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FROM PATHOGENESIS TO FUTURE CURE: A COMPLETE REVIEW OF VITILIGO AND ITS RECENT ADVANCES

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Abstract
Vitiligo is a chronic, acquired skin disorder marked by progressive depigmentation resulting from the selective loss of melanocytes. Though non-life-threatening, it poses significant psychosocial challenges due to its visible impact, particularly in darker-skinned populations. The etiology is multifactorial, with contributions from genetic predisposition, autoimmunity, oxidative stress, and environmental triggers. Vitiligo is now considered a model autoimmune disorder due to its well-characterized immunopathogenesis involving $CD8^+$ T cells, cytokines like IFN-γ, IL-15, and TRM cell persistence. Clinical classification into segmental and non-segmental types, along with diagnostic tools such as Wood's lamp examination, dermoscopy, and scoring systems like VASI and VIDA, aids in tailored management. Therapeutic approaches range from topical corticosteroids and calcineurin inhibitors to phototherapy (NB-UVB, PUVA) and systemic immunosuppressants. Surgical techniques like melanocyte transplantation offer hope for stable vitiligo. Recent advances emphasize targeted immunotherapies (e.g., JAK inhibitors, IL-15 blockade), regenerative strategies using stem cells and melanocyte precursors, and novel delivery systems like nanocarriers and microneedles. Gene therapy and Wnt/β-catenin pathway modulation represent promising futuristic approaches. Preventive strategies include minimizing trauma (Koebner phenomenon), managing psychological stress, antioxidant support, and early intervention. This review integrates classical knowledge with cutting-edge developments, offering a comprehensive perspective on vitiligo. It emphasizes the need for a multidisciplinary and patient-centric approach that combines clinical, psychosocial, and technological strategies. As research deepens, the path toward a potential cure becomes more tangible, supported by innovations in immunology and molecular medicine.
Keywords: Vitiligo; Melanocyte destruction; Autoimmunity; Oxidative stress; Phototherapy; JAK inhibitors; Stem cell therapy; Gene therapy; Clinical classification; Immunomodulation

INTRODUCTION

Vitiligo is an acquired, idiopathic, and often progressive disorder of pigmentation characterized by the selective loss of functional melanocytes from the epidermis, leading to the appearance of well-demarcated, depigmented macules and patches on the skin, mucosa, and occasionally hair. Melanocytes are responsible for melanin production-the pigment that determines skin, hair, and eye color-and their destruction results in the characteristic milky-white lesions seen in vitiligo [1]. While the condition itself is not life-threatening or contagious, it is often chronic, unpredictable in its course, and can significantly impact quality of life, especially in visible or extensive cases.

Globally, vitiligo affects approximately 0.5% to 2% of the population, with no significant gender predilection. However, its psychosocial burden is more pronounced in individuals with darker skin tones due to higher contrast between affected and unaffected areas. The condition can manifest at any age, but the majority of cases occur before the age of 30. Its prevalence varies by geographic region and ethnicity, with a slightly higher incidence reported in India, Mexico, and certain African countries. Vitiligo can also be associated with autoimmune disorders such as autoimmune thyroiditis, type 1 diabetes, alopecia areata, and pernicious anemia, further complicating its clinical management [2].

The psychosocial implications of vitiligo are profound. Patients often report low self-esteem, anxiety, depression, social withdrawal, and even stigmatization in certain cultural contexts. The cosmetic disfigurement caused by vitiligo can interfere with interpersonal relationships, employment, and overall psychological well-being,

necessitating a multidisciplinary approach that includes both medical and psychological support [3, 4].

Given the complex pathogenesis involving genetic predisposition, immune dysregulation, oxidative stress, and environmental triggers, vitiligo has emerged as a model disease for understanding autoimmune skin disorders. The objective of this review is to provide a comprehensive and updated synthesis of current knowledge on vitiligo, ranging from its etiology and pathophysiology to diagnostic criteria and therapeutic strategies. Furthermore, the review highlights recent advancements in immunotherapy, gene editing, and innovative drug delivery systems that offer promising directions for future curative treatments. By integrating clinical experience with cutting-edge research, this review aims to serve as a valuable resource for clinicians, researchers, and healthcare professionals involved in the management of vitiligo.

