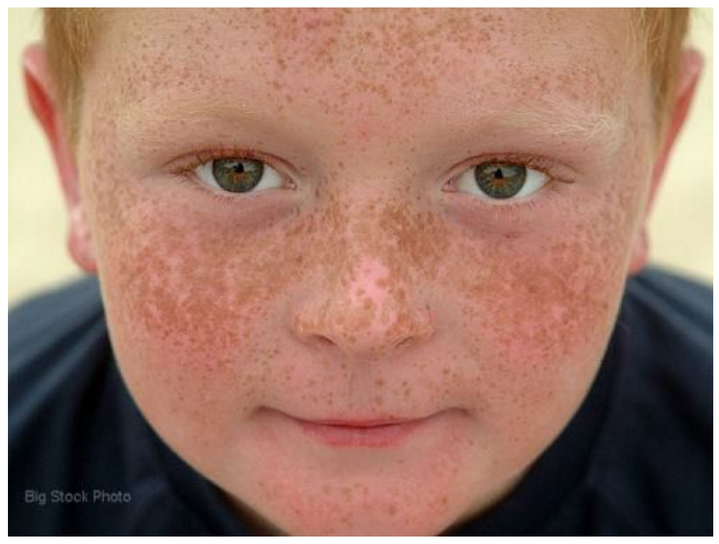


No changes with time
Light but not very white

Hyperpigmentation:

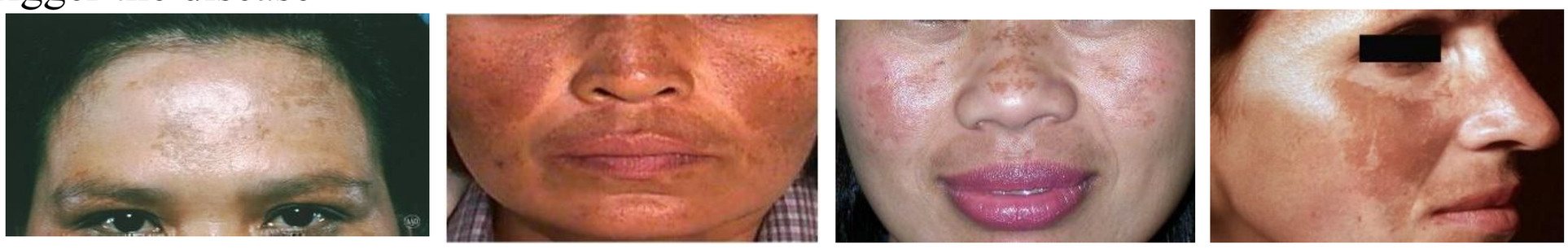
Freckle (Lentigo)

- **Overactivity** of an **increased number of melanocytes**.
- Fair individuals
- **Sun exposure** in genetically predisposed individuals
- Management:
  - Sun block, Pigmented laser (best choice), Bleaching cream we give the cream with the laser.



Melasma:

- Acquired symmetrical blotchy hyperpigmentation mostly on face
- Present as sharply marginated **macules** and patches with irregular borders on cheeks and forehead.
- Epidermal, dermal, mixed (most common)
- Mostly in young females (20-40), only 10% males
- Genetic predisposition, excessive sun exposure, pregnancy, oral contraceptives, hypothyroidism can trigger the disease



These are normal Demarcation lines (not melasma) there is problem of migration in melanocytes:

Melasma Management:

- Sun protection
- Kligman's formula: الخلطة الثلاثية drug of choice
  - Hydroquinone + Tretinoin + corticosteroid (does hypopigmentations)
- Hydroquinone 4% cream
- Glycolic acid, azelaic acid, kojic acid
- Chemical peels:
  - glycolic acid, TCA, phenol, resorcinol
- Fractional laser late choice

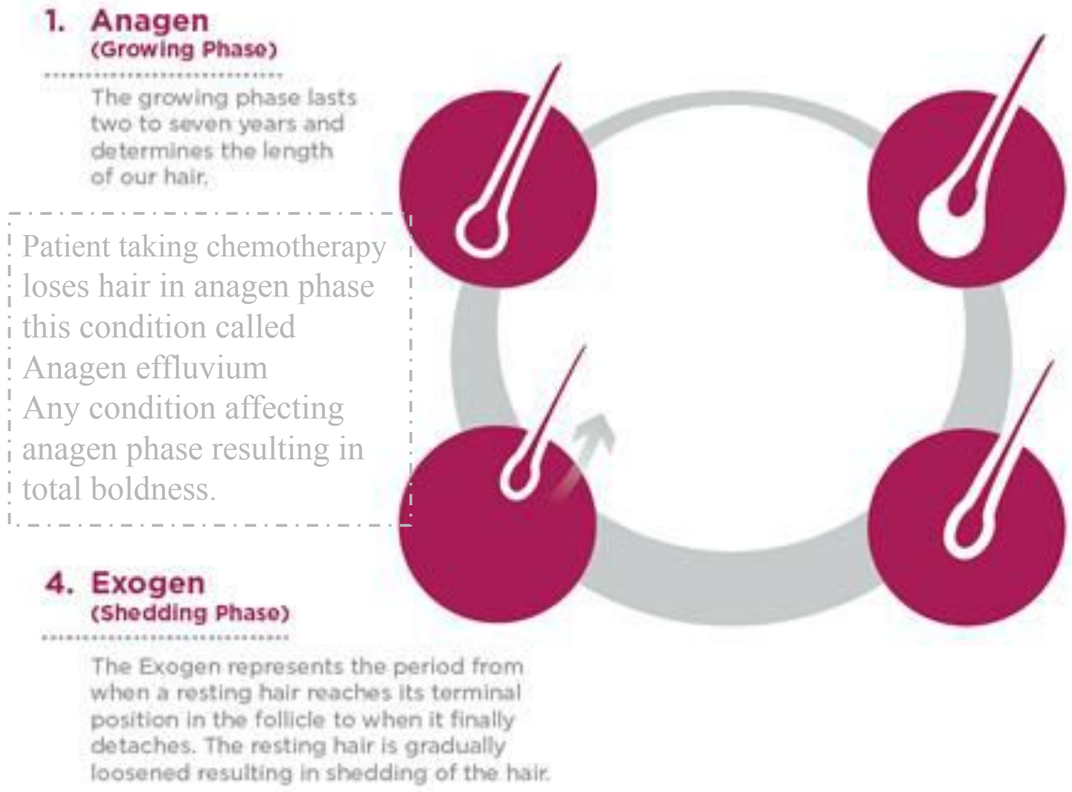
Post Inflammatory Hyperpigmentation:

