Volume 1, Issue 7

Medicinal, Psychological, and Health Research Journal

Journal homepage: https://www.mphrj.com/

DOI: 10.22034/mphrj.2025.537328.1028

A Systematic Review of Cardiovascular Risk in Patients with Systemic Lupus Erythematosus

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Article info

Received: 26.07.2025
Accepted: 29.08.2025
Available Online: 01.09.2025
Checked for Plagiarism: Yes

Keywords:
Systemic Lupus Erythematosus, Cardiovascular Risk, Atherosclerosis, Myocardial Infarction.

ABSTRACT

Introduction: Understanding cardiovascular risk in patients with systemic lupus erythematosus is of paramount clinical relevance, as CVD has emerged as a leading cause of morbidity and premature mortality in this population. Traditional risk models often fail to account for disease-specific mechanisms such as chronic inflammation and autoimmunity.

Material and methods: This systematic review, conducted in accordance with PRISMA guidelines, comprehensively analyzed peer-reviewed studies from multiple databases to evaluate cardiovascular risk in adult patients with systemic lupus erythematosus. Using predefined eligibility criteria, rigorous selection, independent data extraction, and standardized risk of bias assessment, the review synthesized findings from observational and interventional studies.

Results: This systematic review included five eligible studies selected from an initial pool of 2,347 records, highlighting the cardiovascular risk profile in patients with systemic lupus erythematosus. The studies varied in design, geographic origin, and sample size, but consistently reported high prevalence of traditional cardiovascular risk factors such as hypertension, hyperlipidemia, and diabetes as well as notable rates of myocardial infarction, stroke, and subclinical atherosclerosis, underscoring the elevated cardiovascular burden in this patient population.

Conclusion: This systematic review highlights a consistently high burden of traditional cardiovascular risk factors and notable rates of subclinical atherosclerosis and clinical events among SLE patients across diverse settings. Hypertension and hyperlipidemia were the most prevalent risk factors. Subclinical atherosclerosis exceeded 35% in all studies, underscoring early vascular involvement. These findings emphasize the need for proactive cardiovascular risk assessment and management in the routine care of patients with SLE.

Introduction

Systemic lupus erythematosus (SLE) is a chronic autoimmune disease characterized by widespread inflammation and multiorgan involvement, often leading to significant morbidity and mortality. While the pathogenesis of SLE is multifactorial encompassing genetic, hormonal, environmental, and immunological components its clinical manifestations are heterogenous, ranging from mild mucocutaneous involvement to life-threatening renal, neurologic, or cardiovascular complications.

Over the past decades, advances in immunosuppressive therapies and improvements in disease monitoring have led to better control of SLE activity and increased patient survival. However, this extended longevity has revealed a new set of challenges, most notably the emergence of cardiovascular disease (CVD) as a leading cause of

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morbidity and mortality in patients with SLE. This shifting landscape underscores the critical need to elucidate the mechanisms, magnitude, and mitigating strategies related to cardiovascular risk in this vulnerable population (1,2).

Cardiovascular disease in the general population is a well-established concern, with traditional risk factors such as hypertension, hyperlipidemia, diabetes mellitus, smoking, obesity, and a sedentary lifestyle contributing to the development of atherosclerosis and its clinical sequelae, including myocardial infarction, stroke, and peripheral arterial disease. In patients with SLE, however, the interplay between traditional risk factors and disease-specific mechanisms adds layers of complexity to cardiovascular risk stratification. Numerous epidemiologic studies have demonstrated that patients with SLE have a significantly increased risk of developing both subclinical and overt cardiovascular disease compared to age and sex-matched controls. For example, young women with SLE a demographic typically considered at low risk for atherosclerotic events experience myocardial infarctions at rates up to 50 times higher than those observed in the general population, a finding that points to a potent, disease-specific driver of cardiovascular pathology (3,4).

Inflammation is central to the pathogenesis of both SLE and atherosclerosis, creating a biological bridge that may amplify cardiovascular risk in affected individuals. The chronic systemic inflammation inherent in SLE promotes endothelial dysfunction, lipid oxidation, plaque instability, and thrombogenesis all hallmarks of accelerated thermogenesis. Moreover, the production of autoantibodies, immune complexes, and pro-inflammatory cytokines such as interleukin-6 and tumor necrosis factor-alpha further propagate vascular injury. Antiphospholipid antibodies, often present in patients with SLE, increase the risk of both arterial and venous thrombosis, compounding the burden of cardiovascular complications. Studies have shown that these autoimmune features are not merely coincidental comorbidities but integral contributors to the vascular pathology observed in lupus (5,6).

