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Hospital Admissions and Disease Outcomes in Systemic Lupus Erythematosus: A Retrospective Study from a Tertiary Rheumatology Care Unit

Eiman Soliman
Alexandria University
Rania Menesy
Alexandria University
Shirene Abdelkhalek
lilisamy@yahoo.com

Alexandria University

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Abstract

Objective:

To evaluate the clinical and laboratory characteristics associated with disease outcomes and mortality in systemic lupus erythematosus (SLE) patients admitted to a specialized rheumatology care unit.

Methods:

A retrospective cohort study was conducted on 89 patients with SLE admitted between May 2022 and May 2023. Demographic data, comorbidities, clinical presentations, laboratory parameters, and disease activity (SLEDAI) and damage (SLICC/ACR) indices were analyzed. Causes of admission, types of infections, therapeutic interventions, and in-hospital outcomes were also assessed.

Results:

The majority of patients were female (97.8%) with a mean age of 34.96 ± 13.78 years. Infection was the leading cause of admission (51.7%), with bacterial infections accounting for 84% of all infections. Hypertension was the most common comorbidity (37.1%). Disease activity was high, with 51.7% experiencing a severe flare (SLEDAI > 12). Irreversible damage was noted in 70.8% of patients (SLICC/ACR score ≥ 1). The in-hospital mortality rate was 6.7%, with cardiovascular complications responsible for 50% of deaths. Glucocorticoids were administered to nearly all patients (98.9%), while immunosuppressants such as mycophenolate mofetil and cyclophosphamide were used selectively.

Conclusion:

SLE hospitalizations are frequently driven by infectious complications, with bacterial sepsis being predominant. Cardiovascular complications remain the primary cause of death in this population. Improved infection monitoring and proactive cardiovascular risk management are essential to reduce morbidity and mortality in SLE patients requiring hospitalization.

INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, multisystem autoimmune disorder characterised by a wide range of clinical manifestations and fluctuating disease activity. It arises from complex interactions among genetic, epigenetic, and environmental factors that disrupt immune tolerance, resulting in the activation of autoreactive B and T lymphocytes and the formation of pathogenic immune complexes [1].

The clinical course of SLE is heterogeneous, varying from mild cutaneous and musculoskeletal symptoms to life-threatening involvement of major organs such as the kidneys, lungs, and central nervous system [2]. Disease flares, interspersed with periods of remission, can lead to progressive organ damage and disability if not optimally managed [3].

Mucocutaneous involvement is observed in approximately 90% of patients during the disease course [4], followed by joint complaints in up to 50% of cases [5]. Renal manifestations, particularly lupus nephritis (LN), are seen in 15–20% of patients at diagnosis and in up to 50% over time [6]. Cardiac involvement may occur later and includes pericarditis, myocarditis, and endocarditis, often accompanying other systemic features [7]. Pulmonary complications such as pleuritis and pneumonitis are also common, with pleuritis affecting up to 50% of patients [8].

Haematological abnormalities—such as anaemia, thrombocytopenia, and leukopenia—are frequently seen in the early disease stages [9], and may be associated with secondary antiphospholipid syndrome (APS), which predisposes patients to thrombosis [10].

These diverse manifestations often necessitate hospitalization, particularly during disease flares or due to complications, such as infections, or treatment-related complications. Infections are particularly prevalent due to both intrinsic immune dysfunction and immunosuppressive therapies [11, 12]. Admissions to intensive care or high-dependency rheumatology units occur in up to 37% of cases and are associated with variable mortality depending on disease severity, comorbidities, and access to specialised care [13, 14].

While previous studies have examined hospital admissions and outcomes in SLE patients, regional variations in disease patterns, health care access and treatment protocols often necessitate localized research. Studies specifically from tertiary rheumatology care units in Egypt, focusing on the interplay between infectious complications, cardiovascular events and patient outcomes within a recent timeframe, remain limited. This study aims to address this gap by comprehensively analyzing the clinical characteristics, comorbidities and outcomes of SLE patients admitted to specialized units at our University hospital, providing valuable insights relevant to our local context and potentially informing targeted intervention.

