Hair And Pigmentary Disorders

Objectives :

- To know the anatomy and physiology of hair
- To recognize the primary presentation of main types of hair loss
- To understand the possible pathogenesis of each type.
- To know the scheme of managements lines
- To know the anatomy and physiology of melanocytes
- To recognize the primary presentation of vitiligo
- To understand the possible pathogenesis of vitiligo
- To know the scheme of managements lines

***Sources**: doctor's slides and notes +FITZPATRICK color atlas +433team male and female +434team doctors slides and notes*

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[Color index : Important | Notes | Extra]

Introduction

- Hair is made of a tough protein called keratin
- The hair follicle undergoes life-long cyclic transformations into three primary phases: anagen, catagen, and telogen see below hair cycle
- The human body has around 5 millions hairs
- Around 100k in the the scalp
- The hair grows at a rate 0.3 mm/day and around 1 cm per month

Types of Hair

1-Lanugo Hair: Soft fine pigmented hair that covers much of **fetus** usually shed before birth.

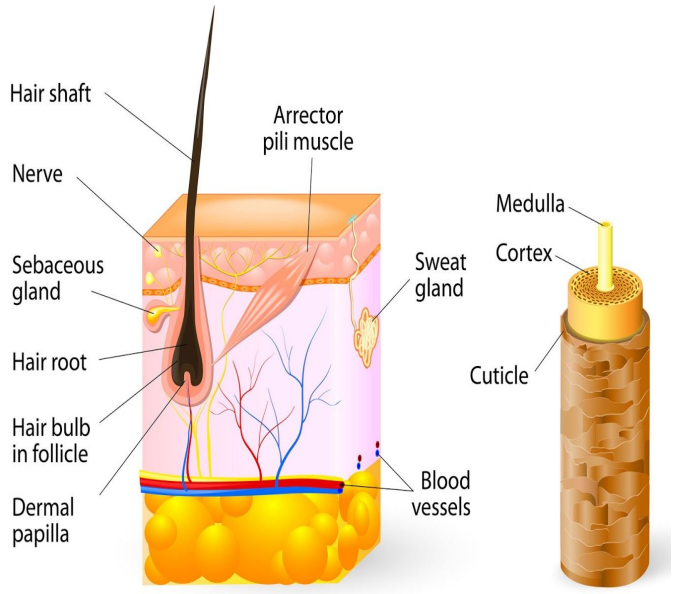
2-VELLUS HAIR: thin, Fine, nonpigmented hair; growth not affected by hormones.

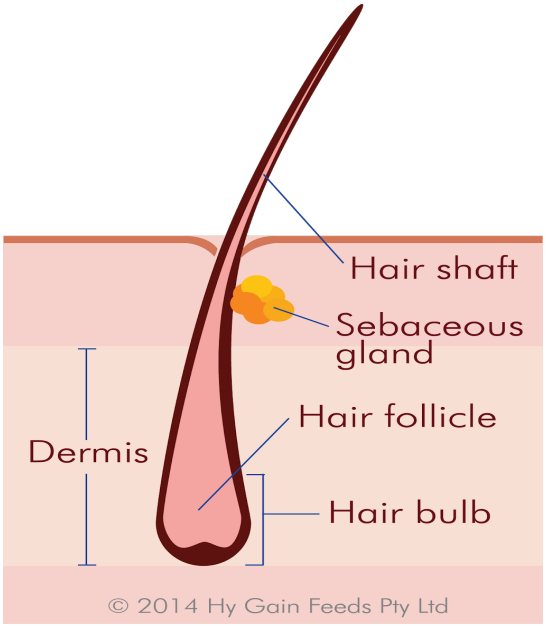
3-TERMINAL HAIR: **Thick**, **pigmented** hair found on scalp, eyebrow/eyelashes, beard, axillae, pubic area; growth is influenced by hormones (e.g. androgenic hair)

Laboratory Examinations

- ***Hair Pull test*** Scalp is gently pulled. Normally, three to five hairs are dislodged; shedding more hair suggests pathology.
- TRICHOGRAM Determines the number o anagen and telogen hairs and is made by epilating (plucking) 50 hairs or more and counting the number of anagen and telogen hairs.
- Scalp Biopsy Offers insight into pathogenesis of alopecia.

