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Provocating factors of vitiligo

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Abstract

Vitiligo is an acquired, chronic depigmenting disorder of the skin. Although the exact cause is still under debate, the disease results from the selective loss of melanocytes, which in turn causes pigment dilution in the affected areas of the skin and/or mucosa. Melanocyte precursors can be found in the hair follicle bulge; differentiated, pigment-producing melanocytes reside in the basal layers of the epidermis and the hair matrix. Depending on the disease course, skin and hair are affected to different degrees. Clinically, skin lesions present as milky white, non-scaly patches with distinct margins. The researchers are trying to explain the pathogenesis of vitiligo with different hypotheses mainly; genetic, autoimmune, autocytotoxic, neurogenic, microenviromental,viral, apoptotic cell, adhesion disorders and multivariate theories .The most popular theory of vitiligo development is a multifactorial hypothesis according to which genetic conditions predispose vitiligo macules to occur as a result of specific environmental factors.In this study , was aimed to evaluate the provocating factors of vitiligo in vitiligo patients .

Keywords: Provocating, vitiligo.

1. Introduction

Vitiligo is the commonest depigmenting disorder worldwide.The largest epidemiological study was carried out in 1977 on the island of Bornholm in Denmark, where the disease was found to influence 0.4% of the population. [1]

Although vitiligo does not result in any restrictions on life expectancy or a patient's capacity to work, it can cause cosmetic disfigurement and may affect the psychological well-being of patients. [2] Patients often complain from stigmatization such as curiosity by other people, rejection and discrimination at work, low self-esteem, embarrassment, impaired quality of life, and higher prevalence of sexual difficulties, especially in women. [3]

Vitiligo predisposition is defined by variant sequences at loci associated with both the innate and adaptive immune system as well as to loci associated with melanogenesis and apoptosis. [4] Precipitating factors have been acknowledged, including exposure to sunlight or skin trauma, leading to oxidative stress in melanocytes .[5] and T-cell mediated autoimmune responses. Indeed, while vitiligo has been established as an autoimmune, entitythe mechanism connecting the initiating event(s) to the induction of anti-melanocyte T-cell immunity is unknown. [6]

Physical or environmental stressors are reported in the onset and disease progression of vitiligo , In the event of a sunburn or exposure to certain chemicals or skin trauma, free radicals and hydrogen peroxide are generated, and in individuals who are predisposed to vitiligo. Psychological stressors also play a role in vitiligo , Events such as death of a family member, work and financial problems have been associated as preceding factors to the onset of vitiligo. [7] In addition, vitiligo patients experience severe psychological effects .[8] and exhibit anxiety ,depression, social stigma ,and impaired quality of life [9] . Stress increases the levels of catecholamines, neuropeptides, and cortisol that are higher in vitiligo patients. [10]

2. Subjects and Methods

This study was a case-control study. This study included (100) patients suffering from vitiligo. In addition to (100) healthy control group consisted of sex and age matched vitiligo free individuals .All patients were selected from the outpatient clinic of Dermatology, Venereology and andrology Department of Benha University Hospitals.

An informed consent was obtained from all participants. The study was approved by the local ethics committee on research involving human subjects of Benha Faculty of Medicine.

All patients included in the study have vitiligo . patients with skin disease other than vitiligo and subjects with acute or chronic infections , malignancies, autoimmune disorders,hepatic or renal diseases were excluded.

2.1. VASI score

Vitiligo area severity index (VASI): involves breaking the body into hand units, each of which is approximately 1% of the total surface area of the body. One hand unit encompasses the palm plus the volar surface of all digits. Within each hand unit, the degree of depigmentation was estimated and quantized into one of 6 levels. The extent of depigmentation within each hand unit-measured patch (possible values of 0, 10, 25, 50, 75, 90 or 100%). The total body VASI was calculated using the following formula by considering the contributions of all body regions. VASI = Σ (all body size) (hand units) x (depigmentation). [11]

2.2. Lab analysis

Venous blood samples was collected under complete aseptic conditions in tubes with EDTA anticoagulant from all participants,and do lab

ananalysis, CBC,ESR,CRP,ANA,LFT,KFT and thyroid function tests T3,T4,TSH.

2.3. Statistical Analysis

- Analytical statistics:
- Student T Test was used to assess the statistical significance of the difference between two study group means.
- For the comparison of the three groups' means, one way analysis of variance (ANOVA) was used.
- Chi-Square test was used to examine the relationship between two qualitative variables
- Fisher's exact test: was used to examine the relationship between two qualitative variables when the expected count is less than 5 in more than 20% of cells.
- Regression analysis: Logistic and linear regression analyses were used for prediction of risk factors. Odds ratio and 95% confidence interval were calculated. Odds ratio and 95% confidence interval were calculated. Odds ratios are used to determine whether a particular exposure is a risk factor for a particular outcome, OR=1 Exposure does not affect the outcome, OR>1 Exposure associated with higher risk of outcome; OR<1 Exposure associated with lower risk of outcome.
- Deviations from Hardy–Weinberg equilibrium expectations were determined using the chi-squared test.
- The HaploView program (version 4.2) was applied to estimate the haplotypes (Barrett et al 2005).
- All reported p values were two-tailed and p <0.05 was considered to be significant.

3. Results

Demographic data of studied groups

There was insignificant difference between patients and control subjects regarding age (p = 0.104) and sex (p = 0.834) Table (1).

Table (1) Demographic data of the study groups.

		Control N=100	Vitiligo N=100	p
Age (years)	**mean±SD**	43.6 ± 8.4	40.2 ± 11.3	0.101
Males	**N (%)**	36 (36)	34 (34)	0.767
Females	**N (%)**	64 (64)	66(66)	

SD, standard deviation, p≤0.05 is significant.