METHODS

Study Design and Setting

A retrospective observational cohort study was conducted at the Rheumatology and Clinical Immunology Unit, Department of Internal Medicine, our Main University Hospital. It is a tertiary referral center equipped with 40 inpatient beds, dedicated outpatient clinics, access to advanced imaging and laboratory services. It is staffed by specialized rheumatologists, fellows and nurses, providing comprehensive care for complex autoimmune diseases including SLE. Patients admitted to this unit typically present with severe disease manifestations, active flares requiring intensive management, or complex complications not manageable in general medical wards. The study period extended from May 2022 to May 2023.

Patient Selection

Eligible participants included adult patients (aged ≥ 18 years) with a confirmed diagnosis of systemic lupus erythematosus (SLE) according to the Systemic Lupus International Collaborating Clinics (SLICC) 2012 classification criteria [15]. Patients were included if they were admitted to the SLE specialised care unit during the study period. Exclusion criteria included incomplete clinical data or overlapping autoimmune conditions.

Ethical considerations

The study protocol was reviewed and approved by the Ethics Committee of the Faculty of Medicine, Alexandria University (Approval No. 0107738). Given the retrospective nature of the study, written informed consent was obtained from all participants prior to their inclusion, as per institutional guidelines. All patients' data were anonymized and handled with strict confidentiality to protect participant privacy, in accordance with the principles of declaration of Helsinki.

Data Collection

Data were extracted from patient records using a structured data collection form built on RED Cap (Research Electronic Data Capture), a secure, web-based software platform designed for research data management [16]. Demographic information (age, sex, marital status, smoking history, family history of autoimmune disease, and obstetric history) and clinical data (comorbidities, previous disease manifestations, and current clinical presentation) were recorded. Information on therapeutic interventions administered during hospitalization was extracted including types of glucocorticoid (intravenous methylprednisolone pulse therapy, oral prednisone), immunosuppressants (e.g. mycophenolate mofetil, cyclophosphamide, Azathioprin), antimalarials (Hydroxchlorochine) and other treatment (biologics, intravenous immunoglobulin). Dosages and duration of key medications, as well as any changes in treatment regimen, were recorded.

Disease Activity and Damage Assessment

The Systemic Lupus Erythematosus Disease Activity Index (SLEDAI-2K) was used to assess disease activity at admission [17]. Irreversible organ damage was assessed using the SLICC/ACR Damage Index, which accounts for cumulative damage from disease or treatment [18].

Laboratory Investigations

Routine laboratory tests included:

- Complete blood count (CBC)
- Renal function: serum urea, serum creatinine, urine protein/creatinine ratio
- Liver function: AST, ALT

- Coagulation profile: international normalized ratio (INR)
- Thyroid-stimulating hormone (TSH)
- Inflammatory markers: erythrocyte sedimentation rate (ESR), C-reactive protein (CRP)
- Microbiological analysis in case of suspected infection.

Immunological tests included:

- Antinuclear antibody (ANA) by immunofluorescence
- Anti-double stranded DNA (anti-dsDNA) by ELISA
- Complement levels (C3, C4)
- Antiphospholipid antibody panel: lupus anticoagulant, anticardiolipin IgG/IgM, and beta-2 glycoprotein I antibodies IgG/IgM

Outcomes

The primary outcomes were cause of admission, length of hospital stay, disease flare severity, organ damage score, ICU referral, and in-hospital mortality. Causes of admission were grouped as infection-related, organ-specific manifestations, or haematological complications.

Statistical Analysis

Data were analysed using SPSS software, version 20.0 (IBM Corp., Armonk, NY). Categorical variables were expressed as frequencies and percentages. Continuous variables were reported as means ± standard deviation (SD) or medians with interquartile range (IQR), depending on distribution. Associations between variables and outcomes were explored using appropriate statistical tests; significance was set at $p < 0.05$.

RESULTS

Demographic and Clinical Characteristics

A total of 89 patients with SLE were included in the study, of whom 87 (97.8%) were female and 2 (2.2%) were male. The mean age at admission was 34.96 ± 13.78 years (range: 10–69 years). The majority of patients (83.1%) were married, and only 2.2% were current smokers. A family history of SLE was reported in 7.9% of cases, and 11.2% had a family history of other rheumatologic diseases.

Obstetric history was available for 74 female patients. The mean gravidity was 2.85 ± 1.64, and the mean parity was 2.23 ± 1.26. A history of abortion was reported in 33.8% of patients.

