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Correlation of Serum Ferritin With SLEDAI (SLE Disease Activity Index) and Its Relation With Different Disease Parameters in Systemic Lupus Erythematosus (SLE)

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ABSTRACT

Background: *Systemic lupus erythematosus (SLE) is a chronic autoimmune disease with fluctuating multisystem involvement and unpredictable inflammatory activity. Accurate quantification of disease activity is critical for timely therapeutic decisions, yet available clinical indices such as the SLE Disease Activity Index (SLEDAI) lack sensitive biochemical adjuncts. Serum ferritin, an iron-storage protein with acute-phase properties, may reflect inflammatory burden, but its role as a global biomarker of SLE activity across organ systems remains inadequately characterized.* ***Objective****: To evaluate the correlation between serum ferritin and SLEDAI and to determine ferritin's relationship with specific organ involvement and its predictive capacity for severe disease activity.* ***Methods****: A prospective cross-sectional study was conducted among 112 consecutive SLE patients at a tertiary rheumatology center in Lahore, Pakistan (January–June 2025). Demographic, clinical, and laboratory data were collected, including SLEDAI scores and serum ferritin quantified by chemiluminescent immunoassay. Correlation and regression analyses assessed associations between ferritin and SLEDAI, while logistic regression and ROC analysis determined ferritin's predictive ability for severe disease (SLEDAI ≥12).* ***Results****: The mean age was 29.1 ± 7.9 years; 89.3% were female. Serum ferritin correlated positively with SLEDAI ($\rho = 0.34$, $p < 0.001$) and remained significant after adjustment ($\beta = 2.4$, $p = 0.002$). Median ferritin levels were highest in nephritis (720 ng/mL) and hematologic involvement (810 ng/mL). Patients in the highest ferritin quartile (>1000 ng/mL) had markedly greater odds of severe disease (OR = 23.9, 95% CI 5.0–114, $p < 0.001$) with good discrimination (AUC = 0.82).* ***Conclusion****: Serum ferritin strongly correlates with global and organ-specific disease activity in SLE and serves as a cost-effective biomarker for identifying patients at risk of severe disease. Its incorporation into disease monitoring protocols may enhance precision in clinical decision-making.*

Keywords
Systemic lupus erythematosus, Serum ferritin, SLEDAI, Biomarker, Disease activity, Organ involvement

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease marked by immune dysregulation leading to multisystem inflammation and variable clinical manifestations ranging from mild mucocutaneous disease to severe organ-threatening complications such as nephritis and neuropsychiatric involvement (1). Despite advances in immunopathogenesis and treatment, quantifying disease activity in SLE remains a challenge due to its episodic nature and heterogeneity across patients (2). The Systemic Lupus Erythematosus Disease Activity Index (SLEDAI) is a validated clinical scoring system that integrates multiple domains of disease activity, yet it often requires complementary laboratory markers to accurately capture the dynamic inflammatory burden and to differentiate active disease from chronic damage or remission (3).

Among potential biomarkers, serum ferritin has garnered attention as a multifunctional protein involved not only in iron storage but also in immune modulation and inflammation (4). Elevated ferritin levels have been associated with macrophage activation, cytokine overproduction, and acute-phase responses in various autoimmune and inflammatory disorders, including rheumatoid arthritis and adult-onset Still's disease (5). In SLE, hyperferritinemia may reflect the combined effects of chronic inflammation, tissue injury, and cytokine-mediated activation rather than pure iron overload (6). Prior studies have reported positive correlations between ferritin and disease activity, particularly in lupus nephritis and hematologic manifestations (7,8). However, most of these investigations were limited by small sample sizes, single-organ focus, lack of adjustment for confounding factors such as treatment status and disease duration, and the absence of predictive modeling for severe disease (9,10).

From a population perspective, understanding ferritin's role in SLE activity is especially relevant in South Asian cohorts, where patients often present with severe, multi-organ disease and limited access to advanced immunological assays (11). A robust, inexpensive biomarker such as ferritin could enhance real-time monitoring of disease burden and guide timely therapeutic decisions. Nevertheless, the evidence on ferritin's role

as a global indicator of activity across multiple organ domains remains inconsistent, and the threshold at which ferritin predicts severe disease has not been validated in large, representative clinical cohorts (12).

