REVIEW ARTICLE

Vitiligo: Clinical Implications and Cell Line Models for Formulation Evaluation

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Abstract

A chronic autoimmune disorder called vitiligo, with a prevalence rate of 0.004–2.28, is encouraged worldwide and is characterized by increasing skin pigmentation loss due to melanocyte destruction, resulting in depigmented skin patches. There are two primary categories of this condition: Segmental Vitiligo (SV), which is less prevalent and usually only affects one side of the body, and Non-SV, the most pervasive form and frequently associated with other autoimmune disorders. An autoimmune reaction that targets melanocytes is the outcome of the delicate interaction between genetic susceptibility and environmental variables in the pathophysiology of vitiligo. The clinical evaluation and family history are the basis for the diagnosis, which might additionally include skin biopsies and blood tests for assessing melanocyte loss. Vitiligo has a major psychological influence on one's quality of living and can cause problems, including fret, low self-esteem, and social stigma. This summary highlights the necessity of having a thorough awareness of vitiligo to enhance management techniques and assist those suffering.

Key words: Cell line studies, cellular Vitiligo treatment, melanocytes, ruxolitinib, Vitiligo

INTRODUCTION

Vitiligo is a long-lasting autoimmune disorder in this condition, the body's immune system malfunctions and it starts to attack the body rather than fighting with the virus and infection that causes the loss of color and depigmented patches to the skin this generally happens when the color producing cells are destroyed (melanocytes) from the epidermis but there is no any itching and skin itself feel normal.[1,2]

The main function of the epidermal unit, which is made up of surrounding keratinocytes and melanocytes in the basal layer of the epidermis, is the complex process of melanogenesis,[3-5] which produces and disseminates melanin. Melanin is a pigment that exists in two different forms: pheomelanin (reddish-yellow)[6,7] and eumelanin (brown-black or black). Its ability to absorb light gives it photoprotection.[8] While several internal and external factors may influence melanogenesis, it is mostly governed by heredity.[9] Surrounding cells comprising keratinocytes, fibroblasts, inflammatory, neural, and endocrine cells release the intrinsic factors.[10] Perhaps the extrinsic influences are pharmaceuticals and ultraviolet (UV) radiation.

This condition of the skin is an idiopathic dermatological disorder that is distinguished by the appearance and development of white marks related to apoptosis or disruption of the melanocytes. Vitiligo affects around 1.5% of the world's residents. In India, the rate of vitiligo is found to be 0.25–2.5%.[11,12] However, both males and females may be affected by the vitiligo in equal measure. This disease can appear at any age of the individual.[13]

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Received: 15-02-2025
Revised: 08-05-2025
Accepted: 23-05-2025

Vitiligo skin (includes white patches on the different regions of the skin), which lacks melanocytes (melanin-producing neural crest-derived cells) in the epidermis region, and this condition also leads to sunburn when the skin is exposed to the sun [Figure 1] shows the effect of vitiligo resulting in loss of melanocytes and depigmentation causes sunburn.

PATHOGENESIS OF VITILIGO

The immune system ruptures the color-producing cells (melanocytes), which is also called Hypopigmentation (lowering the amount of sufficient pigmentation), which forms the white patches on the normal skin, which refers to vitiligo skin.[14-18]

