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Review article on vitiligo

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ABSTRACT -
Vitiligo, a common depigmenting skin disorder, has an esti-mated prevalence of 0.5–2% of the population worldwide.Vitiligo is an acquired, chronic condition characterised by depigmentation of the epidermis or by destruction/loss of melaninVitiligo, whose treatment remains a serious concern and challenge, is an autoimmune skin disease characterized by patches of depigmentation.The disease is characterized by the selective loss of melano-cytes which results in typical nonscaly, chalky-white macules.Vitiligo is often dismissed as a cosmetic problem, although its effects can be psychologically devastating, often with a considerable bur-den on daily life. In 2011, an international consensus classi-
fied segmental vitiligo separately from all other forms of vit-iligo, and the term vitiligo was defined to designate all forms
of nonsegmental vitiligo.
Keywords-vitiligo,depigmentingskin,cosmetic problem

I. INTRODUCING:

Vitiligo, a depigmenting skin disorder, is character-ized by the selective loss of melanocytes, which in turn leads to pigment dilution in the affected areas of the skin. The characteristic lesion is a totally amelanotic, nonscaly,chalky-white macule with distinct margin. Considerable recent progress has been made in our understanding of the pathogenesis of vitiligo, and it is now clearly classified as autoimmune disease, associated with genetic and en-vironmental factors together with metabolic, oxidative stress and cell detachment abnormalities.[1, 2]Vitiligo is a primary, circumscribed, or generalized depigmentation of the skin andmucosa, related to genetic factors, self-destruction of melanocytes, cytokines,autoimmunity, and oxidative stress.[3]The skin loses colour due to a disorder calledvitiligo (pronounced vit-il-EYE-go). [4].In the Aushooryan era, roughly 2200 B.C., vitiligo was first mentioned in writing under the name Kilăsa. Further, the Egyptian Ebers Papyrus also has information on vitiligo that dates back to 1550 B.C.[5].. As a result, several areas of the body, including the skin, hair, and mucous membranes, develop discoloured white marks. The lesion is known as a macule if the area of the skin losing colour is less than 1 centimetre wide and as a patch if it is greater than that[6].To date, available treatments for vitiligo remain limited, and therapeutical strategies rely on nonspecific therapies targeting the inflam-matory and immune responses, such as topical or systemic steroids or topical calcineurininhi-bitors, both associated with ultraviolet (UV)light to promote melanocyte regeneration. This limitation in treatment possibilities highlights the need to improve vitiligo management.Vitiligo can be treated by different modalities of phototherapy, surgical procedures, and topical therapies, such as glucocorticosteroids, immunosuppressive agents, calcineurin inhibitors, and vitamin D. found that the prevalence of vitiligo in the world's population overall ranges between 0.06% and 2.28%and between 0.0 and 2.16% in children and adolescent popumlations.Geographically, prevalence rates vary and are frequently greater in Africa and India[7].

Classification-

type	subtype
segmental	Bisegmental/unisegmental/ plurisegmental
Nonsegmental	Generalized/acrofacial/universal/mucosal
unclassified	Undetermined/focal/mucosal[one site]

Vitiligo canappear clinically in three different ways (according to the evaluation carried out between 2011 and 2012 by the Vitiligo Global Issues Consensus Conference.

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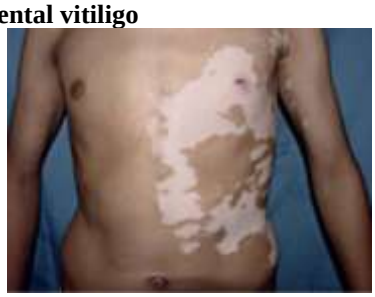
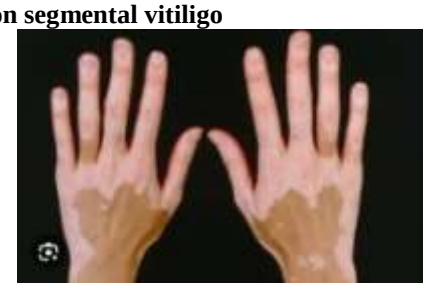
1)Segmental vitiligo



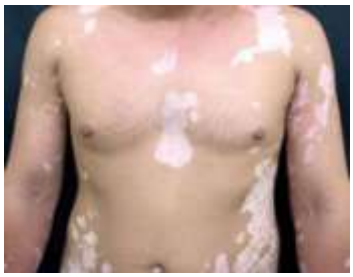
An acquired chronic pigmentation condition called segmental vitiligo is identified bywhite patches that have a unilateral distribution and may completely or partially resemble a dermatome.The most frequently impacted area is the face, followed by the trunk and extremities.Leukotrichia is frequently observed and manifests early in the disease's course. For a period of six months to two years, the disease progresses before stabilising without intervention.It has a poor response to medical treatment when compared to other subtypes of the illness, which could be explained by the frequent occurrence of leukotrichia. [8, 9,10]

