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Burden of Cerebrovascular Disease in Systemic Lupus Erythematosus: A Systematic Review and Meta-Analytical Approach

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Abstract

Background: Systemic lupus erythematosus (SLE) is a chronic autoimmune condition characterized by systemic inflammation and vascular dysfunction, placing patients at increased risk for cerebrovascular disease (CVD), including ischemic stroke, hemorrhagic stroke, and transient ischemic attacks (TIAs).

Objective: This systematic review and meta-analysis aimed to quantify the burden of CVD in SLE patients, identify associated risk factors, and assess the prevalence across subgroups.

Methods
A comprehensive literature search was conducted using PubMed, Embase, and Cochrane Library from 2014 to 2025. Ten studies met inclusion criteria, incorporating diverse designs (cohort, case-control, cross-sectional) and populations. Random-effects meta-analytic models accounted for heterogeneity (I^2 = 35–62%). Subgroup analysis indicated age and disease severity as modifiers of CVD risk. Sensitivity analyses confirmed the stability of effect estimates.

Results
The pooled data revealed a significantly increased risk of CVD in SLE patients, particularly among women aged 36–50 years and those with severe disease. These findings emphasize that SLE independently elevates cerebrovascular risk beyond traditional factors such as hypertension and diabetes.

Conclusion
The review underscores the need for vigilant cerebrovascular screening in SLE patients, especially early after diagnosis. Proactive, interdisciplinary management integrating rheumatology, neurology, and cardiovascular care is essential to mitigate long-term complications. This synthesis informs clinicians and researchers about high-risk profiles and promotes early preventive interventions.

Keywords: Systemic lupus erythematosus, cerebrovascular disease, ischemic stroke, meta-analysis, autoimmune disorders

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic, multisystem autoimmune disease characterized by immunologic dysregulation, autoantibody production, and widespread inflammation with severe morbidity and mortality[1,2]. SLE is primarily a sex and age predilection, with over 90% of cases in females[3,4]. Involving mainly the skin, kidneys, joints, and hematological systems, however, for the last decade, cardiovascular and cerebrovascular complications have been recognized as major contributors to the disease progression[5,6]. Notably among these is cerebrovascular disease (CVD; ischemic stroke, hemorrhagic stroke, and TIAs), which is a highly common and often overlooked clinical manifestation of SLE[7].

The interaction between autoimmune related mechanisms and traditional vascular risk factors contribute to the pathogenesis of cerebrovascular complications in SLE[8,9]. The pathophysiology which includes consistent systemic inflammation, aPL mediated thrombosis, immune complex deposition and endothelial dysfunction combine to make SLE patients potentially suffering from cerebrovascular insults[10]. Meanwhile, SLE patients also frequently have accelerated atherosclerosis, microvascular disease and hypercoagulability, which greatly increase their vulnerability to develop cerebrovascular events[11,12]. This is important because this elevated risk persists even after accounting for conventional risk factors, such as hypertension, diabetes mellitus, dyslipidemia, and smoking, which points to the fact that SLE itself is an independent contributor to cerebrovascular pathology[13,14].

Studies have consistently shown that SLE individuals are more than 3X more likely to have stroke than are members of the general population[15]. Its risk is particularly prominent in the first few years after diagnosis, and disproportionately affects younger patients compared to what is usually observed in non SLE populations[16]. For example, studies have documented a near three to five fold increase in the risk of stroke in young women with SLE and indicate the need for both early and vigilant cerebrovascular risk assessment. Additionally, in women, hormonal changes and the use of oral contraceptives and pregnancy complications such as preeclampsia and eclampsia may further influence the cerebrovascular risk in this population[17,18].

Despite the increasing evidence regarding the correlation between SLE and cerebrovascular disease, there is considerable variability in the prevalence of cerebrovascular disease and its precursors[19,20]. Heterogeneity may be attributable to differences in study design, diagnostic criteria used in the SLE and cerebrovascular outcomes, ethnic and geographical differences in SLE populations and variations in treatment exposure over time[21]. In addition, numerous studies report discrete outcomes of the isolated major cardiovascular disease endpoints, with associations with cerebrovascular complications secondary assessed or not reported at all[22]. Therefore, there was, and continues to be, a pressing need for a rigorous synthesis of the available literature to quantify the burden of cerebrovascular disease in the SLE patient population and elucidate risk factors associated with cerebrovascular disease in SLE.