Pathogenesis of vitiligo-(a) function of interleukins and the primary cellular population in the vitiligo pathogenesis. Arrows indicate corresponding pathways that are predominantly involved in cell-to-cell interactions.[19] (b) In vitiligo perilesional skin, it was first discovered that HMGB1 was released from the nucleus of melanocytes. Moreover, when treated with hydrogen peroxide, cultured normal human melanocytes might produce HMGB1. Furthermore, by attaching to the receptor for advanced glycation end products and triggering the nuclear factor kappa B and extracellular signal-regulated kinase signaling pathways, HMGB1 promoted the release of CXCL16 and IL-8 from keratinocytes. HMGB1 then increased the production of CXCL16 from keratinocytes, which in turn formed chemotaxis for the migration of CD8+ T lymphocytes from vitiligo patients. Furthermore, in vitiligo patients, HMGB1 aided in the development of dendritic cells. Overall, our research shows that the release of HMGB1 from melanocytes plays a role in the development of oxidative stress-induced inflammation in vitiligo.[20] (c) Forkhead Box D3, NLR family pyrin domain containing 1, Platelet Derived Growth Factor Receptor Alpha, human leukocyte antigen, X-box binding protein 1, Tyrosinase, Cytotoxic T-Lymphocyte Antigen 4, Antigen converting enzyme, Catalase, Protein Tyrosine Phosphatase Non-Receptor Type 22, IKAROS Family, Vascular endothelial growth factor, MYG1 Exonuclease, Melanocyte Inducing Transcription Factor, KIT Proto-Oncogene, Receptor Tyrosine Kinase, Estrogen Receptor 1, Autoimmune Regulator, Catechol-O-methyltransferase, Nucleotide-binding oligomerization domain, Leucine-rich Repeat and Pyrin domain containing, Fas Cell Surface Death Receptor, Endothelin 1, Cyclooxygenase 2, Vacuolar iron transporter 1, Discoidin domain receptor 1 these all genes are responsible in vitiligo pathogenesis.[21] (d) various environmental factors may lead to the formation of vitiligo, such as stress, sunburn, and skin trauma, and exposure to strong chemicals such as phenol derivatives, nickel, and hair dyes can disrupt the melanocytes.[22-25]

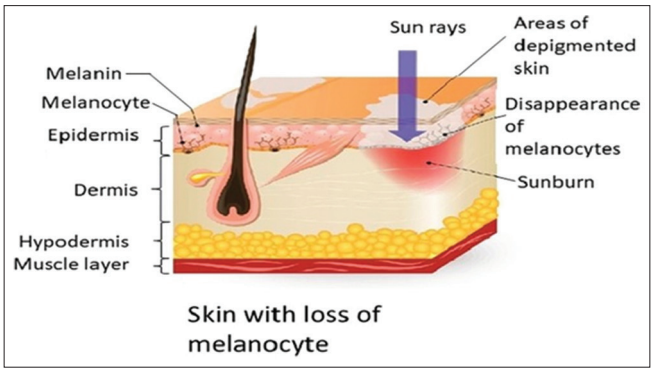

Figure 1: Illustration of Vitiligo skin cell

MANIFESTATION OF VITILIGO

1. White patches and marks that appear primarily on the hand, face, and usually genital areas of the affected person[26]
2. Premature whitening of the hair scalp, eyelashes, eyebrows, or beards
3. The depigmented mucous membrane in the mouth
4. A sudden change in the frequency of emotions may lead to a change in the amount of melanocyte production this can generally occur after an injury
5. The several causes also include environmental factors, which include UV radiation and harmful chemical exposure that can lead to dysfunction of the melanocyte-producing cells, and oxidative stress, which leads to the number of risk factors associated with vitiligo.[27,28]

TYPES OF VITILIGO

Vitiligo is classified based on clinical grounds into 2 broad categories, Segmental vitiligo (SV) and non-SV (NSV).[29-31]

Various types and subtypes of vitiligo with their remarks are mentioned in Table 1.

PSYCHOLOGICAL IMPLICATION OF VITILIGO AND ASSOCIATED DISORDERS

Since the destruction of melanocytes influences over 1.5% of the global community; however, most occurrences may go unreported.[42,43] There are several ways that patients' quality of life is impacted, including physical, social, and mental, as this disease is associated with social stigmatization. As an outcome, the specific person deals with a variety of issues such as poor confidence level[44] low self-esteem, and anxiety, which can lead to depression.[45-53] Skin illnesses may trigger psychological symptoms, including depression as well as dissenting emotions such as culpability, humiliation, concern, and self-doubt.[54,55] Individuals who experience it might be suffering from severe depression manifestation and poor self-depreciation. They might additionally find social

Table 1: Lists the many forms and subtypes of vitiligo[41]

