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HALP Score and Disease Activity in Psoriatic Arthritis: Comparison with Healthy Controls

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ABSTRACT

Objectives: This study evaluated the relationship between the haemoglobin-albumin-lymphocyte-platelet (HALP) score and disease activity in patients with psoriatic arthritis (PsA), and compared HALP scores between PsA patients and healthy controls.

Methods: This single-centre, cross-sectional study included 73 PsA patients and 59 healthy controls. Demographic, clinical and laboratory data were collected. Disease activity was assessed using the Ankylosing Spondylitis Disease Activity Score (ASDAS) based on C-reactive protein (CRP), the Disease Activity index for Psoriatic Arthritis (DAPSA), the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI), the visual analogue scale (VAS), and the Psoriasis Area and Severity Index (PASI). HALP was calculated as haemoglobin × albumin × lymphocyte / platelet. Group comparisons, correlation analyses and ROC analyses were performed.

Results: Compared with controls, PsA patients had higher CRP, erythrocyte sedimentation rate and platelet values, and lower albumin (all $P<0.05$). HALP scores did not differ significantly between groups ($P=0.232$). HALP correlated positively with age at diagnosis ($r = 0.250$; $P=0.031$) and negatively with ASDAS-CRP ($r = -0.259$; $P=0.026$). ROC analysis showed limited diagnostic performance (AUC = 0.561, $P=0.228$).

Conclusions: Although HALP showed a significant inverse correlation with ASDAS-CRP, the association was weak and its diagnostic performance was poor. HALP alone has limited value in reflecting PsA disease activity or distinguishing patients from controls. This may relate to PsA's heterogeneous inflammation and treatment status. Larger prospective studies in different PsA subgroups are needed to clarify the potential role of HALP as an objective biomarker.

Keywords: Psoriatic Arthritis, HALP Score, Inflammation, Biomarker, Disease Activity

Psoriatic arthritis (PsA) is a chronic, immune-mediated inflammatory arthropathy characterized by inflammation of the joints and entheses, including the axial skeleton [1]. The incidence of PsA in patients with psoriasis ranges from 0.27 to 2.7 per 100 person-years, depending on study design and outcome definitions. PsA typically affects individuals between 30 and 60 years of age and occurs equally in men and women [2]. Peripheral manifestations include polyarthritis, oligoarthritis, distal inter-

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phalangeal arthritis and, less commonly, the arthritis mutilans subtype. Periarticular features such as dactylitis and enthesitis are frequent. Axial PsA, also known as the spondylitis subtype, can be limited to the spine and sacroiliac joints or coexist with peripheral disease [3].

Regular and accurate assessment of disease activity is essential in PsA because uncontrolled inflammation leads to structural damage, functional loss and reduced quality of life [4]. Several clinical indices have been developed for this purpose. The Disease Activity Index for Psoriatic Arthritis (DAPSA) primarily focuses on peripheral joint involvement, while the Psoriatic Arthritis Disease Activity Score (PASDAS) includes a broader range of domains but is complex and time-consuming, limiting its routine use [5–7]. For axial involvement, the Bath Ankylosing Spondylitis Disease Activity Index (BASDAI) and the Ankylosing Spondylitis Disease Activity Score (ASDAS) are commonly used, but they are not specific to PsA and do not fully capture peripheral or skin manifestations [5]. Minimal Disease Activity (MDA) criteria take a multidomain approach but rely on binary outcomes, which may not reflect the full spectrum of disease activity [8]. These limitations highlight the need for more practical and objective biomarkers to assess PsA disease activity.