Hair Anatomy





Arrector (erect) Pilli (hair): contraction of this muscle causes the hair to "stand up straight" on the skin (goosebumps).

1-hair bulb. forms the base of the hair follicle. In the hair bulb, living cells divide and grow to build the hair shaft

2-hair shaft.

3-dermal papillae (blood supply). (connective tissue + capillary loop)

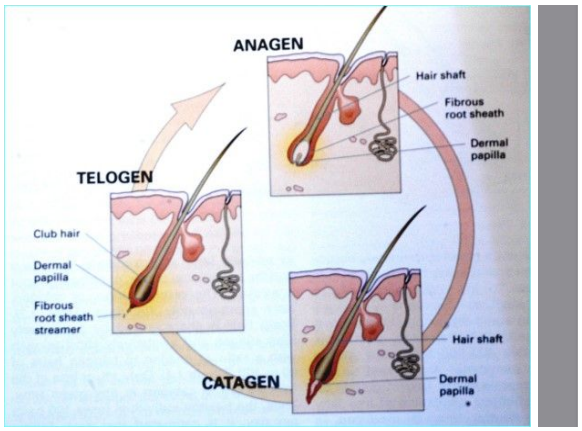
4-hair bulge (reservoir of stem cells)

Attached to the hair follicle are muscle fibers called **arrector pilli muscle**.

also attached to the follicle is **sebaceous glands** which produces the oily or waxy substance.(sebum)

Hair Cycle

1. **ANAGEN Growth phase**: **determines the ultimate length of hair.**
   About 85 to 99% of hairs will be in this phase, with some individual variation.
   Usually Takes 2-6 years
2. **CATAGEN phase:** Apoptosis-driven phase between telogen and anagen phase. Only about 1% of hairs are seen in this phase (**degenerative stage**)
   and usually takes around **2 weeks**
3. **TELOGEN phase:** the Period prior to shedding (‘resting phase’)only about 10% and usually takes around 2-3 months



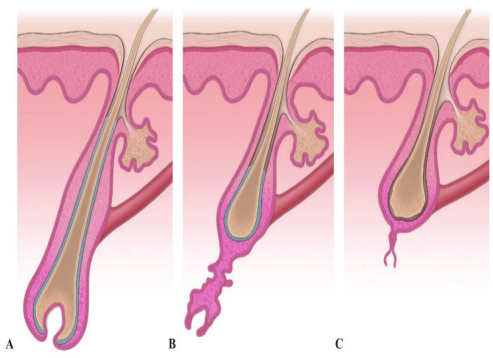


FIGURE 31-1 **Hair growth cycle** Diagrammatic representation of the changes that occur to the follicle and hair shaft during the hair growth cycle. **(A)** Anagen (growth stage); **(B)** Catagen (degenerative stage); **(C)** Telogen (resting stage). (Used with permission from Lynn M. Klein, MD.)

Alopecia (Hair Loss)

