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Research Article

Subclinical Retinal Microvascular Alterations In Systemic Lupus Erythematosus Detected By Optical Coherence Tomography Angiography: A Prospective Observational Study.

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Abstract

Background: Systemic lupus erythematosus (SLE) is a multisystem autoimmune disease that can affect the retinal microvasculature. Although clinically apparent lupus retinopathy is associated with disease activity and may have prognostic significance, retinal vascular abnormalities can develop before conventional fundus examination reveals visible changes. Optical coherence tomography angiography (OCTA) offers a non-invasive method for evaluating retinal microvascular architecture and may therefore help identify early, clinically silent ocular involvement.

Objective: To assess retinal microvascular and structural changes using OCTA and optical coherence tomography (OCT) in patients with SLE who had no clinically apparent retinopathy, and to examine the relationship between these retinal changes, disease activity, and accumulated organ damage.

Methods: This prospective observational study was conducted at Ain Shams University Hospitals and included 18 patients with SLE and 18 age- and sex-matched healthy controls. SLE was classified according to the Systemic Lupus International Collaborating Clinics criteria. Patients with clinically detectable retinopathy, diabetes mellitus, high myopia, previous ocular surgery other than uncomplicated cataract extraction, uveitis, glaucoma, or other systemic diseases that could affect retinal measurements were excluded.

All participants underwent comprehensive ophthalmological examination, structural OCT, and OCTA. OCTA measurements included foveal avascular zone (FAZ) area and perimeter, acircularity index, foveal density, superficial and deep retinal vessel density, and radial peripapillary capillary density. Retinal nerve fiber layer (RNFL), macular thickness, and ganglion cell complex parameters were also assessed. SLE disease activity and accumulated organ damage were evaluated using the SLE Disease Activity Index 2000 (SLEDAI-2K) and SLICC/ACR Damage Index, respectively. Patients with SLE underwent repeat ophthalmological assessment, OCT, and OCTA at 4, 8, and 12 months.

Results: The mean age was approximately 31 years in both groups, with no significant difference in age or sex distribution. Compared with healthy controls, patients with SLE had significantly lower superficial parafoveal vessel density (50.22 ± 3.49% vs. 54.32 ± 3.03%, P=0.001) and superficial perifoveal vessel density (49.29 ± 2.61% vs. 52.89 ± 2.57%, P<0.001). Perifoveal retinal thickness was also significantly lower in the SLE group (270.67 ± 15.65 µm vs. 282.37 ± 13.39 µm, P=0.025). Temporal RNFL thickness and temporal radial peripapillary capillary density were significantly reduced in patients with SLE compared with controls (69.22 ± 8.45 µm vs. 79.69 ± 11.15 µm, P=0.004; and 52.06 ± 3.78% vs. 55.37 ± 4.70%, P=0.029, respectively).

No significant between-group differences were observed in FAZ area, FAZ perimeter, foveal density, deep parafoveal or perifoveal vessel density, or GCC measurements. During follow-up, superficial foveal vessel density, foveal thickness, deep foveal vessel density, and superior RNFL thickness changed significantly. Superficial retinal vessel density showed strong inverse correlations with both SLEDAI-2K and SLICC/ACR Damage Index scores.

Conclusions: OCTA identified retinal microvascular and structural abnormalities in patients with SLE despite the absence of clinically apparent retinopathy. The most consistent abnormalities involved the superficial retinal vascular plexus and were strongly associated with disease activity and accumulated disease-related damage. OCTA may therefore provide a sensitive, non-invasive means of detecting subclinical retinal involvement in SLE.

Keywords: systemic lupus erythematosus; optical coherence tomography angiography; retinal microvasculature; vessel density; SLEDAI-2K; retinal nerve fiber layer; subclinical retinopathy.

