Pigmentary and Hair disorders

By:
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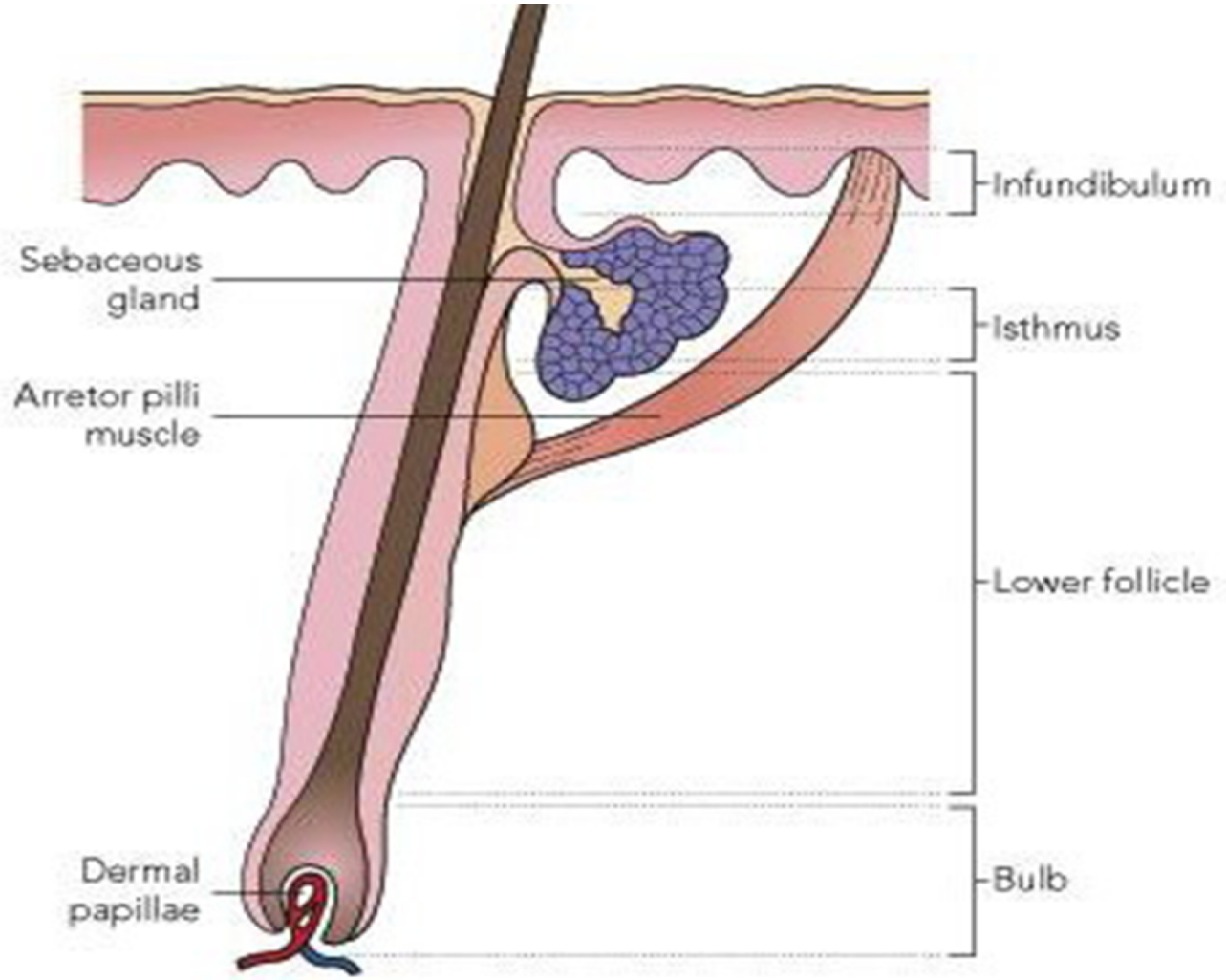
Hair Disorders


Infundibulum
Sebaceous gland
Isthmus
Arretor pilli muscle
Lower follicle
Dermal papillae
Bulb

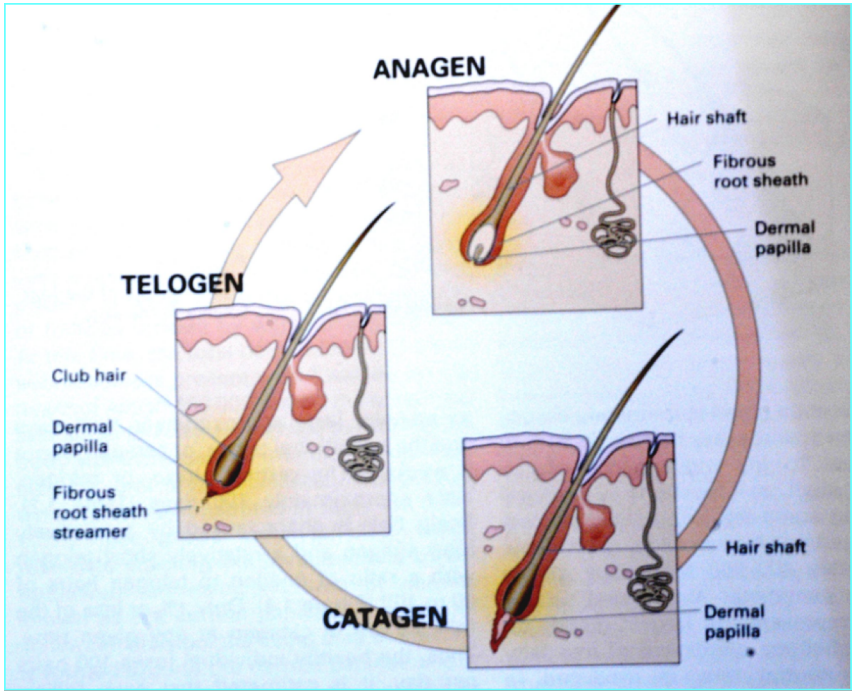
Hair cycle

Hair are in asynchronous continuous cycle

-Anagen phase (growth phase) (3-6 years) : about 85 % of scalp hair are anagens

-Telogen phase (shedding) (3-6 months) , 10 % of scalp hair are telogen

-Catagen phase (transitional) (3-6 weeks) 5% of scalp hair are catagen



Facts

How many hairs in the body?

5 millions hairs; 100,000 in the scalp

Hair growth rate?

0.3mm/day for scalp hair

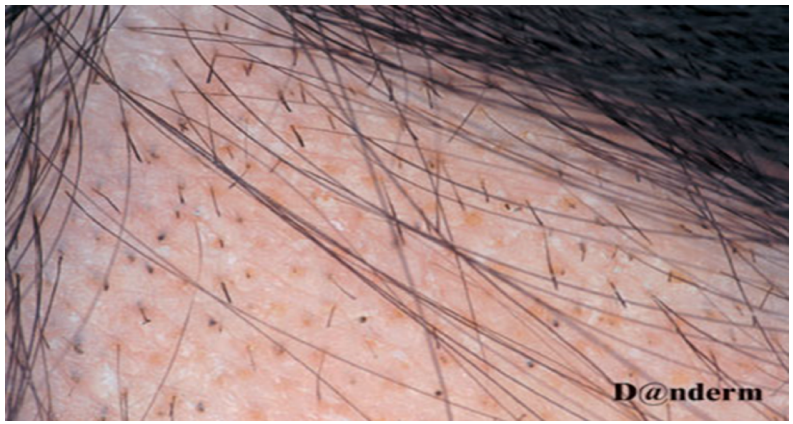
Types of Hair ?

Vellous

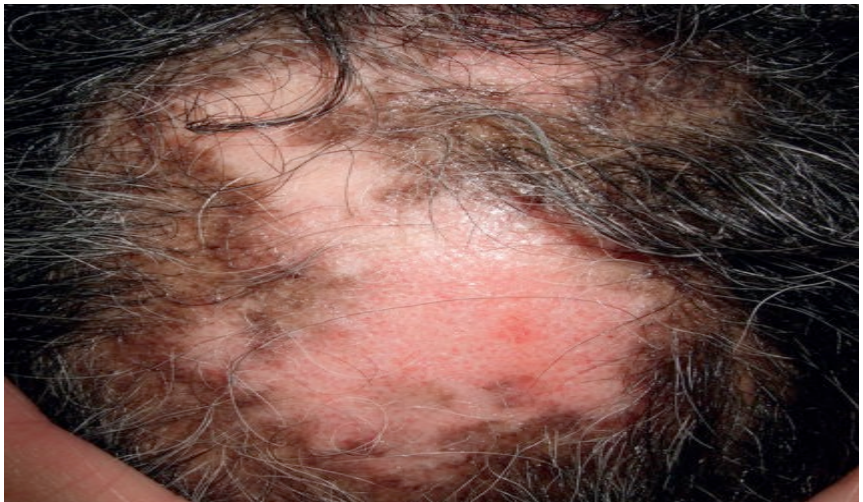
Terminal

Alopecias

Non-Scarring (Reversible)



Scarring (Irreversible)



Nonscarring alopecia	Scarring alopecia
Telogen effluvium	Developmental defects (e.g., Aplasia cutis)
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

BEDSIDE DIAGNOSTIC TESTS FOR THE ASSESSMENT OF ALOPECIA

Gentle hair pull test

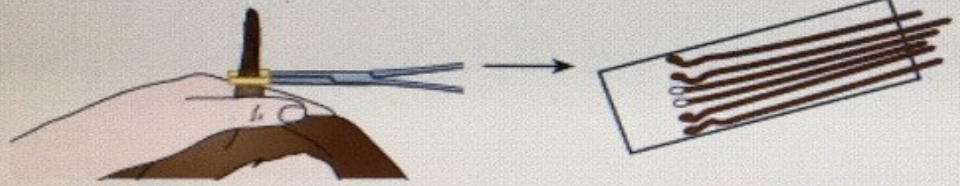
Hair not washed for 24 hours

Telogen hairs

"Tug" test

Assessment for hair shaft fragility

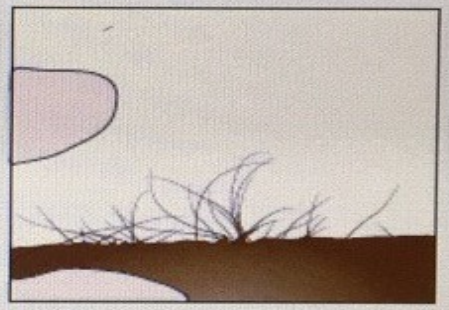
Unit area trichogram



Plucking of hairs within defined area (e.g. 60 mm^2)

Determine ratio of anagen:telogen hairs

Hair card test



Assessment of hair ends (e.g. broken vs tapered), diameters, and growth

Trichoscopy

Visualization of hair shafts, follicular openings, and interfollicular skin (10×; see Fig. 69.7)

Computerized trichometric analysis

In addition to trichoscopy (50×), measures and records density/cm^2 and average hair shaft diameters

Alopecia Areata

- Non-scarring patterned alopecia, most commonly presenting as circular areas of alopecia

- Organ-specific autoimmune disease involving T cells

- Can lead to total scalp hair loss (alopecia totalis) or complete scalp and body hair loss (alopecia universalis)

Alopecia areata

Pathogenesis :

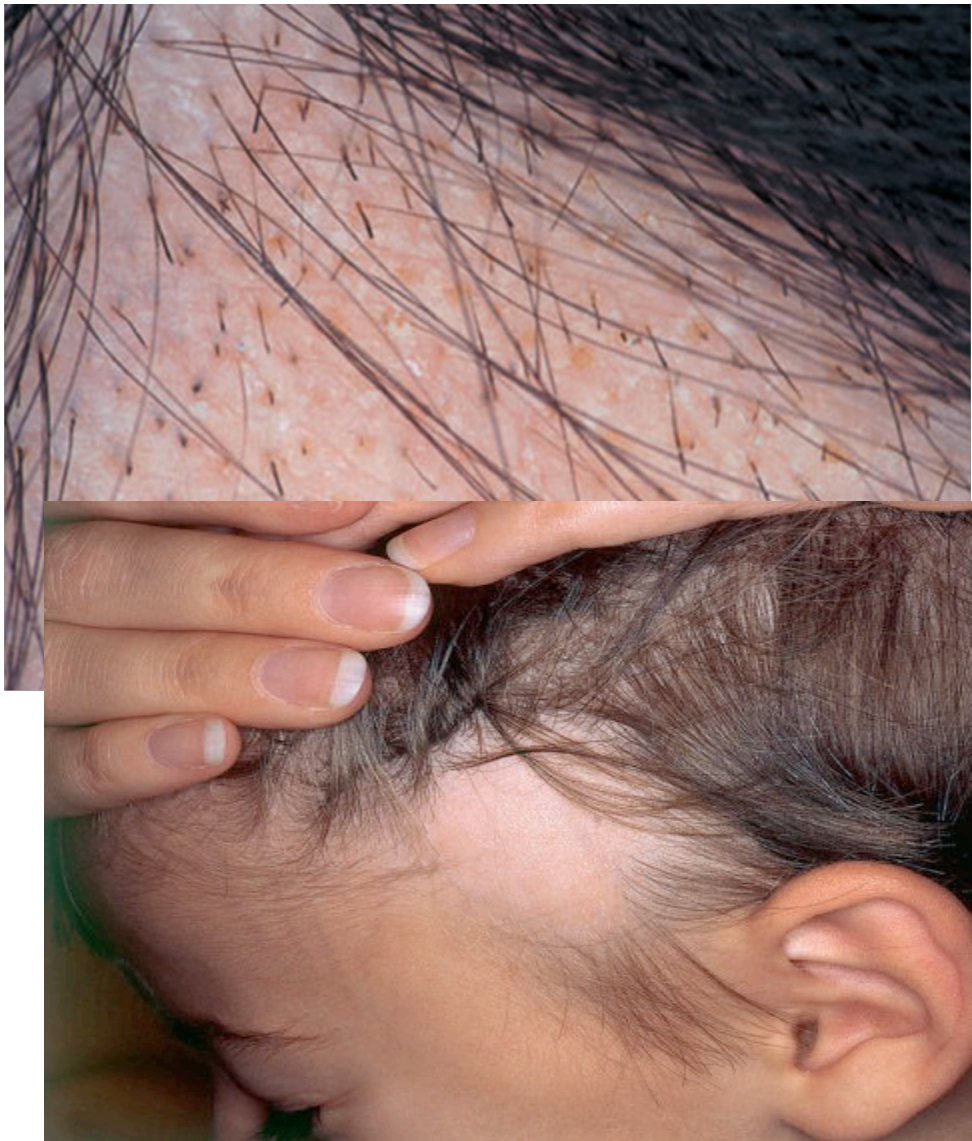
- T-cell-driven autoimmune process
- Genetic susceptibility

Clinical picture :

