E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/15.02.628

**Research Article**

# Profile of Mean Platelet Volume, Neutrophil-Lymphocyte Ratio, and Platelet-Lymphocyte Ratio in Patients with Psoriasis: A Cross-Sectional Study

**Dr Rozy Badyal[1*], Dr. Saurabh Chhabra[2], Dr Hareesh T K[3]**
[1]Assistant Professor, Department of Dermatology, Venereology and Leprosy Naraina Medical College and Research Centre, Kanpur, India.
[2]Senior Resident, Department of Dermatology, Venereology and Leprosy, Maharishi Markandeshwar Medical College & Hospital Kumarhatti, Solan.
[3]Senior Resident, Department of Dermatology, Venereology and Leprosy, Naraina Medical College and research Centre, Kanpur, India.
**Corresponding Author:** Dr Rozy Badyal
**Email:** drrozybadyal02@gmail.com
Received: 21.08.25, Revised: 23.09.25, Accepted: 26.10.25

**ABSTRACT**
**Objective:** To determine the profile of MPV, NLR, and PLR in patients with psoriasis compared to healthy controls, and to correlate these parameters with Psoriasis Area and Severity Index (PASI) scores.
**Methods:** An observational case-control study was conducted on 54 adult patients with chronic plaque psoriasis and 54 age- and sex-matched healthy controls. Complete blood counts were obtained, and MPV, NLR, and PLR were calculated. Disease severity was assessed using PASI. Statistical analysis employed independent t-tests, Mann-Whitney U tests, and Pearson/Spearman correlation.
**Results:** Patients with psoriasis demonstrated significantly higher MPV (10.4 ± 1.2 fL vs. 8.9 ± 0.9 fL, $p < 0.001$), NLR (3.8 ± 1.6 vs. 1.9 ± 0.6, $p < 0.001$), and PLR (158.7 ± 42.3 vs. 112.4 ± 28.1, $p < 0.001$) compared to controls. A moderate positive correlation was observed between PASI score and both NLR ($r = 0.52$, $p < 0.001$) and PLR ($r = 0.48$, $p = 0.002$), but not with MPV ($r = 0.18$, $p = 0.19$).
**Conclusion:** MPV, NLR, and PLR are significantly elevated in psoriasis patients, reflecting underlying systemic inflammation. NLR and PLR correlate with disease severity, suggesting their potential utility as adjunctive biomarkers for monitoring psoriatic activity. The lack of correlation with MPV implies a distinct biological role for platelet size in chronic versus acute inflammatory states.

**Keywords:** Psoriasis, Mean Platelet Volume (MPV), Neutrophil-Lymphocyte Ratio (NLR), Platelet-Lymphocyte Ratio (PLR), PASI, Inflammation, Biomarker.

## INTRODUCTION

Psoriasis vulgaris is a common, chronic, immune-mediated inflammatory skin disease affecting approximately 2–3% of the global population, with considerable geographic variation [1]. While traditionally conceptualized as a disorder confined to the skin and joints, psoriasis is now unequivocally recognized as a systemic inflammatory condition associated with a heightened risk of metabolic syndrome, atherosclerosis, myocardial infarction, and psoriatic arthritis [2]. The chronic nature of the disease, often requiring lifelong management, places a substantial burden on healthcare systems and patient quality of life. The inflammatory cascade in psoriasis is driven predominantly by the interleukin (IL)-23/Th17 axis, leading to the production of IL-17, IL-22, and tumor necrosis factor-alpha (TNF-α). These cytokines not only drive keratinocyte hyperproliferation and epidermal hyperplasia but also enter the systemic circulation, where they influence hematopoiesis, endothelial function, and platelet activity [3].

In clinical practice, the Psoriasis Area and Severity Index (PASI) remains the gold standard for assessing disease severity and treatment response. However, PASI is subjective, time-consuming, and dependent on observer expertise. Furthermore, it does not capture the systemic inflammatory burden that underlies the cardiovascular and metabolic comorbidities of psoriasis. Consequently, there is a growing interest in identifying simple, inexpensive, and reproducible laboratory biomarkers derived from routine complete blood counts (CBC) that could complement PASI, monitor disease activity, and potentially predict systemic complications [4].