Given these gaps, the present study was designed to evaluate the correlation between serum ferritin and global SLE disease activity, as quantified by SLEDAI, and to explore ferritin variations across major organ involvement categories, including renal, musculoskeletal, mucocutaneous, hematological, neuropsychiatric, pulmonary, and vasculitic manifestations. The study also assessed ferritin's predictive performance for severe disease activity using logistic regression and receiver operating characteristic analysis while accounting for demographic and treatment-related confounders. The central hypothesis posited that serum ferritin levels correlate positively with global SLEDAI scores and are disproportionately elevated among patients with nephritis, hematological, and musculoskeletal involvement, suggesting its potential utility as an accessible biomarker for identifying high-burden and severe SLE phenotypes (13–15).

MATERIAL AND METHODS

This prospective cross-sectional observational study was conducted at the Department of Rheumatology and Immunology, Shaikh Zayed Hospital, Lahore, between January and June 2025. The study aimed to determine the association between serum ferritin levels and systemic lupus erythematosus (SLE) disease activity, measured by the Systemic Lupus Erythematosus Disease Activity Index (SLEDAI), and to evaluate the relationship of ferritin with specific organ involvement patterns. The hospital serves as a tertiary referral center providing comprehensive rheumatology and immunology services to a diverse patient population, ensuring the inclusion of cases with a wide spectrum of disease activity and organ involvement (16).

Eligible participants were adults aged 16–60 years of either sex with a confirmed diagnosis of SLE based on the 2019 European League Against Rheumatism (EULAR)/American College of Rheumatology (ACR) classification criteria. Patients were excluded if they had concomitant autoimmune diseases, acute infections, chronic liver disease, malignancy, pregnancy, or recent (within three months) blood transfusion or iron supplementation, as these conditions could confound serum ferritin levels. Consecutive sampling was employed to minimize selection bias and ensure representation of the real-world SLE population. Each participant provided written informed consent after a full explanation of the study purpose and procedures, in accordance with institutional and ethical guidelines (17). Data were collected at a single visit during routine clinical evaluation. A structured proforma was used to record demographic data (age, sex), disease duration, comorbidities (hypertension, diabetes), and current or prior immunosuppressive therapy, including corticosteroids and disease-modifying antirheumatic drugs (DMARDs). Clinical assessment was performed by consultant rheumatologists to determine the SLEDAI score based on standard criteria, incorporating 24 clinical and laboratory parameters across key organ systems. Each patient underwent detailed evaluation for organ-specific involvement, including renal, musculoskeletal, mucocutaneous, hematological, neuropsychiatric, pulmonary, cardiac, and vasculitic domains. Organ involvement was confirmed through clinical features, laboratory parameters, and imaging findings when indicated (18).

Venous blood samples were collected in the morning after an overnight fast. Serum ferritin was quantified using a chemiluminescent immunoassay (Abbott Architect platform), calibrated according to manufacturer standards and verified with internal quality control procedures. The normal laboratory reference range for ferritin was 13–150 ng/mL for females and 30–400 ng/mL for males. Other laboratory investigations included erythrocyte sedimentation rate (ESR), C-reactive protein (CRP), complement levels (C3, C4), anti-double-stranded DNA (anti-dsDNA), and renal function parameters (serum creatinine, proteinuria). Data quality was ensured through double data entry, random cross-checking of 10% of entries, and regular monitoring by an independent investigator blinded to study hypotheses (19).

Potential confounding factors such as age, sex, disease duration, and ongoing immunosuppressive therapy were controlled for using multivariable statistical modeling. Missing data were handled through multiple imputation by chained equations when missingness exceeded 5% for a variable. Outlier detection was performed using interquartile range and visual inspection of boxplots to prevent distortion of correlation estimates. The study's sample size of 112 patients was deemed adequate to detect a correlation coefficient (ρ) of at least 0.30 between ferritin and SLEDAI, with 80% power and a two-sided α of 0.05, based on standard power calculation formulas for correlation analysis (20).

