http://bjas.bu.edu.eg

Intercellular Adhesion Molecule-1 (ICAM-1) gene polymorphism in Patients with Acne Vulgaris

Suzan.E.Amr¹, Adel.A.Ebrahim¹, Amany.I.Mostafa^T and Naglaa.F.Al-Husseini²

¹Dermatology, Venereology and Andrology Dept., Faculty of Medicine, Benha University ²Medical Biochemistry, Dept. Faculty of Medicine, Benha University

²Medical Biochemistry Dept., Faculty of Medicine, Benha University

Abstract

Background: ICAM-Intercellular adhesion molecule-1 (ICAM-1) is a gene that encodes a glycoprotein found on the surface of cells that helps them stick together and respond to immunological stimuli. Several research have looked at whether variations in the ICAM-1 gene have a role in acne vulgaris susceptibility (AV). The purpose of this research was to examine ICAM-1 gene polymorphism in acne vulgaris patients. Methods: Fifty people with AV and 30 people who seemed to be healthy served as controls in this prospective case-control study. Genotyping of ICAM-1 gene polymorphism using PCR necessitated a clinical evaluation and blood sample from all individuals. Results: In regards to the ICAM-1 gene's genotypes and alleles: Everyone, including patients and controls, were genotyped. Two percent of AV patients had the AA genotype, thirty percent had the AG genotype, and fifty-six percent had the GG genotype. Forty percent of healthy controls had the AA genotype, 46.7% the AG genotype, and 13.3% the GG genotype. Acne vulgaris was strongly associated with the GG genotype. In conclusion, ICAM-1 gene polymorphism may increase AV patients' vulnerability.

Acne vulgaris, intercellular adhesion molecule 1, and gene variants are some related terms.

1. Introduction

Acne About 80% of people between the ages of 11 and 30 suffer with acne vulgaris, a chronic inflammatory illness of the pilosebaceous unit. Seborrhea, comedones, papules, pustules, nodules, and cysts, and scarring on the face, neck, chest, and back are all symptoms of this condition [1].

ICAM-1, also known as CD54, is a transmembrane glycoprotein that functions as an adhesion molecule. Glycoprotein with molecular mass ranging from 76 to 110 kilodaltons (kDa) and localised to chromosome 19p13 [2-4].

A increasing body of research has investigated the link between ICAM-1 gene polymorphisms and susceptibility to numerous malignancies, including prostate, colorectal, and breast cancers [5]. Many studies have identified connections between ICAM-1 polymorphisms and chronic inflammatory illnesses.

The goals of the present research were to evaluate the frequency of ICAM-1 gene polymorphism in patients with acne vulgaris, evaluate its clinical importance, and determine its function in acne susceptibility.

2.Methods

The 50 participants in this prospective case control study all had acne vulgaris (Group A). In addition, a control group of 30 people of similar age and sex was selected (Group B). All participants were drawn from the dermatology, venereology, and andrology department's outpatient clinic at Benha University Hospitals. Benha University's Medical School's Human Research Ethics Committee gave its stamp of approval to the project. All individuals who took part voluntarily provided their informed permission. The Global Acne Grading System (GAGS) [6] was used to determine the severity of acne vulgaris to determine eligibility.

Diseases including Crohn's and ulcerative colitis, as well as active hepatitis and a family history of autoimmune disorders like rheumatoid arthritis, were ruled out.

Each patient had a thorough medical history taken, which included the following: Background information (name, age, sex, occupation, residence, habits of medical importance, marital status). Current history (onset, course, and duration of AV; AV in the family). Medication and treatment histories, Diagnostic Procedures: Full medical workup to rule out underlying conditions including thyroid illness; dermoscopy to determine acne's clinical subtype.

Acne was diagnosed by using the Global Acne Grading System (GAGS)[6], which categorised patients into those with mild, moderate, and severe acne vulgaris.

Research studies: ICAM-1 gene polymorphism genotyping was performed on all study participants. Each individual had three millilitres of venous blood drawn using a sterile syringe and deposited in a vaccutainer tube with ethylene diamine tetra-acetic acid (EDTA) (1.2 mg/mL) as an anticoagulant. Aliquots of blood were frozen at -20 degrees Celsius for later ICAM-1 gene polymorphism investigation.

Polymorphism in the ICAM-1 gene identified by polymerase chain reaction

Number crunching

SPSS version 28 was used for data administration and statistical analysis (IBM, Armonk, New York, United States). There were no one-sided statistical analyses. P values below 0.05 were used to indicate statistical significance.

3. Results

This study included 50 patients suffering from acne vulgaris (Group A) and 30 healthy

individuals of matched age and sex as a control group (**Group B**).