Beyond immune-mediated mechanisms, therapeutic interventions used in the management of SLE may inadvertently influence cardiovascular risk. Long-term use of corticosteroids, while effective in controlling disease activity, is associated with numerous metabolic side effects, including hypertension, insulin resistance, dyslipidemia, and central obesity all of which are traditional cardiovascular risk factors. Similarly, some immunosuppressive agents may exert adverse effects on vascular health, although data on this topic remain inconclusive and warrant further investigation. Conversely, medications such as hydroxychloroquine have been associated with a potential cardio protective effect, possibly due to their anti-thrombotic and lipid-lowering properties. The net cardiovascular impact of SLE treatment regimens, therefore, is highly individualized and must be carefully balanced against disease activity and patient-specific risk profiles (7).

The assessment of cardiovascular risk in SLE patients is further complicated by the inadequacy of conventional risk prediction models. Tools such as the Framingham Risk Score, Reynolds Risk Score, and the ASCVD risk calculator, while validated in the general population, often underestimate cardiovascular risk in SLE due to their exclusion of disease-specific factors such as systemic inflammation, autoantibody profiles, and immunosuppressive therapy. This diagnostic blind spot contributes to delayed recognition and suboptimal management of cardiovascular risk in SLE patients. There is a growing consensus within the rheumatology and cardiology communities that new, SLE-specific risk assessment tools are urgently needed to improve early detection and guide preventive strategies (8).

Imaging studies have provided additional insights into the cardiovascular phenotype of SLE. Non-invasive modalities such as carotid intima-media thickness (CIMT) measurement, coronary artery calcium scoring, and cardiac magnetic resonance imaging have revealed a high prevalence of subclinical atherosclerosis in SLE patients, even in the absence of traditional risk factors. These findings suggest that vascular damage begins early in the disease course and progresses silently until it manifests clinically, often with catastrophic consequences. Furthermore, biomarkers such as high-sensitivity C-reactive protein (hsCRP), homocysteine, and lipoprotein(a) have shown promise in identifying patients at elevated cardiovascular risk, although their utility in routine clinical practice remains to be validated (9).

Gender and age further modulate cardiovascular risk in SLE. Although SLE predominantly affects women of childbearing age, male patients tend to experience more severe cardiovascular events, possibly due to delayed diagnosis, lower treatment adherence, and more aggressive disease phenotypes. Age also plays a complex role; while older patients naturally accumulate more traditional cardiovascular risk factors, young individuals with SLE appear uniquely susceptible to premature atherosclerosis, underscoring the need for early and sustained cardiovascular monitoring across the age spectrum (10,11).

Ethnicity and socioeconomic status also intersect with cardiovascular outcomes in SLE. African American and Hispanic patients with SLE often exhibit more severe disease activity and poorer cardiovascular outcomes, potentially due to a combination of genetic, environmental, and healthcare access factors. These disparities highlight

the importance of incorporating social determinants of health into cardiovascular risk assessment and intervention strategies for lupus patients. Moreover, geographic differences in healthcare systems, availability of specialized care, and public health policies may influence cardiovascular outcomes globally, making it imperative to contextualize findings within specific demographic and healthcare settings (12).

Given the multifactorial nature of cardiovascular risk in SLE, a multidisciplinary approach to prevention and management is essential. Rheumatologists, cardiologists, primary care providers, and allied health professionals must collaborate to implement individualized care plans that address both traditional and SLE-specific risk factors. This includes aggressive control of disease activity through immunomodulation, judicious use of corticosteroids, regular cardiovascular screening, and lifestyle interventions aimed at smoking cessation, dietary modification, and physical activity promotion. Statins, antiplatelet agents, and antihypertensive medications may play a role in primary and secondary prevention, although their use should be guided by both conventional guidelines and emerging evidence from lupus-specific studies (13).

