

Risk Factors for Acne Vulgaris Development

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Abstract

Acne vulgaris is a disease of pilosebaceous unit that is most commonly seen during adolescence . Increasing pubertal age, seborrhea, the premenstrual phase, mental stress, and sweet and oily foods were recognized as risk factors for moderate to severe acne. Also gender, spicy foods, and smoking were not associated with acne severity This case-control study included 50 participants; 30 patients suffering from moderate and severe acne vulgaris, in addition to 20 apparently healthy, age and sex matched individuals . Present history including age of onset of acne, disease duration, course of the disease and risk factors (diet, sun exposure , smoking, stress) family histories were obtained from all patients. logistic regression analysis was conducted for prediction of acne vulgaris development, using age, gender, smoking, risk factors,. Positive FH, smoking, higher frequency sun exposure, high caloric diet, excess stress, were associated with risk of acne vulgaris occurrence in univariable analysis. Estimation of risk factors including Positive FH, smoking, higher frequency sun exposure, high caloric diet, excess stress are important in prediction and management of acne vulgaris .

1. Introduction

Acne vulgaris is a disease of pilosebaceous unit that is most commonly seen during adolescence with different severity among individuals [1]. It is an inflammatory skin disease with numerous factors in its etiopathogenesis [2].

The main factors are inflammation, abnormal keratinization, microbial flora changes, and increased sebum production. The sequence in which these events occur is still not certain, but inflammation has been suggested as the initial factor. The mechanisms behind the beginning and maintenance of the inflammatory response are not fully known, but *Propionibacterium acnes* plays an important role in these mechanisms [3].

. Increasing pubertal age, seborrhea, the premenstrual phase, mental stress, and sweet and oily foods were recognized as risk factors for moderate to severe acne. Also gender, spicy foods, and smoking were not associated with acne severity [4].

The heritability of acne is almost 80% in first degree relatives. Acne occurs earlier and is more severe in those with a positive family history [5].

The aims of this study were to assess the Risk factors for acne vulgaris development in patients with acne vulgaris and to evaluate the relation between them and the clinical aspects of the disease.

2. Patient and method

This case-control study was conducted in the Outpatient Clinic of Dermatology, Venereology and Andrology Department of Benha University. The study included 50 participants; 30 patients suffering from moderate and severe acne vulgaris, in addition to 20 apparently healthy, age and sex matched individuals as a control group. The study was approved by the Local Ethics Committee on Research involving human subjects of Benha Faculty of Medicine. Informed consents were obtained from all participants before sample collection.

Subjects with any of the following conditions were excluded from the study:

1- Acneiform eruptions.

2- History of topical or systemic therapy for acne vulgaris two month before the study.

3- Infectious, inflammatory, autoimmune systemic or cutaneous diseases.

4- Serious systemic illness e.g liver, kidney or cardiac disease.

Using systemic drugs which may affect the inflammatory markers e.g systemic steroids.

5- Pregnancy and lactation.

6- Malignancy.

Patients under study were subjected to Full history taking including:

- Personal history including demographic data and habits of related medical importance. Present history including age of onset of acne, disease duration, course of the disease and risk factors (diet, sun exposure , smoking, stress)
- Family history of acne vulgaris: family histories were obtained from all patients.

The patients were examined carefully to detect acne lesions and to determine their types, distribution and grading.

Acne grading was performed using the global acne grading system (GAGS) with the following categories:

1- Moderate: several comedones, papules, pustules and few to several nodules.

2- Severe: numerous comedons, papules, pustules and many nodules.

The collected data was revised, coded, tabulated and introduced to a PC using Statistical package for Social Science (IBM Corp. Released 2011. IBM SPSS Statistics for Windows, Version 20.0. Armonk, NY: IBM Corp.). Data were presented and suitable analysis was done according to the type of data obtained for each parameter. Shapiro test was done to test the normality of data distribution.

Multiple comparisons between ROC curves were used to perform pairwise statistical comparisons for two ROC curves.

Regression analysis

Logistic and linear regression analyses were used for prediction of risk factors, using generalized linear models.

3. Results

There was insignificant difference between patients and controls regarding age, sex and BMI ($p=0.660$, 0.166 , 0.307 respectively) Table (1).

Table (1) Demographic Data of the studied groups.

