International Journal of Research in Medical Sciences
Shivam et al. Int J Res Med Sci. 2025 Aug;13(8):3397-3403
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20252413

Original Research Article

Clinical severity and patterns of organ involvement in acute systemic lupus erythematosus flares and predictors of disease severity: a retrospective study

Shivam[1]*, Gyanamitra Panigrahy[1], Piyali Sengupta[1], Shashi Singh[2], Challagulla M. Babu[1], Dipleshdeep Goyal[1], Miriyala S. Srinivas[1], Lalatendu Mohanty[1]

[1]Department of General Medicine, Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar, Odisha, India
[2]Department of Emergency Medicine, All India Institute of Medical Sciences, Patna, Bihar, India

Received: 04 July 2025
Accepted: 24 July 2025

***Correspondence:**
Dr. Shivam,
E-mail: shivamsrivastava1111@gmail.com

ABSTRACT

Background: Acute, severe flares of systemic lupus erythematosus (SLE) are associated with significant morbidity and mortality, yet robust, accessible predictors for poor outcomes are not fully established. We aimed to identify the clinical and laboratory characteristics associated with in-hospital mortality in patients with severe SLE flares.
Methods: We conducted a retrospective, cross-sectional study of 30 adult patients admitted for a severe SLE flare (SLE disease activity index 2000 [SLEDAI-2K] score >6) to a tertiary care center in Bhubaneswar, India, between January 2024 and January 2025. We extracted demographic, clinical, and comprehensive laboratory data. Pearson's correlation was used to assess the relationship between SLEDAI-2K and laboratory parameters. Baseline characteristics were compared between survivors and non-survivors to identify predictors of the primary outcome, in-hospital mortality.
Results: The cohort was predominantly female (80.0%) with a median age of 28 years and high disease activity at admission (median SLEDAI-2K, 17.5). The overall in-hospital mortality rate was 13.3% (4 of 30). Disease activity strongly correlated with red cell distribution width (r=0.892), anti-dsDNA levels (r=0.760), complement C3 (r=–0.846), and mean platelet volume (r=–0.820; all p<0.001). Compared to survivors, non-survivors had significantly higher median SLEDAI-2K scores (29.0 versus 16.0, p<0.001) and a higher frequency of neurological involvement (75.0% versus 15.4%, p=0.025). Severe leukopenia (median WBC 1.8 versus 7.2×10^3/µl, p<0.001) and thrombocytopenia (median platelets 42.5 versus 172.0×10^3/µl, p=0.005) were also significantly associated with mortality.
Conclusions: In patients hospitalized for severe SLE flares, high baseline disease activity, neurological involvement, and profound cytopenias are key predictors of in-hospital mortality. Readily available hematological indices, particularly RDW and MPV, correlate strongly with disease activity and may serve as useful adjuncts for risk stratification.

Keywords: SLE, Disease activity, prognosis, SLEDAI, Mortality, Neuropsychiatric lupus, Thrombocytopenia, Red cell distribution width, Mean platelet volume

INTRODUCTION

Systemic lupus erythematosus (SLE) is a quintessential chronic autoimmune disorder, defined by its capacity to affect virtually any organ system and its highly variable clinical course.[1] The disease is characterized by periods of flares and remissions, driven by complex interactions between genetic predispositions, environmental triggers, and hormonal factors that result in a loss of immunologic self-tolerance.[2] While many patients experience a relapsing-remitting course, a significant subset suffers from acute, severe flares that constitute medical emergencies and are associated with substantial morbidity and mortality.[3] These life-threatening presentations,

particularly those involving critical organs like the kidneys, heart, lungs, and central nervous system, pose considerable diagnostic and therapeutic challenges for clinicians.[4]

The heterogeneity of severe SLE flares necessitates a multifaceted approach to understanding their clinical patterns. Such episodes can escalate rapidly, requiring immediate and aggressive immunosuppressive strategies to prevent irreversible organ damage and poor outcomes.[5] While major organ involvement, such as lupus nephritis or neuropsychiatric SLE, is a well-established driver of poor prognosis, a complete understanding of how specific clinical phenotypes and laboratory derangements synergize to influence outcomes remains incomplete.[6] Current literature indicates that the pattern of organ involvement and the extent of disease activity during these acute phases are critical determinants of both short-term and long-term patient outcomes.[7]