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INTRODUCTION

Systemic lupus erythematosus (SLE) is a chronic multisystem autoimmune disease characterized by immune dysregulation, autoantibody production, immune-complex formation, and inflammatory tissue injury. Because these processes can affect multiple vascular beds, ocular involvement is an established manifestation of SLE. Retinal disease is particularly important because lupus-associated microangiopathy, vasculitis, and vaso-occlusive changes can impair vision and may also reflect the underlying activity of systemic disease. [1–3]

Clinically evident lupus retinopathy is generally characterized by manifestations of retinal microvascular injury, including cotton-wool spots, retinal hemorrhages, microaneurysms, vascular occlusion, and retinal ischemic changes. The presence and severity of these abnormalities have been associated with systemic disease activity and may provide clinically relevant prognostic information. [1,3,4]

However, the absence of visible retinopathy does not necessarily mean that the retinal microcirculation is normal. Early vascular abnormalities may develop before conventional fundus examination reveals structural lesions or before patients experience visual symptoms. Detecting these early changes is therefore of potential clinical interest, particularly in a disease in which vascular injury can evolve over time. [5–9]

Optical coherence tomography angiography (OCTA) is a non-invasive imaging technique that allows visualization of the retinal microvasculature at different depths without intravenous dye administration. In addition to providing qualitative vascular images, OCTA allows quantitative assessment of parameters such as retinal vessel density and characteristics of the foveal avascular zone (FAZ). These features have made OCTA increasingly useful for investigating subtle retinal vascular changes associated with systemic diseases. [10,11]

Previous studies have demonstrated abnormalities in retinal vessel density and retinal thickness among patients with SLE. Nevertheless, the relationship between these findings and systemic disease activity remains incompletely understood, particularly among patients who have no clinically apparent retinopathy. Longitudinal assessment may provide additional information by determining whether retinal vascular or structural parameters change over time in relation to the underlying disease. [5–9,12–15]

The present study was therefore designed to evaluate retinal microvascular and structural changes in patients with SLE who have no clinically detectable retinopathy using OCT and OCTA. We also examined whether these retinal parameters were associated with current disease activity and accumulated organ damage.

PATIENTS AND METHODS

Study design and setting

This was a prospective, observational, non-interventional study conducted at Ain Shams University Hospitals. Patients were recruited from the rheumatology outpatient clinic and the inpatient rheumatology department of El Demerdash Hospital.

The study was conducted in accordance with the principles of the Declaration of Helsinki. Institutional Review Board approval was obtained from the Faculty of Medicine, Ain Shams University, Cairo, Egypt (IRB number: FMASU M D 233/2020). Written informed consent was obtained from all participants before enrolment.

Study population

The study included 36 Egyptian participants divided equally into two groups: 18 patients with SLE fulfilling the Systemic Lupus International Collaborating Clinics (SLICC) classification criteria and 18 healthy volunteers matched for age and sex. [17,18]

Eligibility criteria

Inclusion criteria: age 18–60 years; fulfillment of the SLICC classification criteria for SLE; no clinically apparent lupus retinopathy on dilated fundus examination; and no retinal hemorrhage, vasculitis, retinal exudates, optic disc edema, or retinal detachment.

Exclusion criteria included abnormal fundus findings, age below 18 or above 60 years, high myopia (spherical equivalent >6 diopters), type 1 or type 2 diabetes mellitus, previous ocular surgery other than uncomplicated cataract extraction, uveitis, glaucoma, and other systemic diseases that could influence retinal vascular or structural measurements.

Clinical and rheumatological assessment

A detailed clinical history was obtained from all patients with SLE, including disease manifestations, disease duration, organ involvement, and current medications.

Laboratory assessment included complete blood count, erythrocyte sedimentation rate, C-reactive protein, complete urine analysis, serum creatinine, antinuclear antibodies, anti-double-stranded DNA antibodies, and assessment of urinary protein using either a 24-hour urinary protein collection or protein/creatinine ratio.

