Bioscientia Medicina: Journal of Biomedicine and Translational Research
https://bioscmed.com e-ISSN: 2598-0580 Vol. 10, No. 8 (2026)

ORIGINAL ARTICLE

Association of Foveal Avascular Zone Diameter with Best-Corrected Visual Acuity in Diabetic Retinopathy: A Cross-Sectional Optical Coherence Tomography Angiography Study

Ramzi Amin[1*], Rezandi Pratama[1], Petty Purwanita[1], Muhammad Aulia Molid Ogest Putra Calisanie[1]

[1]Department of Ophthalmology, Faculty of Medicine, Universitas Sriwijaya / Dr. Mohammad Hoesin Central General Hospital, Palembang, Indonesia

ARTICLE INFO

Article history
Received: June 20, 2026
Accepted: July 19, 2026
Published: August 11, 2026

Keywords:
Diabetic retinopathy
Foveal avascular zone
Optical coherence tomography angiography
Visual acuity
Retinal ischemia

**Corresponding author:*
Ramzi Amin
ramziamin20@gmail.com
ORCID: 0000-0003-1339-7580

DOI:
https://doi.org/10.37275/bsm.v10i8.1659

ABSTRACT

Background: Foveal avascular zone (FAZ) enlargement reflects capillary loss in diabetic retinopathy (DR), but its relationship with visual acuity varies across protocols and populations.

Objective: We assessed the association between swept-source optical coherence tomography angiography (OCTA)-derived FAZ diameter and best-corrected visual acuity (BCVA).

Methods: This cross-sectional study included 32 adults with DR examined from April to December 2024. One eye per participant underwent 3×3-mm swept-source OCTA. Superficial and deep FAZ diameter, superficial vessel density, central subfield thickness, and LogMAR BCVA were assessed using correlations, severity-group comparisons, multivariable regression, and exploratory secondary analyses.

Results: Mean age was 57.8±8.4 years; 17 participants (53.1%) were men. Mean superficial FAZ diameter was 617.6±171.6 µm and BCVA was 0.539±0.341 LogMAR. FAZ diameter correlated with worse BCVA (r=0.582, 95% confidence interval [CI] 0.293–0.774; p<0.001) and increased from 458.3±82.1 µm in mild nonproliferative DR to 812.5±125.8 µm in proliferative DR ($F(3,28)=16.35$; $p<0.001$; $\eta^2=0.637$). FAZ was the largest standardized correlate in the exploratory adjusted model (β=0.485; p<0.001; adjusted $R^2=0.412$). An exploratory 580-µm cutoff yielded an area under the curve of 0.847 (95% CI 0.723–0.971).

Conclusion: Larger superficial FAZ diameter was associated with worse BCVA and greater DR severity. The proposed cutoff and secondary models require independent validation.

1. Introduction

Diabetic retinopathy (DR) is a heterogeneous microvascular complication in which central and peripheral nonperfusion, visible retinal lesions, edema, and neural injury may not advance in parallel. Wider-field swept-source optical coherence tomography angiography (OCTA) studies show that nonperfusion and vascular-density metrics vary across DR severity, while longitudinal imaging demonstrates that microvascular change can add information beyond a single clinical grade.[1–3] Consequently, patients with the same categorical DR stage may still differ in macular perfusion and visual function.

The fovea depends on an organized parafoveal capillary network surrounding the central foveal avascular zone (FAZ). Diabetic capillary closure and remodeling can enlarge or distort this zone, while macular edema and inner-retinal changes may independently affect vision. Recent OCTA studies have distinguished vascular patterns associated with diabetic macular edema, related retinal vascular measures to inner-retinal thickness, and linked OCTA parameters with best-corrected visual acuity (BCVA) in treated DR eyes.[4–6] BCVA is nevertheless an integrated functional measure influenced by optical media, refraction, retinal thickness, photoreceptor integrity, prior treatment, and neural adaptation. FAZ measurements should therefore be interpreted as correlates of concurrent visual status, not isolated determinants of vision.

OCTA provides noninvasive, depth-resolved visualization of retinal and choroidal flow. It can quantify FAZ dimensions, vessel density, nonperfusion, branching, and other geometric features in the superficial capillary plexus (SCP), deeper retinal slabs, and choriocapillaris. Measurement validity depends on acquisition quality, segmentation, projection artifacts, scan size, motion correction, and device-specific algorithms. Multicenter quality assessment has documented substantial OCTA image-quality constraints, ultrawide-field swept-source imaging remains vulnerable to characteristic artifacts, and automated FAZ or nonperfusion algorithms require protocol-specific validation.[7–9] Capillary-only and

large-vessel measures may also differ in their ability to classify DR.[10] Numerical values obtained on one platform, slab definition, or field size cannot therefore be transferred uncritically to another.

Recent work has explored automated DR classification using volumetric OCT/OCTA and deep learning, clinically significant nonperfusion on widefield OCTA, and combined retinal–choroidal metrics.[11–14] These studies support the biological and analytical relevance of quantitative angiographic features but also show that no single central metric fully represents diabetic retinal ischemia. Spatial mapping demonstrates that capillary nonperfusion is not uniformly distributed, and the association between macular perfusion and DR lesions depends partly on lesion location.[15,16] A central 3×3-mm scan is therefore clinically informative but does not characterize peripheral ischemia.

The remaining translational problem is not simply whether OCTA abnormalities accompany DR, but whether a clearly defined, reproducible FAZ measure is associated with vision in a specific clinical setting. Expanded-field biomarkers have shown potential for identifying clinically significant outcomes, and longitudinal OCTA has characterized progression of diabetic macular ischemia; however, these findings use different fields, metrics, populations, and temporal designs.[17,18] Evidence from Southeast Asian tertiary-care populations using diameter-based swept-source FAZ measurements remains limited.

Novelty and aim: The novelty of this study is the severity-stratified evaluation of a diameter-based FAZ metric from 3×3-mm swept-source OCTA in an Indonesian tertiary-care cohort, with simultaneous assessment of SCP and deep capillary plexus (DCP) FAZ diameter, superficial vessel density, central subfield thickness (CST), and LogMAR BCVA. The primary aim was to estimate the cross-sectional association between SCP FAZ diameter and LogMAR BCVA. Secondary aims were to describe ocular measures across DR severity and to evaluate adjusted and explicitly exploratory associations. We hypothesized that larger FAZ diameter and lower vessel density would accompany worse BCVA; any data-derived cutoff, subgroup contrast, or statistical decomposition was treated as hypothesis-generating rather than confirmatory or causal.

2. Methods

Study design, setting, and reporting

This cross-sectional observational study was conducted in the Department of Ophthalmology, RSUP Dr. Mohammad Hoesin, Palembang, Indonesia, from April through December 2024. The analysis included one eye per participant. The study is reported with reference to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) recommendations for cross-sectional studies.[19] Because exposure and outcome were measured at the same clinical time point, all relationships are described as cross-sectional associations. Terms such as prediction, prognosis, mechanism, and mediation are used only when referring to an exploratory statistical model, not to temporal or causal effects.

Ethics and consent

The protocol was approved by the Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya (approval reference 1298/UN9.1.DL/KBK.6.1.3/2024). Written informed consent was obtained before participation. The study was conducted in accordance with the ethical principles applicable to human research and the Declaration of Helsinki. Data used for analysis were de-identified, and no directly identifying participant information is reported.

Participants and analysis eye

Consecutive adults aged 18–75 years with diabetes mellitus and clinically diagnosed DR were considered for enrollment. DR had to be documented by dilated fundus examination and/or multimodal retinal imaging. Participants needed to be able to complete reliable refraction and ETDRS-style BCVA testing, undergo OCTA of sufficient quality for quantitative assessment, and provide informed consent. Reliable measurement required understanding the task, stable refraction, and completion of the acuity examination under standardized conditions.