Furthermore, a diverse array of laboratory abnormalities is central to the diagnosis and monitoring of SLE. Classic serological markers, including anti-dsDNA antibodies and depressed complement levels, are hallmarks of active disease, reflecting underlying immune complex-mediated pathology.[8] Beyond these specific markers, routine hematological parameters are often disturbed during flares. Cytopenias such as leukopenia and thrombocytopenia are included in classification criteria and are known to correlate with disease activity.[9]

More recently, accessible and inexpensive indices like red cell distribution width (RDW) and mean platelet volume (MPV) have emerged as potential indicators of systemic inflammation in various autoimmune conditions, including SLE, yet their role as prognostic tools in severe flares is not fully established.[10,11]

Given the critical need for improved risk stratification in severe SLE, this study was designed to bridge gaps in the current understanding. By systematically analyzing the clinical presentations, patterns of organ involvement, and a comprehensive panel of laboratory markers in a cohort of patients admitted with acute, severe flares, we aimed to identify robust predictors of poor in-hospital outcomes. This research seeks to contribute to the development of more refined management strategies that could potentially improve the survival and quality of life for patients navigating the most dangerous manifestations of SLE.

Aim

The aim of the study was to conduct a comprehensive analysis of acute, life-threatening episodes of systemic lupus erythematosus (SLE) by characterizing the clinical presentations, patterns of organ involvement, and laboratory abnormalities, in order to identify key prognostic factors and enhance the understanding of the pathophysiology of severe disease flares.

Objectives

Primary objectives of the study were to assess the clinical presentations, determine the patterns of organ involvement, and measure disease severity and its correlation with clinical outcomes in patients experiencing acute, life-threatening episodes of SLE.

Secondary objectives were to study the changes in hematological parameters, such as white blood cells, red blood cells, platelets, and complement levels, and their association with disease severity; determine the factors that can predict the prognosis of patients, based on the disease outcomes at the end of hospital stay; and explore the relationships between clinical presentations, laboratory findings, and organ involvement to better understand the pathophysiology of severe SLE flares.

METHODS

Study design and participants

This retrospective cross-sectional study was conducted using data from the Medical Records Department (MRD) of the Kalinga Institute of Medical Sciences (KIMS), Bhubaneswar, India. The study included patient records from admissions that occurred between January 2024 and January 2025.

Patient record selection was done on the basis of specific inclusion and exclusion criteria. The inclusion criteria required adult patients aged 18 years or older with a confirmed diagnosis of SLE, according to the American College of Rheumatology (ACR) or Systemic Lupus International Collaborating Clinics (SLICC) criteria, who were admitted to KIMS for an acute, severe flare-up. An acute, severe flare was defined by a SLE disease activity index 2000 (SLEDAI-2K) score greater than 6, indicating high disease activity.

The exclusion criteria were designed to ensure data quality and comprised patients under 18 years of age, those with incomplete electronic health records detailing the acute flare and its outcomes, admissions for conditions not primarily related to an SLE flare, or duplicate records for the same flare episode.

Sample size

The required sample size was calculated based on prior research reporting differences in SLEDAI-2K scores during severe disease flares. A study by Yazici et al reported mean SLEDAI-2K scores that suggest a large effect size (Cohen's $d \approx 1.0$) when comparing patients with major organ involvement to those without.[17] A minimum of 26 participants was needed to provide 80% power at $\alpha = 0.05$ using a two-sample t-test. Target record selection was set at 38 to allow for a potential data insufficiency or record exclusion rate of approximately 25%.

The final sample size comprised 30 patient records. This group was established from an initial pool of 38 records from the MRD that were assessed for eligibility. Eight records were excluded for the following reasons: incomplete laboratory data required for analysis (n=4), missing definitive outcome details at discharge (n=3), or the admission being a duplicate entry for a previously recorded flare (n=1) as shown in Figure 1.

