Observational Study

Medicine

OPEN

Pan-immune inflammation value and systemic inflammatory index as a measure of systemic inflammation in patients with psoriasis

A retrospective study

Seyma Basar Kilic, MD[a], Huseyin Erdal, PhD[b,*]

Abstract

Psoriasis is a chronic immune-mediated disease characterized by systemic inflammation. In recent years, the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), systemic inflammation response index (SIRI), and pan-immune-inflammation value (PIV) were shown to be important indicators of inflammation. The aim of the present study is to investigate NLR, PLR, SII, SIRI, PIV together in patients with psoriasis. This retrospective case-control study encompassed seventy-one individuals diagnosed with psoriasis and seventy healthy controls who underwent evaluation at the Dermatology clinics of Aksaray University Training and Research Hospital from January 2022 to January 2023. Inflammatory process indicators such as NLR, PLR, SII, SIRI, PIV were computed for analysis. A notable discovery from our research was the indication of a direct relationship between SII and Psoriasis Area Severity Index (PASI) scores. A statistically significant difference was found between the 2 groups in terms of neutrophils, lymphocytes, monocytes, and platelets ($P < .05$). The area under the curve of the SII score for psoriasis was 0.611. The optimal cutoff value of SII to predict psoriasis activation was 442.7, with 55.7% sensitivity and 45.7% specificity (95% confidence interval 0.518–0.704, $P = .024$). A positive correlation was observed between SII, PIV and PASI ($P = .004$, $r = 0.34$; $P = .006$, $r = 0.32$ respectively).There was no statistically significant distinction observed in the PLR indices between the groups ($P > .05$). The present study investigation demonstrates the potential utility of SII, SIRI, and PIV in assessing psoriasis patients. Moreover, the findings suggest that SII and PIV could function as an autonomous prognostic marker for individuals diagnosed with psoriasis.

Abbreviations: AUC = area under the curve, HS = hidradenitis suppurativa, NLR = neutrophil-to-lymphocyte ratio, OS = oxidative stress, PASI = psoriasis area severity index, PIV = pan-immune-inflammation value, PLR = platelet-to-lymphocyte ratio, SII = systemic immune-inflammation index, SIRI = systemic inflammation response index.

Keywords: inflammation, pan-immune-inflammation value, psoriasis, systemic inflammatory response index

1. Introduction

Psoriasis is a prevalent chronic inflammatory condition typified by alternating periods of exacerbation and remission. Its prevalence in the general population is estimated to be between 1.5% to 2%.[1] It is a non-contagious condition that can affect various body parts, including the scalp, elbows, knees, and lower back.[2] Experts suggest that psoriasis arises from a blend of genetic, environmental, and immune system elements.[3] The immune system plays an important role in the occurrence of psoriasis.[4] In a normal immune response, white blood cells, particularly T cells, help protect the body against infections and other threats. However, in individuals with psoriasis, these T cells become overactive and trigger an inflammatory response. This, in turn, accelerates the production of skin cells, leading to the characteristic buildup of thick, scaly patches. In psoriasis, the immune system's abnormal response doesn't just affect the skin; it can also lead to inflammation in other parts of the body.[5] This systemic inflammation has been associated with a higher likelihood of developing additional conditions, including cardiovascular disease, metabolic syndrome, and arthritis. The connection between psoriasis

The authors have no funding and conflicts of interest to disclose.

The datasets generated during and/or analyzed during the current study are available from the corresponding author on reasonable request.

All procedures performed in this study were in accordance with the ethical standards of the institutional and/or national research committee and with the 1964 Helsinki Declaration and its later amendments or comparable ethical standards.

The authors declare that they are responsible for the article's scientific content including study design, data collection, analysis and interpretation, writing, some of the main line, or all of the preparation and scientific review of the contents and approval of the final version of the article.

[a] Department of Dermatology and Venereology, Faculty of Medicine, Aksaray University, Aksaray, Turkey, [b] Department of Medical Genetics, Faculty of Medicine, Aksaray University, Aksaray, Turkey.

** Correspondence: Huseyin Erdal, Department of Medical Genetics, Faculty of Medicine, Aksaray University, Aksaray, Turkey (e-mail: herdalyfa@gmail.com).*

How to cite this article: Basar Kilic S, Erdal H. Pan-immune inflammation value and systemic inflammatory index as a measure of systemic inflammation in patients with psoriasis: A retrospective study. Medicine 2025;104:10(e41715).

