

AN EXHAUSTIVE OVERVIEW OF UNDERSTANDING VITILIGO: CAUSES, TYPES, AND TREATMENT OPTIONS

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Article Received: 30 September 2025, Article Revised: 20 October 2025, Published on: 10 November 2025

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ABSTRACT

Aim: The purpose of this article is to give a thorough overview of vitiligo, covering its pathogenesis, epidemiology, clinical characteristics, and available treatments. **Objectives:** The goals are to outline the many forms of vitiligo, talk about possible causes, go over current treatment methods, and stress the significance of prompt diagnosis and efficient care. **Findings:** There are several kinds of vitiligo, including segmental, non-segmental, and mixed vitiligo. Pharmacological treatments, phototherapy, surgery, and depigmentation techniques

are now available forms of treatment. **Discussion:** Patient outcomes and quality of life can be enhanced by early diagnosis and treatment. To comprehend the underlying causes of vitiligo and create more potent treatments, additional research is required.

KEYWORDS: Vitiligo, Skin depigmentation, Autoimmune, Melanocytes, Treatment options.

INTRODUCTION

A condition known as vitiligo (pronounced vit-il-EYE-go) causes the skin to become less colored.[1] It can impact any area of your body's skin. Hair and the interior of the mouth may also be impacted. Melanin often determines skin and hair color. When the cells that make melanin die or cease to function, vitiligo develops. People of all skin kinds can get vitiligo, but those with darker complexion may notice it more.[2] With an average onset age of around 24, it affects between 0.5% and 1% of the population, and its frequency seems to be equal for men and women. The development of novel treatment approaches has been aided by advancements in understanding the immunological processes that cause melanocyte death. Although the immunological mechanisms behind the two types of vitiligo are basically similar, somatic mosaicism is most likely the primary cause of segmental vitiligo, resulting in a shorter inflammatory phase and distinct treatment possibilities (such as pigment cell transplantation). In the past ten years, alternative theories have received less attention, including the self-destruct theory, melanocytorrhagy, oxidative stress, neurological processes, and melanocyte senescence. Published research has shown intriguing connections between inflammation and other variables, such as oxidative stress, genetics, and neuropeptides.[3]

EPIDEMIOLOGY OF VITILIGO

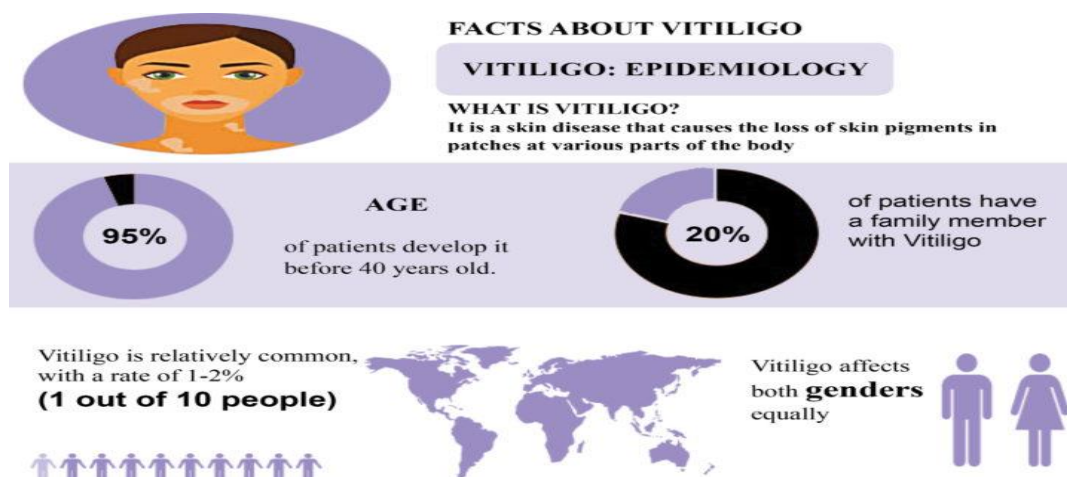
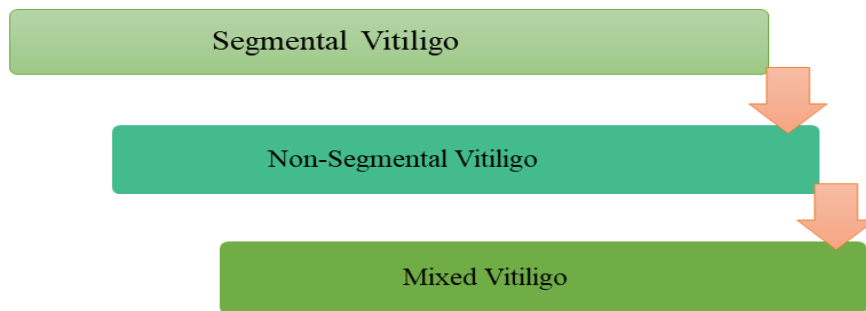