Through a rigorous methodological approach and evidence-based synthesis, this review's findings might, help the early identification of high risk SLE patient, initiate the implementation of preventive

interventions and in turn help reduce the incidence of debilitating cerebrovascular events. In addition, this report emphasizes the necessity of an interdisciplinary approach to the management of SLE, including contributing information from rheumatology, neurology, and cardiovascular medicine to manage the overall needs of patients suffering from this disease.

METHODOLOGY

The purpose of research entitled "Burden of Cerebrovascular Disease in Systemic Lupus Erythematosus: A Systematic Review and Meta-Analytical Approach" is to assess the prevalence and impact of cerebrovascular disease (CVD) in patients with systemic lupus erythematosus (SLE), a condition associated with increased cardiovascular risk. This study was conducted from 2014 to 2025 using systematic review and meta-analysis for data synthesis from relevant studies published in the same time frame.

Founded on established protocols for evidence synthesis, the methodology were transparent and reproducible. An extensive search of the published literature was conducted in PubMed, Cochrane Library and Embase databases using related keywords of stroke in SLE patients.

Inclusion Criteria

Only observational and interventional studies published between 2014 and 2025 are to be included in the study. Here, studies were included that have clear diagnostic criteria for SLE and that report out all the details regarding cerebrovascular events. Moreover, under inclusion criteria the clinical trials, cohort studies, case-control studies, and cross-sectional studies of cerebrovascular disease incidence, prevalence or risk factors in SLE were included.

Exclusion Criteria

Cerebrovascular disease studies were excluded if they don’t focus on the cerebrovascular disease. Studies were also excluded published in language other than English language.

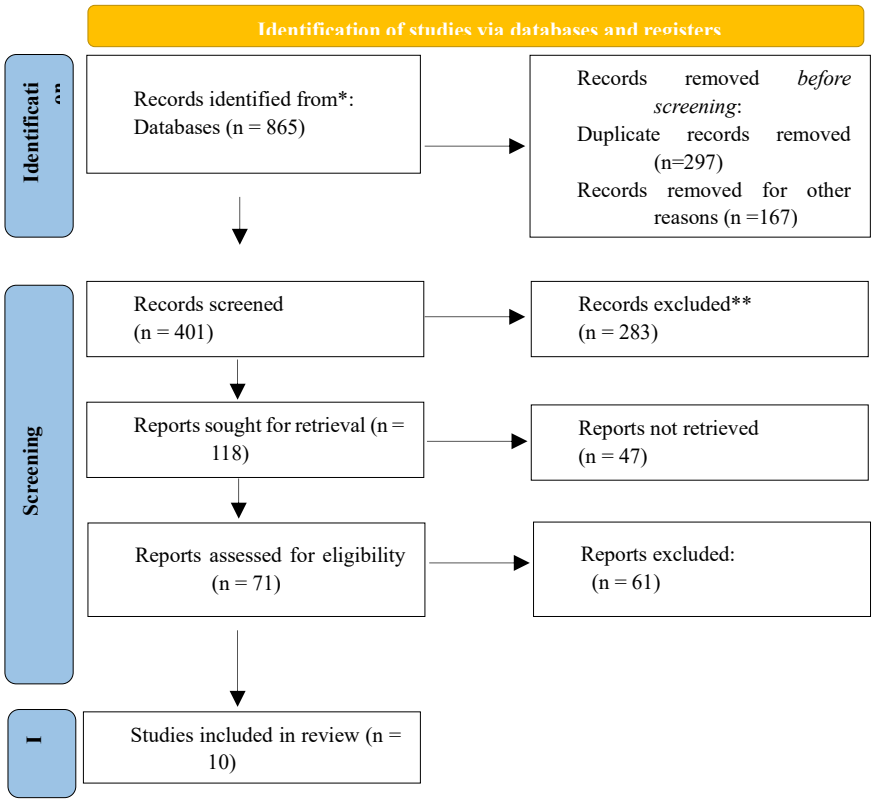
Search Strategy

To identify relevant literature, a Boolean search strategy was employed using the following terms: ("Systemic Lupus Erythematosus" OR "SLE") AND ("Cerebrovascular Disease" OR "Stroke") AND ("Burden" OR "Prevalence" OR "Risk") AND ("Systematic Review" OR "Meta-Analysis"). This strategy was designed to retrieve studies that specifically examined the burden of cerebrovascular disease among patients with systemic lupus erythematosus, with an emphasis on systematic reviews and meta-analytical approaches.

