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Clinicodemographic Characteristics, Hematological Disturbances, and Activity Assessment in Systemic Lupus Erythematosus: An Observational Cross-Sectional Study at a Tertiary Care Hospital.

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Abstract

Background: SLE is an autoimmune disorder of systemic nature, displaying heterogeneous clinical expressions with notable impact on blood cell lineages. Disruptions in hematological parameters serve as reliable indicators of disease severity and are encountered frequently among SLE-affected individuals. ***Objectives:*** This study aimed to describe the clinicodemographic profile of SLE patients, document the range of blood-related abnormalities encountered, and establish their relevance to disease activity as measured by validated scoring tools. ***Methods:*** A prospective cross-sectional design was implemented at a tertiary referral centre over a 10-month study window. Participants meeting the 2019 EULAR/ACR diagnostic criteria were enrolled sequentially. Blood investigations including full blood count, peripheral film review, inflammatory markers, coagulation indices, and complement assays were obtained. The SLEDAI-2K instrument was used for disease activity scoring. Statistical significance was accepted at $p < 0.05$. ***Results:*** One hundred participants were studied, with a mean age of 35.38 ± 9.67 years and a 97:3 female-to-male ratio. Arthralgia was the leading symptom (73%), followed by arthritis (53%) and malar rash (51%); renal involvement affected 47%. Anemia was recorded in 73%, lymphopenia in 67%, leukopenia in 44%, thrombocytopenia in 34%, and pancytopenia in 22%. Anemia, leukopenia, and thrombocytopenia showed statistically significant associations with SLEDAI-2K scores. Hemoglobin and platelet levels correlated negatively with disease activity; ESR and APTT correlated positively. ***Conclusion:*** Hematological parameters constitute accessible and clinically informative biomarkers in SLE, closely linked to disease activity scores, and can meaningfully guide monitoring and therapeutic planning.

Keywords: SLE; Blood Abnormalities; SLEDAI-2K; Autoimmune Hematology; Clinicodemographic Analysis.

INTRODUCTION

Systemic lupus erythematosus (SLE) represents one of the most clinically complex autoimmune disorders, driven by a failure of self-tolerance that results in the generation of pathogenic autoantibodies and inflammatory tissue injury affecting nearly every organ system.[1] Its clinical expression varies considerably — from superficial mucocutaneous changes to life-threatening involvement of the kidneys, central nervous system, and hematopoietic compartment — making disease recognition and monitoring an ongoing challenge.[2]

Involvement of the blood and bone marrow is one of the most consistent features of this condition.[3,4] Cytopenias affecting red cells, white cells, and platelets are each recognized manifestations, with complex presentations such as Evans syndrome also reported.[5] The 2019 revision of the ACR/EULAR classification framework incorporated these blood-based abnormalities as weighted scoring domains, emphasizing their central diagnostic and prognostic role. [6]

The mechanisms underlying these abnormalities are varied. Circulating autoantibodies target blood cell antigens, triggering peripheral destruction through complement fixation and phagocytosis. Cytokine-mediated suppression of marrow progenitors further limits production of red cells, white cells, and megakaryocytes. Concurrently, immunosuppressive agents used in treatment — including glucocorticoids and alkylating agents — contribute iatrogenic myelosuppression. [7] Blood-related disturbances track with inflammatory disease burden, predict flare risk, and correlate with outcomes.[8] Given their routine availability and low cost, hematological parameters are ideally suited as longitudinal monitoring tools. Prior studies from the Indian subcontinent [9,10] have described hematological findings in SLE, but simultaneous characterization of the clinicodemographic context and correlation with disease activity scores remains limited. This study

was therefore undertaken to comprehensively characterize the clinical and hematological profile of SLE patients and to quantify associations between blood parameters and disease activity.

METHODS

A prospective, observational, cross-sectional investigation was conducted at a tertiary-level teaching hospital over 10 months (1st May 2024 to 1st February 2025). Patients presenting to the outpatient rheumatology clinics or admitted under General Medicine and Rheumatology were assessed for eligibility. Using G*Power (version 3.1.9.2) with 95% confidence and 90% power, a sample size of 100 was determined; enrollment proceeded through non-probability convenience sampling. Individuals aged 18 years or above satisfying the 2019 EULAR/ACR criteria for SLE and offering written informed consent were enrolled. Pediatric patients and those declining participation were excluded.

A structured clinical proforma documented baseline characteristics including age, sex, BMI, presenting complaints, comorbid illnesses, and treatment history. A comprehensive physical examination assessed integumentary, articular, cardiorespiratory, neurological, and vascular systems. Every participant underwent complete blood count with differential and peripheral film, ESR, CRP, kidney and liver function tests, APTT, serum C3 and C4 complement levels, Direct Coombs test, reticulocyte production index, serum ferritin, transferrin saturation, and urine microscopy.

