Disorders of skin colour

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Normal skin colour depends on :

- Melanin
- Oxygenated and reduced Hb
- Carotenoid substances

Melanin :

Melanocytes : Dentritic pigment producer cells in the epidermis and dermis. **two types:**

- Secretory melanocytes
- non secretory M. (Melanophor)

Skin colour disorders :

- Hypomelanosis .
- Hyper melanosis .

Causes of Hypomelanosis :

A- Gentically predisposed :

1- Piebaldism : A.D. inhereted disorder in which there is a complete lack of melanocytes probably due to a defect in the migration or differentiation of melanoblast to melanocytes and presented since birth as a depigmented patch in the frontal , median, paramedian areas with a mesh of white hairs **(Fore look) .**

2- Albinism: A.R. due to a defect in synthesis of tyrosinase

↓ melanin production :

Child borns with white hair , red skin and iris with R. Nystagmus , photophobia .

Two types : Partial or complete

→ T. +ve : Pt. gain some pigment with age so hair become golden yellow , with Light brown colour skin that Freckles with age .

T- ve: No change with age .

→

B- Acquired Hypomelanosis :

1- Autoimmune disorders as in vitiligo .

2- Endocrinal causes as in Hypothyrodism , hypopituiturism .

3- Nutritional factors as in chronic protein deficiency and pernicious aneamia .

4- Post inflammatory hypopig . Which is either due to .

A- Damage of melanocytes by inf. Procese as in L.P. and L.E.

B- Failure of melanosomes transfers from melanocytes to the k. cytes due to rapid turn over as in psoriasis and eczema .

5- Physical agents :

- → Thermal burn .
- → Lonizing radiation .
- → Chemicals → Phenols .

6- Malignant → Halo nevus .

7- Chemical → latrogenic

8- Miscellaneous → idiopathic guttate hypomelanosis .

Vitiligo :

common skin dis , characterized by depigmented macular and patchy skin lesion , affecting 1% of the population .

Histopath :

Shows absence of secretory or functioning molanocytes .

Aetilogy : Many theories are suggested for the aetilogy of vitiligo.

1-30-40% of the pts have +ve f. history . In Irag: FH +ve 80-90% .

2- Auto immune theory .

- Association with A.I. disorder .
- Presence of organic specific Abs.

3- Neurogenic theory .

4- Melanocyte self distraction theory . Melanin precursors as phenol. In the presence of defective natural protective mechanism .

5- Mixed theory .

Immunological mediated dis. In genetically predisposed person .

Emotional Factor in vitiligo .

Clinical features :

- **Age :** Any age but 50% before 20 yrs.
- Onset : Is gradual and course is progressive .
- Males and females are equally affected .

Sites : → Around body orifices

→ Sun exposed areas.

→ Normally hyper pigmented areas like genitalia , areola , groins and axillae .

→ sites subjected to trauma and frictions as knees , elbows , back of the hands and fingers .

Lesions : Asymptomatic bilateral symmetrical white de pigmented macules and patches with convex out lines and hyper pigmented or normal edges, not scaly.

Normally sweating slowly progressing to fuse with other patches .

Hair → Normal pigment .

→ Become white in chronic lesions .

Clinical Variants :

- Ordinary
- Linear → koebner ph.
- Prematurs greying of the hair .
- Extensive vitiligo with small normal areas . Universal
- Segmental .
- Halonevus .
- Ocular
- Chemical iatrogenic

DDx → **piebaldism .**

→ post inf. Hypomlanosis

→ pit. alba.

→ T. V.

→ leprosy .

Treatment :

Rx: Is unsatisfactory :

1- Systemic psoralens : In adose of 0.6 mg/kg body wt. with exposure to sun light 2hrs later for increasing time till faint erythema is acheieved → exposure time fixed .

2- Topical :

A- Psoralens : PUVA ¼ hr and expose to UVL as for systemic .

plant extract , photo sensitizer.