Received: 9 October 2024 / Received in final form: 10 February 2025 / Accepted: 12 February 2025

http://dx.doi.org/10.1097/MD.0000000000041715

and systemic inflammation is not fully understood, but it is believed that inflammatory molecules produced in the skin during a psoriatic flare-up can enter the bloodstream and affect other organs. Additionally, the chronic inflammatory state associated with psoriasis may contribute to developing systemic conditions.[6]

Oxidative stress (OS) stems from a combination of heightened production of reactive oxygen species/reactive nitrogen species and diminished levels or activity of antioxidants responsible for their neutralization. Research indicates that OS plays a significant role in the development of various diseases, including psoriasis.[7–13] OS may act as a trigger for the initiation or exacerbation of psoriasis. Increased levels of reactive oxygen species can activate signaling pathways and immune responses that contribute to the development of psoriatic lesions. This suggests that OS may play a role in the initial stages of the disease. Psoriasis is characterized by an abnormal immune response that leads to chronic inflammation. Psoriasis treatment often involves managing both the skin symptoms and addressing systemic inflammation. Topical treatments, phototherapy, and systemic medications (such as immunosuppressants or biologics) are commonly used to control skin manifestations. Making changes to one's lifestyle, like adhering to a balanced diet, engaging in regular physical activity, and abstaining from smoking and excessive alcohol intake, could also aid in controlling the systemic inflammation linked to psoriasis.[14–16] The systemic inflammatory response index (SIRI) and pan-immune-inflammation value (PIV) are recently developed indices recognized as comprehensive markers of immune response and systemic inflammation. These measures play a significant role in predicting prognosis or treatment outcomes in various diseases among patients.[17–21] The objective of this study was to assess neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), SIRI, and PIV parameters in individuals diagnosed with psoriasis.

2. Materials and methods

This retrospective case-control study included 71 patients with psoriasis and 70 healthy controls from individuals who visited the Dermatology Clinics of Aksaray University Training and Research Hospital from January 2022 to January 2023. The study adhered to the principles of the Helsinki Declaration, and informed consent was obtained from all participants. Patients younger than 18 years old, patients with an systemic infection, inflammatory/autoimmune or chronic disease other than psoriasis, and patients who received topical therapy for psoriasis in the last 2 weeks or systemic therapy in the last 4weeks were not included in the study. Demographic data, hemogram results, and biochemical parameters for both the study and control groups were retrieved from the hospital's automated system.

The complete blood parameters included neutrophil, lymphocyte, platelet, and monocyte levels for each group. Following that, the NLR, PLR, SII, SIRI, and PIV were computed as follows: the ratio of neutrophils to lymphocytes, platelets to lymphocytes, platelets multiplied by (neutrophils divided by lymphocytes), (neutrophils multiplied by monocytes) divided by lymphocytes, and (neutrophils multiplied by platelets multiplied by monocytes) divided by lymphocytes. Psoriasis area severity index (PASI) score were used for the assessment of disease severity.[22]

2.1. Ethical approval

The Ethics Committee of Aksaray University granted approval for this study (Approval Date: 2023-06-22, Approval Number: 72-SBKAEK).

2.2. Statistical analysis

IBM SPSS 22 was utilized for the statistical analysis of the groups. The normal distribution of the data was assessed using the Kolmogorov–Smirnov test. Categorical variables were expressed as numbers and percentages, and group differences were evaluated using the chi-squared test. Continuous variables were compared using Student *t* test and presented as means ± standard deviation. Non-normally distributed values were compared using the Mann–Whitney *U* test. The cutoff value for SII was defined using the receiver operating characteristic (ROC) curve. A P value < .05 was considered statistically significant.

3. Results

In the psoriasis group, the average age was 44.9 years with a standard deviation of 13.3, and 44.3% of the patients were

Table 1

Characteristics of patients and controls, including clinical and demographic data.

Parameter		Psoriasis (n = 71) n%	Control (n = 70) n%	*P*
Gender	Male	39 (55.7%)	37 (52.9%)	.734*
	Female	31 (44.3%)	33 (47.1%)	
Age (yr), mean ± SD		44.9 ± 13.3	45.6 ± 8.6	.717**
PASI†		7.9 (4.15–13.83)		
Scalp skin involvement, n (%)	Present	44 (62.9)		
	Absent	26 (37.1)		
Nail involvement, n (%)	Present	33 (47.1)		
	Absent	37 (52.9)		
Family history of psoriasis, n (%)	Present	16 (22.9)		
	Absent	54 (77.1)		
Duration of psoriasis (yr)†		6.0 (3.5–17)		

IQR = interquartile range, PASI = psoriasis area and severity index.
† Median (IQR).
* Chi-square test.
** Student *t* test.

Table 2

Comparison of laboratory findings between the patient and control group.

