ORIGINAL ARTICLE
Iran J Allergy Asthma Immunol
December 2024; 23(6):651-661.
DOI: 10.18502/ijaai.v23i6.17375

Exploring the Association between Blood Indices and Skin and Joint Activity of Psoriatic Arthritis

Abdolrahman Rostamian[1], Shila Aghayani[2], Seyed Reza Najafizadeh[3], Zahra Saffarian[4], and Maryam Yaseri[3]

[1] Department of Internal Medicine, School of Medicine, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran
[2] Department of Rheumatology, School of Medicine, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran
[3] Rheumatology Research Center, Vali-e-Asr Hospital, Tehran University of Medical Sciences, Tehran, Iran
[4] Department of Dermatology, School of Medicine, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran

Received: 4 April 2024; Received in revised form: 5 July 2024; Accepted: 21 July 2024

ABSTRACT

The exact cause of psoriatic arthritis is still unknown, but hypotheses suggest the role of hematological parameters in the onset and severity of the disease. This study evaluated the hematological indices and their association with skin and joint activity in psoriatic arthritis.

This cross-sectional study included 74 patients with psoriatic arthritis. Demographical and clinical data, blood indices, psoriasis area and severity index (PASI) score and disease activity in psoriatic arthritis (DAPSA) scores were calculated for all patients.

The mean age of the patients was 48.89±12.03 years and most were female (n=49). A significant correlation was observed between age and number of underlying diseases with PASI and DAPSA scores. Mean PASI and DAPSA scores were 5.19 and 15.13, respectively. The severity of psoriasis was mild in 58.1%, moderate in 36.5%, and severe in 4.5% of the cases. The activity of psoriatic arthritis was improved in 2.1%, low in 55.4%, moderate in 24.3%, and high in 1.8% of the patients. A significant association was found between erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), red cell distribution width (RDW), neutrophil-to-lymphocyte ratio (NLR), platelet (PLT) count, mean platelet volume (MPV), and PASI scores, while no statistically significant association was reported for PLR. A significant correlation was observed between ESR, CRP, RDW, NLR, PLR, PLT, and DAPSA scores, while no statistically significant association was found for MPV.

The findings indicated that inflammatory and hematological markers can be helpful factors in evaluating the severity of psoriasis and psoriatic arthritis.

Keywords: Hematologic test; Psoriatic arthritis; Psoriasis

Corresponding Author: Maryam Yaseri, MD; Rheumatology Research Center, Vali-e-Asr Hospital, Tehran University of Medical Sciences, Tehran, Iran. Tel: (+98 911) 1350 675, Email: maryam.yaseri21@yahoo.com

INTRODUCTION

Psoriatic arthritis is a chronic inflammatory condition that affects individuals with psoriasis, a common

autoimmune skin disorder characterized by red, scaly patches.[1,2] Psoriatic arthritis involves inflammation of the joints, leading to pain, stiffness, and swelling, often accompanied by nail changes and other extra-articular manifestations.[3] The prevalence of psoriatic arthritis varies widely across different populations, but it is estimated to affect approximately 20% of individuals with psoriasis.[4] The development of psoriatic arthritis involves multiple factors, including genetic predisposition, environmental influences, cellular immunological mechanisms, and the involvement of various cytokines, chemokines, and secreted proteins.[5] Psoriatic arthritis is an immune-mediated condition initiated by the major histocompatibility complex (MHC) class I antigen, a hypothesis strongly supported by substantial evidence.[5,6] The diverse clinical manifestations observed in psoriatic arthritis can challenge its diagnosis. This variability in presentation underscores the importance of considering psoriatic arthritis as a potential diagnosis, even in cases where the joint involvement appears limited.[7]

Although there are no specific laboratory tests available for the diagnosis of psoriatic arthritis, hematological indices are significant diagnostic and monitoring tools in studying psoriatic arthritis.[8] In individuals with psoriatic arthritis, certain acute phase markers such as erythrocyte sedimentation rate (ESR), mean platelet volume (MPV), red cell distribution width (RDW), and C-reactive protein (CRP) may exhibit elevated levels.[9-12] In previous studies, some blood parameters such as neutrophil-to-lymphocyte ratio (NLR) and platelet-to-lymphocyte ratio (PLR) have played an important role in the diagnosis of diseases such as ankylosing spondylitis, Behçet's disease, rheumatoid arthritis, and psoriasis.[13,14] Therefore, we investigated the association between hematologic parameters with disease severity and skin and joint activity in patients with psoriatic arthritis.

PATIENTS AND METHODS

Study Design

This cross-sectional study was conducted on 74 patients with psoriatic arthritis referred to Imam Khomeini Hospital, Tehran, Iran, in 2023. The classification criteria for diagnosing psoriatic arthritis (CASPAR) were meticulously assessed across all patients.[15] Patients with the presence of prior autoimmune disorders like lupus, rheumatoid arthritis, and Sjogren's syndrome, along with acute or chronic infections, malignancies, hematological disorders, pregnancy, chronic liver and kidney diseases, and recent blood transfusions within the past 4 months were excluded from the study. Data on patients' demographical and clinical characteristics, including age, gender, disease duration, body mass index (BMI), underlying diseases, and types of medication were collected. Also, laboratory parameters, including MPV (normal range, 7–11.5 fL), RDW-cv (normal range, 10.5–14.6 %), NLR (0.43–2.75 in men and 0.37–2.87 in women), PLR (36.63–149.13 in men and 43.36–172.68 in women), ESR (men <50 years, ≤15 mm/hr; women <50 years, ≤20 mm/hr; men>50 years,≤20 mm/hr), platelet (PLT) (normal range, 150–450 $10^3/\mu L$), and CRP (normal range, <6 mg/dL) levels were assessed. NLR was calculated by dividing the neutrophil absolute count by the lymphocyte absolute count, and PLR was calculated by dividing the platelet count by the lymphocyte absolute count. Psoriasis area and severity index (PASI) score (indicating the extent and severity of psoriasis-affected skin area)[16] and disease activity index for psoriatic arthritis (DAPSA) score (reflecting the intensity of psoriatic arthritis activity were subjected to rigorous statistical analysis.[17]