2) Non segmental vitiligo



NSV includes the acrofacial, mucosal, generalized, universal, mixed and rare variants. Generalized and acro-facial vitiligo are the most common subtypesThe most common type of vitiligo, which represents 80–90% of all cases, is non-segmental vitiligo(NSV). It is a chronic acquired pigmentation disorder marked by white patches, bilateral, frequently symmetrical, that enlarge over time and typically reflect a considerable loss of functioning melanocytes in the epidermis and some in the hair follicles.When more than 80% of the body's surface is depigmented, the disorder is considered universal vitiligo.[9,11,12]

3) Unclassified/Mixed vitiligo



Unclassifiable forms or undetermined vitiligo include focal, for isolated white macules without segmental distribution, and mucosal, when only one mucosa is affected. Mixed vitiligo (MV) occurs when SV and NSV coexist. The loss of pigmentation surrounding the pre-existing nevus that creates a halo is known as a halo nevus (Sutton nevus). Many halo nevi are a sign of nested pigment-producing cell autoimmunity, which increases the risk of developing vitiligo. [9, 13,14].

- **Pathogenesis**

Vitiligo is a multifactorial disorder characterized by the loss of functional melanocytes.[9, 15,16].Multiple mecha-nisms have been proposed for melanocyte destruction in Vitiligo.The "convergence theory" or "integrated theory"suggests that multiple mechanisms may work jointly in vit-iligo to contribute to the destruction of melanocytes, ulti-mately leading to the same clinical result.[17, 18,19,20,21].NSV and SV were believed to have distinct underlying pathogenetic mechanisms due to their different clinical presentations, with the neuronal hypothesis or somatic mosaicism favored for the segmental form[22].The destruction of melanocytes and the development of white patches in vitiligo have been linked to a variety of different mechanisms. They include neural, genetic, autoim-mune, oxidative stress, production of inflammatory mediators, and other mechanisms for melanocyte separation[9].

1)Genetics of Vitiligo.
2) Oxidative Stress
3) Innate Immunity
4) Adaptive Immunity
5) Biochemical Theory

Epidemiology-

Vitiligo is the most common depigmenting skin disor-der, with an estimated prevalence of 0.5–2% of the popu-lation in both adults and children worldwide [23, 24].Vitiligo affects ethnic groups and people of all skin types with no predilection[25].. This variability in

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epidemiological data could be accounted for by differences in disease classification due to the lack of consensus in previous years, inconsistent reporting by patients and varied populations.Males and females are equally affected, although wom-en and girls often seek consultation more frequently, pos-sibly due to the greater negative social impact than for men and boys[26, 27].Twenty-five percent of vitiligo patients develop the disease before the age of 10 years, almost half of patients with vitiligo develop the disease before the age of 20 years and nearly 70–80% before the age of 30 years[28, 29].

- **Diagnosis**

The diagnosis of vitiligo is generally straightforward, made clinically based upon the finding of acquired, amel-anotic, nonscaly, chalky-white macules with distinct margins in a typical distribution: periorificial, lips and tips of distal extremities, penis, segmental and areas of frictionfriction[30, 31,32].The diagnosis of vitiligo may be facilitated by the use of a Wood's lamp, a hand-held ultraviolet (UV) irradia-tion device that emits UVA[33].It helps identify focal melanocyte loss and detect areas of depigmentation that may not be visible to the naked eye, particularly in pale skin [34]. Under the Wood's light, the vitiligo lesions emit a bright blue-white fluorescence and appear sharply demarcated.Dermoscopy can be used to differentiate vitiligo from other depigmenting disorders. The differential diagnosis of vitiligo is broad(Table-1)

✓ **Chemically-induced leukoderma (occupational)**

Phenols and other derivatives

✓ **Topical or systemic drug-induced depigmentation**

1. Genetic syndromes
2. Hypomelanosis of Ito
3. Piebaldism
4. Tuberous sclerosis
5. Vogt-Koyanagi-Harada syndrome
6. Waardenburg syndrome
7. Hermanski-Pudlak syndrome
8. Menke's syndrome
9. Ziprkowski-Margolis syndrome
10. Griscelli's syndrome

✓ **Postinflammatory hypopigmentation**

1. Pityriasis alba
2. Atopic dermatitis/allergic contact dermatitis
3. Psoriasis
4. Lichen planus
5. Toxic drug reactions
6. Posttraumatic hypopigmentation (scar)
7. Phototherapy- and radiotherapy-induced