Types of vitiligo	Subtypes	Remarks
Segmental vitiligo (SV)	Mucosal, unisegmental, bi-or multisegmental	This is rarer, not commonly affected, develops one side of the body, and may not spread further.[32-34]
Non-segmental vitiligo (NSV)	Mucosal, acrofacial, generalized, universal	It is most common, appears symmetrically on body parts, and progresses slowly.[35,36]
Mixed vitiligo (SV+NSV)	According to the severity of segmental vitiligo	Usually, the segmental vitiligo part in mixed vitiligo is more dominating, development of both segmental vitiligo and color loss beyond the area with segmental vitiligo.[37,38]
Unclassified vitiligo	Asymmetrical nonsegmental mucosal (one side)	When only one mucosa is affected without segmental distribution, this is unclassified and requires more time to conclude a definitive classification.[39,40]

SV: Segmental vitiligo, NSV: Non-segmental vitiligo

situations uneasy, but they might also have an inferiority complex and become perpetrators of bigotry.[56] Over half of the patients reported gaze by others, 16% faced rude and inappropriate comments,[57] and 13–14% of patients suffered from job discrimination and stigmatization for their vitiligo state. Both quantitative and qualitative methods are used to assess the severity of behavioral and social issues resulting from vitiligo.[58]

TREATMENT OF VITILIGO:[59]

Table 2 shows the numerous pharmaceuticals that are intended for the treatment of vitiligo with their respective bioavailability and half-life. Table 3 indicates that various commercial products have their manufacturer information applications included alongside the degree of their vitiligo efficacy.

CONTEMPORARY ADVANCEMENT IN CELL-BASED THERAPIES FOR VITILIGO[78]

- Target cells for vitiligo: melanocytes and keratinocytes- the skin's melanin-producing cells, or melanocytes are generated from neural cells. An array of processes underlying the etiology of vitiligo has been suggested to be explained by melanocyte cell death. Thus, the implantation of eligible melanocytes bears tremendous potential in the management of individuals suffering from vitiligo. Several techniques,[79-83] such as grafting into dermabraded or laser-abraded sites, have been tried to transport non-cultured melanocytes to the afflicted skin area of the patient.

Consequently, there are several methods for transferring the pure culture. The patient with SV can be treated by cultured analogous melanocytes.[84-87]

In this technique, these melanocytes are transplanted into the laser-denuded area, and the patient can recover up to 90% of the pigmentation within one month. In some cases, a small visible white boundary between the normal skin and the transplanted skin can appear.[88] Benzekri and Gauthier[89] have shown that after observation of 24 h, there is no infection detected, and delivery of melanin cells to the epidermis's underlying layer is detected, and around 40–45% of patients show an excellent response toward this technique. It is also feasible to cultivate these autologous melanocyte cells from the quadriceps or pelvic region with normal skin conditions.

- Melanocyte-keratinocyte cell transplantation (MKCT)- It is important to note that MKCT includes all aspects of clinical grafting modification, including recipient site preparation and cell collection from the donor site. In 1992, non-cultured cell transplantation for the treatment of vitiligo was first seen.[90] After various experimental tests performed on the piebald guinea pig skin,[91] therapy includes each melanocyte and keratinocytes being transplanted simultaneously. It is considered that the melanocyte cells grow perfectly in the presence of keratinocytes and show great pigmentation. Phillips *et al*.[92,93] This MKCT grafting technology has also seen major advancement since Olsson and Juhlin's 1998 revelation.[94]

In this discovery phase, Vázquez-Martínez *et al*.[95] and Quezada *et al*.[96] show the effectiveness of MKCT cell suspension treatment following dermabrasion (DA) versus using DA alone. There is no difference found by the researchers between the MKCT+DA and DA alone, but somehow, medically MKCT+DA shows marginally improved outcomes than DA alone. The methods, techniques, and principles for both studies were used in the same.[97] Method- The site that will be used as the donor site is anesthetized, and a very small proportion is cultured using a blade. The skin is nurtured for 30 min at 37°C alongside the solution containing 0.2% trypsin solution. After a slight wash with DMEM/F-12 medium, the dermis layer was removed from the epidermis and fragmented into small sections, then transferred into the tube which contained the same medium and set to centrifugation for 6 min at 2000 rpm.