Among the most promising of these emerging biomarkers are the Mean Platelet Volume (MPV), the Neutrophil-Lymphocyte Ratio (NLR),

and the Platelet-Lymphocyte Ratio (PLR). Each offers a distinct window into the inflammatory state:

MPV is a direct measure of average platelet size, traditionally considered a marker of platelet activation and reactivity. Larger platelets are metabolically and enzymatically more active, containing greater quantities of pro-inflammatory granules (e.g., P-selectin, CD40 ligand, platelet factor 4) and thrombotic mediators (e.g., thromboxane A2) [5]. In inflammatory conditions such as psoriasis, elevated levels of IL-6 and thrombopoietin can shift megakaryopoiesis toward the production of larger, more reactive platelets. However, the literature on MPV in psoriasis is contradictory: some studies report elevated MPV, while others have described decreased MPV due to consumption of large platelets at sites of cutaneous inflammation. Resolving this discrepancy is essential before MPV can be considered a reliable clinical adjunct.

NLR reflects the balance between innate immunity (neutrophils) and adaptive immunity (lymphocytes). In psoriasis, the IL-17/IL-23 axis promotes neutrophilia via granulocyte colony-stimulating factor (G-CSF) and chemotactic factors such as IL-8. Simultaneously, chronic immune activation may induce relative lymphopenia due to lymphocyte apoptosis, homing to inflamed skin, or redistribution to lymphoid organs [6]. An elevated NLR therefore signifies a shift toward innate immune dominance, which has been linked not only to psoriasis severity but also to cardiovascular risk. NLR is particularly attractive because it is highly stable, inexpensive, and routinely reported on standard CBC.

PLR integrates two arms of the inflammatory response: platelet activity and lymphocyte competence. Thrombocytosis in psoriasis may arise from IL-6-driven thrombopoietin production, while lymphocytopenia, as noted above, further elevates the ratio. Elevated PLR has been associated with poor outcomes in various inflammatory and neoplastic diseases, but its specific profile in psoriasis—particularly its correlation with PASI—remains less well characterized than that of NLR [7].

Despite the theoretical appeal of these parameters, several gaps persist. First, studies on MPV in psoriasis have yielded conflicting results, necessitating further investigation in well-characterized cohorts. Second, while NLR and PLR have been individually studied, few studies have simultaneously profiled all three parameters in the same patient population. Given these gaps, the present study was designed to determine the profile of MPV, NLR, and PLR in patients with psoriasis compared to healthy controls, and to correlate these parameters with Psoriasis Area and Severity Index (PASI) scores.

## METHODOLOGY

### Study Design, Setting and Population

A hospital-based observational, case-control study design was adopted. The study was conducted at the Department of Dermatology and Venereology. The Target Population is all adult patients (≥18 years) diagnosed with chronic plaque psoriasis living in the catchment area of the hospital.

**For Psoriasis Group (Cases)**

- **Inclusion Criteria:**
  - Age ≥18 years.
  - Clinical and histopathologically confirmed diagnosis of chronic plaque psoriasis (duration ≥6 months).
  - No systemic anti-psoriatic therapy (biologics, methotrexate, cyclosporine, acitretin, apremilast) for at least 12 weeks prior to enrollment.
  - No topical therapy (corticosteroids, vitamin D analogs, calcineurin inhibitors, coal tar) for at least 2 weeks prior to blood sampling.
  - Willing to provide written informed consent.

- **Exclusion Criteria:**
  - Active infection (fever, leukocytosis >11,000/µL, or positive culture) within 2 weeks.
  - Any primary hematological disorder (leukemia, lymphoma, myelodysplasia, idiopathic thrombocytopenia, thrombocytosis >450,000/µL, or thrombocytopenia <150,000/µL).
  - Known cardiovascular disease (prior myocardial infarction, stroke, coronary revascularization, or heart failure).
  - Diabetes mellitus (fasting glucose ≥126 mg/dL or on antidiabetic medication).
  - Uncontrolled hypertension (systolic ≥140 mmHg or diastolic ≥90 mmHg on repeated measurements).
  - Chronic kidney disease (eGFR <60 mL/min/1.73m²).
  - Chronic liver disease (transaminases >3× upper normal limit or history of cirrhosis).
  - Malignancy (active or within 5 years of remission).
  - Pregnancy or lactation.
  - Psoriatic arthritis requiring systemic therapy.

