International Journal of Science and Research (IJSR)
ISSN: 2319-7064
SJIF (2022): 7.942

The Role of Homoeopathy in Vitiligo: A Comprehensive Guide in the Treatment of Vitiligo to Maintain Beauty of Skin

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Abstract: ***Background: Vitiligo is an acquired idiopathic depigmentary condition characterized by appearance of white ill - defined patches of varying sizes and shape which though worldwide in distribution, is most common in India, Egypt and other tropical countries. The pigment loss may be localized or generalized. Many patients are embarrassed. Physicians should be especially alert to the effects of disfigurement. Homoeopathy plays a major role in the treatment of vitiligo. Day to day people always think about the beauty of skin. Beauty is not always skin deep, quite often people go for the world of glitters and for maintance so much time, money and resources.***

Keywords: Vitiligo, Skin disorder, drugs infection, Homoeopathy

1. Introduction

Vitiligo results due to stoppage of melanin formation by the melanocytes and presents as asymptomatic depigmented macules anywhere on the body including mucous membranes of lips and genitalia. There is a marked reduction or even absence of melanocytes and melanin in the epidermis [1, 2] Vitiligo is not a rare condition but is difficult to treat and is associated with psychological distress [3] The exact cause of the Vitiligo is not known and different patterns of vitiligo may have different pathogenesis [4, 5] Some studies suggested that Vitiligo is an autoimmune disease in which the body's immune system attacks its own healthy cells. It sometimes runs in family suggesting that genetics may play a role [5]. In some cases people with Vitiligo often report with some sort of triggering factors like injury, sunburn, illness, any emotional stress, pregnancy etc. [5, 6]

Incidence: [7]
Age: Onset between 10 - 30 years
Sex: Equal in both sexes.
Etiology: It is a feeling of many workers that vitiligo is a multifactoral malady. Genetic predisposition is important, its influence varies from 10 - 35 pc. Auto immunity has been blamed but inreality, it is a reaction pattern to drugs, infection and toxin, but whole of melanocyte system is defective. Important known causative factor. [8]

Epidemiology: [9, 10]
The prevalence of vitiligo ranges from 0.5% to 1%.2 Its highest incidence has been reported amongst Indians from the Indian subcontinent. India is considered to have the highest prevalence in the world, at about 8.8%.2 Mexico and Japan also has high prevalence of vitiligo. A small prevalence has also been reported by many studies in India, China and Denmark have reported the prevalence to be 0.093%, 0.005% and 0.38% respectively. The family history has been found to affect the prevalence of Vitiligo amongst such people prevalence is high ranging from 7.7% to more than 50%.

Nutritional: Defects in copper, proteins and Vitamins in diet, digestive upsets like amoebiasis, helminthes, chronic diarrhoea, dysentery etc. [7]

Endocrines: Association with thyrotoxicosis and diabetes**.**

Trophoneurosis and autonomic imbalance: Emotional stress and strain.

Infections and toxic products: Enteric fever, ill - health, focal sepsis.

Drugs and Chemicals: Quinones, guanofuracin, a mylphenol, Chlorthiazide, broael spectrum antibiotics, Betablaeteus and chloroquin. Chemicals are known to inhibit melanogenes is, enzymatic actions and several chain biochemical reactions. They can also cause interference with nutrition of the tissnes. Hence tie - up of the two chemicals and nutrition may provide the answer. Role of food adulterants, Industrial chemicals and dyes, Contaminating water and foods.

Pathology: A defect in enzyme tyrosinase is held responsible for vitiligo. According to some, melatonin, a substance secreted at herve ending inhibits tyrosinase, thus interfering in pigment formation DOPA staining shows that melanocytes are deficient. In active cases, mononuclear hugging at the junction of the lesion and normal skin is a prominent feature (Behl&pradhan) [7]

Mechanism of Repigmentation: [11]