Variables		Control N=20	Patients N=30	P
Age (years)	Range	15:28	14:25	0.660
Male	N (%)	12 (60%)	12 (40%)	0.166
Female	N (%)	8 (40%)	18 (60%)	
BMI (kg/m ²)	Range	0.002:0.005	0.002:0.003	0.307

BMI: body mass index, **P<0.05** is significant, **SD:** standard deviation.

The mean acne duration was 5.5 ± 1.2 years, the mean age of onset was 14.9 ± 2.3 years, 14 patients (46.7%) had stationary course, while 16 patients (53.3%) had remissions and exacerbations Table (2).

Table (2) History findings of the studied cases.

Variables		Patients N=30
Duration (years)	Range	1y:10ys
Age of onset (years)	Range	11ys:17ys
Course	Stationary	N (%) 14 (46.7)
	Remissions and exacerbations	N(%) 16 (53.3)

Regarding the reported risk factors for acne development, twelve patients (40%) were smokers and relation to sun exposure was reported by 22 patients (73.30%). Relation to diet was mentioned by 23

patients (76.70%), relation to stress was reported in 25 patients (83.30). Family history was positive in 19 patients (63.30) Fig (1)

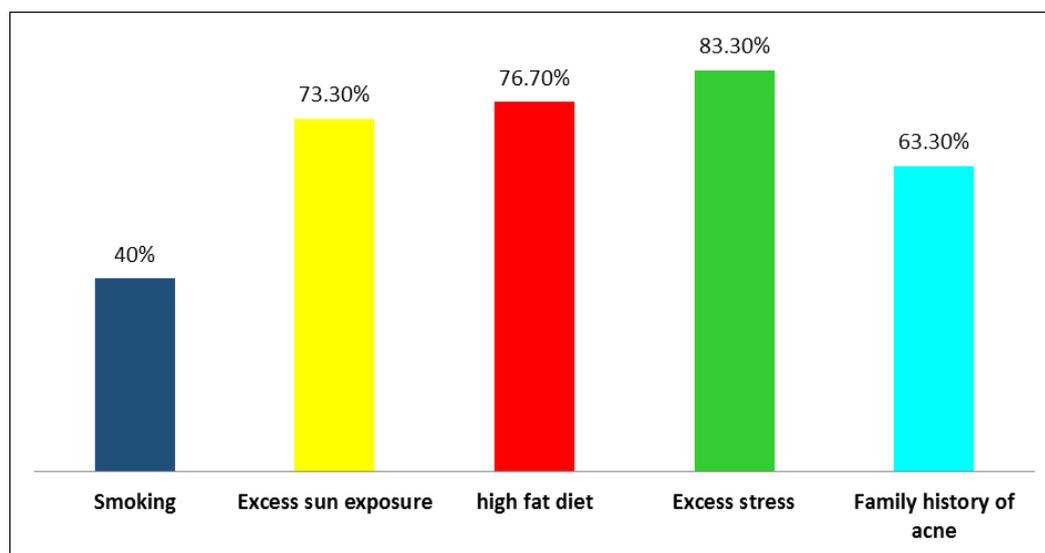


Fig (1) The reported risk factors by the studied cases.

Logistic regression analysis was conducted for prediction of acne vulgaris development, using age, gender, BMI, smoking, FH, risk factors. Positive FH,

smoking, higher frequency sun exposure, high caloric diet, excess stress were associated with risk of Acne Vulgaris occurrence in univariable analysis. Table (3).

Table (3) Regression analysis for prediction of acne vulgaris occurrence.

	Univariable				Multivariable			
	P	OR	95% CI		P	OR	95% CI	
Age	0.653	1.023	0.927	1.129				
Gender	0.166	1.653	0.811	3.366				
BMI	0.291	1.069	0.945	1.209				
Positive family history of acne	<0.001	5.035	2.100	12.075	0.945	0.958	0.288	3.185
Smoking	0.021	2.908	1.171	7.223	0.919	1.046	0.438	2.501
Excess sun exposure	0.001	3.622	1.693	7.748	0.891	1.069	0.412	2.771
high fat diet	<0.001	5.737	2.516	13.079	0.670	1.225	0.481	3.118
Excess stress	<0.001	7.314	3.126	17.112	0.724	1.228	0.394	3.826

CI: confidence interval, OR: odds ratio, P<0.05 is significant.

Logistic regression analysis was conducted for prediction of severe acne vulgaris, using age, gender, BMI, smoking, FH, risk factors, onset, course, duration. Positive family history, smoking, excess sun exposure, high caloric diet, stress, longer duration were

associated with severe acne vulgaris in univariable analysis. However, taking significant risk factors into multivariable analysis revealed that positive family history was considered as predictors for severe acne vulgaris Table (4).