Table 2: Indicates (various drugs used in the management of vitiligo with their respective half-life side effect and marketed formulations)[60-66]

S. No.	Category	Drugs	Bioavailability	Half-life	Side effects	Marketed formulation
1.	Corticosteroid	Clobetasol propionate	High topical bioavailability	1.5–2 h	Skin irritation, burning, itching, long-term use leads to skin thinning	Comply cream, clip cream, detox cream
2.	Corticosteroid (glucocorticoid)	Betamethasone valerate	Hightopical bioavailability	6–9 h	Stretch marks, skin irritation, itching	Betnovate cream, betnowell cream
3.	Corticosteroid (glucocorticoid)	Fluticasone propionate	Low systemic bioavailability	5–7 h	Headache, nausea, fungal infection, adrenaline suppression	Flutivate cream, futica cream, flutiwell cream
4.	Corticosteroid (glucocorticoid)	Mometasone furoate	Low topical bioavailability	5–8 h	Dryness, redness, and change in skin pigmentation, cause many severe side effects.	Mtavil cream, fumom cream, nanomomcream, etc
5.	Corticosteroid, cortisol ester	Hydrocortisone butyrate	96% oral	6 h average	Acneorpimples, burning, itching, pain in hairy areas, lightening normal skin color	Eczacort cream, hydrocortisone butyrate cream by TARO
6.	Bergapten	8-methoxysporalen	50% oral	0.5–2 h	Swelling, severe itching, skin discomfort, and swelling with blisters	Melanocyl ointment, octamoplotion, 8 MOP capsules
7.	4- alkoxy phenols	Monobenzone	Hightopical bioavailability	30–90 min	Mild burning, redness, cracking, or peeling of skin	Albaquin cream, benoquincream
8.	Janus kinase inhibitors (JAK inhibitors)	Ruxolitinib	95% oral	3 h	Burning sensation, dry skin	Opzeluracream
9.	immunosuppressant	Tacrolimus	25% oral bioavailability	12 h	Acne, increased sensitivity to sunlight, skin burning, folliculitis	Tacroz forte ointment, abitec fort ointment
10.	Anti- psoriatic (vit. D)	Calcipotriol	6% systemically ointment	<10 min	Skin irritation, dryness, stining feeling	Pasitrex ointment, calpsor ointment, callove ointment

This technique managed 100% of the repigmentation in 3 patients suffering from SV and 78% in patients suffering from generalized vitiligo (Olsson and Juhlin 1998). Huggins *et al.* (2012) show that the results of MKCT are more in SV patients than the generalized vitiligo.[98]

- ReCell system for treating vitiligo- ReCell is a revolutionary point of care autologous implementation that utilizes the patient's native rejuvenating cells to mend skin imperfections, including minuscule and crucial thermal damage to the skin. This technique makes it feasible to harvest autologous cells,

Table 3: Brand name of some marketed formulations with their applications[67-77]