- **Any inflammatory disease can cause it e.g. after burn**
- Acne, eczema, psoriasis, trauma, laser hair removal, burns, etc.
- More severe with lichen planus
- Improve with time but may persist for years
- **Treatment as melasma**

Disorders of Hair Follicle

Hair Follicle Cycle:

Anagen	Catagen	Telogen	Exogen
• Growth phase. • Hair growth rate? ○ 0.3mm/day for scalp hair • Determines the ultimate length of the hair • 80-85% of hair • 3-6 years every site in the body has its anagen duration.or 2-7 years	• Transitional phase between anagen and telogen (apoptosis driven) • 1% of hair • 1-2 weeks	• **Resting phase** • 5-15% • 3-4 months Hair falls in after shower \ brushing	• Active shedding of the hair.

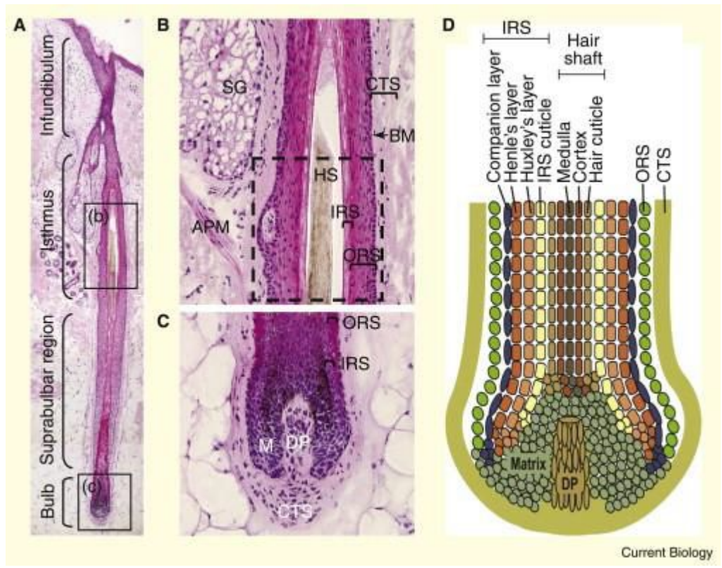


Anagen

- Duration of anagen determines the final length of the hair
- Length varies according to body site
  - Scalp hair 02-08 Y
  - Legs 05-07 M
  - Arms 02-03 M
  - Eyelashes 01-06 M
  - Fingers 01-03 M

Types of Hair:

Growing site

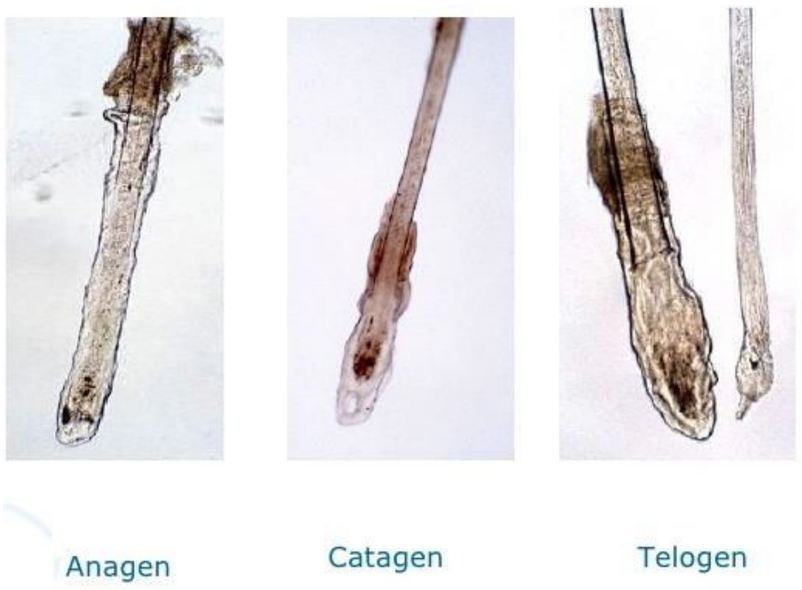


Any chemical changes like hair dying would affect the **matrix** in the hair shaft

- **Lanugo hair:**
  - Fetus, shed before birth
    - (if it did not shed before birth means there is disease.)
- **Vellus hair:**
  - Fine, non-pigmented hair
    - (fingers and arms are is **not** affected by hormones.)
- **Terminal hair:**
  - Thick pigmented hair scalp ,eyebrows/eyelashes,bear ,axilla , pupic area and its growth is **affected** by hormones.(puberty;androgen)

Diagnosis:

- **Hair pull test:**
  - + 6 is positive (there is shedding), Less than 3 is normal
- **Trichogram:**
  - 50 hair pull for anagen/telogen hair ratio 9:1
- **Trichoscopy:** dermatoscope we see hair follicle and scalp
- **Scalp biopsy** for diseases
- **Scanning Electron Microscopy**
- **Wood's Lamp: Fungal Infection**



