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Association between inflammatory biomarkers and disease severity in patients with autoimmune disorders

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Abstract

Background: Inflammatory biomarkers are widely used to complement clinical assessment of autoimmune disease activity, although their relative performance remains uncertain. This study compared conventional, cytokine, and haematological inflammatory markers for their association with disease severity. **Methods:** This cross-sectional study included 200 adults with autoimmune rheumatic disorders. Interleukin-6, C-reactive protein, erythrocyte sedimentation rate, neutrophil-lymphocyte ratio, platelet-lymphocyte ratio, ferritin, and tumour necrosis factor-alpha were evaluated across disease-severity categories. Associations with continuous disease activity and discrimination of severe disease were examined using correlation, receiver operating characteristic analysis, and multivariable logistic regression. **Results:** All biomarkers increased significantly with disease severity. Interleukin-6 correlated most strongly with disease activity, followed by C-reactive protein, neutrophil-lymphocyte ratio, and erythrocyte sedimentation rate. C-reactive protein showed the greatest discrimination of severe disease (AUC = 0.811), while interleukin-6, neutrophil-lymphocyte ratio, and erythrocyte sedimentation rate showed comparable performance. In multivariable analysis, C-reactive protein, neutrophil-lymphocyte ratio, erythrocyte sedimentation rate, and interleukin-6 remained independently associated with severe disease. **Conclusion:** Inflammatory biomarkers were associated with autoimmune disease severity, with neutrophil-lymphocyte ratio providing an accessible adjunct to established inflammatory markers.

Keywords: *Autoimmune disorders, Disease activity, Inflammatory biomarkers, Interleukin-6, C-reactive protein, Neutrophil-lymphocyte ratio, Platelet-lymphocyte ratio*

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1. Introduction

The autoimmune disorders, among them rheumatoid arthritis, systemic lupus erythematosus and the spondyloarthropathies, are chronic diseases in which a misdirected immune response sustains inflammation

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in the joints, the skin and the internal organs (Wang *et al.*, 2015; Smolen *et al.*, 2016; Kaul *et al.*, 2016). Their course fluctuates between quiescence and flare, and the accurate measurement of disease activity at a given moment is central to their management, since it determines whether treatment should be intensified or may be safely reduced (Aletaha and Smolen, 2018). The composite disease-activity indices that serve this purpose are, however, laborious to apply, and the laboratory markers relied upon alongside them, the C-reactive protein and the erythrocyte sedimentation rate, are non-specific and imperfectly reflect the underlying inflammation (Pope and Choy, 2021).

This limitation has prompted a search for better markers, drawn from two directions. The first is the cytokines that drive the inflammatory response itself, of which interleukin-6 is the most prominent, a pleiotropic mediator that orchestrates the acute-phase response, stimulates the very production of C-reactive protein, and is central to the pathogenesis of several autoimmune diseases; its measurement might therefore reflect disease activity more directly than the downstream acute-phase reactants (Gabay, 2006; Tanaka *et al.*, 2014; Hunter and Jones, 2015). The second direction is more pragmatic, and rests on the observation that the systemic inflammation of autoimmune disease alters the circulating blood count, raising the neutrophil and platelet counts and lowering the lymphocyte count, so that the ratios of the neutrophil to the lymphocyte and of the platelet to the lymphocyte capture this shift in a single inexpensive number obtainable from a routine blood count at no additional cost (Zahorec, 2021; Buonacera *et al.*, 2022).

