Original Paper

The Systemic Immune-Inflammation Index Reflects Disease Activity and Functional Limitation in Spondyloarthritis: A Single-Center Cross-Sectional Analysis

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ABSTRACT: Objectives: The Systemic Immune-Inflammation Index (SII) has emerged as a promising biomarker in inflammatory rheumatic diseases, yet its behavior across spondyloarthritis (SpA) subtypes remains poorly characterized. This study aimed to evaluate SII and its associations with disease activity (BASDAI), functional impairment (BASFI), and clinical subgroups in SpA patients. Material and Methods: This cross-sectional study included 64 patients with SpA classified according to ASAS criteria: 32 with axial SpA, 8 with peripheral SpA, and 24 with psoriatic SpA. SII was calculated as (Platelets × Neutrophils)/Lymphocytes. Between-group comparisons were performed using Kruskal-Wallis, Chi-square/Fisher's exact, and Mann-Whitney U tests. Spearman correlations were computed between SII, BASDAI, BASFI, age, and BMI. The study adhered to STROBE guidelines for cross-sectional studies. Results: The mean SII was 834±505, with no significant differences across SpA subtypes ($\chi^2(2)=0.44$, $p=0.80$, $\varepsilon^2=7.05\times10^{-3}$). SII correlated significantly with BASDAI ($\rho=0.350$, $p<0.01$) and BASFI ($\rho=0.316$, $p<0.05$), and inversely with BMI ($\rho=-0.250$, $p<0.05$). ROC analysis for discriminating active disease (BASDAI≥4) yielded an AUC of 0.612 (95% CI 0.421-0.803), with an optimal SII cutoff of 565 (sensitivity 63.5%, specificity 58.3%, PPV 86.8%, NPV 26.9%). SII did not differ significantly by diabetes ($p=0.73$), though hypertension showed a borderline trend ($p=0.09$). Conclusions: SII correlates significantly with disease activity and functional impairment across the SpA spectrum, supporting its potential as a readily accessible, subtype-agnostic inflammatory biomarker. The proposed cutoff of 565 for active disease requires validation in larger cohorts. Longitudinal studies are needed to establish SII's utility in treatment monitoring.

KEYWORDS: *Systemic immune-inflammation index, spondyloarthritis, psoriatic arthritis, BASDAI, BASFI, biomarker.*

Introduction

Spondyloarthritis (SpA) is a family of chronic inflammatory conditions linked by overlapping genetic background, immunopathogenesis, and clinical presentation.

Its main forms, axial SpA (axSpA), peripheral SpA (pSpA), and psoriatic arthritis (PsA), differ in the skeletal and extra-articular sites they preferentially affect [1,2].

Although TNF inhibitors and agents blocking the IL-17 pathway have transformed treatment, many patients still report ongoing symptoms, and there is still no reliable biomarker for tracking disease over time [3,4].

The inflammatory markers most often used in practice, C-reactive protein (CRP) and erythrocyte sedimentation rate (ESR), have well-known shortcomings in SpA. CRP is normal in up to 40-60% of patients with clinically active axial SpA, and neither CRP nor ESR adequately captures the complexity of the inflammatory process across all SpA subtypes [5,6].

Disease activity and function are usually scored with BASDAI and BASFI, the Bath

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Ankylosing Spondylitis Disease Activity and Functional Indices.

Although both are well validated and widely used, they rely entirely on patient self-report and are therefore susceptible to non-inflammatory contributors such as central sensitization, psychological distress, and structural damage [7,8].

The Systemic Immune-Inflammation Index (SII) is derived from a routine complete blood count by multiplying the platelet and neutrophil counts and dividing by the lymphocyte count.

It therefore combines three cell lines that reflect different arms of inflammation: neutrophils (innate immunity), platelets (thromboinflammation), and lymphocytes (adaptive immunity) [9].

Originally developed as a prognostic marker in oncology, SII has since been investigated in a range of autoimmune and inflammatory conditions [10,11].

Wu et al. found higher SII values in patients with ankylosing spondylitis than in healthy individuals, with levels rising in parallel with BASDAI [12].