Table 1
Demographic and Clinical Characteristics (n = 89)

Variable	Value
Female, n (%)	87 (97.8%)
Mean age, years (± SD)	34.96 ± 13.78
Married, n (%)	74 (83.1%)
Smokers, n (%)	2 (2.2%)
Positive family history for SLE, n (%)	7 (7.9%)
Positive family history for other rheumatologic disease, n (%)	10 (11.2%)
Gravidity, mean ± SD	2.85 ± 1.64
Parity, mean ± SD	2.23 ± 1.26
History of abortion, n (%)	25 (33.8%)

Initial patient presentation

By reviewing the previous disease manifestations in admitted patients, 70 patients (78.7%) had cutaneous involvement, 57 patients (64.0%) were complaining from arthritis, anemia was present in 48 patients (53.9%), 41 patients (46.1%) had nephritis, thrombocytopenia was present in 24 patients (27.0%),23 patients (25.8%) were complaining from serositis,13 patients (14.6%) had constitutional manifestations, leukopenia was present in 12 patients (13.5%) and 10 patients (11.2%) had neurological manifestations.

Comorbidities

Comorbidities were observed in 45 patients (50.6%). Hypertension was the most frequent, present in 33 patients (37.1%), followed by diabetes mellitus in 10.1% and hypothyroidism in 9.0%. Malignancy and liver cirrhosis were each reported in 2.2% of patients.

Causes of Admission

Infection was the leading cause of admission, seen in 46 patients (51.7%). Renal involvement was observed in 43.8%, and haematological abnormalities such as anaemia, leukopenia, and thrombocytopenia were common.

Table 2
Primary Causes of Admission to the SLE Care Unit

Cause of Admission	Frequency (%)
Infection and Sepsis	51.7
Renal involvement	43.8
Anaemia	23.6
Thrombocytopenia	28.0
Leukopenia	22.4
Arthritis	18.0
Serositis	11.2
Cutaneous manifestations	10.1
Neurological	4.5
Constitutional symptoms (e.g. fever)	4.5
Autoimmune hepatitis	2.2
Combined causes	65.16

Infectious complications were found as a primary cause of admission. Of the 50 patients diagnosed with infection, 92% acquired the infection prior to admission. Based on microbiological analysis, 84% of all infections were bacterial in origin, 14% was viral and 4% of patients had fungal infection. Lower respiratory tract infections accounted for 46% of infection sources, followed by urinary tract infections (40%) and skin infections (14%).

Hematological abnormalities came next in frequency as a leading cause of admission. We found that 23.6% of patients suffer from anemia of different grades due to different reasons, the least percentage was 4% of the patients due to acute hemolytic anemias, 12% are anemia of chronic illness, and around 7% of anemias are due to element deficiency. Around 15% of patients needed packed red blood cell transfusion. 28% of patients had thrombocytopenia, in half of these patients, thrombocytopenia was severe to be the cause of admission, and the rest of them was combined with other causes of admission. 20% was due to activity of the disease and 8% was due to medications or presence of infection that was improved with treatment of the underlying reason.

Renal involvement was the third cause of admission. We found that 43.8% of patients had abnormal renal functions that needed admission in the special units to determine the cause of these abnormal findings whether to have laboratory analysis, renal biopsy or optimizing treatment or dialysis in some cases. Around 59.6% of the admitted patients with abnormal renal functions had lupus nephritis that

necessitated rapid intervention according to the associated morbidities. While the rest had abnormalities due to associated infections or drug interactions.

Notably, 65.16% of admissions involved a combination of factors. The most frequent combined causes included infection with renal flare 25% of combined causes. Severe hematological abnormalities along with constitutional symptoms 18%. This highlights the multisystemic nature of SLE and often complex clinical scenarios necessitating hospitalization.