Table (4) Regression analysis for prediction of severe acne vulgaris.

	Univariable				Multivariable			
	P	OR	95% CI		P	OR	95% CI	
Age	0.085	1.127	0.984	1.291				
Gender	0.137	2.040	0.797	5.221				
BMI	0.892	1.009	0.888	1.147				
Positive family history	0.001	2.162	1.152	3.841	0.046	0.189	0.037	0.968
Smoking	0.026	1.331	1.125	1.879	0.065	0.267	0.066	1.087
Excess sun exposure	0.029	1.746	1.429	2.296	0.681	0.629	0.069	5.754
High fat diet	0.034	1.754	1.422	2.348	0.955	0.963	0.255	3.633
Excess stress	0.012	1.769	1.395	2.497	0.187	1.630	0.789	3.365
Onset	0.935	1.008	0.825	1.232				
Progressive course	0.464	1.401	0.568	3.459				
Longer duration	0.034	1.166	1.012	1.343	0.445	1.483	1.103	1.937

CI: confidence interval, OR:odds ratio, , P<0.05 is significant .

4. Discussion

Acne vulgaris is an important chronic inflammatory disease of the pilosebaceous unit (comprising the hair follicle, shaft and sebaceous gland) affecting more than 80% of teenagers and young adults [7]. It frequently results in permanent disfigurement, even with appropriate treatment [8].

The aims of this study were to assess the Risk factors for acne vulgaris development in patients with acne vulgaris and to evaluate the relation between them and the clinical aspects of the disease. In the preseny study, logistic regression analysis was conducted for prediction of acne vulgaris development, using age, gender, smoking, risk factors. Positive FH, smoking, higher frequency sun exposure, high caloric diet, excess stress, were associated with risk of acne vulgaris occurrence in univariable analysis.

The comparison of the family history of acne between 220 acne and 1358 non acne twins in the UK resulted that 47% of the acne twins had a family history with acne (P<0.0001) compared to 15% in non acne twins [9].

the prevalence of moderate to severe acne was higher in patients with a positive family history than in those with no family history. Therefore, similar to Evans et al. [10], we suggest that a positive family history is associated with higher risk of developing severe acne. Compatible with our findings, Ballanger et al. [12] reported increased rates of retentional lesions and therapeutic difficulties, indicating more severe grades of acne, in patients with a positive family history. In a large twin study with 458 homozygous and 1099 heterozygous twins, 81% (95% CI: 73–87%) of the disease variance could be attributed to genetic causes and only 19% to environmental factors[13].

as the number of affected family members increased, the risk for moderate to severe acne in our patients grew. Ballanger et al. [12] found more severe acne forms in patients with acne history on the mother's side or on both parental sides than in patients with acne history on the father's side only.

Several epidemiologic studies suggest a dose-dependent correlation between smoking and acne, in both prevalence and severity (not affected by age, sex & social class), citing nicotine as a possible inciting factor behind the pathophysiologic process [12].

The study of Schafer et al. [13] showed acne to be more frequent and severe among smokers, and to follow a dose-dependent association. In contrast, in the study of Klaz et al. [14], active smokers had a significantly lower prevalence of severe acne with an inverse dose-dependent association. This conflicting discrepancy has still to be elucidated. Gender may have an influence on the relation between acne and smoking. According to Rombouts et al. [15], daily cigarette consumption and duration of smoking appeared to be significantly protective in the development of inflammatory acne in girls.

In a case-control study, Chuh et al. [16] concluded that smoking is likely to bear a positive correlation with acne for men. Schafer et al. [13] reported a dose-dependent relationship between acne severity and the quantity of cigarettes consumed daily, which was not affected by gender. In our study, severity of smoking or quantity of cigarettes consumed daily was not investigated.

The perception that diet is strongly associated with the development of acne and its exacerbation is common and geographically widely distributed, not only among acne patients [17]. In a study of Green and Sinclair [18], 41% of final year Australian medical students identified the dietary factors (chocolate, oily and fatty foods, and high sugar content foods) as exacerbating factors in acne.

Evidence showing how diet may directly or indirectly influence the pathogenesis of acne has been provided only currently [19], but the relation between nutrition and acne still needs corroboration.