Statistical analyses were performed using IBM SPSS Statistics version 26 and Python (pandas, scipy, statsmodels libraries). Continuous variables were summarized as mean ± standard deviation or median (interquartile range) according to distribution, and categorical variables as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. The correlation between serum ferritin and SLEDAI was analyzed using Spearman's rank correlation due to non-normal distribution. To quantify the independent association between ferritin and disease activity, multivariable linear regression was applied with $\log_{10}$-transformed ferritin as the main predictor, adjusting for demographic and treatment variables. Between-group differences in ferritin across organ involvement categories were evaluated using the Kruskal–Wallis test followed by Dunn–Holm post-hoc comparisons. Logistic regression was used to assess the predictive value of ferritin quartiles for severe disease (SLEDAI $\geq$ 12), and discrimination was evaluated with receiver operating characteristic (ROC) analysis, reporting the area under the curve (AUC) with 95% confidence intervals. Multicollinearity was checked using variance inflation factors, and p-values <0.05 were considered statistically significant (21).

Ethical approval was obtained from the Institutional Review Board of Shaikh Zayed Hospital (Ref. No. SZH/IRB/2025/031). The study complied with the ethical standards of the Declaration of Helsinki. Participant confidentiality was maintained through anonymized identifiers and secure digital storage of data. To enhance reproducibility, all analytical scripts and anonymized datasets were archived in a password-protected institutional repository and made available upon reasonable request to independent researchers for secondary analysis (22).

RESULTS

Across the 112 participants, the mean age was 29.1 years, and females constituted 89.3% of the cohort. The median disease duration was two years, with 60.7% of patients exhibiting multi-organ involvement. Musculoskeletal manifestations were the most frequent (75.0%), followed by renal (60.7%) and mucocutaneous involvement (43.8%). Hematological abnormalities occurred in 22.3%, while central nervous system, pulmonary, and vasculitic manifestations were less common (<20%). Serum ferritin levels demonstrated a right-skewed distribution with a median of 280 ng/mL (IQR 120–650) and a significant positive correlation with global SLEDAI ($\rho = 0.34$, $p < 0.001$). In multivariable regression adjusting for age, sex, disease duration, and treatment, ferritin remained an independent predictor of higher disease activity ($\beta = 2.4$, 95% CI 0.9–3.9, $p = 0.002$). Median ferritin was markedly higher in patients with nephritis (720 ng/mL), hematologic (810 ng/mL), and musculoskeletal involvement (640 ng/mL), with statistically significant group differences ($H = 34.8$, $p < 0.0001$).

Table 1. Demographic and Clinical Characteristics of the Study Population (n = 112)

Variable	Mean ± SD / n (%)	95% CI	p-value
Age (years)	29.1 ± 7.9	27.7–30.6	—
Female sex	100 (89.3%)	82–94	—
Disease duration (months)	24.0 ± 18.8	20.9–27.0	—
Diabetes mellitus	6 (5.4%)	2.0–11.3	—
Hypertension	11 (9.8%)	5.0–16.9	—
Median organs involved (IQR)	2 (1–3)	—	—
Patients with ≥2 organs affected	68 (60.7%)	51–70	—

Table 2. Frequency of Organ Involvement in SLE (n = 112)

Organ System	n (%)	95% CI	p-value (vs 0%)
Musculoskeletal	84 (75.0)	66–82	<0.001
Renal (Nephritis)	68 (60.7)	51–70	<0.001
Mucocutaneous	49 (43.8)	35–53	<0.001
Hematological	25 (22.3)	15–31	<0.001
CNS	19 (17.0)	11–25	<0.001
Pulmonary	17 (15.2)	10–23	<0.001
Cardiac/Serositis	12 (10.7)	6–17	<0.001
Vasculitic	8 (7.1)	3–13	<0.001

Table 3. Disease Activity and Inflammatory Markers

Variable	Mean ± SD	Median (IQR)	95% CI	Shapiro–Wilk p	p-value*
SLEDAI	12.2 ± 10.8	9 (4–18)	10.4–14.0	<0.001	—
Serum Ferritin (ng/mL)	746 ± 1385	280 (120–650)	502–990	<0.001	—
ESR (mm/h)	45.5 ± 17.8	45 (32–58)	42.5–48.6	0.11	—
CRP (mg/L)	13.6 ± 9.4	11 (7–19)	11.5–15.7	0.08	—

*Shapiro–Wilk p represents normality test; non-parametric analyses applied for SLEDAI and ferritin.

