



## **Hair And Pigmentary Disorders**

**Objectives : not given.**

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[ Color index : **Important** | **Notes** | Extra ]

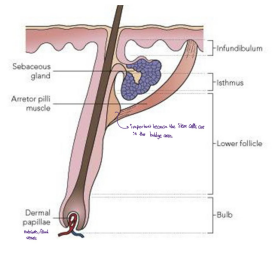
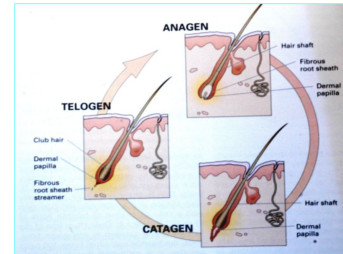
# Hair Disorders

◀ **Hair cycle:** Hair are in asynchronous continuous cycle:

Anagen phase	Telogen phase	Catagen phase
<ul style="list-style-type: none"> <li>- Growth phase.</li> <li>- (3- 6 years)</li> <li>- about 85 % of scalp hair are anagens.</li> </ul>	<ul style="list-style-type: none"> <li>- shedding</li> <li>- (3-6 months )</li> <li>- 10 % of scalp hair are telogen</li> </ul>	<ul style="list-style-type: none"> <li>- Transitional.</li> <li>- (3-6 weeks )</li> <li>- 5% of scalp hair are catagen</li> </ul>

◀ **Facts:**

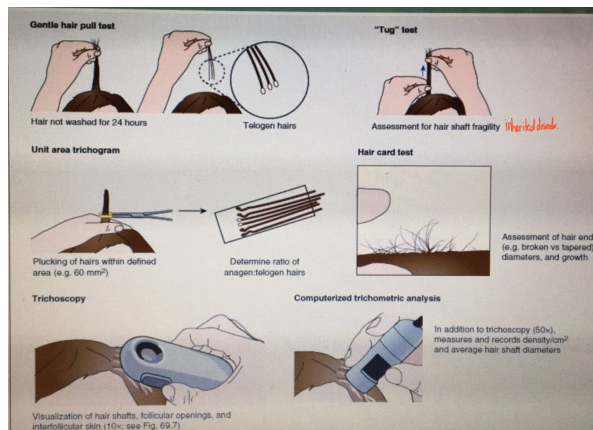
- **How many hairs in the body?** 5 millions hairs; 100,000 in the scalp
- **Hair growth rate?** 0.3mm/day for scalp hair
- **Types of Hair ?** Vellus ويري and Terminal



◀ **Alopecias:**

Non- Scarring	Scarring
<ul style="list-style-type: none"> <li>- Reversible</li> <li>- Intact hair follicles</li> </ul>	<ul style="list-style-type: none"> <li>- Irreversible</li> <li>- Discoid lupus/Lichen planus</li> </ul>

Nonscarring alopecia	Scarring alopecia
<ul style="list-style-type: none"> <li>Telogen effluvium</li> <li>Anagen effluvium</li> <li>Alopecia areata</li> <li>Androgenetic alopecia</li> <li>Hair shaft abnormalities</li> <li>Trauma (e.g., traction)</li> <li>Infectious disorders (e.g., dermatophyte, syphilis)</li> <li>Systemic diseases (e.g., thyroid, systemic lupus erythematosus, iron-deficiency anemia)</li> <li>Intoxications (e.g., vitamin A, Bismuth)</li> <li>Nutritional deficiencies (e.g., zinc, biotin)</li> <li>Medications</li> </ul>	<ul style="list-style-type: none"> <li>Developmental defects (e.g., Aplasia cutis)</li> <li>Infections (bacterial, viral, fungal)</li> <li>Trauma (irradiation, thermal or caustic burns)</li> <li>Neoplastic disorders</li> <li>Lichen planus (lichen planopilaris), lupus erythematosus, morphea, scleroderma, sarcoidosis</li> <li>Keratosis pilaris atrophicans</li> <li>Folliculitis decalvans</li> <li>Dissecting cellulitis of the scalp</li> <li>Acne keloidals</li> <li>Pseudopelade</li> <li>Alopecia mucinosa</li> </ul>