History findings

The mean age of vitiligo onset was 19.6 ± 6.1 years. The mean disease duration was 20.4 ± 5.3 years. **(Table 2)**

Table (2) History findings in all studied cases.

		Cases N=100
Age of onset (years)	**mean±SD**	19.6±5.5
Duration (years)	**mean±SD**	20.4±4.8
Positive family history	**N (%)**	20 (20)

SD, standard deviation, p≤0.05 is significant.

The reported risk factors in the studied patients included stress in 88 patients (88 %) , sun exposure in 4 patients (4 %) and truma in 8 patients (8 %) Figure (1).

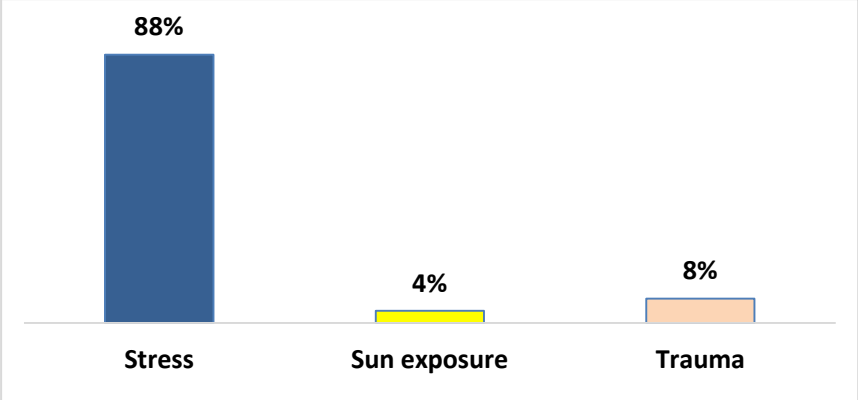

Fig. (1) Provocating factors in the studied cases.

Table (3) Generalized clinical examination in all studied cases.

			Vitiligo N=100	
Site of affection	**Head and neck**	**N, %**	36	36%
	Trunk	**N, %**	4	4%
	Extremities	**N, %**	72	24%
	Generalized	**N, %**	24	72%
Eye or ear affection		**N, %**	16	16%
Hair affection		**N, %**	12	12%
Repigmentation	**Marginal**	**N, %**	36	36%
	Diffuse	**N, %**	12	12%
	Perifollecular	**N, %**	8	8%
	Combined	**N, %**	44	44%
Basal VASI		**mean±SD**	47.3	±14.2

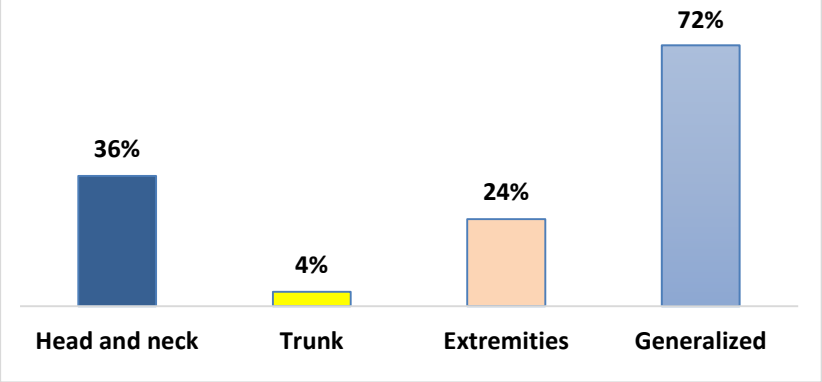

Fig. (2) Generalized clinical examination in all studied cases.

Clinical examination in all studied cases

Head and neck were affected in 36%, trunk in 4%, extremities in 24%, generalized in 72%. Eye or ear were affected in 16%, hair was affected in 12%. Repigmentation was marginal in 36%, diffuse in 12%, perifollecular in 8% and combined in 44%.Mean baseline VASI score was 2.4. Table (3)

4. Discussion

Vitiligo is a skin disease characterized by the loss of pigment in patches of skin. Approximately 1% of the world's population is affected by vitiligo, and in some countries, this percent can be as high as 2–3%. In general, there is no significant difference in gender for susceptibility to vitiligo. Approximately half of vitiligo patients develop this disorder before 20 years old, and the age of onset of vitiligo for most patients is before 40. Vitiligo patients may experience depression and relevant mood disorders due to the potential for discrimination from society. [12]

The pathogenesis of vitiligo remains elusive, although many theories such as autoimmune hypothesis, genetics theory, reactive oxygen species model, zinc-α2-glycoprotein deficiency hypothesis, viral theory, intrinsic theory and biochemical, molecular and cellular alterations accounting for loss of functioning melanocytes in vitiligo were elaborated to clarify vitiligo pathogenesis. [13]

In the current study the reported risk factors in the studied patients included stress in 88 patients (88%),sun exposure in 4 patients (4%) and truma in 8 patients (8%). All of them received previous treatment .None had other diseases. Mean age of onset was 19.6 years.Mean duration was 20.4 years .Most of cases were progressive 80% and only 20% were stable.Head and neck were affected in 36%,trunk in 4%, extremities in 72%, generalized in 24%.Eye or ear were affected in 16%,hair was affected in 12%. All cases had NSV.None had positive koebner sign. Depigmentation was marginal in 36%, diffusion in 12%, perifollicular in 8% and combined in 44%.

5. Conclusion

From this study we can concluded that stressful events,sun exposure and truma trigger the development of vitiligo, . Emotional stress can worsen vitiligo and cause it to become more severe.

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