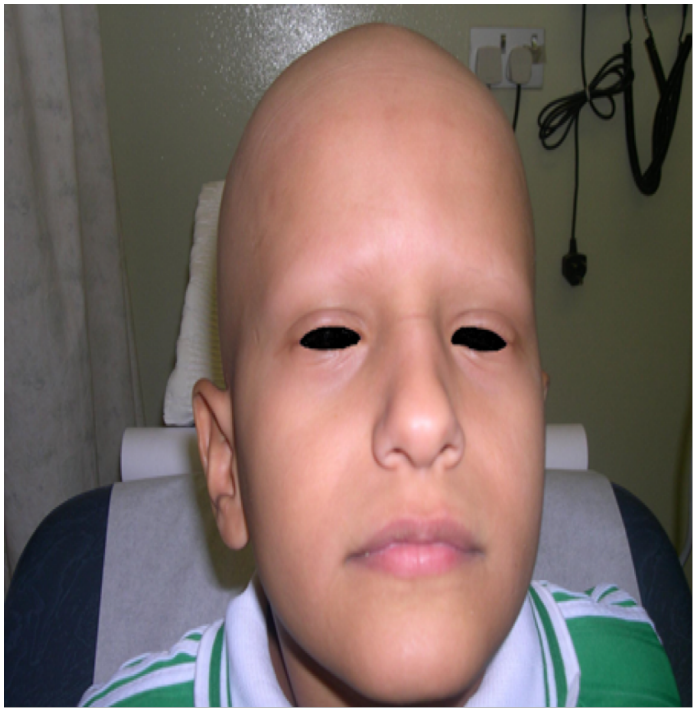
- Well demarcated , non-scarring

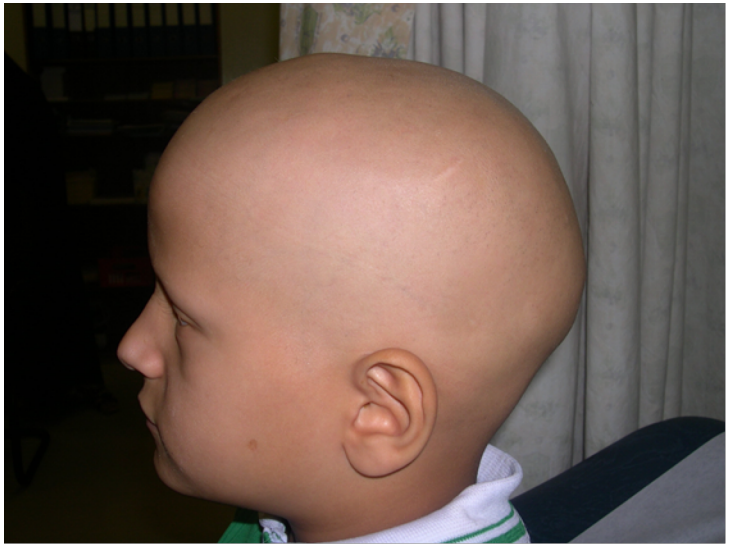
- Exclamation point

- Nail: pitting , trachyonychia (sandpaper-like roughness due to excessive longitudinal ridging), brittle nails, onycholysis

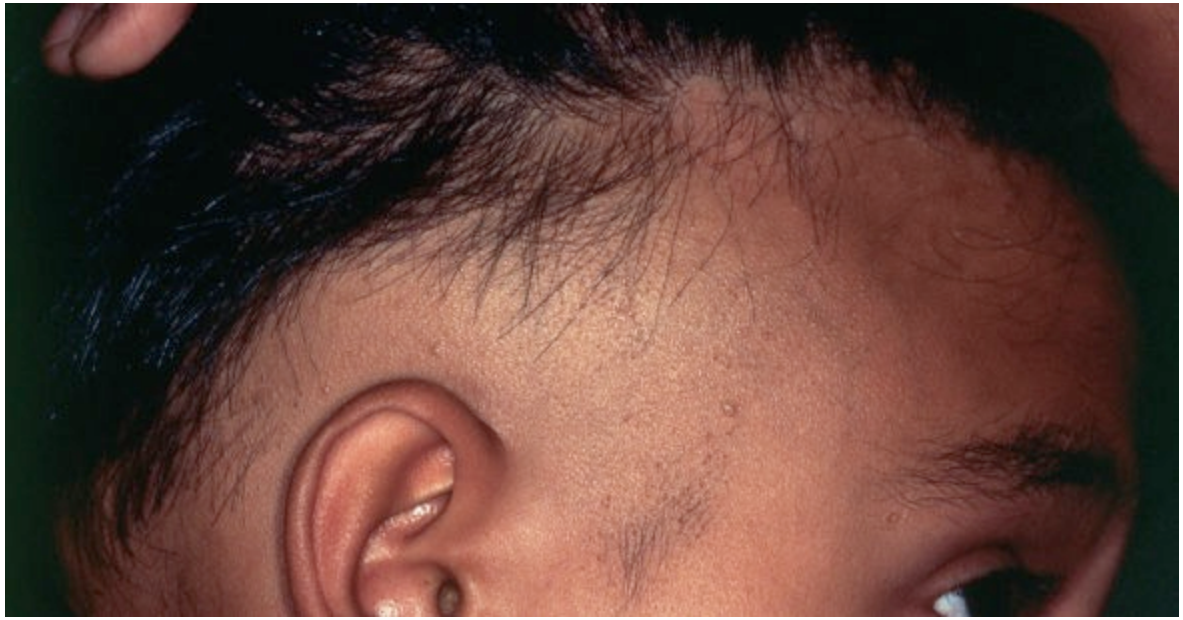


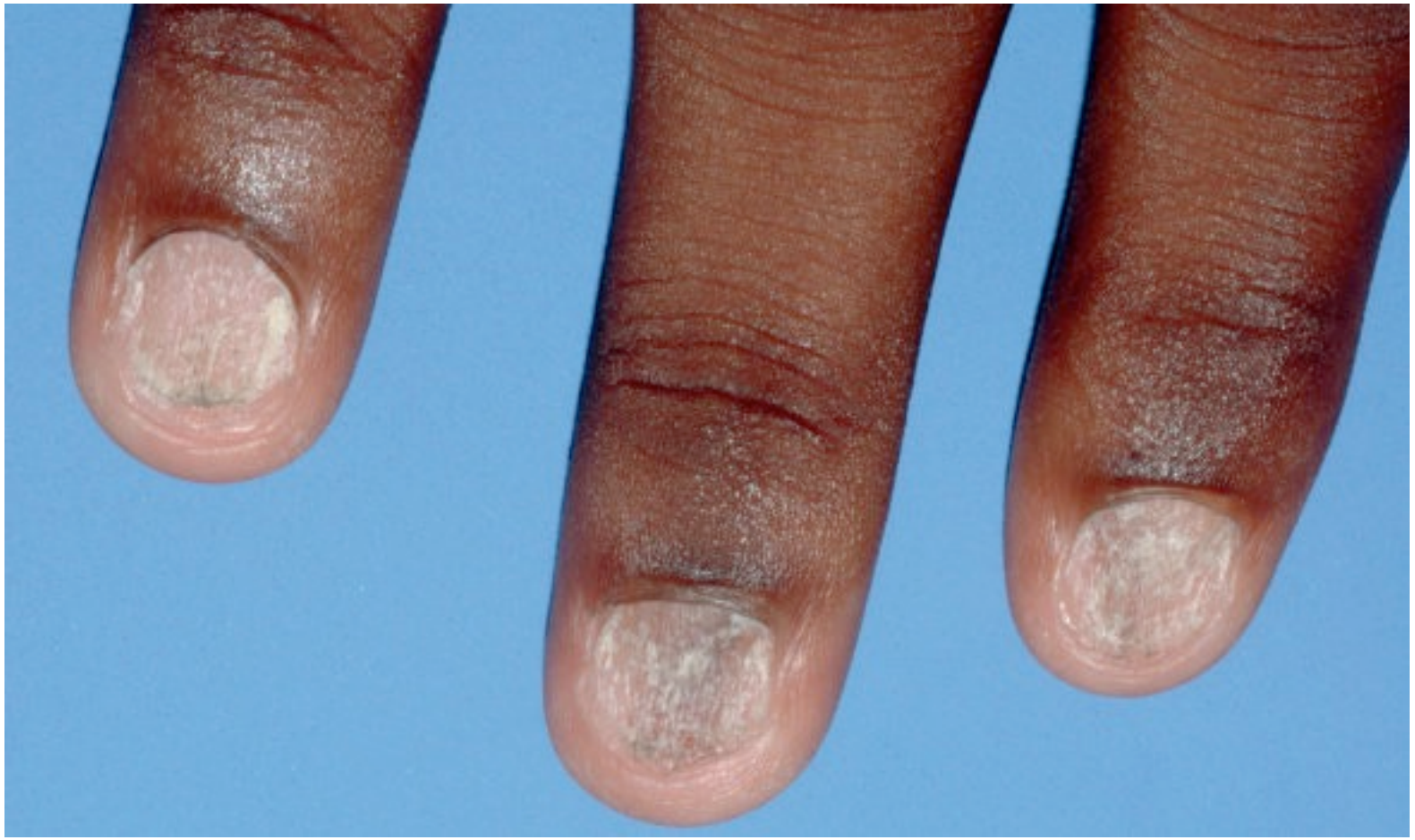
Universalis





Ophiasis





Bad Prognostic signs

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

Diagnosis

- Clinically

-Histology: swarm of bees

-Additional work up : r/o out associated autoimmune disease thyroid function test, CBC , Fasting blood glucose

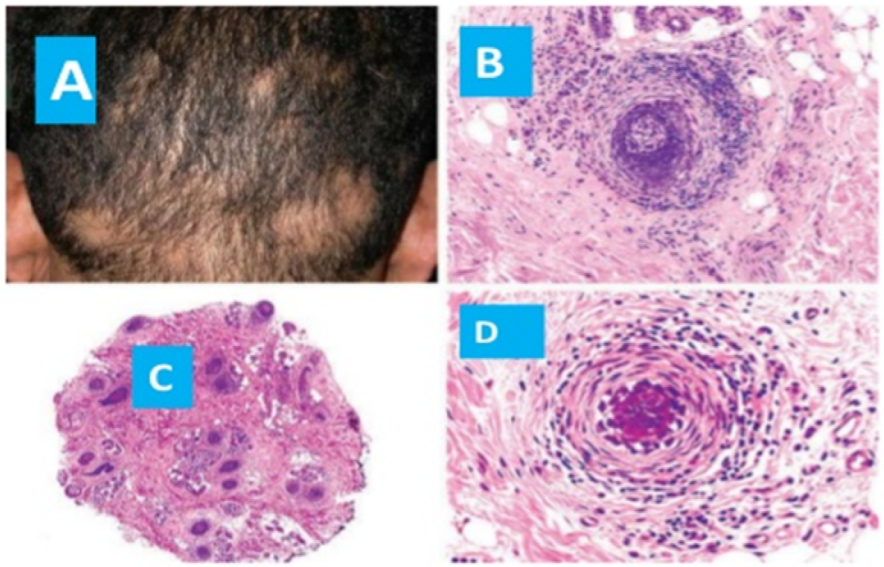


Figure 10. **Alopecia areata.** A, Multiple patches of hair loss with 'exclamation-point' hairs. B, Sub-isthmic region: a hair bulb with peribulbar lymphoid cell infiltrate ('swarm of bees'). C, 'Shift out of anagen': all the hair follicles are in telogen phase. D, Anagen-like nanogen hair follicle with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

Treatment

-Observation (self-limiting)

-Topical/ Intralesional Corticosteroids

-Skin Sensitizers/ irritants : Anthralin , Diphencyclopropenone (DPCP)

-Topical immunotherapy (e.g. squaric acid dibutyl ester)

-Systemic steroid , methotrexate, cyclosporine

-Jak inhibitor tafacitinib

-Minoxidil

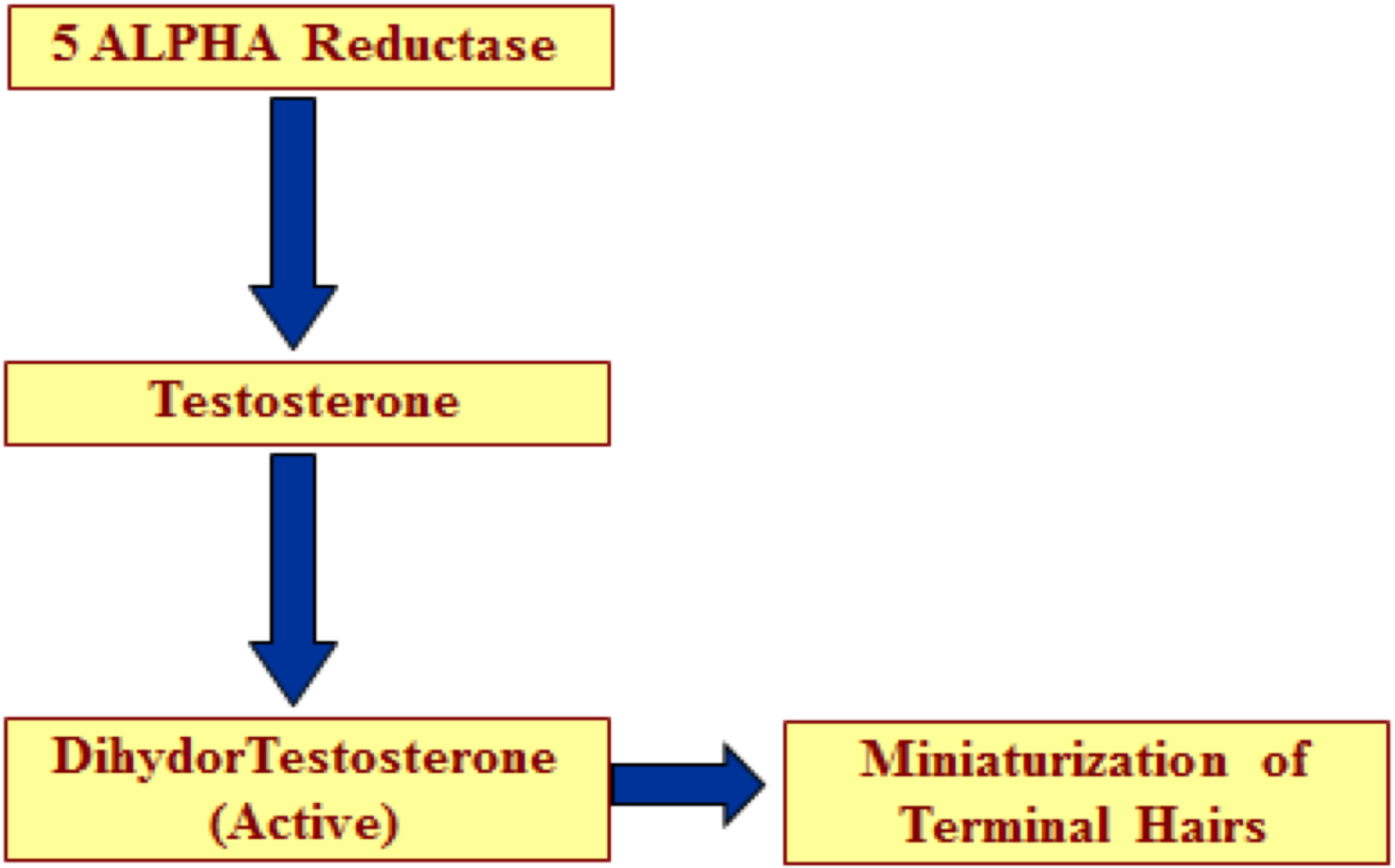
-Phototherapy

Androgenetic Alopecia

- Genetically determined sensitivity of scalp hair follicles to adult levels of androgens

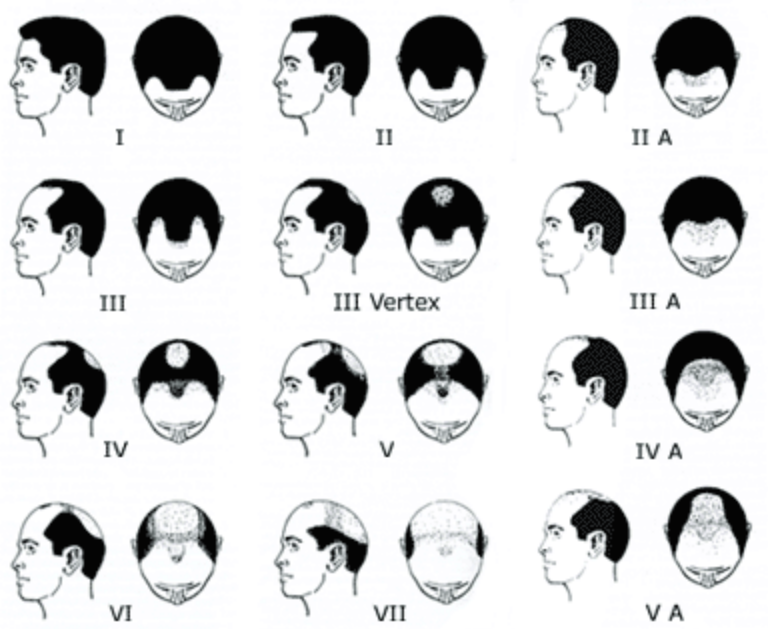
- Miniaturization of hairs in a symmetric pattern on the crown , vertex and frontal regions