Figure 1: Epidemiology of Vitiligo.

TYPES OF VITILIGO

There are three major types of vitiligo are as follows:



1) Segmental Vitiligo: This condition begins and persists on one side of the body. An autoimmune disease is what it is. About 30% of children with vitiligo have it, and it is more noticeable in younger age groups. It reacts favorably to topical therapy.



Figure 3: Segmental Vitiligo.

2) Non-Segmental Vitiligo: This autoimmune condition frequently affects both sides of the body. It is the most prevalent kind of vitiligo, occurring in 90% of cases. They frequently show up on areas of the face, neck, and hands that are frequently exposed to the sun.



Figure 4: Non-Segmental Vitiligo.

3) Mixed Vitiligo: In the rare instances where segmental vitiligo turns non-segmental, there is an overlap of both types.[4, 5]



Figure 5: Mixed Vitiligo.

PATHOPHYSIOLOGY OF VITILIGO

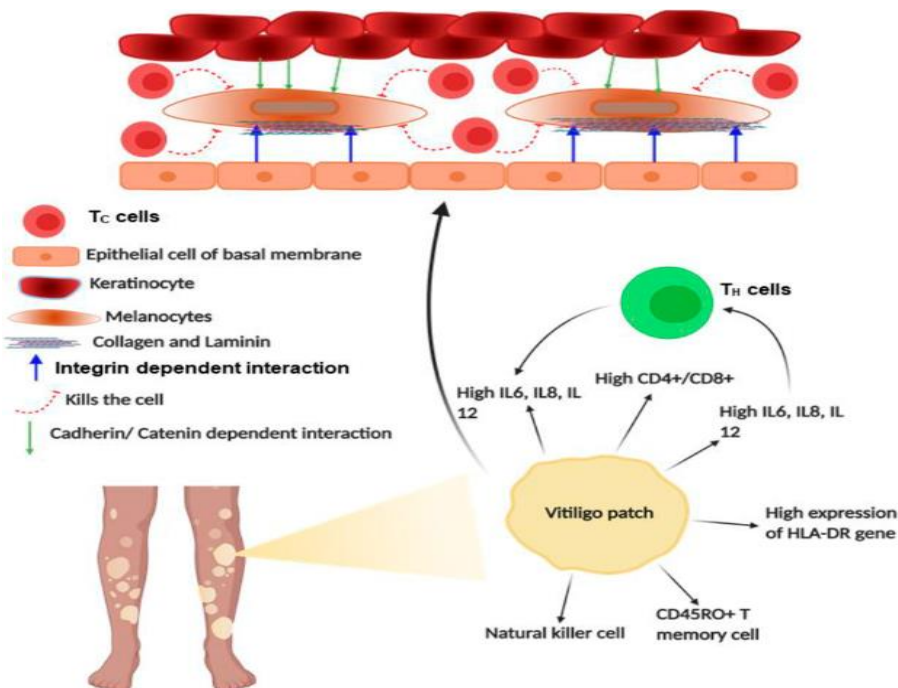


Figure 2: Schematic diagram of vitiligo pathophysiology: the figure provides a schematic diagram of how vitiligo develops and what points of this disease are regulated by integrin. Blue arrows denote signaling or mechanism being regulated by integrins.

