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**Original Research Article**

# Evaluation of potential inflammatory biomarkers of disease severity in psoriasis: a case control study

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## ABSTRACT

**Background:** Psoriasis is a chronic inflammatory skin disorder affecting approximately 2-4% of the global population, characterized by immune dysregulation and keratinocyte hyperproliferation. Although multiple investigations have identified potential biomarkers, no validated panel exists for assessing disease severity. Objectives of the study were to determine levels of serum uric acid (SUA), C-reactive protein (CRP), erythrocyte sedimentation rate (ESR), CRP/albumin ratio (CAR), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) in psoriasis patients versus controls, and assess their correlation with disease severity using psoriasis area severity index (PASI).
**Methods:** This case-control study included 50 psoriasis patients and 50 age- and sex-matched controls. Participants underwent complete blood count, CRP, ESR, serum albumin, and SUA measurements. Disease severity was categorized as mild (PASI <7), moderate (PASI 7-15), or severe (PASI >15).
**Results:** ESR was significantly elevated in cases versus controls (33.24±22.75 versus 19.44±11.50 mm/hour, p<0.001). CAR distribution differed significantly (p<0.001), with 56% of cases having CAR ≤0.1 mg/l versus 2% of controls. SUA showed no overall difference between groups (p=0.468) but significantly correlated with disease severity (p=0.028), increasing from mild (4.76±0.97 mg/dl) to moderate (5.47±1.50 mg/dl) to severe disease (5.77±1.01 mg/dl). CRP, albumin, NLR, and PLR showed no significant differences.
**Conclusions:** ESR and CAR significantly differentiate psoriasis patients from controls. SUA, while not discriminating between cases and controls, correlates with disease severity. These cost-effective biomarkers can complement clinical assessment in psoriasis management.

**Keywords:** Psoriasis, Biomarkers, Disease severity

## INTRODUCTION

Psoriasis is a chronic, immune-mediated inflammatory skin disease that affects approximately 2-4% of the global population, representing one of the most prevalent autoimmune conditions worldwide.[1] This multifactorial disorder is characterized by well- demarcated, erythematous, scaly plaques that typically appear on extensor surfaces, scalp, and other frequently traumatized areas of the body. Beyond its dermatological manifestations, psoriasis has emerged as a systemic inflammatory condition associated with numerous comorbidities, including psoriatic arthritis, cardiovascular disease, metabolic syndrome, and psychological disorders, significantly impacting patients' quality of life and overall health outcomes.[2]

The pathophysiology of psoriasis involves a sophisticated network of inflammatory cascades, with the interleukin (IL)-23/IL-17 axis playing a central role in disease initiation and perpetuation.[3,4]

The clinical assessment of psoriasis severity traditionally relies on scoring systems such as the psoriasis area and severity index (PASI), which evaluates the extent of skin involvement and the intensity of erythema, induration, and desquamation across different body regions.[6] However, these clinical measures have inherent limitations, including inter-observer variability, subjective interpretation, and the inability to capture the underlying molecular and biochemical processes driving disease progression. Furthermore, clinical assessments may not accurately reflect the systemic inflammatory burden associated with psoriasis, which is crucial for understanding disease severity and predicting long-term outcomes.[7] The search for reliable biomarkers in psoriasis has gained significant momentum with advances in molecular biology and high-throughput analytical techniques. Biomarkers represent measurable biological characteristics that can serve as indicators of normal physiological processes, pathogenic mechanisms, or therapeutic responses.[8] Recent research has identified several categories of potential biomarkers in psoriasis, including inflammatory mediators such as C-reactive protein (CRP), acute-phase reactants, and the neutrophil-to-lymphocyte ratio, which has emerged as a simple yet effective biomarker reflecting systemic inflammation.[9,10] Serum uric acid levels have also been investigated as potential biomarkers, with hyperuricemia proposed to enhance oxidative stress and inflammation, potentially promoting atherosclerosis.[11]

The present study aims to address some of these knowledge gaps by conducting a comprehensive case-control investigation of potential biomarkers of disease severity in psoriasis. By employing a systematic approach to evaluate multiple biomarker categories, including inflammatory mediators (ESR, CRP, CRP/albumin ratio), hematological ratios (NLR, PLR), and metabolic markers (serum uric acid), this research seeks to identify reliable, objective measures that correlate with clinical disease severity and could potentially serve as valuable tools for disease monitoring and management.

