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A Review on Vitiligo Disease

Anushka Manekar, Rajlaxmi Deolekar, Sneha Kukade
Students, Final Year, New Montfort Institute of Pharmacy, Ashti, Wardha, India
anushkamanekar7@gmail.com

Abstract: *Vitiligo is a chronic autoimmune disease that cause white patches to appear on the skin. Vitiligo is a long -lasting disease that occurs when the body's immune system attacks and destroys the skin's pigment-producing cells, called melanocytes. It can appear as white patches of varying sizes and shapes, usually well demarcated. The patches can appear anywhere on the body, but often appear symmetrically on both sides. There is no cure for vitiligo, but treatment can help even out skin tone and stop the progression of the disease. people with vitiligo should protect affected areas from the sun with sunscreen and clothing because they are at a higher risk of sunburn and skin cancer. Vitiligo occurs when pigment-producing cells die or stop producing melanin. The Ebers papyrus in 1550 BC mentioned two forms of depigmentation that could be interpreted as leprosy or depigmentation resembling vitiligo.*

Keywords: Vitiligo.

I. INTRODUCTION

Vitiligo is an acquired, usually asymptomatic pigmentary Disorder that results in the loss of functional melanocytes and is often associated with other autoimmune diseases. At the onset of the disease white patches of different sizes Appear on different parts of the body. vitiligo affects Approximately 1% of the world population of all skin types, usually before the age of 20. Its psychological impact on the quality of life can be disastrous, as dissatisfaction with Body image can smother self-esteem and develop a depressive State, especially among dark or tan-skinned patients. [1] Pathogenesis of Vitiligo is an intriguing disorder whose cause has been an Extensive topic of debate. The exact origin of vitiligo is Still unclear, and the pathogenesis is complex and involves the interplay of a series of variables. [2] Vitiligo is an acquired pigmentation disorder, Characterized by depigmented patches, as a result of the disappearance of functioning melanocytes from the epidermis. This condition, cosmetically Disfiguring especially in dark-skinned individuals, Makes the lesional skin more sensitive to sunburns and affects 0.1–2% of the world's population, irrespective of gender and race.[3]

The histologic picture shows loss of melanocytes and melanin in the white patches, and an Inconstant lympho-mononuclear infiltrate in the Advancing margins of vitiligo. Diagnostic criteria are mainly clinical, based on the findings of acquired, well-demarcated white Lesions on the skin, with no associated inflammation that tend to enlarge centrifugally. A recent classification subdivides vitiligo into Two clinical types: segmental (type B) and nonsegmental (type A).[4] Accordingly, specific autoantibodies for melanocytic lineage give no evidence of melanocytes in completely vitiliginous skin. These Histologic data are also supported by electron Microscopy, which fail to identify melanocytic Cells in vitiligo achromic patches.[5]

FIG NO. [1]

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In addition, cultured melanocytes can be Obtained from pigmented skin of vitiligo patients. Vitiligo can be associated with an autoimmune disorder that Can extend beyond the skin. Many observations support the role of altered humoral immunity, cellular immunity, autoimmunity.[6] And cytokines in the pathogenesis of vitiligo. The process of Depigmentation is associated with a T-cell mediated immune Response to melanocytes during disease progression. Many Patients also report that emotional stress, including the death of A first-degree relative or childbirth, is a precipitating factor.[7] Mechanical injury after cuts and burns can induce new lesions due To Koebner's phenomenon, whereby local trauma to the skin can Induce depigmented patches;3 this effect occurs in the majority of Vitiligo patients. Vitiligo is an acquired, idiopathic, and worldwide common depigmentation disorder affecting people of all ages and both sexes equally. Patients lose their skin colour over Time, mostly in a patchy and progressive manner. The Cause of the disease is still under debate.(8)