Anemia was defined per WHO guidelines: hemoglobin below 12 g/dL in women and below 13 g/dL in men. Leukopenia was defined as total white cell count under 4,000 cells/mm³; neutropenia as absolute neutrophil count below 1,800/mm³; thrombocytopenia as platelet count below 100,000/mm³; pancytopenia as simultaneous fulfillment of all three. Anemia typing relied on peripheral film and iron panel: AOCD showed normal or reduced MCV with reduced transferrin saturation and preserved ferritin; IDA showed low MCV with depleted ferritin; AIHA required hemolysis evidence with a reactive Direct Coombs test and elevated reticulocyte count; MCV above 100 fL defined macrocytic anemia.

Disease activity was quantified using the SLEDAI-2K instrument: 0 = inactive; 1–5 = mild; 6–10 = moderate; 11–19 = high; ≥20 = very high activity. Statistical analyses were performed using IBM SPSS software. Continuous variables were expressed as mean ± standard deviation; categorical variables as counts and percentages. Group comparisons employed unpaired Student's t-test. Pearson's r assessed linear relationships between blood parameters and SLEDAI-2K scores. A two-sided $\alpha = 0.05$ was used throughout.

RESULTS

Patient Demographics and Comorbidities

The study enrolled 100 patients with a mean age of 35.38 ± 9.67 years (95% CI: 33.48–37.28). The 31–45-year group was most common (51%), with 31% in the 18–30-year bracket. Women accounted for 97 of 100 participants. Hypertension was the most frequent comorbidity (42%), followed by hypothyroidism (12%), coronary artery disease (11%), tuberculosis (9%), and diabetes mellitus (7%). Among hypertensive patients, 52.4% had coexistent chronic kidney disease..

Table 1: Baseline demographic and comorbidity data (N = 100)

Parameter	Value [n (%) or Mean ± SD]	95% CI
Mean Age (years)	35.38 ± 9.67	33.48–37.28
18–30 years	31 (31.0%)	21.9–40.1
31–45 years	51 (51.0%)	41.2–60.8
46–60 years	16 (16.0%)	8.8–23.2
>60 years	2 (2.0%)	0.0–4.7
Female sex	97 (97.0%)	93.7–100.0
Male sex	3 (3.0%)	0.0–6.3
Hypertension	42 (42.0%)	32.3–51.7
Diabetes mellitus	7 (7.0%)	2.0–12.0
Hypothyroidism	12 (12.0%)	5.6–18.4
Coronary artery disease	11 (11.0%)	4.9–17.1
Tuberculosis	9 (9.0%)	3.4–14.6
Other comorbidities	38 (38.0%)	28.5–47.5
CKD in hypertensives	22/42 (52.4%)	—

Presenting Clinical Features

Joint-related complaints dominated the clinical picture, with arthralgia in 73 patients and frank arthritis in 53. Malar rash was the most prevalent cutaneous finding (51%), followed by oral ulceration (37%) and discoid lesions (26%). Renal

involvement was documented in 47 cases, neurological features in 24, myalgia in 49, pyrexia in 37, lower limb edema in 23, precordial pain in 19, and serositis in 16 patients.

Table 2: Frequency of clinical features at presentation (N = 100)

Clinical Feature	Frequency n (%)
Arthralgia	73 (73%)
Arthritis	53 (53%)
Malar rash	51 (51%)
Discoid rash	26 (26%)
Alopecia	22 (22%)
Oral ulcers	37 (37%)
Myalgia	49 (49%)
Pyrexia	37 (37%)
Precordial pain	19 (19%)
Lower limb edema	23 (23%)
Renal disease	47 (47%)
Neurological features	24 (24%)
Serositis	16 (16%)

Blood Cell Abnormalities and Anemia Subtypes

Hematological abnormalities were nearly universal. Anemia was the most frequent finding, identified in 73 patients (73%), followed by lymphopenia (67%), leukopenia (44%), thrombocytopenia (34%), and pancytopenia (22%). Neutropenia and Evans syndrome were each noted in 3 patients, and 10 patients had a positive Direct Coombs test. Among the 73 anemic patients, anemia of chronic disease (AOCD) was the leading subtype (34 cases), followed by iron deficiency anemia (IDA) in 25, and autoimmune hemolytic anemia (AIHA) in 10. Combined AOCD plus IDA and macrocytic anemia were each found in 2 patients; 27 patients had no anemia.