Serological and Routine Laboratory Results of Admitted SLE Patients TABLE 3

ANA (Positive)	Mean ± SD.	Median	No. (%)
			89 (100.0%)
Anti-ds DNA (IU/ml) (Positive)	310.2 ± 251.6	310.2 ± 251.6	73 (82.0%)
Consumed Complement C3 (mg/dl)	71.72 ± 37.42	69.0	60 (67.4%)
Consumed Complement C4 (mg/dl)	12.94 ± 9.90	12.0	41 (46.1%)
ESR (mm/hr.) ≥ 20 mm/hr.	42.94 ± 30.23	34.0	68 (76.4%)
CRP (mg/L) ≥ 3 mg/L	34.87 ± 53.05	14.40	78 (87.6%)
Anemia < 12 (g/dl)	9.29 ± 1.79	8.90	81 (91.0%)
Thrombocytopenia < 100 (10^3/ul)	221.5 ± 154.7	196.0	22 (24.7%)
Leukopenia < 4 (10^3/ul)	5.88 ± 3.68	4.70	33 (37.1%)
Impaired Renal functions			
Urea (mg/dl) > 50	64.61 ± 56.76	43.0	39 (43.8%)
Creatinine (mg/dl) > 1.2	1.61 ± 1.70	0.90	30 (33.7%)
Urine Protein/creatinine ratio **mg Prot./mg Creat. >0.5**	2.08 ± 2.42	1.0	53 (59.6%)
Elevated Liver enzymes			
AST (U/L) > 32	29.33 ± 33.42	23.0	21 (23.6%)
ALT (U/L) > 33	26.93 ± 29.13	20.0	73 (82.0%)
INR > 1.2	1.12 ± 0.39	1.02	16 (18%)
Antiphospholipid antibodies			
Lupus anticoagulant (positive)			21 (23.6%)
Anticardiolipin antibodies IgG and/or IgM (positive)			22 (24.7%)
Beta-2 glycoprotein 1 antibodies IgG and/or IgM (positive)			27 (30.3%)

Disease Activity and Damage Scores

SLEDAI score ranged from 3–39 (mean 13.09 ± 5.53), where 51.7% of patients had severe flare (SLEDAI > 12). **SLICC/ACR damage index** ranged from 1–5 (mean 1.70 ± 0.98), with 70.8% of patients had documented organ damage.

Treatment and Management

Nearly all patients (98.9%) received glucocorticoids during their admission. Hydroxychloroquine was prescribed in 91% of cases. Mycophenolate mofetil (39.3%) and azathioprine (28.1%) were the most frequently used immunosuppressants. Cyclophosphamide was used in 13.5% of patients, and other treatments included cyclosporine, methotrexate, rituximab, and intravenous immunoglobulin (IVIG).
Figure 1: Bar chart showing distribution of treatments.

Duration of Hospital Stay and ICU Referral

The length of stay in the SLE care unit ranged from 2 to 54 days, with a mean of 12.02 ± 10.18 days. Twelve patients (13.5%) required transfer to the general intensive care unit during their admission.

Patient Outcomes

At discharge, 83 patients (93.3%) had improved clinically, while 6 patients (6.7%) died during admission.
Figure 2: Pie chart of patient outcomes (recovery vs. mortality).

Causes of Mortality

Among the six patients who died, cardiovascular complications were the most common cause (3 patients, 50%). Other causes included: Sepsis (16.7%), Intra-alveolar hemorrhage (16.7%), and CNS vasculitis (16.7%)

DISCUSSION

This study analyzed the clinical features, comorbidities, and outcomes of SLE patients admitted to a specialized rheumatology care unit. The findings highlight the high burden of infection-related admissions and the predominance of cardiovascular complications as the leading cause of in-hospital mortality.

The demographic characteristics of the cohort, including the predominance of female patients and a mean age in the third decade of life, are consistent with the established epidemiology of SLE [19]. A family history of SLE or other rheumatological conditions was present in approximately 19%, supporting a genetic predisposition as previously reported [20].

Notably, spontaneous abortion was documented in one-third of female patients. Pregnancy loss in SLE may result from uncontrolled disease activity or the presence of antiphospholipid antibodies, a pattern

observed in earlier studies [21, 22]. Multidisciplinary management remains critical for improving pregnancy outcomes [23].

Comorbidities were identified in over half of the patients, with hypertension being the most prevalent (37.1%). This aligns with findings from other cohorts, where hypertension is recognized as both a manifestation and a complication of SLE and its treatment [24].

