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Vitiligo: A Review

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ABSTRACT

Vitiligo, a commonde pigmenting skin condition, has an estimated prevalence of 0.5–2% of the population worldwide. The disease is marked by the selective loss of melanocytes which results in typical nonscaly, chalky-white macules. In recent years, tremendous progress has been made in our understanding of the aetiology of vitiligo which is now clearly characterised as an autoimmune disease. Vitiligo is typically overlooked as a cosmetic disease, although its effects can be mentally devastating, often with a major bur- den on everyday living. In 2011, a worldwide consensus classified segmental vitiligo separately from all other forms of vitiligo, and the term vitiligo was defined to designate all kinds of nonsegmental vitiligo. This review highlights the existing knowledge on vitiligo and strives to give an overview of vitiligo.

KEYWORDS: *vitiligo, melanocytes, cosmetic disease, autoimmune disease*

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INTRODUCTION

Vitiligo is a hypopigmentation illness where the loss of functional melanocytes promotes the appearance of white spots on the skin. Vitiligo affects 1% of the world pop ulation however the prevalence has been reported as high as 4% in some South Asian, Mexican and American pop- ulations Vitiligo can develop at any age, but various studies show that 50% of cases appear before the age of 20 Barona discovered that in patients with unilateral vitiligo the mean age at beginning was 16.3 years (95% CI: 12 to 19 years), compared to 24.8 years (95% CI: 22 to 28 years) in patients with bilateral vitiligo

Sixteen to 35% of patients with vitiligo develop significant mental morbidity Depression (10%), dysthymia (7–9%), sleep difficulties (20%), suicidal thoughts (10%), suicidal attempts (3.3%) and anxiety (3.3%) have been identified in persons suffering with vitiligo. Vitiligo can also lead to difficulties in building relation- ships, avoidance of certain social circumstances, and difficulties in sexual relationships. Vitiligo can be confused with leprosy, which likewise causes loss of pigment, thus further stigmatizing sufferers.

Several systematic reviews assessing the clinical efficacy of topical corticosteroids, narrowband ultraviolet light B (UVB), psoralen with ultraviolet light A exposure (PUVA), calcipotriol, l-phenylalanine, topical immunomodulators (tacrolimus and pimecrolimus), excimer laser, and surgical therapy in the management of symptoms associated with vitiligo have recently been published, including in the Journal of the American Medical Association (JAMA) and by the Cochrane Collaboration These evaluations find that there is some evidence that topical steroids are of help, but express concerns about long term negative effects of topical steroids. The review authors also describe some evidence for the efficacy of phototherapy (UVA or UVB) as a monotherapy, or combination with psoralens or calcipotriol. However, concerns are raised about side effects

such as phototoxic responses, blistering, and lack of data on long term skin cancer risk. All three systematic evaluations indicate that use of topical immunomondulators has potential, but that further investigation is needed. In addition, the systematic review by Whitton completed for the Chochrane collaboration and the systematic review published by Forschner in JAMA indicate that L-phenylalanine use with phototherapy is promising, but that more study is essential before definitive conclusions can be drawn. Although several clinical trials exploring the use of natural health product (NHP) therapies (e.g., vitamins, minerals, herbal medications and other supplements) for vitiligo have been published, we were unable to identify any systematic reviews of these. Here we describe the results of a systematic evaluation of controlled clinical studies assessing the efficacy of NHPs in the treatment of vitiligo.

In 2011, the Vitiligo European Taskforce assembled at the Vitiligo Global Issues Consensus Conference and changed the classification of vitiligo. Table 1 below describes the classification of vitiligo according to the taskforce's consensus declaration. The Vitiligo European Taskforce also suggested that the term vitiligo "vulgaris" not be used as it implies a negative connotation, although "vulgaris" is equivalent with common." Many writers have attempted to classify vitiligo; however, for the purposes of this review, one classification was chosen for clarity.