Although there are rarely other physical symptoms involved, many Patients experience stigmatization, unwanted attention, Negative comments, rejection, or bullying. The prevalence of vitiligo is often said to range from 0.09 to 8%, Especially in India. [9] Vitiligo is an acquired pigmentation disorder, Characterized by depigmented patches, as a result Of the disappearance of functioning melanocytes From the epidermis. This condition, cosmetically Disfiguring especially in dark-skinned individuals, Makes the lesional skin more sensitive to sunburns And affects 0.1–2% of the world's population, Irrespective of gender and race.[10]The histologic picture shows loss of melanocytes And melanin in the white patches, and an Inconstant lympho-mononuclear infiltrate in the Advancing margins of vitiligo.[11]Diagnostic criteria are mainly clinical, based on The findings of acquired, welldemarcated white Lesions on the skin, with no associated inflammation that tend (type B) and nonsegmental (type A) . Type B is more and has a Dermatomal distribution; after a rapid onset and Evolution usually exhibit a stable course. Type A is More common, has a potential lifelong evolution, and is associated with Koebner phenomenon and frequently with autoimmune diseases such as Sutton nevus, thyroid disorders, juvenile diabetes Mellitus, pernicious anaemia, and Addison's Disease. Another clinical classification is based on the

Distribution and extension of lesion according to Norland.[12]

Vitiligo is an acquired idiopathic dermatological disorder characterized by the appear acne and development of white macules related to the apoptosis or selective damage of melanocytes. Approximately 0.5–1% of the individuals are diagnosed with vitiligo.[13] The highest reported prevalence has been recorded in India (up to 8.8%), followed by Mexico (2.6–4%) and the Japan (1.68%2). depigmentation usually occurs on exposed areas of the body including the face, neck and arms. The extreme effects of vitiligo often bring dramatic psychological burden to afflicted patients. Although males and females are equally affected by this disease, women more often openly express and address vitiligo for cosmetic purposes and are more likely to seek treatment.[14] The incidence of vitiligo often presents through distinct familial clustering with reports that 20%ofthevitiligopatients also have relative diagnosed with the disorder.4 Individuals with a positive family history usually have an earlier age of onset as well as a longer duration5 compared to individuals that do not have a family history of the disease. Tel +86-571-88013011 Fax +86-571-88018442 Email louxf@zucc.edu.cn Thus far, the ethology of vitiligo has been found to result from multiple factors and has not yet fully been elucidated. In the 1950s, Lerner investigated 600 vitiligo submit your manuscript | www.dovepress.com Dove Press International Journal of Nanomedicine 2020:15 3267–3279 3267 ©2020Sunetal. This work is published and licensed by Dove Medical Press Limited. The full terms of this license are available at https://www.dovepress.com/terms.phphttp://doi.org/10.2147/IJN.S245326andincorporate the Creative Commons Attribution– Non-commercial (unparted, v3.0) License (http://creativecommons.org/licenses/by-nc/3.0/). By accessing the work you hereby accept the Terms. Non-commercial uses of the work are permitted without any further permission from Dove Medical Press Limited, provided the work is properly attributed. For permission for commercial use of this work, please see paragraphs 4.2 and 5 of our Terms (https://www.dovepress.com/terms.php). Sun et al Dove press patients and found that most patients with segmental vitiligo suffered from emotional imbalances or hyperhidrosis, even tally giving rise to the neural theory. [15]

As research pro grassed, factors including stress, autoimmune diseases, melanocyte ternary and autoinflammation have been identified as important factors contributing to vitiligo. Of all theories, autoimmune disease or autoinflammation8–11 and oxidative stress12 as well as interactions among and between them have been accepted as some of the most

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important factor contributing to the disease. [16]In the disease, antigen presenting cells activate T cells through the presentation of melanocyte antigens where T cells then directly kill the melanocytes. It has been reported that endogenous killer and inflammatory dendritic cells are in a hyperactive state in patients with vitiligo.9,10 Various cytokines including INF-γ,14–16 CXCL10,14,17,18 TNF-α, IL-6 and IL-1719–22 are also secreted by innate cells through an autoimmune response. Separate from the autoinflammation theory, oxidative stress is also an important risk factor for vitiligo. Melanin synthesized by melanocytes is toxic which stimulates the cell stress signalling pathway in these cells. Moreover, active energy metabolism in the mitochondria leads to an excessive accumulation of reactive oxygen species (ROS). This also gives rise to the development of vitiligo.[17]