Socio-Demographic characteristics were insignificantly different between both studied groups.

Clinical characteristics of the studied patients were shown in **Table 1**

Table(1) Clinical characteristics of the studied patients

Clinical characteristics				
Oreset	Gradual	28 (56)		
Onset	Sudden	22 (44)		
Cauraa	Stationary	23 (46)		
Course	Progressive	27 (54)		
Duration (24 (1 -84)			
	Face	50 (100)		
Data are presented as number (perce	9 (18)			
	Back	4 (8)		
	Mild	23 (46)		
	Moderate	20 (40)		
Grade	Severe	7 (14)		
Sca	5 (10)			
Relation	18 (36)			
Relation	22 (44)			
Relation	27 (54)			
Positive Previo	43 (86)			
History of syst	1 (2)			
Family l	15 (30)			

 Table (2) Multivariate logistic regression for genotype and allele of I-CAM gene in predicting acne vulgaris

<u>8</u>	$\frac{1}{2}$ Group A (n = $\frac{1}{2}$ roup B* (n =					
		50)	30)	$OR (95\% CI)^{\dagger}$	P-value	
Genotype		7(14)	12 (40)	D		
	AA	/ (14)	12 (40)	ĸ	-	
	GG	28 (56)	4 (13.3)	11.128 (2.657 – 46.597)	< 0.001**	
	G	28 (56.0)	4 (13.3)	8.273 (2.513 – 27.237)	< 0.001**	
Data	were present	ed as number wild and mutant genotypes regarding onset (P				

Data were presented as number (percentage); * Controls follow Hardy-Weinberg equilibrium (P = 0.979); [†] Adjusted for age, gender, and smoking; ** significant; R: Reference; OR: Odds ratio; 95% CI: 95% confidence interval.

Regarding clinical characteristics according to genotypes: No significant differences were observed between those with = 0.117), course (P = 0.689), duration (P = 0.935), face affection, chest affection (P = 0.783), back affection (P= 1.0), grade (P = 0.64), scars (P = 1.0), relation to sun (P = 0.083), relation to diet (P = 0.444), relation to stress (P = 0.43), history of treatment (P = 1.0), history of systemic disease (P = 1.0), and family history (P = 0.415). **Table 3**

		Genotype			
		Wild $(n = 7)$	Mutant $(n = 43)$	P-value	
Onset	Gradual	6 (85.7)	22 (51.2)	0.117	
	Sudden	1 (14.3)	21 (48.8)		
Course	Stationary	4 (57.1)	19 (44.2)	0.689	
	Progressive	3 (42.9)	24 (55.8)		
Duration (months)		24 (2 - 60)	24 (1 - 84)	0.935	
Site affected	Face	7 (100)	43 (100)	-	
	Chest	1 (14.3)	8 (18.6)	0.783	
	Back	0 (0)	4 (9.3)	1.0	
Grade	Mild	3 (42.9)	20 (46.5)		
	Moderate	4 (57.1)	16 (37.2)	0.64	
	Severe	0 (0)	7 (16.3)		
Scars		0 (0)	5 (11.6)	1.0	
Relation to sun		5 (71.4)	13 (30.2)	0.083	
Relation to diet		2 (28.6)	20 (46.5)	0.444	
Relation to stress		5 (71.4)	22 (51.2)	0.43	
History of treatment		6 (85.7)	37 (86)	1.0	
History of systemic disease		0 (0)	1 (2.3)	1.0	
Family history		3 (42.9)	12 (27.9)	0.415	

Table (3) Clinical characteristics according to genotypes

Data are presented with number (percentage) or median (min-max).

4. Discussion

In this work, we made sure that our patients and controls were similar in age and gender. The average age of those receiving treatment was 19.3 against 21.4 in the control group. These results are consistent with the observation that AV is a lifelong condition, since both boys and girls experience hormonal changes throughout puberty and adulthood. In instance, elevated levels of androgens in both sexes stimulate the skin's sebaceous glands to swell and secrete more sebum. Acne lesions form when the follicles get blocked due to an increase in sebum production [7].

Thirty out of fifty patients (60%) are female, whereas twenty out of fifty (40%) are male. Fifteen of the thirty people in the control group are female (50 percent), and fifteen are male (50 percent). El-Hamd et al. [8] reported almost the same thing, with 60% of AV patients being female and 40% being male. The average age of AV patients was reported to be 20.1-2.10 years [9]; 68 percent were female and 32 percent were male.