S. No.	Brand Name	Formula	Manufacturer	Applications	Effectiveness	References
1.	Opzelura cream	Ruxolitinib	Incyte	Treatment of nonsegmental vitiligo	75% cure of vitiligo in 6 months	[67]
2.	Folitrax LP cream	Topical 1% methotrexate	Cipla	provide repigmentation	Restore some pigment over time (melanocytes)	[68]
3.	Abitac Ointment	0.1% w/w Tacrolimus	Abigail healthcare	Topical calcineurin inhibitor for vitiligo	include long-term treatment	[69]
4.	Albaquin cream	20% w/w monobenzone	Puneet laboratories pvt. LTD	Treat the uneven appearance of skin due to loss of color	Works effectively by restoring melanocytes and repigmentation	[70]
5.	Psorisome liposomal gel	Liposomal dithranol (0.5% w/w)	Life care innovation	Action towards vitiligo and other skin disorders	Improve vitiligo over time	[71]
6.	Melgain lotion	Decapeptide	Zydus	It stimulates the movement and growth of melanin-producing cells	Restore color to white patches of skin and hair roots	[72]
7.	Viti-melo cream (day/night cream)	Urea, vit. E, niacinamide	pharmaceris	Improves Hyperpigmentation or uneven tone	It diminishes vitiligo patches on the skin, halts their expansion, and prevents the new area from becoming affected.	[73,74]
8.	Melbild solution	decapeptide	Alkem	Topically used for vitiligo therapy	It functions by inducing skin cells to migrate to the area of the skin with the vitiligo patches.	[75]
9.	Flutivate cream	Fluticasone	GSK	cure vitiligo as well as itchy and inflammation of the skin	This steroidal lotion functions by preventing the synthesis of some chemical signals that cause sensitivity and irritation to the skin.	[76]
10.	Viti pause gel	Madecassoside, melitane GL, greyverse	Fix derma	Antioxidant therapy and pigment-regeneration development	developed to enhance the visual appearance of vitiligo and increase epidermal pigmentation	[73,77]

culture them, and then deliver them via spray therapy, in this a cell fluid incorporating keratinocytes, melanocytes, and Langerhans cells that splashed over the lesion, is used to treat people who had stable vitiligo in three months after the treatment the Mulekar *et al*.[99] compared the success rate of melanocyte-keratinocyte transplant against the ReCell system. This cell suspension had been used to formerly dermabraded zones employing either method, and the effect on healing was equivalent.

The areas that can be coated with the cultivated melanocytes are more expansive than those that can be coated with non-cultivated melanocytes owing to considerations such as expenses, duration, and efficient execution. This is concluded by Cervelli *et al*.[100] (80%) exhibited renewed pigmentation of more than 75%. In 66% of the instances, the investigators revealed a brilliant color match.

- Autologous non-cultured, non-trypsinized epidermal cell grafting - The modulation of autologous non-cultured, non-trypsinized keratinocyte-melanocyte cellular grafting is known as the Jodhpur technique (JT), having been originally implemented in a medical institution in Jodhpur, India. The donor area is dermabraded to obtain the grafting stuff since it's