Exclusion criteria were media opacity that prevented adequate OCTA imaging; concurrent ocular disease likely to materially affect the macula or visual acuity, including age-related macular degeneration, glaucoma, retinal vascular occlusion, macular hole, or epiretinal membrane; intraocular surgery within the preceding three months; anti-vascular endothelial growth factor treatment or retinal laser photocoagulation within the preceding month; estimated glomerular filtration rate below 30 mL/min/1.73 m^2; or inability or unwillingness to participate. These windows limit immediate treatment-related effects but do not eliminate confounding from earlier treatment or chronic ocular changes.

For bilateral DR, the eye with worse BCVA was selected. This rule produced one analysis eye per participant and avoided within-person correlation, but it enriched the sample for poorer function and may have increased the observed association between anatomic damage and BCVA. The rule is therefore treated as a design limitation rather than a neutral sampling choice. A bilateral-eye sensitivity analysis was not available.

Clinical examination and DR severity

All participants underwent a comprehensive ophthalmic examination. Distance BCVA was assessed with an Early Treatment Diabetic Retinopathy Study-style LogMAR protocol at 4 m under standardized illumination. Higher LogMAR values indicate poorer visual acuity. Intraocular pressure was measured by applanation tonometry. The anterior segment was

examined by slit-lamp biomicroscopy. After pharmacologic dilation with tropicamide 1%, the posterior segment was assessed using a 78- or 90-diopter lens and documented clinically.

Two ophthalmologists masked to the OCTA measurements independently graded DR severity using the modified Airlie House/ETDRS framework and clinical categories aligned with the international clinical severity scale.[20] Analysis categories were mild nonproliferative DR (NPDR), moderate NPDR, severe NPDR, and proliferative DR (PDR). No participant in the analyzed cohort was categorized as having no DR. Cohen κ was 0.87 for inter-grader agreement. Because an uncertainty interval was not available, the estimate is presented descriptively without a qualitative category.

Swept-source OCTA acquisition

Macular OCTA was acquired with the DRI OCT Triton Plus swept-source system (Topcon, Tokyo, Japan). The device operates near a 1050-nm center wavelength with a nominal acquisition rate of 100,000 A-scans per second. A 3×3-mm volumetric scan centered on the fovea was used. Small-field scans provide relatively high transverse sampling but do not represent peripheral nonperfusion. The results and any derived threshold therefore apply to this field size and device workflow.

En face angiograms were evaluated separately for the SCP and deep capillary plexus (DCP). Segmentation followed the device-defined retinal slabs. The FAZ boundary was manually traced by one trained observer. Maximum horizontal and vertical chord lengths of the traced polygon were measured, and their mean was recorded as FAZ diameter in micrometers. This diameter measure differs from FAZ area, perimeter, or circularity, which are more commonly reported in parts of the OCTA literature; numerical comparisons across metrics are consequently inappropriate. Obvious boundary or segmentation errors were corrected during image assessment, although a formal artifact count was not available.

Superficial vessel density was expressed as the percentage of pixels with flow signal in the parafoveal annulus from 1 to 3 mm around the foveal center. CST was obtained from the device's structural OCT segmentation. Measurements were repeated twice for each analysis eye. The intraclass correlation coefficient for repeat measurement was 0.92. Metric-specific coefficients and confidence intervals were not available, so this estimate is interpreted as intra-observer repeatability and is not generalized to inter-observer reproducibility.

Outcomes and exposures

The primary outcome was continuous BCVA in LogMAR units. The primary exposure was SCP FAZ diameter in micrometers. Secondary exposure variables were DCP FAZ diameter, superficial vessel density, CST, age, glycated hemoglobin (HbA1c), and diabetes duration. DR severity was used for descriptive stratification and group comparisons. The exploratory ROC outcome was visual impairment defined as BCVA >0.40 LogMAR, and this definition was applied consistently throughout the analysis.

Sample-size rationale

The planned sample size was based on detecting a correlation between FAZ and BCVA. An expected r=0.55, a two-sided α of 0.05, and 90% power gave a target of approximately 32 evaluable participants by the Fisher z approach. The achieved analysis sample was 32. Although adequate for estimating a moderately large bivariate correlation, this number offers limited precision for multivariable modeling, data-derived threshold selection, statistical decomposition, and subgroup comparisons. Those secondary analyses were therefore interpreted as exploratory.

Statistical analysis

Continuous variables were summarized as mean±standard deviation when approximately symmetric and as median with interquartile range for diabetes duration. Categorical variables were reported as counts and percentages. Severity-stratified means, standard deviations, and group counts formed the primary descriptive summaries. Overall means and pooled standard deviations for OCTA and visual variables were calculated from those strata so that the full-cohort and severity-specific estimates had common denominators.

Bivariate associations with BCVA were reported as Pearson correlation coefficients. Two-sided p values were calculated from the t statistic with n−2 degrees of freedom, and 95% confidence intervals were calculated after Fisher z transformation. A positive coefficient indicates that a higher value is associated with poorer visual acuity because higher LogMAR is worse. Correlations were interpreted by magnitude and precision rather than by statistical significance alone. No correction for multiple bivariate comparisons was applied; secondary p values are descriptive.

Group differences across the four DR severity categories were evaluated with one-way analysis of variance based on the group counts, means, and standard deviations. Between-group and within-group sums of squares were calculated, and eta-squared (η^2) was defined as the between-group sum of squares divided by the total sum of squares. The omnibus analysis did not support participant-specific residual diagnostics or adjusted pairwise contrasts; consequently, no post hoc pairwise p values are claimed.

An exploratory multiple linear regression used BCVA as the dependent variable and SCP FAZ diameter, age, superficial vessel density, HbA1c, and diabetes duration as predictors. Standardized coefficients, 95% confidence intervals from 1,000 bootstrap resamples, p values, variance inflation factors, and adjusted R^2 were reported. Unstandardized coefficients and complete

residual and influence diagnostics were not available for secondary evaluation. With 32 observations and five predictors, individual coefficients may be unstable even when variance inflation factors are low.

Exploratory discrimination of BCVA >0.40 LogMAR used the author-supplied ROC analysis for SCP FAZ diameter. The available outputs included the AUC and its 95% confidence interval and a 580-µm cutoff selected with the Youden index. Sensitivity, specificity, positive predictive value, and negative predictive value were calculated from integer classification counts. The sample included 17 impaired and 15 non-impaired participants, with 14 true positives, 3 false negatives, 11 true negatives, and 4 false positives. The graphical summary displays the observed operating point at a false-positive rate of 0.267 and a true-positive rate of 0.824, its vertical distance from the chance line as Youden J=0.557, and the exact 2×2 confusion matrix. Because participant-level FAZ scores and threshold coordinates were unavailable, no continuous empirical ROC curve was reconstructed. Because the threshold was selected and evaluated in the same small sample, performance is apparent rather than externally validated.

An exploratory bootstrap statistical decomposition positioned vessel density between FAZ diameter and BCVA. The total coefficient was partitioned into a direct component and an indirect component, with a 5,000-resample bootstrap interval. Given simultaneous measurement and the absence of temporal ordering, this is reported as an associational decomposition, not causal mediation. The indirect proportion was calculated as 0.312/0.487=64.1%.

For the diabetes-duration subgroup analysis, participants were divided into ≥10 years and <10 years, 16 per group. Correlation p values and 95% confidence intervals were calculated within each subgroup. The difference between independent correlations was tested with Fisher z transformation. The contrast was treated as exploratory because of small groups, loss of information from dichotomization, and absence of multiplicity adjustment. All tests were two-sided with α=0.05, and the summary-level calculations were performed in R 4.5.3.

