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Investigation of systemic inflammation levels in patients followed up with a diagnosis of pneumonia

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ABSTRACT

Aims: Although pneumonia is typically considered a localized infection, it can trigger a systemic inflammatory response. Systemic inflammation is characterized by cytokine release throughout the body, increased acute phase reactants, and immune cell imbalance, which can elevate disease severity and the risk of complications. Recently, hematological parameters such as the systemic immune-inflammation index (SII) and systemic inflammatory response index (SIRI) have gained prominence in assessing this inflammatory response. This study aimed to evaluate systemic inflammation levels in patients diagnosed with pneumonia using SII and SIRI, and to analyze their correlation with classical inflammatory biomarkers such as C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR).

Methods: Between January 1, 2023, and June 1, 2025, a total of 50 patients hospitalized and followed with a diagnosis of pneumonia in the Departments of Pulmonology and Internal Medicine at Yozgat Bozok University Research and Training Hospital, along with 50 healthy individuals who presented to the same clinics during the same period, were included in the study. The study was designed retrospectively, with the pneumonia cases constituting the patient group and healthy volunteers serving as the control group. Demographic characteristics (age, sex), smoking status, clinical histories, laboratory findings, treatment processes, complete blood count (CBC), CRP levels, and ESR were recorded. Patients with active malignancy, hematological or autoimmune diseases, or those receiving immunosuppressive therapy were excluded from the study. For each participant, the SII and SIRI values were calculated.

Results: Of the participants, 66% were male and 34% female, with a mean age of 61.98±16.78 years in the pneumonia group and 59.20±16.10 years in the control group. SII was significantly higher in the pneumonia group (1301.28±886.78) compared to the control group (545.20±488.26), as was SIRI (5.66±6.58 vs. 0.91±1.21) ($p<0.001$ for both). Smokers exhibited significantly higher SII (1535.9±1094.1 vs. 977.2±560.3) and SIRI (7.51±8.13 vs. 3.10±3.72) values than non-smokers ($p=0.031$ and $p=0.014$, respectively).

Conclusion: This study demonstrated that new-generation inflammation indices such as SII and SIRI reflect the intensity of inflammatory response in pneumonia patients and show significant correlation with CRP levels. These findings suggest that integrating these indices into clinical practice may serve as effective tools in evaluating disease prognosis and monitoring treatment response.

Keywords: Pneumonia, systemic immune-inflammation index (SII), systemic inflammatory response index (SIRI)

INTRODUCTION

Community-acquired pneumonia (CAP) is an infectious disease that imposes a substantial global health burden, particularly among elderly individuals and those with comorbidities, often leading to severe clinical courses. The clinical management of CAP varies depending on the early diagnosis and the ability to predict disease severity. Consequently, there is a growing need for objective, rapid, and cost-effective diagnostic tools.[1,2]

Traditional inflammatory markers such as CRP and ESR are indicative of infection but are often insufficient in reflecting disease severity or distinguishing between different pathophysiological processes. Therefore, attention has shifted toward more comprehensive and dynamic indicators of inflammation. In this context, the systemic immune-inflammation index (SII) and systemic inflammatory response index (SIRI) have recently emerged as novel biomarkers for various infectious and inflammatory conditions.[3]

SII and SIRI are calculated using complex formulas that incorporate neutrophil (NEU), lymphocyte (LYM), monocyte (MONO), and platelet (PLT) levels, offering an integrated view of the immune and inflammatory burden within the body. Initially applied in oncology—for example, in gastric cancer[4] and hepatocellular carcinoma[5]—these indices have shown utility in prognostic assessments. More recently, their diagnostic and prognostic value in infectious diseases, including CAP, has gained recognition.

Tortum et al.[6] reported that SII was effective in predicting disease severity among pneumonia patients evaluated in emergency departments. Zeng et al.[7] identified SIRI as a significant predictor in post-ischemic stroke pneumonia. Similarly, Corica et al.[8] highlighted gender-specific differences in inflammatory responses in CAP, noting the potential for these biomarkers to have varying clinical implications across patient subgroups.

Environmental factors also influence the levels of these inflammatory indices. Smoking, in particular, is known to elevate inflammatory cytokines and cellular activity, thereby increasing index values. Lee et al.[9] demonstrated a markedly heightened inflammatory response in smokers. Moreover, a meta-analysis by Yang et al.[10] associated elevated SII with poor prognosis across various cancer types, reflecting the systemic impact of widespread inflammation.