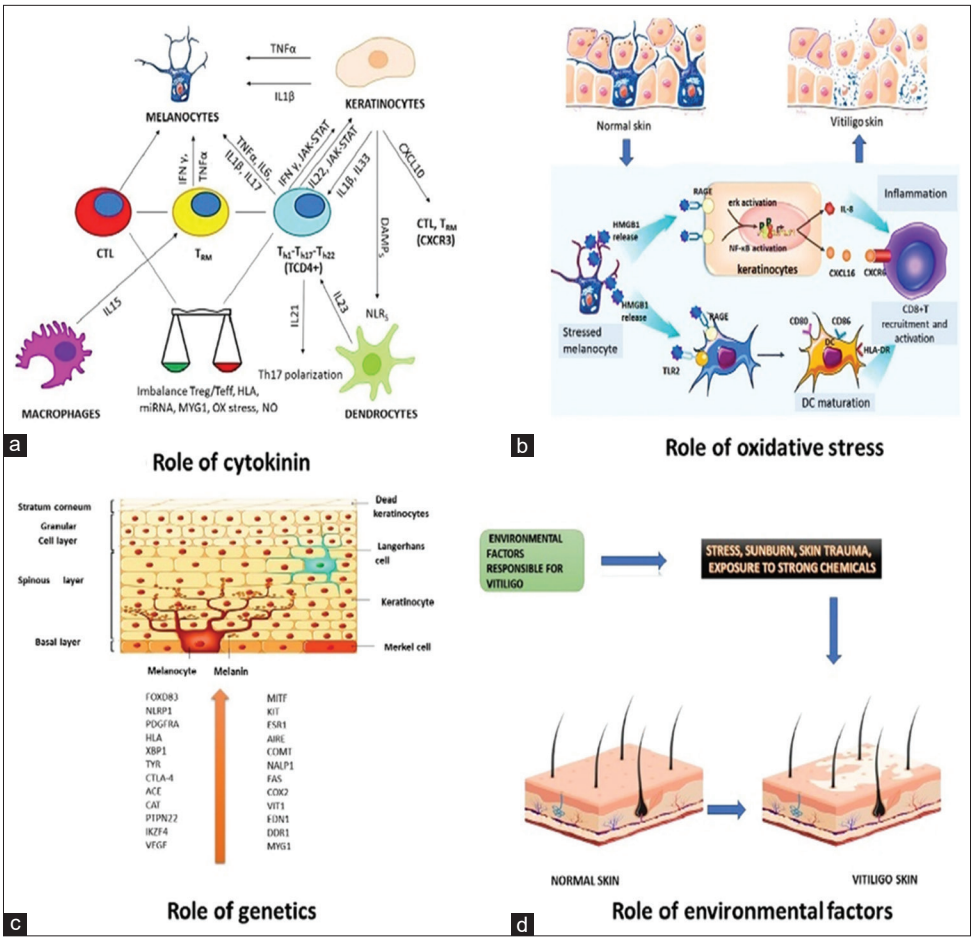

Figure 2: (a-d)Pathogenesis of vitiligo

rich in melanocytes. Although the pathophysiology of vitiligo is complicated and not entirely understood, it is generally agreed that a combination of oxidative stress, immunological reactions, and hereditary susceptibility results in melanocyte destruction. Although autoimmune dysregulation plays a significant role, oxidative stress is believed to be an early trigger that damages melanocytes, initiating the immunological response. This process is depicted in Figure 2. An ointment incorporating the broken-up epidermal granules from DA gets interconnected, generating a paste-like texture. Employing a graft-spreading device, this substance can be spread throughout the recipient lesion area. The earlier observation by Tyagi *et al*.[101] utilizing the JT technique shows that 60% of lesions for both epidermal cell suspension and epidermal curettes exhibited higher than 75% regeneration of colour or melanocytes, a study by Lamoria *et al*.[88] This method works better in terms of the pace of regenerated pigmentation than follicular unit transplant, adverse changes, contentment among patients, and lower dermatological life quality index.

- Combining cell transplantation with autologous platelet-rich plasma or narrowband ultraviolet B (NB-UVB) implementation - Patients with active vitiligo might gain profit from the medical care along NB-UVB.[102] It accelerates cultured melanocyte recruitment and multiplication.

Zhang *et al*.[103] gathered an ensemble of 473 patients to examine the consequences of autologous melanocyte transplantation alongside NB-UVB. In this study the initial group of patients obtained NB-UVB sessions before their melanocyte implantation, the second category got NB-UVB implementation complying with implantation, the third group acquired NB-UVB therapy additionally before and following transplantation, and the final fourth group was not given the NB-UVB sessions although it acquired transplantation, so the studies show the results that group three had the most favorable consequences with 81% of the patient attaining approximately 90% pigmentation regrowth.[104] This indicates that the NB-UVB managed before as well as after melanocyte transplantation provides the best likelihood of repigmentation among people experiencing active vitiligo.[105] Parambath *et al*.[106] researchers demonstrated that 11 of the 38 participants who were chosen for the clinical study declined to undergo surgery.[107]

However, it is impractical to calculate the percentage of vitiligo sufferers who have recovered. Because not every individual undergoes treatment. Moreover, monotherapy is

not much more effective than the combination of the two or more therapies; recurrence takes place in up to 40% of instances.[108]

Two hundred and sixty-nine up-regulating genes were involved in processes such as fatty acid omega oxidation, whilst two hundred and one down-regulated genes have connections to the PPAR and estrogen signaling pathway.[109]

USFDA-APPROVED CELLULAR TREATMENT FOR VITILIGO[110,111]