ETIOLOGY AND RISK FACTORS

Vitiligo is a multifactorial disorder with a complex etiology involving the interplay of genetic, environmental, oxidative, psychological, and autoimmune components. Understanding these contributing factors is essential for comprehending disease onset, progression, and potential therapeutic targets [5].

Genetic Predisposition

Genetic factors play a crucial role in the pathogenesis of vitiligo. Family studies have shown that up to 30% of patients report a positive family history. Genome-wide association studies (GWAS) have identified multiple susceptibility loci, including genes involved in immune regulation and melanocyte function. Notable genes include HLA-DQB1, PTPN22, NLRP1, FOXP3, and TYR, which influence both autoimmunity and pigmentation pathways. These genetic variations do not directly cause vitiligo but predispose individuals to heightened immune responses against melanocytes [6].

Environmental Triggers

Environmental factors are believed to act as precipitating agents in genetically susceptible individuals. Ultraviolet (UV) radiation, chemical exposures (such as phenols and catechols in hair dyes or industrial products), and physical trauma (Koebner phenomenon) have been implicated in the initiation or exacerbation of vitiligo. These triggers may induce oxidative stress or alter melanocyte antigenicity, making them targets of immune attack [7].

Role of Oxidative Stress

A pivotal factor in vitiligo pathogenesis is the accumulation of reactive oxygen species (ROS) within melanocytes. Inadequate antioxidant defences-due to decreased levels of catalase and glutathione peroxidise-render melanocytes particularly vulnerable to oxidative damage. This oxidative stress not only impairs melanocyte function and viability but also serves as a signal for activating the innate and adaptive immune responses against these cells [8].

Psychological Stress and Neurogenic Factors

Psychological stress has long been considered a potential precipitating or aggravating factor in vitiligo. Stress activates the hypothalamic-pituitary-adrenal (HPA) axis, leading to cortisol elevation and increased oxidative burden. Moreover, neurogenic inflammation mediated by neuropeptides (such as substance P) and altered acetylcholine metabolism has been suggested to influence melanocyte survival and immune modulation, particularly in segmental vitiligo [9].

Autoimmune Comorbidities

Vitiligo is frequently associated with other autoimmune disorders, further reinforcing its autoimmune basis. Common comorbid conditions include autoimmune thyroid diseases (e.g., Hashimoto's thyroiditis, Graves' disease), type 1 diabetes mellitus, alopecia areata, pernicious anemia, and systemic lupus erythematosus. The co-occurrence suggests shared immunopathogenic mechanisms and emphasizes the importance of systemic evaluation in vitiligo patients[10].

Table 1. Major Etiological and Risk Factors Associated with Vitiligo

Risk Factor	Mechanism of Contribution
Genetic predisposition	Variants in immune and pigmentation genes enhance susceptibility to melanocyte targeting
UV exposure	Induces oxidative stress and melanocyte antigen presentation
Chemical exposure	Causes melanocyte toxicity and antigenic modification
Trauma (Koebner phenomenon)	Physical injury may induce local inflammation and depigmentation
Oxidative stress	ROS accumulation leads to melanocyte apoptosis and immune activation
Psychological stress	Elevates cortisol and neuropeptides, contributing to immune and oxidative imbalance
Autoimmune comorbidities	Shared autoimmune mechanisms promote cross-reactivity and chronic inflammation

PATHOGENESIS OF VITILIGO

The pathogenesis of vitiligo is a complex, multifactorial process involving genetic predisposition, autoimmune mechanisms, oxidative stress, cellular dysfunction, and environmental triggers. These mechanisms often converge to cause selective melanocyte destruction [11,12].

Genetic Basis

Multiple susceptibility genes have been implicated in vitiligo. These genes are involved in immune modulation, melanocyte function, and oxidative stress responses [13-16]:

- **PTPN22**: A protein tyrosine phosphatase involved in downregulating T-cell signaling; associated with multiple autoimmune diseases.
- **TYR**: Encodes tyrosinase, a critical enzyme in melanin biosynthesis and a major autoantigen in vitiligo.
- **HLA-DQB1**: Associated with antigen presentation to T cells; linked with multiple autoimmune responses.
- **FOXP3**: Critical for the development and function of regulatory T cells (Tregs), which help maintain immune tolerance.