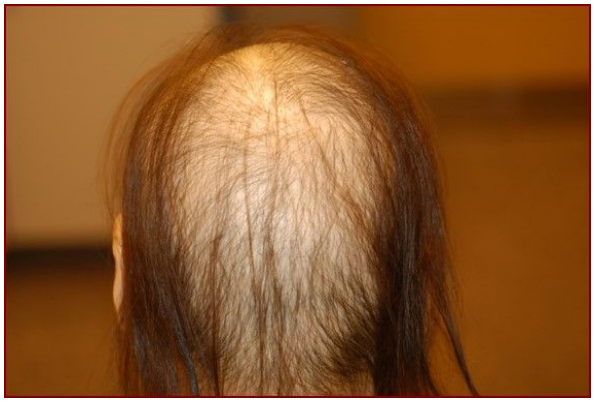
HAIR LOSS: ALOPECIA Ic D-10: 163-166

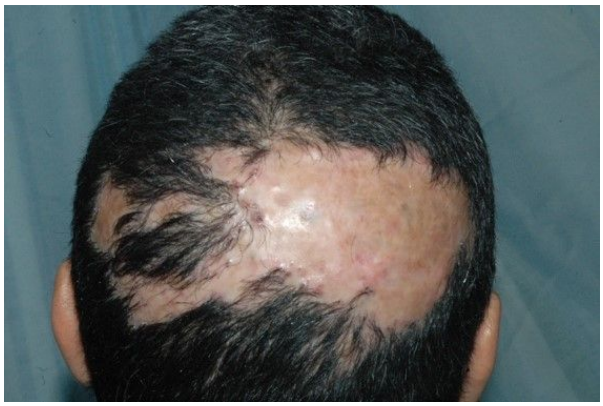
- Shedding of hair is termed effluvium or defuvium, and the resulting condition is called alopecia (Greek alópekia, "baldness").
- Alopecia classified into:
- Noncicatricial alopecia: No clinical sign of tissue inflammation, scarring, or atrophy of skin.
- Cicatricial alopecia: Evidence of tissue destruction such as inflammation, atrophy, and scarring may be apparent.

Divided into:

1-scarring (irreversible) the doctor said that its not included

2-non scarring (reversible)





On the left is non scarring alopecia and the pic on the right represent scarring alopecia

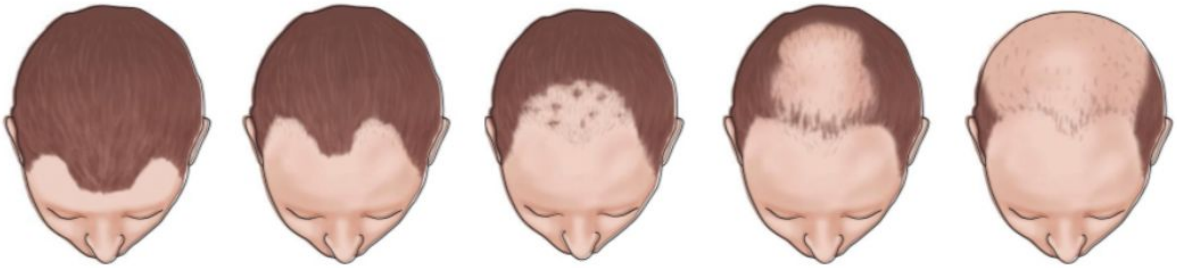
Androgenetic Alopecia: (Male and Female Pattern Hair Loss)

- **Most common type**
- Androgenetic Alopecia affects up to 50% of males and 40% of females
- Autosomal dominant with variable penetrance
- **85% : +ve** family history

pathogenesis;

- 5-alpha reductase enzyme activity which converts testosterone to dihydrotestosterone (DHT) increases in balding scalp
- DHT increases in balding scalp and make the hair villous like hair.
- Number of DHT receptors on the hair follicles increases in balding scalp.
- 5AR not present in the occipital area, explaining the occipital hair remnant.

A. Male (Hamilton classification)



B. Female (Ludwig classification)

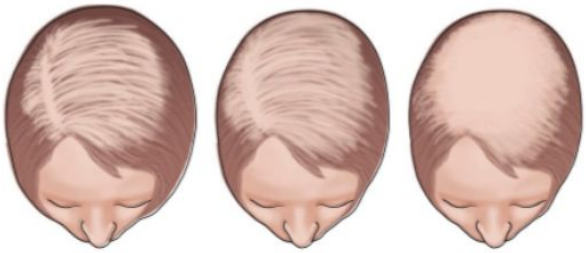


FIGURE 31-3 Androgenetic alopecia: patterns in men and women (A) Hamilton classified the severity and pattern of hair loss in men into types I to V. **(B)** Ludwig classified hair loss in women into types I to III.