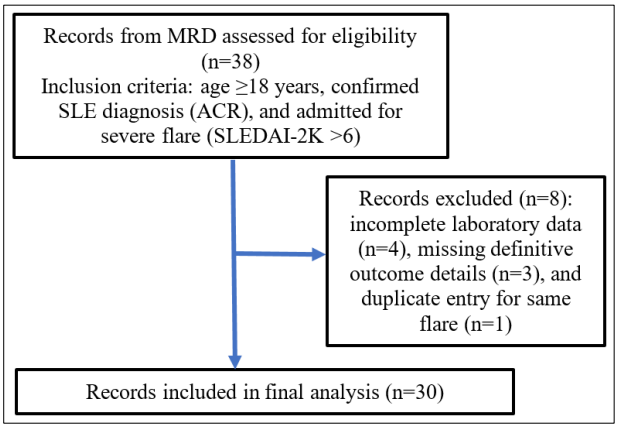

Figure 1: Flow chart for patient selection.

Study procedure

For each selected record, demographic data (age), clinical history (duration of SLE), and specifics of the acute presentation were extracted. Clinical data extraction focused on the frequency and type of symptoms, including fever, rash, arthritis, and severe manifestations such as seizures or psychosis.

Organ involvement was systematically documented, with specific criteria for renal (proteinuria >0.5 g/24 hours, hematuria, or urinary casts), neurological (seizures, psychosis), cardiac (pericarditis), and pulmonary (pleuritis) systems based on the SLEDAI-2K definitions.

Disease severity was quantified using the SLE disease activity index 2000 (SLEDAI-2K), with scores calculated from data corresponding to the time of admission. Hematological profiles, including white blood cell counts, platelet counts, and complement levels (C3, C4), were extracted from the electronic laboratory reports. The primary outcome measures were the clinical symptom profile, the frequency of specific organ involvement, and the mortality rate during the acute hospital stay. Prognostic factors were identified based on the patient's clinical and laboratory data relative to their outcome at discharge.

Statistical analysis

Analyses were conducted using IBM statistical package for the social sciences (SPSS) statistics v20.0. Continuous variables, such as age and SLEDAI-2K scores, were tested for normality with the Shapiro–Wilk test and are reported as mean ± SD or median (interquartile range), as appropriate. Categorical variables, including the presence of specific clinical signs and organ involvement, are reported as count (percentage). Group differences based on clinical outcomes (e.g., survival versus non-survival) were compared using independent-samples t-tests or Mann-Whitney U tests for continuous data, and chi-square or Fisher's exact tests for categorical data. A multivariable logistic regression model was used to assess predictors of in-hospital mortality. Correlational analyses were performed using Pearson or Spearman coefficients to explore relationships between clinical and laboratory data. Statistical significance was set at a two-sided $p<0.05$.

RESULTS

Baseline characteristics of the study cohort

The final study cohort comprised 30 patients meeting the inclusion criteria. The baseline demographic characteristics are summarized in Table 1. The median age of the patients was 28 years (IQR: 22.0–35.5), with a majority being female (24, 80.0%). The median length of hospital stay was 11 days (IQR: 10.0–18.5).

Table 1: Baseline demographic, clinical, and laboratory characteristics (n=30).

Characteristics	Value
Age (years), median [IQR]	28 [22.0–35.5]
Gender, n (%)	
Female	24 (80.0%)
Male	6 (20.0%)
Length of stay (days), median [IQR]	11 [10.0–18.5]

Clinical presentations at admission

The frequency of presenting clinical symptoms is detailed in Figure 2. Joint pain was the most frequently observed symptom, present in 21 (70%) patients. Fever and malar rash were also common, each occurring in 20 (66.7%) patients. Less frequent symptoms included shortness of breath (8 patients, 26.7%), headache (6 patients, 20%), and lower-limb swelling (3 patients, 10%).

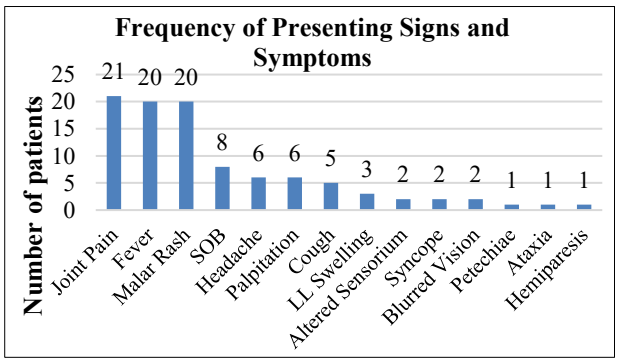

Figure 2: Bar chart showing frequency of presenting signs and symptoms.