Recently, several inflammatory ratios derived from complete blood counts and routine biochemistry have been investigated in rheumatic diseases, including the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR) and monocyte-to-HDL ratio (MHR) [9-11]. The haemoglobin–albumin–lymphocyte–platelet (HALP) score, originally proposed as a prognostic marker in malignancies [12], has more recently been explored in autoimmune and inflammatory diseases [13, 14]. HALP reflects nutritional, inflammatory, immune and haematologic status through a simple calculation, making it a potentially useful biomarker in chronic inflammatory conditions. Psoriatic arthritis is characterized by both systemic inflammation and metabolic alterations that may influence the components of the HALP score. Low haemoglobin and albumin levels are common in chronic inflammation and have been linked to disease activity and poor nutritional status [15]. Reduced lymphocyte counts may reflect immune dysregulation, while elevated platelet counts are associated with systemic inflammation and endothelial activation [16, 17]. Therefore, evaluating the HALP score in PsA may provide an integrated measure of inflammatory and nutritional status, potentially serving as a simple, objective indicator of disease activity.

Given these considerations, there remains a need to investigate the role of HALP in PsA. This study aimed to evaluate the relationship between the HALP score and PsA disease activity, and to compare HALP levels between PsA patients and healthy controls, in order to explore its potential clinical utility as an objective biomarker.

METHODS

This study was designed as a single-centre, cross-sectional, observational study. Patients aged 18-65 years who were being followed up at our rheumatology outpatient clinic and had been diagnosed with PsA according to the CASPAR criteria were included in the study. The control group consisted of healthy individuals who did not have any rheumatic disease and who met the exclusion criteria. Those with liver disease, haematological or other malignancies, kidney diseases such as glomerulonephritis, acute or chronic infections, gastrointestinal diseases causing protein loss, other rheumatological diseases or a history of pregnancy were excluded. Approval for the study was obtained from the İnönü University Clinical Research Ethics Committee (numbered 2025/7251 and dated 11 March 2025). The study was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion in the study.

The age, gender, body mass index (BMI), smoking and alcohol consumption habits, duration of illness, clinical characteristics, and medications used of all participants were recorded. Routine laboratory tests included haemoglobin, white blood cell, lymphocyte, neutrophil and platelet counts, as well as albumin, erythrocyte sedimentation rate (ESR) and CRP levels. Additionally, the NLR and PLR were calculated.

PsA disease activity in participants was assessed using the Visual Analogue Scale (VAS), DAPSA, Psoriasis Area and Severity Index (PASI), ASDAS-CRP and BASDAI. The HALP score was calculated using the formula ‘HALP = haemoglobin (g/L) × albumin (g/L) × lymphocytes (/L)/platelets (/L)’ [18].

Statistical Analysis

According to the theoretical power analysis findings, when the test power is 80%, the effect size is 0.8 and the alternative hypothesis (H1) is two-tailed, the required sample size to detect a statistically significant difference between groups using an independent two-sample t-test is at least 52 per group (104 in total) at a 5% significance level. This analysis was performed using the WSSPAS (Web-Based Sample Size & Power Analysis Software) tool, which was developed by the Department of Biostatistics and Medical Informatics at the Faculty of Medicine, İnönü University [19].

The variables used in the study were summarized using arithmetic mean, standard deviation, median, 25th-75th percentiles, and number and percentage statistics. Quantitative data were analyzed for normal distribution using the Shapiro-Wilk test. The independent samples t-test and Mann-Whitney U test were used for between- group comparisons of quantitative data. Pearson's chi-square and Fisher's exact tests were used for between-group comparisons of qualitative data. Receiver operating characteristic (ROC) analysis was used to calculate the cut-off point and area under the curve (AUC) for quantitative data. P≤0.05 was accepted as the statistical significance level. R Project (version 4.1.2) software was used for analyses.

