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Review Article

Vitiligo: A Comprehensive Review of Epidemiology, Cancer Relationand Emerging Treatments

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ABSTRACT

Vitiligo is a persistent depigmenting skin disorder marked by the appearance of white patches on the skin, caused by the loss of Melanocytes. It affects around 0.5-1% of the global population, regardless of age, gender or race. Although vitiligo is not lethal or life-threatening, it does carry a social stigma that lowers self-esteem in people affected. Recent research suggests that vitiligo is a primary autoimmune illness in which the body's immune system destroys pigment-producing cells, Melanocytes, resulting in depigmentation. Additionally, oxidative stress and genetic predisposition mechanisms are known to contribute to the illness. Even though there is no specific cure for vitiligo, it can be effectively treated with suitable therapies. Vitiligo is widely treated with phototherapy, surgery and topical immunomodulators such as calcineurin inhibitors, corticosteroids and JAK inhibitors. Emerging data points to JAK inhibitors as a possible treatment option, however phototherapy remains the cornerstone of vitiligo management. Studies also show a link between vitiligo and cancer risk. This review summarizes current knowledge on vitiligo, addressing myths and misconceptionsas well as, the psychological difficulties associated with vitiligo, highlighting the significance of tailored treatment strategies and increased awareness in reducing the stigma surrounding this condition.

INTRODUCTION

Vitiligo (pronounced "vit-il-EYE-go") is an acquired skin condition characterized by white, depigmented patches on the skin caused by a deficiency of functional melanocytes. Rarely, the eyes and hair may also lose their pigment. While vitiligo spots can develop anywhere on the body, they are more frequently found near the genitalia, orifices and sun-exposed areas such as the hands and face. [1] The condition does not discriminate

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based on skin colour, age, or gender and the exact cause remains unknown. [2] The term "vitiligo" was coined in the second century A.D., by the Roman physician Celsus.It is sometimes referred to as Kilăsa leukoderma.[3]The first written records of vitiligo, under the name date back to the Aushooryan period around 2200 B.C. Additionally, the Egyptian Ebers Papyrus contains references to vitiligo that date back to 1550 B.C. [4]Although vitiligo is not accompanied by unpleasant sensations such as itching or discomfort, it can significantly impact individual's self-esteem and may lead to anxiety or depression as the disfiguring spots can interfere with social interactions. [5]

The disorder can elicit negative emotions such as shame or humiliation, anxiety, and a loss of confidence, and approximately 75% of vitiligo sufferers experience psychiatric issues.[6]Vitiligo can manifest as segmental, affecting only one area of the body, or widespread, affecting larger areas. It may also be classified as stable or unstable, depending on the emergence of new discoloration. [7]Considerable recent progress has been made in understanding the etiology of vitiligo, which is now firmly characterized as an autoimmune disease associated with genetic, environmental, oxidative stress, metabolic factors, and abnormalities in cell detachment. [8]

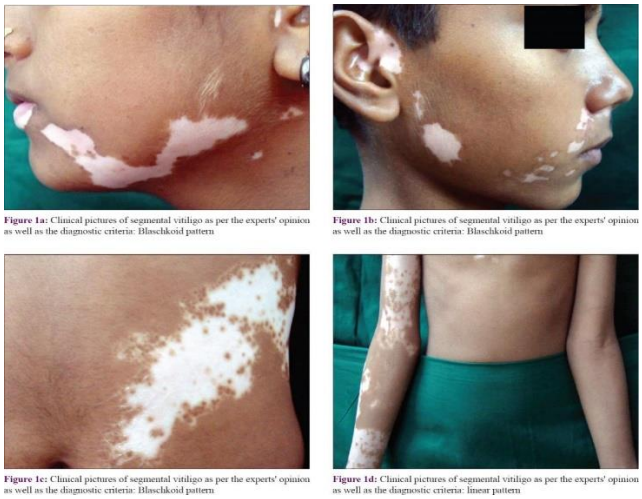

Figure 1: Clinical presentation of Segmental Vitiligo.