Patterns of organ involvement

The pattern of organ system involvement is shown in Table 2 and Figure 3. Renal involvement was the most common major organ manifestation, identified in 15 (50%) patients. Pulmonary complications were present in 10 (33.3%) patients, with pleural effusion being the primary finding. Neurological and cardiac involvement were each documented in 7 (23.3%) cases.

Table 2: Pattern of organ involvement in severe SLE patients (n=30).

Organ system - count (%)	Key involvement	Specific count (%)
Renal - 15 (50)	Lupus nephritis	7 (23.3)
	Proteinuria only	8 (25.8)
Neurological - 7 (23.3)	Stroke/CVA	3 (10.0)
	Headache	3 (10.0)
	Psychosis	1 (3.3)
Cardiac - 7 (23.3)	Pericardial effusion	5 (16.6)
	Pericarditis	2 (6.6)
Pulmonary - 10 (33.3)	Pleural effusion	8 (26.6)
	DAH	2 (6.6)

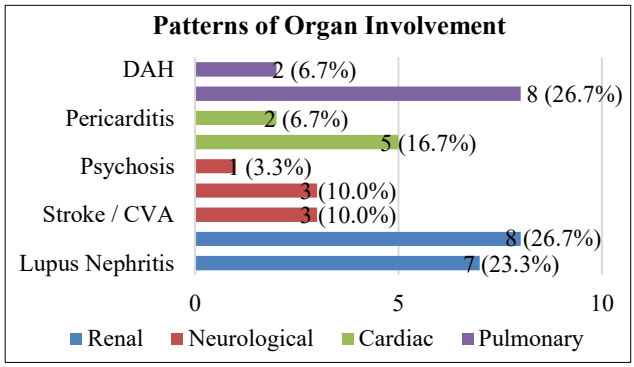

Figure 3: Clustered bar chart showing patterns of organ involvement in severe SLE patients.

Disease activity and laboratory profile at admission

Disease activity at admission was high, with a median SLEDAI-2K score for the cohort of 17.5 (IQR: 13.0–22.0). The detailed laboratory profile is presented in Table 3. The findings indicate significant hematological and serological disturbances, including a tendency towards leukopenia in some patients (median WBC: $7.10\times10^3/\mu l$), elevated inflammatory markers (median ESR: 55.5 mm/hour), and broadly suppressed complement levels (median C3: 58.0 mg/dl).

Correlation of disease activity with laboratory parameters

A bivariate Pearson's correlation analysis was performed to assess the relationship between the SLEDAI-2K score and a comprehensive panel of laboratory parameters. The results, including both statistically significant and non-significant correlations, are detailed in Table 4.

Table 3: Disease activity and laboratory profile at admission (n=30).

Parameter	Value
Disease activity, median [IQR]	
SLEDAI-2K score	17.5 [13.0–22.0]
Hematology, median [IQR]	
WBC ($\times10^3/\mu l$)	7.10 [2.33–9.20]
RBC ($\times10^6/\mu l$)	3.35 [3.20–4.10]
Hemoglobin (g/dl)	10.1 [9.1–10.6]
Platelet count ($\times10^3/\mu l$)	167.5 [54.0–188.0]
RDW (%)	22.5 [18.2–29.8]
MPV (fl)	7.00 [4.25–8.00]
Retic Hb (pg)	37.0 [33.0–42.5]
Biochemistry and serology, median [IQR]	
Creatinine (mg/dl)	1.10 [0.90–1.93]
Complement C3 (mg/dl)	58.0 [35.0–71.5]
Complement C4 (mg/dl)	9.00 [8.00–9.00]
Anti-dsDNA level (IU/ml)	528.0 [405.5–748.8]
ESR (mm/hour)	55.5 [42.0–76.2]
CRP (mg/l)	9.00 [6.50–12.00]
Categorical tests, n (%)	
ANA titre	
Grade 3+	15 (50.0)
Grade 4+	8 (26.7)
Grade 2+	6 (20.0)
Grade 1+	1 (3.3)
DCT (direct Coombs test)	
Negative	22 (73.3)
Positive	8 (26.7)

Strong, significant positive correlations were observed between the SLEDAI-2K score and RDW ($r=0.892$, $p<0.001$), reticulocyte hemoglobin ($r=0.829$, $p<0.001$), and anti-dsDNA level ($r=0.760$, $p<0.001$). Additionally, moderate to strong positive correlations were found with WBC count ($r=0.652$, $p<0.001$) and ANA titre ($r=0.587$, $p<0.001$).