Parameters	Psoriasis patient (n = 71) median (min–max)	Control (n = 70) median (min–max)	*P**
White blood cell (10^3 µL)	7.35 (3.7–14.3)	7.1 (3.7–12.5)	.107
Neutrophil (10^3 µL)	4.4 (0.49–9.9)	3.8 (1.2-6.3)	**.005**
Lymphocyte (10^3 µL)	2.3 (0.1–4.0)	2.6 (0.9–4.5)	**.009**
Monocyte (10^3 µL)	0.46 (0.26–4.6)	0.42 (0.19–3.6)	**.041**
Hemoglobin (g/dL)	14.5 (11.6–18.0)	14.0 (11.2–17.5)	.079
Platelet (10^3 µL)	251.5 (156–490)	278.5 (171–490)	**.002**
CRP (mg/L)	2.7 (0.42–48)	2.4 (0.40–33)	.064
NLR	1.91 (0.21–50)	1.5 (0.52–5.06)	**<.001**
PLR	112.3 (57.8–3366.6)	110.5 (46.5–340.4)	.528
SII	484.2 (53.7–1515)	418.1 (141.8–1532)	**.024**
SIRI	0.90 (0.10–26.5)	0.65 (0.18–4.28)	**<.001**
PIV	223.7 (24.2–2404.9)	181.1 (41.1–958.9)	**.018**

Bold values are statistically significant.
CRP = C-reactive protein, NLR = neutrophil lymphocyte ratio, PIV = pan-immune inflammation value, PLR = platelet lymphocyte ratio, SII = systemic inflammatory index, SIRI = systemic inflammatory response index.
* The Mann–Whitney *U* test was performed for NLR, PLR, SII (calculated as neutrophil multiplied by platelet divided by lymphocyte count), SIRI (calculated as neutrophil multiplied by monocyte divided by lymphocyte count), PIV (calculated as neutrophil multiplied by platelet multiplied by monocyte divided by lymphocyte count), and CRP.

female while 55.7% were male. The control group had an average age of 45.6 years with a standard deviation of 8.6, and 47.1% of the participants were female while 52.9% were male. There were no statistically significant differences between the groups concerning age and gender (see Table 1). The median PASI score was recorded as 7.9.A notable discovery from our research was the indication of a direct relationship between SII and PASI scores. There was a statistically notable contrast observed between the 2 groups concerning neutrophils, lymphocytes, monocytes, platelet ($P < .05$). The median (min–max) results of hemogram parameters and indexes across the groups displays in Table 2.

No notable distinction was observed between the patient and control groups concerning WBC, hemoglobin, and CRP levels ($P > .05$). Moreover, inflammatory indices were computed for both the patient and control groups. There was no statistically significant distinction observed in the PLR indices between the groups ($P > .05$, Table 2). However, NLR, SII, SIRI, and PIV were found to be statistically significant ($P < .05$, Table 2). The area under the curve (AUC) of the SII score for psoriasis was 0.611. The optimal threshold for predicting psoriasis activation using SII was determined to be 442.7, with a sensitivity of 55.7% and specificity of 45.7% (95% confidence interval: 0.518–0.704, $P = .024$; Fig. 1). A positive association was noted between SII and PASI scores ($P = .004$; $r = 0.34$; Fig. 2).

The AUC of the PIV score for Psoriasis was 0.616. The optimal threshold for predicting psoriasis activation using PIV was determined to be 192.9, with a sensitivity of 57.1% and specificity of 44.3% (95% confidence interval: 0.523–0.709, $P = .018$; Fig. 3). A positive association was noted between PIV and PASI scores ($P = .006$; $r = 0.32$; Fig. 4).

4. Discussion

This study represents the initial investigation to collectively analyze hemogram parameters and novel inflammatory indices such as NLR, PLR, SII, SIRI, and PIV in patients with psoriasis. The findings from our current study suggest that both SII and PIV are associated with psoriasis activation. Additionally, this innovative index shows promise for predicting disease activation in psoriasis patients. In this retrospective case-control study, our objective was to contrast hemogram parameters and newly derived inflammatory indices between the patient and control subjects. Lately, clinicians have increasingly turned to inflammation-based indexes like NLR, PLR, SII, SIRI, and PIV to evaluate disease activity and prognostic indicators across different types of inflammatory conditions.[17–21,23–25] Although the etiopathogenesis of psoriasis is a complex disease, it is basically an immune-mediated disease. Therefore, the evaluation of inflammation in this disease is very important for clinicians both