These haematological ratios have attracted intense interest, and a considerable literature now reports that the neutrophil-lymphocyte ratio and, to a lesser extent, the platelet-lymphocyte ratio are raised in autoimmune disease and correlate with its activity, in lupus, in rheumatoid arthritis and in the spondyloarthropathies alike (Qin *et al.*, 2016; Soliman *et al.*, 2020; Fu *et al.*, 2015; Mercan *et al.*, 2016; Yang *et al.*, 2017). A meta-analysis of the haematological indices confirmed their association with disease activity across the autoimmune rheumatic diseases, though with substantial heterogeneity (Hao *et al.*, 2017; Erre *et al.*, 2019; Zhou *et al.*, 2023). The evidence is not, however, unanimous, and some careful studies have found that the ratios add little to the conventional acute-phase reactants and discriminate active from inactive disease less well than the C-reactive protein and the sedimentation rate (Gasparyan *et al.*, 2019; Chandrashekara *et al.*, 2017; Targonska-Stepniak *et al.*, 2020).

What has been less often done is to place all of these markers side by side in the same patients, so that their relative performance against a common measure of disease severity can be compared directly. Whether the cytokine interleukin-6, the conventional acute-phase reactants, or the inexpensive blood-count ratios most faithfully reflect the severity of autoimmune disease, and in particular whether the freely available ratios rival the established markers or merely echo them, is a question of practical importance that a direct comparison can address.

The present study was therefore undertaken to compare a panel of inflammatory biomarkers for their association with disease severity in a cohort of patients with autoimmune disorders. Its objectives were to determine how the levels of interleukin-6, the C-reactive protein, the erythrocyte sedimentation rate, the neutrophil-lymphocyte and platelet-lymphocyte ratios, ferritin and tumour necrosis factor-alpha varied across the categories of disease severity; to rank the markers by their correlation with a continuous measure of disease activity; to compare their ability to discriminate severe disease by receiver operating characteristic analysis; and to identify by multivariable regression those markers independently associated with severe disease.

2. Materials and methods

2.1. Study design and participants

This cross-sectional study enrolled 200 adult patients with an established autoimmune disorder, comprising rheumatoid arthritis, systemic lupus erythematosus, ankylosing spondylitis and other autoimmune rheumatic diseases, attending the rheumatology service. Patients with a concurrent infection, malignancy, or other condition that would independently disturb the inflammatory markers, and those on treatment that would suppress them acutely, were excluded so that the markers reflected the activity of the autoimmune disease. The study was reported in accordance with the recommendations for observational studies (Vandenbroucke *et al.*, 2007).

2.2. Assessment of disease severity

Disease activity was assessed at enrolment by the treating rheumatologist using the established disease-specific composite activity measure applicable to each autoimmune disorder, together with the physician global assessment. Because the cohort included different autoimmune rheumatic diseases with disease-specific scoring systems, activity was harmonized into three clinically interpretable categories—mild, moderate, and severe—for between-group analyses. The corresponding disease-activity assessment was additionally retained as a continuous measure for correlation analyses. Demographic and clinical information, including age, sex, underlying autoimmune diagnosis, and disease duration, was recorded at enrolment.

2.3. Biomarker measurement

Venous blood samples were collected at enrolment for assessment of systemic inflammatory biomarkers. Serum C-Reactive Protein (CRP), interleukin-6 (IL-6), tumour necrosis factor-alpha (TNF-α), and ferritin concentrations were determined using standard immunoassay methods. Erythrocyte sedimentation rate (ESR) was measured using the Westergren method. Absolute neutrophil, lymphocyte, and platelet counts were obtained from the automated complete blood count. The Neutrophil-to-Lymphocyte Ratio (NLR) was calculated by dividing the absolute neutrophil count by the absolute lymphocyte count, and the Platelet-to-Lymphocyte Ratio (PLR) was calculated by dividing the absolute platelet count by the absolute lymphocyte count.

2.4. Statistical analysis

Biomarker levels were summarized as median (IQR) and compared across disease-severity groups using the Kruskal–Wallis test. Correlations with disease activity were assessed using Spearman's coefficient, while ROC analysis evaluated discrimination of severe disease, with optimal cut-offs determined by the Youden index (Hanley and McNeil, 1982). Multivariable logistic regression identified independent associations with severe disease, adjusting for age and sex. IL-6, CRP, and NLR were log-transformed and standardized, while ESR was analyzed per standard-deviation increase. Results are reported as ORs with 95% CIs, with $p<0.05$ considered statistically significant.