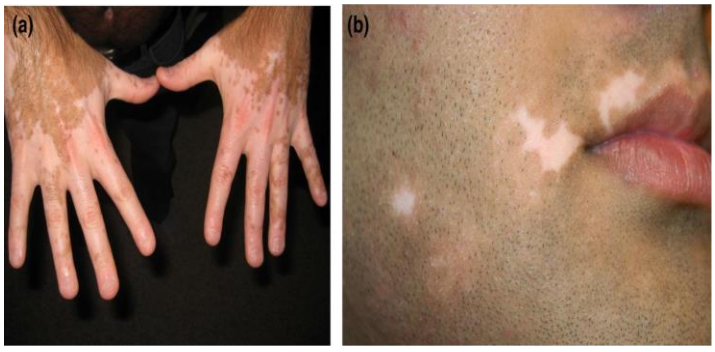

Figure 1: Clinical presentation of Segmental (a) and Non Segmental (b) Vitiligo.

2. Epidemiology: The Reach

Vitiligo affects about 0.5-1% of the global population, regardless of age, gender or skin colour. Vitiligo can affect people of any age, however it is extremely uncommon during birth. The prevalence of Vitiligo varies across different region, with Europe having the highest rates (1.6%), followed by the United States (1.4%) and Japan (0.5%). [9] According to recent investigations, there were 12,709 cases of vitiligo

recorded between 2005 and 2021 (Fig.3).During this time, the total incidence rate rose from 26.3 to 36.8 per 100,000 individuals. Notably, the incidence rates per 100,000 children and adolescents increased from 22.5 and 13.0 in 2005 to 38.1 and 33.0 in 2021, respectively, while the adult rate remained relatively consistent, growing from 29.8 in 2005 to 38.2 in 2021(fig.4).[10]

Vitiligo affects both sexes equally, however there are more reports among females, most likely due to the greater social consequences for women and girls. The majority of cases begin during vigorous growth phases, with approximately half of patients presenting before the age of 20, and 70-80% by the age of 30.[11]It gives clear evident that, younger individuals are more likely to experience active vitiligo than older individuals. The early onset and family history suggest a hereditary component. [12]There is presently no definite cure for vitiligo; however, numerous treatments have shown promising outcomes, with repigmentation occurring in more than 80% of cases. Recent advances in knowledge of its pathophysiology have resulted in new therapy options, pointing to a more promising future for patients.[13]

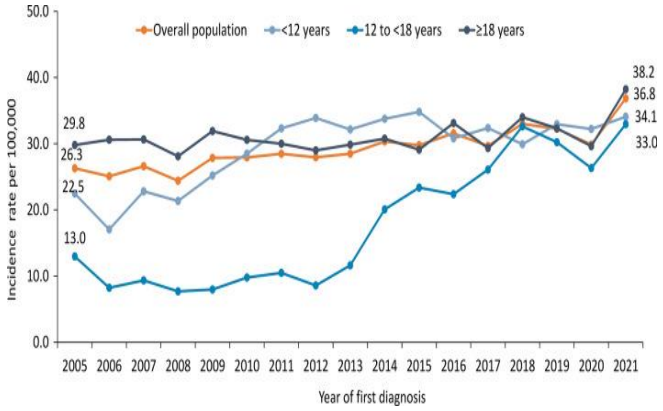

Figure 2: Vitiligo incidence overall and by age group.

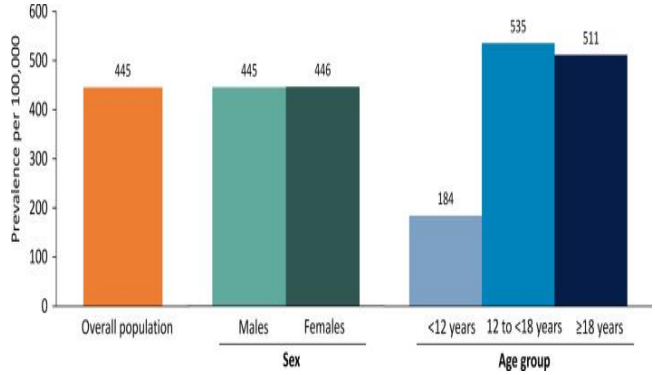

Figure 3: Prevalence of vitiligo in 2021 in overall population, by sex, and by age group

3. Pathophysiology

The Pathogenesis is complex and involves the interplay factors; however, the exact pathogenesis is not well known.