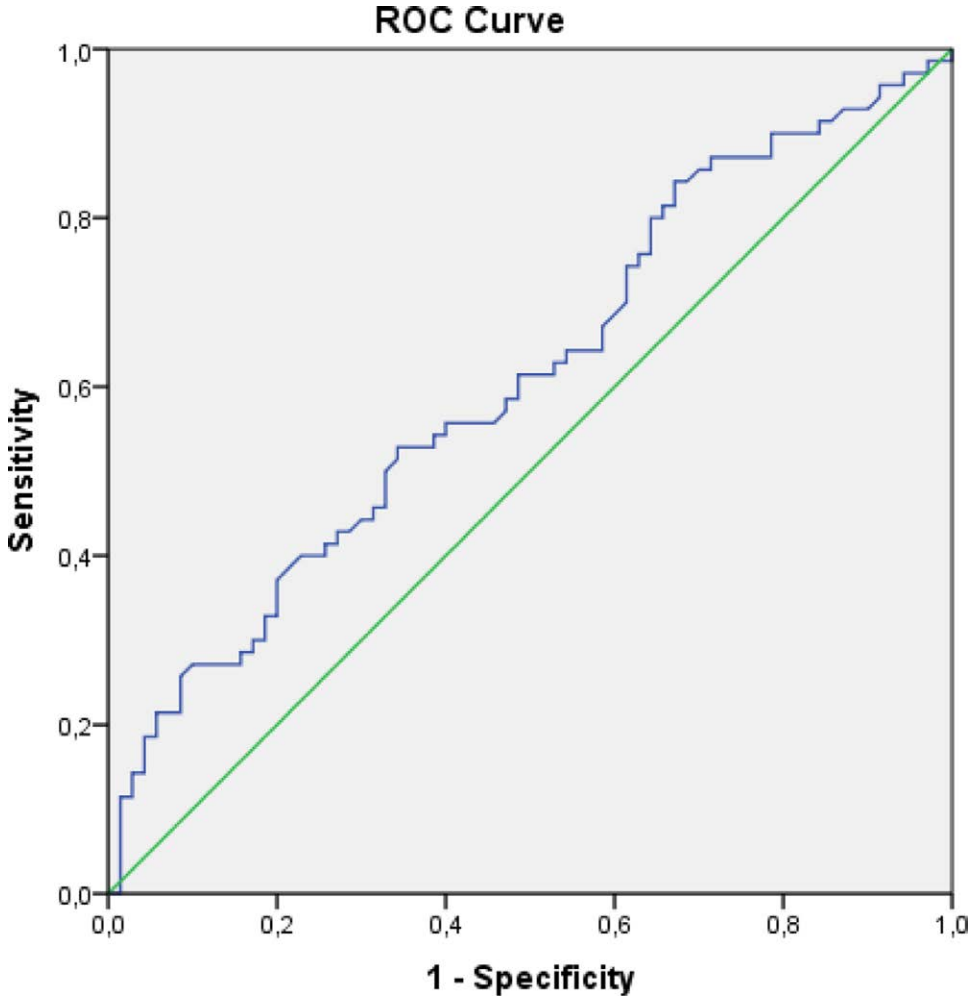

Figure 1. Receiver operating characteristic curve analysis for assessing the performance of SII in the prediction of psoriasis activation. A cutoff value of 442.7 was calculated with 55.7% sensitivity and 45.7% specificity (AUC was 0.611 and $P = .024$). AUC = area under the curve, ROC = receiver operating characteristic, SII = systemic inflammatory index.

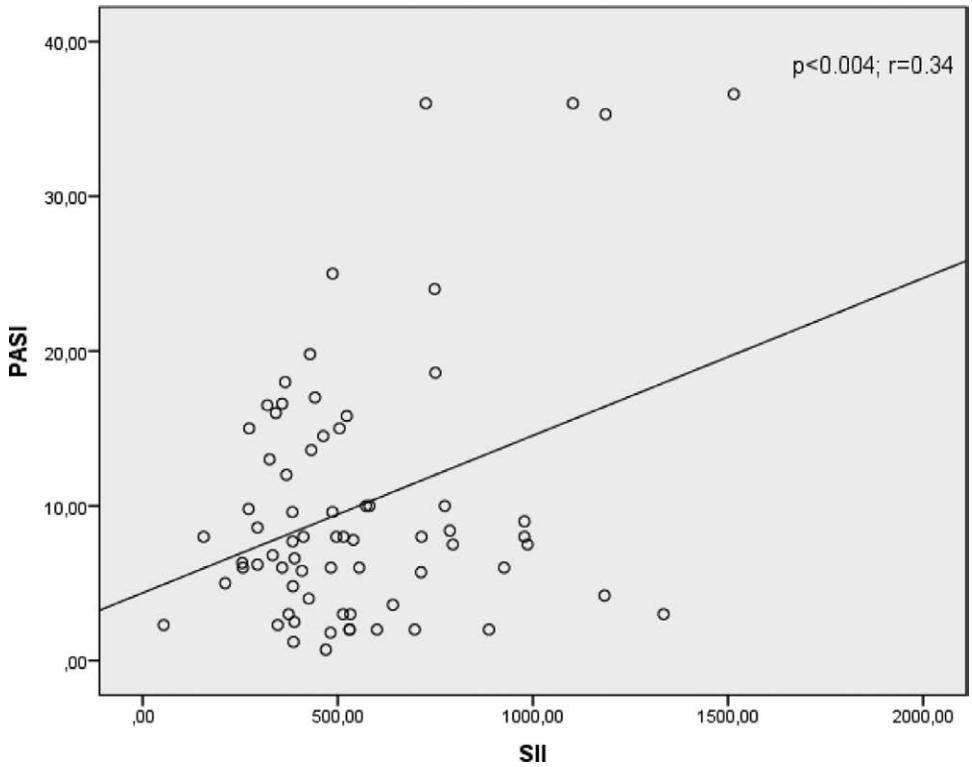

Figure 2. The positive correlation between SII and PASI. A positive association was noted between SII and PASI scores (P = .004; r = 0.34). PASI = psoriasis area and severity index, SII = systemic inflammatory index.

