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ORIGINAL RESEARCH

Evaluation of Pan-Immune-Inflammation Value (PIV) as a Predictive Biomarker for Psoriatic Arthritis in Patients with Psoriasis

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Objective: Psoriasis (PsO) is a chronic inflammatory dermatosis that affects a significant portion of the global population and is associated with various comorbidities, including psoriatic arthritis (PsA). PsA develops in 20–30% of psoriasis patients and significantly impacts patient quality of life. Currently, early detection of PsA remains challenging, with no reliable, non-invasive biomarker for risk assessment. This study aims to evaluate the utility of pan-immune-inflammation value (PIV), along with other inflammatory indices, in predicting the development of PsA in patients with PsO.

Methods: A retrospective analysis was conducted on 101 psoriasis patients (51 PsO, 50 PsA). Hematological parameters, including neutrophil, lymphocyte, monocyte, and platelet counts, were collected, and various inflammatory indices (PIV, SIRI, SII, NLR) were calculated. The diagnostic performance of these indices was assessed using Receiver Operating Characteristic (ROC) curve analysis. As a retrospective, double-center study with a limited sample size, the findings should be interpreted with caution and validated in larger, prospective cohorts.

Results: The study found significant differences between PsA and PsO patients in terms of inflammatory indices, with higher PIV, SII, SIRI, and NLR levels observed in PsA patients. The diagnostic performance of PIV (AUC 0.63), SII (AUC 0.70), SIRI (AUC 0.64), and NLR (AUC 0.71) indicated that elevated levels of these indices could serve as potential markers for PsA risk. Additionally, PIV was significantly correlated with SII, SIRI, and NLR levels.

Conclusion: PIV and other inflammatory indices, particularly NLR and SII, show promise as biomarkers for predicting the onset of PsA in patients with PsO. These findings may aid in early identification and timely intervention for PsA, improving patient outcomes and informing treatment strategies.

Keywords: psoriasis, psoriatic arthritis, pan-immune-inflammation value, biomarker

Introduction

Psoriasis (PsO) is a chronic inflammatory dermatosis characterized by erythematous, scaly plaques, affecting approximately 2–3% of the global population.[1] The etiopathogenesis of psoriasis is highly complex, involving genetic predisposition, environmental factors, and multiple inflammatory pathways.[2] The most commonly affected anatomical sites include the scalp, knees, elbows, lumbosacral region, nails, and genital area; however, any region of the skin may be involved. The activation of inflammatory pathways by various triggering factors results in an exaggerated T-cell response.[2] Recent studies indicate that this aberrant inflammatory reaction extends beyond cutaneous lesions, affecting other tissues, thereby supporting the classification of psoriasis as a systemic inflammatory disease, termed psoriatic disease. The association of psoriasis with multiple comorbidities including metabolic disorders, cardiovascular diseases, malignancies, chronic kidney disease, psychiatric disorders, and inflammatory bowel disease further substantiates this perspective.[3,4]

Psoriatic arthritis (PsA) is a seronegative spondyloarthropathy that affects approximately 20–30% of individuals with psoriasis, characterized by inflammation of the tendons, joints, and entheses.[5] Early diagnosis and timely intervention are

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crucial in preventing irreversible joint damage and mitigating disease severity. In nearly 80% of cases, PsA develops following a prior diagnosis of psoriasis.[6] Consequently, a simple and scalable method for assessing PsA risk in psoriasis patients could facilitate early intervention, potentially halting disease progression, improving quality of life, and informing treatment strategies. However, no non-invasive, widely applicable, and reliable biomarker has yet been identified for the early detection of PsA. In current clinical practice, the diagnosis of psoriatic arthritis (PsA) primarily relies on clinical symptoms, laboratory markers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), and imaging modalities. However, these conventional inflammatory biomarkers lack both specificity and sensitivity, often showing significant elevations only in advanced disease stages, thereby limiting their utility in the early detection of PsA. In response to this limitation, we have expanded the introduction to highlight the shortcomings of CRP and ESR as diagnostic tools and to emphasize the potential value of novel, composite inflammatory indices—particularly the Pan-Immune-Inflammation Value (PIV)—which may more comprehensively capture systemic inflammatory activity and provide earlier insights into the transition from psoriasis to PsA.[7]