Autoimmune Theory

According to genetic research, 85% of the genes linked to vitiligo susceptibility encode molecules involved in innate and adaptive immunity as well as apoptosis, supporting the autoimmune hypothesis as the primary Pathogenic mechanism of vitiligo. [14]The fact that numerous autoimmune conditions, including autoimmune thyroid disease, pernicious anemia, Addison's disease, systemic lupus erythematosus, are linked to vitiligo provides broad support for this theory. The reasoning behind routine thyroid screening in

vitiligo patients is the strong correlation between vitiligo and thyroid dysfunction and thyroid antibodies. [15] It was discovered that the killing of melanocytes in vitiligo is mediated by CD8+ T lymphocytes that are unique to melanocytes. DCs carry antigenic proteins from stressed melanocytes, which invading T lymphocytes especially identify. There is patchy T cell infiltration at the leading edge of vitiligo depigmentation. [16]Other research showed that IFN-γ generated in the lesional skin mediates the recruitment of CD8 + T cells to active vitiligo lesions. In the skin and blood of vitiligo patients, IFN-γ locally raises CXCL9 and CXCL10 levels, which draw pathogenic T cells that express the CXCR3A receptor. Evidence suggests that some of these cells persist in the afflicted skin as resident memory T cells , which could account for the return of pigmentation loss following vitiligo repigmentation.[17] Since the number of melanocyte-reactive T cells in vitiligo lesions is ten times lower when CXCL9 is absent, it appears to be predominantly responsible for bulk T cell recruitment. The severity of vitiligo remains unaltered in spite of this decrease in T cell count, indicating that T cells are over-recruited during vitiligo. On the other hand, vitiligo incidence and severity are decreased when CXCL10 is absent indicating that T cell localization and potentially activity within the skin depend on CXCL10.[18]

Melanocyte-specific CD8+ T cells are both required and sufficient for the destruction of melanocytes, as evidenced by elegant studies that showed perilesional CD8+ T cells isolated from vitiligo skin could kill melanocytes from normal pigmented skin isolated from the same patient when cultured ex-vivo.[19]

Neural Theory

According to the neural hypothesis, vitiligo arises from peripheral nerve endings in the skin producing more melatonin, a chemical that lightens pigment cells colour and inhibits the production of new melanin. [20]

Biochemical Theory

The reactive oxygen species model in the biochemical theory the oxidative stress theory proposes that the vitiliginous skin's redox (reduction-oxidation) state is out of equilibrium.This causes a significant increase in reactive oxygen species (ROS), such as H_2O_2. When ROS oxidize cell components, melanocytes are destroyed, resulting in the formation of depigmented macules.[21]

Many theories have been put out to explain the pathophysiology of vitiligo. These theories examine the involvement of somatic mosaicism, autoimmunity, microvascular abnormalities, melanocyte adhesion problems, oxidative stress-induced melanocyte degeneration, innervation, and hereditary factors.While some are largely unproven but still being argued, others have substantial evidence to back them up. [22]

4. Vitiligo And Cancer: An Unlikely Connection

"It has been traditionally believed that skin pigmentation is the most critical Photo-protective factor, as melanin not only functions as a broad-spectrum UV absorber but also act radical-scavenger with antioxidant properties. Epidemiological studies consistently show that people with darker skin had a lower incidence of skin cancer than people with fair skin, owing to the fact that UV exposure is a well-established risk factor for skin cancer, and melanin provides protection from damaging UV rays.[23] In the case of vitiligo, the absence of melanocytes led to the assumption that people with this illness are more likely to develop melanoma and non-melanoma skin malignancies due to a lack of this protective pigment. Interestingly, multiple studies have discovered that vitiligo is actually connected with a decreased risk of both melanoma and non-melanoma skin cancers, despite the lack of melanin. While the mechanism is not described