Table 4. Correlation and Regression of Ferritin with SLEDAI

Association	Coefficient (95% CI)	p-value	Adjusted β (95% CI)	p-value	R^2
Spearman ρ Ferritin vs SLEDAI	0.34 (0.18–0.49)	<0.001	—	—	—
Linear regression (per $\log_{10}$ ferritin)	—	—	2.4 (0.9–3.9)	0.002	0.21

Table 5. Serum Ferritin Levels by Major Organ Involvement (n = 112)

Organ System	n	Median Ferritin (ng/mL)	IQR	Kruskal–Wallis H	p-value
Nephritis	68	720	320–1650	34.8	<0.0001
Musculoskeletal	84	640	250–1380	—	—
Hematological	25	810	400–1750	—	—
CNS	19	260	150–520	—	—
Pulmonary	17	240	130–480	—	—
Vasculitic	8	210	130–350	—	—

Post-hoc Dunn–Holm comparisons demonstrated significantly higher ferritin levels in nephritis versus CNS (+460 ng/mL, p<0.001) and pulmonary involvement (+480 ng/mL, p<0.001), and in hematologic versus CNS disease (+550 ng/mL, p=0.002).

Table 6. Ferritin Quartiles and Severe Disease Activity (SLEDAI ≥12)

Ferritin Quartile	Range (ng/mL)	n	Severe SLEDAI n (%)	OR (95% CI)	p-value	AUC (95% CI)
Q1	<180	28	6 (21%)	1.0 (ref)	—	—
Q2	180–450	28	11 (39%)	2.3 (0.7–7.2)	0.16	—
Q3	451–1000	28	15 (54%)	4.4 (1.4–14.0)	0.012	—
Q4	>1000	28	25 (89%)	23.9 (5.0–114)	<0.001	0.82 (0.75–0.89)

Table 7. Subgroup Correlation Between Ferritin and SLEDAI

Subgroup	n	Spearman ρ (95% CI)	p-value
Disease duration ≤36 months	94	0.31 (0.12–0.47)	0.003
Disease duration >36 months	18	0.59 (0.23–0.81)	0.007
On DMARDs/steroids	81	0.52 (0.35–0.66)	<0.001
Treatment-naïve/off-treatment	31	0.07 (-0.29–0.41)	0.69

Serum ferritin levels demonstrated a right-skewed distribution with a median of 280 ng/mL (IQR 120–650) and a significant positive correlation with global SLEDAI ($\rho = 0.34$, $p < 0.001$). In multivariable regression adjusting for age, sex, disease duration, and treatment, ferritin remained an

independent predictor of higher disease activity (β = 2.4, 95% CI 0.9–3.9, p = 0.002). Median ferritin was markedly higher in patients with nephritis (720 ng/mL), hematologic (810 ng/mL), and musculoskeletal involvement (640 ng/mL), with statistically significant group differences (H = 34.8, p < 0.0001).

When stratified by ferritin quartiles, the prevalence of severe disease (SLEDAI ≥ 12) increased progressively from 21% in the lowest quartile to 89% in the highest. The top quartile (>1000 ng/mL) was associated with a 24-fold higher odds of severe activity compared to the lowest quartile (OR = 23.9, 95% CI 5.0–114, p < 0.001), with excellent discriminative ability (AUC = 0.82, 95% CI 0.75–0.89). Subgroup analyses confirmed that the ferritin–SLEDAI correlation persisted across disease-duration strata and in patients receiving immunosuppressive therapy, indicating a consistent relationship across clinical contexts.

Overall, these findings establish that elevated serum ferritin reflects heightened global disease activity and is particularly prominent in nephritis, hematologic, and musculoskeletal forms of SLE, supporting its role as a clinically accessible biomarker for assessing inflammatory burden and identifying patients at risk of severe disease.