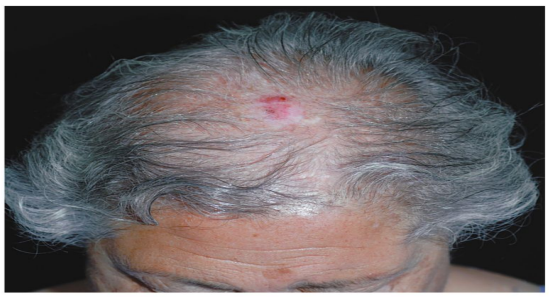


FIGURE 31-7 Pattern hair loss: female, Lugwig type III with basal cell carcinoma (BCC) A 67-year-old Greek female with advanced alopecia of the crown with BCC arising within it.

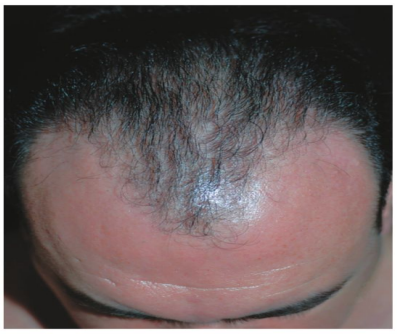


FIGURE 31-4 Pattern hair loss: male, Hamilton type III A 46-year-old male with bitemporal recession of hairline and frontal thinning of hair.

- There is usually a preservation of frontal hair in female, as shown in figure above unlike male.
- Sometimes we see female type in male and the opposite.

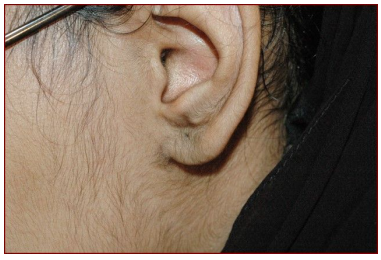
Treatment:

Topical Minoxidil (enhances the anagen phase) 2% and 5% *

Systemic finasteride 5 alpha reductase inhibitor

And there is a new promising hair treatment which is JAK inhibitors

*side effect of minoxidil (increases growth of villous hair (hypertrichosis) + might cause headache +initial hair loss)



Alopecia Areata *(no inflammation)*

ALOPECIA AREATA Ic D-10:163.9

- A localized loss of hair in round or oval areas with no apparent inflammation of the skin.
- Nonscarring; hair follicle intact; hair can regrow.
- Clinical findings: Hair loss ranging from solitary patch to complete loss of all terminal hair.
- Prognosis: good for limited involvement. Poor for extensive hair loss.

Etiology and prevalence:

-Etiology is Unknown. But is Associated with other autoimmune diseases; 30% of persons with alopecia areata (AA) have a familial history of AA. (30%: +ve Family history)

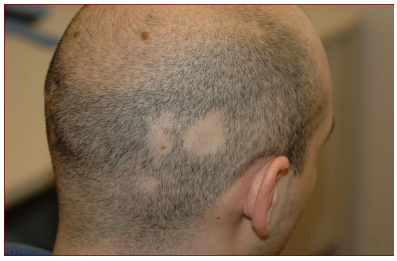
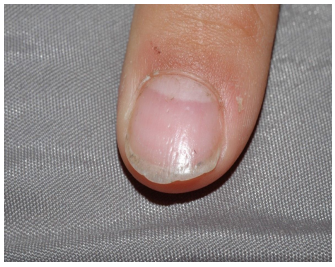
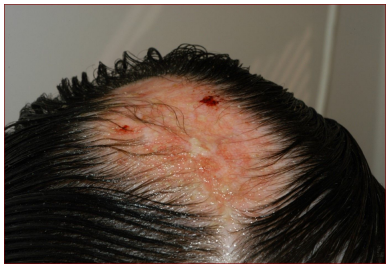
-AGE OF ONSET: Young adults (<25 years); children are affected more frequently.