briefly. [24] The Association between vitiligo and cancer has been questioned. Several case reports and research have revealed a link between vitiligo and internal malignancies. Large-scale national cohort studies of cancer risk in vitiligo patients have been rare. Bae et al. found a significant reduction in the risk of internal malignancies in patients with vitiligo (hazard ratio [HR] 0.86), particularly colon and rectal cancer (HR 0.62), lung cancer (HR 0.75), and ovarian cancer (HR 0.62). Li et al. previously demonstrated increased risks of the thyroid (standardized incidence ratio 3.39) in vitiligo patients. [25]Numerous studies suggest that the genetic and immunological profiles of patients with vitiligo may provide significant protection against melanoma and non-melanoma skin cancers. Tyrosinase (TYR) gene polymorphisms were detected in vitiligo patients during genetic examinations. This gene expresses tyrosinase, a melanin-producing enzyme. The TYR mutation, which raises the chance of vitiligo, also lowers the risk of melanoma. Patients with melanoma, on the other hand, have been shown to have a higher 5-year survival rate after developing vitiligo. [26]The decreased incidence of skin cancer in patients with vitiligo may stem from heightened immune surveillance.The primary mediators of this immunological surveillance are Natural Killer (NK) cells. It has been noted that vitiligo patients have higher blood levels of NK cells. High production of granzyme B and proteases is indicative of an active state in these NK cells.Recent research has demonstrated that, in comparison to healthy controls, both lesional and non-lesional skin exhibit higher numbers of NK cells and overexpression of several NK cell genes. One important function of NK cells is to decrease cutaneous squamous cell carcinoma (SCC). [27] Therefore, it is evident that vitiligo is linked to patients increased immunity against melanoma, even though the mechanisms underlying these phenomena are still being studied.[28]

5. Advance Treatment Of Vitiligo

1) Topical Treatment

A) Jak Inhibitors

Currently, rufolitinib cream is the first vitiligo treatment approved by the Food and Drug Administration (FDA) for the topical treatment of non-segmental vitiligo in adults and pediatric patients older than twelve. [29] it's been demonstrated that JAK inhibitors reduce IFN-gamma signaling, which helps vitiligo sufferers regain their pigmentation. The three most widely known JAK inhibitors used to treat vitiligo are tofacitinib (Pfizer, New York, NY, USA), ruxolitinib (Celgene, Summit, NJ, USA), and baricitinib (Indianapolis, IN, USA). [30] Since the JAK-STAT pathway is used by IFN-γ signaling, vitiligo may benefit from JAK inhibitor treatment. For instance, a patient with generalized vitiligo treated with tofacitinib saw nearly total repigmentation of the hands, forearms, and face over the course of five months; however, depigmentation returned after stopping tofacitinib. [31]The JAK/STAT system is a fast membrane-to-nucleus signaling mechanism that triggers the production of several important inflammatory and cancer mediators. A Growing body of research indicates that autoimmune disorders and a number of malignancies are linked to deregulation of the JAK/STAT pathway. [32]

B) Calcineurin Inhibitor

Some of the newest topical medications in dermatology are calcineurin inhibitors, such as tacrolimus and pimecrolimus. But only vitiligo lesions in the head and neck area respond well to tacrolimus and pimercrolimus. [33]

C) Corticosteriods

The primary treatment benefit of corticosteroids for vitiligo is the suppression and regulation of inflammation. First-line treatment for vitiligo is thought to be topical corticosteroids (TCS), which can be either very potent (clobetasol propionate) or potent (betamethasone valerate). Acral regions

typically respond poorly to treatment, whereas sun-exposed areas respond well. [34]

2) Phototheraphy:

The use of sunlight to cure skin conditions has existed from 2000-1400 BC in India and Egypt, where vitiligo was treated with psoralen and the plant Ammi majus in conjunction with sun exposure. This is despite the more recent emergence of focused UV therapies. [35]A specific spectrum of UV light, classified into ultraviolet A (UVA), psoralen ultraviolet A (PUVA), and ultraviolet B (UVB) irradiation ranges, is used in UV phototherapy. UVB contains narrowband UVB (NB-UVB) at 311-313 nm and broadband UVB (BB-UVB) at 280-320 nm. In contrast to other UV treatments, NB-UVB has fewer adverse effects and is more clinically acceptable.[36]

