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MODERN APPROACHES TO THE TREATMENT OF VITILIGO: INNOVATIVE METHODS AND THEIR CLINICAL APPLICATION

Djanaev Gayrat Yusupovich
Associate Professor, Department of Pharmacology,
Tashkent State Medical University
Ravshanova Mubinabonu Sanjar kizi
Abdusalomova Gulsevara Olimjon kizi
Mekhmonova Zarina Davlatovna
Erkaboyeva Zuhra Davlatali kizi
Ungbayeva Sabrina Adham kizi
Students, Tashkent State Medical University

Abstract

This article provides a comprehensive analysis of the etiology, pathogenesis, and clinical features of vitiligo, as well as modern therapeutic approaches developed in recent years. Particular attention is given to advanced treatment strategies, including Janus kinase (JAK) inhibitors (ruxolitinib, baricitinib, povorcitinib), narrowband UVB phototherapy, cellular transplantation using the RECELL system, and gene therapy. The review also evaluates the efficacy and safety of these interventions based on recent global clinical trials involving patients with vitiligo. The advantages and limitations of emerging therapeutic modalities are discussed, and their role in contemporary clinical practice is critically assessed. This work serves as a relevant source of information for specialists in dermatology and immunology, as well as for researchers engaged in scientific investigations in this field. In addition, current developments in diagnostic and therapeutic strategies are highlighted, including phototherapy (PUVA and narrowband UVB), novel treatment approaches developed in South Korea (JAK inhibitors), regenerative and transplantation techniques (RECELL), immunotherapy, and melanocyte stimulation methods.

Keywords: Vitiligo, autoimmune disease, melanocytes, JAK inhibitors, ruxolitinib, phototherapy, RECELL, depigmentation, genetic factors, oxidative stress, modern therapy, regenerative medicine, dermatology

Introduction

Vitiligo is a chronic dermatological disorder characterized by the appearance of depigmented patches on the skin due to the loss of melanocytes, the cells responsible for melanin production. The disease affects individuals of all age groups worldwide and has significant cosmetic and psychological consequences.

Currently, there is no definitive cure for vitiligo; however, modern medicine offers several effective strategies aimed at disease control and repigmentation. This article focuses on the analysis of contemporary therapeutic approaches and their clinical applications in the management of vitiligo.

Pathogenesis and Key Mechanisms of Vitiligo

Understanding the pathogenesis of vitiligo plays a crucial role in the development of modern therapeutic strategies. The primary pathological mechanism involves the destruction or dysfunction of melanocytes, leading to impaired melanin production in the skin.

Several key factors contribute to the development of this process:

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Autoimmune reactions: The immune system targets and destroys melanocytes, primarily through cytotoxic T-cell activity.

Genetic predisposition: Vitiligo often exhibits familial aggregation, suggesting a hereditary component.

Environmental factors: External triggers such as stress, ultraviolet radiation, and chemical exposures may initiate or exacerbate the disease.

Modern Therapeutic Approaches

Currently, several advanced therapeutic strategies are available for vitiligo management. These approaches aim to slow disease progression, restore pigmentation, and improve patients' quality of life [2].

Topical Corticosteroids

Potent topical corticosteroids, such as betamethasone and clobetasol, are widely used in the treatment of vitiligo. These agents help restore melanocyte activity and slow the progression of depigmented lesions. They are particularly effective in newly developed patches.

Application: Applied to the affected skin once daily or several times per week [3].

Phototherapy (UVB Therapy)

Narrowband UVB (NB-UVB) phototherapy is one of the most effective and commonly used treatment modalities for vitiligo. In this approach, the skin is exposed to controlled ultraviolet B radiation, which stimulates melanocyte proliferation and enhances repigmentation.

Treatment regimen: Typically administered 2–3 times per week, with a treatment duration of 3–6 months.

Efficacy: NB-UVB therapy is particularly effective in cases involving extensive skin areas.

Photochemotherapy (PUVA Therapy)

Psoralen plus ultraviolet A (PUVA) therapy involves the administration of psoralen followed by exposure to UVA radiation.

