Dermatology Practical & Conceptual

Neutrophil Albumin Ratio, Platelet Albumin Ratio, and Certain Inflammatory Parameters in Acne Vulgaris

Neşe Göçer Gürok[1], Zekiye Çatak[2]

1 Department of Dermatology, Fethi Sekin City Hospital, Elazig, Turkey
2 Department of Medical Biochemistry, Fethi Sekin City Hospital, Elazig, Turkey

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Corresponding Author: Neşe Göçer Gürok, Associate Professor, MD, Fethi Sekin Şehir Hastanesi, 23119, Elazig/Turkey. ORCID: 0000-0001-7069-0447. E-mail: dr.n_g@hotmail.com

ABSTRACT

Introduction: Acne vulgaris is a prevalent, chronic inflammatory disease. Systemic inflammation may be evaluated with various biochemical and hematological markers.

Objectives: The present study was conducted to determine an easy-to-identify and low-cost disease activity marker. We investigated the neutrophil/albumin ratio (NAR), platelet/albumin ratio (PAR), C-reactive protein/albumin ratio (CAR), hemoglobin/albumin ratio (HAR), platelet/hemoglobin ratio (PHR), neutrophil/hemoglobin ratio (NHR), systemic immune response index (SIRI), and monocyte/high-density lipoprotein cholesterol ratio (MHR) in patients with moderate or severe acne vulgaris and the correlations between these markers and the disease.

Methods: The study was conducted on 50 moderate-to-severe acne vulgaris patients and 69 healthy controls. The patient and control groups were similar in age and sex. Patient and control group demographics and laboratory data were collected retrospectively from the patient files.

Results: It was determined that albumin, HDL, and CRP levels, and CAR, NAR, PAR, MHR, SIRI, HAR, PHR, and NHR were similar in acne patients and the control group (P=0.672; P=0.265; P=0.318; P=0.303; P=0.991; P=0.096; P=0.972; P=0.325, P=0.337; P=0.051; P=0.057). Furthermore, specific hemogram parameters were compared between the patient and control groups, with no statistically significant difference determined (P>0.05). However, subgroup analysis by sex showed that female patients had higher HDL and HAR values, while male patients had higher albumin, PHR, NHR, and MHR levels (P<0.05).

Conclusion: No statistically significant association was found between the evaluated systemic inflammation biomarkers and acne vulgaris. Nevertheless, sex-based differences in certain parameters among patients indicate possible sex-specific pathophysiological mechanisms. Prospective, large-scale studies are warranted to further investigate these findings and clarify their clinical implications.

Introduction

Acne vulgaris is a prevalent, chronic inflammatory disease. While it is generally observed on the face, it can also be observed on the upper arms, back, and front of the body. It progresses with blackheads, papules, and pustules, and, in more severe forms, nodules and cysts. The prognosis of the disease varies from the mild form, with a few blackheads, to the severe form, with inflammatory lesions, hyperpigmentation, and scarring that negatively affect the psychology of the patient. Although it is common among adolescents, it can also be observed in adults [1].

Inflammation occurs early in acne pathogenesis and plays a key role in the pathogenesis [2]. The immunochemical pathways underlying the induction and spread of inflammation in acne are complex and are still being investigated [3]. Systemic inflammation could be determined via various biochemical and hematological markers. Biomarkers are significant in clinical practice since they allow objective and quantitative assessment of diagnosis, disease processes, and treatment response [4].

Although hematologic-derived inflammatory markers have been extensively studied in various dermatological inflammatory diseases, accumulating evidence highlights their mechanistic relevance in acne vulgaris, a disease increasingly recognized for its systemic inflammatory components [4-6]. Various hematologic ratios have been investigated in recent studies on acne vulgaris, with significant associations identified between these markers and disease activity [7-9]. These findings suggest that composite indices integrating immune cell counts and biochemical parameters may better capture the complex systemic inflammatory pathways involved in acne pathogenesis and its related comorbidities. Thus, investigating these markers can provide a more comprehensive assessment of systemic inflammation beyond single-parameter measures, enhancing understanding of disease activity and prognosis.

Monocytes are known sources of proinflammatory and oxidative cytokines [5,10]. High-density lipoprotein cholesterol (HDL-C) has anti-inflammatory and antioxidant properties. HDL-C inhibits the oxidation of low-density lipoprotein cholesterol (LDL-C) and prevents its adverse effects on the endothelium [5,11,12]. The monocyte/HDL-C ratio (MHR) has been suggested as a marker of systemic inflammation in several inflammatory diseases [4,5].