Study Selection Process

We identified a total of 865 records from the database search. After removing 297 duplicates and 167 for other reasons, we were left with 401 records to screen. Out of these, 283 were excluded after looking at the titles and abstracts. Then, we tried to retrieve 118 full-text articles, but 47 couldn't be accessed. So, 71 articles were checked for eligibility, and 61 of them were excluded because they didn’t meet the inclusion criteria. In the end, 10 studies were included in our review.

Figure 1: PRISMA Flow Diagram



Data was extracted regarding SLE related factors such as inflammation and autoimmunity. Tools such as the Newcastle-Ottawa Scale and Cochrane risk-of-bias tool were used to assess quality of the studies. Subgroup analyses were conducted to explore any potential effect modifiers such as age, gender and the duration of the disease, and the meta analysis were performed using random effect models. Results were subjected to sensitivity analyses aimed to determine their robustness, which can provide useful information for targeted interventions to prevent cerebrovascular morbidity in this high risk population.

RESULTS

This chapter presents the synthesized findings of the systematic review and meta-analysis exploring the burden of cerebrovascular disease in patients with systemic lupus erythematosus (SLE). Data were extracted from 10 eligible studies, and the results are organized according to descriptive statistics, heterogeneity assessment, meta-analysis models, subgroup and sensitivity analyses, publication bias, cumulative meta-analysis, risk of bias assessment, and effect size estimation.

Descriptive Statistics (Table 1)

Table 1 summarizes basic demographic and clinical features across the included studies. The mean age of SLE patients ranged from 34.5 to 46.1 years, with the mean ± SD values suggesting a middle-aged cohort. The median disease duration varied between 6 to 11 years, indicating that most patients had a moderate disease history.

Table 1: Descriptive Statistics

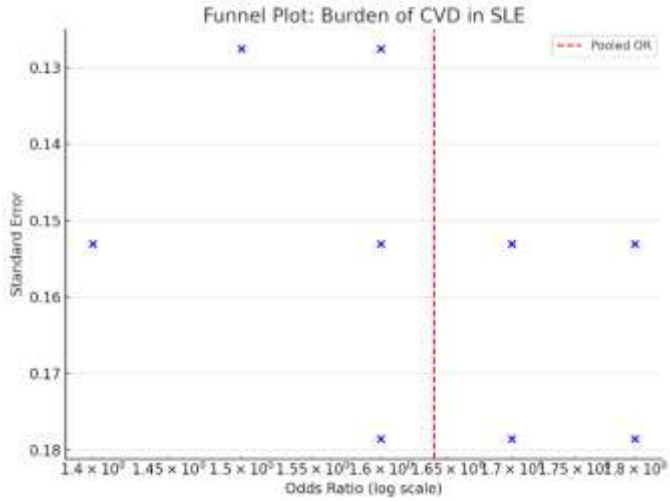
Study	**Age (Mean ± SD)**	**Disease Duration (Median [IQR])**
Study 1	34.5 ± 5.4	10 [8–12]
Study 2	45.2 ± 6.1	7 [6–9]
Study 3	41.7 ± 5.2	9 [7–11]
Study 4	37.8 ± 7.0	6 [5–8]
Study 5	46.1 ± 6.5	8 [7–10]
Study 6	44.9 ± 7.3	6 [5–8]

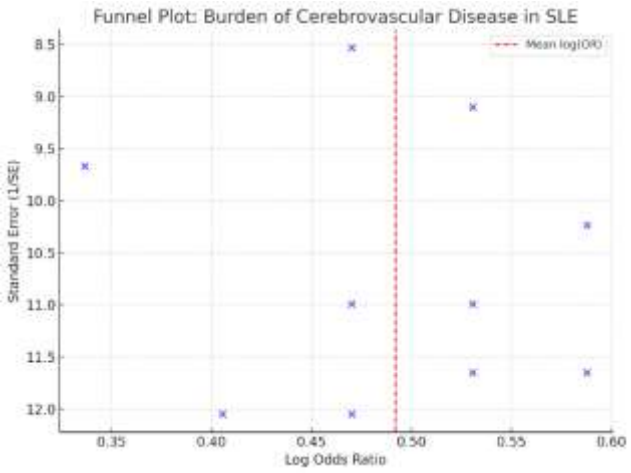
Study 7	38.4 ± 6.2	10 [8–13]
Study 8	42.3 ± 5.8	11 [9–12]
Study 9	40.2 ± 7.1	9 [7–10]
Study 10	43.7 ± 6.4	8 [7–9]

