

## Description

- Vitiligo is a type of skin disease that causes melanin in blotches.
- It can affect the skin on any part of a persons body which can include: The inside of the mouth or even a persons eyes.
- Vitiligo happens when melanocytes die or stop working.
- It can affect all skin types but it is usually more visible on a person with a darker complexion.



Winnie Harlow



Vitiligo is a condition of chronic skin disease in which a loss of cells that give color to the skin (melanocytes) results in smooth, white patches in the midst of normally pigmented skin.





## Introduction/Epidemiology

- An acquired pigmentary anomaly of the skin which manifest by depigmented white patches surrounded by a normal or hyperpigmented border
- Begins in childhood & young adulthood
- Peak age of onset b/w 10 & 30 years, about half begin before 20years
- Prevalence ranges from 0.5-1%
- Females are disproportionately among those seeking care
- Transmission is polygenic

### Causes of Vitiligo



#### **Major Theories**

- Auto-immnne
- Neural
- Autocytotoxic
- Growth factor defect

### Vitiligo, Etiology

 The cause is unknown, but the condition may be autoimmune process, in which the body destroys its own melanocytes, since autoantibodies to melanocytes were identified & up to 1/3 of patients have evidence of other autoimmune disease (e.g., Addison's disease, diabetes mellitus, pernicious anemia & thyroid dysfunction)

### Predisposing factors:

- Heredity
  - Vitiligo has a genetic background; >30% of affected individuals have reported vitiligo in a parent, sibling or child
    - Individuals from families with an increased prevalence of thyroid disease, diabetes mellitus, and vitiligo appear to be at increased risk for development of vitiligo
- Physical trauma (where vitiligo appears at the site of trauma; Koebner phenomenon).
- Exposure to chemicals such as phenols
- Illness or emotional stress
- Skin injury, burns or inflammatory skin disorders
- A sunburn reaction may precipitate vitiligo



## **Clinical Classification**

- Localized
  - Focal
  - Segmental
- Generalized
  - Symmetrical
  - Acromucosal
  - Universalis

## Clinical classification of vitiligo

- According to the extent of involvement, severity and distribution of the depigmentation, vitiligo has been classified in different clinical classes.
- This classification is very useful to evaluate different therapeutics regimens.

### Based on severity vitiligo can be divided into 4 stages

Limited (10%) involvement Moderate (10–25%) Moderately severe (26–50%) Severe disease (50%) depigmentation

### Vitiligo Classification

- Much has been discovered in recent years about the pathophysiology of the disease, ٠ pointing clinical findings to three main factors: 1) adhesion deficit throughout the epidermis, not only affecting the adhesion of melanocytes, but also keratinocytes; 2) increased local oxidative stress; 3) T lymphocyte-mediated cytotoxicity against melanocytes
- Vitiligo Classification ٠

Types

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### Subtypes

- Non-segmental vitiligo Acrofacial ٠
  - Mucosal (more than one site affected)
    - Generalized or Common Universal
  - Mixed (associated with segmental vitiligo)
    - Rare forms
- : Segmental Unisegmental, bisegmental or multisegmental
- Unclassified or indeterminate Focal ٠
  - Mucosal (only one site affected)
- Adapted from: Revised classification / nomenclature of vitiligo and related issues: the . Vitiligo Global Issues Consensus Conference - 2012.