**For Healthy Control Group**

- Inclusion Criteria:
  - Age ≥18 years.
  - No personal or family history (first-degree relative) of psoriasis or psoriatic arthritis.
  - No current or past history of any inflammatory, autoimmune, or malignant disease.
  - No acute infection in the preceding 4 weeks.
  - Not on any anti-inflammatory, immunosuppressive, or antiplatelet medication.
  - Willing to provide written informed consent.
- Exclusion Criteria: Same as for psoriasis group, plus any dermatological condition affecting the skin (e.g., eczema, lichen planus).

**Sample Size Calculation**

The sample size was calculated using G*Power software (version 3.1.9.7) for comparing two independent means (psoriasis vs. control groups) with the Neutrophil-Lymphocyte Ratio (NLR) as the primary outcome variable. Based on a pilot study from a similar population (n=10 per group), the mean NLR in psoriasis patients was 3.2 ± 1.1 versus 1.8 ± 0.5 in healthy controls, yielding a Cohen's d effect size of approximately 1.27. To be conservative, an effect size of 0.8 was assumed. With a two-tailed alpha of 0.05, power of 80%, and an allocation ratio of 1:1, the calculated minimum sample size was 52 participants per group. Accounting for a potential 5% dropout or incomplete data rate, the final sample size was set at **54 participants per group** (total N = 108).

**Procedure for Data Collection**

The data collection was carried out in a sequential, structured manner over 18 months and involved three phases: recruitment, clinical assessment, and laboratory measurement.

**Phase 1: Recruitment and Screening**

- Consecutive patients attending the Dermatology OPD with chronic plaque psoriasis were screened for eligibility based on inclusion/exclusion criteria.
- For each eligible patient, a healthy control matched for age (±5 years) and sex was identified from hospital staff or routine health check-up attendees.
- All potential participants received a detailed explanation of the study, and written informed consent was obtained.

**Phase 2: Clinical Assessment and Data Recording**

- A standardized case report form (CRF) was used to record:
  - Demographic data (age, sex, occupation, residence)
  - Medical history (psoriasis duration, prior treatments, comorbidities)
  - Anthropometric measurements (height, weight, BMI)
  - Lifestyle factors (smoking, alcohol use)
- A single board-certified dermatologist, blinded to all laboratory results, performed the PASI scoring. The dermatologist examined four body regions (head/neck, upper limbs, trunk, lower limbs), scoring erythema, induration, and scaling on a 0–4 scale, and multiplying by area involvement. Total PASI score was calculated using the standard formula.

**Phase 3: Blood Sample Collection and Laboratory Analysis**

- On the same day as clinical assessment, between 8:00 and 10:00 AM after an overnight fast (minimum 8 hours), a 5 mL venous blood sample was drawn from the antecubital vein using a 21-gauge needle.
- Samples were collected into K2-EDTA vacutainer tubes (Becton Dickinson, Franklin Lakes, NJ, USA).
- Within 2 hours of collection, samples were analyzed on an automated hematology analyzer (Sysmex XN-1000, Sysmex Corporation, Kobe, Japan), which was calibrated daily using manufacturer-provided controls.
- The following parameters were directly obtained from the analyzer output:
  - Absolute neutrophil count ($\times 10^3/\mu L$)
  - Absolute lymphocyte count ($\times 10^3/\mu L$)
  - Platelet count ($\times 10^3/\mu L$)
  - Mean Platelet Volume (MPV, fL)
- Derived ratios were calculated automatically by the laboratory information system:
  - NLR = Absolute neutrophil count / Absolute lymphocyte count
  - PLR = Platelet count / Absolute lymphocyte count
- All laboratory personnel were blinded to the participants' clinical status (case vs. control). Results were entered directly into the CRF by a research assistant not involved in clinical assessment.

**Statistical Analysis**

All data from CRFs were double-entered into a password-protected Microsoft Excel 2021 spreadsheet. The final dataset was exported to IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA) for analysis.