C-reactive protein (CRP) is an easily detectable acute-phase protein with a short half-life, synthesized by the liver in response to infection, trauma, and other inflammatory conditions. Thus, clinicians employ CRP as an inflammation marker [13-15]. Albumin is a negative acute-phase protein synthesized by the liver, and its concentration decreases in acute inflammation. Certain studies have suggested that it could serve as a prognostic marker in inflammatory diseases [15-17]. The CRP/albumin ratio (CAR) has recently been studied as an independent prognostic marker in patients with systemic inflammation such as infection, malignancy, and other inflammatory diseases. It has been suggested that CAR measures inflammatory response better than does CRP or albumin. It has been suggested that high CAR levels could be a warning for poor prognosis in systemic disease patients [18,19].

Neutrophil count/albumin ratio (NAR), a novel inflammatory marker, was recently investigated and confirmed as a predictor of clinical outcomes in patients with certain inflammatory diseases, pancreatic cancer, and COVID-19 [20].

Platelet/albumin ratio (PAR) is a clinical biomarker that determines systemic inflammatory and nutritional status with routine laboratory tests and could provide prognostic clues in certain diseases [21,22].

The Systemic Inflammatory Response Index (SIRI) is a novel inflammation marker derived from the counts of neutrophils, lymphocytes, and monocytes. Its prognostic value has been reported in certain malignancies and inflammatory diseases [23]. It demonstrates the balance between the immune status and the inflammatory response of the individual [24].

In addition to the commonly studied indices, we also included hemoglobin/albumin ratio (HAR), platelet/hemoglobin ratio (PHR), and neutrophil/hemoglobin ratio (NHR), which have been investigated in limited studies as potential indicators of systemic inflammation or cancer prognosis. These parameters were derived from routine CBC values and may provide additional insight into inflammatory status in dermatological conditions [25-27].

Objective

The present study aimed to determine NAR, PAR, CAR, HAR, PHR, NHR, SIRI, and MHR in patients with moderate-to-severe acne vulgaris and to analyze their correlation with the disease.

Methods

The study was approved by the Firat University ethics committee (2023/07-10). The study was conducted on 50 patients who attented the Fethi Sekin City Hospital Dermatology Clinic between March 2022 and March 2023 and were diagnosed with moderate-to-severe acne vulgaris on clinical examination. The severity of the disease was determined by the Global Acne Rating System [28]. The patient group included individuals aged 18–30 years old who were diagnosed with acne vulgaris, were not pregnant, had no concomitant dermatological disease nor any systemic disease registration in the system, especially cardiovascular diseases, cancer, systemic inflammatory diseases, chronic degenerative neurological disease, did not use alcohol or any substance or any systemic medication. The control group included 69 healthy individuals aged 18–30 years who attended the hospital for routine check-ups and had a similar age and sex distribution. These individuals had no dermatological disease, no recorded chronic systemic disease, were not receiving systemic medication, and were not pregnant. All samples were analyzed with the electrical impedance method on Beckman Coulter brand Unicel DxH800 model devices. The devices were maintained per the annual maintenance schedule. The devices were maintained with a valid external quality control program (KBUDEK). Internal quality control was conducted before routine analyses. MHR, CAR, NAR, PAR, HAR, PHR, NHR, and SIRI figures were determined in both the patient and control groups and then compared. Patient and control demographics and laboratory data were collected retrospectively based on the patient files. The study was conducted in accordance with the Declaration of Helsinki and good clinical practices.

Statistical Analysis

Statistical analysis was conducted with the SPSS v. 22 software. The normal distribution of the variables was tested with visual (histogram and probability plots) and analytical methods (Kolmogorov-Smirnov/Shapiro-Wilk tests). Pairwise comparisons were conducted with Student's t-test or the Mann-Whitney U test when appropriate. The cases where *P* was lower than 0.05 were considered statistically significant. A priori power analysis was conducted using G*Power 3.1. A moderate effect size (Cohen's d=0.5) was assumed, consistent with both statistical convention and recent findings in similar studies evaluating inflammatory markers in acne vulgaris [7]. For a two-tailed t-test, with α=0.05 and power=0.95, a minimum of 100 participants (50 per group) was required [29].

Results

The study was conducted with 119 individuals, including 50 acne patients (mean age 22±3.6, 11 male and 39 female) and 69 healthy controls (mean age 22±3.0; 22 male and 47 female), with similar age and sex distribution (*P*=0.535 and *P*=0.235, respectively) (Table 1). Albumin, HDL, CRP levels, CAR, NAR, PAR, MHR, SIRI, HAR, PHR, and NHR values were similar across the acne patients and the controls (*P*=0.672; *P*=0.265; *P*=0.318; *P*=0.303; *P*=0.991; *P*=0.096; *P*=0.972; *P*=0.325; *P*=0.337; *P*=0.051; *P*=0.557). Furthermore, certain hemogram parameters were compared between the patient and control groups, and the differences were not statistically significant (Tables 2, 3).