TABLE 1. Classification and Consensus Nomenclature of Vitiligo According to the VETF, 2011

Category of Vitiligo	Subtype
Nonsegmental vitiligo	Acrofacial
	Mucosal (more than one mucosal site)
	Generalized
	Universal
	Mixed
Segmental vitiligo	Unisegmental, bisegmental, or plurisegmental
Undetermined/ unclassified vitiligo	Focal
	Mucosal (one mucosal site in isolation)

Vitiligo is defined based on clinical appearance into two major kinds, segmental vitiligo (SV) and NSV. Rare clinical forms of vitiligo such as localised, inflammatory, confetti-like or "punctate," and trichrome have been recorded but are difficult to definitively define because they may fall into the general clinical spectrum of illness). Trichrome, inflammatory boundaries, and confetti-like lesions are regarded potential indications of active lesions.

NSV has been used as umbrella name for numerous clinical subtypes of vitiligo that are obviously distinct from SV, including acrofacial, generalized, mucosal, and universal. NSV is characterized by depigmented macules that vary in size from a few to several centimeters in diameter, occurring on both sides of the body with a trend towards symmetrical distribution. Acrofacial vitiligo affects the distal extremities and face. Generalized vitiligo means more extensive dispersion and more areas of involvement Universal vitiligo is total or virtually complete depigmentation of skin; sometimes, body hair, oral mucosa, and/or genital mucosae are implicated. Mixed vitiligo is when elements of SV and NSV are present, frequently first appearing as SV and then developing to NSV (Goh & Pandya, 2017). SV is divided into unisegmental, bisegmental, and plurisegmental subtypes. SV is generally associated with fast onset and leukotrichia

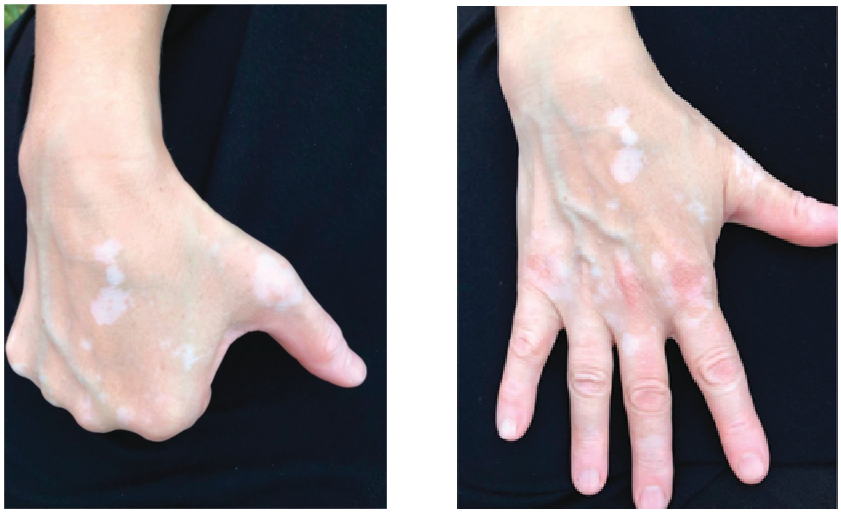

Fig no1 Non-segmental vitiligo of the hand.

Other writers have defined SV as a unilateral patch of depigmentation that does not span the midline The face is most usually afflicted in SV, followed by the trunk, neck, extremities, and scalp When coinciding with NSV, punctate vitiligo should be classed as NSV; however, if noticed in isolation, it should be referred to as punctate vitiligo Inflammatory vitiligo depicts erythema sometimes found around the boundaries of lesions and is regarded to be an indication of disease activity. Trichrome vitiligo is characterized by a combination of intermixed hypopigmented, normal, and depigmented skin. Trichrome vitiligo has been observed as an indication of fast development.

Symptoms:

The most important symptoms of vitiligo known is the depigmentation of regions of skin. Initially, the patches are little but they will be enlarged with time. The skin lesions are dominantly detected on the face, hands and wrists. often patients who are suffering from this disease also suffer from depression.