Similarly, studies in psoriatic arthritis and broader SpA cohorts have suggested associations between SII and musculoskeletal inflammation assessed by ultrasound [13,14].

However, several gaps persist.

Most studies have focused on single SpA subtypes rather than examining SII across the full SpA spectrum.

The relationship between SII and functional outcomes (BASFI) has been insufficiently explored.

The influence of demographic factors, and cardiovascular comorbidities on SII in SpA remains poorly defined [15].

The aim of this study was to evaluate SII across axial, peripheral, and psoriatic SpA subtypes and to assess its associations with disease activity (BASDAI), functional impairment (BASFI), and clinically relevant subgroups defined by disease activity threshold, hypertension, and diabetes.

Material and Methods

Study design and population

We performed an observational, cross-sectional study in the Rheumatology Department of the University of Medicine and Pharmacy of Craiova, Romania, including 64 consecutive adults with spondyloarthritis.

Using the Assessment of SpondyloArthritis International Society (ASAS) classification criteria [16], patients were assigned to axial (n=32), peripheral (n=8), or psoriatic (n=24) SpA.

Inclusion required age ≥18 years and a diagnosis of SpA established at least 3 months before enrolment.

The protocol was approved by the Ethics Committee of the University of Medicine and Pharmacy of Craiova (No. 201/24.11.2021) and complied with the Declaration of Helsinki.

All participants gave written informed consent. The manuscript was prepared according to the STROBE recommendations for cross-sectional studies [17].

Data collection

For each patient we recorded age, sex, body mass index (BMI), and the presence of hypertension or diabetes mellitus. BASDAI, the mean of six self-reported items each rated 0-10, was used to measure disease activity; a score ≥4 defined active disease [7].

Physical function was assessed with BASFI (range 0-10), in which a larger score reflects worse function [8].

Laboratory measurements and SII calculation

Fasting morning venous blood was drawn from all participants, and platelet, neutrophil, and lymphocyte counts were measured on automated hematology analyzers as part of a complete blood count. SII was then computed as platelets × neutrophils / lymphocytes [9].

Statistical analysis

Continuous data are given as mean±SD and as median with interquartile range (IQR); categorical data as counts and percentages.

Normality was checked with the Shapiro-Wilk test.

The three SpA subtypes were compared with one-way ANOVA or its non-parametric equivalent (Kruskal-Wallis) for continuous data, and with the chi-square test (or Fisher's exact test when expected counts were small) for categorical data.

SII was compared between two clinical subgroups with the Mann-Whitney U test, and the magnitude of each difference was expressed as a rank-biserial correlation.

Relationships between SII, BASDAI, BASFI, age, and BMI were explored with Spearman's rank correlation.

To test how well SII distinguished active (BASDAI ≥4) from inactive disease, we built a receiver operating characteristic (ROC) curve,

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reported the area under it (AUC) with its 95% confidence interval, and chose the threshold that maximized the Youden index (sensitivity+specificity-1).

At that threshold we report sensitivity, specificity, and positive and negative predictive values (PPV, NPV).

Statistical significance was set at a two-sided $p < 0.05$.

Analyses were run in R version 4.5.1 (R Foundation for Statistical Computing, Vienna, Austria).

Results

Baseline characteristics

Table 1 summarizes the characteristics of the cohort by SpA subtype.

Patients were on average 53.5±12.8 years old, and age differed between subtypes ($p<0.001$): the mean age was 53.5±12.8 years, with significant differences across subtypes ($p<0.001$): psoriatic SpA patients were the oldest (62±11.5 years), while peripheral SpA patients were the youngest (41.5±13.4 years).

Males predominated in axial SpA (62.5%) but constituted only 25% of both peripheral and psoriatic SpA groups ($p=0.010$).

Hypertension was significantly more prevalent in psoriatic SpA (54.2%) compared with axial (28.1%) and peripheral SpA (12.5%) ($p=0.045$), while diabetes did not differ across subtypes ($p=0.57$).

Mean BASDAI was 5.26±1.44, within the active range for the cohort as a whole, and was comparable across subtypes ($p=0.247$), as was BASFI (4.91±1.57; $p=0.233$).