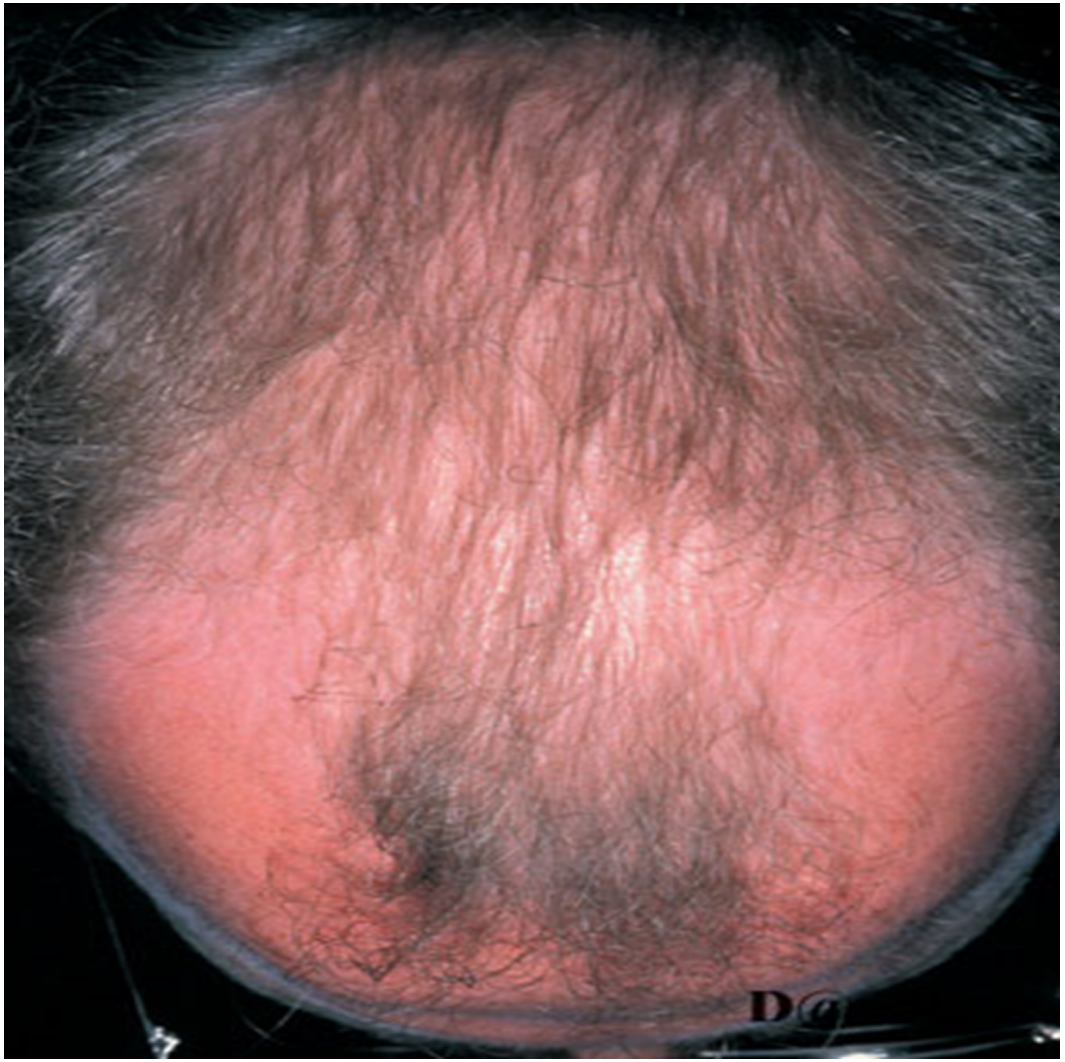
- High concordance of MPHL amongst monozygotic twins indicates a strong genetic predisposition. The inheritance is polygenic .


5 ALPHA Reductase
Testosterone
DihydorTestosterone
(Active)
Miniaturization of
Terminal Hairs

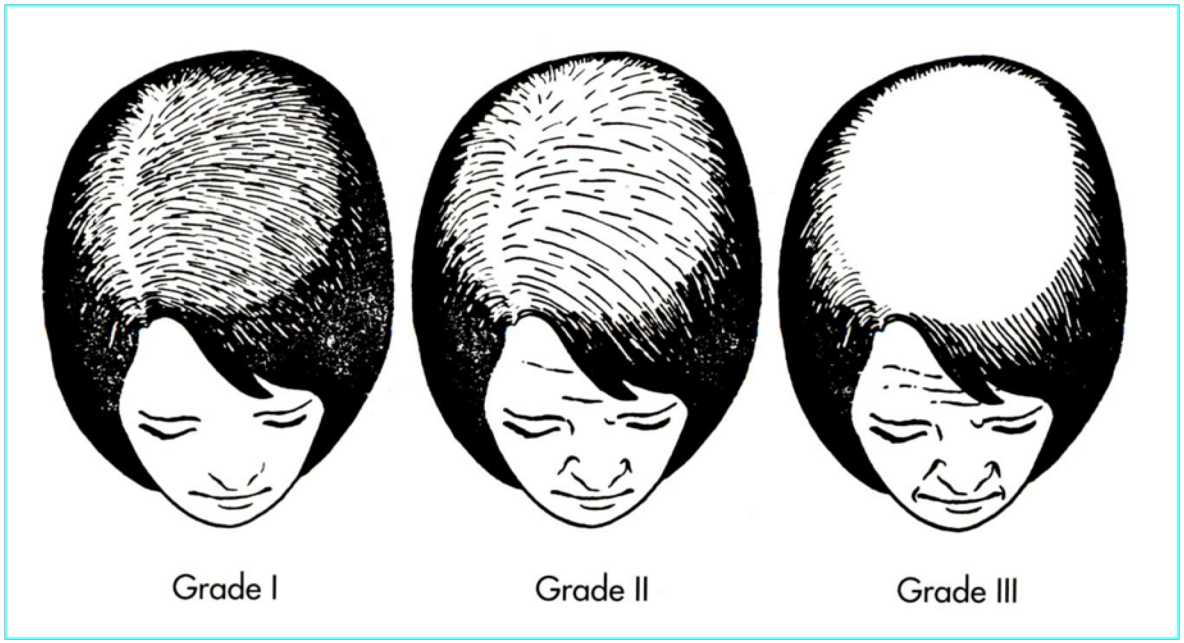
Male pattern

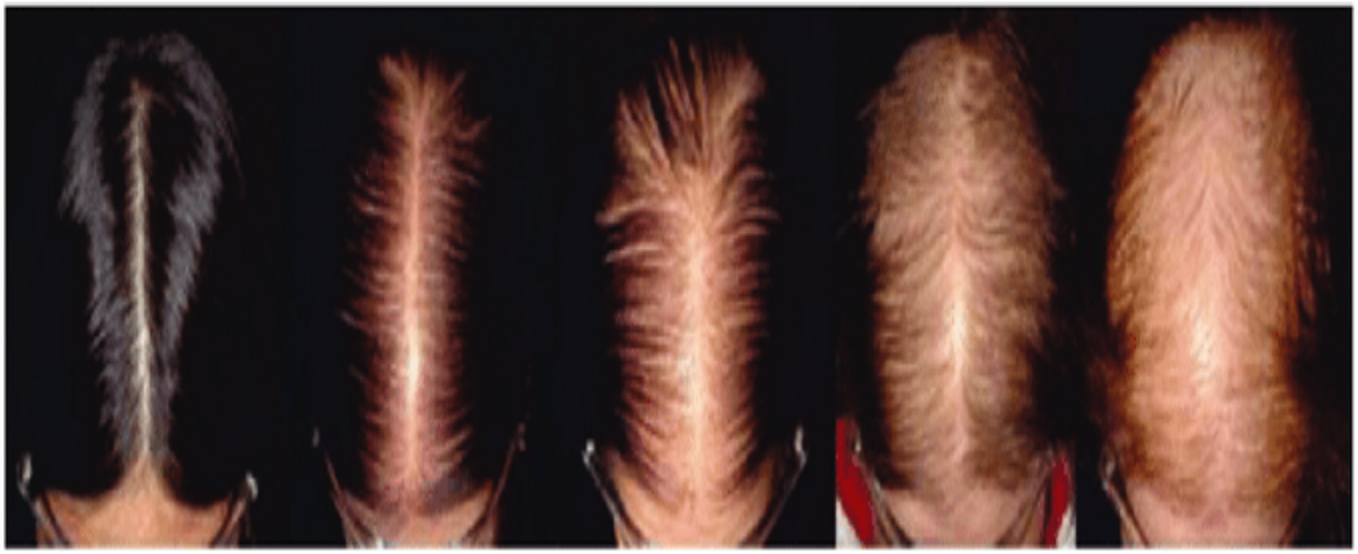
Norwood-Hamilton Scale of Male Pattern Baldness





Female pattern





The Sinclair scale for female pattern hair loss.Stage1–normal; Stage2–widening of the central part line ; Stage3–widening of the part line with translucency of the hair at its border; Stage4– development of a bald area anteriorly along the part line; and Stage5–advanced hair loss

Treatment

-Topical: Minoxidil 2%- 5% solution

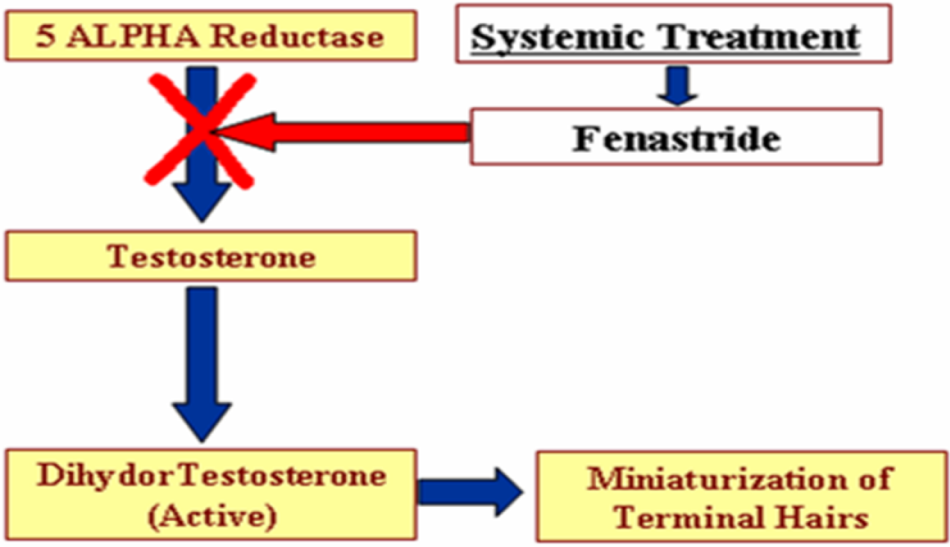
- Systemic:

Finasteride (type II 5α-reductase inhibitor)

Dutasteride (is a combined type I and type II 5α-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

CAUSES OF TELOGEN EFFLUVIUM

- Shedding of the newborn (physiologic)
- Postpartum (physiologic)
- Chronic telogen effluvium[29] (no attributable cause or illness)
- Postfebrile (extremely high fevers, e.g. malaria)
- Severe infection
- Severe chronic illness (e.g. HIV disease[137], systemic lupus erythematosus)
- Severe, prolonged psychological stress
- Postsurgical (implies major surgical procedure)
- Hypothyroidism and other endocrinopathies (e.g. hyperparathyroidism, hyperthyroidism)
- Crash or liquid protein diets; starvation/malnutrition
- Drugs:
  - discontinuation of oral contraceptives
  - retinoids (acitretin, isotretinoin) and vitamin A excess
  - anticoagulants (especially heparin)
  - antithyroid (propylthiouracil, methimazole)
  - anticonvulsants (e.g. phenytoin, valproic acid, carbamazepine)
  - interferon-α-2b
  - heavy metals
  - β-blockers (e.g. propranolol)

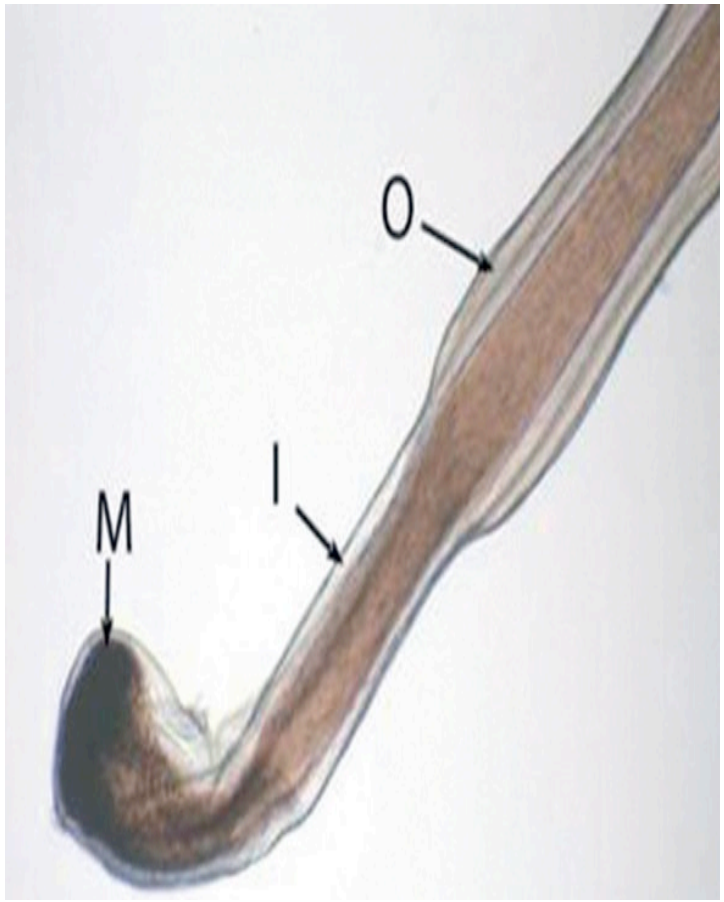
Clinical Picture

- Thinning of the hair

- A gentle hair pull may be 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%

