



## **Different Homoeopathic Medicines Use in Case of Vitiligo**

***Thesiya Hardi Vipulbhai , Savjani Vrushti Rajendrabhai***

From BHMS INTERN BATCH 2024 - 25

Working from.

Gardi Vidhyapith Rajkot – kalawad highway, village Anandpar Rajkot , Gujrat , India Pin code- 361 162

---

### **Vitiligo**

Vitiligo is chronic pigmentary disease of the skin, which develop slowly and progressively. Occurrence of vitiligo is increasing in India, especially in children and middle-aged group who has positive family history or associated with other autoimmune diseases. This article deals with an overview of vitiligo, aetiopathogenesis, clinical feature and its homoeopathic approach in improving quality of life of the patient.

It is pigmentary characterised by circumscribed loss of melanin pigment secondary to melanocyte attrition. It is an acquired, sometimes familial condition, an autoimmune disease in its background.

It is characterised by milky white macules, histochemically there is a lack of DOPA- positive melanocytes in the basal layer of epidermis. The macules vary in size and shape as well as in colour. Some of the lesions or some parts of the lesions may be hypopigmented rather than depigmented. The course of the disease is very variable. The lesions in some patients may remain static or progress very slowly.

It affects around 0.5%–1% of the world population. As high as 8.8% prevalence have been reported in India.

---

### **AGE:**

Vitiligo may develop at any age—birth to 81 years of age. Fifty percent of cases appear before the age of 20, with the disfigurement resulting in psychiatric morbidity in 16%–35% of those affected. All races are affected. Both sexes are affected equally. Vitiligo patches can appear anywhere on the skin, but commonly affected sites include the area around the orifices, the genitals or any sun-exposed areas such as the face and hands. The hair and rarely, the eyes may also be affected.

Genetic predisposition: Inherited disease transmitted as an autosomal dominant. About 35% of the patients with a family history of vitiligo develop this disorder. Nutritional: defects in copper, proteins and vitamins in diet. Endocrinology: Association with thyrotoxicosis, diabetes, hypothyroidism and acromegaly. Emotional stress and strain. Infections and toxic products: Enteric fever, ill health, focal sepsis.

Though vitiligo is mostly a single entity, the aetiology is complex. Several hypotheses to explain its aetiopathogenesis have been propounded which include-genetic, autoimmune, self-destruct (Reactive Oxygen Species Model), neural and melanocytorrhagy hypothesis.

Familial cases of vitiligo are common, suggesting a genetic basis. Studies demonstrate that a family history for vitiligo exists in 6.25-38% of patients. individuals who maintain recessive homozygosity at these loci are affected by vitiligo.

Self-destruct hypothesis, formation of hydrogen peroxide (H<sub>2</sub>O<sub>2</sub>), certain tyrosine analogues and intermediates in melanin synthesis are thought to cause the destruction of melanocytes.

The theory of oxidative stress is a cause for vitiligo suggests that patients with vitiligo have an imbalanced redox (reduction-oxidation) state of the skin, resulting in the excess production of reactive oxygen species. These disturbances and Reactive Oxygen Species accumulation can have toxic effects on all components of the cell (proteins, lipids etc.) and could potentially result in the destruction of melanocytes creating the depigmented macules observed in vitiligo.

Neural Theory asserted that depigmentation in vitiligo results from increased discharge of a specific substance like melatonin at peripheral nerve endings in the skin. One that lightens pigment and discourages formation of new melanin. This theory suggesting of pathogenesis of segmental type of vitiligo.

---

### **TYPES :**

Non-segmental, or generalized vitiligo, the pathogenesis may be better explained by autoimmune mechanisms. One of the most apparent correlations between vitiligo and autoimmunity is the finding that patients with vitiligo often have autoimmune comorbidities. The mechanisms of immunity are humoral (antibody-mediated), cell- mediated, or mediated by cytokines.

The role of humoral immunity suggesting of presence of autoantibodies against tyrosine hydroxylase have potential target on Melanin Concentrating Hormone Receptor1 (MCHR 1). Cell-mediated immunity plays a role in inflammatory vitiligo. It is hypothesized that the inflammatory process may play a role in the elimination of melanocytes. The epidermis-infiltrating T cells found in perilesional skin demonstrated an increased CD8:CD4 ratio, and increased interleukin-2 receptor (IL-2) expression.

