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Original Article

The relationship between neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, systemic immune-inflammation index, and halp score with disease activity in patients with rheumatoid arthritis

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Abstract

Aim: Rheumatoid arthritis (RA) is a chronic autoimmune disorder characterized by persistent joint inflammation driven by dysregulated immune responses. Identifying reliable biomarkers of disease activity remains crucial for optimizing patient management. This study investigated whether inflammatory indices derived from routine complete blood count (CBC) parameters—namely, the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and hemoglobin–albumin–lymphocyte–platelet (HALP) score—are associated with RA disease activity.

Materials and Methods: This single-center, cross-sectional study enrolled 72 patients with rheumatoid arthritis who fulfilled the 2010 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria. Disease activity was assessed using the Disease Activity Score in 28 joints with C-reactive protein (DAS28–CRP), with remission defined as ≤2.6 and active disease as >2.6. The neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), systemic immune-inflammation index (SII), and hemoglobin–albumin–lymphocyte–platelet (HALP) score were calculated from routine hematologic and biochemical data. Associations between these indices and disease activity were examined using correlation and comparative statistical analyses.

Results: Among the 72 patients (73.6% female; mean age, 53.8 ± 11.3 years), disease activity was categorized according to DAS28–CRP (remission ≤2.6; active >2.6). Patients with active disease demonstrated significantly higher NLR, PLR, and SII values compared with those in remission. Disease activity showed a positive correlation with these indices, as well as with CRP, whereas the HALP score exhibited an inverse association with inflammatory markers.

Conclusion: NLR, PLR, and SII appear to be practical, cost-effective biomarkers for evaluating disease activity in rheumatoid arthritis. The HALP score may provide additional value by reflecting both inflammatory and nutritional status.

Keywords: Rheumatoid arthritis, neutrophil-to-lymphocyte ratio, platelet-to-lymphocyte ratio, systemic immune-inflammation index, HALP score

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic autoimmune disease that primarily affects the joints, leading over time to progressive deformities, restricted mobility, and reduced quality of life (1). Inflammation is a key driver of disease progression and a major determinant of therapeutic decisions; therefore, accurate assessment of disease activity is critical for enabling timely intervention and preventing long-term complications.

Disease activity in RA is commonly evaluated using composite indices such as the Disease Activity Score-28 (DAS28), which integrates findings from physical examinations with laboratory parameters (2). In addition to DAS28, other validated indices

include the Simplified Disease Activity Index (SDAI) and the Clinical Disease Activity Index (CDAI), both of which rely more heavily on clinical assessments and, in the case of CDAI, exclude laboratory markers entirely (3). These indices have gained popularity due to their practicality in daily clinical settings and their ability to provide complementary information regarding disease activity.

The immunopathogenesis of RA involves complex interactions among neutrophils, lymphocytes, and platelets, which contribute to both local and systemic inflammation (4). In the early stages of disease, neutrophils dominate the synovial fluid, where they exacerbate inflammation through the generation of reactive oxygen species, the release of proinflammatory cytokines, and the formation of neutrophil extracellular traps (NETs) (5). Platelets further amplify the inflammatory cascade by secreting cytokines, chemokines, and growth factors, thereby sustaining the immune response.

Microparticles circulating in the bloodstream can carry autoantigens that influence coagulation and immune regulation (6). In rheumatoid arthritis, lymphocytes exhibit altered functionality, increased proliferation, and dysregulated cytokine signaling (7). These immune alterations underscore the relevance of hematologic biomarkers for assessing disease progression.

Readily available and cost-effective indices derived from complete blood count (CBC) parameters are under investigation as potential tools for improving the accuracy and comprehensiveness of disease activity assessment in RA. Among these, immune cell ratios such as the neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and monocyte-to-lymphocyte ratio (MLR) have been widely used to reflect systemic inflammation and are significantly associated with RA disease activity (8,9).

The systemic immune-inflammation index (SII), calculated from neutrophil, platelet, and lymphocyte counts, quantifies overall inflammatory burden by capturing the balance among these immune cell types (10). Similarly, the hemoglobin–albumin–lymphocyte–platelet (HALP) score, derived from hematologic and biochemical parameters, reflects both systemic inflammation and nutritional status and has been recognized as a prognostic biomarker in several systemic diseases, particularly malignancies (11,12).