In recent years, hematological parameters and inflammatory indices have garnered increasing attention for their prognostic and predictive value in autoimmune diseases. Among these, the pan-immune-inflammation value (PIV), systemic inflammatory response index (SIRI), neutrophil-to-lymphocyte ratio (NLR), and systemic immune-inflammation index (SII) have emerged as notable markers. PIV, first introduced by Fuca et al in 2020 in the context of metastatic colorectal cancer, is calculated using neutrophil, platelet, monocyte, and lymphocyte counts and has been identified as a potential predictor of disease prognosis and treatment response.[8] PIV has also been investigated in various rheumatologic conditions, including rheumatoid arthritis, familial Mediterranean fever, vasculitis, and Behçet's disease.[9–11] However, the limited number of studies examining PIV in psoriasis and PsA means that its potential role in predicting the transition from PsO to PsA remains uncertain.

The link between PsA development and systemic inflammation is well established. Elevated levels of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-17 (IL-17), and interleukin-23 (IL-23) have been observed in PsA patients, suggesting that systemic inflammation may be reflected in peripheral blood parameters.[12] It is hypothesized that PIV, as an index representing both cellular and humoral immune responses, may serve as a useful predictor of PsA development in individuals with psoriasis.

This study aims to assess the utility of PIV in predicting the onset of PsA in patients with PsO. By comparing PIV values between individuals with PsA and those with cutaneous PsO only, this investigation explores the potential role of PIV as a pre-diagnostic and risk assessment tool in clinical practice. The findings may contribute to the advancement of novel approaches for the early diagnosis and management of PsA in psoriasis patients.

Materials and Methods

Design and Data Collection

This retrospective study included patients aged 18–65 years diagnosed with psoriasis (PsO) or psoriatic arthritis (PsA) who were followed up at the Dermatology and Venereal Diseases Clinic of Sivas Numune Hospital and the Rheumatology Department of Sivas Cumhuriyet University Faculty of Medicine between January 1, 2020, and August 1, 2024. PsO patients were selected from newly diagnosed, treatment-naïve individuals, whereas PsA patients were selected from newly diagnosed individuals who had not received systemic treatment for at least six weeks. Patients with malignancies or active infections were excluded from the study. Only routine laboratory tests performed during follow-up and treatment were analyzed, and no additional investigations were requested from the participants. Additionally, the diagnostic validation process for PsA was explicitly addressed: all PsA diagnoses were confirmed independently by two experienced dermatologists or rheumatologists in accordance with the CASPAR criteria.

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Demographic data and blood parameters at the time of diagnosis, prior to the initiation of systemic treatment including neutrophil (N), lymphocyte (L), monocyte (M), platelet (Plt) counts, C-reactive protein (CRP), and erythrocyte sedimentation rate (ESR) were retrieved from the hospital information system. Based on these criteria, 51 PsO patients and 50 PsA patients were included in the study. Disease severity was assessed using the Psoriasis Area and Severity Index (PASI) score.

Calculation of Inflammatory Indices

The inflammatory indices were calculated using the following formulas:

Pan-Immune-Inflammation Value (PIV): (Neutrophil-to-Lymphocyte Ratio) × Platelets × Monocytes.[8]

Systemic Inflammatory Response Index (SIRI): (Neutrophil-to-Lymphocyte Ratio) × Monocytes.[13]

Systemic Immune-Inflammation Index (SII): (Neutrophil-to-Lymphocyte Ratio) × Platelets.[13]

Neutrophil-to-Lymphocyte Ratio (NLR): Neutrophils / Lymphocytes.[13]

Statistical Analysis

The required sample size was determined using the G*Power 3.1.9.7 software. Based on an effect size of 0.35, a Type I error rate of 0.05, and a test power of 85%, a minimum of 90 patients was deemed necessary. However, to enhance statistical robustness, the study was designed to include 101 patients. Statistical analyses were conducted using IBM SPSS Statistics for Windows, version 27 (IBM Corp., Chicago, IL, USA). The normality of data distribution was assessed using the Kolmogorov–Smirnov test. Continuous variables were expressed as mean ± standard deviation if normally distributed or as median (min–max) if not. The diagnostic performance of PIV, SIRI, SII, and NLR in detecting PsA was evaluated using Receiver Operating Characteristic (ROC) curve analysis. Optimal cut-off values for these indices were determined using the Youden index method. Associations between PIV and clinical/laboratory parameters were analyzed using the Chi-square test, Fisher's exact test, Mann–Whitney *U*-test, and Kruskal–Wallis test. A p-value <0.05 was considered statistically significant.