CAUSES

Melanocyte death or dysfunction results in vitiligo. Melanocytes are nothing but cells producing melanin. It is pigment that gives color to eye skin and hair.

Vitiligo may cause due to:

- A condition where the skin's melanocytes are attacked and destroyed by your immune system.
- Family history, or inheritance.
- A trigger event, including being exposed to industrial toxins, stress, or sunburn
- Events that cause stress
- An infection
- It is not possible to spread vitiligo. It cannot be fastened from an alternate by one individual.[5,6]

Genetics

Vitiligo is a complex condition that is influenced by genetics. Even though vitiligo affects between 0.1 and 2% of the general population, there is a 6% chance that a patient's sibling will also get the condition, and a 23% chance for an identical twin.[7]

Oxidative stress

Melanocytes from vitiligo sufferers may have innate abnormalities that impair their ability to cope with cellular stress, according to mounting data. [8] While healthy melanocytes can counteract these stressors, vitiligo patients' melanocytes appear to be more susceptible.[7]

Environment

Multiple studies suggest that exposure to specific environment factors may play a central role in disease onset. A history of exposure to phenolic and catecholic compounds, which are present in dyes, resin/adhesives, etc., was validated by subsequent research. [7]

Autoimmune associations

Vitiligo is sometimes associated with autoimmune and inflammatory disease such as Addison's disease, alopecia areata, scleroderma, psoriasis, pernicious anemia and celiac disease. [9]

Immunity

Immune system mistakenly attacks and destroys the melanocytes of the skin causing autoimmune destruction of melanocytes.[10]

TREATMENTS OF VITILIGO

1. Pharmacological treatment

The pharmacological treatment also known as pharmacotherapy or drug therapy includes the use of Corticosteroids and Calcineurin inhibitors which helps to suppress or decrease the immune response activity because in Vitiligo the body's immune cells kills the melanocytes which in turn decreases the production of melanin and causes depigmentation.

2. Corticosteroids therapy

Creams containing corticosteroids can be effective in returning the pigmentation. The topical corticosteroid therapy is regarded as the first line therapy for Vitiligo. But these corticosteroids cream can cause breakdown and thinning of the skin or can cause the stretch marks, telangiectasias and they are likely to cause systemic side effects so they must be used under dermatologists care.[11,12]

Calcineurin inhibitors therapy

Calcineurin inhibitors includes cyclosporin, tacrolimus and pimecrolimus which are in the class of Immunosuppressive agents and are generally used in the patients who undergone organ transplantation as well as in auto-immune disorders so as to suppress the immune response. [13]

Combination therapy

The combination therapy includes the use of Calcipotriol and topical corticoids like betamethasone dipropionate. This combination therapy is used in the patients who are resistant to previous treatments like tacrolimus and topical corticoids with 75% of repigmentation rate observed in a series of cases.[11]

2. PHOTOTHERAPY

✓ Photochemotherapy with psoralen plus UVA radiation (PUVA) combines the use of psoralen with long-wave (320–340 nm) UVA radiation, photo chemotherapy with khellin plus UVA (KUVA) combines the use of khellin with long-wave (320–340 nm) UVA radiation, photo chemotherapy with L-phenylalanine (L-phe) combines the use L-phe with

UVA (PAUVA) or UVB, 311 nm narrow band UVB (NB-UVB) phototherapy, and laser therapy (targeted phototherapy devices) including: 308 nm excimer laser (MEL), Bioskin laser which transmits focused 311 nm wave length, helium neon laser (HeNe) which transmits 632.8 nm wavelength.