PASI and DAPSA Scores

PASI was calculated using the formula: PASI = 0.1 (E × A + I × B + D × C), where E represents erythema (redness), A denotes the affected area, I stands for induration (thickness), B indicates scaling (flaking), D represents desquamation (shedding of skin), and C represents the head, the trunk, and the limbs (each part is assigned a value from 0 to 4, depending on the severity and extent of involvement). The percentage of body surface area (BSA) affected by psoriasis is usually estimated in increments of 10, with each palm-sized area being roughly 1% of the BSA. The scores for each component range from 0 to 4, where 0 indicates no involvement, and 4 indicates severe involvement. The PASI score can range from 0 to 72, with higher scores indicating more severe psoriasis.

DAPSA score was derived from a combination of clinical and laboratory parameters. It was calculated using the formula: DAPSA=TJC28 + SJC28 + 0.56 × √ (Tender Entheseal Points) + 0.28 × ln (CRP mg/dL), where TJC28 represents the tender joint count out of 28 joints assessed, SJC28 represents the swollen joint count out of 28 joints assessed, Tender Entheseal Points represent the number of tender entheses out of 6

assessed, and CRP is the C-reactive protein level in milligrams per deciliter. The DAPSA score served as a comprehensive measure of disease activity in psoriatic arthritis, encompassing joint and skin involvement and inflammatory markers. Lower DAPSA scores signified lower disease activity, whereas higher scores indicated higher disease activity.

Statistical Analysis

The presentation of variables depicts their frequencies as percentages, mean±standard deviation (SD), and median (interquartile range). The normality of the quantitative variables was assessed using the Kolmogorov-Smirnov test. To explore the relationship between clinical-demographic characteristics and PASI and DAPSA scores, Spearman's correlation coefficient, Mann-Whitney test, and Johnkheer-Terpstra test were employed. Additionally, Spearman's correlation coefficient was utilized to examine the association between blood indices and PASI and DAPSA scores. Correlation coefficients falling within 0.1–0.3, 0.3–0.5, and <0.5 were interpreted as weak, moderate, and strong correlations, respectively. To evaluate the diagnostic efficacy of blood indices in predicting disease activity, the receiver operating characteristic (ROC) curve was constructed, yielding the area under the curve (AUC). Youden's J index determined the optimal cut-off point, with sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio (LR+), and negative likelihood ratio (LR–) calculated accordingly. AUC values range from 0 to 1, with interpretations of 0.5–0.6, 0.6–0.7, 0.7–0.8, 0.8–0.9, and <0.9 representing negligible, weak, moderate, good, and outstanding diagnostic accuracy, respectively. Data analysis was performed using SPSS version 16 and MedCalc software version 19.5.3, with statistical significance set at $p<0.05$. GraphPad Prism version 8.0.1 was employed for graphical representations.

RESULTS

Out of the 74 participants, 49 were females, and the mean age and mean duration of disease were 48.89±12.03 and 7.20±6.12 years, respectively. The average BMI of the patients was 27.91±3.24 kg/m^2. Among the patients, 26 (35.1%), 23 (31.1%), and 16 (21.6%) presented with hyperlipidemia, hypertension, and diabetes, respectively. About 10 individuals (13.5%) were using nonsteroidal anti-inflammatory drugs (NSAIDs), 66 (89.2%) used disease-modifying antirheumatic drugs (DMARDs), and 27 (36.5%) received antitumor necrosis factor-alpha (anti-TNF-α) drugs, with 45 patients (60.8%) receiving single-drug and 29 (39.2%) receiving multidrug regimens. Also, a high mean of ESR and CRP was observed among patients, 20.91±12.22 mm/hr and 7.61±9.51 mg/L, respectively. Regarding the psoriasis assessment, the average PASI score was 5.19±2.32. Notably, 75% of patients exhibited PASI scores >3.5, 50% had scores >4.5, and 25% had scores >6.5. According to cutoff points, psoriasis severity was mild in 43 individuals (58.1%), moderate in 27 (36.5%), and severe in 4 (5.4%) cases. The mean DAPSA score was 13.15±8.41. Notably, 75% of patients recorded DAPSA scores >8, 50% had >12, and 25% had >16. Based on the recommended cutoff points, disease activity in 9 individuals (12.2%) improved, 41 (55.4%) exhibited low activity, 18 (24.3%) were moderate, and 6 (1.1%) presented high activity (Table 1).