Indications: Primarily used in generalized forms of vitiligo. However, due to the increased risk of phototoxicity and skin damage, PUVA therapy should be applied with caution.

Surgical Approaches

Melanocyte Transplantation

This technique involves the transfer of melanocytes from unaffected skin areas to depigmented regions. It is recommended for stable vitiligo cases, where lesions have remained unchanged for several years.

Skin Grafting

Skin grafting demonstrates high success rates and is commonly used for small to medium-sized vitiligo lesions.

Pharmacotherapy: JAK Inhibitors

Janus kinase (JAK) inhibitors represent a novel class of therapeutic agents currently being investigated for vitiligo treatment. Drugs such as tofacitinib and ruxolitinib have shown promising results in clinical studies.

Mechanism of action: JAK inhibitors modulate the immune response, promote melanocyte survival, and facilitate repigmentation by targeting inflammatory pathways.

Depigmentation Therapy

Depigmentation therapy, also known as skin lightening, is used in patients with extensive vitiligo to achieve uniform skin coloration by removing the remaining pigment.

This approach is generally reserved for severe cases where other treatment modalities have failed.

Future Perspectives and Emerging Research

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Recent advances in vitiligo research focus on innovative therapeutic strategies, including targeted immunotherapy, regenerative medicine, and gene-based approaches [4].

Etiology

Vitiligo is a chronic pigmentary disorder characterized by the loss of color in the skin, hair, and other pigmented tissues, primarily due to the destruction or dysfunction of melanocytes. The etiology of the disease is complex and multifactorial, and several major theories have been proposed to explain its development [5].

Autoimmune Theory

The autoimmune mechanism plays a central role in the etiology of vitiligo. In this process, the body produces autoantibodies against its own melanocytes, and cytotoxic T cells contribute to their destruction. This condition is often associated with other autoimmune diseases such as Hashimoto's thyroiditis, type 1 diabetes mellitus, and others.

Genetic Predisposition

Approximately 20–30% of patients with vitiligo have a positive family history, indicating a genetic component. Mutations in genes such as HLA, NLRP1, PTPN22, TYR, and other immune-regulating genes increase susceptibility to the disease. Vitiligo is considered a polygenic disorder [6].

Oxidative Stress Theory

Reactive oxygen species (ROS) play a significant role in melanocyte function. When antioxidant defense systems are impaired, excessive ROS accumulation leads to cellular damage and melanocyte destruction.

Neurogenic Factors

Neuroimmune interactions between the skin and the central nervous system also contribute to the development of vitiligo. Certain neuropeptides and mediators, such as norepinephrine, may disrupt melanocyte function.

Environmental and Occupational Factors

Exposure to phenolic compounds, resorcinol derivatives, ultraviolet radiation, and mechanical injury (Koebner phenomenon) can exert toxic effects on melanocytes and trigger disease onset.

Thus, the etiology of vitiligo results from a combination of immunological, genetic, oxidative, and environmental factors. The relative contribution of each factor may vary among individuals [7].

Epidemiology

Vitiligo is a globally prevalent condition affecting individuals of all races, genders, and age groups. The worldwide prevalence ranges from approximately 0.5% to 2%, with higher rates reported in certain regions such as Africa and India, where prevalence may reach 3–4%. Although vitiligo can develop at any age, approximately 60–70% of cases are diagnosed between the ages of 10 and 30. Onset before the age of 12 is referred to as early-onset vitiligo [8]. The disease affects males and females equally; however, women are more likely to seek medical attention due to cosmetic concerns. A positive family history is observed in 20–30% of cases, indicating a genetic predisposition. Additionally, 10–20% of patients have concomitant autoimmune diseases such as thyroid disorders, alopecia areata, or type 1 diabetes mellitus. In recent years, increased awareness and improved diagnostic capabilities have led to a rise in the detection rate of vitiligo [9].

Pathophysiology

The pathophysiology of vitiligo is complex and involves multiple interrelated processes leading to the loss of melanocytes in the skin. The main mechanisms include:

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Autoimmune Reactions

The autoimmune response is considered the primary mechanism in vitiligo pathogenesis. Cytotoxic T lymphocytes, particularly CD8+ T cells, target and destroy melanocytes, resulting in depigmentation. Autoantibodies against melanocytes have also been identified.