Emerging evidence suggests that SII is strongly associated with disease severity in rheumatologic disorders, including adult-onset Still's disease, ANCA-associated vasculitis, and ankylosing spondylitis (13–15). In this context, CBC-based inflammatory indices represent promising, accessible tools for the timely and comprehensive assessment of pathological processes in RA.

Accordingly, the present study aimed to investigate the relationship between RA disease activity and four hematologic indices: SII, HALP, NLR, and PLR.

MATERIALS AND METHODS

Study Design and Ethical Approval

This single-center, cross-sectional study was conducted at Ankara Bilkent City Hospital. Ethical approval was obtained from the Scientific and Ethical Evaluation Board for Medical Research (TABED) of Ankara Bilkent City Hospital on January 8, 2025 (Protocol No: TABED 2-25-810). The study was performed in accordance with the principles of the Declaration of Helsinki.

Participants

Patients diagnosed with rheumatoid arthritis according to the 2010 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria were enrolled. Exclusion criteria included immunodeficiency, hematologic disorders, active infections, malignancies, other concurrent inflammatory conditions, and pregnancy or breastfeeding.

Data Collection

Demographic and clinical data, including age, gender, smoking status, disease duration (months), and comorbidities, were recorded. Laboratory assessments included routine CBC parameters—neutrophils ($\times 10^9$/L), lymphocytes ($\times 10^9$/L), platelets ($\times 10^9$/L), hemoglobin (g/L), and albumin (g/L)—as well as erythrocyte sedimentation rate (ESR, mm/h) and C-reactive protein (CRP, mg/L).

Based on these laboratory data, the following indices were calculated:

- Disease Activity Score-28 with C-reactive protein (DAS28–CRP)
- Neutrophil-to-lymphocyte ratio (NLR)
- Platelet-to-lymphocyte ratio (PLR)
- Systemic immune-inflammation index (SII)
- Hemoglobin–albumin–lymphocyte–platelet (HALP) score

The SII was calculated as (neutrophil count × platelet count) ÷ lymphocyte count.

The HALP score was calculated as (hemoglobin × albumin × lymphocyte count) ÷ platelet count.

The NLR and PLR were calculated as neutrophils ÷ lymphocytes and platelets ÷ lymphocytes, respectively.

To assess the association between hematologic indices and disease activity, patients were classified into two groups according to DAS28–CRP values:

1. Remission group: DAS28–CRP ≤ 2.6
2. Active disease group: DAS28–CRP > 2.6

Comparisons of hematologic and biochemical parameters were performed between these groups.

Statistical Analysis

Statistical analyses were performed using SPSS software (version 21.0; IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed using the Kolmogorov–Smirnov test. Descriptive statistics were reported as mean ± standard deviation (SD) for continuous variables and as frequencies and percentages for categorical variables. Group comparisons were conducted using the Student's t-test or Mann–Whitney U test, depending on distributional characteristics. The chi-square test was applied for categorical variables. Correlations between hematologic indices and disease activity were evaluated using Spearman's rank correlation coefficient. Statistical significance was set at $p < 0.05$, with 95% confidence intervals (CIs) reported where applicable.

RESULTS

Seventy-two patients with RA were included in the study, of whom 53 (73.6%) were female. The mean age was 53.79 ± 11.25 years, and the median disease duration was 110 months. Based on DAS28–CRP values, 31 patients (43.1%) were classified as being in remission (≤2.6), while 41 (56.9%) had active disease (>2.6, including low, moderate, and high activity levels) (Table 1).

Patients with active disease (DAS28–CRP > 2.6) exhibited significantly higher NLR, PLR, and SII values compared with those in remission ($p = 0.001$, $p = 0.027$, and $p < 0.001$, respectively). Although the HALP score was lower in the active disease group, the difference did not reach statistical significance ($p > 0.05$) (Table 2).

Correlation analyses demonstrated that NLR showed a moderate positive correlation with DAS28-CRP ($r=0.624$, $p<0.001$) and a strong correlation with CRP ($r=0.603$, $p<0.001$). PLR and SII exhibited weak but significant positive correlations with disease activity parameters, although the PLR–CRP correlation did not reach statistical significance ($p=0.079$). Conversely, the HALP score was inversely correlated with ESR, CRP, and DAS28-CRP (all $p<0.05$), indicating its potential role as a negative marker of inflammation (Table 3).