Analysis reveals a weak positive correlation between age and PASI score among the participants ($p=0.010$, $\rho=0.297$), indicating a general increase in PASI scores with advancing age. Similarly, the disease duration showed a comparable trend ($p=0.036$, $\rho=0.245$), with longer durations correlating with higher PASI scores. Notably, a significant relationship emerged between the number of comorbidities and PASI score ($p=0.047$), where comorbidities were associated with higher PASI scores. However, no significant associations were found between PASI scores and gender ($p=0.202$), BMI ($p=0.476$), and number of medication intakes ($p=0.764$) with PASI scores. In terms of DAPSA scores, a significant weak positive correlation with age was observed ($p=0.030$, $p=0.253$), indicating an increase in DAPSA scores with patient age. Moreover, a weak but significant positive correlation was found between BMI and DAPSA score ($p=0.014$, $\rho=0.284$), suggesting that higher BMI levels corresponded to higher DAPSA scores. The number of comorbidities also displayed a significant relationship with the DAPSA score ($p=0.020$), indicating that an increase in comorbidities was associated with higher DAPSA scores. However, DAPSA scores did not exhibit significant relationships with gender ($p=0.881$), disease duration ($p=0.813$), or type of drug treatment ($p=0.863$).

Table 1. Demographical data and clinical characteristics of patients with psoriatic arthritis.

Variables		Frequency n(%)	Mean±SD (min-max)
Age (year)			48.89±12.03
	≤40	22 (29.7)	
	41–50	16 (21.6)	
	51–60	22 (29.7)	
	≥60	14 (18.9)	
Gender	Male	25 (33.8)	
	Female	49 (66.2)	
BMI (kg/m^2)			27.91±3.24
	20–25	15 (20.3)	
	25–30	40 (54.1)	
	≥30	19 (25.7)	
Duration of disease (year)			7.20±6.12
Underlying diseases	Hyperlipidemia	26 (35.1)	
	Hypertension	23 (31.1)	
	Diabetes	16 (21.4)	
	Hypothyroidism	11 (14.9)	
	Ischemic heart diseases	5 (6.8)	
	Pulmonary diseases	4 (5.4)	
	Kidney diseases	1 (1.4)	
	Autoimmune diseases	0 (0.0)	
Number of underlying diseases	0	27 (36.5)	
	1	17 (23.0)	
	2	21 (28.4)	
	3	9 (12.2)	
Medication	NSAIDs	10 (13.5)	
	DMARDs	66 (89.2)	
	Anti-TNF	27 (36.5)	
	JAK inhibitor	0 (0.0)	
Types of therapy	One medication intake	45 (60.8)	
	Multiple medication intake	29 (39.2)	
Hematologic indices	WBC (×10^9/L)		7.67±2.24 (4.30–12.40)
	Neutrophils (×10^9/L)		4.80±1.83 (2.25–9.67)
	Lymphocytes (×10^9/L)		12.22 (1.15–5.21)
	PLT (×10^9/L)		263.8±79.5 (32–597)
	ESR (mm/h)		20.91±12.22 (3–56)
	CRP (mg/L)		7.61±9.51 (1–60)
	RDW (%)		13.21±0.91 (11.50–17.40)
	MPV (fL)		9.31±0.64 (7.30–11.60)
	NLR		2.13±0.91 (0.70–5.37)
	PLR		119.2±48.1 (14.8–318.4)
PASI score			5.19±2.32 (2.3–12.0)
DAPSA score			13.15±8.41 (3–44)

BMI: body mass index; CRP: C-reactive protein; DAPSA: Disease Activity in Psoriatic Arthritis; DMARDs: disease-modifying antirheumatic drugs; ESR: erythrocyte sedimentation rate; fL: femtoliter; JAK: Janus kinase; MPV: mean platelet volume; NLR: neutrophil-to-lymphocyte ratio; NSAIDs: nonsteroidal anti-inflammatory drugs; PASI: Psoriasis Area and Severity Index; PLR: platelet-to-lymphocyte ratio; PLT: platelets; RDW: red cell distribution width; TNF: tumor necrosis factor; WBC: white blood cell count.

A strong positive correlation between ESR and PASI scores (p=0.569, p<0.001), also for CRP (p=0.560, p<0.001) was found. Furthermore, PASI scores exhibited significant positive correlations with PLT count (p=0.391, p<0.001) and RDW (p=0.318, p=0.006). At a lower level, PASI scores also correlated positively with MPV (p=0.250, p=0.031) and NLR (ρ=0.295, p=0.011). However, no significant correlation was observed between PLR and PASI scores (p=0.106, p=0.371). Moreover, a positive correlation was evident between ESR and DAPSA scores (ρ=0.639, p<0.001) and also for CRP (p=0.767, p<0.001). DAPSA scores similarly correlated positively with PLT count (p=0.430, p<0.001) and RDW (ρ=0.408, p<0.001). However, the correlation between MPV and DAPSA scores was insignificant (ρ=0.227, p=0.052). Nevertheless, DAPSA scores demonstrated significant positive correlations with NLR (p=0.322, p=0.005) and PLR (p=0.231, p=0.048) (Table 2).

Table 2. Association between variables and PASI and DAPSA scores of patients with psoriatic arthritis.