in evaluating the severity of the disease and in guiding more effective management of the disease.[26]

In this study, significant statistical differences were observed in NLR, SII, SIRI, and PIV between the groups. Furthermore, there was a significant correlation observed between SII, PIV and PASI scores in individuals diagnosed with psoriasis. Our findings are strongly correlate with the evidence that psoriasis is a systemic inflammatory disease.[27–29] In a population-based study by Zhao et al examining the latent link between psoriasis and SII, it was noted that a notable and positive correlation between SII and psoriasis and as SII tertiles increased, the risk of psoriasis demonstrated an upward trend. Furthermore, they found that a significant association between SII and psoriasis, characterized by 2 consecutive inverted U-shaped patterns and their analysis revealed the most prominent inflection point at a specific value of 797.067. Their conclusion suggested that the results indicate a significant correlation between elevated SII levels and the presence of psoriasis.[30]

Yorulmaz et al's research on psoriasis patients indicated that SII levels were elevated in individuals with moderate/severe psoriasis compared to those with mild psoriasis. In addition, they also reported that SII, NLR, and PLR levels were higher in patients with arthritis than in those without. They concluded that SII could serve as an autonomous prognostic marker for individuals with psoriasis and psoriatic arthritis.[31] In our current investigation, SII, SIRI, PIV, and NLR levels exhibited statistically significant elevations in psoriasis patients compared to the control group. We indicated that SII and PIV could potentially serve as a prognostic marker. In a retrospective case-control study by Dincer Rota and Tanacan examining the utility of the systemic immune inflammation index (SII) in treating psoriasis patients, it was noted that individuals with psoriasis exhibited significantly elevated SII values. Additionally, they found that a cutoff value of 575.8 yielded 66.7% sensitivity and 66% specificity for identifying psoriasis activation. Their conclusion suggested that SII could potentially serve as a predictor for psoriasis activation.[32] Another study conducted by Kelesoglu Dincer and Sezer reported that the NLR, PLR, MLR, and SII were significantly higher in psoriatic arthritis (PsA) patients compared to healthy control. They demonstrated that SII values were notably elevated in PsA patients exhibiting moderate to severe disease activity, as per DAPSA scores, compared to those in remission or with low disease activity. Their findings suggested that SII could potentially serve as a convenient, practical, cost-effective, and easily accessible means of monitoring disease activity and treatment effectiveness in PsA patients.[33] Melikoglu and Pala showed significantly higher levels of SII in patients with psoriasis than in healthy controls. They also reported that there was no difference between SII and types of psoriasis. In addition, they indicated that systemic immune inflammation index > 510, 69 was determined as a cutoff point with a sensitivity of 52.5% and specificity of 82.5%. They hypothesized that SII may be a supportive method in clinical settings.[34]

The significance of PIV has been explored extensively in malignant diseases as well as rheumatologic conditions such as rheumatoid arthritis, familial mediterranean fever, and vasculitis.[21,23–25] However, its role as a marker in dermatologic diseases is relatively novel. In a study by Öksüm Solak et al, investigating the association between systemic immune inflammation markers and disease severity in patients with hidradenitis suppurativa (HS) compared to healthy controls, it was observed that neutrophil-to-lymphocyte ratio (NLR), systemic immune-inflammation index (SIII), and PIV values were notably elevated in HS patients compared to the control group. Furthermore, these markers exhibited a positive correlation with disease severity.[35] Similarly, Erdal and Gunaydin examined the relationship between systemic immune-inflammation response index (SIRI) and PIV values in patients with chronic spontaneous urticaria and healthy controls, revealing significantly higher SIRI and PIV levels in chronic spontaneous urticaria patients.[17] Notably, literature on the association between PIV and dermatologic diseases is limited, with only 1 study identified for psoriasis. In