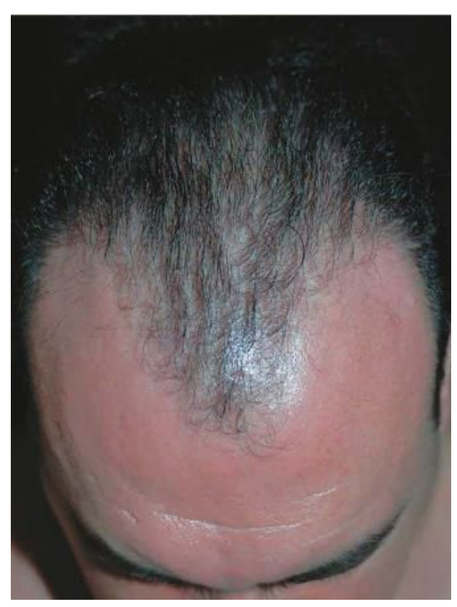
Alopecia classification:

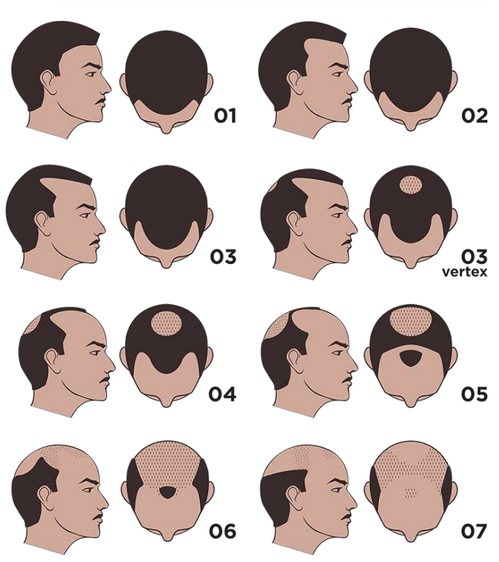
- **Noncicatricial** Non scarring**/ alopecia:**
  - No clinical sign of tissue inflammation, scarring, or atrophy of skin.
  - Male/Female Pattern hair loss, Alopecia Areata, Telogen Effluvium, Anagen Effluvium, Trichotillomania.
- **Cicatricial** Scarring **alopecia:**
  - Evidence of tissue destruction such as inflammation, atrophy, and scarring may be apparent.

Non-Scarring Alopecia

Pattern hair loss: Male Pattern Hair Loss

- Most common type in adult men
- Genetic predisposition (Autosomal dominant) and androgen hormones
- Susceptibility genes inherited from both mother and father
- Genetic sensitivity of hair follicle receptors to Dihydrotestosterone (DHT)
- DHT decrease **anagen** phase from years to months or weeks
- DHT is regulated by 5 alpha reductase
  - **Testosterone → (5alpha reductase I&II)→ DHT**
- Type II alpha reductase:
  - Scalp & beard hair, seminal vesicle, prostate, epididymis, scrotum
- Present as **receding hairline** and hair loss on **frontal area and vertex. With time will be completely bold.**





Hamilton classification helps in diagnosis and treatment progression Follow up and diagnosis

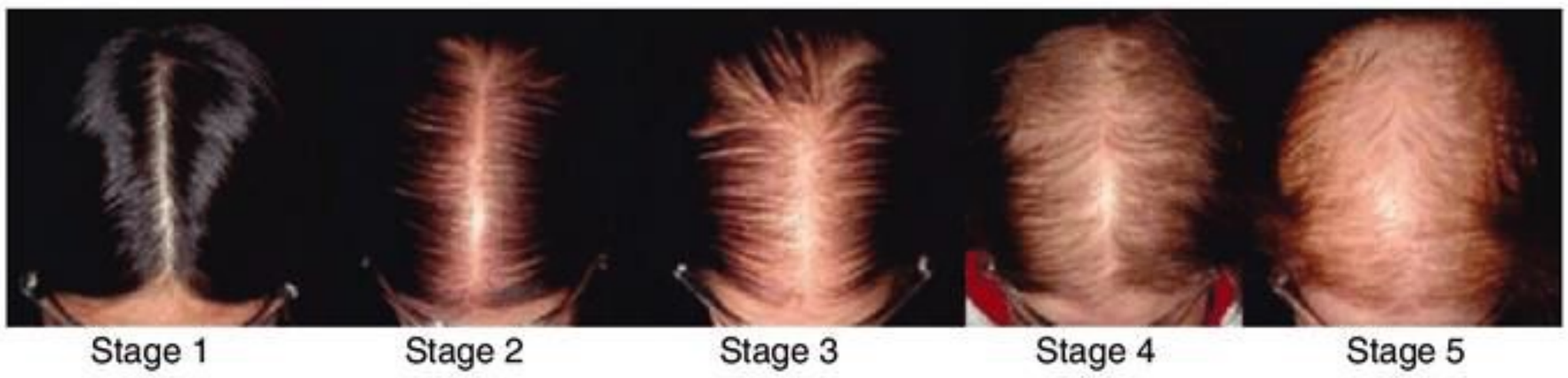
Non-Scarring Alopecia

Pattern hair loss: Male Pattern Hair Loss

- **Management**
  - Minoxidil solution 2% and 5%
  - **Finasteride** 1mg/d (**type II 5 alpha reductase inhibitor**)
  - Dutasteride 2.5mg/d (type I & II)
  - Hair transplant not better by systemic, but after it we still give systemic/topical
  - Hair piece, Tattoo, powder

Pattern hair loss: Female Pattern Hair Loss:

- 40% of women ages 50
- Diffuse thinning of hair begin at the vertix mainly over the crown but NEVER bold
- Usually with preservation of the frontal hair
- Genetic predisposition, polygenic, either parent
- Normal androgen level
- More common after menopause
- **Polycystic Ovarian Syndrome** (PCOS) , Congenital Adrenal Hyperplasia (CAH)



