fortune Journals

Research Article

JOURNAL OF OPHTHALMOLOGY AND RESEARCH

ISSN: 2644-0024

Systemic Lupus Erythematosus (SLE) Pericarditis as a Predictor of Ophthalmic Inflammation

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Abstract

Purpose: To assess the probability that pericarditis in patients with systemic lupus erythematosus (SLE) is associated with ophthalmic inflammation using data from the TriNetX Global Research Network.

Methods: Patients with SLE with and without pericarditis were identified in the TriNetX database, using ICD-10 codes. Propensity score matching delineated two cohorts based on age, sex, race, hydroxychloroquine prescription and already existing disorders. Outcomes evaluated included the development of anterior uveitis, posterior uveitis, panuveitis, retinal vasculitis, episcleritis, scleritis, keratitis, and optic neuritis. Cerebrovascular disease risk, and mortality were collected over a 10-year period. Statistical analysis included Kaplan-Meier graphs as well as risk rates, used to compare the likelihood of getting a cerebrovascular disease.

Results: Four out of the eight inflammatory diseases being assessed in SLE pericarditis patients demonstrated significantly increased risks. The relative risk of posterior uveitis was 1.803 compared with matched SLE controls without pericarditis (95% CI: 1.154–2.815, $p < .01$). Retinal vasculitis risk was approximately doubled (RR = 2.002, 95% CI: 1.171–3.423, $p < .01$). Patients with SLE pericarditis had a 1.609-fold increased risk of episcleritis (95% CI: 1.100–2.350, $p < .05$) and a 1.742-fold increased risk of scleritis (95% CI: 1.213–2.502, $p < .01$). Mortality did not differ significantly between groups.

Conclusions: The presence of pericarditis and SLE has an association to long-term risk of posterior uveitis, retinal vasculitis, episcleritis, and scleritis. These findings emphasize the need for early detection using ophthalmologic screening for patients diagnosed with pericarditis.

Keywords: SLE; Pericarditis; Ophthalmic inflammation; Uveitis; Vasculitis; Scleritis; Keratitis; Neuritis

Introduction

Systemic lupus erythematosus (SLE) is a chronic, relapsing autoimmune disease characterized by immune-complex-mediated injury and complement activation across multiple organ systems [1]. It manifests with multisystem involvement-including the skin, kidneys, CNS, cardiovascular system, and women's health [1]. Its heterogeneous clinical course stems from organ-threatening inflammatory flares, which drive morbidity and mortality [1]. Prevalence is higher in women of childbearing age and disproportionately affects African American, Latina, and Indigenous women, reflecting the interplay of genetic and environmental factors [1].

Cardiovascular involvement is common in SLE, and pericarditis is a well-recognized manifestation of systemic vascular inflammation [2]. It is often the first

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Citation: Marina Gad El Sayed, Lucy Yu, Russell Johl, Ayden Johl, Verina Samir, Maria Zakhary, Michael Zakhary, Ibrahim Abboud, Rose Bishay, Hasson Abdel, Jaskarn S Johl. Systemic Lupus Erythematosus (SLE) Pericarditis as a Predictor of Ophthalmic Inflammation. Journal of Ophthalmology and Research. 9 (2026): 20-26.

Received: February 06, 2026
Accepted: February 18, 2026
Published: February 27, 2026

sign of SLE, reflecting early cardiac involvement [3]. Data show that pericarditis occurs in 20-50% of patients and was reported in 20% of individuals in a recent cohort of 2,900 patients [1], with recurrence in one of five [1]. Recurrence is more often seen in young adults with active disease rather than isolated cardiac pathology [2].

Pericarditis episodes are closely associated with elevations in SLE disease activity indices, including SLEDAI, hypocomplementemia, and anti-dsDNA [2]. Autoimmune pericarditis research shows that SLE pericardial inflammation arises from immune-complex injury and often clusters with renal or CNS involvement [4], marking more severe systemic immune dysregulation [4]. Autoimmune pericarditis in SLE and systemic sclerosis demonstrates that pericardial inflammation often precedes systemic flares [4,5]. Shiozawa emphasizes that SLE is not benign because chronic inflammation can damage organs and cause long-term problems [6]. Studies also show that pericarditis may recur later in life, supporting its autoinflammatory nature [5]. SLE-associated pericarditis therefore signals heightened vascular inflammation [4]. Clinically, it presents with chest pain and a systemic inflammatory response of the pericardial layers [4,5].