Infections were the leading cause of hospital admission (51.7%), with bacterial infections accounting for the majority. This finding is supported by earlier studies indicating that SLE patients are particularly susceptible to bacterial pathogens due to disease-related immune dysfunction and immunosuppressive therapy [11, 25]. In contrast, other reports have cited disease activity as the primary cause of hospitalisation [19], suggesting that admission drivers may vary by region, access to care, or timing of intervention. This discrepancy might be attributed to differences in patients demographics, local healthcare practices, the stage of disease at presentation, or the specific criteria used to define disease activity versus infection- related admissions in different regions.

The extensive use of glucocorticoids (98.9%) is consistent with their central role in acute flare management [26], although their long-term use is associated with organ damage and increased infection risk [3]. Immunosuppressants were also widely used, tailored to individual disease manifestations.

In terms of disease activity, over half of the patients presented with a severe flare (SLEDAI > 12), which is higher than previously reported medians [25, 27]. Furthermore, 70.8% of patients had accrued irreversible organ damage (SLICC/ACR ≥ 1), reflecting the cumulative burden of active disease and treatment over time [18].

Despite the disease severity, the in-hospital mortality rate was relatively low (6.7%), possibly reflecting improved access to specialised care, early intervention, and supportive management. Cardiovascular complications were the primary cause of death (50%), reinforcing their role as a leading contributor to SLE morbidity and mortality [28]. While some studies have identified infection as the predominant cause of death [29], our results underscore the growing importance of cardiovascular risk monitoring in SLE care.

Serological findings supported disease activity: anti-dsDNA positivity was found in 82% of cases, and complement consumption (low C3 and/or C4) was noted in over 70% — both well-established markers of active SLE [30, 31]. Anemia (91%) and proteinuria (59.6%) were also frequent and consistent with multisystem involvement.

Antiphospholipid antibodies were detected in 37.1% of patients, consistent with global reports ranging from 30–40% [32]. These antibodies are associated with thrombotic complications and pregnancy morbidity, necessitating early recognition and appropriate anticoagulation when indicated.

Limitations

This study has several limitations that warrant considerations. Its retrospective, single center- design may limit generalizability of our findings to other populations or healthcare settings. The relatively small sample size (n = 89) might affect the statistical power to detect associations for less common outcomes or specific subgroups. Furthermore, the study focused solely on in- hospital outcomes, thus not capturing long-term morbidity or mortality trends post-discharge. The reliance on medical record data also inherently carries the risk of incomplete documentation, although REDCap was used to standardize data extractions.

Future Directions

To put upon the findings, prospective, multicenter studies with larger cohorts are warranted to validate the identified risk factors and outcomes across diverse SLE populations. Longitudinal studies tracking patients post- discharge would also provide crucial insights into long-term morbidity and mortality. Further research could also focus on developing and evaluating targeted interventions, such as enhanced infection prevention protocols or personalized cardiovascular risk management strategies, within specialized SLE units,

CONCLUSION

This study demonstrates that systemic lupus erythematosus presents with a broad clinical spectrum, often requiring hospitalization for infectious or immunologic complications. Infection — primarily bacterial — was the leading cause of admission, while cardiovascular complications were the primary driver of in-hospital mortality.

The high rates of disease activity, comorbid conditions, and irreversible organ damage underscore the need for early detection, personalised therapy, and integrated management strategies. Rheumatologists should remain vigilant regarding infection risk, carefully balance immunosuppressive therapy, and implement proactive cardiovascular risk reduction to improve survival outcomes in hospitalized SLE patients.

Abbreviations

Abbreviation
Full Term
ANA
Antinuclear Antibody
ALT
Alanine Aminotransferase
APS
Antiphospholipid Syndrome
AST
Aspartate Aminotransferase

CBC
Complete Blood Count
CNS
Central Nervous System
CRP
C-Reactive Protein
C3
Complement Component 3
C4
Complement Component 4
ELISA
Enzyme-Linked Immunosorbent Assay
ESR
Erythrocyte Sedimentation Rate
ICU
Intensive Care Unit
IgG
Immunoglobulin G
IgM
Immunoglobulin M
INR
International Normalized Ratio
IVIG
Intravenous Immunoglobulin
LN
Lupus Nephritis
REDCap
Research Electronic Data Capture
SD
Standard Deviation
SLE
Systemic Lupus Erythematosus
SLEDAI-2K
Systemic Lupus Erythematosus Disease Activity Index 2000
SLICC
Systemic Lupus International Collaborating Clinics
SLICC/ACR DI
Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index
SPSS
Statistical Package for the Social Sciences

TSH
Thyroid-Stimulating Hormone

Declarations

Consent to participate

all authors contributed equally to the study’s conception, design, data collection, analysis, interpretation, and manuscript preparation. All authors read and approved the final version of the manuscript.