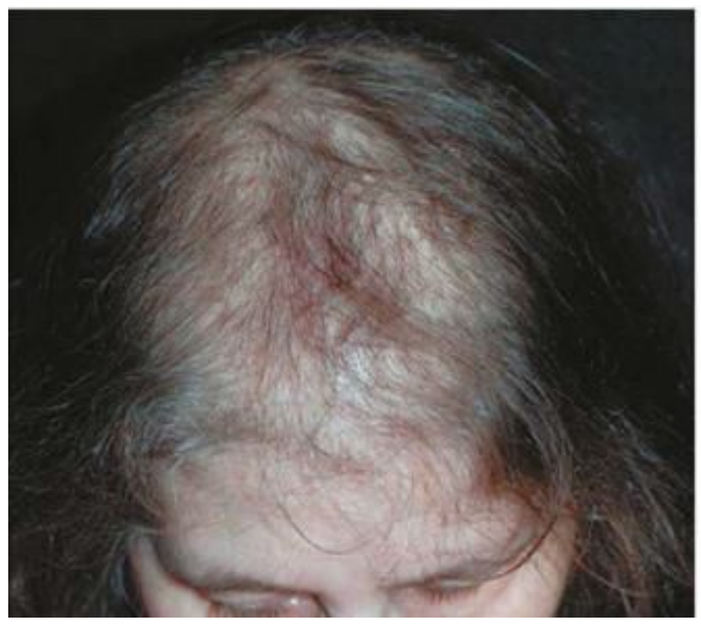
Ludwig staging: widening of frontal part (parting) الفرقة.
كل ما صارت أوسع و شعرها خفيف كل ما كان advanced

Investigation:

- Trichogram if v. early
- Very important we ask about other signs of hormonal abnormalities; Period, acne, hairsutism.
- DHEAS most imp
- Prolactin
- Free testosterone most imp
- LH/FSH

Generally:

- CBC, Ferritin
- Thyroid function test
- Scalp biopsy rarely



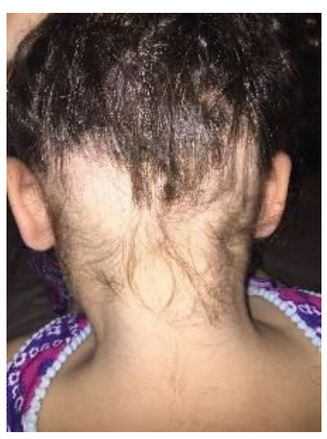
Management:

- Minoxidil 2%, 5%
  - May cause hypertrichosis on face and neck
- Finasteride
- Dutasteride
- Spironolactone
- Flutamide
- Cyproterone acetate
- Hair spray, powder, hair piece
- Hair transplant Male or female pattern hair loss

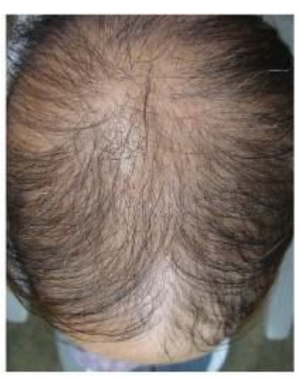
Non-Scarring Alopecia

Alopecia Areata

- Autoimmune with T- cells around hair follicles
- 50% in childhood, 80% before age of 40
- **Genetic predisposition:**
  - 10-20% positive family history
- **Association with:**
  - Vitiligo, thyroid disease, atopic dermatitis and Down syndrome
- **Triggers:**
  - Viral infection, trauma, hormonal changes, severe emotional stress
- Very well defined **Patchy hair total loss** of scalp, beard, eyebrow, eyelash hair, body.
- **Sudden onset**
- Regrowth of white hair then pigment comes back and sometimes not.
- **Exclamation marks** are 2-3 mm **broken hair** with distal end broader than proximal ! at the margin of the hairless patch
- **Nail pitting** and **ridging** in 10-50% of patients
- **Types of Alopecia Areata**
  - Localized AA (The most common)
  - Alopecia totalis: all scalp **hair**
  - Alopecia universalis: whole **body** hands and legs
  - Ophiasis: occipital and lateral scalp bad prognosis
  - Diffuse alopecia areata

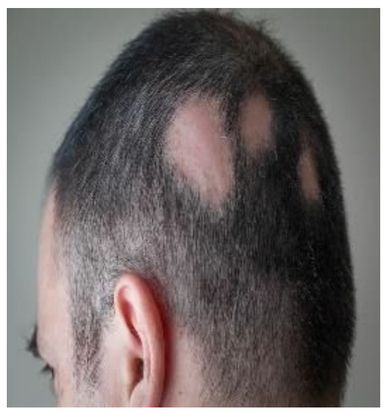


Ophiasis



diffuse

Exclamation marks broken hair
diagnostic feature very unique shape
Pathognomonic



Patchy
2 or 3 spots

Alopecia universalis
كل الشعر و الوجه و الحواجب

Management:

- **Intralesional** corticosteroids (topical) patchy, young.
- Minoxidil solution adjuvant therapy
- Dithranol
- Diphencyprone (DPCP)
- Phototherapy totalis, universalis, extensive
- Systemic corticosteroids, pulse therapy
- Methotrexate, azathioprine
- JAK inhibitors (Tofacetinib, Ruxolitinib)
- Artificial eyelashes, eyebrow tattoo, hair piece

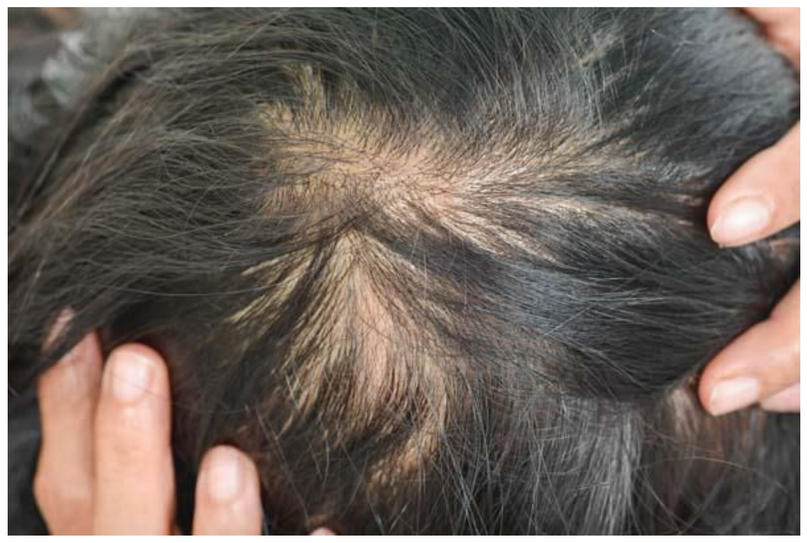
Prognosis:

