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CURRENT UNDERSTANDING OF VITILIGO: A COMPREHENSIVE LITERATURE REVIEW

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ABSTRACT

Introduction and Purpose: Vitiligo is a chronic, acquired autoimmune disorder characterized by the selective destruction of melanocytes, leading to the appearance of well-defined depigmented patches affecting the skin and mucous membranes. It is estimated to affect approximately 0.5 – 2% of the global population. Even though it was once regarded as mainly a cosmetic condition, now it is increasingly being recognized as a systemic disease with substantial psychological consequences. This review aims to summarize current knowledge regarding its genetic background, key immunological pathways and the emerging therapeutic approaches, constantly reshaping patient outcomes.

Description of The State of Knowledge: Recent research has highlighted the complex nature of vitiligo pathogenesis, involving oxidative stress, genetic predisposition and immune-mediated mechanisms. In particular, the IFN-γ-chemokine axis, including CXCL9 and CXCL10, appears to play a central role in melanocyte loss. Clinically, Segmental Vitiligo usually follows a unilateral, dermatomal pattern and is often linked to somatic mosaicism, whereas Non-segmental Vitiligo remains as the most common form and typically presents with symmetrical distribution. Diagnostic precision has also advanced, through the introduction of non-invasive imaging techniques, including reflectance confocal microscopy and molecular biomarkers. From a therapeutic perspective, treatment strategies have moved beyond traditional phototherapy and topical agents to targeted interventions, most notably Janus Kinase inhibitors with ruxolitinib, representing one of the most promising options. Surgical procedures may also be considered in carefully selected patients with stable disease.

Conclusions: Despite remaining a challenging dermatological condition, the progress made in understanding the immunological and molecular mechanisms underlying vitiligo, has opened new possibilities for more targeted and effective treatment. The modern approach increasingly combines precise diagnostic evaluation with individualized therapeutic strategies. Effective management now requires a holistic approach that balances both medical and psychological aspects faced by patients.

KEYWORDS

Vitiligo, Depigmentation Disorders, Melanocyte Autoimmunity, JAK Inhibitors, IFN-γ-Chemokine Axis, Quality of Life

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Introduction

Vitiligo is defined as a chronic, acquired depigmenting skin disorder caused by the selective autoimmune destruction of melanocytes. This process results in the development of distinct, milky-white patches on the skin and mucous membranes [1]. Recognized as the most common depigmenting disorder globally, it affects an estimated 0.5% to 2% of the population worldwide, impacting individuals of all age groups with no clear predilection for sex or ethnic background [1]. Despite being historically regarded primarily as a "cosmetic" concern, current clinical understanding now defines vitiligo as a complex, systemic autoimmune disease with potentially significant psychological consequences that can be devastating to a patient's quality of life and interpersonal functioning [1]. For years, inconsistencies in vitiligo terminology and classification complicated clinical practice methods and research. A major international consensus reached in 2011 standardized the nomenclature by defining two primary categories: Non-segmental Vitiligo (NSV) – the more common type presenting as symmetrical and bilaterally distributed patches usually in an acrofacial pattern with potential progression to the generalized or universal form and Segmental Vitiligo (SV) the less common type, generally characterized by unilateral and segmental lesions [2]. SV often features an early onset and abrupt stabilization, whereas NSV is typically associated with a later onset and is strongly linked to systemic autoimmune conditions [2].

Recent years of research have led to a strong shift in the understanding of vitiligo's pathogenesis. The comprehension of the central role of cell-specific autoimmunity, particularly involving the IFN-γ–chemokine pathway has paved the way for specific therapies which were unattainable before [3]. Simultaneously the

implementation of non-invasive imaging, such as reflectance confocal microscopy and ultraviolet photography has enabled precision in assessment and treatment monitoring [4]. Finally, new data suggests that for patients suffering from vitiligo, healthy lifestyle choices regarding vitamin supplementation, diet and sun protection are increasingly important [5]. Despite these advances, vitiligo remains one of the most significant dermatological challenges to this day. This review aims to provide a comprehensive overview of the current understanding of vitiligo, from its intricate genetic and immunological roots to the latest advancements in clinical management and lifestyle-based interventions.

Methodology

This literature review on vitiligo utilized articles published between 2011 and 2025, sourced from the PubMed database. The review focused mainly on studies addressing the pathophysiology, clinical presentation, diagnosis and treatment. A total of 38 articles were analyzed to provide a comprehensive synthesis of current knowledge.