Briefly, when tiny lesions generated through a sunburn, viral infection or physical trauma occur, epidermal damage associated molecules are released into the body and oxidative stress levels are increased. The adhesion of melanocytes is lost and inflammasomes are activated by the release and induction of molecular and oxidative stress products. After a series of immunoreactions, specific cytotoxic T cells are accumulated on the skin, regulatory T cell activity is down regulated and inflammatory cytokines as well as autoantibody dies are produced, ultimately leading to immune-based melanocyte destruction.[18] Likewise, oxidative stress contributes to the onset of depigmentation, and sub sequent undesired autoimmune responses lead to the pro grissino of vitiligo.[19]

Graph 1: Age distribution of 100 patients with vitiligo followed up for 10 years
anos = years

FIG NO: [2]

HISTORY OF VITILIGO DISEASE

Historical background Koebner's phenomenon (KP) is a well-known phenomenon in dermatology. The reaction is named after a German dermatologist, Heinrich Koebner (1838–1904), who observed in his patients with psoriasis the development of new lesions at sites of skin trauma (Ko, 1877). Over the years, numerous reports have been published describing this phenomenon in a wide range of dermatological disorders after diverse environmental stimuli. The experimental induction of this reaction is termed 'the Koebner experiment' (Miller, 1982a). Since Koebner's discovery, there have been several attempts to provide insight into the pathogenesis and clinical relevance of this reaction. Definition Koebner's phenomenon (KP), also called 'isomorphic response', has been defined as 'the development of lesions at sites of specifically traumatized uninvolved skin of patients with cutaneous diseases Identification of vitiligo susceptibility genes Three very different approaches have been used to identify genes that might mediate susceptibility to vitiligo. Gene expression analyses have attempted to identify genes that are differentially over- or under expressed in cells or tissue from vitiligo patients versus controls, or from disease tissue versus normal tissue from patients. These studies can generate lists of candidate genes, but cannot distinguish genes with primary effects from the many more genes whose expression may be dysregulated on a secondary basis or that show 'differential' expression due to individual variation due to outbred genetic background among humans. Genetic association studies have been used to test specific candidate genes, considered to perhaps be involved in vitiligo susceptibility on the basis of an a priori biological hypothesis. the inheritance of vitiligo has been suggested as polygenic.

More than 50 loci have been associated with vitiligo, and many genetic loci are common to other autoimmune disorders. (Spritz & Andersen, 2017) A meta-analysis found a significant association of HLA-A2 with vitiligo and subsequently associations with other class I and II genes of major histocompatibility complex were found. (Liu et al., 2007; Spritz & Andersen, 2017) Other genetic association include CTLA4, PTPN22, NLRP1, GZMB, IL2RA, FOXP1, FOXD3, XPB1, MC1R, and TYR, supporting the role of innate/ adaptive immunity, melanocyte stress, and apoptosis in the pathogenesis of vitiligo. (Spritz & Andersen, 2017).

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FIGNO:[3]

SYMPTOMS OF VITILIGO DISEASE

The most important symptom of vitiligo known is the depigmentation of patches of skin. Initially, the patches are small but they will be enlarged over time. The skin lesions are dominantly observed on the face, hands and wrists. Often patients who are suffering from this disease also suffer from depression.[20]

Vitiligo signs include:

Patchy loss of skin colour, which usually first appears on the hands, face, and areas around body openings and the genitals

Premature whitening or greying of the hair on your scalp, eyelashes, eyebrows or beard

Loss of colour in the tissues that line the inside of the mouth and nose (mucous membranes)

Vitiligo can start at any age, but usually appears before age 30.