✓ Narrow band UVB radiation currently represents the phototherapy of choice for active and/or widespread vitiligo. Side effects are less frequent than in PUVA especially regarding the risk of melanoma and non-melanoma skin cancers, and the efficacy is at least equivocal. Because oral KUVA causes severe liver damage, it has been mainly discontinued. [14]

3. MELANOCYTE TRANSFER

✓ The patients who are unresponsive to other therapies are recommended for surgical melanocyte transfer. The surgical melanocyte transfer technique is based on the principle that to transplantation of autologous melanocytes from normal pigmented donor skin to the depigmented skin of recipient. Many surgical techniques are available for the surgical transplantation of melanocytes which includes miniature punch grafting, suction blister grafting, transfer of non-cultured epidermal suspension, and transfer of cultured melanocytes.

✓ The vitiligo was categorized according to extent of stability and severity. The area of the macules was calculated based on the radius of the circular macules in cm scale. The large sized irregular macules are calculated by dividing the lesions into geometrical shapes and then determining the area.[15]

4. LASER THERAPY (TARGETED PHOTOTHERAPY)

Monochromatic excimer laser (MEL) 308 nm

MEL is the best-studied and most popular targeted phototherapy for the treatment of vitiligo with less total body irradiation, and less side effects on normal skin. MEL is used one to three times weekly for an initial course of 12 weeks and is approved by the FDA for treating vitiligo.[16] The outcome of the treatment with MEL is improved if it is combined with other treatment modalities, such as topical hydrocortisone, 17-butyrate,[17] topical tacrolimus.[18] The benefit of combining topical vitamin D3 analogs is not clear but topical tacalcitol may induce earlier repigmentation, requiring less cumulative radiation, and it may not affect the final repigmentation.[19,20]

Bioskin

Bioskin is a new device which transmits a focused 311-nm UVB radiation, Bioskin phototherapy (monotherapy) had repigmentation rates of more than 75%in 72% of patients and

better results were achieved if it was combined with other treatment modalities. The best result was achieved if combined with betamethasone dipropionate.[21]

Helium neon laser

The helium neon (HeNe) laser emits 632.8 nm radiation and is used for the treatment of segmental vitiligo. It modifies adrenergic dysregulation of cutaneous blood flow seen in SV, [22] and promotes melanogenesis, melanocyte growth, migration and survival in the skin. [23, 24]

5. DEPIGMENTATION

In patients with extensive vitiligo with remaining pigmented islands of normal skin or patients with refractory vitiligo when satisfactory repigmentation is not attainable, depigmentation may provide pleasing cosmetic outcomes. Monobenzone ethyl ester (MBEH) is a derivative of hydroquinone (HQ), unlike hydroquinone, MBEH almost always causes irreversible depigmentation. [25, 26] The Q-switch ruby laser has been extensively used for depigmentation in vitiligo universalis, although Q-switch Alexandrite laser is also effective. [27] Combination between topical methoxyphenol and Q-switched ruby laser has been proposed by Njoo et al.[26] and achieved complete depigmentation in 69% of patients with universal vitiligo.