Consent for publication

The authors confirm that this manuscript is original, has not been published previously, and is not under cosideration for publication elsewhere. All authors have reviewed and approved the final version of the manuscript and agreed with its submission to the Egyptian Rheumatology and Rehabilition Journal.

Ethics approval and consent to participate

The study was approved by the Ethics Committee of the Faculty of Medicine, Alexandria University (Approval No. 0107738). Written informed consent was obtained from all participants prior to their inclusion in the study.

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Author Contribution

R.m. and S.A. collected the dataS.A. wrote the manuscriptE.S. designed and supervised the workall authors reviewed the manuscript

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Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request

References

1. Ameer MA, Chaudhry H, Mushtaq J et al (2022) An overview of systemic lupus erythematosus (SLE) pathogenesis, classification, and management. Cureus. 2022;14(10):e30341
2. Doria A, Iaccarino L, Ghirardello A et al (2006) Long-term prognosis and causes of death in systemic lupus erythematosus. Am J Med. 2006;119(8):700–706
3. Tarr T, Papp G, Nagy N et al (2017) Chronic high-dose glucocorticoid therapy triggers the development of chronic organ damage in systemic lupus erythematosus. Clin Rheumatol. 2017;36(2):327–333
4. Gatto M, Saccon F, Zen M et al (2019) Preclinical and early systemic lupus erythematosus. Best Pract Res Clin Rheumatol. 2019;33(4):101422
5. Dörner T, Vital EM, Ohrndorf S et al (2022) Musculoskeletal involvement in rheumatoid arthritis and systemic lupus erythematosus: a literature review. Rheumatol Ther. 2022;9(3):781–802
6. Cervera R, Khamashta MA, Font J et al (2003) Morbidity and mortality in SLE: a 10-year study of 1,000 patients. Medicine (Baltimore). 2003;82(5):299–308
7. Mohamed AAA, Hammam N, El Zohri MH et al (2019) Cardiac manifestations in SLE: subclinical echocardiographic features. Biomed Res Int. 2019;2019:2437105
8. Vincze K, Kovats Z, Cseh A et al (2014) Pulmonary manifestations in SLE and peripheral CD4 + cells. Respir Med. 2014;108(5):766–774
9. Santacruz JC, Mantilla MJ, Rueda I et al (2022) Hematologic manifestations of SLE. Cureus. 2022;14(3):e22902
10. Bashal F (2013) Hematological disorders in SLE. Open Rheumatol J. 2013;7:87–95
11. Kaul A, Gordon C, Crow MK et al (2016) Systemic lupus erythematosus. Nat Rev Dis Primers. 2016;2:16039
12. Suárez-Avellaneda A, Quintana JH, Aragón CC et al (2020) SLE in the ICU: a systematic review. Lupus. 2020;29(11):1364–1376
13. Zamir G, Haviv-Yadid Y, Sharif K et al (2018) Mortality of SLE patients admitted to the ICU. Best Pract Res Clin Rheumatol. 2018;32(5):701–709
14. Namendys-Silva SA, Baltazar-Torres JA et al (2009) Prognostic factors in SLE patients in the ICU. Lupus. 2009;18(13):1252–1258
15. Gladman DD, Ibañez D, Urowitz MB (2002) SLEDAI-2000. J Rheumatol. 2002;29(2):288–291
16. Harris PA, Taylor R, Thielke R et al (2009) Research electronic data capture (REDCap)—a metadata-driven methodology and workflow process for providing translational research informatics support. J Biomed Inform. 2009;42(2):377–381