Oxidative Stress

During melanin synthesis, melanocytes produce reactive oxygen species. If antioxidant defenses are insufficient, these molecules cause cellular damage and apoptosis, contributing to disease initiation and progression.

Genetic Factors

As a polygenic disorder, vitiligo involves multiple genes, including HLA, NLRP1, PTPN22, and TYR. These genes regulate immune responses, oxidative stress defense, and melanogenesis.

Neurogenic Mechanisms

Neuroimmune interactions influence melanocyte function through mediators such as neuropeptides and norepinephrine. These mechanisms are particularly relevant in segmental vitiligo.

Apoptosis and Autophagy Dysregulation

Recent studies have demonstrated increased apoptotic activity in melanocytes. In addition, dysfunction of autophagy—the cellular system responsible for removing damaged components—contributes to melanocyte loss.

These mechanisms collectively lead to melanocyte destruction, functional impairment, and subsequent depigmentation of the skin. The extent to which each mechanism contributes varies among patients and determines disease severity and clinical presentation [10].

Histopathology

The histopathological features of vitiligo are identified through microscopic examination of skin biopsy specimens obtained from depigmented areas. These changes are primarily characterized by a reduction in the number or complete absence of melanocytes. The main histological findings include the following:

Loss of Melanocytes

Melanocytes located in the basal layer of the epidermis are either completely absent or significantly reduced in number. This finding can be confirmed using immunohistochemical staining techniques, such as HMB-45, Melan-A, and S100 markers [11].

Loss of Pigmentation

There is a marked reduction or complete absence of melanin pigment in the epidermis. This decrease can be demonstrated using special stains such as the Fontana–Masson stain.

Inflammatory Changes

In the active stage of the disease, mild interface dermatitis may be observed, characterized by lymphocytic infiltration in the basal layer, particularly involving CD8+ T cells. These immune cells exert cytotoxic effects on melanocytes.

Preservation of Epidermal Architecture

Despite the loss of melanocytes, the overall structure of the epidermis remains preserved. Features such as acanthosis, parakeratosis, and spongiosis are typically absent. The primary histopathological alteration is the loss of pigment-producing cells [12].

Dermal Changes

In some cases, mild perivascular lymphocytic infiltration may be observed in the papillary dermis; however, this finding is not consistently present.

Diagnostic Significance of Histopathology

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Histopathological examination plays a crucial role in the diagnostic process, particularly in differentiating vitiligo from other depigmenting disorders such as post-inflammatory depigmentation, pityriasis alba, and lepromatous lesions.

Vitiligo is an autoimmune disorder characterized by depigmentation of the skin, and skin biopsy remains an essential tool for confirming its histopathological features.

The key histopathological characteristics include:

Loss or marked reduction of melanocytes in the basal layer

Decrease or absence of melanin pigment

Mild lymphocytic infiltration in active lesions

Preservation of overall epidermal structure

Given that vitiligo may clinically resemble other dermatological conditions, histopathological analysis is essential for accurate differential diagnosis and appropriate clinical management [13].

Treatment

In recent years, several novel approaches have been developed for the treatment of vitiligo. In modern medical practice, the following methods are widely used in the management of this condition:

Phototherapy (PUVA and Narrowband UVB)

Phototherapy, particularly narrowband UVB (NB-UVB), is considered one of the most effective and safest treatment modalities for vitiligo. NB-UVB therapy utilizes ultraviolet light with a wavelength of 311 nm, which stimulates melanocyte activity and promotes repigmentation.

According to international studies, NB-UVB therapy has demonstrated a success rate of approximately 60–80% in many patients (*Dermatology Research and Practice*, 2023). However, there are differing opinions regarding the long-term safety and duration of PUVA and NB-UVB therapies, as prolonged exposure may increase the risk of skin malignancies (*PubMed*, 2022).

Topical Medications

Topical corticosteroids, tacrolimus, and pimecrolimus are commonly used immunosuppressive agents in vitiligo treatment. These medications reduce excessive immune responses targeting melanocytes and facilitate the restoration of skin pigmentation.

