Original Article

Paraoxonase Enzyme Activity and Determination of Phenotypic Polymorphism in Patients with Acne Vulgaris

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Abstract

Aim: This study aims to compare serum paraoxonase 1 (PON1) activity between patients diagnosed with acne vulgaris (AV) and a healthy control group, and to determine the PON1 phenotype.

Materials and Methods: The study sample consists of 50 patients with moderate to severe AV who presented to the Clinic of Dermatology at Balıkesir University Faculty of Medicine. Additionally, healthy volunteers (n = 52) with similar age and gender characteristics to the patient group were included as the control group. The diagnosis of AV was made using the Global Acne Grading System. The serum PON1 activities in both AV patients and healthy volunteers were measured by spectrophotometric methods, and statistical comparisons were made among the groups. Furthermore, the PON1 phenotypic polymorphism was determined.

Results: In our study, serum PON1 activity levels were found to be significantly lower in AV patients (36,149±14,536) compared to the control group (48,173±18,753) ($P = 0.001$). The distribution of PON1 phenotypes demonstrated a trimodal pattern. In the patient group, the QQ phenotype was observed in 48%, odds ratio in 24%, and risk ratio in 28%. In the control group, these rates were 5.8%, 44.2%, and 50%, respectively.

Conclusion: The notably reduced activity of the antioxidant enzyme PON1 in patients with AV, as compared to the control group, indicates that oxidative stress could be a significant factor in its etiopathogenesis.

Keywords: Acne vulgaris, paraoxonase, paraoxonase phenotypic polymorphism

Introduction

Acne vulgaris (AV) is a condition characterized by prolonged inflammation of the pilosebaceous follicles, predominantly found on the face, back, and torso. AV is a common condition, affecting approximately 80% of adolescents and young adults.[1] Although the exact etiopathogenesis of acne remains unclear, four main factors are widely recognized: sebaceous gland hyperplasia with elevated sebum production, hyperkeratinization of the pilosebaceous ducts, abnormal colonization primarily by *Cutibacterium acnes* (*C. acnes*), and inflammation.[2] Many researchers have examined the role of oxidative stress in the development of AV, investigating the connections between inflammation, oxidative stress, and acne pathogenesis. Although the roles of oxidative and antioxidative system parameters in acne pathogenesis are not definitively established, these parameters have been widely examined in various studies.[3-5]

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The link between acne and oxidative stress is often attributed to *C. acnes* colonization. *C. acnes* is thought to initiate inflammation by producing low-molecular-weight chemotactic factors and triggering neutrophil chemotaxis. Some researchers have suggested that AV is a disease related to oxidative stress, primarily driven by increased sebum production.[6,7]

Paraoxonase 1 (PON1), a calcium-dependent ester hydrolase, has recently gained attention for its antioxidant properties. Serum PON1 is found in plasma associated with high-density lipoproteins (HDL), playing a critical role in preventing the oxidation of plasma lipoproteins. PON1 enzyme activity can vary significantly due to polymorphism, and many clinical studies have explored the relationship between PON1 enzyme activity and various diseases.[8-11]

This study aims to investigate the role of oxidative stress in AV pathogenesis by assessing PON1 enzyme activity and identifying the phenotypic polymorphism of this enzyme in acne patients.

Materials and Methods

Study Groups

Our study included 50 patients diagnosed with moderate to severe AV according to the Global Acne Grading System, who presented to the Clinic of Dermatology at Balıkesir University Faculty of Medicine between November 18, 2014, and May 18, 2015. These patients, aged between 18 and 40, provided informed consent. A control group of 52 healthy individuals with similar age and gender characteristics, presenting to the dermatology clinic for non-acne-related issues, was also included. Exclusion criteria included individuals who had received systemic or topical acne treatment in the past three months, had systemic diseases that could influence PON1 activity, were on systemic medications, had smoking, alcohol, or substance dependence, engaged in regular exercise beyond daily activities, and pregnant women. The study received ethical approval from the Ethics Committee of Balıkesir University Faculty of Medicine (approval number: 2014/94, date: 17.11.2014) for the study titled "Measurement of Paraoxonase Enzyme Activity and Determination of P/Q192 Polymorphism in AV Patients." This research was also funded by the Balıkesir University Scientific Research Projects Unit under project number 2015/135.

Blood Samples

Blood samples collected from the patient and control groups were left to sit for 30 minutes, then centrifuged at 4000 rpm for 10 minutes. The separated sera were stored at -20 °C.