Telogen VS Anagen



Treatment

Treat the underlying cause

Minoxidil

Anagen effluvium

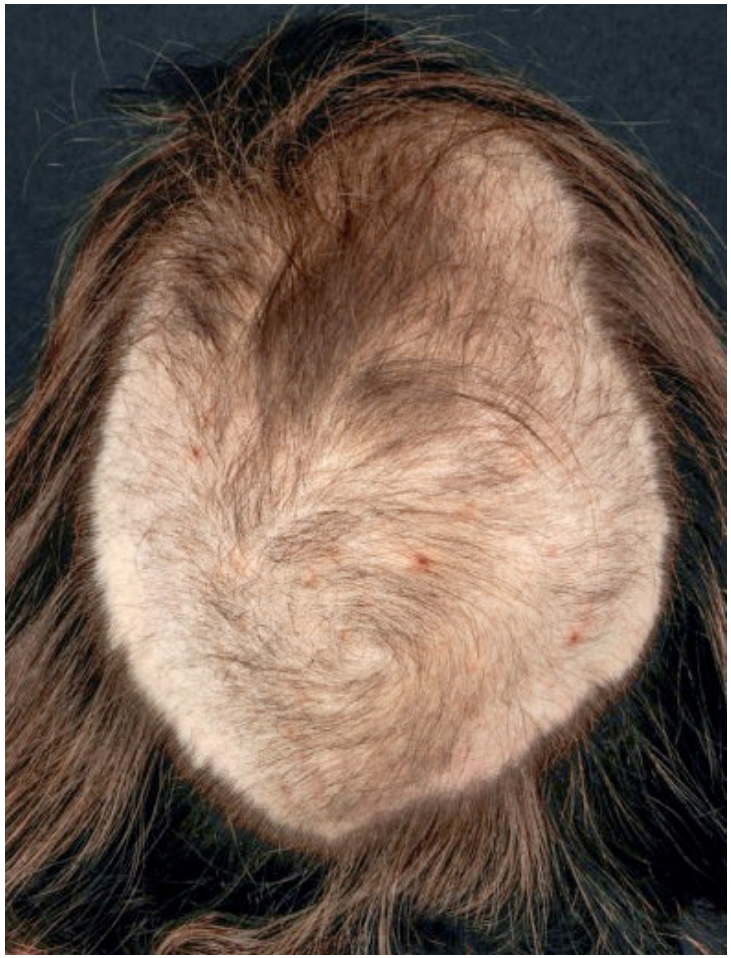
- Always related to cytotoxic chemotherapy
- Acute and severe alopecia
- Mostly reversible but not always

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



CICATRICIAL (SCARRING) ALOPECIAS

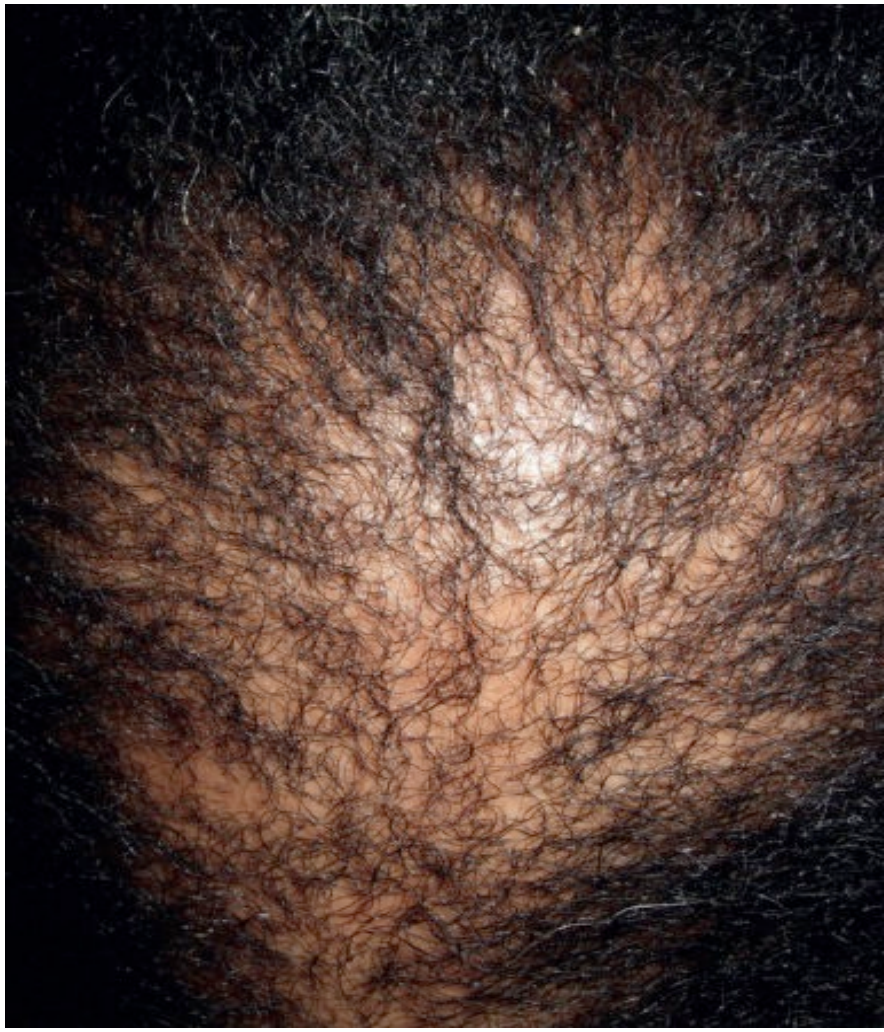
- Central centrifugal cicatricial alopecia
- Lichen planopilaris
- Discoid lesions of lupus erythematosus
- Acne keloidalis (folliculitis keloidalis, acne keloidalis nuchae)
- Dissecting cellulitis (perifolliculitis abscedens et suffodiens)

Central Centrifugal Cicatricial Alopecia

- Slowly progressive, symmetric cicatricial alopecia centered on the crown or vertex

-Most often found in black women of African heritage

- Early and mild disease can be effectively treated; even severe disease may be significantly improved with appropriate therapy