The validity of these indices has also been confirmed in chronic inflammatory diseases. Wu et al.[11] showed that SII correlated with disease activity in ankylosing spondylitis, while Shao et al.[12] demonstrated their diagnostic and prognostic utility in pediatric *Mycoplasma pneumoniae* infections. Elmeazawy et al.[13] reported that SII and SIRI could predict necrotizing pneumonia development in children. Miao et al.[14] found that SII could estimate the risk of postoperative pneumonia in patients undergoing lung resection.

Furthermore, Suwadi et al.,[15] in a systematic review, reported that SII could potentially serve as a diagnostic tool even in other respiratory conditions such as acute pulmonary embolism. These findings suggest that beyond infectious diseases, these biomarkers have broad applicability in evaluating systemic inflammation.

METHODS

Ethics

Ethical approval for the study was obtained from the Non-interventional Clinical Researches Ethics Committee of Yozgat Bozok University Faculty of Medicine (Date: 04.06.2025, Decision No: 2025-GOKAEK-2511_2025.06.04_524). All procedures were carried out in accordance with the ethical rules and the principles of the Declaration of Helsinki.

Study Design and Participants

Between January 1, 2023, and June 1, 2025, data were collected from 50 patients who were hospitalized and followed with a diagnosis of pneumonia in the Departments of Pulmonology and Internal Medicine at Yozgat Bozok University Research and Training Hospital. Patients with active malignancy, hematological disease, autoimmune disease, or a history of immunosuppressive therapy were excluded from the study. During the same period, individuals who presented to the same clinics and were found to have no pathological findings on clinical evaluation and diagnostic work-up were accepted as the healthy control group. The study was conducted retrospectively. Demographic data (age, sex), smoking status, clinical histories, laboratory results, treatment processes, complete blood count (CBC), CRP levels, and ESR were recorded.

Data Collection

Data were retrieved from the hospital information management system. Comprehensive clinical data, including participants' demographic information (age, sex), smoking status, disease history, hemogram, and biochemical parameters, were recorded. Among the laboratory data, hemogram parameters (PLT, NEU, LYM, MONO values) formed the primary variables of the study. To determine the level of systemic inflammation, the SII and SIRI were calculated using the following formulas:

SII: (PLT×NEU)/LYM

SIRI: (NEU×MONO)/LYM

SII and SIRI are composite indicators reflecting the degree of systemic inflammation based on peripheral blood cell counts. SII reflects the inflammatory burden by accounting for increased NEUs and PLTs along with decreased LYMs, while SIRI assesses the cellular immune response by comparing NEU and MONO levels with LYMs. These indices, calculated from routinely performed CBC tests, are cost-effective, rapid, and accessible, and offer valuable clinical insight into infection severity and prognosis.

No diagnostic or therapeutic intervention was performed in this study; data were obtained solely from existing medical records. Accordingly, the study was conducted as a retrospective observational study in line with ethics committee approval.

Statistical Analysis

The data analyses were performed using SPSS version 22.0. Continuous variables were expressed as mean±standard deviation (SD). The distribution of variables was evaluated using the Kolmogorov-Smirnov test. Independent samples t-test was used for comparing normally distributed variables, while the Mann–Whitney U test was applied for non-normally distributed variables. Categorical variables were analyzed using the Chi-square test. The correlation between SII and SIRI indices and CRP and ESR levels was assessed using the Spearman correlation test. A p-value of <0.05 was considered statistically significant in all tests.

RESULTS

Among the 100 individuals included in the study, 66 were male and 34 were female. No statistically significant difference was observed between the groups in terms of gender (p=1.00). The participants' ages ranged from a minimum of 19 to a maximum of 84 years. The mean age was calculated as

61.98±16.78 years in the pneumonia group and 59.20±16.10 years in the control group, with no statistically significant difference between the two groups in terms of age (p=0.106).

When evaluating comorbidities in the patients, chronic obstructive pulmonary disease (COPD), hypertension, and diabetes mellitus were the most common conditions. Additionally, in some cases, coronary artery disease, asthma, hyperthyroidism, heart failure, valvular heart diseases, benign prostatic hyperplasia, vertebrobasilar insufficiency, a history of tuberculosis, and chronic kidney disease were also observed.