Overall SII averaged 834±505 (median 627, IQR 498-1054) and did not vary by subtype ($p=0.801$).

Table 1. Demographic and clinical data of the study cohort (n=64) by spondyloarthritis subtype.

Characteristics	Total (N=64)	Axial SpA (N=32)	Peripheral SpA (N=8)	Psoriatic SpA (N=24)	p
Age, years					
mean±SD	53.5±12.8	50.1±9.4	41.5±13.4	62±11.5	
median (IQR)	53 (43.8-62.3)	47.5 (43-55.3)	42.5 (37-45.8)	59.5 (53-70.5)	< 0.001*
Sex, male (n, %)	28 (43.8%)	20 (62.5%)	2 (25%)	6 (25%)	0.010*
Diabetes (n, %)	7 (10.6%)	3 (9.4%)	1 (12.5%)	3 (12.5%)	0.57
Hypertension (n, %)	23 (35.9%)	9 (28.1%)	1 (12.5%)	13 (54.2%)	0.045*
SII					
mean±SD	834±505	875±549	770±321	800±505	
median (IQR)	627 (498-1054)	802 (523-1091)	606 (538-962)	612 (465-942)	0.801
BASDAI					
mean±SD	5.26±1.44	5.5±1.65	5.64±1.24	4.81±1.12	
median (IQR)	5 (4.07-6.23)	5 (4.9-6.75)	5.7 (4.9-6.05)	4.6 (4-5.62)	0.247
BASFI					
mean±SD	4.91±1.57	5.16±1.71	5.34±1.82	4.43±1.19	
median (IQR)	4.2 (3.7-6)	4.3 (3.7-6.7)	4.9 (4.52-6.05)	4 (3.68-4.82)	0.233

Data presented as mean±SD, median (IQR), or n (%). *p<0.05. SII: Systemic Immune-Inflammation Index; BASDAI: Bath Ankylosing Spondylitis Disease Activity Index; BASFI: Bath Ankylosing Spondylitis Functional Index.

SII distribution across SpA subtypes

The distribution of SII across the three SpA subtypes is illustrated in Figure 1.

Axial SpA patients had the highest mean SII (875.31), followed by psoriatic SpA (800.22) and peripheral SpA (770.31).

However, these differences were not statistically significant (Kruskal-Wallis $\chi^2(2)=0.44$, $p=0.80$).

The effect size was negligible ($\varepsilon^2=7.05 \times 10^{-3}$, 95% CI 3.53×10^{-3} to 1.00), confirming that SpA subtype explains virtually none of the variance in SII.

Violin plots revealed a right-skewed distribution with substantial interindividual variability within all three subgroups, with SII values ranging from below 200 to above 2000 across the cohort.

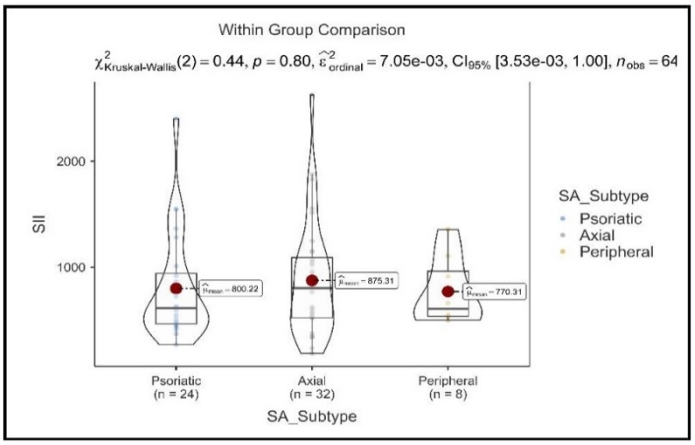


Figure 1. Comparison of SII by spondyloarthritis subtype.

SII across clinical subgroups

Mean SII was higher in the 52 patients with active disease (BASDAI ≥4) than in the 12 with inactive disease (868.58 vs. 684.29), although this did not reach statistical significance (Mann-Whitney W=242.00, p=0.23, r_{ml}=−0.22, 95% CI −0.53 to 0.14; Figure 2).