Etiology And Pathophysiology

The pathophysiology of vitiligo is currently explained by the convergence theory, which states that several coexisting mechanisms such as metabolic stress, autoimmune responses and genetic predisposition contribute to the selective destruction of melanocytes [6,7]. Recent years have shown a growth in evidence that highlights the influence of neuroendocrine signaling and epigenetic modifications in shaping these pathogenic mechanisms [8,9]. Vitiligo is considered a polygenic disorder with a complex, genetic architecture. Genome-wide association studies (GWAS) have recognized more than 50 loci associated with the disease. Many of these genes encode proteins connected to melanocyte biology and survival, as well as proteins engaged in immune regulation within both innate and adaptive immunity [10,11]. Aside from inherited genetic variants, the epigenetic modifications such as methylation, histone acetylation and the activity of non-coding RNA play pivotal roles in altering genes responsible for immune balance and melanocyte survival [7,8]. These alterations could serve as a bridge linking environmental exposures and the clinical manifestation of the disease [6,8]

The early phase of vitiligo development is largely associated with oxidative stress within the epidermis. Patients with vitiligo often exhibit a particular sensitivity of melanocytes to reactive oxygen species (ROS), which promotes the accumulation of misfolded proteins in the endoplasmic reticulum while impairing mitochondrial function [7,12]. This oxidative imbalance triggers a cellular stress response and the release of damage-associated molecular patterns (DAMPs), such as heat shock proteins (HSP70i). These molecules activate innate immune receptors, which creates a link between metabolic stress and the inflammatory cascade in contributing to melanocyte damage [3,12].

The main cause of progressive depigmentation in vitiligo is a cell-specific autoimmune response. Among immune cells CD8+ cytotoxic T lymphocytes serve as the main mediators of melanocyte damage. Their migration to the skin is regulated by the interferon-gamma signaling pathway, specifically through the CXCL9 and CXCL10 chemokines [3,11]. After activation these lymphocytes recognize antigens associated with melanocytes and lead to their apoptosis. Although generalized vitiligo revolves around systemic autoimmunity, segmental vitiligo appears to involve a more localized autoimmune mechanism. Current evidence shows a large contribution of somatic mosaicism or interactions between immune and neural pathways in dictating the dermatomal distribution observed in the SV subtype [13,14].

Neural mechanisms also contribute to the clinical manifestations of vitiligo, especially in the segmental form. Disturbances within neuroendocrine pathways involving increased release of neuropeptides such as Substance P and Neuropeptide Y, along with catecholamines can modulate local immune responses while intensifying oxidative stress within the skin [9,13]. Furthermore, in addition to internal factors, environmental triggers, including physical trauma (Koebner phenomenon), and chemicals – particularly phenolic compounds may act as catalytic events that amplify pathogenic mechanisms in genetically predisposed individuals [6,15].

Epidemiology

Recent epidemiological studies have substantially refined our understanding of the global distribution of vitiligo. Earlier estimates generally suggested that the condition affected between 0.5% and 2% of the population. However, findings from the Global Vitiligo Atlas, together with recent systematic reviews, indicate the prevalence of this disease varies considerably between regions and demographic groups, demonstrating a more nuanced distribution of vitiligo across different geographic regions and age groups [4,16].

Vitiligo is the most common cause of acquired depigmentation worldwide. Reported prevalence rates differ markedly between populations, partly because of differences in diagnostic approaches and the populations being studied. Some regions appear to show higher rates of the disease, which could be related to environmental influences, local genetic factors or more frequent clinical recognition in individuals with darker skin phototypes, where depigmented lesions are more readily visible [3,16]. While both sexes are affected equally, women are often more likely to seek medical intervention due to the heightened psychosocial impact in these demographics. Younger patients also tend to present more frequently for treatment, highlighting the substantial psychological burden vitiligo may impose during adolescence and early adulthood [16,17].

Vitiligo can develop at any stage of life, ranging from early childhood to late adulthood, yet half of all affected individuals experience the first clinical signs before the age of 20 [1,2]. In pediatric patients, this disease often demonstrates distinct clinical characteristics. Segmental vitiligo is relatively more common in children, and it often has a stronger correlation with a family history of autoimmune disorders [2,18]. Conversely, epidemiological studies indicate that cases with late-onset vitiligo, which manifests after the age of 50, are more likely to be associated with non-segmental forms. In these patients, vitiligo often concurs with systemic autoimmune pathologies, particularly thyroid dysfunction [1,19].

