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ADVANCES IN VITILIGO TREATMENT A SYSTEMATIC REVIEW

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ABSTRACT

Vitiligo is a common skin disorder characterized by depigmentation due to the loss of melanocytes. This systematic review examines recent advances in vitiligo treatments, highlighting therapeutic options, their efficacy, and emerging trends in management. Skin cells (melanocytes) are responsible for producing melanin, the substance that gives pigmentation to the skin. This review aims to provide a comprehensive overview of the current state of knowledge about vitiligo. Although there is no specific ethnic group, gender, or skin type that is more prone to vitiligo than others, it can affect anyone. The most commonly prescribed treatments for vitiligo are systemic and topical phototherapy and Immunomodulators such as corticosteroids, calipering inhibitors, and vitamin D analogues.

KEYWORDS: Vitiligo, autoimmunity, immune resident memory Melanocytes, Skin disorder, Repigmentation Treatment.

INTRODUCTION

Vitiligo is a disease that causes the loss of skin color in spots In the Aushooryan era, roughly 2200 B.C., vitiligo was first mentioned in writing under the name Kilăsa. Further, the Egyptian Ebers Papyrus also has information on vitiligo that dates back to 1550 B.C.[1] It can affect the skin on any part of your body. It may also affect hair and the inside of the mouth. Usually, the color of hair and skin is determined by melanin. Vitiligo occurs when the cells that produce melanin die or stop functioning. Like all other skin Disorders, patients with vitiligo are considered social outcasts in various societies.[2] which Has psychological and physical impacts [Vitiligo affects people of all skin types, but it may be more noticeable in people with darker skin. Vitiligo normally marks about 1% of the world residents. It does not embrace racial, sexual or regional differences amongst the people. Certain intelligences propose that incidence of Vitiligo in India, Egypt and Japan is higher. It ranges from 1.25% to 6% of the population. The most communal type is non-segmental generalized vitiligo (referred to as vitiligo), According to various studies From India, vitiligo prevalence among dermatology outpatients ranges between 0.25 and4%, with the states of Gujarat and Rajasthan having a maximum frequency of 8.8%.[3] Males and females are equally affected; however, few studies have indicated a female Predominance, which may be related to women's higher tendency for autoimmune disor-Ders or because women tend to be more concerned with their appearance whenseeking Advice and treatment.[4,5] which presents with extensively dispersed, typically symmetric, as well as progressive lesions. Vitiligo has an articulate impact on the physical as well as mental health of patients, including loss of skin photo protection, compromised cutaneous immunity, as well as an appreciable decrease in quality of life that is unswervingly correlated with the early age of onset. Vitiligo affects approximately 1% of the global population and can significantly impact quality of life. Traditional treatments include topical corticosteroids, phototherapy, and surgical options. Recent advancements aim to enhance repigmentation and address underlying pathophysiology.[6,7]

Symptoms and causes

Vitiligo is loss of skin color. irst shows on sun-exposed areas like Hands, lips, arms and face.

Signs of vitiligo

- Loss of skin colour
- Whitening on your scale and eyebrows or beard
- Change colour of inner layer of eyeball
- Vitiligo can start at any age, nevertheless frequently seems before age 20. It is tough to predict how your Diseases will growth. Sometimes the patches stop forming without treatment. In most cases, pigment loss Suppers and ultimately involves most of your skin. Hardly, the skin gets its color back.

Causes

Vitiligo devlop when the melanocytes die or stop functioning. Melanocytes are nothing but cells producing Melanin. It is pigment that gives color to eye skin and hair.

- Family history (heredity).
- Stressful events
- Vitiligo is not contagious. One person cannot fasten it from alternative.[8,9] - Immune system attacks and

destroys the melanocytes in the skin - Due to Sunburn, stress or exposure to industrial chemicals.