Significant sex-related differences were observed in the patient group but not among healthy controls. Female patients had higher HDL and HAR values, whereas male patients had higher albumin, PHR, NHR, and MHR levels (*P*<0.05 for all) (Table 4). No significant sex-related differences were detected for PAR, NAR, CRP, NLR, SIRI, or CAR.

Discussion

Recently, interest in low-cost, easily accessible biomarkers that can assist in diagnosis, prognosis, and severity assessment of diseases has increased [17-27]. Several studies have been conducted on biomarkers for inflammatory diseases, malignancy, and cardiovascular diseases [5,15,19,23,30].

Acne vulgaris is an inflammatory disease commonly diagnosed in dermatology clinics that leads to suffering in

Table 1. Sex distribution of groups.

			Groups		Total	*P*
			Acne	Control		
Sex	Male	Count	11	22	33	
		% within	33.3%	66.7%	100.0%	0.235
	Female	Count	39	47	86	
		% within	45.3%	54.7%	100.0%	
Total		Count	50	69	119	
		% within	42.0%	58.0%	100.0%	

Table 2. Comparison of basic hemogram and biochemical parameters between the acne and control groups.

	Group	N	Mean	Std. Deviation	*P*
Albumin	Acne	50	43.72	2.69	0.672□
	Control	69	43.95	3.20	
HDL	Acne	50	48.94	8.47	0.265□
	Control	69	50.92	10.27	
HCT	Acne	50	41.60	3.31	0.425□
	Control	69	41.02	4.52	
LYM#	Acne	50	2.17	0.62	0.767□
	Control	69	2.21	0.65	
MON#	Acne	50	0.53	0.14	0.592□
	Control	69	0.55	0.15	
NEU#	Acne	50	4.30	1.11	0.859□
	Control	69	4.34	1.45	
PLT	Acne	50	278.3	55.1	0.115□
	Control	69	260.3	64.5	
CRP	Acne	50	2.8174	1.458	0.318†
	Control	69	2.6228	1.496	
HGB	Acne	50	14.214	1.346	0.834†
	Control	69	14.230	1.697	
	†: Mann-Whitney U test; □: Student T-test;				

HDL: High-density lipoprotein cholesterol, HCT: Hematocrit, LYM#: Absolute lymphocyte count, MON#: Absolute monocyte count, NEU#: Absolute neutrophil count, PLT: Platelet count, CRP: C-reactive protein, HGB: Hemoglobin.

Table 3. Comparison of composite hematological and inflammatory ratios between acne and control groups.

	Group	N	Mean	Std. Deviation	*P*
NAR	Acne	50	0.0987	0.0258	0.991□
	Control	69	0.0985	0.0310	
HAR	Acne	50	1.1239	0.2109	0.337□
	Control	69	1.1677	0.2660	
CAR	Acne	50	0.0644	0.0332	0.303†
	Control	69	0.0600	0.0352	
PAR	Acne	50	6.3886	1.3493	0.096†
	Control	69	5.9379	1.4859	
PHR	Acne	50	5.8945	1.8146	0.051□
	Control	69	5.2824	1.5595	
NHR	Acne	50	0.0919	0.0345	0.557†
	Control	69	0.0897	0.0398	
	Acne	50	.0114	.00433	0.972†
MHR	Control	69	.0114	.00425	
	Acne	50	587.77	265.76	0.325†
SIRI	Control	69	559.99	303.87	
	†: Mann-Whitney U test; □: Student T-test;				

NAR: neutrophil-to-albumin ratio, HAR: hemoglobin-to-albumin ratio, CAR: C-reactive protein-to-albumin ratio, PAR: platelet-to-albumin ratio, PHR: platelet-to-hemoglobin ratio, NHR: neutrophil-to-hemoglobin ratio, MHR: monocyte-to-HDL cholesterol ratio, SIRI: Systemic Inflammation Response Index.

Table 4. Sex-based differences in parameters.

Parameter	Male (Mean ± SD)	Female (Mean ± SD)	p-value
HDL (mg/dL)	42.6 ± 9.0	50.7 ± 7.5	0.004
Albumin (g/L)	45.6 ± 2.9	43.2 ± 2.4	0.006
HAR	0.93 ± 0.17	1.18 ± 0.19	0.0003
PHR	6.96 ± 2.42	5.59 ± 1.51	0.040
NHR	0.12 ± 0.04	0.084 ± 0.03	0.003
MHR	0.0148 ± 0.0062	0.0104 ± 0.0031	0.007

HDL, high-density lipoprotein; HAR, HDL-to-albumin ratio; PHR, platelet-to-HDL ratio; NHR, neutrophil-to-HDL ratio; MHR, monocyte-to-HDL ratio.

patients. The present study investigated various parameters that had not previously been examined, and the ratios of certain parameters were analyzed to determine a prospective clinical biomarker of acne vulgaris. However, these parameters did not exhibit statistically significant differences between the patient and control groups.