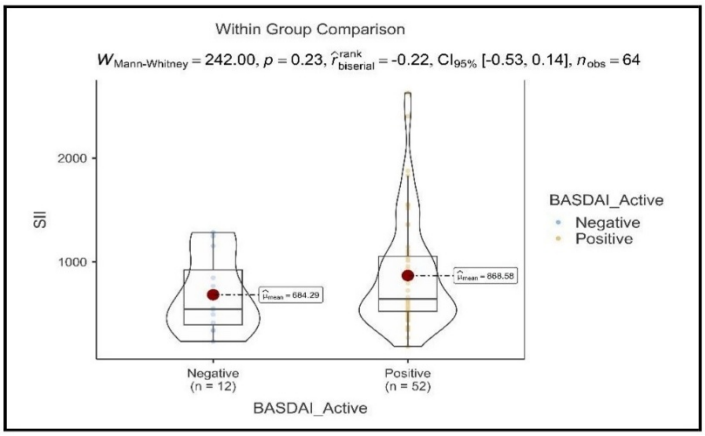


Figure 2. Comparison of SII by BASDAI (Active disease defined as BASDAI ≥4).

Mean SII was also higher in diabetic than in non-diabetic patients (1140.05 vs. 796.44), but with only seven diabetic patients and a wide dispersion of values, the difference was not significant (p=0.73; Figure 3A).

Hypertensive patients showed a trend toward higher SII compared with normotensive patients (956.36 vs. 765.40; W=595.00, p=0.09, r_{ml}=0.26), as shown in Figure 3B.

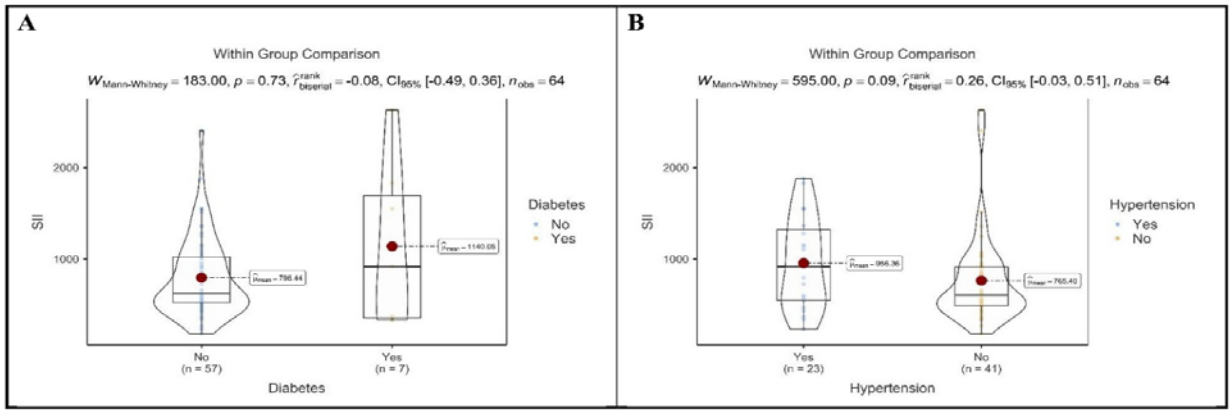


Figure 3. Comparison of SII by comorbidities.
A. By diabetes. B. By hypertension. r_{ml}: rank-biserial correlation.

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Correlation analysis

The correlation matrix is shown in Figure 4.

SII increased with both BASDAI (ρ=0.350, p<0.01) and BASFI (ρ=0.316, p<0.05) and decreased as BMI increased (ρ=−0.250, p<0.05), but showed no relationship with age (ρ=−0.133, p>0.05).

BASDAI and BASFI were closely related to each other (ρ=0.787, p<0.001).

Older patients tended to have a higher BMI (ρ=0.337, p<0.01) and a lower BASDAI (ρ=−0.264, p<0.05).

