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Original Research Article

Evaluation of Serum Inflammatory Biomarkers and Disease Severity Correlation in Patients with Chronic Plaque Psoriasis: A Cross-Sectional Study

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Abstract

Background: Chronic plaque psoriasis is a systemic immune-mediated inflammatory dermatosis characterized by dysregulated cytokine networks and persistent cutaneous inflammation. Serum inflammatory biomarkers have emerged as potential objective indicators of disease activity, yet their precise correlation with clinical severity indices remains incompletely characterized.

Methods: This cross-sectional study enrolled 120 patients with chronic plaque psoriasis and 60 age- and sex-matched healthy controls from the dermatology outpatient department of a tertiary care hospital. Patients were stratified into mild (PASI <7), moderate (PASI 7–12), and severe (PASI >12) groups. Serum biomarker levels were quantified using enzyme-linked immunosorbent assay (ELISA). Correlations between biomarker levels and PASI scores were analyzed using Spearman's rank correlation coefficient.

Results: Mean serum levels of CRP (14.82 ± 6.41 mg/L vs. 2.14 ± 1.03 mg/L; $p<0.001$), IL-6 (28.67 ± 11.54 pg/mL vs. 5.23 ± 2.18 pg/mL; $p<0.001$), TNF-α (45.39 ± 18.72 pg/mL vs. 8.91 ± 3.44 pg/mL; $p<0.001$), and IL-17A (38.56 ± 14.83 pg/mL vs. 6.47 ± 2.76 pg/mL; $p<0.001$) were significantly elevated in psoriasis patients compared to controls. All biomarkers demonstrated significant positive correlations with PASI scores, with IL-17A exhibiting the strongest correlation ($r=0.784$, $p<0.001$).

Conclusion: Serum inflammatory biomarkers are significantly elevated in chronic plaque psoriasis and correlate positively with disease severity. IL-17A demonstrated the strongest association with PASI, supporting its utility as a reliable serological indicator of disease activity and therapeutic monitoring.

Keywords: Psoriasis; inflammatory biomarkers; interleukin-17A; PASI; C-reactive protein; disease severity.
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Introduction

Psoriasis is a chronic, relapsing, immune-mediated inflammatory skin disease affecting approximately 2–3% of the global population, with chronic plaque psoriasis representing the most prevalent clinical subtype, accounting for nearly 80–90% of all cases [1]. The disease is characterized by well-demarcated, erythematous, scaly plaques resulting from epidermal hyperproliferation, abnormal keratinocyte differentiation, and extensive dermal inflammatory infiltration [2].

Beyond its cutaneous manifestations, psoriasis is increasingly recognized as a systemic inflammatory disorder associated with significant comorbidities, including cardiovascular disease, metabolic syndrome, psoriatic arthritis, and psychological morbidity [3]. The immunopathogenesis of psoriasis involves a complex interplay between innate and adaptive immune responses, with the interleukin-23/interleukin-17 (IL-23/IL-17) axis occupying a central role in disease initiation and perpetuation [4]. T-helper 17 (Th17) cells, upon activation by IL-23, produce proinflammatory cytokines including IL-17A, IL-17F, and IL-22, which drive keratinocyte proliferation and amplify the inflammatory cascade [5]. Additionally, tumor necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6) serve as critical mediators linking local cutaneous inflammation to systemic immune dysregulation [6].

The Psoriasis Area and Severity Index (PASI) remains the gold standard clinical tool for assessing disease severity; however, it is inherently subjective and demonstrates significant inter-observer variability [7]. Consequently, there has been growing interest in identifying objective serological biomarkers that can reliably reflect disease activity, predict treatment response, and facilitate personalized therapeutic strategies [8]. C-reactive protein (CRP), an acute-phase reactant synthesized hepatically in response to IL-6 stimulation, has been extensively investigated as a systemic inflammatory marker in psoriasis, with several studies reporting elevated levels correlating with disease severity [9].