A study that investigated CBC parameters and MHR in acne vulgaris patients reported no significant difference between the MHR of the patient and control groups, similar to the present study findings [8]. Also, a study conducted on rosacea patients reported no significant difference between MHR findings of the patient and healthy control groups [31].

Contrary to our findings, a recent study conducted on acne vulgaris patients reported significantly higher CAR in the patient group when compared to healthy controls [7].

Among dermatological diseases, NAR has only been investigated in Behçet's disease. A study investigating NAR and CAR in Behçet patients found that both were elevated. It was suggested that NAR and CAR could be inexpensive markers for the identification of Behçet disease activity [32]. In our study, there was no significant difference between the groups in SIRI. There is only one study in the literature that investigated SIRI in acne vulgaris patients. However, that study was a pretreatment/posttreatment comparison. It investigated the variations between isotretinoin treatments in acne patients and reported that SIRI decreased after the treatment [33]. In a study that investigated SIRI and various inflammatory parameters in hidradenitis suppurativa, it was reported that SIRI was significantly elevated in the patients when compared to healthy controls [34].

There are a limited number of studies on PAR in the current literature. A survey conducted with axial spondylarthritis patients concluded that PAR elevation could be a new and reliable marker of disease activity [35]. In another study, PAR was reported to be associated with ankylosing spondylitis disease activity but not with axial psoriatic arthritis [36]. Although PAR and PHR did not reach statistical significance, both exhibited p-values close to the conventional threshold (PAR: P=0.096; PHR: P=0.051). Notably, the mean PAR and PHR values were higher in the acne group compared to controls. This pattern may suggest an underlying trend toward elevated platelet activity or hemoglobin-related inflammatory modulation in acne vulgaris. These trends could become statistically significant in larger, adequately powered studies.

Although previous studies have reported significant associations between systemic inflammatory biomarkers and various inflammatory dermatoses such as psoriasis and Behçet's disease, we could not detect any statistically significant difference in these indices between acne vulgaris patients and healthy controls in the present study. This discrepancy may be attributed to the localized nature of inflammation in acne, which is primarily confined to the pilosebaceous unit and may not consistently provoke a measurable systemic immune response [37, 38]. Furthermore, acne vulgaris is a multifactorial disease influenced by hormonal fluctuations, individual variability in cutaneous microbiota, and dietary factors affecting pathways such as IGF-1 signaling and mTORC1 activation. These processes, together with individual variations in immune and metabolic responses, may promote localized inflammatory changes without inducing systemic alterations detectable through hematological indices [39,40].

In our study, significant sex-related differences in lipid and hematological inflammatory markers were observed among patients with acne vulgaris, but not in healthy controls. Female patients exhibited higher HDL and HAR, whereas male patients showed elevated albumin, PHR, NHR, and MHR, suggesting sex-specific pathophysiological responses in the context of disease. The higher HDL and HAR in females may reflect estrogen-mediated enhancement of hepatic HDL synthesis and anti-inflammatory effects [41], while increased albumin and pro-inflammatory ratios in males may result from androgen-driven protein synthesis and greater neutrophil/monocyte activation [42,43]. These differences were not detected in healthy controls, indicating that they are likely disease-specific rather than physiological. Notably, when all patients were analyzed together regardless of sex, these differences lost statistical significance, likely due to the relatively small number of male patients (N=11) compared to

females (N=39), which reduced statistical power, and the increased variability introduced by pooling the sexes. These findings underscore that acne vulgaris elicits sex-specific metabolic and inflammatory responses, highlighting the importance of considering sex as a biological variable in understanding disease mechanisms and developing personalized therapeutic strategies.

Conclusions

Systemic inflammatory biomarkers did not significantly differ between moderate-to-severe acne vulgaris patients and healthy controls. However, distinct sex-related differences were observed, suggesting that acne vulgaris may elicit sex-dependent metabolic and inflammatory responses. These findings underscore the importance of accounting for sex in both clinical management and future research on acne pathophysiology.

Limitations

This study has several limitations. Its retrospective design and relatively small sample size may have reduced statistical power. Confounding factors such as dietary habits, metabolic status, subclinical hormonal changes, and skin microbiota composition were not evaluated. Additionally, only systemic inflammatory markers were assessed, without measurement of local skin-level inflammatory mediators. Another limitation is that although all included patients had moderate-to-severe acne, the distinction between moderate and severe cases could not always be reliably made due to inconsistencies in the original records. Therefore, a subgroup analysis according to acne severity could not be performed. Future prospective, large-scale, and sex-balanced studies incorporating both systemic and local biomarker analyses are needed to provide a more comprehensive understanding of acne pathogenesis.

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