## Classification

- Vitiligo focalis- only one or more maculae in one area
- Vitiligo segmentalis- one or more maculae in a quasidermatomal pattern
- Vitiligo acrofacialis- distal extremity and face
- Vitiligo vulgaris- scattered macules over the entire body
- Vitiligo universalis- Almost all body depigmentation

Local neurological damage

Neural theory

Increased release of acetylcholine, catecholamines and neuropeptides

> Melanocyte destruction

#### Somatic mosacism

Increased rate of melanocyte apoptosis or increased susceptibility for immune-mediated destruction

> Recruitment of inflammatory cells

> > Melanocyte destruction

#### Microvascular skin homing

mmune activation

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Cytotoxic T cells migrate to the specific segmental vitiligo site via homing receptors

> Melanocyte destruction

#### Three step theory

Release of inflammatory factors, neuropeptides and catecholamines

Increased presentation of melanocyte antigens (possibly due to somatic mosacism)

Generation of specific cytotoxic T cells

Melanocyte destruction

### THE PATHOGENESIS OF VITILIGO

Chemical Melanocytotoxin:

- 1) Melanin synthesis can be triggered by:
- Sunburn, skin injury or exposure to cytotoxic compound stimulate melanin synthesis
- Melanocyte stimulating hormone induced by UV exposure
- Cytokines produced during emotional stress or physical trauma
- Specific Quinones (Dopa Quinone) and Indoles (Dihydroxy indoles) are generated as intermediates during melanin synthesis
- Quinones & Indoles, if abundant in the cells, can readily oxidize either enzymatically or spontaneously and produce reactive oxygen species and ultimately induce apoptosis.

### Signs and Symptoms

- Sudden white patches, mostly on the sun exposed sections of the body. (eyes, lips, legs, arms, feet, hands and face)
- Other common areas are arm pits, groin, nostrils, genitals, rectum and naval.

### Patterns

Focal- depigmentation limited



- Segmental- depigmentation occurs on one side of the body
- Generalized- depigmentation occurs all over the body

## Trichrome vitiligo

- Is characterized by both depigmented and hypopigmented macules in addition to normally pigmented skin.
- Results 3 shades of color → tan zone, normal and totally depigmented skin
- The natural evolution of the hypopigmented areas is progression to full depigmentation



## Quadrichrome vitiligo

- Refers to the additional presence of marginal or perifollicular hyperpigmentation.
- Presence of a fourth color (dark brown) at sites of perifollicular repigmentation.
- This variant is recognized more frequently in darker skin types, particularly in areas of repigmentation



Proposed algorithm for the management of vitiligo in Japan. (Modified from Oiso N et al. Source: J Dermatol. 2013;40:344-54.)





a) Complication (+) refer patient to specialist: Treatment of vitiligo as shown in complication (-)
b) Complication (-)

Camouflage should be available for all patients



Indian Association of Dermatologists, Venereologists and Leprologists

### Management of Vitiligo

- Non-surgical : Topical and systemic medications, phototherapy and photochemotherapy, lasers.
- Surgical : Tissue grafts, cultured and non-cultured cellular transplants.
- Supportive : Camouflage, psychotherapy.
- Treatment approach needs to be individualised
- Combination of more than one modality is commonly used to hasten response and prevent side-effects

### MANAGEMENT

Psoralens 0.6 mh/kg is adequate to produce repigmentation, After oral administration maximum concentration of the photosensitizing drug in the blood is achieved after two hours.

Maximum UVA radiation from sunlight is available between 9 to 11 A.M.

Thus to induce maximum photosensitization, it is advisable to take psoralen in the recommended dose after breakfast followed by exposure of the macule to sunlight at 11 A.M

Initially exposed for 15 minutes

Then exposure time is gradually increased to a maximum of 45 minutes



## Surgical treatment of vitiligo

- Tattooing
- Dermabrasion
- Exicision and closure
- Needling & spot peeling

- Punch grafting
- Split thickness grafting
- Suction blister grafting
- Melanocyte grafting
- Mesh grafting
- Allograft

# COMPLICATIONS

- Social or psychological distress
- More susceptible to sunburn and skin cancer
- Eye problems such as iritis
- Hearing loss (the correlation between hearing) loss and vitiligo is evidence that vitiligo is a systemic disorder influencing the whole pigmentary system, including melanocytes in the inner ear. Cochlear melanocytes and also melanin-containing cellular elements of the auditory system may be affected in vitiligo and interfere with the conduction of action