IMMUNE DYSREGULATION

Vitiligo is primarily considered a T-cell-mediated autoimmune disease [18]

- $CD8^+$ cytotoxic T cells recognize melanocyte-specific antigens and secrete IFN-γ, a key cytokine that upregulates CXCL10 in keratinocytes, creating a chemotactic loop that recruits more T cells to the site.
- IL-15 promotes the persistence of tissue-resident memory T cells (TRM) in the skin, contributing to disease relapse and chronicity.

OXIDATIVE STRESS AND MITOCHONDRIAL DYSFUNCTION

Oxidative stress is considered a triggering factor in vitiligo onset. A deficiency in antioxidant enzymes like catalase and glutathione peroxidase leads to reactive oxygen species (ROS) accumulation. Melanocytes are particularly vulnerable to oxidative damage, and mitochondrial dysfunction further impairs their survival.

Melanocyte Adhesion Defects (Melanocytorrhagy Theory)

This theory posits that melanocytes in vitiligo have weak adherence to the basal membrane due to defective expression of adhesion molecules (e.g., integrins). Physical trauma or microinjury results in their detachment and loss, a phenomenon that supports the Koebner effect.

Environmental Amplification Mechanisms

Environmental insults such as UV radiation, mechanical injury, and chemical exposure may act as triggers or accelerators by promoting melanocyte stress or by revealing cryptic antigens, leading to immune activation in genetically predisposed individuals.

CLINICAL CLASSIFICATION AND FEATURES

Vitiligo presents in diverse clinical patterns, which are important for diagnosis, prognosis, and treatment planning [19-24].

TYPES OF VITILIGO

- **Non-Segmental Vitiligo (NSV)**: The most common form, characterized by symmetrical, bilateral lesions with progressive course and relapsing episodes.
- **Segmental Vitiligo (SV)**: Unilateral, dermatomal lesions with earlier onset and rapid stabilization.
- **Mixed Type**: Presence of both segmental and non-segmental lesions in the same patient.

Subtypes

- **Vulgaris**: Scattered macules across multiple regions.
- **Acrofacial**: Affects extremities and facial orifices.
- **Mucosal**: Confined to lips, genitalia, and other mucosal areas.
- **Focal**: Limited to a few areas without specific distribution.
- **Universal**: Involves over 80% of the body surface.

SPECIAL CLINICAL SIGNS

- **Leukotrichia**: White hair in depigmented areas, indicating follicular involvement and poor prognosis.
- **Trichrome Sign**: Presence of three zones-normal skin, hypopigmented skin, and depigmented center-suggests active progression.
- **Koebner Phenomenon**: Development of lesions at sites of trauma, reflecting melanocyte vulnerability.

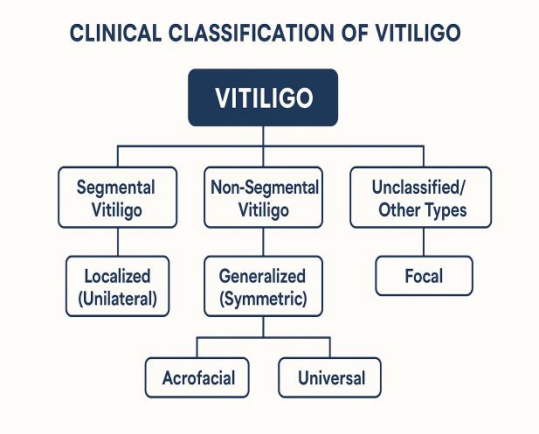

Fig 1: Clinical Classification of Vitiligo

DIAGNOSTIC APPROACHES

Accurate diagnosis of vitiligo is based on clinical examination, supported by investigative tools to confirm the disease and rule out mimicking conditions [25-28].

Clinical Evaluation

- Careful history regarding onset, progression, family history, and associated symptoms.
- Physical examination focusing on lesion symmetry, pattern, and presence of Koebner phenomenon or leukotrichia.

Wood's Lamp Examination

- Utilizes ultraviolet (UV) light (365 nm) to enhance visualization of depigmented areas.
- Vitiligo lesions appear as sharply demarcated, bright white patches, helpful in early or subtle cases.

Dermoscopy and Skin Biopsy

- Dermoscopy may show reduced pigment network, perifollicular depigmentation, and telangiectasia.
- Histopathology confirms absence of melanocytes, lymphocytic infiltrate, and sometimes basal cell vacuolization.