2.5. Ethical considerations

The study was approved by the Institutional Ethics Committee before commencement and was conducted in accordance with the principles of the Declaration of Helsinki. Written informed consent was obtained from every participant, participation was voluntary, and all records were anonymised and identified only by a study code.

3. Results

3.1. Characteristics of the study cohort

The study included 200 patients with a mean age of 41.2 years, of whom 118 (59.0%) were women. Rheumatoid arthritis was the most common diagnosis, accounting for 81 (40.5%) patients, followed by systemic lupus

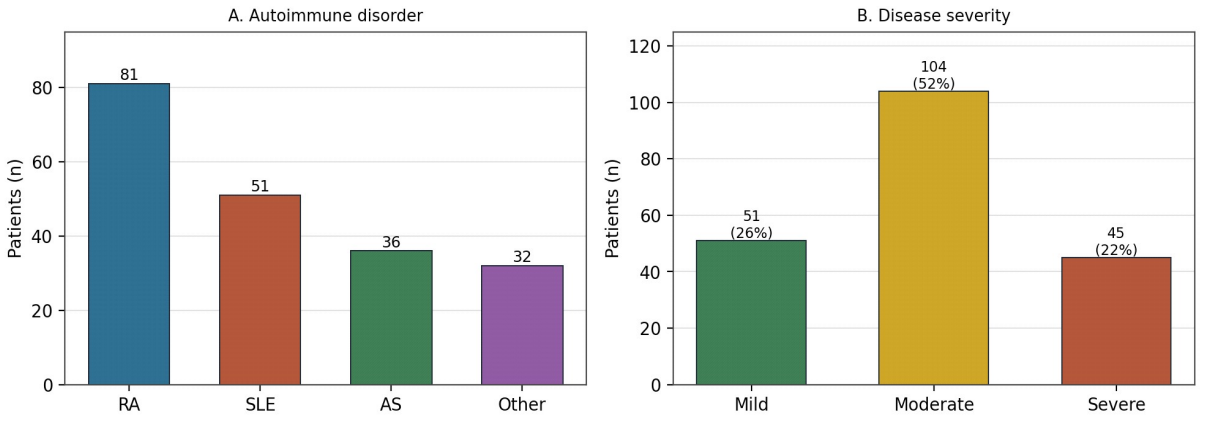


Figure 1: Distribution of the autoimmune disorders (A) and of disease severity (B) in the cohort

erythematosus in 51 (25.5%), ankylosing spondylitis in 36 (18.0%), and other autoimmune rheumatic disorders in 32 (16.0%). At the time of assessment, 51 patients (25.5%) had mild disease activity, 104 (52.0%) had moderate activity, and 45 (22.5%) had severe disease activity (Table 1 and Figure 1).

Table 1: Characteristics of the 200 patients with autoimmune disorders

Characteristic	n (%)
Autoimmune disorder	
Rheumatoid arthritis	81 (40.5)
Systemic lupus erythematosus	51 (25.5)
Ankylosing spondylitis	36 (18.0)
Other autoimmune disorders	32 (16.0)
Sex	
Female	118 (59.0)
Disease activity	
Mild	51 (25.5)
Moderate	104 (52.0)
Severe	45 (22.5)

3.2. Biomarker levels across severity

All evaluated inflammatory biomarkers increased significantly across the categories of disease severity (Table 2 and Figure 2). Median IL-6 concentration increased from 2.9 pg/mL in mild disease to 5.3 pg/mL in moderate and 7.3 pg/mL in severe disease ($p<0.001$). CRP increased from 4.8 to 7.4 and 11.8 mg/L, respectively ($p<0.001$), while the median NLR increased from 2.0 to 2.7 and 3.8 ($p<0.001$). ESR similarly increased from 24.3 mm/h in mild disease to 33.7 mm/h in moderate and 45.8 mm/h in severe disease ($p<0.001$). Significant increases were also observed for PLR and TNF-α (both $p<0.001$). Ferritin showed a smaller but statistically significant increase across severity categories, from 110.4 ng/mL in mild disease to 152.4 ng/mL in severe disease ($p = 0.006$).