Since phototherapy causes T-cell death, down regulation of inflammatory cytokines, and over expression of interleukin, it is essential for stopping the course of active disease. Additionally, it lowers the quantity of antigen-presenting cells called epidermal Langerhans cells. In stable diseases, phototherapy is also helpful. It promotes the migration and development of melanocytes from hair follicles to the epidermis, which causes keratinocytes to secrete endothelin-1 and basic fibroblast growth factor.[37] Psoralen with UVA (PUVA) was the primary form of photo-chemotherapy for vitiligo for a long time. However, narrowband ultraviolet B (NB-UVB) therapy was more advantageous due to the adverse effects of psoralen, which include nausea, vomiting, photo-toxicity, and photo-carcinogenicity**.** [38] While excimer laser therapy and other topical medications are used to treat localized vitiligo, phototherapyincluding psoralen–UV-A (PUVA) and narrowband UV-B (NB-UVB) therapy—is the main therapeutic option for generalized vitiligo. However, phototherapy might have unsatisfactory results because it requires regular clinic visits and lengthy treatment periods ranging from months to years. [39]

6. MYTHS & Misconseptions:

"Myths and Misconceptions" regarding vitiligo, We can clear up some frequent misconceptions that frequently lead to stigma or ignorance regarding the illness such as social misconceptions, cultural attitudes, inadequate educationand ignorance of the illness. In addition to cosmetically deforming the patient, vitiligo is associated with a number of myths that differ depending on the region of the world. The disease is often misunderstood to be communicable, incurable, linked to a particular food or beverage. (40)

MYTH 1: Adults are primarily affected with vitiligo. [41]

Vitiligo appears in about half of its sufferers before the age of 20.

MYTH 2: Only Skin of colour populations experience the psychological disease burden of vitiligo.[41]Regardless of their skin type, all patients are affected by the psychosocial effects of vitiligo. According to a recent study by Ezzedine et al.6, there are some variations in the issues that patients are most concerned about depending on their skin tone, even if the psychosocial burden of disease is the same for all patients. Darker-skinned patients were more worried about looks, while fair-skinned patients were more worried about the emergence of skin cancer.

MYTHS 3: Poor hygiene is linked to vitiligo. For instance: "Some people think that vitiligo is caused by bad lifestyle choices or inadequate cleanliness. However, vitiligo is completely independent to personal hygiene and is caused by immunological and genetic factorisation. Stressing that vitiligo has nothing to do with lifestyle choices or cleanliness. Numerous problems have been identified by the study as hindring the delivery of safe and efficient therapy for vitiligo. Patients are forced to stop their treatment because they do not

have enough knowledge about the illness and its treatment. Patients experience additional burdens as a result of side effects and drug interactions, which erodes their confidence in getting the prescription. Most people think that vitiligo is an incurable condition. According to this study, the healthcare system shouldn't disregard this illness, and more research in this area is necessary. [42]

CONCLUSION:

Vitiligo is a complex disease that affects not just skin pigmentation but also patientsemotional well-being and social interactions. This review analysed its global occurrence, emphasizing its diverse presentation across different groups and highlighting essential elements of its pathophysiology, such as autoimmune processes, oxidative stress, and genetic predispositions. Treatment advances, such as the development of JAK inhibitors, Phototherapy, and surgical treatments, have given patients new hope for effective treatment. However, obstacles remain in attaining complete repigmentation, long-term stability, and inexpensive access to these treatments. Furthermore, knowing the dual link between vitiligo and cancer emphasizes the importance of taking a cautious but optimistic approach to therapy and research. While medical research advances, cultural views about vitiligo demands equal consideration. Public awareness campaigns, improved educational programs, and support systems can all contribute to dispelling falsehoods, reducing stigma, and promoting diversity. Future research should concentrate on individualized therapy, which targets individual triggers and treatment responses while also investigating the role of environmental variables and genetics. With ongoing scientific developments and an increasing cultural emphasis on acceptance and diversity, there is real hope that people with vitiligo will have access to better care and live without stigma. Medical advancements and social change can work together to provide a better future for those suffering from vitiligo.

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