Recent investigations have underscored the significance of serum IL-17A as a particularly promising biomarker, given its pivotal pathogenic role in psoriatic inflammation [10]. However, studies simultaneously evaluating multiple inflammatory biomarkers and their comparative correlative strength with standardized severity indices remain limited, particularly in diverse clinical populations [11]. Furthermore, discrepancies in reported findings across different geographic and ethnic cohorts necessitate additional investigation to establish robust biomarker-severity associations [12].

The present study aimed to evaluate serum levels of CRP, IL-6, TNF-α, and IL-17A in patients with chronic plaque psoriasis, compare these levels with healthy controls, and determine the correlation between each biomarker and disease severity as measured by PASI scores.

Materials and Methods

Study Design and Setting: This cross-sectional, observational study was conducted at the Department of Dermatology, Venereology, and Leprology of a tertiary care teaching hospital.

Study Participants: A total of 120 patients diagnosed with chronic plaque psoriasis and 60 age- and sex-matched healthy volunteers (controls) were recruited. The sample size was calculated based on a previous pilot study estimating a correlation coefficient of 0.30 between serum IL-17A and PASI, with 80% power and a 5% significance level, yielding a minimum requirement of 84 patients. To account for potential dropouts and subgroup analyses, enrollment was expanded to 120 patients.

Inclusion and Exclusion Criteria

Inclusion Criteria: Patients aged 18–65 years with clinically and/or histopathologically confirmed chronic plaque psoriasis of at least six months duration were included.

Exclusion Criteria: Patients with erythrodermic, pustular, or guttate psoriasis variants; those receiving systemic immunosuppressive therapy, biologics, or phototherapy within the preceding three months; patients with concurrent autoimmune diseases, active infections, malignancies, hepatic or renal insufficiency, pregnancy, or lactation were excluded. Controls with any history of chronic inflammatory conditions, autoimmune disorders, or ongoing anti-inflammatory medication use were similarly excluded.

Clinical Assessment: All patients underwent comprehensive dermatological examination by a single experienced dermatologist to minimize inter-observer variability. Disease severity was assessed using the PASI score (range: 0–72). Patients were stratified into three severity groups: mild (PASI <7; n=38), moderate (PASI 7–12; n=44), and severe (PASI >12; n=38). Demographic data, disease duration, body mass index (BMI), family history, and comorbidity profiles were recorded using a standardized proforma.

Sample Collection and Biomarker Analysis: Venous blood samples (5 mL) were collected from all participants under aseptic conditions following overnight fasting. Serum was separated by centrifugation at 3000 rpm for 10 minutes and stored at −80°C until analysis. Serum CRP levels were measured using latex-enhanced immunoturbidimetric assay (Roche Diagnostics). Serum IL-6, TNF-α, and IL-17A levels were quantified using commercially available sandwich ELISA kits (R&D Systems, Minneapolis, MN, USA) according to the manufacturer's instructions. All assays were performed in duplicate, and mean values were used for statistical analysis. Intra-assay and inter-assay coefficients of variation were maintained below 8% and 12%, respectively.

Statistical Analysis: Data were analyzed using Statistical Package for Social Sciences (SPSS) version 26.0 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables were expressed as frequencies and percentages. Normality of data distribution was assessed using the Shapiro-Wilk test.

Comparisons between two groups were performed using the independent samples t-test or Mann-Whitney U test, as appropriate. Comparisons among three or more groups were conducted using one-way ANOVA with post-hoc Tukey's test or Kruskal-Wallis test. Correlations between serum biomarker levels and PASI scores were evaluated using Spearman's rank correlation coefficient. Multiple linear regression analysis was performed to identify independent predictors of PASI scores. A two-tailed p-value <0.05 was considered statistically significant.

Results

Demographic and Clinical Characteristics: Among the 120 psoriasis patients, 72 (60%) were male and 48 (40%) were female, with a mean age of 42.56 ± 12.34 years.

The control group comprised 36 (60%) males and 24 (40%) females, with a mean age of 40.83 ± 11.67 years. No significant differences were observed between groups regarding age (p=0.372), sex distribution (p=0.918), or BMI (p=0.214). The mean disease duration was 7.84 ± 5.21 years, and the mean PASI score was 12.47 ± 7.63. A positive family history of psoriasis was present in 28 (23.3%) patients (Table 1).