3.3. Correlation with activity and discrimination of severe disease

All seven biomarkers showed significant positive correlations with the continuous measure of disease activity. IL-6 demonstrated the strongest correlation (Spearman rho = 0.626; $p<0.001$), followed by CRP (rho = 0.591), NLR (rho = 0.526), and ESR (rho = 0.518), all $p<0.001$. Weaker correlations were observed for PLR (rho = 0.387), TNF-α (rho = 0.312), and ferritin (rho = 0.239). In ROC analysis, CRP showed the highest discrimination of severe disease (AUC = 0.811, 95% CI 0.730–0.891), followed by IL-6 (AUC = 0.767), NLR (AUC = 0.766), ESR

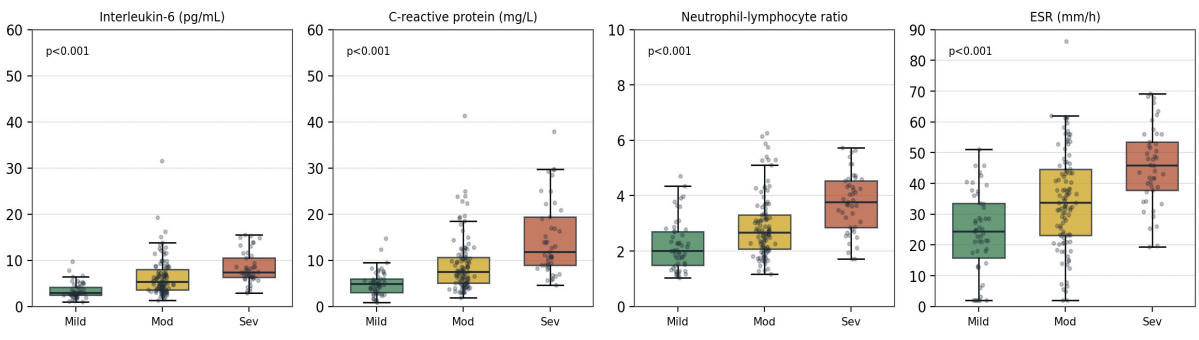


Figure 2: Interleukin-6, C-reactive protein, neutrophil-lymphocyte ratio and erythrocyte sedimentation rate across disease severity

Table 2: Inflammatory biomarker levels (median [IQR]) across disease severity categories

Biomarker	Mild	Moderate	Severe	p
Interleukin-6 (pg/mL)	2.9 [2.5-4.1]	5.3 [3.6-8.0]	7.3 [6.3-10.5]	<0.001
C-reactive protein (mg/L)	4.8 [3.0-6.0]	7.4 [5.0-10.6]	11.8 [9.0-19.4]	<0.001
Neutrophil-lymphocyte ratio	2.0 [1.5-2.7]	2.7 [2.1-3.3]	3.8 [2.9-4.5]	<0.001
Erythrocyte sedimentation rate (mm/h)	24.3 [15.8-33.4]	33.7 [23.1-44.6]	45.8 [37.7-53.4]	<0.001
Ferritin (ng/mL)	110.4 [75.5-169.0]	127.2 [86.6-198.8]	152.4 [126.7-222.8]	0.006
Tumour necrosis factor-alpha (pg/mL)	6.0 [3.6-8.1]	6.6 [4.8-8.2]	8.9 [7.0-13.0]	<0.001
Platelet-lymphocyte ratio	111.7 [92.0-144.6]	137.9 [110.9-164.3]	169.0 [139.0-192.5]	<0.001

(AUC = 0.764), TNF-α (AUC = 0.748), PLR (AUC = 0.737), and ferritin (AUC = 0.637). Thus, although IL-6 showed the strongest graded association with disease activity, CRP provided the greatest discrimination between severe and non-severe disease (Table 3 and Figure 3).