→ Side effects of psoralens :

mode of action .

B- potent steroid :

1- If lesions are localized to :

A- Critical sites around the eye. And M.C. junct.

B- Flexural areas , Face .

2- With systemic psoralens .

Intra lesional steroid for localized or resistant lesion.

C-Depigment the residual pigment in extensive vitiligo by bleaching agents .

Signs of response

Continue Rx for 6/12 .

80% response .

Prognosis : → Spont resolution in 10-20% of cases.

→ 60-70% repipment with Rx .

→ After stopping the Rx .

Bad prognostic points :

→ Longstanding → M. cut areas .

→ Hair less areas . → Extensive type .

→ Of the hair → premature greying .

→ Linear type → with A.I. disease .

Causes of hyper melanosis :

A- gentically predisposed :

1- Freckles : Brown macules on sun exposed areas , increase in pigment , No. and size in summer times .

2- Lentigo : Brownish macule at both covered and uncovered areas, not change at different times .

B- Acquired :

1- Due to physical agents :

- Photo toxic and allergic
- Post irridations .
- Post traumatic .

2- Endocrine :

A- Pregnancy , Menstruation , contraceptive pills → **Melasma**

Blotchy facial hyperpigmention . **(chloasma) .**

Treatment

B- Addison’s disease .

C- Hyperthyroidisn , Hyperpit .

3- Systemic diseases . Chronic inf. , malignances , chr. Renal diseases.

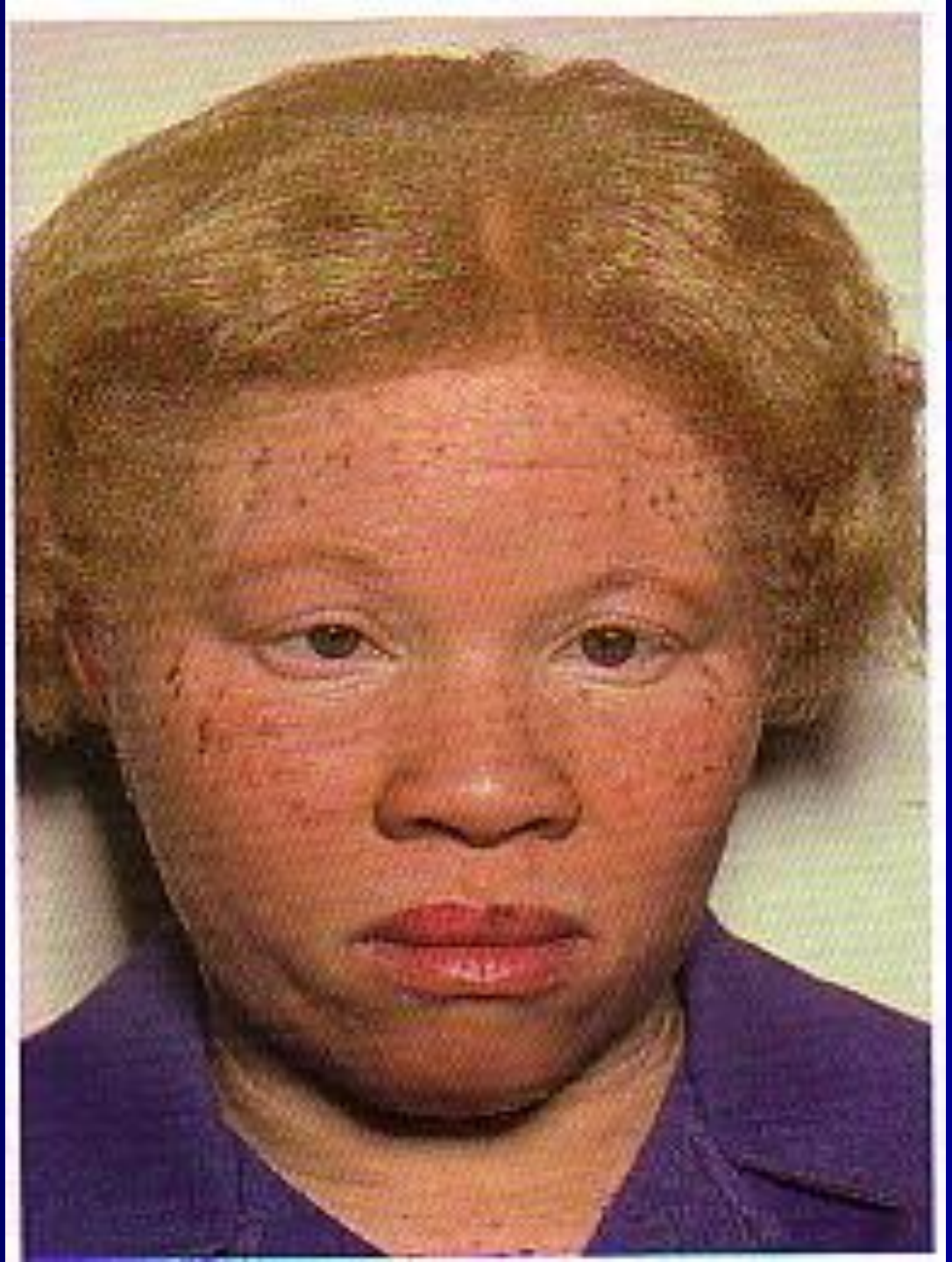
4- Drug induced : (psoralens) Chlorpromazine , phentoin .