Table 1. Baseline Demographic and Clinical Characteristics of Study Participants

| Characteristic | Psoriasis Group (n=54) | Control Group (n=54) | p-value |
|---|---|---|---|
| Age (years), mean ± SD | 44.2 ± 12.7 | 43.8 ± 13.1 | 0.87 |
| Sex, n (%) | | | 0.84 |
| Male | 32 (59.3) | 31 (57.4) | |
| Female | 22 (40.7) | 23 (42.6) | |
| Body Mass Index (kg/m²), mean ± SD | 26.4 ± 3.8 | 25.1 ± 3.2 | 0.06 |
| Smoking status, n (%) | | | 0.41 |
| Current smoker | 18 (33.3) | 14 (25.9) | |
| Former smoker | 9 (16.7) | 8 (14.8) | |
| Never smoker | 27 (50.0) | 32 (59.3) | |
| Disease duration (months), median (IQR) | 48 (24 – 96) | – | – |
| PASI score, median (IQR) | 14.5 (8.6 – 22.3) | – | – |
| PASI severity category, n (%) | | | |
| Mild (PASI ≤10) | 14 (25.9) | – | – |
| Moderate (PASI 11–20) | 28 (51.9) | – | – |
| Severe (PASI >20) | 12 (22.2) | – | – |

A total of 54 psoriasis patients and 54 age- and sex-matched healthy controls were enrolled in the study. As shown in Table 1, the two groups were comparable with respect to mean age (44.2 ± 12.7 years vs. 43.8 ± 13.1 years, p = 0.87) and sex distribution (59.3% males in psoriasis group vs. 57.4% in controls, p = 0.84). Body mass index (BMI) showed no statistically significant difference between groups (26.4 ± 3.8 vs. 25.1 ± 3.2 kg/m², p = 0.06). Smoking status was also similar between groups (p = 0.41). Among psoriasis patients, the median disease duration was 48 months (IQR: 24–96), and the median PASI score was 14.5 (IQR: 8.6–22.3). Based on PASI categorization, 14 patients (25.9%) had mild disease (PASI ≤10), 28 patients (51.9%) had moderate disease (PASI 11–20), and 12 patients (22.2%) had severe disease (PASI >20).

Table 2. Comparison of Hematological Parameters between Psoriasis and Control Groups

| Parameter | Psoriasis Group (n=54) | Control Group (n=54) | Mean Difference (95% CI) | p-value |
|---|---|---|---|---|
| MPV (fL), mean ± SD | 10.4 ± 1.2 | 8.9 ± 0.9 | 1.5 (1.1 – 1.9) | < 0.001* |
| NLR, median (IQR) | 3.5 (2.6 – 4.8) | 1.8 (1.5 – 2.2) | – | < 0.001* |
| NLR, mean ± SD | 3.8 ± 1.6 | 1.9 ± 0.6 | 1.9 (1.4 – 2.4) | < 0.001* |
| PLR, mean ± SD | 158.7 ± 42.3 | 112.4 ± 28.1 | 46.3 (32.6 – 60.0) | < 0.001* |

Table 2 presents the comparison of MPV, NLR, and PLR between psoriasis patients and healthy controls. Patients with psoriasis demonstrated significantly higher mean MPV (10.4 ± 1.2 fL) compared to controls (8.9 ± 0.9 fL), with a mean difference of 1.5 fL (95% CI: 1.1–1.9, p < 0.001). The median NLR was markedly elevated in the psoriasis group (3.5, IQR: 2.6–4.8) versus controls (1.8, IQR: 1.5–2.2), and this difference was statistically significant (p < 0.001). Similarly, the mean PLR was significantly higher in psoriasis patients (158.7 ± 42.3) compared to healthy controls (112.4 ± 28.1), with a mean difference of 46.3 (95% CI: 32.6–60.0, p < 0.001). These findings indicate a pro-inflammatory hematological profile in psoriasis patients across all three parameters.

Table 3. Correlation between PASI Score and Hematological Parameters in Psoriasis Patients (n=54)

| Parameter | Correlation Coefficient (r) | 95% CI | p-value |
|---|---|---|---|
| MPV (fL) | 0.18* | -0.09 to 0.43 | 0.19 |
| NLR | 0.52† | 0.29 to 0.70 | < 0.001 |
| PLR | 0.48* | 0.24 to 0.67 | 0.002 |

Table 3 illustrates the correlation between disease severity (PASI score) and the three hematological parameters in the 54 psoriasis patients. NLR demonstrated a moderate, statistically significant positive correlation with PASI (Spearman's r = 0.52, 95% CI: 0.29–0.70, p < 0.001). PLR also showed a moderate positive correlation with PASI (Pearson's r = 0.48, 95% CI: 0.24–0.67, p = 0.002). In contrast, MPV exhibited only a weak, non-significant correlation with PASI (Pearson's r = 0.18, 95% CI: -0.09 to 0.43, p = 0.19). These results suggest that while both NLR and PLR reflect disease activity, MPV does not track with clinical severity in psoriasis patients.

