Vitiligo

Acquired, primary, usually progressive melanocytopenia.

→ circumscribed achromic macules often associated ± leukotrichia & histologically by degeneration & disappearance of melanocyte

Etiology - unknown

Epidemiology: All races but highest India & Mexico

Incidence & risk factors -

→ prolonged consumption of diet poor in proteins & cupric minerals.

→ M:F ≈ 1:1

→ Mostly affects people ± skin Type III & IV

→ May start at any age.

→ Onset of VIL dermatomal type within 10 years.

→ B/L non-dermatomal
↳ 2nd - 4th decade

Hereditofamilial aspects:-

Genetic predisposition (+)

↓ AD ↓ AR ↓ polygenic

- No HLA Ag.
- Catalase gene & vitiligo
  VIT1 gene → 2p16
- Atopy - familial association

CIF :-

1) well defined depigmented macule + depigmented hair w/o △ in skin texture.

2) Sometimes, margin - hyperpigmented

3) Trichome vitiligo

4) Quadrichrome - marginal perifollicular hyperpigmentation characteristic of repigmenting vitiligo

5) Onset - asymptomatic

6) Site: Exposed area - dorsal surface of hands, elbow, feet, legs, knee, neck, face

Ext. surfaces of body such as pretibial region, sides of ankles, knees, elbows, skin overlying the digits, periorificial area, anogenital area.

⇒ Achromotrichia - diff. from other hypopigmented nevi due to poor repigmentation capacity

⇒ Koebner's phenomenon : V. vulgaris
Minor trauma → linear depigmented macule.

• Emotional trauma & repeated
↳ sudden onset & rapid e

↓

Vulcano type vitiligo

Classification

Vitiligo vulgaris
B/L macules of various size & symmetry
Acral pts of limbs
↳ Acrofacial or Acrosegmental
Entire body surface is achromatic & few normal or hyperpigmented
↓
Vitiligo universalis

Vitiligo pseudo segmental
Early age of onset
Stable course
⊖ of Koebner
Less response to PUVA

⇒ Vitiligo focalis or areata
Ages 1
⇒ Tigroid appearance.
scars dt destruction of pigment layers.

Associated skin:-
Cavities
Alopecia areata
Atopic eczema
psoriasis

seleroderma
LP
Lichen striatus
DLE
halo nevus
ichthyosis

Segel

Systemic :- PA

Addison, Graves, Thyper, hypo
Thyroiditis, Thyroiditis hyperpara
DM

Prognosis:

- lesions on so called resistant sites - ↓ bony prominences, non flashy area.
- white hair ↑ poor progn.
- long standing dz.

Ethiopathy:-

1) Immune hypo
2) Neural hypo
3) Autochemic self destruction
4) Composite
5) Melanocyte AF (I)
6) Anti oxidant def.

HP: Absence of melanin granules

Diff:- DDx:

B/L lesions - Piebaldism
Waardenburg's syndrome
Woolly
Ziprkowski
Naegelis

Segmental: Congenital nevus depigmentosus.

• Achromic lesion of TS
than cutaneous & neurologic
abnormality.

Management

General Aspects :-

1) Diet - Adequate protein
Vitamin B complex
Cu, Zn, Fe

2) Avoidance of trauma

3) Avoidance of soaps & detergents ~~&~~ phenolic
compound.

Medical :- NBUVB → 311 nm

Psoralen & UVA therapy.

↓

PUVA → 320 - 400 nm

PUVASOL : when sun → UVA.

Topical :- 1% vehicle < 6 sq cm²
usually 0.1% applied
weekly ~~to~~ 2-3 hours later
by solar UVA irradiation
for 30 - 60 seconds.

Oral :- 0.6 mg/kg/body wt
of TMP ingested ~~in~~ food
2 hours before sun exposure

Exposure time :- Prescribed UV dose
0.06 × Irradiance