Results

A total of 130 patients newly diagnosed with psoriasis were initially included in the study; however, 29 patients were excluded due to incomplete clinical and laboratory data. The median age of the remaining participants was 49 years (range: 19–76), with 49 patients (48.5%) aged 50 years or older. Of the total cohort, 41 (40.6%) were male, and 60 (59.6%) were female. Comorbidities were present in 48 patients (47.5%). Regarding disease manifestations, 78 patients (77.2%) had widespread skin lesions, 19 (18.8%) had hand-foot involvement, and 4 (4.0%) had scalp-only involvement. Additionally, 16 patients (15.8%) had palmoplantar psoriasis, while 85 (84.2%) had psoriasis vulgaris. Nail involvement was observed in 33 patients (32.7%), and psoriatic arthritis (PsA) was diagnosed in 50 patients (49.5%). The median disease duration was 10 months (range: 1–40), and the median Psoriasis Area and Severity Index (PASI) score was 10 (range: 0–27). Systemic therapy was administered to 81.2% of the patients. The clinical and demographic characteristics of the study participants are summarized in Table 1.

The diagnostic performance of various laboratory parameters in predicting PsA in psoriasis patients was evaluated through Receiver Operating Characteristic (ROC) curve analysis (Figure 1).

The area under the curve (AUC) values were as follows: PIV (0.63, 95% CI: 0.52–0.74), SII (0.70, 95% CI: 0.59–0.80), SIRI (0.64, 95% CI: 0.52–0.75), and NLR (0.71, 95% CI: 0.61–0.82). The optimal cutoff values (maximum Youden index) for these indices were determined as follows: PIV = 329.1, SII = 681, SIRI = 1.07, and NLR = 2.17 (Table 2).

Significant differences were observed between patients with and without PsA in terms of sex, nail involvement, disease manifestation, systemic therapy administration, and inflammatory index levels. Specifically, female sex, absence

Table 1 Clinical and Demographic Characteristics of the Patients

Variable	n (%)
Age	
< 50 years	52 (51.5%)
≥ 50 years	49 (48.5%)
Sex	
Male	41 (40.6%)
Female	60 (59.4%)
Psoriasis subtype	
Palmoplantar	16 (15.8%)
Vulgaris	85 (84.2%)
Localization	
Widespread	78 (77.2%)
Hand-foot lesions	19 (18.8%)
Scalp only	4 (4.0%)
Nail involvement	
Absent	68 (67.3%)
Present	33 (32.7%)
Comorbidity	
Absent	53 (52.5%)
Present	48 (47.5%)
Treatment	
Systemic	82 (81.2%)
Topical	19 (18.8%)
Disease duration (months)	Median: 10 (1–40)
PASI	Median: 10 (0–27)

Abbreviation: PASI, Psoriasis Area and Severity Index.

of nail involvement, presence of widespread skin lesions, systemic therapy administration, and elevated levels of PIV, SII, SIRI, and NLR were more common in PsA patients compared to those without PsA ($p<0.05$) (Table 3).

In the study cohort, 54 patients (53.5%) had low PIV, while 47 patients (46.5%) had high PIV. High PIV was more frequently observed in younger patients, females, individuals with the vulgaris subtype, those with widespread skin lesions, those without nail involvement, those without comorbidities, those receiving systemic therapy, and patients with longer disease durations; however, these associations did not reach statistical significance ($p>0.05$). The mean PASI score did not show a statistically significant difference between the PsO and PsA groups ($p > 0.05$).

In contrast, high PIV was significantly correlated with elevated SII, SIRI, and NLR levels ($p<0.001$, Table 4).

A strong positive correlation was found between PIV and SII ($r=0.682$, $p<0.001$), PIV and SIRI ($r=0.769$, $p<0.001$), and PIV and NLR ($r=0.566$, $p<0.001$).

