

DISORDERS OF HYPERMELANOSIS

Melasma
Freckles
Lentigenes

Etiology of Hypermelanosis

Genetically determined

i.e. ↑ no. of melanocytes in skin eg. naevi

Acquired

i.e. ↑ melanogenesis

triggered by UVR

Hormones

Chemicals

Others

- Vit. A deficiency
- Chronic infections
- Anaemia
- Malnutrition/pellagra

Systemic disorders

Drugs

- Phenothiazine
- Chlorpromazine
- Arsenic

MELASMA

More common in female patient

Appears during pregnancy

Patients on OCP

Males also affected

Clinically

- Light to dark brown pigmentation
- Upper lip, cheeks, chin, forehead
brown macular pigmentation with well
defined margins occurring symmetrically
- Pigmentation increases on sun
exposure

Treatment

- Photoprotection

Hydroquinones

- retinoids
- Topical corticosteroids
- Alpha hydroxy acid
- Azaleac acid

TANNING

Increased melanin pigmentation of human
skin following sun exposure

Immediate Pigment Darkening

UVA light can be induced within few minutes

- Maximum within 1-2 hours
- Slowly decreases between 3-24 hours

Delayed Pigment Darkening

Occurs after 48 – 72 hours of exposure

Clinical types of hypermelanosis

- Freckles
- Lentigenes
- Post inflammatory

Ephelides (Freckles)

Autosomal dominant

Pathology - No increase in no. of
melanocytes pigmented
layer but their melanosomes
are long /rod shaped like
those in dark skinned people

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Clinically

Appear at about 5 years of age

Light brown, pigmented macules on
light exposed skin

Increased in depth of pigmentation
during summer/sunlight

Treatment

- Depigmenting agents
- Sunscreens



Difference in ethnic groups

Amt. & arrangement of
melanosomes in melanocytes

Lentigenes

- Benign pigmented macules
(darker/larger than freckles)
- Different sites

Post inflammatory

After acute/chronic infection

Following trauma

Sun exposure

Even preceding illness may be absent

Skin conditions responsible:

- Eczema
- Lichen planus, amyloidosis
- Tumours





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