In the pneumonia group, the mean NEU value was 9.15±8.24×10^9/L, compared to 3.95±1.62 ×10^9/L in the control group. The mean LYM value was 1.93±1.77×10^9/L in the pneumonia group and 2.04±0.62×10^9/L in the control group. The MONO value was 0.94±0.43×10^9/L in the pneumonia group and 0.37±0.11×10^9/L in the control group. The hemoglobin (Hb) level was 13.83±2.12 g/dl in the pneumonia group and 13.89±2.62 g/dl in the control group. The PLT count was 265.34±103.29×10^9/L in the pneumonia group and 239.02±47.83×10^9/L in the control group (**Table 1**).

Table 1. Comparative parameter table between pneumonia and control groups

Parameter	Pneumonia mean	Pneumonia SD	Control mean	Control SD
NEU (10^3/µL)	9.15	8.24	3.95	1.62
LYM (10^3/µL)	1.93	1.77	2.04	0.62
MONO (10^3/µL)	0.94	0.43	0.37	0.11
Hb (g/dl)	13.83	2.12	13.89	2.62
PLT (10^3/µL)	265.34	103.29	239.02	47.83
ESR (mm/h)	42.00	31.81	6.95	4.76
CRP (mg/L)	93.84	71.84	2.07	2.67

SD: Standard deviation, NEU: Neutrophil, LYM: Lymphocyte, MONO: Monocyte, Hb: Hemoglobin, PLT: Platelet, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate

The mean SII was calculated as 1301.28±886.78 in the pneumonia group and 545.20±488.26 in the control group, with this difference being statistically significant (p=0.000). Similarly, the mean SIRI was 5.66±6.58 in the pneumonia group and 0.91±1.21 in the control group, and the difference between the groups was also statistically significant (p=0.000) (**Figure 1, 2**).

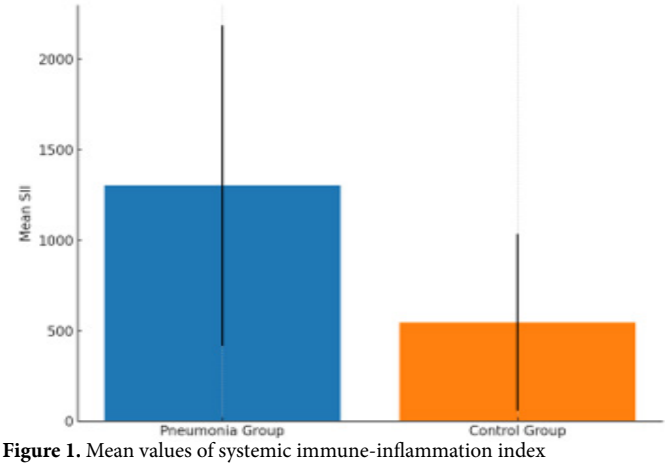

Figure 1. Mean values of systemic immune-inflammation index

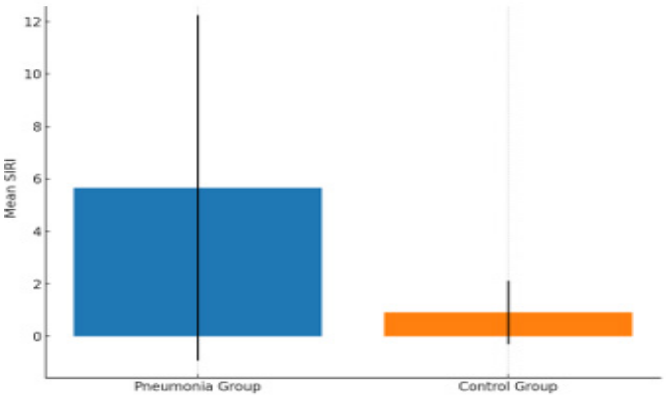

Figure 2. Mean values of systemic inflammatory response index

Among the biochemical markers of inflammation, the CRP level was found to be 93.84±71.84 mg/L in pneumonia patients, whereas it was 3.46±2.78 mg/L in the control group; this difference was also considered statistically significant (p=0.000). Furthermore, the ESR was 42.00±31.81 mm/h in the pneumonia group and 7.03±4.83 mm/h in the control group, which was also found to be statistically significant (p=0.000) (**Table 1, 2**).