Table 1. Demographic and clinical characteristics of patients with rheumatoid arthritis

Parameter	Demographics, n (%)	RA Patient Group, n:72
	Female, n (%)	53(73.6)
	Age, years, mean ± SD	53.79±11.25
	Disease duration, months, median (IQR)	110(123)
	Smoking, n (%)	27(37.5)
Comorbidities, n (%)	Hypertension	24(33.3)
	Diabetes mellitus	14(19.4)
	Coronary artery disease (CAD)	6(8.3)
	Congestive heart failure (CHF)	1(1.4)
	COPD/asthma	4(5.6)
	Malignancy	2(2.8)
DAS28–CRP, n (%)	Remission (≤2.6)	31(43.1)
	Active disease (> 2.6)	41(56.9)

RA, rheumatoid arthritis; CRP, C-reactive protein; DAS28–CRP, Disease Activity Score based on 28 joints and CRP; CAD, coronary artery disease; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease.

Table 2. Comparison of hematologic indices and laboratory parameters according to disease activity in patients with rheumatoid arthritis

	Remission (n = 31), median (IQR)	RA patients with active (n=41)	p-value
NLR	2.11(1)	4.12(2)	**0.001**
PLR	149.22(67)	188.42(115)	**0.027**
SII	565.08(368)	924.09(1022)	**<0.001**
HALP score	39.65(30)	33.79(23)	0.058
Leukocytes (/µL)	6220(2040)	7710(3860)	**0.002**
Leukocytes (/µL	3700(2050)	5300(3665)	**<0.001**
Lymphocytes (/µL)	1800(650)	1480(655)	**0.008**
Platelets (×10³/µL)	253000(94000)	285000(114000)	**0.048**

RA, rheumatoid arthritis; **NLR**, neutrophil-to-lymphocyte ratio; **PLR**, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index; **HALP**, hemoglobin–albumin–lymphocyte–platelet score; **CRP**, C-reactive protein; **DAS28–CRP**, Disease Activity Score-28 based on CRP.

Table 3. Correlation of hematologic indices with activation parameters in patients with rheumatoid arthritis

	NLR		PLR		SII		HALP score	
	rh	p	rh	p	rh	p	rh	p
ESR	0.293	**0.014**	0.265	**0.025**	0.339	**0.004**	-0.348	**0.003**
CRP	0.603	**<0.001**	0.208	0.079	0.357	**0.002**	-0.284	**0.016**
DAS28-CRP	0.624	**<0.001**	0.292	**0.013**	0.441	**<0.001**	-0.324	**0.006**

RA, rheumatoid arthritis; NLR, neutrophil-to-lymphocyte ratio; PLR, platelet-to-lymphocyte ratio; SII, systemic immune-inflammation index; HALP, hemoglobin–albumin–lymphocyte–platelet score; ESR, erythrocyte sedimentation rate; CRP, C-reactive protein; DAS28–CRP, Disease Activity Score-28 based on CRP.

DISCUSSION

Our findings demonstrate a significant association between RA disease activity and specific hematologic indices, namely the NLR, PLR, SII. These markers were markedly elevated in patients with active RA. Although the HALP score was lower in the active group, the difference did not reach statistical significance ($p > 0.05$). Nevertheless, the HALP score may hold clinical relevance, as it integrates markers of both nutritional status and systemic inflammation—two domains frequently altered in chronic autoimmune disorders.

In RA, persistent inflammatory stimuli disrupt the function and activation of neutrophils, platelets, and lymphocytes. This immune dysregulation is further amplified by B-cell hyperactivity and T-cell dysfunction (16). Recent evidence highlights the role of platelets as active participants in inflammation beyond their established function in hemostasis. Upon activation, platelets facilitate leukocyte recruitment, promote endothelial adhesion, and release pro-inflammatory mediators such as TNF-α, IL-1β, IL-6, and IL-8 (17,18). Cytokines such as TNF-α and IL-6, primarily produced by monocytes and macrophages, sustain the chronic inflammatory microenvironment characteristic of RA (19,20). This prolonged inflammatory state drives joint damage and synovial destruction, underscoring the potential of hematologic indices to reflect systemic inflammatory burden.