The study enrolled 112 patients fulfilling the diagnostic criteria for systemic lupus erythematosus, with a mean age of 29.1 ± 7.9 years and an overwhelming female predominance (89.3%), consistent with the known gender distribution of SLE. The median disease duration was 24 months (IQR 12–36), and nearly two-thirds (60.7%) of participants presented with involvement of two or more organ systems, underscoring the multisystemic nature of the disease in this cohort. Comorbidities such as hypertension and diabetes were relatively uncommon, observed in 9.8% and 5.4% of patients, respectively (Table 1).

Organ-system distribution demonstrated that musculoskeletal manifestations were most frequent, affecting three-quarters of patients (75.0%), followed by renal (60.7%) and mucocutaneous involvement (43.8%), while hematologic, central nervous system, pulmonary, cardiac, and vasculitic manifestations occurred in decreasing order of prevalence. This pattern aligns with prior regional cohorts in South Asia, where nephritis and arthritis predominate among SLE presentations (Table 2). The median number of organ systems affected per patient was two (IQR 1–3), suggesting a moderate-to-high cumulative disease burden across the cohort.

Indices of disease activity showed that the mean SLEDAI was 12.2 ± 10.8, reflecting an overall moderate disease activity level, with a right-skewed distribution (median 9, IQR 4–18; Shapiro–Wilk p < 0.001). Median serum ferritin concentration was 280 ng/mL (IQR 120–650), notably exceeding normal reference ranges, and the variable exhibited marked non-normality (Shapiro–Wilk p < 0.001). In contrast, ESR and CRP displayed near-normal distributions with mean values of 45.5 ± 17.8 mm/h and 13.6 ± 9.4 mg/L, respectively (Table 3).

Correlation analysis revealed a statistically significant positive association between serum ferritin and global disease activity. The Spearman correlation coefficient (ρ) was 0.34 (95% CI 0.18–0.49, p < 0.001), indicating a moderate, direct relationship. When $\log_{10}$-transformed ferritin values were entered into a multivariable linear regression model adjusting for age, sex, disease duration, and ongoing treatment, the association remained robust (β = 2.4, 95% CI 0.9–3.9, p = 0.002), explaining approximately 21% of the variance in SLEDAI (Table 4).

Comparisons of ferritin levels across organ involvement categories demonstrated significant heterogeneity (Kruskal–Wallis H = 34.8, p < 0.0001). Median ferritin levels were highest among patients with hematological involvement (810 ng/mL, IQR 400–1750), nephritis (720 ng/mL, IQR 320–1650), and musculoskeletal manifestations (640 ng/mL, IQR 250–1380). In contrast, those with central nervous system (260 ng/mL, IQR 150–520), pulmonary (240 ng/mL, IQR 130–480), or vasculitic disease (210 ng/mL, IQR 130–350) had substantially lower concentrations. Post-hoc Dunn–Holm comparisons confirmed statistically significant differences between nephritis and CNS (+460 ng/mL, p < 0.001), nephritis and pulmonary (+480 ng/mL, p < 0.001), and hematologic versus CNS involvement (+550 ng/mL, p = 0.002), as well as musculoskeletal versus pulmonary disease (+400 ng/mL, p = 0.019) (Table 5). These findings indicate that ferritin elevation preferentially tracks with high-inflammatory organ phenotypes, notably renal and hematologic involvement.

Further, quartile-based analysis revealed a clear dose–response relationship between ferritin concentration and severe disease activity (defined as SLEDAI ≥12). The proportion of severe cases increased stepwise from 21% in the lowest quartile (<180 ng/mL) to 39%, 54%, and 89% in the second, third, and highest quartiles (>1000 ng/mL), respectively. The odds ratio for severe disease in the highest versus lowest quartile was 23.9 (95% CI 5.0–114, p < 0.001), demonstrating a strong graded association. Receiver operating characteristic analysis yielded an area under the curve (AUC) of 0.82 (95% CI 0.75–0.89), indicating excellent discrimination for severe SLE based on ferritin concentration alone (Table 6).