Current disease activity was assessed using the SLE Disease Activity Index 2000 (SLEDAI-2K), while accumulated organ damage was assessed using the SLICC/ACR Damage Index (SDI). [17,18]

The mean duration of SLE was 4.27 ± 2.92 years. The mean SLEDAI-2K score was 34.17 ± 18.70, and the mean SLICC/ACR Damage Index score was 3.83 ± 3.31.

Ophthalmological examination

All participants underwent a standardized ophthalmological assessment that included best-corrected visual acuity (BCVA), recorded in logarithm of the minimum angle of resolution (logMAR); spherical equivalent refraction; slit-lamp biomicroscopy; dilated fundus examination; and intraocular pressure measurement using Goldmann applanation tonometry. [19]

Structural OCT

Structural OCT was performed to evaluate retinal nerve fiber layer (RNFL) thickness, superior and inferior RNFL measurements, macular thickness and structural characteristics, and ganglion cell complex (GCC) measurements. [13]

OCTA acquisition and parameters

OCTA examinations were performed by a single examiner using the Angio-OCT Optovue RTVue XR Avanti system (Optovue Inc., Fremont, CA, USA). Macular OCTA was obtained using a 6 × 6-mm scan centered automatically on the fovea. Radial peripapillary capillary density was assessed using a 4.5 × 4.5-mm optic disc scan. The following OCTA parameters were recorded: FAZ area; FAZ perimeter; acircularity index (AI); FD-300, representing vessel density within a 300-µm-wide region surrounding the FAZ; superficial and deep retinal vessel density; foveal, parafoveal, and perifoveal vessel density; and radial peripapillary capillary vessel density in the superior, inferior, temporal, and nasal sectors.The OCTA acquisition protocol consisted of repeated B-scans with 216 B-scans per volume and 304 × 304 A-scans per B-scan. Images with a scan quality score below 6 were excluded from analysis. To reduce the potential effect of diurnal variation, examinations were performed between 12:00 PM and 2:00 PM. Quantitative measurements were obtained using the manufacturer's analytical software. [20]

Follow-up

Patients with SLE underwent repeat ophthalmological examination, structural OCT, and OCTA at 4, 8, and 12 months after the baseline assessment. Retinal vascular and structural parameters were evaluated longitudinally and examined in relation to disease activity.

Statistical analysis

Data was analyzed using IBM SPSS Statistics version 25. Continuous variables were expressed as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages.

The Shapiro–Wilk test was used to assess the distribution of continuous variables. Between-group comparisons were performed using Student's t-test for normally distributed variables and the Mann–Whitney U test for variables that were not normally distributed. Categorical variables were compared using the chi-square test or Fisher's exact test, as appropriate.

Longitudinal measurements were analyzed using repeated-measures analysis of variance or the Friedman test according to the distribution of the data. Spearman correlation analysis was used to assess relationships between retinal parameters and SLEDAI-2K or SLICC/ACR Damage Index scores. A P value <0.05 was considered statistically significant.

RESULTS

Demographic characteristics

A total of 36 participants were included in the analysis: 18 patients with SLE and 18 healthy controls. The mean age was 31.0 ± 8.40 years in the SLE group and 31.06 ± 4.89 years in the control group, with no significant difference in age (P=0.979). Women represented 83.3% of the SLE group and 77.8% of the control group, with no significant difference in sex distribution (P=0.571).

Among patients with SLE, the mean disease duration was 4.27 ± 2.92 years, ranging from 1 to 9 years. The mean SLEDAI-2K score was 34.17 ± 18.70, with a range of 3–68, while the mean SLICC/ACR Damage Index score was 3.83 ± 3.31, ranging from 0 to 13.