## METHODS

This study was approved by the institutional ethics committee (Approval no: RRMCH-IEC/42/2024; Date of Approval:11/03/2024). Written informed consent was obtained from each participant willing to be included in the study.

### *Study design and population*

This case-control study was conducted in the Department of Dermatology, Venereology and Leprosy, Raja Rajeswari Medical College and Hospital, Bangalore, India from March 2024 to September 2025. Based on the Yamane formula, 50 patients clinically diagnosed with psoriasis and 50 age- and sex-matched controls were enrolled. Cases included patients of both sexes, aged 18-65 years, with clinically diagnosed psoriasis who provided written informed consent.

Controls were individuals attending the outpatient department for health check-ups, matched for age and sex, with no clinical signs of psoriasis. Exclusion criteria for cases included patients currently on or who had received topical and systemic therapy in the past month, those receiving corticosteroids, allopurinol, or thiazide-type diuretics, patients with diabetes mellitus, hypertension, cardiovascular diseases, active infection, malignancy, liver or renal diseases, and pregnant or lactating women. Controls with any known underlying disease were excluded.

### *Clinical assessment and laboratory investigations*

After obtaining informed consent, all participants underwent comprehensive evaluation including detailed history taking, physical examination, and demographic documentation. For psoriasis patients, thorough dermatological examination was performed and disease severity was assessed using the psoriasis area and severity index (PASI) score. Patients were categorized as mild (PASI <7), moderate (PASI 7-15), or severe (PASI >15). Ten milliliters of venous blood were collected from each participant after overnight fasting under aseptic conditions. Laboratory parameters measured included complete blood count (white blood cell count, neutrophil count, lymphocyte count, platelet count), erythrocyte sedimentation rate (ESR), CRP, serum albumin, and serum uric acid (SUA). The CRP to albumin ratio (CAR), neutrophil to lymphocyte ratio (NLR), and platelet to lymphocyte ratio (PLR) were calculated from obtained value.

### *Statistical analysis*

Data were entered into Microsoft excel and analyzed using statistical package for the social sciences (SPSS) version 23. Quantitative data were presented as mean±standard deviation, while categorical data were expressed as frequencies and percentages. Chi-square tests examined associations between categorical variables. Independent t-tests were employed for normally distributed continuous variables. Pearson's correlation coefficient assessed relationships between biomarkers and PASI scores. Statistical significance was set at $p<0.05$.

## RESULTS

The study included 50 psoriasis cases (48% females, 52% males) with mean age 42.10±11.72 years and 50 controls (48% females, 52% males) with mean age 35.12±11.66 years. The demographic characteristics of the study population are summarized in tables 1 and 2. Disease severity distribution showed 30 patients (60%) with mild psoriasis (PASI <7), 10 patients (20%) with moderate psoriasis (PASI 7-15), and 10 patients (20%) with severe psoriasis (PASI >15).

Table 1 shows the age distribution of psoriasis cases and controls. The majority of psoriasis patients (50.0%) belonged to the 41-60 years age group, whereas most controls (70.0%) were in the 21-40 years age group. The age distribution differed significantly between the two groups (p=0.029). In addition, the mean age of cases was significantly higher than that of controls (42.10±11.72 years vs. 35.12±11.66 years; p=0.004).

Table 2 shows the sex distribution of the study population. Both the case and control groups had identical sex distribution, with 26 (52.0%) males and 24 (48.0%) females in each group (p=1.0), confirming successful matching of cases and controls by sex.