Depending on the type of vitiligo you have, it may affect:

Nearly all skin surfaces. With this type, called universal vitiligo, the discoloration affects nearly all skin surfaces.

Many parts of the body. With this most common type, called generalized vitiligo, the discoloured patches often progress similarly on corresponding body parts (symmetrically).

Only one side or part of the body. This type, called segmental vitiligo, tends to occur at a younger age, progress for a year or two, then stop.

One or only a few areas of the body. This type is called localized (focal) vitiligo.

The face and hands. With this type, called acrofacial vitiligo, the affected skin is on the face and hands, and around body openings, such as the eyes, nose and ears.

It's difficult to predict how this disease will progress. Sometimes the patches stop forming without treatment. In most cases, pigment loss spreads and eventually involves most of the skin. Occasionally, the skin gets its colour back.

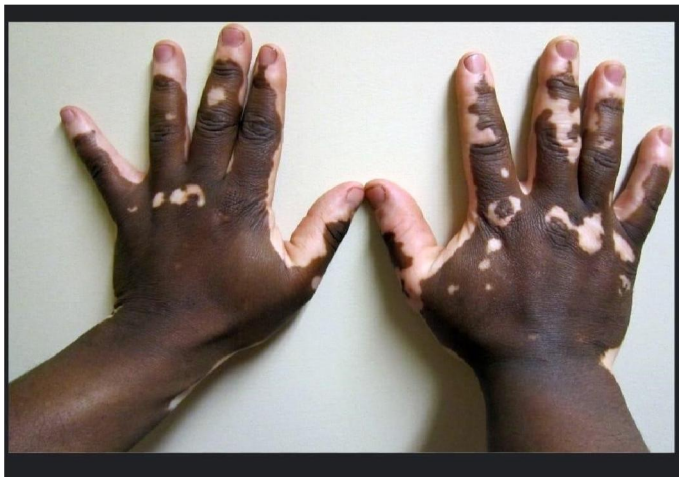

FIG NO:[4]

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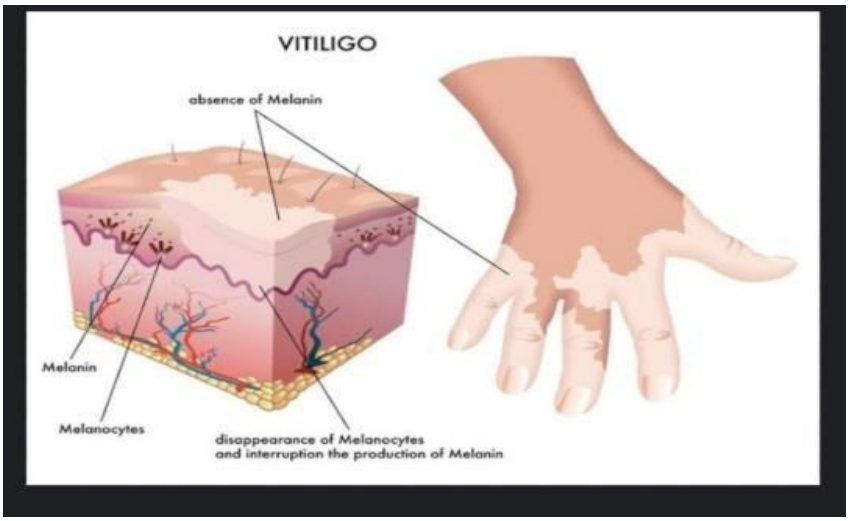

FIG NO:[5]

CAUSES OF VITILIGO DISEASE

FIG NO.[6]

Vitiligo is caused by the lack of pigment called melanin in the skin.