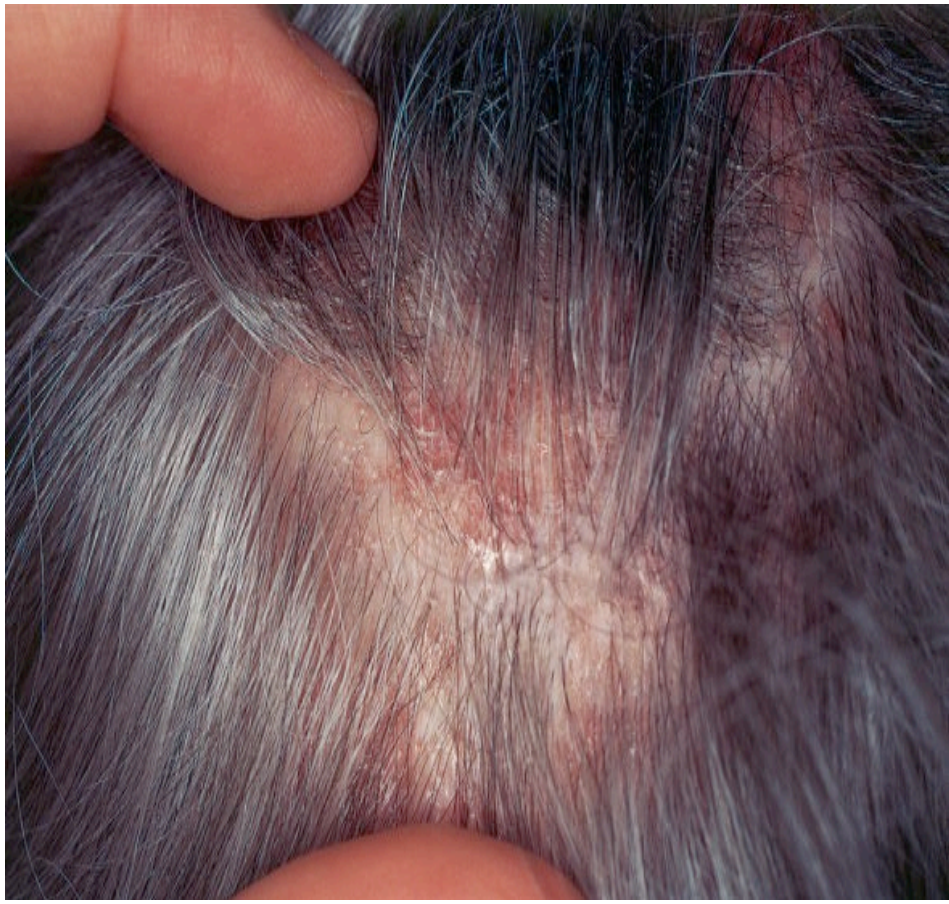


Lichen Planopilaris

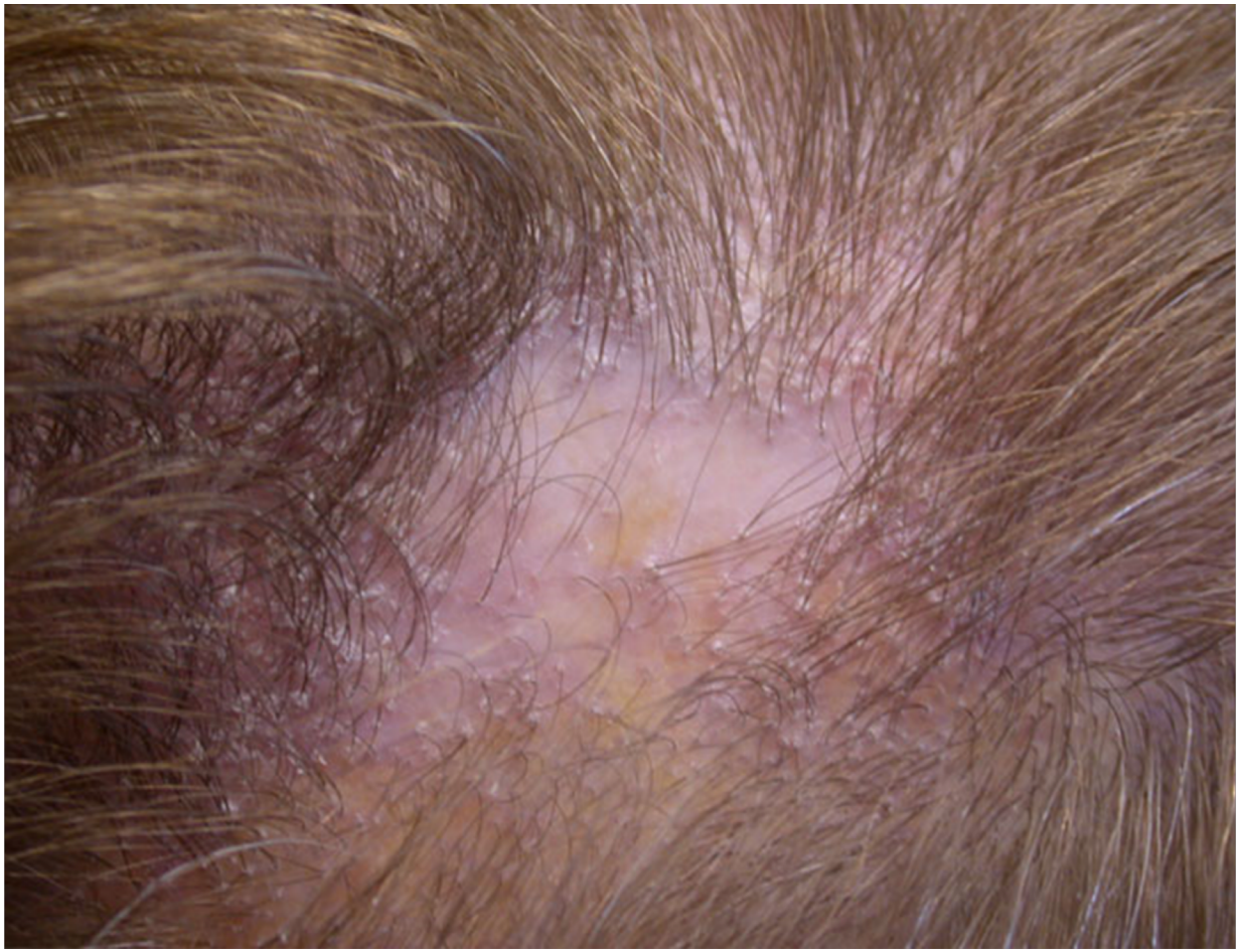
-Inflammatory, cicatricial alopecia

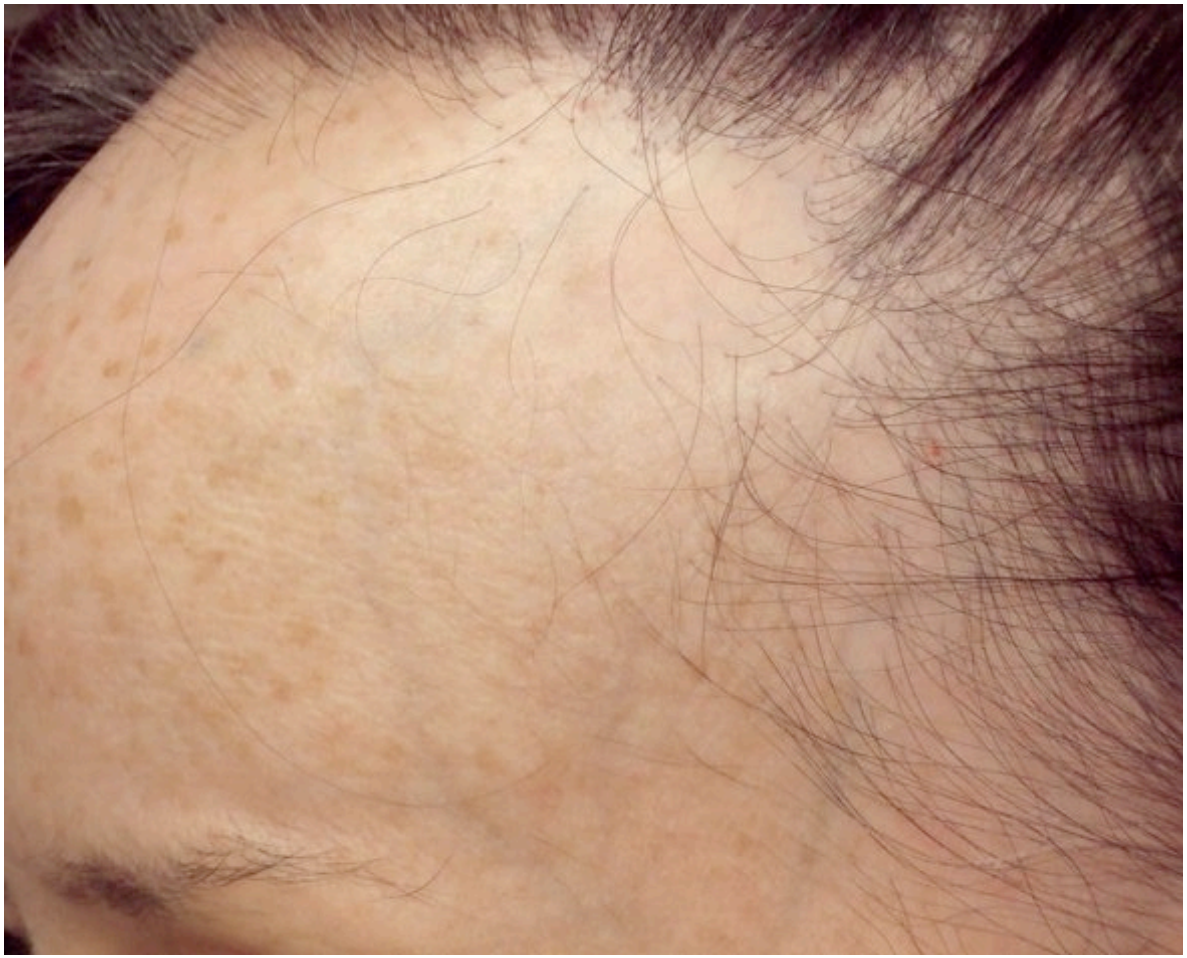
-Perifollicular scales and erythema

- Can be very resistant to treatment



Lichen planopilaris



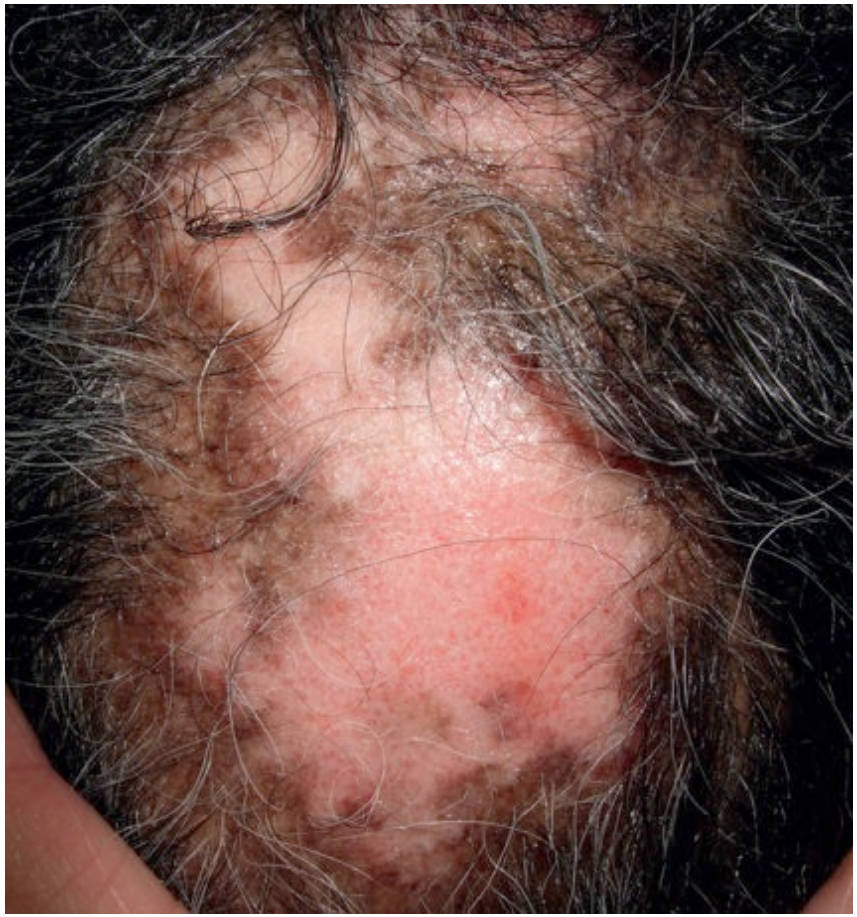


Frontal fibrosing alopecia. Progressive hair loss along the frontotemporal hairline. Note eyebrow hair loss as well as the presence of isolated “lonely” hairs on the upper forehead

DLE

- Lesions of discoid lupus erythematosus occur most commonly on the face, ears, and scalp

- Histopathologic findings can resemble those found in lichen planopilaris

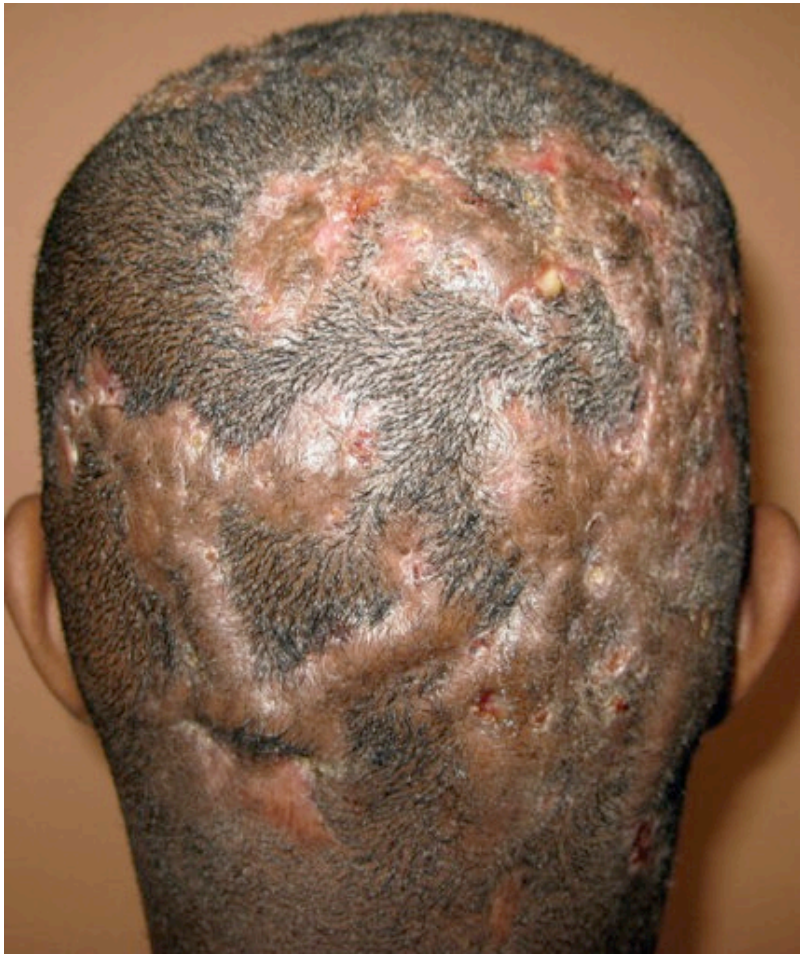


Dissecting Cellulitis of the Scalp

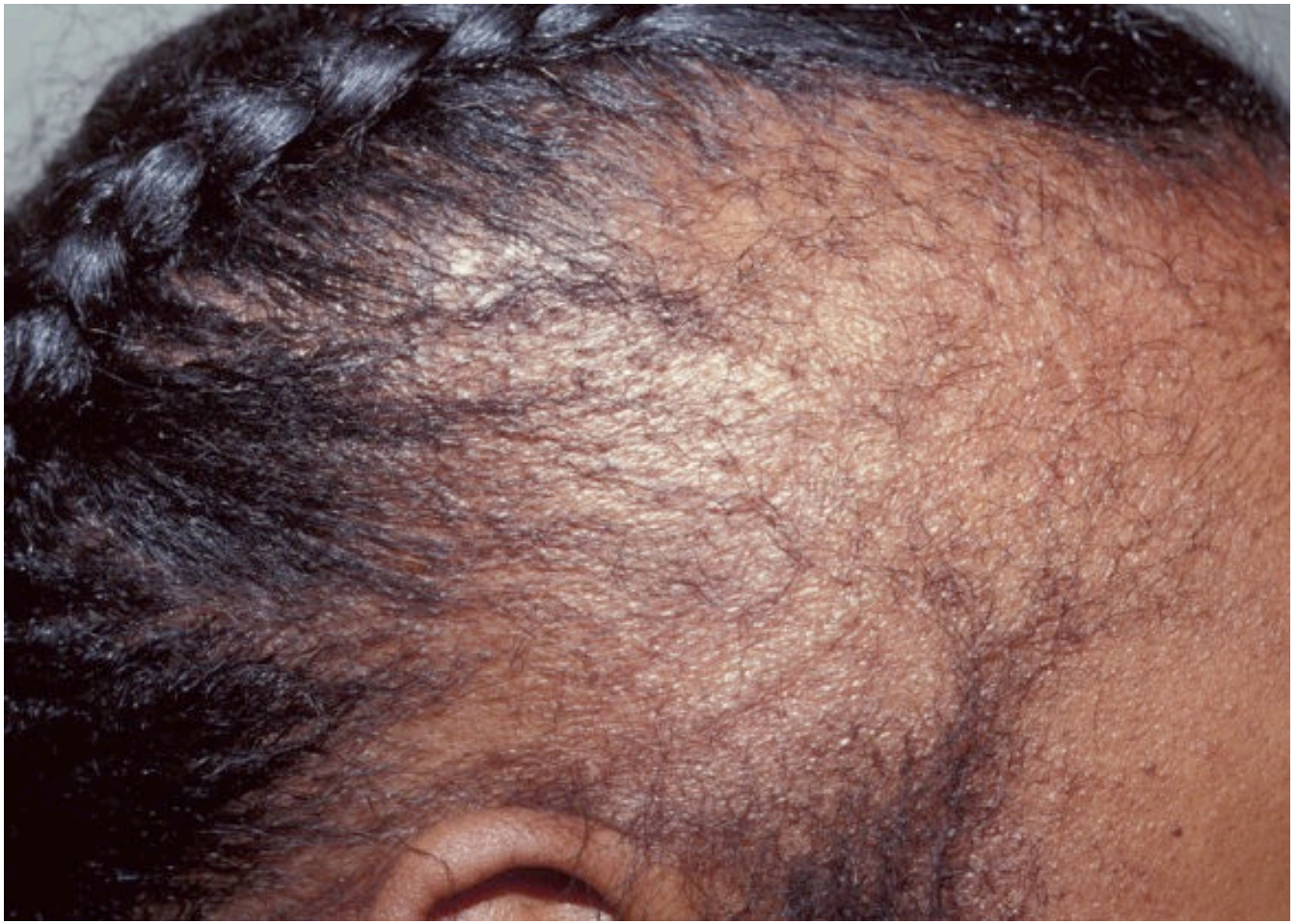
- A component of the “follicular occlusion tetrad”

- Early disease spares the hair follicles

- Inflammation is deep (subcutaneous fat and deep dermis)



Traction alopecia



Pigmentary disorders

Vitiligo

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

Vitiligo

Pathogenesis :

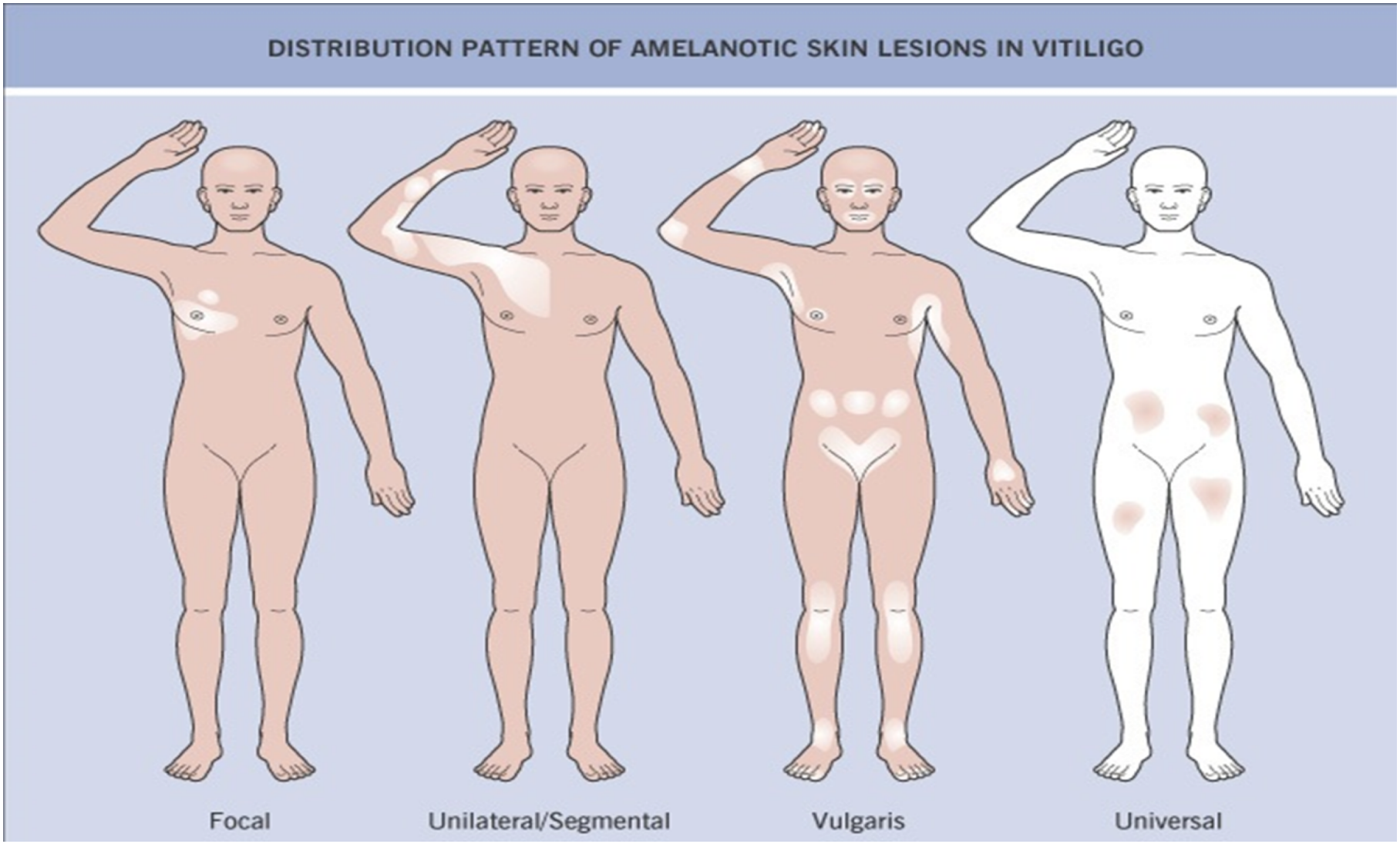
-Genetic : 7% of the first-degree relatives of vitiligo patients had vitiligo