SLE may also affect the eyes, producing vision-threatening inflammatory conditions because any ocular structure can become inflamed [7]. Common ocular problems include episcleritis and keratitis, while more serious conditions-scleritis, uveitis, retinal vasculitis, and optic neuritis-can cause permanent vision loss [7]. Posterior segment disease reflects systemic activity, since inflammation in the eyes may signal active disease in other organs, including the CNS [7,8]. If not treated early, this can lead to irreversible damage [7].

Pericarditis and ophthalmic inflammation share immunopathogenic pathways involving immune-complex deposition, complement activation, and vascular endothelial inflammation [4,7,9]. Patients with SLE pericarditis often exhibit heightened systemic vasculitic activity [4], which may also affect ocular vessels and lead to inflammation or blindness [7,9]. Articles on inflammatory eye disease show that SLE causes blood-vessel damage systemically, including in ocular vessels, particularly when pericarditis is active [7]. Studies also show that dry eye and other ocular conditions often occur alongside systemic autoimmune disease [10], suggesting that pericarditis may act as a signal to evaluate ocular health [9]. Although pericarditis and ocular disease appear linked, no large-scale studies have evaluated whether SLE pericarditis predicts ophthalmic inflammatory involvement. Most prior research has focused on renal, neuropsychiatric, or hematologic markers, while cardiac inflammation has been overlooked [8]. This study examines whether SLE patients with pericarditis are more likely to develop ophthalmic inflammation-retinal vasculitis, scleritis, episcleritis, keratitis, and optic neuritis-compared with SLE patients without pericarditis.

Methodology

Data for this retrospective cohort study was collected from TriNetX Global Research Network. TriNetX is a global research database that contains de-identified patient records, provided by over 220 healthcare organizations (HCOs) from over 30 countries [2]. In exchange for providing data to TriNetX, contributing HCOs receive data visualization and analysis capabilities using the platform's software. Additionally, TriNetX is a federated ecosystem which secures the data through storage on hardware located within the HCO's data center. This study analyzes data within the TriNetX database, using the platform's data visualization and query functions to investigate SLE and pericarditis as a predictor of ophthalmic inflammation PSM.

Patients with SLE pericarditis were selected from the TriNetX database using ICD-10 codes corresponding to SLE (M32.X) and pericarditis (I30, I31, I32). Exclusion criteria for the study included drug-induced SLE (M32.0) as this was not the focus of the current study. See supplementary table S1 for detailed inclusion and exclusion criteria. In order to take other risk factors for SLE and ophthalmic inflammation into account, a 1:1 nearest neighbor matching algorithm was used to build two matching cohorts. Cohorts were matched based upon demographics (age, sex, race), hypertension, disorders of lipoprotein metabolism, type 2 diabetes, glomerular disease in SLE, personal history of nicotine dependence, and antiphospholipid syndrome. Lastly, cohorts were matched based upon hydroxychloroquine prescription. (see matching criteria in supplemental material S2).

The primary outcome of interest was ophthalmic inflammation, which was assessed by determining the risk of developing eight different forms of ophthalmic inflammation within a ten-year time window. These outcomes of interest included: anterior uveitis, posterior uveitis, paneuveitis, retinal vasculitis, episcleritis, scleritis, keratitis, and optic neuritis.

The secondary outcome explored was mortality, which was assessed over a ten-year window after the index date via a Kaplan-Meier plot (see Supplemental material S3). The index date was established as the first date of SLE pericarditis recorded in the affected patients, and the corresponding date in the controls. Extracted cohort data was collected between Oct 15, 2015-Oct 15, 2025. Patients with recorded ophthalmic inflammation prior to the incidence of SLE pericarditis were excluded from the study analysis.