3. Results

Participant characteristics

Thirty-two participants contributed 32 analysis eyes. Mean age was 57.8±8.4 years, and 17 participants (53.1%) were men. Median diabetes duration was 9 years (interquartile range 5–14), and mean HbA1c was 8.2±1.6%. Hypertension was present in 18 participants (56.3%) and dyslipidemia in 12 (37.5%). Mean intraocular pressure was 14.8±2.9 mmHg. Eight eyes had mild NPDR, nine had moderate NPDR, eight had severe NPDR, and seven had PDR. The complete participant profile is detailed in Table 1.

Table 1. Demographic and clinical characteristics (n=32).

Characteristic	Value
Age, years	57.8±8.4
Male sex	17 (53.1)
Diabetes duration, years	9 (5–14)
HbA1c, %	8.2±1.6
Hypertension	18 (56.3)
Dyslipidemia	12 (37.5)
Intraocular pressure, mmHg	14.8±2.9
Mild NPDR	8 (25.0)
Moderate NPDR	9 (28.1)
Severe NPDR	8 (25.0)
PDR	7 (21.9)

Values are mean±standard deviation, median (interquartile range), or n (%). HbA1c, glycated hemoglobin; NPDR, nonproliferative diabetic retinopathy; PDR, proliferative diabetic retinopathy.

Ocular measures across DR severity

Ocular measures changed progressively across the severity categories. The overall and severity-stratified values are detailed in Table 2. For the full cohort, mean SCP FAZ diameter was 617.6±171.6 µm, mean DCP FAZ diameter was 567.1±158.5 µm, mean BCVA was 0.539±0.341 LogMAR, mean superficial vessel density was 38.0±14.4%, and mean CST was 323.4±61.8 µm.

Mean SCP FAZ diameter rose from 458.3±82.1 µm in mild NPDR to 535.8±106.4 µm in moderate NPDR, 698.2±118.9 µm in severe NPDR, and 812.5±125.8 µm in PDR. The omnibus comparison was $F(3,28)=16.35$, $p<0.001$, with $\eta^2=0.637$. DCP FAZ diameter showed a similar ordered pattern. Mean BCVA worsened from 0.18±0.12 to 0.96±0.18 LogMAR across the same categories ($F(3,28)=29.56$; $p<0.001$; $\eta^2=0.760$). Superficial vessel density decreased from 52.3±6.8% to 18.7±5.3% ($F(3,28)=33.29$; $p<0.001$; $\eta^2=0.781$). CST increased across categories, with a smaller omnibus effect ($F(3,28)=3.27$; $p=0.036$; $\eta^2=0.260$). These are unadjusted, cross-sectional comparisons and do not demonstrate within-person disease progression.

Table 2. Ocular measures by diabetic retinopathy severity.

Measure	Overall (n=32)	Mild NPDR (n=8)	Moderate NPDR (n=9)	Severe NPDR (n=8)	PDR (n=7)	F; p; η^2
BCVA, LogMAR	0.539±0.341	0.18±0.12	0.38±0.18	0.71±0.21	0.96±0.18	29.56; <0.001; 0.760
FAZ diameter, SCP, µm	617.6±171.6	458.3±82.1	535.8±106.4	698.2±118.9	812.5±125.8	16.35; <0.001; 0.637
FAZ diameter, DCP, µm	567.1±158.5	412.1±68.5	498.2±92.1	635.7±104.2	754.3±118.6	—
Vessel density, SCP, %	38.0±14.4	52.3±6.8	45.6±8.2	32.1±7.4	18.7±5.3	33.29; <0.001; 0.781
Central subfield thickness, µm	323.4±61.8	287.1±31.2	304.6±45.3	341.2±62.8	368.9±78.4	3.27; 0.036; 0.260

Values are mean±standard deviation. Overall values were reconstructed from the displayed group counts, means, and standard deviations. Omnibus ANOVA statistics use the same severity-group summaries; a DCP omnibus statistic was not evaluated. BCVA, best-corrected visual acuity; DCP, deep capillary plexus; FAZ, foveal avascular zone; LogMAR, logarithm of the minimum angle of resolution; NPDR, nonproliferative diabetic retinopathy; PDR, proliferative diabetic retinopathy; SCP, superficial capillary plexus.

Bivariate associations with BCVA

SCP FAZ diameter was positively correlated with LogMAR BCVA (r=0.582, 95% CI 0.293–0.774; p<0.001), indicating that a larger FAZ was associated with poorer visual acuity. DCP FAZ diameter was also positively correlated with BCVA (r=0.534, 95% CI 0.228–0.744; p=0.002). Superficial vessel density was inversely associated with BCVA (r=−0.521, 95% CI −0.736 to −0.211; p=0.002), so lower perfusion density accompanied worse acuity. All seven bivariate estimates are detailed numerically in Table 3 and visualized with their 95% CIs in Figure 1.

Diabetes duration showed a smaller positive correlation with BCVA (r=0.378, 95% CI 0.034–0.642; p=0.033). The intervals for age (r=0.312), HbA1c (r=0.289), and CST (r=0.184) included zero. The intervals in Figure 1 are wide, consistent with the modest sample, and the secondary associations were not adjusted for multiple testing.

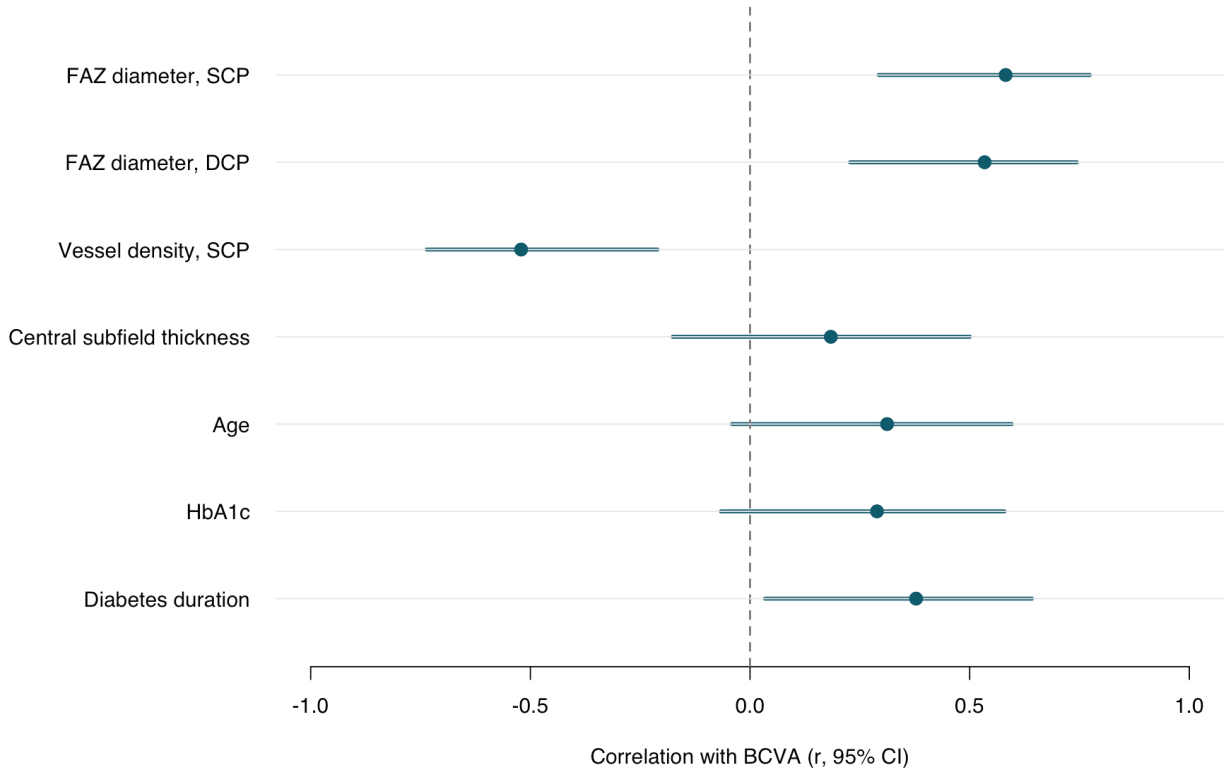

Figure 1. Bivariate associations with LogMAR best-corrected visual acuity. Points are Pearson correlation coefficients; horizontal lines are 95% confidence intervals calculated by Fisher z transformation (n=32). Positive coefficients indicate association with worse acuity.