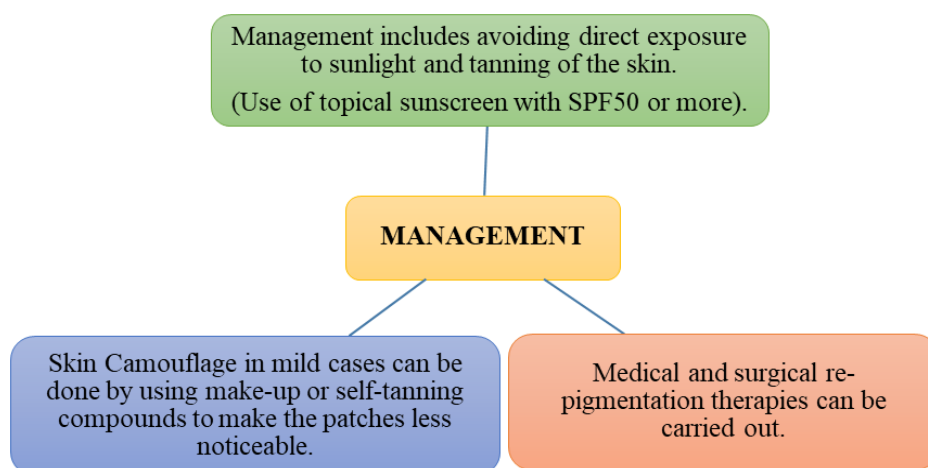
6. PSYCHOTHERAPY

Depigmentation exerts a negative impact on the patient's appearance and self-esteem. [28] Levels of disability vary according objective factors such as, extension and site of the disease, skin type, ethnicity, and cultural background. [29, 30] Perceived severity of the disease seems to be influenced by the patient's personality more than objective factors.[31] Lack of a universally effective treatment adds to the psychological burden of the disease.[32]

7. BIOLOGICS AND IMMUNOSUPPRESSANTS

Tumor necrosis alpha inhibitors, [33] immune suppressants like azathioprine, [34] cyclophosphamide and cyclosporine were studied in the treatment of vitiligo, but the current data do not provide enough evidence to recommend their use in patients with vitiligo. Moreover, the potential side effects of these agents do not justify their use in vitiligo. [35]

MANAGEMENT[36]



DIFFERENTIAL DIAGNOSIS

There are other conditions that make the skin change or lose its pigmentation. Differential diagnosis is made versus.

- 1. Piebaldism;** this is a rare depigmentation disorder due to a mutation of c-kit migration of melanocytes. It is characterized by stable and circumscribed white patches present at birth, affecting the face, sternal and abdominal zones, knees and elbows.
- 2. Achromic nevus:** This is a well-limited depigmented area, stable and evident at birth, in which melanocytes are either normal or reduced.
- 3. Post-inflammatory leukoderma:** The condition in which patients have a history of pre-existing dermatosis.
- 4. Pityriasis Alba:** This condition starts off with red and scaly areas of skin, which fades into scaly lighter patches of skin.
- 5. Depigmented lesions in leprosy:** The condition which shows anesthetic disturbance of sensibility.
- 6. Albinism:** It is a genetic disorder that causes the skin, hair, eyes to have little or no color.[37]

MARKETED FORMULATION

- Lukoskin used for treatment of vitiligo. It is an innovation by DRDO.
- Anti-vitiligo kit treatment by Dr. Ravish Kamal.
- Drug like methoxasalen, triosalen and posarlen are used in treatment of vitiligo with minimum side effects.
- Potent corticosteroids like betamethasone, valerate, triamcinolone and very potent corticosteroids like alobetasol, fluticasone propionate are helpful for repigmentation of skin.

HERBAL FORMULATION COMPONENTS

Following components have good properties and will used to treat vitiligo.

- Cucumis melo
- Ginkgo biloba
- Khellin
- Green tea Polyphenols
- Traditional Chinese Medicine (TCM)
- Capsaicin, Curcumin
- Picrorhiza kurroa. [38,39]

RECENT ADVANCEMENT IN TREATMENT OF VITILIGO

- Depigmentation therapy using monobenzone is approved by the FDA.
- Minocycline, a broad-spectrum antibiotic, has also been evaluated in vitiligo owing to its anti-inflammatory, antioxidant, and immunomodulator properties.
- Afamelanotide, a longer-acting synthetic analogue of alpha melanocyte motivating hormone, has also shown promise in initial clinical studies. Through binding to the melanocortin-1 receptor, Afamelanotide may battle melanocortin system defects in vitiligo patients by stimulating melanocyte proliferation and melanogenesis.[39]

CHEMICAL VITILIGO AND VITILIGO- INTERLINKING

There is no difference between vitiligo and vitiligo caused by chemicals.[40,41]It was determined by Cummings and Norland [42] that certain types of vitiligo vulgaris are chemical leucoderma brought on by unknown melanocytotoxic substances in the ecosystem. The pathophysiology of chemical leucoderma and the function of external oxidants in vitiligo were both highlighted by Glassman. [43] This pathomechanism may help to explain the

causes of idiopathic vitiligo's progression and chronicity. Analogously to generalized idiopathic vitiligo, depigmentation in chemical vitiligo spreads to distant venues.