Table 1: Demographic and Clinical Characteristics of Study Participants

Parameter	Psoriasis Patients (n=120)	Healthy Controls (n=60)	p-value
Age (years), mean ± SD	42.56 ± 12.34	40.83 ± 11.67	0.372
Male, n (%)	72 (60.0)	36 (60.0)	0.918
Female, n (%)	48 (40.0)	24 (40.0)	—
BMI (kg/m²), mean ± SD	26.73 ± 4.18	25.84 ± 3.92	0.214
Disease duration (years), mean ± SD	7.84 ± 5.21	—	—
PASI score, mean ± SD	12.47 ± 7.63	—	—
Family history positive, n (%)	28 (23.3)	—	—
Psoriatic arthritis, n (%)	18 (15.0)	—	—

Serum Biomarker Levels: Patients versus Controls: All four serum inflammatory biomarkers were significantly elevated in psoriasis patients compared to healthy controls (p<0.001 for all comparisons). CRP levels were approximately seven-fold higher, IL-6 approximately five-fold higher, TNF-α approximately five-fold higher, and IL-17A approximately six-fold higher in the psoriasis group (Table 2).

Table 2. Serum Inflammatory Biomarker Levels in Psoriasis Patients versus Controls

Biomarker	Psoriasis Patients (n=120)	Healthy Controls (n=60)	p-value
CRP (mg/L), mean ± SD	14.82 ± 6.41	2.14 ± 1.03	<0.001
IL-6 (pg/mL), mean ± SD	28.67 ± 11.54	5.23 ± 2.18	<0.001
TNF-α (pg/mL), mean ± SD	45.39 ± 18.72	8.91 ± 3.44	<0.001
IL-17A (pg/mL), mean ± SD	38.56 ± 14.83	6.47 ± 2.76	<0.001

Biomarker Levels Stratified by Disease Severity: Serum biomarker levels demonstrated a statistically significant stepwise increase corresponding to disease severity categories. Patients with severe psoriasis (PASI >12) exhibited markedly higher biomarker concentrations compared to those with moderate and mild disease (p<0.001, one-way ANOVA). Post-hoc analysis revealed significant differences between all pairwise severity group comparisons for IL-17A and TNF-α, while CRP and IL-6 showed significant differences primarily between mild and severe groups (Table 3).

Table 3. Serum Biomarker Levels Stratified by Disease Severity

Biomarker	Mild (PASI <7; n=38)	Moderate (PASI 7–12; n=44)	Severe (PASI >12; n=38)	p-value (ANOVA)
CRP (mg/L)	8.23 ± 3.14	14.61 ± 4.27	22.18 ± 5.63	<0.001
IL-6 (pg/mL)	17.42 ± 6.38	28.34 ± 8.91	41.26 ± 10.47	<0.001
TNF-α (pg/mL)	26.18 ± 9.53	44.72 ± 12.86	67.43 ± 16.29	<0.001
IL-17A (pg/mL)	21.34 ± 7.62	38.14 ± 10.25	58.72 ± 12.41	<0.001

Correlation between Biomarkers and PASI Scores: Spearman's rank correlation analysis revealed significant positive correlations between all four biomarkers and PASI scores. IL-17A demonstrated the strongest correlation (r=0.784, p<0.001), followed by TNF-α (r=0.721, p<0.001), IL-6 (r=0.658, p<0.001), and CRP (r=0.612, p<0.001). Multiple linear regression analysis identified IL-17A (β=0.392, p<0.001), TNF-α (β=0.274, p=0.002), and disease duration (β=0.168, p=0.018) as independent predictors of PASI scores, with the model explaining 68.4% of the variance (adjusted R^2=0.684).

Discussion

The present study comprehensively evaluated four serum inflammatory biomarkers in chronic plaque psoriasis and demonstrated their significant elevation compared to healthy controls, with progressive increases corresponding to disease severity. These findings reinforce the conceptualization of psoriasis as a systemic

inflammatory condition extending beyond the cutaneous compartment.