Hematologic Indices		PASI score		DAPSA score	
		Mean ρ (IQR)	*p* value	Mean ρ (IQR)	*p* value
Age		0.297	0.010†	0.253	0.030†
Gender			0.202‡		0.881‡
	Male	5.3 (3.4–7.8)		12.0 (6.5–16.0)	
	Female	4.0 (3.5–6.0)		12.0 (8.0–16.0)	
BMI (kg/m^2)		0.084	0.467†	0.284	0.014†
Duration of disease (year)		0.245	0.036†	0.028	0.813†
Number of			0.047§		0.020§
underlying	**0**	4.0 (3.5–4.7)		8.0 (6.0–14.0)	
diseases	**1**	4.6 (3.5–6.5)		8.0 (8.0–15.5)	
	≥2	5.2 (3.5–7.6)		13.0 (9.8–0.2)	
Types of therapy			0.764‡		0.863‡
	One medication intake	4.5 (3.5–7.0)		4.5 (3.5–6.5)	
	Multiple medication intake	12.0 (6.5–19.0)		12.0 (8.0–15.0)	
WBC		0.419	<0.001†	0.403	<0.001†
Neutrophils		0.437	<0.001†	0.407	<0.001†
Lymphocytes		0.188	0.108†	0.056	0.633†
PLT		0.391	<0.001†	0.430	<0.001†
ESR		0.569	<0.001†	0.639	<0.001†
CRP		0.560	<0.001†	0.767	<0.001†
RDW		0.318	0.006†	0.408	<0.001†
MPV		0.250	0.031†	0.227	0.052†
NLR		0.295	0.011†	0.322	0.005†
PLR		0.106	0.371†	0.231	0.048†

† Spearman's correlation coefficient; ‡ Mann-Whitney test; § Johnkheer-Terpstra test

Correlation coefficient values of 0.1–0.3, 0.3–0.5, and ≥0.5 indicate weak, moderate, and strong correlation, respectively.

BMI: body mass index; CRP: C-reactive protein; DAPSA: Disease Activity in Psoriatic Arthritis; ESR: erythrocyte sedimentation rate; IQR: interquartile range; MPV: mean platelet volume; NLR: neutrophil-to-lymphocyte ratio; PASI: Psoriasis Area and Severity Index; PLR: platelet-to-lymphocyte ratio; PLT: platelets; RDW: red cell distribution width; WBC: white blood cell count.

ROC curve analysis showed that ESR (AUC=0.879), CRP (AUC=0.864), and PLT count (AUC=0.769) exhibited the highest predictive potential for disease activity in psoriatic arthritis. ESR and CRP demonstrated good predictive power, while PLT count showed relatively good predictive capability. The optimal ESR cutoff point was identified as 13 for the J index. This threshold yielded a sensitivity of 76.9%, specificity of 88.9%, positive predictive value of 98.0%, negative predictive value of 34.8%, positive likelihood ratio of 6.92, and negative likelihood ratio of 0.26. Cutoff point values for other blood parameters are comprehensively presented in Table 3 and Figure 1.