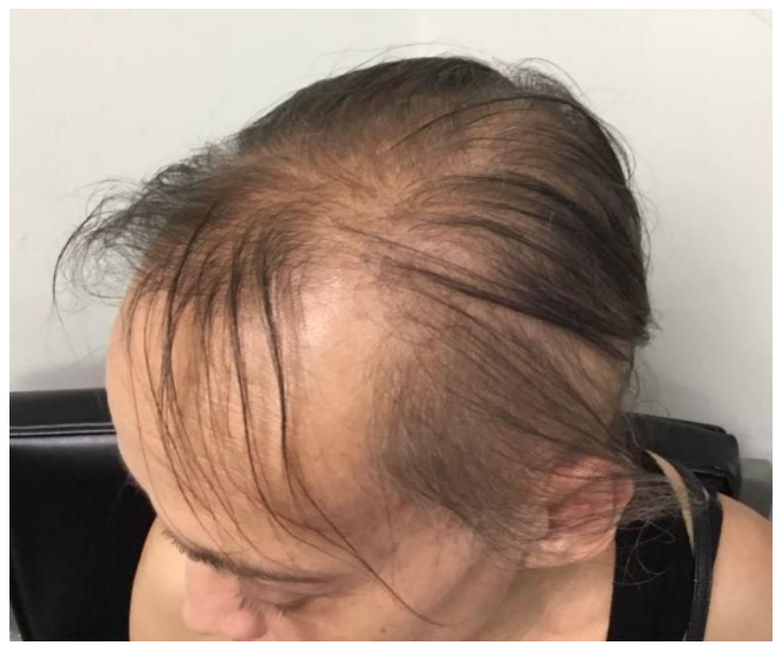
- **Single patch:**
  - 80% resolution in 1 year spontaneously
- **Poor prognostic factors:** can not get better or does but relapse
  - Extensive disease
  - Duration >1 year
  - Ophiasis pattern
  - Nail involvement
  - Childhood onset
  - Positive family history
  - Other concomitant autoimmune diseases
  - Atopy
  - Down syndrome

Non-Scarring Alopecia

Telogen Effluvium:

- Temporary hair loss of telogen hair
- **System shock:** change anagen hair to telogen
- Diffuse hair fall but in pattern hair loss it is more on the crown.
- Might take 2 months after shock to start losing hair
- Usually last for 6-9 months with incomplete recovery
- Could be chronic, but doesn't cause complete baldness



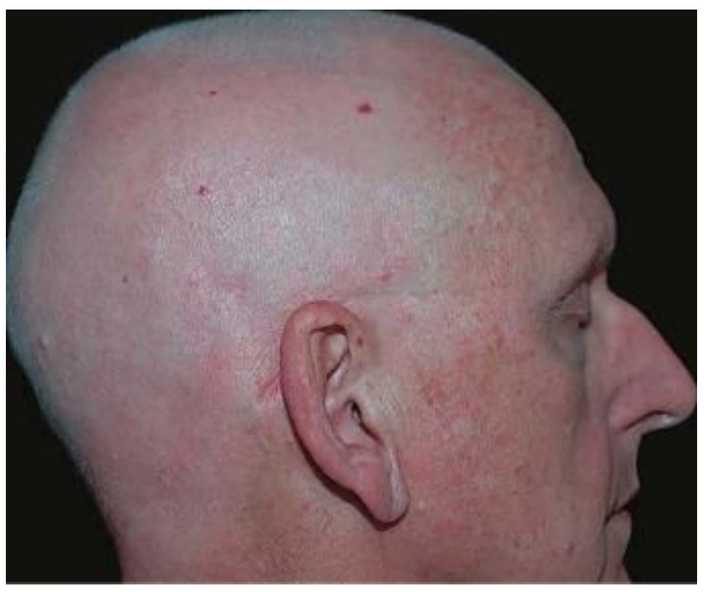


Diffuse thinning (not localised on crown as if female pattern hair loss). Thinning , + hair pull

- **Causes**
  - Mostly postpartum
  - Fever, surgery with general anesthesia, childbirth, severe emotional trauma, severe weight loss. Crash diets
  - **Drugs:**
    - Heparin, warfarin, B-blockers, Ace-inhibitors, lithium, anticonvulsants (especially valproic acid), isotretinoin.
  - **Nutrition:** Vitamins deficiencies
  - **Anemia:** Iron deficiency or precious
  - Thyroid disease
  - Ferritin store deficiency
- **Diagnosis**
  - HAIR PULL: +ve. Female pattern hair loss is -
  - CBC, Ferritin.
  - TSH Rule out thyroid disease.
  - Vitamin D level
  - Histology: **swarm of bees**
- **Treatment**
  - Treat the cause
  - Minoxidil 2% adjuvant. If alone it will not help.
  - Takes a while. If iron deficiency we wait for the iron to go back to normal then after 1 or 2 months till the hair fall stops.

Anagen Effluvium:

- **Etiology:**
- Radiation therapy to head; **chemotherapy** with alkylating agents; Intoxications. It kills the cells.
- Onset is usually rapid and extensive within 2 weeks to a month
- Pathogenesis: Occurs after any insult to the hair follicle that impairs its mitotic/metabolic activity. Falls in its abnormal phase
- Regrowth is usually rapid after discontinuation of chemotherapy.