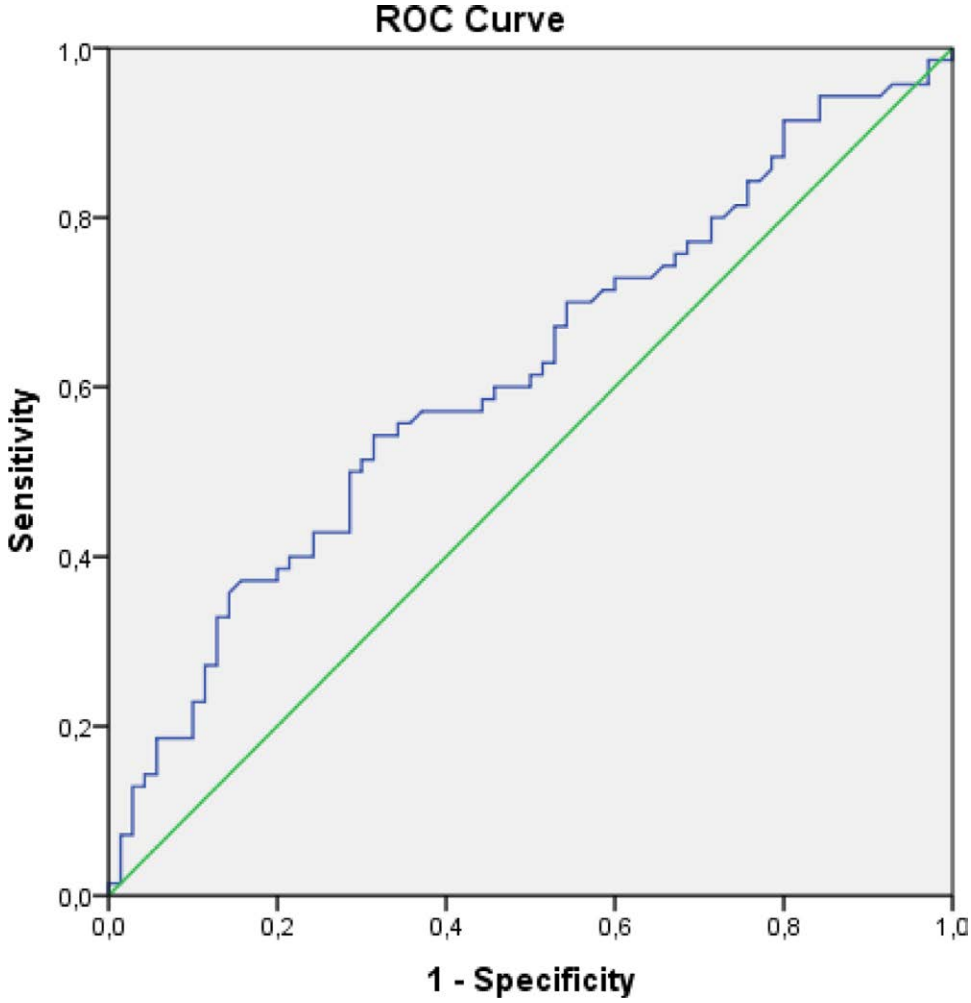

Figure 3. Receiver operating characteristic curve analysis for assessing the performance of PIV in the prediction of psoriasis activation. A cutoff value of 192.9 was calculated with 57.1% sensitivity and 44.3% specificity (AUC was 0.616 and *P* = .018). AUC = area under the curve, ROC = receiver operating characteristic, PIV = pan-immune inflammation value.

this study Gambichler et al examining the systemic immune-inflammation biomarkers and the novel pan-immune-inflammation value (PIV) in healthy controls, HS and psoriasis patients under treatment with interleukin 17A-inhibitors, it was noted that significantly higher NLR, SII, and PIV values in psoriasis and HS patients when compared to HC. Furthermore, they found that a PIV (*P* = .032, odds ratio 1.17 95% CI 1.001–1.01) were independent predictors for generalized pustular psoriasis subtype. Their conclusion suggested that the results indicate that In psoriasis patients, high levels of systemic inflammation and impaired quality of life are reflected by high SIIB values and PIV appears to be independent predictor for the generalized pustular psoriasis-subtype among psoriasis patients.[36]

5. Conclusion

In conclusion, SII, SIRI, and PIV emerge as novel inflammatory indices applicable for assessing psoriasis patients. SII holds promise for predicting disease activation, presenting a valuable, cost-effective, and practical aid for clinicians in treatment planning. Consequently, incorporating hemogram parameters and combined inflammatory indices may offer valuable insights into monitoring psoriasis progression.

Author contributions

Conceptualization: Seyma Basar Kilic, Huseyin Erdal.
Data curation: Seyma Basar Kilic, Huseyin Erdal.
Formal analysis: Seyma Basar Kilic, Huseyin Erdal.
Funding acquisition: Seyma Basar Kilic, Huseyin Erdal.
Investigation: Seyma Basar Kilic, Huseyin Erdal.
Methodology: Seyma Basar Kilic, Huseyin Erdal.
Project administration: Seyma Basar Kilic, Huseyin Erdal.
Resources: Seyma Basar Kilic, Huseyin Erdal.
Software: Seyma Basar Kilic, Huseyin Erdal.
Supervision: Seyma Basar Kilic, Huseyin Erdal.
Validation: Seyma Basar Kilic, Huseyin Erdal.
Visualization: Seyma Basar Kilic, Huseyin Erdal.
Writing – original draft: Seyma Basar Kilic, Huseyin Erdal.
Writing – review & editing: Seyma Basar Kilic, Huseyin Erdal.