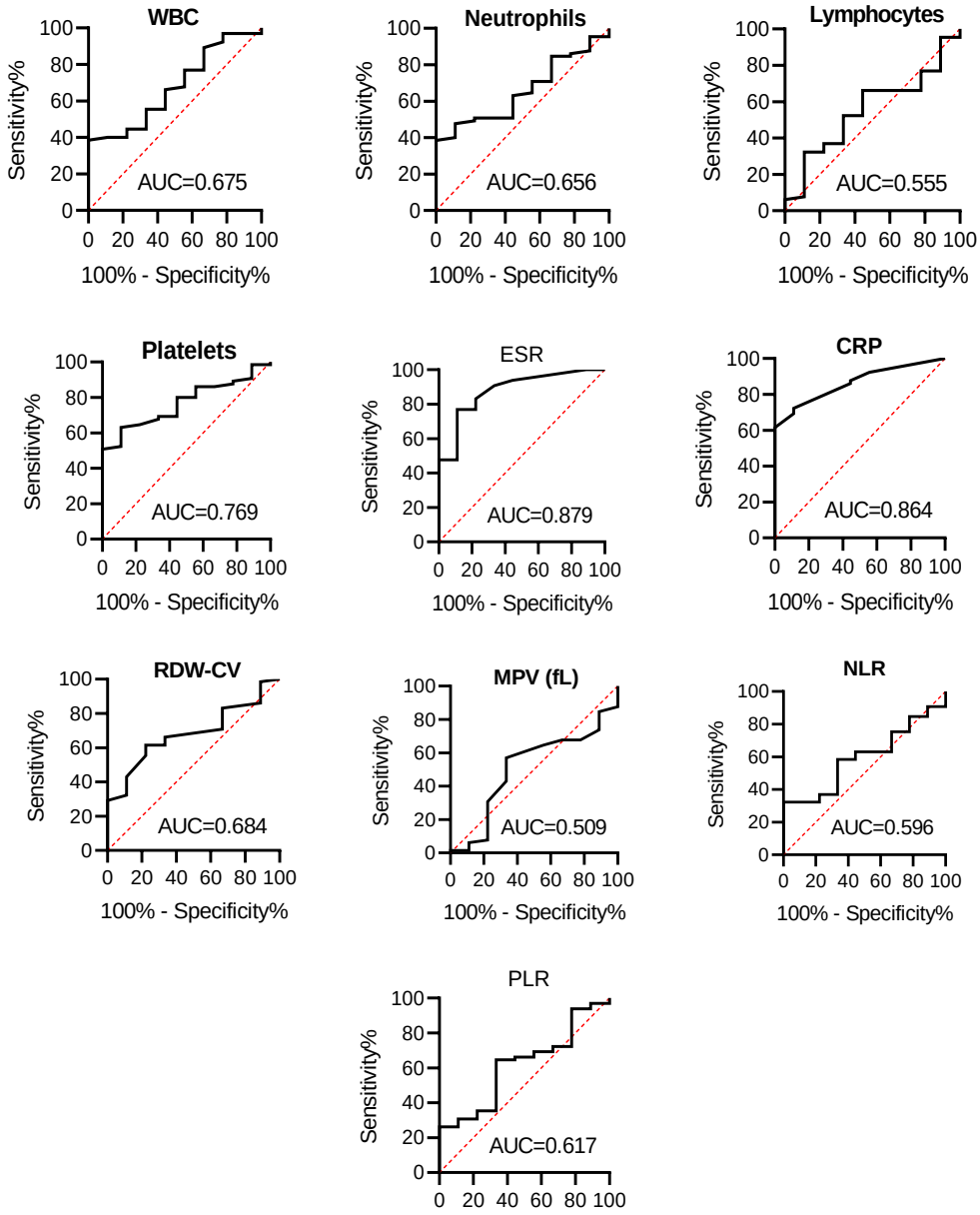

Figure 1. The predictive power of laboratory indicators in predicting psoriatic arthritis disease activity. The possible range of the area under the curve (AUC) value is between 0 and 1, and AUC values are 0.5–0.6, 0.6–0.7, 0.7–0.8, 0.8–0.9, and <0.9 representing no discriminatory ability, weak, relatively good, good, and excellent recognition power, respectively. CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; MPV: mean platelet volume; NLR: neutrophil-to-lymphocyte ratio; PLR: platelet-to-lymphocyte ratio; RDW-CV: red cell distribution width coefficient of variation; WBC: white blood cell count.

Table 3. The predictive power of laboratory indicators in predicting psoriatic arthritis disease activity.

Hematologic Indices	AUC (95% CI)	*p*	Cutoff	Sensitivity	Specificity	PPV	NPV	LR+	LR–
WBC	0.675 (0.509–0.842)	0.090	8.34	38.5	100	100	18.4	–	0.62
Neutrophils	0.656 (0.506–0.807)	0.130	5.44	38.5	100	100	18.4	–	0.62
Lymphocytes	0.55 (0.371–0.738)	0.597	2	66.2	55.6	91.5	18.5	1.49	0.61
PLT	0.769 (0.648–0.891)	0.009	233	63.1	88.9	97.6	25.0	5.68	0.42
ESR	0.879 (0.766–0.993)	<0.001	13	763.9	88.9	98.1	34.8	6.92	0.26
CRP	0.864 (0.771–0.957)	<0.001	4	61.5	100	100	26.5	–	0.38
RDW	0.684 (0.533–0.834)	0.075	13	61.5	77.8	95.2	21.9	2.77	0.49
MPV	0.509 (0.310–0.707)	0.934	9.2	56.9	66.7	92.5	17.6	1.71	0.65
NLR	0.596 (0.434–0.757)	0.354	2.27	32.2	100	100	17.0	–	0.68
PLR	0.617 (0.438–0.796)	0.257	98.5	64.6	66.7	93.3	20.7	1.94	0.53

The possible range of AUC values is between zero and one, and AUC values are 0.5–0.6, 0.6–0.7, 0.7–0.8, 0.8–0.9, and <0.9, representing no discriminatory ability, weak, relatively good, good, and excellent recognition power, respectively.

AUC: area under the curve; CI: confidence interval; CRP: C-reactive protein; ESR: erythrocyte sedimentation rate; LR+: positive likelihood ratio; LR–: negative likelihood ratio; MPV: mean platelet volume; NLR: neutrophil-to-lymphocyte ratio; NPV: negative predictive value; PLR: platelet-to-lymphocyte ratio; PLT: platelets; PPV: positive predictive value; RDW: red cell distribution width; WBC: white blood cell count.

DISCUSSION

Psoriatic arthritis as a chronic inflammatory musculoskeletal disease has variable clinical manifestations and treatment responses influenced by demographic and social factors. In the present study, the majority of patients were women, and the most common comorbidities observed among the participants were hyperlipidemia, hypertension, and diabetes, respectively. Our study found a significant positive correlation between increasing age and higher PASI and DAPSA scores. A study by Gisondi et al demonstrated that hypertension, dyslipidemia, and diabetes, respectively, had higher frequencies among comorbidities, and the majority of patients were males.[18] Another study reported a higher frequency of female gender and received monotherapy among patients with psoriatic arthritis with a mean age of 39 years, and the average duration of the disease was 3 years.[19] Similar to their findings, we observed that monotherapy was more frequent among patients. The patients' mean age and disease duration were higher in the current study. Kılıç et al reported that the prevalence of female gender and comorbidities in patients with psoriatic arthritis was higher than in the psoriasis group.[20] These differences among studies may result from diverse geographic, cultural, and healthcare system contexts, inclusion criteria, and distinctive characteristics of study populations.

