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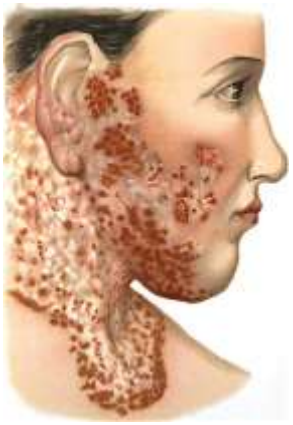
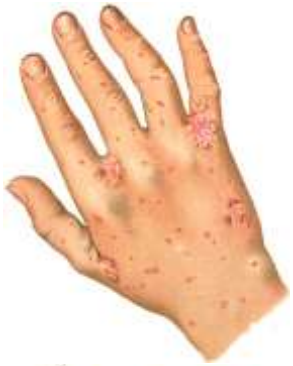
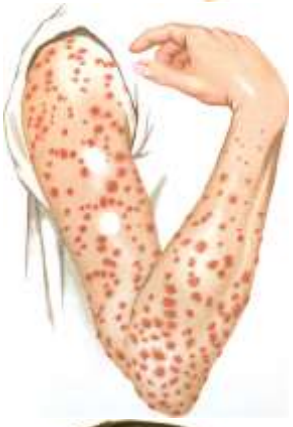
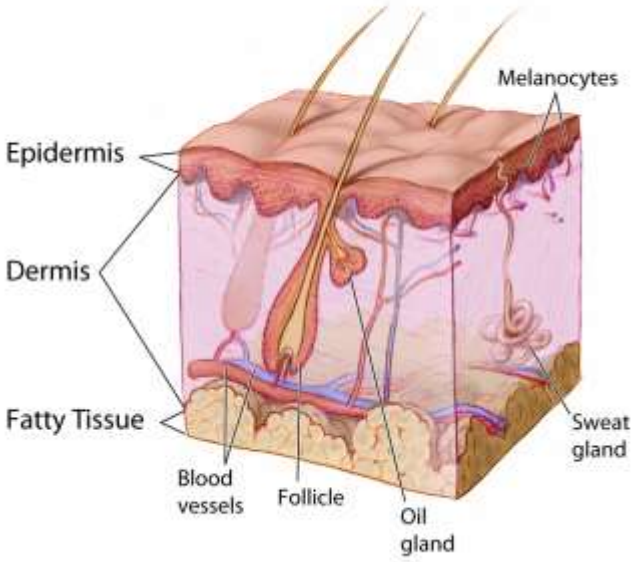






# 432 Teams
# Dermatology




## *Pigmentary Disorders*

***Color Code: Original, Team's note, Important, Doctor's note, Not important, Old teamwork***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*Reviewer:* ***Lama AlTawil***

*Team Leader:* ***Basil Al Suwaine and Lama AlTawil***

# Objectives

1. Pathogenesis, features and management of different pigmentary disorders including:
    - Freckle
    - Different types of Melanocytic naevi
    - Melasma
    - Vitiligo

## *Freckle (Lentigo)*

**1-Overactivity** of an increased2- **number of melanocytes** (number more than activity)

- Fair individuals (white people)
- Affect sun exposed area (face, forearm)
- Sun exposure in genetically predisposed Individuals
- Sun sensitivity "they get burnt easily"

**Treatment:**

Sun block "to minimize the risks of pigmentation and sun burning"

bleaching creams (not that effective) and pigmented laser (high tendency of recurrence) mainly if it is very deep >> chemical peel

## *Melanocytic Naevi (Mole)*

Common.

Concern: early malignant melanoma.

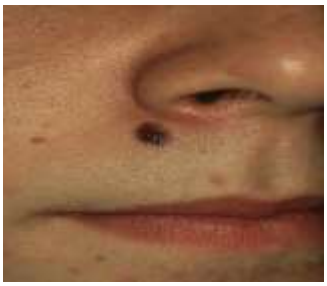
**ABCD: "features of MM"**

1-**A**ssymetery
2-irregular **B**order
3-irregular **C**olor
4-**D**imeter more the **6mm**
5-bloody and pinful