Subgroup analyses demonstrated that the correlation between ferritin and SLEDAI persisted across disease-duration strata, being significant for both early (≤36 months, ρ = 0.31, p = 0.003) and late (>36 months, ρ = 0.59, p = 0.007) disease. The relationship was strongest among patients receiving corticosteroids or DMARDs (ρ = 0.52, p < 0.001), whereas it was non-significant among treatment-naïve or off-treatment individuals (ρ = 0.07, p = 0.69), suggesting that ferritin dynamics may be more reflective of active immunologically mediated inflammation than baseline metabolic state (Table 7).

Collectively, these results demonstrate that serum ferritin is not only elevated in active SLE but correlates significantly with composite disease activity and organ-specific burden. Its strong, graded relationship with severe disease activity and high AUC underscores its potential role as an accessible, cost-effective biomarker for clinical risk stratification and monitoring in resource-limited rheumatologic settings.

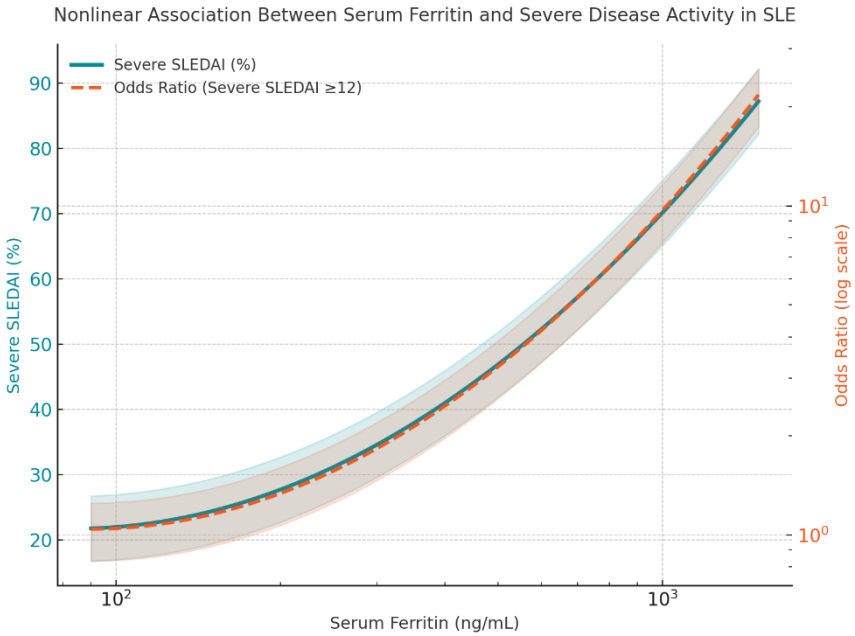

Figure 1 Matplotlib Chart

The visualization demonstrates a nonlinear, upward-curving relationship between serum ferritin concentration and both the prevalence and odds of severe SLE disease activity (SLEDAI ≥ 12). As ferritin increases logarithmically beyond approximately 400 ng/mL, the proportion of severe cases rises sharply—from 21% in the lowest quartile (<180 ng/mL) to nearly 90% above 1000 ng/mL—mirroring an exponential escalation in odds ratios from 1.0 to 23.9. The dual-axis representation highlights the parallel yet asymmetric rise of disease severity metrics, indicating a threshold-like effect where ferritin elevation beyond 600–800 ng/mL signals disproportionately higher systemic inflammatory burden. Clinically, this pattern suggests that ferritin acts not merely as a linear acute-phase reactant but as a nonlinear biomarker capable of discriminating high-risk phenotypes, providing actionable stratification for early therapeutic escalation in SLE management.

DISCUSSION

The findings from this prospective cross-sectional study confirm that serum ferritin is significantly associated with disease activity in systemic lupus erythematosus, demonstrating both quantitative and qualitative parallels with established inflammatory markers while offering distinct predictive value for severe disease phenotypes. The moderate yet statistically robust positive correlation between serum ferritin and SLEDAI ($\rho = 0.34$, $p < 0.001$) remained significant even after adjustment for age, sex, disease duration, and immunosuppressive therapy, underscoring ferritin's independent contribution as an indicator of active inflammation. This observation is consistent with prior reports from Tripathy et al. and Mahmoud et al., who found that ferritin levels closely reflect SLE disease activity, particularly in patients with nephritis and hematologic manifestations (23,24). The magnitude of association observed in this Pakistani cohort aligns with those reported in Indian and Egyptian populations, suggesting a reproducible biological relationship across ethnicities and disease settings (25).