Exploratory adjusted model

In the five-predictor regression, SCP FAZ diameter had the largest standardized coefficient (β=0.485, bootstrap 95% CI 0.298–0.672; p<0.001). Age had a smaller positive coefficient (β=0.228, 95% CI 0.012–0.444; p=0.042). The intervals for vessel density, HbA1c, and diabetes duration included zero. Adjusted R^2 was 0.412. Variance inflation factors ranged from 1.14 to 1.31, which does not suggest problematic linear collinearity among the included variables. However, low collinearity does not address overfitting, residual misspecification, or influential observations; the model remains exploratory because n=32 is small for five predictors and complete diagnostics were unavailable. The bivariate and adjusted coefficients are detailed together in Table 3 without implying that standardized coefficients establish independent causation.

Table 3. Bivariate and exploratory adjusted associations with LogMAR BCVA.

Parameter	r or β	95% CI	p	VIF
Panel A. Bivariate correlations with BCVA (n=32)				
FAZ diameter, SCP	0.582	0.293 to 0.774	<0.001	—
FAZ diameter, DCP	0.534	0.228 to 0.744	0.002	—
Vessel density, SCP	−0.521	−0.736 to −0.211	0.002	—
Central subfield thickness	0.184	−0.176 to 0.501	0.313	—
Age	0.312	−0.041 to 0.596	0.082	—
HbA1c	0.289	−0.066 to 0.579	0.109	—
Diabetes duration	0.378	0.034 to 0.642	0.033	—
Panel B. Exploratory multivariable model for BCVA				
FAZ diameter, SCP	0.485	0.298 to 0.672	<0.001	1.24
Age	0.228	0.012 to 0.444	0.042	1.18
Vessel density, SCP	−0.186	−0.389 to 0.017	0.078	1.31
HbA1c	0.142	−0.074 to 0.358	0.196	1.22
Diabetes duration	0.108	−0.089 to 0.305	0.278	1.14

Positive values indicate association with worse BCVA. Correlation intervals and p values were calculated from r and n=32. Regression values are standardized coefficients with 1,000-resample bootstrap intervals; adjusted R^2=0.412. BCVA, best-corrected visual acuity; CI, confidence interval; DCP, deep capillary plexus; FAZ, foveal avascular zone; SCP, superficial capillary plexus; VIF, variance inflation factor.

Exploratory ROC, statistical decomposition, and subgroup analyses

The apparent AUC for SCP FAZ diameter to discriminate BCVA >0.40 LogMAR was 0.847 (95% CI 0.723–0.971). At the data-derived 580-µm cutoff, 14 of 17 participants with impairment were classified positive and 11 of 15 participants without impairment were classified negative. This corresponds to sensitivity 82.4%, specificity 73.3%, positive predictive value 77.8%, and negative predictive value 78.6%. Youden J was 0.557 (0.824+0.733−1). The complete confusion-count-derived performance measures are detailed in Table 4, while the ROC operating point and exact confusion matrix are shown in Figure 2. These values describe the same sample used to choose the cutoff and should not be interpreted as validated clinical performance.

The exploratory FAZ–vessel-density–BCVA decomposition produced a total coefficient of 0.487, a direct coefficient of 0.175, and an indirect coefficient of 0.312. The bootstrap interval for the indirect component excluded zero (95% CI 0.148–0.512), whereas the direct component interval included zero. The indirect component represented 64.1% of the total coefficient by arithmetic division. This partition is consistent with shared variation among FAZ, vessel density, and BCVA; it does not establish a temporal or biological pathway.

For diabetes duration ≥10 years, the FAZ–BCVA correlation was r=0.724 (95% CI 0.356–0.898; p=0.002). For duration <10 years, it was r=0.412 (95% CI −0.105 to 0.754; p=0.113). Although the point estimate was larger in the longer-duration group, the between-group Fisher z test was not significant (z=1.22; p=0.223). The data therefore do not support a claim that diabetes duration modified the association. The ROC analysis, associational decomposition, and subgroup results are consolidated sequentially as Panels A–C in Table 4.

Table 4. Exploratory ROC, statistical decomposition, and subgroup analyses.

Analysis/metric	Estimate	95% CI	p or count	Details
Panel A. ROC analysis for BCVA >0.40 LogMAR				
Area under the curve	0.847	0.723 to 0.971	—	Apparent performance
Cutoff	580 µm	—	—	Youden-selected
Sensitivity	82.4%	67.2% to 92.1%	14/17	True positives / impaired
Specificity	73.3%	56.8% to 85.2%	11/15	True negatives / non-impaired
Positive predictive value	77.8%	62.4% to 88.0%	14/18	—
Negative predictive value	78.6%	61.8% to 89.3%	11/14	—
Panel B. Exploratory associational decomposition				
Total coefficient	0.487	0.298 to 0.676	<0.001	—
Direct component	0.175	−0.048 to 0.398	0.125	—
Indirect component via vessel density	0.312	0.148 to 0.512	0.001	64.1% of total
Panel C. Diabetes-duration subgroup analysis				
≥10 years (n=16)	r=0.724	0.356 to 0.898	0.002	—
<10 years (n=16)	r=0.412	−0.105 to 0.754	0.113	—
Between-group difference	z=1.22	—	0.223	Fisher z test

The cutoff was selected and evaluated in the same sample and is not externally validated. The statistical decomposition is cross-sectional and non-causal. BCVA, best-corrected visual acuity; CI, confidence interval; LogMAR, logarithm of the minimum angle of resolution; ROC, receiver operating characteristic.