Table 3 compares the inflammatory biomarkers between psoriasis patients and controls. ESR was significantly higher among psoriasis patients than controls (33.24±22.75 versus 19.44±11.50 mm/hour; p<0.001). The distribution of CRP/albumin ratio (CAR) categories also differed significantly between the two groups (p<0.001). However, no statistically significant differences were observed for CRP, serum albumin, neutrophil count, lymphocyte count, platelet count, NLR, PLR, or serum uric acid.

Table 4 demonstrates the association between biomarkers and psoriasis severity based on PASI categories. Serum uric acid showed a significant positive association with disease severity, with mean values increasing from 4.76±0.97 mg/dl in mild psoriasis to 5.47±1.50 mg/dl in moderate psoriasis and 5.77±1.01 mg/dl in severe psoriasis (p=0.028). No statistically significant association was observed between disease severity and the remaining biomarkers.

**Table 1: Baseline demographic characteristics of cases and controls- age category distribution by groups.**

| Age category (years) | Cases, N (%) | Controls, N (%) | P value |
|---|---|---|---|
| **≤20** | 1 (2.0) | 3 (6.0) | 0.029* |
| **21-40** | 23 (46.0) | 35 (70.0) | |
| **41-60** | 25 (50.0) | 12 (24.0) | |
| **>60** | 1 (2.0) | 0 (0.0) | |
| **Total** | 50 (100.0) | 50 (100.0) | |
| **Mean±SD** | 42.10±11.72 | 35.12±11.66 | 0.004* |

*P value statistically significant

**Table 2: Baseline demographic characteristics of cases and controls- sex category distribution by groups.**

| Sex | Cases, N (%) | Controls, N (%) | P value |
|---|---|---|---|
| **Female** | 24 (48.0) | 24 (48.0) | 1.0 |
| **Male** | 26 (52.0) | 26 (52.0) | |
| **Total** | 50 (100.0) | 50 (100.0) | |

**Table 3: Comparison of biomarkers between cases and controls.**

| Variables | Cases (n=50), mean±SD | Controls, mean±SD | P value |
|---|---|---|---|
| **CRP (mg/dl)** | 3.00±11.45 | 5.94±4.64 | 0.096 |
| **Albumin (g/dl)** | 4.24±0.41 | 4.11±0.57 | 0.185 |
| **Neutrophil (%)** | 57.92±10.37 | 60.32±12.65 | 0.302 |
| **Lymphocyte (%)** | 29.78±8.23 | 31.78±11.81 | 0.328 |
| **Platelet (lakhs/cumm)** | 2.94±0.66 | 2.73±0.80 | 0.148 |
| **ESR (mm/hour)** | 33.24±22.75 | 19.44±11.50 | <0.001* |
| **CRP/albumin ratio** | 0.92±2.88 | 1.39±1.14 | 0.282 |
| **Neutrophil/lymphocyte ratio** | 2.31±1.48 | 2.41±1.69 | 0.740 |
| **Platelet/lymphocyte ratio** | 10.83±5.05 | 10.10±6.03 | 0.514 |
| **CAR category, N (%)** | | | |
| 1. ≤0.1 mg/l | 28 (56.0) | 1 (2.0) | <0.001* |
| 2. 0.31-1.0 mg/l | 0 (0.0) | 23 (46.0) | <0.001* |
| 3. >1.0 mg/l | 22 (44.0) | 26 (52.0) | <0.001* |
| Serum uric acid (mg/dl) | 5.10±1.16 | 5.26±1.00 | 0.468 |

*P<0.05 statistically significant; CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, SD: standard deviation