TriNetX platform's statistical analysis software was used to analyze resulting data from the search query. Data analysis for the study included calculation of risk ratios (RRs) for ophthalmic inflammation and mortality, including a 95%

confidence interval (C.I.). Kaplan-Meier survival analysis was performed to analyze and compare time-to-event outcomes between the cohorts. Furthermore, log-rank tests were performed to determine the statistical significance of the Kaplan-Meier findings. Finally, a Cox proportional hazards ratio (HR) was generated to adjust for residual confounders and estimate the HR over time for inflammation outcomes and mortality. This study was considered non-humans subject research by the University of California, Riverside Institutional Review Board due to its exclusive use of de-identified TriNetX data.

Results

A total of 17,050 patients with SLE and pericarditis and 351,081 patients without pericarditis were identified (Figure 1). After propensity score matching, 16,113 matched pairs were identified (Table 1).

Table 1: Propensity score matching for patients with SLE and no pericarditis, and those with pericarditis. After matching, both groups included 16,113 patients.

Demographics / Diagnoses / Medications	SLE with no pericarditis (Before)	SLE with pericarditis (Before)	P-Value (Before)	SLE with no pericarditis (After)	SLE with pericarditis (After)	P-Value (After)
Age at Index (years)			<.0001			
Mean ± SD	48.3 ± 16.9	47.3 ± 18.2	<.0001	48.1 ± 17.9	47.8 ± 18.2	.16530
Female (%)	86.329%	84.211%	<.0001	84.069%	84.236%	.6804
White	51.677%	43.619%	<.0001	45.293%	44.542%	.1754
Black or African American	19.980%	33.993%	<.0001	33.340%	32.886%	.3875
Male	11.593%	13.763%	<.0001	13.920%	13.703%	.5720
Hispanic or Latino	8.839%	11.486%	<.0001	10.737%	11.239%	.1491
Asian	6.346%	5.060%	<.0001	4.723%	5.15%	.0760
Diagnoses						
Essential Hypertension	31.006%	56.434%	<.0001	55.111%	54.788%	.5604
Disorders of Lipid Metabolism	20.076%	35.221%	<.0001	34.810%	34.537%	.6066
Type 2 Diabetes Mellitus	11.572%	19.497%	<.0001	19.301%	19.202%	.8212
Glomerular Disease in SLE	3.696%	20.850%	<.0001	17.849%	17.737%	.7932
Personal History of Nicotine Dependence	7.745%	18.019%	<.0001	17.669%	17.328%	.4200
Antiphospholipid Syndrome	2.736%	8.595%	<.0001	6.963%	7.478%	.0741
Medications						
Hydroxy-chloroquine	41.806%	72.140%	<.0001	71.129%	70.992%	.7870

Out of all eight ophthalmic inflammation outcomes of interest, half of them yielded a statistically significant difference in risk between the two cohorts within the ten-year time frame. These four statistically significant ophthalmic inflammation outcomes were: posterior uveitis, retinal vasculitis, episcleritis, and scleritis (Figures 1-4).

The relative risk of developing posterior uveitis was 1.803 times greater for SLE patients with pericarditis than patients without pericarditis (95% C.I.: 1.154 – 2.815, $p < .01$). For retinal vasculitis, the relative risk among SLE patients with pericarditis was 2.002 times greater than patients with no pericarditis (95% C.I.: 1.171-3.423, $p < .01$). The risk of developing episcleritis within the ten-year time frame was 1.609 times greater for SLE patients with pericarditis than SLE patients without pericarditis (95% C.I.: 1.1 - 2.35, $p < .05$). Lastly, the relative risk of developing scleritis was 1.742 times greater for SLE patients with pericarditis than SLE patients without pericarditis (95% C.I.: 1.213 - 2.502, $p < .01$).

