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VITILIGO IN FOCUS: A COMPREHENSIVE REVIEW OF ETIOLOGY, DIAGNOSIS AND MANAGEMENT

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ABSTRACT
In world about 0.5% -2% populations got affected by Vitiligo depigmenting cutaneous disorder. Vitiligo is characterized by loss of melanocytes like chalky white patches, non scaly macules. There are following main causes of Vitiligo first is reduced reaction to touch allergen second is the white epidermis people with albinism. The overall cause of Vitiligo is immune system illness has been topic of discussion after many years. It is an autoimmune disease with Even though many people think vitiligo is just a skin-color issue, it can actually cause serious emotional pain and make daily life very difficult. This review aims to provide study of current state of Vitiligo.the main treatment of Vitiligo are phototherapy, anti-inflammatory creams and surgical grafting of healthy skin cells to white patches.

KEYWORD: Vitiligo, albinism, phototherapy, depigmentation.

1. INTRODUCTION

Vitiligo is a hypo pigmented condition marked by sporadic skin pigmentation loss. Increasing melanin synthesis and modifying immune system responses are the main goals of current treatment approaches. Such hypopigmentation problems may be treated with a combination of topical medicines at the right doses.[1] The Roman physician Celsus in the first century AD is credited with coining the term vitiligo, which is most likely derived from the Latin word Vitilus, which means calf. Various body parts develop white patches on their skin. Both the retina (the inner layer of the eyeball) and the mucous membranes (the tissues lining the inside of the mouth and nose) have similar patches. On vitiligo-affected areas, hair can occasionally turn white.

Fig. 1: Vitiligo disease.

Although it can occur at any age, 50% of cases begin before the age of 20.[2] In the presence of an enzyme known as a melanosome, melanocytes produce melanin from the amino acid tyrosine. Melanin is the pigment required to produce skin color.[3] Vitiligo, often known as bars, is a skin pigmentation condition that has several risk factors and triggers. Milky white macules that expand, spread, and can affect the entire body's skin are its clinical hallmark.[4] Vitiligo affects about 0.5–1% of the population. India has the greatest reported prevalence (up to 8.8%), followed by Mexico (2.6–4%) and Japan (1.68%2). The face, neck, and arms are among the exposed body parts that typically experience depigmentation. Patients with vitiligo frequently experience a significant psychological load as a result of the severe symptoms. While both sexes are equally affected by this condition, women are more inclined to seek treatment and to openly discuss vitiligo for cosmetic reasons.[5]

Two clinical kinds of vitiligo are distinguished by a recent classification: segmental (type B) and no segmental (type A). Type B is more uncommon, has a dermatomal distribution, and typically has a stable course following a quick beginning and evolution. Type A is more prevalent, may evolve over the course of a lifetime, and is often linked to autoimmune conditions such Sutton nevus, thyroid issues, juvenile diabetes mellitus, pernicious anemia, and Addison's disease.[6] The following are the two most typical causes of white skin. After producing melanin, melanocytes pack it into melanosomes, which are then injected into nearby keratinocytes by the surrounding keratinocytes via their dendritic processes. Melanins and melanosomes are carried by keratinocytes from the basal layer of the epidermis to the stratum corneum, where cells are desquamated and released into the environment.[13]

Although vitiligo can develop at any age, it typically starts in childhood or early adulthood and peaks between the ages of 10 and 30.15, 20, 26–28 Fifty percent of instances occur before the age of twenty, according to several studies.20, 26–28 In infancy or elderly age, it is uncommon.17 As age increases, the incidence declines.25 Barona discovered that the mean age at onset was 16.3 years (95% CI = 12–19 years) for patients with unilateral Vitiligo and 24.8 years (95% CI = 22–28 years) for those with bilateral Vitiligo.[7]

2. PATHOGENESIS OF VITILIGO

Pathogenesis A number of theories have been proposed to explain the pathogenic processes that result in melanocyte death and the formation of hypo pigmented patches. The most widely recognized theories include neurological, genetic, autoimmune, and autocytotoxic.[8]

Autoimmune hypothesis: An relationship with other autoimmune disorders and the identification of blood autoantibodies to melanocytes, particularly to melanosomal proteins, in patients with active disease lend credence to this. Additionally, T-cell analyses in peripheral blood and histological and immunohistochemical investigations in perilesional skin suggest the role of cellular immunity in Vitiligo.

Autocytotoxic hypothesis: Melanocytes may be cytotoxically affected by melanin precursors like tyrosine. Contact Vitiligo is linked to phenolic and catecholic compounds, which share structural similarities with tyrosine.