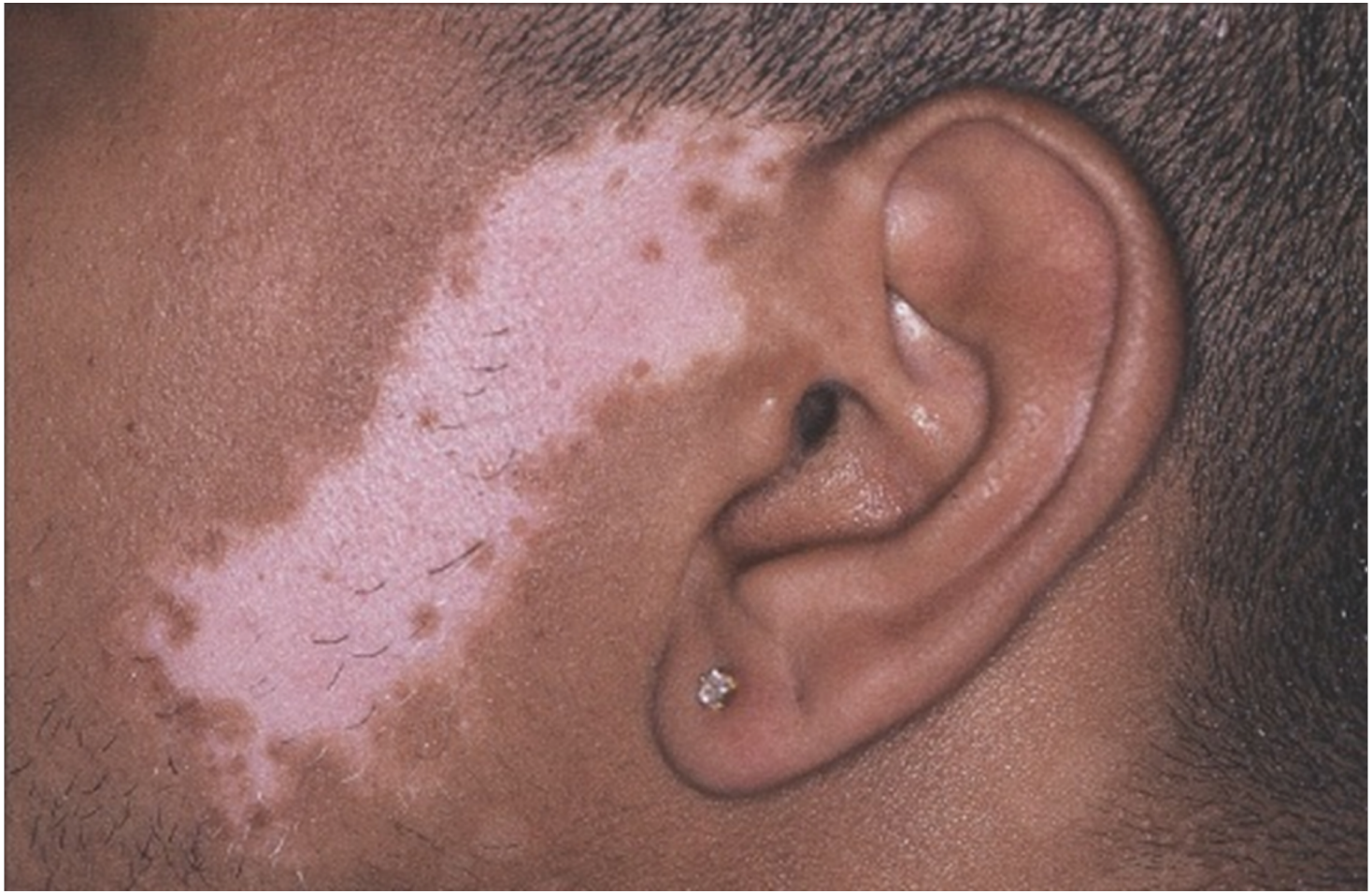
-Autoimmune

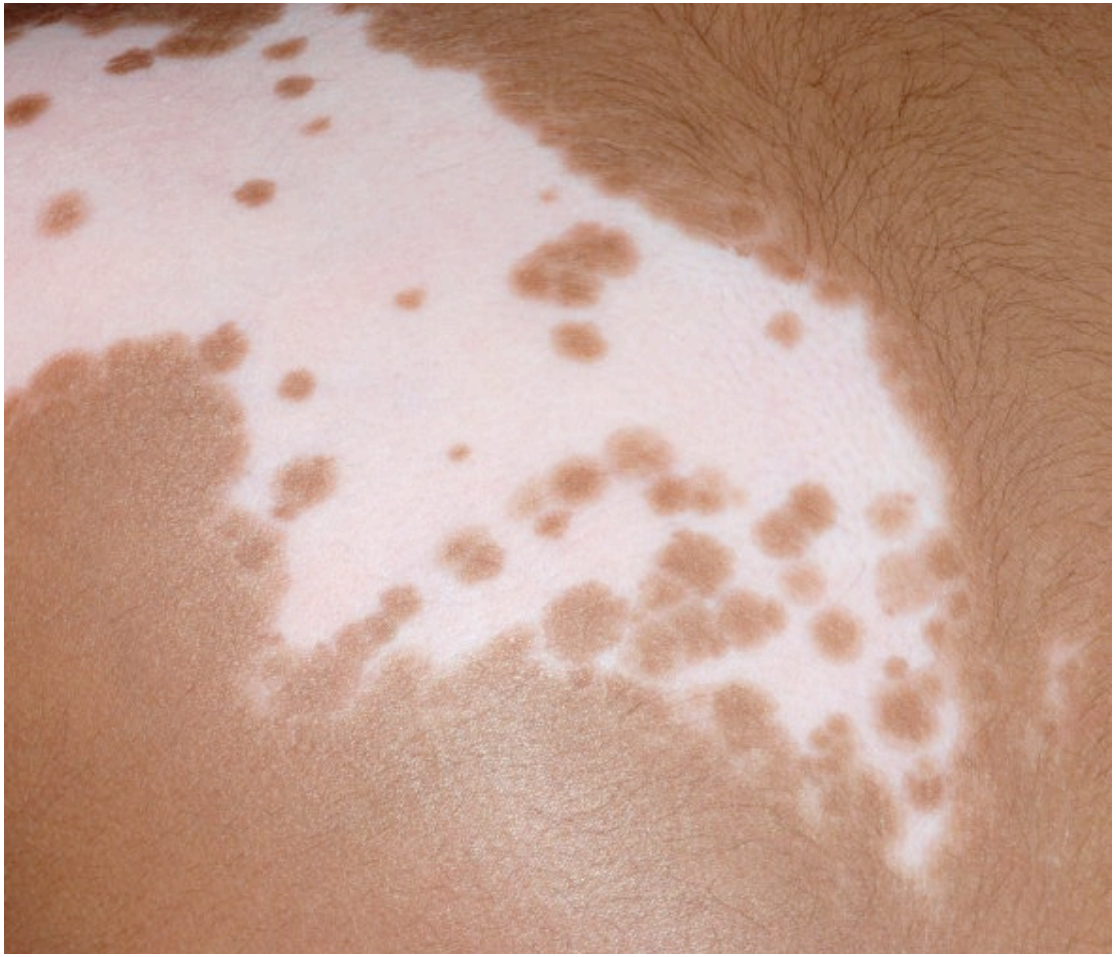
Clinical picture

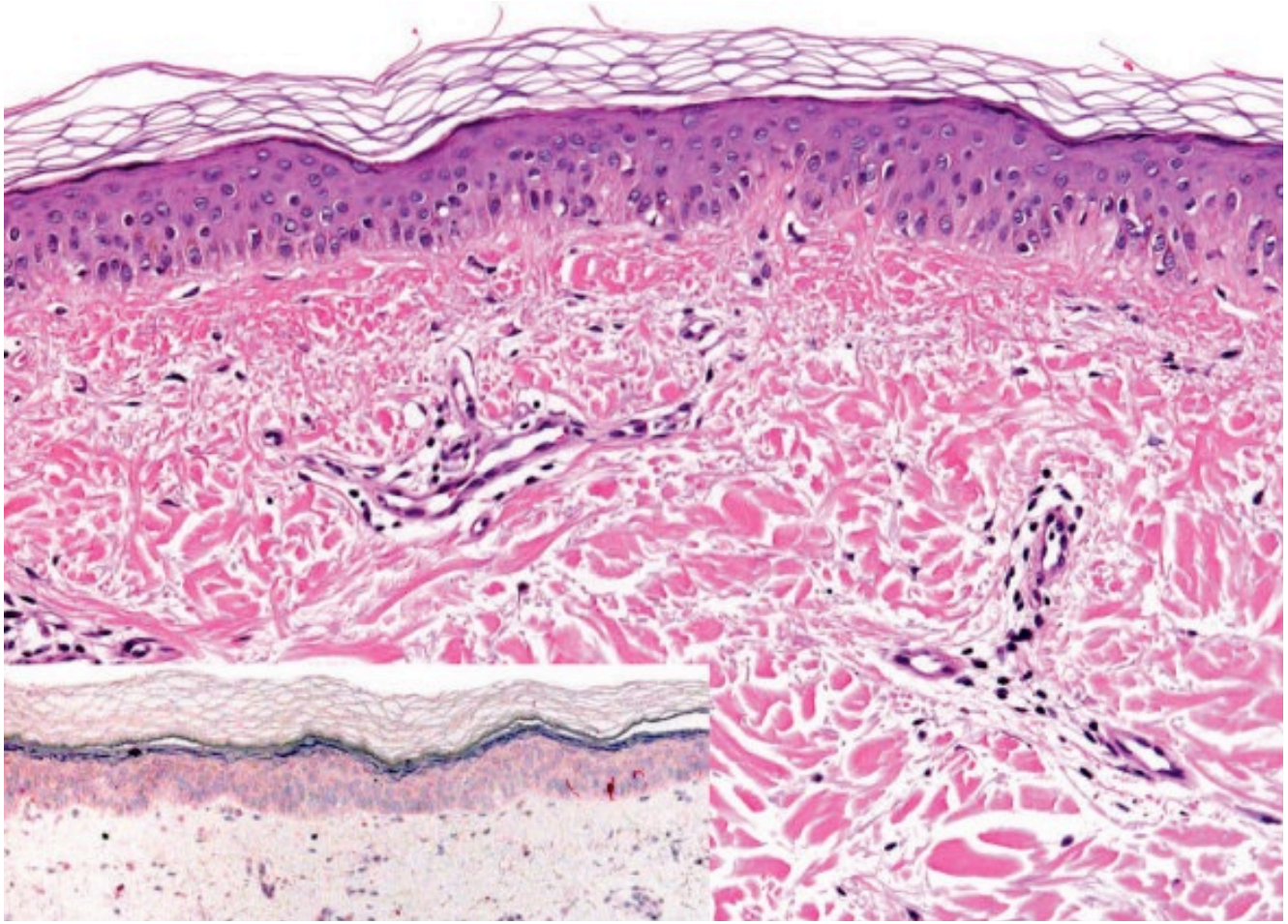
- The most common presentation of vitiligo is totally amelanotic (milk or chalk-white) macules or patches surrounded by normal skin

- koebner phenomena


DISTRIBUTION PATTERN OF AMELANOTIC SKIN LESIONS IN VITILIGO
Focal
Unilateral/Segmental
Vulgaris
Universal







Diagnosis

-clinical

-R/o associated autoimmune disease

CBC , T4, TSH, FBS

Treatment

Localized vitiligo :

Topical steroid

Topical calcineurin inhibitors

Topical PUVA

Excimer laser

Resistant, Stable of 2 years : Surgical

Generalized :

Phototherapy

Bleaching agent (Monobenzyl ether of hydroquinone)

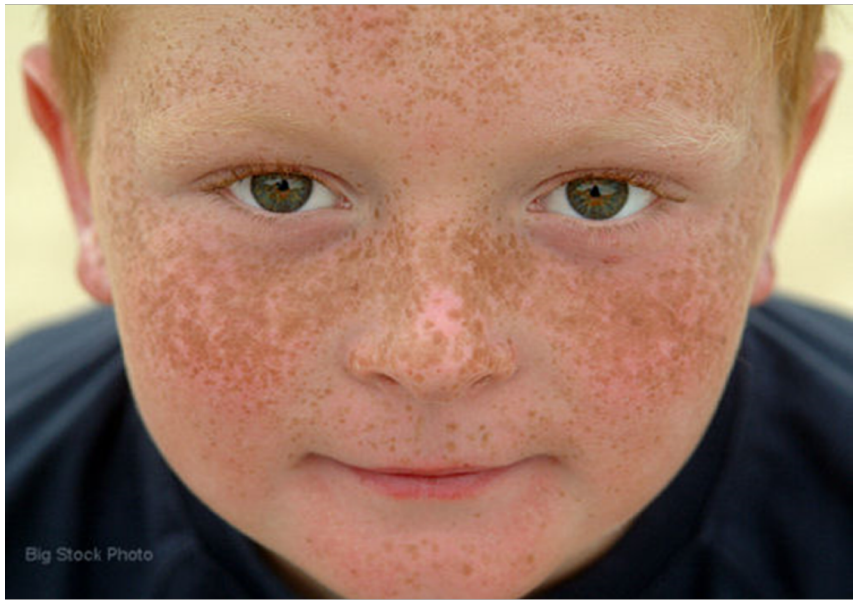
Freckle (EPHELIDES)

- 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

- Acquired MN : very common, small, uniform, no need for treatment except ABCDE

(Asymmetry , irregular borders, non-homogenous color , diameter more than 5 mm , evolution)

- Congenital MN: variable size could be Giant CMN (Bathing trunk) could harbor “Malignant melanoma”

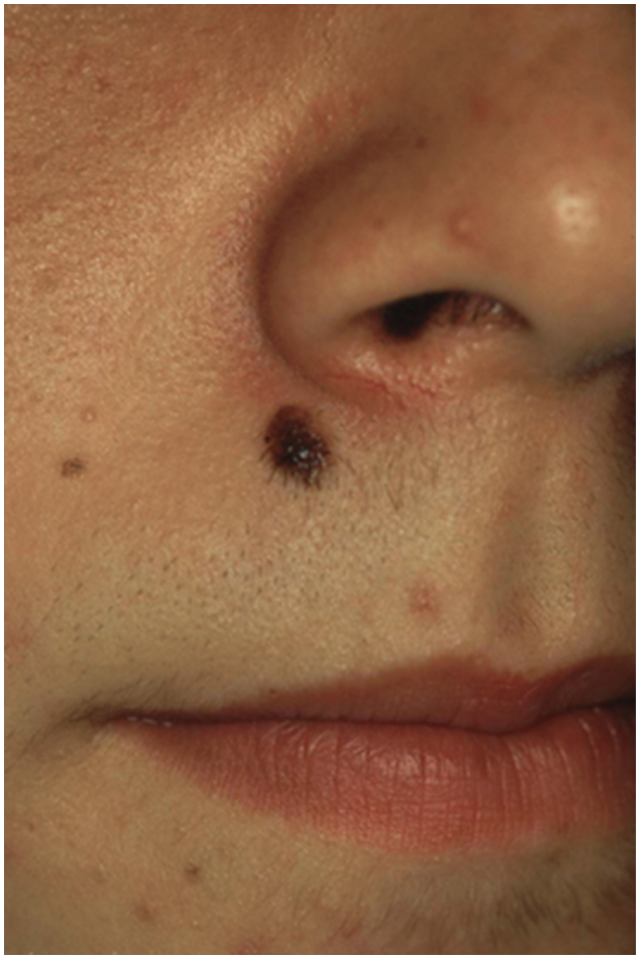
- Atypical nevus (dysplastic): larger with one or more atypical signs “4 or more: risk of malignant melanoma in the subject”.

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

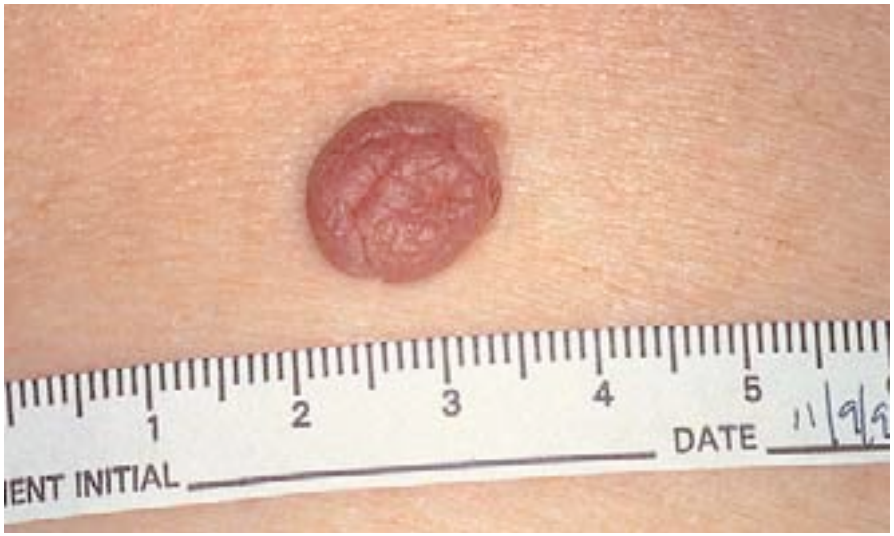
- Halo nevus: compound nevus with halo of depigmentation (association with vitiligo , atypia, melanoma)