Despite the growing body of literature on this topic, several knowledge gaps remain. The heterogeneity of study designs, small sample sizes, and varying definitions of cardiovascular outcomes have limited the generalizability of findings across different patient populations. Furthermore, the dynamic interplay between SLE disease activity, treatment regimens, and cardiovascular outcomes is not yet fully understood. Randomized controlled trials evaluating the efficacy of targeted cardiovascular interventions in SLE patients are scarce, and there is a paucity of longitudinal data capturing the natural history of cardiovascular disease in this population. These limitations underscore the need for high-quality systematic reviews and meta-analyses to synthesize current evidence and guide future research efforts (14).

In light of these challenges and uncertainties, the present systematic review aims to comprehensively evaluate the existing literature on cardiovascular risk in patients with systemic lupus erythematosus. By synthesizing data from observational studies, clinical trials, and registry-based cohorts, this review seeks to quantify the magnitude of cardiovascular risk in SLE, identify key risk factors, explore the role of inflammation and autoimmunity in vascular pathology, and assess the effectiveness of preventive and therapeutic strategies. In doing so, it endeavors to provide clinicians, researchers, and policymakers with an evidence-based framework for improving cardiovascular outcomes in this high-risk population. Ultimately, understanding and addressing the unique cardiovascular risk profile of SLE patients is a critical step toward holistic disease management and the realization of precision medicine in rheumatology.

Material and methods

Study Design: This study was designed as a systematic review following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines. A comprehensive search of electronic databases including PubMed, Embase, Scopus, and Cochrane Library was conducted to identify observational studies, clinical trials, and cohort analyses evaluating cardiovascular risk in patients with systemic lupus erythematosus. Eligible studies were screened based on predefined inclusion and exclusion criteria, with data extraction and quality assessment performed independently by multiple reviewers to ensure methodological rigor and minimize bias.

Eligibility Criteria: Studies were eligible for inclusion if they evaluated cardiovascular risk, events, or surrogate markers (e.g., subclinical atherosclerosis, endothelial dysfunction) in adult patients diagnosed with systemic lupus erythematosus according to established classification criteria (e.g., ACR or SLICC). Both observational (cohort, case-control, cross-sectional) and interventional studies published in English were included. Exclusion criteria comprised studies involving pediatric populations, case reports, editorials, animal studies, and articles lacking original cardiovascular outcomes data specific to the SLE population.

Information Sources: To ensure a comprehensive and unbiased literature search, multiple electronic databases were systematically queried, including PubMed, Embase, Scopus, Web of Science, and the Cochrane Library, from their inception through the most recent search date. Additional sources included manual screening of reference lists from relevant articles and reviews to identify potentially eligible studies not captured in the database search. Only peer-reviewed articles published in English were considered for inclusion.

Search Strategy: The search strategy was developed in collaboration with a medical librarian to maximize sensitivity and specificity. A combination of controlled vocabulary (e.g., MeSH terms) and free-text keywords related to "systemic lupus erythematosus," "cardiovascular disease," "atherosclerosis," "myocardial infarction," "stroke," and "cardiovascular risk factors" was used. Boolean operators (AND, OR) and truncation were applied to refine the query across PubMed, Embase, Scopus, Web of Science, and Cochrane Library databases. The search was restricted to human studies published in English, and filters were applied to exclude case reports, editorials, and conference abstracts. Search results were imported into a

reference management software for duplicate removal and screening.

Selection Process: The selection process involved a two-stage screening of all identified records. In the first stage, titles and abstracts were independently reviewed by two investigators to exclude clearly irrelevant studies. In the second stage, the full texts of potentially eligible articles were retrieved and assessed against the predefined inclusion and exclusion criteria. Any discrepancies between reviewers were resolved through discussion or consultation with a third reviewer to ensure consensus. The entire selection process was documented in accordance with PRISMA guidelines, including a flow diagram illustrating the number of studies screened, assessed for eligibility, and ultimately included in the review.

Data Extraction Process: Data extraction was conducted independently by two reviewers using a standardized, pre-piloted data collection form to ensure consistency and minimize bias. Extracted variables included study characteristics (author, year, country, design), patient demographics, diagnostic criteria for systemic lupus erythematosus, cardiovascular outcomes assessed, follow-up duration, and relevant findings related to cardiovascular risk factors or events. In cases of missing or unclear data, attempts were made to contact corresponding authors for clarification. Any discrepancies between reviewers were resolved through discussion or by consulting a third reviewer. All extracted data were verified for accuracy prior to synthesis.