The melanocytorrhagy hypothesis is a relatively new approach to explaining the pathogenesis of vitiligo. This describes the pathogenesis of non-segmental vitiligo as from the result of a chronic detachment and loss of melanocytes resulting from altered melanocytes responses to trauma and other stressors. There is marked absence of melanocytes and melanin in the epidermis. Histological studies show a lack of dopa-positive melanocytes in the basal layer of epidermis. Area around vitiligo margin shows abnormalities of the keratinocytes. Increased cellularity of the dermis and occasional colloid amyloid bodies are found. In inflammatory vitiligo, there is raised erythematous border, infiltrate of lymphocytes and histiocytes seen.

Focal Vitiligo: There is either a single or few macules limited in both size and number. Segmental Vitiligo: The lesions are distributed in a dermatomal or quasi-dermatomal pattern. This is earlier in onset but is considered a stable type of vitiligo.

Generalised Vitiligo: It is characterised by few to many to widespread macules. The lesions are often symmetrical and involve extensor surfaces, areas over small and large joints. Periungual involvement may occur alone or with simultaneous involvement of lips, distal penis and nipples. The names lip tip and acrofacial vitiligo are given to this peculiar distribution of lesions. Universal Vitiligo: It indicates almost total involvement of the body with few remaining areas of pigmentation and has been associated with various endocrinopathy syndromes. Typical vitiligo macule has a chalk or milky white colour, round to oval in shape often with convex margins which are usually well defined, varying from few millimetres to many centimetres in diameter. Areas subjected to repeated friction and trauma are likely to be affected for example feet, elbow, knees and ankle. Rarely itching in the absence of sunburn. Isomorphic or Koebner phenomenon- Damage to the normal skin frequently results in an area of depigmentation.

Trichrome vitiligo refers to the presence of an intermediate colour, an interface between the white lesions of vitiligo and the normally pigmented skin.

Quadrichrome refers to fourth colour—perifollicular or marginal pigmentation seen in some cases of re-pigmenting vitiligo. Inflammatory vitiligo has an erythematous raised border similar to the one sometimes seen in pityriasis versicolour. Vitiligo may have other symptoms like leukotrichia, prematurely gray hair, halo nevi, and alopecia areata. Rarely vitiligo patients may have pigmentary abnormalities in iris and retina.

Vitiligo usually associated with diseases like Thyroid abnormality, pernicious anaemia, systemic lupus erythematosus, diabetes mellitus, Addison's disease, inflammatory bowel disease, rheumatoid arthritis, etc.

Active stage (V1): – New lesions developing

- Lesions increasing in size – Border ill defined.

Quiescent/ stable (V2): – No new lesions developed

- Lesions stationary in size
- Border hyper-pigmented and well defined.

Improving (V3):

- Lesions decreasing in size
- No new lesions developing
- Border defined and signs of spontaneous re-pigmentation.

Clinically vitiligo can be diagnosed by chalky white macules, with distinct margins.

Wood's lamp examination enhances the contrast between pigmented and non-pigmented skin.

Topical application of ointment or lotion on alternate days followed by exposure to the phototherapy PUVA.

Narrowed band UVA exposure.

Corticosteroids like Prednisolone 0.5 mg/kg body weight.

Other drugs like Levamisole may arrest slowly spreading vitiligo.

According to the German New Medicine theory, Skin consists of two layers namely Epidermis and corium skin (dermis and under skin). Corium skin composed largely of melanocytes, which make the pigment Melanin. Melanin acts as an effective absorber of light to shield the skin from the UV radiation.

---

## HOMOEOPATHIC APPROACH :

ALUMINA: General condition corresponding to this drug is dryness of skin and mucous membrane. Low spirited, fears loss of reason. Confusion of personality. Hasty, hurried



Time passes slowly. Suicidal tendency when seeing knife or blood. Lack of vital heat and characteristic constipation.

**ARSENICUM ALBUM:** profoundly acting remedy on every organ and tissue. Itching, burning of skin. Worse cold and scratching. Great exhaustion after slightest exertion. Unquenchable thirst. Great anguish and restlessness. Despair drives him from place to place.