Neural hypothesis: The neuronal theory was initially proposed by Lerner. Catecholamine and metabolite levels were considerably greater in vitiligo patients than in controls in a study looking for a connection between vitiligo and monoaminergic system activity. This could be a result of monoaminergic systems being more active during the early stages of the illness.

Genetic factors: There are a number of genetically related loci and candidate genes that seem to mediate susceptibility to generalized Vitiligo.

A thorough examination of the borders of active generalized Vitiligo lesions has frequently revealed sparse infiltrates of cytotoxicity T cells, and many patients with generalized Vitiligo have circulating auto reactive T cells and serum auto antibodies directed against melanocytes and melanocyte components.[9]

Oxidant stress: In Vitiligo, oxidative stress may potentially have a significant pathogenic impact. It is hypothesized that vitiligo may be caused by an imbalance in the oxidant-antioxidant system rather than oxidative stress. Molecular studies have also shown that vitiliginous skin lacks antioxidant compounds. The free radicals block tyrosinase and are harmful to melanocytes.[10]

Innate Immunity: As was previously reported, GWA investigations in Vitiligo patients identified several susceptibility loci associated with the genes governing innate immunity. As evidenced by the recruitment of innate populations such as natural killer (NK) cells and the production and release of high levels of pro-inflammatory proteins and cytokines such as heat-shock proteins (HSP), IL-1β, IL6, and IL-8, this probably results in deregulated innate activation in response to melanocyte stress. Inducible HSP70 (HSP70i) is distinct from other bigger HSP molecules in that it can be released to chaperone peptides that are particular to the original host cells. Through the activation of inflammatory dendritic cells (DCs), which may be cytotoxic or carry and present melanocyte-specific antigens to T cells in lymphoid tissues, HSP70i has recently been demonstrated to be crucial for Vitiligo pathogenesis in a mouse model. It has been suggested that this is a crucial interaction between innate and adaptive immunity that results in the autoimmune death of melanocytes by T cells.[11]

Adaptive Immunity: Melanocytes are ultimately destroyed by cytotoxic CD8+ T lymphocytes. These autoreactive T cells use the skin's produced cytokines as an early signal to find stressed melanocytes. This is likely significant since the epidermis lacks vascularization, necessitating the utilization of active processes to effectively detect melanocytes. Chemokines are tiny, secreted proteins that direct T cell movement by acting as chemo attractants. In Vitiligo sufferers' skin and blood, as well as in a mouse model, IFN-γ and IFN-γ-induced chemokines (CXCL9 and CXCL10) are substantially produced. Additionally, in a mouse model of the disease, IFN-γ and CXCL10 are necessary for both disease maintenance and development. A different study recently showed that serum CXCL10 levels were not only higher in Vitiligo patients than in healthy controls, but they were also linked to disease activity and dramatically dropped following successful treatment. This suggests that serum CXCL10 could be used as a biomarker to track treatment response and disease activity.[11]

3. SYMPTOMS OF VITILIGO

Depigmented Patches- The primary symptom of vitiligo is the appearance of white or light-colored patches on the skin. These patches can vary in size and shape and may appear on any part of the body.[16]

Symmetrical Distribution -Vitiligo patches typically appear symmetrically on both sides of the body. For instance, if a patch appears on one elbow, it can also show up on the other. Progressive Spreading: Over time, vitiligo patches may progressively get bigger or spread. Further depigmentation may result from the development of new spots.

Hair Discoloration - In addition to affecting the skin, vitiligo can also cause hair in the affected areas to turn white or lose pigment.

Sensitivity to Sunlight -Some vitiligo sufferers may be more sensitive to sunlight in their depigmented patches. These spots may burn more readily or become more apparent when exposed to the sun.

Changes in Pigmentation of Mucous Membranes -In rare instances, vitiligo may also have an impact on the pigmentation of mucous membranes, including the tissues in the nose and mouth.[12]

4. TYPES OF VITILIGO

Nonsegmental vitiligo (NSV) - This is the most prevalent kind, sometimes referred to as generalized vitiligo. It has a symmetrical, bilateral pattern.[13] Depending on the distribution and degree of depigmentation, there are multiple subtypes of non-segmental vitiligo. The face, head, hands, and feet—especially the perioral area and distal digits—are all affected by the acrofacial subtype.[23]

Segmental vitiligo (SV) - This kind has a dermatomal or linear pattern, affecting only one side (unilateral) of the body.[13] One or more body segments are usually affected by segmental vitiligo, with unisegmental involvement being the most prevalent. It can cause hair depigmentation (leukotrichia) and manifests as distinct depigmented macules that are limited to one side of the body, frequently along the midline. The illness normally develops quickly, stabilizes in 6 to 12 months, and most patients don't experience any new lesions or additional progression. Rarely, bilateral or multisegmental patterns could appear.[23]