The chance of conversion to **malignant melanoma** (killer)

most common site for malignant melanoma is acral

**Acquired MN: commonest**

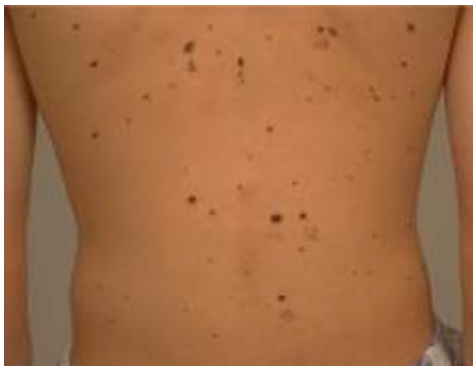


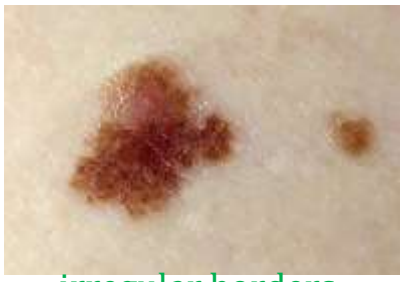
very common, small, uniform, no need for treatment except ABCD (Change in size shape, edge, color)

- It is better to leave the mole untreated so if there is a sign of melanoma, we detect it so avoid pigment laser removal
- Best treatment option: ablative laser "remove it as a whole" or surgical (resurfacing) .
- If the mole has a hair shaft, it should be cut only "not removed" to minimize any risk of inflammation
- Moles usually increase in number, With **aging and estrogen they become more prominent**

**Atypical naevi (dysplastic, premalignant):**

larger with one or more atypical signs (ABCD); risk of malignant melanoma in the subject. " Needs closer observation"





irregular borders

Dysplastic naevus syndrome, Multiple dysplastic naevi
These patients must be seen **every 6 months**.
Whenever in doubt, we take biopsy to rule out MM

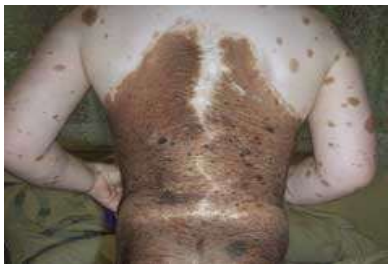
**Congenital MN:**

variable size could be:

- Small.
- Medium.
- Large "Giant CMN" >20 cm "up to 20% risk of MM" so these patients need very close observation and examination annually

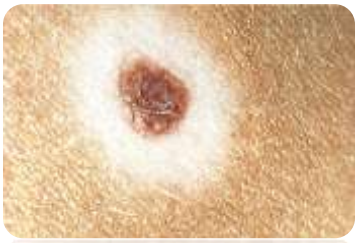
Sometimes it covers the whole body and these patients may present with multiple malignant melanoma.

(Bathing trunk) could harbor "Malignant melanoma"

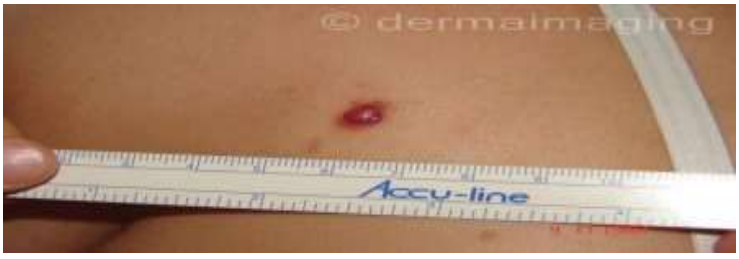


**Blue naevi** : deep-blue color and common on face, hand or feet.

**Halo naevi**: compound naevi with halo of depigmentation.

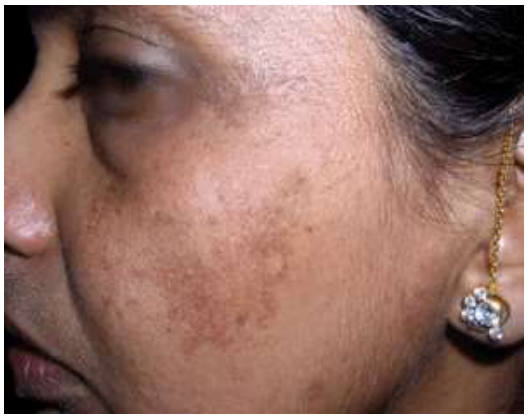


**Spitz naevi**: common on **children** face with pink or pale brown color and in adult carry the risk of transformation to malignant melanoma



## *Melasma (chloasma)*

- Genetically programmed, hormonally stimulated increase in **melanogenesis**
- (increase in activity more than number) Hyperpegmintation
- Affecting the Face Mainly in the lateral cheeks but may be very extensive covering the forehead
- Could be induced by Pregnancy "mask of pregnancy", OCP and excessive Sun exposure
- Affects darkly pigmented skin "in contrast to freckle"



**Treatment**: sun block & bleaching "Creams, chemical peel or laser"

Melasma could be at two levels:

- Epidermis: treated with any of the above
- Dermis: severe, so should be treated with deep chemical or fraxel laser

To differentiate between melasma and skin demarcation:
Skin demarcation line occurs in teens as it is hormonally induced and it has a very clear shape and sharp line. 1 or 2 triangle variant .usually we give sunblock