**Table 4: Association of biomarkers with disease severity (PASI categories).**

| Biomarkers | Mild (<7), n=30 mean±SD | Moderate (7-15), n=10 mean±SD | Severe (>15), n=10 mean±SD | P value |
|---|---|---|---|---|
| **CRP/albumin ratio** | 0.67±3.02 | 1.97±2.63 | 0.60±2.70 | 0.440 |
| **Platelet/lymphocyte ratio** | 10.01±4.17 | 11.11±2.78 | 13.01±8.21 | 0.264 |
| **Neutrophil/lymphocyte ratio** | 2.06±1.14 | 2.19±0.65 | 3.17±2.50 | 0.118 |
| **CRP (mg/dl)** | 1.40±11.33 | 8.40±11.38 | 2.40±11.38 | 0.246 |
| **Albumin (g/dl)** | 4.14±0.44 | 4.43±0.35 | 4.33±0.27 | 0.114 |
| **Neutrophils (%)** | 55.60±10.47 | 60.50±6.95 | 62.30±11.70 | 0.142 |
| **Lymphocytes (%)** | 31.43±8.42 | 28.90±4.84 | 25.70±9.38 | 0.151 |
| **Platelets (lakhs/cumm)** | 2.90±0.64 | 3.12±0.56 | 2.87±0.80 | 0.612 |
| **ESR (mm/hour)** | 31.87±22.21 | 34.30±26.48 | 36.30±22.58 | 0.800 |
| **Serum uric acid (mg/dl)** | 4.76±0.97 | 5.47±1.50 | 5.77±1.01 | 0.028* |

*P<0.05 statistically significant; PASI: psoriasis area severity index, SD: standard deviation CRP: C-reactive protein, ESR: erythrocyte sedimentation rate, SD: standard deviation

**Table 5: Comparison of significant biomarkers in psoriasis across previous studies and the present study.**

| Study | ESR | CRP | CAR | (SUA) | NLR | PLR |
|---|---|---|---|---|---|---|
| **Kemeriz et al, 2020[28]** | ↑ in cases; correlated with PASI (p<0.05) | ↑ in cases (p>0.05) | Significantly ↑ strong PASI correlation (p<0.001) | NS (p>0.05) | NS (p>0.05) | NS (p>0.05) |
| **Solak et al, 2016[29]** | NS (p>0.05) | ↑ in cases (5.9±5.3 versus 4.0±1.5 mg/l; p<0.001) | Not assessed | ↑ in cases (5±1.3 versus 4.3±1.1 mg/dl; p =0.001); no PASI correlation | ↑ in cases (2.19±0.81 versus 1.74±0,56; p<0.001) with PASI | NS |
| **Mishra et al, 2022[30]** | Not assessed | NS | Not assessed | ↑ in cases (5.28±1.20 versus 4.65±0.98 mg/dl; p<0.01); independent PASI predictor (p<0.05) | NS (p>0.05) | Not assessed |
| **Present study** | ↑ in cases (33.24±22.75 versus 19.44±11.50 mm/hour; p<0.001) | NS (p=0.096) | Significant distribution difference (p<0.001); no PASI correlation | NS between cases/ controls (p=0.468); ↑ PASI severity (p=0.028) | NS (p=0.740); trend with severity | NS (p=0.514) |

↑ indicates statistically significant increase in psoriasis patients and/ or disease severity, NS=not statistically significant

## DISCUSSION

The present case-control study systematically evaluated multiple potential biomarkers in 50 psoriasis patients compared to 50 age- and sex-matched healthy controls, yielding several important findings that contribute to understanding psoriasis as a systemic inflammatory disorder. The age distribution showing higher mean age in psoriasis patients (42.10 years) compared to controls (35.12 years) reflects the epidemiological pattern of psoriasis with bimodal peaks of onset, the second occurring in the fifth to sixth decade of life, consistent with large epidemiological studies.[13] The sex distribution with slight male preponderance (1.08:1) is comparable to other Indian studies.[14] ESR emerged as the most significant discriminatory marker, with marked elevation in psoriasis patients versus controls (p<0.001), As mentioned in table 3.[15,16] These findings are comparable to those reported by Kemeriz et al, who also demonstrated significantly elevated ESR levels in psoriasis patients compared to controls; however, similar to our study, ESR did not consistently correlate with PASI scores, suggesting that ESR reflects systemic inflammatory burden rather than cutaneous disease severity alone.