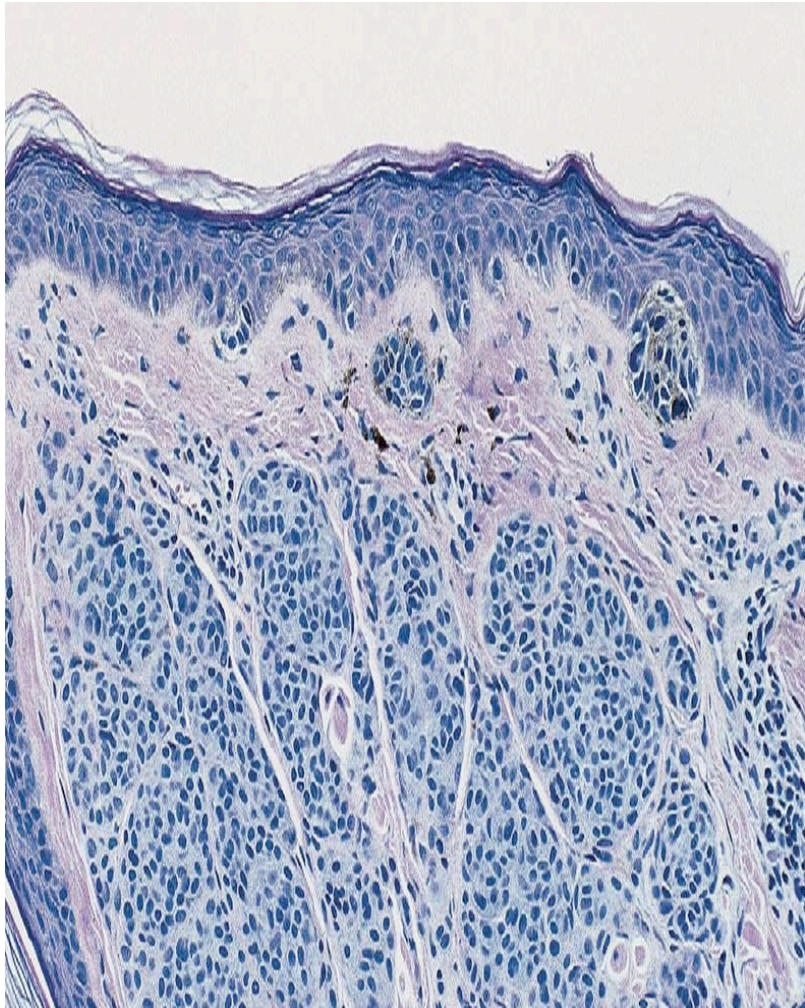
- Spitz nevus: common on children face with pink or pale brown color , common in children

Acquired Melanocytic nevus



Acquired Melanocytic nevus





Dysplastic nevus

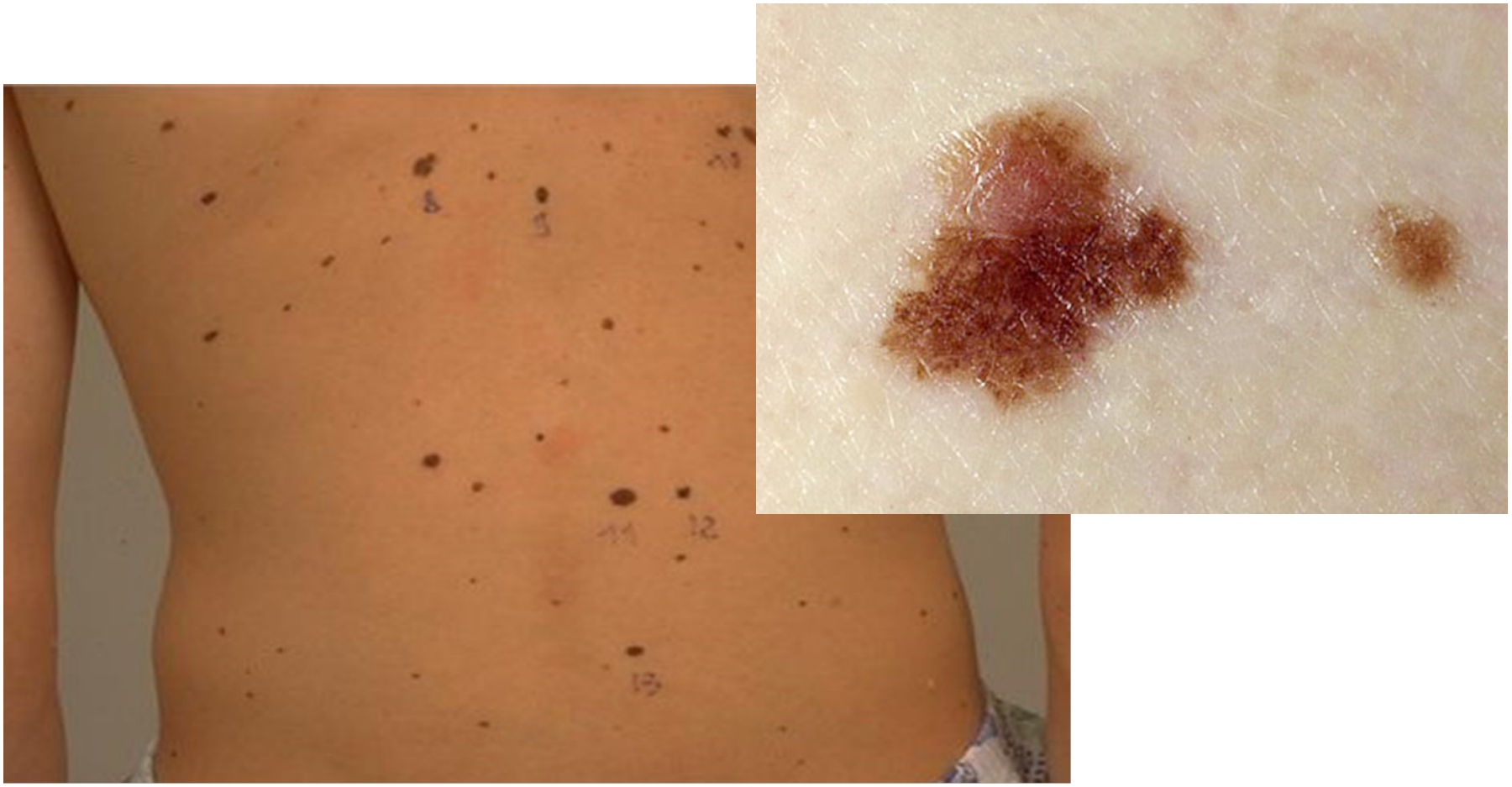
- controversial clinical designation for various nevi that have morphologic changes such as asymmetry, irregular borders and color variation (ABCDE role)

- Also a controversial pathologic term used for nevi with certain architectural changes and/or cytologic atypia

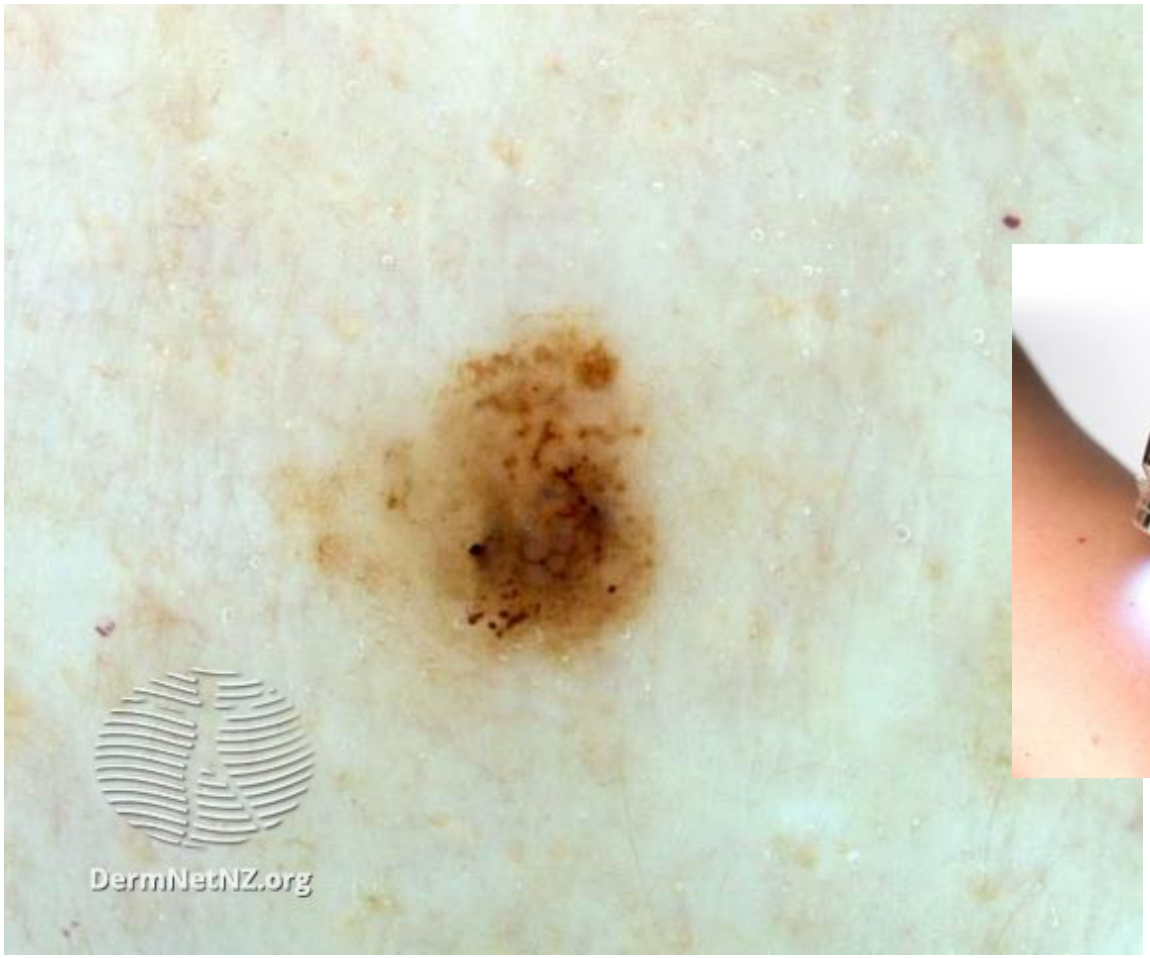
- The relationship to melanoma is complex

- Examine with dermoscopy and biopsy all suspicious lesions

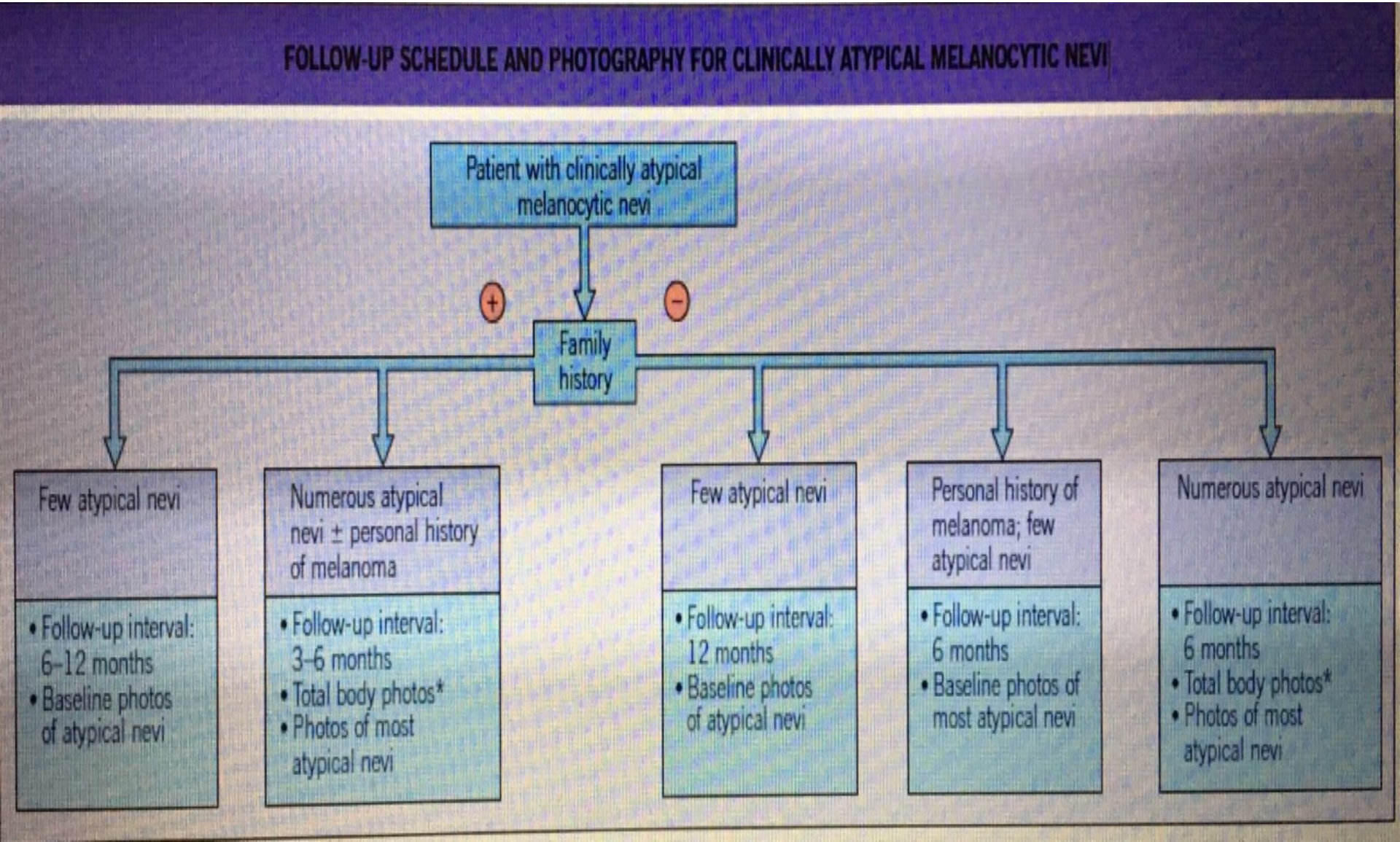
Dysplastic nevus



Dysplastic nevus



Dysplastic nevus



Congenital melanocytic nevus

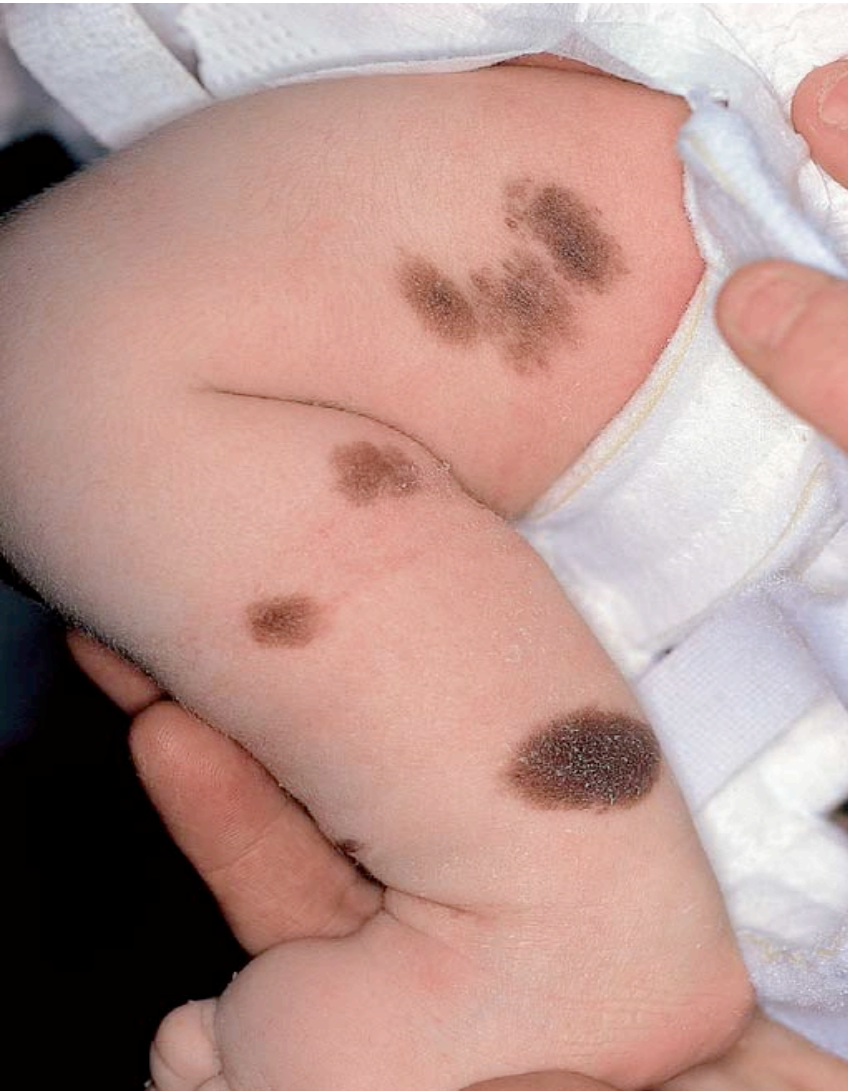
- Small congenital nevi are less than 1.5 cm in diameter