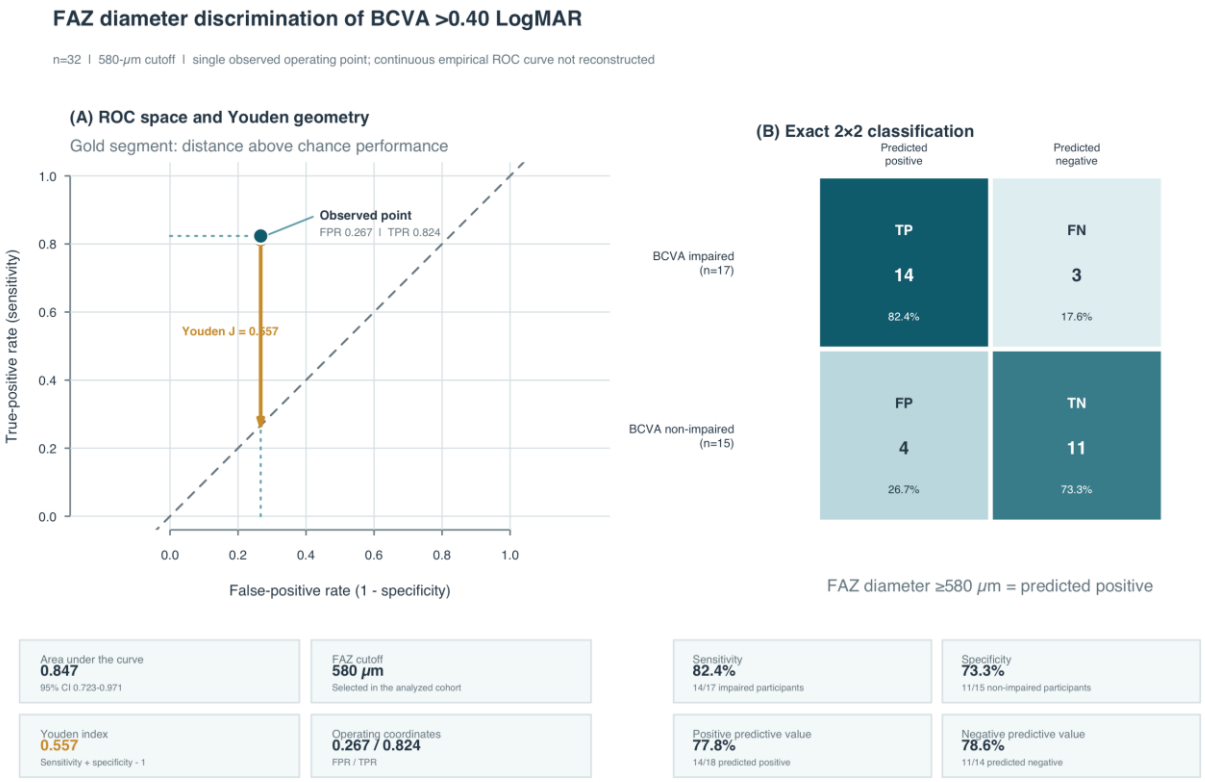

Figure 2. ROC/Youden operating-point summary for discrimination of BCVA >0.40 LogMAR by superficial FAZ diameter. (A) The observed 580-µm operating point is plotted at a false-positive rate of 0.267 and sensitivity of 0.824; the gold segment represents Youden J=0.557. The author-supplied AUC was 0.847 (95% CI 0.723–0.971). (B) Exact classification counts at the same cutoff. Only the supported operating point is displayed; a continuous empirical ROC curve was not reconstructed because participant-level predictor scores and threshold coordinates were unavailable.

4. Discussion

Principal findings

In this cross-sectional cohort of 32 Indonesian adults with DR, a larger SCP FAZ diameter was associated with worse LogMAR BCVA. The bivariate coefficient of 0.582 was moderately large, but its 95% confidence interval ranged from 0.293 to 0.774, illustrating meaningful uncertainty. FAZ diameter also increased markedly across clinical severity categories, while superficial vessel density decreased and BCVA worsened. The group comparison yielded $F(3,28)=16.35$ and $\eta^2=0.637$ for SCP FAZ diameter. These results provide a coherent anatomic pattern: eyes selected for poorer visual function and classified with more severe DR had a larger central capillary-free diameter and lower parafoveal perfusion density.

The multivariable model was directionally consistent with the primary analysis. SCP FAZ diameter had the largest standardized coefficient after inclusion of age, vessel density, HbA1c, and diabetes duration, and the model explained 41.2% of the observed variation in BCVA after adjustment for predictor count. This does not mean that FAZ diameter alone caused or predicted 41.2% of vision loss. Adjusted R^2 applies to the full fitted model, and standardized coefficients are sample-dependent. With only 32 observations and five predictors, the model has limited stability. The strongest defensible conclusion is therefore that the FAZ–BCVA association persisted in the exploratory adjustment, not that FAZ is an independently validated prognostic factor.

The subgroup analysis did not demonstrate effect modification. The correlation was numerically higher among participants with diabetes for at least 10 years, but the formal difference between subgroup correlations was $z=1.22$ with $p=0.223$. One subgroup's statistically significant correlation and the other subgroup's non-significant correlation do not themselves prove that the two correlations differ. The wide and overlapping confidence intervals further argue against an interaction claim. This distinction is especially important in small studies, where dichotomization reduces power and apparent differences may reflect sampling variation.

Relation to previous evidence

The present severity gradient is consistent with contemporary OCTA studies across advanced DR phenotypes. Quantitative imaging in proliferative DR has described marked angiographic abnormalities; widefield follow-up has visualized progressive neovascularization; multimodal work has linked diabetic lesions with retinal nonperfusion; and OCTA-detected intraretinal microvascular abnormalities have been associated with DR severity.[21–24] These findings support the direction of the changes detailed in Table 2, but they do not make the current cross-sectional groups a surrogate for longitudinal progression.

Recent investigations also show why FAZ diameter should not be interpreted in isolation. Perivenular capillary rarefaction, dilated deep capillaries, perfused versus nonperfused microaneurysms, and vascular branching or fragmentation each capture distinct components of diabetic microvascular injury.[25–28] In the present cohort, both SCP and DCP FAZ diameters correlated with BCVA, while superficial vessel density showed an inverse association, as detailed in Table 3 and Figure 1. The convergence of these directions supports a structure–function relationship, whereas the differing metrics caution against declaring average FAZ diameter the universally optimal biomarker.

Longitudinal and cross-platform research further emphasizes protocol dependence. Widefield OCTA has been used to infer capillary nonperfusion progression, progression detection improves when OCTA metrics are matched to DR severity, projection-resolved analyses reveal choriocapillaris flow deficits, and OCTA measures have been evaluated in the management context of diabetic macular edema.[29–32] Together with the acquisition-quality studies cited earlier, this evidence indicates that scan field, slab definition, artifact handling, and clinical phenotype materially influence the measured signal.

The direct comparison most relevant to the functional outcome comes from recent treated-eye data showing relationships between visual acuity and multiple OCTA parameters.[6] The present study extends that general observation to an Indonesian tertiary-care cohort and a diameter-based 3×3-mm swept-source FAZ protocol. Numerical comparison across studies would be inappropriate because populations, treatment status, devices, segmentation, and metrics differ. Accordingly, the observed $r=0.582$ should be interpreted as a cohort-specific estimate with its 95% CI, not as a portable calibration constant.

The central 3×3-mm field also leaves a known anatomical gap. Wider-field studies have shown that clinically significant nonperfusion and lesion distribution extend beyond the macula.[1,13,15,16] An eye with substantial peripheral nonperfusion may therefore have a different central OCTA profile from another eye with the same clinical grade. This unmeasured heterogeneity may contribute to residual variation in BCVA and reinforces the need to integrate OCTA with structural OCT, clinical examination, and systemic or ocular covariates.

Interpretation of the severity gradient

The ordered increase in FAZ diameter from mild NPDR through PDR is one of the most internally coherent results. Capillary closure, nonperfusion, and remodeling plausibly enlarge the central avascular region as DR becomes more advanced. Simultaneous loss of superficial vessel density strengthens this interpretation. Nevertheless, the analysis compares different participants at one visit. It does not show that the FAZ of an individual eye expanded by the observed

group difference over time. Cohort composition, prior therapy, edema, cataract, and other covariates may differ between severity groups and contribute to the gradient.

The effect sizes for vessel density and BCVA were large. Large group separation in a small, selected tertiary cohort can arise when categories are clinically distinct and when the worse eye is chosen. It can also be accentuated by sparse groups, extreme observations, or restricted variation within categories. Complete residual information was unavailable for independent assessment of normality and equal variance. The omnibus p values should therefore be read together with the group means, standard deviations, and sample sizes rather than as stand-alone evidence.