Pathogenesis:

While the exact cause of vitiligo remains obscure, there are three potential explanations for its origin. The pathogenesis involves biochemical/cytotoxic, neurological, and immunological mechanisms. The biochemical/cytotoxic hypothesis posits that vitiligo occurs when melanocytes are destroyed by cytotoxic substances involved in melanin synthesis. The neutral hypothesis, on the other hand, suggests that nerve injury in specific areas leads to segmental vitiligo, with neurons interacting with substances that are toxic to melanocytes. Lastly, the autoimmune hypothesis... The convergence theory suggests that various factors, such as stress, accumulation of toxic substances, infection, autoimmunity, mutations, altered cellular environment, and impaired melanocyte migration and proliferation, can all contribute to the development of vitiligo in different degrees. There is substantial data indicating that vitiligo is mostly caused by autoimmune. A study compared skin biopsies from patients with vitiligo to those from normal healthy donors and relevant disease controls. The study found that skin-homing T cells play a significant role in the death of melanocytes observed in vitiligo. Genetic factors have a significant role in the development of vitiligo, as evidenced by a family history of the condition in 20% to 30% of individuals. Nevertheless, there is insufficient data to substantiate the precise aetiology of vitiligo.

TREATMENT:

Vitiligo is a dermatological condition distinguished by the presence of white patches on the skin, which is why it is also known as leukoderma (leuko meaning white and derma meaning skin). The absence of melanin, the skin pigment, causes these areas of skin to lack colour. In the majority of cases, sunburn is the primary cause of this disorder. Normally, excessive sun exposure initially causes skin reddening, followed by peeling of the outer skin layers, and production of darker skin in the exposed area ("tanned" skin). However, in rare circumstances, a reaction occurs in which the melanin formation is stopped and the skin loses its color. The patches of white are normally at the site of the bum, but it is also possible for further patches to begin forming elsewhere. Genetic factors are considered to contribute to the sensitivity towards vitiligo, and the condition now utilised more regularly than PUVA as it is less destructive to the skin. As with PUVA, therapy is carried out twice weekly in a clinic or every day at home, and there is no need to use psoralen. Immune mediators Tentative evidence supports a function for tacrolimus. There is short-term evidence for pimecrolimus but long-term data is missing. Skin camouflage In moderate cases, vitiligo patches can be covered using makeup or other cosmetic camouflage techniques. If the affected person is pale-skinned, the patches might be rendered less noticeable by avoiding tanning of the affected skin. Depigmenting In cases of widespread vitiligo the option to depigment the unaffected skin with topical medications such monobenzone, mequinol, or hydroquinone may be considered to render the skin an even hue. The removal of all the skin pigment with monobenzone is permanent and strong. Sun-safety must be adhered to for life to avoid serious sunburn and melanomas.

Depigmentation takes roughly a year to complete could be produced by factors other than sunburn, such as viral infections or physical stress to the skin. The condition appears to have an autoimmune characteristic, in which melanocytes (melanin generating cells) are attacked and killed. Vitiligo usually initially emerges by the age of 20, however it can develop later. There are a lot of therapies for vitiligo with the best evidence for applied steroids and the use of ultraviolet light in combination with creams. Due to the elevated risks of skin cancer, the NHS recommended phototherapy only be used if primary therapies are inadequate.

Chemically-inducedleukoderma(occupational)

Phenolsandotherderivatives

Topicalorsystemicdrug-inducedepigmentation

Geneticsyndromes HypomelanosisofIto Piebaldism

Tuberoussclerosis

Vogt-Koyanagi-Haradasyndrome
Waardenburgsyndrome Hermanski-Pudlaksyndrome
Menke'ssyndrome

Ziprkowski-Margolissyndrome Griscelli'ssyndrome

Postinflammatoryhypopigmentation

Pityriasisalba

Atopicdermatitis/allergiccontactdermatitis Psoriasis

Lichenplanus

Toxicdrugreactions

Posttraumatic hypopigmentation (scar) Phototherapy- and radiotherapy-induced

Neoplasm-relatedhypomelanoses

Melanoma-associatedleukoderma Mycosisfungoides

Infection-relatedhypomelanoses Leprosy

Pityriasisversicolor Leishmaniasis Onchocerciasis

Treponematoses(pintaandsyphilis)