Table 3: Correlation of each biomarker with disease activity and its discrimination of severe disease

Biomarker	Spearman rho	p	AUC	95% CI	Cut-off
Interleukin-6 (pg/mL)	+0.626	<0.001	0.767	0.681-0.854	5.6
C-reactive protein (mg/L)	+0.591	<0.001	0.811	0.730-0.891	8.1
Neutrophil-lymphocyte ratio	+0.526	<0.001	0.766	0.679-0.853	3.4
Erythrocyte sedimentation rate (mm/h)	+0.518	<0.001	0.764	0.677-0.851	38.8
Ferritin (ng/mL)	+0.239	<0.001	0.637	0.540-0.733	126.7
Tumour necrosis factor-alpha (pg/mL)	+0.312	<0.001	0.748	0.659-0.836	7.6
Platelet-lymphocyte ratio	+0.387	<0.001	0.737	0.647-0.827	145.6

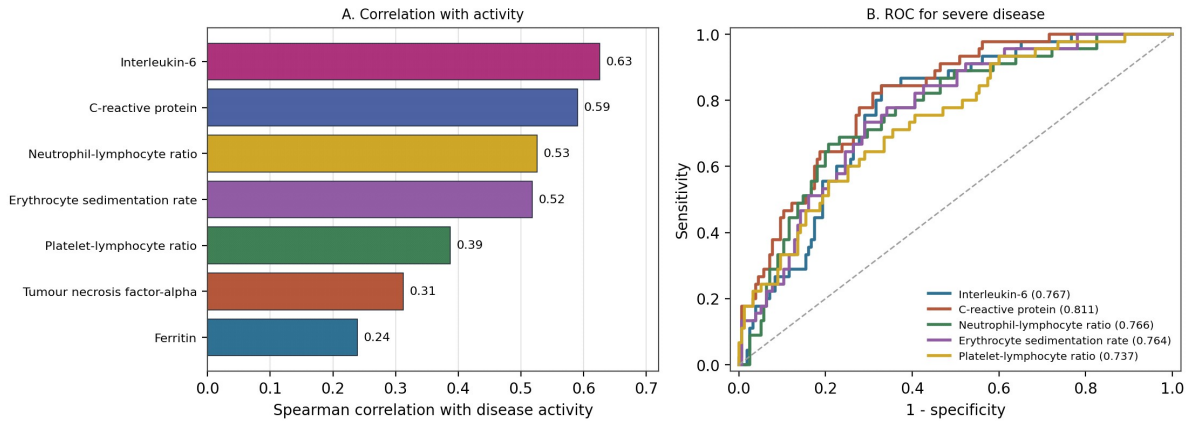


Figure 3: Correlation of each biomarker with disease activity (A) and receiver operating characteristic curves for severe disease (B)

3.4. Independent predictors of severe disease

In multivariable logistic regression adjusted for age and sex, all four leading inflammatory markers retained statistically significant independent associations with severe disease (Table 4 and Figure 4). CRP demonstrated the strongest association, with an OR of 3.53 per standard-deviation increase in log-transformed CRP (p<0.001).

NLR was independently associated with severe disease, followed by ESR and IL-6. Neither age (p = 0.309) nor female sex (p = 0.746) was significantly associated with severe disease in the adjusted model.