Table 2. Comparative table of inflammatory markers

Parameter	Pneumonia mean	Pneumonia SD	Control mean	Control SD
SII	1301.28	886.78	545.20	488.26
SIRI	5.66	6.58	0.91	1.21
CRP (mg/L)	93.84	71.84	3.46	2.78
ESR (mm/h)	42.00	31.81	7.03	4.83

SD: Standard deviation, SII: Systemic immune-inflammation index, SIRI: Systemic inflammatory response index, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate

When evaluating the relationship between smoking status and systemic inflammation indices, the mean SII value in smokers was 1535.9±1094.1 and the SIRI value was 7.51±8.13. In non-smokers, these values were calculated as 977.2±560.3 for SII and 3.10±3.72 for SIRI. Statistical analysis revealed significant differences between the groups in terms of both SII (p=0.031) and SIRI (p=0.014). These findings indicate that smoking increases the systemic inflammatory response (**Table 3, Figure 3**).

Table 3. Comparison of SII and SIRI values according to smoking status in pneumonia patients

Parameter	Smokers (mean±SD)	Non-smokers (mean±SD)	p-value
SII	1535.9±1094.1	977.2±560.3	0.031
SIRI	7.51±8.13	3.10±3.72	0.014

SD: Standard deviation, SII: Systemic immune-inflammation index, SIRI: Systemic inflammatory response index

The relationship between the SII and SIRI indices and the levels of CRP and erythrocyte sedimentation in pneumonia patients was evaluated using the Spearman correlation coefficient. A correlation coefficient (r) between 0.1–0.3 was interpreted as weak, 0.3–0.5 as moderate, and ≥0.5 as strong. Spearman correlation analysis revealed a moderate positive correlation between SII and CRP (r=0.41, p=0.003), and a stronger positive correlation between SIRI and CRP (r=0.48,

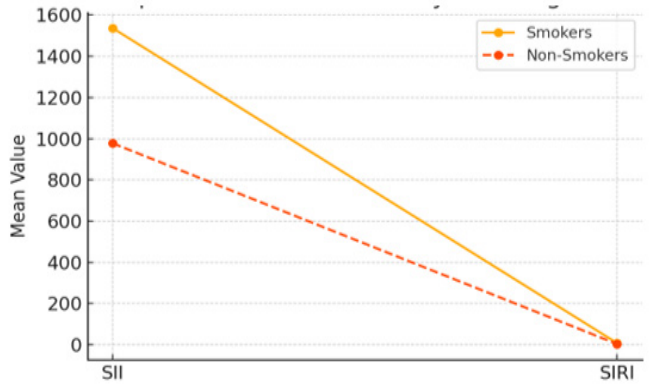

Figure 3. Comparison of SII and SIRI according to smoking status
SII: Systemic immune-inflammation index, SIRI: Systemic inflammatory response index

p<0.001). In contrast, no statistically significant relationship was found between SII and sedimentation (r=0.21, p=0.149) or between SIRI and sedimentation (r=0.10, p=0.505) (**Table 4, Figure 4**).

Table 4. Correlation analysis between SII and SIRI with CRP and sedimentation

Correlation pair	Correlation (r)	p-value
SII–CRP	0.41	0.003
SII–ESR	0.21	0.149
SIRI–CRP	0.48	<0.001
SIRI–ESR	0.10	0.505

SII: Systemic immune-inflammation index, SIRI: Systemic inflammatory response index, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate

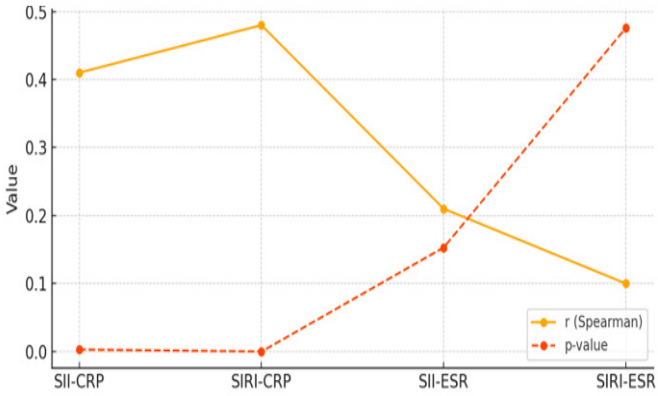

Figure 4. Relationship between SII/SIRI and CRP and sedimentation in the pneumonia group
SII: Systemic immune-inflammation index, SIRI: Systemic inflammatory response index, CRP: C-reactive protein, ESR: Erythrocyte sedimentation rate