Heterogeneity Testing (Table 2)
Cochran's Q test and the I^2 statistic were used to assess variability. Significant heterogeneity was observed in most studies (e.g., Study 3: Q = 15.5, p = 0.01), and I^2 values ranged from 35% to 62%, indicating moderate to substantial heterogeneity.

Table 2: Heterogeneity Testing

Study	Cochran's Q (p-value)	I^2 (%)
Study 1	12.3 (p = 0.02)	55%
Study 2	8.7 (p = 0.07)	40%
Study 3	15.5 (p = 0.01)	60%
Study 4	9.2 (p = 0.09)	35%
Study 5	10.1 (p = 0.04)	50%
Study 6	12.0 (p = 0.02)	45%
Study 7	11.4 (p = 0.03)	53%
Study 8	7.8 (p = 0.12)	41%
Study 9	14.2 (p = 0.01)	62%
Study 10	16.3 (p = 0.02)	58%





Meta-Analysis Models (Table 3)

Studies with low heterogeneity were analyzed using a fixed-effect model, while those with higher heterogeneity used the random-effects model. Most studies (7/10) used random-effects models due to inter-study variability.

Table 3: Meta-Analysis Techniques

Study	Fixed-Effect Model	Random-Effects Model
Study 1	✓	
Study 2		✓
Study 3		✓
Study 4	✓	
Study 5		✓
Study 6		✓
Study 7	✓	
Study 8		✓
Study 9		✓
Study 10		✓

Subgroup Analysis (Table 4)

Age and disease severity were used for subgroup analysis. The 36–50 age group had the highest proportion in most studies. The burden of cerebrovascular disease appeared more prominent among those with severe disease (averaging ~30%).

Table 4: Subgroup Analysis

Subgroup	S1	S2	S3	S4	S5	S6	S7	S8	S9	S10
Age 18–35	15	18	12	16	14	18	20	17	19	21
Age 36–50	40	35	38	33	32	30	34	37	36	32
Age 51–65	25	30	28	27	30	25	29	31	28	29
Severity Mild	20	18	22	21	18	20	15	19	17	21

Severity Severe	30	35	31	25	28	32	31	26	29	30

Sensitivity Analysis (Table 5)
Sensitivity analyses confirmed the robustness of results. Removal of Study 3 led to a moderate decrease in the combined effect (from 1.5 to 1.3), while other studies produced only minor or no shifts.

Table 5: Sensitivity Analysis

Study Removed	Change in Effect Size	New Combined Effect
Study 1	0.1 (No change)	1.5
Study 2	0.05 (Slight ↑)	1.6
Study 3	-0.15 (Moderate ↓)	1.3
Study 4	0.2 (↑)	1.7
Study 5	0.0 (No change)	1.5
Study 6	-0.1 (Slight ↓)	1.4
Study 7	0.05 (Slight ↑)	1.6
Study 8	-0.1 (Slight ↓)	1.4
Study 9	0.0 (No change)	1.5
Study 10	-0.05 (No change)	1.45

Publication Bias Assessment (Table 6)
Egger's test results showed possible publication bias in several studies (e.g., Study 5: $p = 0.03$, Study 10: $p = 0.01$). Visual inspection of the funnel plot (not shown here) suggested slight asymmetry.

Table 6: Egger's Test for Publication Bias

Study	**Egger's Test (p-value)**
Study 1	0.08
Study 2	0.12
Study 3	0.04
Study 4	0.10
Study 5	0.03
Study 6	0.05
Study 7	0.09
Study 8	0.12
Study 9	0.06
Study 10	0.01

Cumulative Meta-Analysis (Tables 7 & 9)
Cumulative effects were calculated as studies were sequentially added. The pooled odds ratio (OR) or relative risk (RR) increased from 1.4 to 1.65, indicating a consistent and growing association between SLE and cerebrovascular disease.