CST differed less strongly across groups than FAZ, vessel density, or BCVA. This may indicate that central thickness and ischemic morphology capture partly different aspects of DR. Thickness can increase with edema, remain near normal when ischemia is present without edema, or decrease in chronic atrophic change. The weak bivariate CST–BCVA correlation in this sample does not establish that edema was clinically unimportant; it may reflect heterogeneous mechanisms or limited power. Dedicated edema grading, intraretinal-fluid quantification, and photoreceptor-layer assessment would be needed to clarify this relationship.

Exploratory discrimination and clinical meaning

The apparent AUC of 0.847 suggests that SCP FAZ diameter separated participants above and below the study's BCVA threshold reasonably well within this dataset. At 580 μm, sensitivity was 82.4%, specificity was 73.3%, and Youden J was 0.557. Figure 2 emphasizes that J is the vertical separation of the selected operating point from the chance line; it is not a validation statistic. Importantly, sensitivity is the proportion of impaired participants who tested positive; it is not the probability that a person with FAZ above the cutoff has impairment. That latter quantity is closer to the positive predictive value, which was 77.8% in this sample and depends on the prevalence of impairment. Predictive values will change in screening, general ophthalmology, and tertiary retina populations.

The cutoff was chosen by maximizing the Youden index and assessed on the same 32 participants. This creates optimism because random sample features influence both selection and evaluation. The AUC confidence interval is wide, and the threshold may be unstable with only 17 impaired and 15 non-impaired participants. Device calibration, scan size, magnification, segmentation, and use of diameter rather than area further restrict transportability. The result is best considered a candidate threshold for external evaluation. It should not direct treatment, referral urgency, or follow-up intervals without independent validation and comparison with established clinical variables.

Recent OCTA classification studies have reported strong discrimination for DR severity categories, but that task differs from discriminating concurrent visual impairment among patients who already have DR.[11,12] The present endpoint also combines all causes of poorer BCVA remaining after eligibility exclusions. A future diagnostic study should prespecify the threshold, use masked evaluation, enroll a representative target population, report calibration, and compare incremental value over DR grade, CST, lens status, and other routine measurements.

Associational decomposition and biological interpretation

The statistical decomposition assigned 0.312 of a total 0.487 coefficient to an indirect component involving vessel density, approximately 64.1%. This arithmetic is compatible with the observation that larger FAZ and lower vessel density occur together and both relate to BCVA. It does not establish that FAZ enlargement causes loss of vessel density, which then causes visual impairment. FAZ diameter and parafoveal vessel density are simultaneous features of the same vascular image, and BCVA was measured at the same visit. Reverse direction, shared causes, measurement coupling, and unmeasured confounding remain possible.

Biologically, capillary dropout can reduce oxygen and metabolite delivery to neural retina, while chronic hyperglycemia promotes endothelial dysfunction, pericyte loss, inflammation, and barrier failure. These processes can influence both central nonperfusion and visual function. Yet the magnitude of a cross-sectional indirect coefficient should not be translated into a percentage of biological mechanism. Longitudinal data with repeated OCTA, structural OCT, and BCVA measurements would be needed to establish temporal sequence. A causal mediation analysis would additionally require well-defined exposure timing, mediator timing, confounder control for each path, and assumptions that cannot be verified here.

Integrated interpretation of central perfusion and vision

The results are most informative when the displays are interpreted together rather than as separate hypothesis tests. Table 2 shows three parallel severity gradients: mean SCP FAZ diameter increased, superficial vessel density decreased, and LogMAR BCVA worsened from mild NPDR through PDR. Table 3 then shows that the same directions were present across participants: larger SCP and DCP FAZ diameters accompanied worse BCVA, whereas higher superficial vessel density accompanied better BCVA. Figure 1 makes the relative magnitudes and uncertainty visible on a common correlation scale. This concordance supports internal coherence, but it does not remove the

effects of eye selection, severity-group composition, or unmeasured confounding.

The magnitude of the primary correlation also requires clinical context. An r of 0.582 indicates a moderately large linear association in this sample, not a fixed increase in LogMAR for a specified increase in micrometers. A correlation is dimensionless and symmetric; it does not identify which variable precedes the other. The standardized regression coefficient likewise does not provide a patient-level conversion from FAZ diameter to expected acuity. Such a conversion would require an unstandardized coefficient, a valid linear functional form, and external calibration. Therefore, the current results support association and prioritization for further study, but not individualized forecasting.

The severity-group means should not be interpreted as normative cut points. The transition from 458.3±82.1 µm in mild NPDR to 812.5±125.8 µm in PDR was observed in selected tertiary-care eyes measured with one device and a specific diameter definition. Standard deviations within groups show that individual distributions overlap even when group averages are well separated. Clinical classification should continue to use the full retinal examination rather than replacing visible lesions with a single central OCTA measure.

Table 4 addresses a different question from Tables 2 and 3. The ROC analysis dichotomizes continuous BCVA and estimates apparent discrimination, while the subgroup analysis asks whether two duration-defined correlations differ. Figure 2 makes the classification trade-off and exact error counts visible without implying a participant-level ROC reconstruction. Neither analysis strengthens the causal interpretation of the primary association. The non-significant subgroup contrast is particularly important because the larger point estimate in the ≥10-year group could otherwise be overread. Taken together, these analyses define the boundaries of the finding: the FAZ–BCVA association is present, but threshold performance and heterogeneity remain uncertain.

Measurement reproducibility and transportability

OCTA metrics are jointly determined by anatomy and the measurement pipeline. Scan centering, signal strength, motion, segmentation boundaries, projection removal, thresholding, magnification, and manual correction can change a calculated FAZ boundary or vessel-density value. Multicenter and ultrawide-field quality studies have documented these sources of variation, while automated and capillary-specific approaches still require validation against an appropriate reference standard.[7–10] Consequently, a statistically precise result from one workflow can remain clinically nontransportable if acquisition and processing are not sufficiently comparable.

The present protocol used the average of maximum horizontal and vertical FAZ chord lengths. This choice is easy to communicate but differs geometrically from area, perimeter, circularity, or irregularity. Two FAZ shapes can share a similar average diameter while differing materially in contour and area. Conversely, small boundary changes may affect area and perimeter differently from diameter. Comparative studies should calculate several prespecified metrics from the same segmentation so that incremental information, repeatability, and redundancy can be quantified rather than assumed.

Device and field-size effects are equally important. The central 3×3-mm acquisition offers dense sampling of the macula, but it excludes most peripheral nonperfusion. Wider-field studies demonstrate that severity-related information can reside outside the central field, and recent work suggests that metric performance depends on matching the angiographic feature to the severity range under study.[1,13,15,16,30] A central FAZ measure may therefore be most useful as one component of a broader imaging profile, not as a complete ischemia index.

Reproducibility should be established before external threshold validation. Repeated acquisitions are needed to separate scan-to-scan variation from grader variation; repeated segmentation by multiple masked observers is needed to estimate inter-grader agreement. The statistical model for the intraclass correlation coefficient, confidence interval, and unit of analysis should be prespecified. Bland–Altman limits of agreement can then show whether measurement error is small enough for the intended use. These steps are especially important near the 580-µm candidate cutoff, where modest measurement variation could change classification.

Transportability also requires evaluation across clinical spectra. A threshold derived in a tertiary-care sample selected by the worse eye may behave differently in screening populations, early diabetes, or clinics with different distributions of cataract, edema, treatment exposure, and systemic control. External validation should preserve the original threshold and endpoint, report calibration and discrimination with uncertainty, and document image exclusions. Only after such validation should investigators test whether adding FAZ diameter to DR grade, BCVA, structural OCT, and systemic variables improves decisions or patient outcomes.