Enzyme Activity Measurement

PON enzyme activity was measured using a spectrophotometric method. For the activity measurement, 0.05 mL of serum sample (enzyme source) was added to a previously prepared 1 mL buffer solution (100 mM Tris-base, pH 8.0) along with substrate (2 mM paraoxon) and coenzyme (2 mM $CaCl_2$). The absorbance change at a wavelength of 412 nm was recorded over one minute at 37 °C. This method determined the rate of enzymatic conversion of paraoxon to p-Nitrophenol. The procedure was repeated with a control sample lacking the enzyme, and the difference between the two readings was calculated as enzyme activity. One unit of PON activity was defined as the amount of p-Nitrophenol formed per minute, in nanomoles.

Determination of Q and R-Types

For the activity measurement, 0.05 mL of serum sample was rapidly added to a 1 mL pre-prepared buffer (100 mM Tris-base, pH 10.5) and substrate (1 mM paraoxon) solution. The baseline activity value at 412 nm was recorded over one minute at 37 °C. To measure salt-stimulated activity, 1 M NaCl was added as a coenzyme to the same solutions, and the salt-stimulated activity value was recorded. This method assessed the enzymatic conversion rate of paraoxon to p-Nitrophenol. The procedure was repeated with a control sample lacking the enzyme, and the difference between the two values was calculated as enzyme activity. PON activity was defined as the amount of p-Nitrophenol produced per minute, measured in micromoles (µmol). After measuring the basal and salt-stimulated activities of PON, the following formula was used for phenotype determination (Figure 1).[12]

Hydrolysis of Paraoxon

When high-activity PON enzyme is stimulated by 1 M NaCl, the low-activity form is inhibited, resulting in a trimodal distribution in this analysis. Values up to 60% represent homozygous Q (A), values between 60% and 200% correspond to QR (AB), and values above 200% indicate homozygous R (B) individuals.

$$\frac{\text{Paraoxonase activity in the presence of 1 M NaCl} - \text{Basal paraoxonase activity}}{\text{Basal paraoxonase activity}} \times 100$$

Figure 1. Percentage of Paraoxonase Activity Induced by NaCl

Biochemical Parameters

Serum levels of PON1, total cholesterol (TC), HDL, low-density lipoprotein (LDL), very low-density lipoprotein (VLDL), and triglycerides (TG) were analyzed in both the patient and control groups, and their intergroup differences were compared. Additionally, the correlation between PON1 activity and TG, HDL, LDL, and TC levels was examined within each group.

Statistical analysis

Statistical analysis of the study findings was performed using the SPSS 21.0 software package. Pearson's chi-square test was applied to compare categorical parameters. A t-test was used for comparing parameters between two groups. For comparisons involving more than two groups, the One-Way ANOVA test was used, followed by the Bonferroni test to identify which groups contributed to the observed differences. Pearson correlation analysis was conducted to examine relationships between parameters. The results were analyzed at a 95% confidence level, with statistical significance set at $P < 0.05$.

Results

The study included 50 patients aged 18-33, with an average age of 21.7±2.96 years. The control group consisted of 52 individuals aged 18-27, with an average age of 22.5±2.53 years. Among the patients, 29 (58.0%) were female and 21 (42.0%) were male, while in the control group, 32 (61.5%) were female and 20 (38.5%) were male. There were no significant differences in age or gender between the groups ($P = 0.128$, $P = 0.435$).

Levels of serum PON1, TC, HDL, LDL, VLDL, and TG were assessed in both the patient and control groups, and the differences between the groups were analyzed. No statistically significant differences were found between the two groups for TC, TG, HDL, or LDL levels ($P > 0.05$). However, PON1 activity was significantly lower in the patient group (36,149±14,536) compared to the control group (48,173±18,753) ($P = 0.001$; $P < 0.05$). Statistical comparison of PON1 activity indicated that it was markedly lower in AV patients compared to the control group (Table 1).

When the correlation between PON1 activity and TG, HDL, LDL, and TC levels was analyzed in both the patient and control groups, no significant relationship was found between PON1 activity and these parameters ($P > 0.05$) (Table 2).

Phenotypic Distribution

After measuring basal and salt-stimulated PON1 activities in both the patient and control groups, phenotypic distributions were determined as described in the materials and methods section. The phenotypic polymorphism of PON1 showed a trimodal distribution: QQ, QR, and RR. The effects of polymorphism on PON1 activity, as well as TG, TC, HDL, and LDL levels, were evaluated. A significant difference in phenotype distribution was observed between the groups ($X^2 = 23,360$; $P = 0.001$; $P < 0.05$). In the patient group, 24 individuals (48.0%) had the QQ phenotype, 12 (24.0%) had the QR phenotype, and 14 (28.0%) had the RR phenotype. In the control group, 3 individuals (5.8%) had the QQ phenotype, 23 (44.2%) had the QR phenotype, and 26 (50.0%) had the RR phenotype (Table 3).

