Ankyra Medical Journal
Original Article
DOI: 10.51271/ANKMJ-0046

Relationship between pan-immune-inflammation value, systemic immune inflammation index, and systemic inflammation response index in patients with rheumatoid arthritis

Emre Ersoy[1], Duygu Felek[2]
[1]*Department of Physical Therapy and Rehabilitation, Faculty of Medicine, Yozgat Bozok University, Yozgat, Turkiye*
[2]*Department of Internal Medicine, Faculty of Medicine, Yozgat Bozok University, Yozgat, Turkiye*

Cite this article: Ersoy E, Felek D. Relationship between pan-immune-inflammation value, systemic immune inflammation index, and systemic inflammation response index in patients with rheumatoid arthritis. *Ank Med J.* 2025;4(3):45-50.

Received: 03/07/2025 ◆ **Accepted**: 26/07/2025 ◆ **Published**: 29/07/2025

ABSTRACT

Aims: The aim of this study was to develop easily applicable tools that reflect systemic inflammation in rheumatoid arthritis (RA). In this context, the relationship between RA disease activity and pan-immune-inflammation value (PIV), systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI) was examined.

Methods: Patients and healthy controls who applied to Yozgat Bozok University Physical Medicine and Rehabilitation and Internal Medicine clinics between 01.01.2020 and 04.01.2025 were included in the study. Visual Analog Scale (VAS), Disease Activity Score-28 (DAS28), hemogram, and biochemistry parameters—including ALT, AST, fasting glucose, C-reactive protein (CRP), erythrocyte sedimentation rate, uric acid, creatinine, calcium, magnesium, alkaline phosphatase, parathyroid hormone, lipid profile, albumin, total protein, T4, TSH, rheumatoid factor (RF), and anti-cyclic citrullinated peptide (anti-CCP)—were retrospectively recorded from patient files. PIV, SII, and SIRI were calculated using complete blood count data from both the RA and control groups. Data were analyzed using SPSS, and a significance level of 0.05 was considered statistically significant.

Results: SII, SIRI, and PIV values were significantly higher in the RA group compared to the control group (p=0.002, p=0.001, and p=0.001, respectively). Among the three disease activity groups, SII, SIRI, and PIV levels were highest in the active disease group. A positive correlation was found between DAS28 and SII (r=0.305, p=0.012), and between DAS28 and PIV (r=0.270, p=0.028). However, no significant correlation was observed between DAS28 and SIRI (p=0.111). The difference among the activity groups was statistically significant for SII and PIV (p=0.016 and p=0.039, respectively), but not for SIRI (p=0.171). Furthermore, SII and PIV levels were significantly higher in patients receiving anti-TNF-α treatment compared to those using DMARDs (p=0.001 and p=0.003, respectively).

Conclusion: The significantly higher SII and PIV values in the RA group compared to controls, and their positive correlation with DAS28, suggest that these indices may be associated with RA disease activity. Additionally, the lower levels of SII and PIV in patients receiving anti-TNF-α treatment support their potential role in monitoring treatment response.

Keywords: Rheumatoid arthritis, disease activity, pan-immune-inflammation value, systemic immune-inflammation index, systemic inflammation response index

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic, systemic inflammatory disorder that predominantly targets small joints, resulting in both structural damage and functional limitations, and ultimately diminishing quality of life.[1] The precise cause of RA remains unknown; however, the disease is characterized by inflammation that initially affects the synovial membrane and progressively damages the underlying subchondral bone and cartilage. This pathological process promotes the development of pannus tissue, which plays a key role in joint deformities and irreversible damage. Although the exact mechanisms underlying RA pathogenesis are not fully clarified, immune system dysregulation is believed to be central to disease progression. Accurate assessment of disease activity is essential not only to avoid serious complications but also to initiate timely and effective therapeutic interventions.[2] DAS28 is a widely used assessment tool for determining disease activity in RA and monitoring response to treatment.[3]