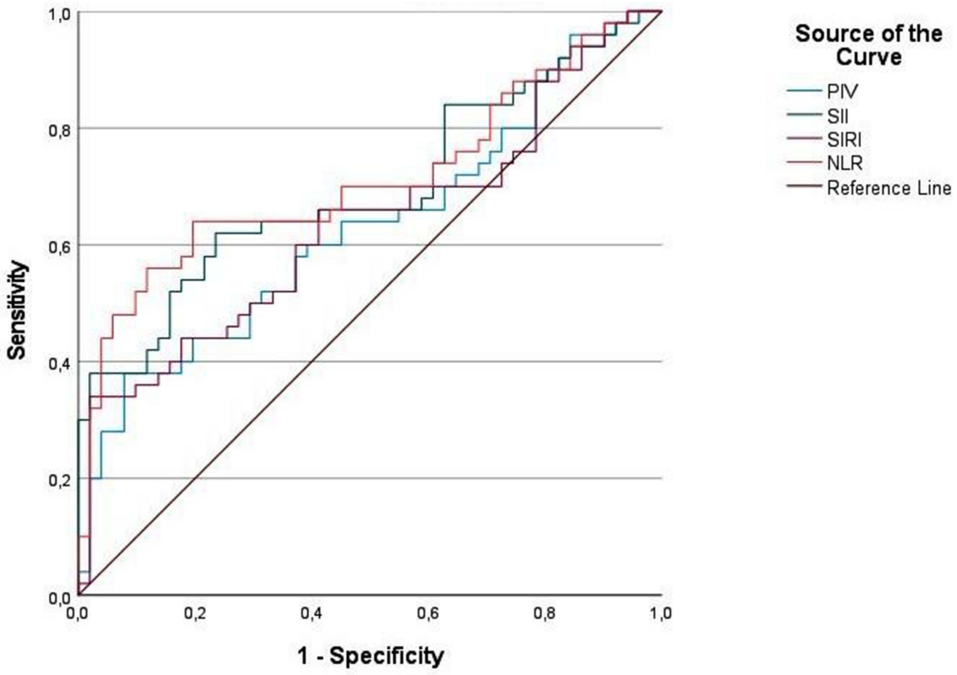

Figure 1 ROC curve analysis results demonstrating the relationship between PIV, SII, SIRI, and NLR values and the presence of arthritis.

Discussion

PsO is a chronic inflammatory dermatosis characterized by erythematous, scaly plaques, with arthritis affecting 20–30% of psoriasis patients. PsA can develop at any age, although it is most frequently observed in individuals aged 30 to 50, with no significant gender predilection. In our study, the median age of participants was 49 years, and no significant gender differences were noted between male and female patients.

Table 2 Diagnostic Performance and Cut-off Values of Laboratory Parameters for Arthritis Prediction

Variable	AUC	95% CI	p	Sensitivity (%)	Specificity (%)	Cut-off Value
PIV	0.630	0.521–0.740	0.024	58.0	62.7	329.1
SII	0.696	0.592–0.800	0.001	62.0	76.5	681
SIRI	0.635	0.524–0.745	0.020	66.0	58.8	1.07
NLR	0.712	0.608–0.816	0.000	64.0	80.4	2.17

Abbreviations: AUC, Area under the curve; PIV, Pan immune-inflammation value; SII, Systemic immune inflammation index; SIRI, Systemic inflammatory response index; NLR, Neutrophil-lymphocyte ratio.

Table 3 Comparison of Categorical Variables Between Patients with and without Psoriatic Arthritis

Variable	PsA Absent (n, %)	PsA Present (n, %)	p-value
Age			
< 50 years	27 (52.9%)	25 (50.0%)	
≥ 50 years	24 (47.1%)	25 (50.0%)	0.462

(*Continued*)

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Table 3 (Continued).

Variable	PsA Absent (n, %)	PsA Present (n, %)	p-value
Sex			
Male	27 (52.9%)	14 (28.0%)	
Female	24 (47.1%)	36 (72.0%)	0.009
Psoriasis subtype			
Palmoplantar	11 (21.6%)	5 (10.0%)	
Vulgaris	40 (78.4%)	45 (90.0%)	0.093
Localization			
Widespread	32 (62.7%)	46 (92.0%)	
Hand-foot	15 (29.4%)	4 (8.0%)	<0.001
Scalp	4 (7.8%)	0 (0.0%)	
Nail involvement			
Absent	40 (78.4%)	28 (56.0%)	
Present	11 (21.6%)	22 (44.0%)	0.014
Comorbidity			
Absent	29 (56.9%)	24 (48.0%)	
Present	22 (43.1%)	26 (52.0%)	0.244
Treatment			
Systemic	32 (62.7%)	50 (100.0%)	
Topical	19 (37.3%)	0 (0.0%)	<0.001
PIV			
< 329.1	33 (64.7%)	21 (42.0%)	
≥ 329.1	18 (35.3%)	29 (58.0%)	0.014
SII			
< 681	39 (76.5%)	19 (38.0%)	
≥ 681	12 (23.5%)	31 (62.0%)	<0.001
SIRI			
< 1.07	31 (60.8%)	17 (34.0%)	
≥ 1.07	20 (39.2%)	33 (66.0%)	0.006
NLR			
< 2.17	32 (62.7%)	18 (36.0%)	
≥ 2.17	19 (37.3%)	32 (64.0%)	0.006

Abbreviations: PsA, Psoriatic arthritis; PIV, Pan immune-inflammation value; SII, Systemic immune inflammation index; SIRI, Systemic inflammatory response index; NLR, Neutrophil-lymphocyte ratio.