Figure 6: Chemical vitiligo on feet from rubber.

Table 1: Causative common household products among 864 chemical leucoderma Patients.[44]

S.No	Consumer	Percentage
1	Hair dye	27.4
2	Deodorant/perfume	21.6
3	Detergent/cleanser	15.4
4	Adhesive 'bindi' (decorative color on forehead)	12.0
5	Rubber sandal	9.4
6	Black socks/shoes	9.1
7	Eyeliner	8.2
8	Lip liner	4.8
9	Rubber condom	3.5
10	Lipstick	3.3
11	Fur toys	3.1
12	Toothpaste	1.9
13	Insecticide	1.7
14	Alta (decorative color on feet)	1.2
15	Amulet (holy material) string color	0.9
16	Multiple chemicals	67.8

REFERENCES

1. Khadeejeh AL-smadi et al., Vitiligo: A Review of Aetiology, Pathogenesis, Treatment, and Psychosocial Impact, 2023, 10, 84.
2. Meghraj Suryawansh, Introduction to Vitiligo and Its Treatment: A Review, 2018, Vol. 1 (4), 72-75.
3. Alkhateeb A, Fain PR, Thody A, Bennett DC, Spritz RA. Epidemiology of vitiligo and associated autoimmune diseases in Caucasian probands and their relatives. *Pigment Cell Res.* 2003;16: 208–214.

4. Lakhani, D.M. and Deshpande, A.S. (2014), "Various Treatments for Vitiligo: Problems Associated and Solutions", *Journal of Applied Pharmaceutical Science*, Vol, 4(11), 101-105.
5. Talia, K., (2009), "Vitiligo in children: a review of classification, hypotheses of pathogenesis and treatment", *World J Pediatric*, 4, 265-268.
6. Craiglow, B.G. and King, B.A. (2015), "Tofacitinib citrate for the treatment of vitiligo: a pathogenesis-directed therapy, *JAMA Dermatology*, 151(10), 1110-1112.
7. Mehdi R, John E. Harris. Vitiligo Pathogenesis and Emerging Treatments. *Dermatologic Clinics*, 2017; 30(2): 257-265.
8. Prashiela M, Nada E, Seth J. et al. Recent advances in understanding Vitiligo. *F1000Research*, 2016; 1-9.
9. Birlea SA, Serota M, Norris DA. Nonbullous Skin Diseases: Alopecia Areat a, Vitiligo, Psoriasis, and Urticaria. In: *The Autoimmune Diseases Academic Press*, 2020; 6: 1211-34.
10. Anuradha B, Davinder P. Clinical and Molecular Aspects of Vitiligo Treatments. *International Journal of Molecular Science*, 2018; 19(5):1509.
11. Faria AR, Tarlé RG, Dellatorre G, Mira MT, Castro CC. Vitiligo-Part 2-classification, histopathology and treatment. *Anais brasileiros de dermatologia*, 2014; 89(5): 784-90.
12. Eleftheriadou V, Thomas K, van Geel N, Hamzavi I, Lim H, Suzuki T, Katayama I, Anbar T, Abdallah M, Benzekri L, Gauthier Y. Developing core outcome set for vitiligo clinical trials: International e-Delphi consensus. *Pigment cell & melanoma research*, 2015; 28(3): 363-9.
13. Kostovic K, Pasic A. New treatment modalities for vitiligo. *Drugs*, 2005; 1, 65(4): 447-59.
14. Pacifico A, Leone G. Photo (chemo) therapy for vitiligo. *Photodermatology, photoimmunology & photomedicine*, 2011; 27(5): 261-77.
15. Birinder SG, Manmohan SB, Neha C, et al. Non-cultured melanocyte transfer in the management of stable Vitiligo. *Journal of Family Medicine and Primary Care*, 2019; 8(9): 2912- US Food and Drug Administration web site. Available from: www.fda.gov.
16. Sassi F, Cazzaniga S, Tessari G, Chatenoud L, Reseghetti A, Marchesi L, Girolomoni G, Naldi L. Randomized controlled trial comparing the effectiveness of 308-nm excimer laser alone or in combination with topical hydrocortisone 17-butyrate cream in the treatment of vitiligo of the face and neck. *Br J Dermatol*. 2008; 159:1186–1191.