RESULTS

The study included 59 individuals in the control group and 73 patients in the PsA group. There was no significant difference in mean age or BMI between the two groups (P=0.148 and P=0.059, respectively). Serum albumin levels were lower in the PsA group than in the control group (4.3 [4.1-4.6] vs. 4.4 [4.3-4.6] g/dL,

TABLE 1. Comparison of Clinical and Laboratory Parameters Between Groups

Variables		Control Group (n=59)	PsA Group (n=73)	P-value
Age (years),		44±12	47±11	0.148*
BMI (kg/m²)		27.06 (24.2-30.4)	28.6 (25.5-32.8)	0.059**
Gender	Female	40 (67.8%)	55 (75.3%)	0.337†
	Male	19 (32.2%)	18 (24.7%)	
Smoking	No	39 (66.1%)	52 (71.2%)	0.526†
	Yes	20 (33.9%)	21 (28.8%)	
Alcohol	No	58 (98.3%)	70 (95.9%)	0.628††
	Yes	1 (1.7%)	3 (4.1%)	
Albumin (g/dL)		4.4 (4.3–4.6)	4.3 (4.1-4.6)	**0.029****
CRP (mg/dL)		0 (0-0.0)	0.3 (0-1.05)	**<0.001****
Haemoglobin (g/dL)		13.90±1.62	13.36±1.78	0.075*
Lymphocyte (cells/µL)		2220 (1860-2470)	2390 (1870-2940)	0.067**
Neutrophil (cells/µL)		4130 (3160-5260)	4090 (3310-5640)	0.449**
NLR		1.95 (1.58-2.39)	1.76 (1.35-2.45)	0.335**
PLR		123.12 (100.3-140.5)	119 (95.2-151.1)	0.814**
ESR (mm/h)		6 (2-9)	12 (5-24)	**<0.001****
Platelets (x10³/µL)		249 (218-307)	303 (244-349)	**0.006****
HALP Score		50.73 (42.5-63.56)	48 (34.4-62.64)	0.232**

Data are shown as mean±standard deviation or number (percent) or median (25th-75th percentiles) where appropriate.

BMI, body mass index; CRP, C-reactive protein; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; ESR, erythrocyte sedimentation rate; HALP, hemoglobin-albumin lymphocyte-platelet.

*Independent-samples t-test, **Mann-Whitney U test, †Pearson chi-square, ††Fisher's exact test.

Statistically significant P-values are shown in bold.

P=0.029). CRP and ESR levels were higher in the PsA group (CRP: 0.3 [0-1.05] vs. 0 [0-0.0] mg/dL, P<0.001; ESR: 12 [5-24] vs. 6 [2-9] mm/h, P<0.001). Platelet counts were also higher in the PsA group (303 [244-349] vs. 249 [218-307] $\times 10^3/\mu L$, P=0.006). Other parameters, including lymphocyte and neutrophil counts, the neutrophil-to-lymphocyte ratio, and the platelet-to-lymphocyte ratio, did not differ significantly between the two groups (P>0.05 for all). Gender distribution, smoking status, and alcohol consumption were similar between groups (P=0.337, P=0.526, and P=0.628, respectively). No significant difference was found in HALP scores between PsA and control groups (48 [34.4-62.64] vs. 50.73 [42.5-63.56], P=0.232). These findings are summarized in Table 1.

Among the 73 PsA patients, 53.4% (n= 39) had peripheral involvement, 42.5% (n=31) had both axial and peripheral disease, and 4.1% had purely axial involvement. Regarding treatment, 20.5% were untreated, 43.8% (n=32) were on conventional synthetic DMARDs (csDMARDs), and smaller proportions received anti-TNF agents (11.0%) or IL-17 inhibitors (9.6%). Corticosteroids were used by 28.8% (n=21) of patients. The median disease duration was 4.0 years (P25–P75: 0.3–7.0). The median VAS, DAPSA, PASI, and BASDAI scores were 6.0 (5.0-8.0), 16.0 (10.0-21.0), 0.3 (0.0-2.4), and 6.1 (4.5-7.1), respectively. The mean ASDAS-CRP score was 2.65± .06 (Table 2).