17. Gladman DD, Goldsmith CH, Urowitz MB et al (2000) The Systemic Lupus International Collaborating Clinics/American College of Rheumatology Damage Index for systemic lupus erythematosus international comparison. J Rheumatol. 2000;27(2):373–376
18. Ghazali WSW, Daud SMM, Mohammad N et al (2018) SLICC Damage Index in SLE patients and its associated factors. Medicine (Baltimore). 2018;97(42):e12787
19. Barreneche MF, Tamayo WD, Roldan DM et al (2019) Clinical and epidemiologic characterisation of SLE ICU patients. Adv Rheumatol. 2019;59:29
20. Sestan M, Kifer N, Arsov T et al (2023) Genetic risk in childhood-onset SLE. Curr Issues Mol Biol. 2023;45(7):5981–6002
21. Roussou E, Weerakoon A, Iacovou C et al (2013) Ethnic differences in pregnancy outcomes in SLE. Clin Rheumatol. 2013;32:1223–1228
22. Zamani B, Shayestehpour M, Esfahanian F et al (2020) Factors associated with pregnancy outcomes in SLE. BMC Res Notes. 2020;13:185
23. Yamamoto Y, Aoki S (2016) Strategies to improve pregnancy outcomes in SLE. Int J Womens Health. 2016;8:265–272
24. Gamal SM, Mokbel A, Niazy MH et al (2023) Comorbidities among Egyptian SLE patients: the COMOSLE-EGYPT study. Chronic Illn. 2023;19(4):791–803
25. Aragón CC, Ruiz-Ordoñez I, Quintana JH et al (2020) Outcomes in SLE ICU admissions. Lupus. 2020;29(9):1133–1139
26. Northcott M, Morand EF (2021) Glucocorticoids. In: Lahita RG, Tsokos G, Buyon J, Koike T, editors. Lahita's Systemic Lupus Erythematosus. London: Academic Press; 2021. pp. 611–622
27. Zen M, Salmaso L, Amidei CB et al (2023) Mortality in SLE: a population-based study. Eur J Intern Med. 2023;112:45–51
28. Bhargav A, Ramanathan V, Ramadoss R et al (2023) Outcomes of critically ill SLE patients in Southern India. Lupus. 2023;32(12):1462–1470
29. Pisetsky DS, Bossuyt X, Meroni PL (2019) ANA as an entry criterion for SLE classification. Autoimmun Rev. 2019;18(12):102400
30. Momtaz OM, Senara SH, Zaky SH et al (2019) Critically ill SLE patients in Fayoum University Hospital. Egypt Rheumatol. 2019;41(2):129–133
31. Alza YS, Sánchez-Bautista J, Alarcón-Gómez ZM et al (2023) Anaemia in Colombian SLE patients. CES Med. 2023;37(2):25–33
32. Bruce IN, Clark-Soloninka CA, Spitzer KA et al (2000) Beta2-glycoprotein I antibodies in SLE and APS. J Rheumatol. 2000;27(12):2833–2837