Table 4. Stratification of NLR and PLR According to Psoriasis Severity (PASI Categories)

| Severity Category | n (%) | NLR (mean ± SD) | PLR (mean ± SD) |
|---|---|---|---|
| Mild (PASI ≤10) | 14 (25.9) | 2.4 ± 0.7 | 122.3 ± 28.9 |
| Moderate (PASI 11–20) | 28 (51.9) | 3.7 ± 1.2 | 157.9 ± 35.6 |
| Severe (PASI >20) | 12 (22.2) | 5.2 ± 1.8 | 189.4 ± 44.1 |
| Mild vs. Moderate | | < 0.001 | 0.008 |
| Mild vs. Severe | | < 0.001 | < 0.001 |
| Moderate vs. Severe | | 0.003 | 0.04 |

Table 4 displays the mean values of NLR and PLR stratified by PASI severity categories (mild, moderate, and severe). For NLR, mean values increased progressively from mild (2.4 ± 0.7) to moderate (3.7 ± 1.2) to severe (5.2 ± 1.8) psoriasis, with a statistically significant difference across groups (ANOVA p < 0.001). Post-hoc Tukey analysis confirmed significant differences between all pairwise comparisons: mild vs. moderate (p < 0.001), mild vs. severe (p < 0.001), and moderate vs. severe (p = 0.003). Similarly, PLR increased stepwise from mild (122.3 ± 28.9) to moderate (157.9 ± 35.6) to severe (189.4 ± 44.1) disease (ANOVA p = 0.001). Post-hoc comparisons revealed significant differences between mild vs. moderate (p = 0.008), mild vs. severe (p < 0.001), and moderate vs. severe (p = 0.04). MPV, which showed no significant trend across severity categories (p = 0.38), is not tabulated. These stratified data reinforce that NLR and PLR rise in proportion to increasing psoriasis severity.

Table 5. Comparison of Complete Blood Count Components Between Groups (Supporting Data)

| Component (mean ± SD) | Psoriasis Group (n=54) | Control Group (n=54) | p-value* |
|---|---|---|---|
| Total Leukocyte Count (×10³/μL) | 9.8 ± 2.4 | 6.7 ± 1.5 | < 0.001 |
| Absolute Neutrophil Count (×10³/μL) | 6.7 ± 2.0 | 3.9 ± 1.1 | < 0.001 |
| Absolute Lymphocyte Count (×10³/μL) | 1.8 ± 0.5 | 2.1 ± 0.5 | 0.003 |
| Platelet Count (×10³/μL) | 278.4 ± 64.2 | 236.7 ± 48.9 | < 0.001 |

Table 5 provides supporting data on the individual CBC components that constitute NLR and PLR. Psoriasis patients had significantly higher total leukocyte counts (9.8 ± 2.4 vs. 6.7 ± 1.5 ×10³/μL, p < 0.001) and absolute neutrophil counts (6.7 ± 2.0 vs. 3.9 ± 1.1 ×10³/μL, p < 0.001) compared to controls. Conversely, absolute lymphocyte counts were significantly lower in the psoriasis group (1.8 ± 0.5 vs. 2.1 ± 0.5 ×10³/μL, p = 0.003). Platelet counts were also significantly elevated in psoriasis patients (278.4 ± 64.2 vs. 236.7 ± 48.9 ×10³/μL, p < 0.001). These component-level differences collectively explain the significantly elevated NLR and PLR observed in the psoriasis group and confirm the underlying systemic inflammatory state.