17. Kawalek AZ, Spencer JM, Phelps RG. Combined excimer laser and topical tacrolimus for the treatment of vitiligo: a pilot study. *Dermatol Surg.* 2004; 30:130–135.
18. Goldinger SM, Dummer R, Schmid P, Burg G, Siefert B, Lauchli S. Combination of 308-nm xenon excimer laser and topical calcipotriol in vitiligo. *J Eur Acad Dermatol Venereol.* 2007; 21:504–508.
19. Lu-yan T, Wen-wen F, Lei-hong X, Yi J, Zhi-zhong Z. Topical tacalcitol and 308-nm monochromatic excimer light: a synergistic combination for the treatment of vitiligo. *Photodermatol Photoimmunol Photomed.* 2006;22:310–314.
20. Lotti T, Buggiani G, Troiano M, Assad GB, Delescluse J, De Giorgi V, Hercogova J. Targeted and combination treatments for vitiligo. Comparative evaluation of different current modalities in 458 subjects. *Dermatol Ther.* 2008;21(suppl 1): s20–s26.
21. Wu CS, Hu SC, Lan CC, Chen GS, Chuo WH, Yu HS. Low-energy helium-neon laser therapy induces repigmentation and improves the abnormalities of cutaneous microcirculation in segmental-type vitiligo lesions. *Kaohsiung J Med Sci.* 2008;24:180–189.
22. Lan CC, Wu CS, Chiou MH, Hsieh PC, Yu HS. Low-energy helium-neon laser induces locomotion of immature melanoblasts and promotes melanogenesis of the more differentiated melanoblasts: recapitulation of vitiligo repigmentation in vitro. *J Invest Dermatol.* 2006; 126:2119–2126.
23. Yu HS, Wu CS, Yu CL, Kao YH, Chiou MH. Heliumneon laser irradiation stimulates migration and proliferation in melanocytes and induces repigmentation in segmental-type vitiligo. *J Invest Dermatol.* 2003;120:56–64.
24. Mosher DB, Parrish JA, Fitzpatrick TB. Monobenzylether of hydroquinone. A retrospective study of treatment of 18 vitiligo patients and a review of the literature. *Br J Dermatol.* 1977; 97:669–679.
25. Njoo MD, Vodegel RM, Westerhof W. Depigmentation therapy in vitiligo universalis with topical 4-methoxyphenol and the Q- switched ruby laser. *J Am Acad Dermatol.* 2000; 42:760–769.
26. Rao J, Fitzpatrick RE. Use of the Q-switched 755-nm alexandrite laser to treat recalcitrant pigment after depigmentation therapy for vitiligo. *Dermatol Surg.* 2004; 30:1043–1045.
27. Linthorst Homan MW, Spuls PI, De Korte J, Bos JD, Sprangers MA, van der Veen JP. The burden of vitiligo: patient characteristics associated with quality of life. *J Am Acad Dermatol.* 2009; 61:411–420.