The SII, which integrates neutrophil, lymphocyte, and platelet counts, serves as a robust indicator of both innate immune activation and suppression of adaptive immunity. Prior research has established its prognostic value in malignant and inflammatory conditions, including gastrointestinal, breast, renal, and gynecological cancers, as well as cardiovascular disease (21,22). More recently, SII has demonstrated clinical utility in chronic inflammatory disorders such as COVID-19, osteoporosis, Behçet's disease, Still's disease, and ANCA-associated vasculitis (23–29). These findings, together with our results, suggest that SII may provide a reliable and cost-effective measure of systemic inflammation in RA.

In rheumatologic diseases, SII has been shown to correlate significantly with disease severity, particularly in psoriatic arthritis and RA (30). Bai et al. reported that RA patients who responded favorably to TNF-α inhibitors had significantly lower SII levels, which also correlated with conventional inflammatory markers such as CRP, rheumatoid factor (RF), and ESR (31). Similarly, another study demonstrated that RA patients with elevated high-sensitivity CRP (hs-CRP ≥ 3 mg/L) had higher SII values, and that SII was positively associated with both NLR and PLR (32). Collectively, these findings support the growing evidence that SII may serve as a practical and accessible marker for assessing disease activity in RA.

Beyond SII, several studies have examined the diagnostic and prognostic significance of other systemic inflammatory indices, particularly NLR and PLR. A recent meta-analysis confirmed that patients with RA exhibit higher NLR and PLR levels compared with healthy controls, and both indices showed a modest but consistent positive correlation with disease activity (33). Zengin et al. further proposed that NLR may aid in the early diagnosis of RA, whereas PLR demonstrated limited diagnostic value in this context (34). In a systematic review and meta-analysis, Mangoni and Zinellu assessed the diagnostic performance of these indices, reporting that NLR yielded an area under the curve (AUC) of 0.76 for RA diagnosis and 0.70 for disease activity, while PLR achieved a slightly higher diagnostic accuracy (AUC 0.80) (35). However, evidence specifically linking PLR with RA disease activity remains limited. Taken together, these observations highlight the potential of NLR and PLR as cost-effective and widely available biomarkers that may complement established inflammatory indices in the evaluation of RA activity. In line with these findings, our study demonstrated a moderate correlation between NLR and DAS28-CRP, whereas the correlations of PLR and SII with disease activity were weaker. This suggests that while all indices reflect systemic inflammation, NLR may represent a more robust surrogate marker of RA activity in routine clinical practice.

Calculated using hemoglobin, albumin, lymphocyte, and platelet levels, the HALP score serves as a composite indicator that integrates both systemic inflammation and nutritional status. It has been validated as a reliable prognostic factor across multiple malignancies (36). Among its components, serum albumin is particularly noteworthy, as it reflects not only nutritional reserve but also systemic inflammatory activity and overall disease severity, especially during acute clinical events (37). Beyond oncology, the HALP score has demonstrated prognostic value in non-malignant conditions such as stroke, where both inflammation and nutritional status are key determinants of patient outcomes (38).

CONCLUSION

Given its simplicity and cost-effectiveness, the HALP score offers a unique approach for simultaneously capturing inflammatory burden and nutritional condition, thereby providing a more holistic view of patient status. Its capacity to reflect the interplay between these processes highlights its potential role in guiding prognosis and therapeutic strategies. Although the present study is the first to investigate the relevance of the HALP score in RA, no statistically significant association with disease activity was observed. Nonetheless, the observed trend toward lower HALP values in patients with active RA suggests that this index may still have clinical relevance. Further large-scale, longitudinal studies are warranted to clarify its prognostic significance and potential role in disease monitoring within rheumatology.

Strengths and Limitations of the Study

This study is the first to examine the clinical relevance of the HALP score in rheumatoid arthritis, introducing a novel composite biomarker that reflects the interplay between systemic inflammation and nutritional status. A key strength lies in its focus on simple, inexpensive, and widely available hematologic indices, underscoring their potential applicability in routine clinical practice. The significant associations observed between disease activity and indices such as SII, NLR, and PLR support their use as adjunctive tools for disease monitoring.

Several limitations should be acknowledged. First, the cross-sectional design precludes conclusions regarding causality. Second, the relatively small sample size and single-center setting may limit the generalizability of the findings. Third, the absence of longitudinal follow-up prevents assessment of how these indices change over time or in response to treatment interventions. Another limitation of our study is the absence of SDAI and CDAI assessments.