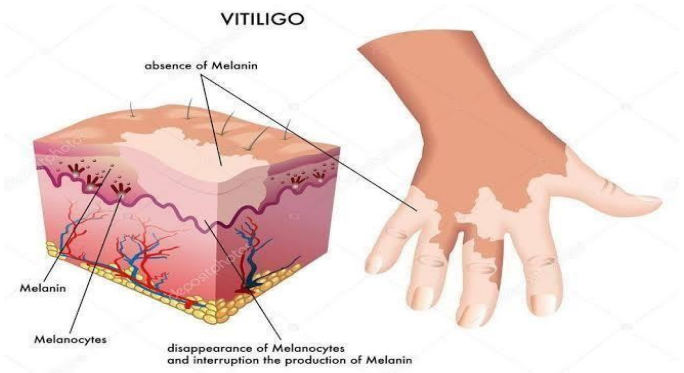

Fig. no. 1: Vitiligo occurs due to absence of melanin.[10]

Pathogenesis of Vitiligo

Pathogenesis of vitiligo is multifactorial, involving genetic, autoimmune, and environmental factors. Melanocytes are attacked and destroyed by autoreactive T cells. This destruction leads to the appearance of depigmented patches. New research also highlights the role of oxidative stress and the inflammatory cascade in disease progression.

METHODOLOGY

A systematic review was conducted using databases such as PubMed, Scopus, and Web of Science. Studies published between 2015 and 2023 were included, focusing on clinical trials, meta-analyses, and reviews. A total of 20 references were analysed.

DIAGNOSIS

Medical History

If your doctor respondents you have vitiligo, he or she will inquire about your medical history, scrutinize you and try to rule out other medical problems, such as dermatitis or psoriasis. He or she can use a special lamp to shine ultraviolet light onto the skin to determine whether you have vitiligo.

Skin Examination and Blood test

-Take a small sample (biopsy) of the affected skin.

-Draw blood for lab tests to look for underlying autoimmune conditions, such as anaemia or diabetes.

-Wood's light is very useful to diagnose the vitiligo.

Current treatment

Many treatments are accessible to benefit restore skin color or even out skin tone. Topical, systemic treatment, and phototherapy are useful for stabilization and repigmentation of vitiligo. Treatment modalitiesare chosen in the individual patient, based on disease severity, disease activity (stable versus progressive disease), patient preference (including cost and accessibility), and response evaluation. For rapidly progressive disease, low-dose oral glucocorticoids and phototherapy are useful in stabilizing the disease. Therapeutic options for stable, segmental vitiligo include topical therapies (eg, topical corticosteroids, topical calcineurininhibitors), targeted phototherapy, and surgical therapy (tissuegrafts and cellular grafts.[11] Some treatments have serious side effects. No drug can stop the process of vitiligo treatment are posible using light therapy. Help to restore some skin tone

- Use of corticosteroid cream
- Depigmentation
- Skin grafting
- Blister grafting
- Tattooing (Micro pigmentation)
- An arthritis drug – Tofacitinib citrate – has shown some promise. It inhibits Janus kinase, an enzyme
- Kapalbhati and various yogas are helpful in the treatment of vitiligo. –Photochemistry.

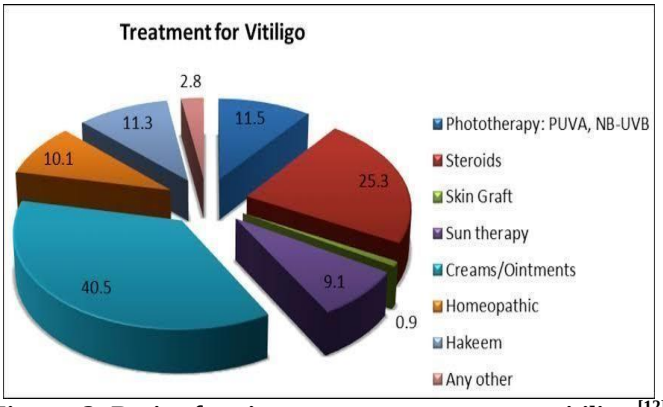

Fig. no. 2: Ratio of various treatment use to treat vitiligo.[12]

Marketed Formulation

- Lukoskin ointment used for treatment of vitiligo. It is developed by DRDO.
- Drug like methoxasalen, triosalen and posarlen are used in treatment of vitiligo with minimum side effects.
- Potent corticosteroids like betamethasone, Momentasone, methoxsalen, clobetasol, betamethsone are used in treatment of vitiligo.
- Ruxolitinib (Opzhelura)
- Afamelanotide (16 mg subcutaneous implant) along with Narrowband UVB has given promising results.[13]

Ayurveda Formulation

- Savarnakara Yoga (Bakuchi, Haratala and Gomutra
- Kanakabindwarishta
- Rasayana Churna
- Manjishthadi Kwatha
- Arogyavardhini Rasa - Vidanga Churna.