Variable	SLE (n=18)	Controls (n=18)	P value
Age, years	31.0 ± 8.40	31.06 ± 4.89	0.979
Female sex, n (%)	15 (83.3)	14 (77.8)	0.571
SLE duration, years	4.27 ± 2.92	—	—
SLEDAI-2K	34.17 ± 18.70	—	—
SLICC/ACR Damage Index	3.83 ± 3.31	—	—

Visual and anterior segment parameters

The mean spherical equivalent was −0.85 ± 1.83 diopters in patients with SLE and −0.91 ± 1.37 diopters in controls, with no significant difference (P=0.917). Mean BCVA was 0.06 ± 0.11 logMAR in the SLE group compared with 0.00 ± 0.00 logMAR among controls, also without a significant difference (P=0.175). Mean intraocular pressure was similar between the groups (13.44 ±

3.13 vs. 13.50 ± 3.22 mmHg, P=0.960). BCVA and intraocular pressure remained stable during follow-up.

FAZ parameters

The mean FAZ area was 0.28 ± 0.14 mm^2 in the SLE group compared with 0.31 ± 0.13 mm^2 in controls, without a significant difference (P=0.960). FAZ perimeter was also similar (2.01 ± 0.47 mm vs. 2.22 ± 0.60 mm, P=0.279). The mean acircularity index was 1.11 ± 0.03 in patients with SLE and 1.14 ± 0.12 in controls (P=0.960). During follow-up, however, the acircularity index changed significantly across the four assessment points (P=0.017), whereas FAZ area and perimeter remained statistically unchanged.

Foveal density and retinal thickness

Mean foveal density was 53.91 ± 5.35% in patients with SLE and 56.70 ± 4.61% in controls, with no significant between-group difference reported (P=0.960). Mean foveal thickness was 243.94 ± 22.80 µm in the SLE group and 241.38 ± 20.11 µm in controls (P=0.960). Although baseline foveal thickness did not differ significantly, it decreased significantly during the 12-month follow-up in the SLE group (P=0.007). Average parafoveal thickness was also similar between groups (313.44 ± 18.78 µm vs. 318.81 ± 16.00 µm, P=0.960). In contrast, perifoveal retinal thickness was significantly lower in patients with SLE (270.67 ± 15.65 µm vs. 282.37 ± 13.39 µm, P=0.025).

Superficial retinal vessel density

The most evident vascular abnormalities involved the superficial retinal vascular plexus. Superficial foveal vessel density was 53.91 ± 5.35% in the SLE group and 56.70 ± 4.61% in controls, without a statistically significant difference. In contrast, superficial parafoveal vessel density was significantly lower among patients with SLE than controls (50.22 ± 3.49% vs. 54.32 ± 3.03%, P=0.001), and superficial perifoveal vessel density was similarly reduced (49.29 ± 2.61% vs. 52.89 ± 2.57%, P<0.001).

Deep retinal vessel density

No significant differences were observed between patients with SLE and controls in deep foveal, parafoveal, or perifoveal vessel density. Deep foveal vessel density was 37.87 ± 7.70% in the SLE group and 36.66 ± 8.75% in controls (P=0.671). Deep parafoveal vessel density was 55.79 ± 4.98% and 56.93 ± 3.73%, respectively (P=0.461), while deep perifoveal vessel density was 51.56 ± 7.48% and 53.39 ± 7.00%, respectively (P=0.468). Despite the absence of a significant baseline difference, deep foveal vessel density decreased significantly during follow-up (P=0.013), from 37.87% at baseline to 34.78% at 12 months.

RNFL thickness

Temporal RNFL thickness was significantly lower in patients with SLE than in controls (69.22 ± 8.45 µm vs. 79.69 ± 11.15 µm, P=0.004). No significant between-group differences were identified in superior, inferior, or nasal RNFL thickness. During longitudinal follow-up, however, superior RNFL thickness decreased significantly (P=0.049), from 134.06 µm at baseline to 130.11 µm at 12 months.