Laboratory Investigations

These help detect autoimmune associations and systemic involvement:

- Thyroid function tests (TSH, anti-TPO antibodies)
- Fasting glucose (for diabetes screening)
- ANA and vitamin B12 (for other autoimmune conditions)

Scoring Systems

- VASI (Vitiligo Area Scoring Index): Calculates extent of depigmentation using body surface area.
- VIDA (Vitiligo Disease Activity score): Measures disease activity based on recent lesion development.

Table 2: Diagnostic Tools in Vitiligo

Method	Utility
Clinical examination	Primary diagnosis, lesion pattern, family history
Wood's lamp	Enhances visualization of early or subtle lesions
Dermoscopy	Evaluates pigment loss and perifollicular involvement
Skin biopsy	Confirms absence of melanocytes
Lab investigations	Screens for autoimmune diseases

CURRENT TREATMENT MODALITIES

Topical Therapies

Corticosteroids

Topical corticosteroids are the first-line treatment in localized vitiligo, especially in early or progressive stages. They reduce inflammation and immune response that cause melanocyte destruction. Potent steroids like clobetasol are often used, but long-term use is associated with side effects such as skin atrophy, striae, and telangiectasia, particularly in sensitive areas [29].

Calcineurin Inhibitors

Tacrolimus and pimecrolimus are topical immunomodulators effective in facial and intertriginous vitiligo. They inhibit T-cell activation without the skin-thinning effects seen with steroids. These agents are especially preferred in pediatric patients and can be used long-term with a favorable safety profile [30].

Vitamin D Analogs

Calcipotriol and calcitriol, analogs of vitamin D3, help regulate melanocyte differentiation and immune response. They are often combined with corticosteroids for synergistic effects, particularly in resistant patches. Their efficacy as monotherapy is limited, but they are well-tolerated [31].

PHOTOTHERAPY

Narrowband UVB (NB-UVB)

NB-UVB is the gold standard for generalized vitiligo. It works by stimulating melanocyte proliferation and migration, as well as modulating local immune activity. It is safe for long-term use and can be applied to large body areas [32].

Excimer Laser

This laser delivers a focused beam of 308 nm UVB light to targeted depigmented patches. It is suitable for localized lesions and offers quicker repigmentation with fewer sessions, although it's more expensive than NB-UVB [33].

PUVA (Psoralen + UVA)

PUVA involves the use of psoralen (oral or topical) followed by UVA light. It induces melanocyte proliferation and migration. Due to higher risks like phototoxicity and long-term carcinogenic potential, its use has declined in favor of NB-UVB.

SYSTEMIC THERAPIES

Oral Corticosteroids

Systemic corticosteroids like prednisolone can halt the rapid progression of vitiligo through immunosuppression. They are generally used short-term due to side effects such as hyperglycemia, weight gain, and osteoporosis.[34]

Immunomodulators (Methotrexate, Azathioprine)

These agents suppress autoimmunity and are used in progressive, refractory vitiligo. Methotrexate is often preferred due to its immunosuppressive and anti-inflammatory properties. Regular monitoring is necessary due to potential hepatic and hematological toxicity.

SURGICAL INTERVENTIONS

Autologous Melanocyte Transplantation

Involves harvesting melanocytes from a pigmented donor area and grafting them onto depigmented patches. This is suitable for stable vitiligo and offers permanent repigmentation with cosmetic satisfaction.

Split-thickness Skin Grafting

Here, thin sections of pigmented skin are grafted to affected areas. It is useful for large or resistant lesions but may lead to donor site complications and uneven pigmentation.

SUPPORTIVE THERAPIES

Antioxidants

Supplementation with antioxidants (e.g., vitamin C, E, alpha-lipoic acid) may help counteract oxidative stress, a key factor in vitiligo pathogenesis. They are often used adjunctively with other therapies [36].

Counselling and Psychological Support

Given the visible nature of vitiligo and its psychological impact, counseling, cognitive-behavioural therapy (CBT), and support groups are crucial for patient well-being and treatment adherence [37].