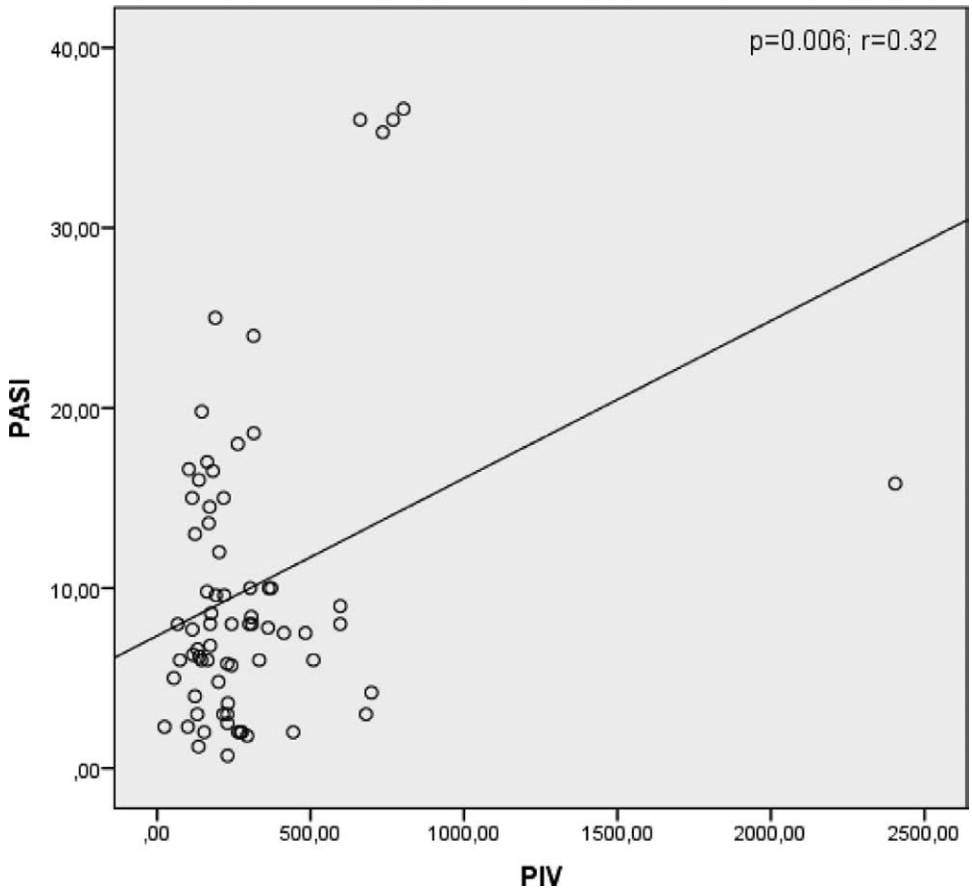

Figure 4. The positive correlation between PIV and PASI. A positive association was noted between PIV and PASI scores (P = .006; r = 0.32). PASI = psoriasis area and severity index, PIV = pan-immune inflammation value.

References

[1] Pathirana D, Ormerod AD, Saiag P, et al. European S3-guidelines on the systemic treatment of psoriasis vulgaris. J Eur Acad Dermatol Venereol. 2009;23(Suppl 2):1–70.

[2] Griffiths CE, Barker JN. Pathogenesis and clinical features of psoriasis. Lancet. 2007;370:263–71.

[3] Kamiya K, Kishimoto M, Sugai J, Komine M, Ohtsuki M. Risk factors for the development of psoriasis. Int J Mol Sci. 2019;20:4347.

[4] Singh R, Koppu S, Perche PO, Feldman SR. The cytokine mediated molecular pathophysiology of psoriasis and its clinical implications. Int J Mol Sci. 2021;22:12793.

[5] Hawkes JE, Chan TC, Krueger JG. Psoriasis pathogenesis and the development of novel targeted immune therapies. J Allergy Clin Immunol. 2017;140:645–53.

[6] Tashiro T, Sawada Y. Psoriasis and systemic inflammatory disorders. Int J Mol Sci . 2022;23:4457.

[7] Aksoy M, Kirmit A. Thiol/disulphide balance in patients with psoriasis. Postepy Dermatol Alergol. 2020;37:52–5.

[8] Bacchetti T, Campanati A, Ferretti G, Simonetti O, Liberati G, Offidani AM. Oxidative stress and psoriasis: the effect of antitumour necrosis factor-α inhibitor treatment. Br J Dermatol. 2013;168:984–9.

[9] Erdal H, Turgut F. Thiol/disulfide homeostasis as a new oxidative stress marker in patients with Fabry disease. J Investig Med. 2023;71:865–70.

[10] Genc SO, Erdal H. Effect of mode of delivery on neonatal oxidative stress and dynamic thiol-disulfide homeostasis. J Int Med Res. 2023;51:3000605231202145.

[11] Demirtas MS, Kilicbay F, Erdal H, Tunc G. Oxidative stress levels and dynamic thiol-disulfide balance in preterm newborns with bronchopulmonary dysplasia. Lab Med. 2023;54:587–92.

[12] Demirtas MS, Erdal H, Kilicbay F, Tunc G. Association between thiol-disulfide hemostasis and transient tachypnea of the newborn in late-preterm and term infants. BMC Pediatr. 2023;23:135.