El Sayed MG, et al., J Opthalmol Res 2026
DOI:10.26502/fjor.2644-002400110

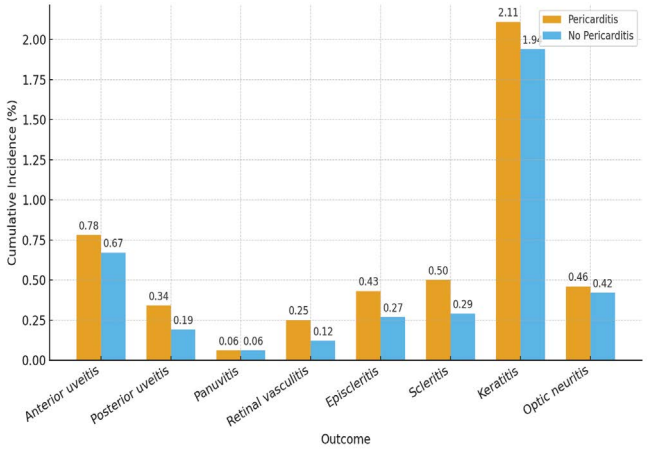

Figure 1: Bar graph depicting incidence risk of eight ophthalmic inflammation outcomes in patients diagnosed with systemic lupus erythematosus with and without pericarditis. SLE: systemic lupus erythematosus.

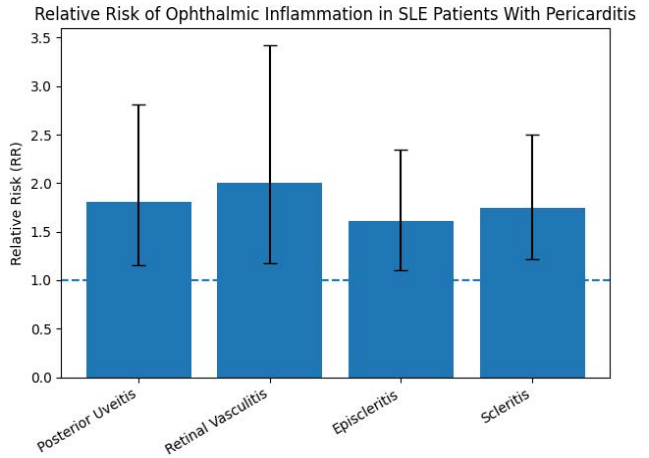

Figure 2: Bar Graph of Relative Risk for Ophthalmic Inflammation Outcomes. Relative risk of select ophthalmic inflammatory diseases in patients with systemic lupus erythematosus and pericarditis compared with matched controls without pericarditis.

Discussion

This study strongly highlights systemic lupus erythematosus (SLE) and its connection to ophthalmic inflammation. Through several studies, including our own, we concluded that SLE patients are more likely to have higher relative risks for retinal vasculitis, scleritis, episcleritis, keratitis, and optic neuritis. The diseases most strongly associated with this condition are posterior uveitis, episcleritis, scleritis, and retinal vasculitis. We observed the strongest associations with a relative risk 1.803 times greater for posterior uveitis and 2.002 for retinal vasculitis. These findings suggest that pericarditis may serve as a warning sign for vascular or endothelial inflammation. Visual impairment appears when systemic disease occurs, and in some SLE patients, ocular pain was directly linked to disease activity [11-13].

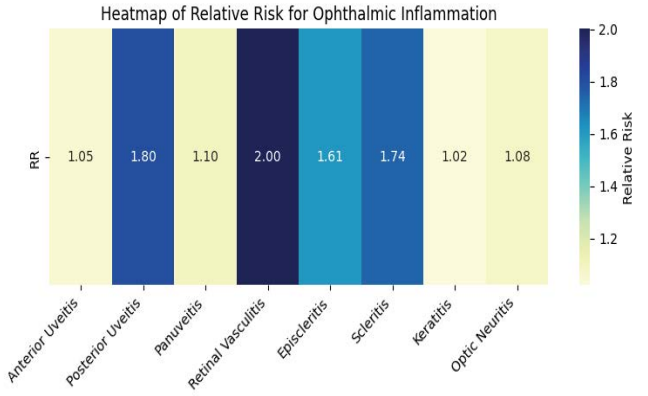

Figure 3: Heatmap of Relative Risk for All Outcomes showing the relative risk (RR) of developing eight ophthalmic inflammatory conditions in patients with systemic lupus erythematosus (SLE) and pericarditis compared with matched SLE controls without pericarditis.