Unclassified- Focal vitiligo refers to one or more acquired lesions that do not fit a segmental distribution. This form may evolve into either SV or vitiligo/NSV. Mucosal vitiligo is classified as undetermined/unclassified vitiligo when oral and/or genital mucosal lesions occur without skin involvement. If skin involvement exists, mucosal vitiligo is included in vitiligo/NSV.[25]

5. HISTORY

Vitiligo is derived from the Latin word "vitilus," which means calf. Celsus, a Roman physician, originally used the phrase in the first century of the Common Era. He claimed that the disease's white patches were similar to a spotted calf's white spots. The Bible, the Veda, and the Holy Quran all make reference to vitiligo, an old illness. The illness is even referred to as "Bai Dian Feng" in traditional Chinese medicine, "Shewetakusta" in the ancient Indian Atharva Veda, "Kilas" in the Buddhist holy book Vinay Pitah, "Bars," and "Phulbehri" in Arabic and Punjabi.[14]

Lesions started off small and isolated, became larger over time, and eventually migrated to the back and abdomen. There was a slight itching. This patient does not have a burning feeling. After receiving treatment with contemporary medicine for a year and a half, the patient showed no discernible progress. Historical background: Not noteworthy Personal history: The patient was intolerant of heat and had a craving for hot foods like beef and ice cream. Psychological history: A family issue caused the patient to experience stress for almost five months. During this time, white spots grew in size. Family background Vitiligo was not inherited.[15]

Local analysis

1. Lesion site: B/L iliac spine, extending to the abdomen and back
2. Symmetrical Distribution
3. Itching: Not too severe
4. Absent Discharge
5. Feeling: Current
6. Lesion characteristics: widespread, clustered, white lesion
7. There is no inflammation.

6. DIAGNOSIS OF VITILIGO

The increasing loss of melanocytes that results in white patches on the skin is the hallmark of vitiligo. Typically, Wood's light examination is used in conjunction with clinical examination to make the diagnosis. Pityriasis Alba, hypopigmented mycosis fungoides, tinea versicolor, idiopathic guttate hypomelanosis, and other hypo- or depigmented conditions are among the differential diagnosis for vitiligo.[17]

One must be able to distinguish between various skin conditions, such as total depigmentation, hypopigmentation, and normal skin color, in order to accurately diagnose vitiligo. It is particularly challenging to diagnose vitiligo in patients with light skin tones. When diagnosing vitiligo in patients with skin types I and II, Wood's light is quite helpful. Vitiligo can be diagnosed with Wood's light lamp equipment, a sound-treated room, a Cochlear Emission Analyzer Madsen, an Immittance meter, an Evoked Response Audiometer Nickolet Compact four, and a pure tone and speech audiometer.[3]

7. TREATMENT OF VITILIGO

The Etiology of Vitiligo is still a mystery. However, it incorporates a number of ideas, including autoimmunity, cytotoxicity, triggering, neurology, free radicals, and genetics. In mild cases, Vitiligo can be treated with oral or topical drug formulations alone, but in severe cases, light therapy is also used in addition to medicine to increase skin pigmentation. In order to achieve an equitable distribution of the typical quantity of skin coloring, treatment for leukoderma or Vitiligo necessitates both the deposition of pigment in the depigmented areas and the redistribution of pigment from hyper pigmented borders. Additionally, it is dependent on the sort of cell that is present. The presence of keratinocyte stem cells in a comparable area reduces the possibility of melanocyte development in interfollicular epidermis.

Herbal medicines

Treatment of vitiligo with herbal medicines from the list of Chinese herbs acting for the treatment of vitiligo decoction of xiaobailing changyee powder and three yellow powders are most effective. These medicines include xanthumstramanum, sophoraflavescens, atractylodes japonica, arisaemaamurense.[3]

7.1 Traditional Treatment

Red clay: Vitiligo can also be treated with red clays that are found on hill slopes or along rivers. It can be administered by combining it with ginger juice. Ginger increases blood flow to the area, which aids in the repigmentation of the spots, while copper in the clay restores skin pigmentation. A paste is created by powdering radish seeds with vinegar. Vitiligo can be treated with this paste. White patches can also be treated using a mixture of turmeric and mustard oil made by heating two of them.