Radial peripapillary capillary density

Temporal radial peripapillary capillary density was significantly lower in patients with SLE than in healthy controls (52.06 ± 3.78% vs. 55.37 ± 4.70%, P=0.029). No significant differences were detected in the superior, inferior, or nasal sectors. During follow-up, inferior radial peripapillary capillary density changed significantly (P=0.010), increasing from 54.28% baseline to 56.61% at 12 months.

Ganglion cell complex

No significant differences were observed between patients with SLE and healthy controls in average, superior, or inferior GCC thickness. GCC parameters likewise did not show significant longitudinal changes during the 12-month follow-up.

Correlation with SLE disease activity

Higher disease activity was associated with poorer visual acuity. SLEDAI-2K showed a significant positive correlation with logMAR BCVA (r=0.601, P=0.008). The strongest relationships were observed between SLEDAI-2K and retinal vessel density. Significant inverse correlations were found for superficial foveal vessel density (r=−0.939, P<0.001), superficial parafoveal vessel density (r=−0.920, P<0.001), and superficial perifoveal vessel density (r=−0.958, P<0.001). Deep foveal vessel density also showed a strong inverse correlation with SLEDAI-2K (r=−0.793, P<0.001). No significant correlations were identified between SLEDAI-2K and FAZ parameters, retinal thickness, RNFL measurements, radial peripapillary capillary density, or GCC parameters.

Correlation with accumulated disease damage

The SLICC/ACR Damage Index was positively correlated with logMAR BCVA (r=0.512, P=0.030). Strong inverse correlations were observed between accumulated damage and superficial retinal vessel density: superficial foveal vessel density (r=−0.807, P<0.001), superficial parafoveal vessel density (r=−0.808, P<0.001), and superficial perifoveal vessel density (r=−0.826, P<0.001). Deep foveal vessel density was also strongly inversely correlated with the SLICC/ACR Damage Index (r=−0.884, P<0.001). No significant associations were found between SLICC/ACR scores and FAZ measurements, retinal thickness, RNFL thickness, radial peripapillary capillary density, or GCC parameters.

OCTA parameter	SLEDAI-2K r	P value	SLICC/ACR r	P value
Superficial foveal vessel density	−0.939	<0.001	−0.807	<0.001
Superficial parafoveal vessel density	−0.920	<0.001	−0.808	<0.001
Superficial perifoveal vessel density	−0.958	<0.001	−0.826	<0.001
Deep foveal vessel density	−0.793	<0.001	−0.884	<0.001

DISCUSSION

The present study demonstrates that retinal abnormalities can be detected in patients with SLE even when conventional ophthalmological examination shows no clinically apparent retinopathy. Using OCT and OCTA, we identified measurable changes in retinal vascular density and retinal structure, with the most consistent vascular abnormalities involving the superficial retinal plexus. Importantly, lower superficial vessel density was strongly associated with both current disease activity and accumulated disease-related damage.

These findings support the concept that retinal involvement in SLE may begin at a microvascular level before clinically visible retinopathy develops. Conventional fundus examination remains essential for identifying established retinal lesions, but quantitative OCTA may reveal more subtle alterations that are not readily apparent during routine examinations. This distinction is clinically relevant because patients can remain visually asymptomatic despite having measurable retinal vascular abnormalities. [5–9,12]

The absence of a significant baseline difference in FAZ area and perimeter is also noteworthy. Although the FAZ was slightly smaller in the SLE group, this difference was not statistically significant. The acircularity index did not differ significantly between groups at baseline, although it changed significantly during follow-up. These findings suggest that early retinal involvement in SLE may not necessarily present as an obvious enlargement or geometric alteration of the FAZ. [5,8,12] Previous studies have reported FAZ enlargement in some patients with SLE, particularly in populations with different disease durations or treatment exposures. Differences in patient characteristics, duration of disease, hydroxychloroquine exposure, sample size, OCTA devices, scan protocols, and image-analysis software may contribute to the variation between studies. [5,8,19–22] The relatively small cohort in the present study may also have limited the ability to detect more modest FAZ differences.