Tacrolimus has shown significant efficacy in several clinical trials, particularly in patients with mild to moderate vitiligo (*JAMA Dermatology*, 2021).

However, prolonged use of topical corticosteroids may lead to skin atrophy; therefore, they are recommended primarily for short-term use (*British Journal of Dermatology*, 2022).

Emerging Therapeutic Approaches from South Korea (JAK Inhibitors)

One of the most promising modern therapeutic strategies involves the use of Janus kinase (JAK) inhibitors. These agents regulate immune system activity, protect melanocytes, and promote repigmentation.

JAK inhibitors such as ruxolitinib and tofacitinib have demonstrated encouraging clinical outcomes in patients with vitiligo, particularly in segmental forms of the disease (*JAMA Dermatology*, 2022).

Regenerative and Transplantation Techniques (RECELL Technology)

RECELL technology, which involves melanocyte regeneration and transplantation, has emerged as an effective treatment option in recent years. This method uses small skin samples from the patient to isolate melanocytes, which are then applied to depigmented areas.

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Clinical studies have reported significant repigmentation in 70–80% of patients, especially in facial and hand regions. This technique is notable for its minimal adverse effects (*Clinical, Cosmetic and Investigational Dermatology*, 2023).

Immunotherapy and Melanocyte Stimulation

Immunotherapy and melanocyte-stimulating techniques are also increasingly used in vitiligo treatment. These approaches aim to prevent melanocyte destruction and restore their pigment-producing function.

Platelet-rich plasma (PRP) therapy and stem cell-based treatments have been investigated in multiple studies and have shown promising results in melanocyte regeneration (*European Journal of Dermatology*, 2022).

Modern Pharmacological Approaches

Recent research has focused on novel pharmacological agents, including bimatoprost (a prostaglandin analog) and calcipotriol (a vitamin D analog). These agents contribute to melanocyte stimulation and repigmentation of the skin (*International Journal of Dermatology*, 2021).

Modern Approaches to Vitiligo Treatment: Evidence from International Literature

Recent international studies highlight significant progress in the development of innovative therapeutic strategies for vitiligo. Modern clinical practice increasingly incorporates phototherapy, immunomodulatory agents, regenerative technologies, and targeted molecular therapies as part of a comprehensive treatment approach [14].

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Conclusion

Vitiligo is a complex autoimmune disorder in which the immune system targets and destroys melanocytes, leading to the development of depigmented patches on the skin. In recent years, significant progress has been made in understanding the pathogenesis of vitiligo and in developing novel therapeutic approaches.

Janus kinase (JAK) inhibitors, particularly ruxolitinib (topical) and tofacitinib (oral), have demonstrated promising results in the treatment of vitiligo. Clinical studies have shown that combining these agents with phototherapy significantly enhances repigmentation outcomes. For instance, a 52-week clinical study involving upadacitinib reported that 63% of patients achieved at least 75% facial repigmentation.

Afamelanotide, an analog of α-melanocyte-stimulating hormone, has also shown efficacy in enhancing repigmentation when used in combination with narrowband UVB phototherapy, especially in individuals with Fitzpatrick skin phototypes IV–VI.

In addition, phosphodiesterase-4 (PDE-4) inhibitors, such as apremilast, are being explored as potential therapeutic options due to their anti-inflammatory effects, although current evidence regarding their efficacy remains limited.

According to the 2023 recommendations of the International Vitiligo Task Force, treatment strategies should be individualized based on disease activity and extent. In active vitiligo, a combination of topical therapy, phototherapy, and/or systemic treatment is recommended, whereas in stable cases, maintenance therapy plays a key role in preventing relapse.

Furthermore, addressing the psychological impact of vitiligo is essential. Psychological support and increasing public awareness are important components of comprehensive patient care.

In conclusion, substantial advances have been achieved in vitiligo management through immunomodulatory therapies, phototherapy, and targeted molecular approaches. However, further large-scale studies are required to evaluate the long-term efficacy and safety of these treatment modalities.

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