The current study identified a significant association between escalating BMI and rising DAPSA scores. Leung et al documented that individuals with psoriatic arthritis and obesity exhibited a reduced likelihood of recovery or attaining low disease activity, estimating a 2.5- to 3-fold difference compared to their non-obese counterparts. Consequently, psoriatic arthritis patients with concomitant obesity may present diverse disease profiles necessitating tailored management strategies.[21] Klingberg et al also noted heightened psoriatic arthritis disease activity among obese individuals relative to non-obese counterparts.[22] Psoriatic arthritis, obesity, and atherosclerosis share chronic inflammation involving cytokines like TNF-α, IL23, IL-17, IL-6, and IL-1β. On the other hand, white adipose tissue is a key source of these cytokines and proinflammatory adipokines. Elevated levels of these substances may provoke autoimmune inflammation in psoriatic arthritis .[23-25]

We observed that the mean PASI score among patients was 5.2, and more patients had mild psoriasis. Also, the average DAPSA score was 13.15, and low psoriatic arthritis disease activity was more common among patients, in which both PASI and DAPSA scores were comparatively lower than those reported in prior investigations.[26,27] Kasiem et al reported that most patients with psoriatic arthritis experienced mild psoriasis during the first year of follow-up. Patients without psoriasis increased from 17% at baseline to 34% at the 12-month follow-up, while the number of patients with severe psoriasis remained consistent. Their findings indicate an overall improvement in psoriasis severity during the initial year of follow-up, albeit the majority still presented with mild psoriasis.[28]

Our results illustrated an association between ESR and CRP levels with PASI and DAPSA scores. Various studies reported similar findings, indicating a significant positive correlation between ESR and CRP levels with DAPSA.[29,30] Haroon et al reported that 56.5% of patients exhibited elevated CRP levels during their initial evaluation. Furthermore, 24% of patients never manifested elevated CRP levels during follow-up, suggesting mild disease severity .[31] Regarding treatment response to biologic DMARDs, Magee et al highlighted the clinical significance of CRP levels in psoriatic arthritis patients .[32] We found no statistically significant association between PASI and DAPSA scores with the type of medication in psoriatic arthritis patients. Notably, DAPSA scoring may be influenced by patient-reported disease activity, which can vary due to individual sensitivity. In a study by Gialouri et al CRP exhibited no correlation with disease-related parameters and patient-reported outcomes but with ESR alone.[33]

Our study findings revealed an association between RDW and PLT with PASI and DAPSA scores, while no significant association was observed between MPV and DAPSA scores. Nageen et al reported a significant correlation between PLT and PASI scores but not for MPV and RDW.[34] Previous studies showed that MPV was significantly associated with DAPSA score.[35-37] On the other hand, in a meta-analysis by Liu et al PASI scores showed a weak correlation with PLT and MPV.[38] Individuals with psoriasis exhibit elevated plasma levels of PLT chemokine beta-thromboglobulin compared to a control group that correlated with PASI scores and are notably reduced following successful treatment.[39] Furthermore, we found a significant association between NLR and both PASI and DAPSA scores. Furthermore, the PLR exhibited a significant relationship with DAPSA scores, while no statistically significant association was observed with PASI scores. Nguyen et al demonstrated a significant correlation between NLR and PASI scores in the psoriasis vulgaris group and between NLR and PLR with PASI scores in patients with psoriatic arthritis.[13] Moreover, Dongyun et al identified NLR and PLR as strong predictors of psoriatic arthritis.[40] Similarly, Albayrak observed significant decreases in NLR, neutrophil-to-monocyte ratio (NMR), PLR, and immune-inflammatory index in the third month of follow-up compared to baseline.[41]

According to the ROC curve analysis, ESR, CRP, and PLT indices demonstrated the highest predictive power for psoriatic arthritis disease activity, respectively. Kelesoglu Dincer and Sezar utilized DAS28-ESR, DAS28-CRP, and DAPSA to assess inflammatory parameters for evaluating psoriatic arthritis disease activity. They observed significant correlations between all 3 indicators of disease activity and NLR and PLR.[42] However, some studies have highlighted that while ESR and CRP are commonly employed as inflammation markers, they exhibit low sensitivity and specificity for diagnosing psoriatic arthritis, elevated in only 40% of psoriatic arthritis patients.[43,44] The presence of psoriatic arthritis complicates the overall management of psoriasis. Using biomarkers as complementary tools alongside clinical signs can assist in earlier and more accurate diagnosis of psoriatic arthritis for patients. Easy access to hematologic parameters in diagnosis, treatment, and monitoring, alongside clinical symptom assessment, can significantly aid clinical management in patients with psoriatic arthritis. While the current study comprehensively investigated the association between hematologic indices besides inflammatory markers with PASI and DAPSA scores among patients with psoriatic arthritis, it has limitations, Firstly, its findings may not be generalizable due to being conducted at a single center. Secondly, the small sample size limits the study's statistical power, resulting in small effect sizes for many hematologic indices, but lacking statistical significance. Thirdly, the limited sample size prevented the performance of multivariable (adjusted) analyses. Lastly, the cross-sectional design of the study restricts the ability to infer causal relationships between hematological indices and the disease activity of psoriatic arthritis.

The current study elucidated the relationship between blood indices and PASI and DAPSA scores in patients with psoriatic arthritis. Our findings underscore a notable positive correlation of indices such as ESR, CRP, and PLT with PASI and DAPSA scores. These results underscore the potential utility of blood indices as valuable markers for assessing and prognosticating the activity of psoriatic arthritis in conjunction with clinical symptoms.

STATEMENT OF ETHICS

The research protocol received approval from the Ethics Committee of Tehran University of Medical Sciences, Tehran, Iran (IR.NASRME.REC.1402.238) and conforms to the Declaration of Helsinki. All patients consented to participate in the study.

FUNDING

Not applicable.