The majority of cases in this research (56%) have a gradual beginning, whereas 44% have a fast onset, and 54% have a progressive course, while 64% have a stagnant course. Disease severity was classified as mild in 46% of patients, moderate in 40% of cases, and severe in 14%

of cases in the present research. Abdel-Hafez et al. [9] also found that 35% of patients are considered mild, 39% are considered intermediate, and 26% are considered severe with this condition. All of the instances in this research included the face, and 10% of them had a scar. This is consistent with the findings of Nast et al. [10], who identified facial affection in 99 percent of cases, and Kiprono and Wamburu [11], who did so in 93.2% of instances.

Three-sixths of AV cases were shown to have a positive association with sun exposure, 44% had a positive association with food, 54% had a positive association with psychological stress, and 8% of AV patients were smokers. Almost identical findings were made by other researchers. In 57.5% of instances, Tan et al. [12] found a positive correlation with psychological strain. Fourtyfive percent of the cases had a positive correlation with sun exposure [13]. In 49.5 percent of instances, a favourable relationship was seen between diet and Kiprono and wamburu [11].

The current investigation confirmed the findings of a previous study by Barnes et al. [14] that demonstrated an increased risk of acne among twins and first-degree relatives, suggesting that a hereditary component may explain the tendency to acne for some people. Various clinical indicators are compared with the frequency of ICAM-1 genotypes in our investigation. There was no correlation between ICAM-1 genotype frequency and patient age, gender, or smoking status. The same lack of significance was seen for the disease's onset, severity, and persistence.

The following clinical factors were analysed for their correlation with the frequency of ICAM-1 genotypes and were found to have no statistical significance.

5. Conclusions

From Based on the findings of the current study, it was determined that the frequency of the ICAM-1 GG genotype and the G allele was considerably greater in AV patients. Therefore, ICAM-1 gene polymorphism may increase AV patients' vulnerability.

References

.

- [1] Leung AK, Barankin B, Lam JM, Leong KF, Hon KL. Dermatology: how to manage acne vulgaris. Drugs Context. 2021;10.
- [2] Mohammadzadeh Z, Omidkhoda A, Chahardouli B, Hoseinzadeh G, Moghaddam KA, Mousavi SA, et al. The impact of ICAM-1, CCL2 and TGM2 gene polymorphisms on differentiation syndrome in acute promyelocytic leukemia. BMC Cancer. 2021;21:46.
- [3] Bui TM, Wiesolek HL, Sumagin R. ICAM-1: A master regulator of cellular responses in inflammation, injury resolution, and tumorigenesis. J Leukoc Biol. 2020;108:787-99.
- [4] Nogales A, M LD. Host Single Nucleotide Polymorphisms Modulating Influenza A Virus Disease in Humans. Pathogens. 2019;8.
- [5] Liu A, Wan A, Feng A, Rui R, Zhou B. ICAM-1 gene rs5498 polymorphism decreases the risk of coronary artery disease. Medicine (Baltimore). 2018;97:e12523.

- [6] Alsulaimani H, Kokandi A, Khawandanh S, Hamad R. Severity of Acne Vulgaris: Comparison of Two Assessment Methods. Clin Cosmet Investig Dermatol. 2020;13:711-6.
- [7] Kong YL, Tey HL. Treatment of acne vulgaris during pregnancy and lactation. Drugs. 2013;73:779-87.
- [8] El-Hamd MA, Nada EEA, Moustafa MA, Mahboob-Allah RA. Prevalence of acne vulgaris and its impact of the quality of life among secondary school-aged adolescents in Sohag Province, Upper Egypt. J Cosmet Dermatol. 2017;16:370-3.
- [9] Abdel-Hafez K, Mahran AM, Hofny ER, Mohammed KA, Darweesh AM, Aal AA. The impact of acne vulgaris on the quality of life and psychologic status in patients from upper Egypt. Int J Dermatol. 2009;48:280-5.
- [10] Nast A, Dréno B, Bettoli V, Degitz K, Erdmann R, Finlay AY, et al. European evidence- based (S3) guidelines for the treatment of acne. J Eur Acad Dermatol Venereol. 2012;26 Suppl 1:1-29.
- [11] Kiprono SK, Wamburu G. Acne vulgaris in general population of rural western Kenya: cross-sectional community survey. Int J Dermatol. 2016;55:e212-4.
- [12] Tan HH, Tan AW, Barkham T, Yan XY, Zhu M. Community-based study of acne vulgaris in adolescents in Singapore. Br J Dermatol. 2007;157:547-51.
- [13] Gupta A, Sharma YK, Dash KN, Chaudhari ND, Jethani S. Quality of life in acne vulgaris: Relationship to clinical severity and demographic data. Indian J Dermatol Venereol Leprol. 2016;82:292-7.
- [14] Barnes LE, Levender MM, Fleischer AB, Jr., Feldman SR. Quality of life measures for acne patients. Dermatol Clin. 2012;30:293-300, ix.