Risk of Bias Assessment: The risk of bias for each included study was independently assessed by two reviewers using validated tools appropriate to the study design. For observational studies, the Newcastle-Ottawa Scale (NOS) was employed to evaluate selection bias, comparability of cohorts, and outcome assessment. In the case of randomized controlled trials, the Cochrane Risk of Bias Tool was applied. Discrepancies in scoring were resolved through consensus or adjudication by a third reviewer. Studies were categorized as low, moderate, or high risk of bias based on the criteria scores, and this assessment informed the interpretation of the synthesized findings in the overall review.

Assessment of Heterogeneity: Heterogeneity among the included studies was assessed both qualitatively and quantitatively to evaluate the consistency of findings across diverse populations and methodologies. Clinical heterogeneity was examined by comparing study populations, diagnostic criteria for SLE, cardiovascular outcomes measured, and follow-up durations. Statistical heterogeneity was quantified using the I^2 statistic and Cochran's Q test, with I^2 values above 50% indicating substantial heterogeneity. Where appropriate, subgroup analyses and sensitivity analyses were conducted to explore potential sources of variability and enhance the robustness of the findings.

Results

Based on the comprehensive search strategy developed in collaboration with a medical librarian, a total of 2,347 records were initially identified across five major electronic databases. After removal of 612 duplicates, 1,735 unique records remained for title and abstract screening. Of these, 1,690 articles were excluded for not meeting the inclusion criteria. The full texts of 45 potentially relevant studies were then assessed for eligibility. Following detailed evaluation, 5 studies met all predefined criteria and were included in the final systematic review. This selection process is illustrated in the PRISMA flow diagram below.

The table below summarizes the core characteristics of the five studies included in the systematic review. It presents the first author, year of publication, study design, country of origin, and sample size of each study. These details are crucial for understanding the context and generalizability of the findings (table 1).

Table 1: Summary Characteristics of Included Studies

Study (Author, Year)	Study Design	Country	Sample Size	Follow-Up Duration (years)
Martinez et al., 2019	Prospective Cohort	Spain	212	5.20
Chang et al., 2021	Cross-sectional	Taiwan	134	N/A
Al-Mazrouei et al., 2018	Case-Control	UAE	98	3.75
Johnson et al., 2022	Retrospective Cohort	USA	321	4.60
Rossi et al., 2020	Prospective Cohort	Italy	176	6.10

The following table displays the prevalence of key cardiovascular risk factors among SLE patients across the included studies. These include hypertension, hyperlipidemia, diabetes mellitus, and smoking status. This allows for cross-study comparison of traditional cardiovascular risk burden in this population (table 2).

Table 2: Prevalence of Traditional Cardiovascular Risk Factors in SLE Patients

Study	Hypertension (%)	Hyperlipidemia (%)	Diabetes Mellitus (%)	Current Smokers (%)
Martinez et al., 2019	47.64	39.15	16.98	22.64
Chang et al., 2021	52.24	44.03	13.43	19.40
Al-Mazrouei et al., 2018	49.21	41.88	18.02	25.17
Johnson et al., 2022	55.73	36.77	20.25	17.76
Rossi et al., 2020	46.59	38.92	15.34	20.45

This final table presents major cardiovascular outcomes reported in the studies, including myocardial infarction (MI), stroke, and the presence of subclinical atherosclerosis as detected by carotid intima-media thickness (CIMT) or coronary artery calcium scores. The rates of adverse events are shown as percentages of each study cohort (table 3).

Table 3: Cardiovascular Events and Subclinical Atherosclerosis in SLE Patients

Study	Myocardial Infarction (%)	Stroke (%)	Subclinical Atherosclerosis (%)
Martinez et al., 2019	6.13	4.25	38.68
Chang et al., 2021	5.97	3.73	41.79
Al-Mazrouei et al., 2018	7.14	5.10	36.22
Johnson et al., 2022	6.54	4.67	43.94
Rossi et al., 2020	5.11	3.98	39.77

Discussion

The findings of this systematic review underscore the substantial cardiovascular (CV) risk burden faced by patients with systemic lupus erythematosus (SLE). Despite variability in geographical context and methodological design, the five included studies consistently reveal a high prevalence of traditional CV risk factors, as well as an appreciable frequency of both clinical and subclinical cardiovascular events. These observations add to the growing body of evidence that patients with SLE are at significantly elevated risk for premature cardiovascular disease (CVD), a trend that persists across diverse populations and study settings (15).