Other causes white patches to develop on your skin or hair. Vitiligo occurs when pigmentproducing cells (melanocytes) die or stop producing melanin — the pigment that gives your skin, hair and eyes colours. The involved patches of skin become lighter or white. It's unclear exactly what causes these pigment cells to fail or die. It may be related to:

A disorder of the immune system (autoimmune condition)

Family history (heredity)

A trigger event, such as stress, severe sunburn or skin trauma, such as contact with a chemical

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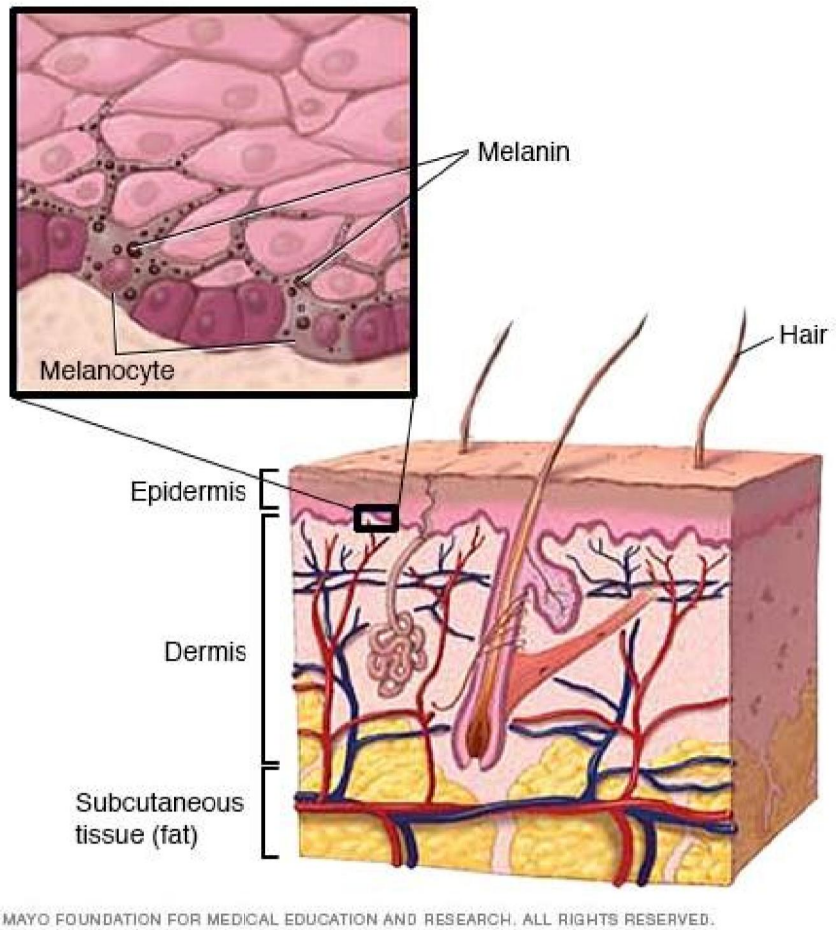

FIG NO.[7]

Skin layers and melanin is a natural pigment that gives your skin its colour. It's produced in cells called melanin.

PATHOGENESIS OF VITILIGO DISEASE

Although the cause of vitiligo is unknown, there are three hypotheses for its pathogenesis including biochemical/cytotoxic, neural and autoimmune: the biochemical/cytotoxic hypothesis emphasizes that vitiligo occurs when the melanocyte is killed by cytotoxic precursors to melanin synthesis; the neural hypothesis is based on nerve injury development with effected sites that leads to segmental vitiligo with neurons that interact with melanocytes and release melanocyte toxic substrates; the autoimmune hypothesis is based on genetic data which are more associated to autoimmune disease.[21]

Vitiligo is an intriguing disorder whose cause has been an extensive topic of debate. The exact origin of vitiligo is still unclear, and the pathogenesis is complex and involves the interplay of a series of variables.[22] There is a multifactorial genetic component predisposing certain individuals to vitiligo and family history is a variable found in approximately one-third of the people with the disease. There is also strong genetic evidence of a link between vitiligo and other autoimmune diseases. [23] Autoimmune Diseases According to the neural theory, segmental vitiligo follows the same path as dermatome, and dysfunction of the sympathetic nervous system can curb melanin production and lead to depigmentation. The intrinsic theory suggests that defects in vitiligo melanocytes lead to their death. These include morphologic defects, decreased adhesive properties, and deficient melanocyte growth factors.[24] Increased oxidative stress has also proved to be an important cause for melanocytes destruction. [25].