Corresponding Author: Duygu Felek, d.kocamemik@gmail.com

RA is an inflammatory condition involving the immune system. Currently, there is no single laboratory test that definitively confirms the diagnosis of RA. While erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP) are frequently used to assess inflammation in RA, their diagnostic accuracy is limited due to low sensitivity and specificity.[4] As a result, recent research has focused on identifying new immune-based prognostic indicators such as the monocyte-to-lymphocyte ratio (MLR), neutrophil-to-lymphocyte ratio (NLR), and platelet-to-lymphocyte ratio (PLR) in the context of RA.[5] Although each of these immune cell types contributes to the inflammatory process, none is sufficient on its own to accurately reflect the overall inflammatory status. Therefore, more integrated indices have been developed that combine these parameters. One such index is the pan-immune-inflammation value (PIV), which is calculated using complete blood count (CBC) data including neutrophils, platelets, monocytes, and lymphocytes and is used to assess the degree of systemic inflammation. PIV has been shown to serve as a prognostic marker in several types of cancer.[6]

The systemic immune inflammation index (SII) increases with relatively high neutrophil and platelet counts and low lymphocyte counts, which is considered an indicator of a strong inflammatory response.[7] SII has been evaluated in diseases such as lupus, psoriatic arthritis, and RA, and is associated with disease activity levels.[6,8,9] The systemic inflammation response index (SIRI) represents the interplay between inflammatory activity and immune function.[10] Several studies have emphasized the importance of SIRI as a biomarker in both the onset and progression of various types of cancer.[11,12]

The aim of our study is to develop easily applicable tools that reflect systemic inflammation in RA. Despite the growing interest in systemic inflammation markers, studies that specifically compare the relationship between RA and indices such as PIV, SII, and SIRI remain limited; therefore, the present study aims to investigate the association between rheumatoid arthritis disease activity and the calculated values of SII, SIRI, and PIV.

METHODS

Ethics

The study was carried out with the permission of the Yozgat Bozok University Non-interventional Clinical Researches Ethics Committee (Date: 04.06.2025, Decision No: 2025-GOKAEK-2511_2025.06.04_526). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki. This study included patients who presented to the Physical Medicine and Rehabilitation and Internal Medicine clinics of Yozgat Bozok University between January 1, 2020, and January 4, 2025, and was retrospectively analyzed.

Inclusion Criteria

Diagnosis of RA according to the American College of Rheumatology (ACR) 2010 classification criteria.[13]

- Age between 18 and 75 years

Exclusion Criteria

- Age under 18 or over 75 years
- Historyof malignancy (cancer) or presence of active malignany
- Active infection
- Immunodeficiency
- Presence of hematological diseases or other systemic inflammatory diseases
- Use of steroids or cytotoxic drugs

A healthy control group was also included for comparison. In addition, during the same years, individuals who had applied to the mentioned clinics and tested negative for RA were also included in the study as healthy male and female control subjects.

Inclusion Criteria

- Age between 18 and 75 years

Exclusion Criteria

- Age under 18 or over 75 years
- Historyof malignancy (cancer) or presence of active malignany
- Active infection
- Immunodeficiency
- Presence of hematological diseases or other systemic inflammatory diseases
- Use of steroids or cytotoxic drugs

Inflammatory Indices

PIV, SII, and SIRI values were calculated from CBC data in patients diagnosed with RA and in the control group.[14]

These markers are formulated as follows:

PIV=Neutrophils (10^9/L)×platelets (10^9/L)×monocytes (10^9/L)/lymphocytes (10^9/L)

SII=Neutrophils (10^9/L)×platelets (10^9/L)/lymphocytes (10^9/L)

SIRI=Neutrophils (10^9/L)×monocytes (10^9/L)/lymphocytes (10^9/L)

Demographic Data and Laboratory Parameters

For the patients diagnosed with RA included in the study, data were retrospectively collected from patient records, including age, gender, disease history, medications used, and available clinical scores such as The Visual Analog Scale (VAS) and DAS28. Laboratory results recorded included CBC and biochemical parameters such as ALT (U/L), AST (U/L), fasting glucose (mg/dl), CRP (mg/L), sedimentation rate, uric acid (mg/dl), creatinine (mg/dl), calcium, magnesium, alkaline phosphatase, parathormone, lipid profile, albumin, total protein, thyroid function tests (T4 and TSH), rheumatoid factor (RF), and anti-citrullinated peptide antibody (Anti-CCP). Only the parameters that were reviewed and documented in the patient files were included in the study.