TABLE 2. Clinical Characteristics, Treatment Distribution and disease Activity Scores of Patients

Characteristics		Value
Involvement type	Axial	3 (4.1)
	Peripheral	39 (53.4)
	Axial + Peripheral	31 (42.5)
	Total	73 (100)
DMARD treatment	No treatment	15 (20.5)
	csDMARD	32 (43.8)
	>1 csDMARD	4 (5.5)
	anti-TNF	8 (11.0)
	IL-17 inhibitor	7 (9.6)
	csDMARD+anti-TNF	6 (8.2)
	csDMARD+IL-17 inhibitor	1 (1.4)
	Total	73 (100)
Steroid use	No	52 (71.2)
	Yes	21 (28.8)
	Total	73 (100)
Age at diagnosis (years)		4.0 (0.3-7.0)
VAS score,		6.0 (5.0-8.0)
DAPSA score		16.0 (10.0-21.0)
PASI score		0.3 (0.0-2.4)
ASDAS-CRP score		2.65±1.06
BASDAI score		6.1 (4.5-7.1)

Data are shown as mean±standard deviation or number (percent) or median (25th-75th percentiles) where appropriate. DMARD, disease-modifying antirheumatic drug; csDMARD, conventional synthetic DMARD; TNF, tumor necrosis factor; IL-17, interleukin-17; VAS, visual analogue scale; DAPSA, disease activity in psoriatic arthritis; PASI, psoriasis area and severity index; ASDAS-CRP, ankylosing spondylitis disease activity score using C-reactive protein; BASDAI, bath ankylosing spondylitis disease activity index.

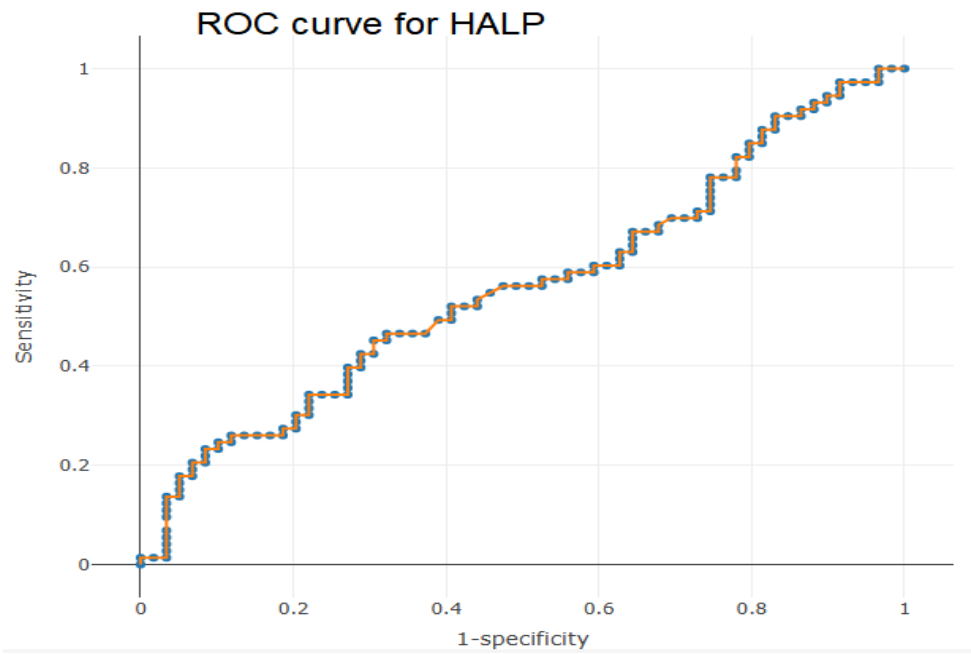


FIGURE 1. Receiver operating characteristic (ROC) curve of the HALP score for distinguishing PsA patients from healthy controls. The area under the curve (AUC) was 0.561 (95% CI: 0.462-0.659), with a sensitivity of 23.3% and a specificity of 91.5%, indicating limited diagnostic discrimination.