✓ **Neoplasm-related hypomelanoses**

1. Melanoma-associated leukoderma
2. Mycosis fungoides
3. Infection-related hypomelanoses
4. Leprosy
5. Pityriasis versicolor
6. Leishmaniasis
7. Onchocerciasis
8. Treponematoses (pinta and syphilis)
9. =

✓ **Idiopathic**

1. Idiopathic guttatehypomelanosis
2. Progressive (or acquired) macular hypomelanosis

✓ **Congenital**

1. Nevus anemicus
2. Nevus depigmentosus

✓ **Others**

1. Lichen sclerosus et atrophicus
2. Melasma (caused by contrast between lighter and darker skin).

- **Treatment:**

Phototherapy and topical and oral immunomodulators such as corticosteroids and calcineurin inhibitors are common vitiligo therapies[35].Psychosocial therapies, depigmentation therapy, non-traditional therapy, and surgical therapy are also some of the other therapeutic options for vitiligo[36]Topical, systemic treatment, and phototherapy are useful for stabilization and repigmentation of vitiligo. Therapeutic options for stable, segmental vitiligo include topical therapies (eg, topical corticosteroids, topical calcineurin inhibitors), targeted phototherapy, and surgical therapy (tissue grafts and cellular grafts)[37].

- **Herbal treatment**

1) Ginkgo biloba

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Ginkgo biloba (also known as "maidenhair tree"), is belong to familyGinkgoaceae. It is one of the oldest trees on Earth and its leaves and seeds had been largely used in medicine for a very long time. In the last few years, gingko extracts have also been used for the treatment of vitiligo. The drug is formulated into a tablet of different dosage, which must be taken orally once to three times daily, for more than three months.

Mechanism of action

The exact mechanism of action of Ginkgo biloba in vitiligo is still unknown, but it seems to be related to the anti-inflammatory, immunomodulatory and antioxidant properties of the drug[38].

2) Cucumis melo

cucumismelo (also known as "Muskmelon") is a species of Cucumis, plants of the belongs to family Cucurbitaceae.

Cucumis melo extract is rich in antioxidants that naturally contain a high superoxide dismutase (SOD) (Table 1) activity, which has been proposed to be important in stopping the melanocytes d e construction by the oxidative stress in the first step of vitiligo. Recently, preliminary studies were conducted to evaluate the efficacy of a topical preparation, containing Cucumis melo superoxide dismutase (SOD) and catalase, in the treatment of vitiligo [39, 40].

Herbs	Active components
Cucumis melo	Cucumis melo superoxide dismutase
Green Tea	Epicatechin, epicatechin-3-gallate, epigallocatechin
Picrorhiza kurroa	Picroside I and picroside II
Polypodium leucotomos	p-coumaric, ferulic, caffeic, vanillic, 3,4 - dihydroxybenzoic, 4 - hydroxybenzoic, 4 - hydroxycinnamic, 4 - hydroxycinnamoyl - quinic, chlorogenic acids

3)Khellin

Khellin is a naturally occurring furanochromone, derived from the plant Amnivisnaga. It is developed and introduced in medicine in the last decades for the treatment of vitiligo, where they provide good results in combination with UVA phototherapy.

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Mechanism of action
Even if the exact mechanism of action is unclear, khellin acts by stimulating melanocytes proliferation and melanogenesis.

4) Picrorhizakurroa

Ayurvedic medicine had tried to treat vitiligo with herbal products, such as Picrorhizakurroa. Picrorhizakurroa (also known as "Kutki" or "Kutaki").It is belongs to family Plantains.It is another khellin extract, with well - known hepatoprotective properties. More recently, researchers have proposed how the herbal extract has antioxidant and immune-modulating activities too (Table 1).

4) Capsaicin

Capsaicin is one of the active component of chili peppers, plants of the genus Capsicum. Because its antinflammatory and antioxidant properties, the drug has been proposed as a therapeutic tool for vitiligo treatments.

II. CONCLUSION:

Current models of treatment for vitiligo are often nonspecific and general. Vitiligo is a common multifactorial skin disorder with a very complex pathogenesis. Uncertainties remain about what ultimately causes the destruction of melanocytes, and further studies are needed to completely elucidate vitiligo pathogenesis.Vitiligo can affect anyone, regardless of gender, ethnicity, age, or skin colour. Most vitiligo patients desire to hide their visible lesions by using clothing, camouflage, shade cream bases, and other methods that can help them improve their quality of life and social functioning. Recent research advances in our understanding of the pathogenesis of vitiligo has led to the development of targeted therapies for this dis-ease, which has a high impact on patients'quality of life. JAK inhibition is showing promising results, and ongoing clinical trials could lead to a first approved treatment for vitiligo.

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