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The theory of autoimmune mediated destruction of melanocytes is well accepted and seems to have currently become the leading hypothesis in vitiligo pathogenesis. The immune reaction can be mediated by cellular immunity, humoral antibody-mediated immunity, and the action of cytokines. The action of antibodies against different melanocyte associated antigens was confirmed in vitiligo. The main antigen recognized by these antibodies is tyrosinase, but antibodies against tyrosine hydroxylase, pigment cell surface antigens, and antithyroid antigens have also been found.[26]

Cell-mediated immunity in vitiligo is demonstrated by the presence of inflammatory infiltrates in perilesional vitiligo skin. Decreased CD4+ to CD8+ lymphocytes ratio in vitiligostricken skin compared to healthy skin and CD8 T cells directed against melanocytic antigens have been found both in perilesional skin and in the blood of vitiligo patients. [27]

This shows that the elimination of melanocytes by cytotoxic T cells is a mechanism leading to depigmentation in vitiligo. Cytokines also seem to play an important role in vitiligo pathogenesis. There is an increase in the expression of tumour necrosis alpha (TNF-α) and interferon-gamma (IFN-γ), suggesting that vitiligo is mediated by a T helper cell-1 (Th1) response.

TREATMENT OF VITILIGO DISEASE

There are no treatments ensuring the complete cure of vitiligo. The main goal of the current treatments is either to control the autoimmune destruction of melanocytes or stimulate their growth on affected areas. Treatment can encomia pass pharmacological, physical, and surgical approaches or still a combination of different procedures. Pharmacological treatment consists of a topical and systemic corticosteroid therapy, topical calcineurin inhibitors such as tacrolimus and pimecrolimus, pseudo catalase, mela genuine (human placental exact) and vitamin derivative. NB-UVB), and excimer laser. These treatments are free quaintly combined with topical treatment and transplantation processes.[29] Surgical treatment can bean option and is us uallyrecom mended for patients with stable vitiligo (with no change in lesions numbers or morphology), for which other treatments had no significant results. There are various techniques, but the most usual technique consists of transplanting a pigmented skin graft from a donor onto the patient's affected areas. Surgical treatments include suction blister grafts, punch grafts, Mini grafts, and split thickness skin grafts.[30]

Melanocytes and keratinocytes transplantation is also a surgical intervention by which cell suspension is trans ferried from a donor's pigmented skin onto a previously pre pared recipient, usually by means of dermabrasion. Cultured and non cultured cells can be used. Non cultured melanocytes-keratinocytes cellular grafting yields significant pigmentation results in patients with stable forms of vitiligo, as seen for data previously collected by our group.[31] vitiligo, the results are different for each patient and hardly ever satisfying, as these people can be affected by a series of unknown factors. Knowledge of the immune parameters associated with vitiligo pathogenesis is therefore vital in predicting and suggesting the right type of treatment for each patient.Several studies pointed out the involvement of T cells in vitiligo pathogenesis, and a CD8 T- cell infiltrate is consistently found in vitiligo skin. Furthermore, after regimentation, vitiligo lesions often recur on similar anomic sites, suggesting the involvement of TRM. We recently identified subsets of CD69+ CD103+ and CD69+ CD103- TRM that accumulate both in the epidermis and the dermis of perilesional skin of vitiligo patients with stable or active disease, CD103+ TRM being more abundant within the epidermis. Melanocyte-specific TRM are characterized by the expression of CXCR3 and mount under acti ovation high levels of the pro-inflammatory cytokines TNFα and inter Ferron IFNY. TRM expressing CD49a were also observed both in the dermis and epidermis of vitiligo lesional skin and were characterized by production of IFNY, perforin and granzyme B,[11] notably when TRM cells were cultured in the presence of IL- 15, a cytokine that has been shown to be involved in TRM differentiation and maintenance. In line with the involvement of TRM in vitiligo, melanoma- associated vitiligo supports TRM that can provide anti- tumour immunity in vivo. Soluble factors produced by TRM will be further involved in me lipocytes loss observed in vitiligo. Indeed, TNFα or IFNY have been previously shown to inhibit melanocyte function, among other in amatory cytokines.[4] Regulatory T cells (Treg) dysfunction has also been previously reported in vitiligo skin and their replenishment could be of interest to limit depigmentation. Interestingly, skin resident regulatory T cells have been shown to be critical for hair follicle stem cell regeneration.[32]