VAS

VAS is a reliable and valid tool used to measure pain intensity on a single continuum. This scale consists of a 10-centimeter horizontal line with endpoints labeled “no pain” and “the worst imaginable pain.” Patients mark a point on the line

that best represents their current level of pain. The score is determined by measuring the distance in centimeters from the "no pain" end to the patient's mark, yielding a value between 0 and 10.[15]

DAS28

The DAS28 is a clinical scoring system used to assess disease activity in patients with RA. This score evaluates how active the disease is by considering the number of tender and swollen joints out of 28 specified joints, the patient's self-assessment of their health (usually measured on a visual analog scale ranging from 0 to 100), and inflammatory markers such as ESR or CRP. In our study, we used the DAS28 score calculated with ESR. The formula used for DAS28 calculation was:

DAS28=0.56×√(number of tender joints out of 28)+0.28×√number of swollen joints out of 28)+0.70×ln(ESR)+0.014×patient's global health assessment.

Based on DAS28 scores, patients were categorized into three groups:

DAS28≤3.2: Low disease activity or remission

3.2<DAS28≤5.1: Moderate disease activity

DAS28>5.1: High disease activity or active disease.[16]

Statistical Analysis

The data analyses were performed using SPSS version 20.0 (Statistical Package for the Social Sciences, SPSS Inc., Chicago, IL). Descriptive statistics were presented as mean±standard deviation for continuous variables and as percentages for categorical variables. The Kolmogorov-Smirnov test was used to assess the normality of distribution for the groups. For comparisons between groups, independent samples t-test and ANOVA were used for normally distributed continuous variables, while the chi-square test was applied for categorical variables. In cases where the data were not normally distributed, the Mann-Whitney U test and Kruskal-Wallis test were employed. Correlation analyses (Pearson or Spearman) were conducted to evaluate relationships between quantitative variables. A p-value less than 0.05 was considered statistically significant.

RESULTS

The study included 63 patients with RA (42 females [66%], 21 males [34%]) and 50 healthy controls (32 females [64%], 18 males [36%]). There was no statistically significant difference in gender distribution between the groups (p>0.05). The mean age of all participants was calculated as 63.50±2.12 years. No statistically significant difference was observed between the patient and control groups regarding mean age (p=0.15). In the patient group, the mean disease duration was 12.95±8.94 years; the mean RF level was 171.61±50.60; and the mean Anti-CCP level was 339.33±66. In the patient group, SII, SIRI, and PIV values were found to be statistically higher compared to the control group, and the differences were statistically significant (p=0.002, p=0.001, and p=0.001, respectively) (Table 1).

When patients were classified according to their DAS28 scores into remission, moderate activity, and active disease groups, 24 patients (38%) were in remission, 18 patients (29%) had moderate disease activity, and 21 patients (33%) had active disease. Approximately 69% of the patients were using DMARDs (disease-modifying anti-rheumatic drugs), while 31% were receiving anti-TNF-α therapy. Among those on DMARDs, 80% were using methotrexate, 10% were on other DMARDs besides methotrexate, and 10% were on combination DMARD therapy with methotrexate and another DMARD. Among the patients receiving Anti-TNF-α treatment, 9 were using etanercept, 5 infliximab, 3 golimumab, and 2 adalimumab. Figure 1 displays distribution of disease activity according to DAS Scores. Figure 2 displays distribution of medications used.

Table 1. Mean SII, SIRI, and PIV values of patient and control groups

		Mean	SD	p
SII	Patient	971.11	913.848	**0.002**
	Control	567.22	476.282	
SIRI	Patient	1.594	1.3895	**0.001**
	Control	1.000	.8814	
PIV	Patient	497.15	484.944	**0.001**
	Control	239.36	178.742	

SD: Standard deviation, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index, PIV: Pan-immune-inflammation value

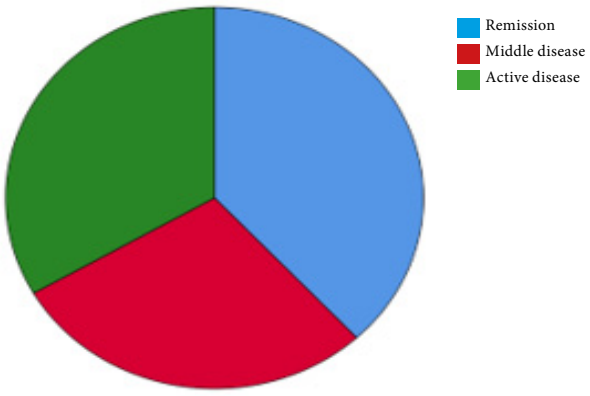

Figure 1. Distribution of disease activity according to DAS scores
DAS: Disease Activity Score