Strengths

The study has several strengths. It provides data from an Indonesian tertiary-care population, a setting less represented in the OCTA structure–function literature. A single swept-source platform and a standardized 3×3-mm acquisition were used. One eye per participant avoided statistical dependence between fellow eyes. DR severity was graded independently by two ophthalmologists, with a reported κ of 0.87. Both SCP and DCP FAZ dimensions, superficial vessel density, CST, and ETDRS-style LogMAR acuity were included, allowing the primary association to be considered alongside complementary anatomic measures.

The use of one eye per participant, common severity-stratified denominators, and explicit integer ROC classifications makes the results readily interpretable. Effect estimates are presented with confidence intervals, and the subgroup conclusion is based on a formal between-correlation test rather than on comparison of separate significance labels. Consolidating complementary results into four tables and two figures emphasizes the primary FAZ–BCVA relationship while keeping secondary findings visible and clearly marked as exploratory.

Limitations

The principal analytical limitation is that complete participant-level diagnostics and reusable analysis code were not available for independent secondary evaluation. The reported summaries cannot identify the influence of individual observations, nonlinear patterns, unequal variance, or associations between image quality and the measured outcomes. The adjusted regression, AUC, and bootstrap decomposition should therefore be interpreted as exploratory. Future sharing of de-identified data and executable code, when permitted by institutional governance and participant confidentiality, would strengthen reproducibility and enable alternative model specifications.

The study is small, single-center, and cross-sectional. The sample-size basis addressed the primary correlation but not the more demanding adjusted, ROC, decomposition, or subgroup analyses. Confidence intervals are therefore more informative than binary p-value labels. Cross-sectional timing prevents prognosis or causal inference. No longitudinal change or treatment response was observed. Statements that FAZ could guide anti-vascular endothelial growth factor therapy, laser, surgery, or monitoring intensity are not supported by these data and have been removed.

Selection of the worse eye may inflate the prevalence of impairment and increase separation across disease categories. Excluding eyes with inadequate media clarity or OCTA image quality may produce a cohort in whom imaging is easier than in routine practice. Detailed screened and excluded counts, image-quality failures, and variable-specific missingness were not available. Diabetes type, lens status, axial length, refractive error, detailed blood-pressure measurements, smoking, and remote treatment history were not fully reported. Residual confounding is likely.

Measurement limitations include use of average FAZ chord diameter rather than area, perimeter, or circularity; a single observer; a small scan field; and incomplete reporting of segmentation boundaries, artifact correction frequency, and magnification adjustment. The reported repeatability coefficient lacked a confidence interval and metric-specific estimates. Values from this protocol should not be treated as interchangeable with other devices or algorithms. DCP measures may be particularly sensitive to projection and segmentation artifacts.

Finally, multiplicity and model development were not fully controlled. Seven bivariate correlations, several group comparisons, a five-predictor regression, ROC optimization, decomposition, and subgroup analysis were conducted in 32 participants. Secondary findings may include chance results. The corrected duration interaction was not significant, and the study does not establish effect modification. The ROC and decomposition results are hypothesis-generating. External validation in a larger, prospectively specified cohort is essential before clinical translation.

Implications and future research

For current practice, the data support viewing FAZ diameter as an adjunctive quantitative description of concurrent macular ischemic change. It may help clinicians communicate anatomic findings when interpreted together with visual acuity, structural OCT, DR grade, and the full clinical examination. The study does not support using 580 µm as a stand-alone treatment threshold. A clinically useful OCTA marker would need standardized acquisition, demonstrated repeatability, incremental value over routine predictors, and evidence that its use improves decisions or outcomes.

Future studies should be multicenter and prospectively define the analysis eye, scan protocol, segmentation boundaries, artifact correction, and primary FAZ metric. Participant flow, missingness, and image-quality exclusions should be reported explicitly. Area, diameter, circularity, perimeter, vessel density, and extrafoveal nonperfusion should be compared in the same eyes. Models should include clinically justified confounders and report unstandardized effects in meaningful units, such as change in LogMAR per 100-µm increase in FAZ. Internal validation should be followed by external validation on another center or device.

An additional priority is to define measurement agreement before testing clinical thresholds. Repeated scans should separate acquisition variability from grader variability, report prespecified intraclass-correlation models with confidence intervals, and quantify the frequency and direction of manual segmentation corrections. Axial-length or image-scale correction should be considered because magnification can influence geometric measurements. Studies that compare devices should use the same analysis eyes and distinguish systematic calibration differences from random repeatability error. For a candidate decision threshold, investigators should report the distribution of FAZ values around the cutoff, not only sensitivity and specificity, because small measurement error can reclassify eyes near the boundary. Decision-curve analysis may then determine whether adding an OCTA metric to BCVA, CST, and clinical DR grade provides net benefit at plausible clinical risk thresholds. These steps

would move the field from statistically detectable association toward a reproducible test whose incremental value can be assessed in the intended population.

Longitudinal repeated-measures designs are needed to determine whether FAZ change precedes or tracks change in BCVA. Such studies should measure diabetic macular edema, photoreceptor integrity, lens status, systemic control, and treatment exposure. If causal pathways are of interest, temporal ordering and confounder control must be planned before data collection. Rather than dichotomizing diabetes duration, investigators should ordinarily examine a continuous interaction with adequate power and a prespecified functional form. These steps would clarify whether OCTA adds clinically actionable information beyond established retinal assessment.

5. Conclusion

In this tertiary-care cross-sectional cohort, larger superficial and deep FAZ diameters and lower superficial vessel density were associated with worse LogMAR BCVA. SCP FAZ diameter increased across DR severity categories and remained the largest standardized correlate in the exploratory adjusted model. The subgroup comparison did not demonstrate that diabetes duration modified the association. The 580-µm cutoff and vessel-density decomposition are hypothesis-generating results from a small, selected sample, not validated clinical rules or causal mechanisms. Larger prospective studies with reproducible analysis, standardized OCTA measurements, and external validation are required before FAZ diameter can be used for prognosis or treatment decisions.

Declarations

Ethics approval and consent to participate

The Research Ethics Committee of the Faculty of Medicine, Universitas Sriwijaya approved the study (approval reference 1298/UN9.1.DL/KBK.6.1.3/2024). All participants provided written informed consent.

Consent for publication

The manuscript contains no identifiable individual participant information.

Competing interests

The authors declare no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Author contributions

RA conceived the study, recruited participants, performed clinical examinations, and drafted the manuscript. RP performed clinical examinations, independently graded DR severity, and contributed to manuscript revision. PP performed OCTA acquisition and analysis, including FAZ and vessel-density measurements. MAPOC conducted the statistical analyses, created the analytical figures, and assisted with participant recruitment and data management. All authors critically reviewed the manuscript and approved the final version.

Data availability

De-identified data may be requested from the corresponding author and are subject to institutional data-governance requirements and participant confidentiality protections.

Acknowledgments

None.

Generative AI disclosure

The authors declare that no generative artificial intelligence tools were used in the design, analysis, or writing of this manuscript.