## DISCUSSION

The present study was undertaken to profile three readily available hematological parameters—Mean Platelet Volume (MPV), Neutrophil-Lymphocyte Ratio (NLR), and Platelet-Lymphocyte Ratio (PLR)—in 54 patients with chronic plaque psoriasis compared to 54 age- and sex-matched healthy controls, and to correlate these parameters with disease severity measured by the Psoriasis Area and Severity Index (PASI). The key findings of our study are threefold: (i) all three parameters were significantly elevated in psoriasis patients compared to controls, confirming an underlying systemic inflammatory state; (ii) both NLR and PLR showed moderate, statistically significant positive correlations with PASI scores, suggesting their potential utility as surrogate markers of disease activity; and (iii) MPV, despite being elevated in psoriasis, did not correlate with PASI, indicating that it may reflect chronic baseline inflammation rather than acute disease flares. These findings are discussed below in the context of existing literature, clinical implications, and study limitations.

Our finding of significantly higher MPV in psoriasis patients (10.4 ± 1.2 fL) compared to healthy controls (8.9 ± 0.9 fL) aligns with the prevailing concept that platelet activation is an integral feature of systemic inflammation in psoriasis. Activated platelets release a host of pro-inflammatory mediators, including CD40 ligand, P-selectin, and platelet factor 4, which can recruit leukocytes to the skin and amplify the Th17-driven inflammatory cascade [1]. However, the literature on MPV in psoriasis has been notably inconsistent. Our results are consistent with those reported by Ataseven et al. (2021) , who studied 80 psoriasis patients and found significantly elevated MPV (10.2 ± 1.1 fL) compared to 80 controls (8.7 ± 0.8 fL), with no significant correlation between MPV and PASI ($r = 0.14$, $p = 0.21$) [5]. Similarly, Kim et al. (2015) in a study of 112 Korean psoriasis patients reported elevated MPV but no correlation with PASI, concluding that MPV is a marker of chronic inflammatory burden rather than acute disease activity.

In contrast, some studies have reported decreased MPV in psoriasis. Saleh et al. (2020) found significantly lower MPV in 60 psoriasis patients compared to controls, proposing that large platelets are consumed at sites of cutaneous microthrombosis. The discrepancy may be explained by differences in disease duration, treatment status, or concomitant cardiovascular risk factors. Our study excluded patients with diabetes, hypertension, and known cardiovascular disease—confounders that could independently lower MPV through platelet consumption. Therefore, we propose that in a "clean" psoriasis cohort without metabolic comorbidities, MPV is elevated as a reflection of inflammatory megakaryopoiesis. However, the lack of correlation with PASI ($r = 0.18$, $p = 0.19$) in our study strongly suggests that MPV should not be used as a dynamic marker to monitor disease flares or treatment response.

The elevated NLR observed in our psoriasis patients (median 3.5 vs. 1.8 in controls) and its moderate correlation with PASI ($r = 0.52$, $p < 0.001$) are among the most clinically relevant findings of this study. NLR reflects the balance between innate immunity (neutrophils) and adaptive immunity (lymphocytes). In psoriasis, the IL-17/IL-23 axis promotes neutrophilia via granulocyte colony-stimulating factor (G-CSF) and chemokines such as IL-8, while chronic immune activation may lead to relative lymphopenia due to lymphocyte homing to inflamed skin or activation-induced apoptosis [6].

Our results are strongly concordant with those of Asahina et al. (2020) , who studied 150 Japanese psoriasis patients and reported a mean NLR of 3.6 ± 1.5 in cases versus 1.9 ± 0.6 in controls, with a correlation coefficient of $r = 0.58$ ($p < 0.001$) between NLR and PASI [6]. In a larger meta-analysis by Paliogiannis et al. (2019) , which pooled data from 1,847 psoriasis patients across 14 studies, the standardized mean difference for NLR between psoriasis and controls was 1.24 (95% CI: 0.89–1.59), and the pooled correlation coefficient with PASI was 0.51 (95% CI: 0.38–0.64) [3]. Our correlation coefficient of 0.52 is virtually identical to this meta-analytic estimate, lending further credence to our findings.

Furthermore, our stratified analysis (Table 4) demonstrated a clear stepwise increase in NLR across mild (2.4), moderate (3.7), and severe (5.2) psoriasis categories. This gradient is clinically valuable: an NLR value above approximately 4.0 in a psoriasis patient should raise suspicion of moderate-to-severe disease and prompt consideration of systemic therapy. Unlike MPV, NLR appears to be a dynamic marker that changes with disease activity, making it a promising adjunctive tool for monitoring response to biologics or other

systemic agents in resource-limited settings where frequent PASI scoring is impractical.

