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ORIGINAL ARTICLE

Serum level of serum amyloid A1 protein in patients with acne vulgaris

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Abstract

Introduction: The serum amyloid A1 (SAA1) protein is involved in many pathological diseases. The increased secretion of SAA1 can maintain inflammatory conditions. Acne vulgaris (AV) is a chronic inflammatory disease of pilosebaceous glands and may be associated with systemic manifestations.

Aim of the work: This study aimed to evaluate the serum level of SAA1 in patients with AV.

Subjects and Methods: 120 participants were included in this study: 60 patients with AV and 60 apparently healthy volunteers as a control group. These participants were subjected to dermatological examination and assessments of lipid profile, fasting blood glucose (FBG), and serum SAA1.

Results: Both serum SAA1 and FBG are significantly elevated in patients with AV than control ($p < 0.0001$ and $p < 0.001$, respectively). Furthermore, there are positive correlations between AV severity and SAA1 and FBG ($p < 0.001$ and $p < 0.0001$, respectively).

Conclusions: Serum amyloid A1 is increased in AV, and this elevation may play a role in the inflammatory milieu of AV.

KEYWORDS
acne vulgaris, body mass index, fasting blood glucose, serum amyloid A1

1 | INTRODUCTION

Acne vulgaris (AV) is a common inflammatory disease affecting pilosebaceous glands.[1] Sebocytes responsible for sebum production of the sebaceous glands are resembling adipocytes in secretion and affection by different inflammatory mediators.[2] Several adipokines had been associated with AV pathogenesis.[3] Furthermore, this association may participate in the inflammatory milieu of AV.[4]

The serum amyloid proteins are inflammatory-related molecules.[5] They are markers for acute and chronic inflammatory status.[6] Serum amyloid A1 (SAA1) is linked to insulin resistance, weight gain, and pro-inflammatory cytokine expression.[7]

2 | AIM OF THE WORK

To evaluate the serum level of serum amyloid A1 in AV.

3 | SUBJECTS AND METHODS

3.1 | Ethical consideration

This study was done after approval of the IRC at Faculty of Medicine, Benha University, and according to the Helsinki Declaration. The included subjects were not subjected to any harmful procedures and had given informed consent before participation in this study.

TABLE 1 Demographic data of the participants

	Patients (60)	Control (60)	Test	*p*-value
Age (year)	20 ± 3	19 ± 4	$t = 1.549$	0.086
Sex (male/female)	28/32	31/29	$\chi^2 = 0.3$	0.583
BMI (kg/m^2)	26.48 ± 5.26	25.63 ± 4.1	$t = 0.987$	0.326
Family history of AV (yes/no)	37/23	14/46	$\chi^2 = 18.039$	<0.0001
Duration of AV (year)	3 ± 2			
GAGS score	25 ± 5			
Presence of acne scar	40 (66.66%)			

Note: $p \leq 0.05$ is significant.

Abbreviations: AV, acne vulgaris; BMI, body mass index; GAGS, Global Acne Grading System.

TABLE 2 Laboratory data of the participants

	Patients (60)	Control (60)	Test (*t* test)	*p*-value
Cholesterol (mg\dl)	152 ± 33	147 ± 25	0.935	0.351
Triglycerides (mg\dl)	91 ± 20	89 ± 12	0.664	0.508
HDL (mg\dl)	37 ± 7	38 ± 6	0.84	0.403
LDL (mg\dl)	95.5 ± 25.5	90.5 ± 20.7	1.179	0.241
FSG (mg\dl)	105 ± 21	91 ± 14	4.297	< 0.001
SAA1(ng/ml)	5.33 ± 2.41	1.73 ± 1.47	9.878	< 0.0001

Note: $p \leq 0.05$ is significant.

Abbreviations: FSG, fasting serum glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SAA1, serum amyloid A1.

3.2 | Participant selection

This study was done from March 2020 to March-February 2021. Overall, 120 subjects were recruited in this study: 60 were patients complaining of AV from both sexes attending Dermatology Outpatients Clinic of Benha University Hospitals, and the other 60 were both age- and sex-matched apparently healthy volunteers as a control group.