## Alopecia Areata الثعلبة

<b>Definition:</b>	<ul style="list-style-type: none"> <li>● <b>Non-scarring</b> reversible patterned alopecia, most commonly presenting as circular areas of alopecia.</li> <li>● Organ-specific autoimmune disease involving T cells</li> <li>● Can lead to total scalp hair loss (alopecia totalis) or complete scalp and body hair loss (alopecia universalis)</li> </ul>
<b>Pathogenesis:</b>	<ul style="list-style-type: none"> <li>● T-cell-driven autoimmune process</li> <li>● Genetic susceptibility</li> </ul>
<b>Clinical picture:</b>	<ul style="list-style-type: none"> <li>● Well demarcated , non- scarring</li> <li>● <b>Exclamation point</b> كأنها علامة تعجب</li> <li>● Nail: pitting , trachyonychia <b>20 layer dystrophy</b> (sandpaper-like roughness due to excessive longitudinal ridging), brittle nails, onycholysis</li> </ul>
<b>Bad Prognostic signs:</b>	<ul style="list-style-type: none"> <li>● Young age</li> <li>● Atopy</li> <li>● Alopecia totalis, universalis, ophiasis</li> <li>● Nail changes and resistance to Tx</li> </ul>
<b>Diagnosis:</b>	<ul style="list-style-type: none"> <li>● Clinically</li> <li>● Histology: <b>swarm of bees</b></li> <li>● Additional work up : r/o out associated autoimmune disease thyroid function test, CBC , Fasting blood glucose</li> </ul>
<b>Treatment:</b>	<ul style="list-style-type: none"> <li>● Observation (self-limiting )</li> <li>● Topical/ Intralesional Corticosteroids</li> <li>● Skin Sensitizers/ irritants : Anthralin , Diphenylcyclopropenone (DPCP)</li> <li>● Topical immunotherapy (e.g. squaric acid dibutyl ester)</li> <li>● Systemic steroid , methotrexate, cyclosporine</li> <li>● Jak inhibitor tofacitinib</li> <li>● Minoxidil <b>Anti-HTN</b></li> <li>● Phototherapy</li> </ul>



universalis



ophiasis

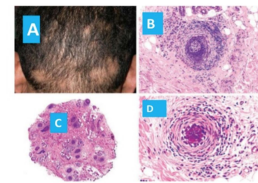
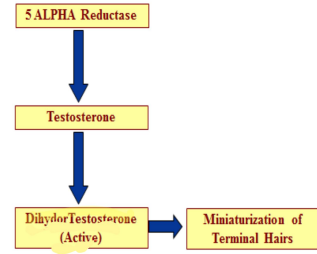


Figure 10. **Alopecia areata.** A, Multiple patches of hair loss with 'exclamation-point' hairs. B, Sub-isthmic region: a hair bulb with peribulbar lymphoid cell infiltrate ('swarm of bees'). C, 'Shift out of anagen': all the hair follicles are in telogen phase. D, Anagen-like nanogen hair follicle with no central hair shaft, and perifollicular lymphocytes.

# Androgenetic Alopecia

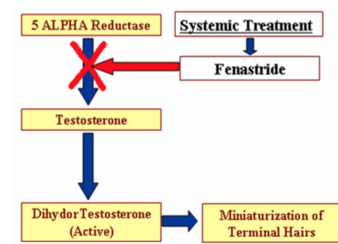
- Genetically determined sensitivity of scalp hair follicles to adult levels of androgens
- Miniaturization of hairs in a symmetric pattern on the crown , vertex and frontal regions. **The terminal hair shifts into vellus hair**
- High concordance of MPHL amongst monozygotic twins indicates a strong genetic predisposition. The inheritance is polygenic .



Male pattern	Female pattern
<p><b>Norwood-Hamilton Scale of Male Pattern Baldness</b></p> 	<p><b>Sinclair scale for female pattern hair loss</b></p> <p>Stage 1    Stage 2    Stage 3 (Christmas tree)    Stage 4    Stage 5</p> <p>The Sinclair scale for female pattern hair loss. Stage 1—normal; Stage 2—widening of the central part line ; Stage 3—widening of the part line with translucency of the hair at its border; Stage 4—development of a bald area anteriorly along the part line; and Stage 5—advanced hair loss</p>
<ul style="list-style-type: none"> <li>• Male pattern</li> </ul>	<ul style="list-style-type: none"> <li>• Anterior hairline is intact</li> <li>• Stage 3 = christmas tree</li> </ul>

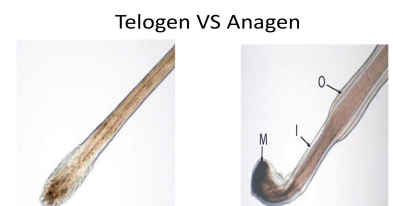
## ◀ Treatment:

- **Topical:** Minoxidil 2%- 5% solution S/E: headache, hypertrichosis in unwanted sites.
- **Systemic:** Finasteride (type II 5  $\alpha$  -reductase inhibitor) **impotence in males + feminization of genitalia "caution in pregnancy"** , Dutasteride (is a combined type I and type II 5  $\alpha$  -reductase inhibitor), Spironolactone
- **Hair transplant**