Remember:
Increase in **number** more than activity of melanocyte → lentigo
Increase in **activity** more than number of melanocyte → chloasma

## *Vitiligo*

- Low pigments >> hypopigmentation Loss of pigments >> depigmentation
- Acquired cutaneous depigmentation "chocky white" (loss of melanocyte)
- Kobner phenomena: Trauma induced dermatological lesions and it is mainly seen in Psoriasis, warts, vitiligo, lichen planus. mcq
- More clear when sun exposure or tanned

**Causes:** "multifactorial"

1. Genetic→ not mendelian "2-3 times Higher risk than normal population, but not a must"
2. Autoimmune disease (the most prominent).
3. Neural "Segmental vitiligo, following dermatomes"
4. Cytotoxicity.

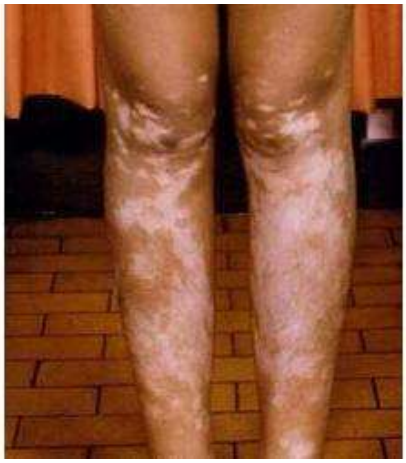
**Natural course**

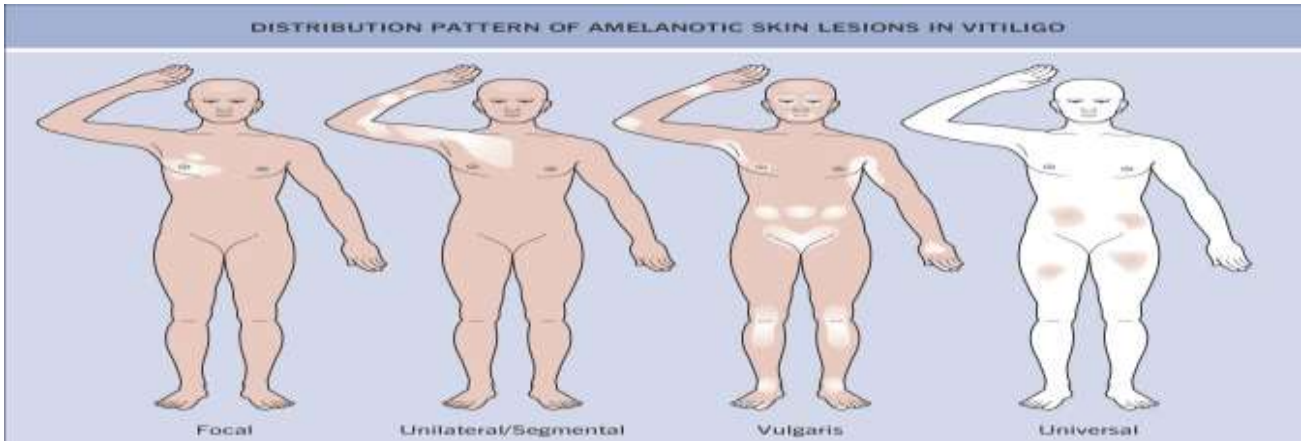
Variable "Some patients present with one patch, treated and disappear
Others present with one patch, never get treated" it can be controlled but not cured

- In case of vitiligo always ask about previous inflammation;

Post inflammatory, patients may have hyper, hypo or depigmentation(e.g.deep burn , chemical)

- DDX: Depigmentation in case of chemicals.

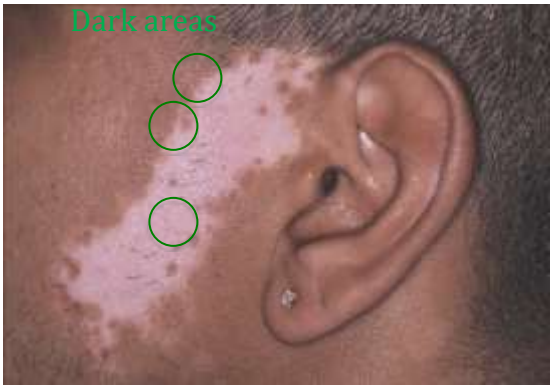





- Vulgaris mainly affects areas around the eyes and orifices, elbows and knees "lip tip trauma" it's the most common
- Universal affects more than 90% of the body surface area.
- Treatment:
  - Focal and segmental: topical treatment or laser.
  - Vulgaris: if less than 15%-20% topical, more phototherapy (narrow band UVB or less commonly PUVA(ADR on liver ).