Strong, significant negative correlations were identified with complement C3 ($r=-0.846$, $p<0.001$) and MPV ($r=-0.820$, $p<0.001$), along with a moderate negative correlation with complement C4 ($r=-0.575$, $p<0.001$).

Other parameters, including RBC count, hemoglobin, platelet count, creatinine, ESR, and CRP, did not show a statistically significant correlation with the SLEDAI-2K score in this cohort.

Clinical outcomes and identification of prognostic factors

During the hospital stay for the acute flare, the overall mortality rate in this cohort was 13.3% (4 of 30 patients).

The remaining 26 patients (86.7%) showed clinical improvement and were discharged.

To identify factors associated with in-hospital mortality, baseline clinical and laboratory parameters were compared between patients who survived and those who did not (Table 5).

The analysis revealed that non-survivors had a significantly higher disease activity at admission, with a median SLEDAI-2K score of 29.0 compared to 16.0 for survivors (p<0.001).

Furthermore, the presence of neurological involvement was significantly associated with mortality (75.0% in non-survivors versus 15.4% in survivors, p=0.025). Non-survivors also had significantly lower median Complement C3 levels (32.5 mg/dl versus 60.5 mg/dl, p=0.005) and platelet counts (42.5 versus 172.0×10^3/µl, p=0.005). Other baseline characteristics did not show a statistically significant difference between the two groups.

Table 4: Bivariate Pearson's correlation between SLEDAI-2K and all laboratory parameters (n=30).

Parameter	Correlation coefficient (r)	P value
White blood cell count	0.652	<0.001
Red blood cell count	-0.076	0.689
Hemoglobin	-0.106	0.576
Platelet count	-0.017	0.931
Red cell distribution width	0.892	<0.001
Mean platelet volume	-0.82	<0.001
Reticulocyte hemoglobin	0.829	<0.001
Creatinine	0.125	0.509
Complement C3	-0.846	<0.001
Complement C4	-0.575	<0.001
Anti-dsDNA level	0.76	<0.001
Erythrocyte sedimentation rate	0.324	0.081
C-reactive protein	0.158	0.405
ANA titre	0.587	<0.001

Table 5: Comparison of baseline factors between survivors and non-survivors.

Variable	Survivors (n=26)	Non-survivors (n=4)	P value
Demographics			
Age (years), median [IQR]	28.0 [22.0–35.0]	32.5 [26.5–39.5]	0.315
Disease activity and organ involvement			
SLEDAI-2K score, median [IQR]	16.0 [12.8–19.0]	29.0 [25.0–40.5]	<0.001
Renal involvement, n (%)	12 (46.2)	3 (75.0)	0.568
Neurological involvement, n (%)	4 (15.4)	3 (75.0)	0.025
Laboratory parameters, median [IQR]			
Complement C3 (mg/dl)	60.5 [44.0–73.0]	32.5 [23.8–38.5]	0.005
Platelet count (×10^3/µl)	172.0 [157.5–190.0]	42.5 [33.8–50.0]	0.005
WBC (×10^3/µl)	7.2 [4.9–9.4]	1.8 [1.0–2.2]	<0.001
Creatinine (mg/dl)	1.1 [0.9–1.8]	2.6 [1.2–4.3]	0.063

DISCUSSION

In this retrospective analysis of 30 patients admitted with severe SLE flares, we identified a significant burden of multi-organ disease, high disease activity, and a notable in-hospital mortality rate of 13.3%. Our principal findings highlight that a high baseline SLEDAI-2K score, the presence of neurological involvement, and profound hematological disturbances—specifically thrombocytopenia and leukopenia—were significantly associated with poor outcomes. Furthermore, disease activity demonstrated a strong correlation not only with established serological markers like low C3 and high anti-dsDNA but also with readily available hematological indices such as elevated RDW and decreased MPV.