28. Thompson AR, Clarke SA, Newell RJ, Gawkrödger DJ, Appearance Research Collaboration. Vitiligo linked to stigmatization in British South Asian women: a qualitative study of the experiences of living with vitiligo. *Br J Dermatol.* 2010; 163:481–486.
29. Kostoupolou P, Taieb A. Psychological interventions. In: Picardo M, Taieb A, eds. *Vitiligo*. Berlin: Springer; 2009:433–435.
30. Kostopolou P, Jouary T, Quintard B, Ezzedine K, Marques S, Boutchnei S, Taieb A. Objective vs. Subjective factors in the psychological impact of vitiligo: the experience from a French referral center. *Br J Dermatol.* 2009;161:128–133.
31. Talsania N, Lamb B, Bewley A. Vitiligo is more than skin deep: a survey of members of the Vitiligo Society. *Clin Exp Dermatol.* 2009; 35:736–739.
32. Birol A, Kisa U, Kurtipek GS, Kara F, Kocak M, Erkek E, Caglayan O. Increased tumor necrosis factor alpha (TNF-alpha) and interleukin 1 alpha (IL1-alpha) levels in the lesional skin of patients with nonsegmental vitiligo. *Int J Dermatol.* 2006; 45:992–994.
33. Radmanesh M, Saedi K. The Efficacy of combined PUVA and low-dose azathioprine for early and enhanced repigmentation in vitiligo patients. *J Dermatol Treat.* 2006; 17:151–153.
34. Taieb A, Alomar A, Böhm M, Dell'anna ML, De Pase A, Eleftheriadou V, Ezzedine K, Gauthier Y, Gawkrödger DJ, Jouary T, Leone G, Moretti S, Nieuweboer- Krobotova L, Olsson MJ, Parsad D, Passeron T, Tanew A, van der Veen W, van Geel N, Whitton M, Wolkerstorfer A, Picardo M, Vitiligo European Task Force (VETF); European Academy of Dermatology and
35. Venereology (EADV); Union Européenne des Médecins Spécialistes (UEMS). Guidelines for the management of vitiligo: the European Dermatology Forum consensus. *Br J Dermatol.* 2012; 168:5–19.
36. Minhaj Sohail Shakil Patel Et Al., A Comprehensive Review On Current Treatments of Vitiligo, Vol 9, and Issue 10, 2020.
37. Moretti S. Vitiligo. *Orphanet Encyclopedia*, 2003: 1-5.
38. Benzekri, L. and Gauthier, Y., (2014), “New Insights in Mixed Vitiligo: Initial Non Segmental Vitiligo can precede the Onset of Segmental Vitiligo. (Cases Report and Review of Theories)”, *Pigmentary Disorders*, 1,114.
39. Soni, P., (2010), “A Review on Traditional and Alternative Treatment for Skin Disease Vitiligo”, *International Journal of Pharmaceutical & Biological Archive*, 1(3). 220-227.

40. Harris JE. Chemical-induced vitiligo. *Dermatol Clin*. 2017;35:151–61. doi: 10.1016/j.det.2016.11.006.
41. Alikhan A, Felsten LM, Daly M, Petronic-Rosic V. Vitiligo: a comprehensive overview Part I.Introduction, epidemiology, quality of life, diagnosis, differential diagnosis, associations, histopathology, etiology, and work-up. *J Am Acad Dermatol*. 2011;65:473–91. doi: 10.1016/j.jaad.2010.11.061.
42. Cummings MP, Norlund JJ. Chemical leukoderma: Fact or fancy. *Am J Contact Dermatitis*. 1995; 6:122–6.
43. Glassman SJ. Vitiligo, reactive oxygen species and T-cells. *Clin Sci*. 2011;120:99–120. doi: 10.1042/CS20090603.
44. Ghosh S, Mukhopadhyay S. Chemical leucoderma: A clinico- aetiological study of 864 cases in the perspective of a developing country. *Br J Dermatol*. 2009;160:40–7. doi: 10.1111/j.1365-2133.2008.08815.x.