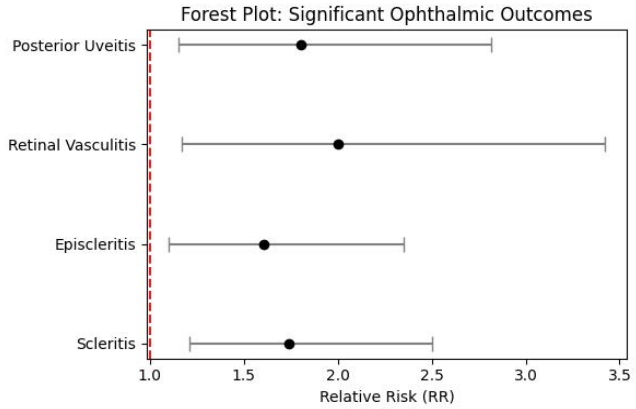

Figure 4: Forest Plot of Significant Outcomes showing relative risks (RR) and 95% confidence intervals for posterior uveitis, retinal vasculitis, episcleritis, and scleritis in SLE patients with pericarditis compared with matched controls. The red dashed line indicates RR = 1

Our results align with prior studies reporting links between SLE and ophthalmic complications [14,15]. This suggests that patients experiencing pericarditis should monitor their retinal function, as pericarditis, a cardiovascular condition, may increase the likelihood of ophthalmic disease, potentially leading to vision loss [11,16]. Previous studies addressed this association but differed in methodology, such as inclusion criteria and living conditions. Heightened systemic inflammation may contribute to additional comorbid conditions, making the association statistically significant [17,18]. Some differences in our study may explain variations compared to prior work. For example, previous researchers may not have accounted for pre-existing autoimmune diseases, which can independently cause ocular inflammation [12,14]. Our study specifically examined the likelihood of all eight retinal inflammatory diseases in patients with and without pericarditis, finding that four out of eight had high interaction rates between pericarditis and ocular inflammation [11,13].

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These associations may be explained by the immunopathology of SLE. Autoimmune activation contributes to both SLE and pericarditis [15,16]. SLE has also been linked to pulmonary arterial hypertension [19], illustrating the broader impact of systemic inflammation. The strongest associations in our study, posterior uveitis and retinal vasculitis, may result from shared pathways connecting the cardiovascular system to ophthalmic immune responses via pro-inflammatory signaling [11,13]. SLE patients are also at increased risk for central nervous system infections, representing a serious non-autoimmune complication.

Our study had several strengths. A large sample size enabled detection of variations and long-term trends. Controls accounted for demographics, smoking, and comorbidities [11,17]. Prior research shows that inflammatory signaling in SLE affects multiple organs, including the eyes, and that biologic therapies can improve disease control [11,21].

However, limitations exist. Diagnosis relied on ICD-10 codes, which may omit disease severity details. Patient medication use was not fully captured, which could affect inflammation outcomes [11,15]. The severity of SLE was not measured using validated scales, limiting interpretation of disease impact [15,17].

Despite these limitations, our findings provide strong evidence linking pericarditis with eye disease. We examined all eight inflammatory eye diseases over a ten-year period, enhancing reliability and accounting for confounding factors such as age, sex, and race [11,13,16]. Our study suggests that early eye screening is warranted for SLE patients with pericarditis or chronic autoimmune disease. Recent guidelines emphasize early monitoring and treatment to reduce organ damage and disease flares [15,19-21].

In conclusion, SLE patients with pericarditis have higher risk for posterior uveitis, retinal vasculitis, episcleritis, and scleritis. Clinicians should perform early eye screenings for these patients to prevent vision loss and optimize outcomes [11,13,15]. Overall, our study adds further evidence that SLE-mediated systemic inflammation can affect multiple organs, including the eyes.