**ARSENICUM-SULFURATUM-FLAVUM:** Indicated in leucoderma. Skin chafed about genitals. Needle pricks from within outwards in chest, forehead, right side. Sticking behind ear.

**CALCAREA CARBONICA:** Unhealthy skin, small wounds do not heal readily. Impaired nutrition being the keynote of its action, the glands, skin, bone being instrument in the changes wrought. Apprehensive, worse towards evening. Fears loss of reasons, misfortune, contagious diseases. Slightest mental effort produces hot head with much perspiration. Aversion to meat, craving for indigestible things like-chalk, coal and also for the eggs, salt and sweets. Persons of scrofulous type, who take cold easily.

**CALCAREA SILICATA:** It is the remedy for the complaints which come on slowly and reach their final development after long period. Itching, burning cold and blue skin. Patient is irresolute, lack of self-confidence. Very sensitive to the cold. Patient is weak, emaciated, cold and chilly, but worse from being overheated.

**IGNATIA:** Itching of the skin, very sensitive to the draught of cold air. Marked hyperesthesia of the all the senses. Alert, nervous, apprehensive, rigid, trembling patient who suffer acutely in the mind or body, worse by drinking coffee. Effects of grief and worry. Silently brooding. Sighing and sobbing.

**LYCOPodium:** Symptoms characteristically run from right to left, acts especially on right side of the body. Pre-senility. Patient is thin, withered, full of gas and dry. Lacks vital heat. Melancholy, fear of being alone. Averse to undertaking new things. Weak memory, confusion of thoughts. Desire for sweet things. Eating ever so little creates fullness.

**MERCURIUS:** Skin is almost moist. Excessive odorous viscid perspiration, worse at night not relieved thereby. Itching worse from warmth of bed. Weary of life. Intense thirst for cold drinks. Sensitive to heat and cold.

**NATRUM-CARBONICUM:** Inclination to perspire easily, or dry rough, cracked skin. Great debility created by summer heat. Exhaustion, anaemia, milky, watery skin. Very weak ankle. Very sensitive to noises. Anxious and restless during thunderstorm. Aversion to milk.

**NATRUM-MURIATICUM:** Greasy, oily skin. Emaciation most notable in neck. Diseases due to ill effects of grief, fright, anger. Consolation aggravation. Awkward, hasty. Headaches as if thousand little hammers were knocking on the brain. Crave for salt.

**PHOSPHORUS:** Wounds bleed easily, even if small. They heal and break again. Tall slender persons, narrow chest, with thin, transparent skin, weakened by loss of vital fluids, with great nervous debility, emaciation. Great susceptibility to the external impressions. Clairvoyance. Thirst for cold water.

**SEPIA:** Itching not relieved by scratching. Worse in bends of elbow and knees. Sweat on feet, intolerable odour. Hot flashes at menopause with weakness and perspiration. Indifferent to loved ones. Aversion to occupation, to family. Dread to be alone. Weeps when telling symptoms. Feeling of goneness, not relieved by eating.

**SILICIA:** Skin is delicate, pale, waxy. Rose coloured blotches. Every little injury suppurates. Imperfect assimilation and consequent defective nutrition. Patient is cold, wants plenty of warm clothing. Obstinate, head strong. Fixed ideas. Profuse sweat on head, offensive and extends to neck.

**SULPHUR:** Skin is dry, scaly, unhealthy. Even little injury suppurates. Itching, burning worse after scratching, warm. Sinking feeling in the stomach about 11a.m. standing is always uncomfortable. Aversion to being washed. Delusions, thinks rags beautiful things, that he is immensely wealthy. Very selfish, no regard for others. Great desire for sweet.

Vitiligo is chronic skin disease prevailing among middle aged group affecting persons quality of life. Various factors like genetic, oxidative stress, neural and autoimmune conditions play a role in development of the disease. Holistic treatment in Homoeopathy will not only treat the vitiligo but also remove the miasmatic sting runs in the family.

## BIBLIOGRAPHY

Pocket Manual of Homoeopathic Materia Medica & ReRepertory. By William Boericke  
Materia Medica of Homoeopathic Medicines. By. S.R. Phatak