Our observation of significantly elevated CRP levels in psoriasis patients is consistent with the findings reported by Coimbra et al., who demonstrated that CRP serves as a reliable indicator of systemic inflammation in moderate-to-severe psoriasis and reflects underlying subclinical atherosclerotic risk [13]. The approximately seven-fold elevation in CRP observed in our cohort underscores the magnitude of systemic inflammatory burden in these patients and aligns with the well-established association between psoriasis and cardiovascular comorbidities [14].

The significant elevation of serum IL-6 and its positive correlation with PASI scores corroborate the findings of Dowlatshahi et al., whose systematic review and meta-analysis confirmed consistently elevated IL-6 levels in psoriasis patients compared to controls [15]. IL-6 plays a multifaceted role in psoriatic pathogenesis, driving acute-phase protein synthesis, promoting Th17 cell differentiation, and contributing to insulin resistance and metabolic derangements frequently observed in psoriasis patients [16]. The strong correlation between IL-6 and disease severity observed in our study ($r=0.658$) supports its utility as a monitoring biomarker, although its lack of disease specificity limits its diagnostic application.

TNF-α demonstrated a robust correlation with PASI scores ($r=0.721$), consistent with the pivotal role of this cytokine in psoriatic inflammation and the demonstrated therapeutic efficacy of TNF-α inhibitors in managing moderate-to-severe disease [17]. Arican et al. similarly reported significantly elevated serum TNF-α levels in psoriasis patients with positive correlations to severity indices, supporting the notion that circulating TNF-α concentrations reflect the overall inflammatory disease state [18].

Notably, IL-17A exhibited the strongest correlation with PASI scores ($r=0.784$) among all biomarkers evaluated, a finding that is particularly relevant given the central pathogenic role of the IL-23/IL-17 axis in psoriasis. This observation aligns with the work of Kyriakou et al., who reported significant associations between serum IL-17 levels and disease severity in psoriasis patients [19].

The superior correlative performance of IL-17A compared to other biomarkers may reflect its direct involvement in keratinocyte activation, neutrophil recruitment, and the maintenance of the psoriatic inflammatory loop [20]. Furthermore, the remarkable clinical efficacy of anti-IL-17A biological agents, including secukinumab and ixekizumab, in achieving rapid and sustained PASI improvement provides indirect validation of IL-17A as a pathogenically relevant and clinically meaningful biomarker [21].

Multiple linear regression analysis identified IL-17A as the strongest independent predictor of PASI scores, further supporting its potential as a primary serological indicator for disease monitoring. This finding extends the observations of Takahashi et al., who reported that serum IL-17A levels not only correlated with disease severity but also demonstrated significant reductions following successful therapeutic intervention, suggesting their utility in longitudinal treatment monitoring [22].

The progressive stepwise elevation of all biomarkers across severity categories—mild, moderate, and severe—observed in our study provides a framework for potential biomarker-based severity stratification. Such objective serological stratification could supplement clinical assessment tools and facilitate more precise therapeutic decision-making, particularly in the context of escalating to biologic therapy [23].

Several limitations of this study merit consideration. The cross-sectional design precludes assessment of temporal relationships and biomarker dynamics during treatment. The sample was derived from a single tertiary care center, potentially limiting generalizability. Additionally, other emerging biomarkers such as IL-22, IL-23, and β-defensin-2 were not evaluated, and their inclusion in future studies may provide a more comprehensive biomarker profile. The absence of longitudinal follow-up data prevents assessment of biomarker responsiveness to therapeutic interventions [24].

Conclusion

This study demonstrates that serum levels of CRP, IL-6, TNF-α, and IL-17A are significantly elevated in patients with chronic plaque psoriasis compared to healthy controls and exhibit progressive increases corresponding to disease severity. Among the evaluated biomarkers, IL-17A displayed the strongest correlation with PASI scores and emerged as the most robust independent predictor of disease severity. These findings support the incorporation of serum inflammatory biomarkers, particularly IL-17A, as objective adjunctive tools for disease severity assessment and therapeutic monitoring in chronic plaque psoriasis. Future prospective longitudinal studies evaluating the dynamic changes of these biomarkers in response to targeted biologic therapies are warranted to validate their clinical utility in personalized management strategies.

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