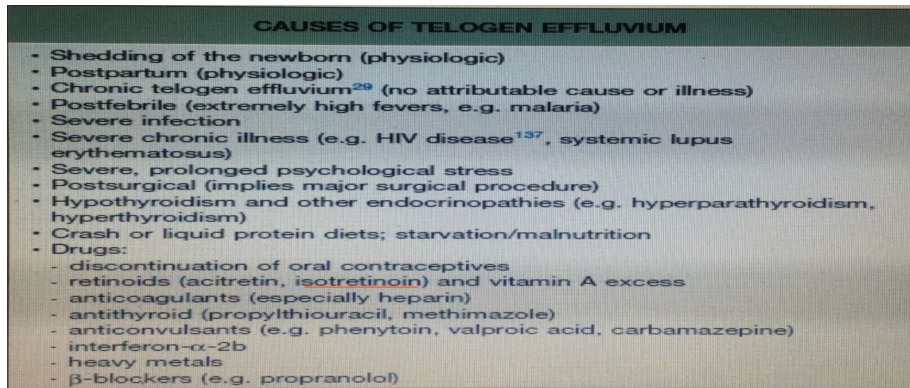
# Telogen effluvium

- Increased shedding of otherwise normal telogen hairs in response to a pathologic or normal physiologic change in health status
- A chronic form with no known precipitating factor is observed in some women
- Laboratory evaluation is directed by history, physical examination, and microscopic evaluation of shed or plucked hairs





## ◀ Causes:



## ◀ Clinical Picture:

- Thinning of the hair
- A gentle hair pull maybe positive for two or more normal telogen hairs
- A forcible hair pluck(trichogram)will show a mixture of anagen and telogen hairs with the percentage of telogen in excess of 20%

## ◀ Treatment:

- Treat the underlying cause
- Minoxidil

## Anagen effluvium

- **Always related to cytotoxic chemotherapy.**
- Acute and severe alopecia
- Mostly reversible but not always **cooling and banding decrease the hair loss**


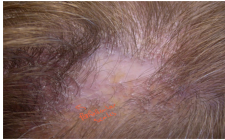
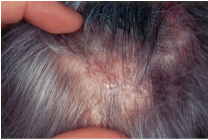

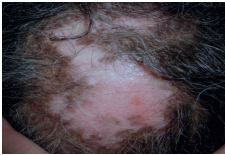
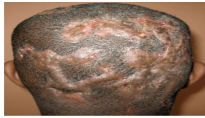

## Trichotillomania

- 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.**



## Cicatricial (Scarring) Alopecias

- ◆ Central centrifugal cicatricial alopecia Lichen planopilaris
- ◆ Discoid lesions of lupus erythematosus
- ◆ Acne keloidalis (folliculitis keloidalis, acne keloidalis nuchae)
- ◆ Dissecting cellulitis (perifolliculitis abscedens et suffodiens)

<b>Central Centrifugal Cicatricial Alopecia</b>	<ul style="list-style-type: none"> <li>● Slowly progressive, symmetric cicatricial alopecia centered on the crown or vertex</li> <li>● Most often found in black women of African heritage</li> <li>● Early and mild disease can be effectively treated; even severe <b>hot comb alopecia</b></li> <li>● disease may be significantly improved with appropriate therapy</li> </ul>	
<b>Lichen Planopilaris</b>	<ul style="list-style-type: none"> <li>● Inflammatory, cicatricial alopecia</li> <li>● <b>Perifollicular scales and erythema</b></li> <li>● Can be very resistant to treatment</li> </ul>	 
<b>Frontal fibrosing alopecia</b>	<p>Progressive hair loss along the frontotemporal hairline. Note eyebrow hair loss as well as the presence of isolated “lonely” hairs on the upper forehead. <span style="color: blue;">:) كأنها موناليزا</span></p>	
<b>DLE</b>	<ul style="list-style-type: none"> <li>● Lesions of discoid lupus erythematosus occur most commonly on the face, ears, and scalp</li> <li>● Histopathologic findings can resemble those found in lichen planopilaris</li> </ul>	
<b>Dissecting Cellulitis of the Scalp</b>	<ul style="list-style-type: none"> <li>● A component of the “follicular occlusion tetrad”</li> <li>● Early disease spares the hair follicles</li> <li>● Inflammation is deep (subcutaneous fat and deep dermis)</li> </ul>	
<b>Traction alopecia</b>	<p style="color: blue;">Non scarring initially but can progress to scarring</p>	<p style="color: red; font-size: small;">Traction alopecia Non-scarring initially, leads to scarring</p> 