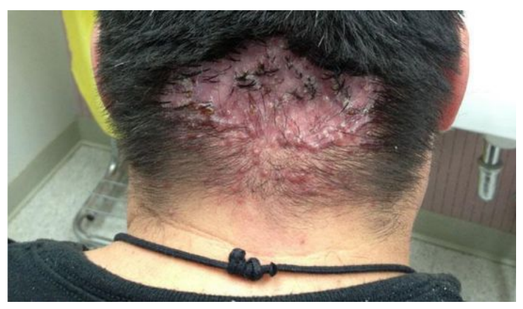
Anogen in our hair is 80%- 85% so the person can go bold. Unlike talogen 15% so doesn't matter how much hair loss he/she will never go bald because they will not lose more than 10% of their hair,

Trichotillomania:

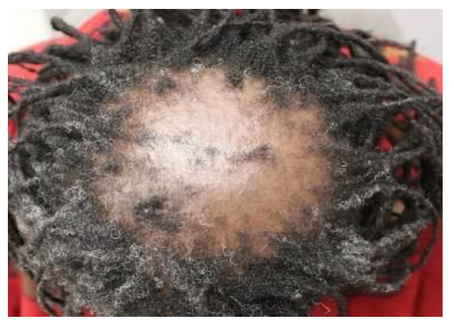
- Self-induced plucking or breakage of hair
- Often associated with psychological stress or a personality disorder
- Incomplete and distorted follicular anatomy is a histologic hallmark
- Key to diagnose: **variable size hair**
- Tx: behavioral therapy, hair hat or band.

Scarring Alopecia loss of hair follicle opening

- Lichen planopilaris LPP
- Frontal fibrosing alopecia: post menopausal women
- Central centrifugal cicatricial alopecia
- Discoid lupus erythematosus of scalp
- Traction alopecia when u pull or comb hair aggressively. Interlocking n the african americans
- Trichotillomania it starts as non scarring but can progress to scarring
- Acne keloidalis nuchae
- Kerion (tinea capitis) Scalp infection; boggy inflamed

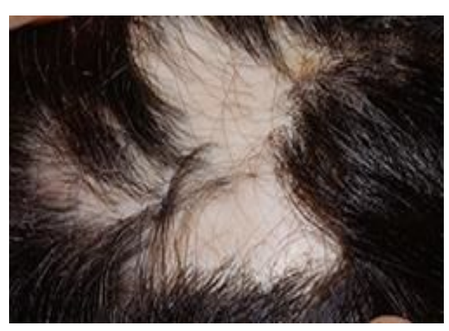


acne keloidalis nuchae



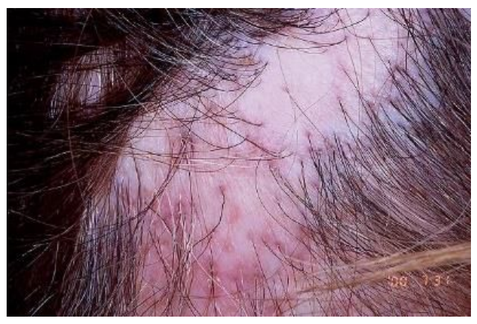
central centrifugal cicatricial with itching