Idiopathic

Idiopathicguttatehypomelanosis

Progressive(oracquired)macularhypomelanosis

Congenital

Nevusanemicus Nevusdepigmentosus

Others

Lichen sclerosuset atrophicus

Melasma(causedbycontrastbetweenlighteranddarkersk in)

Management

The treatment of vitiligo is still one of them ost tough dermatological issues. An key step in the man-agement of vitiligois to first accept that it is not merely cosmetic disease and that there are safe and effective therapies available. These therapies include phototherapy, topical and systemic immunosuppressants, and surgical methods, which collectively may help in arresting the disease, stabilizing depigmented lesions and inducing repigmentation. Choice of treatment depends on numerous aspects including: the subtype of the disease, the size, location and activity of diseases well as the patient'sage, photo- type, influence on quality of life and motivation for treatment. The face, neck, trunk and mid-extremities respond best to therapy, while the lips and distal extremities are more resistive. Repigmentation emerges first in a perifollicular pattern or at the periphery of the lesions. Treatment for at least 2–3months is needed to establish efficacy of treatment. UVlight-based therapy is the most prevalent treatment for vitiligo and, when paired with an additional therapy, is related with.

Management involves a specific therapeutic approach where by patients should always be consulted, as most of the therapeutic alternatives are time demanding and require long-term follow up. Advice on cosmetic concealment by a cosmetician ora professional nurse should be offered and can be advantageous for people with vitiligo affecting exposed regions. These include foundation-based cosmetics and self-tanning products containing dihydroxy acetone which offers permanent color for up to several days. Several guidelines have been developed for the management of vitiligo. In 2008, the British Association of Dermatologists produced user-friendly clinical recommendations for the diagnosis and therapy of vitiligo which were established based on the first Cochrane review and expert consens us on vitiligo reflect

Conclusion

Vitiligo is a frequent multifactorial skin condition with a very complex pathophysiology. Although tremendous progress has lately been made in our understanding of vitiligo, the origin and pathogenesis of vitiligo remain un lucid. Uncertainties remain concerning what finally causes the death of melanocytes, and further investigations are needed to completely explain vitiligo pathophysiology. Uncovering the biological mediators and the molecular mechanisms that contribute to metabolic abnormalities and hence melanocyte degeneration and autoimmunity is important in order to develop new therapeutic targets and medications that could prevent, stop disease progression or perhaps cure vitiligo. Experience with systemic biological therapies that target cytokines such as in psoriasis suggests that a similar approach might be successfully used in vitiligo. As such targeting the IFN-γ-chemokine axis with existing or novel medication is empting and promising. Furthermore, another crucial is suein vitiligo is enhancing their levance offuture vitiligo clinical trials and the ability to compare them. There is a significan the terogeneity of out comemea suresuse dinRCT s for vitiligo. Indeed, Ele ther ia douetal. Reported that 48 different outcome assessment tools have been utilised to measure repigmentation in 54 controlled trials. There are 11 outcome measurement instruments for measuring features of vitiligo Following the foregoing, two worldwide Delphiconsensusonac ore out comeset for vitiligo were undertaken. They defined the successful proportion of repigmentation as being ≥80%. Finally, three workshops with patients with vitiligo have recently been undertaken following the guidance from the Cochrane Skin Group Core Outcome Set Initiative and the Vitiligo Global Issues Consensus Group. The authors advocated the use of percentage of repigmentation quartiles (0–25, 26–50, 51–79, 80–100%) and the Vitiligo Noticeability Scale. This continual endeavour to produceac ore out come setwill increase the ability to use trial data formeta-analyses and will ultimately lead to more confidence

indecisions regarding the optimal care of patients with vitiligo.

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