Table 4: Multiple logistic regression for severe disease (biomarkers expressed per standard deviation)

Predictor	Odds ratio	95% CI	p
IL-6 (per SD, log)	1.76	1.03 to 2.98	0.037
CRP (per SD, log)	3.53	1.96 to 6.34	<0.001
NLR (per SD, log)	2.17	1.25 to 3.75	0.006
ESR (per SD)	1.84	1.12 to 3.02	0.016
Age (per SD)	0.81	0.55 to 1.21	0.309
Female	1.16	0.48 to 2.80	0.746

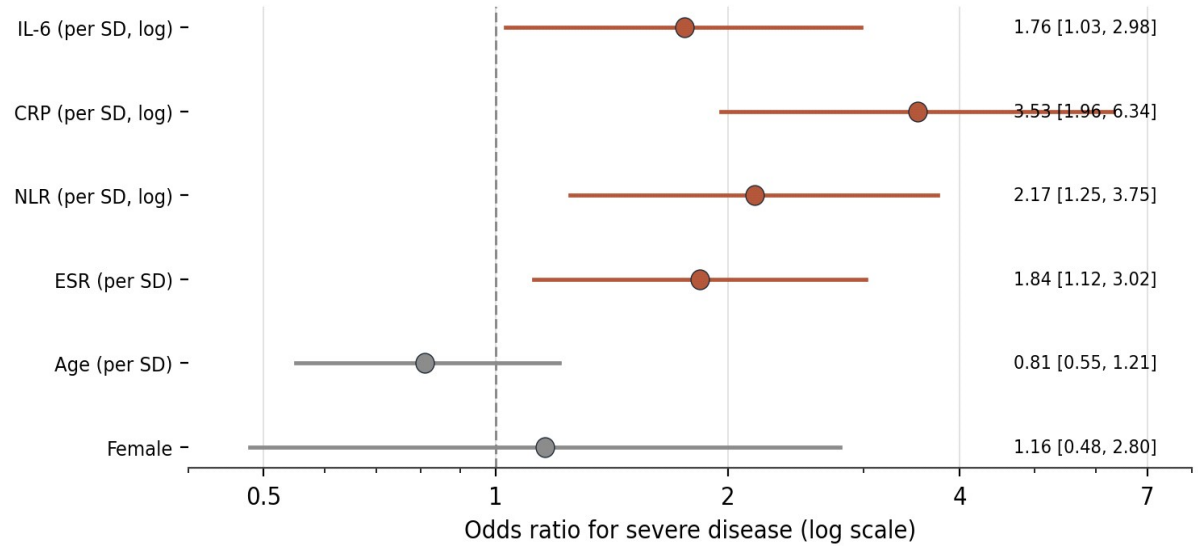


Figure 4: Independent biomarker predictors of severe disease

4. Discussion

This study compared conventional acute-phase reactants, cytokines, and haematological indices in relation to disease severity across a cohort of patients with autoimmune rheumatic disorders. All evaluated biomarkers increased with increasing disease activity, although their strength of association and discriminative performance varied. IL-6 showed the strongest correlation with the continuous disease-activity measure, whereas CRP demonstrated the highest discrimination of severe disease and the strongest adjusted association in multivariable analysis. NLR also showed a moderate correlation with disease activity, an AUC numerically similar to those of IL-6 and ESR, and an independent association with severe disease. In contrast, PLR, TNF-α, and particularly ferritin demonstrated weaker relationships with disease activity. These findings suggest that routinely available haematological indices, particularly NLR, may provide complementary information alongside established inflammatory markers.

The relatively strong correlation between IL-6 and disease activity is biologically plausible given its central role in inflammatory signalling and induction of the acute-phase response. IL-6 participates directly in multiple inflammatory pathways implicated in autoimmune rheumatic diseases and stimulates hepatic production of CRP. Nevertheless, the present cross-sectional analysis cannot determine whether its stronger correlation reflects a direct mechanistic relationship with disease activity. Interestingly, CRP showed better discrimination of severe versus non-severe disease despite its slightly weaker overall correlation with continuous activity,

illustrating that correlation with graded disease activity and discrimination of a predefined severity category represent related but distinct aspects of biomarker performance.