The goal of treatment is to restore melanocytes to the skin. Therapy involves stimulating melanocytes within the hair

follicle to proliferate and migrate back into depigmented skin. Depigmented skin is devoid of melanocytes in the epidermis. Melanotic melanocytes in the bulb and infundibulum of the hair follicle are absent in vitiliginous skin. Repigmentation is caused by activation and migration of melanocytes from a melanocytic reservoir located in the hair follicles. Therefore skin with little or no hair (hands and feet) or with white hair responds poorly to treatment. Inactive amelanotic melanocytes in the middle and lower parts of the follicle and the outer root sheath are still present. These cells can be activated by treatment to acquire enzymes for melanogenesis. They proliferate and mature as they migrate up the hair follicle into the epidermis and spread centrifugally. When a vitiliginous spot repigments, it repigments from the follicle and spreads outward. This process is slow and requires at least 6 to 12 months of treatment. The face, arms, trunk, and legs respond best. Melanocytes divide rapidly after any inflammatory process or after UV irradiation. PUVA produces inflammation in the skin at the depth of the hair follicle. Cytokines released by the inflammatory process may stimulate melanocytes to proliferate and migrate outward.

Classification: [8]
Active progressive stage (V_1), Quiescent stage (V_2), Repigmenting stage (V_3) &Zosteriformis / segmental stage

Active progressive stage (V_1):
New lesions developing, Lesions increasing in size & Border ill defined.

Quiescent stage / stable (V_2):
No new lesion developing., Lesion stationary in size. Border hyperpigmented and well defined

Repigmenting stage / Improving stage (V_3)
Lesions decreasing in size. No new lesions developing. Border defined and signs of spontaneous repigmentation (Folicular and Peripheral)

Zosteriformis / segmental stage:
Unilateral distribution of lesions, Preferally along the course of nerves.

Clinical Manifestations: [11]
There are two types of vitiligo (A and B). In the more common type A (generalized), there is a fairly symmetric pattern of white macules with well - defined borders. The borders may have a red halo (inflammatory vitiligo) or a rim of hyperpigmentation. The loss of pigmentation may not be apparent in fair - skinned individuals, but it may be disfiguring in black people. Initially the disease is limited; it then progresses slowly over years. Commonly involved sites include the backs of the hands, the face, and body folds, including axillae and genitalia. White areas are common around body openings such as the eyes, nostrils, mouth, nipples, umbilicus, and anus. The palms, soles, scalp, lips, and mucous membranes may be affected. Genital vitiligo - like depigmentation following use of imiquimod 5% cream is reported. Vitiligo occurs at sites of trauma (Koebner phenomenon), such as around the elbows and in previously sunburned skin. Many patients with vitiligo develop halo nevi. An acrofacial or lip - tip type (involving lips and digits) also occurs. Segmental vitiligo (type B) occurs in an asymmetric distribution. The segments do not correspond to a dermatomal distribution. It is common in segmental forms for the hair follicles to be depigmented, indicating an absence of follicular melanocytes. The onset is earlier than that for the generalized form. There is a decreased association with autoimmune disease.

Onset: Insidious.

Location: Common sites, to start with, are pressure points, ie. Knuckles, elbows, lips.

Symptoms:
Lesions do not itch. Lesions start as white localized macules. Rounded, well defined at onset. Adjacent lesions coalesce. As they increase in size, become irregular. Spread to involve greater parts of body. Depigmented lesion is surrounded by area of apparent hyperpigmentation. Depigmented hairs in vitiliginous areas. No sensory loss in lesion. Every little trauma heals with depigmentation. Lesions are hypersensitive to sun light. Skin is susceptible to minor trauma. It may develops around pigmented moles Hulonaevus. Hair may or may not become depigmented in vitiligious areas.

Diagnosis: [13]
In doubtful and early cases, Wood's lamp is of great help in diagnosis. Usually in macular leprosy, anetoderma, seborrhoeides, pityriasisversicolor and nervoidconditions. Vitiligo areas are milky white while others lack this milky white coloration. Pinta and syphilitic leucomelanoderma can be differentiated by other clinical stigmata and positive blood serology. Haemoglobin content of the blood is low and sometimes intestinal parasites and infections can be defected. Patient complaint to easy fatiguability.