1. Systemic Therapies

a. JAK Inhibitors: Recent RCTs show promising results with agents like tofacitinib and ruxolitinib, leading to significant repigmentation, particularly in patients with active disease.[14,15]

b. Immunomodulators: Agents like azathioprine and mycophenolate mofetil have been used, but evidence is mixed regarding their efficacy and safety profiles.[16]

c. Monoclonal Antibodies: Emerging data on anti-PD-1 therapies (e.g., pembrolizumab) suggest a potential role in repigmentation, though longterm safety remains uncertain.

2. Surgical Interventions

a. Skin Grafting: Techniques such as punch grafting and melanocyte-keratinocyte transplantation have shown effectiveness, particularly for stable vitiligo, with variable success rates depending on the technique and site.[17]

b. Laser Therapies: Excimer laser has shown efficacy in localized vitiligo, enhancing repigmentation when combined with topical treatments.[18]

3. Emerging Therapies

a. Gene Therapy: Preliminary studies indicate potential in targeting specific pathways involved in pigmentation.[19]

4. Cytokine-targeted therapies

Multiple monoclonal antibodies are available for vitiligoTreatment, targeting IFN-g, CXCL10, CXCR3, HSP70i, IL-15,IL-17/23, and TNF. In addition to full-size immunoglobulin, Affibodies and nan bodies, composed of considerably smallerProteins, are currently being developed, which have higherBioavailability as well as affinity and specificity to the Targeted molecules.

Stress response in vitiligo

The onset of vitiligo can be instigated by various triggers, including sunburn and exposure to phenolic chemicals; however, the trigger is not known in most cases. The triggers are all thought to induce oxidative stress in melanocytes.[19]

Oxidative stress extends to the endoplasmic reticulum (ER), which is frequently dilated in perilesional melanocytes from patients with vitiligo.[20]

The key role of antioxidants in vitiligo has been suggested by a candidate gene association study, which found a significant association between single nucleotide polymorphism (SNP) rs3565214 within NRF2 and vitiligo in the Chinese population.[21]

The autoimmune component

Autoimmunity has long been suspected to feature significantly in the pathogenesis of vitiligo, and multiple studies published in the past few years increasingly shed light on the role of the immune system in vitiligo. CD8 + T cells specific to, and capable of killing, melanocytes are increased in the blood of those with vitiligo compared to healthy controls, and numbers correlate with disease activity. Using an engineered mouse model of vitiligo, Harris and coworkers had previously found that interferon (IFN)-γ played a central role in the spread of vitiligo lesions.[22]

Specifically, they showed that IFN-γ caused an increase in the expression of CXCL10, a chemokine which regulates the invasion of epidermal and follicular tissues by CD8 + T cells. IFN-γ was also identified as part of a "signature cytokine profile" in an avian model of vitiligo. The Smyth line (SL) of chickens develops a spontaneous, depigmentation disorder that shares several key clinical and pathologic features with human vitiligo. For example, melanocytes that pigment the feathers are lost in an autoimmune-driven process. As the disease progresses, there is an increase in the expression of IFN-γ.[23]

Recently, however, a study by Yang et al. suggested that IFN-γ could play an even more direct role in vitiligo pathogenesis by demonstrating that the IFN-γ derived from cytotoxic T cells could itself cause apoptosis in melanocytes.[24]

DISCUSSION

Recent advancements in vitiligo treatment reflect a shift towards personalized medicine. The introduction of JAK inhibitors and biologics marks a significant evolution, addressing both the cosmetic and psychological impacts of the condition. However, long-term efficacy and safety data are still needed.

CONCLUSION

Advances in vitiligo treatment are promising, with a range of therapeutic options now available. Continued

research is essential to optimize treatment protocols and improve patient outcomes.

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