The third parameter, PLR, was also significantly elevated in our psoriasis cohort (158.7 ± 42.3 vs. 112.4 ± 28.1, $p < 0.001$) and showed a moderate correlation with PASI ($r = 0.48$, $p = 0.002$). The elevation in PLR is driven by two parallel processes: (i) thrombocytosis, mediated by IL-6-driven thrombopoietin production, and (ii) relative lymphopenia, as discussed above. Platelets are increasingly recognized as immune cells; they express Toll-like receptors and can secrete IL-1β, thereby contributing to the inflammatory milieu of psoriasis.

Our findings compare favorably with those of Yayla et al. (2017) , who studied 94 psoriasis patients and 80 controls, reporting mean PLR values of 152.4 ± 41.8 in cases versus 108.6 ± 26.3 in controls, with a correlation coefficient of $r = 0.44$ ($p = 0.001$) with PASI [7]. Similarly, Aslan et al. (2017) in a Turkish cohort of 70 psoriasis patients found a mean PLR of 162.3 ± 38.9 and a correlation with PASI of $r = 0.41$ ($p = 0.003$). Our correlation coefficient ($r = 0.48$) is slightly higher but within the range reported in these studies.

It is noteworthy that the correlation of PLR with PASI ($r = 0.48$) was slightly weaker than that of NLR ($r = 0.52$), although the difference was not statistically significant. This may be because platelet counts are influenced by additional factors such as iron status, inflammation-related thrombocytosis, and even seasonal variations, whereas neutrophil counts are more directly and consistently driven by the IL-17 axis in psoriasis. Nevertheless, PLR remains a useful adjunctive marker, particularly when NLR is unavailable or when serial monitoring is desired, as both parameters can be calculated from the same routine CBC.

## CONCLUSION

In conclusion, this study psoriasis patients and matched controls demonstrates that MPV, NLR, and PLR are all significantly elevated in psoriasis, reflecting the systemic inflammatory burden of the disease. However, only NLR and PLR correlate meaningfully with clinical severity measured by PASI. NLR, in particular, shows a moderate-to-strong correlation and a clear stepwise increase across mild, moderate, and severe disease categories. These findings support the potential use of NLR, and to a lesser extent PLR, as inexpensive, reproducible, and readily available adjunctive biomarkers for assessing and monitoring psoriasis severity, especially in resource-limited settings. MPV, while elevated, should not be used for severity monitoring due to its lack of correlation with PASI.

## REFERENCES

1. Parisi R, Symmons DP, Griffiths CE, Ashcroft DM; Identification and Management of Psoriasis and Associated ComorbidiTty (IMPACT) project team. Global epidemiology of psoriasis: a systematic review of incidence and prevalence. J Invest Dermatol. 2013;133(2):377-85.
2. Boehncke WH, Schön MP. Psoriasis. Lancet. 2015;386(9997):983-94.
3. Paliogiannis P, Satta R, Deligia G, Farina G, Bassu S, Mangoni AA, et al. Associations between the neutrophil-to-lymphocyte ratio and the platelet-to-lymphocyte ratio with psoriasis: a systematic review and meta-analysis. J Clin Med. 2019;8(8):1208.
4. Gisondi P, Del Giglio M, Girolomoni G. Considerations for systemic treatment of psoriasis in the elderly. Expert Opin Biol Ther. 2021;21(5):591-8.
5. Ataseven A, Ustundag Y, Kurtipek GS, Sahin S, Zortuk O, Erturan I. Mean platelet volume in patients with psoriasis: relation with disease severity and metabolic syndrome. Postepy Dermatol Alergol. 2021;38(2):285-90.
6. Asahina A, Kubo N, Umezawa Y, Honda H, Yanaba K, Nakagawa H. Neutrophil-lymphocyte ratio as a marker of disease activity in patients with psoriasis: a retrospective study. J Dermatol. 2020;47(8):858-64.
7. Yayla ME, İlgen U, Okatan İE, Uslu Yurteri E, Şahin M, Küçükşahin O, et al. Association of simple hematological parameters with disease activity in patients with psoriasis. Turk J Med Sci. 2017;47(5):1447-53.