3.3 | Exclusion criteria

Any participant with other dermatological diseases, autoimmune disorder, endocrinal disorder, menstrual cycle irregularity, psychiatric disease, hepatic, renal, past history of chemotherapy, or receiving immunosuppressive medication was excluded from this study.

3.4 | Clinical assessment

A complete history was taken from each patient and included the onset, course, family history, and previous therapy. Each participant was subjected to a generalized dermatological examination with an assessment of body mass index (BMI). The Global Acne Grading System (GAGS) was the assessment tool used for AV scoring in this study.[8]

3.5 | Laboratory assessment

A fasting 8 ml venous blood sample was withdrawn from each participant at 10 am. This venous sample was left to clot for 45 min at 25°C, then centrifuged for 20 min at 1000 × g. Serum was aspirated and separated into two parts. The first part was used to assess serum cholesterol, triglycerides, high-density lipoprotein, and fasting serum glucose, while low-density lipoprotein was calculated using Frigewal's formula (LDL-C = TC [mg/dl]–TG/5 [mg/dl] + HDL-C [mg/dl]). The second serum sample was stored at −80°C till the time of assay. SAA1 was assessed using an enzyme-linked immunosorbent assay kit provided by R&D Systems, Minneapolis, MN\USA [Product No. DY3019-05] with a detection range from 1.6–100 ng/ml.

3.6 | Statistical analysis

All data were collected and statistically analyzed using the Statistical Package for the Social Sciences (SPSS®) version 26 program (IBM®) for Windows 10 64 bit®.

Means and standard deviation (SD) were used to represent numerical data, and numbers and percentages were used to express categorical data. For numerical data, the independent *t* test for normally distributed variables was used to compare the two groups. The chi-square test (χ^2) was used to evaluate differences between categorical variables. The Spearman correlation was used to perform the correlation analysis. The statistically significant cut-point was chosen at *p*-value ≤0.05.

4 | RESULTS

The demographic and clinical data of the participants are summarized in Table 1. The results of this study showed that a family history of AV is common in patients with AV than in the control group ($p < 0.0001$; Table 1). The laboratory results revealed non-significant differences regarding lipid profile between patients with AV and control group. However, patients with AV had significant elevations of fasting serum glucose ($p < 0.001$) and SAA1 ($p < 0.001$) than controls (Table 2).

In this study, 35 patients were suffering from AV scar (with a mean serum level of SAA1 of 7.03 ± 4.86 ng/ml), while 25 of the patients had no AV scar (with a mean serum level of SAA1 of 4.41 2.58 ng/ml). There was a significant elevation of SAA1 in patients with AV scars than in those without AV scars ($p = 0.014$; Figure 1).

The results of this study showed significant positive correlations between AV severity and BMI ($r = 0.520$, $p < 0.001$), and FBG ($r = 0.87$, $p < 0.0001$), and mean serum level of SAA1 level ($r = 0.696$, $p < 0.001$; Table 3).

5 | DISCUSSION

The acute-phase response markers reflect the changes occurring secondary to inflammation or infection. Among these markers, the expression of the amyloid A family is prominent.[9] The expression of amyloid A proteins is induced by several inflammatory cytokines and lipopolysaccharide,[6] and even systemic glucocorticoids.[10] SAA1 is produced mainly by hepatocytes, but other cells can also secrete SAA1 such as adipocytes.[11] SAA1 is increased in different disorders linked with AV such as polycystic ovary,[12] insulin resistance,[13] and hyperandrogenism.[14]

The results of this study show significant elevation of SAA1 in patients with AV, with a positive correlation with the severity of AV. The exact role of SAA1 in AV has not previously been investigated. Serum amyloid A1 may have a role in Av pathogenesis through different mechanisms. SAA1 is known for its lipophilicity, with a strong effect on sebocyte activation.[15] In addition, SAA1 can maintain tissue inflammation and can induce remolding through metalloproteinase release.[16] *Cutibacterium* acnes (previously known as *Propionibacterium* acnes) can also increase the release of SAA1.[17] The effect of SAA1 is also mediated through activation of Toll-like receptors, especially Toll-like receptor 2.[18,19] SAA1 can increase the expression of peroxisome proliferator-activated receptor γ and subsequent induction of inflammation.[20] Furthermore, SAA1 is a strong initiator Akt/mTOR inflammatory cascade.[21] All these factors are involved in the pathophysiological process of AV.[22]

The results of this study showed a significant elevation in serum level of SAA1 in patients with AV scar than in those without a scar.

