

# Vitiligo

acquired, primary, usually  
progressive melanocytopenia.

- circumscribed achromic  
macules often associated  
w/ leukoecithria & histologically  
by degeneration & disappearance  
of melanocyte

Etiology - unknown

Epidemiology: All races but  
highest India & Mexico

Incidence & host factors -

- prolonged consumption of  
diet poor in protein  
& cupro minerals.

→ M:F  $\approx$  1:5

→ Mostly affects people w/  
skin type III & IV

→ May start at any age.

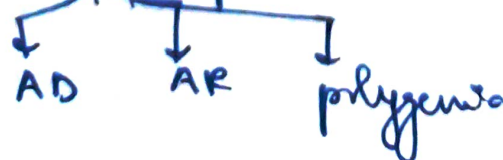
→ Onset of VIL dermatomal  
type within 10 years.

→ B/L non-dermatomal

↳ 2nd - 4th decade

Hereditary aspects:-

Genetic predisposition ⊕



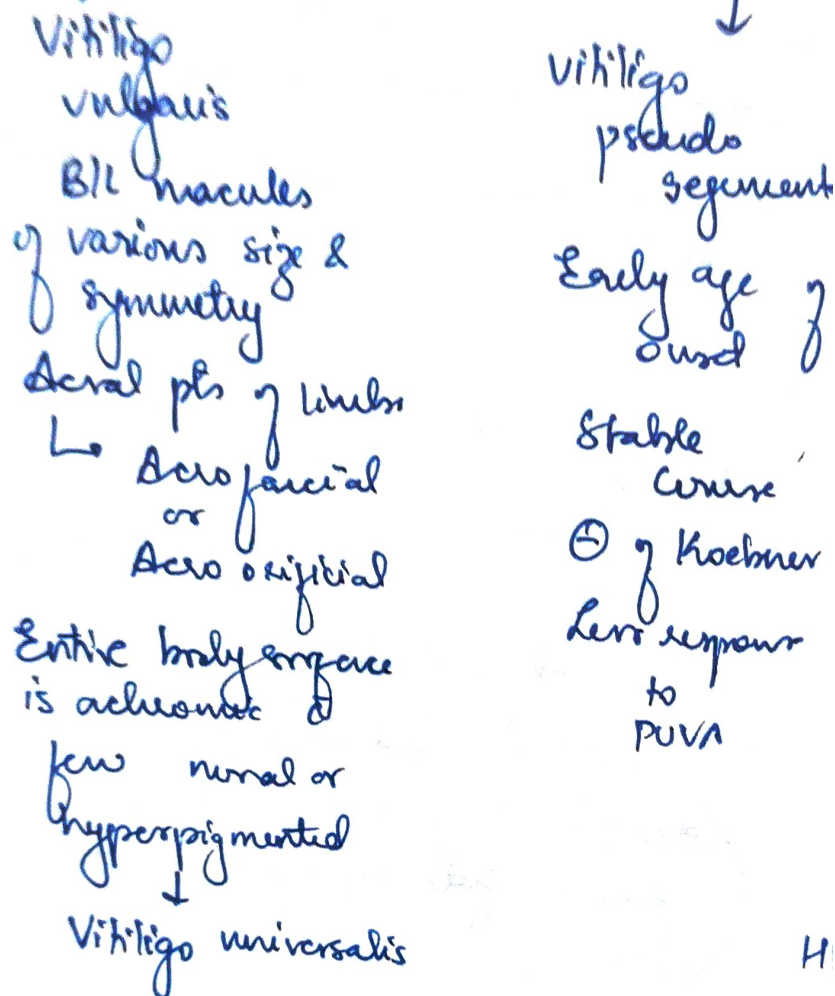
- No HLA Ag.
- Catalase gene & vitiligo  
VIT1 gene  $\rightarrow$  2p16
- Atopy - familial association

ClF:-

- 1) well defined ~~hyper~~ depigmented macule + depigmented hair w/o  $\Delta$  in skin texture.
  - 2) Sometimes, margin - hyperpigmented
  - 3) Trichome vitiligo 
  - 4) Quadrichrome - marginal perifollicular hyperpigmentation characteristic of repigmenting vitiligo
  - 5) Onset - insidious
  - 6) Site: Exposed area -  
dorsal surface of hands,  
elbows, feet, legs, knees, neck,  
face  
Ext. surfaces of body such  
as periorbital region, sides of ankles,  
knees, elbows, skin overlying the  
digits, periorificial areas,  
anogenital areas.
- $\Rightarrow$  Achromotrichia - diff. from  
other hypopigmented nevi  
w/ poor repigmentation capacity
- $\Rightarrow$  Koebner's phenomenon: V. vulgaris  
Minor trauma  $\rightarrow$  linear depigmented  
macule.

- Emotional trauma & repression  
 ↳ sudden onset & rapid e  
 ↓  
 vitiligo type vitiligo

## Classification



⇒ Vitiligo focalis or areata  
 Eyes  
 ⇒ Tigmoid appearance.  
 scars d/t destruction of pigment layers.

Associated skin:-

|                 |                |            |
|-----------------|----------------|------------|
| Canities        | Scleroderma    | Sebor      |
| Alopecia areata | LP             |            |
| Atopic eczema   | Lichen simplex |            |
| Psoriasis       | DLE            |            |
|                 | halo nevus     | ichthyosis |

Systemic:- PA

Addison's, Graves, Hyper, hypo  
thyroidism, thyroiditis, hyperparathyroidism

Prognosis:-

- lesions on so called resistant sites - & many prominences, non fleshy areas.
- dis • white hair ↑ poor progn.
- long standing dis.

Etiopathia:-

- 1) Immune hypo
- 2) Neural hypo
- 3) Auto toxic self destruction
- 4) Congenital
- 5) Melanocyte CF ⊕
- 6) Anti oxidant def.

HP: Absence of melanin granules

Dis:- Dis:

B/L lesions - Piebaldism

Waardenburg's syndrome

Woolf

Ziporkowski

Margolis

Segmental: Congenital nervous depigmentation.

- Acute lesions of TS  
has cutaneous & neurologic  
abnormalities.

## Management

### General Aspects:-

- 1) Diet - Adequate protein  
Vitamin B complex  
Cu, Zn, Fe
- 2) Avoidance of trauma
- 3) Avoidance of soaps &  
detergents & phenolic  
compounds.

Medical:- IVBUB  $\rightarrow$  311 nm

Psoralen & UVA therapy.

$\downarrow$

PUVA  $\rightarrow$  320 - 400 nm

PUVAOL : when sun  $\rightarrow$  UVA.

Topical:- 1g macule = 6 sq cm  
usually 0.1% applied  
weekly for 2-3 hrs later  
by solar UVA irradiation  
for 30 - 60 seconds.

Oral:- 0.6 mg/kg body wt  
of TMP ingested  $\bar{c}$  food  
2 hrs before sun exposure

Exposure time:- Prescribed UVA dose  
0.06 x irradiance