-Can occur at any age.

-PREVALENCE 1.7% of the US population experiences at least one episode of AA in a lifetime

-75% : Self recovery

Poor prognosis associated with onset in childhood, loss of body hair, nail involvement, atopy, and family history of AAs

NAIL features: Fine pitting ("hammered brass") of dorsal nail plate

Follicular stem cell is spared; hair follicles are not destroyed (there is no scarring).

Also alopecia areata spares white hair

Treatment:(no curative treatment is currently available @from the book 0_0) **(Rx is imp in exam according to doctor)**

1.No Rx and **wait** for spontaneous recovery.(observe)

2. Intralesional corticosteroid don't give to children

3. **Skin Sensitizers** ثوم "Possible MCQ"
(a substance that will induce an allergic response following skin contact)

4.Immune modulating Rx

5.Topical steroids

6. Systemic Steroids

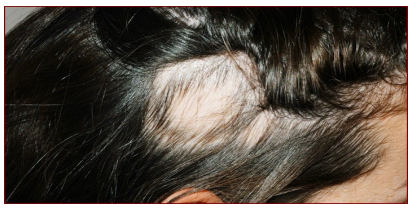
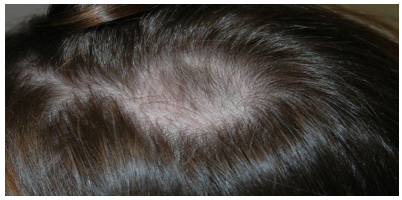
7. Cytotoxic Rx

8. Phototherapy

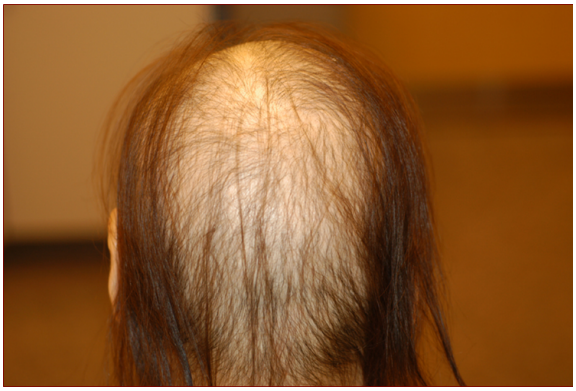
9. Minoxidil might cause hypotension

Hair Transplant is **not used**

Alopecia areata is an autoimmune disease in which the body
attacks its own hair follicles. hair transplantation is not likely to be effective because the transplanted hair would be subject to the same problem

Anagen effluvium (chemotherapy):



- Always related cytotoxic chemotherapy
- (Severity is generally dose dependent)
- (Regrowth is usually rapid after discontinuation of chemotherapy)
- **Onset** is usually rapid (**acute**) and extensive
- Mostly reversible but not always

Could it be preventable? **No effective preventive measures are available** (**book**)

Telogen effluvium

-Telogen effluvium is the transient increased shedding of hair Secondary to accelerated shift of anagen (growth phase) into catagen and telogen (resting phase). It is common and none specific

-Most common after androgenetic hair loss

Hair loss occurs diffusely throughout the scalp

Causes: (rule out thyroid causes and iron deficiency anemia)

- **Chronic alopecia/Anemia with chronic disease**
- Drugs
- Weight loss
- Acute blood loss
- General anasthesia
- Low iron
- Stress

Reversible (but may become chronic)

Treatment:

No intervention is needed or required. The patient should be reassured that the process is part of a normal cycle of hair growth.