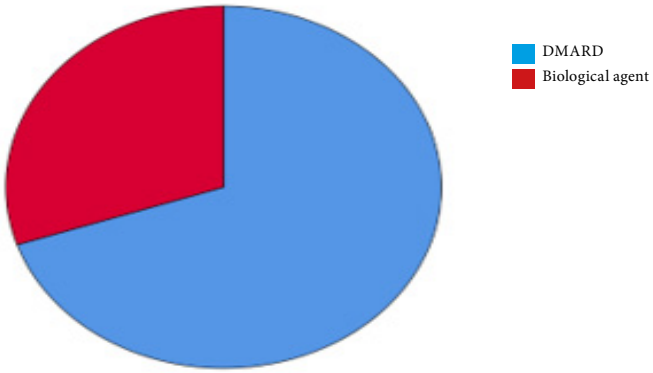

Figure 2. Distribution of medications used

When evaluating the three groups according to disease activity in terms of inflammatory indices, the active disease group showed the highest levels of SII, SIRI, and PIV. The differences between groups were statistically significant for SII and PIV (p=0.016 and p=0.039, respectively), whereas the difference in SIRI levels was not statistically significant (p=0.171) (Table 2). In pairwise comparisons, there were no statistically significant differences in SII, SIRI, and PIV values between the remission and moderate disease activity

groups (p=0.170, 0.819, and 0.322, respectively). Comparing remission and active disease groups, significant differences were observed in SII and PIV values (p=0.013 and p=0.022, respectively), but no significant difference was found for SIRI (p=0.099). Between the moderate and active disease groups, SII levels differed significantly (p = 0.032), while no statistically significant differences were found for SIRI and PIV (p=0.119 and p=0.065, respectively) (Table 2).

Table 2. SII, SIRI, and PIV values according to disease activity

Disease activity	SII (mean±SD)	SIRI (mean±SD)	PIV (mean±SD)
Remission	799.44±909.98	1.35±1.13	404.24±484.44
Moderate	747.93±387.35	1.33±0.90	389.97±221.21
Active	1358.60±1127.	2.10±1.85	695.20±596.38

SD: Standard deviation, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index, PIV: Pan-immune-inflammation value

Patients receiving anti-TNF-α therapy had significantly higher SII and PIV levels compared to those using DMARDs (p=0.001 and p=0.003, respectively). However, no significant difference was observed between the two groups in terms of SIRI levels (p=0.116) (Table 3).

Table 3. SII, SIRI, and PIV values according to current treatments in RA patients

Treatment	Index	Mean	SD
DMARD	SII	1148.44	1028.46
	SIRI	1.75	1.51
	PIV	585.59	585.15
Anti-TNF-α	SII	580.47	307.99
	SIRI	1.23	1.01
	PIV	293.37	203.4

DMARD: Disease-modifying anti-rheumatic drugs, SD: Standard deviation, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index, PIV: Pan-immune-inflammation value, RA: Rheumatoid arthritis

A moderate positive correlation was found between DAS28 and SII, and a weak positive correlation between DAS28 and PIV (r=0.305, p=0.012; r=0.270, p=0.028, respectively). However, no significant correlation was observed between DAS28 and SIRI (p=0.111). CRP levels showed a moderate positive correlation with SII (r=0.321, p<0.001). RF demonstrated a weak positive correlation with both SII and SIRI, and a moderate positive correlation with PIV (r=0.250, p=0.043; r=0.291, p=0.017; r=0.333, p=0.006, respectively). Additionally, RF levels showed a moderate positive correlation with DAS28 (r=0.341, p=0.005) (Table 4).

DISCUSSION

In this study, we analyzed and compared several inexpensive, simple, and easily accessible inflammation markers derived from CBC, focusing on PIV, SII, and SIRI. While markers like NLR and PLR have been extensively studied in RA, data on PIV, SII, and SIRI remain limited. Our findings showed that systemic inflammation indices SII, PIV, and SIRI were significantly elevated in RA patients compared to controls, with the highest levels observed in the active disease group. Importantly, positive correlations between SII and PIV values and the DAS28 disease activity score suggest their potential utility in assessing disease activity. Given the increasing need for simple, cost-effective, and reliable markers to monitor RA and predict complications early, these inflammation indices hold considerable promise for disease management. In our comparisons between remission and active disease groups, SII and PIV demonstrated potential predictive value, while only SII significantly differentiated between moderate and active disease activity.