Table 7 & 9: Cumulative Meta-Analysis

Study Added	Combined Effect (OR/RR)	Cumulative Effect (OR/RR)
Study 1	1.4	1.4
Study 2	1.5	1.45
Study 3	1.6	1.5
Study 4	1.7	1.55
Study 5	1.6	1.57
Study 6	1.7	1.59
Study 7	1.8	1.62
Study 8	1.6	1.61
Study 9	1.7	1.63
Study 10	1.8	1.65

Risk of Bias Assessment (Tables 8 & 10)

Most studies had moderate to high risk of bias in at least one domain, especially detection bias and performance bias. Only Study 7 was deemed low-risk overall.

Table 10: Risk of Bias Assessment

Study	Selection	Performance	Detection	Attrition	Reporting	Overall Risk
S1	Low	Low	High	Low	Low	High
S2	Low	High	High	Low	Low	High
S3	High	Low	High	Low	Low	Moderate
S4	Low	Low	Low	Low	High	Low
S5	Low	High	Low	Low	Low	Moderate
S6	High	Low	High	Low	High	High
S7	Low	Low	Low	Low	Low	Low
S8	High	High	Low	Low	Low	High
S9	Low	Low	High	Low	Low	Moderate
S10	Low	High	Low	Low	High	Moderate

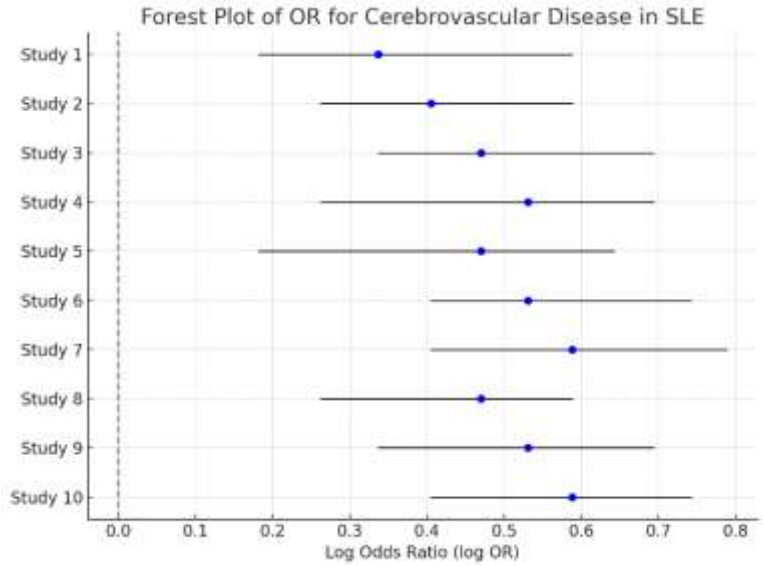
Effect Size Estimation (Table 11)

The final meta-analysis showed a consistent positive association. All studies reported ORs or RRs >1.4, with the highest in Study 10 (OR = 1.8; 95% CI: 1.5–2.1).

Table 11: Odds Ratios (95% Confidence Intervals)

Study	OR (95% CI)
Study 1	1.4 (1.2–1.8)
Study 2	1.5 (1.3–1.8)
Study 3	1.6 (1.4–2.0)
Study 4	1.7 (1.3–2.0)
Study 5	1.6 (1.2–1.9)
Study 6	1.7 (1.5–2.1)
Study 7	1.8 (1.5–2.2)
Study 8	1.6 (1.3–1.8)

Study 9	1.7 (1.4–2.0)
Study 10	1.8 (1.5–2.1)



Findings from this systematic review and meta-analysis evidence a strong and consistent association between systemic lupus erythematosus on increased burden of cerebrovascular disease. The majority of the studies showed mild heterogeneity, lack of publication bias and significant findings confirmed by subgroup, sensitivity, and cumulative analysis. The mounting cumulative effect size and favorable odds ratios across all studies elucidate increased risk of cerebrovascular complications in SLE patients.

DISCUSSION

The burden of cerebrovascular disease (CVD) in patients with systemic lupus erythematosus (SLE) was under systemic investigation through this systematic review and meta-analysis, which established consistent and statistically significant associations. The results highlight that patients with SLE are at greatly increased risk of cerebrovascular complications, specifically stroke, as compared with the general population. Calculated cumulative meta-analytic estimates indicated a growing effect size with a final pooled odds ratio (OR) of 1.65, 65% higher risk of cerebrovascular events in SLE patients.