Frontal fibrosing alopecia

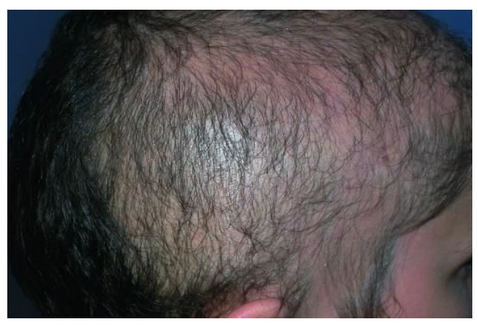


Pseudopelade

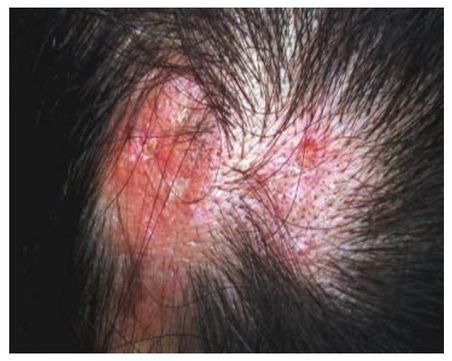
traction alopecia



lichen Planopilaris, triangular, perifollicular edema



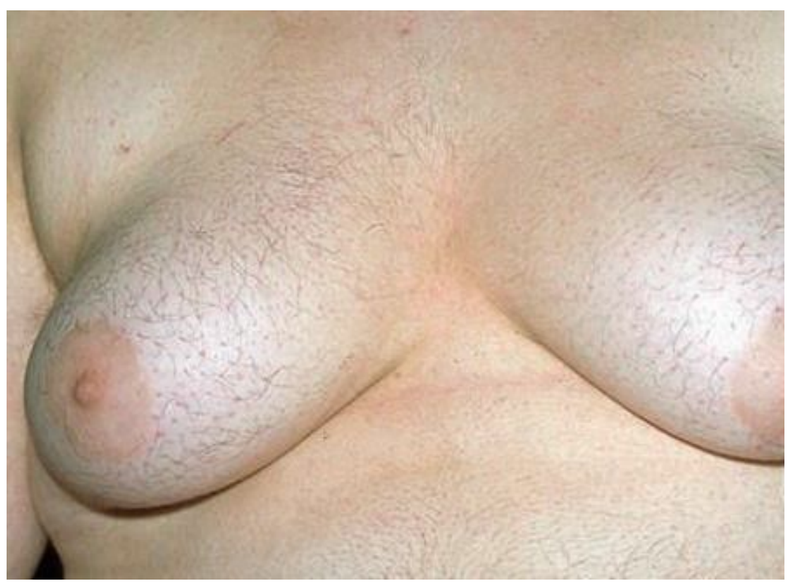
trichotillomania

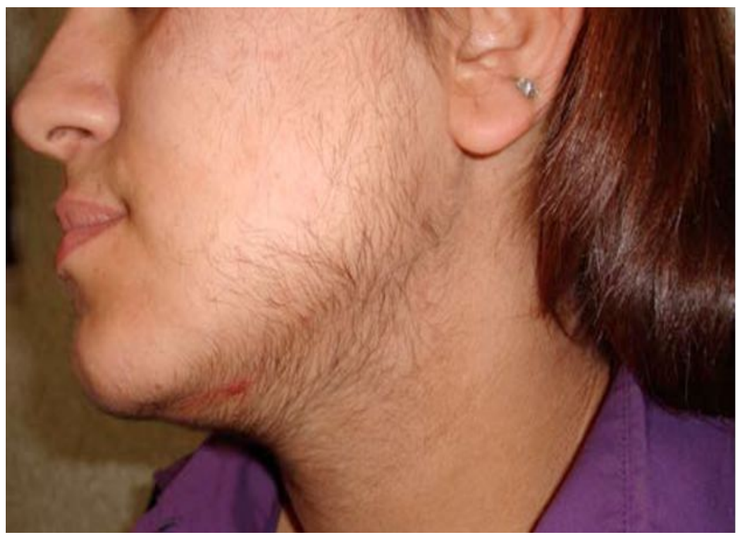


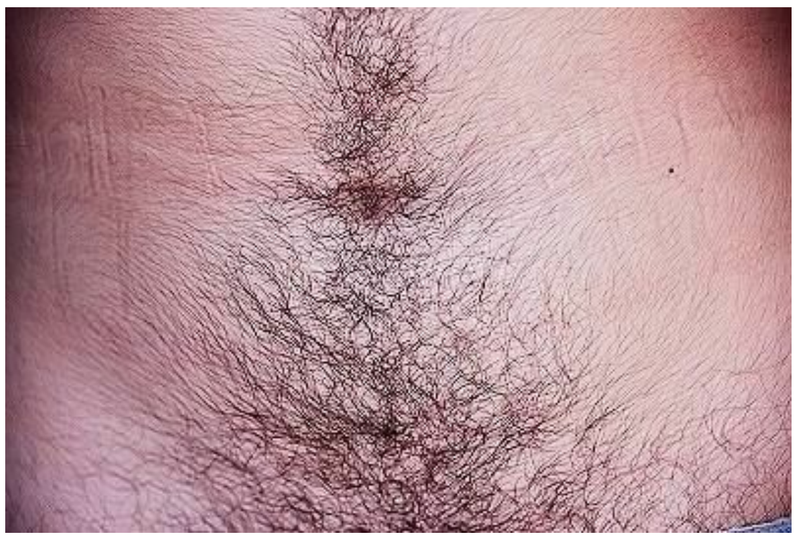
Discoid lupus erythematosus

Hirsutism

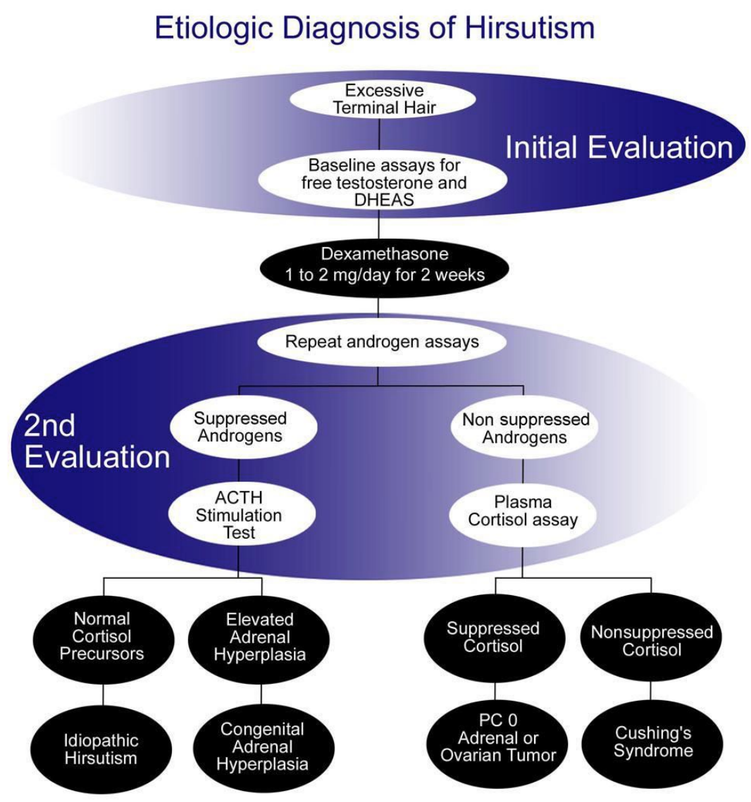
- Male pattern hair growth in women
  - Facial hair, below umbilicus, around nipple, upper back, inner thighs.
- **Associated findings:**
  - Acanthosis nigricans, galactorrhea, striae, acne, virilization (increase muscle bulk & size of clitoris, deep voice, baldness, menstrual irregularities , infertility
- **Causes:**
  - Genetically determined, 10% of women affected rule out others to say this.
  - PCOS
  - Stopping oral contraceptives
  - Cushing syndrome
  - CAH (21 hydroxylase deficiency)
  - Tumors of adrenal glands (almost always malignant)
  - Ovarian tumors (arrhenoblastoma)
  - Pituitary gland (ACTH dependent)







Investigation:



- **Initial screening:** 1st thing; most imp
  - Free testosterone
  - Dehydroepiandrosterone sulfate (DHEAS)
  - **Adrenal** (↑testo+↑DHEAS)
  - **Ovary** (↑testo + normal DHEAS)
- **Thyroid function**
- **Prolactin**
- **LH/FSH ratio:** PCOS
- **17-hydroxyprogesterone:** CAH
- Urinary and serum cortisol
- Dexamethasone suppression test: to **exclude ACTH dependent hirsutism**
- Prostate specific antigen(PSA): a marker for increase androgens
- U/S pelvis, CT-Scan, MRI