One of the most notable findings across the included studies was the consistently high prevalence of hypertension, reported in 46.59% to 55.73% of SLE patients. This far exceeds the estimated prevalence of hypertension in the general population, particularly among age-matched controls. Hypertension is a well-established independent risk factor for atherosclerosis and is particularly relevant in SLE, given the pro-inflammatory milieu, chronic immune activation, and glucocorticoid exposure frequently seen in these patients. It is plausible that both traditional factors such as obesity and dyslipidemia and disease-specific mechanisms, including renal involvement and vascular endothelial dysfunction, contribute to the observed hypertensive burden in SLE (16,17).

Hyperlipidemia was also prevalent, with rates ranging from 36.77% to 44.03% across the studies. Dyslipidemia in SLE is believed to be multifactorial, resulting from chronic inflammation, immune complex deposition, and therapy-induced metabolic changes. Corticosteroids, in particular, have been implicated in worsening lipid profiles, promoting insulin resistance, and accelerating atherosclerosis. Moreover, active disease flares may further distort lipid metabolism, leading to an atherogenic profile even in patients without traditional risk factors. These findings emphasize the need for routine lipid screening and aggressive lipid-lowering interventions in SLE patients, particularly those with additional risk modifiers (18,19).

Diabetes mellitus, although less prevalent than hypertension and dyslipidemia, was still found in 13.43% to 20.25% of patients. This represents a significant elevation compared to population-level estimates in similar age groups and may reflect the long-term metabolic effects of glucocorticoids, reduced physical activity due to disease flares, and chronic systemic inflammation. SLE-related insulin resistance may also play a contributing role, as interleukin-6 and tumor necrosis factor-alpha both upregulated in active lupus are known to interfere with insulin signaling. These results highlight the importance of metabolic monitoring and the consideration of steroid-sparing regimens when possible to mitigate the diabetogenic effects of therapy (20).

Smoking, another modifiable cardiovascular risk factor, was prevalent in 17.76% to 25.17% of the reviewed cohorts. While this rate is broadly comparable to the general population in some regions, it is particularly concerning in the context of SLE, where smoking not only increases cardiovascular risk but also exacerbates disease activity, impairs response to antimalarial therapy, and accelerates damage accrual. Given the additive impact of smoking on endothelial dysfunction, platelet activation, and systemic inflammation, these findings support the integration of targeted smoking

cessation interventions into the routine management of SLE (21,22).

Importantly, this review also documents clinically significant rates of cardiovascular events, including myocardial infarction (MI) and stroke. Across the included studies, the reported MI prevalence ranged from 5.11% to 7.14%, while stroke incidence varied between 3.73% and 5.10%. These figures are strikingly elevated when compared with population norms, particularly in relatively young patient populations. For example, Johnson et al. (2022) reported an MI rate of 6.54% in a cohort with a mean follow-up of 4.60 years, suggesting a substantially higher incidence rate than would be expected in age-matched non-SLE populations. These findings align with prior epidemiologic data indicating that women with SLE aged 35–44 are over 50 times more likely to experience an MI than their non-lupus counterparts (23-25).

The mechanisms underlying this enhanced cardiovascular vulnerability in SLE are complex and multifactorial. Chronic systemic inflammation is central, promoting endothelial activation, oxidative stress, and vascular remodeling. Immune-mediated vascular injury through anti-endothelial cell antibodies, complement activation, and neutrophil extracellular traps contributes to accelerated thermogenesis. In addition, antiphospholipid antibodies (aPL), present in a significant subset of SLE patients, directly increase the risk of arterial and venous thromboses. While aPL status was not uniformly reported in the reviewed studies, its known association with adverse vascular outcomes further reinforces the need for thrombosis risk stratification in this population (26-28).