In the correlation analysis conducted on the PsA group, a positive and significant correlation was found between the HALP score and age at diagnosis (r=0.250, 95% CI: 0.022-0.456, P=0.031). By contrast, a negative and significant correlation was found between the HALP score and the ASDAS-CRP score (r=−0.259, 95% CI: -0.462 to -0.034, P=0.026).

ROC analysis showed an AUC of 0.561 (95% CI: 0.462-0.659), with an optimal cut-off value of 31.25, corresponding to a sensitivity of 23.3% and a specificity of 91.5% (P=0.228). These values indicate that the discriminative performance of HALP for distinguishing PsA from controls was not statistically significant. The ROC curve is presented in Figure 1.

DISCUSSION

This study examined the prognostic value of the HALP score in relation to the clinical and laboratory characteristics of patients with PsA. Our findings showed that there were significant differences in CRP, ESR, platelet count and serum albumin levels in the PsA group compared to the control group, but the HALP score did not show a statistically significant difference. Furthermore, ROC curve analysis showed that the HALP score had limited discriminatory power. These results suggest that the HALP score may not be a reliable PsA biomarker on its own.

In cases of systemic inflammation, the number of platelets often increases, while haemoglobin, albumin and lymphocyte levels tend to decrease. Inflammatory cytokines increase the number of neutrophils in circulation while decreasing lymphocyte count and albumin levels [20, 21]. Interleukin (IL)-1β and tumour necrosis factor (TNF)-α suppress the release of erythropoietin from the kidneys. Additionally, IL-6 increases hepcidin production in the liver. This reduces iron absorption from the intestine and iron release from macrophages, leading to decreased haemoglobin production and chronic disease anaemia in patients [22, 23]. Therefore, a decrease in the HALP score is expected in cases of systemic inflammation.

Previous studies have demonstrated that the HALP score may be useful for predicting outcomes in different disease groups. In recent years, attention has been drawn to the HALP score due to its ability to assess inflammation, nutrition, immune response and coagulation processes simultaneously. The ease with which it can be calculated also makes it attractive for

use, particularly in diseases involving intense inflammation [24, 25]. Notably, a low HALP score has been reported to be strongly associated with survival in solid tumours, including gastric and colorectal cancer [12, 18]. Furthermore, the HALP score has been identified as a meaningful biomarker for predicting renal relapses in lupus nephritis and for determining disease activity in patients with ankylosing spondylitis [13, 14]. These findings suggest that the HALP score could play a role in both malignant and inflammatory processes. In our study, HALP showed a statistically significant negative correlation with ASDAS-CRP, which may indicate a partial relationship with inflammatory activity in PsA. However, this association was weak, and no clear correlations were observed with other disease activity indices. These results imply that the clinical utility of HALP in PsA may be more limited than in other diseases.

One possible explanation for the limited performance of the HALP score in PsA is that systemic inflammatory responses tend to be less pronounced in PsA compared to other inflammatory arthritides such as rheumatoid arthritis (RA). Recent studies have shown that levels of CRP, serum amyloid A (SAA), and adhesion molecules (sICAM-1, sVCAM-1) are significantly higher in RA than in PsA [26]. In PsA, the inflammatory response is often localized to tissues such as the entheses and synovium, and type 3 immune pathways, particularly IL-23/IL-17 signaling, are thought to play a central role in this process. As a result, classical systemic inflammatory markers are not always elevated [1, 2]. This pathophysiological difference may partly explain the limited performance of systemic inflammation-based indices, such as the HALP score, in PsA.