[13] Erdal H, Demirtas MS, Tuncer SC, Ozcan O. Thiol/disulfide homeostasis as a new oxidative stress marker in patients with neonatal transient tachypnea. Ann Clin Anal Med. 2023;14:208–11.

[14] Bakshi H, Nagpal M, Singh M, Dhingra GA, Aggarwal G. Treatment of psoriasis: a comprehensive review of entire therapies. Curr Drug Saf. 2020;15:82–104.

[15] Elmets CA, Leonardi CL, Davis DMR, et al. Joint AAD-NPF guidelines of care for the management and treatment of psoriasis with awareness and attention to comorbidities. J Am Acad Dermatol. 2019;80:1073–113.

[16] Golbari NM, Porter ML, Kimball AB. Current guidelines for psoriasis treatment: a work in progress. Cutis. 2018;101(3S):10–2.

[17] Erdal H, Gunaydin FE. Are pan-immune inflammation value, systemic inflammatory response index clinically useful to predict in patients with chronic spontaneous urticaria? Ann Clin Anal Med. 2023;14:897–900.

[18] Genc SO, Erdal H. Are pan-immune-inflammation value, systemic inflammatory response index and other hematologic inflammatory indexes clinically useful to predict first-trimester pregnancy loss? Ann Clin Anal Med. 2023;14:473–7.

[19] Gunaydin FE, Erdal H. Is there a relationship between systemic immune-inflammatory indices and asthma? Ann Clin Anal Med. 2023;14:840–3.

[20] Akbulut Yagcı B, Erdal H. Can pan-immune inflammation value and systemic inflammatory response index be used clinically to predict inflammation in patients with cataract? Ann Clin Anal Med. 2023;14:1064–7.

[21] Chen JH, Zhai ET, Yuan YJ, et al. Systemic immune-inflammation index for predicting prognosis of colorectal cancer. World J Gastroenterol. 2017;23:6261–72.

[22] Langley RG, Ellis CN. Evaluating psoriasis with psoriasis area and severity index, psoriasis global assessment, and lattice system physician's global assessment. J Am Acad Dermatol. 2004;51:563–9.

[23] Lee LE, Ahn SS, Pyo JY, Song JJ, Park YB, Lee SW. Pan-immune-inflammation value at diagnosis independently predicts all-cause mortality in patients with antineutrophil cytoplasmic antibody-associated vasculitis. Clin Exp Rheumatol. 2021;39:88–93.

[24] Ulutaş F, Muhammed A. Pan-immune-inflammation value in FMF patients. Med Sci Discov. 2023;10:364–7.
[25] Başaran PO, Dogan M. The relationship between disease activity with pan-immune-inflammatory value and systemic immune-inflammation index in rheumatoid arthritis. Medicine (Baltimore). 2024;103:e37230.
[26] Branisteanu DE, Cojocaru C, Diaconu R, et al. Update on the etiopathogenesis of psoriasis (Review). Exp Ther Med. 2022;23:201.
[27] Zhou X, Chen Y, Cui L, Shi Y, Guo C. Advances in the pathogenesis of psoriasis: from keratinocyte perspective. Cell Death Dis. 2022;13:81.
[28] Babaie F, Omraninava M, Gorabi AM, et al. Etiopathogenesis of psoriasis from genetic perspective: an updated review. Curr Genomics. 2022;23:163–74.
[29] Grän F, Kerstan A, Serfling E, Goebeler M, Muhammad K. Current developments in the immunology of psoriasis. Yale J Biol Med. 2020;93:97–110.
[30] Zhao X, Li J, Li X. Association between systemic immune-inflammation index and psoriasis: a population-based study. Front Immunol. 2024; 15:1305701.
[31] Yorulmaz A, Hayran Y, Akpinar U, Yalcin B. Systemic immune-inflammation index (SII) predicts increased severity in psoriasis and psoriatic arthritis. Curr Health Sci J. 2020;46:352–7.
[32] Dincer Rota D, Tanacan E. The utility of systemic-immune inflammation index for predicting the disease activation in patients with psoriasis. Int J Clin Pract. 2021;75:e14101.
[33] Kelesoglu Dincer AB, Sezer S. Systemic immune inflammation index as a reliable disease activity marker in psoriatic arthritis. J Coll Physicians Surg Pak. 2022;32:773–8.
[34] Melikoglu M, Pala E. Systemic immune-inflammation index as a biomarker of psoriasis severity. Arch Basic Clin Res. 2023;5:291–5.
[35] Öksüm Solak E, Baran Ketencioglu B, Cinar SL, Kartal D, Borlu M. The role of new inflammatory markers in determining disease activation and severity in patients with hidradenitis suppurativa. Int J Dermatol. 2023;62:1076–81.
[36] Gambichler T, Würfel L, Abu Rached N, Mansour R, Bechara FG, Scheel CH. Systemic immune-inflammation biomarkers in psoriasis patients under interleukin 17A-inhibitor treatment. J Eur Acad Dermatol Venereol. 2023;37:e837–9.