Figures

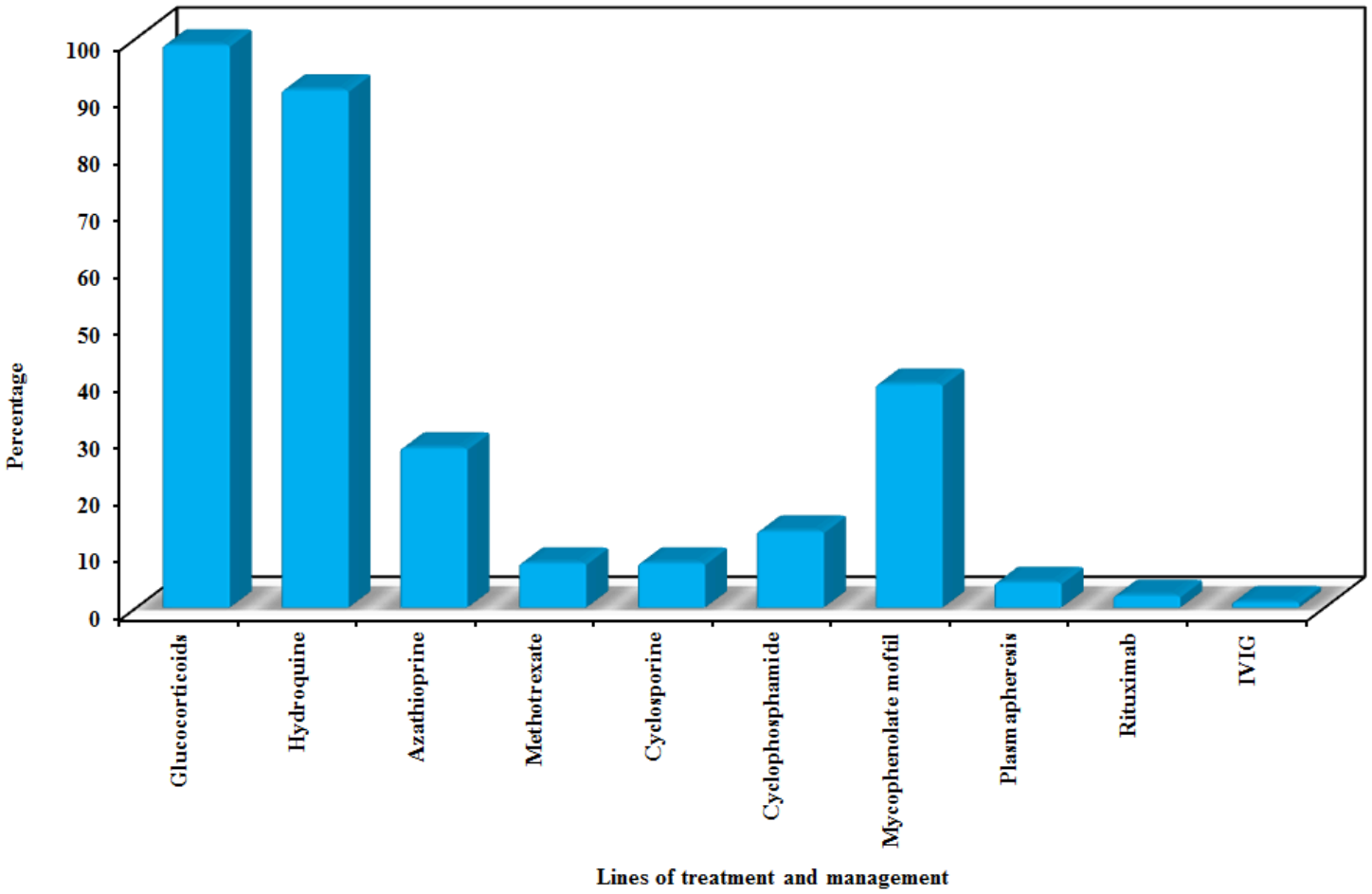


Figure 1

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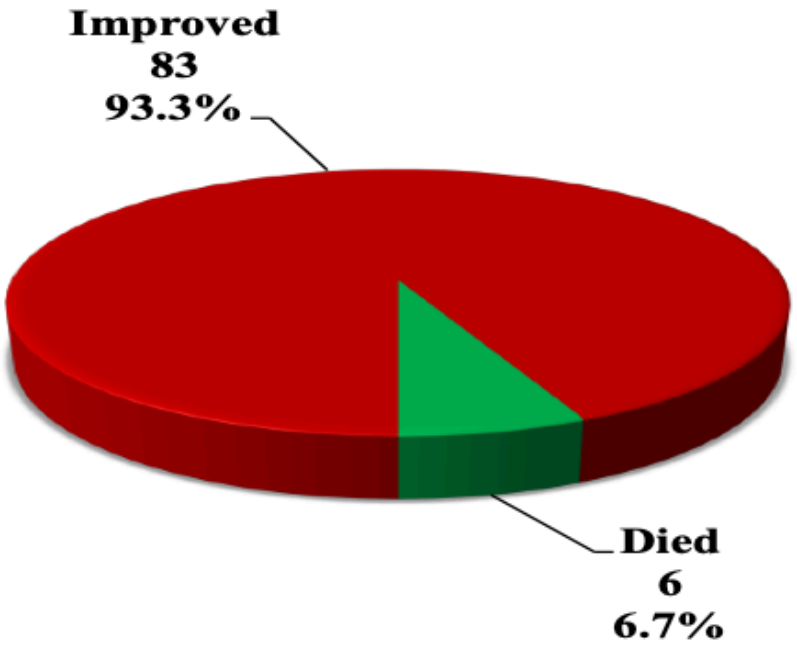


Figure 2

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