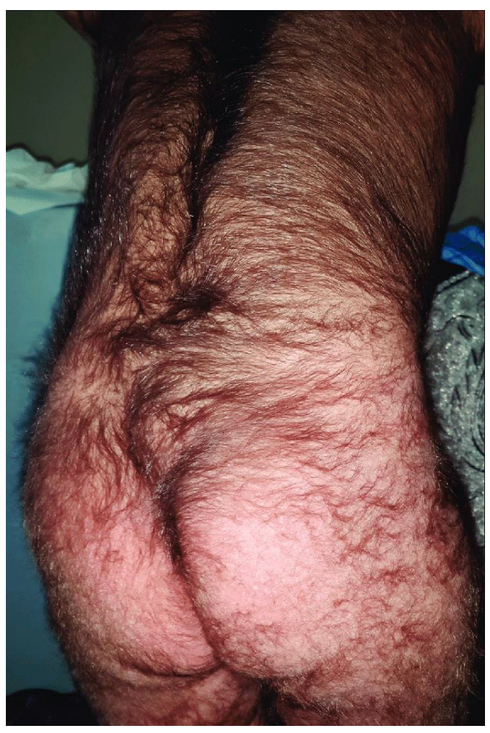
1st thing; TST, DHEAS.
+/- TSH , prolactin , LH//FSH Ratio

- **Treatment of Hirsutism** Based on cause

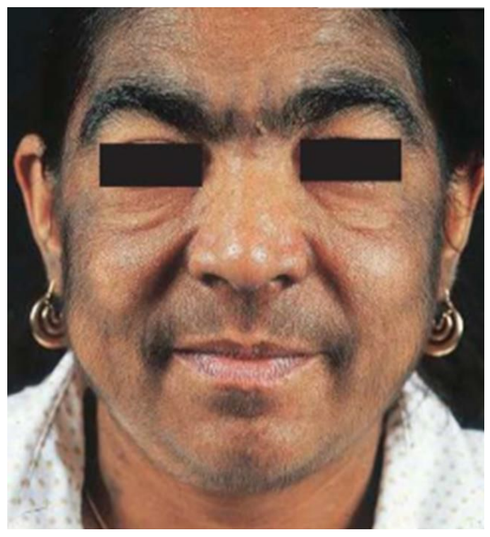
Medications	Cosmetic treatments
Birth control pills	Shaving
Androgen receptor blockers	Eflornithine cream
Spironolactone	Waxing
Flutamide	Bleaching
Glucocorticosteroids	Plucking
Dexamethasone	Depilatory agents
Prednisone	Electrolysis
Methylprednisolone	Laser
Enzyme inhibitors	
Finasteride	
GnRH analogs	

The cosmetics treatment doesnt stop the growth it just decreases its frequency. We have to treat th cause to stop the hairsutism

Hypertrichosis



- **Werewolf syndrome** Affects males and females
- Excessive growth of lanugo, vellus, or terminal hair excess hair in general no specific pattern unlike hirsutism (male pattern in female)
- Congenital or acquired
- <u>Generalized hypertrichosis:</u>
  - **Porphyria cutanea tarda**, Malnutrition, Malignancy, Hypothyroidism, Drugs (Minoxidil, cyclosporine, phenytoin, androgenic steroids, Oral corticosteroids).
- <u>Localized</u>:
  - Chronic itching and rubbing, topical minoxidil (instruct to not drop it on the face) topical corticosteroids, PUVA
- <u>Hypertrichosis associated with nevi:</u>
  - Congenital melanocytic nevus, Becker's nevus, Vascular malformation
- <u>Trichomegaly</u> (Bimatoprost medication) الشعرة نفسها تضخم



Questions:

1) Which of the following causes Anagen effluvium?
   A) Colchicine
   B) Lithium
   C) Warfarin
   D) Heparin

2) Patient have sudden hair loss after 4 months of bariatric surgery. What's your diagnosis?
   A) Telogen effluvium
   B) Anagen effluvium
   C) Female pattern hair loss
   D) Alopecia acreta

3) 25 year old female presented to the dermatology clinic with hair loss after an unbalanced diet. There was hair loss and hair thinning. What is the most common cause?
   A) Telogen effluvium
   B) Anagen effluvium
   C) Alopecia areata
   D) Drug induced hair loss

4) A 25-year old male presented to the dermatology clinic complaining of hair loss. On examination, there were 2 well-defined hairless non-scarring smooth patches over the occipital area of his scalp. What is the most likely diagnosis?
   A) Alopecia Areata.
   B) Anagen Effluvium
   C) Telogen Effluvium.
   D) Androgenetic Alopecia.

5) Club hair is a feature of which one of the following?
   A) Anagen
   B) Catagen
   C) Telogen
   D) Vellus

6) What's the best initial therapy for a localized vitiligo?
   A) Phototherapy
   B) Topical steroids
   C) Infliximab
   D) Methotrexate

7) Alopecia Areata is related with?
   A) Vitiligo
   B) Melasma

8) What is the connection between vitiligo and psoriasis?
   A) Autoimmune
   B) Joe nerd phenomenon

9) Which of the following is a promising treatment of vitiligo in the future?
   A) JAK inhibitors
   B) Cyclosporine
   C) Azathioprine
   D) Methotrexate

10) A 45 year old female presented with decreased hair density over the vertex in non-scarring alopecia pattern- The anterior hairline was preserved- Which one of the following is the most likely diagnosis ?
   A) Discoid lupus erythematosus
   B) Lichen Planus
   C) Traumatic alopecia
   D) Female pattern hair loss

1 A
2 A
3 A
4 A
5 C
6 B
7 A
8 A
9 A
10 D