The possible significance of mental stress in acne and the induction of seborrhea has been indicated by previous own experimental studies in tissue and in vitro [20].

References

- [1] A.L. Zaenglein, A.L. Pathy, B.J. Schlosser, Guidelines of care for the management of acne vulgaris. *J Am Acad Dermatol*, Vol.174(5), PP.945-973,2016.
- [2] Y.J. Bhat, I. Latief, I. Hassan, Update on etiopathogenesis and treatment of Acne. *Indian J Dermatol Venereol Leprol*, Vol.83(3), pp,298-305,2017.
- [3] S. Korkmaz, S.K. Fıçıcıoğlu, Calprotectin can play an inflammatory role in acne vulgaris. *Postepy Dermatol Alergol*, Vol. 35(4), PP. 397-399,2018.
- [4] S. Zahra Ghodsi, H. Orawa, C. C. Zouboulis, Prevalence, Severity, and Severity Risk Factors of Acne in High School Pupils: A Community-Based Study. *Journal of Investigative Dermatology*, Vol.129(9), PP.2136-2141,2009.
- [5] C.C. Zouboulis, H. Seltmann, N. Hiroi, Corticotropin releasing hormone: an autocrine hormone that promotes lipogenesis in human sebocytes. *Proc Natl Acad Sci USA*, Vol.99, PP.7148-53,2002
- [6] S.F. Witchel, A. K. Topaloglu, Puberty: gonadarche and adrenarche. *Yen and Jaffe's reproductive endocrinology. Content Repository Only*, vol 25(5), PP. 394-446,2019.
- [7] M. Alper, F.A. Khurami, Histopathologic evaluation of acneiform eruptions: practical algorithmic proposal for acne lesions. *Acne and Acneiform Eruptions. InTech*, Vol.139, PP.160,2017.
- [8] S.L. Kolar, C.M. Tsai, J. Torres, Propionibacterium acnes-induced immunopathology correlates with health and disease association. *JCI Insight*, Vol.4(5), PP. 3-7,2019.
- [9] R. Lichtenberger, M.A. Simpson, C. Smith, Genetic architecture of acne vulgaris. *J Eur Acad Dermatol Venereol*, Vol. 31(12), PP.1978-1990, 2017.
- [10] D.M. Evans, K.M. Kirk, D.R. Nyholt, Teenage acne is influenced by genetic factors. *Br J Dermatol*, Vol. 152, PP.579-81, 2005
- [11] F. Ballanger, P. Baudry, J.M. Nouguyen, Heredity: a prognostic factor for acne. *Dermatology*, Vol.212, PP.145-9
- [12] V. Bataille, H. Snieder, A.J. MacGregor, The influence of genetics and environmental factors in the pathogenesis of acne: a twin study of acne in women. *J Invest Dermatol*, Vol. 119, PP. 1317-22, 2002.
- [13] T. Schafer, A. Nienhaus, D. Vieluf, Epidemiology of acne in the general population: the risk of smoking. *Br J Dermatol*, Vol. 145, PP.100-4, 2001.
- [14] I. Klaz, I. Kochba, T. Shohat, Severe acne vulgaris and tobacco smoking in young men. *J Invest Dermatol*, Vol.126:, PP.1749-52, 2006
- [15] S. Rombouts, T. Nijsten, J. Lambert, Cigarette smoking and acne in adolescents: results from a cross-sectional study. *J Eur Acad Dermatol Venereol*, Vol. 21, PP.326-33,2007.
- [16] A.A.T. Chuh, V. Zawar, W.C.W. Wong, The association of smoking and acne in men in Hong Kong and in India: a retrospective case-control study in primary care settings. *Clin Exp Dermatol*, Vol. 29, PP.597-9,2004.
- [17] T.M. Tallab, Beliefs, perceptions and psychological impact of acne vulgaris among patients in the Assir region of Saudi Arabia. *West Afr J Med*, Vol. 23, PP.85-7, 2004.

- [18]J .Green, R.D. Sinclair , Perception of acne vulgaris in final year medical student written examination answers. Austral J Dermatol , Vol.42, PP..98–101, 2001
- [19]F.W. Danby, Acne and milk, the diet myth, and beyond. J Am Acad Dermatol , Vol.52, PP..360–2,2005
- [20]R . Ganceviciene, V. Graziene, S. Fimmel, Involvement of the corticotropin-releasing hormone system in the pathogenesis of acne vulgaris. Br J Dermatol Vol. 160, PP..345–52, 2009.