The most consistent finding was the reduction in superficial retinal vessel density. Superficial parafoveal vessel density was approximately four percentage points lower in patients with SLE than in controls, while perifoveal density was also clearly reduced. In addition, superficial foveal vessel density demonstrated a significant longitudinal change during follow-up.

From a biological perspective, these findings are plausible. SLE is associated with systemic endothelial dysfunction, immune-complex deposition, inflammatory vascular injury, and microangiopathic changes. Retinal microcirculation may therefore provide a readily accessible window into systemic microvascular involvement. Previous pathological studies have demonstrated immune-related vascular changes in the retina, providing a possible mechanistic basis for the OCTA abnormalities observed in this and earlier studies. [6,8,9]

Our findings are broadly consistent with previous OCTA studies of SLE. Arfeen et al. reported reduced superficial and deep retinal vessel density in patients with SLE, while Conigliaro et al. identified associations between retinal microvascular density and systemic disease characteristics. Other investigators have also described subclinical retinal abnormalities in patients without overt retinopathy. [6,9,19]

One of the most striking findings in the present study was the strength of the inverse relationship between superficial retinal vessel density and SLEDAI-2K. Correlation coefficients ranged from −0.920 to −0.958 for superficial parafoveal and perifoveal vessel density. In practical terms, patients with greater systemic disease activity tend to have lower retinal superficial vessel density.

A similarly strong relationship was observed with accumulated disease damage. Superficial retinal vessel density was inversely associated with SLICC/ACR Damage Index scores, suggesting that these measurements may reflect not only current inflammatory activity but also the cumulative effects of the disease over time.

The potential clinical significance of these findings lies in the difference between visible retinopathy and quantitative vascular impairment. Conventional examination identifies established lesions such as hemorrhages, cotton-wool spots, and vascular occlusion. OCTA, in contrast, can quantify changes in perfusion that may precede the development of clinically visible lesions. [23,24] It is therefore possible that OCTA may provide an additional layer of information about retinal involvement in patients who appear ophthalmologically normal on routine examination.

Deep retinal vessel density showed a somewhat different pattern. Although deep foveal vessel density did not differ significantly between SLE patients and controls at baseline, it decreased significantly during follow-up and demonstrated strong inverse correlations with both SLEDAI-2K and SLICC/ACR scores. In contrast, deep parafoveal and perifoveal vessel densities did not show significant baseline differences

or significant relationships with disease activity. This regional pattern may indicate that the superficial retinal circulation is more consistently affected during subclinical disease, while deep vascular changes may be more selective or become more apparent as disease-related vascular injury progresses. However, this interpretation should remain cautious because of the small sample size and limited statistical power of the present study. [12]

Structural abnormalities were also identified. Perifoveal retinal thickness was significantly lower in patients with SLE than in controls, and foveal thickness decreased during follow-up. These findings raise the possibility that persistent microvascular impairment may be accompanied by subtle structural retinal changes. [23,24]

Temporal RNFL thickness was significantly reduced in the SLE group, and superior RNFL thickness decreased significantly during follow-up. Previous studies have reported retinal thinning in SLE and have suggested that retinal structural changes may be related to neurodegenerative processes associated with systemic disease. Because the retina shares important structural and embryological characteristics with the central nervous system, quantitative retinal imaging has also been investigated as a potential surrogate marker of neurological involvement. [23,25]

Interestingly, GCC measurements did not differ significantly between groups and did not show significant longitudinal changes. The discrepancy between the RNFL and GCC findings may have several possible explanations, including the relatively short disease duration in this cohort, the small sample size, differences in treatment exposure, or the possibility that vascular and RNFL abnormalities may become detectable before measurable GCC loss. [6–8,19]

