



Vitiligo is a skin condition which results in loss of epidermal melanocytes, leading to depigmented lesions surrounded by normal skin.

About 20% of patients develop vitiligo before age 8, with a mean age of onset of 4-5 years of age.

PRESENTATION

- **Depigmented macules and patches:** various shapes and sizes, well-circumscribed
- **Stable or progressive**
- **Segmental, non-segmental** or **mixed types**
- **Common locations:** face, scalp, acral surfaces, extensor surfaces, genitalia
- **Leukotrichia:** white hair *indicator of poor prognosis



ETIOLOGY

*The etiology of vitiligo is unclear, but likely multifactorial:

- **Autoimmune destruction of melanocytes:** ~20% of patients have autoimmune diseases
- **Genetic predisposition:** up to 50% of patients have +FHx
- **Skin injury:** sunburn, exposure to chemicals in household products and dyes
- **Intrinsic defects of melanocytes:** early apoptosis, defective cell-to-basement membrane adherence

DIAGNOSIS

***Clinical diagnosis** in most cases. If diagnosis is uncertain:

- **Dermoscopy:** white structureless areas with a typical glowing appearance, perilesional and perifollicular hyperpigmentation, and/or leukotrichia
- **Wood lamp examination:** vitiligo lesions appear as well-defined bright blue-white areas
- **Skin biopsy:** absence of melanocytes, perilesional lymphocytes



Dermoscopic features of vitiligo

MANAGEMENT

Vitiligo that begins in childhood may cause **psychological trauma**. Children with vitiligo can experience **depression, anxiety, body-image concerns, low self-esteem, and rejection among peers**. Therefore, treating vitiligo involves medical interventions that can **restore pigmentation**, and **psychosocial supports** to improve quality of life.

Limited Disease:

- **Topical Calcineurin Inhibitors (TCI):**
 - Immunomodulators, inhibit cytokine expression
 - Used on sensitive areas (face, skin folds)
 - i.e. tacrolimus 0.1% or pimecrolimus 1%
- **Topical Corticosteroids:**
 - Anti-inflammatory and immunosuppressive
 - Short-term use, or alternating with TCI
 - i.e. clobetasol 0.05%, mometasone 0.1% cream or ointment

*Consider intralesional corticosteroids for small areas

Extensive or Refractory Disease:

- **UVBnb Phototherapy**
 - Reduces inflammation and stimulates melanocyte production
- **Topical JAK inhibitors (i.e. Ruxolitinib)**
 - blocks JAK-STAT pathway, allowing melanocytes to repopulate
- **Oral Corticosteroids**
 - Short term use, for widespread disease
- **Skin graft or melanocyte transplant**
 - for refractory disease and significant cosmetic concerns

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