Conclusion

This study demonstrates an increased risk of ocular damage associated with systemic immune-mediated inflammation. Presence of pericarditis and SLE has an association to long-term risk of posterior uveitis, retinal vasculitis, episcleritis, and scleritis. These findings emphasize the need for early detection using ophthalmologic screening for patients diagnosed with pericarditis. Within the ten year follow up period, the risk of retinal vasculitis as well as posterior uveitis were determined to be the highest findings within SLE patients with pericarditis. However, no statistically significant differences were detected for anterior uveitis, panuveitis, keratitis, or optic neuritis. Further studies are needed to determine more strategies for ophthalmologic surveillance, as well as to determine how to improve a patient's outcome with this disease. This altogether aligns with other scientific findings that dictate towards pericarditis being a warning of heightened disease activity within ophthalmic pathways. This determines the need for ocular follow ups to reduce the risk of irreversible blindness.

Limitations: This study contains a variety of limitations inherent to the database used to create this paper. First, the diagnostic codes may be subject to misinterpretation or misclassification creating room for issues regarding accuracy of disease identification. Next, the TriNex Research Network does not provide specific information regarding the disease severity as well as the period, giving a broad amount of data used for clinical findings. Lastly, the treatments found were unable to be fully evaluated. Information on the amount of dosage as well as duration were not provided. Further studies with diverse samples are needed to confirm these findings.

Acknowledgements

The authors would like to acknowledge the TriNex Research Network for providing the data and utilities to create this scientific article, as well as being a part of the Healthcare organization. We would also like to thank the clinicians that provided the data to make this research possible. This research received no external funding.

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Citation: Marina Gad El Sayed, Lucy Yu, Russell Johl, Ayden Johl, Verina Samir, Maria Zakhary, Michael Zakhary, Ibrahim Abboud, Rose Bishay, Hasson Abdel, Jaskarn S Johl. Systemic Lupus Erythematosus (SLE) Pericarditis as a Predictor of Ophthalmic Inflammation. Journal of Ophthalmology and Research. 9 (2026): 20-26.

Table S1: Inclusion criteria for both cohorts on the TriNetX database using ICD-10 Codes

Inclusion Criteria	Cohort 1: SLE Without Pericarditis	Cohort 2: SLE With Pericarditis
Must have One of the Following ICD-10 Codes	M32.1 – Systemic lupus erythematosus with organ or system involvement M32.8 – Other forms M32.9 – unspecified M32.10 – organ or system involvement unspecified	M32.1 – Systemic lupus erythematosus with organ or system involvement M32.8 – Other forms M32.9 – unspecified M32.10 – organ or system involvement unspecified
And Must Not Have	M32.0 – Drug-induced systemic lupus erythematosus	M32.0 – Drug-induced systemic lupus erythematosus
And Must Not Have	M32.12 – Pericarditis in systemic lupus erythematosus I30 – Acute pericarditis I31 – Other diseases of pericardium I32 – Pericarditis in diseases classified elsewhere	-
And Must Have	-	M32.12 – Pericarditis in systemic lupus erythematosus I30 – Acute pericarditis I31 – Other diseases of pericardium I32 – Pericarditis in diseases classified elsewhere

Table S2: Propensity (1:1) matching criteria for SLE pericarditis and ophthalmic inflammation risk factors based on ICD-10 codes

ICD-10/Other code	Description
Demographics	
AI	Age at Index Event
F/M	Sex (Male, Female)
2106-3, 2054-5, 2028-9	Race (White, Black/African-American, Asian)
2135-2	Ethnicity (Hispanic/Latino, Not Hispanic/Latino)
Ophthalmic Risk Factors	
I10	Essential (primary) hypertension
E78	Disorders of lipoprotein metabolism and other lipidemias
E11	Type 2 diabetes mellitus
M32.14	Glomerular disease in systemic lupus erythematosus
D68.61	Antiphospholipid syndrome
Smoking	
Z87.891	Personal history of nicotine dependence
Medications	
5521	Hydroxychloroquine

Table S3: Outcome measures selection based on ICD-10 codes

ICD-10 Code	Description
H20	Anterior uveitis (must have iridocyclitis)
H30	Posterior uveitis (must have chorioretinal inflammation)
H44.11	Panuveitis
H35.06	Retinal vasculitis
H15.1	Episcleritis
H15.0	Scleritis
H16	Keratitis
H46	Optic neuritis