The effects of QQ, QR, and RR phenotypes on PON1 activity, as well as TG, TC, HDL, and LDL levels, were evaluated in both the patient and control groups. In the patient group, the QQ, QR, and RR phenotypes had a statistically significant effect on PON1 activity ($P = 0.003$; $P < 0.05$). PON1 activity levels in individuals with the RR phenotype (46,556±9,400) were higher than those with the QQ phenotype (30,342±15,650).

Similarly, in the control group, the impact of QQ, QR, and RR phenotypes on PON activity was significant ($P = 0.003$, $P < 0.05$). Individuals with the RR phenotype in the control group had higher PON activity levels (55,560±21,103) compared to those with the QR phenotype (41,808±12,887).

Discussion

Recent research suggests that inflammation and immune responses may be among the most crucial factors in acne pathogenesis. However, there is still no consensus on what triggers this inflammatory process.[13,14]

Table 1. Comparison of TC, TG, HDL, LDL, and PON1 activity between groups

	Patient (n = 50)	Control (n = 52)	t	*P*
TC (mg/dL)	159,740±28,722	165,100±28,383	-0.947	0.346
TG (mg/dL)	69,820±22,370	78,020±36,872	-1,351	0.176
HDL (mg/dL)	55,840±12,151	55,289±11,053	0.240	0.811
LDL (mg/dL)	89,340±23,407	93,800±23,515	-0.960	0.340
PON1 activity	36,149±14,536	48,173±18,753	-3,609	**0.001**

TC: Total cholesterol, TG: Triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, PON1: Paraoxonase 1

Table 2. Effect of PON1 activity on TC, TG, HDL, and LDL levels in patients and control group

	Patient		Control	
	r	p	r	*P*
TC	-0.035	0.810	0.082	0.565
TG	-0.221	0.123	0.139	0.326
HDL	0.107	0.459	0.049	0.731
LDL	-0.046	0.749	0.057	0.690

PON1: Paraoxonase 1, TC: Total cholesterol, TG: Triglycerides, HDL: High-density lipoprotein, LDL: Low-density lipoprotein

Table 3. Phenotypic distribution among different groups

		Patient		Control		
		n	%	n	%	*P*
Phenotype	QQ	24	48.0%	3	5.8%	X^2=23,360 **$P = 0.001$**
	QR	12	24.0%	23	44.2%	
	RR	14	28.0%	26	50.0%	

Studies highlighting oxidative stress in acne pathogenesis have examined the relationship between inflammation, oxidative stress, and acne, with growing evidence suggesting that oxidative stress may play a significant role in the development of AV.[4,6,15]

The association between acne and oxidative stress is often linked to the colonization of *C. acnes* bacteria on the skin. *C. acnes* produces several low-molecular-weight chemotactic factors that initiate inflammation and attract neutrophils to the area. In addition, it secretes various enzymes, including protease, lecithinase, hyaluronidase, and neuraminidase.[16]

These enzymes, produced by *C. acnes* and neutrophils, can damage the follicle wall. As a result of this damage, follicular contents leak into the dermis, creating a foreign body reaction that intensifies inflammation. Activated neutrophils release proteolytic enzymes and reactive oxygen species (ROS), further exacerbating tissue damage and deepening the inflammatory response. This tissue damage is often described as autoxidative injury.[16]

Some researchers also propose that AV is a disease driven by oxidative stress, with increased sebum production playing a key role. The rise in sebum production, along with changes in its composition, contributes to sustained inflammation through irritation and damage to the follicular wall by enzymes and ROS released from neutrophils.[3,4,6,15]

Many studies investigating oxidative stress in AV pathogenesis have identified significant differences in oxidative stress parameters between acne patients and healthy controls.[3]

PON1, a calcium-dependent ester hydrolase, is one of the enzymes associated with HDL and is the first among these enzymes with well-studied structural characteristics. One of PON1's primary functions is hydrolyze organophosphate neurotoxins, aromatic carboxylic acid esters, and insecticides, which is considered its initial physiological role.[17]

In recent years, PON1 activity has been evaluated as a marker of oxidative stress in several dermatological diseases which involve oxidative stress in their etiology, including alopecia areata, recurrent aphthous stomatitis, rosacea, androgenetic alopecia (AGA), and psoriasis.[10,18-21]

In 2015, Takci et al.[21] compared serum PON1 activity and lipid hydroperoxide levels in 39 patients with rosacea and 40 healthy controls. Their study found that serum PON1 activity was lower, while serum lipid hydroperoxide levels were significantly higher in rosacea patients compared to controls. Based on these findings, the authors suggested that reduced PON1 levels may indicate a role of oxidative stress in the etiology of rosacea.[21]