6. References

1. Garg I, Uwakwe C, Le R, et al. Nonperfusion area and other vascular metrics by wider field swept-source optical coherence tomography angiography as biomarkers of diabetic retinopathy severity. Ophthalmol Sci. 2022;2(2):100144. doi:10.1016/j.xops.2022.100144.
2. Yuan M, Wang W, Kang S, et al. Peripapillary microvasculature predicts the incidence and development of diabetic retinopathy: an SS-OCTA study. Am J Ophthalmol. 2022;243:19–27. doi:10.1016/j.ajo.2022.07.001.
3. Marques IP, Ribeiro ML, Santos T, et al. Patterns of progression of nonproliferative diabetic retinopathy using non-invasive imaging. Transl Vis Sci Technol. 2024;13(5):22. doi:10.1167/tvst.13.5.22.
4. Mirshahi R, Riazi-Esfahani H, Khalili Pour E, et al. Differentiating features of OCT angiography in diabetic macular edema. Sci Rep. 2021;11(1):23398. doi:10.1038/s41598-021-02859-y.
5. Temel E, Demirel S, Batıoğlu F, et al. Association between optical coherence tomography angiography findings and inner retinal thickness in diabetic patients. Turk J Ophthalmol. 2022;52(5):331–337. doi:10.4274/tjo.galenos.2021.52722.
6. Wei-Zhang S, He K, Zhou W, et al. Relationship between visual acuity and OCT angiography parameters in diabetic retinopathy eyes after treatment. Eur J Ophthalmol. 2024;34(5):1521–1531. doi:10.1177/11206721241228010.
7. Lujan BJ, Calhoun CT, Glassman AR, et al. Optical coherence tomography angiography quality across three multicenter clinical studies of diabetic retinopathy. Transl Vis Sci Technol. 2021;10(3):2. doi:10.1167/tvst.10.3.2.
8. Wang XN, Li S, Cai X, et al. Imaging artifacts and quality evaluation with ultrawide-field swept-source OCTA in diabetic retinopathy. Curr Eye Res. 2024;49(4):410–416. doi:10.1080/02713683.2023.2296362.
9. Le Boité H, Bonnin S, Gallardo M, et al. Deep learning for retinal non-perfusion and foveal avascular zone analysis in wide-field OCTA in diabetic retinopathy. Sci Rep. 2025;15(1):30225. doi:10.1038/s41598-025-15712-3.
10. Abtahi M, Le D, Ebrahimi B, et al. Differential capillary and large vessel analysis improves OCTA classification of diabetic retinopathy. Invest Ophthalmol Vis Sci. 2024;65(10):20. doi:10.1167/iovs.65.10.20.
11. Zang P, Hormel TT, Wang X, et al. A diabetic retinopathy classification framework based on deep-learning analysis of OCT angiography. Transl Vis Sci Technol. 2022;11(7):10. doi:10.1167/tvst.11.7.10.
12. Ryu G, Lee K, Park D, et al. A deep learning algorithm for classifying diabetic retinopathy using optical coherence

tomography angiography. Transl Vis Sci Technol. 2022;11(2):39. doi:10.1167/tvst.11.2.39.
13. Kawai K, Murakami T, Mori Y, et al. Clinically significant nonperfusion areas on widefield OCT angiography in diabetic retinopathy. Ophthalmol Sci. 2023;3(1):100241. doi:10.1016/j.xops.2022.100241.
14. Tan B, Lim NA, Tan R, et al. Combining retinal and choroidal microvascular metrics improves discriminative power for diabetic retinopathy. Br J Ophthalmol. 2023;107(7):993–999. doi:10.1136/bjophthalmol-2021-319739.
15. Falavarjani KG, Mirshahi R, Riazi-Esfahani H, et al. Spatial distribution of diabetic capillary non-perfusion. Microcirculation. 2021;28(7):e12719. doi:10.1111/micc.12719.
16. Manafi N, Oncel D, Verma A, et al. Relationship between macular perfusion and lesion distribution in diabetic retinopathy. Eye (Lond). 2024;38(14):2724–2730. doi:10.1038/s41433-024-03105-1.
17. Ding X, Romano F, Garg I, et al. Expanded field OCT angiography biomarkers for predicting clinically significant outcomes in non-proliferative diabetic retinopathy. Am J Ophthalmol. 2025;270:216–226. doi:10.1016/j.ajo.2024.10.016.
18. Yoshida M, Murakami T, Ishihara K, et al. Clinical characterization of 3-year progression of diabetic macular ischemia on OCT angiography. Jpn J Ophthalmol. 2026;70(1):106–116. doi:10.1007/s10384-025-01257-5.
19. von Elm E, Altman DG, Egger M, et al. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. Ann Intern Med. 2007;147(8):573–577. doi:10.7326/0003-4819-147-8-200710160-00010.
20. Wilkinson CP, Ferris FL 3rd, Klein RE, et al. Proposed international clinical diabetic retinopathy and diabetic macular edema disease severity scales. Ophthalmology. 2003;110(9):1677–1682. doi:10.1016/S0161-6420(03)00475-5.
21. Kilani A, Werner JU, Lang GK, et al. Optical coherence tomography angiography findings in proliferative diabetic retinopathy. Ophthalmologica. 2021;244(3):258–264. doi:10.1159/000516525.
22. Shiraki A, Sakimoto S, Eguchi M, et al. Analysis of progressive neovascularization in diabetic retinopathy using widefield OCT angiography. Ophthalmol Retina. 2022;6(2):153–160. doi:10.1016/j.oret.2021.05.011.
23. Stino H, Huber KL, Niederleithner M, et al. Association of diabetic lesions and retinal nonperfusion using widefield multimodal imaging. Ophthalmol Retina. 2023;7(12):1042–1050. doi:10.1016/j.oret.2023.07.020.
24. Ding X, Xu J, Romano F, et al. Association of OCT angiography-detected intraretinal microvascular abnormalities with diabetic retinopathy severity. Ophthalmol Retina. 2026;10(2):185–193. doi:10.1016/j.oret.2025.08.011.
25. Crincoli E, Colantuono D, Miere A, et al. Perivenular capillary rarefaction in diabetic retinopathy: interdevice characterization and association to clinical staging. Ophthalmol Sci. 2023;3(2):100269. doi:10.1016/j.xops.2023.100269.
26. Terada N, Murakami T, Ishihara K, et al. Quantification of dilated deep capillaries in diabetic retinopathy on optical coherence tomography angiography. Sci Rep. 2023;13(1):17884. doi:10.1038/s41598-023-44848-3.
27. Gao M, Hormel TT, Guo Y, et al. Perfused and nonperfused microaneurysms identified and characterized by structural and angiographic OCT. Ophthalmol Retina. 2024;8(2):108–115. doi:10.1016/j.oret.2023.08.019.
28. Yu Y, Cui S, He Y, et al. Early diabetic retinopathy evaluation with OCTA: a study on vascular branching and fragmentation. Invest Ophthalmol Vis Sci. 2024;65(14):21. doi:10.1167/iovs.65.14.21.
29. Yoshida M, Murakami T, Kawai K, et al. Inference of capillary nonperfusion progression on widefield OCT angiography in diabetic retinopathy. Invest Ophthalmol Vis Sci. 2023;64(13):24. doi:10.1167/iovs.64.13.24.
30. Kakihara S, Zhuang K, AbdelSalam M, et al. The importance of matching optical coherence tomography angiography metrics to diabetic retinopathy severity for detecting progression. Invest Ophthalmol Vis Sci. 2025;66(11):49. doi:10.1167/iovs.66.11.49.
31. Wang J, Hormel T, Park DW, et al. Quantifying choriocapillaris flow deficits in diabetic retinopathy using projection-resolved OCT angiography. Invest Ophthalmol Vis Sci. 2025;66(12):13. doi:10.1167/iovs.66.12.13.
32. Basiony AI, Mohamed Gad Marey H, Ezzat Abdel Fattah AM, et al. Predictive value of optical coherence tomography angiography in management of diabetic macular edema. BMC Ophthalmol. 2024;24(1):429. doi:10.1186/s12886-024-03540-4.