Despite these limitations, this study provides important preliminary evidence that hematologic indices could complement conventional measures such as CRP, ESR, and DAS28 in evaluating RA disease activity. Although HALP did not demonstrate a statistically significant association with disease activity in this cohort, its ability to integrate nutritional and inflammatory status warrants further investigation in larger, multicenter, and longitudinal studies.

Conflict of Interests
The authors declare that there is no conflict of interest in the study.

Financial Disclosure
The authors declare that they have received no financial support for the study.

Ethical Approval
Ethical approval was obtained from the Scientific and Ethical Evaluation Board for Medical Research (TABED) of Ankara Bilkent City Hospital on January 8, 2025 (Protocol No: TABED 2-25-810).

REFERENCES

1. Zhou H, Hu B, Zhao Z, et al. Elevated circulating T cell subsets and cytokines expression in patients with rheumatoid arthritis. Clin Rheumatol. 2019;38:1831-9.
2. Sparks JA. Rheumatoid arthritis. Ann Intern Med. 2019;170:ITC1-ITC16.
3. Colglazier CL, Sutej PG. Laboratory testing in the rheumatic diseases: a practical review. South Med J. 2005;98:185-91.
4. Birkelund S, Bennike TB, Kastaniegaard K, et al. Proteomic analysis of synovial fluid from rheumatic arthritis and spondyloarthritis patients. Clin Proteomics. 2020;17:29.
5. Wright HL, Lyon M, Chapman EA, et al. Rheumatoid arthritis synovial fluid neutrophils drive inflammation through production of chemokines, reactive oxygen species, and neutrophil extracellular traps. Front Immunol. 2020;11:584116.
6. Olumuyiwa-Akeredolu OO, Page MJ, Soma P, Pretorius E. Platelets: emerging facilitators of cellular crosstalk in rheumatoid arthritis. Nat Rev Rheumatol. 2019;15:237-48.
7. Schulze-Koops H. Lymphopenia and autoimmune diseases. Arthritis Res Ther. 2004;6:178-80.
8. 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:e13037.
9. Sargin G, Senturk T, Yavasoglu I, Kose R. Relationship between neutrophil-lymphocyte, platelet-lymphocyte ratio and disease activity in rheumatoid arthritis treated with rituximab. Int J Rheum Dis. 2018;21:2122-7.
10. Hu B, Yang XR, Xu Y, et al. Systemic immune-inflammation index predicts prognosis of patients after curative resection for hepatocellular carcinoma. Clin Cancer Res. 2014;20:6212-22.
11. Dincer ABK, Sezer S. Systemic immune inflammation index as a reliable disease activity marker in psoriatic arthritis. J Coll Physicians Surg Pak. 2022;32:773-8.
12. Tian M, Li Y, Wang X, et al. The hemoglobin, albumin, lymphocyte, and platelet (HALP) score is associated with poor outcome of acute ischemic stroke. Front Neurol. 2021;11:610318.
13. Kim JW, Jung JY, Suh CH, Kim HA. Systemic immune-inflammation index combined with ferritin can serve as a reliable assessment score for adult-onset Still's disease. Clin Rheumatol. 2021;40:661-8.
14. Kim Y, Choi H, Jung SM, et al. Systemic immune-inflammation index could estimate the cross-sectional high activity and the poor outcomes in immunosuppressive drug-naïve patients with antineutrophil cytoplasmic antibody-associated vasculitis. Nephrology. 2019;24:711-7.
15. Wu J, Yan L, Chai K. Systemic immune-inflammation index is associated with disease activity in patients with ankylosing spondylitis. J Clin Lab Anal. 2021;35:e23964.
16. El-Tanbouly GS, Rehab SA. Novel anti-arthritic mechanisms of trans-cinnamaldehyde against complete Freund's adjuvant-induced arthritis in mice: involvement of NF-κB/TNF-α and IL-6/IL-23/IL-17 pathways in the immuno-inflammatory responses. Inflammopharmacology. 2022;30:1769-80.

17. Gawaz M. Role of platelets in coronary thrombosis and reperfusion of ischemic myocardium. Cardiovasc Res. 2004;61:498-511.