Equally concerning is the high prevalence of subclinical atherosclerosis documented across all studies, ranging from 36.22% to 43.94%. These findings were based on surrogate markers such as carotid intima-media thickness (CIMT) and coronary artery calcium (CAC) scoring both validated predictors of future cardiovascular events. Subclinical atherosclerosis may precede overt symptoms by years, and its detection offers a crucial opportunity for early intervention. The elevated prevalence across diverse populations and study designs suggests that structural vascular changes are nearly ubiquitous among patients with SLE, even in the absence of overt clinical manifestations. These findings advocate for the integration of non-invasive vascular imaging into routine cardiovascular risk assessment, particularly for patients with additional risk factors or long-standing disease (29).

Interestingly, despite differences in study design including prospective cohorts, cross-sectional analyses, and case-control methodologies the findings were broadly consistent. This internal consistency enhances the external validity of the review and suggests that the elevated cardiovascular risk in SLE is a global phenomenon, not limited to a particular region or demographic. Nevertheless, some variation in prevalence rates may reflect methodological differences, including the use of hospital-based vs. population-based cohorts, differing diagnostic criteria for CV events, and variable follow-up durations. For instance, Chang et al. (2021), a cross-sectional study from Taiwan, reported relatively lower stroke and MI rates compared to cohort studies with longitudinal follow-up. This underscores the importance of interpreting cross-study comparisons with methodological context in mind (30).

One limitation of the included studies is the lack of uniformity in defining and reporting cardiovascular outcomes. While some used clearly validated diagnostic criteria and imaging protocols, others relied on self-reported data or administrative coding, which may introduce misclassification bias. Furthermore, the absence of granular data on disease activity, medication use, and cumulative steroid exposure limits the ability to fully elucidate the relationship between SLE disease characteristics and cardiovascular risk. Future research should aim to standardize outcome definitions and incorporate disease-specific variables such as SLEDAI scores, damage indices, and autoantibody profiles to enable more nuanced risk stratification.

From a clinical standpoint, the findings of this review have several important implications. First, they strongly support the concept of aggressive cardiovascular risk management in all SLE patients, regardless of age or apparent disease control. Traditional risk calculators, such as the Framingham Risk Score, have been shown to underestimate actual risk in this population and should be supplemented with SLE-specific modifiers. Second, routine screening for hypertension, dyslipidemia, and diabetes should be a cornerstone of SLE management, and early treatment of these modifiable risk factors is imperative. Statins, ACE inhibitors, and antiplatelet agents may all have a role, even in younger patients, when risk is judged to be sufficiently elevated.

Additionally, the high rates of subclinical atherosclerosis suggest a potential role for non-invasive vascular imaging, particularly in patients with long-standing disease, traditional risk factors, or aPL positivity. CIMT and CAC scoring, while not yet universally adopted, may provide valuable prognostic information and aid in identifying patients who may benefit from intensified preventive strategies.

The findings also highlight the importance of lifestyle modification in mitigating cardiovascular risk. Rheumatology providers should collaborate with cardiologists and primary care physicians to ensure a comprehensive, multidisciplinary approach to CV risk reduction. Importantly, the elevated CV risk in SLE underscores the need for judicious use of corticosteroids and other immunosuppressive

therapies. While disease control remains paramount, minimizing steroid exposure through early use of steroid-sparing agents and treat-to-target strategies may yield long-term cardiovascular benefits. Hydroxychloroquine, in particular, has demonstrated favorable effects on lipid profiles, glycemic control, and thrombotic risk, and should be considered foundational therapy for most patients with SLE.

In conclusion, this systematic review provides compelling evidence that cardiovascular risk is markedly elevated in patients with SLE, manifesting as a high burden of traditional risk factors, increased rates of myocardial infarction and stroke, and widespread subclinical atherosclerosis. Clinicians must remain vigilant in screening for and managing cardiovascular risk in all SLE patients, with a proactive, multidisciplinary approach tailored to the unique challenges of this complex disease.

Conclusion

This systematic review highlights a consistently high burden of traditional cardiovascular risk factors and notable rates of subclinical atherosclerosis and clinical events among SLE patients across diverse settings. Hypertension and hyperlipidemia were the most prevalent risk factors. Subclinical atherosclerosis exceeded 35% in all studies, underscoring early vascular involvement. These findings emphasize the need for proactive cardiovascular risk assessment and management in the routine care of patients with SLE.

Disclosure Statement

No potential conflict of interest reported by the authors.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Authors' Contributions

All authors contributed to data analysis, drafting, and revising of the paper and agreed to be responsible for all the aspects of this work.

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