The predictive value of SII and PIV regarding disease activity has been explored in previous studies.[14,17] In the study conducted by Yoshikawa et al.[18] involving 574 RA patients, a significant positive correlation was found between SII and

Table 4. Correlation of SII, SIRI, and PIV with other parameters

	DAS28	VAS	ESR	CRP	RF	CCP	SII	SIRI	PIV
DAS28	1	.555**	.420**	.431**	.341**	-.018	.305*	.203	.270*
		.000	.001	.000	.183	.891	.015	.111	.032
VAS		1	.096	.132	-.024	.022	.151	.028	.098
			.455	.302	.851	.864	.237	.828	.444
ESR			1	.516**	.336**	.303*	.432**	.250**	.388**
				.000	.007	.016	.000	.008	.000
CRP				1	.069	.006	.321**	.173	.258**
					.589	.962	.001	.067	.006
RF					1	.547**	.250*	.291*	.333**
						.000	.048	.021	.008
CCP						1	.030	.102	.050
							.818	.425	.700
SII							1	.693**	.877**
								.000	.000
SIRI								1	.864**
									.000
PIV									1

DAS28: Disease Activity Score-28, VAS: Visual Analog Scale, ESR: Erythrocyte sedimentation rate, CRP: C-reactive protein, RF: Rheumatoid factor, CCP: Citrullinated peptide antibody, SII: Systemic immune-inflammation index, SIRI: Systemic inflammation response index, PIV: Pan-immune-inflammation value, **. Correlation is significant at the 0.01 level (2-tailed), *. Correlation is significant at the 0.05 level (2-tailed).

DAS28-ESR. When patients were divided into three groups—remission, low, and high disease activity—it was observed that SII levels significantly increased with rising disease activity. The authors highlighted that, for the first time, SII demonstrated a stronger association with disease activity compared to NLR. Similarly, another study by Okutan and colleagues[19] reported that SII and PIV were significantly higher in RA patients compared to the control group, and these indices showed a positive correlation with the DAS28 score. Additionally, subgroup analyses based on disease activity revealed that SII had a significant predictive value for disease activity. The results of our study largely align with these two studies, with particular interest in emphasizing the predictive role of SII.

In our study, a moderate positive correlation was observed between CRP levels and SII. This finding suggests that SII may serve as an alternative inflammatory marker to CRP in the monitoring of RA. Indeed, in the study conducted by Dervisevic et al.,[20] SII values were significantly higher in RA patients compared to healthy individuals and showed positive correlations with hs-CRP (high sensitivity CRP), ESR, NLR, MLR, PLR, the number of tender joints, and the swollen-to-tender joint ratio. These results support the findings of our study and indicate that SII could be a meaningful tool reflecting the degree of inflammation in patients with RA.

According to current research, both SII and PIV levels were found to be lower in the group receiving anti-TNF-α therapy compared to those treated with DMARDs, suggesting that these two indices may serve as potential tools for evaluating treatment response. SII and PIV are not only associated with disease activity but may also be valuable indicators for assessing treatment effectiveness. It has been proposed that SII, alongside CRP and ESR, is an effective tool for monitoring response to TNF-α inhibitors in RA patients, with SII showing the highest predictive value among these markers for evaluating the efficacy of TNF-α inhibitors. However, in the retrospective study conducted by Bai et al,[21] PIV was not evaluated.In our study, PIV was also able to distinguish between the anti-TNF group and the DMARD group. When conventional treatments cause severe side effects or fail to achieve the desired clinical response, TNF-α inhibitors are considered alternative options for RA therapy. Widely used TNF-α inhibitors include infliximab, adalimumab, etanercept, golimumab, and certolizumab pegol, all of which aim to neutralize TNF-α and alleviate symptoms. In recent years, these agents have been shown to provide significant benefits in controlling disease activity and reducing treatment-related adverse effects.[22]

Using data from the National Health and Nutrition Examination Survey (NHANES) between 1999 and 2018, the relationship between SII and RA was investigated. A total of 37.604 individuals were included in the study, of whom 2.642 (7.03%) had an RA diagnosis. After adjusting for potential confounding variables, multivariate logistic regression analysis showed that higher SII levels were significantly associated with an increased risk of RA.[23]