Table 3: Current Treatment Modalities

Category	Treatment	Mechanism	Key Notes
Topical therapies	Corticosteroids	Immunosuppression	Effective but long-term use limited

	Calcineurin inhibitors	T-cell suppression	Preferred for sensitive skin
	Vitamin D analogs	Modulates melanocyte function	Often used in combination
Phototherapy	NB-UVB	Stimulates melanocytes	Gold standard for generalized vitiligo
	Excimer laser	Targeted UVB stimulation	Suitable for localized lesions
	PUVA	DNA crosslinking, immune modulation	Higher risk; less commonly used
Systemic therapies	Oral corticosteroids	General immunosuppression	Short-term for progressive vitiligo
	Methotrexate, Azathioprine	T-cell and B-cell suppression	Monitor toxicity
Surgical interventions	Melanocyte transplantation	Restores melanocytes	For stable, localized vitiligo
	Skin grafting	Physical repopulation of melanocytes	Risk of scarring
Supportive therapies	Antioxidants	Reduces oxidative stress	Adjunctive only
	Counseling	Psychosocial support	Enhances quality of life

CONCLUSION

Vitiligo, though often underestimated in its clinical severity, presents a significant burden through its psychosocial consequences and complex pathogenesis. Advances in our understanding of the immunological basis-particularly involving $CD8^+$ T cells, IFN-γ, and IL-15-mediated memory T-cell persistence-have opened avenues for targeted interventions. Current treatment modalities such as corticosteroids, NB-UVB phototherapy, and surgical grafting provide varying degrees of repigmentation, particularly in early or stable disease. However, the unpredictable nature of progression and relapse necessitates long-term strategic management. Emerging therapies such as JAK inhibitors, IL-15 blockade, and stem cell-based regeneration show promise in overcoming resistance to conventional treatments. Gene therapy and Wnt pathway modulation hold futuristic potential but require rigorous clinical validation. Importantly, the integration of antioxidant therapy, psychological counseling, and preventive lifestyle strategies enhances the overall quality of life for affected individuals. A patient-centric, multidisciplinary approach that combines medical, psychological, and emerging technological strategies is essential. Future research must aim at identifying robust biomarkers for disease activity and treatment response while exploring curative therapies. This review underscores that with sustained research and innovation, vitiligo is transitioning from being a cosmetic challenge to a treatable immune-mediated condition with curative potential on the horizon.

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CONFLICT OF INTEREST

Authors are declared that no conflict of interest.

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AUTHOR CONTRIBUTIONS

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REFERENCES

1. Rodrigues M, Ezzedine K, Hamzavi I, Pandya AG, Harris JE. New discoveries in the pathogenesis and classification of vitiligo. J Am Acad Dermatol. 2017;77(1):1–13.
2. Taieb A, Picardo M. The definition and assessment of vitiligo: a consensus report. Pigment Cell Res. 2007;20(1):27–35.
3. Bergqvist C, Ezzedine K. Vitiligo: a focus on pathogenesis and its therapeutic implications. J Dermatol. 2021;48(3):252–270.
4. Dell'Anna ML, Ottaviani M, Kovacs D, Mirabilii S, Brown DA, Cota C, et al. Membrane lipid peroxidation and altered caveolin-1 expression in vitiligo melanocytes: A comparative study. Pigment Cell Melanoma Res. 2010;23(2):270–278.