The only drug that the Food and Drug Administration (FDA) has authorized up to this point (FDA) involves ruxolitinib (opzelura cream) the first drug that is approved in several countries which involve USA, UK, and EU for NSV under age older than 12 years,[112-115] which aids in the restoration of the pigment to the NSV-affected skin. This drug is a JAK1/JAK2 inhibitor.[116] This demonstrates the target approach as ruxolinitib cream as compared to the oral administration of the drug, as the patient endures an assortment of oral complications.[117,118]

This topical cream was authorized by the USFDA in July 2022 for the restoration of the colour to the depigmented area and helps in the enhancement of melanocytes, including both pediatric and adult individuals. Ruxolinitib (opzerula) cream for vitiligo treatment- Ruxolinitib cream contains a white oil-in-water solubilizing emulsion which contains a maximum dose of 60 g/week.[119,120] It is a JAK1 and 2 inhibitor that can suppress IFN-y- signaling through the JAK-STAT pathway,[121] and is a chimeric anti-CD20 antibody that adheres exclusively to pre-B and mature B cells that have been identified as CD20-positive, leading to the breakdown of cells. Associations among B cells and immune cells restrict the generation of mediators that modify T cells, impede the processing of autoantigens, and diminish the onset of auto-inflammatory disorders. Furthermore, it restricts human dendritic cells from maturing and proliferating (DCs). This leads to reducing the expansion of CD8+ cytotoxic T cells, T cell responses specific to CD4 and CD8 antigens, and various critical cell reactions that are associated with pathogenesis.[81,122]

RECELL system (autologous cell harvesting device) - This is the first therapeutic device approved by the USFDA for treating vitiligo lesions that possess stable depigmentation.

Autologous means the cells used for the treatment, harvested from the patient's own body, which gives a beneficial effect with a lower chance of adverse effects or any allergic reactions to the patient's skin. The skin is harvested through abiopsy. In case of stable vitiligo, the regenerative epidermal suspension is created from the patient's normal skin. This treatment can restore the pigmentation over weeks to months. This is the minimally invasive method (about 25 cm^2 can treat up to 80 times the size of a vitiligo mark or any other skin condition), it is also used in various skin conditions, including burn injuries and acute skin defects. USFDA approval for vitiligo specifically recognizes its potential to meet an unmet need for an effective, safe for patient, and autologous treatment (self-derived). The approval of this device was made based on the clinical trials performed, which identify the efficacy and safety of the technique (clinical trial, govidentifier): NCT04547998. Results show pigmentation in 80% of the area in 6 months, with a 36% success rate of the RECELL therapy.[123]

CONCLUSION

For individuals who suffer from this, vitiligo is a complex autoimmune disease that poses major physical and psychological difficulties. Awareness of the cause of the disease and its accompanying risks–particularly its connections to other autoimmune conditions–requires an awareness of the distinction between segmental and NSV. There are still investigations being conducted on the pathophysiology of vitiligo to clarify the genetic and environmental components at play. Vitiligo has a significant psychological impact that frequently results in low self-esteem, social stigma, and mental health problems such as anxiety and depression. Consequently, enhancing the quality of life for those suffering from vitiligo requires an all-encompassing treatment approach that incorporates both medical and psychological support. To lessen the stigma attached to this illness, future research should concentrate on creating personalized therapy for individuals with proper studies of genetic conditions and diagnosis of the actual cause of the vitiligo or loss of melanocytes and regeneration of the skin color and raising awareness in society and enhancement of the self-estimation of the individual, there should also FDA approved drugs and some other techniques other than drugs which are very helpful for this skin condition and future research should also look forward for such FDA approved light therapies, drugs and other cellular techniques.

AUTHOR'S CONTRIBUTIONS

TW, the paper's primary author, came up with the review topic and wrote the article. APK, SKM, MT, and AK contributed insights into specific sections and reviewed every step, and APK was involved as the corresponding author. SR and AKY contributed by reviewing the FDA regulations regarding the management of vitiligo. All the authors have read and approved the article.

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Source of Support: Nil. **Conflicts of Interest:** None declared.