Differential Diagnosis: [11]

The differential diagnosis of vitiligo:
Chemically - induced leukoderma (often occupational), Phenols and other derivatives, Infections, Leishmaniasis, Leprosy, Onchocerciasis, Tineaversicolor, Treponematoses (pinta and syphilis)

Genetic syndromes
Hypomelanosis of Ito, Piebaldism, Tuberous sclerosis, Vogt - Koyanagi - Harada syndrome, Waardenburg syndrome

Postinflammatory hypopigmentation
Atopic dermatitis/allergic contact dermatitis, Nummular dermatitis, Phototherapy - and radiotherapy - induced, hypopigmentation, Pityriasis alba

Posttraumatic hypopigmentation (scar)
Psoriasis & Systemic lupus erythematosus

Topical or systemic drug - induced depigmentation

Neoplastic
Amelanotic melanoma, Halo nevus, Melanoma - associated leukoderma, Mycosis fungoides

Idiopathic
Idiopathic guttatehypomelanosis, Lichen sclerosusetatrophicus, Lichen striatus–like leukoderma, Melasma (caused by contrast between lighter and darker skin), Progressive (or acquired) macular hypomelanosis

Malformations
Nevus anemicus& Nevus depigmentosus/hypopigmentosus

Common disorders that resemble vitiligo are tineaversicolor, idiopathic guttatehypomelanosis, and nevus depigmentosus. Chemical leukoderma is caused by dyes, cleansers, insecticides, and many other products. Occupational vitiligo occurs with exposure to several different industrial chemicals.

Childhood Vitiligo:
Childhood vitiligo is a distinct subset of vitiligo. There is an increased incidence of segmental vitiligo (type B vitiligo), of autoimmune and/or endocrine disease, of premature graying in immediate and extended family members, and of organ - specific antibodies, in addition to a poor response to topical PUVA therapy.

Psychologic Impact:
Vitiligo can have a major impact on personality. Feelings of stress, embarrassment, self - consciousness, and low self - esteem can occur. Patients claim the disease interferes with sexual relationships. The psychologic impact can be profound in deeply pigmented races. The disease can have serious social stigma in some cultures.

Eye, Ear, and Meningeal Findings:
Vitiligo affects all melanocytes. Depigmented areas in the pigment epithelium of the retina and choroid occur in up to 40% of vitiligo patients. The incidence of uveitis is elevated. The membranous labyrinth of the inner ear contains melanocytes. Minor hearing problems can occur. The Vogt - Koyanagi - Harada syndrome consists of vitiligo with many other associated findings; the most common are meningismus, hearing loss, alopecia, tinnitus, and poliosis. The aseptic meningitis may be due to destruction of leptomeningeal melanocytes. The disease appears in the fourth to fifth decade and is more common in women and in persons with dark pigmentation.

Associated Diseases:
Most patients with vitiligo have no other associated findings; however, vitiligo has been reported to be associated with alopecia areata, hypothyroidism, Graves' disease, Addison's disease, pernicious anemia, insulin - dependent diabetes mellitus, uveitis, chronic mucocutaneous candidiasis, the polyglandular autoimmune syndromes, and melanoma. Thyroid disorders have been reported in as many as 30% of vitiligo patients. Circulating autoantibodies such as antithyroglobulin and antimicrosomal and antiparietal cell antibodies have been found in more than 50% of patients.

Wood's Light Examination:
Examination with the Wood's light in a dark room accentuates the hypopigmented areas and is useful for examining patients with light complexions. The axillae, anus, and genitalia should be carefully examined. These areas are frequently involved but often clinically nonapparent without the Wood's light. Vitiligo may be a predictor of metastases in melanoma patients, and a Wood's light examination may show early subtle changes in these patients.

Studies and Referral:
Obtain a thyroid - stimulating hormone level and complete blood count with indices and blood glucose level to rule out thyroid disease, pernicious anemia, and diabetes mellitus. Screen for autoimmune disease with an antinuclear antibody test. Consider histopathologic evaluation. Biopsy lesional and normal skin. Vitiligo may be part of a syndrome with multisystem dysfunction. Evaluation by a geneticist is recommended for those patients.