5. Picardo M, Dell'Anna ML, Leone G, et al. Vitiligo: Treatments and therapeutic perspectives. Dermatol Clin. 2007;25(2):233–239.
6. Harris JE. Cellular stress and innate inflammation in organ-specific autoimmunity: lessons learned from vitiligo. Immunol Rev. 2016;269(1):11–25.
7. Ezzedine K, Eleftheriadou V, Whitton M, van Geel N. Vitiligo. Lancet. 2015;386(9988):74–84.
8. Lotti T, Buggiani G, Prignano F. The importance of early treatment in vitiligo: new insights. Dermatol Clin. 2007;25(3):435–442.
9. Le Poole IC, van den Wijngaard RM, Westerhof W, Das PK. Presence or absence of melanocytes in vitiligo lesions: An immunohistochemical investigation. J Invest Dermatol. 1993;100(6):816–822.
10. Kim YC, Kim YJ, Kim HJ, et al. Vitiligo and associated autoimmune diseases: A review and meta-analysis. J Am Acad Dermatol. 2020;82(5):1099–1110.
11. Mulekar SV. Long-term follow-up study of segmental and focal vitiligo treated by autologous, non-cultured melanocyte–keratinocyte cell transplantation. Arch Dermatol. 2004;140(10):1211–1215.
12. Birlea SA, Costin GE, Norris DA. New insights on the pathogenesis of vitiligo: Reverse transcriptase inhibitors, oxidative stress and stress-response signaling pathways. Exp Dermatol. 2009;18(9):779–790.
13. Rashighi M, Harris JE. Interfering with the IFN-γ–CXCL10 axis to develop new treatments for vitiligo. Ann Transl Med. 2015;3(21):343.
14. Passeron T. The mechanisms of action of narrowband UVB phototherapy in vitiligo. J Invest Dermatol. 2022;142(1):10–12.
15. Speeckaert R, Speeckaert MM, van Geel N. CD8+ T cells in vitiligo: A review. Autoimmun Rev. 2017;16(9):937–945.
16. Gauthier Y, Benzekri L, Doré MA, Cario-André M, Taïeb A. Melanocyte transplantation in vitiligo: A review of techniques. Indian J Dermatol Venereol Leprol. 2003;69(1):51–58.
17. Falabella R. Surgical therapies for vitiligo: An evidence-based review. Clin Dermatol. 2007;25(5):556–563.
18. Tembhre MK, Sharma VK. Efficacy of oral mini-pulse dexamethasone therapy in vitiligo: A randomized placebo-controlled trial. Br J Dermatol. 2021;185(4):791–799.
19. Parsad D, Kanwar AJ. Topical immunomodulators in vitiligo: A review. Br J Dermatol. 2002;147(3):393–400.
20. Kang HY. Advances in medical and surgical treatments for vitiligo. J Clin Med. 2020;9(11):3688.
21. Grimes PE. New insights and new therapies in vitiligo. JAMA. 2005;293(6):730–735.
22. Ezzedine K, Spritz RA, Lim HW, et al. Vitiligo: A comprehensive overview Part II: Treatment options and approach to treatment. J Am Acad Dermatol. 2012;67(1):19–40.
23. Gupta S, Olsson MJ, Kanwar AJ, et al. Surgical management of vitiligo: Consensus analysis and recommendations. Indian J Dermatol. 2017;62(3):284–294.
24. Lee AY. Role of keratinocytes in the development of vitiligo. Ann Dermatol. 2012;24(2):115–125.
25. Whitton ME, Ashcroft DM, González U, et al. Interventions for vitiligo. Cochrane Database Syst Rev. 2015;2:CD003263.
26. Mashayekhi S, Bahrami A, et al. Vitamin D in vitiligo: A comprehensive review. J Dermatolog Treat. 2021;32(4):407–414.
27. Van Geel N, Speeckaert R. Advances in vitiligo: An update on pathophysiology and treatment. Dermatol Clin. 2017;35(2):181–192.
28. Singh RK, Lee KM, et al. Therapeutic potential of JAK inhibitors in vitiligo. J Invest Dermatol. 2020;140(3):617–623.
29. Hamzavi IH, Jain H, McLean D, et al. Parametric modeling of narrowband UV-B phototherapy for vitiligo. Arch Dermatol. 2004;140(6):677–681.
30. AlGhamdi KM. Topical tacrolimus for vitiligo: A review. J Dermatolog Treat. 2010;21(5):251–254.
31. Alshiyab DM, et al. IL-15 blockade and memory T cells in vitiligo: Future therapeutic directions. Clin Immunol. 2023;248:109259.
32. Mohan GC, Silverberg JI. Association of vitiligo with smoking, alcohol, and stress: A systematic review. JAMA Dermatol. 2015;151(11):1170–1176.
33. Singh S, et al. Psychological impact of vitiligo: A review. Dermatol Ther. 2022;35(6):e15338.
34. Strassner JP, Harris JE. Understanding mechanisms of immune-mediated tissue damage in vitiligo. J Invest Dermatol. 2018;138(1):15–23.
35. Chen J, et al. Nanotechnology in dermatology: Emerging treatment options for vitiligo. Int J Nanomedicine. 2021;16:6895–6908.
36. Kim N, et al. Gene therapy approaches in dermatology. Curr Opin Pediatr. 2021;33(4):466–471.
37. Senapati S, et al. Diagnostic and therapeutic biomarkers in vitiligo. Indian J Dermatol. 2019;64(2):101–106.