Treat the cause

Minoxidil

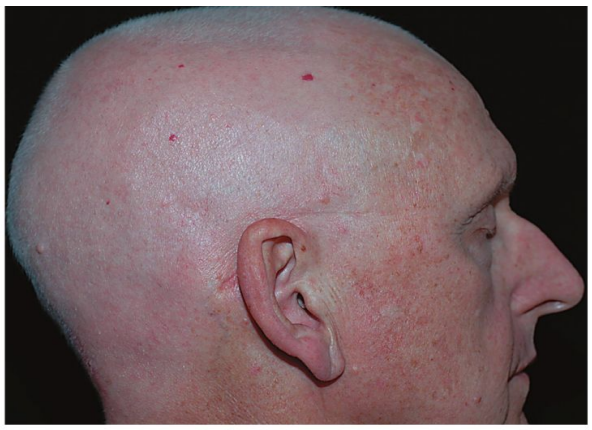


FIGURE 31-12 Anagen effluvium: chemotherapy All scalp, facial, and bodily hair have fallen out. Close inspection reveals that scalp hair has begun to regrow.

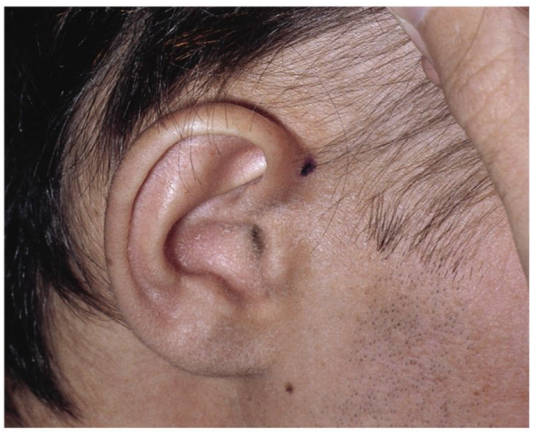


FIGURE 31-11 Telogen effluvium A clump of hair in the hand, associated with striking thinning of scalp hair. Using the fingers as shown, 30 to 40 hairs could be removed with each "hair pull."

Vitiligo (Pigmentary Disorders)

VITILIGO ICD-10: L80

- Worldwide occurrence; 1% of population affected.
- A major psychological problem for brown or black persons, resulting in severe dif culties in social adjustment.
- A chronic disorder with multifactional predisposition and triggering factors.
- Clinically characterized by totally white macules, which enlarge and can affect the entire skin.
- Microscopically: complete absence of melanocytes.
- Rarely associated with systemic autoimmune and/or endocrine disease.

Definition: it is an autoimmune disease results in a **well defined *depigmented* (complete loss of melanin)** macules and patches that can be localized or generalized

Types:

1. **Localized**
2. **Generalized**

Epidemiology:

AGE OF ONSET: May begin at any age, but in 50% o cases it begins between the ages of 10 and 30 years.
INCIDENCE: Common worldwide. Affects up to 1% o the population
RACE: affects All races.
INHERITANCE: Vitiligo has a genetic background;

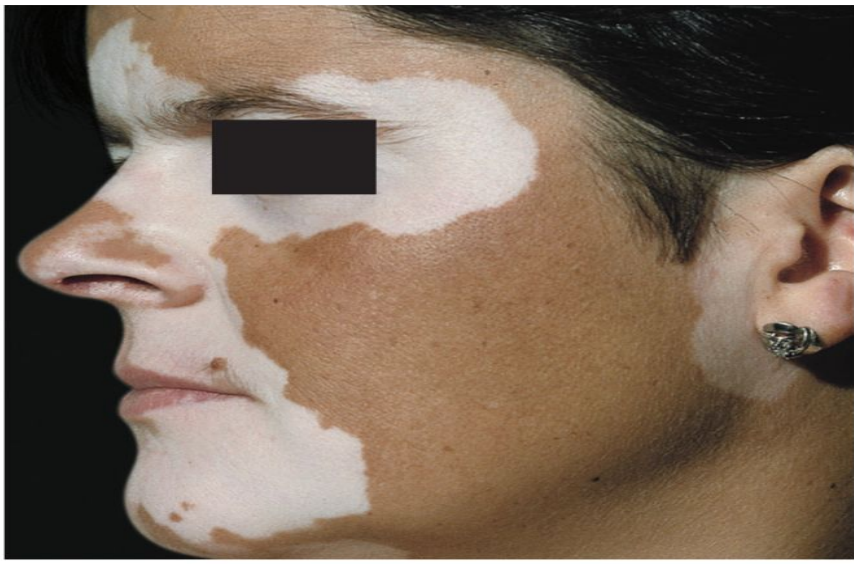
Pathogenesis: three theories most accepted is the autoimmune theory