Modern epidemiological evidence increasingly supports the concept of viewing vitiligo as a systemic disease and not solely a localized skin condition [19]. Individuals affected by this disorder show a higher prevalence of several other autoimmune diseases, most notably Graves' disease, Hashimoto's thyroiditis and type 1 diabetes mellitus [19,20]. Furthermore, recent research has also identified possible metabolic associations, including increased rates of dyslipidemia and insulin resistance in vitiligo patients, which raises the necessity of metabolic screening in certain cases [18].

Surprisingly, epidemiological data also suggest that vitiligo may be associated with reduced risk of certain skin cancers. Large-scale cohort studies have demonstrated lower rates of both melanoma and keratinocyte-derived skin cancers among patients with vitiligo, a phenomenon attributed to the overactive immune surveillance and the IFN-gamma-driven inflammatory pathways [21,22].

Beyond its physical manifestations, vitiligo can have a substantial psycho-social impact. More importantly, the degree of emotional distress experienced by patients often does not correlate directly with the physical extent of the disease, measured by body surface area (BSA). Even limited disease affecting visible areas such as the face and hands may significantly impair self-esteem and social functioning. Meta-analyses indicate that approximately one-third of patients with vitiligo also suffer from anxiety disorders, with significantly elevated rates of depression compared to the general population [17,23]. The impact on quality of life is particularly severe in patients with easily visible lesions or those affecting sensitive areas, such as the genital region – which can further contribute to sexual dysfunction, relationship difficulties and social withdrawal [17,24].

Clinical Presentation

Vitiligo typically manifests as sharply defined areas of depigmentation that appear chalk-white or milky in color. These macules and patches are usually asymptomatic and result from the total loss of epidermal melanocytes, which may appear on any part of the body surface. Although certain locations are affected more frequently, including periorificial regions, the dorsum of the hands, and areas exposed to repeated mechanical stress, this is often described as the Koebner phenomenon [1,2,6]. According to the International Vitiligo Consensus, vitiligo can be broadly divided into two principal clinical patterns. Non-segmental vitiligo represents the most prevalent form. It generally displays a bilateral and often symmetrical distribution. Several subtypes are listed within this category, including generalized, acrofacial, mucosal and universal forms. In universal vitiligo, depigmentation may involve more than 80 – 90% of the body surface area (BSA) [2,3,6]. On the other hand, Segmental Vitiligo is distinguished by lesions with a unilateral distribution, frequently following a dermatomal or quasi-dermatomal pattern. This form often develops rapidly and tends to affect the hair follicle pigment reservoir early in the course of the disease, which may lead to premature leukotrichia [13,14]. In some patients, a mixed pattern can develop when non-segmental lesions appear in individuals who previously presented with segmental lesions [6,13].

A critical aspect of clinical evaluation is determining whether the disease is currently progressing or has entered a stable phase. Active vitiligo may be suggested by several characteristic morphological features. These include "confetti-like" depigmentation, consisting of numerous small satellite macules near lesion margins, as well as a "trichrome pattern", where intermediate zones of lighter pigmentation are seen between normal skin and completely depigmented areas. In some rare cases, faint inflammatory borders may also be observed [1,3,6]. These morphological markers are thought to correspond to increased infiltration of cytotoxic

CD8+ T lymphocytes at the expanding edges of lesions [3,11]. Conversely, stable vitiligo is generally defined by the absence of new lesions and the lack of enlargement of existing ones for at least 6 – 12 months. Establishing stability is particularly important when considering surgical therapeutic approaches [25, 26,27].

Although vitiligo is primarily recognized as a disorder affecting the skin, evidence increasingly suggests that the disease may involve pigment-containing cells in other tissues as well. Ocular changes such as iris depigmentation or uveitis have been reported, but are often asymptomatic in presentation [19,28]. A separate but related entity is chemical-induced vitiligo, resulting from exposure to certain phenolic or catecholic compounds. These lesions can mimic generalized NSV, despite underlying mechanistic differences [15]. In addition, vitiligo-like patches have been described as an adverse effect of some modern immunomodulatory treatments. Cases have been reported in patients receiving biologic agents, particularly TNF-α inhibitors and immune checkpoint inhibitors, highlighting the complex relationship between immune regulation and melanocyte survival [29].