CONFLICT OF INTEREST

The authors declare no conflicts of interest.

ACKNOWLEDGEMENTS

Not applicable.

Data Availability

Upon reasonable request from the corresponding author via email.

AI Assistance Disclosure

We used ChatGPT to improve the grammar and language of the manuscript.

REFERENCES

1. Harrison SR, Aung Din BNR, Marzo-Ortega H, Helliwell PS. Recent advances in the management of psoriatic arthritis: practical considerations. Polish Arch Intern Med. 2024;134(1).
2. Zhao SS, Marzo-Ortega H. Advances in psoriatic arthritis six decades on. Clin Ther. 2023;45(9):808-9.
3. Kishimoto M, Deshpande GA, Fukuoka K, Kawakami T, Ikegaya N, Kawashima S, et al. Clinical features of psoriatic arthritis. Best Pract Res Clin Rheumatol. 2021;35(2):101670.
4. Alinaghi F, Calov M, Kristensen LE, Gladman DD, Coates LC, Jullien D, et al. Prevalence of psoriatic arthritis in patients with psoriasis: A systematic review and meta-analysis of observational and clinical studies. J Am Acad Dermatol. 2019;80(1):251-65.e19.
5. Carvalho AL, Hedrich CM. The Molecular Pathophysiology of Psoriatic Arthritis—The Complex Interplay Between Genetic Predisposition, Epigenetics Factors, and the Microbiome [Internet]. Vol. 8, Frontiers in Molecular Biosciences. 2021.
6. Azuaga AB, Ramírez J, Cañete JD. Psoriatic Arthritis: Pathogenesis and Targeted Therapies. Int J Mol Sci. 2023;24(5).
7. Rida MA, Chandran V. Challenges in the clinical diagnosis of psoriatic arthritis. Clin Immunol. 2020;214:108390.
8. Amer AS, Al Shambaky AY, Ameen SG, Sobih AK. Hematological indices in psoriatic enthesopathy: relation to clinical and ultrasound evaluation. Clin Rheumatol. 2024 Jun;43(6):1909-1917. doi: 10.1007/s10067-024-06951-2. Epub 2024 Apr 8. PMID: 38584198; PMCID: PMC11111547. .
9. Korendowych E, Owen P, Ravindran J, Carmichael C, McHugh N. The clinical and genetic associations of anti-cyclic citrullinated peptide antibodies in psoriatic arthritis. Rheumatology (Oxford). 2005;44(8):1056–60.
10. Punzi L, Podswiadek M, Oliviero F, Lonigro A, Modesti V, Ramonda R, et al. Laboratory findings in psoriatic arthritis. Reumatismo. 2007;(59 Suppl 1):52–5.
11. Hackett S, Ogdie A, Coates LC. Psoriatic arthritis: prospects for the future. Ther Adv Musculoskelet Dis. 2022;14:1759720X221086710.
12. Conic RR, Damiani G, Schrom KP, Ramser AE, Zheng C, Xu R, et al. Psoriasis and Psoriatic Arthritis Cardiovascular Disease Endotypes Identified by Red Blood Cell Distribution Width and Mean Platelet Volume. J Clin Med. 2020;9(1).
13. Nguyen HT, Vo LDH, Pham NN. Neutrophil-to-lymphocyte and platelet-to-lymphocyte ratios as inflammatory markers in psoriasis: a case-control study. Dermatology reports. 2023;15(1):9516.
14. Soliman WM, Sherif NM, Ghanima IM, EL-Badawy MA. Neutrophil to lymphocyte and platelet to lymphocyte ratios in systemic lupus erythematosus: Relation with disease activity and lupus nephritis. Reumatol Clínica. 2020;16(4):255–61.