Table 4 Comparison of Clinical and Laboratory Parameters Based on PIV Groups

Variable	PIV < 329.1 (n, %)	PIV ≥ 329.1 (n, %)	p-value
Age			
< 50 years	26 (48.1%)	26 (55.3%)	
≥ 50 years	28 (51.9%)	21 (44.7%)	0.302
Sex			
Male	18 (33.3%)	23 (48.9%)	
Female	36 (66.7%)	24 (51.1%)	0.082
Psoriasis subtype			
Palmoplantar	9 (16.7%)	7 (14.9%)	
Vulgaris	45 (83.3%)	40 (85.1%)	0.514
Localization			
Widespread	38 (70.4%)	40 (85.1%)	
Hand-foot	14 (25.9%)	5 (10.6%)	0.177
Scalp	2 (3.7%)	2 (4.3%)	
Nail involvement			
Absent	37 (68.5%)	31 (66.0%)	
Present	17 (31.5%)	16 (34.0%)	0.475
Comorbidity			
Absent	26 (48.1%)	27 (57.4%)	
Present	28 (51.9%)	20 (42.6%)	0.232
Treatment			
Systemic	43 (79.6%)	39 (83.0%)	
Topical	11 (20.4%)	8 (17.0%)	0.432
Arthritis			
Absent	33 (61.1%)	18 (38.3%)	
Present	21 (38.9%)	29 (61.7%)	0.018
SII			
< 681	48 (88.9%)	10 (21.3%)	
≥ 681	6 (11.1%)	37 (78.7%)	<0.001
SIRI			
< 1.07	45 (83.3%)	3 (6.4%)	
≥ 1.07	9 (16.7%)	44 (93.6%)	<0.001

(*Continued*)

Table 4 (Continued).

Variable	PIV < 329.1 (n, %)	PIV ≥ 329.1 (n, %)	p-value
NLR			
< 2.17	41 (75.9%)	9 (19.1%)	<0.001
≥ 2.17	13 (24.1%)	38 (80.9%)	

Abbreviations: PsA, Psoriatic arthritis; PIV, Pan immune-inflammation value; SII, Systemic immune inflammation index; SIRI, Systemic inflammatory response index; NLR, Neutrophil-lymphocyte ratio.

Arthritis, which develops in 80% of psoriasis patients, is a key factor in predicting patient outcomes, as early detection can significantly improve patient comfort.[6] This is especially important given that the presence of arthritis exacerbates the impact of the disease on quality of life. Therefore, identifying biomarkers that predict the risk of arthritis and assist clinicians in selecting appropriate treatment strategies is crucial. Several studies have explored biomarkers such as the SII, SIRI, and NLR.[14–19] However, our study is the first to assess the PIV index in PsA patients.

The NLR, a simple, cost-effective, and widely used hematological parameter, serves as a marker of systemic inflammation by comparing neutrophil and lymphocyte counts in peripheral blood. Numerous studies have demonstrated that NLR is significantly elevated in psoriasis and PsA patients compared to controls. One study identified NLR as a robust predictor of PsA and observed a positive correlation between NLR and the Psoriasis Area and Severity Index (PASI).[14] Similarly, in our study, NLR was significantly higher in PsA patients compared to those with PsO alone. In another study of psoriasis patients undergoing biological therapy, NLR, along with CRP, decreased post-treatment, suggesting that NLR may serve as a useful marker for assessing response to systemic therapies.[15]

The SII offers a comprehensive evaluation of systemic inflammation by simultaneously measuring immune system activation (neutrophils), platelet-mediated inflammation, and lymphocyte-mediated immune regulation, utilizing parameters derived from complete blood counts. A study by Dinçer et al, which compared patients with PsA to a control group, revealed significantly elevated SII levels in PsA patients.[18] Additionally, when PsA patients were stratified according to disease activity scores, statistically significant positive correlations were observed between DAS28-ESR, DAS28-CRP, DAPSA, and SII.[18] These findings suggest that SII may serve as a valuable tool for assessing disease activity and monitoring treatment response.