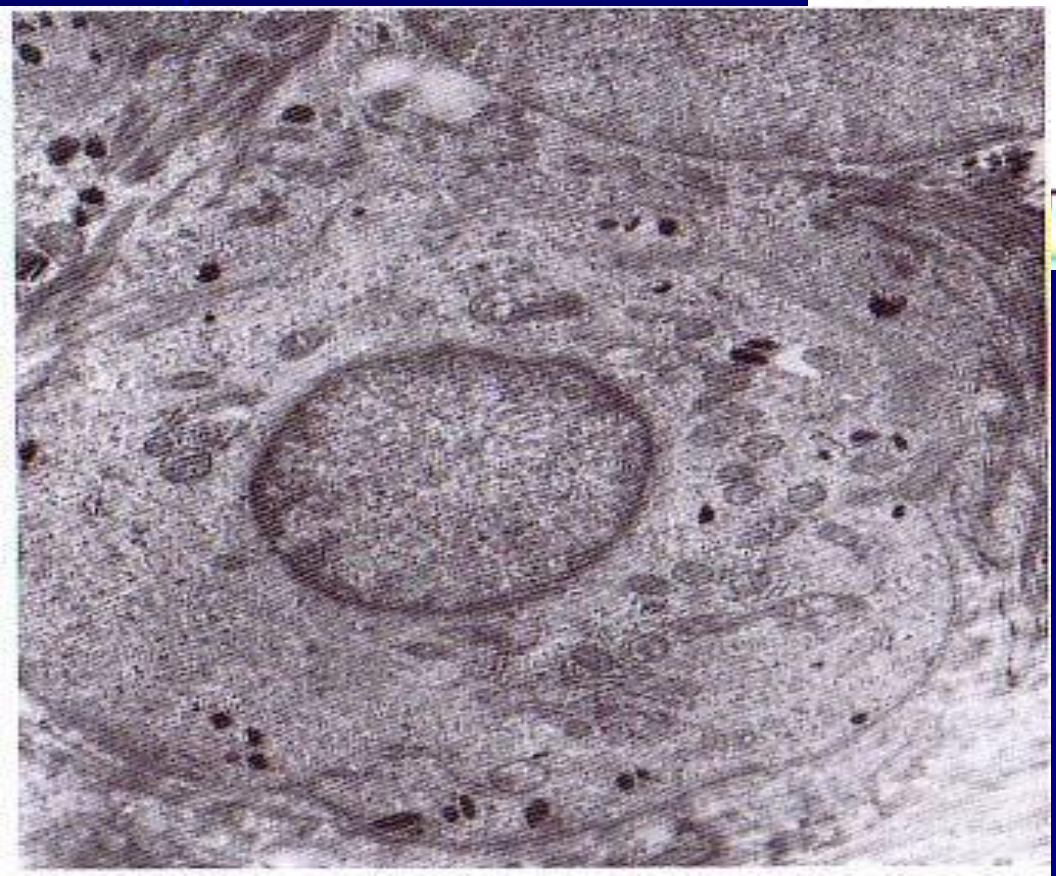
5- Post inf. Hyperpigmentation .