DISCUSSION

In this study, systemic inflammation parameters in pneumonia patients were comprehensively examined, revealing significant differences compared to the control group. Of the 100 participants, 66% were male and 34% were female, with no statistically significant difference between sexes (p=1.00). While this finding suggests that gender may not be a determining factor in the development of pneumonia, the literature indicates that in CAP, males often present with higher hospitalization rates, more frequent comorbidities such as COPD, and a more pronounced pro-inflammatory response (Cillóniz et al.[2]). In contrast, female patients may display differences in clinical presentation and treatment response.[8]

The mean age of participants was 61.98±16.78 years (range: 19–84), indicating that pneumonia can occur not only in the elderly but also in younger adults. However, with increasing age, the incidence and risk of complications rise, and in elderly patients, immune senescence, the presence of comorbidities, reduced functional capacity, and atypical presentations complicate diagnosis and management (Cillóniz et al.[2]).

The most common comorbidities were COPD, hypertension, and diabetes mellitus. COPD, through its effects on both lung parenchyma and systemic inflammatory balance, represents a major risk factor for pneumonia. Additionally, the use of inhaled corticosteroids (ICS) may increase infection risk. Lee et al.[18] reported that ICS use significantly increased the risk of pneumonia and tuberculosis in a dose-dependent manner.

Laboratory analysis revealed a significantly higher NEU count in the pneumonia group (9.15±8.24×10^9/L) compared to controls (3.95±1.62×10^9/L), reflecting an acute immune response and systemic inflammation. Elevated NEUs directly influence composite inflammatory indices such as SII and SIRI, which correlate with disease severity (Yang et al.[3]).

The pneumonia group also exhibited a lower lymphocyte count (1.93±1.77×10^9/L) compared to controls (2.04±0.62×10^9/L), suggesting possible immunosuppression during infection. Lymphocytopenia is associated with infection extent and immune insufficiency. Tortum et al.[6] demonstrated that low lymphocyte counts, in combination with elevated SII, were associated with increased intensive care requirements and mortality in pneumonia patients. Our findings support the view that pneumonia patients exhibit weakened immune function, with lymphocyte counts serving as an important biomarker in this context.

In the pneumonia group, the mean MONO count was 0.94±0.43×10^9/L, compared to 0.37±0.11×10^9/L in the control group, representing a significant increase indicative of a systemic inflammatory response to infection. MONOs, as key cells of innate immunity, migrate to infected tissues, perform phagocytosis, and regulate the release of inflammatory mediators. Elmeazawy et al.[13] reported that elevated MONO counts are critical in predicting necrotizing pneumonia in pediatric patients, correlate with increases in SII and SIRI, and reflect disease severity. Our findings similarly suggest that MONO elevation in pneumonia may serve not only as a diagnostic biomarker but also as an indicator of clinical progression and complication risk.

Hb levels averaged 13.83±2.12 g/dl in the pneumonia group and 13.89±2.62 g/dl in the control group, with no significant difference regarding anemia. In acute infections, Hb typically remains stable in the early phase. Hu et al.[5] demonstrated that in hepatocellular carcinoma, Hb was not directly associated with systemic inflammation, whereas composite indices such as SII provided more sensitive markers. This may explain the preserved Hb levels observed in our study cohort.

PLT counts were 265.34±103.29×10^9/L in the pneumonia group and 239.02±47.83×10^9/L in the control group. Beyond their hemostatic function, PLTs are active participants in immune responses. Prina et al.[26] found that thrombocytosis in CAP was associated with poor prognosis, higher mortality,

and complications. In our study, the elevated PLT counts may similarly represent part of the inflammatory response.

SII, SIRI, CRP, and ESR values were significantly higher in the pneumonia group than in controls (p=0.000). Among pneumonia patients who smoked, SII (1535.9±1094.1 vs. 977.2±560.3; p=0.031) and SIRI (7.51±8.13 vs. 3.10±3.72; p=0.014) were notably elevated. The mean SII was 1301.28±886.78 in the pneumonia group compared to 545.20±488.26 in controls (p=0.000), indicating a markedly stronger systemic inflammatory response. This aligns with Tortum et al.,[6] who reported that higher SII values in pneumonia patients presenting to the emergency department predicted greater disease severity, intensive care requirements, and increased complication risk.