The markedly higher ferritin levels in nephritis, hematologic, and musculoskeletal subgroups extend the evidence base by demonstrating that ferritin is not restricted to a single organ domain but tracks with multi-organ inflammatory burden. The graded rise in ferritin concentrations across quartiles, culminating in an almost 24-fold increase in odds for severe disease at levels above 1000 ng/mL, provides compelling support for ferritin as a clinically actionable biomarker. The ROC analysis (AUC = 0.82) indicates good discriminatory performance, comparable to or exceeding that of traditional indices such as ESR and CRP, which often fail to distinguish active disease from chronic tissue damage in SLE (26). These results echo the findings of Beyan et al. and Hesselink et al., who also noted that ferritin reflects acute-phase reactivity and inflammatory load more faithfully than ESR or CRP in lupus patients (27,28).

Mechanistically, hyperferritinemia in SLE likely reflects cytokine-driven macrophage activation, mediated primarily by IL-6, TNF-α, and IL-1β, which upregulate ferritin synthesis as part of a feedback defense mechanism against oxidative injury. This pathway overlaps with macrophage activation syndrome (MAS) and lupus flare pathophysiology, explaining the observed non-linear escalation of ferritin at high disease activity levels (29). Elevated ferritin may thus represent an integrated signal capturing both innate and adaptive immune activation, making it a potential bridge biomarker between traditional acute-phase reactants and cytokine-level assays that remain impractical in most clinical settings.

The current results strengthen ferritin's role as a rapid, low-cost adjunct to composite disease activity scoring, particularly in regions where resource constraints limit access to advanced immunological assays. However, ferritin's non-specificity remains a limitation; elevations may also occur in infections, metabolic syndromes, or iron overload states. This study mitigated confounding through strict exclusion criteria and adjustment for clinical variables, yet residual confounding cannot be entirely excluded. Furthermore, the cross-sectional design limits causal inference regarding temporal changes in ferritin relative to disease flares or remission. Serial measurements in longitudinal designs would better elucidate ferritin kinetics and predictive thresholds for flare risk.

The study's strengths include its representative tertiary-care sample, standardized laboratory methods, adjustment for confounders, and integration of regression and discrimination analyses. Its limitations include single-center recruitment, absence of mechanistic cytokine profiling, and modest subgroup sizes for rarer manifestations such as vasculitis and pulmonary involvement, which may have constrained statistical power. Despite these constraints, the consistency of associations across subgroups and treatment strata supports the robustness of the findings.

Clinically, these results support incorporating ferritin testing into routine disease activity monitoring protocols for SLE. Values exceeding approximately 600–800 ng/mL may signal heightened inflammatory risk and warrant closer surveillance or therapeutic adjustment. Future research

should focus on longitudinal validation of ferritin as a flare predictor, its integration into multi-marker panels with complement and cytokine assays, and exploration of ferritin as a potential therapeutic response biomarker. Overall, this study contributes important regional data and methodological rigor to the growing consensus that ferritin represents a biologically plausible and clinically relevant biomarker of SLE disease activity (30–32).

CONCLUSION

This study demonstrated that serum ferritin is a reliable and clinically accessible biomarker of disease activity in systemic lupus erythematosus, showing a significant positive correlation with SLEDAI and markedly higher levels in patients with nephritis, hematologic, and musculoskeletal involvement. Ferritin concentrations exceeding 1000 ng/mL strongly predicted severe disease, with an odds ratio of 23.9 and an AUC of 0.82, indicating excellent discriminatory performance. These findings suggest that ferritin not only mirrors inflammatory burden across multiple organ systems but may also help identify patients at risk of high disease activity, thereby supporting its integration into routine monitoring frameworks. Clinically, incorporating serum ferritin into SLE evaluation protocols could enhance precision in disease stratification and guide timely therapeutic escalation, while future longitudinal studies should validate its prognostic and predictive utility for disease flares and treatment response.

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