Treatment Perspective: [11]
All treatment options have limited success. The face and neck respond best to all therapeutic approaches; the acral areas are least responsive. For generalized vitiligo, phototherapy with narrow - band UVB (NB - UVB) radiation is most effective with the fewest side effects. Topical corticosteroids are the preferred drugs for localized vitiligo. They may be replaced by topical immunomodulators, which display comparable effectiveness and fewer side effects. The effectiveness of vitamin D analogues is controversial with limited data but they are felt to be the least effective topical treatment. The excimer laser is an alternative to UVB therapy especially for localized vitiligo of the face. Surgical therapy can be very successful, but requires an experienced surgeon and is very demanding of time and facilities, thus limiting its widespread use.

Indications for Treatment: [12]
Treatment is necessary for patients in whom the disease causes emotional and social distress. Vitiligo in individuals with fair complexions is usually not a significant cosmetic problem. The condition becomes more apparent in the summer months when tanning accentuates normal skin. Tanning may be prevented with sunscreens that have an SPF of 15 or higher. Vitiligo is a significant cosmetic problem in people with dark complexions, and repigmentation with psoralens may be worthwhile. Todiagnose the exact vitiligo one should be able to differentiate between different conditions of the skin like complete depigmentation, hypo pigmentation and normal colour of the skin. Diagnosis of vitiligo is very difficult in patients having lightcomplexion of the skin colour. Wood's light is very useful todiagnose the vitiligo in the patients having skin type I and II. Puretone and speech audiometer, Sound treated room, Cochlear Emission Analyzer Madsen, Immittance meter, Evoked Response Audiometer Nickolet Compact four, Wood's light lampequipments can be used for the diagnosis of vitiligo. [12]

General Management:
Reassurance, Treat associated disorder, If iatrogenic, identify and withdraw offending agent, Avoid physical trauma, Adegnate mental rest, Improve general health, Diet consisting of sprouteel grams, Cheese. Etc., Not to eat fish along with curd.

Homoeopathic approach in the treatment for vitiligo:
The Homoeopathic literature subsequently suggested a number of medicines for the conditions mentioned as 'vitiligo, ''hypopigmented spots'. [14 - 16]Homoeopathy provides individualized patient treatment, which includes a holistic approach to the understanding of the patient. [17]Homoeopathy treats the patient not the disease. Homoeopathy does not focus on end results i. e. the white spots, but on the cause of Vitiligo. The best way to treat Vitiligo is through internal medication of Homoeopathy rather than by application of local medicines. It is believed that Homeopathic medicines moderate the overactive immune system and recovers the destroying melanocyte cells in the skin. [18]

Homoeopathy has given innumerable successful results in the treatment of Vitiligo when prescribed on the basis of totality of symptoms depicting individualization. This reaffirms the importance of individualization approach of Homoeopathy in treating the patients. This also confirms the observations of Samuel Hahnemann in aphorism 18 Organon of Medicine "From this indubitable truth, that besides the totality of the symptoms with consideration of the accompanying modalities (§ 5) nothing can by any means be discovered in disease wherewith they could express their need of aid, it follows undeniably that the sum of all the symptoms and conditions in each individual case of disease must be the sole indication, the sole guide to direct us in the choice of a remedy" in treating the chronic illnesses, which laid emphasis on totality of symptoms of a patient which ultimately becomes the only guide to select a similar medicine from MateriaMedica. [18]

Therapeutic aim:
Control disease activity, Maintain skin beauty &To cure

Miasmatic cleavage:
Psoro - Syphilitic disorder

Homoeopathic Medicine for Vitiligo: [19]
Homeopathic medication involves a holistic approach for the understanding of the patient. Certain diseases are manifested when genetic predisposition combing psychological, physical and physiological factors and, homeopathy recognizes these factors. Homoeopathic medicines such as Calcareacarbonica, Arsenicum album, Sulphur, Phosphorus, Natrummuriaticum, Thujaofficenalis, Lycopodiumclavatum, Mercuriussolubilis and Arsenicumsulphuratumflavum were found to be most frequently prescribed in the Vitiligo cases, which are mostly the constitutional medicines with the exception of Ars. Sulphflavum.