Başaran and colleagues[24] investigated the association between disease activity and the levels of PIV and SII in patients with RA, aiming to determine which of these two inflammatory indices offers greater diagnostic utility. Their findings indicated that both PIV and SII levels were significantly higher in the active RA group compared to both the remission and control groups. PIV and SII levels were significantly higher in the remission group compared to the controls. In the ROC analysis for predicting remission, CRP did not show significant discriminatory ability. In contrast, both PIV and SII showed statistically significant results. Among them, PIV demonstrated higher sensitivity and specificity.[24]

In our study, a weak positive correlation was found between RF and SII, whereas a moderate positive correlation was observed between RF and PIV. It is well established that RF levels are associated with disease activity in patients with RA.[25] Moreover, fluctuations in RF titers are considered useful for monitoring both disease activity and treatment response.[26] Consistent with these findings, a positive correlation was also observed between DAS28 scores and RF levels. The associations between RF and both SII and PIV indicate that these inflammatory indices may serve as potential alternative markers for evaluating inflammatory status in RA.

No significant correlation was found between SIRI and DAS28 scores, nor was there a difference in SIRI levels between patients treated with DMARDs and those receiving anti-TNF therapy. In contrast, both PIV and SII showed significant associations with disease activity and treatment response. These findings suggest that PIV and SII are more reliable markers for monitoring RA, while the utility of SIRI appears limited.

Limitations

Our study has several limitations. We evaluated both newly diagnosed and long-term patients together. In general, most of the participants were patients receiving long-term treatment. Therefore, we were unable to assess the relationship between these markers and disease activity in patients with a shorter disease duration. The retrospective design of the study limited the ability to evaluate the prognostic significance of inflammatory indices and their utility in monitoring treatment response. Additionally, the single-center nature of the study and the relatively small sample size can also be considered as further limitations.

CONCLUSION

As a result, SII and PIV appear to be potential biomarkers capable of reflecting disease activity and monitoring treatment response in patients with RA. Notably, the sensitivity of SII to different levels of disease activity and the reduction of both indices with anti-TNF therapy highlight their clinical relevance and potential utility in practice.

ETHICAL DECLARATIONS

Ethics Committee Approval

The study was carried out with the permission of the Yozgat Bozok University Non-interventional Clinical Researches Ethics Committee (Date: 04.06.2025, Decision No: 2025-GOKAEK-2511_2025.06.04_526).

Informed Consent

Because the study was designed retrospectively, no written informed consent form was obtained from patients.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