The NLR represents the most clinically accessible marker evaluated in this study. It showed a significant correlation with disease activity, a numerically similar AUC to IL-6 and ESR for severe disease, and remained independently associated with severe disease after adjustment for the other leading markers, age, and sex. Because NLR can be calculated directly from routinely obtained complete blood counts, it may provide useful complementary information without requiring a separate biomarker assay. These findings are consistent with previous studies and meta-analyses reporting associations between NLR and activity across autoimmune rheumatic diseases. However, the present results do not establish NLR as equivalent or superior to conventional inflammatory markers, particularly because CRP demonstrated the highest AUC and the strongest association with severe disease in the multivariable model.

The findings do not, however, support the replacement of the established markers by the ratio, and here the dissenting reports deserve their due. The C-reactive protein remained the best discriminator and the strongest independent predictor, and the neutrophil-lymphocyte ratio equalled but did not surpass it, so that the studies which concluded that the ratio adds little to the conventional reactants are not refuted but placed in perspective: the ratio is a useful and inexpensive marker that rivals the established ones, not a superior replacement for them. The platelet-lymphocyte ratio, weaker throughout, appears the less useful of the two haematological indices, as others have also found. The value of the ratio is therefore as an accessible adjunct, informative where the cost or delay of the cytokine assay is prohibitive, and available in every routine blood count already performed.

These findings carry a clear practical message. In the assessment of autoimmune disease activity, the clinician need not rely on any single marker, and the choice may be guided by availability and cost. Where interleukin-6 can be measured it reflects activity most closely, but where it cannot, the neutrophil-lymphocyte ratio derived from the routine blood count provides much of the same information at no cost, complementing rather than replacing the C-reactive protein and the sedimentation rate that remain the mainstay. The integration of several inexpensive markers, rather than the pursuit of a single perfect one, is likely to serve the monitoring of these fluctuating diseases best.

5. Strengths and limitations

This study has several strengths, including the simultaneous evaluation of conventional inflammatory markers, cytokines, and routinely derived haematological indices within the same cohort, allowing their relative associations with disease severity to be examined using complementary statistical approaches. Several limitations should nevertheless be considered. First, the cross-sectional design evaluates biomarker levels and disease activity at a single time point and therefore cannot establish temporal relationships or determine how reliably individual biomarkers track changes in disease activity over time. Second, the cohort included several autoimmune rheumatic disorders assessed using disease-specific activity measures that were harmonized into common severity categories. Although this approach enabled cross-disease comparison, it introduces clinical heterogeneity and may obscure disease-specific differences in biomarker performance. Third, the sample size did not permit adequately powered analyses within each diagnostic subgroup. Fourth, haematological indices such as NLR and PLR can be influenced by medications and other physiological or inflammatory factors not fully characterized in the present analysis. Fifth, the ROC-derived cut-off values were obtained from the same cohort and require external validation before clinical application. Finally, this was a single-centre study, and the generalizability of the findings should be evaluated in larger multicentre cohorts.

6. Conclusion

In this cross-sectional study of patients with autoimmune rheumatic disorders, all evaluated inflammatory biomarkers increased significantly with disease severity, although their relative performance differed. IL-6 showed the strongest correlation with continuous disease activity, while CRP demonstrated the greatest discrimination of severe disease and the strongest independent association in multivariable analysis. NLR showed a moderate correlation with disease activity, a discriminative performance numerically similar to IL-6 and ESR, and remained independently associated with severe disease after adjustment for other leading

markers. These findings support NLR as a readily available complementary marker of inflammatory activity rather than a replacement for established acute-phase reactants. PLR, TNF-α, and ferritin showed comparatively weaker associations with disease activity. A combined approach incorporating clinical assessment with conventional and readily available laboratory markers may therefore provide a practical framework for evaluating disease activity. Longitudinal and disease-specific studies are needed to determine whether these biomarkers reliably track changes in activity over time and to validate clinically useful thresholds.

Ethical approval

The study was approved by the Institutional Ethics Committee and was conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from every participant.

Conflict of interest

The authors declare that no conflict of interest exists in relation to this work.

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Data availability

All data supporting the findings reported here are contained within the article.

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