Several studies have investigated the NLR and PLR as markers of systemic inflammation in PsA. For example, Kim *et al*. [27] reported in a retrospective Korean study that both NLR and PLR were significantly higher in PsA patients compared to healthy controls and patients with psoriasis alone, and that NLR was particularly effective in predicting the presence of PsA. Similarly, a Japanese study demonstrated that pre-treatment NLR and PLR levels were significantly higher in PsA than in psoriasis, and that both ratios decreased in parallel with CRP following biologic therapy [28]. These findings suggest that NLR and PLR may serve as useful indicators of systemic inflammation in PsA. In our study, most patients were receiving treatment, and ESR and CRP levels were not markedly elevated. This may partly explain the limited performance of both NLR/PLR and the HALP score. Although HALP reflects not only inflammation but also nutritional and hematologic status, the localized inflammatory response characteristic of PsA, together with treatment-related suppression, may have reduced its ability to distinguish disease activity. These findings suggest that HALP alone may not be sufficient as a biomarker in PsA, but its combined use with other hematological ratios (e.g., NLR, PLR, MHR) or novel biological markers could enhance its clinical relevance.

Furthermore, recent studies in other rheumatologic diseases have demonstrated a growing interest in inflammation-related hematologic and biochemical ratios. For example, one study evaluated monocyte-to-HDL and CRP-to-albumin ratios in Takayasu arteritis, demonstrating that these markers may reflect disease activity and vascular inflammation [29]. From this perspective, HALP can be viewed as part of a broader group of composite ratios being investigated in various rheumatologic conditions to quantify inflammatory burden and monitor disease activity.

Strengths and Limitations

To our knowledge, this is the first study to directly examine the relationship between the HALP score and disease activity in patients with PsA, offering a new perspective to the current literature. A major strength of our study is its comparative design with a healthy control group and the multidimensional evaluation of disease activity using different clinical indices. However, several limitations should be considered. The relatively small sample size, cross-sectional design, and heterogeneity of treatment regimens may have influenced our findings. In addition, evaluating HALP in different PsA subtypes (axial, peripheral, dactylitis-dominant) or across various treatment groups (e.g., bDMARDs, IL-17 inhibitors) may help to better define its potential clinical applications. These findings also underline the limited value of the HALP score in PsA and emphasize the need for cautious interpretation of systemic inflammation markers in clinical practice. Therefore, further confirmation in larger, prospective cohorts is warranted. Future multicentre studies will help to more clearly define the biomarker potential of the HALP score in psoriatic arthritis.

CONCLUSION

In conclusion, the HALP score demonstrated a limited association with disease activity in PsA, likely reflecting the unique inflammatory profile of this disease. Although it may not serve as a stand-alone biomarker, its accessibility and simplicity suggest potential value as an exploratory or hypothesis-generating parameter rather than a clinically established marker. Larger, prospective, and multicentre studies are needed to confirm these preliminary findings and to better define the role of HALP in disease monitoring and assessment in PsA.

Ethics Approval and Consent to Participate

This study was approved by the the İnönü University Scientific Research and Publication Ethics Committee (Decision No: 2025/7251; date: 11.03.2025). All procedures were conducted in accordance with the ethical standards of the institutional and national research committee and with the 1964 Helsinki Declaration and its later amendments. Written informed consent was obtained from all participants prior to inclusion in the study.

Data Availability

All data generated or analyzed during this study are included in this published article. The data that support the findings of this study are available on request from the corresponding author, upon reasonable request.

Authors' Contribution

Study Conception: SZ, SY, MBB; Study Design: SZ, SY; Supervision: SZ, SY; Funding: N/A, BS; Materials: SZ, EŞ, Eİ; Data Collection and/or Processing: SZ, Eİ, EŞ; Statistical Analysis and/or Data Interpretation: AKA, SZ; Literature Review: SZ; Manuscript Preparation: SZ, AKA; and Critical Review: SY, Eİ, EŞ, AKA.

Conflict of Interest

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Generative Artificial Intelligence Statement

The author(s) declare that no artificial intelligence-based tools or applications were used during the preparation process of this manuscript. The all content of the study was produced by the author(s) in accordance with scientific research methods and academic ethical principles.

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