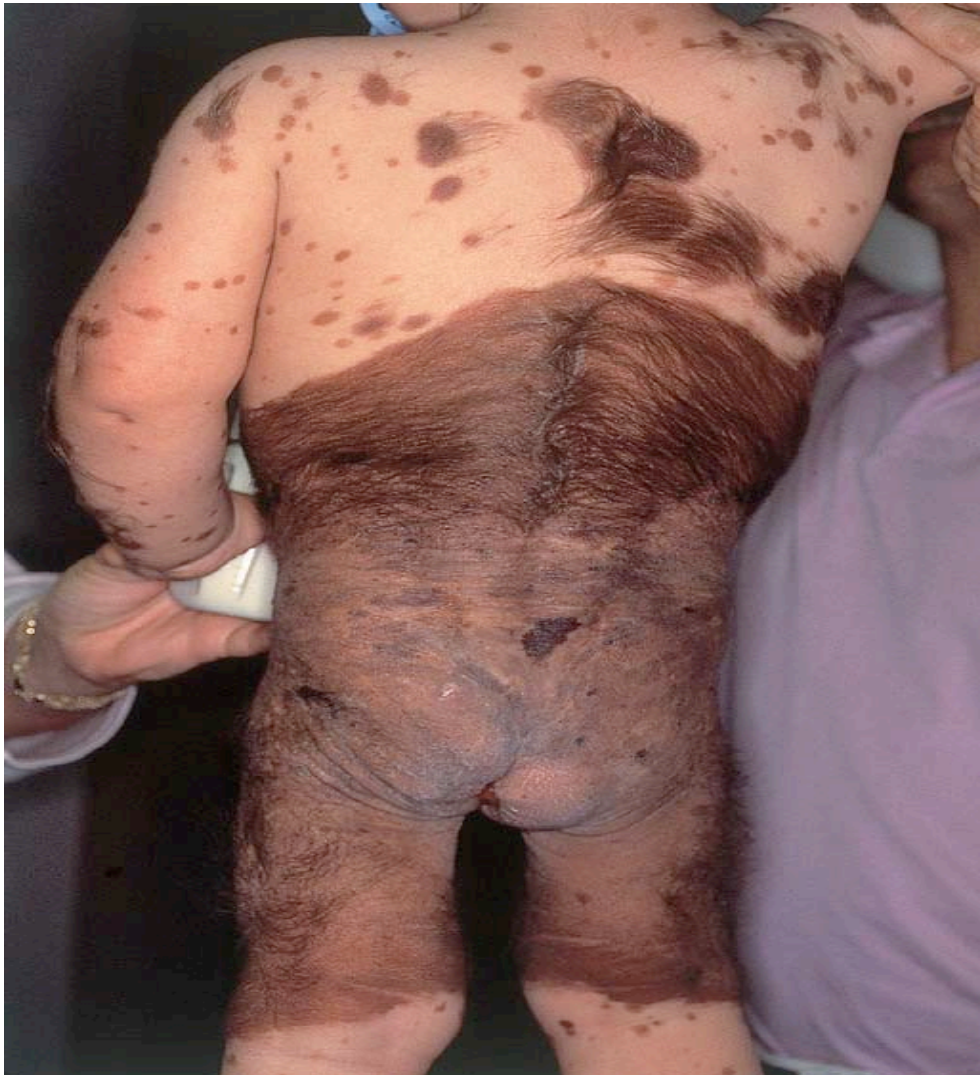
-Medium congenital nevi are between 1.5 and 19.9 cm in diameter

- 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 ,

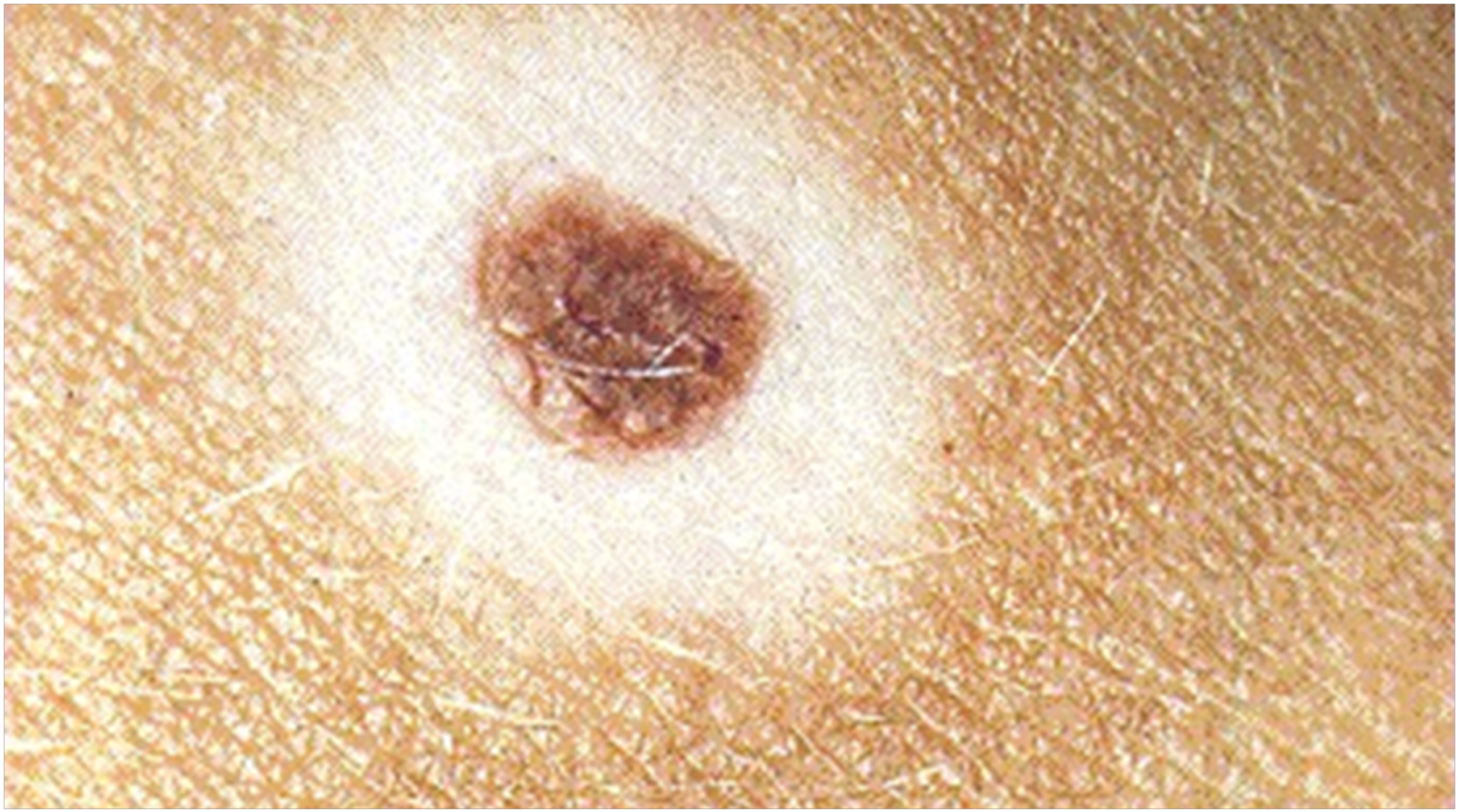
- Consider neuro-cutaneous melanosis in bathing trunk distribution

Congenital melanocytic nevus

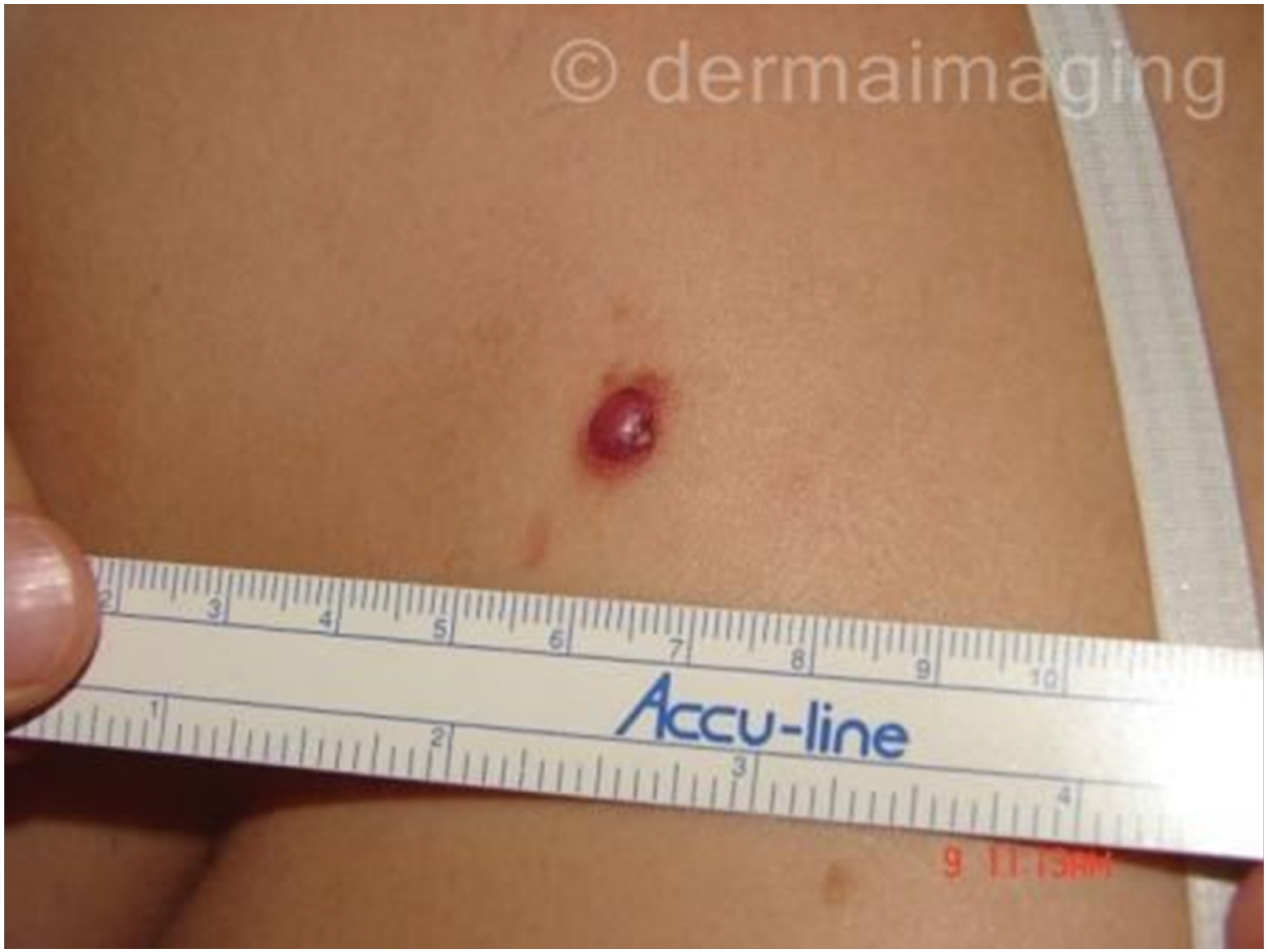




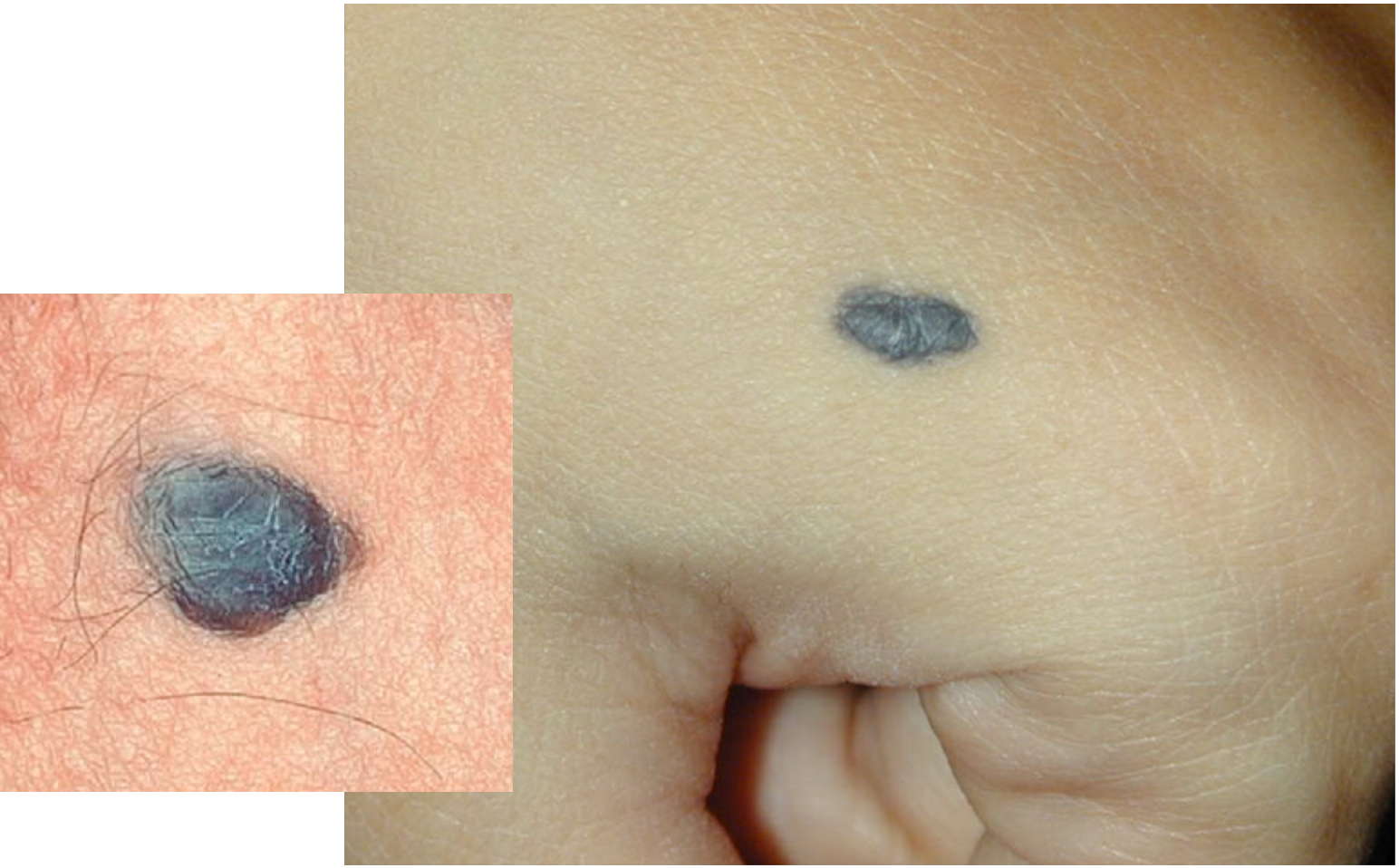
Halo nevus



Spitz nevus



Blue nevus



Melasma

- Genetically programmed increase in melanogenesis affecting the Face

- Could be induced by Pregnancy, OCP and excessive Sun exposure

- Treatment: sun block & bleaching cream