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

The nature of repigmentation is classified into 3 types:




1. From periphery to the center "marginal"
2. The color faints gradually "diffuse"
3. Perifollicular repigmentation: "the commonest"

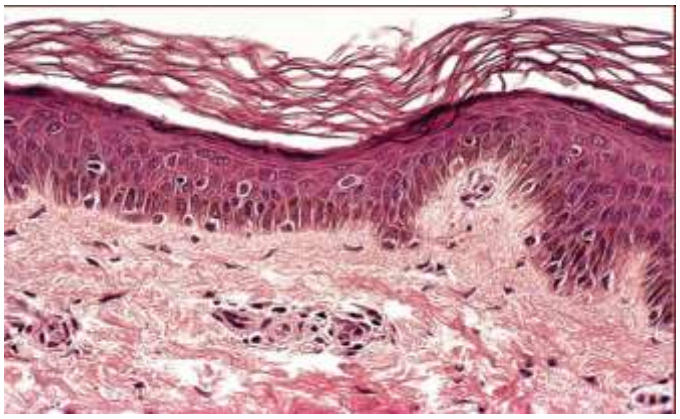
Pigments in the hair follicle go from the hair bulb to the dermis and skin starts to regain its pigmentation.

Pigmentations start as perifollecular and then it expands and covers the whole area. thats why laser hair removal wont regain pigment easier as when hair preserved

**Workup:**

Biopsy with dopa stain →is specific for the melanocyte

Shows loss of normal melanoctyes. At the dermoepidermal junction



T4, TSH, FBS "for other autoimmune diseases"

Look for other systemic disorders like DM

ANA/Ro/La (prior to PUVA) "to rule out photosensitivity" to avoid getting burnt

## Treatment

Only control the disease, no cure

Sunscreens to avoid sunburn and koebnerization

Avoid tanning "the contrast will be worse"

**1-Limited:**

- Class 3 topical corticosteroids "drug of choice"
- Topical Tacrolimus "immunomodulators, less effective but safer, so it is used on the face" no steroid ADR
- Topical PUVA
- Excimer laser

Resistant but Stable of 2 years Surgical treatment:

The disease didn't progress and the patient didn't develop new vitiligo even after trauma. If not stable you will depigment the area you took the graft from

1. Melanocyte Transplant (only in fix inactive vitiligo)
2. Cosmetic Tattoo

NBUVB:
Narrowband UVB

PUVA:
UVA + Psoralen

Psoralen: drug increasing skin sensitivity to ultraviolet light

**Generalized:** more than 20%

- Phototherapy (NBUVB, PUVA)+ topical

Start with NBUVB, if no response >> PUVA, but make sure you rule out photosensitivity first

**Universal**

- Bleaching agent: Depigment all skin by **Benoquin** (Used as final solution when the vitiligo is **more than 50% of the skin**)→ may repigment patches when exposed to sun or on steroid due to renal failure therapy

## Summary

- **Freckle:**
  - Overactivity of an increased **number of melanocytes** (number more than activity).
  - Treatment: Sun block, bleaching cream and pigmented laser (recurrence)
- **Melanocytic naevi (mole):**
  - **Acquired MN**: very common, small, uniform, no need for treatment except **ABCD**.
  - **Congenital MN**: variable size could be Giant CMN (Bathing trunk) could harbor "Malignant melanoma" Higher risk of developing malignant melanoma than the Acquired MN.
- **Melasma (chloasma):**
  - Genetically programmed increase in **melanogenesis** (increase in activity more than number) Affecting the Face Could be induced by Pregnancy, OCP and excessive Sun exposure
- **Vitiligo:**
  - Acquired depigmentation (loss of melanocyte) "Kobner phenomena": dermatologic disease occur in the site of the truma could be (vitiligo, psoriasis, eczema , wart , lichen planus ).

# Questions:

1) Thirty years old patient presented with multiple bilateral symmetrical depigmented patches over Face, trunk and extremities for 3 months. What is the melanocyte pathology?

A. decrease activity
B. Increase activity
C. Increase in melanocyte number
D. Absence of melanocytes

2) A 50 year---old male who had vitiligo more than 30 years. His vitiligo involving more than 97% of His body. What is the best treatment option for this patient?

A. Topical steroids.
B. systemic steroids.
C. Melanocyte transplant
D. Depigmentation.

3) A 6 year---old girl presented with bilateral white patches. In case it is vitiligo, what you see under Wood'slamp?

A. A color whiter than normal skin
B. A color darker than normal skin
C. Similar color to normal skin
D. A Golden green color

**1-D**
**2-D**
**3-A**