The CRP/albumin ratio demonstrated a highly distinctive distribution pattern significantly differentiating cases from controls (p<0.001), As mentioned in Table 3. This novel finding suggests CAR may capture aspects of the inflammatory-metabolic profile not reflected by individual parameters alone. Recent studies have highlighted CAR as a prognostic marker in various inflammatory conditions.[17-

[19] While CRP alone showed no significant differences in our study (Table 3), contrasting with numerous reports of elevated CRP in psoriasis.[20]

In contrast, Kemeriz et al reported significantly elevated CRP levels, reduced albumin levels, and markedly increased CAR values in psoriasis patients, with CAR showing the strongest correlation with PASI scores.[27] The weaker association observed in our study may be attributable to the predominance of mild disease and lower overall inflammatory burden in our cohort compared to the higher proportion of moderate-to-severe cases included in other study.

Serum uric acid, while showing no significant difference between cases and controls overall (p=0.468), demonstrated crucial significant association with disease severity (p=0.028), progressively increasing from mild to severe disease. Similar findings have been reported by Mishra et al, who identified serum uric acid as the only biomarker independently associated with PASI scores despite no significant difference between cases and controls. In contrast, Solak et al demonstrated significantly higher serum uric acid levels in psoriasis patients compared to controls but did not observe an independent association with disease severity.[28] These differences suggest that while hyperuricemia is common in psoriasis, its relationship with disease severity may vary depending on population characteristics, metabolic profile, and disease distribution.[21,22] From a clinical perspective, monitoring uric acid may identify patients at higher risk for metabolic complications, as hyperuricemia associated with metabolic syndrome, cardiovascular disease, and gout-comorbidities more prevalent in psoriasis patients.[23]

The NLR and PLR showed no statistically significant differences between cases and controls in this study, though trends toward higher NLR in severe disease were observed. While psoriasis involves immune dysregulation, absolute cell counts may remain within ranges not producing significantly altered ratios compared to healthy individuals.[24,25] Though a meta-analysis showed an association with presence but not disease severity.[26] Our findings align more closely with those of Mishra et al, suggesting that NLR may not serve as a consistent marker of disease severity across all psoriasis populations.[29]

Table 5 summarizes the comparison of the present study findings with previously published studies. The findings of the present study are largely comparable with earlier studies, particularly regarding the significant association of ESR and CRP/albumin ratio with psoriasis and the association of serum uric acid with disease severity, while NLR and PLR did not demonstrate statistically significant differences.

### *Limitations*

The study has limitations including cross-sectional design that cannot establish temporal relationships, sample size that may be underpowered for subgroup analyses, predominantly mild disease limiting generalizability to severe cases, and lack of accounting for disease duration or treatment history. Future studies should include larger cohorts with balanced representation across all severity grades and incorporate additional biomarkers including specific cytokines and metabolic parameters.

## CONCLUSION

This study confirms psoriasis as a systemic inflammatory disorder with measurable biomarker alterations. ESR emerges as a significant, readily available marker significantly elevated in psoriasis patients that could be incorporated into routine assessment. Serum uric acid, while not distinguishing cases from controls, demonstrates significant correlation with disease severity, serving as a valuable objective marker to complement clinical assessment and identify patients at risk for metabolic complications. The CRP/albumin ratio shows a distinctive distribution pattern warranting further investigation as a potential novel biomarker. These cost-effective biomarkers can complement clinical evaluation in psoriasis management. Future research should focus on validating these biomarkers in larger cohorts, establishing their value for predicting treatment response and long-term outcomes, and exploring relationships to psoriasis-associated comorbidities to develop evidence-based algorithms incorporating biomarkers into clinical decision-making for optimal patient care.

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