18. Kaplanski G, Farnarier C, Kaplanski S, et al. Interleukin-1 induces interleukin-8 secretion from endothelial cells by a juxtacrine mechanism. Blood. 1994;84:4242-8.

19. Cooles FA, Isaacs JD. Pathophysiology of rheumatoid arthritis. Curr Opin Rheumatol. 2011;23:233-40.

20. Haynes J, Garcia B, Stollar EJ, et al. The biologically relevant targets and binding affinity requirements for the function of the yeast actin-binding protein 1 Src-homology 3 domain vary with genetic context. Genetics. 2007;176:193-208.

21. Yang R, Chang Q, Meng X, et al. Prognostic value of systemic immune-inflammation index in cancer: a meta-analysis. J Cancer. 2018;9:3295-302.

22. Çirakoğlu ÖF, Yılmaz AS. Systemic immune-inflammation index is associated with increased carotid intima-media thickness in hypertensive patients. Clin Exp Hypertens. 2021;15:1-7.

23. Muhammad S, Fischer I, Naderi S, et al. Systemic inflammatory index is a novel predictor of intubation requirement and mortality after SARS-CoV-2 infection. Pathogens. 2021;10:58.

24. Kim JW, Jung JY, Suh CH, Kim HA. Systemic immune-inflammation index combined with ferritin can serve as a reliable assessment score for adult-onset Still's disease. Clin Rheumatol. 2021;40:661-8.

25. Fang H, Zhang H, Wang Z, et al. Systemic immune-inflammation index acts as a novel diagnostic biomarker for postmenopausal osteoporosis and could predict the risk of osteoporotic fracture. J Clin Lab Anal. 2020;34:e23016.

26. Wang ZC, Jiang W, Chen X, et al. Systemic immune-inflammation index independently predicts poor survival of older adults with hip fracture: a prospective cohort study. BMC Geriatr. 2021;21:155.

27. Du YN, Chen YJ, Zhang HY, et al. Inverse association between systemic immune-inflammation index and bone mineral density in postmenopausal women. Gynecol Endocrinol. 2021;2:1-5.

28. Kim Y, Choi H, Jung SM, et al. Systemic immune-inflammation index could estimate the cross-sectional high activity and the poor outcomes in immunosuppressive drug-naïve patients with antineutrophil cytoplasmic antibody-associated vasculitis. Nephrology (Carlton). 2019;24:711-7.

29. Tanacan E, Dincer D, Erdogan FG, Gurler A. A cutoff value for the systemic immune-inflammation index in determining activity of Behçet disease. Clin Exp Dermatol. 2021;46:286-91.

30. 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;46:352-357.

31. Bai J, Tian Y, Wang F, et al. Role of systemic immune-inflammation index (SII) in assessing clinical efficacy of TNF-α inhibitors for rheumatoid arthritis. Am J Transl Res. 2023;15:6524-33.

32. Dervisevic A, Fajkic A, Jahic E, et al. Systemic immune-inflammation index in evaluation of inflammation in rheumatoid arthritis patients. Medeni Med J. 2024;39:183-91.

33. Lee YH. Association between the neutrophil-to-lymphocyte ratio, and platelet-to-lymphocyte ratio and rheumatoid arthritis and their correlations with the disease activity: a meta-analysis. J Rheum Dis. 2018;25:169.

34. Zengin O, Onder ME, Kalem A, et al. New inflammatory markers in early rheumatoid arthritis. Z Rheumatol. 2018;77:144-50.

35. Mangoni AA, Zinellu A. Diagnostic accuracy of the neutrophil-to-lymphocyte ratio and the platelet-to-lymphocyte ratio in rheumatoid arthritis: a systematic review and meta-analysis. Clin Exp Med. 2024;24:207.

36. Peng D, Zhang CJ, Tang Q, et al. Prognostic significance of the combination of preoperative hemoglobin and albumin levels and lymphocyte and platelet counts (HALP) in patients with renal cell carcinoma after nephrectomy. BMC Urol. 2018;18:20.

37. Eckart A, Struja T, Kutz A, et al. Relationship of nutritional status, inflammation, and serum albumin levels during acute illness: a prospective study. Am J Med. 2019;133:713-22.e7.

38. Fu Y, Liu Q, Anrather J, Shi FD. Immune interventions in stroke. Nat Rev Neurol. 2015;11:524-35.