Table 3: Hematological abnormalities and anemia subtypes (N = 100)

Abnormality	n (%)	95% CI
Anemia	73 (73.0%)	64.3–81.7
Leukopenia	44 (44.0%)	34.3–53.7
Lymphopenia	67 (67.0%)	57.8–76.2
Neutropenia	3 (3.0%)	0.0–6.3
Thrombocytopenia	34 (34.0%)	24.7–43.3
Pancytopenia	22 (22.0%)	13.9–30.1
Direct Coombs reactive	10 (10.0%)	4.1–15.9
Evans syndrome	3 (3.0%)	0.0–6.3
Anemia Subtypes (among 73 anemic patients)		
Anemia of Chronic Disease (AOCD)	34 (34.0%)	24.7–43.3
Iron Deficiency Anemia (IDA)	25 (25.0%)	16.5–33.5
Autoimmune Hemolytic Anemia (AIHA)	10 (10.0%)	4.1–15.9
AOCD combined with IDA	2 (2.0%)	0.0–4.7
Macrocytic Anemia	2 (2.0%)	0.0–4.7
No Anemia	27 (27.0%)	18.3–35.7

Laboratory Findings

The mean hemoglobin was 9.86 ± 1.89 g/dL, reflecting the high anemia burden. Red cell sizing was near-normal (MCV 83.90 ± 7.38 fL), though RDW was elevated (16.16 ± 1.89), indicating cell size variability. Mean ESR was 47.65 ± 32.74 mm/hr; CRP was elevated in 59% of patients. Complement C3 was reduced in 60% and C4 in 44%, consistent with active immune complex consumption. APTT was prolonged in 19%, and RPI exceeded 2 in 10%, indicating hemolytic activity. Renal involvement was biochemically supported by elevated creatinine (mean 1.69 ± 1.31 mg/dL) and reduced albumin (mean 3.16 ± 0.65 g/dL).

Table 4: Quantitative and categorical laboratory results (N = 100)

Test	Mean ± SD	95% CI
Hemoglobin (g/dL)	9.86 ± 1.89	9.49–10.23
MCV (fL)	83.90 ± 7.38	82.4–85.4
RDW	16.16 ± 1.89	15.79–16.53
Total White Cell Count (/mm³)	6015.20 ± 4423.96	5148–6882
Platelet Count (/µL)	176048.60 ± 119760.34	152508–199589
Neutrophil fraction (%)	78.93 ± 8.50	77.25–80.61
Lymphocyte fraction (%)	15.99 ± 8.70	14.27–17.71
ESR (mm/hr)	47.65 ± 32.74	41.20–54.10
Creatinine (mg/dL)	1.69 ± 1.31	1.43–1.95
Albumin (g/dL)	3.16 ± 0.65	3.03–3.29
Categorical Laboratory Abnormalities		
Elevated CRP		59 (59%)
Low C3		60 (60%)
Low C4		44 (44%)
Prolonged APTT		19 (19%)
RPI > 2		10 (10%)

Blood Abnormalities and Disease Activity

Patients with anemia, leukopenia, or thrombocytopenia had substantially higher SLEDAI-2K scores compared to those without ($p < 0.05$ for each). Mean SLEDAI in anemic patients was 17.13 ± 13.80 versus 6.43 ± 4.90 in non-anemic patients. Leukopenic patients scored 20.16 ± 14.82 versus 10.73 ± 10.00. Thrombocytopenic patients had the highest mean SLEDAI of 22.94 ± 15.03 versus 10.73 ± 11.00 in those with normal platelets. Lymphopenia was not a statistically significant predictor of SLEDAI-2K (15.55 ± 13.70 vs. 13.51 ± 12.00; $p > 0.05$).

Table 5: Mean SLEDAI-2K scores by blood abnormality status (N = 100)

Blood Abnormality	Score if Present (Mean ± SD)	Score if Absent (Mean ± SD)
Anemia	17.13 ± 13.80	6.43 ± 4.90
Leukopenia	20.16 ± 14.82	10.73 ± 10.00
Thrombocytopenia	22.94 ± 15.03	10.73 ± 11.00
Lymphopenia	15.55 ± 13.70	13.51 ± 12.00

Correlation Analysis

Table 6: Pearson correlation between laboratory values and SLEDAI-2K scores (N = 100)

Laboratory Parameter	Pearson r	p-value
Hemoglobin	−0.55	0.01
Platelet Count	−0.35	0.01
ESR	+0.42	< 0.01
APTT	+0.21	0.04