1) **Arssulph Flavatum:** Leucoderma especially at mucocutaneousjunction. The skin is very dry cracked, scaly and blackish in appearance. It may occur along with eruption. especially on the outer as ell as inner side of the left wrist.
2) **Arsenic album**: Dry, dirty roughskin> warmth, Burning sensation in varying body. Great anxiety and marked restlessness. < cold scratching
3) **Baryta carb**: Leucoderma with burning sensation intolerable itching and fingling over the whole body at night. Sensations like pricks of burning needles along with itching and crawling on the skin. <at night > warm atmosphere < cold
4) **Barytamur:** Leucoderma in small spots. Small spots and whitish patches all around the head, nape of the neck, abdomen and thigs. White spots associated with itchy eruptions all over the body.
5) **Boran:** Un healthy skin. Itching on back of finger. Eruptions on fingers and hands < Warm weather, motion. White skin and red patches
6) **Calc. Carb:** Un healthy, Petechial eruptions, Vitiligo with milky white spots on skin. It appear's anywhere on the body. Craving for egg, lime and pencile. Fat fair and flabby persons. Tendency to catch cold easily <erertion mental (or) physical, Fullmoon
7) **Causticum**: Leucoderma from burns. Soreness in foldsd of skin, back of ears, between thigh. <dry cold winds
8) **Hydrocotyleasoatica:** Dry eruptions, Great thickening of epidermoid layer, Cellular proliferation in any parts. "If arssulphflav fails this remedy may be tried"
9) **Nitric acid:** Vitiligo or Leucoderma occurs especially at mucocutaneous junction. Discolouredzigzagpatcheson the skin. < Cold climate & hot weather
10) **Psoraleacoryl**: It is a specific remedy for Leucrderma. White patch on dorsum of the feet and face.
11) **Staphysagria**: Vitiligo at the tip of penis. Itching scratching changes location of itching occur's. Night sweat <Grief, anger

2. Conclusion

`Vitiligo is one of the most common skin disorders today. It makes a significant impact on the patient's psychological development gradually. The word vitiligo may be derived from the Greek vitellius, signifying a "calf's white patches. " Vitiligo is an acquired loss of pigmentation characterized histologically by the absence of epidermal melanocytes. It may be an autoimmune disease associated with antibodies (vitiligo antibodies) to melanocytes, but the pathogenesis is still not understood. Studies suggest there is some genetic mechanism involved in the etiology of vitiligo and that it is polygenic in nature. There is a positive family history in at least 30% of cases. Both genders are affected equally. Approximately 1% of the population is affected; 50% of cases begin before age 20. Homoeopathy helps to reduce this tension by responding to the patient's physical and psychological necessities. Homoeopathy treats the patient as a whole, not simply the disease's symptoms. All of the patient's symptoms are taken into account before homoeopathic drugs are recommended. The range of action of Homoeopathic medicine aims to strengthen the action of the immune system through the primary understanding that symptoms are an attempt by the immune system to achieve balance. 'Through the application of the principle of resonance, the basis of homeopathic medicine is that if a substance is capable of producing a similar symptom profile in a healthy organism, then the likelihood of its strengthening of the body's defense mechanisms in a diseased body with the same symptoms is great. The fundamental pillar of the science of Homoeopathy is that 'like cures like. The basis for Homoeopathy is that any substance (plant, animal, mineral, or metal) that can affect the human health can serve as a medication, when in the right form. Homoeopathic medicines are prepared through

serial dilutions and using a frictional process called 'succussion' or potentization, resulting in no traceable 'material' left in the solution, therefore enabling a safe use of toxic substances that may otherwise prove fatal. The symptoms obtained through 'proving' of the Homoeopathic compounds on healthy humans serve as the basis for their prescription in diseased individuals. Because the main therapeutic rule in homeopathy is SimiliaSimilibusCurentur (let like be treated by like) Homoeopathy has the advantage of taking the causes of diseases and their effects into account. Therefore, Homoeopathic treatment, when given in a timely fashion, may bring lasting improvement for autoimmune disease, when Homoeopathy is applied in the early stages.

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