# Pigmentary disorders

## Vitiligo

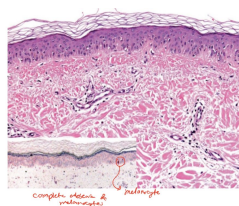
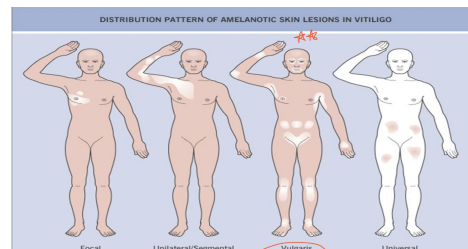
**Definition:** Is an acquired disorder characterized by circumscribed depigmented macules and patches that result from the loss of functional melanocytes

**Pathogenesis:**

- Genetic : 7% of the first-degree relatives of vitiligo patients had vitiligo
- Autoimmune **T-cell mediated**

**Clinical picture:**

- The most common presentation of vitiligo is totally amelanotic (milk or chalk-white) macules or patches surrounded by normal skin
- **Koebner phenomenon**



Leukotrichia\*

Perifollicular pigmentation

\*Leukotrichia: (the new hair is white, also seen in A. areata)

**Diagnosis:**

- Clinical, R/o associated autoimmune disease CBC **pernicious anemia** , T4, TSH, FBS

**Treatment:**

**Localized vitiligo :**

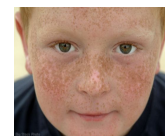
- ❖ Topical steroid
- ❖ Topical calcineurin inhibitors **Tacrolimus**
- ❖ Topical PUVA / UVB
- ❖ Excimer laser
- ❖ Resistant, Stable of 2 years : Surgical **melanocyte transplant**

**Generalized :**



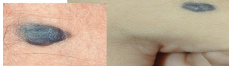


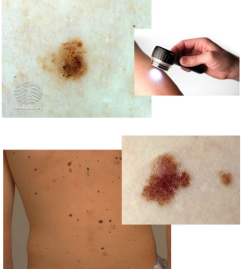
- ❖ Phototherapy
- ❖ Bleaching agent (Monobenzyl ether of hydroquinone ) **مایکل جاکسون استفاده**

## Freckle (Ephelides) النمش

- Small, well-circumscribed, pigmented macules found on sun-exposed skin of individuals with fair skin
- Over-activity of melanocyte , melanocytes are larger and have more branching of dendrites
- Sun block & bleaching cream - Pigmented laser (recurrence)



## Melanocytic nevi

<b>Acquired MN</b>	very common, small, uniform, no need for treatment except ABCDE (Asymmetry , irregular borders, non-homogenous color , diameter more than 5 mm , evolution).	<p>Acquired Melanocytic nevus</p> 
<b>Congenital MN</b>	variable size could be Giant CMN (Bathing trunk) could harbor “Malignant melanoma” <ul style="list-style-type: none"> <li>• Small congenital nevi are less than 1.5 cm in diameter-Medium congenital nevi are between 1.5 and 19.9 cm in diameter</li> <li>• Large (or giant) congenital nevi 20 cm or more in diameter (in adults) have a significantly higher risk for developing melanoma than do ordinary nevi</li> <li>• Consider neurocutaneous melanosis in <b>bathing trunk distribution</b> bathing trunk ddx (meningeal involvement “order MRI”, melanoma)</li> </ul>	
<b>Blue nevus</b>	Deep-blue color and common on face, hand or feet.	
<b>Halo nevus</b>	Compound nevus with halo of depigmentation (association with vitiligo atypia, melanoma) <b>3 associations: vitiligo, melanoma, dysplastic nevus</b>	
<b>Spitz nevus</b>	Common on children face with pink or pale brown color , common in children.	
<b>Dysplastic (Atypical) nevus</b>	<ul style="list-style-type: none"> <li>• Larger with one or more atypical signs “4 or more: risk of malignant melanoma in the subject”.</li> <li>• Controversial clinical designation for various nevi that have morphologic changes such as asymmetry, irregular borders and color variation (ABCDE role)</li> <li>• Also a controversial pathologic term used for nevi with certain architectural changes and/or cytologic atypia</li> <li>• The relationship to melanoma is complex</li> <li>• Examine with <b>dermoscopy</b> and biopsy all suspicious lesions</li> </ul>	

## Melasma الكلف

- Genetically programmed increase in melanogenesis affecting the Face
- Could be induced by Pregnancy, OCP and excessive Sun exposure
- Treatment: sun block & bleaching cream **tranexamic acid could be used**