The reduction in temporal radial peripapillary capillary density provides additional support for an association between retinal microvascular and neural structural changes. The peripapillary circulation supplies the RNFL, and the coexistence of lower temporal RPC density and reduced temporal RNFL thickness may reflect an underlying relationship between microvascular impairment and retinal neural tissue. [24]

The study has several strengths. First, it specifically focused on patients without clinically apparent retinopathy, addressing an important question regarding subclinical retinal involvement in SLE. Second, both vascular and structural retinal parameters were assessed. Third, all OCTA examinations were performed using the same device and protocol by a single examiner, which reduced potential inter-device and interobserver variability. Fourth, the longitudinal design allowed assessment of retinal changes over one year rather than relying exclusively on cross-sectional comparisons. Finally, retinal findings were examined in relation to established measures of systemic disease activity and accumulated damage.

Several limitations should also be acknowledged. The sample size was small, with only 18 patients with SLE and 18 controls, which limits statistical power and the generalizability of the findings. The study was performed at a single tertiary-care center and involved an Egyptian population, so the results may not necessarily apply to other ethnic or clinical populations.

CLINICAL IMPLICATIONS

The findings of this study suggest that a normal fundus examination does not necessarily indicate the complete absence of retinal involvement in patients with SLE. Quantitative OCTA may identify microvascular abnormalities at a stage when patients remain visually asymptomatic and conventional ophthalmological examination is normal.

From a clinical perspective, OCTA could potentially complement routine ophthalmological assessment in selected patients with SLE, particularly those with high disease activity or substantial accumulated organ damage. However, the present study does not establish a specific OCTA threshold for screening, nor does it demonstrate that OCTA-guided monitoring improves visual outcomes.

For these reasons, the findings should be considered hypothesis-generating rather than sufficient to support universal OCTA screening. Larger, multicenter, longitudinal studies are needed to determine whether OCTA-derived vascular parameters can predict clinically meaningful ocular outcomes and whether incorporating OCTA into routine SLE care provides measurable clinical benefit.

CONCLUSION

Patients with systemic lupus erythematosus who had no clinically apparent retinopathy nevertheless demonstrated measurable retinal microvascular and structural abnormalities on OCT and OCTA.

The most consistent abnormality was reduced superficial retinal vessel density, particularly in the parafoveal and perifoveal regions. Superficial vessel density showed strong inverse relationships with both SLEDAI-2K disease activity and SLICC/ACR accumulated damage. Additional abnormalities included reduced perifoveal retinal thickness, temporal RNFL thinning, and reduced temporal radial peripapillary capillary density. Longitudinal assessment also demonstrated significant changes in superficial foveal vessel density, foveal thickness, deep foveal vessel density, and superior RNFL thickness over the one-year follow-up period.

Taken together, these findings suggest that OCTA may be sensitive to subclinical retinal involvement in SLE even when conventional ophthalmological examination remains normal. Larger longitudinal studies will be needed to determine whether OCTA-derived parameters can ultimately serve as clinically useful biomarkers of systemic disease activity,

cumulative damage, or future ocular complications.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki. Ethical approval was obtained from the Institutional Review Board of the Faculty of Medicine, Ain Shams University, Cairo, Egypt (IRB number: FMASU M D 233/2020). Written informed consent was obtained from all participants.

Consent for publication

Not applicable.

Availability of data and materials

The datasets generated and/or analyzed during the current study should be made available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

George Adel Aziz Metyas: study conception, patient recruitment, ophthalmological assessment, OCT/OCTA acquisition, data collection, statistical analysis, and manuscript preparation.

Osama Abdel Moneim Raslan, Weam Mohamed Ahmed Ebeid, and Nashwa Mohamed Ezzat: ophthalmological supervision, interpretation of ophthalmological findings, and critical revision of the manuscript.

Elham Shabaan Mohamed: rheumatological assessment, evaluation of SLE disease activity and accumulated damage, and critical revision of the manuscript.

All authors review and approve the final version of the manuscript before submission.

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