The SIRI is a biomarker that reflects the intensity of systemic inflammation, particularly through neutrophils and monocytes, contrasting with the balancing effect of lymphocytes on immune regulation. Current literature on SIRI values in psoriasis and PsA is limited. A study utilizing National Health and Nutrition Examination Survey (NHANES) data categorized SII and SIRI values into low, medium, and high levels, with medium and high levels associated with an increased risk of psoriasis (13). Additionally, Sugimoto et al found elevated SII and SIRI values in PsA patients compared to those with PsO, with high SII scores correlating with lower continuation rates of traditional systemic therapies.[20]

PIV, a relatively novel hematological biomarker, has gained attention in oncology and chronic inflammatory diseases as an indicator of systemic inflammation and immune response. In oncology, elevated PIV levels are linked to poor prognosis, increased tumor burden, and decreased survival.[21–24] Although studies on PIV in dermatological inflammatory diseases are limited, one study on hidradenitis suppurativa found that both SII and PIV levels were significantly higher in patients compared to healthy controls, with a correlation between these values and disease severity.[25] In the retrospective observational study conducted by Kilic et al, it was demonstrated that the PIV and the SII could serve as predictive markers for assessing disease activity and prognosis in patients with psoriasis.[26] Additionally, in a study on ANCA-associated vasculitis, patients with elevated PIV levels at diagnosis exhibited poorer survival outcomes.[11] In a comparable study conducted by Tutan et al on patients with rheumatoid arthritis (RA), it was observed that the PIV was elevated in the active RA group. Additionally, the PIV value was found to be higher in RA patients in remission when compared to the control group. Consequently, the authors suggested that PIV could serve as a valuable biomarker for distinguishing between active and remission phases of RA in comparison to healthy individuals.[9] In our investigation, a similar pattern was noted, where patients with PsA exhibited significantly higher PIV levels compared to those with

PsO without arthritis. It is well-established that psoriasis patients with nail involvement are at an increased risk of developing associated psoriatic arthritis.[27] However, in our study, elevated PIV levels and the presence of arthritis were observed in patients without nail involvement. This discrepancy may be attributed to the relatively small sample size, the retrospective design of the study, and the potential lack of comprehensive data within the medical records.

Expanding the clinical relevance of PIV requires determining its practical utility for dermatologists. Given that NLR and SII demonstrated higher AUC values, further clarification is needed to establish whether PIV offers additional prognostic value or serves as a complementary biomarker. Dermatologists could consider integrating PIV as part of baseline inflammatory screening in psoriatic patients to stratify systemic inflammation risk.

Despite the insightful findings, several limitations of this study should be noted. The retrospective and double-center design, along with the relatively small and imbalanced sample distribution between the PsO and PsA groups, may limit the generalizability of the results. Additionally, potential confounding factors such as comorbid conditions, variations in systemic therapy, and differences in lesion extent or nail involvement were not fully controlled, which may have influenced inflammatory marker levels. Future prospective, multicenter studies with larger and more homogeneous cohorts are warranted to validate these findings and establish standardized cutoff values for clinical use.

Conclusions

PIV is a simple, cost-effective, and readily accessible biomarker that can be assessed using peripheral blood parameters. Although its discriminatory power was lower than that of NLR and SII, it still holds potential as a supportive tool for assessing the risk of arthritis development in psoriasis patients. Moreover, PIV may provide complementary insights for treatment selection and patient monitoring, thereby assisting clinicians in the overall decision-making process.

Abbreviations

PsO, Psoriasis; PsA, Psoriatic arthritis; PIV, Pan-immune-inflammation value; NLR, Neutrofil-lymphocyte ratio; SII, Systemic Inflammatory Index; SIRI, Systemic Inflammatory Response Index; ROC, Receiver Operating Characteristic; SPSS, Statistical Package for Social Sciences.

Data Sharing Statement

The datasets used in this study can be made available by the corresponding author upon reasonable request, with permission from the Rheumotology Department of Sivas Cumhuriyet University School of Medicine.

Ethical Approval

The study was conducted in accordance with the Declaration of Helsinki, and approved by the Non-Interventional Clinical Research Ethics Committee of Sivas Cumhuriyet University on October 17, 2024 (Approval Number: 2024/10-20). Ethical approval was obtained from the relevant institutional ethics committee, and written informed consent was obtained from all patients included in the study.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no conflicts of interest.

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