In our study, the SIRI value was found to be significantly higher in the pneumonia group compared to the control group (5.66±6.58 vs. 0.91±1.21; p=0.000). Calculated as the ratio of NEU and MONO counts to lymphocyte counts, SIRI reflects both the extent of infection and the magnitude of the immune response. In the literature, Zeng et al.[7] reported that SIRI is an effective biomarker for predicting the development of pneumonia following ischemic stroke, whereas Zheng et al.[16] demonstrated that elevated SIRI in patients undergoing thrombectomy for acute ischemic stroke was an independent risk factor for pneumonia, with each unit increase raising the risk by 17% (OR=1.169, p=0.014) and showing significant associations with intensive care requirement, complications, and mortality. Our findings indicate that the elevated SIRI levels observed in the pneumonia group reflect the severity of infection and systemic inflammatory burden, aligning closely with the existing literature.

Among classical inflammatory markers, CRP levels averaged 93.84±71.84 mg/L in the pneumonia group and 3.46±2.78 mg/L in the control group, while ESR values were 42.00±31.81 mm/h and 7.03±4.83 mm/h, respectively (both p=0.000). Marrie[1] and Cillóniz et al.[2] have emphasized the diagnostic and therapeutic follow-up value of these markers in CAP, while Wang et al.[4] noted their role in calculating systemic inflammation indices.

A significant association was also found between smoking status and inflammatory parameters. Both SII and SIRI values were markedly higher in smokers (SII: 1535.9±1094.1 vs. 977.2±560.3; p=0.031 / SIRI: 7.51±8.13 vs. 3.10±3.72; p=0.014). Lee et al.[9] demonstrated that toxic components in cigarette smoke activate alveolar macrophages, epithelial cells, and dendritic cells, thereby enhancing the release of pro-inflammatory cytokines such as TNF-α, IL-1β, and IL-6, which in turn augment peripheral NEU activation, suppress lymphocyte function, and increase MONO activation. Similarly, Tabakoğlu et al.[19] reported that a smoking history of at least 20 pack-years significantly increased SII values (B=180.053, p=0.034), attributing this to chronic inflammatory stimulation, oxidative stress, and immune cell activation. Our findings are consistent with these mechanisms, further highlighting the amplifying effect of smoking on systemic inflammatory responses.

Correlation analysis revealed significant positive associations between SII and CRP (r=0.41, p=0.003) and between SIRI and CRP (r=0.48, p<0.001). These results demonstrate that both indices are closely related to biochemical inflammatory markers. Wu et al.[11] and Miao et al.[14] similarly reported that these indices correlate with CRP and reflect the severity of inflammation. Shao et al.[12] emphasized the utility of SII and SIRI in the diagnosis and follow-up of pediatric *Mycoplasma pneumoniae* infections.

Taken together, these findings suggest that SII and SIRI are robust diagnostic and prognostic biomarkers in pneumonia, and that evaluating them alongside classical markers such as CRP and ESR may significantly enhance clinical decision-making. Furthermore, integrating these indices into established clinical risk stratification models, such as those by Fine et al.[20] and Chalmers et al.,[21] may allow earlier and more accurate prediction of mortality and complication risk.

Limitations

However, our study has certain limitations. First, its single-center design may restrict the generalizability of the findings. Second, the relatively small sample size could reduce statistical power, particularly in subgroup analyses. Lastly, the retrospective design limits the ability to establish causal relationships. Therefore, to validate our results and obtain stronger evidence, larger-scale, multicenter, prospective studies are warranted.

CONCLUSION

This study demonstrates that the systemic inflammation indices SII and SIRI may serve as valuable diagnostic and prognostic biomarkers in patients with pneumonia. The findings reveal that classical inflammatory parameters—such as NEUs, MONOs, CRP, and ESR—are significantly elevated in the pneumonia group. Moreover, the positive correlation between SII and SIRI with CRP supports the role of these indices in reflecting the systemic impact of infection. The observation that SII and SIRI levels are higher among smokers underscores the pro-inflammatory effect of tobacco use on the immune response.

In light of these results, the routine use of systemic inflammation indices such as SII and SIRI in infectious diseases like pneumonia may offer substantial benefits in early diagnosis, severity assessment, and monitoring of treatment response. Future prospective studies with larger sample sizes are warranted to provide stronger evidence for the integration of these parameters into routine clinical practice.

ETHICAL DECLARATIONS

Ethics Committee Approval

Ethical approval for the study was obtained from the Non-interventional Clinical Researches Ethics Committee of Yozgat Bozok University Faculty of Medicine (Date: 04.06.2025, Decision No: 2025-GOKAEK-2511_2025.06.04_524).

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.

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