Yoga therapy: Kapalbhati is useful for treating vitiligo. Kapalbhati aerates blood and cleanses blood circulation by inhaling and expiration. This helps with a variety of skin conditions, including psoriasis, vitiligo, and other allergies.[3][19]

Homeopathic treatment: Homeopathy is an alternative medicine that was developed in Germany in the 18th century and has since spread to many other nations. Homeopathic medicine can employ drugs such as Arsach, Bacillinum, Graphites, Mercasol, Nat mur, nuxvom, sil, sulph, thuja, etc.[19]

Ayurvedic treatments: According to Ayurveda, vitiligo is referred to as Switra and is mostly brought on by Pitta Dosha. When Pitta is inflamed, toxins (ama) build up in the skin's deep layers, causing vitiligo.

7.2 Herbal treatment

TURMERIC: Curcuma, a substance found in turmeric, is recognized for its antioxidant and anti-inflammatory qualities. Turmeric (Curcuma longa), a golden spice, is the source of curcumin, a polyphenol. Curcumin has been used to treat a variety of illnesses due to its many qualities, including antioxidant, anti-proliferative, anti-inflammatory, antiviral, antibacterial, and antifungal effects.[18] Turmeric contains a molecule called curcumin, which inhibits the generation of inflammatory mediators by controlling several signaling pathways, including NF-κB, MAPK, AP-1, and NLRP3 inflammasome.[20]

Fig. 2: Turmeric.

NIGELLA SATIVA

Active phytochemical, the main ingredient in Nigella sativa seeds, thymoquinone, protects organs from oxidative damage brought on by different diseases that produce free radicals.[20] Nigella sativa, also known as black seed or black cumin, is an annual plant with a number of therapeutic uses. It has been demonstrated that a variety of N. sativa's active ingredients, including thymoquinone (TQ) (30%–40%), alkaloids (nigellicines and nigelledine), saponins (alpha-hederin), flavonoids, proteins (26.7%), fatty acids, and others, improve vitiligo patients' therapy when taken together.[24]

Fig. 3: Nigella Sativa.

NEEM: Compounds found in neem have the ability to reduce autoimmune attacks on pigment-producing cells (melanocytes) by calming the immune system. Nimbin and nimbidin, for example, assist minimize skin inflammation, which may prevent the development of depigmented patches. Antioxidants found in neem may shield melanocytes from oxidative damage.

Fig. 4: Neem.

ALOE VERA Gel: Skin Healing and Regeneration:′ Aloe vera is known for its skin healing properties. It promotes cell regeneration, which can be beneficial for vitiligo patients, as it may help in the regeneration of depigmented skin areas and support overall skin health.[16] Anti-inflammatory Effects: Vitiligo can sometimes cause skin irritation or inflammation around the depigmented patches. Aloe vera has′ strong antiinflammatory properties that can help soothe and calm irritated skin, reducing redness and swelling.[16]

Moisturizing: Aloe vera is a natural moisturizer that helps in hydrating the skin. This is especially useful for vitiligo patients who might′ have dry skin. Proper hydration can improve the skin's appearance and texture.

Antioxidant Properties: Aloe vera contains antioxidants like vitamins A, C, and E, which can protect the skin from free radicals and′ oxidative stress. This may help in preventing further damage to the skin and promote the overall health of skin cells. Lightening and Evening Out

Fig. 5: Aloe Vera.

Skin Tone: Aloe vera contains compounds that can help in lightening dark spots and even out skin tone,′ which may be beneficial for people with vitiligo, particularly in areas where the skin has developed patches of different pigmentation.[16]

Supports Immune System: Aloe vera has immunomodulatory effects, meaning it can help regulate immune responses. This is relevant in′ vitiligo because it is an autoimmune disorder where the immune system attacks melanocytes (pigmentproducing cells). Aloe vera may help modulate immune function to support skin health.[16]

8. Cream: "A semisolid dosage form containing one or more drug substances dissolved or dispersed in a suitable base" is the definition of cream. Compared to real ointment, creams are semi-solid emulsions of oil with a lighter body and softer consistency.

Creams are homogenous, semi-fluid substances that are applied externally to the skin or specific mucous membranes for preventative, therapeutic, or protective reasons, particularly where occlusive dispersions of effect are not required. Usually made up of solutions or one or more medications in appropriate bases, they are semisolids. To create medicines that are practically miscible with the skin secretion, hydrophilic or hydrophobic bases are used in their formulation.[21]

Semisolid Emulsions of either O/W or W/O type

- Oil in Water O/W
- Water in Oil W/O
- Cosmetics cream
- Medicated cream

8.1 Herbal Cream: The newest and safest trend in the beauty and fashion industries is herbal creams. These herbal creams are becoming more and more popular because most people these days prefer natural products over synthetic ones for their personal care to improve their appearance. These products provide the body with nutrients, improve health, and provide satisfaction because they are free of synthetic chemicals.[21]

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