1. The autoimmune theory holds that selected melanocytes are destroyed by cytotoxic lymphocytes that have somehow been activated.
2. The self-destruct hypothesis suggests that melanocytes are destroyed by toxic substances formed as part of normal melanin biosynthesis. This could then activate mechanisms mentioned in the autoimmune hypothesis.
3. The neurogenic hypothesis is based on an interaction of the melanocytes and nerve cells. This probably holds only for segmental vitiligo.

- Other associated diseases: thyroiditis, Addison's disease, pernicious anemia
- **Natural course:** rarely repigment without treatment

Wood's lamp:

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.



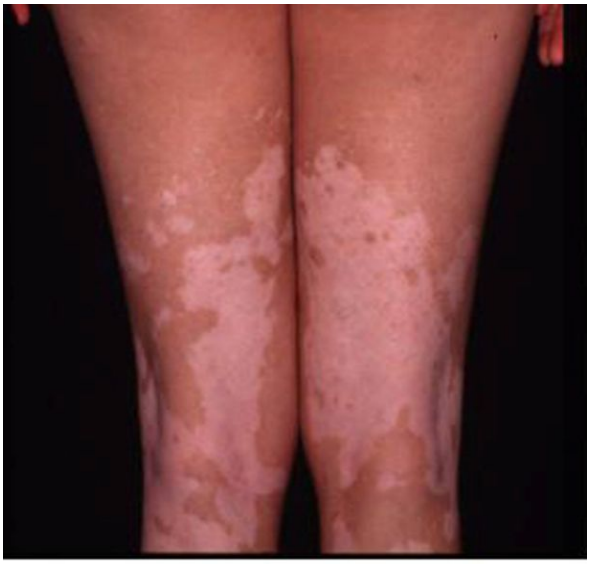


FIGURE 13-1 Vitiligo: face Extensive depigmentation of the central face. Involved vitiliginous skin has convex borders, extending into the normal pigmented skin. Note the chalk-white color and sharp margination. Note also that the dermal melanocytic nevus on the upper lip has retained its pigmentation.

- the white area (left) is described as "depigmented/chalky white", while the right pic is showing hypopigmented area.

Treatment IMP for exam

don't use systemic steroids (it will improve in the beginning but will cause severe aggressive rebound when we stop it)

No single effective Rx

1-Topicals:

- **Corticosteroids** "most common"
- **Immunomodulators tacrolimus**

2-Light Therapy (tanning):

a. UVA or UVB.

b. UVA + Psoralen = PUVA(Topical and Systemic).

(PSORALEN تخلي الجسم يمتص اكبر كمية من الضوء "its local")

c. UVB = Laser.

3-Surgical Rx: .Melanocyte Transplant

.Skin Grafts Transplant

4-Bleaching Agents: Depigment all skin

Treatment Pearls

- **they think the hair is the reservoir of melanocyte**

Thus areas with no hair like the Tips of fingers, toes and the Lips are hard to treat

also **Bony prominence (due to koebner phenomenon)**

- After being treated there is a high **Recurrence rate**
- Treatment **Rarely 100% repigmentation for large area the larger the area the harder**
- Adverse effect of Rx