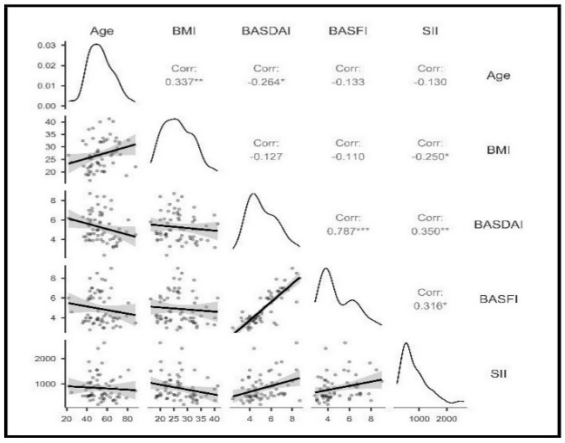


Figure 4. Spearman rank correlation matrix.

ROC curve analysis of SII for active disease

Figure 5 shows the ROC curve for SII in distinguishing active from inactive disease.

The AUC of 0.612 (95% CI 0.421-0.803) indicates only poor-to-fair discrimination.

The Youden index was maximized at an SII of 565.01, giving 63.5% sensitivity and 58.3% specificity.

At this threshold, 86.8% of patients classified as positive truly had active disease (PPV), a figure inflated by the 81.3% prevalence of active disease in the sample, whereas only 26.9% of those classified as negative were truly inactive (NPV).

Because the confidence interval of the AUC (0.421-0.803) includes 0.5, the value expected by chance, there is considerable uncertainty around this estimate, consistent with the limited sample size and the highly unbalanced distribution of active versus inactive patients (52 vs. 12).

The relatively high PPV (86.8%) is driven primarily by the high baseline prevalence of active disease rather than by the intrinsic discriminative power of SII, whereas the low NPV (26.9%) indicates that a SII below 565 does not reliably exclude active disease.

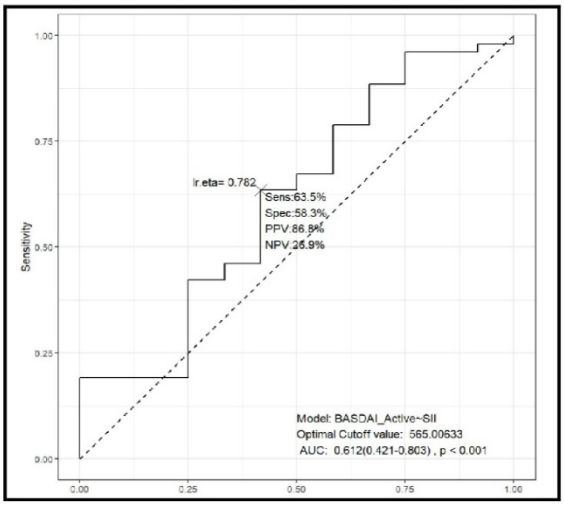


Figure 5. ROC curve of SII for active disease of spondylarthritis.

Discussion

This cross-sectional study evaluated SII across the full spectrum of spondyloarthritis and its associations with disease activity, functional impairment, and clinically relevant subgroups.

The principal findings are: (1) SII correlates significantly with both BASDAI and BASFI, establishing it as an objective hematologic index that reflects patient-reported disease burden; (2) SII values are comparable across axial, peripheral, and psoriatic SpA subtypes, with a negligible effect size; (3) ROC curve analysis yields an AUC of 0.612 for discriminating active from inactive disease, with an optimal cutoff of 565; and (5) SII is inversely correlated with BMI.

The significant correlations between SII and BASDAI (ρ=0.350, p<0.01) and BASFI (ρ=0.316, p<0.05) represent the central finding of this study.

These moderate associations indicate that approximately 12% and 10% of the variance in disease activity and functional impairment, respectively, can be explained by SII.

While modest, this magnitude is consistent with what conventional markers typically achieve against BASDAI: CRP-BASDAI correlations in SpA cohorts generally range from 0.15 to 0.40 [5,6].

The fact that an objective laboratory composite correlates significantly with subjective patient-reported outcomes supports the biological validity of SII as a systemic inflammation marker in SpA.