15. Taylor W, Gladman D, Helliwell P, Marchesoni A, Mease P, Mielants H. Classification criteria for psoriatic arthritis: development of new criteria from a large international study. Arthritis Rheum. 2006;54(8):2665–73.
16. Kerschbaumer A, Smolen JS, Aletaha D. Disease activity assessment in patients with psoriatic arthritis. Best Pract & Res Clin Rheumatol. 2018;32(3):401–14.
17. Smolen JS, Schoels M, Aletaha D, Schoels M, Aletaha D. DAPSA revisão. 2015;
18. Gisondi P, Geat D, Lippi G, Montagnana M, Girolomoni G. Increased red blood cell distribution width in patients with plaque psoriasis. J Med Biochem. 2021;40(2):199–201.
19. Ozisler C, Sandikci SC. Evaluation of red blood cell distribution width in patients with psoriatic arthritis. Egypt Rheumatol. 2020;42(4):309–12.
20. Kılıç S, Reşorlu H, Işık S, Oymak S, Akbal A, Hiz MM, et al. Association between mean platelet volume and disease severity in patients with psoriasis and psoriatic arthritis. Adv Dermatology Allergol Dermatologii i Alergol [Internet]. 2017;34(2):126–30.
21. Leung YY, Eder L, Orbai A-M, Coates LC, de Wit M, Smolen JS, et al. Association between obesity and likelihood of remission or low disease activity status in psoriatic arthritis applying index-based and patient-based definitions of remission: a cross-sectional study. RMD open. 2023;9(3).
22. Klingberg E, Bilberg A, Björkman S, Hedberg M, Jacobsson L, Forsblad-d'Elia H, et al. Weight loss improves disease activity in patients with psoriatic arthritis and obesity: an interventional study. Arthritis Res Ther. 2019;21(1):17.
23. Ivanov S, Merlin J, Lee MKS, Murphy AJ, Guinamard RR. Biology and function of adipose tissue macrophages, dendritic cells and B cells. Atherosclerosis. 2018;271:102–10.
24. Chehimi M, Vidal H, Eljaafari A. Pathogenic Role of IL-17-Producing Immune Cells in Obesity, and Related Inflammatory Diseases. J Clin Med. 2017;6(7).
25. Wolk K, Sabat R. Adipokines in psoriasis: An important link between skin inflammation and metabolic alterations. Rev Endocr Metab Disord. 2016;17(3):305–17.
26. Hägg D, Sundström A, Eriksson M, Schmitt-Egenolf M. Severity of Psoriasis Differs Between Men and Women: A Study of the Clinical Outcome Measure Psoriasis Area and Severity Index (PASI) in 5438 Swedish Register Patients. Am J Clin Dermatol. 2017;18(4):583–90.
27. Duruöz MT, Gezer HH, Nas K, Kılıç E, Sargın B, Kasman SA, et al. Gender-related differences in disease activity and clinical features in patients with peripheral psoriatic arthritis: A multi-center study. Jt Bone Spine. 2021;88(4):105177.
28. Kasiem FR, Kok MR, Luime JJ, Tchetverikov I, Wervers K, Korswagen L-A, et al. The burden of psoriasis in patients with early psoriatic arthritis. Rheumatology [Internet]. 2022;61(4):1570–8.
29. Mustafa TA, Esho MI. Association Between Mean Platelet Volume And Disease Activity In Patients With Psoriatic Arthritis. Iraqi J Pharm. 2022;19(2):46–54.
30. Izci Duran T, Pamukcu M. Relationship between disease impact scores and C-reactive protein/albumin ratio in patients with psoriatic arthritis. Croat Med J. 2022;63(2):141–7.
31. Haroon M, Gallaghar P, Ahmad M, FitzGerald O. Elevated CRP even at the first visit to a rheumatologist is associated with long-term poor outcomes in patients with psoriatic arthritis. Clin Rheumatol. 2020;39(10):2951–61.
32. Magee C, Jethwa H, FitzGerald OM, Jadon DR. Biomarkers predictive of treatment response in psoriasis and psoriatic arthritis: a systematic review. Ther Adv Musculoskelet Dis. 2021;13:1–14.
33. Gialouri CG, Evangelatos G, Pappa M, Karamanakos A, Iliopoulos A, Tektonidou MG, et al. Normal C-reactive protein in active psoriatic arthritis: results from real-world clinical practice. Ther Adv Musculoskelet Dis. 2022;14:1759720X221122417.
34. Nageen S, Shah R, Sharif S, Jamgochian M, Waqas N, Rao B. Platelet Count, Mean Platelet Volume, and Red Cell Distribution Width as Markers for Psoriasis Severity. J Drugs Dermatol. 2022 Feb;21(2):156–61.
35. Olumuyiwa-Akeredolu O-O, Page MJ, Soma P, Pretorius E. Platelets: emerging facilitators of cellular crosstalk in rheumatoid arthritis. Nat Rev Rheumatol. 2019;15(4):237–48.
36. Talukdar M, Barui G, Adhikari A, Karmakar R, Ghosh UC, Das TK. A Study on Association between Common Haematological Parameters and Disease Activity in Rheumatoid Arthritis. J Clin Diagn Res. 2017;11(1):EC01–4.
37. Tekeoğlu İ, Gürol G, Harman H, Karakeçe E, Çiftçi İH. Overlooked hematological markers of disease activity in rheumatoid arthritis. Int J Rheum Dis. 2016;19(11):1078–82.
38. Liu Z, Perry LA, Morgan V. The association between platelet indices and presence and severity of psoriasis: a

systematic review and meta-analysis. Clin Exp Med. 2023;23(2):333–46.

39. Tamagawa-Mineoka R, Katoh N, Ueda E, Masuda K, Kishimoto S. Elevated platelet activation in patients with atopic dermatitis and psoriasis: increased plasma levels of beta-thromboglobulin and platelet factor 4. Allergol Int Off J Japanese Soc Allergol. 2008;57(4):391–6.
40. Kim DS, Shin D, Lee MS, Kim HJ, Kim DY, Kim SM, et al. Assessments of neutrophil to lymphocyte ratio and platelet to lymphocyte ratio in Korean patients with psoriasis vulgaris and psoriatic arthritis. J Dermatol [Internet]. 2016;43(3):305–10.
41. Albayrak H. Neutrophil-to-Lymphocyte Ratio, Neutrophil-to-Monocyte Ratio, Platelet-to-Lymphocyte Ratio, and Systemic Immune-Inflammation Index in Psoriasis Patients: Response to Treatment with Biological Drugs. J Clin Med. 2023;12(17).
42. Bahar A, Dincer K, Sezer S. Systemic Immune Inflammation Index as a Reliable Disease Activity Marker in Psoriatic Arthritis. 2022;32(06):773–8.
43. Ritchlin CT, Colbert RA, Gladman DD. Psoriatic Arthritis. N Engl J Med. 2017 Mar;376(10):957–70.
44. Wu J, Yan L, Chai K. Systemic immune-inflammation index is associated with disease activity in patients with ankylosing spondylitis. J Clin Lab Anal. 2021;35(9):e23964.