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FIG NO :[8]

RISK FACTORS OF VITILIGO DISEASE

Lifestyle Risk Factors

There's no way to know for sure whether a person will develop vitiligo. However, experts have identified several risk factors that are thought to increase the chances— particularly if the person has a genetic predisposition to the condition.

Skin Damage or Trauma

Research has shown that vitiligo development might be more likely in areas where there has been frequent sun exposure and severe sunburns (commonly on the face, neck, and hands).

Areas of the skin that have been affected by trauma, such as a deep cut or repeated rubbing, friction, scratching, or pressure, may also be more likely sites for vitiligo to occur. Related: Skin Changes Caused by Trauma

Stress

Studies have shown that stressful events or chronic emotional and physical stress may trigger both the development and progression of vitiligo, particularly in patients who are predisposed because of their genes.

It's thought that the skin changes are prompted, at least in part, by the hormonal changes that happen when a person experiences extreme stress. Evidence has also shown that trauma and significant life stressors are linked to autoimmune disease.

Song H, Fang F, Tomasson G, et al.Association of Stress-Related Disorders With SubsequentAutoimmune Disease.*JAMA*. 2018;319(23):2388-2400. doi:10.1001/jama.2018.7028

Related: Autoimmune Skin Disorders

Chemical Exposure

Contact with or exposure to certain chemicals might be another environmental risk factor for developing vitiligo. Some experts have hypothesized that the chemicals accelerate stress pathways that are already present in melanocytes, leading to autoimmune inflammation.

In addition, genetic influences may increase cellular stress in melanocytes or set a lower threshold for stress that the immune system can handle.

One of the chemicals that has been studied is monobenzone, which is found in certain products like rubber, leather, and cosmetic dyes. Research has found that monobenzone can prompt skin depigmentation to develop and worsen in people who already have vitiligo.

Another category of chemicals that might play a role in vitiligo is phenols, which are thought to disrupt melanocyte function. These chemicals are often ingredients in products such as adhesives, disinfectants, paints, insecticides, and more.

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Related: 8 Ways Environmental Pollutants Can Harm the Body

FIG NO.[9]

II. CONCLUSION

BY these thesis I would like to conclude that vitiligo is an acquired, usually asymptomatic pigmentary Disorder that results in the loss of functional melanocytes and is often associated with other autoimmune diseases. At the onset of the disease white patches of different sizes Appear on different parts of the body.(Melanocytes are the cells that produce the pigment that gives skin and hair their colour) This disease affects Approximately 1% of the world population of all skin types, usually before the age of 20.Basically it is a skin disease that cause white patches .it is cause by lack of pigment called melanin in skin ,but its exact cause is unknown and it may cause by combination of genetic and environmental factors. pathogenesis including three hypothesis that is biochemical/cytotoxic, neural and autoimmune. There are different theories about the pathogenesis of vitiligo but exact etiology is still unknown. symptoms are loss of skin colour, loss of colour in muscus membrane, sensitivity to sunlight,rapid pigment loss. AGE:it can start in any age but it usually appears before age of 30. It may diagnosis bye wood's lamp examination skin biopsy ,blood test . There are no treatments to complete cure of vitiligo. The main goal of the current treatments is either to control the autoimmune destruction of melanocytes or stimulate their growth on affected areas.

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