Our findings are concordant with those of Wu et al., who reported significant SII-BASDAI correlations in ankylosing spondylitis (n=136) [12], and with Predescu et al., who demonstrated associations between cellular immune

inflammation markers and disease activity indices in a mixed SpA cohort [13].

Importantly, our study extends these observations to the SII-BASFI relationship, which has been insufficiently explored.

The parallel correlation pattern suggests that SII captures the inflammatory component that drives both symptom perception and functional limitation, consistent with the strong BASDAI-BASFI coupling observed in our cohort (ρ=0.787).

From a biological standpoint, these correlations are coherent. SII integrates neutrophil counts (reflecting IL-17/IL-23 axis activation and innate immune engagement at entheseal and synovial sites), platelet counts (a marker of IL-6-driven thromboinflammation), and lymphocyte counts (which may decline during chronic immune activation due to redistribution to inflamed tissues) [9,10].

By combining these parameters, SII captures a broader inflammatory signal than any individual component.

The absence of significant SII differences across axial, peripheral, and psoriatic SpA ($\chi^2(2)$=0.44, p=0.80, $\varepsilon^2=7.05 \times 10^{-3}$) is one of the most conceptually important findings of this study.

Despite well-known differences in pathogenesis, primary inflammatory sites, and genetic architecture among SpA subtypes [1,2], the systemic immune-inflammatory milieu-as captured by circulating blood cell ratios-appears remarkably comparable.

The negligible effect size confirms that this is not merely a power issue; SpA subtype genuinely explains virtually none of the SII variability.

This uniformity may reflect a shared upstream inflammatory drive consistent with the concept of a unified SpA spectrum in which the IL-23/IL-17 axis plays a central role across phenotypes [18].

Alternatively, SII may predominantly capture systemic rather than local inflammation; subtype-specific differences may manifest at the tissue level (enthesitis, synovitis, sacroiliitis, skin inflammation) without proportionally affecting circulating blood cell ratios.

From a clinical standpoint, this finding supports the potential utility of SII as a subtype-agnostic marker, simplifying laboratory monitoring in mixed SpA clinics.

The ROC curve analysis revealed that SII has poor to fair discriminative ability for identifying patients with active disease (BASDAI≥4), with an AUC of 0.612 (95% CI 0.421-0.803).

The optimal cutoff of 565 yielded a sensitivity of 63.5% and specificity of 58.3%, with a high PPV of 86.8% but a low NPV of 26.9%.

Several considerations contextualize this finding.

First, the wide confidence interval crossing 0.5 reflects substantial uncertainty, driven by the small inactive disease group (n=12).

Second, the high PPV is largely a function of the high prevalence of active disease (81.3%) rather than intrinsic SII accuracy-in a population with more balanced disease activity distribution, the PPV would be considerably lower.

Third, the moderate sensitivity and specificity are not unexpected for a single hematologic index attempting to discriminate a complex clinical state defined by a multidimensional patient-reported instrument.

Notably, CRP and ESR have shown similarly modest AUC values (0.55-0.70) for discriminating active from inactive axial SpA in prior studies [5,12].

The SII cutoff of 565 identified here should be considered hypothesis-generating rather than definitive, and requires external validation in larger, independent cohorts with more balanced active-to-inactive ratios.

Among the subgroup comparisons, the borderline elevation of SII in hypertensive patients (mean 956.36 vs. 765.40, p=0.09, r_{ml}=0.26) is the most clinically interesting finding.

This is biologically plausible given the bidirectional relationship between systemic inflammation and hypertension through pro-inflammatory cytokines, endothelial dysfunction, and vascular stiffness [19,20].

The finding aligns with the increasingly recognized cardiovascular-inflammation axis in SpA, particularly in psoriatic arthritis [21].

The comparison between active and inactive disease showed a 27% higher mean SII in the active group (868.58 vs. 684.29, p=0.23), but the highly asymmetric distribution (52 vs. 12 patients) yielded estimated power of only 25-30%, suggesting an underpowered rather than negative finding.

The significant negative correlation between SII and BMI (ρ=−0.250, p<0.05) is unexpected. In the context of chronic active SpA (mean BASDAI >5), TNF-α-mediated cachexia may produce lower BMI in the most inflamed patients.