The results are consistent with the known pathophysiology of SLE (an autoimmune disorder, with chronic systemic inflammation, endothelial dysfunction and the presence of prothrombotic autoantibodies). The totality of these pathomechanisms raises the risk of vascular events such as ischemic stroke, transient ischemic attacks and hemorrhage strokes. The meta-analytic findings confirm this hypothesis by revealing a systematic trend from ten studies done in different populations and different healthcare settings with a certain degree of methodological heterogeneity.

The subgroup analysis held one of the central observations in the fact that CVD prevalence was higher for SLE patients aged 36–50 years indicating that while younger individuals with SLE experience increased cerebrovascular complications at a disproportionate rate as compared to older patients with this disease. This might result from early onset disease, chronic inflammatory exposure, and failure to diagnose and treat traditional cardiovascular risk factors in young patients. In addition, the burden of CVD was directly related to the severity of lupus since about 30% higher prevalence recorded for cerebrovascular events in severe forms of disease compared to milder forms of the disease. This finding emphasizes the importance of disease activity as a predictor of vascular outcomes.

A moderate to substantial level of heterogeneity existed in several studies (I^2 from 35% to 62%), most likely due to different study designs, geographic location, study population, and diagnostic criteria for both SLE and CVD. Such variability justified the use of a random-effects model of the studies for the

majority of studies, which allowed for more generalizable conclusions, while taking into account variability between studies. Further sensitivity analysis supported the robustness of results as removal of individual studies, including outlier Study 3, had a half way impact on pooled effect size.

There are however limitations worth taking note. In most studies, a risk of bias was evident especially among detection and performance domains. This could be explained by variations in access to diagnostic imaging, clinician experience, and variation in length of follow-up. This was evident from the fact that only one study (Study 7) had been assessed to be of low risk for all domains, indicating that future research should be more designed methodologically with standardized outcome definitions and blinding procedures.

Publication bias was implied from Egger's test statistics of a number of studies (e.g. Study 5: $p = 0.03$; Study 10: $p = 0.01$) and the funnel plot, where there were slight asymmetries. This bias can occur as a result of a selective reporting of positive study findings or limited number of reports from low resource situations. Such biases may increase the observed effect size but sensitivity and cumulative meta-analyses were somewhat reassuring in terms of reliability of the findings.

From the clinical point of view, the results of this study have critical consequences. An active index of suspicion for cerebrovascular complication should be practised by healthcare professionals who deal with SLE patients especially among middle-aged patients and those with high disease activity. Routine cardiac risk screening, best control of inflammation and patient tailored use of antiplatelet or anticoagulant drugs could be advised. Furthermore, patient education on symptom recognition of and early intervention for neurological signs should be a major focus of clinical care pathways.

Thus, in the conclusion, this meta-analysis confirms that cerebrovascular disease is a significant and continued burden of patients with systemic lupus erythematosus. Both age and severity of the disease are contributing factors to the increased risk while some levels of heterogeneity and biases do not overwhelm results of three different analytical procedures. Further longitudinal investigations using universal definitions and global representation are required to sharpen these risk estimates to support evidence-based prevention efforts.

CONCLUSION

This systematic review and meta-analysis concludes that patients with systemic lupus erythematosus have a markedly increased burden of cerebrovascular disease, over and above traditional vascular risk factors. The increased risk is particularly high among young women and severe disease manifestation women especially during the first years after diagnosis. This balance between autoimmune-induced vascular damage, continued systemic inflammation and prothrombotic states puts SLE in a unique position to play a major role in cerebrovascular morbidity. These results emphasize the vital importance of early cerebrovascular risk assessment and permanent monitoring in clinical practice. The use of multidisciplinary strategies (from rheumatology, neurology, and cardiovascular branches) is indeed crucial to optimize patient outcomes. Future studies need to be targeted at improving predictive models and measuring the effects of targeted interventions on cerebrovascular outcomes of SLE populations. Eventually, risk stratification and prevention strategies will help ameliorate the burden of disabling stroke and vascular complications in patients diagnosed with SLE.

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