Diagnostic Approach

The diagnosis of vitiligo is primarily based on clinical evaluation, yet in recent years it has increasingly incorporated additional diagnostic tools, advanced imaging and molecular biomarkers to help improve accuracy and allow better monitoring of disease progression and treatment outcomes. While careful physical examination remains the key element of assessment, the use of a Wood's lamp (365 nm) is broadly used in routine practice to enhance the contrast of depigmented lesions. It can reveal early or subclinical patches, not yet easily visible under normal lighting conditions, while also helping differentiate vitiligo from other hypopigmented dermatoses [1,2]. To obtain more detailed information about the skin changes, non-invasive imaging techniques such as dermoscopy and reflectance confocal microscopy are increasingly utilized. These methods function as in-vivo microscopic assessments that demonstrate inflammatory changes at lesion borders. They can also evaluate whether a functional melanocyte reservoir remains in hair follicles, or surrounding tissue [4,27]. In both research settings and clinical practice, standardized assessment scales, such as the Vitiligo Area Scoring Index (VASI), which estimates the extent of depigmentation and the Vitiligo Signs of Activity (VSA), which focuses on indicators of ongoing progression, are commonly used to objectively measure disease severity and activity [2,22,27]. Furthermore, modern diagnostic strategies also extend beyond the skin itself. They now incorporate patient evaluation via systemic screening for associated metabolic or autoimmune conditions, including thyroid-stimulating hormone (TSH) and fasting glucose levels. At the same time molecular biomarkers, particularly CXCL10, have garnered attention, which serves as a highly specific indicator of IFN-γ mediated immune activity and may provide useful information regarding the chances of response to targeted treatments and disease activity [3,18,30].

Treatment

Management strategies for vitiligo have evolved considerably in recent years, shifting from empirical symptom targeting toward treatments that aim at specific pathogenic pathways. The choice of therapy depends mainly on disease activity, distribution and overall extent of skin involvement. Initial management of localized or limited lesions typically utilizes potent topical corticosteroids or topical calcineurin inhibitors. These treatments are often used in synergy with narrow-band ultraviolet B (NB-UVB) phototherapy, which overall remains the gold standard of treatment for generalized vitiligo because it promotes melanocyte proliferation and migration to the hair follicle reservoir, resulting in repigmentation [25,30,31,32].

Supportive therapies are usually added to improve treatment response. Topical vitamin D analogues and systemic antioxidants are used as adjuncts with the aim of reducing oxidative stress and improving the cutaneous microenvironment, which should lead to improved melanocyte survival and recovery [7,12,33]. However, in recent years, the introduction of targeted immunomodulatory agents has been the most significant advance in the current treatment approach. In particular, inhibitors of the Janus kinase (JAK) pathway, most notably ruxolitinib, which disrupts the IFN-γ-CXCL10 signaling axis, that drives cytotoxic T-cell activity against melanocytes, resulting in slowing disease progression and promoting repigmentation [3,30,34,35]. For patients with disease resistant to medical therapies but in a stable phase, defined by the absence of new lesions or Koebner phenomenon for at least 12 months, surgical interventions could be taken into consideration. Procedures such as autologous melanocyte-keratinocyte transplantation (MKTP) or suction blister grafting aim to offer a new path for restoring pigment in areas where the natural follicular melanocyte reservoir is absent or insufficient [25,26,27]. Additional methods, including microneedling and laser-assisted drug delivery are constantly being explored as new ways to enhance positive treatment outcomes. Alongside these medical and procedural interventions, comprehensive management should also take into account lifestyle factors and psychological well-being, given the significant psychosocial burden associated with the disease [5,36,37,38].

Conclusions

Over the past decade, the understanding of vitiligo has changed substantially. Rather than being considered only a cosmetic condition, it is now recognized as a complex autoimmune disease involving genetic susceptibility, oxidative stress and immune mediated melanocyte destruction by CD8+ T cells. Recent research has identified the IFN-γ-CXCL9/10 signaling pathway as a key mechanism in disease progression and an important therapeutic agent. This has contributed to a shift toward more targeted treatments, such as Janus Kinase inhibitors, mainly ruxolitinib, which has shown promising repigmentation results in patients unresponsive to conventional therapies. Modern management also emphasizes more comprehensive evaluation, including non-invasive imaging techniques and screening for associated autoimmune or metabolic disorders. Because vitiligo affects approximately 0.5 – 2% of the global population and often has a significant psychosocial impact, effective treatment should address both clinical symptoms and patients' quality of life. And even though challenges remain, ongoing advances in targeted therapies and surgical techniques have improved treatment options and may lead to more personalized approaches in the future.

Disclosure

Authors Contribution:

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