Additionally, hemodilution in patients with higher adipose mass could reduce blood cell concentrations.

This inverse relationship has been described in other chronic inflammatory conditions as part of the “obesity paradox” [22] and suggests that

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BMI should be considered as a covariate when interpreting SII.

This study has several limitations.

The cross-sectional design precludes causal inferences. The peripheral SpA subgroup (n=8) was small, limiting statistical power.

The ROC analysis was constrained by the highly unbalanced distribution of active versus inactive disease (52 vs. 12), which inflates PPV and limits the reliability of cutoff derivation.

Treatment data were not collected, preventing adjustment for pharmacological effects on SII.

The absence of joint counts, enthesitis scores, PASI, imaging data, and disease duration limits phenotypic characterization.

BASDAI was developed for axial SpA and may not optimally capture disease activity in peripheral and psoriatic forms [7].

A healthy control group was not included, precluding assessment of whether SII is elevated in SpA relative to the general population.

Among strengths, this is one of the first studies to evaluate SII simultaneously across all three major SpA subtypes within a single cohort, and effect sizes with confidence intervals are reported alongside p-values.

Conclusions

In this cross-sectional cohort of 64 SpA patients, SII demonstrated significant positive correlations with both BASDAI ($\rho=0.350$, $p<0.01$) and BASFI ($\rho=0.316$, $p<0.05$), supporting its role as an objective hematologic biomarker of disease activity and functional impairment.

SII values were remarkably uniform across axial, peripheral, and psoriatic SpA subtypes ($\varepsilon^2=7.05 \times 10^{-3}$), suggesting that it captures a shared systemic inflammatory process.

ROC analysis identified an optimal SII cutoff of 565 for discriminating active from inactive disease (AUC=0.612), with moderate sensitivity (63.5%) and specificity (58.3%); however, the wide confidence interval and unbalanced group sizes indicate that this cutoff requires external validation.

The borderline association with hypertension highlights the inflammation-cardiovascular interface in SpA.

These findings position SII as a promising, readily accessible complement to patient-reported outcomes in spondyloarthritis monitoring.

Larger longitudinal studies incorporating treatment data, objective disease assessments, healthy controls, and balanced disease activity distributions are needed to refine SII cutoff values and define its clinical utility.

Abbreviations

ASAS=Assessment of SpondyloArthritis International Society; AUC=area under the curve; axSpA=axial spondyloarthritis; BASDAI=Bath Ankylosing Spondylitis Disease Activity Index; BASFI=Bath Ankylosing Spondylitis Functional Index; BMI=body mass index; CI=confidence interval; CRP=C-reactive protein; DM=diabetes mellitus; ESR=erythrocyte sedimentation rate; HTN=hypertension; IL=interleukin; IQR=interquartile range; NPV=negative predictive value; PPV=positive predictive value; PsA=psoriatic arthritis; pSpA=peripheral spondyloarthritis; ROC=receiver operating characteristic; SD=standard deviation; SII=Systemic Immune-Inflammation Index; SpA=spondyloarthritis; STROBE=Strengthening the Reporting of Observational Studies in Epidemiology; TNF=tumor necrosis factor.

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None to declare.

Author Contributions

Conceptualization, F.A.V. C.E.B..; Methodology, R.D.U., M.N.V., A.T. M.T.S.; Investigation, R.D.U., S.C.F, S.C.D., A.T.S.; Data analysis, M.T.S and A.T.S..; Manuscript writing and initial draft preparation, R.D.U, A.L.B, C.E.B., A.T.; Manuscript review and editing, A.T.S., S.C.D. F.A.V; Supervision, F.A.V.

All authors read and approved the final manuscript.

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Conflicts of interest

The authors declare no competing interests.

Institutional Review Board

The protocol was approved by the Ethics Committee of the University of Medicine and Pharmacy of Craiova (No. 201/24.11.2021) and complied with the Declaration of Helsinki.

Consent Statement

All participants gave written informed consent.

Data availability

The original data presented in the study are openly available in at DOI https://doi.org/10.5281/zenodo.18763989.

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