The clinical profile of our cohort is largely consistent with other studies of severe SLE. The predominance of young female patients reflects the well-known epidemiology of the disease.[12] The high frequency of renal involvement (50%) aligns with reports that identify lupus nephritis as one of the most common and severe manifestations of SLE, often precipitating hospital admission.[13] The observed mortality rate of 13.3% underscores the life-threatening nature of acute flares. This figure is comparable to mortality rates reported in other international cohorts of hospitalized SLE patients, which range from 9% to 20% depending on the severity of the cohort and the presence of major organ damage.[3]

A key finding of our study is the strong correlation between the SLEDAI-2K score and routine hematological parameters. The robust positive correlation with RDW (r=0.892) and negative correlation with MPV (r=-0.820) are particularly noteworthy. Elevated RDW, a measure of variability in red blood cell size, is increasingly recognized as a non-specific but potent marker of systemic inflammation, thought to be driven by inflammatory cytokines that impair erythropoiesis.[10,14]

Similarly, a lower MPV during active inflammation may reflect the consumption of larger, more reactive platelets at sites of endothelial injury, a central process in SLE vasculopathy.[11,15] These findings suggest that RDW and MPV, which are part of a standard complete blood count, could serve as inexpensive and accessible adjuncts to traditional serological tests for gauging the intensity of a flare.

The identification of prognostic factors for in-hospital mortality provides critical, clinically relevant insights. While an elevated SLEDAI score is an expected predictor of poor outcomes, our analysis specifically implicated neurological involvement as a significant risk factor. Neuropsychiatric SLE is known to carry high morbidity, and our finding that 75% of non-survivors presented with neurological symptoms, compared to just 15% of survivors, reinforces its status as a harbinger of a potentially fatal course.[6,16]

The strong association of mortality with severe thrombocytopenia and leukopenia is also clinically significant. These cytopenias are not merely markers of disease activity but are direct contributors to risk, predisposing patients to life-threatening hemorrhage and infection, respectively.[9]

The clinical implications of our findings are twofold. First, they emphasize the need for heightened vigilance and aggressive management in patients presenting with high SLEDAI scores and any evidence of neurological dysfunction. Second, they support the integration of a broader panel of laboratory markers, including RDW and MPV, into the routine assessment of flare severity. While this study provides valuable data from an Indian cohort, the findings must be interpreted in the context of its limitations.

Strengths and limitations

The primary strength of this study is its focus on a well-defined and homogenous cohort of patients with confirmed severe, acute SLE flares, which reduces the confounding effects of including patients with mild or moderate disease. The comprehensive data collection, encompassing a wide array of clinical, serological, and detailed hematological parameters, allowed for a robust correlational analysis. Furthermore, by analyzing predictors of a definitive clinical endpoint—in-hospital mortality—the study provides clinically actionable information.

However, several limitations must be acknowledged. The retrospective design is inherently susceptible to information bias and dependent on the accuracy and completeness of existing medical records. The small sample size of 30 patients limits the statistical power of our analyses, particularly for identifying prognostic factors, and precludes a more complex multivariable regression model. As a single-center study, the findings may have limited generalizability to other populations or healthcare settings with different patient demographics or management protocols. Future large-scale, prospective multicenter studies are warranted to validate these findings.

CONCLUSION

In patients hospitalized for severe SLE flares, high baseline disease activity, the presence of neurological involvement, and profound cytopenias are strongly associated with in-hospital mortality. This study advances current knowledge by demonstrating that readily available hematological indices, such as RDW and MPV, correlate robustly with disease severity alongside traditional serological markers in an Indian cohort. These findings underscore that a comprehensive assessment integrating clinical presentation—especially neurological status—with a broad panel of laboratory markers is crucial for accurately stratifying risk and may represent an opportunity to improve outcomes in this vulnerable patient population.