In a study conducted by Bilgili et al.[22] with 39 alopecia areata patients and 39 healthy controls, serum PON1 activity was reported to be lower in the patient group. The same research team also examined serum PON1 levels in 31 patients with recurrent aphthous stomatitis and 31 healthy controls, finding that PON1 activity was significantly reduced in the patient group. These findings suggest that oxidative stress may play a role in the etiology of these diseases.[22]

Tantawy et al.[10] conducted a study to explore the potential role of ROS in the pathogenesis of AGA by assessing serum PON1 levels in AGA patients and examining their association with disease severity. Their findings indicated that serum PON1 levels were significantly reduced in AGA patients compared to the control group, with a notable decline in PON1 levels correlating with increased severity of AGA ($P < 0.001$). These results suggest that PON1 may serve as a sensitive and specific biomarker for AGA and could be useful as a predictive indicator for this condition in healthy individuals.[10]

In a similar context, Oszukowska et al.[20] investigated the atherogenic potential in psoriasis by analyzing both antioxidant and pro-oxidant factors, including PON-1, α-tocopherol, uric acid, and homocysteine, comparing these parameters between psoriasis patients and a healthy control group. Their research revealed that PON-1 activity in psoriasis patients was significantly lower than in healthy subjects ($P < 0.001$).[20]

In our study, we investigated PON1 enzyme activity and phenotypic polymorphism in serum samples from 50 AV patients and 52 controls with similar age and gender distributions. We observed that the serum PON1 activity levels in AV patients (36,149±14,536) were significantly lower compared with the levels in the control group (48,173±18,753).

PON1 enzyme activity varies significantly due to polymorphisms. Although its exact physiological role in the body is not entirely understood, many clinical studies have explored the relationship between PON1 activity and various diseases. Additionally, some studies have examined whether there is a link between PON1 polymorphism and specific diseases.[11,23,24]

The second part of our research focused on determining the PON1 phenotype in AV patients, an area that has not been extensively studied. In the patient group, the QQ phenotype was observed at 48%, QR at 24%, and RR at 28%, while in the control group, these frequencies were 5.8%, 44.2%, and 50%, respectively.

Our results show a higher prevalence of the QQ allele and a lower frequency of the RR allele in the AV group compared to the control group. The elevated rate of the QQ allele, associated with lower enzyme activity, is one of the most notable findings of our study.

The primary rationale for selecting PON1 as a focus in this study was the lack of prior investigation into its relationship with AV. Previous studies have established that PON1 activity is associated with inflammatory and oxidative stress-related diseases.[10,19,21,25] In this context, we hypothesized that exploring the potential effects of PON1 on AV could provide novel insights and enhance the originality of our research. Furthermore, establishing a link between PON1 activity and AV may suggest that this enzyme could serve as a potential biomarker, aiding in the assessment of disease severity or the monitoring of treatment efficacy.

Study Limitations

Among the limitations of our study is the relatively small sample size. To better elucidate the relationship between AV and PON enzyme activity, studies with a larger and more diverse population are needed. Additionally, the limited geographic and genetic diversity of the patient and control groups restricts the generalizability of the results. Furthermore, the cross-sectional design of our study prevents drawing definitive conclusions about the causal relationship regarding changes in PON1 activity. We believe that these limitations should be considered in more comprehensive, future studies.

Conclusion

This study investigated PON1 activity and determined its phenotypic polymorphism in AV cases, which had not been previously explored. PON1 activity levels in the patient group (36,149±14,536) were found to be significantly lower than the control group (48,173±18,753) ($P < 0.05$). The finding of reduced activity of PON1, an enzyme with antioxidant properties, in acne patients suggests that oxidative stress may play a role in the etiology of AV.

The second part of our study revealed that the high prevalence of the low-activity QQ allele in the acne group is one of the most striking results. We believe that our study is significant because it is the first to investigate the PON1 phenotype in patients with AV. Based on our findings, we propose that individuals with the low-activity QQ allele may have increased susceptibility to acne development due to oxidative stress.

Ethics

Ethics Committee Approval: This study was approved by Balıkesir University Faculty of Medicine Ethical Committee (approval number: 2014/94, date: 03.11.2021).

Informed Consent: It was obtained.

Footnotes

Authorship Contributions

Surgical and Medical Practices: İ.C., Ç.B., N.G., S.Ö., A.K., Concept: İ.C., Ç.B., N.G., S.Ö., A.K., Design: İ.C., Ç.B., N.G., S.Ö., A.K., Data Collection or Processing: İ.C., Ç.B., N.G., S.Ö., A.K., Analysis or Interpretation: İ.C., Ç.B., N.G., S.Ö., A.K., Literature Search: İ.C., Ç.B., N.G., S.Ö., A.K., Writing: İ.C., Ç.B., N.G., S.Ö., A.K.

Conflict of Interest: The authors declared that they have no conflict of interest.

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