REFERENCES

1. Rashid T, Darlington G, Kjeldsen-Kragh J, et al. Proteus IgG antibodies and C-reactive protein in English, Norwegian and Spanish patients with rheumatoid arthritis. *Clin Rheumatol*. 1999;18(3):190-195. doi:10.1007/s100670050208
2. Aletaha D, Smolen JS. Diagnosis and management of rheumatoid arthritis: a review. *JAMA*. 2018;320(13):1360-1372. doi:10.1001/jama.2018.13103
3. England BR, Tiong BK, Bergman MJ, et al. 2019 update of the American College of Rheumatology recommended rheumatoid arthritis disease activity measures. *Arthritis Care Res (Hoboken)*. 2019;71(11):1540-1555. doi:10.1002/acr.23831
4. Colglazier CL, Sutej PG. Laboratory testing in the rheumatic diseases: a practical review. *South Med J*. 2005;98(2):185-189. doi: 10.1097/01.smj.0000153103.06303
5. Erre GL, Paliogiannis P, Castagna F, et al. Meta-analysis of neutrophil-to-lymphocyte and platelet-to-lymphocyte ratio in rheumatoid arthritis. *Eur J Clin Invest*. 2019;49(10):e13037. doi:10.1111/eci.13037
6. Ulutaş F, Çobankara V. Pan-immune-inflammation value (PIIV) in lupus nephritis. *Med Sci Discov*. 2023;10(3):234-238. doi:10.17546/msd.1200
7. Baba Y, Nakagawa S, Toihata T, et al. Pan-immune-inflammation value and prognosis in patients with esophageal cancer. *Ann Surg Open*. 2021;3(1):e113. doi:10.1097/AS9.0000000000000113
8. Dincer ABK, Sezer S. Systemic immune inflammation index as a reliable disease activity marker in psoriatic arthritis. *J Coll Physicians Surg Pak*. 2022;32(6):773-778. doi:10.29271/jcpsp.2022.06.773
9. Ozdemir A, Baran E, Kutu M, et al. Could systemic immune inflammation index be a new parameter for diagnosis and disease activity assessment in systemic lupus erythematosus? *Int Urol Nephrol*. 2023;55(1):211-216. doi:10.1007/s11255-022-03355-4
10. Zhang P, Li Y, Zhang H, et al. Prognostic value of the systemic inflammation response index in patients with aneurysmal subarachnoid hemorrhage. *Br J Neurosurg*. 2020;34(1):1-7. doi:10.1080/02688697.2019.1632117
11. Chao B, Ju X, Zhang L, et al. A novel prognostic marker, systemic inflammation response index (SIRI) for operable cervical cancer patients. *Front Oncol*. 2020;10:766. doi:10.3389/fonc.2020.00766
12. Pacheco-Barcia V, Solís RM, France T, et al. A systemic inflammation response index (SIRI) correlates with survival in metastatic pancreatic cancer. *Pancreatology*. 2020;20(2):254-264. doi:10.1016/j.pan.2019.11.013
13. Aletaha D, Neogi T, Silman AJ, et al. 2010 rheumatoid arthritis classification criteria. *Arthritis Rheum*. 2010;62(9):2569-2581. doi:10.1002/art.27584
14. Tutan D, Doğan AG. Pan-immune-inflammation index as a biomarker for RA progression. *Cureus*. 2023;15(4):e46609. doi:10.7759/cureus.46609
15. Delgado DA, Lambert BS, Boutris N, et al. Validation of digital visual analog scale pain scoring with a traditional paper-based visual analog scale in adults. *J Am Acad Orthop Surg Glob Res Rev*. 2018;2(3):e088. doi:10.5435/JAAOSGlobal-D-18-00088
16. de Bandt M, Fautrel B, Maillefert JF, et al. Determining a low disease activity threshold for DMARD continuation in RA. *Arthritis Res Ther*. 2009;11(4):R157. doi:10.1186/ar2796
17. Ulutaş F, Aydın M. Pan-immune-inflammation value in FMF patients. *Med Sci Discov*. 2023;10(4):364-367. doi:10.17546/msd.1212
18. Yoshikawa T, Furukawa T, Tamura M, et al. Systemic immune-inflammation index in RA patients: relation to disease activity. *Ann Rheum Dis*. 2019;78(3):325. doi:10.1136/annrheumdis-2018-eular.3741
19. Okutan İ, Aci R, Keskin Â, et al. New inflammatory markers in RA: PIV, SII, and SIRI. *Reumatologia*. 2024;62(6):439-446. doi:10.5114/reum.2024.124231
20. Dervisevic A, Fajkic A, Jahic E, et al. Systemic immune-inflammation index in RA patients. *Medeni Med J*. 2024;39(3):183-191. doi:10.5222/MMJ.2024.183
21. Bai J, Tian Y, Wang F, et al. Role of systemic immune-inflammation index in assessing tumor necrosis factor-α inhibitor efficacy in rheumatoid arthritis. *Am J Transl Res*. 2023;15:6524-6533. doi:10.7150/ajtr.86597
22. Jang DI, Lee AH, Shin HY, et al. The role of TNF-alpha in autoimmune disease and therapeutics. *Int J Mol Sci*. 2021;22(5):2719. doi:10.3390/ijms2205271
23. Liu B, Wang J, Li YY, Li KP, Zhang Q. The association between systemic immune-inflammation index and rheumatoid arthritis: evidence from NHANES 1999–2018. *Arthritis Res Ther*. 2023; 25(1): 34. doi:10.1186/s13075-023-03018-6
24. Başaran PÖ, Doğan M. Relationship between disease activity and PIV/SII in RA. *Medicine (Baltimore)*. 2024;103(9):e37230. doi:10.1097/MD.0000000000037230
25. Peng XC, Yin R, Luo LP, Xu S, Shuai Z. Rheumatoid factor titer as an indicator of the risk of rheumatoid arthritis activity: dose-effect analysis with the restricted cubic spline model. *J Inflamm Res*. 2024;17:10699-10709. doi:10.2147/JIR.S488605
26. Mikuls TR, O'Dell JR, Stoner JA, et al. Association of rheumatoid arthritis treatment response and disease duration with declines in serum levels of IgM rheumatoid factor and anti-cyclic citrullinated peptide antibody. *Arthritis Rheum*. 2004;50(12):3776-3782. doi:10.1002/art.20659