ACKNOWLEDGEMENTS

The authors would like to express their sincere gratitude to Dr. Shubhransu Patro, Professor and Head of the Department of General Medicine, KIMS, for his continuous support. They also extend heartfelt thanks to Dr. Purusottam Misra, Dr. Rabinarayan Rout and Dr. Sarada Priyadarshini Suna from Department of General Medicine for their valuable insights, clinical expertise, and encouragement throughout the course of this work.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Tsokos GC. Systemic lupus erythematosus. N Engl J Med. 2011;365(22):2110-21.
2. Hochberg MC. Updating the American College of Rheumatology revised criteria for the classification of systemic lupus erythematosus [letter]. Arthritis Rheum. 1997;40(9):1725.
3. Tektonidou MG, Dasgupta A, Ward MM. Risk of hospitalization for serious infections in patients with systemic lupus erythematosus: a population-based study. Arthritis Care Res (Hoboken). 2016;68(7):930-6.
4. Bertsias GK, Tektonidou M, Amoura Z, Flöel A, Boumpas DT, Jayne D, et al. Joint European League Against Rheumatism and European Renal Association-European Dialysis and Transplant Association (EULAR/ERA-EDTA) recommendations for the management of adult and paediatric lupus nephritis. Ann Rheum Dis. 2012;71(11):1771-82.

5. Petri M. Epidemiology of systemic lupus erythematosus. Best Pract Res Clin Rheumatol. 2002;16(5):847-58.
6. Ainiala H, Hietaharju A, Loukkola J, Peltola J. The diverse clinical manifestations of neuro-psychiatric systemic lupus erythematosus. J Neurol. 2007;254(9):1227-33.
7. Ruiz-Irastorza G, Ramos-Casals M, Brito-Zeron P, Khamashta MA. Clinical efficacy and side effects of antimalarials in systemic lupus erythematosus: a systematic review. Autoimmun Rev. 2010;9(4):271-8.
8. Frieri M, Stampfl H. Systemic lupus erythematosus and the role of autoantibodies in its pathogenesis. Curr Allergy Asthma Rep. 2003;3(5):395-402.
9. Fayyaz A, Igoe A, Kurien BT, Danda D, James JA, Stafford HA, et al. Haematological manifestations of lupus. Lupus Sci Med. 2015;2(1):e000078.
10. Scharte M, Fink MP. Red blood cell physiology in critical illness. Crit Care Med. 2003;31(12 Suppl):S651-7.
11. Gasparyan AY, Ayvazyan L, Mikhailidis DP, Kitas GD. Mean platelet volume: a link between thrombosis and inflammation? Curr Pharm Des. 2011;17(1):47-58.
12. Rees F, Doherty M, Grainge MJ, Lanyon P, Zhang W. The worldwide incidence and prevalence of systemic lupus erythematosus: a systematic review of epidemiological studies. Rheumatology (Oxford). 2017;56(11):1945-61.
13. Maroz N, Mossey PA. The evidence for the use of corticosteroids in the management of lupus nephritis. J Am Soc Nephrol. 2006;17(4 Suppl 2):S123-7.
14. Lippi G, Targher G, Montagnana M, Salvagno GL, Zoppini G, Guidi GC. Relation between red blood cell distribution width and inflammatory biomarkers in a large cohort of unselected outpatients. Arch Pathol Lab Med. 2009;133(4):628-32.
15. Vizioli J, Muscari S, Muscari A. The relationship of mean platelet volume with the risk and prognosis of cardiovascular diseases. Int J Clin Pract. 2009;63(10):1509-15.
16. Hanly JG, Urowitz MB, Su L, Bae SC, Gordon C, Wallace DJ, et al. Prospective analysis of neuropsychiatric events in a national cohort of patients with systemic lupus erythematosus. Ann Rheum Dis. 2010;69(3):529-35.
17. Yazici Y, Biyikli M, Erkan D, Akman-Demir G, Ozbek S, Yurdakul S, et al. The systemic lupus erythematosus disease activity index 2000 is valid and practical for routine use in busy clinical settings. Ann Rheum Dis. 2003;62(5):483-4.

Cite this article as: Shivam, Panigrahy G, Sengupta P, Singh S, Babu CM, Goyal D, et al. Clinical severity and patterns of organ involvement in acute systemic lupus erythematosus flares and predictors of disease severity – a retrospective study. Int J Res Med Sci 2025;13:3397-403.