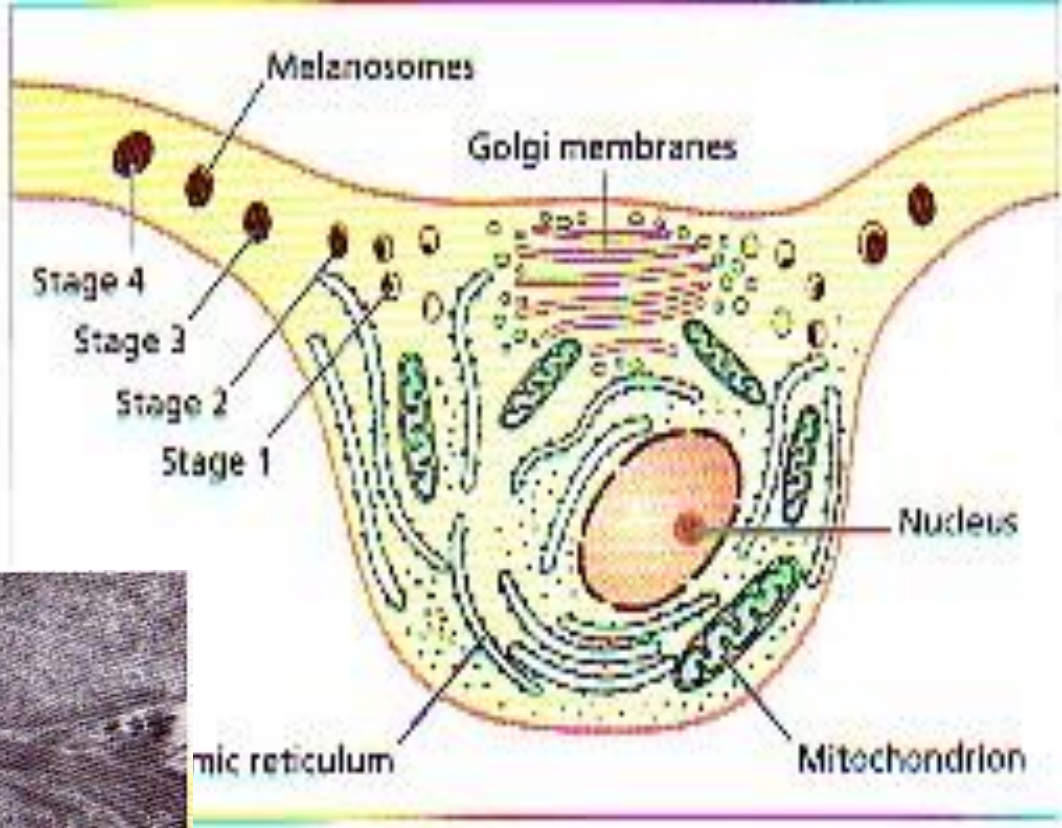
6- Nutritional → Pellagra and vit diff.

7- Tumours → M.M.


Thank you






Melanosomes
Golgi membranes
Stage 4
Stage 3
Stage 2
Stage 1
Nucleus
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Mitochondrion