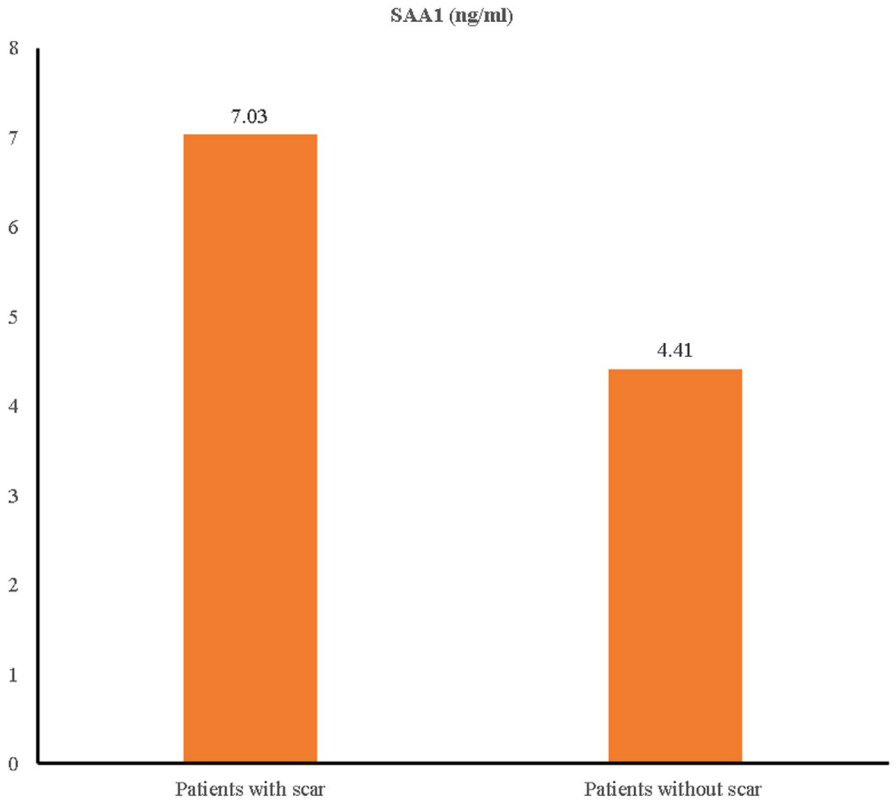

FIGURE 1 Comparison of serum level of SAA1 between patients with AV scars and those without scars

TABLE 3 Correlation between AV severity and different parameters

	Age	BMI	Cholesterol	Triglyceride	HDL	LDL	FSG	SAA1-13
r	−.220	0.520	−.130	−.073	−.118	−.105	0.87	0.696
p	0.091	<0.001	0.321	0.578	0.37	0.423	<0.0001	<0.001

Note: $p \leq 0.05$ is significant.

Abbreviations: BMI, body mass index; FSG, fasting serum glucose; HDL, high-density lipoprotein; LDL, low-density lipoprotein; SAA1, serum amyloid A1.

SAA1 has a direct effect on dermal fibroblast,[23] and its serum level is elevated in diseases characterized by fibrosis.[6]

There are positive correlations between AV severity and both BMI and FBG found in this study. The association between AV and obesity is not fully well established. A recent meta-analysis highlights this association,[24] and another recent meta-analysis did not show this association.[25] On the contrary, the role of hyperglycemia is documented in the pathogenesis of AV.[26,27]

6 | CONCLUSION

Serum amyloid A1 is increased in AV, and this elevation may play a role in the inflammatory milieu of AV.

7 | LIMITATIONS

This study did not examine the effect of therapy on the serum level of SAA1 in patients with AV.

CONFLICT OF INTEREST

The authors report no conflict of interest.

ETHICAL APPROVAL

This study was done after approval of the Ethical Committee of Research on Human at Faculty of Medicine, BenhaUniversity.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are available on request from the corresponding author. The data are not publicly available due to privacy or ethical restrictions.

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