Table 6 presents the Pearson correlation coefficients between key laboratory parameters and SLEDAI-2K disease activity scores. Hemoglobin showed the strongest inverse correlation ($r = −0.55$, $p = 0.01$), confirming that lower hemoglobin directly corresponds to higher disease activity. Platelet count demonstrated a similar negative relationship ($r = −0.35$, $p = 0.01$), reflecting the association between thrombocytopenia and worsening lupus burden. ESR exhibited a moderate positive correlation ($r = +0.42$, $p < 0.01$), underscoring its value as a real-time marker of systemic inflammation. APTT showed a mild but statistically significant positive correlation ($r = +0.21$, $p = 0.04$), suggesting that coagulation prolongation — likely mediated by antiphospholipid antibodies — tracks with increasing immunological activity. The total white cell count did not show a significant linear correlation with SLEDAI-2K, which is attributable to the opposing influences of steroid-induced neutrophilia and autoimmune-mediated lymphopenia within the same leukocyte pool, masking a true directional relationship.

DISCUSSION

This investigation examined 100 SLE patients at a tertiary care setting, documenting their clinical and hematological profiles relative to disease activity. Our patient cohort had a mean age of 35.38 ± 9.67 years, consistent with reports from other South Asian and global populations including Alarfaj et al. (34.3 ± 11.9 years) and Gulati et al. (39.85 ± 12.83

years).[9,10] This age profile reflects the well-recognized predilection of SLE for women of reproductive age. [8,9] The 97% female predominance aligns with prior data[10,11] and is attributed to the immunomodulatory influence of estrogen, which augments autoreactive immune responses.[9]

Articular symptoms predominated, with arthralgia and arthritis in 73% and 53% of patients respectively,consistent with prior literature.[11,12] Malar rash was the leading cutaneous feature (51%). Renal disease affected 47%, comparable to established benchmarks,[11] and neurological involvement occurred in 24%. Variation in clinical presentation across populations is expected, given differences in disease chronicity, treatment exposure, and genetic factors.

Anemia was the dominant hematological finding at 73%, matching Gulati et al. (72%) and exceeding Alarfaj et al. (63%).[10,11] Multiple overlapping mechanisms converge to produce this burden: inflammatory suppression of erythropoiesis, autoantibody-directed hemolysis, nutritional iron depletion, and renal EPO deficiency.[13] AOCD was the leading subtype, reflecting the central role of inflammatory cytokines in disrupting iron cycling. The concurrent presence of IDA and AIHA highlights the etiological diversity that must be considered when evaluating anemia in SLE.

Leukopenia occurred in 44%, exceeding prior reports [10], possibly reflecting more aggressive immune-mediated marrow suppression. Lymphopenia was identified in 67%, higher than earlier data, [11] stemming from heightened lymphocyte apoptosis, redistribution, and autoantibody-mediated cell destruction. Despite its frequency, lymphopenia did not predict SLEDAI-2K scores, suggesting it represents a stable background immunological phenotype rather than a dynamic marker of acute flares.

Thrombocytopenia was present in 34%, surpassing comparable studies,[9,10] reflecting immune-mediated platelet destruction associated with bleeding risk and worse clinical outcomes. Pancytopenia in 22% implies diffuse hematopoietic failure from combined peripheral destruction and marrow suppression.[14] Evans syndrome in 3% represents a rare extreme of immune dysregulation.[15] Biochemically, reduced complement levels indicated active immune complex formation and consumption. Prolonged APTT in 19% may reflect antiphospholipid antibody activity, connecting autoimmune dysregulation to thrombotic risk.[17]

The robust associations between anemia, leukopenia, thrombocytopenia and higher SLEDAI-2K scores align with prior work demonstrating that cytopenias track with disease burden. [8,18,19] Hemoglobin ($r = -0.55$) and platelet count ($r = -0.35$) inversely track disease activity, while ESR ($r = +0.42$) and APTT ($r = +0.21$) move in the same direction. Worsening blood counts and rising inflammatory indices are therefore reliable signals of increasing disease activity. The failure of total white cell count to correlate with SLEDAI-2K reflects the compositional heterogeneity of the leukocyte pool, where steroid-induced neutrophilia may offset lymphopenia.

CONCLUSION

SLE exhibits a marked predilection for women during the third and fourth decades of life, producing a wide variety of clinical and hematological abnormalities. Key indices — particularly hemoglobin, platelet count, and ESR — show clinically meaningful associations with SLEDAI-2K scores and serve as accessible, cost-efficient surrogates for disease activity monitoring. Anemia, leukopenia, and thrombocytopenia are most closely tied to active disease, while lymphopenia, despite being pervasive, lacks independent predictive value for disease severity. Routine blood evaluation should be incorporated into the clinical management of SLE patients for early detection of flares, risk categorization, and outcome prediction.

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